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SYNAPSES: INSIGHTS ACROSS THE DISCIPLINES

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PRAGMATIC CHALLENGES IN ENGLISH–UZBEK TRANSLATION

Gaziyeva Dilshodai Nosir
Termez University of Economics and Service
Student of the Department of Philology and Language Teaching

ABSTRACT: Translation is a complex intercultural and communicative activity that involves much more than the transfer of words, grammatical structures, and lexical meanings from one language into another. In the process of translation, the translator is expected to reproduce the communicative intention of the source text, preserve its pragmatic meaning, and create an appropriate communicative effect on the target audience. This requirement becomes particularly challenging when translating between English and Uzbek, as the two languages represent different linguistic systems, cultural traditions, patterns of social interaction, and conventions of politeness. The present study investigates the major pragmatic challenges encountered in English–Uzbek and Uzbek–English translation. Particular attention is given to contextual meaning, speech acts, politeness strategies, forms of address, directness and indirectness, implicature, presupposition, modality, irony, sarcasm, culture-specific expressions, and pragmatic equivalence. The study employs descriptive, comparative, contextual, and functional approaches to analyze the ways in which pragmatic meaning is constructed and transferred between English and Uzbek. The findings demonstrate that literal translation does not always guarantee communicative adequacy because the same linguistic structure may perform different pragmatic functions in different cultural contexts. The research also emphasizes the importance of contextual adaptation, pragmatic compensation, explicitation, functional substitution, and cultural adaptation as effective translation strategies. It is argued that successful English–Uzbek translation requires an integrated combination of linguistic competence, pragmatic awareness, intercultural competence, and professional translation skills. The study concludes that pragmatic equivalence should be regarded as one of the fundamental criteria for assessing translation quality, particularly in contexts where cultural and communicative differences significantly influence the interpretation of meaning.

KEYWORDS: pragmatics, translation, English, Uzbek, pragmatic equivalence, speech acts, context, politeness, implicature, presupposition, intercultural communication, translation strategies.

Translation has played a fundamental role in the development of communication between different linguistic and cultural communities throughout human history. In the

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modern era, the importance of translation has increased considerably as a result of globalization, international education, scientific cooperation, diplomacy, tourism, business, migration, and technological development. People from different linguistic backgrounds communicate with one another more frequently than ever before, creating an increasing demand for accurate, natural, and culturally appropriate translation.

However, translation cannot be understood as a simple process of replacing words in one language with corresponding words in another. Languages are embedded in cultural systems, social relationships, historical experiences, and patterns of communication. As a result, the meaning of an utterance is often determined not only by its lexical and grammatical structure but also by the context in which it occurs, the relationship between the participants, the speaker's intentions, shared knowledge, and culturally established conventions.

Pragmatics is the branch of linguistics that investigates meaning in relation to language use and context. It examines how speakers communicate intended meanings and how listeners interpret meanings that may not always be explicitly expressed. In translation, pragmatic meaning is particularly important because a translator must transfer not only what the source text says but also what it means in a particular communicative situation.

The distinction between semantic and pragmatic meaning is essential in this respect. Semantic meaning is generally associated with the conventional meaning of linguistic expressions, whereas pragmatic meaning emerges from the interaction between linguistic form and contextual factors. For example, the sentence "Can you pass me the salt?" is grammatically structured as a question about ability. Pragmatically, however, it normally functions as a request. A translator who focuses exclusively on the literal meaning may fail to reproduce the communicative function of the utterance.

The problem becomes even more complex in intercultural translation. English and Uzbek belong to different linguistic and cultural traditions and demonstrate differences in patterns of politeness, forms of address, social hierarchy, directness, indirectness, and the expression of interpersonal relationships. These differences may create pragmatic gaps that cannot be overcome through literal translation alone. Uzbek communication may use forms such as professional titles and kinship-based honorifics. Expressions such as "aka," "opa," and "ustoz" can carry pragmatic meanings that extend beyond their basic lexical meanings.

For example, "ustoz" may literally refer to a teacher or mentor, but in certain contexts it can also function as a respectful form of address. A translator should therefore determine whether the term is being used descriptively or as a form of interpersonal recognition.

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RESULTS

The research identifies several major findings. First, pragmatic equivalence is an essential component of translation quality. Second, linguistic equivalence does not necessarily guarantee pragmatic equivalence. Third, contextual interpretation is necessary for understanding indirect and implicit meanings. Fourth, English and Uzbek differ significantly in their use of politeness strategies and forms of address. Fifth, culture-specific expressions require careful adaptation and contextual interpretation. Sixth, pragmatic challenges are particularly significant in literary, audiovisual, academic, diplomatic, and business translation. Seventh, translators can overcome pragmatic difficulties through contextual adaptation, explicitation, implicitation, pragmatic compensation, functional substitution, and cultural adaptation. Finally, effective translation requires the integration of linguistic, pragmatic, cultural, and communicative competence.

IMPLICATIONS FOR TRANSLATOR EDUCATION

The findings of the study have important implications for translator education. Traditional translation training often focuses heavily on vocabulary, grammar, terminology, and translation techniques. While these components are essential, they are insufficient for developing complete translation competence. Students should be trained to analyze communicative situations and identify pragmatic meanings. Translation exercises should include:

- indirect requests;
- culturally specific greetings;
- forms of address;
- ironic expressions;
- implied meanings;
- presuppositions;
- politeness strategies;
- modality;
- culturally bound expressions.

Students should also compare multiple possible translations and discuss how each version changes the pragmatic effect. For example, learners may analyze the following sentence: "Would you mind waiting outside?" They should identify:

1. the literal meaning;
2. the speech act;
3. the level of politeness;
4. the social relationship;
5. possible target-language equivalents;

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6. differences in pragmatic effect.

Such activities can help students develop practical pragmatic awareness.

CONCLUSION

Pragmatic challenges constitute a fundamental issue in English–Uzbek translation. Translation is not merely a linguistic process involving the replacement of words and grammatical structures. It is an intercultural communicative activity that requires the transfer of meaning, intention, social relationships, cultural references, and communicative effects. The study demonstrates that pragmatic differences between English and Uzbek may create considerable difficulties in translating speech acts, politeness strategies, forms of address, indirect expressions, implicatures, presuppositions, modal meanings, irony, sarcasm, and culture-specific elements. Context plays a central role in the interpretation of pragmatic meaning. The same linguistic expression may perform different communicative functions depending on the situation, relationship between participants, and cultural background.

The research also demonstrates that pragmatic equivalence should be considered an essential criterion of translation quality. A translation may be grammatically accurate and lexically appropriate while still failing to communicate the intended pragmatic meaning. To achieve pragmatic equivalence, translators may employ contextual adaptation, explicitation, implicitation, pragmatic compensation, functional substitution, and cultural adaptation. The choice of strategy depends on the type of text, communicative purpose, target audience, and cultural context.

The findings emphasize that successful English–Uzbek translation requires a combination of linguistic competence, pragmatic awareness, intercultural competence, and professional translation skills. Translators must understand not only what the source text says but also why it is said, to whom it is addressed, under what circumstances it is communicated, and what effect it is intended to produce.

In the context of globalization and increasing intercultural communication, pragmatic competence has become increasingly important. Future research may investigate pragmatic challenges in specialized areas such as literary translation, audiovisual translation, diplomatic discourse, academic writing, legal translation, and digital communication.

Ultimately, the successful translation of pragmatic meaning contributes significantly to effective intercultural communication. A translator who can recognize and reproduce both explicit and implicit meanings is better equipped to create translations that are accurate, natural, culturally appropriate, and communicatively effective.

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MACHINE TRANSLATION AND HUMAN TRANSLATION: A COMPARATIVE ANALYSIS

Gaziyeva Dilshodai Nosir
Termez University of Economics and Service
Student of the Department of Philology and Language Teaching

ABSTRACT: The rapid development of artificial intelligence and language technologies has significantly transformed the field of translation. Machine translation has evolved from rule-based systems to sophisticated neural machine translation models capable of processing large amounts of multilingual data and generating increasingly fluent translations. At the same time, human translation continues to play a crucial role in professional, literary, academic, legal, diplomatic, and culturally sensitive communication. This article presents a comparative analysis of machine translation and human translation, focusing on their strengths, limitations, functional differences, and potential for cooperation. The study examines major aspects of translation quality, including accuracy, fluency, contextual interpretation, cultural adaptation, pragmatic meaning, terminology, creativity, consistency, speed, and ethical responsibility. A qualitative comparative methodology is employed to analyze the capabilities of machine and human translators in different communicative contexts. The findings indicate that machine translation provides significant advantages in terms of speed, accessibility, consistency, and the ability to process large volumes of text. However, human translators demonstrate greater flexibility in interpreting ambiguous meanings, understanding cultural references, preserving stylistic nuances, and reproducing pragmatic and emotional effects. The study also emphasizes the growing importance of human post-editing and human–machine collaboration. Rather than replacing human translators entirely, modern translation technologies are increasingly becoming supportive tools that can improve productivity and efficiency. The article concludes that the future of translation is likely to involve a collaborative model in which artificial intelligence and human expertise complement one another. Such a model can combine the computational efficiency of machine translation with the cultural, pragmatic, creative, and ethical competence of human translators.

KEYWORDS: machine translation, human translation, artificial intelligence, neural machine translation, translation quality, post-editing, translation technology, cultural adaptation, pragmatics, artificial intelligence, human–machine collaboration.

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Translation has historically served as one of the most important means of communication between different linguistic and cultural communities. It has contributed significantly to the exchange of knowledge, literature, science, education, technology, and cultural values. In the contemporary world, the demand for translation has increased dramatically as a result of globalization, international trade, academic cooperation, digital communication, migration, tourism, and the rapid expansion of online content.

At the same time, technological developments have fundamentally changed the way translation is performed. The emergence of machine translation has introduced new possibilities for processing multilingual information quickly and efficiently. Modern translation systems can translate large amounts of text within seconds, making multilingual communication more accessible to individuals and organizations around the world.

Machine translation refers to the use of computer systems and computational methods to translate texts from one natural language into another without the direct involvement of a human translator during the initial generation of the translation. The development of machine translation has passed through several major stages, including rule-based machine translation, statistical machine translation, and neural machine translation. The emergence of neural machine translation has significantly improved the fluency and naturalness of automated translations. Neural systems use artificial neural networks to identify patterns in large collections of multilingual data. Unlike traditional rule-based systems, neural models are capable of processing broader contextual relationships and generating translations that often appear more natural.

Despite these developments, human translation remains essential. Human translators possess cognitive, cultural, pragmatic, and creative abilities that are difficult to reproduce through automated systems. They can interpret ambiguous expressions, recognize irony and humor, understand cultural references, evaluate the communicative intentions of authors, and adapt texts to specific audiences. The comparison between machine and human translation is therefore not simply a question of determining which one is "better." Instead, it is necessary to examine the particular contexts in which each approach is most effective.

For example, machine translation may be highly useful for translating large amounts of technical documentation, preliminary information, online communication, and repetitive texts. Human translators, in contrast, may be more appropriate for literary works, legal documents, diplomatic communication, advertising, academic publications, and texts that require cultural adaptation. The central question of the present study is how machine translation and human translation differ in terms of

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quality, efficiency, cultural competence, contextual interpretation, and communicative effectiveness.

The objectives of the research are:

1. to examine the development and characteristics of machine translation;
2. to analyze the main characteristics of human translation;
3. to compare machine and human translation in terms of accuracy and fluency;
4. to investigate the role of context and cultural knowledge;
5. to examine the treatment of pragmatic meaning and ambiguity;
6. to analyze the advantages and limitations of artificial intelligence in translation;
7. to explore the role of human post-editing;
8. to examine the future relationship between human translators and translation technologies.

The significance of the research lies in the increasing role of artificial intelligence in professional and educational translation. Understanding the strengths and limitations of both machine and human translation is essential for developing effective translation practices in the digital era.

CONSISTENCY AND TERMINOLOGY

Machine translation offers significant advantages in terminology management. Large-scale translation projects often require consistent use of technical terms. A machine translation system can apply terminology databases and translation memories to maintain consistency across thousands of pages. Human translators may unintentionally use different terms for the same concept. However, human translators are better able to determine whether a term should be translated consistently or adapted according to context. Therefore, the strongest approach may involve combining automated terminology management with human review.

HUMAN CREATIVITY AND MACHINE TRANSLATION

Creativity is particularly important in literary translation, advertising, poetry, slogans, and creative writing.

Human translators can recreate:

- humor;
- wordplay;
- rhythm;
- emotional tone;
- literary style;
- cultural associations.

For example, a slogan may depend on a pun that cannot be translated literally. A human translator may create a new expression that differs from the original wording but

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produces a similar communicative effect. Machine translation systems are increasingly capable of generating creative language, but their ability to understand the deeper cultural and rhetorical purpose of creative texts remains limited. Therefore, human creativity continues to be highly valuable.

HUMAN POST-EDITING

Human post-editing has become an important part of modern translation workflows. Post-editing refers to the process of reviewing and correcting machine-generated translations. The post-editor may need to correct:

- grammatical errors;
- mistranslations;
- terminology;
- word order;
- cultural references;
- stylistic problems;
- pragmatic errors;
- inconsistencies.

There are generally two broad approaches to post-editing. Light post-editing focuses on correcting major errors and making the text understandable. Full post-editing aims to produce a polished translation suitable for professional publication. The increasing use of post-editing demonstrates that human and machine translation are not necessarily competing processes. They can operate together in an integrated workflow.

DISCUSSION

The comparative analysis demonstrates that machine translation and human translation possess distinct advantages and limitations. Machine translation has transformed the accessibility and speed of multilingual communication. It allows individuals and organizations to process enormous quantities of text rapidly and at relatively low cost. The development of neural machine translation has significantly improved the fluency of automated output. In many routine translation tasks, machine-generated texts may be sufficiently accurate for general communication. However, high fluency can sometimes conceal errors. A machine-generated sentence may appear natural while containing an incorrect interpretation of the original meaning.

Human translation remains essential when texts require complex interpretation, cultural adaptation, creativity, or high levels of responsibility. The analysis also suggests that the distinction between machine and human translation is becoming less rigid. Modern translation practice increasingly involves cooperation between human translators and technological systems. The translator of the future may not simply translate texts manually. Instead, the translator may manage technological tools,

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evaluate machine output, correct errors, and ensure cultural and pragmatic appropriateness.

CONCLUSION

Machine translation and human translation represent two complementary approaches to the complex task of multilingual communication. Machine translation has developed rapidly and now provides impressive levels of speed, accessibility, consistency, and fluency. It has become an essential tool for organizations and individuals who need to process large quantities of multilingual information. However, human translation continues to demonstrate significant advantages in areas that require contextual interpretation, cultural competence, pragmatic awareness, creativity, emotional sensitivity, and ethical responsibility.

The comparison presented in this study demonstrates that machine translation should not be viewed simply as a replacement for human translators. Instead, it should be understood as a powerful technological tool that can support and enhance human translation. Human translators remain essential for interpreting ambiguous expressions, understanding cultural references, preserving stylistic nuances, reproducing pragmatic meanings, and adapting texts to specific audiences. The most promising model for the future is therefore a collaborative approach in which artificial intelligence and human expertise complement each other. In such a model, machines can perform repetitive, large-scale, and time-consuming tasks, while human translators focus on complex interpretation, cultural adaptation, quality control, and creative decision-making.

The growing importance of artificial intelligence also requires a transformation in translator education. Future professionals must combine advanced linguistic competence with technological literacy, intercultural awareness, pragmatic competence, and critical thinking. Ultimately, the goal of translation is not merely to produce grammatically correct sentences in another language. The fundamental purpose is to communicate meaning effectively across linguistic and cultural boundaries. Machine translation can accelerate this process, but human intelligence, cultural understanding, and professional judgment remain essential for ensuring that communication is accurate, meaningful, natural, and appropriate.

The future of translation is therefore unlikely to be defined by a simple competition between humans and machines. Instead, it will increasingly be shaped by collaboration between human expertise and artificial intelligence. Such collaboration has the potential to make translation faster, more accessible, and more efficient while preserving the human qualities that are essential to meaningful intercultural communication.

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YASHIL IQTISODIYOTGA O‘TISH SHAROITIDA INVESTITSIYALAR VA BARQAROR IQTISODIY RIVOJLANISH: XALQARO TAJRIBA VA O‘ZBEKISTON UCHUN IMKONIYATLAR

Xolsaidova Dilnoza Chorshanbi qizi

Annotatsiya. Mazkur maqolada yashil iqtisodiyotga o‘tish jarayonida investitsiyalarning barqaror iqtisodiy rivojlanishga ta’sir mexanizmlari, xalqaro tajriba va O‘zbekiston uchun amaliy imkoniyatlar tahlil qilinadi. Tadqiqotda Xalqaro energetika agentligi (IEA), Jahon banki, IRENA, O‘zbekiston Respublikasi Energetika vazirligi hamda normativ-huquqiy hujjatlardagi ma’lumotlar asosida qiyosiy, trend va institutsional tahlil usullari qo‘llanildi. Natijalar global energetika investitsiyalari tarkibida toza energiyaga yo‘naltirilayotgan kapitalning qazilma yoqilg‘ilarga nisbatan qariyb ikki baravar yuqori ekanini, O‘zbekistonda esa 2025-yilda energetika sohasiga 11 mlrd AQSh dollari xorijiy investitsiya o‘zlashtirilgani va yashil elektr energiyasi ishlab chiqarish hajmi 16,8 mlrd kVt·soatga yetganini ko‘rsatadi. Tadqiqot O‘zbekiston uchun yashil moliya instrumentlarini kengaytirish, davlat-xususiy sheriklik mexanizmlarini chuqurlashtirish, elektr tarmoqlariga investitsiyalarni ko‘paytirish, energiya samaradorligi va iqlimga mos qishloq xo‘jaligini ustuvorlashtirish zarurligini asoslaydi. Yashil investitsiyalarning iqtisodiy samarasi faqat yangi generatsiya quvvatlari bilan emas, balki xususiy kapitalni safarbar etish, institutsional islohotlar, tarif signallari va risklarni taqsimlash sifati bilan belgilanadi.

Kalit so‘zlar: yashil iqtisodiyot, yashil investitsiya, barqaror rivojlanish, qayta tiklanuvchi energiya, iqlim moliyasi, davlat-xususiy sheriklik, energiya samaradorligi, O‘zbekiston.

Аннотация. В статье исследуются механизмы влияния инвестиций на устойчивое экономическое развитие в условиях перехода к зеленой экономике, международный опыт и практические возможности для Узбекистана. На основе данных Международного энергетического агентства (IEA), Всемирного банка, IRENA, Министерства энергетики Республики Узбекистан и нормативно-правовых документов применены сравнительный, трендовый и институциональный методы анализа. Результаты показывают, что глобальные инвестиции в чистую энергетику почти в два раза превышают капиталовложения в ископаемое топливо, а в энергетическом секторе Узбекистана в 2025 году было освоено 11 млрд долларов США иностранных инвестиций, при этом

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производство «зеленой» электроэнергии достигло 16,8 млрд кВт·ч. Обоснована необходимость расширения инструментов зеленого финансирования, развития государственно-частного партнерства, инвестиций в электрические сети, энергоэффективность и климатически устойчивое сельское хозяйство.

Ключевые слова: зеленая экономика, зеленые инвестиции, устойчивое развитие, возобновляемая энергетика, климатическое финансирование, государственно-частное партнерство, энергоэффективность, Узбекистан.

Abstract. This article examines how investment supports sustainable economic development during the transition to a green economy, drawing on international experience and identifying practical opportunities for Uzbekistan. Comparative, trend and institutional analyses are applied using data from the International Energy Agency (IEA), the World Bank, IRENA, the Ministry of Energy of Uzbekistan and national policy documents. The results show that global clean-energy investment is nearly twice the level of fossil-fuel investment. In Uzbekistan, foreign investment absorbed in the fuel and energy complex reached USD 11 billion in 2025, while green electricity generation reached 16.8 billion kWh. The study argues that Uzbekistan should expand green-finance instruments, deepen public-private partnership mechanisms, increase investment in power grids, and prioritize energy efficiency and climate-smart agriculture. The economic effect of green investment depends not only on additional generation capacity, but also on the ability to mobilize private capital, improve institutions, provide credible tariff signals and allocate risks efficiently.

Keywords: green economy, green investment, sustainable development, renewable energy, climate finance, public-private partnership, energy efficiency, Uzbekistan.

KIRISH

Jahon iqtisodiyotida yashil transformatsiya endi faqat ekologik siyosat yo‘nalishi emas, balki kapital qo‘yilmalarining tarkibi, sanoat raqobatbardoshligi, energiya xavfsizligi va uzoq muddatli iqtisodiy o‘sishni belgilovchi asosiy omillardan biriga aylandi. Elektrlashtirish, qayta tiklanuvchi energiya, energiya saqlash, tarmoq infratuzilmasi, past uglerodli transport va energiya samaradorligiga yo‘naltirilayotgan investitsiyalar bir vaqtning o‘zida ishlab chiqarish xarajatlarini pasaytirish, import qilinadigan yoqilg‘iga qaramlikni kamaytirish va yangi texnologik zanjirlarni shakllantirish imkonini beradi. Shu sababli yashil investitsiya tushunchasi ekologik loyihalarni moliyalashtirishdan kengroq bo‘lib, iqtisodiyotning kapital bazasini resurs tejamkor va past emissiyali texnologiyalar tomon qayta yo‘naltirish jarayonini anglatadi.

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IEA ma'lumotlariga ko'ra, 2026-yilda jahon energetika sektoriga yo'naltiriladigan investitsiyalar 3,4 trln AQSh dollariga yetishi, shundan 2,2 trln dollari toza energiya segmentlariga to'g'ri kelishi kutilmoqda (IEA, 2026). Bu nisbat kapital bozorlarida energiya xavfsizligi va dekarbonizatsiya vazifalari tobora bir-biriga yaqinlashayotganini ko'rsatadi. Shu bilan birga, rivojlanayotgan iqtisodiyotlarda kapital qiymatining yuqoriligi, valyuta tavakkali, tarmoq infratuzilmasining yetishmasligi va siyosiy-regulyator noaniqlik yashil loyihalarning bankability darajasini pasaytirishi mumkin. Demak, kapital miqdoridan tashqari uning narxi, muddati, tavakkalchiliklarni taqsimlash mexanizmi va institutsional muhit ham hal qiluvchi ahamiyatga ega.

O'zbekiston uchun yashil o'tish ayniqsa muhim. Mamlakat iqtisodiyotining energiya va resurs sig'imi yuqori, elektr energiyasiga talab tez o'smoqda, suv taqchilligi va iqlim xatarlari esa iqtisodiy o'sish sifatiga bevosita ta'sir ko'rsatadi. Jahon banki baholashiga ko'ra, yetarli moslashuv choralarisiz iqlim o'zgarishi ta'sirida O'zbekiston iqtisodiyoti 2050-yilda potensial ssenariyga nisbatan 10 foizgacha kichikroq bo'lishi mumkin; shu bilan birga, moslashuv investitsiyalarining foydasi xarajatlardan ikki-uch baravar yuqori bo'lishi mumkin (World Bank, 2023). Shuning uchun yashil investitsiyalar masalasi O'zbekiston uchun ekologik maqsad emas, balki makroiqtisodiy barqarorlik, energiya xavfsizligi va uzoq muddatli unumdorlik masalasidir.

Tadqiqotning maqsadi yashil iqtisodiyotga o'tish sharoitida investitsiyalar va barqaror iqtisodiy rivojlanish o'rtasidagi bog'liqlikni xalqaro tendensiyalar hamda O'zbekistonning amaldagi transformatsiya jarayonlari asosida baholash, shuningdek, mamlakat uchun eng yuqori iqtisodiy multiplikativ ta'sirga ega investitsiya yo'nalishlarini aniqlashdan iborat. Mazkur maqsad doirasida uchta savol qo'yildi: birinchidan, global yashil investitsiyalar tarkibi qanday o'zgarmoqda; ikkinchidan, O'zbekistonning energetika va iqlim siyosati ushbu global trendga qanchalik mos keladi; uchinchidan, mavjud kapital oqimlarini barqaror o'sishga aylantirish uchun qaysi institutsional mexanizmlar ustuvor bo'lishi kerak?

METODOLOGIYA VA ADABIYOTLAR TAHLILI

Tadqiqot desk-research formatida amalga oshirildi. Ma'lumotlar bazasini 2023–2026-yillarda e'lon qilingan IEA, Jahon banki va IRENA hisobotlari, O'zbekiston Respublikasi Energetika vazirligining 2025-yil yakunlari bo'yicha rasmiy axboroti, shuningdek, 2030-yilgacha yashil iqtisodiyotga o'tish dasturining maqsadli ko'rsatkichlari tashkil etdi. Statistik tahlilda mutlaq qiymatlar, o'sish sur'atlari va tarkibiy nisbatlar qo'llanildi. 2024-yil uchun ayrim ko'rsatkichlar 2025-yil rasmiy

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o‘sish sur’atlaridan teskari hisoblash orqali baholandi; bunday hisoblar jadval va diagrammalarda alohida qayd etildi.

Metodologik jihatdan tadqiqot to‘rtta usulga tayandi. Birinchisi, qiyosiy tahlil bo‘lib, global toza energiya va qazilma yoqilg‘i investitsiyalari nisbatini O‘zbekistonning investitsiya dinamikasi bilan solishtirishda qo‘llanildi. Ikkinchisi, trend tahlili bo‘lib, elektr ishlab chiqarish va yashil generatsiyadagi o‘zgarishlarni aniqlashda ishlatildi. Uchinchisi, institutsional tahlil bo‘lib, yashil taksonomiya, davlat-xususiy sheriklik, tarif islohoti, davlat moliya institutlari va xususiy kapitalni jalb qilish mexanizmlarining rolini baholadi. To‘rtinchisi, sababiy-mantiqiy model bo‘lib, investitsiya manbalaridan real sektor natijalarigacha bo‘lgan uzatish kanallarini konseptual tarzda ifodaladi.

Adabiyotlarda yashil investitsiyalar va iqtisodiy o‘sish o‘rtasidagi munosabat bir nechta kanallar orqali izohlanadi. Birinchi kanal — kapital yangilanishi: yangi energiya va ishlab chiqarish aktivlari eski, energiya sig‘imi yuqori uskunalarni almashtiradi. Ikkinchi kanal — innovatsiya va o‘rganish effekti: texnologiya miqyoslashgani sari birlik xarajatlari kamayadi. Uchinchi kanal — tashqi xarajatlarni qisqartirish: havo ifloslanishi, sog‘liq, yoqilg‘i importi va iqlim zararlarining kamayishi ijtimoiy-iqtisodiy foyda yaratadi. To‘rtinchi kanal — investitsiya krowding-in effekti: ishonchli regulyator muhit va davlatning risklarni qisman kamaytirishi xususiy kapitalni jalb qiladi.

IEA (2026) energetika investitsiyalarida elektr tizimlari, qayta tiklanuvchi energiya, saqlash va samaradorlik segmentlari ustuvorligini ko‘rsatadi. IRENA (2025) esa qayta tiklanuvchi energiya quvvatlarining tez kengayayotganini qayd etib, yangi energiya tizimlari uchun generatsiya bilan bir qatorda tarmoq, moslashuvchanlik va moliyaviy arxitektura muhimligini ta’kidlaydi. Jahon bankining O‘zbekiston bo‘yicha CCDR hisobotida energiya sektori mamlakat issiqxona gazlari emissiyasining qariyb 75 foiziga javob berishi, dekarbonizatsiya va eskirgan energetika infratuzilmasini almashtirish uchun 2060-yilgacha taxminan 340 mlrd AQSh dollari talab qilinishi qayd etilgan (World Bank, 2023). Bu raqamlar davlat byudjeti bunday transformatsiyani yakka o‘zi moliyalashtira olmasligini va xususiy kapitalning markaziy rolini ko‘rsatadi.

O‘zbekistonning 2030-yilgacha yashil iqtisodiyotga o‘tish dasturida 2010-yil darajasiga nisbatan YAIM birligiga issiqxona gazlari emissiyasini 35 foizga kamaytirish, qayta tiklanuvchi energiya quvvatlarini 15 GVtga yetkazish va elektr ishlab chiqarishdagi ulushini 30 foizdan oshirish kabi maqsadlar belgilangan. Ushbu maqsadlar investitsiya siyosatini energetika, sanoat, transport, suv va qishloq xo‘jaligidagi texnologik modernizatsiya bilan birlashtirishni talab qiladi. Bunda

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muammo faqat kapital yetishmasligi emas; loyiha tayyorlash sifati, bankable shartnomalar, valuta riskini boshqarish, xarid jarayonlarining shaffofligi va elektr tarmog‘ining yangi quvvatlarni qabul qilish imkoniyati ham cheklov bo‘lishi mumkin.

NATIJALAR

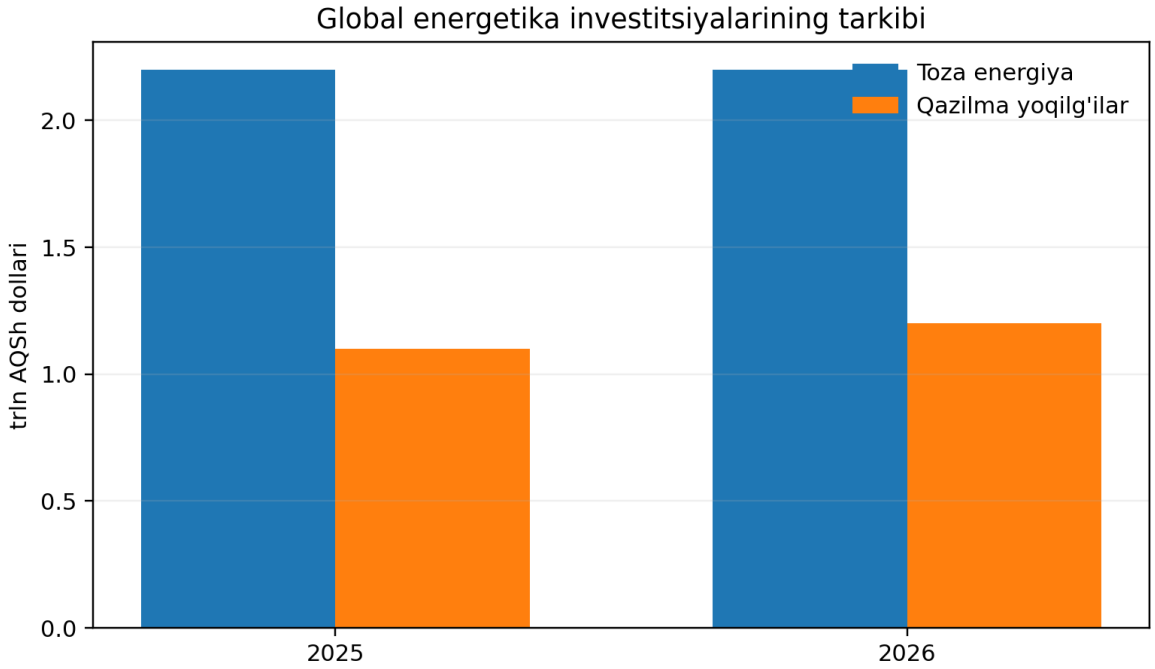
Global investitsiya tendensiyalari shuni ko‘rsatadiki, energetika kapitali tarkibi strukturaviy ravishda toza texnologiyalar foydasiga siljigan. IEA 2025-yil uchun global energetika investitsiyalarini 3,3 trln dollar, shundan 2,2 trln dollarini toza energiya va 1,1 trln dollarini qazilma yoqilg‘i segmentlariga baholagan. 2026-yilda jami hajm 3,4 trln dollarga o‘sishi, toza energiya investitsiyalari 2,2 trln dollar atrofida qolishi va qazilma yoqilg‘iga investitsiyalar taxminan 1,2 trln dollarni tashkil etishi kutilmoqda. Demak, yangi kapitalning asosiy qismi elektrlashtirish, qayta tiklanuvchi energiya, tarmoqlar, saqlash va samaradorlik infratuzilmasiga yo‘nalmoqda.

1-jadval. Global energetika investitsiyalarining asosiy ko‘rsatkichlari

Ko‘rsatkich	2025	2026	Tahliliy mazmun
Jami energetika investitsiyasi, trln $	3,3	3,4	Kapital oqimi yuqori darajada saqlanmoqda
Toza energiya, trln $	2,2	2,2	Energetika kapitalining asosiy qismi
Qazilma yoqilg‘ilar, trln $	1,1	≈1,2	Toza energiyadan sezilarli past
Toza/qazilma nisbati	2,0 marta	≈1,8 marta	Strukturaviy yashil siljish saqlanmoqda

Manba: IEA (2025, 2026) ma’lumotlari asosida muallif hisob-kitobi.

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1-diagramma. Global toza energiya va qazilma yoqilg‘i investitsiyalari, 2025–2026 Manba: IEA, World Energy Investment 2025 va World Energy Investment 2026 asosida tuzildi.

O‘zbekiston bo‘yicha 2025-yil ko‘rsatkichlari yashil transformatsiyaning investitsion bosqichi tezlashganini ko‘rsatadi. Energetika vazirligi ma’lumotiga ko‘ra, 2025-yilda yoqilg‘i-energetika kompleksida 11 mlrd AQSh dollari xorijiy investitsiya o‘zlashtirilgan bo‘lib, bu respublika bo‘yicha o‘zlashtirilgan xorijiy investitsiyalarning 25 foiziga teng. Shu yil elektr energiyasi ishlab chiqarish 86,7 mlrd kVt·soatni tashkil etib, 2024-yildagi 81,5 mlrd kVt·soatga nisbatan 6 foizga oshgan. Quyosh, shamol va gidro manbalardan ishlab chiqarilgan yashil energiya 16,8 mlrd kVt·soatga yetib, bir yil ichida 29 foizga ko‘paygan. Quyosh va shamol generatsiyasining o‘zi 10,5 mlrd kVt·soatni tashkil etib, 2,1 baravar o‘sgan.

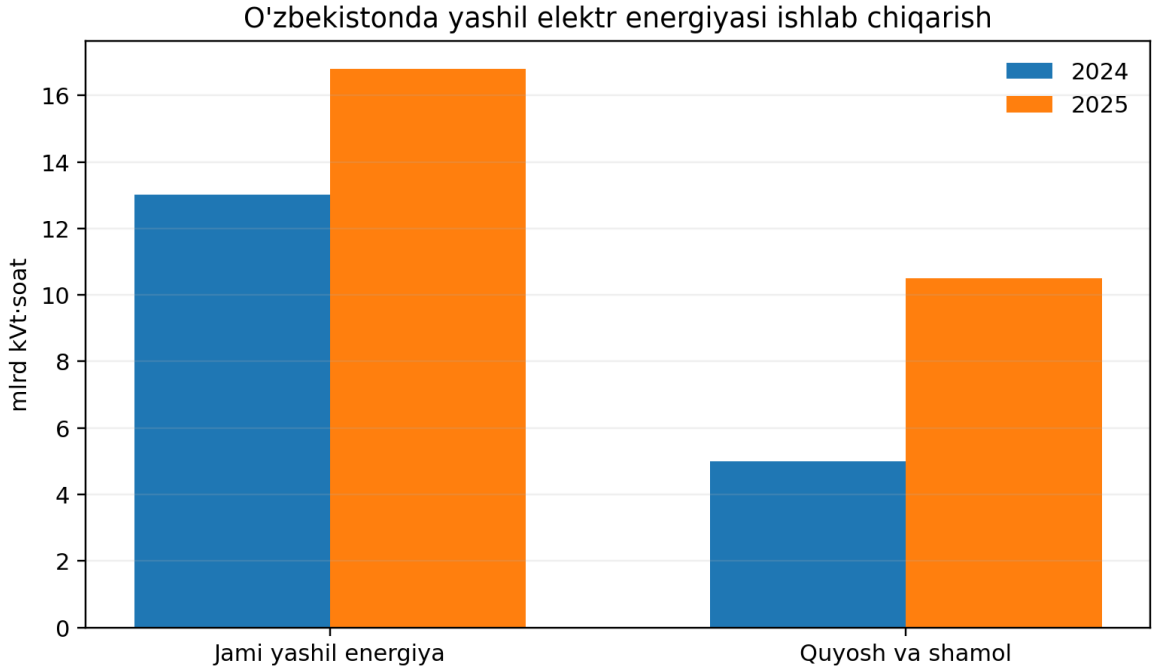
2-jadval. O‘zbekiston energetika sektorida yashil transformatsiyaning tanlangan ko‘rsatkichlari

Ko‘rsatkich	2024	2025	O‘zgarish
Elektr energiyasi ishlab chiqarish, mlrd kVt·soat	81,5	86,7	+6%
Yashil energiya, mlrd kVt·soat	≈13,0*	16,8	+29%
Quyosh va shamol, mlrd kVt·soat	≈5,0*	10,5	2,1 baravar

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Tejalgan tabiiy gaz, mlrd m³	—	3,2	2025-yil natijasi
Oldi olingan zararli tashlanmalar, mln tonna	—	4,7	2025-yil natijasi
Energetikada xorijiy investitsiya, mlrd $	—	11,0	Jami xorijiy investitsiyaning 25%i

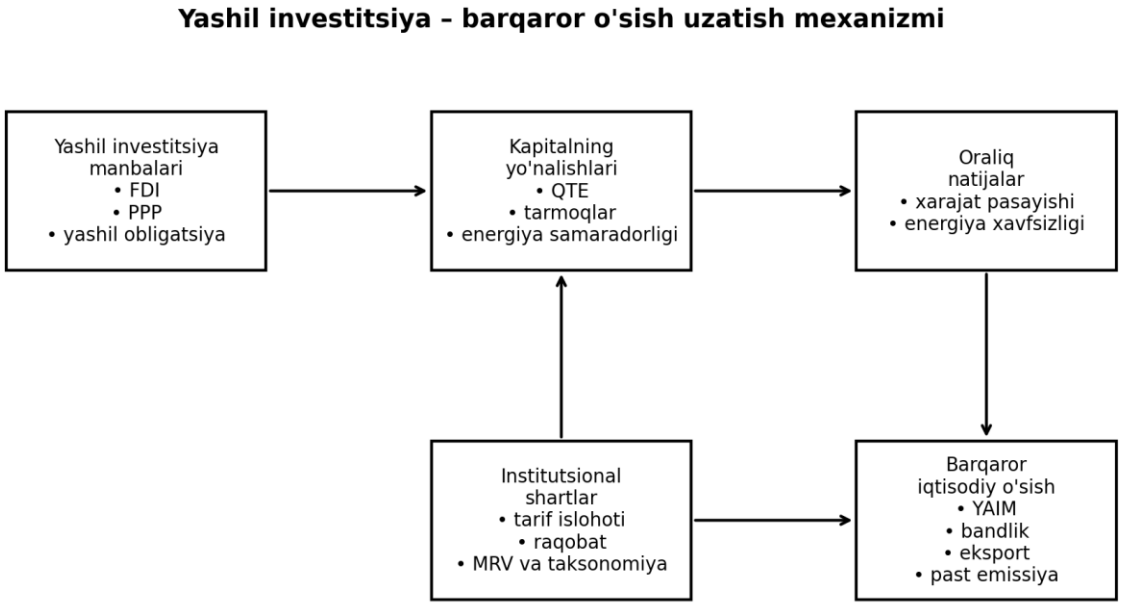
*Manba: O‘zbekiston Respublikasi Energetika vazirligi (2026). *2024-yil qiymatlari 2025-yil o‘sish ko‘rsatkichidan teskari hisoblangan.*



2-diagramma. O‘zbekistonda yashil elektr energiyasi ishlab chiqarish dinamikasi Manba: Energetika vazirligining 2025-yil yakunlari bo‘yicha rasmiy ma’lumotlari asosida hisoblandi.

Yashil elektr ishlab chiqarishning o‘sishi tabiiy gazni ichki bozorda tejash, eksport yoki yuqori qo‘shilgan qiymatli qayta ishlash uchun resurs bo‘shatish imkonini beradi. 2025-yildagi yashil generatsiya natijasida 3,2 mlrd kub metr tabiiy gaz tejalgan va 4,7 mln tonna zararli tashlanmalarning oldi olingan. Bu ikki tomonlama dividend — energiya resurslarini tejash va ekologik tashqi xarajatlarni kamaytirish — yashil investitsiyaning makroiqtisodiy qiymatini faqat elektr sotish daromadi bilan o‘lchash yetarli emasligini ko‘rsatadi.

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1-rasm. Yashil investitsiyalarning barqaror iqtisodiy o‘sishga ta’sir mexanizmi
Manba: Muallif ishlanmasi.

Konseptual modeldan ko‘rinadiki, investitsiyaning o‘zi yakuniy natija emas. Kapital birinchi bosqichda qayta tiklanuvchi generatsiya, elektr tarmoqlari, saqlash, energiya samaradorligi va iqlimga mos texnologiyalarga yo‘naltiriladi. Ikkinchi bosqichda ishlab chiqarish xarajatlari, resurs sarfi va import qaramligi kamayadi; energiya ta’minotining ishonchliligi oshadi. Uchinchi bosqichda esa unumdorlik, xususiy investitsiya, bandlik va eksport salohiyati orqali barqaror iqtisodiy o‘sish shakllanadi. Ushbu uzatish zanjiri tarif islohoti, raqobat, shaffof xaridlar, yashil taksonomiya va ishonchli monitoring tizimi mavjud bo‘lgandagina to‘liq ishlaydi.

3-jadval. O‘zbekiston uchun ustuvor yashil investitsiya yo‘nalishlari va kutiladigan iqtisodiy ta’sir

Yo‘nalish	Asosiy muammo	Investitsiya instrumenti	Kutiladigan ta’sir
Elektr tarmoqlari va saqlash	Yangi QTE quvvatlarini qabul qilish cheklovi	PPP, DFI krediti, infratuzilma obligatsiyasi	Yo‘qotishlar kamayishi, tizim barqarorligi
Quyosh va shamol	Kapital qiymati va valuta riski	Auksion, PPA, kafolat, blended finance	Arzonroq generatsiya, gaz tejalishi

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Energiya samaradorligi	Kichik va tarqoq loyihalar	ESCO, yashil kredit liniyalari	Sanoat tannarxi va energiya sig‘imi pasayishi
Suv va qishloq xo‘jaligi	Suv taqchilligi va hosildorlik riski	Natijaga bog‘liq moliya, imtiyozli kredit	Suv tejalishi, hosildorlik va rezilyentlik
Yashil transport	Infratuzilma va boshlang‘ich xarajat	PPP, municipal yashil obligatsiya	Havo sifati, transport xarajati va emissiya yaxshilanishi
Mahalliy yashil sanoat	Texnologiya va ta’minot zanjiri zaifligi	FDI, maxsus investitsiya rejimi	Mahalliylashtirish, eksport, yuqori malakali ish o‘rinlari

Manba: IEA, World Bank va O‘zbekiston siyosiy maqsadlari asosida muallif taklifi.

MUHOKAMA

Olingan natijalar yashil investitsiyalarni iqtisodiy rivojlanishning mustaqil drayveri sifatida ko‘rish uchun asos beradi, biroq bitta muhim shart mavjud: investitsiya oqimlari iqtisodiy samaradorlik mezonlari asosida boshqarilishi kerak. Katta hajmdagi kapital har doim ham yuqori ijtimoiy qaytimni anglatmaydi. Masalan, yangi quyosh yoki shamol stansiyasi tarmoqning o‘tkazuvchanligi yetarli bo‘lmasa, ishlab chiqarishni cheklash (curtailment) xavfi paydo bo‘ladi. Xuddi shuningdek, PPA shartnomalarida valuta va talab risklari asosan davlat zimmasida qolsa, xususiy investitsiya bugungi byudjet xarajatini kamaytirgani bilan kelajakdagi fiskal majburiyatni oshirishi mumkin. Shuning uchun loyiha tanlashda kVt quvvat narxi bilan bir qatorda tizim xarajati, tarmoq investitsiyasi, saqlash ehtiyoji va shartli majburiyatlar ham hisobga olinishi kerak.

Xalqaro tajribada uchta institutsional xulosa ajralib turadi. Birinchidan, raqobatli auksionlar va uzoq muddatli shaffof PPA shartnomalari kapital narxini tushiradi. Ikkinchidan, davlat yoki xalqaro moliya institutlarining kafolatlari faqat xususiy investor boshqara olmaydigan siyosiy, offtaker yoki valuta risklariga nisbatan qo‘llanganda samarali bo‘ladi; aks holda riskning haddan tashqari ijtimoiylashtirilishi yuzaga keladi. Uchinchidan, yashil moliya instrumentlari aniq taksonomiya va MRV — monitoring, reporting and verification tizimisiz “greenwashing” xavfini tug‘diradi. O‘zbekiston uchun ham yashil obligatsiya va yashil kredit bozorini kengaytirish bilan

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parallel ravishda loyihalarning iqlim ta'sirini o'lchash standartlari mustahkamlanishi kerak.

Jahon bankining O'zbekiston bo'yicha tahlili davlat moliya institutlari va davlat korxonalarining yashil transformatsiyadagi ulkan rolini ko'rsatadi. Davlatga tegishli moliya institutlari iqtisodiyot kreditlarining katta qismini ta'minlaydi, davlat investitsiya fondlari esa sezilarli kapital oqimlarini boshqaradi. Bu holat ikki xil imkoniyat yaratadi. Bir tomondan, yashil kredit standartlari va investitsiya mandatlarini tez joriy etish mumkin. Ikkinchi tomondan, noto'g'ri rag'batlar yoki siyosiy tanlovlar kapitalning past samarali loyihalarga yo'nalish xavfini oshiradi. Shu bois yashil maqsadlar moliyaviy intizomni almashtirmasligi, balki loyiha qaytimi, emissiya kamayishi va iqtisodiy qo'shimcha qiymat bilan birgalikda baholanishi kerak.

O'zbekiston uchun eng katta imkoniyatlardan biri — energetika transformatsiyasini sanoat siyosati bilan bog'lash. Quyosh panellari, kabel, transformator, energiya saqlash tizimlari, raqamli hisoblagichlar, energiya auditi va muhandislik xizmatlari atrofida mahalliy qiymat zanjirlarini rivojlantirish mumkin. Ammo to'liq mahalliylashtirishni ma'muriy talab bilan tezlashtirish noto'g'ri bo'lishi mumkin: kichik bozor va texnologiyaning tez yangilanishi mahsulot tannarxini oshiradi. Eng maqbul model — xalqaro investorlarni mahalliy yetkazib beruvchilarni rivojlantirish, kadr tayyorlash, texnologik hamkorlik va eksportga yo'naltirilgan klasterlar orqali bog'lashdir.

Yashil investitsiyalar hududiy siyosat nuqtai nazaridan ham differensial yondashuvni talab qiladi. Quyosh resursi yuqori hududlarda generatsiya va saqlash, shamol salohiyati yuqori zonalarda yirik shamol parklari, suv tanqis hududlarda tomchilatib sug'orish va suv hisobini raqamlashtirish, yirik shaharlarda elektr jamoat transporti va bino energiya samaradorligi ustuvor bo'lishi mumkin. Bir xil instrumentni barcha hududlarga qo'llash kapital samaradorligini pasaytiradi. Shuning uchun yashil investitsiya dasturlari hududiy resurs bazasi, tarmoq cheklovlari va ish o'rinlari salohiyatiga asoslangan portfel yondashuvi orqali shakllantirilishi zarur.

Tahlilning cheklovi shundaki, O'zbekiston bo'yicha ochiq ma'lumotlar investitsiyalarning texnologiyalar, hududlar va moliyalashtirish manbalari kesimidagi to'liq vaqt qatorini bermaydi. Shu sababli maqolada asosan rasmiy agregat ko'rsatkichlar va xalqaro institutlarning baholari qo'llanildi. Kelgusidagi tadqiqotlarda yashil investitsiyalar, hududiy YAIM, bandlik, energiya sig'imi va eksport o'rtasidagi bog'liqlikni panel ma'lumotlar yoki input-output modeli asosida ekonometrik baholash maqsadga muvofiq.

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XULOSA

Tadqiqot natijalari yashil iqtisodiyotga o‘tish sharoitida investitsiya siyosati barqaror iqtisodiy rivojlanishning markaziy instrumentiga aylanayotganini ko‘rsatadi. Global miqyosda toza energiyaga yo‘naltirilayotgan kapital qazilma yoqilg‘i investitsiyalaridan qariyb ikki baravar yuqori. Bu jarayon ekologik maqsadlarning o‘zi bilan emas, balki quyosh va shamol texnologiyalarining raqobatbardoshligi, elektrlashtirish, energiya xavfsizligi va sanoat siyosati bilan ham izohlanadi.

O‘zbekistonda 2025-yilda energetika sohasiga 11 mlrd dollar xorijiy investitsiya o‘zlashtirilishi, yashil energiya ishlab chiqarishning 16,8 mlrd kVt·soatga yetishi va quyosh-shamol generatsiyasining keskin o‘sishi yashil transformatsiyaning miqyosi ortganini ko‘rsatadi. Biroq keyingi bosqichda yangi generatsiya quvvatlarini qurishning o‘zi yetarli bo‘lmaydi. Elektr tarmoqlari, saqlash tizimlari, energiya samaradorligi, suv infratuzilmasi va institutsional sifat kapitalning iqtisodiy qaytimini belgilaydi.

O‘zbekiston uchun eng muhim amaliy yo‘nalishlar quyidagilar: yashil loyihalar uchun yagona va xalqaro standartlarga mos taksonomiya hamda MRV tizimini mustahkamlash; qayta tiklanuvchi energiya auksionlari va PPPlarda risklarni optimal taqsimlash; elektr tarmoqlari va energiya saqlashga generatsiya bilan parallel investitsiya qilish; tijorat banklari, davlat moliya institutlari va kapital bozorida yashil moliya instrumentlarini ko‘paytirish; sanoat va binolarda ESCO kabi energiya samaradorligi mexanizmlarini joriy etish; suv va qishloq xo‘jaligida iqlimga moslashuv investitsiyalarini kengaytirish; hamda yashil sanoat zanjirlarida mahalliy kadrlar va yetkazib beruvchilar salohiyatini oshirish.

Asosiy xulosa shuki, yashil investitsiyaning hajmi emas, balki uning sifati va iqtisodiy tizimga integratsiyasi hal qiluvchi omildir. Kapital oqimi raqobatli bozor, oqilona tariflar, shaffof xaridlar, barqaror regulyator muhit va xususiy sektor uchun aniq risk-qaytim nisbatiga tayansa, yashil o‘tish O‘zbekiston uchun xarajat emas, balki energiya xavfsizligi, unumdorlik, yangi ish o‘rinlari va uzoq muddatli iqtisodiy o‘sish manbaiga aylanishi mumkin.

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THE BLOOD–BRAIN BARRIER: THE BODY'S ULTIMATE SECURITY SYSTEM

Ayush Sharma , Medical Student, Samarkand State Medical University, Uzbekistan

Rishima Katoch, Medical Student, Samarkand State Medical University, Uzbekistan

Khushi , Medical Student, Samarkand State Medical University, Uzbekistan

Somashekar Deepak , Medical Student, Samarkand State Medical University, Uzbekistan

Abstract

Few biological structures guard their territory as jealously as the blood–brain barrier. A continuous wall of specialized endothelial cells, welded together by tight junctions and backed up by pericytes and astrocytes, it admits the nutrients the brain needs and repels nearly everything else — including, inconveniently, the majority of medicines we would like to deliver to it. This narrative review examines the blood–brain barrier as a living security system: its historical discovery, its cellular and molecular architecture, and the tightly regulated transport routes that allow a small, privileged set of molecules to cross. We then consider what happens when that security fails — in stroke, multiple sclerosis, Alzheimer's disease, brain tumours, and central nervous system infection — and the therapeutic consequences of both a barrier that is too tight and one that is too leaky. Finally, we survey the creative armamentarium scientists have assembled to breach or bypass the barrier on demand: prodrugs, molecular Trojan horses, nanoparticles, focused ultrasound, and intranasal delivery. Understanding the barrier not as a static wall but as a dynamic, responsive interface is now central to treating the brain's most stubborn diseases.

Keywords: Blood–brain barrier; tight junctions; neurovascular unit; drug delivery; transcytosis; neurodegeneration.

1. Introduction

The brain is a demanding tenant. Though it accounts for only about two percent of body weight, it commands a fifth of the body's circulating blood supply and consumes energy at a rate surpassed by no other organ. Yet the bloodstream that feeds it is kept at a careful arm's length. Between the capillary lumen and the neural tissue lies the blood–brain barrier (BBB) — arguably the most selective border in the human

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body, and the reason why treating diseases of the brain has frustrated medicine for over a century [1].

The barrier's reputation is well earned. It is estimated that only about two percent of small-molecule drugs and essentially no large biologics cross into brain tissue in any meaningful amount, a statistic that has made the BBB the single greatest bottleneck in central nervous system (CNS) drug development [4]. For a pharmaceutical company, a brilliant molecule that cures a brain tumour in a dish but cannot reach its target is not a drug; it is an expensive failure. But the barrier is not a defect of design. It is the price of admission for the extraordinary chemical stability the brain requires — an organ with almost no regenerative capacity can ill afford exposure to toxins, pathogens, or wild fluctuations in the chemistry of the blood [5]. This review traces the story of the barrier from its accidental discovery to the frontiers of modern delivery science, treating it throughout as what it truly is: the body's ultimate security system, complete with walls, guards, checkpoints, and — as every good security system has — ways to be defeated.

2. A Brief History of an Accidental Discovery

The barrier revealed itself through a series of beautiful accidents. In 1885, the German microbiologist Paul Ehrlich injected aniline dyes into the bloodstream of animals and noticed, to his puzzlement, that virtually every organ took up the stain except the brain. Ehrlich concluded, incorrectly, that the brain simply had no affinity for the dye. It was his student, Edwin Goldmann, who performed the decisive experiment in 1913: injecting the dye directly into the cerebrospinal fluid, he found that the brain stained vividly while the rest of the body remained untouched. The two compartments were sealed off from one another — something stood between blood and brain [6].

What that something was took another half-century to see. The breakthrough came in 1967, when Thomas Reese and Morris Karnovsky injected the electron-dense enzyme horseradish peroxidase into the circulation of mice and examined brain capillaries under the electron microscope. The tracer halted abruptly at the endothelial cell junctions, never squeezing between them as it did in capillaries elsewhere. The barrier, they showed, was not a membrane but the endothelial wall itself — sealed by structures that would come to be called tight junctions [3]. Six decades later, the neurovascular unit they glimpsed remains the conceptual foundation of the field.

3. Anatomy of the Fortress: Cellular and Molecular Architecture

The BBB is best understood not as a single wall but as a coordinated community of cells — the neurovascular unit — each contributing to security (Figures 1 and 2). The

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first and decisive layer is the endothelium. Brain capillary endothelial cells differ profoundly from endothelial cells elsewhere in the body: they are connected by continuous belts of tight junction proteins, they display very few pinocytotic vesicles, and they rest on a dense basement membrane. The tight junctions — molecular stitches formed by claudins, occludin, and the zonula occludens scaffolding proteins — fuse adjacent cells into a membrane with electrical resistance many times higher than that of ordinary capillaries, effectively sealing the paracellular route [10].

Wrapped around the endothelium are pericytes, contractile cells embedded in the basement membrane that regulate capillary diameter, stabilize the vessel wall, and help maintain the junctional seal; loss of pericytes alone is enough to make vessels leaky, in both mice and human disease [5]. Covering the outer surface like insulating mesh are astrocyte end-feet — bulbous processes of star-shaped glial cells that transfer chemical signals from neurons to the vasculature and help induce the barrier phenotype during development [1]. Microglia, the brain's resident immune cells, patrol the perivascular space as a mobile security detail, and a thin layer of perivascular macrophages completes the ensemble [11].

Adding another layer of selectivity, the endothelial surface bristles with transport machinery: nutrient carriers such as the glucose transporter GLUT1 and the large neutral amino acid transporter LAT1 admit essential supplies, while ATP-dependent efflux pumps — most famously P-glycoprotein and the breast cancer resistance protein — actively expel lipophilic molecules that slip into the endothelial membrane, ejecting them back into the blood [13]. The barrier is therefore not merely a wall; it is a wall with active bouncers.

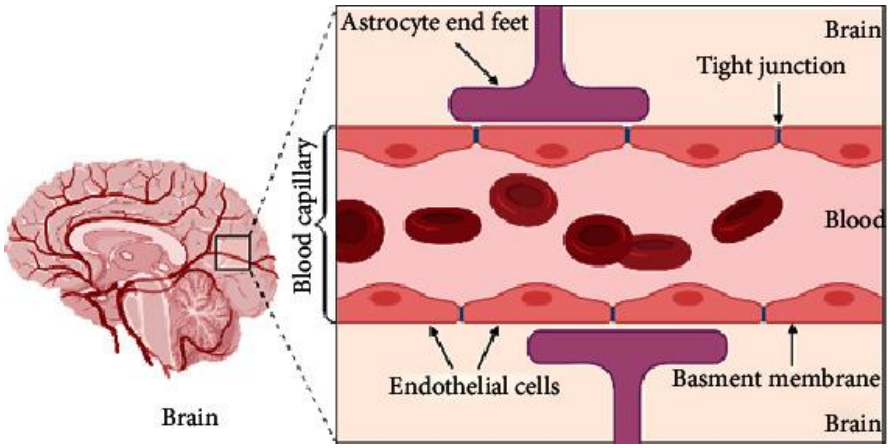


Figure 1. Structural overview of the blood–brain barrier: brain capillary endothelium joined by tight junctions, ensheathed by the basement membrane and astrocyte end-feet. (Image source: PMC, open access)

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4. How Guests Are Admitted: Transport Across the Barrier

That a barrier so tight still nourishes the brain is a feat of logistics. Small lipophilic molecules such as oxygen and carbon dioxide diffuse straight across the endothelial membrane, as do some anaesthetics — the reason a surgical patient drifts off within seconds of an intravenous injection. Water moves through dedicated water channels (aquaporin-1), and charged ions cross via selective channels. Glucose, amino acids, nucleosides, and vitamins, however, are too polar to diffuse and too precious to exclude; they ride specific carrier proteins in a process of facilitated transport [1].

Larger cargo — iron-bearing transferrin, insulin, low-density lipoprotein — enters by receptor-mediated transcytosis: the molecule binds its receptor on the blood-facing membrane, the complex is internalized into a vesicle, and the vesicle shuttles across the cell to be released on the brain side. This Trojan-horse mechanism has become one of the most exploited routes in delivery science (Figure 3) [7]. Finally, the efflux transporters work in reverse, constituting a fourth, subtractive category of transport that keeps the brain clear of lipophilic intruders. Table 1 summarizes the principal routes and their therapeutic significance.

Transport route	**Mechanism**	**Representative cargoes**	**Therapeutic significance**
Passive (lipid) diffusion	Dissolution through the endothelial lipid bilayer; driven by concentration gradient	O2, CO2, ethanol, general anaesthetics, some small lipophilic drugs	Basis of CNS penetration for many sedatives and antidepressants; efflux pumps can expel even these
Carrier-mediated transport	Facilitated diffusion or active transport via membrane proteins	Glucose (GLUT1), neutral amino acids (LAT1), nucleosides, monocarboxylates	LAT1 exploited to ferry L-DOPA and investigational drugs; essential for brain nutrition

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Receptor-mediated transcytosis	Ligand–receptor binding, vesicular shuttling across the endothelium	Transferrin, insulin, low-density lipoprotein, IgG (FcRn)	Molecular Trojan horses: antibodies and enzymes fused to BBB-targeting ligands
Adsorptive transcytosis	Cationic molecules bind the luminal membrane and trigger vesicular uptake	Certain peptides and cationized proteins	Route for some cell-penetrating peptides; less specific than RMT
Efflux transport	ATP-dependent pumps expel substrates back into blood	P-glycoprotein, BCRP, MRPs	Major cause of multidrug resistance in brain tumours and of poor CNS penetration

Table 1. Principal routes of molecular traffic across the blood–brain barrier.

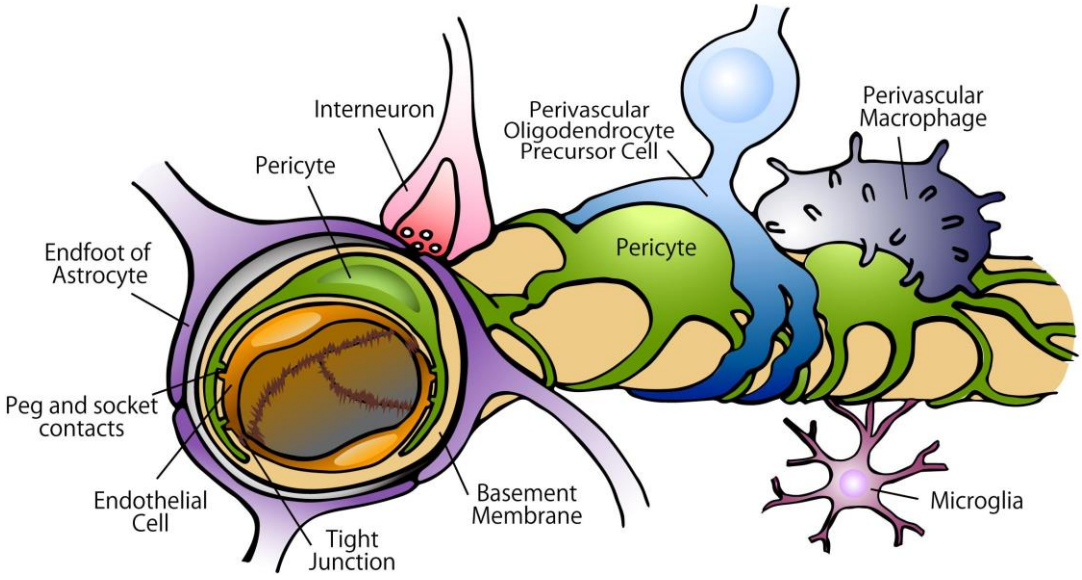


Figure 2. Constituents of the neurovascular unit: endothelial cells sealed by tight junctions, pericytes in the basement membrane, astrocyte end-feet, microglia, and perivascular macrophages. (Image source: Wikimedia Commons)

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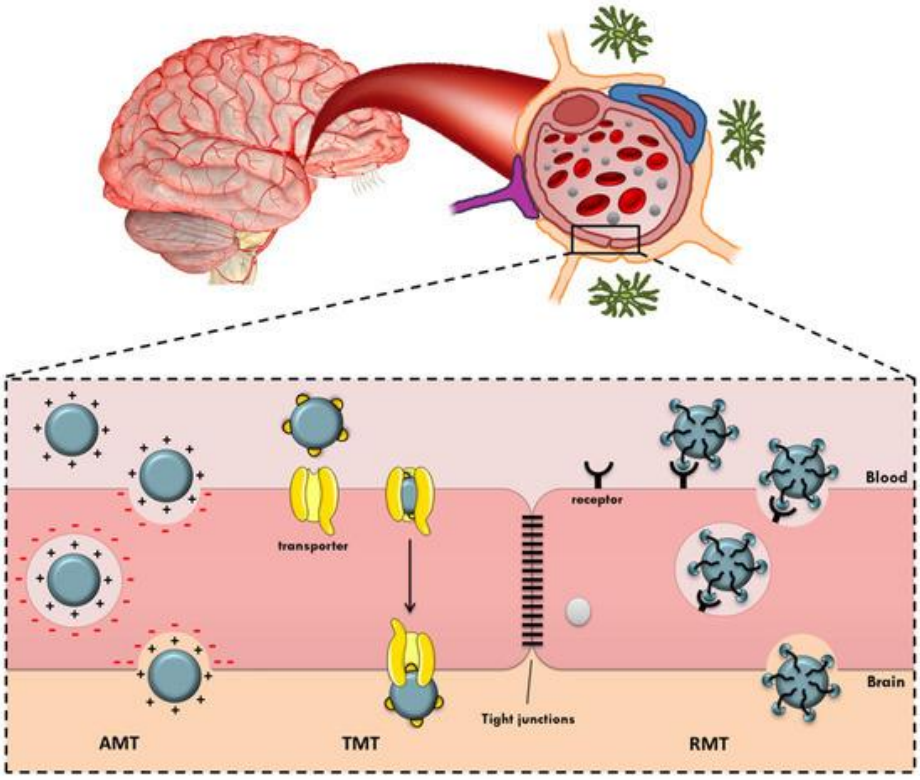


Figure 3. Strategies for nanoparticle crossing of the blood–brain barrier: adsorptive-mediated (AMT), transporter-mediated (TMT), and receptor-mediated (RMT) transcytosis. (Image source: MDPI Int. J. Mol. Sci., open access)

5. The Barrier at Work: Security, Maintenance, and Loopholes

Viewed as a security operation, the barrier performs at least three duties. First, exclusion: it filters plasma proteins, toxins, and circulating pathogens, maintaining the low-protein, tightly controlled ionic environment that neurons require for electrochemical signalling. Second, surveillance and response: the barrier is not passive but dynamic, relaxing its junctions locally in response to neuronal activity to increase blood flow where it is needed — a coupling so precise that imaging it (the BOLD signal) underpins all of functional MRI [5]. Third, selective recruitment: immune traffic into the healthy brain is deliberately minimal, with patrolling T-cells screened at the border rather than admitted wholesale, a policy that limits inflammation but complicates defence against CNS pathogens [15].

No security system is without service entrances, and the brain has several. The circumventricular organs — small midline structures such as the area postrema, the subfornical organ, and the neurohypophysis — sit outside the barrier, their leaky capillaries allowing the brain to sample the blood for toxins (triggering vomiting) and to secrete hormones directly into the circulation [6]. Elsewhere, the blood–cerebrospinal fluid barrier, formed by the choroid plexus epithelium, and the arachnoid

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barrier layer provide complementary lines of defence. Together these specialized borders partition the CNS into compartments, each with its own customs regime — a fact of direct clinical consequence, since antibiotics that treat meningitis must cross different barriers than those that treat brain abscesses [14].

6. When Security Fails: Barrier Breakdown in Disease

A wall that never fails is a wall that never gets tested; the barrier is tested constantly. In acute ischaemic stroke, energy failure collapses the endothelial ion gradients that tight junctions depend on, and vessels leak within minutes — which is why gadolinium-enhanced MRI after stroke lights up the damaged territory, and why reperfused tissue swells dangerously [2]. In multiple sclerosis, autoimmune T-cells breach the barrier with the help of adhesion molecules and matrix proteases, and the resulting perivenular inflammation visible on contrast MRI has become a radiological signature of the disease [15].

In chronic neurodegeneration the story is subtler but no less consequential. Long before plaques accumulate, Alzheimer's disease shows evidence of microvascular dysfunction and barrier leakage; pericyte loss and impaired amyloid-β clearance across the vessel wall are now considered part of the disease's early biology, not merely its aftermath [9]. Glioblastoma exploits the barrier differently, co-opting pericytes and vessels to build a blood–tumour barrier that is both leaky in places and selectively impermeable — excluding chemotherapy while the tumour's tortuous, compressed vessels hypoxia-drive its aggressive growth [8]. And in meningitis, pathogens such as Neisseria meningitidis and Streptococcus pneumoniae actively remodel the junctions to gain entry, turning the host's own border crossing against it [14]. Table 2 summarizes these pathological patterns. The unifying lesson is that barrier breakdown is rarely a simple matter of a wall falling: it is a change in customs policy, and the brain pays the price either way.

Disease	**Blood–brain barrier alteration**	**Clinical consequence**
Ischaemic stroke	Energy-dependent tight junction failure; rapid vasogenic oedema	Haemorrhagic transformation risk; cytotoxic and vasogenic swelling

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Multiple sclerosis	Autoimmune cell trafficking; focal perivenular leakage	Demyelinating lesions; contrast enhancement on MRI tracks activity
Alzheimer's disease	Microvascular degeneration, pericyte loss, impaired amyloid-β clearance	Chronic leakage; reduced perfusion; possible contribution to neurodegeneration
Glioblastoma	Blood–tumour barrier; heterogeneous leakiness with retained efflux pumps	Chemotherapy exclusion; resistance; oedema from abnormal vessels
Bacterial meningitis	Pathogen-induced junction disruption and inflammation	Pathogen entry; altered antibiotic penetration; vasogenic oedema
Epilepsy	Barrier opening at seizure foci; altered transporter expression	May perpetuate seizure activity; drug resistance via efflux pumps
Brain metastases	Disrupted barrier at core, relatively intact at invasive rim	Protection of microscopic disease; variable contrast enhancement

Table 2. Disease states associated with blood–brain barrier disruption.

7. Defeating the Wall: Strategies for CNS Drug Delivery

For the drug developer, the barrier is a formidable adversary, and the history of CNS pharmacology is in large part a history of schemes to outwit it. The crudest approach is brute force: raising the systemic dose until enough drug crosses — a strategy that buys brain exposure at the cost of systemic toxicity. Osmotic disruption, in which a hypertonic mannitol infusion temporarily shrinks endothelial cells and opens the junctions, is its surgical cousin, still occasionally used to deliver chemotherapy in brain tumours but limited by seizures and non-selective opening [4].

Smarter schemes work with the barrier's own machinery. Prodrugs disguise a polar molecule in lipid-friendly clothing — the classic example is L-DOPA, which rides the LAT1 amino acid transporter and is decarboxylated to dopamine inside the brain,

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transforming the treatment of Parkinson's disease. Molecular Trojan horses graft a therapeutic (an enzyme, an antibody, a nanoparticle) onto a ligand such as transferrin or an anti-transferrin receptor antibody, tricking the endothelium into ferrying the cargo across by receptor-mediated transcytosis; this approach has carried enzyme replacement into the brain in clinical trials for lysosomal storage disorders [7,13]. Efflux pump inhibition and transporter-targeted prodrugs extend the same philosophy to small molecules.

7.1 Nanoparticles and physical methods

Nanotechnology has given the field its most versatile toolkit (Figure 4). Liposomes, polymeric nanoparticles, and solid lipid carriers can be coated with polyethylene glycol to evade the immune system and decorated with targeting ligands — transferrin, insulin, lactoferrin, or low-density lipoprotein receptor binders — to engage transcytosis; some cationic particles cross by adsorptive mechanisms. More than a decade of optimization has improved brain accumulation, though translating elegant rodent data into consistent human benefit remains the field's honest challenge [16].

Perhaps the most dramatic method is focused ultrasound. Low-intensity ultrasound beams, combined with intravenously injected microbubbles, mechanically perturb the vessel wall and open the barrier reversibly at a precise, MRI-guided location — a temporary, localised loosening of border controls that has already been tested in patients with Alzheimer's disease and brain tumours, with encouraging safety signals [12]. Intranasal delivery offers a complementary back door: drugs deposited along the nasal mucosa can travel along olfactory and trigeminal pathways into the CNS, bypassing the bloodstream entirely — though uptake is variable and best suited to small, lipophilic agents. Each strategy trades among three currencies: how much drug reaches the brain, how selectively, and at what risk.

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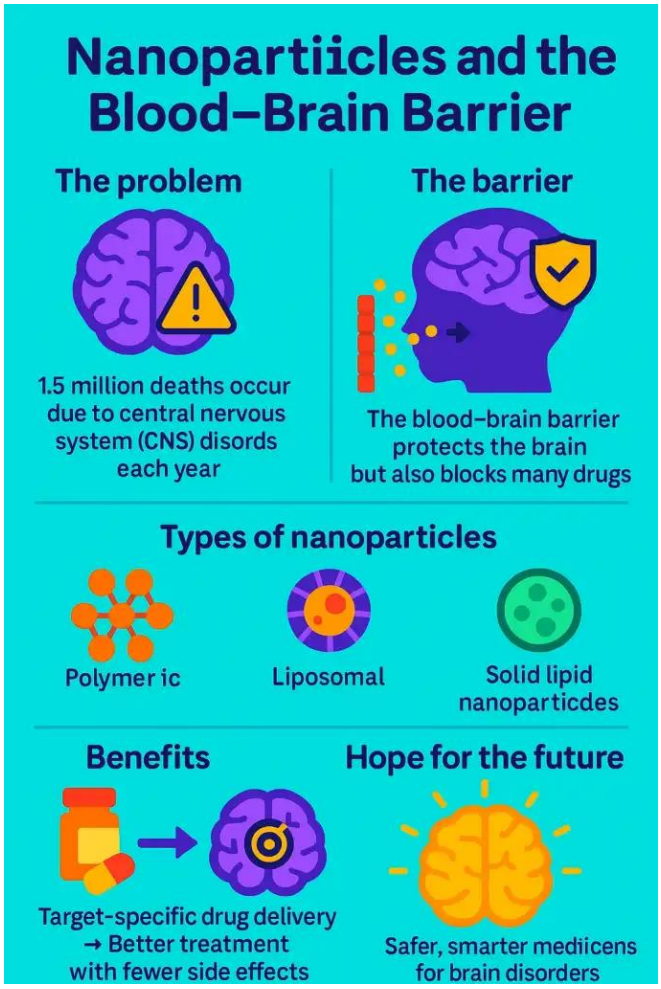


Figure 4. Nanoparticle-based approaches to blood–brain barrier crossing and their promise for central nervous system disorders. (Image source: Premier Science, open access)

8. Treating the Barrier Itself

An emerging insight of the last decade is that the barrier need not always be the obstacle to be overcome — sometimes it is the therapeutic target itself. Because barrier leakage and inflammation feed one another in stroke, multiple sclerosis, and neurodegeneration, stabilizing the junctions or restoring pericyte function may slow disease independently of any drug delivered across them. Modulators of Wnt and Sonic hedgehog signalling — pathways that construct the barrier during embryogenesis — are being explored to repair it in adulthood [5]. In brain tumours, normalizing the chaotic tumour vasculature with anti-angiogenic therapy (bevacizumab) reduces oedema and may transiently improve drug delivery by restoring pressure gradients [8]. And in epilepsy, where barrier opening at seizure foci may itself be pro-ictogenic, sealing the leak is a genuine therapeutic hypothesis rather than a metaphor [2].

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9. Conclusion

The blood–brain barrier is neither the impenetrable wall of pharmacology textbooks nor the simple leaky sieve of pathology atlases. It is a dynamic, intelligent border — built by endothelium, pericytes, and astrocytes; policed by tight junctions and efflux pumps; opened on demand for nutrients and, when disease strikes, breached at our peril. Its very existence explains the slow progress of CNS therapeutics, and its molecular anatomy now points the way forward: prodrugs that ride its transporters, antibodies that hijack its receptors, nanoparticles that negotiate its transcytosis, and ultrasound that opens its gates under image guidance, locally and reversibly. The next generation of brain medicines will likely succeed not by smashing the barrier down but by learning its rules well enough to slip past its guards — respecting the security system even as they outsmart it. Important questions remain: how heterogeneous the barrier is between brain regions and individuals, how it changes with age, and how best to measure its function in living patients [17]. Answering them will determine whether the body's ultimate security system becomes, at last, a cooperative partner in medicine.

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THE 'DEFAULT-TO-STEROIDS' PITFALL IN ACUTE CARE

Dr Saloni Jain , Assistant, Samarkand state medical university, Uzbekistan

Parth , Medical Student, Samarkand state medical university, Uzbekistan

Punith , Medical Student, Samarkand state medical university, Uzbekistan

Jemimah , Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Corticosteroids are among the most potent anti-inflammatory drugs in the acute-care armamentarium, and they are also among the most casually prescribed. In emergency departments, urgent-care centres and out-of-hours clinics around the world, short courses of prednisone, dexamethasone or methylprednisolone are routinely dispensed for sore throats, acute bronchitis, nonspecific back pain and undifferentiated respiratory symptoms, conditions for which the evidence of benefit is weak, absent or outright negative. This narrative article examines the phenomenon that clinicians increasingly call the 'default-to-steroids' pitfall: the reflexive reach for a steroid prescription when a patient is uncomfortable, a diagnosis is uncertain, and time is short. The article traces the clinical and psychological roots of the reflex, reviews the evidence base for steroids across the most common acute indications, catalogues the harms that accumulate in populations exposed to repeated short courses, and proposes practical strategies for safer, more deliberate prescribing. The central argument is not that corticosteroids lack value; conditions such as asthma exacerbation, anaphylaxis, adrenal crisis, croup and acute COPD exacerbation are unequivocal indications. The argument is that the reflex has quietly outgrown the evidence, and that each apparently harmless prescription erodes both patient safety and the culture of careful prescribing. A framework of five questions for the acute-care clinician is offered as a practical antidote.

Keywords: corticosteroids; glucocorticoids; prednisone; dexamethasone; prescribing cascade; antimicrobial stewardship; emergency medicine; urgent care; medical overuse; deprescribing

1. Introduction

Every emergency physician knows the moment. A patient sits on the gurney with a scratchy throat and a miserable expression, or a persistent cough after a cold, or a back that seized up that morning. The examination is unremarkable. There is no bacterial infection to treat, no fracture to splint, no procedure to perform. Time pressure hums in the background: the waiting room is full, the next patient is on the tracker board, and

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the current one looks up hopefully. In that moment, the path of least resistance is a small, white, familiar prescription slip: prednisone 40 milligrams for five days. It feels like doing something. It often is also doing harm.

Corticosteroids occupy a peculiar place in acute medicine. They are genuinely lifesaving in a short, well-defined list of emergencies, anaphylaxis, severe asthma, adrenal crisis, raised intracranial pressure, spinal cord compression, and their anti-inflammatory power is unmatched at the bedside. Yet the same pharmacologic potency that makes them valuable makes them hazardous when applied indiscriminately. Over the past two decades, observational studies from North America, Europe and Australasia have documented rising rates of short-course systemic steroid prescriptions issued in ambulatory and acute-care settings, including for conditions with no supporting evidence. Critics have coined a term for the underlying habit: the 'default-to-steroids' pitfall, the reflexive prescribing of a steroid course where the indication is absent, dubious or unexamined.

This article offers a narrative exploration of that pitfall: where it comes from, what it costs, and how acute-care clinicians can keep steroids where they belong, powerful tools in a short list of diseases, rather than a placebo-grade reflex for human misery.



Figure 1. Prednisone remains one of the most dispensed prescriptions in acute and ambulatory care worldwide.

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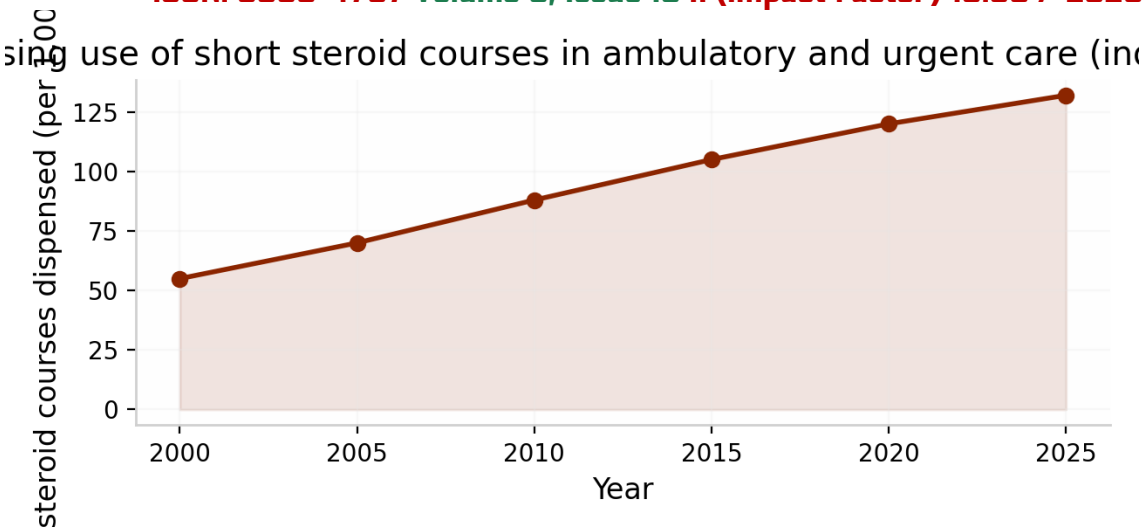


Figure 2. Indicative trend of short-course systemic steroid dispensing in ambulatory and urgent-care settings.

2. The Anatomy of a Reflex

2.1 The tyranny of time and the pressure to act

Acute care runs on minutes. The median emergency-department encounter in many health systems lasts well under an hour, and urgent-care consultations are shorter still. Within that window the clinician must assess, diagnose, reassure, treat and document. A steroid prescription offers a seductively efficient package: it is cheap, familiar, perceived as safe, and it gives the patient a tangible token of medical attention. When the alternative is a careful explanation that most sore throats and coughs simply need time, the prescription can feel like the more compassionate option, even when the evidence says otherwise.

2.2 Diagnostic uncertainty and prescribing as punctuation

A steroid course also functions psychologically as punctuation: it closes an ambiguous encounter. Nonspecific symptoms, a cough, fatigue, low-grade fever, vague chest discomfort, resist precise diagnosis. Prescribing converts uncertainty into action, giving both clinician and patient the sense that the visit has produced something. Research on 'treatment as diagnosis' shows that clinicians often prescribe partly to hedge, reasoning that if the steroid helps, the inflammation was 'real'; if it does not, the illness was self-limiting either way. The patient, meanwhile, learns a lesson that will shape future encounters: severe symptoms merit a steroid, and clinicians who withhold one are withholding care.

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2.3 Cultural momentum and learned habits

The reflex is taught. Trainees watch seniors prescribe dexamethasone for every sore throat and prednisone for every wheeze; the habit replicates across generations of clinicians with little explicit scrutiny. Marketing has played a role as well: single-dose dexamethasone was long promoted for pharyngitis, and a wave of small positive trials in the 2000s, later tempered by larger and better-controlled studies, embedded the practice before the evidence matured. Once established, such habits resist revision because each individual prescription appears trivially small.



Figure 3. Busy emergency and acute-care environments, where time pressure and diagnostic uncertainty make reflexive prescribing especially tempting.

3. The Evidence: Where Steroids Help, and Where They Do Not

3.1 The unequivocal indications

Certain acute conditions demand corticosteroids, and withholding them is a genuine error. In acute asthma exacerbation, systemic steroids shorten recovery and prevent relapse; in anaphylaxis, intramuscular adrenaline remains first-line but steroids are standard adjuncts; in acute exacerbations of COPD, steroids shorten recovery in patients with respiratory acidosis or requiring hospitalisation; in croup, a single dose of dexamethasone is one of the most evidence-supported treatments in all of paediatrics; and in adrenal crisis, septic shock refractory to fluids, and selected

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neurological emergencies, steroids are simply non-negotiable. The pitfall lies not in these indications but in the long tail of conditions where steroids have drifted into routine use without supporting evidence.

3.2 Sore throat

Pharyngitis is perhaps the purest example of the pitfall. Early trials suggested a single dose of dexamethasone reduced pain at 24 to 48 hours; however, the largest and most rigorous trial to date, a multicentre study of adults with moderate-to-severe sore throat, found no meaningful benefit and no reduction in antibiotic prescribing, while guidelines from the American College of Physicians and the Infectious Diseases Society of America recommend against routine steroid use. For most patients, an analgesic, a warm drink and time outperform a prescription, yet steroids for sore throat remain among the most common prescriptions in urgent care.

3.3 Acute bronchitis and the common cold

Acute bronchitis is almost always viral, and evidence is consistent: steroids do not reduce cough duration or severity in non-asthmatic adults. Nevertheless, emergency and urgent-care studies report steroids dispensed to substantial minorities, in some series more than a third, of bronchitis patients, driven by the feeling that the cough 'sounds inflammatory.' For undifferentiated upper respiratory infection, the evidence is similarly negative, and for acute rhinosinusitis trials show at most marginal, clinically insignificant benefit.

3.4 Low back pain and musculoskeletal complaints

For acute nonspecific low back pain, systematic reviews have repeatedly found no clinically meaningful benefit of systemic steroids over placebo, and one well-known emergency-department randomised trial showed no improvement in function or pain at one week or one month. Yet back pain is one of the leading drivers of steroid prescriptions in acute care, often in combination with an opioid, compounding two problematic prescribing cascades in a single visit.

3.5 COVID-19: a case study in indication-discipline

The pandemic offered a natural experiment in the cost of default-to-steroids thinking. When early observational enthusiasm met rigorous randomised trials, the message crystallised with unusual clarity: dexamethasone saves lives in hospitalised patients

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requiring oxygen, but offers no benefit, and possible harm, in mild disease. The contrast between the two populations became a teaching moment about why a steroid that is heroic in one context can be useless or harmful in another.

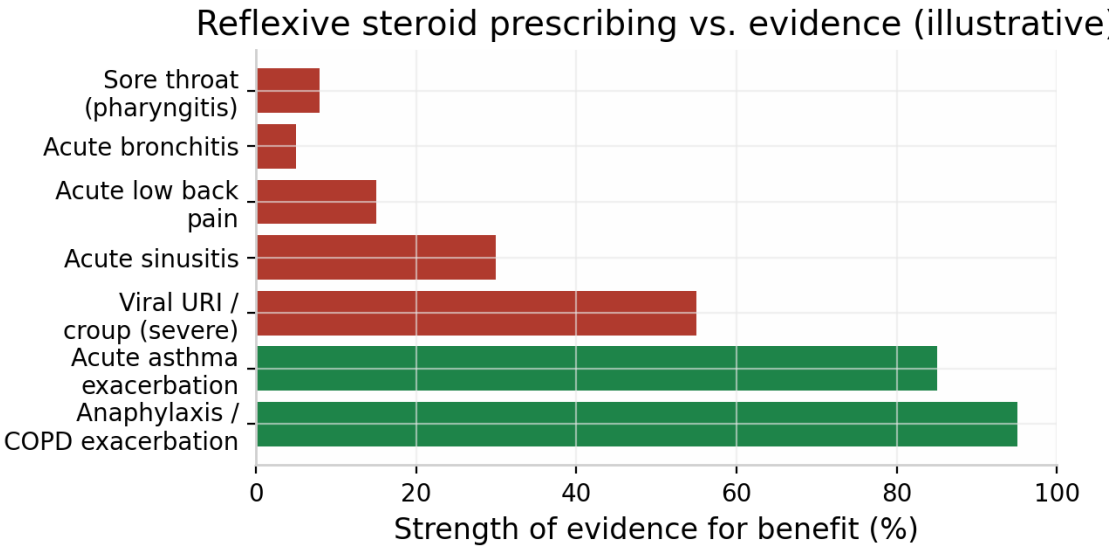


Figure 4. Strength of evidence for steroid benefit across common acute-care presentations (illustrative synthesis).

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Acute presentation	Evidence for systemic steroids	Recommendation	Safer first-line alternative
Sore throat (pharyngitis)	Large RCTs show no meaningful benefit; no effect on antibiotic use	Do not prescribe routinely	Analgesics (paracetamol/NSAID), fluids
Acute bronchitis (non-asthmatic)	Consistently negative trials	Do not prescribe	Symptomatic care; expectant management
Acute sinusitis	At most marginal benefit; not clinically significant	Not recommended	Intranasal steroids if persistent; saline, analgesia
Acute low back pain	RCTs show no improvement in pain or function	Do not prescribe	Stay active; NSAIDs; education/reassurance
Viral URI / common cold	No benefit	Do not prescribe	Symptomatic care
Croup (paediatric)	Strong benefit; single-dose dexamethasone is standard	Recommended	Dexamethasone 0.15–0.6 mg/kg
Acute asthma exacerbation	Strong benefit; reduces relapse and admission	Recommended	Prednisolone 40–50 mg adults; 1–2 mg/kg children
Acute COPD exacerbation	Benefit in hospitalised/acidotic patients	Recommended for severe exacerbations	Prednisolone 40 mg ~5 days
Anaphylaxis	Adjunct only; adrenaline is first-line	Give after adrenaline	IM adrenaline 1 mg/mL, 0.5 mg adult

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Adrenal crisis / septic shock (refractory)	Life-saving	Mandatory	Hydrocortisone 100 mg IV

Table 1. Steroid evidence and recommendations across common acute-care presentations.

4. The Costs of the Reflex

4.1 Immediate and short-term harms

Even a five-day course is not benign. Up to a quarter of patients on short courses report insomnia, agitation, mood disturbance or appetite changes; gastrointestinal upset, fluid retention and transient hyperglycaemia, clinically significant in diabetics, are common. Emergency-department studies of single-dose dexamethasone for pharyngitis documented measurable increases in adverse effects without offsetting benefit. In older adults, steroids compound confusion and falls; in people with diabetes, a 'routine' prednisone course can trigger days of difficult glucose control.

4.2 Population-level harm from repetition

The deeper danger is cumulative. Steroid courses are rarely counted across a lifetime, yet a patient who receives two or three 'short harmless' courses each winter accumulates substantial cumulative exposure. Large observational analyses link repeated short courses with increased risks of osteoporosis and fracture, cataract, glaucoma, type 2 diabetes, cardiovascular events, avascular necrosis and infection, with risks rising with cumulative dose even when no single course exceeds two weeks. Each apparently trivial prescription is a small withdrawal from an account the patient will draw on for decades.

4.3 The prescribing cascade

Steroids also seed further prescriptions. Steroid-induced hyperglycaemia generates antidiabetic treatment; insomnia generates hypnotics; gastritis symptoms generate proton-pump inhibitors; and a patient 'allergic' or intolerant to one NSAID after combination therapy may be labelled incorrectly, distorting future care. The cascade, prescriptions treating the side effects of prescriptions, is one of the most quietly expensive dynamics in acute medicine.

4.4 Erosion of diagnostic and therapeutic discipline

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Finally, the reflex exacts a professional cost. When a steroid is the answer to every inflamed-sounding symptom, the discipline of diagnosis atrophies. Cough becomes 'steroid-worthy' rather than a prompt to consider asthma, reflux, heart failure or infection; a sore throat becomes 'dexamethasone-worthy' rather than an invitation to examine, test for streptococcus, or simply reassure. The default substitutes ritual for reasoning, and both patients and clinicians are poorer for it.



Figure 5. Cushingoid features such as moon facies signal substantial cumulative glucocorticoid exposure.

5. A Five-Question Antidote

Breaking the default does not require more knowledge; it requires a pause. The following five questions, answerable in under a minute at the bedside, separate the genuine indications from the reflexive ones.

1. Is this a recognised steroid-responsive condition? If the diagnosis is not on the short evidence-based list, the burden of proof rests on the prescriber, not the disease.

2. Would I prescribe this if the patient were watching me reason aloud? The reflex survives on autopilot. Articulating the rationale aloud exposes how often it reduces to "they look miserable."

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3. What is the smallest dose and shortest course that could work? For true indications such as asthma, low-dose short-course regimens are often as effective as older, heavier ones.

4. What are this patient's specific risks? Diabetes, older age, osteoporosis, psychosis history, active infection and prior adverse reactions change the calculus dramatically.

5. What will I tell the patient if this does not work? If the honest answer is "come back regardless," the prescription was comfort for the clinician, not treatment for the patient.

Driver of the default-to-steroids reflex	Mechanism	Countermeasure
Time pressure	Prescriptions as efficient closure of brief encounters	Pre-visit scripts for common self-limiting conditions
Diagnostic uncertainty	Steroid as hedge against ambiguous presentations	Structured differential + explicit "treat vs. wait" decision
Patient expectation	Perceived demand for 'something strong'	Shared decision-making; explain natural history with timelines
Cultural habit	Prescribing patterns replicated from seniors	Local audit & feedback; explicit departmental guidelines
Defensive practice	Fear of appearing to withhold treatment	Document non-prescription decisions and follow-up plans
Marketing & legacy trials	Small positive studies entrenching practice	Regular evidence reviews; de-adoption campaigns

Table 2. Drivers of reflexive steroid prescribing and corresponding countermeasures.

6. Systems Solutions and Stewardship

Individual discipline matters, but the pitfall is also a system problem, and systems respond to system levers. Antimicrobial stewardship programmes have demonstrated

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that audit-and-feedback, order sets and clinical decision support can shift prescribing behaviour at scale; the same infrastructure can be extended to steroids. Emergency departments that instituted indication-tagged steroid prescribing, with hard stops or warnings for non-evidence-based indications such as pharyngitis and low back pain, have reported measurable reductions in inappropriate courses without worsening patient satisfaction. Education of the public is equally important: if patients arrive expecting a steroid for every cough, clinicians will keep reaching for the prescription pad.

Deprescribing and de-adoption deserve a place in acute-care quality agendas alongside antibiotic stewardship. Just as clinicians now hesitate before prescribing antibiotics for viral illness, a parallel norm is emerging for steroids: a brief, explicit justification at the moment of prescribing, recorded in the chart, that distinguishes treatment from reflex.

7. Conclusion

The default-to-steroids pitfall is not a story of careless clinicians. It is a story of good people working under bad conditions, time pressure, diagnostic fog and human suffering, reaching for a tool that is powerful, cheap and emotionally satisfying. But corticosteroids are not a salve for uncertainty, and a prescription written to end an uncomfortable encounter is a treatment for the clinician, not the patient. The evidence is now clear enough to draw a bright line: steroids are indispensable in a short list of acute diseases and unhelpful or harmful across most of the rest. Discipline, a five-question pause, a handful of honest alternatives and systems that make the right choice the easy choice, can return these potent drugs to their rightful place: deployed with intention, counted with care, and reserved for the moments when they genuinely save.

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OBESITY IS A CHRONIC DISEASE: BEYOND BMI

Dr Shahrukh Azmi , Assistant, Samarkand State Medical University, Uzbekistan

Shejal chopal , Medical Student, Samarkand state medical university, Uzbekistan

Riya thakur , Medical Student, Samarkand state medical university, Uzbekistan

Vaishnavi thakur , Medical Student, Samarkand state medical university, Uzbekistan

Ashrith , Medical Student, Samarkand state medical university, Uzbekistan

Abstract

For more than a century, obesity has been weighed, charted, and moralised—reduced to a ratio of height over weight squared and, too often, to a verdict of weak will. Yet since the American Medical Association recognised obesity as a disease in 2013, and with particular force since the Lancet Diabetes & Endocrinology Commission's 2025 redefinition, medicine has begun to treat excess adiposity as what the biology always said it was: a chronic, relapsing, multifactorial illness of dysfunctional adipose tissue. This narrative review traces how obesity crossed the threshold from behaviour to disease; why body mass index (BMI), the instrument that built that threshold, is now recognised as an inadequate ruler for individuals; what adiposopathy and the genetics of energy homeostasis reveal about the condition's mechanisms; and how the new distinction between clinical and preclinical obesity promises to end both overdiagnosis and therapeutic neglect. We examine staging systems, the treatment revolution spearheaded by incretin-based pharmacotherapy, and the unresolved tensions—stigma, medicalisation, equity, and the fate of the "obesity paradox." The evidence supports a clear thesis: obesity qualifies as a chronic disease not because a committee declared it so, but because it behaves like one—progressive, relapsing, genetically and environmentally driven, and treatable only when it is measured beyond the scale.

Keywords: obesity; chronic disease; body mass index; adiposopathy; clinical obesity; preclinical obesity; Lancet Commission; Edmonton Obesity Staging System; GLP-1 receptor agonist

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1. Introduction

Consider two patients in the same clinic. The first is a rugby forward: 1.83 metres tall, 110 kilograms, BMI 32.9—squarely in the "obese" range by every textbook table—whose blood pressure, glucose, and lipids are impeccable and whose body fat hovers near 14%. The second is a retired schoolteacher: 1.60 metres, 63 kilograms, BMI 24.6—"normal weight"—whose waist circumference tells a different story, whose liver shimmers with fat on ultrasound, and whose fasting glucose has quietly crossed into diabetes. Two bodies, two diseases, one number that gets the first man wrong and misses the second woman entirely. For decades this arithmetic was the whole of the diagnosis.

The story of how obesity became a disease is really two stories: one of biology, in which adipose tissue was revealed as an endocrine organ whose dysfunction drives systemic illness; and one of policy, in which medicine's institutions slowly granted the condition the standing of a chronic disease—one that, like hypertension or asthma, requires long-term management rather than willpower and blame. The National Institutes of Health declared obesity a disease in 1998; The Obesity Society followed in 2008; the American Association of Clinical Endocrinologists in 2012; and in June 2013 the American Medical Association's House of Delegates, overriding its own Council on Science and Public Health, voted to recognise obesity as a disease state "with multiple pathophysiological aspects requiring a range of interventions." The pace then accelerated dramatically: in January 2025, a Lancet Commission of 58 experts, endorsed by more than 75 medical organisations worldwide and including people with lived experience of obesity, published a consensus redefinition that demoted BMI from diagnostic instrument to screening tool. This article follows that arc—why BMI was trusted, why it failed, what replaced it, and what the new framework changes for patients and physicians.

Year	Milestone	Significance
1998	NIH declares obesity a disease	Federal research and treatment legitimacy; begins shift from behaviour to pathology.
2004–2006	CMS reclassifies obesity; covers bariatric surgery under Medicare (2006)	Insurance reimbursement for obesity treatment becomes possible in the United States.

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2008	The Obesity Society position paper: obesity as a disease	Scientific society endorsement built on adipose-tissue biology.
2012	American Association of Clinical Endocrinologists position statement	Endocrinology leadership formally adopts disease framing.
June 2013	AMA House of Delegates recognises obesity as a disease state	Most influential medical association; catalyses coverage, research, and de-stigmatisation.
January 2025	Lancet Diabetes & Endocrinology Commission consensus (58 experts, 75+ organisations)	Redefines obesity around excess adiposity and organ function; creates clinical vs preclinical obesity; relegates BMI to population screening.

Table 1. Milestones in the recognition of obesity as a chronic disease.

2. The Blunt Instrument: Why BMI Cannot Bear the Weight of a Diagnosis

BMI was never designed to measure fat. Adolphe Quetelet devised the index in the 1830s to describe the "average man" of a population, not to diagnose anyone; it was adopted in clinical practice largely because height and weight are cheap to obtain. But a ratio of mass to height squared cannot distinguish adipose from muscle, visceral from subcutaneous fat, or a metabolically healthy fat depot from an inflamed, dysfunctional one. The consequences are systematic and well documented. Elite athletes are misclassified as obese. Conversely, the "TOFI" phenotype—thin on the outside, fat on the inside—passes through every screening net, particularly common in South and East Asian populations, in whom cardiometabolic risk rises at BMI and waist values far below the conventional cut-offs. The same BMI also means different things at different ages and in different ethnic groups, which is why the Commission insisted on ethnicity-, sex-, and age-appropriate thresholds. And at the far end of the spectrum, large epidemiological cohorts produced the so-called obesity paradox—overweight and class-I-obese patients with heart failure or chronic kidney disease sometimes outliving normal-weight ones—largely, critics argue, because BMI obscures which patients carry

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dangerous visceral adiposity and muscle wasting versus which carry metabolically quieter fat. A measure that can be wrong in both directions, in every population, at every extreme, is a screening statistic, not a diagnosis. The Lancet Commission's core recommendation follows directly: BMI should be used only as a surrogate measure of health risk at the population level, for epidemiology, or for initial screening—never as an individual measure of health.

3. The Biology That Earned the Diagnosis

What transformed the classification was not rhetoric but mechanism. Adipose tissue, it turns out, is an endocrine and immune organ, and in excess it becomes sick—"adiposopathy," in the coinage of obesity medicine. As adipocytes enlarge beyond their vascular supply, they become hypoxic, recruit macrophages, and secrete a pro-inflammatory soup of cytokines and adipokines that drives insulin resistance, endothelial dysfunction, and ectopic fat deposition in liver, muscle, and pericardium. The disease is also, crucially, a disease of the brain's energy-regulation circuitry. Twin, family, and adoption studies attribute 40–70% of the population variance in adiposity to heredity; more than twenty genes cause monogenic obesity, most acting on hypothalamic satiety pathways; and analyses of genome-wide association studies have implicated over a thousand loci whose variants nudge appetite and expenditure upward in obesogenic environments. Add leptin resistance, gut-brain signalling, sleep, stress, medications, and the built environment, and the stereotype of simple gluttony collapses. The physiology explains the clinical signature of a chronic disease: weight defended by powerful homeostatic forces, with roughly nine in ten people who lose weight regaining most of it as appetite hormones surge and resting metabolism falls. That pattern— progression, relapse, multifactorial causation—is precisely what defines chronic disease, and it is why the Obesity Medicine Association describes obesity as a "chronic, relapsing, multifactorial, neurobehavioural disease."

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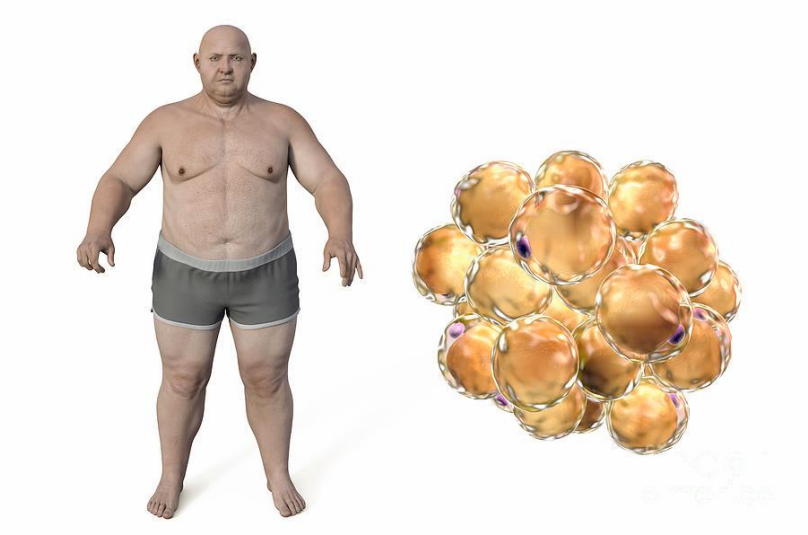


Figure 1. Obesity understood as adipocyte disease: excess, enlarged, and metabolically dysfunctional fat cells—not merely excess kilograms. (Photograph: K. Kon / Science Photo Library.)

4. The New Grammar: Clinical and Preclinical Obesity

The 2025 Commission report supplies what BMI never could: a definition of illness. Obesity is defined as excess or abnormally distributed adiposity, confirmed not by weight alone but by direct measurement (DXA or bioimpedance) or by anthropometry—waist circumference, waist-to-hip ratio, or waist-to-height ratio—using validated, ethnicity-specific cut-offs. Within that umbrella sit two distinct states. Preclinical obesity is excess adiposity with preserved organ function: a state of increased risk, warranting monitoring, risk counselling, and treatment when risk is substantial—but not yet an illness. Clinical obesity is excess adiposity that is already injuring tissues: obesity-induced heart failure, obesity-related breathlessness from reduced lung function, knee and hip disease with stiffness and lost range of motion, obstructive sleep apnoea, or metabolic-organ dysfunction—codified in eighteen diagnostic criteria for adults and thirteen organ-system criteria for children and adolescents. The distinction does real clinical work. It ends the absurdity in which insurers demand a second disease, such as diabetes, before paying for obesity treatment; clinical obesity is itself the indication. It protects people with high BMI but healthy organs from unnecessary pharmacotherapy and surgery. And it reframes treatment goals away from the scale—where weight cycling awaits—toward the restoration of organ function and daily living, echoing the older Edmonton Obesity Staging System, which grades patients 0–4 by metabolic, functional, and psychosocial impairment rather than by BMI alone.

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Traditional (BMI-centric) approach	Lancet Commission framework (2025)
Sole reliance on BMI ≥ 30 kg/m² (or ethnicity-specific cut-offs) for diagnosis.	Diagnosis requires confirmation of excess/abnormal adiposity: direct measurement (DXA, bioimpedance), or BMI plus at least one anthropometric criterion, or two anthropometric criteria (WC, WHR, WHtR) regardless of BMI; excess fat pragmatically assumed when BMI ≥ 40.
BMI treated as an individual measure of health and illness.	BMI demoted to a population-level, epidemiological, or screening tool—not an individual diagnostic measure.
No distinction between illness and risk; "obese or not" as the whole question.	Two categories: preclinical obesity (excess fat, preserved organ function—increased risk, not yet illness) and clinical obesity (excess fat with organ dysfunction or major limitation of daily activities—an illness in its own right).
Treatment typically justified only after a comorbidity (e.g., type 2 diabetes) emerges.	Clinical obesity itself justifies treatment; preclinical obesity managed by risk reduction and monitoring, with treatment if risk is very high.
Success defined as percentage of weight lost.	Success defined by restored organ function, improved daily living, cardiometabolic risk reduction, and prevention of progression—weight change as one marker, not the goal.

Table 2. The traditional BMI-centric model versus the Lancet Commission diagnostic framework.

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5. The Treatment Revolution the Redefinition Made Possible

Disease status matters most when it changes what can be prescribed. Recognition of obesity as a chronic disease legitimated the armamentarium that now exists: intensive behavioural therapy, metabolic and bariatric surgery (which remains the most durable intervention for clinical obesity), and—most consequentially—the incretin-based pharmacotherapy era. Semaglutide, a GLP-1 receptor agonist, produces sustained mean weight loss of roughly 15% in trials, and tirzepatide, combining GIP and GLP-1 activity, has exceeded 20% in head-to-head studies, with encouraging signals for cardiovascular, renal, and hepatic outcomes. These are not diet pills; they act on the very neurobehavioural pathways the disease definition describes, restoring satiety signalling against a biology that defends excess weight. Yet the gap between capability and delivery remains scandalous: fewer than one in four people living with obesity receives any evidence-based treatment, hampered by cost, access, and lingering policy that still treats the condition as cosmetic. Precision medicine—phenotyping patients by genotype, adipose distribution, and metabolic phenotype to match diet, drug, device, or surgery—is the horizon the new definition points toward.

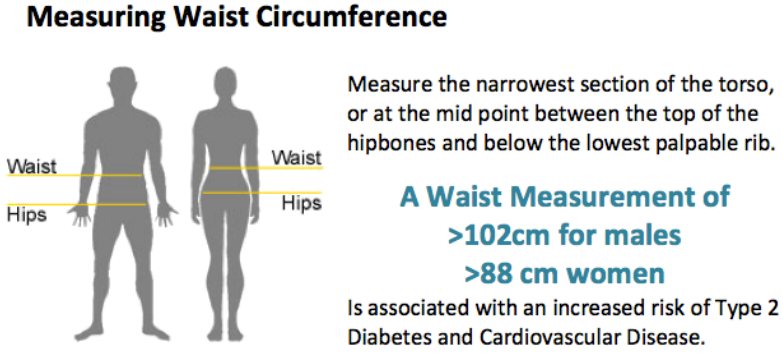


Figure 2. Waist circumference measurement: the narrowest torso point or midpoint between lowest rib and iliac crest. Values above 102 cm (men) and 88 cm (women) signal elevated visceral adiposity and cardiometabolic risk. (Infographic: Fijian Ministry of Health.)

6. Honest Tensions: Medicalisation, Stigma, and the Paradox That Will Not Die

The disease framing is not uncontested, and its critics raise questions worth keeping in view. Medicalisation can pathologise body diversity and hand pharmaceutical markets a mandate; the Commission itself acknowledged the overdiagnosis risk, which is precisely what the clinical/preclinical divide is designed to contain. Labels can cut both ways on stigma: some studies show that a disease attribution reduces blame for individuals, while others warn that treating body size as pathology deepens societal weight bias—language such as "person living with obesity" rather than "obese person"

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is the small daily countermeasure. The obesity paradox, though much of it dissolves when waist circumference and body composition replace BMI, reminds us that the relationship between adiposity and outcomes is a continuum, not a cliff edge, and that patients reclassified as "non-obese" under the new definition still carry intermediate risk and deserve counsel rather than dismissal. And equity remains the unfinished chapter: the medications and surgeries that the chronic-disease model legitimises are least available where obesity prevalence and its complications are highest.

7. Conclusion

The case for obesity as a chronic disease now stands on three converging legs. Mechanistically, adiposopathy, neurobehavioural dysregulation of energy balance, and strong heritability make obesity behave like—and be—an illness rather than a habit. Epidemiologically, BMI's twin failures of overdiagnosis and underdiagnosis have forced better instruments—waist measures, body composition, staging—that describe the disease the way it actually is. And clinically, treatment has outgrown the old paradigm, with pharmacotherapy that acts on the disease's own pathways and surgical options with decades of outcome data, both awaiting the coverage that disease recognition was designed to unlock. For the rugby forward, the new framework says: high BMI, healthy organs—monitor, counsel, no stigmatising label of illness. For the schoolteacher, it says: modest BMI, sick liver—diagnosed, staged, treated. That is what "beyond BMI" means in practice: the end of weighing people to decide whether they are ill, and the beginning of measuring them to find out. The scale never deserved the final word. It kept it for a century anyway. Medicine is finally taking it back.

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MICROPLASTICS AND HUMAN HEALTH: AN EMERGING MEDICAL-ENVIRONMENTAL PROBLEM

Mely Taku , Medical Student, Samarkand state medical university, Uzbekistan

Mezoon Hameed , Medical Student, Samarkand state medical university, Uzbekistan

Bengia Kartik , Medical Student, Samarkand state medical university, Uzbekistan

Kenjom Bagra , Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Plastic materials are widely used in packaging, clothing, transport, construction and healthcare. As larger plastic items weather, fragment or shed fibres, small particles known as microplastics can enter air, water, soil and food. Human exposure may occur mainly through ingestion and inhalation, while the contribution of dermal contact remains less clear. Researchers have detected plastic particles in several human samples, including stool, lung tissue, blood and placental tissue, raising important questions about possible biological effects. This narrative review summarises the sources and pathways of exposure, current evidence on potential health consequences, and practical approaches to prevention. Laboratory and animal studies suggest possible mechanisms including oxidative stress, inflammation, changes in immune responses, disruption of intestinal barriers and effects related to chemicals associated with some plastics. Human studies are growing, but they remain limited by small samples, different analytical methods, possible contamination and difficulty establishing cause and effect. Therefore, detection of microplastics in a tissue should not automatically be interpreted as proof that the particles caused disease. Evidence is not yet sufficient to quantify an individual's risk or to attribute specific common diseases to microplastic exposure. A precautionary public-health response is nevertheless reasonable: reducing avoidable plastic pollution, improving waste and wastewater management, limiting unnecessary single-use plastics, and strengthening validated monitoring and long-term human research. The issue calls for collaboration between clinicians, environmental scientists, public-health authorities, industry and communities.

Keywords: microplastics; nanoplastics; human health; environmental pollution; ingestion; inhalation; oxidative stress; public health

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1. Introduction

Plastic has become part of everyday life because it is lightweight, durable and relatively inexpensive. These same qualities create a long-term environmental challenge. Plastic bags, bottles, synthetic textiles, tyre wear and packaging can break into smaller pieces, while washing synthetic clothing and the use of some industrial products can release fibres or particles. Once released, particles may move through waterways, soil, indoor dust and the atmosphere. Their small size makes them difficult to remove completely and challenging to measure consistently.

Microplastics are commonly defined as plastic particles smaller than 5 millimetres. Nanoplastics are smaller still, although definitions and size cut-offs vary across studies. Size, shape, polymer type, surface chemistry and attached substances may all influence how particles behave in the environment and interact with biological systems. A fibre, a fragment and a spherical laboratory particle should not automatically be assumed to have identical effects.

The medical concern is straightforward: people encounter these particles through everyday environments, and researchers increasingly report them in biological samples. However, a careful distinction is essential between exposure, biological response and clinical disease. Finding particles in a human sample shows that contact or internal presence may occur; it does not, by itself, demonstrate that the particles caused a particular illness. The World Health Organization (WHO) has emphasised major knowledge gaps and the need for better-quality research on exposure through food, water and air [1,2].

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Figure 1. Conceptual illustration of possible microplastic exposure through air, water and food. The image is illustrative and does not represent measured particle concentrations or proven organ injury.

2. Literature overview and approach

This narrative review was prepared from selected authoritative assessments and peer-reviewed reviews and studies identified through targeted searches of PubMed-linked literature and WHO publications. Priority was given to sources discussing environmental exposure, human biomonitoring, health outcomes and methodological limitations, including the WHO reports on drinking water and dietary/inhalation exposure, a 2024 rapid systematic review of digestive, reproductive and respiratory outcomes, and a review of human biomonitoring [1–5]. The purpose is to provide an accessible overview rather than a formal systematic review.

The literature is difficult to compare because studies differ in sampling methods, particle-size detection limits, polymer identification, contamination controls and the types of biological samples examined. Airborne fibres from clothing or laboratory materials can contaminate samples if collection and analysis are not carefully controlled. In addition, studies may count particles, estimate mass, or report polymer signals—measurements that are not interchangeable. These differences limit direct comparisons between studies and make estimates of daily exposure uncertain.

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3. Sources and routes of human exposure

3.1 Food and drinking water

Microplastics have been reported in various foods and drinking-water samples. Potential sources include environmental contamination, processing and packaging, and particles deposited during preparation or storage. Seafood has received attention because aquatic organisms can ingest plastic particles, but exposure is not limited to seafood: drinking water, salt, other foods and household dust may also contribute. The relative importance of each source depends on local conditions and on the methods used to measure particles.

WHO's 2019 assessment concluded that available evidence at that time suggested a low health risk from microplastics in drinking water, while stressing that the data were limited and further research was needed [1]. Its 2022 report broadened the assessment to dietary and inhalation exposure and identified substantial uncertainties in exposure measurement and toxicity [2]. These assessments should be interpreted as evaluations of the evidence available at the time, not as proof that all particle types, sizes or exposure levels are harmless.

3.2 Air and indoor dust

People may inhale airborne particles released from synthetic textiles, household dust, industrial processes, vehicle tyre wear and the breakdown of plastic materials. Indoor environments deserve attention because people spend substantial time inside buildings, where fibres can be released from clothing, furnishings and other materials. Larger inhaled particles may be trapped in the upper airways and cleared, whereas smaller particles may behave differently. The amount that reaches the lower respiratory tract and the fraction retained in tissue depend on particle characteristics and breathing conditions.

3.3 Occupational and other exposure settings

Workers in plastic production, recycling, textile manufacturing, waste sorting and related industries may encounter higher concentrations of plastic dust or fibres than the general population. Actual exposure varies with tasks, ventilation, material handling and protective measures. Occupational hygiene—including source enclosure, effective ventilation, housekeeping that avoids resuspending dust, and appropriate respiratory protection based on risk assessment—can reduce inhalation exposure. More studies are

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needed to define the contribution of occupational microplastics to long-term health outcomes.

4. Potential biological effects and health concerns

4.1 Inflammation and oxidative stress

Experimental studies have proposed several mechanisms by which some micro- and nanoplastics could affect cells. Particles may interact with cell membranes, trigger inflammatory signalling or contribute to oxidative stress, a condition in which reactive molecules exceed the body's capacity to neutralise them. These effects depend strongly on particle size, shape, dose, polymer, surface characteristics and experimental conditions. Many experiments use concentrations or exposure routes that may not closely reflect everyday human exposure, so results should not be translated directly into a prediction of clinical harm.

4.2 Digestive system and gut microbiome

Ingested particles may pass through the digestive tract, while a smaller fraction of certain particles may interact with intestinal surfaces or potentially cross biological barriers. Animal and laboratory studies have reported changes in intestinal inflammation, barrier function and gut microbial patterns under some experimental conditions. Human evidence is still limited, and it is not established that ordinary environmental exposure to microplastics causes a specific digestive disease. Future studies need reliable exposure estimates and validated clinical outcomes, rather than relying on particle detection alone.

4.3 Respiratory system

Inhalation is a plausible route of exposure, particularly for airborne fibres and dust. Experimental research suggests that some particles can provoke local inflammatory responses or cellular stress. Human studies have detected plastic particles in respiratory samples and lung tissue, including in occupational contexts, but the extent to which environmental microplastic exposure independently contributes to asthma, chronic obstructive pulmonary disease or other respiratory conditions remains uncertain. Smoking, air pollution, workplace chemicals and other factors must be carefully considered in any clinical study.

4.4 Reproductive and developmental health

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Plastic-associated chemicals and some particle exposures have raised concerns about reproductive and developmental health. Laboratory and animal studies have investigated sperm quality, reproductive hormones, placental interactions and developmental endpoints. Some observational human studies have reported particles in reproductive-related samples, but sample sizes are often small and confounding is difficult to exclude. It would be premature to conclude that microplastics are a proven cause of infertility, miscarriage or adverse birth outcomes. Research should distinguish effects of the plastic particle itself from effects of additives or other chemicals associated with plastic products.

4.5 Cardiovascular and other systemic outcomes

Emerging studies have reported micro- or nanoplastics in blood and in some vascular specimens, and investigators are exploring possible associations with inflammation and cardiovascular events. These findings are important signals for further investigation, but they do not yet establish a causal relationship. Potential confounders, analytical contamination and the difficulty of quantifying particle burden must be addressed. Evidence concerning neurological, metabolic and long-term cancer outcomes is also incomplete, and strong claims should be avoided until replicated human studies are available.

5. Summary of evidence

Topic	What the literature suggests	Main limitation
Exposure	People can encounter microplastics through food, water and air.	Sources and doses are difficult to compare.
Human detection	Particles or polymer signals have been reported in several human samples.	Contamination control and analytical methods vary.
Inflammation / oxidative stress	Mechanisms are supported mainly by experimental evidence.	Dose and particle type may not reflect real-world exposure.
Digestive / respiratory health	Animal and laboratory findings raise plausible concerns.	Causal human evidence remains limited.

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Reproductive / cardiovascular outcomes	Early human studies are investigating possible associations.	Small samples, confounding and uncertain exposure measures.
Population-level risk	Reducing unnecessary plastic pollution is a reasonable preventive goal.	Individual clinical risk cannot yet be quantified reliably.

Table 1. Overview of current evidence. This is a qualitative synthesis, not a formal grading of certainty for every outcome.

6. Prevention and public-health response

Because the science is still developing, prevention should focus on sensible measures that reduce avoidable plastic release without creating unnecessary fear. Individuals can use durable alternatives to disposable products where practical, avoid heating food in plastic containers unless the product is specifically designed for that use, follow local guidance for food storage, and reduce littering. These actions may reduce some exposures, but no household measure can guarantee complete avoidance of microplastics. Bottled water should not be assumed to be universally safer than tap water; water quality depends on local supply and treatment.

At community and policy levels, the largest opportunities often lie upstream: reducing unnecessary single-use plastics, improving collection and recycling systems, preventing pellet and industrial dust loss, upgrading wastewater treatment where feasible, and designing products that shed fewer particles. Healthcare facilities can review avoidable disposable plastic use while preserving infection prevention and patient safety. Environmental monitoring should use harmonised methods so results can be compared across regions and over time.

Clinicians should respond to questions with balanced communication. There is currently no validated routine clinical test that can diagnose a person's illness as being caused by microplastic exposure, and testing for particles in a biological sample is not a standard screening procedure. Patients should not be encouraged to buy unproven detoxification products or undergo unnecessary tests. Instead, clinicians can support evidence-based environmental health practices and explain what is known, what remains uncertain, and why reducing pollution is worthwhile even before every health effect is resolved.

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7. Research priorities

Future research should use standard definitions and validated methods to identify particle size, shape, polymer type and concentration. Field and laboratory blanks, strict contamination control, transparent reporting and independent replication are essential. Longitudinal human studies are needed to test whether measured exposure precedes health outcomes, and researchers should control for age, occupation, smoking, diet, socioeconomic conditions and other environmental pollutants. Studies should also distinguish the effects of particles from additives, plastic-associated chemicals and contaminants carried on particle surfaces. Finally, researchers should prioritise outcomes that matter clinically, rather than treating detection alone as evidence of disease.

8. Discussion

Microplastics illustrate a wider challenge in environmental medicine: a contaminant may be widespread before its long-term health effects are fully understood. Public concern is understandable, especially when particles are reported in human tissues. Yet uncertainty cuts both ways. It is not scientifically appropriate to claim that microplastics are harmless under all conditions, nor to state that they have been proven to cause a wide range of chronic diseases. Current evidence supports concern, improved monitoring and prevention, while requiring restraint in causal claims.

The issue cannot be solved by individual behaviour alone. People have limited control over the particles present in ambient air, municipal water, food systems and workplaces. Manufacturers, regulators, waste-management services, researchers and healthcare professionals all have roles. A proportionate approach combines reduced plastic leakage with better evidence and transparent communication. This can protect environmental quality now while building the knowledge needed for future clinical guidance.

9. Conclusion

Microplastics are an emerging environmental-health concern because exposure through food, water and air is widespread and particles have been reported in several human samples. Experimental evidence suggests possible inflammatory, oxidative and barrier-related effects, but the clinical significance of everyday exposure and the causal links to specific human diseases remain uncertain. The most responsible response is neither alarmism nor dismissal. It is to reduce avoidable plastic pollution, strengthen occupational and environmental controls, standardise analytical methods and invest in well-designed human studies. Clear communication should acknowledge uncertainty while supporting practical, evidence-informed prevention.

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THE FIRST 1,000 DAYS: BUILDING HEALTH BEFORE DISEASE

Dr. Konika Singh, Medical Student, Samarkand state medical university, Uzbekistan

Dr. Farha Naaz, Medical Student, Samarkand state medical university, Uzbekistan

Dr. Mohammad Shahid Ahmad, Medical Student, Samarkand state medical university, Uzbekistan

Dr. Arsh Arif, Medical Student, Samarkand state medical university, Uzbekistan

Dr. Rishabh Yadav, Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Between conception and a child's second birthday lies a window of barely three years during which the human body and brain are built at a pace they will never repeat—a window the nutrition community calls the first 1,000 days. This narrative review examines why that window holds such extraordinary power and fragility: how the developing brain forges up to a thousand new neural connections each second, reaches 80% of its adult size by age two, and consumes two-thirds of the body's resting energy; how maternal nutrition, stress, and health behaviours are written into the fetus through epigenetic mechanisms; and how the developmental origins of health and disease (DOHaD) framework traces adult cardiometabolic illness back to events before birth. We review the critical micronutrients of the window—folate, iron, iodine, zinc, choline, vitamin D, and long-chain omega-3 fatty acids—the three stages of the journey (pregnancy, exclusive breastfeeding, and complementary feeding), and the non-nutritional determinants of nurturing care, toxic stress, water and sanitation, and maternal mental health. The economic arithmetic is stark: roughly a quarter of children under five globally are stunted, malnutrition drains about $1 trillion in yearly productivity, and every dollar invested in early nutrition can return up to $23. The review concludes that the first 1,000 days are medicine's only genuine opportunity to prevent, rather than merely treat, the chronic diseases of adulthood—and that much of the world's future health is decided at a dinner table, a clinic visit, and a kitchen sink long before a child can speak.

Keywords: first 1,000 days; developmental origins of health and disease (DOHaD); maternal nutrition; stunting; epigenetics; early childhood development; breastfeeding; complementary feeding

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1. Introduction

In a maternity ward at dawn, a newborn is weighed for the first time. The number—2.4 kilograms, say—will follow this child in ways no one in the room can yet imagine. It will shape her risk of stunting and infection in childhood, her performance at school, her adult height, her earnings, her blood pressure at fifty, and perhaps even the health of grandchildren she will never meet. Medicine has a name for this long shadow: the first 1,000 days, the period from conception to a child's second birthday, during which the human body and brain are assembled faster than at any other time of life. During roughly 270 days of gestation and two years of infancy, an organism builds its skeleton, its immune repertoire, its gut microbiome, and a neural network that will perform a lifetime of thought—all from raw materials supplied almost entirely by the mother's body, her diet, her milk, and her care.

The concept entered global health consciousness through two converging streams. The first was epidemiological: in the 1980s David Barker, studying early-twentieth-century birth records in Hertfordshire, noticed that the districts with the highest infant mortality also had the highest rates of adult death from coronary heart disease, and that low birth weight predicted cardiovascular disease decades later. His "fetal origins" hypothesis—now the Developmental Origins of Health and Disease (DOHaD)—proposed that undernutrition in utero permanently programmes metabolism toward thrift, raising later risk of obesity, type 2 diabetes, and hypertension. The second stream was practical nutrition science, which showed that a small set of nutrients at precise moments—folate in the first weeks, iron and iodine through gestation, vitamin K at birth, long-chain fats during lactation—could prevent specific, devastating harms. Together they reframed public health: the greatest returns on the health of any population are earned before that population can vote, work, or even walk.

2. An Anatomy of the Window: Why Timing Is Everything

The biology of the first 1,000 days explains both its power and its unforgiving deadlines. The neural tube—the embryonic forerunner of brain and spinal cord—closes between the third and fourth weeks of pregnancy, often before a woman knows she is pregnant; folate deficiency at exactly that moment produces neural tube defects, and adequate folate can prevent roughly seven in ten of them. By the end of the first trimester the brain is already producing neurons at a furious rate; the organ grows from an estimated ten thousand cells at four weeks to ten billion by mid-gestation. After birth the tempo remains extraordinary: by age two the brain has reached about 80% of its adult size, and across the whole window it can forge on the order of a thousand new synaptic connections per second—twice as many, at the peak, as the adult brain

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manages—consuming two-thirds of the body's resting energy budget while doing so. Each of these feats has a nutritional prerequisite, and each has a closing date. The immune system is similarly time-bound: antibody transfer across the placenta, colonization at delivery, and the calibration of tolerance in the first months all depend on maternal diet, mode of delivery, breastfeeding, and environmental hygiene. The body's metabolic set-points—blood pressure, appetite regulation, insulin sensitivity—are likewise dialled during gestation and early infancy under epigenetic control: methylation and histone modifications that switch genes on or off without altering DNA sequence, and some of which appear transmissible to the next generation. This is the molecular meaning of DOHaD: environment becomes biology, and biology becomes biography. Stunting—chronic undernutrition in these years—perfectly captures the irreversibility: it leaves survivors shorter as adults, with lower schooling attainment, lower adult income, and reduced offspring birth weight, a transgenerational echo of a nutritional failure that ended before the child's third birthday.



Figure 1. Maternal nutrition during pregnancy: the raw materials of the first 1,000 days are supplied by what a mother eats long before her child's first meal. (Image: UNC Health Talk.)

Nutrient	Role in the first 1,000 days	Consequence of deficiency	Key sources
Folate	Neural tube closure and early brain/spine development; cell division; blood formation	Neural tube defects (spina bifida, anencephaly)—preventable in ~70% of cases	Leafy greens, beans, fortified grains; supplementation before and in early pregnancy

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		with adequate intake	
Iron	Oxygen delivery; myelin formation; dopamine metabolism; child cognition and behaviour	Maternal anaemia; irreversible deficits in learning, attention, and social-emotional behaviour	Meat, eggs, legumes, fortified cereals; supplements as indicated
Iodine	Thyroid hormone synthesis—essential to fetal brain development	Impaired brain function, delayed development, preventable intellectual disability	Iodized salt, dairy, eggs, seafood
Zinc	Cell growth, immune function, taste perception; supports neurodevelopment	Increased infection risk, poor growth, developmental delay	Meat, shellfish, legumes, nuts, whole grains
Choline	Cell membranes; neural tube closure; lifelong memory function	Impaired brain development, neural tube defects, memory deficits	Eggs, meat, liver, breastmilk
Vitamin D	Bone mineralisation; immune regulation; calcium absorption	Rickets, thin/brittle bones, impaired immune function; breastmilk is low—supplementation advised	Sunlight, fortified milk, fish, supplements

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Vitamin A	Vision, cell differentiation, organ formation, immune function	Eye damage, poor growth, infection susceptibility	Egg yolks, orange and dark-green vegetables, liver
Long-chain omega-3 (DHA)	Structural fat of brain and retina; synapse formation; attention and learning	Poor weight gain, lowered immunity, attention and learning problems	Oily fish (salmon, sardines), breastmilk; maternal intake during pregnancy/lactation
Protein	Construction of all tissues, muscle, organs, and neurotransmitters	Fatigue, infection, failure to thrive	Eggs, dairy, fish, legumes, meat
Vitamin K	Blood clotting; all infants are born deficient	Vitamin K deficiency bleeding—potentially fatal; single injection at birth is standard	Single prophylactic dose at birth

Table 1. Critical nutrients of the first 1,000 days, their roles, deficiency consequences, and sources.

3. The Journey in Three Stages

The window is best understood as three linked chapters. In pregnancy, maternal nutrition, weight gain, and health behaviours set the trajectory. The evidence includes some surprises: the "Goldilocks" principle applies to gestational weight gain—too little raises the risk of small babies, too much programmes childhood obesity—and maternal diet shapes taste preferences before birth, as flavours of the mother's meals reach the fetus through amniotic fluid. Smoking, alcohol, chronic stress, and untreated infection each leave measurable imprints, including paradoxes such as the higher later obesity risk in babies of mothers who smoked during pregnancy despite their smaller size at birth. In infancy, the first six months belong to exclusive breastfeeding wherever possible: breastmilk delivers perfectly matched nutrition, immune factors, and long-chain fats, and is associated with lower infection rates and better cognitive outcomes. At around six months, complementary feeding must arrive on time—delay wastes a

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period of rapid growth, while early introduction of sugary or salty foods can programme a lifelong preference for them. The toddler years complete the arc: children aged one to three need about 7 mg of iron daily, and without iron-rich foods roughly a quarter fail to meet requirements—one reason this window, not the school years, is when iron deficiency does its permanent damage to cognition. Table 2 summarises the stage-by-stage priorities.



Figure 2. Breastfeeding in the first 1,000 days: breastmilk is the single most complete nutritional intervention of early childhood, and its benefits depend on support—maternity leave, lactation care, and workplace flexibility. (Image: Ethos Hospital.)

Stage	What is being built	Priority interventions
Pregnancy (conception to birth)	Neural tube and brain; organs; skeleton; immune transfer; metabolic set-points	Preconception folate; balanced diet with iron, iodine, DHA, choline; "Goldilocks" weight gain; treat anaemia and infection; avoid tobacco and alcohol; manage stress; prenatal care

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Infancy: 0–6 months	Rapid brain growth to 80% of adult size; immune system maturation; gut colonisation	Exclusive breastfeeding (or appropriate formula); vitamin D for breastfed infants; vitamin K injection at birth; responsive caregiving; timely immunisation
Infancy and toddlerhood: 6–24 months	Synaptic density and myelination; motor and language skills; food preferences; iron stores; social-emotional development	Timely, nutrient-dense complementary feeding (iron- and zinc-rich foods); continued breastfeeding to 2 years and beyond; limit added sugar and salt; nurturing care and early stimulation; safe water, sanitation, and hygiene

Table 2. The three stages of the first 1,000 days and their priority interventions.

4. Beyond the Plate: Care, Stress, Water, and the Microbiome

Nutrition is necessary but not sufficient. The developmental science of the window identifies three fundamental supports every child needs—healthy nutrition, consistent responsive interactions with caregivers, and safe, caring environments—and shows that when one fails, the others cannot fully compensate. Chronic poverty and maternal depression degrade the quality of early interaction just as surely as food insecurity degrades growth: fetal exposure to elevated stress hormones can alter the child's own stress-response system for life, and families in economic crisis have measurably fewer of the back-and-forth exchanges that build language. Water, sanitation, and hygiene form the second silent pillar—diarrhoeal disease in these years creates a vicious cycle in which infection strips nutrients faster than feeding can replace them, and stunting tracks environmental enteropathy as much as calorie counts. The microbiome, seeded at birth and shaped by breastfeeding and diet, mediates immune training and even metabolic programming. And the cascade runs both ways: maternal mental health is itself a nutrition issue, since depression suppresses breastfeeding initiation and caregiving energy. Modern early-childhood frameworks therefore speak of "nurturing

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care"—a package of nutrition, health, responsive caregiving, early learning, and safety—rather than calories alone. A child can be fed and still fail to thrive.



Figure 3. Food safety and dietary guidance for pregnancy—an illustration of how clinical nutrition counselling operationalises the first 1,000 days in everyday practice. (Infographic: Johns Hopkins Medicine.)

5. The Arithmetic of Neglect

The numbers convert tragedy into policy. Globally, about 23% of children under five are stunted—chronic undernutrition made visible as short stature but measured in lost cognition, weakened immunity, and curtailed schooling. The Food and Agriculture Organization of the United Nations estimates the annual productivity loss from malnutrition at roughly $1 trillion. Set against this, the economics of intervention are almost embarrassingly favourable: analyses across nutrition-sensitive programmes find that every dollar invested in nutrition during the first 1,000 days can return up to $23—a benefit-cost ratio that infrastructure, education, and most health interventions never approach. The failure is not knowledge but delivery: prenatal care that arrives late, breastfeeding abandoned for lack of maternity protection, micronutrient programmes that cover only a fraction of the vulnerable, and food systems that make nutritious diets more expensive than calorie-dense ones. In high-income countries the picture is subtler but real—iron deficiency in a quarter of toddlers, one in five babies never breastfed,

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rising childhood overweight before the second birthday—and the same logic applies: the cheapest diseases to prevent are the ones that begin before the first word is spoken.

6. Conclusion

The first 1,000 days is where prevention stops being a slogan and becomes anatomy. A neural tube that closes on folate or fails without it; a brain that builds a thousand connections a second or builds them on iron-deficient myelin; an immune system trained by breastmilk and clean water or diarrhoea-battered into stunting; a stress-response system calibrated by maternal calm or poverty—these are not metaphors but mechanisms, documented from Hertfordshire birth ledgers to cord-blood methylomes. The science has done its part: it has identified the window, the nutrients, the deadlines, and the returns. What remains is the translation—clinics that reach mothers before conception, food systems that make healthy choices affordable, workplaces that let mothers breastfeed, and water that does not steal a child's growth. A newborn's birth weight will indeed follow her all her life. The question the first 1,000 days asks of every health system is simply this: will that number have been built for her, or merely happen to her?

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HYGIENE IN CROWDED AREAS: RISKS, PREVENTIVE MEASURES, AND PRACTICAL PRECAUTIONS

Dr Saloni Jain , Assistant, Samarkand state medical university, Uzbekistan

Mohd Aleem , Medical Student, Samarkand state medical university, Uzbekistan

Mohd Tohid , Medical Student , Samarkand state medical university, Uzbekistan

Abideen Zainul , Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Crowded settings such as schools, public transport, markets, workplaces, shelters, dormitories, and healthcare waiting rooms bring people into close contact and can make basic hygiene harder to maintain. When clean water, toilets, handwashing facilities, waste collection, ventilation, or cleaning services are inadequate, the risk of infectious disease transmission and environmental contamination may increase. The level of risk depends on the pathogen, duration and closeness of contact, indoor air quality, sanitation infrastructure, and the health of people present. This narrative review summarizes practical hygiene measures for crowded environments, emphasizing hand hygiene, respiratory etiquette, ventilation, safe water, sanitation, food safety, environmental cleaning, waste management, and organization of shared spaces. Evidence-informed guidance from the World Health Organization and the Centers for Disease Control and Prevention supports a layered approach rather than reliance on any single precaution. Facility managers should make hygiene resources accessible, develop cleaning and maintenance routines, communicate in clear and inclusive language, and plan for outbreaks without stigmatizing affected people. The article includes implementation tables and a qualitative priority graph. The graph is a planning aid, not a measured comparison of intervention effectiveness. Safer crowded environments depend on both individual habits and the systems that make those habits possible.

Keywords: crowded areas; hygiene; infection prevention; hand hygiene; ventilation; sanitation; respiratory etiquette; public health; environmental cleaning

1. Introduction

Crowded places are part of everyday life. People gather in classrooms, buses, railway stations, markets, offices, dormitories, shelters, religious venues, and healthcare facilities. These settings support education, work, travel, and social connection, but high occupancy can complicate hygiene and infection prevention. Shared surfaces are

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touched by many people, toilets may be used frequently, waste can accumulate, and indoor air may become stale when ventilation is poor. Crowding itself does not guarantee that illness will spread; risk depends on the type of exposure and whether practical safeguards are available.

Hygiene in crowded settings should be understood as a shared responsibility. Individuals can clean their hands and cover coughs, but these actions are difficult if soap, water, bins, or tissues are unavailable. Asking people to avoid crowding is not always realistic where transport, housing, or essential services are limited. Public-health planning must therefore combine personal habits with infrastructure, maintenance, communication, and fair access to facilities [1–4].

2. Why hygiene becomes challenging in crowded areas

Infectious agents spread through different routes. Respiratory pathogens may be transmitted through particles released when an infected person breathes, talks, coughs, or sneezes; poorly ventilated indoor spaces can allow infectious particles to accumulate. Some gastrointestinal infections spread through contaminated hands, food, water, or surfaces. Other pathogens may spread through direct contact or contaminated equipment. Because routes differ, a single intervention cannot prevent every infection.

Crowded facilities may face operational problems: too few handwashing points, unreliable water supplies, toilets that are not cleaned often enough, insufficient waste bins, and queues that bring people into close contact. Inadequate ventilation can be less visible than dirty surfaces but is especially relevant for airborne respiratory infections. Effective prevention starts with identifying the specific risks in each setting and choosing a combination of controls that is realistic and consistently maintained [1,3,4].

Challenge	Potential consequence	Practical response
High occupancy and close contact	More opportunities for close-range exposure	Improve flow and queue design; reduce avoidable crowding where feasible.
Poor indoor ventilation	Respiratory particles may accumulate indoors	Bring in outdoor air where safe; maintain ventilation systems; consider appropriate air cleaning.
Limited handwashing access	Hands may remain contaminated after key activities	Provide convenient sinks, soap, clean water, drying materials, or sanitizer where suitable.

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Overused toilets and shared facilities	Contamination and unpleasant or unsafe conditions	Set inspection and cleaning schedules; promptly repair water and plumbing failures.
Poor waste collection	Overflow, pests, odors, and contaminated surroundings	Provide suitable bins and reliable collection.
Unclear communication	Precautions may be misunderstood or ignored	Use short, visible, multilingual instructions and demonstrate key practices.

3. Core hygiene precautions

3.1 Hand hygiene

Hand hygiene is one of the most practical ways to reduce the transfer of germs. In community settings, people should wash with plain soap and water before preparing or eating food, after using the toilet, after coughing or sneezing or blowing the nose, when hands are visibly dirty, and after handling waste or contaminated materials. Hands should be rubbed thoroughly so that all surfaces are cleaned, then rinsed and dried. When hands are not visibly dirty and a sink is unavailable, an alcohol-based hand rub can be a useful alternative; soap and water remain important when hands are visibly soiled and for some specific pathogens [1,2].

Managers should place handwashing facilities near toilets, food preparation and eating areas, entrances, and other high-use points. Supplies need to be replenished rather than installed only for appearance. A sink without water or soap does not provide effective access. Where alcohol-based sanitizer is supplied, it should be stored and used safely.

3.2 Respiratory hygiene and masks

People should cover their mouth and nose with a tissue when coughing or sneezing, discard the tissue into a bin, and clean their hands afterward. If a tissue is unavailable, coughing or sneezing into the elbow is preferable to using bare hands. Anyone who develops respiratory symptoms should, where possible, limit close contact with others, improve ventilation, and consider wearing a well-fitting mask in shared indoor spaces, particularly around people at increased risk. Mask recommendations can vary with

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local transmission, setting, and public-health guidance; masks work best as part of a layered approach rather than as a substitute for ventilation or staying away from others when ill [2–4].

3.3 Ventilation and indoor air

Fresh air reduces the concentration of some airborne contaminants. Depending on the building and outdoor conditions, ventilation may be improved by opening windows or doors, using properly maintained mechanical ventilation, or arranging activities outdoors when feasible. Air cleaners with appropriate filtration may provide additional benefit in some rooms, but they do not replace adequate outdoor-air ventilation or other precautions. Fans should be positioned thoughtfully so that air is not simply blown from one person toward another. Building operators should maintain systems according to qualified technical advice and local requirements [3,4].

3.4 Safe water, toilets, and sanitation

Reliable access to safe water and usable toilets is fundamental to public hygiene. Facilities should have a clear schedule for inspection, cleaning, replenishment of soap and toilet paper, and repair of broken taps, flushes, drains, or ventilation. Toilets should protect privacy, accessibility, and dignity. Drinking water should be obtained from a safe source and stored in clean, covered containers where needed. If water safety is uncertain, local public-health authorities should advise on appropriate treatment and use.

3.5 Cleaning shared surfaces and equipment

Frequently touched surfaces such as door handles, railings, desks, taps, counters, shared keyboards, and transport handholds should be cleaned regularly, with frequency adjusted to use and contamination risk. Cleaning with detergent removes dirt and many germs. Disinfection may be needed for certain settings, pathogens, or contamination events; it should follow product-label instructions and local guidance. More chemical use is not automatically better: products must be used safely, never mixed unless the label explicitly allows it, and kept away from food and children. Shared equipment should be cleaned between users when appropriate.

3.6 Food hygiene and waste management

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Crowded food areas need safe sourcing, clean preparation surfaces, separation of raw and ready-to-eat foods, adequate cooking and storage temperatures, and staff access to handwashing. Food handlers who are ill with symptoms that may contaminate food should follow workplace and public-health rules before returning to food preparation. Waste bins should be easy to reach, emptied before overflowing, and cleaned when soiled. Waste should be separated and handled according to local rules, especially in healthcare settings where regulated waste may require special procedures.

Measure	Recommended practice	Who can support it
Hand hygiene	Soap and water at key locations; sanitizer where appropriate; regular supply checks.	Facility management, cleaners, users.
Respiratory etiquette	Tissues, bins, visible reminders, and practical guidance for symptomatic people.	All visitors and staff.
Air quality	Ventilation checks, fresh-air pathways, maintenance, and appropriate filtration where useful.	Building operators and trained technicians.
Toilets and water	Documented inspection, cleaning, replenishment, and repair process.	Facility management and sanitation staff.
Surface cleaning	Risk-based schedule, appropriate products, staff training, and safe storage.	Cleaning teams and supervisors.
Food and waste	Safe handling, temperature control, suitable bins, and timely collection.	Food handlers, vendors, waste services.

4. Organizing crowded spaces more safely

Good hygiene is easier when the environment supports it. Where practical, facilities can stagger appointments or breaks, use clear entry and exit routes, reduce bottlenecks, provide waiting areas with adequate space, and place signs before people enter a queue. People with respiratory symptoms should be able to notify staff and wait separately from others when feasible, without being shamed or denied necessary services. In

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healthcare facilities, staff should follow current infection-prevention guidance, including appropriate precautions for the suspected pathogen.

Communication should be simple and respectful. Posters should use readable text and clear illustrations; messages may need translation or accessible formats. Staff should explain not only what to do but why the measure matters. Supplies should be available to people who cannot easily purchase them, and policies should consider older adults, people with disabilities, children, and those who rely on crowded transport or shared housing.

5. Implementation checklist for facility managers

Area	Routine check	Action if a gap is found
Entrances and waiting zones	Are signs, bins, tissues, and hand hygiene supplies available?	Restock and place supplies where people can reach them.
Air and ventilation	Are windows, vents, and mechanical systems functioning as intended?	Report faults; consult qualified building staff; use safe temporary measures.
Handwashing stations	Are water, soap, and drying materials available?	Repair or replenish promptly; provide an alternative nearby.
Toilets	Are facilities clean, accessible, stocked, and functioning?	Clean, restock, and log repairs or recurring capacity problems.
Shared surfaces	Is a written, risk-based cleaning schedule followed?	Clarify responsibility, train staff, and record completion.
Food and waste	Are food handling and bin collection practices safe?	Correct unsafe storage, remove overflowing waste, and review collection frequency.
Outbreak readiness	Is there a process for symptoms, reporting, and local guidance?	Update contact lists and procedures with public-health advice.

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6. Graph: prioritizing hygiene measures

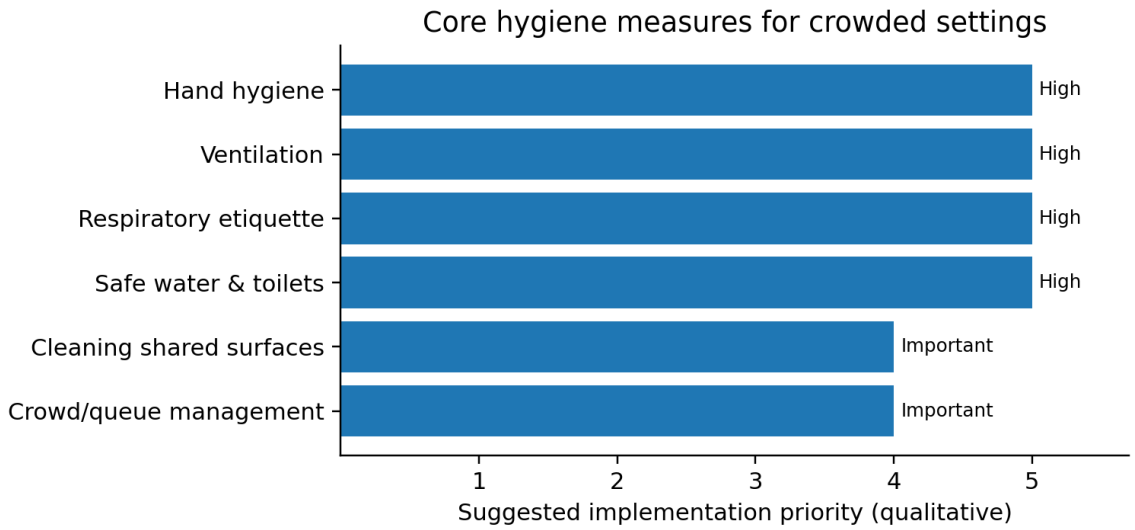


Figure 1. Qualitative planning illustration of core measures for crowded settings. The ratings are not research-derived effect sizes and should not be interpreted as a numerical ranking of efficacy. Actual priorities should be set after a site-specific risk assessment.

7. Barriers and practical solutions

Common barriers include limited budgets, unreliable water supply, staff shortages, poor building design, low risk awareness, and inconsistent maintenance. These barriers are often connected: an overloaded toilet block may need both more frequent cleaning and improved capacity. A realistic plan assigns responsibility, records supply and cleaning checks, and includes a route for reporting problems. Small changes can be useful, but structural problems require investment rather than repeated reminders to individuals.

Hygiene measures should also be proportionate. Excessive disinfection, unnecessary restrictions, or blaming particular groups can waste resources and undermine trust. Facilities should choose measures based on likely transmission routes, local public-health advice, and the needs of people using the space. During outbreaks, guidance may change as evidence develops; managers should use current official recommendations instead of relying on old posters or informal messages.

8. Conclusion

Maintaining hygiene in crowded areas requires a combination of personal precautions, suitable infrastructure, reliable cleaning, and thoughtful organization of shared spaces. Hand hygiene, respiratory etiquette, clean air, safe water and toilets, food safety, and

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effective waste management address different routes by which infections and environmental hazards may spread. No single measure is sufficient in every situation, and the most appropriate approach depends on the setting and the risks present. Institutions should make good hygiene practical by providing supplies, maintaining buildings, training staff, and communicating clearly and respectfully. A planned, layered approach can make schools, transport hubs, markets, workplaces, shelters, and healthcare facilities safer while preserving access and dignity.

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MICROPLASTICS AS POTENTIAL VECTORS OF ANTIMICROBIAL RESISTANCE IN HOSPITAL WASTEWATER: FROM RESISTOME DETECTION TO EVOLUTIONARY RISK

Nori Leeza Abdulhamid , Medical Student, Samarkand state medical university, Uzbekistan

Kumhar Avinash , Medical Student, Samarkand state medical university, Uzbekistan

Kabra Vishal , Medical Student, Samarkand state medical university, Uzbekistan

Sharma Prince , Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Antimicrobial resistance (AMR) in hospital wastewater develops within a highly heterogeneous environment. Along with resistant bacteria and antimicrobial resistance genes, these waste streams can contain mobile genetic elements, residual antimicrobials, disinfectants, heavy metals, and other chemical pollutants. The presence of microplastics adds another potentially important component to this system. Their surfaces can be colonized by microorganisms and support biofilm development, while the particles may also retain extracellular DNA and interact with chemicals present in the surrounding wastewater. These properties suggest that microplastics could alter the local conditions under which resistant microorganisms persist and resistance determinants are exchanged.Studies indicate that microplastic-associated biofilms can harbour antimicrobial resistance genes, virulence-associated determinants, mobile genetic elements, and prophage-associated sequences. Microplastic-associated biofilms may increase opportunities for horizontal gene transfer by promoting cell proximity and retention of extracellular genetic material.

Finding resistance genes on microplastics does not necessarily mean that they pose a clinical risk. What matters is whether the particles help resistant bacteria survive, exchange resistance genes, or maintain these genes in the wastewater environment. Microplastic-associated biofilms therefore deserve attention as active sites of resistance exchange. Current research is examining how pollutants, wastewater treatment, and microbial communities interact to shape these resistance patterns.

Current data suggest that Current evidence suggests that microplastics can provide surfaces that concentrate microorganisms, extracellular genetic material, and chemical

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contaminants within localized biofilm-associated microenvironments. However, the extent to which these interactions increase clinically relevant antimicrobial resistance under environmentally realistic conditions remains uncertain. Future research should move beyond just finding new genes and instead focus on tracking real risks. This means studying how plastic properties, certain bacteria, gene movement, protein activity, and environmental persistence relate to actual clinical threats.

Keywords: antimicrobial resistance, hospital wastewater, microplastics, plastisphere, resistome, mobilome, horizontal gene transfer, genomic epidemiology, wastewater treatment, One Health.

1. Introduction

Antibiotic resistance is no longer limited to hospitals and clinical settings. Resistant bacteria and resistance genes are now being detected in many different environments, including wastewater systems, rivers and lakes, soil, food production, and other natural settings. Hospital wastewater is particularly important because it contains a complex mixture of antibiotics, resistant bacteria, resistance genes, strong disinfectants, pharmaceuticals, metals, free DNA, and other biologically active pollutants. Together, these conditions create an environment where bacterial selection, survival, close microbial contact, and genetic exchange can occur simultaneously.

Microplastics can act as surfaces on which wastewater microorganisms attach and grow. Once present in wastewater, these particles become coated with organic material and are gradually colonized by bacteria and other microorganisms. The resulting biofilm is often described as the plastisphere.

The importance of this plastic-associated environment has increased as accumulating evidence links microplastics to the dissemination of antimicrobial resistance. Experimental studies show that biofilms growing on plastic surfaces can increase uptake of extracellular DNA carrying resistance genes, while smaller, weathered plastic particles may promote substantially higher rates of gene transfer. Recent reviews have identified horizontal gene transfer, the accumulation of selective chemical compounds, and chemical co-selection as important mechanisms through which microplastics may contribute to the spread of antimicrobial resistance.

Recent research on hospital wastewater has advanced this field. A 2026 study combining incubation experiments, metagenomics, and genome reconstruction uncovered 68 resistance genes, 443 mobile elements, 414 virulence factors, and 129 prophages living right on colonizing microplastics. In addition, the researchers

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reconstructed 360 metagenome-assembled genomes and found that mobile genetic elements accounted for a major share of the variation in those resistance genes. e.g. **[2]**

Nevertheless, an important conceptual problem remains. The presence of an ARG does not necessarily mean it is mobile or can be transmitted to another bacterial host**.** An ARG may be extracellular, incomplete, transcriptionally inactive, located in a non-mobile genomic region, or present in a microorganism with little opportunity to transmit to clinically relevant hosts.

ARG detection → bacterial host → MGE association → genetic mobility → expression → phenotypic resistance → persistence → successful transfer → clinical linkage

This framework distinguishes environmental resistome detection from actual evolutionary and epidemiological risk.

This review therefore aims not simply to summarise ARG abundance on microplastics, but to examine whether the plastisphere can alter the biological conditions that govern AMR persistence, mobility, and dissemination.

2. Hospital Wastewater and the Plastisphere as an Evolutionary Interface

Hospital wastewater brings together antibiotics, disinfectants, metals, resistant bacteria, and free DNA. Microplastics can become colonized by these bacteria and develop biofilms. The resulting matrix can keep cells, DNA, and chemicals in close proximity, which may favour the transfer of resistance genes between bacteria.

Three potential ecological roles can therefore be distinguished:

1. **Reservoir** — accumulation of pre-existing ARGs and resistant organisms.
2. **Selective niche** — enrichment of microorganisms able to tolerate local chemical and ecological conditions.
3. **Evolutionary interface** — modification of the physical and chemical conditions that facilitate genetic exchange.

Evidence for the reservoir and selective-niche roles is currently stronger than evidence for a general evolutionary-amplification effect. The distinction is critical. If microplastics merely concentrate organisms that are already present in wastewater, their ecological role differs fundamentally from a situation in which they independently increase HGT or the emergence of resistant phenotypes.

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Recent reviews suggest several plausible mechanisms for AMR selection within the plastisphere, including increased cell proximity, retention of extracellular DNA and adsorption of antimicrobial or co-selective compounds.

Researchers need to determine whether ARGs behave differently in microbial communities growing on microplastics compared with those growing without them.

3. Microplastic Aging as an Emerging AMR Determinant

Microplastics do not remain chemically or physically unchanged after entering wastewater. Environmental aging can produce oxidation, fragmentation, changes in surface roughness, altered surface charge, and changes in functional groups. These modifications can influence microbial attachment, biofilm development and contaminant adsorption.

Importantly, a particle's environmental history may therefore be more informative than its polymer identity alone.

A useful experimental classification is:

pristine particle → wastewater-exposed particle → aged particle → fragmented particle

These stages should be evaluated for:

- microbial colonisation;
- biofilm succession;
- ARG abundance;
- ARG–MGE association;
- extracellular DNA retention;
- bacterial host composition;
- HGT frequency;
- resistance phenotype.

Experiments already show that a plastic particle's age and size can strongly affect how easily resistance genes are transferred. For instance, one study found that smaller, weathered microplastics increased gene transfer frequencies much more than larger, brand-new plastic particles.

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A 2024 review further proposed that photoageing can increase surface functional groups and sorption capacity, potentially modifying interactions between antibiotics, ARGs and microbial communities.

However, increased adsorption does not automatically mean increased biological risk. A chemical compound adsorbed to a particle must remain sufficiently bioavailable to exert selective pressure. Similarly, increased ARG adsorption does not necessarily translate into successful genetic transfer.

Therefore, aging research should move beyond adsorption measurements and ask whether aging changes the **probability of functional resistance transfer**.

4. Spatial Structure of the Plastisphere

A major limitation of conventional plastisphere research is its reliance on bulk community measurements.

A mature biofilm is spatially heterogeneous. Oxygen, nutrients, pH, redox potential and contaminant concentrations can differ over very small distances.Consequently, bacterial populations occupying different microenvironments on the same particle may experience substantially different physicochemical conditions.

This spatial organization may influence AMR in at least three ways.

First, localized high cell density can increase donor–recipient contact.

Second, extracellular polymeric substances may retain DNA and modify its accessibility.

Third, chemical conditions may vary across the particle surface, creating small areas where resistant bacteria have a selective advantage. Gene transfer may therefore be more common in some parts of the biofilm than others. Future studies should combine metagenomic data with:

- confocal microscopy
- single-cell genomics
- fluorescence-based localization
- microscale chemical mapping
- spatial transcriptomics where technically feasible.

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The critical objective is to determine whether **ARG-bearing organisms, recipient cells and MGEs spatially converge** within the same microenvironments.

This would provide stronger mechanistic evidence than bulk ARG abundance alone.

5. From Resistome to Mobilome

The most important conceptual limitation of ARG-centred studies is that **gene abundance does not equal gene mobility**.

An ARG may be abundant but genetically isolated. Conversely, a low-abundance ARG located on a highly mobile plasmid in a clinically relevant bacterial host may represent a greater evolutionary concern.

An ARG count does not show its host or genetic location. Microplastic-associated communities can contain resistance genes within different bacterial and mobile-element backgrounds. Linking these components requires methods such as long-read sequencing, genome reconstruction, and Hi-C analysis.

Therefore, **culture-based validation, conjugation assays, transformation experiments and phenotypic antimicrobial susceptibility testing** remain important where feasible.

A particularly useful future endpoint would be the identification of ARGs that are simultaneously:

1. present
2. associated with a bacterial host
3. linked to an MGE
4. expressed
5. phenotypically functional
6. transferable
7. persistent under environmentally relevant conditions.

6. Prophages: An Understudied Route of Resistance Mobility

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Most environmental AMR studies have concentrated on plasmids and integrons. Phages and prophages have received much less attention, despite their potential role in resistance-gene transfer.

That picture is changing. A recent hospital wastewater study identified 129 prophage-associated sequences within microplastic-associated microbial communities. In addition, independent large-scale analyses of hospital wastewater have shown a wide variety of phages, found resistance genes linked directly to them, and mapped potential phage-and-host connections.

Finding prophage DNA is not enough to confirm that phages are transferring resistance genes. Experiments are needed to test phage activation, resistance-gene packaging, release of phage particles, and transfer after treatment.

Resolving these questions matters because plasmid-mediated mechanisms alone may not capture the full range of genetic pathways driving environmental AMR dissemination.

7. Chemical Co-selection in the Plastisphere

- **potential enhancement of horizontal gene transfer within dense biofilms**
- **concentration of chemicals capable of selecting or co-selecting for resistance**
- **retention or gradual release of plastic-associated chemical additives**

Experimental studies have also found complex interactions among heavy metals, disinfectants, and resistance genes within these plastisphere communities.

However, the interpretation requires caution.

Total chemical burden and microbial exposure are not equivalent. Selection depends on the fraction of a compound that remains accessible to microorganisms and is present at a biologically active level.

Therefore, future studies should examine environmentally realistic mixtures such as:

antibiotics + disinfectants + metals + pharmaceuticals + organic contaminants

and measure:

- gene expression
- ARG enrichment
- MGE abundance

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- HGT frequency
- microbial succession
- resistance phenotype
- bacterial fitness.

Mixture experiments should also test antagonistic and synergistic interactions because environmental chemical effects are not necessarily additive.

8. Wastewater Treatment: Are We Destroying Resistance, or Just Moving It Around?

Looking only at the liquid effluent does not give a complete picture of what happens to ARGs during wastewater treatment. A decrease in ARGs in the water phase may not mean that they have been destroyed. Some may instead become associated with suspended solids, sludge, or microbial communities growing on microplastics. For this reason, studies of ARG removal should consider the water, particulate matter, microplastic-associated biomass, and sludge together. This can help determine whether resistance determinants are actually degraded or simply removed from the water and redistributed elsewhere in the treatment system.

raw influent→ biological tanks→ disinfection→ sludge→ treated discharge

9. The Phenotype Gap: Finding DNA Doesn't Mean Finding Trouble

Most environmental AMR studies rely heavily on molecular detection of resistance-associated DNA in wastewater, sludge, and associated particulate compartments. However, DNA detection alone does not establish functional resistance.

DNA detection ≠ gene expression ≠ resistance phenotype

Genomics — identification of resistance-associated genetic determinants and their genomic context.

Transcriptomics — assessment of resistance-gene transcription and functional activity.

Culture-based and phenotypic testing — determination of antimicrobial susceptibility and functional resistance.

A stronger framework combines:

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metagenomics + metatranscriptomics + culture-based susceptibility testing

where feasible.

This is especially important for novel ARGs whose clinical relevance cannot be established through sequence similarity alone.

For a resistance determinant to be considered high-confidence environmental AMR evidence, ideally there should be evidence of:

sequence → host → mobility → expression → phenotype

10. The Environmental-to-Clinical Transmission Gap

The presence of ARGs on microplastics does not establish a clinical connection. Evidence is still needed to identify the bacterial hosts, confirm gene activity and transfer, and trace the same resistance determinants between environmental and clinical isolates.

11. Particle-Specific AMR Risk

Treating all microplastics as one exposure category may obscure important biological differences.

Relevant characteristics include:

Polymer composition	Surface chemistry and microbial attachment
Particle size	Surface-to-volume ratio and cell concentration
Ageing	Surface transformation and contaminant adsorption
Roughness	Biofilm attachment and spatial organization
Biofilm maturity	Cell proximity and genetic exchange
Chemical loading	Local selective pressure
Morphology	Hydrodynamic behaviour and colonization

Experimental evidence indicates that polymer characteristics, size, and aging can influence transformation and ARG-associated processes.

Therefore, future risk assessments should develop **particle-specific AMR profiles** rather than treating all microplastics as biologically equivalent.

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A particle with high surface roughness, advanced aging, mature biofilm, and substantial chemical loading may have a different ecological function from a pristine particle with limited colonization.

12. Establishing Causality

Methodological Challenge

Wastewater studies alone make it difficult to determine whether microplastics drive changes in resistance or simply provide another surface on which the existing microbial community can accumulate. A useful way to address this problem would be to examine the same microbial system without particles and after exposure to pristine microplastics, aged microplastics, and non-plastic particles. Alongside ARG abundance, the analysis should consider transcriptional activity, ARG linkage to mobile genetic elements, HGT, antimicrobial susceptibility, bacterial fitness, and persistence.

It is still unclear whether microplastics have a role in AMR beyond that of ordinary particles found in wastewater. Bacteria can colonize almost any suitable surface, so the presence of a plastisphere alone is not evidence of a unique resistance effect. The comparison becomes more informative when microplastics and natural particles are exposed to the same wastewater conditions and examined for differences in contaminant binding, microbial composition, and gene transfer. Such experiments would help determine whether the plastic surface itself changes resistance dynamics or simply provides another place for microorganisms to accumulate.

13. Priority Future Research

Priority Areas for Future Research

Several areas require further investigation before the contribution of microplastics to environmental AMR can be clearly established. First, studies should examine whether aging changes microplastics' ability to support ARG transfer, rather than simply increasing microbial attachment or adsorption of genetic material. Comparing pristine and environmentally aged particles under similar conditions would help resolve this question.

A second priority is to improve the resolution of ARG–MGE–host relationships. Identifying which resistance genes are physically associated with mobile genetic

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elements and which bacterial hosts carry them would provide a more meaningful indication of their potential for dissemination than ARG abundance alone.

The possible contribution of prophages also deserves greater attention. Detecting prophage-associated resistance genes does not demonstrate active transduction, and experimental studies are needed to determine whether these elements can contribute to resistance transfer within plastisphere communities.

The spatial organization of the plastisphere is another important area for study. Microscale measurements could help determine whether local conditions such as nutrient availability, chemical gradients and high cell density bring potential donor and recipient bacteria into closer contact and thereby influence HGT.

Chemical interactions should also be investigated under environmentally relevant conditions. Antibiotics, disinfectants, metals and other contaminants may occur together in hospital wastewater, and their combined effects on resistance selection, microbial community structure and bacterial fitness may differ from their individual effects.

Wastewater-treatment studies should follow resistance determinants through the entire treatment process, including water, microplastic and sludge compartments. Tracking ARGs together with their bacterial hosts and associated MGEs would help distinguish actual degradation from physical removal or redistribution.

An ARG identified by sequencing is only a genetic observation. Its biological relevance requires evidence that the gene is transcribed and associated with a resistance phenotype. Researchers can explore a possible link to clinical AMR by comparing environmental samples with patient-derived or healthcare isolates for matching resistance variants and their surrounding plasmid or mobile-element sequences. A genetic match would strengthen the association, but would not establish that the gene moved from the environment into a patient.

The same limitation applies to microplastic-associated AMR. An ARG count says little about the biological context in which the gene occurs. Relevant factors include the particle type, its colonizing organisms, the genetic location and mobility of the ARG, its persistence and expression, and the pathogenic characteristics of its host. Taken together, these data are needed to determine whether microplastics are simply accumulating resistance determinants or are associated with conditions that favour their dissemination.

14. A Next-Generation Research Framework

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A comprehensive investigation of plastisphere-associated AMR can be organized into five analytical levels.

Level 1 — Particle ecology

Polymer, size, morphology, ageing and surface chemistry.

Level 2 — Plastisphere structure

Biofilm development, microbial succession and spatial organization.

Level 3 — Resistance architecture

ARGs, MGEs, prophages, bacterial hosts and virulence determinants.

Level 4 — Functional validation: Assessment should include gene expression, horizontal gene transfer, antimicrobial susceptibility, bacterial fitness, and persistence.

Level 5 — Clinical linkage: Compare environmental findings with contemporaneous clinical isolates using high-resolution genomic epidemiology.

An ARG measurement does not reveal its biological behaviour. The same gene may be inactive in one host and clinically relevant in another, depending on its expression, genetic context, mobility, and persistence. Functional assays and genomic analyses are needed to resolve these differences. Risk assessment should consequently give greater weight to resistance determinants supported by evidence of activity, transfer, persistence, and clinical relevance.

15. One Health Implications and Environmental Infection Prevention

Hospital wastewater provides an important connection between infection control within healthcare facilities and the environmental dimensions of AMR. Once discharged from healthcare facilities, wastewater carries a complex mixture of resistant microorganisms, resistance determinants, antimicrobial compounds, disinfectants, and other chemical residues into environments where these biological and chemical pressures interact with established microbial communities before and during treatment. Microplastics add another component to this environment because they can provide persistent surfaces for microbial colonization and may carry associated microorganisms and genetic material through different stages of the wastewater system.

This relationship can be considered along a broader pathway:

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Healthcare facility → hospital wastewater → treatment system → receiving environment → environmental microbial communities → potential human and animal exposure

Viewing the problem in this way places the hospital-wastewater plastisphere within a One Health context. AMR arising in clinical settings does not necessarily remain confined to the hospital, and resistance detected in the environment cannot be understood independently of its possible human, animal, and healthcare sources. Wastewater therefore represents an important interface where these compartments meet.

Microplastics are only one part of the AMR environment found in hospital wastewater. The same wastewater carries antibiotic residues, resistant bacteria, extracellular DNA, disinfectants, metals, and other pollutants, all of which can alter microbial survival and selection. Reducing the plastic fraction alone would therefore leave many of the processes involved in resistance persistence and dissemination unchanged.

Reducing microplastics addresses only one part of the wastewater AMR problem. Resistance is also shaped by antibiotic exposure, hospital infection-control practices, sanitation, pharmaceutical residues, treatment performance, and the organisms released with treated waste. These factors interact along the route from the healthcare facility to the receiving environment. Monitoring resistance at several points along this route can therefore show where selection or release is occurring and where intervention is most needed.

Hospital wastewater can consequently be viewed as part of a wider environmental infection-prevention system. Monitoring this compartment may provide information not only on the release of resistant organisms and resistance determinants from healthcare facilities, but also on their persistence and redistribution during treatment. Integrating wastewater surveillance with clinical and environmental data could therefore provide a more complete picture of how AMR moves across interconnected One Health compartments.

16. Limitations of the Current Evidence

Despite rapidly increasing research interest, several limitations prevent definitive conclusions regarding the clinical importance of microplastics in AMR dissemination.

First, many studies remain observational or based on laboratory systems that may not reproduce the chemical, microbial and hydrodynamic complexity of hospital wastewater.

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Second, ARG abundance is frequently used as the primary outcome even though abundance does not demonstrate mobility or phenotypic resistance.

Third, metagenomic association between an ARG and an organism does not necessarily demonstrate that the organism carries a functional, transferable resistance determinant.

Fourth, laboratory transformation or HGT experiments may use concentrations, particle sizes or chemical conditions that differ from environmentally realistic exposure.

Environmental studies have detected ARGs and resistant bacteria on microplastics, but clinical relevance has not been established. The missing evidence is a demonstrated link between plastisphere-associated resistance and resistance occurring in patients or healthcare settings. Until that link is shown, the clinical implications of microplastic-associated AMR should remain an open research question.

17. Conclusion

Microplastics are part of the microbial and chemical environment of hospital wastewater, where resistance genes and resistant bacteria can accumulate. Their presence alone does not show that they make resistance spread more efficiently than other particles. The unresolved question is whether the plastisphere has a measurable effect on the behaviour and movement of resistance genes.

This can only be addressed by comparing microplastics with relevant non-plastic particles and examining the organisms, genetic elements, activity, and persistence involved. Clinical genomic data may provide additional evidence about the relevance of environmental findings. Until these links are demonstrated, microplastics should be considered a possible component of environmental AMR rather than a proven cause of clinical resistance.

For now, microplastics should be viewed as one part of the wider wastewater resistome. Their contribution to AMR will become clearer only when ecological observations are supported by functional and epidemiological evidence.

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ANTIMICROBIAL RESISTANCE AND THE ROLE OF HYGIENE IN ITS PREVENTION

Kunnummal Sahla Shirin , Medical Student, Samarkand state medical university, Uzbekistan

Jilani Alia Amir , Medical Student, Samarkand state medical university, Uzbekistan

Mohammed Aysath Rafna , Medical Student, Samarkand state medical university, Uzbekistan

Ifra Ayesha , Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Antimicrobial resistance (AMR) has quietly become one of the most serious threats to modern medicine. Bacteria, viruses, fungi, and parasites are evolving faster than our ability to treat them, turning once-curable infections into dangerous and sometimes deadly conditions. While the development of new drugs is slow and costly, an often-underestimated weapon already exists in our hands — quite literally. This article explores how basic hygiene practices, from handwashing in hospitals to safe food handling at home, interrupt the chain of infection and reduce the need for antibiotics in the first place. Drawing on landmark studies and global reports, it argues that hygiene is not a secondary measure but a frontline strategy in the fight against resistance. The article reviews the mechanisms of resistance, examines evidence for hygiene interventions in healthcare and community settings, and highlights the One Health approach that links human, animal, and environmental health. It concludes that preventing infection through hygiene is one of the simplest, cheapest, and most immediately available tools we have to slow the march of resistant microbes.

Keywords: antimicrobial resistance, hygiene, hand hygiene, infection prevention, antibiotics, One Health, healthcare-associated infections

1. Introduction

Imagine a world where a simple cut on your finger, a routine surgery, or a child's ear infection could once again become life-threatening. For most of human history, that was the reality. The discovery of penicillin in 1928 changed everything, ushering in an era in which bacterial infections were routinely curable. Yet less than a century later, we are drifting back toward that older, darker world — not because antibiotics have

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stopped working altogether, but because the microbes they target have learned to fight back.

Antimicrobial resistance occurs when microorganisms such as bacteria, viruses, fungi, and parasites evolve mechanisms that protect them from the drugs designed to kill them or stop their growth. The World Health Organization (WHO) has declared AMR one of the top ten global public health threats facing humanity. Estimates suggest that drug-resistant infections already cause more than a million deaths each year, and without decisive action, that number could climb dramatically by 2050. Modern medicine itself is at stake: organ transplantation, cancer chemotherapy, caesarean sections, and the care of premature infants all depend on antibiotics that still work.

In the race to solve this crisis, attention has naturally focused on developing new drugs. But antibiotics are expensive to develop and easy for bacteria to outsmart. There is, however, a far older and humbler line of defence — hygiene. Keeping hands, surfaces, water, and food clean stops infections before they begin, which means fewer antibiotics are prescribed, less selection pressure is placed on microbes, and resistant strains have fewer chances to spread. This article examines how hygiene serves as a cornerstone of AMR prevention, weaving together evidence from hospitals, communities, farms, and households around the world.



Figure 1. Antibiotic susceptibility testing: discs impregnated with different antibiotics are placed on a bacterial culture. Clear zones around discs indicate where bacteria

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were killed; small or absent zones signal resistance. (Image: University of Queensland, IMB)

2. How Microbes Become Resistant

Resistance is not a deliberate act by microbes but the result of evolution happening at breathtaking speed. Bacteria reproduce in minutes, and each round of reproduction carries a small chance of random genetic mutations. When an antibiotic enters this picture, it acts as a ruthless filter: susceptible bacteria die, while any mutant that happens to survive multiplies freely. Over time, the surviving population becomes dominated by resistant strains.

Microbes acquire resistance through several routes. Some alter the target site of the drug so the antibiotic can no longer bind effectively. Others produce enzymes that destroy the drug outright — beta-lactamase enzymes that break down penicillins are a famous example. Some bacteria develop efflux pumps that actively push the drug out of the cell, while others simply reduce the permeability of their cell walls. Resistance genes can also jump between bacteria through horizontal gene transfer, meaning one resistant strain can share its survival tools with entirely different species. This is why resistance in a harmless gut bacterium can eventually reach a dangerous pathogen.

Perhaps the most worrying development is multidrug resistance, in which a bacterium resists several classes of antibiotics simultaneously. Pathogens such as methicillin-resistant Staphylococcus aureus (MRSA), carbapenem-resistant Enterobacterales, and extensively drug-resistant Mycobacterium tuberculosis have turned treatable conditions into clinical nightmares. The table below summarizes some of the most concerning resistant pathogens today.

Table 1. Major resistant pathogens of global concern

Pathogen	Notable resistance	Clinical impact
Staphylococcus aureus (MRSA)	Methicillin and other beta-lactams	Severe skin, wound, and bloodstream infections; hard to treat in hospitals
Escherichia coli	Extended-spectrum beta-lactams (ESBL), fluoroquinolones	Urinary tract and bloodstream infections; common in community and hospitals

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Klebsiella pneumoniae	Carbapenems	Pneumonia and sepsis; very limited treatment options remain
Mycobacterium tuberculosis	Rifampicin, isoniazid (MDR/ XDR-TB)	Months-long, toxic treatment regimens; significant mortality
Neisseria gonorrhoeae	Cephalosporins, fluoroquinolones	Rising threat of untreatable sexually transmitted infection

3. What Drives Resistance?

Resistance genes exist in nature, but human behaviour amplifies them dramatically. The overuse and misuse of antibiotics is the primary driver: drugs are prescribed for viral illnesses like colds and flu against which they are powerless, courses are stopped early as soon as patients feel better, and antibiotics are widely available without prescriptions in many countries. In agriculture, antibiotics are used not only to treat sick animals but also to promote growth and prevent disease in healthy herds, creating vast reservoirs of resistant bacteria that can reach humans through food, water, and direct contact.

Poor infection control adds fuel to the fire. In settings where clean water, sanitation, and hygiene are lacking, infections spread more easily, which in turn drives greater antibiotic use — and greater selection of resistant strains. This vicious cycle explains why the burden of AMR falls hardest on low- and middle-income countries, where both the need for hygiene and the consequences of its absence are greatest. Breaking this cycle requires attacking resistance on multiple fronts, and hygiene sits at the centre of that effort.

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Figure 2. Antibiotics remain one of the most powerful tools in medicine — but every unnecessary prescription gives bacteria another chance to adapt.

4. Hygiene as the First Line of Defence

The logic behind hygiene as an AMR strategy is elegantly simple: if fewer people get infected, fewer antibiotics are used, and bacteria face less pressure to evolve resistance. Hygiene works at two levels. First, it reduces the incidence of infection directly. Second, it reduces the transmission of resistant organisms between people, which stops local outbreaks from becoming regional epidemics. Unlike new drug development, which takes decades and billions of dollars, hygiene interventions are inexpensive, immediately deployable, and benefit everyone regardless of income.

A landmark example comes from the hospital ward. Healthcare-associated infections affect hundreds of millions of patients worldwide every year, and they are frequently caused by resistant organisms. In the 1990s, Didier Pittet and colleagues in Geneva demonstrated that a sustained hand hygiene campaign among healthcare workers could slash infection rates within months. The programme, built on the simple idea of alcohol-based hand rubs placed at every bedside, became the model for the WHO's global hand hygiene strategy. Studies since then have consistently shown that hand hygiene alone can reduce healthcare-associated infections by 20–40%, and that even modest improvements translate into fewer antibiotic prescriptions and slower spread of resistant strains.

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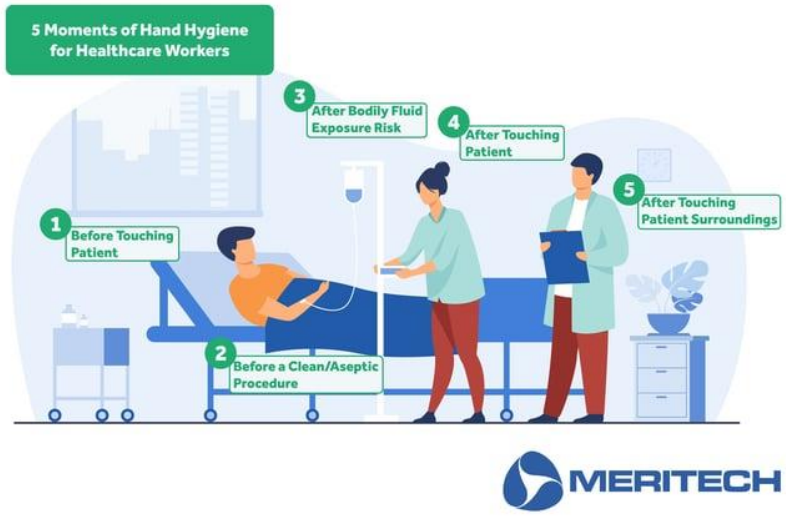


Figure 3. The WHO 'Five Moments' of hand hygiene for healthcare workers. Targeting hand cleaning to these critical moments maximizes protection for both patients and staff. (Image: Meritech)

5. Hygiene Beyond the Hospital

Hospitals are only one battlefield. The majority of antibiotic use actually happens in the community, which makes household and public hygiene equally important. Handwashing with soap after using the toilet and before preparing food is one of the most cost-effective health interventions ever studied. Trials in low-income settings have shown that handwashing campaigns reduce diarrhoeal disease by roughly a third and respiratory infections by about a fifth — both major reasons for antibiotic prescriptions, especially in children.

Water, sanitation, and hygiene (WASH) infrastructure extends these benefits to whole populations. Safe drinking water prevents cholera, typhoid, and dysentery; sanitary toilets interrupt the faecal-oral route of transmission; and clean surfaces in schools, clinics, and homes reduce the background level of infectious disease. When people fall ill less often, the demand for antibiotics falls with it, easing the selection pressure that breeds resistance. This is why global AMR action plans increasingly integrate WASH programming alongside antibiotic stewardship.

Food hygiene completes the picture. Thorough cooking, safe storage, separation of raw and cooked foods, and clean kitchen surfaces prevent foodborne infections that would otherwise send people to doctors — and to antibiotics. In the food production chain, good hygiene on farms can reduce the need for routine antibiotic use in animals, cutting off another major route by which resistant bacteria enter the human food supply.

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Table 2. Hygiene interventions and their role in preventing AMR

Intervention	Setting	AMR prevention mechanism
Hand hygiene (soap / alcohol rub)	Hospitals, clinics	Breaks transmission of resistant pathogens between patients and staff
Handwashing education	Households, schools	Reduces diarrhoea and respiratory infections, cutting antibiotic demand
Safe water and sanitation (WASH)	Communities	Prevents faecal-oral disease; lowers overall antibiotic consumption
Food hygiene (cooking, storage)	Homes, food industry	Prevents foodborne illness; limits antibiotic use and farm-to-human spread
Environmental cleaning / disinfection	Healthcare facilities	Removes resistant organisms from surfaces, devices, and equipment
Farm biosecurity and hygiene	Animal agriculture	Reduces routine antibiotic use; protects the food chain

6. The One Health Perspective

Microbes do not respect boundaries between humans, animals, and the environment. Resistant bacteria bred on a farm can travel through meat to a kitchen, through manure to a river, and through the water supply to a city. Recognizing this, the One Health approach — promoted by the WHO, the Food and Agriculture Organization, and the World Organisation for Animal Health — calls for coordinated action across all three domains. Hygiene is the thread that connects them: clean farms, clean food, clean water, clean hospitals, and clean hands form a continuous shield against resistance. The evidence table below summarizes key findings from the literature on hygiene and AMR-related outcomes.

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Table 3. Selected evidence from the literature

Study / Report	Finding	Significance
Pittet et al. (2000), Geneva	Hand hygiene campaign cut healthcare-associated infection rates substantially within months	Foundation of WHO's global hand hygiene strategy
WHO (2014), Global report on AMR	Resistance documented in every region of the world for common infections	AMR framed as a universal, urgent threat
O'Neill Review (2016), UK	Projected ~10 million deaths annually from AMR by 2050 without action	Drove global investment in AMR economics
CDC (2019), AR Threats Report	Drug-resistant infections cause millions of illnesses yearly in the USA alone	Documented the domestic burden in high-income settings
Lancet Series on WASH (2014)	Hygiene and sanitation among the most cost-effective health interventions	Linked WASH to reduced infectious disease burden
Global Research on AMR Project (2022)	AMR directly linked to ~1.27 million deaths globally in 2019	Established the true scale of the crisis

7. Challenges and the Way Forward

If hygiene is so effective, why is resistance still rising? The answer lies in the gap between knowing and doing. Hand hygiene compliance among healthcare workers, even in well-resourced hospitals, often hovers below 50% despite decades of campaigns. In many low-income settings, clean water and soap are simply unavailable. Behaviour change is difficult to sustain, funding for preventive measures is scarce compared to treatment, and hygiene is rarely as visible or celebrated as a new drug.

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Closing this gap demands a shift in mindset. Governments must treat hygiene infrastructure as a core part of their AMR national action plans, not an optional extra. Hospitals need continued investment in infection prevention teams, hand hygiene supplies, and environmental cleaning. Schools and communities need sustained education that makes handwashing a habit rather than a chore. And the public health community must communicate a message that is both honest and hopeful: every infection prevented is a small victory against resistance.



Figure 4. A bundle of hygiene practices — hand hygiene, surface disinfection, and protective equipment — working together to reduce infection risk in healthcare settings.

8. Conclusion

Antimicrobial resistance is often described as a slow pandemic, moving quietly through hospitals, farms, and households. The response, however, does not always require futuristic science. The most powerful tools we have — soap, clean water, sanitation, and disciplined hygiene — have been with us for over a century. Every act of hand hygiene, every clean surface, and every safe food practice is a vote for a future in which antibiotics still work. In the fight against resistance, cleanliness is not just next to godliness; it is next to survival.

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OCCUPATIONAL HEALTH HAZARDS AMONG MEDICAL STUDENTS DURING CLINICAL TRAINING

Mohd Sohel SULEMANI , Medical Student, Samarkand state medical university, Uzbekistan

Mohd Umar saifi , Medical Student, Samarkand state medical university, Uzbekistan

Mohammad bishr shakil , Medical Student, Samarkand state medical university, Uzbekistan

Shanu saifi , Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Medical students occupy an unusual position in the hospital: they are learners, yet they work in the same wards, theatres, and emergency rooms as fully trained staff, often with the least experience, the least authority, and the fewest protective habits. This narrative article reviews the occupational health hazards they face during clinical training and argues that these hazards are neither rare nor incidental, but structural. The hazards are organized into five families — biological (needlestick injuries and blood-borne pathogens such as HIV, hepatitis B and C, and tuberculosis), chemical (antineoplastic drugs, anaesthetic gases, disinfectants), physical (radiation, noise, slips and falls), ergonomic (patient handling, long standing hours), and psychological (sleep deprivation, burnout, depression, and bullying) — each illustrated with examples and, where possible, figures from the published literature. Tables summarize the hazard categories and the reasons students are disproportionately exposed. The article concludes that protecting students requires more than personal protective equipment: it demands vaccination, safety-engineered devices, enforced reporting systems, duty-hour limits, and above all a clinical culture in which asking for help is a sign of professionalism rather than weakness.

Keywords: medical students; clinical training; occupational health; needlestick injury; burnout; blood-borne pathogens; workplace safety

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1. Introduction

There is a peculiar moment in the life of every medical student: the first time a white coat is handed over in a hospital corridor, and the realization dawns that the coat is less a uniform than a passport — into other people's illnesses, other people's emergencies, and, quietly, other people's risks. From that day forward, the student is exposed to the same biological, chemical, physical, and emotional hazards as the professionals beside them, but without their years of conditioning, their instincts, or sometimes even their gloves.

Occupational health is the discipline concerned with preventing work-related injury and illness, and for two centuries it has asked the same sensible question of every workplace: what does this environment do to the people inside it? Hospitals, oddly, have been slow to turn that question inward. We screen patients meticulously for infections we might transmit; we audit hand hygiene with near-religious devotion; yet the tally of injuries suffered by the students themselves — the needlestick in a crowded casualty bay, the needle prick during a nervous first venepuncture, the month of night shifts that erases sleep and mood alike — has historically been kept less carefully than the ward inventory.

This article examines the occupational health hazards of medical students during clinical training in a narrative rather than purely technical style. It walks through the major categories of hazard, pausing on the ones the literature identifies as most common and most consequential, and then asks why students are so consistently overexposed. It closes with prevention — because every hazard described here already has a countermeasure, and the real scandal is how rarely those countermeasures are applied with consistency.

2. The Clinical Years: When the Classroom Meets the Ward

The pre-clinical years carry their own pressures, but it is the clinical phase — ward rotations, operating theatres, emergency departments, and community postings — that converts theoretical risk into daily exposure. A student drawing blood for the first time performs a procedure that staff perform hundreds of times a month; a student standing through a twelve-hour theatre list absorbs the same anaesthetic gases, radiation scatter, and fatigue as the surgical team, but with no say in the schedule. The teaching hospital is, in effect, a live laboratory in which students are both observers and, increasingly, participants. That participation is precisely what makes training valuable — and what obliges institutions to treat student safety as a duty rather than an afterthought.

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Figure 1. Medical students in the clinical environment — wards and theatres are where classroom learning becomes hands-on exposure to occupational hazards. (Photo: in-Training, online publication for medical students.)

3. Biological Hazards: Needles, Blood, and Invisible Enemies

Ask any group of senior clinicians about their training and a surprising number will roll up a sleeve or hold out a hand to show a small pale scar — the signature of a needlestick injury sustained as a student. The global numbers justify the storytelling: it has been estimated that sharps injuries expose millions of health workers to blood-borne pathogens every year, and large cohort studies have found that trainees sustain a disproportionate share. The mechanics are unglamorous — a needle passed from nurse to doctor between patient curtains, a syringe recapped in a hurry, a sharps box that overflows because nobody checked it — but the consequences can be life-altering. A single hollow-bore needle from an infectious patient can transmit hepatitis B in up to roughly a third of susceptible exposures, hepatitis C in a smaller but still substantial fraction, and HIV in around one in three hundred. Those odds sound comforting until one remembers that students perform and assist at hundreds of procedures, often on patients whose infection status is unknown.

Beyond sharps, students face the same airborne and contact risks as any hospital worker: tuberculosis from the undiagnosed cough in a crowded outpatient department, influenza and — as the COVID-19 pandemic made brutally clear — novel respiratory pathogens for which the student may lack adequate personal protective equipment. Latex allergy, scabies, and drug-resistant organisms complete the biological portfolio. The recurring theme is unpredictability: the student rarely knows which patient carries which risk, and is often the last person in the chain to be told.

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4. Beyond Infection: Chemical, Physical, Ergonomic, and Psychological Hazards

The word 'hazard' in a hospital summons images of needles, but some of the most stubborn occupational threats carry no syringe. Antineoplastic drugs handled during oncology rotations can be absorbed through skin or inhaled as dust; glutaraldehyde and other disinfectants irritate airways; waste anaesthetic gases accumulate in poorly ventilated theatres. Physically, students absorb scattered radiation during fluoroscopy lists, endure noise levels that would trouble an industrial inspector, and navigate slippery corridors on too little sleep. Ergonomically, the profession seems almost designed to injure the young: hours of standing, awkward positioning at operating tables, and patient transfers performed with technique learned by imitation rather than instruction.

The psychological hazards, however, may be the heaviest of all. Systematic reviews report that roughly a quarter to a third of medical students screen positive for depressive symptoms, while burnout rates climb higher still as training progresses. The drivers are structural: chronic sleep deprivation on call nights, the fear of making a fatal mistake, examinations that never quite end, and — too often — humiliation from seniors who mistake cruelty for teaching. Add exposure to death, suffering, and occasional abuse from patients, and it becomes clear that the clinical years tax the mind as reliably as the body.

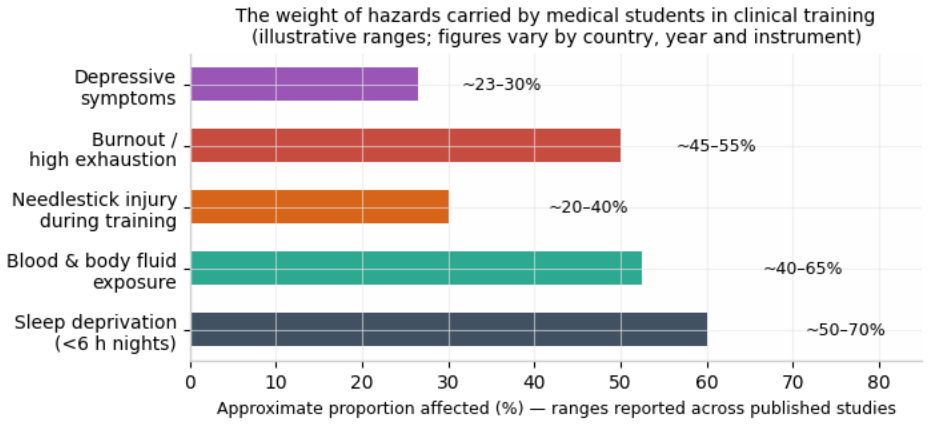


Figure 2. Approximate prevalence ranges for selected hazards among medical students, as reported across published studies; figures vary widely by country and measurement instrument and are illustrative rather than definitive.

Hazard category	Representative examples	Potential consequences

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Biological	Needlestick/sharps injuries; blood & body-fluid exposure; tuberculosis; influenza and other respiratory infections; latex	HIV, hepatitis B/C; chronic infection; TB disease; allergy
Chemical	Antineoplastic drugs; waste anaesthetic gases; glutaraldehyde and disinfectants; formalin	Organ toxicity; mucosal irritation; dermatitis; reproductive harm
Physical	Ionizing radiation; noise; wet floors; needles in laundry; electric equipment	Cumulative radiation dose; hearing strain; falls and lacerations
Ergonomic	Prolonged standing; awkward surgical postures; manual patient handling; heavy file/equipment loads	Musculoskeletal pain; varicose veins; chronic back injury
Psychosocial	Sleep deprivation; examination pressure; bullying; witnessing death; work–study imbalance	Depression, anxiety, burnout; reduced empathy; impaired learning

Table 1. The five families of occupational hazards encountered by medical students during clinical training.

5. Why Students Are Disproportionately Exposed

It would be tempting to dismiss student injuries as the ordinary tuition of a dangerous craft. The evidence suggests the opposite: students are exposed not merely because they work in risky places, but because of who they are within those places. They are inexperienced, performing procedures for the first time under observation, often with unfamiliar equipment and in settings where supervision competes with service demands. They occupy the bottom of an unforgiving hierarchy: the student who asks for gloves, questions a sharps practice, or refuses an unsafe task risks being labelled difficult at precisely the moment they need referees for their careers. Reporting systems, where they exist, are frequently designed for employees rather than students, so incidents go unrecorded, unprophylaxed, and uncounted. And because students rotate between departments and hospitals, nobody's dashboard accumulates their total exposure. Each of these factors is fixable; together, they explain why the newest hands in the hospital so often collect its oldest hazards.

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Key exposure	Why students are at higher risk	Core protective measure
Needlestick / sharps injury	First-time, supervised procedures; crowded settings; recapping habits	Safety-engineered devices; no-recapping rule; sharps boxes at arm's reach
Blood & body-fluid exposure	Emergency first assists; unknown patient serostatus; inconsistent glove use	Universal precautions; gloves + eye protection; hepatitis B vaccination
Airborne infection (TB, flu)	Prolonged ward contact; shared rooms; variable respirator fit-testing	Fit-tested respirators; ventilation; prompt isolation protocols
Antineoplastic / chemical agents	Oncology rotations; handling without training	Chemotherapy-competency training; closed-system drug transfer
Radiation	Standing close during fluoroscopy 'to learn', unbadged	Personal dosimeters; distance and shielding; justified standing zones
Sleep loss & fatigue	Night calls plus next-day classes; no duty-hour limits	Enforced rest rules; protected post-call time; scheduling limits
Stress, burnout, bullying	Exams + service demands; power imbalance; stigma around help-seeking	Confidential counselling; anti-bullying policies; mentoring; peer support

Table 2. Principal exposures in clinical training, the student-specific reasons risk is amplified, and the measures shown to reduce them.

6. Prevention: Building a Culture of Safety

The countermeasures for nearly everything described above are known, affordable, and embarrassingly simple. Hepatitis B vaccination — with post-vaccination titre confirmation — converts one of the gravest sharps-injury fears into a manageable risk. Safety-engineered needles and syringes, combined with the iron rule of never recapping, remove the single most common mechanism of injury. A post-exposure protocol that a student can reach within minutes — wash, report, test the source patient, begin prophylaxis where indicated — is the difference between a frightening afternoon

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and a life sentence of anxiety. For the mind, the prescriptions are equally concrete: confidential mental-health services that students actually trust, duty-hour limits honoured rather than merely published, and a culture in which seniors model the asking of help.

Yet the deepest prevention is cultural. A teaching hospital that audits student exposures with the same seriousness it audits central-line infections is telling its learners that their safety is part of the curriculum. Institutions that welcome incident reports instead of punishing them, that fit-test respirators for students and not only for staff, that teach safe patient handling as rigorously as they teach anatomy — these are the places where the next generation of physicians learns, from day one, that caring for oneself is not the opposite of caring for patients. It is a prerequisite.

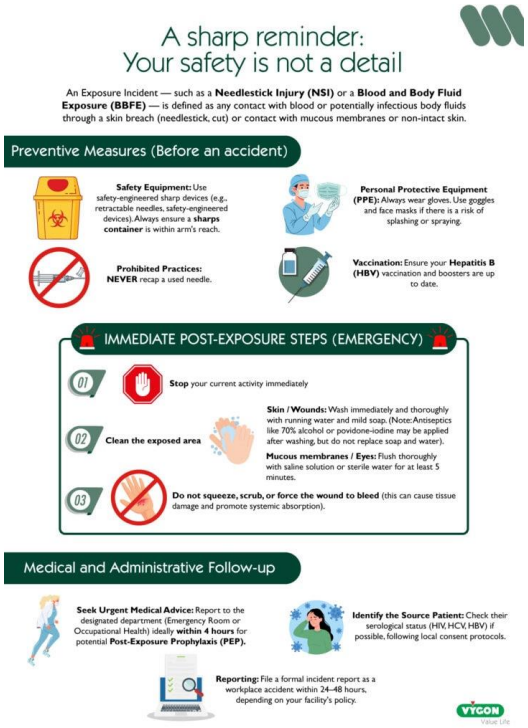


Figure 3. Standard prevention and post-exposure protocol for sharps injuries: engineered devices, PPE, vaccination, immediate first aid, and prompt reporting for post-exposure prophylaxis. (Infographic: Campus Vygon.)

7. Review of the Literature

The modern evidence base on these hazards rests on several sturdy pillars. Global burden estimates for sharps injuries, notably the work of Prüss-Üstün and colleagues published in the Bulletin of the World Health Organization, established that percutaneous exposures among health-care workers number in the millions annually, with the majority occurring in low- and middle-income countries where protective

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systems are weakest. Surgical cohort studies have sharpened the picture: Makary and co-workers, following surgeons in training in the United States, documented how frequently trainees are injured and how rarely their injuries are formally reported, even in well-resourced systems.

On the psychological side, the systematic reviews of Dyrbye and colleagues and, later, the meta-analysis by Rotenstein and co-workers in JAMA quantified what students had long said in corridors — that depressive symptoms affect roughly a quarter to a third of medical students worldwide, with burnout, anxiety, and suicidal ideation travelling close behind. Management guidance converges from public-health institutions: the World Health Organization and national public-health services publish exposure-management protocols and infection-prevention standards that apply with equal force to trainees. Read together, the literature tells a consistent and quietly optimistic story — the hazards are well characterized, the countermeasures are known, and the remaining gap is one of implementation and culture rather than knowledge.

8. Conclusion

Medical students enter clinical training to learn how to protect others; the least a teaching hospital can do in return is teach them — systematically, explicitly, and by example — how to protect themselves. The hazards of the clinical years are real and, at times, severe: infected needles, invisible viruses, toxic chemicals, ionizing radiation, wrecked sleep, and a burden of psychological strain that too many students carry in silence. None of this should be romanticized as a rite of passage, because rites of passage are supposed to end. The way forward is neither mysterious nor expensive: vaccination, engineered safety devices, honest reporting, enforced rest, accessible mental-health care, and a hierarchy humble enough to listen to its most junior members. The students who walk through hospital doors tomorrow are the workforce that will staff them for the next forty years. Their safety is not a student issue. It is the future of the profession, measured in unbroken skin, unbroken sleep, and unbroken spirits.

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THE TIRED BRAIN ON FIRE: THE RELATIONSHIP BETWEEN SLEEP DEPRIVATION AND MIGRAINE IN YOUNG ADULTS

Dr. Anjali Raj , Medical Student, Samarkand state medical university, Uzbekistan

Pavithira Radhakrishnan , Medical Student, Samarkand state medical university, Uzbekistan

M. Bhagya Harini , Medical Student, Samarkand state medical university, Uzbekistan

Aftab saifi , Medical Student, Samarkand state medical university, Uzbekistan

Shariq Ali , Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Migraine is among the most prevalent and disabling neurological disorders worldwide, and young adulthood is precisely the age when it strikes hardest. At the same time, no generation of young adults has ever slept less. This article examines the two-way relationship between sleep deprivation and migraine in people aged roughly 18 to 35, drawing on epidemiological studies, neuroimaging findings, and clinical trials. The evidence points to a self-reinforcing cycle: insufficient and irregular sleep lowers the migraine threshold, while migraine attacks and the medications used to treat them further erode sleep. Several overlapping mechanisms appear to link the two conditions, including hypothalamic dysregulation, heightened trigeminal pain signaling, glymphatic clearance failure, and stress-axis overactivation. Notably, sleep regularity may matter as much as sleep duration. The article concludes that sleep assessment and behavioral sleep treatment should be considered first-line, low-risk companions to standard migraine care — and that for many young adults, protecting the night may be one of the most powerful migraine preventions available.

Keywords: *sleep deprivation; insomnia; migraine; headache disorders; young adults; sleep hygiene; circadian rhythm; CGRP; trigeminal system; cognitive behavioral therapy for insomnia (CBT-I)*

1. Introduction

Picture a twenty-four-year-old finalizing a presentation at 1 a.m., collapsing into bed, and waking four hours later with a throbbing, one-sided headache that light and sound

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make unbearable. If this scene feels familiar, it is because it is playing out across campuses, offices, and apartments everywhere. Migraine affects roughly one in seven people globally, with onset peaking in young adulthood — and in large epidemiological studies, disturbed sleep consistently ranks among the strongest lifestyle triggers patients report.

The timing could hardly be worse. Survey after survey finds that people aged 18 to 35 sleep less and keep more irregular schedules than any other age group, trading sleep for screen time, shift work, study, and social life. What makes this pairing more than a coincidence is a growing body of neuroscience suggesting that sleep and migraine are not merely neighbors but roommates — sharing circuitry, chemistry, and, all too often, misery. Understanding that relationship may be the key to easing a burden that steals some of young adulthood's most productive years.

2. Methodology

This article is a narrative review rather than a systematic meta-analysis. Evidence was gathered through targeted searches of PubMed and Google Scholar using combinations of the terms 'sleep deprivation,' 'sleep quality,' 'insomnia,' 'migraine,' 'headache,' and 'young adults,' prioritizing publications from the last fifteen years. The selection emphasized large population cohorts, prospective diary studies, neuroimaging work, and randomized trials of behavioral sleep interventions in migraine populations. Foundational mechanistic papers and international headache classification guidelines were included for context. Because the article aims for synthesis and accessibility rather than exhaustive coverage, findings are interpreted narratively, and the limits of each evidence type are acknowledged where relevant.

3. The Sleep-Deprived Generation

Ask a room of young adults how they slept and a shrug is the most common answer. Chronically short sleep — typically defined as under seven hours a night — is now the norm rather than the exception in this age group. The causes are well rehearsed: glowing screens that delay the body clock, academic and professional pressure, economic necessity that demands shift work, and a culture that quietly treats exhaustion as a badge of commitment. Sleep regularity has suffered even more than duration; bedtimes drift by hours between weekdays and weekends, creating a kind of weekly jet lag.

The brain pays for this immediately. Sleep deprivation dulls attention, amplifies emotional reactivity, and raises inflammatory signaling. Neuroimaging reveals that after a short night, the brain's pain-processing regions become more reactive while its

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pain-dampening regions grow quieter — a neurological double hit that leaves the sleeper, in effect, tuned to a louder pain frequency.

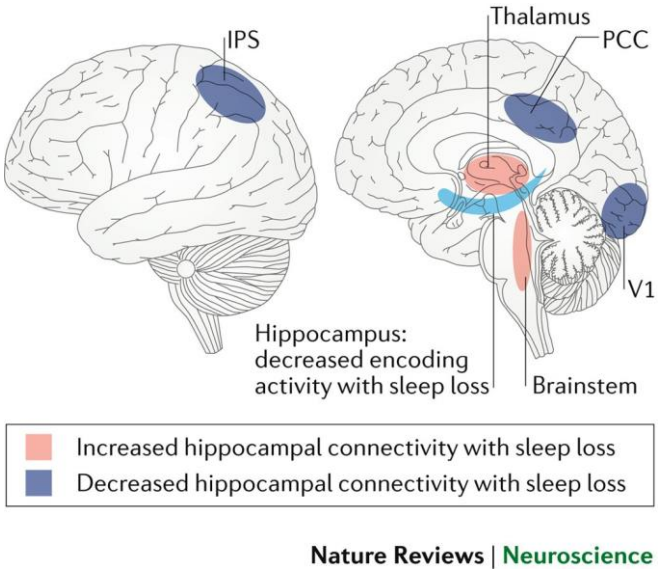


Figure 1. The sleep-deprived brain: altered connectivity between the hippocampus, thalamus, and pain-processing networks after sleep loss.

4. Migraine: A Disorder of the Whole Brain, Not Just a Bad Headache

Migraine deserves a better reputation than it has. Far from a severe headache, it is a complex neurological event that unfolds in phases. Hours before pain begins, the hypothalamus — the brain's master regulator of sleep, appetite, and stress — already shows altered activity, producing food cravings, yawning, and mood shifts. In some patients, a wave of altered electrical activity sweeps the cortex, generating the visual disturbances known as aura. Then the trigeminal pain system ignites, flooding meninges with inflammatory peptides such as CGRP, and the sufferer retreats to a dark, silent room.

Two features of this cascade matter for our story. First, the hypothalamus — the very structure that governs sleep — sits at the center of migraine biology. Second, migraine brains are hypersensitive: they respond more strongly to sensory input, stress hormones, and — crucially — changes in sleep. A night of deprivation does not create migraine from nothing; it lowers a threshold that was already set precariously low.

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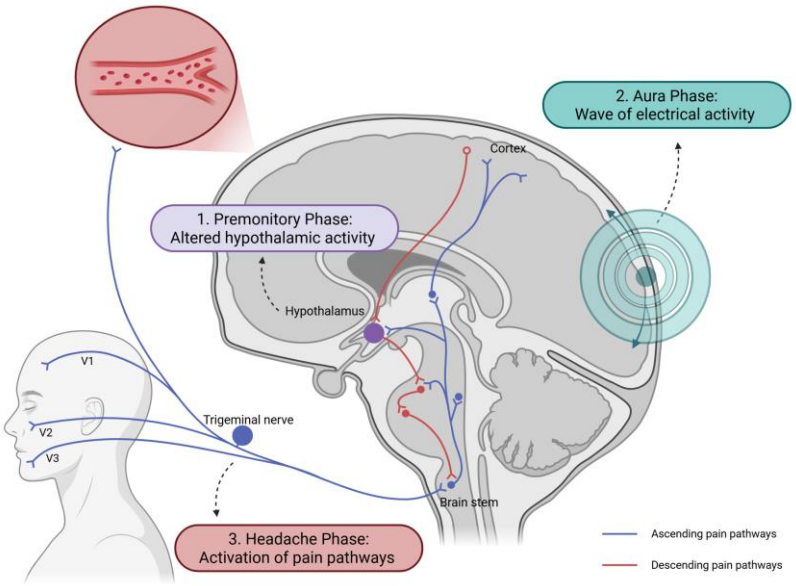


Figure 2. The phases of a migraine attack: altered hypothalamic activity, cortical spreading waves, and activation of trigeminal pain pathways.

5. Two Ways, One Vicious Cycle

The relationship between sleep and migraine runs in both directions, and understanding this loop is essential. Going forward, sleep loss triggers attacks: prospective diary studies show that both short sleep and, interestingly, excessive sleep predict next-day migraine, and that weekday–weekend shifts in sleep timing are followed by increased attack risk. Going backward, migraine wrecks sleep: nocturnal attacks fragment rest, and frequent use of acute pain medications can itself erode sleep architecture and, at worst, contribute to medication-overuse headache — a second disorder built on top of the first.

Several mechanisms plausibly link the two conditions. During deep sleep, the brain's glymphatic system flushes metabolic waste; curtailed deep sleep may leave behind inflammatory byproducts that sensitize trigeminal pathways. The hypothalamus, short on sleep, releases more orexin — an alertness peptide that also promotes CGRP release. Sleep loss amplifies the body's stress axis, flooding tissues with cortisol and inflammatory signals that prime pain. And serotonin, which helps regulate both sleep cycles and cranial blood vessels, falls out of balance in both conditions. None of these mechanisms alone explains the link; together, they form a plausible conspiracy.

Table 1. Evidence snapshot: what studies link sleep loss to migraine

Evidence type	Representative finding	Takeaway

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Population cohorts	Poor sleep quality doubles to triples risk of frequent headache/migraine	Strong, consistent association
Prospective diary studies	Short sleep and oversleep both predict next-day attacks	U-shaped relationship; timing matters
Neuroimaging	Sleep loss heightens reactivity of pain-processing regions	Plausible biological mechanism
RCTs of behavioral sleep treatment	Improved sleep regularity reduces headache frequency and disability	Sleep intervention itself is therapeutic
Clinical observation	Treating comorbid insomnia reduces medication overuse	Sleep care may de-escalate treatment

Narrative synthesis of epidemiological, neuroimaging, and intervention literature; effect sizes vary across studies.

6. Why Young Adults Sit at the Epicenter

Young adulthood concentrates every risk factor in one life stage. Migraine onset peaks in the early twenties, especially in women, whose attacks fluctuate with hormonal cycles that sleep disruption further destabilizes. This is also the age of maximal circadian disruption: delayed biological clocks collide with early obligations, caffeine and energy drinks substitute for rest, and mental-health conditions such as anxiety and depression — each independently linked to both poor sleep and migraine — reach their own peak prevalence. Add academic pressure and precarious work schedules, and the young adult brain becomes a perfect storm of triggers arriving all at once.

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Figure 3. For many young adults, the small hours have become study hours, screen hours, and worry hours — with consequences the next morning.

7. Breaking the Cycle: Sleep as Migraine Medicine

Here lies the hopeful twist: if poor sleep feeds migraine, then treating sleep should treat migraine — and the clinical evidence increasingly says it does. Behavioral programs that stabilize sleep schedules, most notably cognitive behavioral therapy for insomnia (CBT-I), have reduced headache frequency and disability even when administered by therapists without headache specialization. The operative principle appears to be regularity: going to bed and waking at consistent times seems to protect against attacks more reliably than simply logging more hours. Traditional sleep hygiene — dim evenings, cool dark rooms, screens off before bed — remains sensible groundwork, and melatonin, which both governs circadian timing and shows low levels in migraineurs, is being actively studied as a preventive agent.

Table 2. Practical sleep strategies for young adults with migraine

Strategy	What to do	Why it helps
Anchor your wake time	Same wake-up time every day, even weekends	Stabilizes circadian rhythm; reduces "social jet lag"
Protect a wind-down	30–60 min of dim, screen-free activity before bed	Allows natural melatonin rise
Watch the caffeine curfew	No caffeine after early afternoon	Caffeine lingers and fragments deep sleep
Treat insomnia behaviorally	CBT-I before or alongside headache drugs	Improves sleep without medication-overuse risk

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Track both sleep and attacks	Simple daily diary of bed/wake times and headaches	Reveals personal trigger patterns
Discuss melatonin	Ask a clinician about low-dose evening melatonin	Addresses circadian and migraine biology together

General guidance synthesized from headache-society recommendations and behavioral sleep-medicine literature; individual care varies.

8. Conclusion

Sleep deprivation and migraine are bound together in a relationship that is bidirectional, biological, and — most importantly — modifiable. For young adults living at the intersection of the two, the message is neither complicated nor hopeless: the night is not an extension of the day to be spent at will, but a physiological necessity on which the brain's pain thresholds depend. Restoring sleep will not cure every migraine, just as better migraine control will not fix every sleep problem. But protecting one powerfully protects the other. In a generation taught to burn the midnight oil, the evidence now suggests that the most radical thing a young migraineur can do is, quite simply, go to bed.

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PATHOGENETIC INTERRELATIONSHIP BETWEEN INSULIN RESISTANCE AND HYPERANDROGENISM IN POLYCYSTIC OVARY SYNDROME: MOLECULAR MECHANISMS, CLINICAL EVIDENCE, REAL-WORLD DATA, AND TRANSLATIONAL OPPORTUNITIES

Xolboyeva Mashhura Yoqutjon qizi

Email – yoqutjonovnam@gmail.com

Termez University of Economics and Service

Faculty of Medicine

Abstract

Background: Polycystic ovary syndrome (PCOS) is a heterogeneous reproductive–metabolic disorder in which insulin resistance, hyperinsulinemia, androgen excess, altered adipose biology and ovarian dysfunction interact rather than operate as independent abnormalities. Objective: To critically synthesize the mechanistic bidirectionality of insulin resistance and hyperandrogenism and examine how randomized trials, real-world evidence and computational methods may refine disease stratification and treatment evaluation. Methods: A structured, non-systematic evidence synthesis prioritized the 2023 International Evidence-based PCOS Guideline, indexed mechanistic reviews, randomized-trial syntheses and regulatory documents concerning real-world evidence and artificial intelligence. This article does not report new patient-level analyses or a completed PRISMA systematic review. Results: Insulin may amplify luteinizing-hormone-dependent theca-cell steroidogenesis and decrease hepatic sex hormone-binding globulin, increasing circulating bioavailable testosterone. Conversely, androgen excess may alter adipocyte function, body-fat distribution, skeletal-muscle insulin signalling and insulin secretion. Neither mechanistic direction is universally dominant; phenotype, adiposity, age and treatment exposure alter the observable association. The 2023 international guideline estimates PCOS prevalence at approximately 10–13% in reproductive-age women, conditional on diagnostic criteria, and discourages routine clinical insulin-resistance assays because available measures lack reliability. Therapeutic and observational evidence must therefore distinguish metabolic endpoints, reproductive outcomes and patient-centred effects. Real-world registries and cautiously validated machine learning can complement controlled trials, but confounding, assay heterogeneity, selection bias and subgroup underrepresentation complicate causal interpretation. Conclusion: PCOS is more accurately conceptualized as a network of reciprocal endocrine and metabolic feedback

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loops than as a single linear insulin-to-androgen pathway. Translational progress requires standardised androgen measurement, longitudinal metabolic phenotyping, prospectively registered causal analyses, equity-conscious algorithms and clinically meaningful outcomes.

Keywords: polycystic ovary syndrome; insulin resistance; hyperinsulinemia; hyperandrogenism; ovarian theca cells; sex hormone-binding globulin; adipose dysfunction; real-world evidence; causal inference; machine learning; precision medicine; pharmacovigilance.

1. Introduction

Clinical and biological framing

PCOS cannot be inferred from an ultrasound image alone: diagnostic criteria require appropriate combinations of androgen excess, ovulatory dysfunction, and polycystic ovarian morphology or, in adults, an eligible anti-Müllerian hormone alternative, after exclusion of competing diagnoses. Phenotypes differ in insulin sensitivity, adiposity and reproductive expression. Women with normal body mass index may have clinically important metabolic abnormalities, while weight gain can accentuate pre-existing susceptibility. The consensus guideline underscores that diagnostic decisions should not depend on an insulin assay routinely offered in commercial panels. This distinction matters when mechanistic biomarkers are translated into clinical pathways [1,2].

Non-linear causation

The conventional account of PCOS begins with insulin resistance and compensatory hyperinsulinemia, proceeding to ovarian androgen synthesis, suppressed hepatic sex hormone-binding globulin and anovulation. Yet longitudinal causal sequence is unsettled: insulin hypersecretion or altered hepatic insulin clearance could precede measurable peripheral resistance in some individuals. Excess androgens may themselves worsen insulin action, providing a feedback explanation for heterogeneity rather than requiring a single initiating lesion. An association between fasting insulin and free testosterone cannot identify which process began first; medication exposure, adiposity and pubertal maturation may confound both [3,4].

Meaning of RWD and RWE

Real-world data (RWD) are routinely generated observations about health and healthcare delivery, including clinical records, claims, registries, pharmacy dispensing

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and patient-generated measurements. Real-world evidence (RWE) refers to clinical evidence derived from appropriate analysis of RWD concerning use, benefits or risks of an intervention. The distinction is epistemic: a data repository becomes evidence only through a clearly defined research question, trustworthy measurements, a valid design and transparent inference. The FDA has issued guidance on data fitness, registries, submissions and non-interventional studies; none implies that an uncontrolled association automatically establishes drug effectiveness [7–11].

Efficacy versus effectiveness

Randomised controlled trials (RCTs) usually estimate treatment efficacy under protocol-specified selection, monitoring and adherence. In contrast, effectiveness describes performance under ordinary conditions where discontinuation, access barriers, comorbidities and co-interventions operate. PCOS illustrates why this is clinically consequential: improvement in menstrual regularity, hirsutism, conception, weight, glycaemia and patient-reported wellbeing may differ in magnitude or timing. RCT evidence is indispensable for treatment decisions, but observational follow-up can reveal persistence, treatment switching and infrequent adverse outcomes excluded by short follow-up. Observational emulation must approximate baseline randomization and control biases rather than treating scale as an antidote to confounding [1,5,7,8].

Regulatory and ethical context

WHO guidance requires autonomy, safety, transparency, accountability, inclusion and sustainability for health-related artificial intelligence. FDA oversight applies to qualifying AI-enabled medical-device functions and evaluates safety and effectiveness in the context of intended use. EMA initiatives have developed infrastructure for observational evidence, including the Data Analysis and Real World Interrogation Network (DARWIN EU). These bodies do not endorse deploying unvalidated PCOS prediction algorithms. Their relevance is governance: clinically derived evidence must remain auditable, fair, reproducible and proportionate to risk [12–16].

Why PCOS is a useful translational model

Few common disorders so clearly juxtapose endocrine regulation, metabolic disease and reproductive outcomes. The ovarian theca cell, liver, muscle, pancreas, adipose tissue and hypothalamic–pituitary axis each receive different insulin and androgen signals. This creates a natural test case for multimodal precision medicine, but also a setting where plausible biological stories may outrun actual evidence. This review

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therefore separates well-supported clinical recommendations, experimental pathway models, methodological proposals and areas of genuine uncertainty rather than interpreting all mechanistic hypotheses as proven in patients [1–4].

2. Materials and Methods

Design: critical integrative narrative review incorporating methodological appraisal. The evidence base was assembled from identifiable indexed publications and official organizational documents; it was not generated by a prospective study protocol, original database download or formal, reproducible PRISMA workflow. Searches were oriented to PubMed-indexed PCOS guidelines and mechanistic reviews, with contextual consideration of Cochrane Library, Embase, Scopus, Web of Science and publishers such as JAMA, Lancet, NEJM and Nature Medicine; coverage of each commercial database was not independently verified. No invented screening totals, pooled estimates, confidence intervals or heterogeneity statistics are reported.

Eligibility logic: prioritize human PCOS cohorts, experimental studies that identify tissue-specific mechanisms, international clinical practice guidelines, RCTs and systematic reviews, with emphasis on literature published from 2020 through October 2026. Older methodological or seminal sources were retained where indispensable. Exclude articles with unverifiable provenance, abstracts lacking ascertainable endpoints, studies conflating phenotype definitions, and prediction algorithms lacking outcome validation. Evidence is narrated by design, applicability, biological plausibility, confounding risk and transferability. The lack of dual-reviewer screening and preregistration limits claims of comprehensiveness.

Propensity score matching (PSM)

Estimate treatment probability from pretreatment covariates, establish common support, match observations and assess standardized mean differences rather than relying only on significance tests. PSM cannot balance unmeasured endocrine severity or clinician preferences.

Inverse probability weighting (IPW)

Weight treatment groups by inverse propensity probabilities to emulate a balanced pseudo-population. Inspect extreme weights, positivity, time-varying confounding and truncation. In longitudinal PCOS care, treatment decisions often respond to evolving weight and glycaemia.

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Cox proportional-hazards regression

Estimate relative hazard for time-to-event endpoints such as incident diabetes under proportional hazards assumptions; check time interactions, competing events and delayed entry. Hazard ratios should not be described as cumulative risk ratios.

Kaplan–Meier and survival analysis

Display event-free survival with correct censoring and risk sets. Switching therapy, loss to follow-up and reproductive status can generate informative censoring; competing-risk curves may be preferable for some outcomes.

Target-trial emulation

Specify eligibility, treatment strategies, assignment time, grace periods, follow-up, outcomes and estimand before analysis. Align time zero for exposure and comparator groups to prevent immortal-time bias.

Causal inference

Use directed acyclic graphs to identify confounders and mediators, distinguish total from direct effects, consider sensitivity analyses for unmeasured confounding, and avoid conditioning on colliders such as specialist referral.

Machine learning and deep learning

Evaluate penalized regression, tree ensembles, neural architectures and temporal models with patient-level splits and external-site testing. Compare with a transparent baseline, calibrate probabilities and assess clinically consequential thresholds.

Natural language processing and LLMs

Extract symptoms, menstrual-cycle narratives, adverse effects and family history from unstructured notes. Require manual adjudication samples, temporal negation handling, privacy protections and error monitoring; generative text is not independently verified clinical evidence.

Research reporting

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For future original observational analyses use STROBE and RECORD where relevant; use TRIPOD-compatible predictive reporting and risk-of-bias assessment for models; declare protocol deviations and prespecified subgroups.

3. Results: Integrated Evidence Synthesis

The following results constitute analytical findings from published clinical and mechanistic evidence, not outcomes of newly enrolled patients. Each subsection distinguishes observations that are incorporated into guidelines from hypotheses requiring prospective validation.

3.1. Molecular coupling of insulin signalling and ovarian steroidogenesis

Theca-cell steroidogenic response

Insulin augments the response of ovarian theca cells to luteinizing hormone, favouring activity within the steroidogenic cascade that generates androgen precursors. The pathway is not equivalent to the action of insulin in skeletal muscle glucose transport: selective insulin resistance may allow ovarian and steroidogenic responses to persist despite impaired systemic glucose disposal. The degree to which individual intracellular branches explain phenotype variation in vivo remains uncertain. LH pulsatility, genetic susceptibility, local growth factors and follicular stage provide alternative or interacting influences [3,4].

Hepatic SHBG suppression

The liver connects metabolic status to circulating free-androgen exposure by regulating sex hormone-binding globulin (SHBG). Insulin-linked metabolic perturbation can lower SHBG, increasing the proportion of bioavailable testosterone even without a matching increase in total testosterone. Consequently, apparent hyperandrogenemia depends partly on assay choice and binding-protein biology. In clinical practice, high-quality total testosterone measurement and properly calculated free testosterone are preferred; direct immunoassays may misclassify mild androgen excess. This mechanism demonstrates why insulin and androgen laboratory variables cannot be interpreted in isolation [1,2].

Pancreatic insulin secretion and clearance

Hyperinsulinemia is often treated as a compensation for defective insulin-mediated glucose uptake, but insulin secretion and hepatic clearance are separately regulated.

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Individuals with similar sensitivity estimates may experience distinct insulin exposure and divergent ovarian responses. Contemporary scholarship contests a universally fixed temporal ordering between hypersecretion and resistance; different phenotypes may have different proximal drivers. Studies using a single fasting sample are poorly equipped to determine dynamic secretion, clearance or feedback loops, emphasizing a need for longitudinal and physiological measurement [3].

Androgens as metabolic effectors

Androgens can affect adipose distribution, adipocyte differentiation, inflammatory signalling, skeletal-muscle substrate use and aspects of pancreatic function. Human and animal evidence supports biological plausibility but effect sizes vary by tissue, exposure concentration and developmental stage. Obesity should neither be treated as a compulsory intermediary nor excluded as an important amplifier. Strong causal claims require experiments isolating androgen exposure and longitudinal analyses that distinguish an androgen-driven metabolic change from shared antecedents [3,4].

Evidence interpretation: these observations should be read against differences in PCOS definitions, selection into specialty care, analytical platforms, background metabolic risk and follow-up duration. Mechanistic plausibility alone does not establish a treatment indication, and observational convergence does not remove the need for design-specific bias appraisal. Whenever a specific numeric treatment effect is absent, this synthesis intentionally refrains from supplying an invented estimate.

3.2. Phenotypes, diagnosis and clinical stratification

Phenotype heterogeneity

PCOS is an umbrella diagnosis rather than a homogeneous metabolic entity. The combination of menstrual disturbance, biochemical or clinical hyperandrogenism and ovarian morphological features affects apparent prevalence and prognosis. The guideline summary cites approximately 10–13% prevalence in reproductive-age women, varying with criteria and setting. It would be erroneous to assign this figure to Uzbekistan or to every age stratum without population-based ascertainment [1,2].

Differential diagnosis

Thyroid dysfunction, hyperprolactinemia, non-classic congenital adrenal hyperplasia, Cushing syndrome where clinically suspected, androgen-secreting tumours and medication effects should be considered according to presentation. Rapid virilization,

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marked androgen elevations or sudden severe progression require investigation beyond a routine PCOS label. Menstrual irregularity after menarche also needs age-appropriate interpretation because adult ultrasound thresholds and anti-Müllerian hormone criteria cannot simply be applied to adolescents [1,2].

Insulin resistance testing caveat

Insulin resistance is a key pathophysiological feature, but guideline authors caution that routine clinical measures are inaccurate and do not currently recommend their routine use as a diagnostic test. HOMA-IR may be useful in explicitly designed research, but no universal cutoff can be exported across laboratories and populations. Clinical assessment of glycaemic risk requires established metabolic testing and consideration of diabetes risk factors rather than reliance on fasting insulin as a diagnostic shortcut [1,2].

Outcome prioritization

Fertility, menstrual regularity, hirsutism, acne, androgen biochemistry, dysglycaemia, blood pressure, adiposity, mental health and quality of life describe different outcome domains. Improvement in one domain cannot be assumed to imply improvement in the others. Patient-reported satisfaction and therapeutic burden are particularly relevant when agents are continued for years. A research report should specify the exact endpoint and observation window and distinguish symptomatic benefit from reversal of underlying pathophysiology [1,5].

Evidence interpretation: these observations should be read against differences in PCOS definitions, selection into specialty care, analytical platforms, background metabolic risk and follow-up duration. Mechanistic plausibility alone does not establish a treatment indication, and observational convergence does not remove the need for design-specific bias appraisal. Whenever a specific numeric treatment effect is absent, this synthesis intentionally refrains from supplying an invented estimate.

3.3. Real-world clinical data sources and interpretive value

Electronic health records

Longitudinal encounter histories can link PCOS diagnosis, body-mass trajectories, glycaemic investigations, prescriptions and pregnancy outcomes. Yet clinician coding may privilege classic phenotypes and miss patients primarily treated for acne or infertility. A robust case definition should combine diagnosis codes with menstrual and

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biochemical information and, when possible, undergo chart adjudication. The date of first recorded diagnosis is not the date of biological onset; conflating them will distort causal models and natural-history estimates [7–10].

Electronic prescribing and pharmacy records

Prescribing datasets clarify the intended clinical strategy, while dispensing histories better approximate access. Neither is a direct measure of medicine ingestion. Combining orders, fills and treatment changes can describe adherence and persistence, but differences in insurance coverage, public supply chains and over-the-counter use must be addressed. For metformin, contraceptives and incretin-based medicines, classification of indication is crucial because the same agent may be used for other conditions [7–10].

Laboratory repositories

Glucose challenge tests, glycated haemoglobin, lipids and hormone results provide objective longitudinal variables when units, reference ranges and assay platforms are harmonized. Testosterone assays are especially vulnerable to interchangeability assumptions; an LC–MS/MS result and a low-range immunoassay value may not be directly comparable. Missing endocrine assays may indicate selective testing of symptomatic patients rather than random absence; interpretation therefore requires missingness diagnostics [1,2].

National registries and claims

Population registries can establish linkage across facilities and assess long-term diabetes, cardiovascular or reproductive events. Claims extend geographic reach but usually provide sparse physiology and may be biased by reimbursement pathways. Registry transparency requires defined inclusion rules, coding validation, linkage accuracy and secure governance. Apparent increases in PCOS incidence can reflect changed diagnostic access or recording rules, not necessarily altered biological disease frequency [7–10].

Pharmacovigilance datasets

Spontaneous safety reports can identify unusual patterns after exposure to treatment, particularly when an event is uncommon. They cannot provide incidence rates or treatment-specific causal risk by themselves because denominator data, duplicate

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reports and stimulated reporting may be absent. A reported association must be evaluated against background risk, co-medications, age and pregnancy status before any clinical claim is made [7,9].

Wearables and mobile applications

Step counts, sleep timing, heart-rate metrics and symptom diaries may describe trajectories inadequately captured by periodic appointments. But device adoption is socially patterned, menstrual tracking may be intermittent, and algorithm-generated features may change after software updates. The data support contextual assessment rather than PCOS diagnosis unless instruments are validated for the specific purpose. Research protocols should document sensor model, missingness, adherence and participant consent [12,13].

Evidence interpretation: these observations should be read against differences in PCOS definitions, selection into specialty care, analytical platforms, background metabolic risk and follow-up duration. Mechanistic plausibility alone does not establish a treatment indication, and observational convergence does not remove the need for design-specific bias appraisal. Whenever a specific numeric treatment effect is absent, this synthesis intentionally refrains from supplying an invented estimate.

3.4. Drug effectiveness, trial evidence and causal comparison

Lifestyle intervention and metformin

Individualized lifestyle measures remain central across PCOS phenotypes, while metformin is used for selected metabolic indications within guideline recommendations. A 2023 systematic review and meta-analysis evaluated randomized comparisons of metformin with or without lifestyle modification against placebo, underscoring that treatment effects must be appraised outcome by outcome rather than advertised as global syndrome resolution. Gastrointestinal adverse effects and patient preferences affect real-world persistence; randomized efficacy cannot predict adherence without further evidence [1,5].

Antiandrogenic and contraceptive strategies

Combined oral contraceptive pills can address hyperandrogenic symptoms and cycle concerns where appropriate, whereas antiandrogens require pregnancy-prevention consideration because of potential fetal risks. Because these therapies suppress signs of androgen excess through distinct mechanisms, changes in hormone measures during

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treatment cannot be equated with elimination of intrinsic PCOS susceptibility. Observational comparative effectiveness studies must control baseline symptom burden and contraindications, since the choice of medication is inherently confounded by indication [1,2].

Incretin-based treatment and metabolic endpoints

GLP-1 receptor agonists may be relevant in selected patients with excess weight or metabolic comorbidities under applicable care pathways. Changes in weight, insulin sensitivity, cycle parameters and androgen indices are related yet distinct; extrapolation of diabetes and obesity trial findings to reproductive PCOS outcomes requires PCOS-specific evidence. Treatment discontinuation, access, tolerability and pregnancy planning complicate extended follow-up, particularly in observational cohorts without reliable fertility-intention documentation [1,2].

Comparators across other diseases

In diabetes, treatment effectiveness is typically measured with glycaemia, metabolic events and complications; cardiovascular medicine uses hard events and risk-factor trajectories; oncology frequently focuses on progression and survival; infectious disease studies emphasize microbiological or clinical resolution; neurology uses disease-specific function or relapse endpoints. These templates illustrate transferable causal methods, not evidence that PCOS medications prevent cancer, infections or neurologic disease. Mechanistically plausible cross-disease models must not silently merge incompatible outcome definitions [7–10].

Target-trial design for PCOS

A realistic target trial could compare initiation of metformin versus non-initiation among eligible adults with a defined metabolic profile, assigning time zero to the same clinical decision point. Prespecify treatment strategies, crossover rules, one-year glycaemic outcomes and relevant tolerability endpoints. A propensity score can balance measured pretreatment predictors; inverse weighting or clone-censor-weight designs address treatment switching under appropriate assumptions. Explicit negative controls and quantitative bias analysis help demonstrate how much residual confounding might alter findings [7–10].

Evidence interpretation: these observations should be read against differences in PCOS definitions, selection into specialty care, analytical platforms, background metabolic

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risk and follow-up duration. Mechanistic plausibility alone does not establish a treatment indication, and observational convergence does not remove the need for design-specific bias appraisal. Whenever a specific numeric treatment effect is absent, this synthesis intentionally refrains from supplying an invented estimate.

3.5. Artificial intelligence for prediction and evidence generation

Supervised machine learning

A classifier may integrate cycle history, measured androgens, metabolic tests, demographics and treatment history to predict dysglycaemia or loss to follow-up. Predictors collected after the clinical decision must be excluded from a baseline model to prevent leakage. Performance should be evaluated against logistic regression and guideline-based rules, emphasizing calibration, sensitivity in underrepresented phenotypes and net clinical benefit rather than area-under-curve alone. No single published algorithm justifies universal PCOS deployment without external clinical validation [12–16].

Deep learning and multimodal fusion

Neural architectures can handle ultrasound imaging, sequential notes and dense physiological measures, but imaging recognition of polycystic morphology is not equivalent to syndrome diagnosis. Models trained on images selected because a specialist suspected PCOS risk incorporating referral or operator signals rather than disease biology. Multi-centre acquisition variation, image quality and hidden patient overlap are central hazards. A sensible pathway begins with transparent image annotations, independently held-out institutions and clinical reader comparisons [12–16].

Reinforcement learning

Sequential decision algorithms could theoretically suggest treatment adjustments to improve metabolic and reproductive trajectories. Nevertheless, reinforcement learning depends on reliable counterfactual outcomes, well-defined states and safe action constraints. Retrospective healthcare data offer limited exploration of untried actions and may reward recording intensity rather than health. Offline policy evaluation cannot substitute for prospective safety testing; autonomous therapy modification in PCOS would require stringent governance and clinical oversight [12–16].

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NLP and large language models

Natural language processing can recover menstrual irregularity, hirsutism descriptions, lifestyle barriers and adverse-effect narratives omitted from structured fields. Large language models may draft phenotype summaries or support chart abstraction; however, hallucinated medication history, imprecise temporal grounding and leakage of sensitive reproductive information create unacceptable risks without review. Validation should compare extracted variables with clinician-adjudicated records and measure subgroup-specific error and abstention quality [12,13].

Clinical decision support

A meaningful clinical decision-support system combines evidence-based thresholds, explicit uncertainties and understandable explanations. It should alert to metabolic screening, review contraindications and support shared decisions, rather than assign causal authority to correlational risk scores. Workflow studies must show that the system changes decisions beneficially without creating alert fatigue or widening gaps between urban specialty care and resource-limited settings. Prospective impact evaluation is a different claim from algorithmic benchmark performance [12–16].

Evidence interpretation: these observations should be read against differences in PCOS definitions, selection into specialty care, analytical platforms, background metabolic risk and follow-up duration. Mechanistic plausibility alone does not establish a treatment indication, and observational convergence does not remove the need for design-specific bias appraisal. Whenever a specific numeric treatment effect is absent, this synthesis intentionally refrains from supplying an invented estimate.

3.6. Pharmacovigilance, genetics and precision medicine

Adverse drug reactions

Treatment choices for PCOS require clinically contextualized safety monitoring. Metformin-related gastrointestinal symptoms, contraindications to estrogen-containing regimens, pregnancy-related concerns with antiandrogens and adverse reactions to weight-management agents have different monitoring pathways. Pharmacovigilance signals are hypothesis-generating; signal detection statistics are not event incidence estimates. Causal assessment should consider exposure duration, dechallenge, competing causes and reporting dynamics [1,5,7].

Pharmacogenetics and pharmacogenomics

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Genetic heterogeneity may influence insulin action, ovarian steroidogenesis and individual treatment response, but broad genomic profiling is not yet a validated routine method for selecting all PCOS therapies. Discriminative power, ancestry transferability and clinical utility must be demonstrated before genetic associations are used as treatment rules. A marker predicting biochemical response does not automatically predict live birth, glycaemic complications or quality of life. Pharmacogenetic claims should distinguish replicated pharmacodynamic effects from exploratory signals [1,3].

Biomarker hierarchy

Useful biomarker candidates include accurately measured testosterone, SHBG-derived free-androgen assessment, anthropometric variables, glucose tolerance and selected investigational metabolic or inflammatory signatures. Clinical biomarker status depends on analytical validity, clinical validity and evidence that acting on the result improves outcomes. The complexity of insulin physiology makes it unlikely that a single fasting value will universally partition PCOS into causal subtypes. Multi-marker panels require validation that survives changes in age, body composition and treatment exposure [1–4].

Precision medicine as intervention

A precision strategy should begin by defining an actionable patient subgroup, the available treatment choices and an outcome that matters to patients. Clustering phenotypes can be useful descriptively, yet cluster boundaries may be unstable across measurement protocols. A treatment-benefit model must predict the difference between potential outcomes under alternative therapies, not just prognosis under a chosen therapy. Personalization therefore requires robust trial or trial-emulation evidence of heterogeneous treatment effects [7–10].

Evidence interpretation: these observations should be read against differences in PCOS definitions, selection into specialty care, analytical platforms, background metabolic risk and follow-up duration. Mechanistic plausibility alone does not establish a treatment indication, and observational convergence does not remove the need for design-specific bias appraisal. Whenever a specific numeric treatment effect is absent, this synthesis intentionally refrains from supplying an invented estimate.

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3.7. Uzbekistan: infrastructure and research implementation

Healthcare digitalization context

For Uzbekistan, the immediate opportunity is to develop reliable reproductive–metabolic care pathways that can eventually be audited across public and private providers. Institutional initiatives for electronic records, digital prescriptions and longitudinal registries require confirmation of their current scope and implementation status through official Ministry of Health documents; this review does not assert a nationwide level of completion. A PCOS registry should use consent-compatible data governance and standard fields for cycle history, androgen results, metabolic status and treatment exposure.

Electronic prescriptions and longitudinal linkage

A structured e-prescription record can document therapeutic initiation, switching and possible drug–drug interactions, but the ability to link prescriptions to dispensing, laboratory results and clinical endpoints depends on legal permissions and software interoperability. Before causal evaluation, health authorities should publish data dictionaries, standardized identifiers, data-retention policies and quality-control procedures. A pilot may be conducted across selected centres; claims about broader deployment must follow public audit data rather than promotional descriptions.

Laboratory and clinical pharmacology capacity

Variation in hormone assays is a serious barrier to comparing PCOS diagnoses across regions. A harmonization programme could prioritize total testosterone assay methods, reference ranges, glucose testing procedures, body-mass units and systematic treatment-reaction documentation. Clinical pharmacology services could coordinate adverse-effect reviews, medication-use evaluation and research governance, particularly for therapies prescribed off-label. The first deliverables should be validation studies of recorded variables rather than complex predictive models.

AI implementation roadmap

Stage one should audit missingness and assay consistency; stage two should establish a deidentified multicentre dataset with independent ethics review; stage three should externally validate a simple, transparent risk model; stage four should prospectively test decision support under clinician oversight. Each stage needs predefined stopping

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criteria, adverse-event reporting, public performance summaries and governance for model revision. This incremental approach is more credible than deploying generative models into reproductive care before demonstrating safety and patient benefit.

Evidence interpretation: these observations should be read against differences in PCOS definitions, selection into specialty care, analytical platforms, background metabolic risk and follow-up duration. Mechanistic plausibility alone does not establish a treatment indication, and observational convergence does not remove the need for design-specific bias appraisal. Whenever a specific numeric treatment effect is absent, this synthesis intentionally refrains from supplying an invented estimate.

4. Discussion

Convergence and tension in international evidence

International guidance and recent mechanistic accounts converge on the importance of a reciprocal endocrine–metabolic network, but they differ in the strength of causal language attached to the initial abnormality. A conceptual model centered on insulin resistance is clinically useful; a model that also permits early hyperinsulinemia, altered clearance and androgen-mediated metabolic effects is more faithful to heterogeneity. The evidence does not establish that every woman enters the feedback loop through the same organ. Clinical recommendations should consequently prioritize verified outcomes rather than mechanistic certainty not yet demonstrated [1–4].

Selection, confounding and collider bias

Women referred for specialist care tend to have more severe symptoms or infertility concerns, and treatment choice reflects perceived severity, access and comorbidity. Conditioning on specialty attendance can induce collider bias between biological severity and healthcare-seeking behaviour. Propensity matching cannot repair unrecorded indications, while machine learning can excel at predicting who is tested rather than who truly has disease. Diagnostic and therapeutic models should therefore explicitly represent the care pathway that produced their data [7–10].

Missing data and measurement error

Androgen assays, menstrual records and metabolic investigations are often missing not at random: clinicians order them selectively, and patients facing financial or geographic barriers may not obtain testing. Multiple imputation requires assumptions whose plausibility must be discussed; complete-case analysis may preferentially retain

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patients receiving intensive care. Even an apparently high-performing model can be miscalibrated where testosterone assays change. Missingness indicators must not be mistaken for disease-specific biology without validation [1,2,12].

Explainable and ethical AI

Attribution maps or feature importance plots can describe a model without proving causal influence. Explanations must be checked for stability, clinical plausibility and possible encoding of protected or sensitive attributes. WHO emphasizes autonomy, safety, transparency, accountability, inclusiveness and sustainability; compliance requires human review and meaningful consent in reproductive health settings. Performance audits should include age, BMI, geography and clinically appropriate groups while safeguarding privacy [12,13].

Regulatory science and DARWIN EU

FDA RWE guidance focuses on whether the data and design are fit to answer a regulatory question, not whether an enormous observational dataset exists. Its AI-enabled medical-device oversight similarly evaluates intended use and lifecycle concerns. EMA real-world evidence initiatives including DARWIN EU provide an instructive organizational model for distributed observational analyses, but do not confer regulatory approval on a hypothetical PCOS tool. Uzbekistan could adapt governance principles while retaining locally appropriate implementation and legal safeguards [7–16].

Implications for future trials

The most informative prospective work would repeatedly characterize insulin sensitivity, secretion or clearance, ovarian and adrenal androgen production, adiposity distribution and clinical outcomes in explicitly defined phenotypes. Pragmatic randomization and causal trial emulation could test whether treatment benefit differs by baseline metabolic profile. Such designs must state pregnancy intentions and medication exposures, since reproductive decisions substantially affect treatment assignments and censoring. Patient-reported burden deserves equal methodological attention to biochemical surrogates [1,3,5].

5. Conclusion

PCOS arises from reciprocal interactions among insulin resistance, variable insulin secretion and clearance, androgen production, hepatic SHBG regulation, adipose

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signalling and the hypothalamic–pituitary–ovarian axis. Insulin resistance is important but neither a universal initiating event nor a sufficiently accurate stand-alone clinical diagnostic measure. The strongest near-term strategy combines guideline-concordant endocrine assessment, individualized metabolic surveillance, patient-prioritized treatment and rigorous appraisal of trial and real-world evidence. Prediction systems should be introduced only after transparent validation, assessment of calibration and subgroup fairness, privacy protections and prospective clinical evaluation. For Uzbekistan, careful standardization of laboratory measures and electronic clinical records is a necessary precursor to credible multi-centre PCOS evidence generation.

Declarations

Funding: Not reported. Conflict of interest: To be completed by the authors. Ethics approval: Not applicable to this critical literature review; no identifiable patient-level data were collected. Data availability: No original dataset was generated. Author contributions: To be supplied. Review limitations: Non-systematic search, no quantitative meta-analysis, and incomplete coverage of paywalled bibliographic services. This manuscript is not an original clinical outcomes study.

6. Extended critical appraisal and methodological research agenda

Temporal ordering of metabolic and ovarian abnormalities

The hypothesis that hyperinsulinemia initiates reproductive dysfunction can only be evaluated if investigators collect measurements before manifestations arise, rather than taking baseline at first diagnosis. Incident PCOS itself is an uncertain endpoint because recognition depends on menstrual maturation and clinician testing. Repeated insulin secretion and clearance assessments, weight changes, steroid profiles and cycle tracking would permit analysis of temporal sequence, but longitudinal associations still require assumptions regarding time-varying shared causes. Pubertal transitions, sleep, glucocorticoid exposure and contraception create changing endocrine environments. A persuasive study would predefine a plausible time scale between metabolic disruption and ovarian response, test alternative lag periods, and report whether androgen levels forecast later insulin changes after prior insulin trajectories are considered. The reverse model should be evaluated symmetrically. Testing only the dominant hypothesis invites confirmation bias, particularly when several interconnected variables are measured with different error rates. Negative findings should not be dismissed solely because the initial model was biologically attractive.

Selective tissue insulin responsiveness

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The phrase selective insulin resistance describes a theoretical distinction between insulin actions that remain responsive and those that become less effective. In PCOS, one clinically important observation is that ovarian steroidogenic responsiveness can coexist with impaired peripheral metabolic control. Translating this into a precise molecular model is complicated by differing cell environments and the participation of insulin-like growth factor signalling. Receptor abundance, intracellular adaptor proteins, inflammatory mediators and gonadotropin availability can alter pathway readouts in cultured cells. In-vitro models rarely reproduce the full concentration gradients and endocrine feedback encountered in vivo. Consequently, a single molecular marker derived from ovarian tissue should not be presented as a validated patient-level diagnostic biomarker. Researchers should triangulate cellular perturbation experiments with physiological endocrine challenges and longitudinal patient observations. Findings concordant across these layers deserve greater weight than associations confined to one experimental platform.

Interpreting anthropometry and adipose dysfunction

Body-mass index is convenient but cannot distinguish visceral adipose accumulation from lean mass or ectopic fat. Regional adiposity, inflammation and liver fat may influence insulin metabolism even when total body weight is not strikingly elevated. Studies that statistically adjust for BMI and declare an androgen effect independent of obesity may still leave residual confounding by adipose distribution and developmental exposures. Conversely, controlling for adiposity can remove part of a biologically meaningful mediated effect if androgen excess alters fat accumulation over time. Whether BMI belongs in an adjustment set therefore depends on the causal question and presumed temporal order, not on a universal covariate checklist. Prospective work should measure waist circumference consistently and consider more detailed body-composition measures when feasible. Investigators should report both absolute metabolic risk and measures of association so that a subgroup with a strong relative effect but low baseline risk is not incorrectly portrayed as requiring identical management.

Genetic architecture and environmental modifiers

PCOS susceptibility appears polygenic and environmentally modified; neither a single gene nor a single obesogenic behaviour explains most clinical presentations. Developmental conditions, family history and population ancestry may influence expression, but familial aggregation alone cannot separate inherited variation from

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shared exposures. Any genetic risk score requires validation in an ancestry-relevant population before it can be promoted as a treatment-selection instrument. A molecular association between a variant and insulin signalling may provide mechanistic clues while yielding little incremental predictive value beyond ordinary clinical examination. Similarly, an association between environmental exposures and PCOS phenotypes requires careful attention to timing, exposure accuracy and socioeconomic confounding. Claims that a lifestyle choice caused an individual PCOS are therefore unjustified. A balanced explanation can recognize modifiable metabolic risk without attributing blame or presenting untested genomic approaches as established care.

Measurement of circulating androgen excess

Testosterone measurement in women presents difficult analytical conditions because concentrations are lower than those typically encountered in adult men. Laboratory methods differ in cross-reactivity, sensitivity, calibration and reference populations; these differences can produce apparent phenotype shifts without any biological change. The international guideline emphasizes validated mass spectrometry for total testosterone and appropriately estimated or measured free testosterone. Overtreatment or missed diagnoses may follow if an immunoassay value is interpreted as exact. Hormonal contraception further complicates interpretation by altering SHBG and gonadotropin regulation. Ideally, investigators record the analytical platform, time since treatment cessation when clinically appropriate, menstrual context and reference interval. For multicentre RWE, laboratory-standardization exercises are not a cosmetic step but a prerequisite for valid causal comparisons. Without them, a model may identify hospital-specific testing equipment rather than endocrine disease.

Cardiovascular risk without overstating event evidence

PCOS is associated with several recognized metabolic and cardiovascular risk factors, yet elevated risk-factor frequency should not be conflated with proven disease-specific effects on myocardial infarction or cardiovascular mortality in all age groups. Many observational studies follow relatively young cohorts for limited periods, and treatment of dyslipidaemia, obesity or diabetes can modify later event rates. A Cox model yielding a hazard ratio for cardiovascular events requires accurate event adjudication, proportional-hazards assessment, careful age control and sufficient follow-up. Selection of symptomatic patients into care may affect both baseline cardiovascular testing and outcome surveillance. Clinically, standardized risk-factor assessment is warranted, but research writing should separate established guidance from uncertain

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long-term absolute risk. This distinction guards against both neglect and exaggerated prediction.

Pregnancy intentions as a time-varying clinical variable

Women with PCOS may change priorities between skin symptoms, menstrual control, infertility care and metabolic prevention. Pregnancy planning strongly affects which medications are acceptable, how menstrual outcomes are interpreted and whether follow-up is continuous. Observational datasets rarely capture these intentions reliably, which can produce apparent differences between treated cohorts that have little to do with pharmacological action. An emulated trial addressing medication persistence must distinguish cessation because of intolerance from cessation because conception is being attempted. Live birth, biochemical pregnancy and ovulation are different estimands; conflating them exaggerates treatment effects. Researchers should consider competing events and pregnancy-related censoring explicitly, rather than treating every termination of follow-up as statistically exchangeable. Shared decision-making should also recognize that not all patients prioritize fertility and that a reproductive diagnosis should not erase mental-health or metabolic concerns.

Comparative evidence for inositol products

Inositol formulations are frequently marketed through mechanistic claims involving insulin-related signalling, but plausibility does not guarantee a clinically meaningful or reproducible effect. A systematic review prepared for the international guideline evaluated the evidence and found uncertainty concerning efficacy across important PCOS outcomes. Products may vary in isomer composition, dose, quality and regulatory status, limiting the transferability of small trials. Observational studies of supplement use are especially prone to healthy-user effects and undocumented co-interventions, because users may simultaneously change diet or take other therapies. Reporting should distinguish study-grade interventions from commercially heterogeneous preparations and should not label all supplements equivalent. A trial should prespecify reproductive and metabolic endpoints, adherence verification, adverse events and clinically credible follow-up; marketing testimonials and within-person pre–post change are insufficient evidence of effectiveness [21].

Comparative effectiveness and the limits of propensity scores

Propensity scores summarize measured covariate information about treatment assignment, not biological similarity in every respect. If women with the most severe

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menstrual disturbances preferentially receive one strategy and symptom severity is poorly captured, matched cohorts may still differ in important ways. Transparent reporting should include distributions before and after matching, numbers discarded, overlap, balance for continuous laboratory variables and follow-up after treatment initiation. Weighting can preserve more patients but creates instability when predicted probabilities approach zero or one. The analyst should identify positivity violations rather than mechanically truncate away clinically meaningful subgroups. Where prescribing depends on unmeasured fertility plans, practice-site preference or contraindications, an observational estimate may remain noncausal. Reporting null results with honest uncertainty is more valuable than presenting superficially precise hazard ratios from poorly aligned comparisons.

Transportability and algorithmic fairness

A model developed in a tertiary reproductive centre may overestimate disease severity when moved to primary care, where symptom prevalence, referral thresholds and access to hormone testing are different. Simple demographic adjustment cannot necessarily correct these shifts. Validation should examine calibration intercepts and slopes, decision thresholds, under-diagnosis among patients without overt obesity and performance across geographic or resource strata. Gender-related and reproductive health variables also require careful privacy governance: adding sensitive covariates should be justified by clinical value and consent, not merely by a small improvement in discrimination. Clinical usability depends on whether a model supplies actionable information at the point of care without increasing clinician workload or stigmatizing patients. Periodic reassessment is essential when clinical guidelines, laboratory methods or electronic coding practices change.

Pharmacovigilance beyond automated signal mining

AI-assisted adverse-event detection can organize text reports and identify unexpected medication–event combinations, but frequency of a reporting term is not an estimate of attributable harm. For example, gastrointestinal complaints may be common reasons for metformin discontinuation, yet an adverse-event record may omit dose titration, formulation, co-medications or intercurrent illness. Automated pharmacovigilance should preserve provenance, de-duplicate records and clearly indicate uncertainty in temporal association. Clinical reviewers must assess alternative explanations, potential pregnancy exposure and whether the event meets a case definition. Robust postmarketing studies can sometimes complement spontaneous reports with

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identifiable denominators and comparators. A safety signal should generate a testable hypothesis and proportionate clinical response, not a headline that a medication has definitively caused a newly recognized syndrome.

Cross-disease extrapolation: methodological gains and biological hazards

Methodological tools developed in diabetes, cardiovascular medicine, oncology, infectious diseases and neurology offer PCOS investigators valuable examples of longitudinal endpoint definition, registry linkage, adaptive monitoring and treatment-benefit modelling. These domains nevertheless differ sharply in natural history, outcome incidence and acceptable treatment risk. A survival model appropriate for death in oncology may be inefficient when the primary PCOS outcome is symptom improvement over months. Antimicrobial resistance dynamics offer little direct biological analogy for ovarian androgen production, even when machine-learning techniques are shared. The transfer should therefore be methodological rather than evidentiary: importing an analytical approach does not imply that findings or treatments transport across diseases. PCOS-specific endpoints and reproductive context should anchor every study, with other disease areas used as examples of design discipline.

A feasible multicentre protocol for Uzbekistan

A pragmatic initial study could prospectively enroll consecutive adults evaluated for possible PCOS in selected gynaecology and endocrinology settings, documenting both confirmed diagnoses and exclusions. The protocol would record menstrual history, standardized clinical androgen features, laboratory test method, anthropometry, relevant medication use and established metabolic assessments. Investigators should preregister a limited number of outcomes and harmonize variable names before recruitment. A short clinician-completed data form, multilingual patient information and secure pseudonymization would be preferable to collecting excessive unvalidated variables. Missingness and assay differences should be reviewed during pilot enrolment, and the dataset should undergo an external quality audit before predictive modelling. The proposal does not imply that a national PCOS registry currently exists or that the Ministry has endorsed a particular protocol; these would require separate institutional authorization.

Implementation science and health system decision-making

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Evidence-based innovations fail when they do not match clinical workflow, staffing and patient access. A national digital-health strategy relevant to PCOS should address the full pathway from first presentation to metabolic follow-up and fertility consultation. A validated laboratory result unavailable to a rural patient does not provide equitable diagnostic benefit. Electronic decision support should be piloted with both specialists and primary-care clinicians, measuring time spent, unnecessary repeat testing, referral patterns, adverse events and patient experience. Procurement criteria ought to include data portability and algorithmic auditing, not merely technical demonstration performance. Training should teach clinicians to distinguish prediction from causal evidence and to interpret missing or unreliable test results. These implementation outcomes are often more consequential than small gains in model discrimination.

Research agenda: high-priority unresolved questions

Several empirical questions remain open: whether early insulin hypersecretion identifies a reproducible causal subgroup; whether androgen lowering can independently improve insulin sensitivity beyond changes in adiposity; which repeated endocrine measures predict meaningful metabolic outcomes; and whether phenotype-tailored treatment exceeds guideline-concordant usual care. High-quality answers will require longitudinal time ordering, analytical standardization, experimental triangulation and sufficiently representative recruitment. Trial emulation can support but not replace randomized intervention where equipoise persists. Investigators should commit to publishing protocols and null findings, reporting effect-size uncertainty and making non-identifying data dictionaries available. Success should be assessed by improvements in patient outcomes and equity, not the number of biomarkers or algorithms produced.

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7. Evidence certainty, reporting transparency and final translational priorities

Evidence certainty and multiplicity

Scientific caution is especially important in the PCOS literature because the same clinical population can yield numerous correlated outcomes: fasting insulin, different indices of insulin sensitivity, total testosterone, free androgen index, cycle frequency, body weight and ovarian morphology. Testing each outcome without a prespecified hierarchy increases the probability of apparently persuasive chance findings. In a review, these results should be contextualized by study design, outcome priority, whether treatment comparisons were randomized, and whether primary analyses were registered. When reports provide only within-group improvement rather than between-group treatment contrasts, a causal conclusion is inappropriate. Likewise, statistical significance cannot repair measurement bias or make a small laboratory difference clinically valuable. Future PCOS trials should separate primary endpoints from exploratory pathway analyses, report adverse outcomes comprehensively and avoid treating post-hoc phenotypic subgroups as definitive responders. Evidence synthesis should preserve the uncertainty of underlying research instead of converting multiple small studies into an overconfident consensus.

Clinical meaning and patient-level communication

An integrated metabolic–reproductive model has practical value only when clinicians can explain it accurately without creating deterministic expectations. A patient with PCOS should not be told that every androgen abnormality results from excess insulin or that body weight alone accounts for her symptoms. The relative contributions of gonadotropins, androgen production, binding proteins, adipose function and genetic predisposition vary. Clinical conversations ought to distinguish risk from diagnosis and

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clarify why individual therapies target particular manifestations. For example, cycle control is not a validated substitute for metabolic screening, and improved glucose-related markers cannot be presumed to resolve infertility. Effective care requires reproductive goal assessment, cardiometabolic review, emotional wellbeing considerations and follow-up adapted to access and preferences. This multidimensional approach is consistent with the international guideline emphasis on shared decision-making and avoidance of weight stigma [1,2].

Data infrastructure and reproducible science

Any proposed national or international PCOS analytic network needs a reproducibility architecture. Protocols should define source populations, dates, ascertainment logic, exposure windows, outcome algorithms, clinically meaningful estimands and precommitted sensitivity analyses. Secure data-linkage rules and shared semantic definitions are more important than collecting unstructured records at scale. Distributed analysis, as illustrated by European regulatory evidence infrastructures, can permit harmonized methods while limiting unnecessary movement of identifiable information [21,23]. Independent validation teams should review coding accuracy and endocrine assay comparability before analyses are interpreted. Algorithm developers should release model cards, evaluation plans and documentation of relevant clinical constraints where law and privacy rules allow. Publication of negative validation results would reduce waste and counter selective reporting. These processes create the foundation for reliable evidence rather than simply adding technical sophistication.

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ANTIBIOTIC RESISTANCE AS AN EMERGING ENVIRONMENTAL HEALTH PROBLEM: SOURCES, PATHWAYS, HEALTH IMPACTS AND HYGIENE-BASED PREVENTION WITHIN A ONE HEALTH FRAMEWORK

Dr.Aatika fahim , Medical Student, Samarkand state medical university, Uzbekistan

Dr.Osama , Medical Student, Samarkand state medical university, Uzbekistan

Dr.Patel Noman , Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Background: Antimicrobial resistance (AMR) is one of the leading global threats to public health. Although it has long been regarded as a problem of hospitals and clinical prescribing, a growing body of evidence shows that the natural environment acts as a reservoir, a transmission route and a breeding ground for antibiotic-resistant bacteria (ARB) and antibiotic resistance genes (ARGs).

Aim: This review explains why antibiotic resistance qualifies as an emerging environmental health problem, describes its main environmental sources and pathways, summarises its health and economic burden, and outlines hygiene-based and policy measures for its control.

Main findings: Bacterial AMR was directly responsible for an estimated 1.14 million deaths in 2021, and attributable deaths are forecast to reach 1.91 million per year by 2050. In 2023, about one in six laboratory-confirmed bacterial infections worldwide was resistant to antibiotics. Major environmental drivers include untreated or partially treated sewage, hospital effluent, pharmaceutical manufacturing waste, antibiotic use in livestock and aquaculture, and co-selection by heavy metals and biocides. Humans are exposed through drinking water, food, recreational water and direct contact.

Conclusion: Safe water, sanitation and hygiene (WASH), infection prevention, improved wastewater treatment, control of manufacturing pollution, prudent antibiotic use and environmental surveillance are essential, mutually reinforcing measures. A One Health approach that links human, animal and environmental health is required to slow the spread of resistance.

Keywords: antimicrobial resistance; antibiotic resistance genes; environmental health; wastewater; One Health; hygiene; sanitation; pharmaceutical pollution

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Key Messages

- Antibiotic resistance genes are now recognised as environmental contaminants, not only clinical concerns.
- Sewage, hospital and factory effluents, and farm run-off carry antibiotics and resistant bacteria into water and soil.
- Poor sanitation and unsafe water help resistant bacteria spread from person to person, which makes hygiene a frontline defence.
- Controlling resistance requires a One Health approach that joins human, animal and environmental action.

1. Introduction

When Alexander Fleming received the Nobel Prize in 1945 for the discovery of penicillin, he warned that careless use of the drug could allow microbes to become resistant to it [1]. Eight decades later, that warning has become one of the defining health challenges of our time. Antimicrobial resistance (AMR), the ability of microorganisms to survive medicines that once killed them, is ranked by the World Health Organization (WHO) among the top global public health and development threats [2]. In 2019, bacterial AMR was directly responsible for an estimated 1.27 million deaths and was associated with 4.95 million deaths, a toll greater than that of HIV/AIDS or malaria [3]. The most recent WHO surveillance report found that one in six laboratory-confirmed bacterial infections worldwide in 2023 was resistant to antibiotic treatment, and that resistance increased in more than 40% of the pathogen-antibiotic combinations monitored between 2018 and 2023 [4].

For most of the antibiotic era, resistance was treated as a clinical problem confined to hospitals and prescribing practice. Over the past two decades, however, research has shown that the environment plays a central role in how resistance emerges and spreads. In 2006, Pruden and colleagues proposed that antibiotic resistance genes (ARGs) should be regarded as emerging environmental contaminants [5], and in 2023 the United Nations Environment Programme (UNEP) called for stronger environmental action as part of the One Health response to AMR [6]. Resistance itself is not new: genes conferring resistance to modern antibiotics have been found in 30,000-year-old permafrost [7]. What is new is the scale at which human activity is releasing antibiotics, resistant bacteria and resistance genes into water, soil and air.

This review has four objectives: (i) to explain why antibiotic resistance qualifies as an emerging environmental health problem; (ii) to describe its major environmental

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sources and pathways of human exposure; (iii) to summarise its health and economic burden; and (iv) to highlight hygiene-based, technical and policy measures for its prevention and control.

2. Methods

This article is a narrative review. Literature was drawn from peer-reviewed articles indexed in PubMed, including systematic analyses and reviews, together with reports and technical guidance from official public health and environmental bodies: WHO, the US Centers for Disease Control and Prevention (CDC), UNEP, the United Nations General Assembly and the World Bank. Search terms included combinations of "antibiotic resistance", "antimicrobial resistance", "environment", "wastewater", "antibiotic resistance genes", "pharmaceutical pollution", "One Health" and "water, sanitation and hygiene". Priority was given to landmark studies, recent global burden estimates and current international policy documents. As this is a narrative rather than a systematic review, no formal quality scoring of individual studies was performed.

3. Understanding Antibiotic Resistance

3.1 How resistance arises and spreads

Bacteria become resistant in two main ways. First, random mutations in their own chromosomes may alter the target of an antibiotic or the way the drug enters or leaves the cell. Second, and more importantly for rapid spread, bacteria can acquire ready-made resistance genes from other bacteria through horizontal gene transfer: conjugation (transfer of plasmids between cells), transformation (uptake of free DNA from the surroundings) and transduction (transfer by bacteriophages). Resistance mechanisms include enzymes that destroy the drug (such as extended-spectrum β-lactamases and carbapenemases), efflux pumps that expel it, modification of the drug's target and reduced permeability of the cell wall. Because many resistance genes sit on mobile genetic elements such as plasmids, transposons and integrons, they can move from harmless environmental bacteria into human pathogens [8].

3.2 Why the environment matters

The environment contributes to resistance in three ways. It is a *reservoir*, holding an enormous diversity of bacteria and resistance genes. It is a *conduit*, carrying resistant organisms between people, animals and places through water, soil, food and air. It is also a *selection site*: even very low concentrations of antibiotics can favour resistant

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bacteria. Laboratory work by Gullberg and colleagues showed that resistant strains can be selected at antibiotic concentrations far below those needed to inhibit bacterial growth [9]. On this basis, Bengtsson-Palme and Larsson proposed concentration limits for individual antibiotics in the environment, designed to stay below the level at which resistance is selected [10].

3.3 Pathogens of greatest concern

The 2024 WHO Bacterial Priority Pathogens List places carbapenem-resistant *Acinetobacter baumannii*, third-generation cephalosporin-resistant and carbapenem-resistant Enterobacterales, and rifampicin-resistant *Mycobacterium tuberculosis* in its critical priority group [11]. Enterobacterales such as *Escherichia coli* and *Klebsiella pneumoniae* normally live in the human and animal gut and are shed in large numbers in faeces. This creates a direct link between antibiotic resistance and sanitation, which is why the problem falls squarely within the field of hygiene.

4. Why Antibiotic Resistance Is an Emerging Environmental Health Problem

An emerging environmental health problem is one that has only recently been recognised, is increasing in scale, poses a credible risk to human health and is not yet adequately covered by monitoring or regulation. Antibiotic resistance meets each of these criteria. ARGs and resistant bacteria are not included in most national drinking-water or effluent standards, yet they are now detected in rivers, sediments, soils and treated wastewater around the world [8].

Several landmark findings illustrate this. In 2011, bacteria carrying the NDM-1 gene, which confers resistance to carbapenems, were detected in public tap water and in seepage water from streets and pools in New Delhi [12]. In 2016, the plasmid-borne *mcr-1* gene, which confers resistance to colistin (a last-resort antibiotic), was reported in pigs, retail meat and patients in China and was subsequently detected in many other countries [13]. A global study of urban sewage from 79 sewage samples from 74 cities in 60 countries found that the abundance of resistance genes varied widely and was associated with socioeconomic, health and environmental factors, including sanitation [14]. Similarly, an analysis of data from over 100 countries found that poor infrastructure and governance were more strongly associated with resistance levels than antibiotic consumption alone, suggesting that the *spread* of resistant bacteria through inadequate sanitation is as important as antibiotic use itself [15].

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5. Environmental Sources and Pathways

Figure 1 summarises how antibiotics, resistant bacteria and resistance genes move from their sources into the environment and back to people. Table 1 summarises the main sources and corresponding control measures.

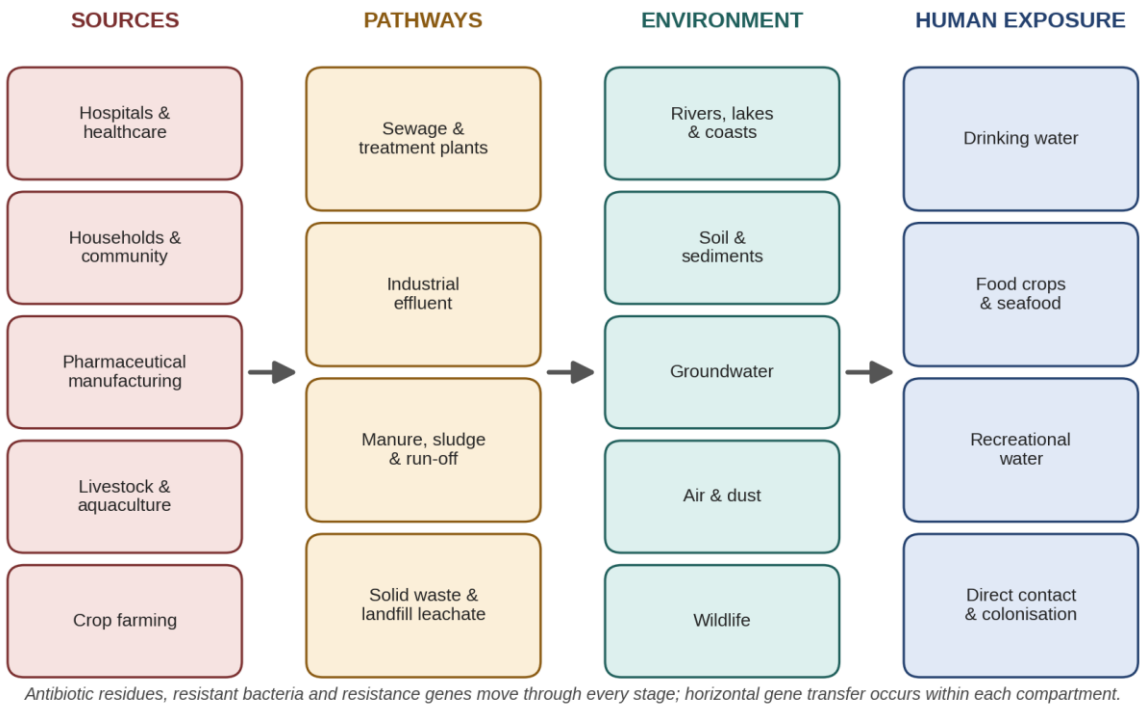


Figure 1. Simplified One Health model of the environmental dimension of antibiotic resistance, showing sources, environmental pathways and routes of human exposure. Original figure prepared by the authors based on [6,8].

5.1 Domestic sewage and wastewater treatment plants

A substantial proportion of many antibiotics taken by people passes through the body unchanged or as active metabolites and enters the sewer [16]. Wastewater treatment plants (WWTPs) therefore receive a continuous mixture of antibiotic residues, resistant bacteria from millions of human guts and free resistance genes. Conventional treatment reduces bacterial numbers but does not eliminate resistant organisms or genes, and the dense, nutrient-rich microbial communities in treatment tanks provide ideal conditions for gene exchange; for this reason WWTPs are described as hotspots of resistance [17]. A study of WWTPs in seven European countries found that the resistance genes in their

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effluents mirrored the pattern of clinical resistance in each country [18], showing that sewage reflects the resistance carried by the population it serves. Where sewage is discharged without any treatment, as still occurs in many low- and middle-income countries, the load reaching rivers and coasts is far higher [6].

5.2 Hospital effluent

Hospitals use large quantities of antibiotics, including last-resort agents, and treat patients colonised or infected with multidrug-resistant bacteria. Their wastewater is often discharged into the municipal sewer with little or no pre-treatment, adding concentrated antibiotic residues and highly resistant organisms to the general sewage stream [16].

5.3 Pharmaceutical manufacturing

Some of the highest antibiotic concentrations ever measured in the environment have come from factory effluents. Treated effluent from a plant near Hyderabad, India, which served around 90 bulk drug manufacturers, contained ciprofloxacin at concentrations exceeding those found in the blood of patients receiving the drug [19]. A global survey of pharmaceutical pollution in 258 rivers across 104 countries found active pharmaceutical ingredients at potentially harmful concentrations at about a quarter of sampling sites, with the greatest pollution in low- and middle-income countries [20]. In 2024, WHO issued its first guidance on wastewater and solid waste management for antibiotic manufacturing, setting targets intended to keep antibiotic concentrations in receiving waters below levels that select for resistance [21].

5.4 Agriculture and aquaculture

A large share of the world's antimicrobials is used in food-producing animals, often for growth promotion or routine disease prevention rather than treatment. Van Boeckel and colleagues estimated that antimicrobial use in food animals would rise by about two-thirds between 2010 and 2030, driven by growing demand for meat in middle-income countries [22]. Animal manure carrying residues and resistant bacteria is spread on fields as fertiliser, from where it can wash into rivers and groundwater or contaminate crops. In aquaculture, antibiotics are frequently added directly to water. The emergence of *mcr-1* in livestock is a clear example of resistance arising in agriculture and reaching humans [13].

5.5 Co-selection by heavy metals and biocides

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Genes for resistance to heavy metals (such as copper, zinc and mercury) and to disinfectants are often located on the same mobile genetic elements as antibiotic resistance genes. Pollution by these substances can therefore maintain and select for antibiotic resistance even where no antibiotic is present, a process known as co-selection [23]. This links antibiotic resistance to wider problems of industrial and agricultural pollution.

5.6 Solid waste and the wider planetary context

Unused medicines discarded in household waste or flushed down toilets, and leachate from landfills, are further sources of contamination. UNEP places AMR within the "triple planetary crisis" of climate change, biodiversity loss and pollution, noting that environmental degradation and resistance are closely interconnected [6].

Table 1. Major environmental sources of antibiotic resistance and corresponding control measures. ARB, antibiotic-resistant bacteria; ARGs, antibiotic resistance genes; IPC, infection prevention and control; WWTP, wastewater treatment plant.

Source	What enters the environment	Key evidence	Hygiene and control measures
Domestic sewage	Antibiotic residues, gut-derived ARB, ARGs	WWTPs are resistance hotspots [17]	Universal safe sanitation; upgraded and advanced treatment; safe sludge handling
Hospitals	Last-resort antibiotics, multidrug-resistant bacteria	Concentrated residues in effluent [16]	On-site effluent pre-treatment; IPC; safe disposal of pharmaceutical waste
Pharmaceutical industry	Very high antibiotic concentrations	Ciprofloxacin above therapeutic levels [19]	WHO manufacturing guidance; discharge limits; responsible procurement [21]

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Source	What enters the environment	Key evidence	Hygiene and control measures
Livestock and aquaculture	Residues, ARB and ARGs via manure and water	Rising animal antimicrobial use [22]	End growth promoters; vaccination; farm biosecurity; manure treatment
Metals and biocides	Co-selecting agents	Shared mobile genetic elements [23]	Industrial pollution control; prudent biocide use

6. Routes of Human Exposure

People come into contact with environmental resistance through several routes. **Drinking water** is a key route wherever water treatment and distribution are inadequate, as illustrated by the detection of NDM-1-carrying bacteria in tap water [12]. **Food** is another: crops irrigated with wastewater or fertilised with manure, as well as meat and seafood, can carry resistant bacteria. **Recreational water** also matters. In the United Kingdom, a study of surfers and other coastal water users found that regular surfers were several times more likely than non-surfers to have their guts colonised by *E. coli* resistant to third-generation cephalosporins [24]. Finally, **occupational exposure** affects farmers, abattoir staff, sanitation workers and wastewater treatment personnel. Colonisation is usually silent, but colonised people can later develop resistant infections or pass resistant bacteria to others.

7. Health and Economic Burden

The Global Research on Antimicrobial Resistance (GRAM) project estimated that, in 2021, 1.14 million deaths were directly attributable to bacterial AMR and 4.71 million were associated with it. Without further action, attributable deaths are forecast to rise to 1.91 million per year by 2050, with a cumulative 39 million deaths attributable to AMR between 2025 and 2050. South Asia is expected to bear the largest share, with 11.8 million attributable deaths over that period [25]. WHO surveillance data show marked regional differences, with resistance highest in the South-East Asia and Eastern Mediterranean Regions (Figure 2) [4]. Even in high-income countries the burden is

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substantial: the CDC estimated that more than 2.8 million antibiotic-resistant infections and over 35,000 deaths occur each year in the United States [26].

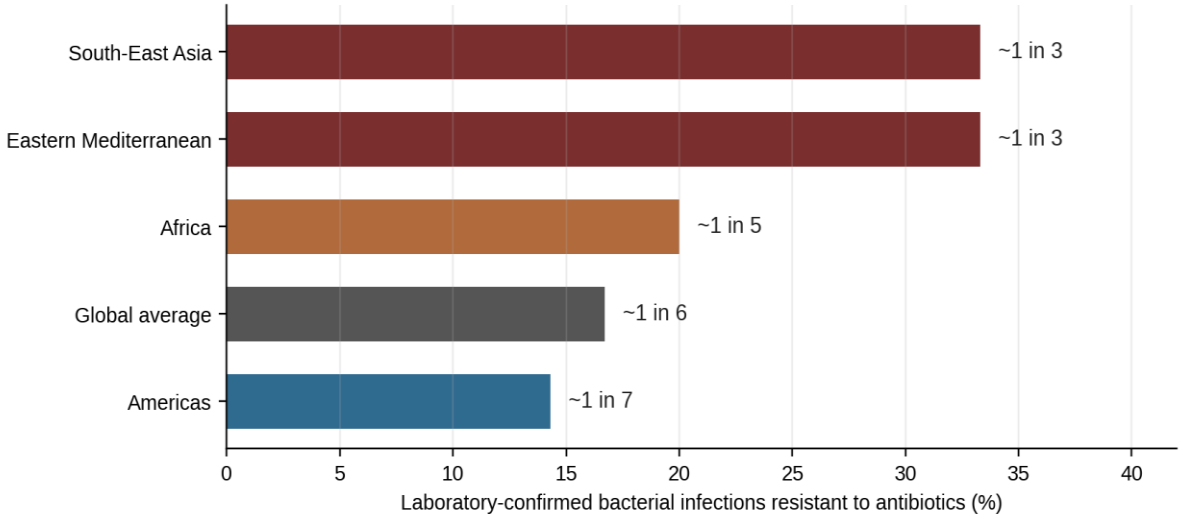


Figure 2. Approximate proportion of laboratory-confirmed bacterial infections resistant to antibiotics in 2023, by WHO region (regions with reported summary estimates shown). Figure prepared by the authors from data in [4].

Resistance also threatens the foundations of modern medicine. Surgery, caesarean sections, cancer chemotherapy, organ transplantation and the care of premature infants all depend on effective antibiotics to prevent and treat infection. Resistant infections lead to longer hospital stays, the use of more expensive and more toxic second-line drugs, and higher mortality. The World Bank estimated that, in a high-impact scenario, AMR could reduce global economic output by 3.8% by 2050 and push up to 28 million additional people into extreme poverty [27]. The earlier Review on Antimicrobial Resistance had projected that AMR could cause 10 million deaths a year by 2050 [28]; although subsequent estimates have refined this figure, it was instrumental in placing AMR on the global political agenda.

8. Prevention and Control: The Central Role of Hygiene

Because resistant bacteria spread through the same routes as other faecal and environmental pathogens, many of the most effective defences against AMR are classic hygiene and public health measures. Every infection prevented is an antibiotic course that is never needed.

8.1 Water, sanitation and hygiene (WASH)

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Universal access to safe drinking water, safely managed sanitation and handwashing with soap reduces the transmission of resistant gut bacteria within communities and lowers the overall burden of infection, and therefore the demand for antibiotics. The association between poor sanitation infrastructure and high resistance levels makes WASH investment one of the most powerful, and most neglected, AMR interventions [6,15].

8.2 Infection prevention and control in healthcare

In hospitals and clinics, hand hygiene, environmental cleaning and disinfection, screening and isolation of patients carrying multidrug-resistant organisms, and safe management of medical and pharmaceutical waste limit both patient-to-patient spread and release into the environment. Pre-treatment of hospital effluent before it reaches municipal sewers is an additional safeguard [16].

8.3 Improved wastewater treatment

Advanced treatment technologies such as ozonation, ultraviolet disinfection, membrane filtration and activated carbon can further reduce resistant bacteria and genes in treated effluent, while careful handling of sewage sludge prevents contamination of agricultural land [17,29]. Where centralised treatment is not yet affordable, expanding basic sanitation coverage remains the priority.

8.4 Controlling pollution from manufacturing

Applying the WHO manufacturing guidance, introducing enforceable discharge limits based on resistance-selection thresholds, and rewarding cleaner production in public procurement can sharply reduce one of the most concentrated sources of environmental antibiotics [10,21].

8.5 Antibiotic stewardship in humans and animals

Prudent use reduces the quantity of antibiotics excreted into the environment. WHO's AWaRe classification groups antibiotics into Access, Watch and Reserve categories to guide prescribing [30], and the 2024 UN political declaration set the target that at least 70% of antibiotics used in human health should come from the Access group [31]. In animal production, ending growth promotion, improving vaccination and farm biosecurity, and restricting antibiotics that are critically important for human medicine are central measures.

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8.6 Environmental surveillance

Monitoring of sewage offers a low-cost, non-invasive way to track resistance across entire populations, including healthy people who are never tested in clinics [14]. Integrating environmental data with human and animal surveillance allows earlier detection of emerging resistance and better targeting of interventions.

8.7 Public awareness and education

Individuals can contribute by:

- not demanding or using antibiotics for viral illnesses such as colds and influenza;
- taking antibiotics only when prescribed by a qualified health professional and following the prescriber's instructions;
- never sharing antibiotics or using leftover medicines;
- returning unused medicines to pharmacies or take-back schemes instead of flushing them or throwing them away;
- practising good hand hygiene and safe food preparation.

9. Global and National Policy Responses

The WHO Global Action Plan on Antimicrobial Resistance, adopted in 2015, set five strategic objectives: improving awareness and understanding, strengthening surveillance and research, reducing infection through sanitation, hygiene and infection prevention, optimising antimicrobial use, and increasing sustainable investment [32]. Its implementation is coordinated through the Quadripartite partnership of the Food and Agriculture Organization (FAO), UNEP, WHO and the World Organisation for Animal Health (WOAH). In September 2024, world leaders at the UN General Assembly adopted a political declaration committing to reduce the estimated 4.95 million deaths associated with bacterial AMR by 10% by 2030, to ensure that at least 60% of countries have funded national action plans, to meaningfully reduce antimicrobial use in agrifood systems, and to prevent and address the discharge of antimicrobials into the environment [31]. Nevertheless, the environmental dimension remains one of the least developed components of many national action plans [6].

10. Challenges and Research Gaps

Important gaps remain. There are as yet no widely adopted regulatory limits for resistance genes in drinking water or effluents, and methods for measuring them are

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not fully standardised. The dose-response relationship between environmental exposure and human infection is still poorly understood [8]. Surveillance capacity is weakest in the regions with the highest burden, and many countries still do not report data to global systems [4]. The pipeline of new antibiotics, particularly against Gram-negative bacteria, remains thin [11]. Addressing these gaps will require sustained funding, interdisciplinary research and closer cooperation between the health, environmental, agricultural and industrial sectors.

11. Implications for Hygiene and Public Health Practice

For students and professionals in hygiene and public health, antibiotic resistance reinforces a simple principle: preventing infection is better than treating it. Health workers can lead by example in hand hygiene and rational prescribing, educate patients and communities, advocate for safe water and sanitation, support environmental monitoring, and promote the One Health perspective in their institutions. Annual campaigns such as World AMR Awareness Week offer opportunities for student-led outreach in schools, communities and healthcare facilities.

12. Conclusion

Antibiotic resistance is no longer only a hospital problem. Antibiotics, resistant bacteria and resistance genes are released into the environment from homes, hospitals, factories and farms, and they return to people through water, food and everyday contact. This makes antibiotic resistance a genuine and growing environmental health problem. The tools to slow it are largely known: safe water and sanitation, good hygiene, infection prevention, cleaner manufacturing, better wastewater treatment, prudent antibiotic use and integrated surveillance. Applying them together, through a One Health approach, is essential to preserve effective antibiotics for future generations.

Declarations

Author contributions: All authors contributed to the conception of the review, literature search, drafting and critical revision of the manuscript. All authors read and approved the final version and agree to be accountable for its content.

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Use of AI tools: During the preparation of this manuscript, the authors used Claude (Anthropic), an AI assistant, to support drafting, language editing and formatting. The authors reviewed and edited the content, verified the cited sources, and take full responsibility for the content of this publication.

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GUT–BRAIN AXIS: A NEW PERSPECTIVE ON EATING BEHAVIOUR

Danish Abdullah , Medical Student, Samarkand state medical university, Uzbekistan

Nayaab Shamim , Medical Student, Samarkand state medical university, Uzbekistan

Anshika Pandey , Medical Student, Samarkand state medical university, Uzbekistan

Mohd Rafe , Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Eating is often described as a simple response to hunger, but everyday food choices are shaped by a much richer conversation between the gastrointestinal tract and the brain. The gut–brain axis integrates mechanical distension, nutrients, gut hormones, microbial metabolites, vagal signals and central reward pathways to influence hunger, satiation, satiety and food preference. This narrative review presents a human-centred view of how that communication may help explain why people sometimes eat when they are not physiologically hungry, stop eating before energy needs are fully restored, or find highly palatable foods unusually difficult to resist. Particular attention is given to ghrelin, glucagon-like peptide-1 (GLP-1), peptide YY (PYY), cholecystokinin (CCK), the vagus nerve and the intestinal microbiota. Current evidence suggests that gut microbes and their metabolites may modify appetite signalling, although human studies remain heterogeneous and do not support the idea that a single 'good' or 'bad' microbiome determines eating behaviour. The gut–brain axis is therefore best understood as a flexible biological network interacting with sleep, stress, food environment, learning and reward.

Keywords: gut–brain axis; microbiota–gut–brain axis; eating behaviour; appetite; satiety; gut hormones; GLP-1; ghrelin; PYY; vagus nerve; food reward; nutrition.

1. Introduction

Why do we feel hungry at one time of day but not another? Why can the smell or sight of food trigger interest even after a meal, while stress or illness can make food unappealing? These everyday experiences show that eating behaviour is not controlled by the stomach alone. It is the outcome of continuous communication among the gastrointestinal tract, nervous system, endocrine signals, immune pathways, microbial communities and the brain. The term gut–brain axis describes this two-way communication network. Recent work places the axis at the centre of research on appetite and food intake because it connects basic physiological needs with motivation, reward and learned behaviour.

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The modern perspective is useful because it replaces a simple 'hungry versus full' model with a dynamic one. The gut reports what has entered the digestive tract, how much is present and which nutrients are being processed. The brain combines these signals with memories, sensory cues, emotions, habits and the availability of food. Eating behaviour therefore reflects both homeostatic regulation—maintaining energy balance—and hedonic regulation, in which food can be rewarding even when energy needs are already met.

This review discusses the gut–brain axis as a new lens for understanding eating behaviour. It focuses on established gut hormones, neural communication and emerging evidence concerning the microbiota, while also highlighting an important limitation: much of the mechanistic literature comes from animal models, and effects observed in experimental settings do not automatically translate into predictable changes in human eating.

2. How the Gut Talks to the Brain

Communication begins as soon as food reaches the gastrointestinal tract. Stretch receptors detect distension, while enteroendocrine cells sense nutrients and release signalling molecules. These messages can reach the brain through the vagus nerve, circulating hormones and metabolites, and neuroendocrine pathways. Brainstem and hypothalamic networks then integrate incoming information with higher brain regions involved in motivation and reward.

A meal can therefore be viewed as a conversation rather than a one-way event. The gut does not merely receive food; it actively reports the meal back to the brain. This helps explain why the same amount of food can produce different experiences of hunger or fullness depending on nutrient composition, eating speed, previous meals, physiological state and environmental cues.

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Figure 1. Conceptual map of gut–brain signalling in eating behaviour

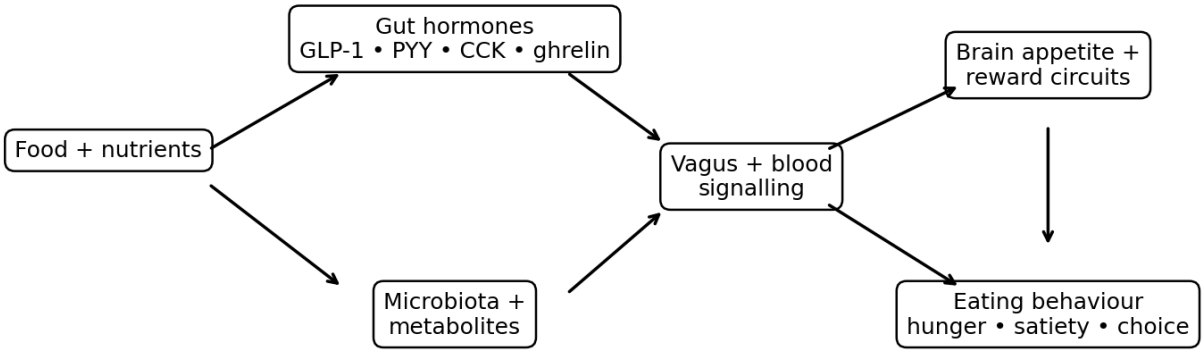


Figure 1. Conceptual map of gut–brain signalling. This is an educational schematic, not a quantitative effect-size model.

3. Gut Hormones: The Chemical Language of Appetite

Several gut-derived hormones are central to appetite research. Ghrelin is commonly described as an orexigenic, or hunger-promoting, signal, while GLP-1, PYY and CCK generally contribute to satiation or satiety. These hormones do not act in isolation; they interact with one another and with glucose, insulin, leptin and neural signals. Brain-imaging research shows that gut-related signals can influence regions involved in appetite, motivation and food-related processing.

Signal	Main source	Typical role	Behavioural relevance
Ghrelin	Stomach and upper gut	Promotes hunger	Helps signal energy need and can increase food motivation.
GLP-1	Intestinal enteroendocrine cells	Promotes satiation/satiety	Links nutrient arrival with reduced food intake and

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			metabolic regulation.
PYY	Distal intestine	Promotes satiety	Helps extend the post-meal period before hunger returns.
CCK	Small intestine	Promotes satiation	Responds to nutrients and contributes to meal termination.

Figure 2. Major gut-hormone signals and appetite

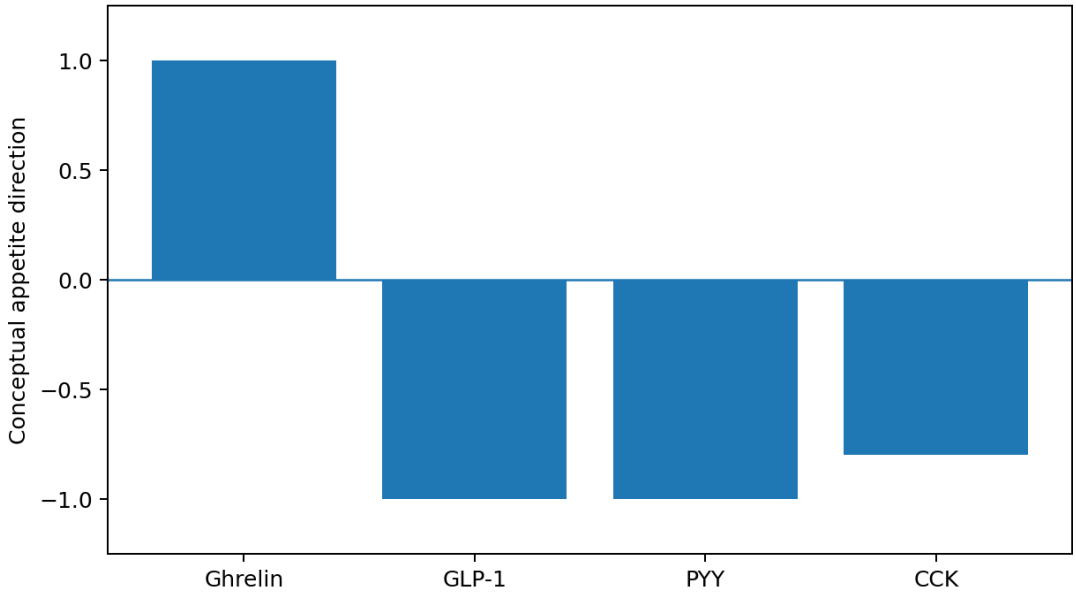


Figure 2. Conceptual direction of major gut-hormone appetite signals; not a measurement of hormone concentrations or effect sizes.

4. From Gut Microbes to Food Motivation

The microbiota adds another layer to the gut–brain conversation. Intestinal microorganisms metabolize dietary components and generate compounds such as short-chain fatty acids, bile-acid derivatives and other signalling molecules. These products can interact with intestinal cells, immune pathways and neural or endocrine signalling. Recent reviews describe microbial metabolites as important potential mediators of appetite regulation, while emphasizing that the field is still developing.

The most interesting possibility is that microbes may influence eating behaviour indirectly rather than acting like a hidden controller of food choice. Changes in

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microbial metabolism can alter the chemical environment of the gut, which may affect hormone secretion, nutrient sensing and communication with the nervous system. However, human evidence is not yet strong enough to say that a particular bacterial species will reliably make a person crave or avoid a particular food. A systematic review of 37 studies found associations between the gut microbiome and appetite, appetite hormones, food intake or weight-related outcomes in many studies, but probiotic and synbiotic findings were inconsistent.

This distinction matters because microbiome research can easily be oversimplified in popular health discussions. Eating behaviour is shaped by biology, but it is also shaped by culture, family routines, food availability, stress, sleep, learning and personal preference. The microbiome is one participant in that network, not a single master switch.

5. Homeostatic Hunger Meets Food Reward

A useful way to understand eating behaviour is to separate homeostatic and hedonic influences. Homeostatic eating is closely tied to energy needs and physiological signals that promote or reduce food intake. Hedonic eating involves pleasure, reward, attention and learned associations. In real life, these systems overlap. A person may be physiologically satisfied but still interested in dessert because its taste, smell, social meaning or previous associations activate reward-related processes.

This perspective makes 'willpower' an incomplete explanation for eating behaviour. Biological signals can change the perceived value of food, while the environment can repeatedly expose people to highly rewarding food cues. The brain must continuously decide whether a food cue is worth acting on, and that decision is influenced by both internal state and context.

6. Gut–Brain Signalling and Modern Eating Environments

Modern food environments can challenge biological systems that evolved to protect against energy shortage. Foods that combine intense sweetness, saltiness, fat, aroma and texture may be especially attention-grabbing. Large portions and constant availability can also reduce the natural boundaries between meals. The gut–brain axis does not create these environmental pressures, but it helps explain why repeated exposure can interact with physiology and learning.

Sleep and stress are additional modifiers. Poor sleep, psychological stress and irregular routines can change appetite, food choice and meal timing, making the overall system even more complex. For this reason, interventions that focus only on a single nutrient or a single hormone may overlook the broader behavioural context.

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7. Factors That Can Shape Eating Behaviour Through the Gut–Brain Network

Factor	Gut-level signal	Brain/behavioural influence	Practical interpretation
Meal size/composition	Distension and nutrient sensing	Changes satiation and post-meal satiety	Meal quality and pace can influence fullness.
Dietary fibre	Fermentation and microbial metabolites	May affect satiety-related signalling	Fibre-rich foods support a broader gut environment.
Highly palatable foods	Nutrient and sensory inputs	Can engage reward and learned cues	Eating is not governed by hunger alone.
Stress	Changes gut function and appetite signalling	May alter cravings or appetite	Emotional state can modify eating patterns.
Sleep disruption	May alter endocrine/metabolic regulation	Can affect appetite and food choice	Regular sleep supports healthy appetite regulation.
Microbiota composition	Metabolites and host signalling	Potential effects on appetite pathways	Associations are promising but not deterministic.

8. Clinical and Public-Health Perspective

Understanding the gut–brain axis may broaden how clinicians and educators talk about eating behaviour. Instead of framing appetite as a simple test of self-control, it may be more helpful to consider sleep, stress, meal patterns, food environment, digestive symptoms, medications and metabolic health alongside dietary intake. This does not mean every appetite change is caused by the microbiome, nor that microbiome-targeted products are universally effective.

Gut-hormone research has already influenced modern metabolic therapeutics. GLP-1-based medicines demonstrate that manipulating physiological appetite pathways can change food intake and metabolic outcomes. At the same time, pharmacological effects should not be confused with proof that ordinary foods or supplements can reproduce

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the same results. Current evidence supports continued research rather than simplistic promises.

9. What the Evidence Still Cannot Tell Us

Three questions remain particularly important. First, causality is difficult to establish: does a change in microbiota cause altered eating behaviour, or does a change in diet reshape the microbiota? In many human studies, both processes may occur at the same time. Second, individual variation is substantial. Age, genetics, medication exposure, habitual diet and health status can all influence gut microbial communities and appetite signalling. Third, animal studies are valuable for mechanism but cannot by themselves establish how a pathway operates in everyday human life. The most useful future research will combine detailed dietary records, objective metabolic measures, microbiome profiling, brain imaging and long-term behavioural follow-up.

10. A Humanized View: Eating Is a Conversation

The most important shift offered by the gut–brain axis is conceptual. Eating is not simply a message sent from the brain to the stomach, nor is it simply a response to an empty stomach. It is an ongoing conversation. The gut senses the meal, hormones carry information, nerves transmit rapid signals, microbes transform nutrients, and the brain interprets all of this alongside memories, emotions and the surrounding food environment.

Seeing eating behaviour in this way can reduce blame and increase curiosity. A person who struggles with appetite is not necessarily lacking discipline; their behaviour may reflect many biological and environmental signals arriving at the same time. That does not remove personal agency, but it places behaviour in a more realistic physiological context. The future of nutrition may therefore depend less on searching for a single perfect diet and more on understanding how whole patterns of eating, sleep, stress, activity and food environments interact with individual biology.

11. Conclusion

The gut–brain axis provides a powerful framework for understanding eating behaviour as a dynamic interaction between physiology and experience. Gut hormones such as ghrelin, GLP-1, PYY and CCK communicate information about nutrient availability and meal completion, while the vagus nerve and circulating signals connect the gastrointestinal tract with brain networks that regulate appetite, satiety and reward. The microbiota adds another layer by producing metabolites that may influence intestinal, endocrine, immune and neural pathways. Current human evidence is promising but heterogeneous, so microbiome–behaviour relationships should be interpreted

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cautiously. Overall, eating behaviour is best viewed as the product of several interacting systems rather than a simple matter of hunger or willpower. A more complete understanding of these systems may support future nutritional and clinical strategies that are more individualized, biologically informed and compassionate.

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SLEEP QUALITY AND ASSOCIATED FACTORS AMONG MEDICAL STUDENTS

Muhammad Moeez, Medical Student, Samarkand state medical university, Uzbekistan

Aryan Tomar , Medical Student, Samarkand state medical university, Uzbekistan

Faisal Khan , Medical Student, Samarkand state medical university, Uzbekistan

Ruthra Akash , Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Sleep is one of the most basic biological requirements, yet it is often treated as negotiable during medical education. Medical students commonly move between lectures, examinations, clinical duties, commuting, study sessions and social responsibilities, creating a routine in which sleep may be shortened or fragmented. This narrative review examines sleep quality among medical students and the factors associated with it, including academic workload, stress and anxiety, clinical schedules, electronic-device use, irregular routines, lifestyle habits and the learning environment. Evidence from large systematic reviews indicates that poor sleep quality is common in this population. A worldwide meta-analysis involving more than 59,000 medical students estimated poor sleep quality in about 55.6% of participants, while average sleep duration was approximately 6.5 hours per night. The Pittsburgh Sleep Quality Index (PSQI) is frequently used to assess sleep quality and provides a structured measure of subjective sleep and related daytime problems. Poor sleep is important not only because students feel tired; it may also affect concentration, memory, mood, academic functioning and overall wellbeing. The review argues that sleep should be treated as part of medical education and student health rather than as an optional personal habit. Practical solutions include sleep-health education, realistic scheduling, early identification of persistent sleep problems, accessible psychological support and a learning culture that does not reward chronic sleep restriction.

Keywords: sleep quality; medical students; sleep deprivation; Pittsburgh Sleep Quality Index; insomnia; daytime sleepiness; academic stress; anxiety; clinical training; sleep hygiene; student wellbeing.

1. Introduction

Medical education is intellectually demanding and emotionally intense. Students are expected to learn large volumes of information, perform in examinations, adapt to

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clinical environments and gradually develop the judgement and communication skills required for healthcare practice. In this setting, sleep can easily become the first thing sacrificed when time feels limited. The problem is that sleep is not simply a period when the body is inactive. It is an active biological process involved in memory consolidation, emotional regulation, attention, immune function and recovery.

Research consistently suggests that sleep problems are common among medical students. A comprehensive meta-analysis of 57 studies involving 25,735 medical students estimated a pooled prevalence of poor sleep quality of 52.7% when the PSQI was used. A much larger worldwide review of 109 studies and 59,427 participants estimated poor sleep quality at 55.64% and average sleep duration at about 6.5 hours per night. These figures should not be interpreted as a diagnosis for every student, but they demonstrate that sleep disturbance is a widespread student-health issue rather than an occasional inconvenience. □cite□turn0search4□turn0search12□

This narrative review therefore looks beyond the question of how many medical students sleep poorly. It asks why sleep quality becomes difficult during medical training, what factors repeatedly appear in the literature, how poor sleep can affect daily functioning, and what medical schools and students can realistically do about it.

2. Understanding Sleep Quality

Sleep quality is broader than sleep duration. A student may spend seven hours in bed but still wake repeatedly, take a long time to fall asleep, feel unrefreshed, or experience excessive daytime sleepiness. The Pittsburgh Sleep Quality Index is one of the most widely used research instruments in this area. Developed by Buysse and colleagues, the PSQI assesses sleep over the previous month using seven components: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication and daytime dysfunction. □cite□turn0search0□

The PSQI is useful because it captures the student's experience rather than focusing only on hours slept. Its measurement properties have been evaluated across clinical and non-clinical populations, with evidence supporting its reliability and validity as a screening measure. □cite□turn0search8□

For medical students, this distinction is particularly important. A timetable may appear to provide enough sleep opportunity, but anxiety before examinations, late-night studying, early clinical rounds, night duties or constant phone notifications can make actual sleep less restorative.

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3. How Common Is Poor Sleep Among Medical Students?

The prevalence estimates vary between studies because researchers use different definitions, populations, countries and questionnaires. Even so, the overall pattern is remarkably consistent: a substantial proportion of medical students report poor sleep. The worldwide meta-analysis found poor sleep quality in 55.64% of students, excessive daytime sleepiness in 33.32%, and an average sleep duration of 6.5 hours per night. □cite□turn0search12□

Figure 1. Estimated prevalence of poor sleep quality among medical students

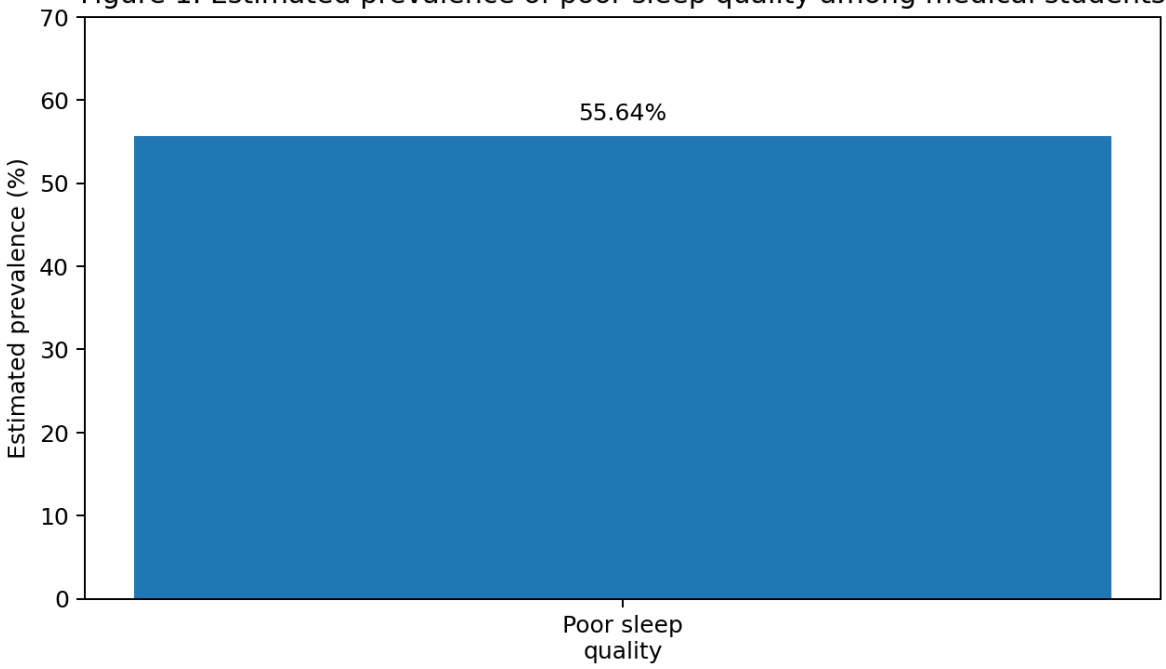


Figure 1. Estimated pooled prevalence of poor sleep quality among medical students from a worldwide meta-analysis. This figure reproduces the published pooled estimate and should not be interpreted as a prevalence estimate for a particular college or country.

Regional evidence also shows substantial sleep disturbance. A systematic review and meta-analysis of medical students in the Middle East and North Africa reported high levels of insomnia, excessive daytime sleepiness and circadian sleep–wake problems, with female sex, later academic years, anxiety, excessive internet use and poor academic performance appearing among significant associated factors in the included studies. □cite□turn0search3□

More recent evidence from Saudi Arabia similarly highlights a high burden of insomnia and excessive daytime sleepiness and identifies psychological distress, academic stage and lifestyle factors among important correlates. These studies reinforce the idea that

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sleep quality is influenced by the entire student environment rather than by one isolated behaviour. □cite□turn0search5□

4. Academic Workload and Examination Pressure

The academic structure of medical education is a major reason sleep becomes vulnerable. The volume of material can encourage students to study late into the night, especially before examinations. Some students use the quiet hours of the night because they believe they can concentrate better without interruptions. Others simply feel that sleeping means losing valuable study time.

This creates a misleading trade-off. An extra hour of studying may feel productive in the moment, but chronic sleep restriction can make attention, working memory, learning efficiency and emotional control more difficult. The relationship can also become circular: a tired student may study less efficiently, fall behind, experience more stress and then stay awake even later trying to catch up.

Clinical training adds another layer. Early morning rounds, long hospital days, commuting and occasional night duties can produce irregular sleep schedules. Students in later years may face a particularly complex combination of academic responsibility and clinical exposure. Regional evidence has repeatedly identified academic stage and clinical demands as relevant correlates of sleep disturbance. □cite□turn0search3□turn0search5□

5. Stress, Anxiety and Emotional Wellbeing

Sleep and psychological wellbeing are closely connected. Medical students may experience examination anxiety, fear of poor performance, uncertainty about career progression, financial concerns, relationship difficulties and the emotional burden of encountering illness in clinical settings. When the mind remains highly alert at night, falling asleep can become difficult even when the body is tired.

The relationship also works in the opposite direction. Poor sleep can leave students more irritable, emotionally reactive and less able to cope with ordinary academic demands. Recent evidence specifically examining medical students has found an association between poor sleep and mental-health outcomes such as depression, anxiety and burnout, while also emphasizing that most available studies are cross-sectional and therefore cannot prove that poor sleep directly causes these conditions. □cite□turn0search7□

This bidirectional relationship is important. Telling a distressed student simply to 'sleep more' may be unrealistic if the underlying problem is severe anxiety or an unhealthy

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workload. Sleep support should therefore be connected to broader student wellbeing and mental-health services.

6. Electronic Devices, Internet Use and Evening Habits

Smartphones, laptops and tablets are now embedded in medical education. Students may use them for lectures, question banks, clinical references and communication. The same devices can also extend study into the night and keep students mentally engaged when they should be winding down.

Evidence from regional reviews identifies excessive internet use as an associated factor for sleep disorders among medical students. The mechanism is probably not one-dimensional: prolonged screen exposure may delay bedtime, social media can increase cognitive arousal, and online studying can blur the boundary between academic and personal time. □cite□turn0search3□

The practical lesson is not that students should avoid technology altogether. Rather, students may benefit from creating a predictable transition between study and sleep, reducing unnecessary notifications near bedtime and avoiding the habit of turning the bed into a permanent study station.

7. Lifestyle and Environmental Factors

Factor	How it may affect sleep	Possible student experience	Helpful response
Academic workload	Delays bedtime and reduces sleep opportunity	Late-night studying and morning fatigue	Plan study blocks earlier; protect a consistent sleep window.
Stress/anxiety	Increases mental arousal and disrupts sleep continuity	Racing thoughts or difficulty settling down	Use counselling/support when stress is persistent; develop a wind-down routine.
Clinical schedules	Create early starts or irregular hours	Short sleep or changing sleep times	Coordinate schedules where possible and protect recovery time.

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Internet/device use	May delay bedtime and maintain cognitive arousal	'Just one more hour' of scrolling/studying	Set a device boundary before sleep and silence non-essential notifications.
Caffeine and stimulants	Can interfere with sleep when used late	Difficulty falling asleep despite fatigue	Keep caffeinated drinks earlier in the day when possible.
Poor sleep environment	Noise, light or uncomfortable conditions interrupt sleep	Frequent waking or unrefreshing sleep	Improve darkness, quiet, temperature and comfort.
Sedentary routine	May reduce healthy day–night rhythm	Low energy and irregular routine	Include regular daytime movement and outdoor light exposure.

8. Commonly Reported Associated Factors

Figure 2. Commonly reported factors associated with poorer sleep

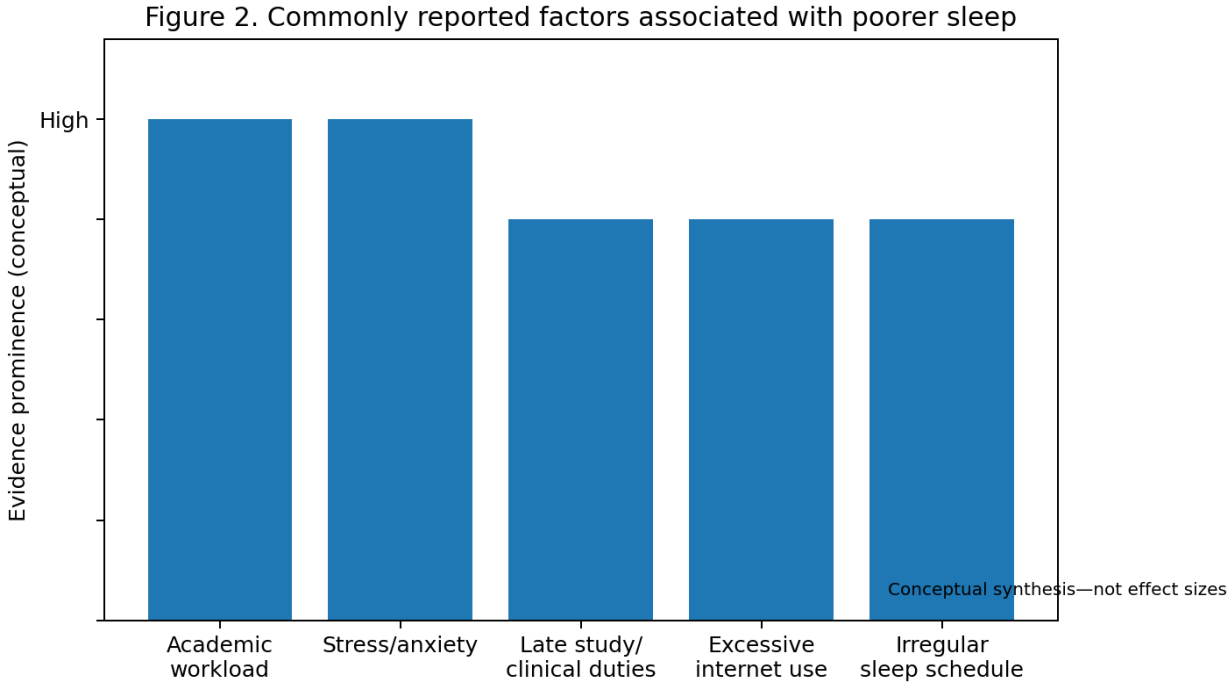


Figure 2. Conceptual synthesis of factors repeatedly reported in the literature as associated with poorer sleep among medical students. The bars represent evidence prominence in this review, not measured effect sizes.

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Across the literature, no single factor explains poor sleep. Instead, medical students often experience several pressures simultaneously. A student may be studying for an examination, worrying about performance, using a laptop late at night, waking early for clinical duties and relying on caffeine the next morning. Each factor can reinforce the others.

This cumulative perspective is more useful than searching for a single culprit. It also explains why interventions need to operate at both individual and institutional levels.

9. Sleep Quality and Academic Performance

Students often sacrifice sleep because they believe it will improve academic performance. The evidence is more nuanced. Poor sleep has been associated with daytime dysfunction and, in some studies, poorer academic outcomes, but findings are not completely consistent. Regional meta-analytic evidence reports that sleep disorders can negatively affect academic performance, while some individual studies have found weak or no significant associations. □cite□turn0search3□turn0search5□

The uncertainty is understandable. Academic performance depends on many factors, including prior knowledge, teaching quality, study technique, mental health, socioeconomic circumstances and examination design. Sleep is one part of this larger picture. Still, the absence of a perfect causal estimate should not be interpreted as evidence that sleep is unimportant. Attention, memory and daytime functioning are biologically linked to adequate sleep, making sleep health a reasonable part of an effective learning strategy.

10. Practical Approaches for Medical Students

A realistic approach to sleep should focus on consistency rather than perfection. Students can begin by protecting a regular sleep opportunity, avoiding unnecessary all-night study sessions, keeping caffeine away from late hours, and creating a short period of reduced academic stimulation before bed. Daytime light exposure and regular physical activity can also support a stable sleep–wake rhythm.

Medical schools have an equally important role. Orientation programmes can teach students how sleep affects learning and health. Timetables can avoid unnecessary scheduling patterns that repeatedly force very early starts after late academic activities. Student counselling should be accessible without stigma, and persistent sleep problems should be evaluated rather than normalized as simply part of medical school.

Importantly, sleep education should not become another demand placed on already overwhelmed students. If the workload is structurally excessive, telling students to

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manage their time better will not solve the problem. Institutional design and student behaviour have to be considered together.

11. Discussion

The evidence portrays sleep quality among medical students as a population-level wellbeing issue. More than half of students in large pooled analyses have been classified as having poor sleep quality, and the average sleep duration reported in the worldwide literature is shorter than the commonly recommended 7–9 hours for adults. □cite□turn0search12□

At the same time, prevalence estimates should be interpreted carefully. Most studies rely on self-reported questionnaires and cross-sectional designs. A PSQI score identifies a pattern of poor sleep but does not by itself establish a specific sleep disorder or prove causality. Differences in culture, curriculum, academic calendars and assessment methods also make direct comparison between countries difficult.

Future research would benefit from longitudinal designs, objective measures such as actigraphy where appropriate, repeated assessment across examination and non-examination periods, and intervention studies. It would also be useful to examine whether changes in curriculum, clinical scheduling or institutional sleep-health programmes produce measurable improvements in student wellbeing and academic functioning.

12. Conclusion

Sleep quality is an important but often underestimated component of medical student health. The available literature shows that poor sleep is common and is associated with a complex mixture of academic pressure, clinical schedules, psychological distress, electronic-device use, lifestyle habits and irregular routines. Large meta-analyses suggest that roughly half of medical students experience poor sleep quality, while many report insufficient sleep or excessive daytime sleepiness. □cite□turn0search4□turn0search12□

The central message is simple: sleep should not be treated as the leftover time after studying is finished. It is part of the process that makes learning, emotional regulation and safe professional development possible. A humanized approach recognizes that students are not machines that can exchange sleep for productivity indefinitely. Protecting sleep requires both individual habits and educational environments that make healthy sleep realistically achievable.

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JAZO TAYINLASHDA ODILLIK VA INSONPARVARLIK PRINSIPLARINING O‘ZARO UYG‘UNLIGI

Xolmo’minova Hilola Toshnazar qizi

Termiz davlat universiteti Yuridik fakulteti 2-bosqich talabasi

Burxonov Dilnur Muxiddin o’g’li

Termiz davlat universiteti Yuridik fakulteti 2-bosqich talabasi

Ilmiy rahbar: **Allanazarova Farida Ilxomovna**

Termiz davlat universiteti o'qituvchisi

ORCID: https://orcid.org/0009-0000-8852-7517

ANNOTATSIYA

Mazkur maqolada O‘zbekiston Respublikasi jinoyat qonunchiligida jazo tayinlash jarayonida odillik va insonparvarlik prinsiplarining o‘zaro uyg‘unligini ta’minlashning nazariy-huquqiy asoslari hamda amaliy ahamiyati tadqiq etilgan. Jinoyat kodeksining 7, 8, 42, 54, 55 va 56-moddalarida belgilangan normalar asosida jazoning mutanosibligi, individuallashtirilishi, mahkumning shaxsiy xususiyatlarini hisobga olish va ozodlikdan mahrum qilish bilan bog‘liq bo‘lmagan jazo choralarini qo‘llash imkoniyatlari tahlil qilingan. Tadqiqotda formal-yuridik, tizimli, qiyosiy-huquqiy va mantiqiy tahlil usullaridan foydalanilgan. Odillik va insonparvarlik prinsiplarining o‘zaro munosabati faqat jazo miqdorini belgilash bilan cheklanmasligi, balki jinoyatning ijtimoiy xavflilik darajasi, aybdorning shaxsi, jabrlanuvchining qonuniy manfaatlari hamda mahkumni qayta ijtimoiylashtirish imkoniyatlarini kompleks baholashni talab etishi asoslangan. Tadqiqot natijasida sud qarorlarining asoslantirilganligini kuchaytirish, muqobil jazolarni tanlash mezonlarini takomillashtirish va jazo tayinlashda individual yondashuvni rivojlantirish yuzasidan ilmiy-amaliy takliflar ishlab chiqilgan.

Kalit so‘zlar: jinoyat huquqi, jazo tayinlash, odillik prinsipi, insonparvarlik prinsipi, jazoning mutanosibligi, jazoni individuallashtirish, sud diskretsiyasi, muqobil jazo, ijtimoiy adolat.

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АННОТАЦИЯ

В статье исследуются теоретико-правовые основы и практическое значение обеспечения взаимосвязи принципов справедливости и гуманизма при назначении уголовного наказания в Республике Узбекистан. На основе анализа статей 7, 8, 42, 54, 55 и 56 Уголовного кодекса рассматриваются вопросы соразмерности и индивидуализации наказания, учёта личности виновного, а также применения мер наказания, не связанных с лишением свободы. В исследовании использованы формально-юридический, системный, сравнительно-правовой и логический методы анализа. Обосновывается, что согласованное применение принципов справедливости и гуманизма предполагает комплексную оценку общественной опасности преступления, личности виновного, законных интересов потерпевшего и возможностей ресоциализации осуждённого. По результатам исследования предложены рекомендации по совершенствованию мотивировки судебных решений, уточнению критериев назначения альтернативных наказаний и развитию индивидуального подхода в судебной практике.

Ключевые слова: уголовное право, назначение наказания, принцип справедливости, принцип гуманизма, соразмерность наказания, индивидуализация наказания, судебное усмотрение, альтернативные наказания, социальная справедливость.

ABSTRACT

This article examines the theoretical and legal foundations and practical significance of harmonizing the principles of justice and humanism in criminal sentencing in the Republic of Uzbekistan. Based on Articles 7, 8, 42, 54, 55, and 56 of the Criminal Code, the study analyzes proportionality, individualized sentencing, consideration of the offender's personal circumstances, and the application of non-custodial sanctions. The research employs formal legal, systematic, comparative legal, and logical methods. It argues that the coordinated application of justice and humanism requires a comprehensive assessment of the seriousness of the offense, the offender's personality, the legitimate interests of victims, and the prospects for rehabilitation and social reintegration. The findings provide practical recommendations for strengthening the reasoning of judicial decisions, improving the criteria for alternative sentencing, and promoting individualized approaches in judicial practice.

Keywords: criminal law, sentencing, principle of justice, principle of humanism, proportionality, individualized punishment, judicial discretion, alternative sanctions, social justice.

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KIRISH

Jinoyat huquqi jamiyatda qonuniylikni ta’minlash, fuqarolarning huquq va erkinliklarini muhofaza qilish, ijtimoiy xavfli qilmishlarning oldini olish hamda buzilgan huquqiy tartibni tiklashga xizmat qiluvchi muhim huquqiy vositalardan biridir. Ushbu vazifalarni amalga oshirishda jinoyat sodir etgan shaxslarga nisbatan jazo tayinlash instituti alohida ahamiyat kasb etadi. Chunki sud tomonidan tayinlanadigan jazo nafaqat sodir etilgan qilmishga davlatning huquqiy munosabatini ifodalaydi, balki jinoyat sodir etgan shaxsning kelgusidagi xulq-atvoriga, uning jamiyatga qayta moslashuviga hamda fuqarolarning odil sudlovga bo‘lgan ishonchiga bevosita ta’sir ko‘rsatadi.

Jazo tayinlashda davlatning majburlov vakolatlari bilan shaxsning konstitutsiyaviy huquqlari o‘rtasida maqbul muvozanatni saqlash talab etiladi. Bunda odillik va insonparvarlik prinsiplari o‘zaro bog‘liq bo‘lgan, ayni paytda mustaqil huquqiy mazmunga ega mezonlar sifatida namoyon bo‘ladi. Odillik sodir etilgan jinoyat bilan qo‘llaniladigan jazo o‘rtasidagi mutanosiblikni talab etsa, insonparvarlik jazoning shaxs qadr-qimmatini kamsitmaydigan, uning axloqan tuzalishiga va yangi jinoyat sodir etishining oldini olishga xizmat qiladigan darajada qo‘llanishini nazarda tutadi.

O‘zbekiston Respublikasi Jinoyat kodeksining 7-moddasida insonparvarlik, 8-moddasida esa odillik prinsipi mustahkamlangan. Ushbu normalar mazmunidan kelib chiqib, jinoyat sodir etgan shaxsga nisbatan zarur va yetarli huquqiy ta’sir chorasini tanlash, jazoning og‘irligini jinoyatning xususiyati hamda aybdorning shaxsiga muvofiqlashtirish talab etiladi.

Mazkur prinsiplarni amalda uyg‘unlashtirish murakkab huquqiy baholash jarayonidir. Bir tomondan, haddan tashqari yengil jazo tayinlanishi jabrlanuvchining qonuniy manfaatlari va jamiyatda adolat haqidagi tasavvurlarga salbiy ta’sir ko‘rsatishi mumkin. Ikkinchi tomondan, jinoyatning aniq holatlari va shaxsning individual xususiyatlarini yetarlicha baholamasdan haddan tashqari og‘ir jazo belgilanishi mahkumning ijtimoiy aloqalari uzilishiga, qayta ijtimoiylashish imkoniyatlari cheklanishiga va jazoning tarbiyaviy salohiyati pasayishiga olib kelishi ehtimoli mavjud.

Shu sababli jinoyat sodir etgan shaxsga nisbatan jazo tayinlashda odillikni faqat qat’iy jazolash, insonparvarlikni esa faqat jazoni yengillashtirish bilan bog‘lash ilmiy jihatdan yetarli emas. Har ikkala prinsipning mazmuni ancha keng bo‘lib, ular jazoning qonuniyligi, asoslantirilganligi, zaruriyligi, mutanosibligi va shaxsga moslashtirilganligini ta’minlashga xizmat qiladi.

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Tadqiqotning maqsadi — O‘zbekiston Respublikasi jinoyat qonunchiligida jazo tayinlashda odillik va insonparvarlik prinsiplarining o‘zaro huquqiy bog‘liqligini ochib berish, ularni birgalikda qo‘llashda yuzaga kelishi mumkin bo‘lgan muammolarni aniqlash hamda sud amaliyotida mazkur prinsiplarning samarali amalga oshirilishiga qaratilgan takliflarni ishlab chiqishdan iborat.

Tadqiqotning ilmiy muammosi odil va insonparvar jazoning umumiy mezonlari qonunchilikda belgilangan bo‘lsa-da, ushbu mezonlarning muayyan jinoyat ishi bo‘yicha qanday muvozanatlashtirilishi suddan alohida huquqiy asoslantirishni talab etishida namoyon bo‘ladi.

TADQIQOT METODOLOGIYASI

Tadqiqot jinoyat-huquqiy normalarni doktrinal tahlil qilishga asoslangan bo‘lib, unda odillik va insonparvarlik prinsiplarining mazmuni, ularning jazo tayinlashning umumiy asoslari bilan munosabati hamda xalqaro standartlarga muvofiqligi o‘rganildi.

Tadqiqotning normativ asosini O‘zbekiston Respublikasi Jinoyat kodeksining 7, 8, 42, 54–56-moddalari, jazo tayinlashning boshqa tegishli qoidalari va O‘zbekiston Respublikasi Oliy sudi Plenumining 2006-yil 3-fevraldagi 1-sonli qarori tashkil etadi. Xalqaro qiyosiy mezon sifatida Birlashgan Millatlar Tashkilotining ozodlikdan mahrum qilish bilan bog‘liq bo‘lmagan choralar to‘g‘risidagi minimal standart qoidalari — Tokio qoidalari o‘rganildi.

Formal-yuridik usul yordamida qonunchilik normalarining mazmuni, huquqiy maqsadi va o‘zaro bog‘liqligi tahlil qilindi. Tizimli tahlil orqali odillik va insonparvarlik prinsiplari jazoning maqsadlari, jazoni individuallashtirish va yengillashtiruvchi holatlarni hisobga olish institutlari bilan birgalikda o‘rganildi. Qiyosiy-huquqiy usul milliy qonunchilikdagi yondashuvlarni xalqaro tavsiyalar bilan solishtirish imkonini berdi.

Mantiqiy tahlil va huquqiy modellashtirish asosida prinsiplar o‘rtasida yuzaga kelishi mumkin bo‘lgan ziddiyatli vaziyatlar ishlab chiqilib, ularni hal qilish mezonlari shakllantirildi.

Tadqiqot empirik sud statistikasi yoki real jinoyat ishlari bo‘yicha yig‘ilgan ma’lumotlarga asoslanmaydi. Shu bois natijalar normativ-huquqiy tahlil va nazariy modellashtirish xulosalari sifatida talqin qilinadi; sud amaliyotida mazkur yondashuvlarning haqiqiy samaradorligini aniqlash alohida empirik tadqiqotni talab etadi.

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TADQIQOT NATIJALARI

O‘tkazilgan normativ-huquqiy tahlil natijasida jazo tayinlashda odillik va insonparvarlik prinsiplarining o‘zaro uyg‘unligi bir nechta muhim huquqiy omillar bilan belgilanishi aniqlandi. Ularning asosiylari jazoning mutanosibligi, sud tomonidan individual yondashuvning ta’minlanishi, jazoning maqsadga muvofiqligi va ozodlikdan mahrum qilish bilan bog‘liq bo‘lmagan choralarni qo‘llash imkoniyatlaridan iborat.

Odillik prinsipining jazo tayinlashdagi huquqiy mohiyati

Odillik jinoyat-huquqiy munosabatlarda davlat majburlovining qonuniy va mutanosib amalga oshirilishini ta’minlovchi asosiy prinsiplardan biri hisoblanadi. Uning mazmuni sodir etilgan jinoyatning og‘irligi, aybning darajasi va aybdor shaxsning ijtimoiy xavflilik xususiyatlariga muvofiq huquqiy ta’sir chorasini belgilashda namoyon bo‘ladi.

O‘zbekiston Respublikasi Jinoyat kodeksining 8-moddasiga muvofiq, jinoyat sodir etgan shaxsga qo‘llaniladigan jazo yoki boshqa huquqiy ta’sir chorasi jinoyatning og‘ir-yengilligiga, aybning va shaxsning ijtimoiy xavflilik darajasiga mos bo‘lishi talab etiladi. Ushbu modda aynan bir jinoyat uchun ikki marta javobgarlikka tortishga ham yo‘l qo‘ymaydi.

Mazkur prinsipning jazo tayinlashdagi asosiy vazifasi sud tomonidan jazoni belgilash jarayonida subyektiv yondashuvlarning huquqiy chegaralarini aniqlashdan iborat. Jazo bir xil turdagi jinoyatlarni sodir etgan barcha shaxslarga mexanik tarzda bir xil qo‘llanilishi kerakligini anglatmaydi. Aksincha, jinoyatlarning tashqi o‘xshashligiga qaramay, ularni sodir etish sabablari, aybning shakli, yetkazilgan zarar miqdori va aybdorning shaxsiy xususiyatlari turlicha bo‘lishi mumkin.

Masalan, mulkiy zarar yetkazish bilan bog‘liq jinoyatni sodir etgan ikki shaxsdan biri o‘z aybini tan olib, yetkazilgan zararni ixtiyoriy qoplashi, ikkinchisi esa qonunda belgilangan tartibda og‘irlashtiruvchi deb topiladigan holatlarda harakat qilishi mumkin. Bunday vaziyatlarda ayni bir jazo turini va miqdorini tayinlash har doim ham individual odillik talabiga mos kelavermaydi.

Shu nuqtayi nazardan, odillik prinsipining amalga oshirilishi jinoyatga berilgan umumiy huquqiy baho bilan cheklanmaydi. U sud tomonidan aniq holatlarni kompleks tekshirish, jazoni tanlash sabablarini ko‘rsatish va tayinlangan jazoning zarurligini asoslashni ham talab etadi.

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Insonparvarlik prinsipining mazmuni va jazoning maqsadlari bilan bog‘liqligi

Insonparvarlik prinsipi jinoyat sodir etgan shaxsning huquqiy maqomi davlat tomonidan jazo qo‘llanilishi natijasida butunlay inkor etilmasligini anglatadi. Shaxs qonuniy jazoga tortilgan taqdirda ham uning insoniy qadr-qimmati, qonun bilan himoyalangan huquqlari va ijtimoiy hayotga qaytish imkoniyatlari e’tibordan chetda qolmasligi kerak.

Jinoyat kodeksining 7-moddasida jazo va boshqa huquqiy ta’sir choralari jismoniy azob berish yoki inson qadr-qimmatini kamsitish maqsadini ko‘zlamasligi mustahkamlangan. Shuningdek, mazkur norma jinoyat sodir etgan shaxsga uning axloqan tuzalishi hamda yangi jinoyat sodir etishining oldini olish uchun zarur va yetarli bo‘lgan jazo tayinlashni nazarda tutadi.

Ushbu qoida insonparvarlik prinsipining ikki jihatini ochib beradi. Birinchi jihat davlatning jinoiy-huquqiy ta’sir choralarini qo‘llashda shaxs qadr-qimmatini hurmat qilish majburiyatini ifodalaydi. Ikkinchi jihat esa jazoning maqsadga muvofiqligi va zaruriylik talabiga bog‘liqdir.

Jinoyat huquqida insonparvarlik javobgarlikdan asossiz ravishda ozod qilish yoki jinoyatning salbiy oqibatlarini e’tiborsiz qoldirish degani emas. Ayrim og‘ir va o‘ta og‘ir jinoyatlar uchun qat’iy jazo tayinlash jamiyat xavfsizligi hamda jabrlanuvchilarning huquqlarini himoya qilish nuqtayi nazaridan zarur bo‘lishi mumkin. Muhim jihat shundaki, har qanday jazo qonuniy maqsadlarga xizmat qilishi, uning og‘irligi esa asoslantirilgan bo‘lishi lozim.

Shu sababli insonparvarlik prinsipi jazoning yengilligini emas, balki uning zarurligi va yetarliligini ustuvor mezon sifatida ilgari suradi.

Odillik va insonparvarlik prinsiplarining qiyosiy-huquqiy tavsifi

1-jadval. Jazo tayinlashda odillik va insonparvarlik prinsiplarining o‘zaro bog‘liqligi

Tahlil mezoni	Odillik prinsipi	Insonparvarlik prinsipi
Asosiy maqsad	Jazoning jinoyatga mutanosibligini ta’minlash	Zarur va yetarli jazo orqali qonuniy maqsadlarga erishish
Huquqiy asos	JK 8-moddasi	JK 7-moddasi

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Tahlil mezoni	Odillik prinsipi	Insonparvarlik prinsipi
Ustuvor yondashuv	Jinoyatning og‘irligi va ayb darajasini baholash	Shaxs qadr-qimmati va tuzalish imkoniyatini hisobga olish
Jazoni individuallashtirish	Aybdorning roli va holatlarini farqlash	Shaxsiy va ijtimoiy sharoitlarni inobatga olish
Jazo tanlash	Qilmish va jazo o‘rtasida muvozanat	Haddan tashqari og‘ir jazoga yo‘l qo‘ymaslik
Kutiladigan natija	Mutanosib va asoslantirilgan jazo	Tuzalishga yo‘naltirilgan huquqiy ta’sir

Manba: O‘zbekiston Respublikasi Jinoyat kodeksining 7, 8 va 54-moddalari asosida muallif tomonidan tizimlashtirilgan.

Jadvaldan ko‘rinadiki, odillik va insonparvarlik bir-biriga qarama-qarshi prinsiplar emas. Ular jazo tayinlashning turli jihatlarini tartibga solgan holda yagona maqsad — qonuniy, mutanosib va samarali huquqiy ta’sirni amalga oshirishga xizmat qiladi.

Biroq bu uyg‘unlik avtomatik tarzda yuzaga kelmaydi. Masalan, sud aybdorning oilaviy sharoitlarini inobatga olib yengilroq jazo tayinlashni maqsadga muvofiq deb hisoblashi mumkin. Agar bunda jinoyatning og‘ir oqibatlari, aybdorning jinoyatdagi roli va jabrlanuvchining manfaatlari yetarli baholanmasa, insonparvarlikka asoslangan qaror odillik talablariga zid kelishi ehtimoli mavjud.

Aksincha, sodir etilgan jinoyatga nisbatan qattiq munosabat bildirish maqsadida shaxsning aybini yengillashtiruvchi holatlarni e’tiborsiz qoldirish ham jazoning individual adolatliligiga putur yetkazishi mumkin.

Demak, prinsiplarning uyg‘unligi sudning jazoni yengillashtirish yoki og‘irlashtirishga moyilligi bilan emas, balki barcha ahamiyatli omillarga asoslangan muvozanatli qaror qabul qilishi bilan belgilanadi.

Jazoni individuallashtirish prinsiplar uyg‘unligining asosiy mexanizmi sifatida Jazo tayinlashda odillik va insonparvarlikni muvofiqlashtirishning asosiy huquqiy vositalaridan biri jazoni individuallashtirish hisoblanadi.

Jinoyat kodeksining 54-moddasiga ko‘ra, sud jazo tayinlashda jinoyatning xususiyati va ijtimoiy xavflilik darajasi, qilmishning sababi, yetkazilgan zararning xususiyati va

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miqdori, aybdorning shaxsi, jazoni yengillashtiruvchi hamda og‘irlashtiruvchi holatlarni hisobga oladi.

Mazkur normalar sudga muayyan ish bo‘yicha jazoning individual xususiyatlarini belgilash imkonini beradi. Shunday bo‘lsa-da, sud diskretsiyasi mutlaq erkinlik emas. Jazoni tanlashda huquqiy asoslar, qonunchilikda belgilangan chegaralar va ish bo‘yicha aniqlangan holatlarga qat’iy rioya etilishi lozim.

Jazoni individuallashtirishda aybdorning shaxsiga oid ma’lumotlarni hisobga olish muhimdir. Bunda uning yoshi, sog‘lig‘i, oilaviy ahvoli, mehnat faoliyati, avvalgi xulq-atvori va jinoyatdan keyingi munosabati tegishli qonuniy mezonlar doirasida ahamiyat kasb etishi mumkin.

Ayniqsa, yetkazilgan zararning qoplanishi, chin ko‘ngildan pushaymon bo‘lish va jinoyatning ochilishiga faol yordam berish kabi holatlarni baholashda aniq, ishonchli dalillarga tayanish talab etiladi. Ushbu omillarning mavjudligi avtomatik ravishda eng yengil jazoni tayinlashni anglatmaydi, ammo ularning huquqiy ahamiyati e’tibordan chetda qolmasligi kerak.

Muqobil jazo choralarini qo‘llashning ahamiyati

Jazo tayinlashda odillik va insonparvarlikning uyg‘unligini ta’minlash imkoniyatlaridan yana biri ozodlikdan mahrum qilish bilan bog‘liq bo‘lmagan jazo choralaridan foydalanishdir.

Bunday choralar ayrim holatlarda shaxsning oilaviy va mehnat aloqalarini saqlash, uning jamiyatdan ajralmagan holda huquqiy majburiyatlarini bajarishi hamda takroriy jinoyat sodir etishining oldini olishga xizmat qilishi mumkin.

Birlashgan Millatlar Tashkiloti Bosh Assambleyasining 1990-yil 14-dekabrdagi 45/110-sonli rezolyutsiyasi bilan qabul qilingan Tokio qoidalarida ozodlikdan mahrum qilish bilan bog‘liq bo‘lmagan choralarni qo‘llash, inson huquqlariga rioya etish va jamiyat xavfsizligi manfaatlarini muvozanatlashtirish masalalariga alohida e’tibor qaratilgan.

Mazkur xalqaro yondashuvdan kelib chiqib, jazoning samaradorligi uning faqat og‘irlik darajasi bilan emas, balki jinoyat-huquqiy maqsadlarga erishish salohiyati bilan baholanishi lozim, degan xulosaga kelish mumkin.

Biroq muqobil jazolarni keng qo‘llash ham har doim ijobiy natijani kafolatlamaydi. Bunda shaxsning oldingi xulq-atvori, jinoyatning xususiyati, qayta jinoyat sodir etish xavfi va jamiyat xavfsizligiga ta’siri individual baholanishi zarur.

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Jazo tayinlashni takomillashtirish bo‘yicha ishlab chiqilgan model

Tadqiqot natijasida odillik va insonparvarlik prinsiplarini muvofiqlashtirishga qaratilgan besh mezonli huquqiy baholash modeli taklif etildi.

2-jadval. Odil va insonparvar jazo tayinlashning taklif etilayotgan baholash mezonlari

Mezon	Baholash mazmuni	Kutiladigan huquqiy natija
Qonuniylik	Jazo turi va miqdorining JK talablariga muvofiqligi	Asossiz jazolashning oldini olish
Mutanosiblik	Jinoyat og‘irligi, ayb va zarar darajasini hisobga olish	Odillikni ta’minlash
Individuallashtirish	Shaxsga oid ahamiyatli holatlarni baholash	Asoslantirilgan individual jazo
Zaruriylik va yetarlilik	Yengilroq choralar bilan jazo maqsadiga erishish imkoniyatini tekshirish	Ortiqcha jazolashni cheklash
Asoslantirilganlik	Jazo tanlash sabablarini sud hujjatida tushuntirish	Qarorning shaffofligini oshirish

Manba: normativ-huquqiy tahlil asosida ishlab chiqilgan mualliflik taklifi. Jadval sud amaliyotida tasdiqlangan statistik natijalarni emas, taklif etilayotgan baholash modelini ifodalaydi.

Taklif qilinayotgan modelga ko‘ra, sud jazo tayinlashda qonunda nazarda tutilgan asoslarni aniqlash bilan bir qatorda, nima sababdan aynan muayyan jazo turi va miqdori tanlanganini izchil asoslantirishi maqsadga muvofiq.

Modelning muhim jihati shundaki, u sud diskretsiyasini mexanik hisoblash tizimiga aylantirmaydi. Aksincha, sudyaning individual huquqiy bahosini saqlagan holda qarorning dalillarga tayanganligini kuchaytirishga qaratilgan.

MUHOKAMA

Tadqiqot natijalari jazo tayinlashda odillik va insonparvarlik prinsiplarining o‘zaro munosabatini jinoyat-huquqiy siyosatning murakkab masalalaridan biri sifatida baholash imkonini beradi. Ushbu prinsiplarning har biri mustaqil normativ ahamiyatga

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ega bo‘lsa-da, ularni bir-biridan ajratilgan holda tatbiq etish jazoning umumiy maqsadlariga erishishni qiyinlashtirishi mumkin.

Jazo tayinlashning amaliy murakkabligi shundaki, sud jinoyat sodir etgan shaxsning javobgarligini belgilash bilan birga, tayinlanayotgan jazoning kelgusidagi huquqiy va ijtimoiy oqibatlarini ham hisobga olishi kerak. Jazoning haddan tashqari qattiqligi mahkumning qayta ijtimoiylashish imkoniyatlarini cheklashi mumkin bo‘lsa, uning asossiz yengilligi jinoiy javobgarlikning ogohlantiruvchi ta’sirini pasaytirishi ehtimoldan xoli emas.

O‘zbekiston Respublikasi Oliy sudi Plenumining 2006-yil 3-fevraldagi 1-sonli qarorida jinoyat uchun jazo tayinlashda qonuniylik, insonparvarlik, odillik va javobgarlikning muqarrarligi prinsiplariga amal qilish zarurligi ta’kidlangan. Mazkur qarorda javobgarlikning muqarrarligi har bir holatda jazo qo‘llanishi shartligini anglatmasligi ham tushuntirilgan.

Mazkur yondashuv jazo tayinlash institutida individual huquqiy baholashning ahamiyatini yaqqol ko‘rsatadi. Jinoyat sodir etgan shaxsga nisbatan qonuniy asoslar mavjud bo‘lganda javobgarlikdan yoki jazodan ozod qilish imkoniyatlarini ko‘rib chiqish insonparvarlikning muhim ko‘rinishlaridan biri hisoblanadi. Biroq bunday qaror qabul qilishda jinoyatning ijtimoiy xavflilik xususiyatlari va jabrlanuvchining qonuniy manfaatlari ham yetarli darajada hisobga olinishi lozim.

Odillik va insonparvarlikning uyg‘unligini ta’minlashga to‘sqinlik qilishi mumkin bo‘lgan omillardan biri jazoni tanlashda qo‘llaniladigan baholash mezonlarining turlicha talqin qilinishidir. Sudyalar bir xil huquqiy normalarga tayangan holda, ishning o‘ziga xos holatlariga turlicha huquqiy baho berishlari mumkin. Bunday farqlarning mavjudligi o‘z-o‘zidan qonunbuzarlikni anglatmaydi. Ammo o‘xshash holatlarga nisbatan keskin farqlanuvchi jazolar tayinlanishi yetarlicha asoslantirilmasa, sud amaliyotining izchilligi va oldindan bashorat qilinuvchanligiga salbiy ta’sir ko‘rsatishi mumkin.

Shu jihatdan sud hukmlarida jazo tanlashga ta’sir qilgan omillarning mazmunini batafsil ko‘rsatish muhim ahamiyatga ega. Sud nafaqat yengillashtiruvchi va og‘irlashtiruvchi holatlar mavjudligini qayd etishi, balki ularning tanlangan jazo turiga qanday ta’sir ko‘rsatganini ham mantiqiy va huquqiy jihatdan asoslantirishi maqsadga muvofiq.

Tadqiqot doirasida insonparvarlik prinsipining yana bir muhim jihati — jazo tayinlashda shaxsning ijtimoiy aloqalarini saqlab qolish imkoniyatlarini baholash masalasi ko‘rib chiqildi. Ayrim hollarda ozodlikdan mahrum qilish jazosi shaxsni

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mehnat faoliyatidan, oilaviy muhitdan va boshqa ijtimoiy munosabatlardan uzoqlashtiradi. Bu esa jazodan keyingi moslashuv jarayonini murakkablashtirishi mumkin. Shu bilan birga, og‘ir yoki yuqori xavflilikka ega jinoyatlarda jamiyat xavfsizligini ta’minlash zarurati bunday oqibatlarga nisbatan ustuvor ahamiyat kasb etishi mumkin.

Shuning uchun ozodlikdan mahrum qilish bilan bog‘liq bo‘lmagan jazo choralarining qo‘llanishi jinoyatning xususiyati va qonunchilik imkoniyatlariga bog‘liq bo‘lishi zarur. Muqobil jazolarni har qanday vaziyatda ustun qo‘yish odillik talabiga muvofiq bo‘lmasligi mumkin.

BMTning Tokio qoidalarida ham jinoyat sodir etgan shaxslarning huquqlari, jabrlanuvchilarning manfaatlari va jamiyat xavfsizligi o‘rtasidagi muvozanatni ta’minlashga e’tibor qaratiladi. Bu esa insonparvarlikni faqat aybdorning manfaatlari bilan cheklab bo‘lmasligini ko‘rsatadi.

Mazkur tadqiqot asosida jazo tayinlash amaliyotini takomillashtirishning quyidagi yo‘nalishlari maqsadga muvofiq deb hisoblanadi:

1. Sud hukmlarining asoslantirilganligini kuchaytirish. Tayinlangan jazoning jinoyatning og‘irligi, aybdorning shaxsi hamda ishning boshqa holatlariga muvofiqligi sud hujjatlarida izchil yoritilishi kerak.
2. Muqobil jazo choralarini tanlash mezonlarini aniqlashtirish. Qonunchilikda imkoniyat mavjud bo‘lgan holatlarda yengilroq jazo bilan qonuniy maqsadlarga erishish mumkinligi alohida baholanishi maqsadga muvofiq.
3. Jazoni individuallashtirish amaliyotini rivojlantirish. Aybdorning shaxsiga oid holatlarni hisobga olishda bir xil va izchil huquqiy yondashuvlarni shakllantirish zarur.
4. Jabrlanuvchi manfaatlarini kompleks baholash. Insonparvarlik prinsipini tatbiq etish jabrlanuvchining huquqlari va jamiyat xavfsizligi hisobiga amalga oshirilmasligi kerak.
5. Sud amaliyotini ilmiy monitoring qilish. O‘xshash jinoyatlar bo‘yicha jazo tayinlash mezonlarini umumlashtirish, tafovutlar sabablarini o‘rganish va ularni huquqiy jihatdan baholash amaliyotning izchilligini kuchaytirishga xizmat qilishi mumkin.

Taklif etilgan yo‘nalishlar odillik va insonparvarlikni muvofiqlashtirish uchun huquqiy asoslarni mustahkamlashga xizmat qilishi mumkin. Biroq ularning amaliy samaradorligi haqidagi qat’iy xulosa sud qarorlari tahlili, qiyosiy huquqiy tadqiqotlar va tegishli empirik ma’lumotlar asosida tekshirilishi lozim.

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XULOSA

O‘zbekiston Respublikasi jinoyat qonunchiligida jazo tayinlashda odillik va insonparvarlik prinsiplarining o‘zaro uyg‘unligi qonuniy, mutanosib va individual jazo belgilashning muhim huquqiy sharti hisoblanadi. Mazkur prinsiplar jinoyat sodir etgan shaxsga nisbatan davlat majburlovini asosli qo‘llash, inson qadr-qimmatini hurmat qilish va jinoyat-huquqiy ta’sir choralarining samaradorligini ta’minlashga xizmat qiladi.

Tadqiqot davomida odillik prinsipining mazmuni sodir etilgan jinoyatning og‘irligi, aybning darajasi va shaxsning ijtimoiy xavfliligiga mos jazo belgilash bilan bog‘liqligi aniqlandi. Insonparvarlik prinsipi esa jazoning zarur va yetarli darajada qo‘llanishini, uning shaxs qadr-qimmatini kamsitishga qaratilmasligini hamda mahkumning axloqan tuzalish imkoniyatlarini hisobga olishni talab etadi.

O‘tkazilgan normativ-huquqiy tahlil asosida odillik va insonparvarlik prinsiplarini bir-biriga qarama-qarshi tushunchalar sifatida emas, balki jazo tayinlashning o‘zaro to‘ldiruvchi asoslari sifatida baholash lozimligi asoslandi. Chunki jinoyatning og‘irligiga mos kelmaydigan haddan tashqari yengil jazo ham, aybdorning individual xususiyatlarini e’tiborsiz qoldiruvchi asossiz og‘ir jazo ham huquqiy muvozanatga salbiy ta’sir ko‘rsatishi mumkin.

Jazoni individuallashtirish ushbu prinsiplarni uyg‘unlashtirishning asosiy huquqiy mexanizmi sifatida namoyon bo‘ladi. Bunda sud jazo tayinlashda jinoyatning obyektiv xususiyatlarini, aybdorning shaxsini, yetkazilgan zarar va uning oqibatlarini, shuningdek qonunchilikda nazarda tutilgan yengillashtiruvchi hamda og‘irlashtiruvchi holatlarni kompleks hisobga olishi zarur.

Maqolaning asosiy ilmiy-amaliy natijasi sifatida qonuniylik, mutanosiblik, individuallashtirish, zaruriylik va yetarlilik hamda asoslantirilganlik mezonlariga tayanuvchi baholash modeli taklif etildi. Ushbu model sud tomonidan tanlanayotgan jazoning huquqiy asoslarini tizimli tekshirish, ortiqcha repressiv yondashuvlarning oldini olish va sud qarorlarining shaffofligini oshirish uchun nazariy asos bo‘lib xizmat qilishi mumkin.

Shunday qilib, odil jazo har doim eng og‘ir jazo degani emas, insonparvar jazo esa har doim eng yengil jazo degani emas. Ushbu prinsiplarning haqiqiy uyg‘unligi sodir etilgan jinoyatga mutanosib, shaxsning individual xususiyatlarini hisobga oluvchi va qonunda belgilangan maqsadlarga erishish uchun zarur hamda yetarli bo‘lgan jazo tayinlanishida namoyon bo‘ladi.

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BANKLARNING INVESTITSIYA FAOLIYATI SAMARADORLIGINI OSHIRISH YO‘NALISHLARI

Bultakov Jamshid Shokir o‘g‘li

O‘zbekiston Respublikasi bank-moliya akademiyasi
2-kurs magistranti

Jahon moliya bozorlarida iqtisodiy beqarorlikning kuchayishi, investitsiya resurslariga bo‘lgan talabning ortib borishi, moliyaviy instrumentlar turlarining kengayishi hamda banklar o‘rtasidagi raqobatning keskinlashuvi tijorat banklarining investitsiya faoliyati samaradorligini oshirish masalasini dolzarb ilmiy-amaliy muammolardan biriga aylantirmoqda.

O‘zbekiston Respublikasida bank tizimini isloh qilish, moliya bozorini rivojlantirish, banklar faoliyatida bozor mexanizmlarini kengaytirish va iqtisodiyotga investitsiyalarni jalb etish bo‘yicha amalga oshirilayotgan islohotlar tijorat banklarining investitsiya faoliyatini takomillashtirish uchun yangi imkoniyatlar yaratmoqda. Banklar faoliyatida moliyaviy resurslardan samarali foydalanish, investitsiya aktivlari hajmini maqbul darajada oshirish, daromad manbalarini kengaytirish va investitsion risklarni nazorat qilish mamlakat bank tizimining raqobatbardoshligini ta’minlashda muhim omillar hisoblanadi. Shu nuqtayi nazardan, tijorat banklarining investitsiya faoliyati samaradorligini oshirish nafaqat alohida banklarning moliyaviy natijalarini yaxshilash, balki milliy moliya bozorining rivojlanishi va iqtisodiyotning investitsion salohiyatini mustahkamlash uchun ham ahamiyatlidir.

Tijorat banklarining investitsion jozibadorligi bevosita ularning aktivlariga, ayniqsa aktivlarning sifati, rentabelligi, likvidligi va boshqaruv samaradorligiga bog‘liqdir. Bank o‘z aktivlaridan samarali foydalansa, bu nafaqat daromadni, balki investorlar ishonchini ham oshiradi. Shu boisdan, har bir tijorat banki aktivlar strategiyasini chuqur tahlil asosida shakllantirishi zarur.

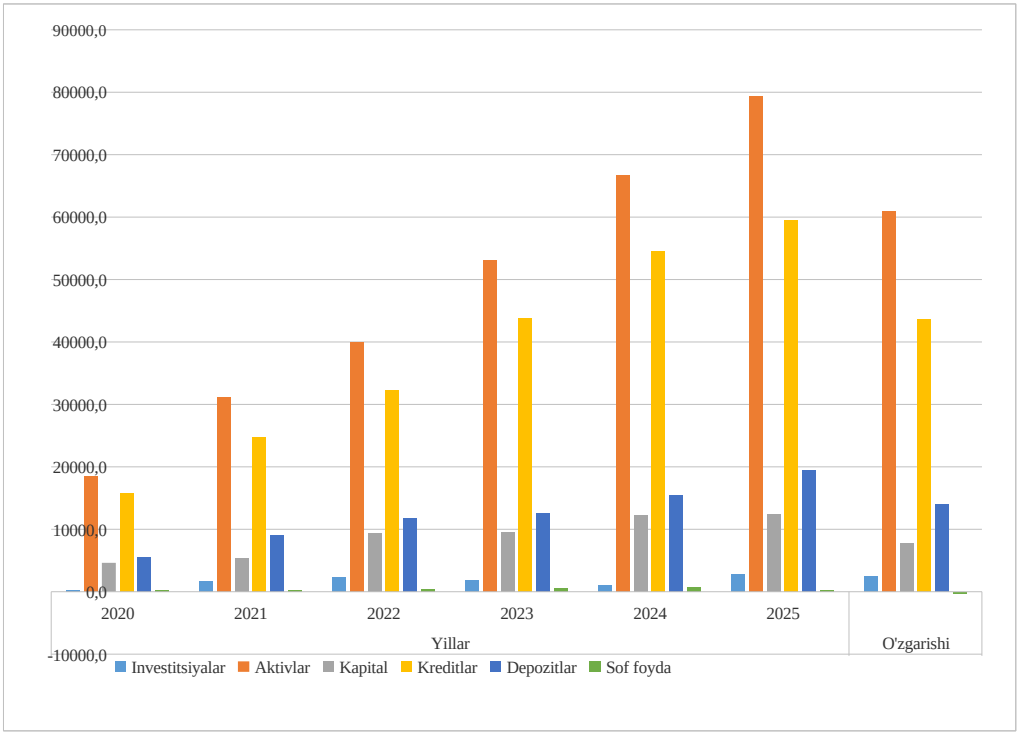
Banklarning investitsion jozibadorligi ularning moliyaviy barqarorligi, daromadliligi va risklarni boshqarish darajasi bilan chambarchas bog‘liq bo‘lib, mazkur omillar bevosita bank aktivlarining tarkibi va samaradorligi orqali shakllanadi. Shu nuqtayi nazardan, bank aktivlari investitsion jozibadorlikni oshirishning asosiy manbai va ko‘rsatkichlar bazasi hisoblanadi.

J.K. Majidovning fikricha, tijorat banklari aktivlarining oqilona tarkibini shakllantirish risklar darajasining oshishiga yo‘l qo‘ymagan holda, aktivlarning

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daromadliligi va likvidliligi o‘rtasidagi mutanosiblikni ta’minlash imkonini beradi.. Diversifikatsiyalangan aktivlar portfeli bankning bozor shoklariga chidamliligini oshirib, investorlar uchun ishonchlilik darajasini kuchaytiradi.

Ma’lumotlariga ko‘ra, 2021–2025-yillar oralig‘ida bank tizimidagi jami aktivlar hajmi 403 153,2 mlrd so‘mga yoki 110,1 foizga oshgan. Bu o‘sish bank sektorida umumiy kapitalning kengayganini, moliyaviy resurslar va sarmoyalar hajmining ortganini ko‘rsatadi. Ayrim banklar (Kapitalbank, Agrobank, Orient Finans) innovatsion moliyalashtirish, raqamli xizmatlar va xususiy kapital qo‘shilishi orqali sezilarli o‘sishga erishgan. Mikrokreditbank, Invest Finance, Davr-bank kabi o‘rta segmentdagi banklar bozor ulushini oshira boshlagan. Raqobatbardoshlikni saqlash uchun past ulushli banklarga xizmatlar diversifikatsiyasi, raqamlashtirish va mijozlar bazasini kengaytirish bo‘yicha chora-tadbirlar zarur.



“Sanoatqurilishbank” ATBning asosiy moliyaviy ko‘rsatkichlari dinamikasi

Manba: https://ahbor-reyting.uz/ru/service sayti ma’lumotlari asosida muallif tomonidan hisoblangan

Yuqoridagi rasm ma’lumotlariga asoslanib shuni alohida ta’kidlash joizki, “Sanoatqurilishbank” ATBning investitsiyalar hajmi 5 yilda 10 barobardan ko‘proq o‘sgan.

Bu bank infratuzilmasini modernizatsiya qilish, raqamli xizmatlar, texnologik yangiliklar va kredit portfelini kengaytirishga yo‘naltirilgan. Bu esa potensial investorlar uchun bankning o‘suvchan bozor ishtirokchisi ekanini ko‘rsatadi (2.12-jadval).

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“Sanoatqurilishbank” ATBning investitsiya faoliyati tahlili (*mlrd so‘m*)

Yil	Investitsiyalar (mlrd so‘m)
01.01.2020	272,4
01.01.2025	2 764,7
O‘sish	**+2 492,3 mlrd so‘m (10 barobardan ko‘proq)**

Bank tizimining moliyaviy barqarorligi, avvalo, uning asosiy moliyaviy ko‘rsatkichlari – aktivlar hajmi va kapital bazasining izchil o‘sishi bilan chambarchas bog‘liq. Ushbu ikki omil bankning nafaqat joriy faoliyat samaradorligini, balki uning uzoq muddatli to‘lovga qobiliyatliligi va investitsion jozibadorligini belgilovchi muhim mezonlardandir.

“Sanoatqurilishbank” ATBning aktivlar va kapital o‘sishi dinamikasi (*mlrd so‘m*)

Ko‘rsatkich	01.01.2020	01.01.2025	O‘sishi
Aktivlar	18 487,9	79 403,8	+60 915,9
Kapital	4 627,1	12 426,8	+7 799,7

“Sanoatqurilishbank” ATBning umumiy aktivlari va kapitali keskin oshgan. Bu kengayish strategiyasini olib borayotganini, risklarni qamrab olish salohiyatining kuchli ekanini, investorlar uchun moliyaviy xavf pastligini bildiradi. Kapitalning o‘sishi – bankning kredit va investitsiya jalb etish imkoniyatlarini oshiradi.

Aktivlar o‘sishi, bir tomondan, bank faoliyatining ko‘lamini, moliyaviy xizmatlar bozoridagi ishtirok darajasini va umumiy iqtisodiy faollikda tutgan o‘rnini ifodalaydi. «Sanoatqurilishbank” ATB misolida olib qaralganda, 2020-yildan 2025-yilgacha bank aktivlari 18 487,9 mlrd so‘mdan 79 403,8 mlrd so‘mgacha, ya’ni 4,2 barobarga o‘sgan. Bunday ko‘rsatkich bank infratuzilmasining kengayganligi, xizmatlar spektrining ortganligi va mijozlar bazasining faol ravishda kengayib borayotganligidan dalolat beradi.

Kapital bazasining o‘sishi esa bankning moliyaviy barqarorligini saqlab qolish, xavflarga nisbatan chidamliligini oshirish va mustaqil faoliyat yuritish salohiyatini mustahkamlashda hal qiluvchi omil hisoblanadi. «Sanoatqurilishbank” ATB kapitali 2020-yilda 4 627,1 mlrd so‘m bo‘lgan bo‘lsa, 2025-yilga kelib bu ko‘rsatkich 12 426,8 mlrd so‘mga yetgan. Bu esa bankning umumiy moliyaviy poydevori

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mustahkamlanayotganini va xatarlarga nisbatan zarur moliyaviy bufer shakllanayotganini ko‘rsatadi.

Shuningdek, kapitalning aktivlarga nisbati (leveraj ko‘rsatkichi; kapital yetarliligi koeffitsienti esa riskka tortilgan aktivlarga nisbatan hisoblanadi)

bankning o‘z majburiyatlarini qoplash imkoniyati, xususan depozitlar va kreditlar bo‘yicha risklarni minimallashtirish darajasini belgilaydi. Yuqoridagi statistikalar asosida aytish mumkinki, «Sanoatqurilishbank” ATB kapitalining yetarli darajada o‘sib borishi bankning barqarorligini ta’minlash va qo‘shimcha investitsiyalarni jalb etish salohiyatini oshiradi.

Aytish mumkinki, bank aktivlari va kapitalining tizimli va muvozanatli o‘sishi – bu bankning moliyaviy sog‘lomligi, ishonchliligi hamda investorlarga nisbatan jozibadorligining muhim indikatoridir. Bu holat, ayniqsa, «Sanoatqurilishbank” ATB misolida yaqqol namoyon bo‘lib, uning moliyaviy salohiyati va kelgusi yillarda investitsiyalarni jalb qilish imkoniyatlari kengayib borayotganini tasdiqlaydi.

«Sanoatqurilishbank” ATB kredit portfelining o‘sishi 2025-yilda 2020-yilga nisbatan 43 665,8 mlrd so‘mga oshgan. Kredit hajmining 3,7 barobar o‘sishi bankning real sektorga (sanoat, xizmatlar, tadbirkorlik) sarmoya yo‘naltirishini bildiradi. Bu samarali aktiv ishlatish va foyda olish imkoniyatlarining yuqoriligini ko‘rsatadi.

Shuningdek, «Sanoatqurilishbank” ATBning depozitlari hajmi 3,5 barobarga oshgan. Bu esa aholi va yuridik shaxslar bankni ishonchli moliyaviy institut deb bilayotganini, resurs bazasi mustahkamligini anglatadi hamda ichki investitsiya manbai mavjudligini ko‘rsatadi – bu esa investorlar uchun muhim omillardan hisoblanadi.

Bank tizimi barqarorligini baholashda asosiy moliyaviy ko‘rsatkichlardan biri bu – sof foyda hisoblanadi. Bank faoliyatining rentabelligini va menejment samaradorligini bevosita aks ettiruvchi bu ko‘rsatkich, bank investitsion jozibadorligini belgilovchi hal qiluvchi omillardan biridir. Sof foydaning dinamikasi, ayniqsa uzoq muddatli trendlarda, bankning umumiy moliyaviy holati va risklarga chidamlilik darajasi haqida muhim ma’lumot beradi.

Agrobank misolida olib qaralganda, 2020-yildan 2024-yilgacha sof foyda muntazam o‘sish tendensiyasini ko‘rsatgan, ammo 2025-yilga kelib sof foyda keskin pasayib, atigi 69 mlrd so‘mni tashkil qilgan. Bu esa oldingi yillarga nisbatan jiddiy orqaga chekinish, ya’ni –101,7 mlrd so‘mlik salbiy o‘zgarishni anglatadi. Sof foydaning keskin pasayishi – bankning moliyaviy barqarorligiga putur yetkazishi mumkin bo‘lgan ogohlantiruvchi omildir. Bu ko‘rsatkich bo‘yicha yuzaga kelgan salbiy dinamika bankning ichki iqtisodiy salohiyati, risklarni boshqarish darajasi va investitsion jozibadorligini izchil tahlil qilishni talab etadi. Shu bois, foyda rentabelligi

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ustida doimiy monitoring olib borish va uni ta’minlovchi omillarni kuchaytirish tijorat banklari oldida turgan dolzarb vazifalardan biridir.

“Sanoatqurilishbank” ATB moliyaviy ko‘rsatkichlarining ta’sir darajasiga qarab investitsion jozibadorlikni baholash

Ko‘rsatkich	O‘sish (2020–2025)	Investitsion jozibadorlikka ta’siri
Aktivlar	+60 915,9 mlrd so‘m	Eng yuqori darajada ijobiy
Kreditlar	+43 665,8 mlrd so‘m	Kuchli darajada ijobiy
Kapital	+7 799,7 mlrd so‘m	Barqaror darajada ijobiy
Depozitlar	+14 014,5 mlrd so‘m	Mijoz ishonchini ifodalaydi
Investitsiyalar	+2 492,3 mlrd so‘m	Faol investitsiya siyosati
Sof foyda	**–101,7 mlrd so‘m**	Xavfli holat, ehtiyot signali

Moliyaviy ko‘rsatkichlarning o‘sishi dinamikasi bankning investitsion jozibadorligini belgilashda hal qiluvchi ahamiyatga ega. Ularning orasida sof foyda, aktivlar, va investitsiyalar o‘zgarishlari eng katta ta’sir kuchiga ega bo‘lib, to‘g‘ridan-to‘g‘ri investorlarning qarorlariga ta’sir ko‘rsatadi. Shuningdek, kapital va depozitlar kabi ko‘rsatkichlar ham bankning ichki barqarorligi va tashqi ishonchliligini ifodalaydi. Shu boisdan, har bir bank o‘z ko‘rsatkichlarining o‘sish sifatini nafaqat miqdoriy, balki sifat jihatidan ham tahlil etib borishi lozim.

“Sanoatqurilishbank” ATBning investitsion jozibadorligi bahosi

Yo‘nalish	Baho	Ta’siri
Aktivlar o‘sishi	Yaxshi	Investitsion salohiyatni oshiradi
Kapital o‘sishi	Barqaror	Moliyaviy xavfsizlik
Kredit portfeli	Kengaymoqda	Riskni nazorat qilish zarur
Depozitlar	Barqaror o‘smoqda	Yaxshi likvidlik
Sof foyda	Pasaygan	Jiddiy ogohlantiruvchi signal
ROA / ROE	O‘rtacha	Investorga yetarli, lekin raqobatbardosh emas

Agrobank moliyaviy barqarorlik, aktivlar va kapital o‘sishi bo‘yicha ijobiy bahoga ega. Biroq foyda rentabelligidagi pasayish va sof foydadagi keskin tushish

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uning investitsion jozibadorligini to‘liq kafolatlamaydi. Shu sababli, bankka investitsiya kiritishdan oldin foyda barqarorligini ta’minlash va ichki operatsion samaradorlikni oshirish bo‘yicha islohotlar zarur.

“Sanoatqurilishbank” ATBning investitsion jozibadorligini oshirishda risklarni kamaytirish yo‘llari

Risk turi	Ehtimoliy ta’siri	Qarshi chora-tadbirlar
Sof foyda pasayishi	Rentabellikka salbiy	Xarajatlarni optimallashtirish, daromad manbalarini diversifikatsiya qilish
Kredit xavflari	Aktivlar sifati yomonlashuvi	Risklarni baholash tizimini kuchaytirish, NPL monitoringi
Raqobat kuchayishi	Mijozlar oqimi kamayishi	Innovatsion xizmatlar, past foizli mahsulotlar taklifi

“Sanoatqurilishbank” ATB investitsion portfeli izchil kengayib borayotgan, faol kredit siyosati yurituvchi va barqaror moliyaviy asosga ega bank hisoblanadi. Sof foydaning pasayishi qisqa muddatli moliyaviy xatarni anglatgan bo‘lsa-da, bankning kapital bazasi va aktivlar tarkibidagi o‘sish, uning uzoq muddatli jozibadorligini saqlab qolmoqda.

Umuman olganda, «Sanoatqurilishbank” ATB investitsion portfeli jozibadorligini oshirishda quyidagi jihatlarga e’tibor qaratishi lozim:

Birinchidan, raqamli bank xizmatlarini kengaytirish.

Ikkinchidan, kichik biznes kreditlari liniyasini kengaytirish.

Uchinchidan, ESG tamoyillariga asoslangan “yashil loyihalar”ni moliyalashtirish.

To‘rtinchidan, iqtisodiy mintaqalar uchun infratuzilma loyihalarini qo‘llab-quvvatlash.

Beshinchidan, xalqaro moliya institutlari (IFC, EBRD, IsDB) bilan sheriklik aloqalarini rivojlantirish.

Oltinchidan, mahalliy va xorijiy investorlar bilan qo‘shma loyihalar amalga oshirish.

Yettinchidan, Sukuk (islomiy obligatsiyalar) va yashil obligatsiyalar orqali investitsiya jalb etish.

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ИШЛАБ ЧИҚАРИШ ЖАРАЁНЛАРИДА ИККИ ФАКТОРЛИ ФУНКЦИЯЛАРДАН ФОЙДАЛАНИШ.

Садатов О.Х., Эшқораев Қ.А.,Тохиров Н.Х., Қурбонмуродов У.Ж.

Термиз муҳандислик ва агротехнологиялар университети

Аннотация: Танланган ишлаб чиқариш жараёнида, максимал маҳсулот ҳажмига эришиш учун икки ишлаб чиқариш факторлари орасидаги боғланишни “Ишлаб чиқариш функциялари, яъни масалан Кообб-Дуглас” орқали аниқлаш мумкин. Ушбу илмий изланишда Кооб-Дуглас ишлаб чиқариш функцияси қаралмоқда.

Калит сўзлар: ҳом ашё, маблағ, ҳажм, ҳаражат, капитал, технологик жараён, эластиклик коэффициентлари, фиксирланган капитал, ер ресурси.

Кириш

Ишлаб чиқариш функциялари, ишлаб чиқаришнинг қандай кўриниши бўлишидан қаттай назар қуйидаги умумий хоссаларни ўзида ифодалаши лозим бўлади:

1) Битта ишлаб чиқариш русурсини сарфлаш билан ишлаб чиқариш ҳажмини ошириб бўлмайди ёки унинг чеки бор(бир иш жойига кўп ишчини ёллаб бўлмайди – қайсики, ҳаммада ҳам жой йўқ).

2) Ишлаб чиқариш факторлари ўзаро тўлдирувчи(ишчилар ва воситалар) ва ўзаро алмашинувчи(автоматлашган ишлаб чиқариш) бўлиши мумкин.

Метод

Умумий ҳолда ишлаб чиқариш функцияларини қуйидагича ҳам қарашимиз мумкин:

$$Q = f(K, L, M, T, N)$$

бу ерда Q - ишлаб чиқариш ҳажми;

K- маблағ (ёки жиҳозлар);

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L- меҳнат боғлами;

M- ҳом ашёлар, материаллар;

T – технологик жараён (ёки ишлаб чиқариш технологиялари);

N –ишбилармонлик услублари.

Биз қарамоқчи бўлган ишлаб чиқариш функцияси қуйидагига эга бўлиши мумкин:

$Q=AK^{\text{ҳажм}}*L^{\text{ҳаражат}}$,

қайсики, бу ерда A – барча функцияларнинг нисбий, ишлаб чиқариш коэффициентлари кўрсаткичлари (бу кўрсатгич базали технология 30-40 йилларда ўзгариши натижасида ўзгариши мумкин);

K, L- маблағ ва меҳнат ресурслари;

ҳажм, ҳаражат – капитал маблағ ва меҳнат ҳаражатларининг ишлаб чиқариш ҳажми эластиклик коэффициентлари.

Агар ҳажм = 0,25 бўлса, капитал маблағ 1% га ва ишлаб чиқариш ҳажми 0,25% га ортади.

Қуйида иккита меҳнат ҳаражажатлари ўзгарувчиларидан ташкил бўлган фирманинг қисқа даврдаги фаолиятини қараймиз. Бундай ҳолатда фирма катта ҳажмдаги меҳнат ҳаражатларидан фойдаланиб ишлаб чиқариш ҳажмини ошириши мумкин.

1) ўсувчи хом –ашё функциясига прпорционал, качонки

об+ зат=1 ($Q = K^{0,5} \cdot L^{0,2}$).

2) пропорционал эмас–ўсувчи

$\alpha + \beta > 1 (Q = K^{0,9} + L^{0,8})$;

3) камаювчи

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$\alpha + \beta < 1 (Q = K^{0,4} \cdot L^{0,2})$.

Натижа

Агар капитал маблағ факторлари фиксирланган (доимий) бўлса, а меҳнат ресурслари эса ўзгарувчан бўлса, фирма катта меҳнат ресурслари ёрдамидан фойдаланиб ишлаб чиқариш ҳажмини ошириш мумкин. Лекин пасаювчи лимитли ҳосилдорлик қонунига кўра, бошқа ўзгарувчан факторлардан фойдалниб кетма-кет ўсувчи ўзгарувчан ресурсларга келиши мумкин, у ҳолда лимитли маҳсулот ҳажми ёки меҳнат ҳосилдорлиги пасайиш ҳолати кузатилади. Агар ёлланган ишчи кучини кўпатириб борсак, охирги натижада улар бир-бирларига ҳалақит бериб қолади (ҳосилдорлик лимитли коэффициенлари тушиб қолади) ва ишлаб чиқариш ҳажмининг камайиб кетишига олиб келади.

Ҳосилдорлик лимити (меҳнат ресурсининг лимитли маҳсулотлари бирлиги – MP_L) – бу кейинги навбатдаги битта меҳнат ресурсининг ўсиш ҳажми коэффициенти.

$MP_i = \frac{\Delta Q_l}{\Delta L}$,

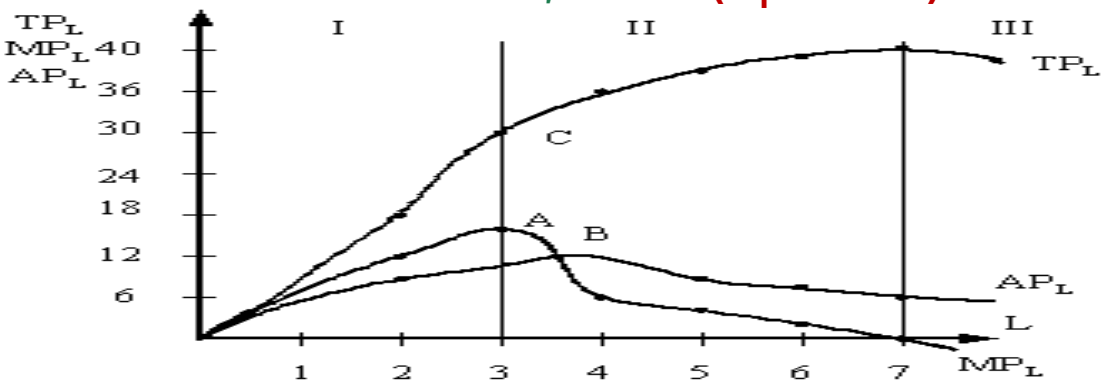
яъни, ҳосилдарлик ўсишининг жами маҳсулотга ўсиши (TP_L)

$MP_L = \frac{\Delta TP_L}{\Delta L}$.

қайсики, бу ерда MP_K лимитли капитал маҳсулот аниқланади.

Пасаювчи ҳосилдорлик қонунига асосланиб, умумий (TP_L), ўртача (AP_L) ва лимитли маҳсулот (MP_L) ларнинг ўзаро боғлиқликларини таҳлил қилсак бўлади.(расмга қаранг).

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Чизмадан кўриниб турибдики, 3-босқичда, ишчилар сони фиксирланган капиталга (дастгоҳлар) нисбатан ортиқроқ бўлса, MP манфий қийматлар олади, шунинг учун TP пасга тушишни бошлайди, ўртача маҳсулот эгри чизиғи AP эса MP эгри чизиғи динамикасига шартли боғлиқ. Биринчи босқичда эса иккала эгри чизиқ ҳам ўсаяпти, қайсики, янги олинган ёлланма ишчилар бўлганлиги учун ишлаб чиқариш ҳажми юқори бўлади, ўртача ҳосилдорлик (AP_L) олдинги қабул қилинган ёлланма ишчилар ишлаб чиқариш ҳажмига нисбатан. Лекин А нуқтадан кейин (max MP), тўртинчи ишчи умумий маҳсулотга (TP) қўшилса, уч кишиникидан кам ҳажм бўлади, MP пасая бошлайди. Шунинг учун тўртта ишчининг ўртача ишлаб чиқариш ҳажми ҳам қисқаради.

Хулосалар

Асосий замонавий иқтисодий ўсиш моделлари, шунингдек бошқа моделлар абстракт характерли, оддий реал иқтисодий жараёнларни тенгламалар ва унинг графиклари формаларида ифодалайди.

Шунинг учун охирги вақтларда ноанъанавий макроиқтисодиёт асосчиларининг асосий тушунтиришлари ишлаб чиқариш функцияларига йўналтирилган.

Макроиқтисодий ишлаб чиқариш функциялари учун аосан иккита таъриф берилган:

1. булар маҳсулот ишлаб чиқариш ҳолатини тенглаштириш функциялари ва уларнинг ишлаб чиқариш факторларини аниқлаш(капитал,меҳнат,ер ресурси ва НТП);

2. булар миллий маҳсулотлар орасидаги муносабатлар яъни, иқтисодиётда

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қўлланиладиган, жамиятга фойда келтирувчи ёки бойлик келтирувчи жамиятлар орасидаги ўзаро боғлиқлик муносабатлари аниқловчи моделлар яратиш.

Демак икки факторли (капитал,меҳнат) ишлаб чиқаришга ишлаб чиқариш функцияларини қўллаб маҳсулот ишлаб чиқариш ҳажмининг боғликлигини аниқлаш мумкин.

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YOSHLAR BANDLIGINI TA'MINLASHDA MEHNAT BOZORINING INSTITUTSIONAL MEXANIZMLARINI TAKOMILLASHTIRISH

Habibullayev Zuxriddin Quddusovich
70410102 – Iqtisodiyot mutaxassisligi magistranti
Ilmiy rahbar: i.f.d., dots. Sultonov B.

ANNOTATSIYA

Ushbu maqolada yoshlar bandligini oshirish maqsadida mehnat bozori institutlari faoliyatini muvofiqlashtirishning tashkiliy-iqtisodiy modeli taklif etiladi. Tadqiqotning asosiy vazifasi alohida bandlik xizmatlarini yagona xizmat zanjiriga birlashtirish, jarayon va natija ko‘rsatkichlarini farqlash hamda dasturlarni baholash uchun o‘lchanadigan mezonlarni ishlab chiqishdan iborat. Normativ hujjatlar, institutsional boshqaruv nazariyasi va xalqaro bandlik siyosati materiallari tizimli hamda funksional tahlil qilindi. Natija sifatida besh bosqichli “tashxis – individual reja – malaka va tajriba – ishga joylashish – keyingi kuzatuv” modeli ishlab chiqildi. Shuningdek, natijadorlikni ishga joylashtirish soni bilan cheklamasdan, olti va o‘n ikki oy davomida ishda qolish, ishning rasmiyligi, kasbiy moslik va xizmat birligi xarajati bo‘yicha baholash tavsiya qilindi. Taklif etilgan tizim konseptual mualliflik ishlanmasi bo‘lib, haqiqiy natijadorlikni tasdiqlash uchun pilot loyiha hamda nazorat guruhi bilan tekshirish zarurligi asoslab berildi.

Kalit so‘zlar: yoshlar bandligi; integratsiyalashgan xizmat; hududiy mehnat bozori; natijadorlik ko‘rsatkichlari; individual bandlik rejasi; raqamli boshqaruv; monitoring

KIRISH

Yoshlarni ishga joylashtirishga qaratilgan siyosatda markaziy masala ko‘pincha mavjud qo‘llab-quvvatlash vositalari sonini ko‘paytirish sifatida talqin qilinadi. Lekin bir maqsadga yo‘naltirilgan xizmatlar o‘zaro bog‘lanmasa, tashkiliy resurslar ko‘payishi bilan yakuniy natija mutanosib ravishda ortmaydi. Shu nuqtayi nazardan xizmatlarning integratsiyasi va natijadorlikka asoslangan boshqaruv yoshlar bandligini ta’minlashning mustaqil ilmiy muammosi hisoblanadi.

Bandlik xizmatlari, o‘quv markazlari, oliy ta’lim tashkilotlari, ish beruvchilar, tadbirkorlikka ko‘maklashish institutlari va mahalliy boshqaruv organlari turlicha maqsadli ko‘rsatkichlar bilan ishlashi mumkin. Ularning har biri o‘z vazifasini bajarsa-da, yosh fuqaroning ta’limdan doimiy va munosib ishga o‘tish jarayoni yakuniga yetmasligi ehtimoli mavjud. Natijada alohida tashkilotlar ko‘rsatkichlari yaxshi, tizimning umumiy samarasi esa cheklangan bo‘lishi mumkin.

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Xalqaro mehnat tashkiloti tadqiqotlarida yoshlarning mehnat bozoridagi holatini baholashda ishsizlik bilan bir qatorda ta'lim va ish bilan qamrab olinmaganlar ulushi, shuningdek ish o'rnining barqarorligi kabi sifat ko'rsatkichlari muhimligi ko'rsatiladi [3]. O'zbekiston qonunchiligida ham bandlikni qo'llab-quvvatlash va yoshlar uchun imkoniyatlar yaratish huquqiy yo'nalish sifatida mustahkamlangan [4; 5]. Tadqiqotning mohiyati shu huquqiy imkoniyatlarni boshqaruv jarayoniga qanday bog'lash mumkinligidadir.

Maqolaning maqsadi hududiy darajada yoshlar bandligini ta'minlashning integratsiyalashgan institutsional modelini ishlab chiqish va uning samaradorligini baholash indikatorlarini taklif qilishdan iborat. Asosiy savol shundan iboratki, bandlikka ko'maklashish resurslari qanday ketma-ketlikda va qanday javobgarlik taqsimoti bilan qo'llanilganda yoshlar uchun barqaror natija ehtimoli oshishi mumkin?

MATERIALLAR VA METODLAR

Tadqiqot konseptual loyihalash va funksional tahlil usullariga asoslanadi. Ma'lumot manbai sifatida O'zbekistonning bandlik va yoshlarga oid huquqiy hujjatlari, institutsional iqtisodiyot adabiyotlari hamda Xalqaro mehnat tashkilotining yoshlar bandligiga doir hisoboti tanlandi [1; 3–5]. Tadqiqotda biror hududdagi dastur tajribasi muvaffaqiyatli deb e'lon qilinmaydi va soxta o'lchov natijalari keltirilmaydi.

Model ishlab chiqishda xizmatlar zanjiri tamoyili qo'llandi: boshlang'ich murojaat yagona identifikator orqali qayd etiladi; ish qidiruvchining ehtiyoji va to'siqlari aniqlanadi; individual xizmat rejasi tuziladi; zarur o'qitish yoki amaliyot taqdim etiladi; nomzod ish beruvchi bilan bog'lanadi; ishga kirgandan keyin bandlik sifati nazorat qilinadi. Har bir bosqich uchun mas'ul institut va nazorat indikatori ajratiladi.

Natijadorlik uch darajaga ajratildi: kirish resurslari (mablag', xodim va vaqt), bevosita mahsulotlar (xizmat olganlar va suhbatga yo'naltirilganlar), yakuniy natijalar (barqaror rasmiy ish va daromad). Ushbu ajratish qayd etilgan murojaatlar soni bilan haqiqatda ishga moslashgan shaxslar sonini adashtirmaslik uchun zarur. Shuningdek, hududlar o'rtasidagi demografik va sanoat tarkibi farqlarini inobatga olish tamoyili kiritildi.

Baholash modeli matematik jihatdan oldindan aniqlangan indikatorlardan iborat, biroq vazn koeffitsiyentlari yoki hududiy natijalar empirik ma'lumotsiz belgilanmadi. Modelni sinash uchun dasturga kirgan va o'xshash xususiyatga ega, lekin hali xizmat olmagan yoshlar guruhlarini taqqoslash, dastlabki holatni hisobga olish va vaqt bo'yicha kuzatuv o'tkazish tavsiya etiladi.

NATIJALAR

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Tadqiqot natijasida besh funksional moduldan iborat boshqaruv konstruktsiyasi shakllantirildi. Birinchi modul – diagnostika. Unda ish izlovchining yoshi, ta’lim holati, ish tajribasi, kasbiy kompetensiyasi, mobilligi va ish qidirishdagi to‘siqlar yagona anketa orqali aniqlanadi. Biroq ma’lumot yig‘ish faqat zarur miqdorda olib borilishi, shaxsiy ma’lumotlardan foydalanishga doir huquqiy talablar bajarilishi lozim.

Ikkinchi modul – individual bandlik rejasi. Rejada maqsadli kasb yoki faoliyat yo‘nalishi, kutilayotgan ko‘nikma, mas’ul tashkilot, vaqt chegarasi va oraliq natija ko‘rsatiladi. Barcha yoshlarni bir xil qisqa kursga yo‘naltirish o‘rniga, ishga tayyor talabgorga to‘g‘ridan-to‘g‘ri vakansiya tavsiya etiladi, malaka bo‘shlig‘i aniqlangan talabgorga qayta tayyorlash, tajribasi yo‘q bitiruvchiga esa amaliyot marshruti belgilanadi.

Uchinchi modul – ish beruvchilar bilan hamkorlik. Bo‘sh ish o‘rni e’lonida lavozim, vazifalar, zarur ko‘nikmalar, ish grafigi, shartnoma shakli va ish haqi doirasi imkon qadar standart ko‘rsatiladi. Ish beruvchilardan kelgan teskari aloqa o‘quv dasturlari mazmunini qayta ko‘rib chiqishda qo‘llanadi. Shartnomaviy amaliyot mexanizmi ish beruvchi mentorligi, amaliyot muddati va natijani tekshirish mezonlarini belgilashi kerak.

To‘rtinchi modul – ishga joylashishni tasdiqlash. Bandlikni tasdiqlash uchun ishga qabul qilish haqidagi dastlabki xabarni real mehnat munosabatlari haqidagi qonuniy va tekshiriladigan ma’lumot bilan solishtirish talab etiladi. Aks holda, bir fuqaroning bir necha xizmat orqali qayta-qayta “ishga joylashtirildi” deb sanalishi yoki norasmiy, qisqa muddatli ishning barqaror natija sifatida qayd etilishi mumkin.

Beshinchi modul – post-bandlik monitoringi. Taklif qilingan modelda ishga kirishdan 6 va 12 oy o‘tgach ish holati, kasbiy moslik va mehnat sharoitlari qonuniy asosda tekshiriladi. Shunday qilib, xizmat ko‘rsatishning yakuniy nuqtasi ishga yo‘llanma berish emas, balki yosh fuqaroning mehnat bozorida mustahkam o‘rnashganini baholashga o‘tadi.

Natijadorlikning taklif qilingan ko‘rsatkichlari quyidagicha ifodalanadi: R6 = (ishga joylashganlardan 6 oy ishda qolganlar soni / kuzatish mumkin bo‘lgan ishga joylashganlar soni) × 100%; R12 xuddi shu tartibda 12 oy uchun hisoblanadi. Bir barqaror ish o‘rnining dastur xarajati esa C = jami dastur xarajatlari / belgilangan davrda tasdiqlangan barqaror ish o‘rinlari soni formulasi orqali aniqlanadi. Maxraj nol bo‘lsa, ko‘rsatkich hisoblanmaydi va bu holat alohida qayd etiladi. Formulalar hisoblash vositasi, real o‘lchangan natija emas.

1-jadval. Integratsiyalashgan modelning funksional bosqichlari

Bosqich	Asosiy mas’ullar	Monitoring natijasi

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Tashxis	Bandlik xizmati / maslahatchi	Ehtiyoj profili shakllanishi
Individual reja	Maslahatchi va talabgor	Reja bo‘yicha bosqichlarning bajarilishi
Malaka va amaliyot	Ta’lim tashkiloti / ish beruvchi	Kompetensiya tasdig‘i
Joylashtirish	Bandlik xizmati / ish beruvchi	Rasmiy ishga kirish
Kuzatuv	Vakolatli monitoring guruhi	R6, R12 va natija xarajati

KENGAYTIRILGAN NAZARIY-TAHLILIY NATIJALAR

1. Yagona murojaat arxitekturasi

Modelning kirish nuqtasi raqamli kabinet bilan cheklanmasligi, jismoniy maslahat punktlari orqali ham ishlashi lozim. Fuqaro bir marta murojaat qilgach, qaysi dastur unga tegishli ekani haqida turli idoralardan takror ma’lumot qidirib yurmasligi kerak. Shu maqsadda dastlabki diagnostika ma’lumotlari kam hajmda yig‘ilib, keyingi maqsadli xizmat faqat zarur maydonlardan foydalanishi taklif etiladi. Bunda yagona axborot muhiti barcha ma’lumotlarni bir tashkilot nazoratiga topshirish degani emas; vakolat va kirish huquqlari qat’iy ajratiladi.

Yagona murojaat arxitekturasi yo‘nalishida boshqaruv qarorining mezoni xizmatning rasmiy bajarilishi emas, foydalanuvchiga yetib borgan amaliy qiymat bo‘lishi zarur. Jarayon uchun mas’ul tashkilot, axborot olish manbasi, belgilangan xizmat muddati va shikoyatni ko‘rib chiqish tartibi bir hujjatda qayd etiladi. Pilotda kutilgan natija kuzatilmasa, avval dastur nazariyasining noto‘g‘ri taxminlari, so‘ng resurs va ijro intizomi tekshiriladi. Hududlar o‘rtasida xulosalar chiqarishda iqtisodiy sharoit va qatnashchilarning dastlabki holati bo‘yicha farqlar hisobga olinishi shart. Har qanday kengaytirish qarori mustaqil baholashdan keyin qabul qilinishi maqsadga muvofiq.

2. Individual yo‘nalish algoritmi

Xizmatlar barcha yoshlarga bir xil ketma-ketlikda berilsa, resurslar samarasiz taqsimlanadi. Masalan, malakasi yetarli, biroq vakansiyalardan xabarsiz shaxsga uzoq o‘quv kursi emas, sifatli axborot kerak; ko‘nikma yetishmaydigan talabgorga esa ishga yo‘llanma berishdan oldin amaliy tayyorgarlik zarur. Individual reja kamida to‘siqning

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turi, kutilayotgan natija, mas'ul ijrochi va qayta ko'rib chiqish muddatini aks ettirishi lozim. Algoritm tavsiya berishi mumkin, ammo yakuniy qaror maslahatchi va fuqaroning ongli tanloviga bog'liq bo'lishi kerak.

Individual yo'nalish algoritmi yo'nalishida boshqaruv qarorining mezoni xizmatning rasmiy bajarilishi emas, foydalanuvchiga yetib borgan amaliy qiymat bo'lishi zarur. Jarayon uchun mas'ul tashkilot, axborot olish manbasi, belgilangan xizmat muddati va shikoyatni ko'rib chiqish tartibi bir hujjatda qayd etiladi. Pilotda kutilgan natija kuzatilmasa, avval dastur nazariyasining noto'g'ri taxminlari, so'ng resurs va ijro intizomi tekshiriladi. Hududlar o'rtasida xulosalar chiqarishda iqtisodiy sharoit va qatnashchilarning dastlabki holati bo'yicha farqlar hisobga olinishi shart. Har qanday kengaytirish qarori mustaqil baholashdan keyin qabul qilinishi maqsadga muvofiq.

3. Xizmat darajasi kelishuvi

Integratsiya deklarativ hamkorlik bilan chegaralansa, murojaatlar muassasalar o'rtasida yo'qolishi mumkin. Hamkorlik kelishuvida murojaatga javob berish, bo'sh ish o'rni ma'lumotini tekshirish, talabgorni suhbatga yo'naltirish va natijani qaytarishning maksimal muddatlari belgilanishi maqsadga muvofiq. Ushbu muddatlar amaliy pilotda imkoniyatlarga qarab belgilanadi; ularni real jarayon tahlilisiz universal me'yor deb e'lon qilish to'g'ri emas. Kelishuv buzilishi holatlari tahlil qilinsa, resurs yetishmasligi bilan jarayonning noto'g'ri qurilganini ajratish mumkin.

Xizmat darajasi kelishuvi yo'nalishida boshqaruv qarorining mezoni xizmatning rasmiy bajarilishi emas, foydalanuvchiga yetib borgan amaliy qiymat bo'lishi zarur. Jarayon uchun mas'ul tashkilot, axborot olish manbasi, belgilangan xizmat muddati va shikoyatni ko'rib chiqish tartibi bir hujjatda qayd etiladi. Pilotda kutilgan natija kuzatilmasa, avval dastur nazariyasining noto'g'ri taxminlari, so'ng resurs va ijro intizomi tekshiriladi. Hududlar o'rtasida xulosalar chiqarishda iqtisodiy sharoit va qatnashchilarning dastlabki holati bo'yicha farqlar hisobga olinishi shart. Har qanday kengaytirish qarori mustaqil baholashdan keyin qabul qilinishi maqsadga muvofiq.

4. Ish beruvchiga qiymat taklifi

Korxona platformaga sifatli vakansiya kiritishi uchun unda aniq rag'bat mavjud bo'lishi kerak. Nomzodlarni dastlabki kompetensiya bo'yicha saralash, amaliyot jarayonini muvofiqlashtirish va ishga qabul qilishdagi hujjat yukini kamaytirish ish beruvchi ishtirokini oshirishi mumkin. Biroq bunday qulayliklar mehnat sharoitlariga qo'yiladigan talablarni yumshatish hisobidan yaratilmasligi lozim. Bandlik xizmati va

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korxona o‘rtasida qayta aloqa mexanizmi bo‘lmasa, malaka talablarining o‘zgarishi ta’lim tashkilotlarigacha yetib bormaydi.

Ish beruvchiga qiymat taklifi yo‘nalishida boshqaruv qarorining mezoni xizmatning rasmiy bajarilishi emas, foydalanuvchiga yetib borgan amaliy qiymat bo‘lishi zarur. Jarayon uchun mas’ul tashkilot, axborot olish manbasi, belgilangan xizmat muddati va shikoyatni ko‘rib chiqish tartibi bir hujjatda qayd etiladi. Pilotda kutilgan natija kuzatilmasa, avval dastur nazariyasining noto‘g‘ri taxminlari, so‘ng resurs va ijro intizomi tekshiriladi. Hududlar o‘rtasida xulosalar chiqarishda iqtisodiy sharoit va qatnashchilarning dastlabki holati bo‘yicha farqlar hisobga olinishi shart. Har qanday kengaytirish qarori mustaqil baholashdan keyin qabul qilinishi maqsadga muvofiq.

5. Modullararo ma’lumot oqimi

Taklif etilgan besh modulning har biri o‘z yakuniy hodisasini qayd etishi kerak: diagnostika yakunlandi, individual reja tasdiqlandi, suhbat o‘tkazildi, mehnat munosabati tasdiqlandi, monitoring yakunlandi. Bitta statusni boshqasi o‘rnida ishlatish muhim xatodir. Masalan, suhbatga yuborish ishga qabul qilishni anglatmaydi. Holat tarixini saqlash foydalanuvchining takror murojaatini yengillashtiradi va qaysi bo‘g‘inda vaqt yo‘qotilayotganini aniqlashga yordam beradi. Ma’lumotlar harakatida ruxsat, maqsadga muvofiqlik va audit izi bo‘lishi shart.

Modullararo ma’lumot oqimi yo‘nalishida boshqaruv qarorining mezoni xizmatning rasmiy bajarilishi emas, foydalanuvchiga yetib borgan amaliy qiymat bo‘lishi zarur. Jarayon uchun mas’ul tashkilot, axborot olish manbasi, belgilangan xizmat muddati va shikoyatni ko‘rib chiqish tartibi bir hujjatda qayd etiladi. Pilotda kutilgan natija kuzatilmasa, avval dastur nazariyasining noto‘g‘ri taxminlari, so‘ng resurs va ijro intizomi tekshiriladi. Hududlar o‘rtasida xulosalar chiqarishda iqtisodiy sharoit va qatnashchilarning dastlabki holati bo‘yicha farqlar hisobga olinishi shart. Har qanday kengaytirish qarori mustaqil baholashdan keyin qabul qilinishi maqsadga muvofiq.

6. Natijadorlik zanjiri

Baholashda resurs, faoliyat, xizmat mahsuloti va yakuniy natija ketma-ketligi saqlanishi zarur. Maslahatchi soni tizim salohiyatini, o‘tkazilgan suhbatlar faoliyatni, tasdiqlangan ishga joylashish bevosita mahsulotni, olti oydan keyin ishda qolish esa nisbatan barqaror natijani aks ettiradi. Ularning hech biri boshqasini avtomatik isbotlamaydi. Shuning uchun boshqaruv panelida ko‘rsatkichlarning turli qatlamlari

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birgalikda, lekin aralashtirilmagan holda berilishi kerak. Tanlanadigan asosiy indikatorlar soni kam va talqini aniq bo‘lsa, monitoring amaliy qarorga ko‘proq xizmat qiladi.

Natijadorlik zanjiri yo‘nalishida boshqaruv qarorining mezoni xizmatning rasmiy bajarilishi emas, foydalanuvchiga yetib borgan amaliy qiymat bo‘lishi zarur. Jarayon uchun mas’ul tashkilot, axborot olish manbasi, belgilangan xizmat muddati va shikoyatni ko‘rib chiqish tartibi bir hujjatda qayd etiladi. Pilotda kutilgan natija kuzatilmasa, avval dastur nazariyasining noto‘g‘ri taxminlari, so‘ng resurs va ijro intizomi tekshiriladi. Hududlar o‘rtasida xulosalar chiqarishda iqtisodiy sharoit va qatnashchilarning dastlabki holati bo‘yicha farqlar hisobga olinishi shart. Har qanday kengaytirish qarori mustaqil baholashdan keyin qabul qilinishi maqsadga muvofiq.

7. Maxrajni to‘g‘ri tanlash

Foiz ko‘rsatkichining ishonchliligi maxrajda kimlar hisobga olinganiga bog‘liq. Olti oylik natijani faqat kuzatuvga javob bergan shaxslar orasida hisoblash bilan barcha dastur qatnashchilariga nisbatan hisoblash o‘rtasida muhim farq mavjud. Kontaktni yo‘qotgan ishtirokchilar tasodifiy bo‘lmasligi mumkin: aynan beqaror ish sharoitidagi yoshlar tizimdan tezroq chiqib ketadi. Shu sababli kuzatuvga yetib bormaganlar ulushi va ular bo‘yicha ehtimoliy og‘ish natijalar bilan birga e’lon qilinishi kerak.

Maxrajni to‘g‘ri tanlash yo‘nalishida boshqaruv qarorining mezoni xizmatning rasmiy bajarilishi emas, foydalanuvchiga yetib borgan amaliy qiymat bo‘lishi zarur. Jarayon uchun mas’ul tashkilot, axborot olish manbasi, belgilangan xizmat muddati va shikoyatni ko‘rib chiqish tartibi bir hujjatda qayd etiladi. Pilotda kutilgan natija kuzatilmasa, avval dastur nazariyasining noto‘g‘ri taxminlari, so‘ng resurs va ijro intizomi tekshiriladi. Hududlar o‘rtasida xulosalar chiqarishda iqtisodiy sharoit va qatnashchilarning dastlabki holati bo‘yicha farqlar hisobga olinishi shart. Har qanday kengaytirish qarori mustaqil baholashdan keyin qabul qilinishi maqsadga muvofiq.

8. Xarajatlarni hisoblash

Bir barqaror ish o‘rni uchun xarajat ko‘rsatkichiga faqat ajratilgan subsidiya emas, maslahatchilar mehnati, axborot tizimiga xizmat ko‘rsatish, kasbiy o‘qitish va monitoring xarajatlari ham tegishli ulushda qo‘shilishi zarur. Aks holda bir hudud arzonroq ko‘ringani bilan u xarajatlarning bir qismini boshqa idora zimmasiga yuklagan bo‘lishi mumkin. Dasturlarni solishtirishdan oldin bir xil hisob davri, narxlar darajasi va xarajat chegaralari belgilanadi. Narx va natija indikatorlari birga ko‘rilmasa, tejamkorlik sifatsiz xizmatga olib kelishi ehtimoli bor.

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Xarajatlarni hisoblash yo‘nalishida boshqaruv qarorining mezoni xizmatning rasmiy bajarilishi emas, foydalanuvchiga yetib borgan amaliy qiymat bo‘lishi zarur. Jarayon uchun mas’ul tashkilot, axborot olish manbasi, belgilangan xizmat muddati va shikoyatni ko‘rib chiqish tartibi bir hujjatda qayd etiladi. Pilotda kutilgan natija kuzatilmasa, avval dastur nazariyasining noto‘g‘ri taxminlari, so‘ng resurs va ijro intizomi tekshiriladi. Hududlar o‘rtasida xulosalar chiqarishda iqtisodiy sharoit va qatnashchilarning dastlabki holati bo‘yicha farqlar hisobga olinishi shart. Har qanday kengaytirish qarori mustaqil baholashdan keyin qabul qilinishi maqsadga muvofiq.

9. Riskga moslashtirish

Hududlar natijalarini xom foizlarda solishtirish murakkab talabgorlar bilan ishlaydigan markazlarni nohaq jazolaydi. Modelda boshlang‘ich bilim, ish qidirish davomiyligi, yashash joyi, mobilitet va mehnat faoliyatiga to‘sqinlik qiluvchi holatlar hisobga olingan ehtiyotkor taqqoslash maqsadga muvofiq. Bunda xatar profili kishini xizmatdan chetlashtirish vositasi emas, zarur yordam hajmini belgilash vositasi bo‘lishi kerak. Algoritmik qarorlar bo‘yicha tushuntirish olish va xatoni tuzatish imkoniyati modelning majburiy elementi sanaladi.

Riskga moslashtirish yo‘nalishida boshqaruv qarorining mezoni xizmatning rasmiy bajarilishi emas, foydalanuvchiga yetib borgan amaliy qiymat bo‘lishi zarur. Jarayon uchun mas’ul tashkilot, axborot olish manbasi, belgilangan xizmat muddati va shikoyatni ko‘rib chiqish tartibi bir hujjatda qayd etiladi. Pilotda kutilgan natija kuzatilmasa, avval dastur nazariyasining noto‘g‘ri taxminlari, so‘ng resurs va ijro intizomi tekshiriladi. Hududlar o‘rtasida xulosalar chiqarishda iqtisodiy sharoit va qatnashchilarning dastlabki holati bo‘yicha farqlar hisobga olinishi shart. Har qanday kengaytirish qarori mustaqil baholashdan keyin qabul qilinishi maqsadga muvofiq.

10. Pilot dizayni

Tizimni bir vaqtning o‘zida barcha hududga tatbiq etish tashkiliy xatolarni kattalashtirishi mumkin. Shu sababli turli iqtisodiy tuzilishga ega kichik hududlarda cheklangan pilot o‘tkazish taklif qilinadi. Pilot boshlanishidan oldin asosiy ko‘rsatkichlar, dasturga kirish qoidalari, monitoring davri va xizmat ko‘rsatish tartibi yozib qo‘yiladi. Ushbu hujjat yakuniy natijaga qarab mezonlarni o‘zgartirish xavfini kamaytiradi. Pilot davomidagi tashkiliy muammolar ham natijaning bir qismi sifatida qayd etilishi lozim.

Pilot dizayni yo‘nalishida boshqaruv qarorining mezoni xizmatning rasmiy bajarilishi emas, foydalanuvchiga yetib borgan amaliy qiymat bo‘lishi zarur. Jarayon

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uchun mas’ul tashkilot, axborot olish manbasi, belgilangan xizmat muddati va shikoyatni ko‘rib chiqish tartibi bir hujjatda qayd etiladi. Pilotda kutilgan natija kuzatilmasa, avval dastur nazariyasining noto‘g‘ri taxminlari, so‘ng resurs va ijro intizomi tekshiriladi. Hududlar o‘rtasida xulosalar chiqarishda iqtisodiy sharoit va qatnashchilarning dastlabki holati bo‘yicha farqlar hisobga olinishi shart. Har qanday kengaytirish qarori mustaqil baholashdan keyin qabul qilinishi maqsadga muvofiq.

11. Muqobil baholash dizayni

Agar tasodifiy taqsimlash amaliy yoki axloqiy jihatdan mumkin bo‘lmasa, bir-biriga o‘xshash hududlar bo‘yicha oldingi va keyingi davrlar ma’lumotlarini solishtirish mumkin. Biroq bunday dizayn iqtisodiy kon’yunktura, migratsiya va hududiy investitsiya kabi tashqi omillardan xoli bo‘lmaydi. Farqlar-farqi yondashuvi tanlansa, davrlar bo‘yicha oldingi tendensiyalar o‘xshashligi va ma’lumotlarning bir xil tarzda yig‘ilishi tekshirilishi kerak. Ushbu shartlar bajarilmasa, natija sababiy ta’sir deb emas, kuzatilgan bog‘liqlik sifatida beriladi.

Muqobil baholash dizayni yo‘nalishida boshqaruv qarorining mezoni xizmatning rasmiy bajarilishi emas, foydalanuvchiga yetib borgan amaliy qiymat bo‘lishi zarur. Jarayon uchun mas’ul tashkilot, axborot olish manbasi, belgilangan xizmat muddati va shikoyatni ko‘rib chiqish tartibi bir hujjatda qayd etiladi. Pilotda kutilgan natija kuzatilmasa, avval dastur nazariyasining noto‘g‘ri taxminlari, so‘ng resurs va ijro intizomi tekshiriladi. Hududlar o‘rtasida xulosalar chiqarishda iqtisodiy sharoit va qatnashchilarning dastlabki holati bo‘yicha farqlar hisobga olinishi shart. Har qanday kengaytirish qarori mustaqil baholashdan keyin qabul qilinishi maqsadga muvofiq.

12. Ma’lumotlar xavfsizligi

Integratsiyalashgan modelda yoshlarning ta’limi, ish tajribasi, aloqa ma’lumotlari va ish haqi haqida sezgir axborot to‘planishi mumkin. Shuning uchun huquqiy asos, foydalanuvchiga tushunarli xabarnoma, minimal yig‘ish prinsipi va muddatli saqlash siyosati boshlang‘ich dizaynda belgilanishi kerak. Kirish huquqlarining lavozim bo‘yicha cheklanishi, ma’lumotga murojaat jurnalining tekshirilishi va shikoyat mexanizmi keyin qo‘shiladigan emas, avvaldan loyihalanadigan nazorat vositalaridir. Tizimning samaradorligi fuqarolarning axborot xavfsizligi hisobidan oshirilmasligi lozim.

Ma’lumotlar xavfsizligi yo‘nalishida boshqaruv qarorining mezoni xizmatning rasmiy bajarilishi emas, foydalanuvchiga yetib borgan amaliy qiymat bo‘lishi zarur.

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Jarayon uchun mas’ul tashkilot, axborot olish manbasi, belgilangan xizmat muddati va shikoyatni ko‘rib chiqish tartibi bir hujjatda qayd etiladi. Pilotda kutilgan natija kuzatilmasa, avval dastur nazariyasining noto‘g‘ri taxminlari, so‘ng resurs va ijro intizomi tekshiriladi. Hududlar o‘rtasida xulosalar chiqarishda iqtisodiy sharoit va qatnashchilarning dastlabki holati bo‘yicha farqlar hisobga olinishi shart. Har qanday kengaytirish qarori mustaqil baholashdan keyin qabul qilinishi maqsadga muvofiq.

13. Inson nazorati

Raqamli tavsiya moduli qaysi ish o‘rni ko‘proq mos ekanini tez hisoblab bera olsa-da, u talabgorning barcha sharoitini bilmaydi. Oilaviy majburiyat, qatnov, ish grafigi yoki shaxsiy afzalliklar algoritmda yetarli aks etmasligi mumkin. Shu sababli tavsiya rad etilganda xizmat ko‘rsatish to‘xtab qolmasligi, maslahatchi muqobil variantni taklif qilishi kerak. Algoritmik moslikni ishga qabul qilish kafolati deb taqdim etish ham noto‘g‘ri. Qonuniy mas’uliyat inson va tashkilot zimmasida qolishi zarur.

Inson nazorati yo‘nalishida boshqaruv qarorining mezoni xizmatning rasmiy bajarilishi emas, foydalanuvchiga yetib borgan amaliy qiymat bo‘lishi zarur. Jarayon uchun mas’ul tashkilot, axborot olish manbasi, belgilangan xizmat muddati va shikoyatni ko‘rib chiqish tartibi bir hujjatda qayd etiladi. Pilotda kutilgan natija kuzatilmasa, avval dastur nazariyasining noto‘g‘ri taxminlari, so‘ng resurs va ijro intizomi tekshiriladi. Hududlar o‘rtasida xulosalar chiqarishda iqtisodiy sharoit va qatnashchilarning dastlabki holati bo‘yicha farqlar hisobga olinishi shart. Har qanday kengaytirish qarori mustaqil baholashdan keyin qabul qilinishi maqsadga muvofiq.

14. Bosqichma-bosqich joriy etish

Birinchi bosqichda mavjud xizmatlar xaritasi va huquqiy vakolatlar inventarizatsiyasi qilinadi. Ikkinchi bosqichda ma’lumotning majburiy maydonlari, almashish protokoli va murojaat statuslari kelishiladi. Uchinchi bosqichda maslahatchilar hamda korxonalar uchun amaliy o‘quv tashkil etiladi. To‘rtinchi bosqichda cheklangan pilot o‘tkazilib, xizmat sifatiga oid shikoyatlar yig‘iladi. Beshinchi bosqichda mustaqil baholash asosida tizimni kengaytirish yoki ayrim elementlardan voz kechish haqida qaror qabul qilinadi. Har bir bosqich keyingisi uchun tekshiriladigan shartlarni ta’minlashi kerak.

Bosqichma-bosqich joriy etish yo‘nalishida boshqaruv qarorining mezoni xizmatning rasmiy bajarilishi emas, foydalanuvchiga yetib borgan amaliy qiymat bo‘lishi zarur. Jarayon uchun mas’ul tashkilot, axborot olish manbasi, belgilangan

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xizmat muddati va shikoyatni ko‘rib chiqish tartibi bir hujjatda qayd etiladi. Pilotda kutilgan natija kuzatilmasa, avval dastur nazariyasining noto‘g‘ri taxminlari, so‘ng resurs va ijro intizomi tekshiriladi. Hududlar o‘rtasida xulosalar chiqarishda iqtisodiy sharoit va qatnashchilarning dastlabki holati bo‘yicha farqlar hisobga olinishi shart. Har qanday kengaytirish qarori mustaqil baholashdan keyin qabul qilinishi maqsadga muvofiq.

15. Boshqaruvga qayta aloqa

Monitoring faqat yuqori tashkilotga hisobot berish amaliyotiga aylanib qolsa, u xizmat sifatini o‘zgartirmaydi. Modelda ish beruvchilar talabining yangilanishi ta’lim tashkilotlariga, ish izlovchining rad javobi sabablari esa maslahatchiga qaytishi kerak. Shu tariqa ma’lumot yig‘ishning o‘zi emas, yig‘ilgan axborot asosida xizmat dizaynini qayta ko‘rish institutsional o‘rganish mexanizmini hosil qiladi. Qaror qabul qilishning davriyligi va mas’ul shaxslari avvaldan yozma belgilanmasa, qayta aloqa formal bosqichga aylanadi.

Boshqaruvga qayta aloqa yo‘nalishida boshqaruv qarorining mezoni xizmatning rasmiy bajarilishi emas, foydalanuvchiga yetib borgan amaliy qiymat bo‘lishi zarur. Jarayon uchun mas’ul tashkilot, axborot olish manbasi, belgilangan xizmat muddati va shikoyatni ko‘rib chiqish tartibi bir hujjatda qayd etiladi. Pilotda kutilgan natija kuzatilmasa, avval dastur nazariyasining noto‘g‘ri taxminlari, so‘ng resurs va ijro intizomi tekshiriladi. Hududlar o‘rtasida xulosalar chiqarishda iqtisodiy sharoit va qatnashchilarning dastlabki holati bo‘yicha farqlar hisobga olinishi shart. Har qanday kengaytirish qarori mustaqil baholashdan keyin qabul qilinishi maqsadga muvofiq.

MUHOKAMA

Modelning afzalligi bandlik siyosatini hujjatlarni qayd qilish jarayonidan natijani boshqarishga yo‘naltirishidadir. O‘zaro mos xizmatlar birligi takroriy murojaatlarni qisqartirishi, fuqaro uchun tushunarli yo‘nalish yaratishi va qaysi bosqichda muammo yuzaga kelganini ko‘rsatishi mumkin. Shunga qaramay, integratsiya barcha vakolatlarni bitta idoraga jamlash degani emas: vakolatlar saqlangan holda ma’lumot almashinuvi va javobgarlik protokoli uyg‘unlashtiriladi.

Taklifning eng muhim xavfi “ko‘rsatkich uchun ishlash” holatidir. Agar tashkilot faqat R6 ni oshirish uchun baholansa, murakkab ijtimoiy to‘siqqa duch kelgan yoshlarni qabul qilishdan qochishi mumkin. Buni kamaytirish uchun natijalar ijtimoiy guruhlar va boshlang‘ich ehtiyoj darajalari bo‘yicha ajratib e’lon qilinishi, shuningdek xizmatga qamrov va teng imkoniyat indikatorlari ham qo‘llanilishi kerak.

Dastur bahosi ichki nazorat bilan cheklanmasligi maqsadga muvofiq. Bosqichma-bosqich joriy etishda hududlar yoki guruhlarning boshlang‘ich

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ko‘rsatkichlari solishtiriladi, kuzatuvdan chiqib ketgan fuqarolar ulushi ochiq ko‘rsatiladi, xarajatlar esa nafaqat ma’muriy, balki o‘qitish va subsidiyalash yo‘nalishlari kesimida hisoblanadi. Sababiy samaradorlikni sinash uchun mos taqqoslash guruhi bo‘lmasa, ishda qolish yuqori bo‘lgani aynan dastur tufayli yuz berdi, deb da’vo qilib bo‘lmaydi.

Raqamli ma’lumot almashinuvi shaxsiy ma’lumotlarni muhofaza qilish majburiyatlarini kuchaytiradi. Kirish huquqlari lavozim bo‘yicha cheklanishi, aralashuvlar uchun rozilik va huquqiy asos hujjatlashtirilishi, ma’lumotlar uzatish jurnali yuritilishi zarur. Bundan tashqari, nogironligi bo‘lgan yoshlar va raqamli xizmatlardan foydalana olmaydigan shaxslar uchun muqobil xizmat kanallari ko‘zda tutilishi kerak.

Ushbu maqolaning birinchi maqoladan asosiy farqi shundaki, unda muammoning tasnifi emas, balki aniq xizmat zanjiri, ijrochi mas’uliyati va natija indikatorlari loyihalashtirilmoqda. Demak, u bandlik siyosatini tashkil etish va baholash metodologiyasini takomillashtirishga qaratilgan mustaqil ilmiy ishlanma sifatida qaralishi mumkin.

XULOSA

Yoshlar bandligi bo‘yicha mavjud tashkiliy vositalarning samaradorligi ular sonidan ko‘ra yagona natijaga xizmat qilish darajasiga bog‘liq. Ishda taklif etilgan diagnostika, individual reja, ish beruvchi bilan moslashtirish, joylashtirishni tasdiqlash va keyingi monitoringdan iborat besh bosqichli model ushbu bog‘lanishni amaliy boshqaruv tartibiga keltiradi.

Modelni joriy etishdan oldin bir yoki ikki hududda cheklangan pilot sinov o‘tkazish, huquqiy ma’lumot almashish tartibini belgilash, bazaviy bandlik ko‘rsatkichlarini yig‘ish va olti-o‘n ikki oylik kuzatuvni tashkil qilish tavsiya etiladi. Yalpi joylashtirish soni bilan birga barqaror bandlik, qamrovdagi adolatlilik va bir barqaror natijaga sarflangan xarajat ham baholanishi kerak. Ushbu model empirik tasdiqqa muhtoj ilmiy-amaliy taklif hisoblanadi.

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BO‘YIN UMURTQALARIDAGI DEGENERATIV O‘ZGARISHLARNING ANATOMIK XUSUSIYATLARI VA YOSH BILAN BOG‘LIQLIGI

Yusupov Fayozbek
Fayozbekyusupo533@gmail.com
Qudratov Ozodbek Ortiq o‘g‘li
Surxondariyo Ilmu-ziyo tibbiyot texnikumi o‘qituvchisi
Kudratovozodbek6765@gmail.com

Annotatsiya: bo‘yin umurtqa pog‘onasi degeneratsiyasining anatomik asoslari va yoshga bog‘liq radiologik ko‘rinishlari adabiyotlar sharhi orqali tahlil qilinadi. Maqsad umurtqalararo disk, umurtqa tanasi, bo‘g‘imlar va umurtqa kanali o‘rtasidagi tarkibiy munosabatlarni degenerativ o‘zgarishlar nuqtayi nazaridan yoritishdir. Usul sifatida tekshirilgan ilmiy manbalarni mavzular bo‘yicha qiyosiy umumlashtirish qo‘llanildi. Natijalar disk degeneratsiyasi yoshi katta guruhlarda ko‘proq aniqlanishini, biroq tasviriy o‘zgarishlar alomatsiz odamlarda ham uchrashini ko‘rsatdi. Anatomik torayishning joylashuvi nerv ildizi yoki orqa miya zararlanishining turli ko‘rinishlarini tushuntiradi.

Kalit so‘zlar: bo‘yin umurtqalari, anatomik tuzilish, umurtqalararo disk, degeneratsiya, spondiloz, osteofit, yosh omili, magnit rezonans tomografiya.

Kirish

Bo‘yin umurtqa pog‘onasidagi degenerativ o‘zgarishlar disklar va ularga tutash suyak hamda bo‘g‘im tuzilmalarini qamrab oladi. Ushbu o‘zgarishlarning ahamiyati faqat tasvirlarda ko‘rinishi bilan cheklanmaydi. Disk balandligining kamayishi va suyak o‘simtalari umurtqalararo teshik yoki umurtqa kanali bo‘shlig‘iga ta’sir qilishi mumkin. Shu sababli anatomik tuzilishlarni bir-biridan ajratib baholash degenerativ jarayonning oqibatlarini to‘liq tushuntirmaydi. Shedid va Benzel disk hamda faset bo‘g‘imlarining yuk uzatish va harakatdagi o‘zaro ishtirokini bo‘yin spondilozining muhim biomekanik asosi sifatida ko‘rsatadi.

Yosh bilan degeneratsiya o‘rtasidagi bog‘liqlikni tushuntirishda radiologik belgi va klinik kasallikni farqlash zarur. Matsumoto va hammualliflarning alomatsiz ko‘ngillilar ishtirokidagi tekshiruvi diskdagi o‘zgarishlar shikoyatsiz odamlarda ham aniqlanishini ko‘rsatgan. Demak, MRT xulosasidagi degeneratsiya og‘riqning manbasini o‘z-o‘zidan isbotlamaydi. Ushbu masala anatomik bilimlarni klinik talqin bilan bog‘lashda alohida ahamiyatga ega.

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Tadqiqotning maqsadi bo‘yin umurtqa pog‘onasida degenerativ o‘zgarishlarning asosiy anatomik ko‘rinishlarini tizimlashtirish, yosh bilan bog‘liq dalillarni tahlil qilish va ularning talqin chegaralarini aniqlashdan iborat.

Materiallar va metodlar

Ish narrativ adabiyotlar sharhi sifatida tayyorlandi. PubMed va PubMed Central orqali bo‘yin spondilozi anatomiyasi, disk degeneratsiyasi, yosh omili va tasviriy tekshiruvlarga oid manbalar ko‘rib chiqildi. Qidiruvda cervical spondylosis anatomy, cervical disc degeneration age hamda asymptomatic cervical MRI iboralari ishlatildi. Mazmuniy tahlil uchun mavzuga bevosita tegishli to‘rtta tekshirilgan nashr tanlandi: alomatsiz shaxslarda MRT natijalarini bayon qilgan bir tadqiqot va anatomik, klinik hamda diagnostik jihatlarni yoritgan uchta sharh nashri.

Manbalar uch yo‘nalishda tahlil qilindi: degeneratsiyaning anatomik joylashuvi, yosh bo‘yicha radiologik farqlar va tasviriy o‘zgarishlarning klinik ahamiyati. Raqamli ma’lumotlar birlamchi nashrdagi birliklar saqlangan holda olindi. Bemorlar soni bilan tekshirilgan disklar ulushi aralashtirilmadi. Natijalar birlashtirilgan statistik baho sifatida hisoblanmadi, yangi korrelatsiya koeffitsiyenti yoki statistik ahamiyat darajasi chiqarilmadi.

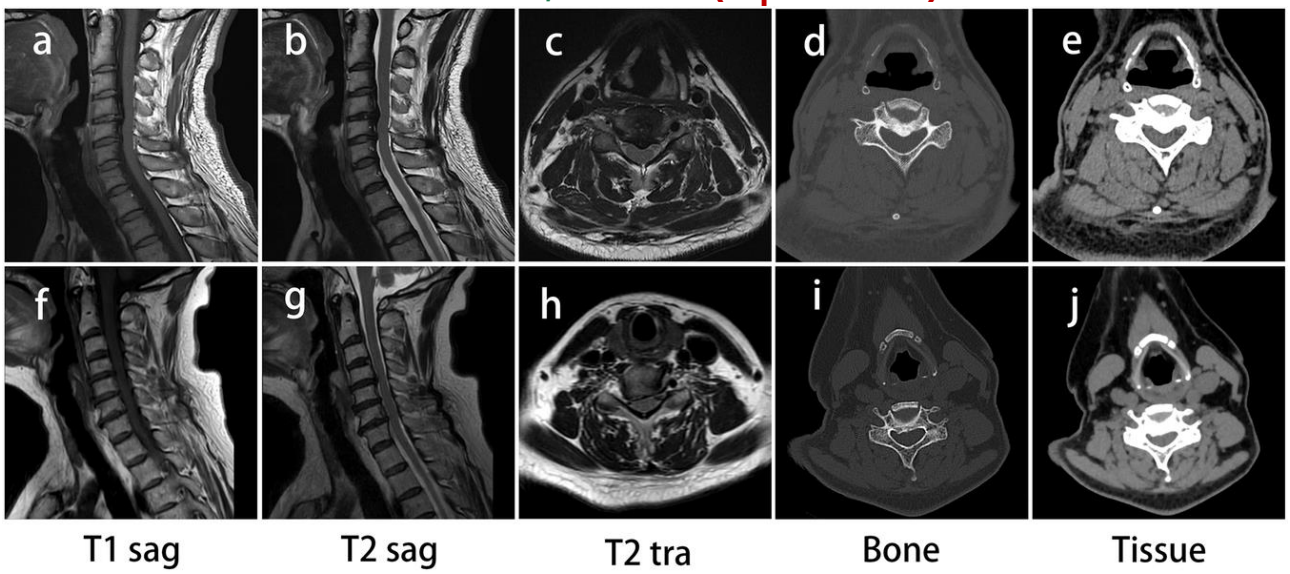
Sharh to‘liq tizimli qidiruv yoki metatahlil hisoblanmaydi. Tadqiqotlar dizayni va tanlanmalari turlicha bo‘lgani sababli raqamlar barcha aholi uchun universal ko‘rsatkich sifatida talqin qilinmadi. Illyustratsiya uchun Yu va hammualliflarning 2025-yilgi ochiq ilmiy nashridan haqiqiy MRT va KT tasvirlari olindi. Ular ushbu sharh muallifining klinik tanlanmasiga tegishli emas.

Natijalar

Degenerativ o‘zgarishlarning anatomik joylashuvi

Adabiyotlar tahlili disk degeneratsiyasi, osteofitlar va bo‘g‘im o‘zgarishlari o‘zaro bog‘langan jarayon sifatida qaralishini ko‘rsatdi. Diskning mexanik xususiyatlari o‘zgarganda umurtqalar orasida yuk taqsimlanishi ham o‘zgaradi. Shedid va Benzelning sharhida disklar va faset bo‘g‘imlari kuchlarni uzatuvchi hamda harakatni ta’minlovchi tuzilmalar sifatida tavsiflanadi. Osteofitlar umurtqa kanali bo‘shlig‘ini kamaytirishi va asab tuzilmalariga bosim o‘tkazishi mumkin. Bunda kanalning dastlabki anatomik kengligi ham ahamiyatlidir.

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1-rasm. Bo‘yin spondilozida haqiqiy MRT va KT tasvirlari. Yuqori qator — 51 yoshli erkak; pastki qator — 57 yoshli ayol. a va f — sagittal T1 MRT; b va g — sagittal T2 MRT; c va h — aksial T2 MRT; d va i — KT suyak oynasi; e va j — KT yumshoq to‘qima oynasi. Manba: Yu va hammualliflar, Scientific Data, 2025, asl nashrdagi 2-rasm.. Tasvir o‘zgartirilmagan.

Kelly va hammualliflar sharhiga ko‘ra, degenerativ o‘zgarishlar ko‘pincha C5–C6 segmentida, undan keyin C6–C7 va C4–C5 darajalarida uchraydi. Ushbu joylashuvni barcha odamlarda bir xil ketma-ketlikda kechadigan qonuniyat deb bo‘lmaydi. Alohida shaxsda qaysi segment zararlangani tasviriy tekshiruv orqali aniqlanadi.

Yoshga bog‘liq radiologik ko‘rsatkichlar

Matsumoto va hammualliflar 497 nafar alomatsiz shaxsning bo‘yin disklarini MRT yordamida tekshirgan. Baholash C2–C3 dan C6–C7 gacha bo‘lgan beshta disk darajasini qamrab olgan. Disk signali pasayishi, oldinga va orqaga protruzion o‘zgarishlar, disk oralig‘i torayishi hamda umurtqalararo teshik stenozi o‘rganilgan. Degenerativ belgilarning uchrash tezligi yosh bilan ortgan.

20–29 yoshli erkaklarda disklarning 17 foizida, ayollarda 12 foizida degeneratsiya qayd etilgan. 60 yoshdan katta guruhda bu ko‘rsatkichlar erkaklarda 86 foizga, ayollarda 89 foizga yetgan. Bu yerda faqat mazkur nashrda berilgan ikki yosh toifasining qiymatlari keltirildi; oraliq yosh guruhlari uchun qiymatlar taxmin qilinmagan. Ushbu natijalar diskdagi strukturaviy o‘zgarishlarning yosh bilan bog‘liqligini ko‘rsatadi, biroq ularning albatta shikoyat keltirib chiqarishini anglatmaydi.

Tasviriy belgilarni anatomik talqin qilish

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Butler va hammualliflarning sharhida MRT yumshoq to‘qimalar tafsilotlarini ko‘rsatishda KTga nisbatan ustun ekani ta’kidlangan. KT ayrim sharoitlarda, jumladan MRTga qarshi ko‘rsatma yoki metall artefaktlari mavjud bo‘lganda, foydali bo‘lishi mumkin. Shu sababli tekshiruv usuli qaysi anatomik tuzilma va qanday savol baholanayotganiga qarab tanlanadi.

Kelly va hammualliflar umurtqalararo teshik torayishini nerv ildizi bilan, umurtqa kanali torayishini esa orqa miya kompressiyasi bilan bog‘laydi. Anatomik joylashuv radikulopatiya va miyelopatiyaning farqini tushuntiradi. Biroq tasvirdagi torayishning o‘zi klinik sindromni tasdiqlash uchun yetarli emas.

Muhokama

Tahlilning asosiy xulosasi shuki, yosh degenerativ o‘zgarishlarning uchrashiga bog‘liq, ammo klinik holatning yagona izohi bo‘la olmaydi. Alomatsiz odamlarning MRT tasvirlarida ham degeneratsiya mavjudligi radiologik tashxisni ehtiyotkorlik bilan talqin qilishni talab etadi. Matsumoto tadqiqoti bu masalada ayniqsa muhim: uning tanlanmasi shikoyati bo‘lmagan shaxslardan tuzilgan. Shuning uchun ushbu ko‘rsatkichlarni davolanishga murojaat qilgan bemorlardagi kasallik tarqalishi deb ko‘rsatish metodologik xato bo‘ladi.

Anatomik jihatdan bir xil nom bilan ifodalangan degeneratsiya turli joylarda turlicha oqibatga olib kelishi mumkin. Kelly sharhida bayon qilingan teshik va kanal torayishi o‘rtasidagi farq bunga misoldir. Mazkur dalillardan kelib chiqib, tasviriy xulosada faqat degeneratsiya borligini emas, balki segment, torayish yo‘nalishi va tegishli asab tuzilmasi bilan munosabatni qayd etish foydali, deb hisoblash mumkin. Bu sharh muallifining manbalar asosidagi metodik xulosasidir.

Yosh guruhlari o‘rtasidagi farqlarni baholashda kuzatuv birligi muhim. Disklar ulushi odamlar ulushiga teng emas: bir odamda bir nechta disk tekshiriladi. Natijalarda aynan disklar ulushi ko‘rsatilgani ushbu xatoning oldini oladi. Jinslar orasidagi ikki ko‘rsatkichning farqi esa yangi statistik tahlilsiz umumiy ustunlik yoki moyillik sifatida talqin qilinmadi.

Sharhning cheklovlari manbalar sonining kamligi, qidiruvning tizimli emasligi va miqdoriy natijaning bitta klassik tadqiqotdan olinganidir. O‘zbekiston aholisi bo‘yicha mustaqil tanlanma o‘rganilmagan. Shu sababli natijalar mahalliy tarqalish darajasini ifodalamaydi. Kelgusi tadqiqotda yosh guruhlari, simptomlar, segmentlar va tasviriy baholash mezonlari oldindan belgilangan holda tekshiruv o‘tkazilishi maqsadga muvofiq.

Xulosa

Bo‘yin umurtqa pog‘onasidagi degeneratsiya disk, bo‘g‘im va suyak tuzilmalarining o‘zaro munosabatlari orqali namoyon bo‘ladi. Tekshirilgan

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adabiyotlarda yosh ulg‘ayishi bilan radiologik o‘zgarishlar ko‘proq uchrashi ko‘rsatilgan. Shu bilan birga, alomatsiz odamlarda ham disk degeneratsiyasi mavjud bo‘lishi mumkin. Shuning uchun anatomik o‘zgarishlar klinik belgilar bilan birgalikda talqin qilinishi kerak.

Ushbu sharh yangi klinik natija bermaydi; mavjud dalillarning anatomik mazmunini umumlashtiradi. Amaliy jihatdan segmentning aniq joylashuvi, umurtqalararo teshik va kanal holati hamda asab tuzilmalari bilan munosabatni baholash yoshning o‘zini asosiy mezon qilib olishdan ko‘ra mazmunliroq ma’lumot beradi.

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TARIX FALSAFASINING JAMIYAT TARAQQIYOTINI ANGLASHDAGI O‘RNI VA AHAMIYATI

Muallif: Mamatqulova Nilufar Xusanovna
Ta’lim muassasasi: Toshkent davlat tibbiyot universiteti Termiz filiali

Annotatsiya. Ushbu maqolada tarix falsafasining mohiyati, uning shakllanishi va rivojlanish bosqichlari, jamiyat taraqqiyotini anglashdagi nazariy hamda amaliy ahamiyati tahlil qilinadi. Shuningdek, tarixiy jarayonlarning qonuniyatlari, inson omilining tarixdagi o‘rni, tarixiy xotira va milliy o‘zlikni anglash masalalariga e’tibor qaratiladi.

Kalit so‘zlar: tarix falsafasi, tarixiy tafakkur, jamiyat taraqqiyoti, tarixiy jarayon, tarixiy xotira, inson omili, sivilizatsiya, milliy o‘zlik.

Kirish

Tarix insoniyatning o‘tmishi, buguni va kelajagini o‘zaro bog‘lab turuvchi muhim ijtimoiy hodisadir. Insoniyat tomonidan yaratilgan moddiy va ma’naviy qadriyatlar, siyosiy tuzumlar, madaniyatlar hamda ijtimoiy munosabatlar tarixiy jarayonlar davomida shakllanib, rivojlanib kelgan. Ushbu jarayonlarning mazmun-mohiyatini chuqur anglash esa tarixga nafaqat voqealar ketma-ketligi sifatida, balki falsafiy tafakkur obyekti sifatida yondashishni talab etadi.

Tarix falsafasi tarixiy voqea va hodisalarning mazmunini, taraqqiyot yo‘nalishlarini, sabab va oqibat munosabatlarini hamda insoniyat rivojlanishining umumiy tamoyillarini o‘rganuvchi falsafiy yo‘nalishdir. U tarixiy jarayonlarning qanday kechganini o‘rganish bilan birga, nima sababdan muayyan voqealar yuz bergani, ularning jamiyat hayotiga qanday ta’sir ko‘rsatgani va kelajak uchun qanday xulosalar chiqarish mumkinligini ham tahlil qiladi.

Bugungi globallashuv sharoitida tarix falsafasining ahamiyati yanada ortib bormoqda. Chunki tarixiy tajribani to‘g‘ri anglash jamiyatning ma’naviy barqarorligini mustahkamlash, milliy o‘zlikni anglash va kelajak taraqqiyotining ustuvor yo‘nalishlarini belgilashda muhim ahamiyat kasb etadi.

1. Tarix falsafasining mohiyati va shakllanishi

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Tarix falsafasi falsafiy bilimlarning muhim tarkibiy qismi bo‘lib, tarixiy jarayonlarning umumiy mazmuni va qonuniyatlarini tushuntirishga intiladi. Uning asosiy vazifasi tarixiy voqealarni oddiy bayon qilish emas, balki ularning ichki bog‘liqligini, rivojlanish omillarini va insoniyat hayotidagi o‘rnini aniqlashdan iborat.

Tarix falsafasining shakllanishi qadimgi davrlarga borib taqaladi. Qadimgi yunon mutafakkirlari Gerodot va Fukidid tarixiy voqealarni o‘rganish hamda izohlashga katta hissa qo‘shganlar. Gerodot tarixiy voqealarni tasvirlash va turli xalqlarning urf-odatlarini o‘rganishga e’tibor qaratgan bo‘lsa, Fukidid tarixiy hodisalarning siyosiy sabablarini tahlil qilishga intilgan.

O‘rta asrlarda tarixni anglashda diniy va ma’naviy qarashlar muhim o‘rin egalladi. Sharq mutafakkirlari, jumladan, Abu Nasr Forobiy, Abu Rayhon Beruniy va Ibn Xaldun inson, jamiyat va tarixiy taraqqiyot masalalarini chuqur tahlil qilganlar. Ayniqsa, Ibn Xaldunning jamiyatlar rivoji, davlatlarning vujudga kelishi va tanazzuli haqidagi qarashlari tarix falsafasining rivojlanishida alohida ahamiyatga ega.

Yangi davrda tarixiy taraqqiyot masalalari Giambattista Viko, Georg Vilgelm Fridrix Gegel va Karl Marks kabi mutafakkirlarning asarlarida keng yoritildi. Gegel tarixni erkinlik va insoniyat ongining rivojlanishi bilan bog‘liq jarayon sifatida talqin qilgan. Marks esa jamiyat taraqqiyotida moddiy ishlab chiqarish, iqtisodiy munosabatlar va ijtimoiy ziddiyatlarning roliga alohida e’tibor qaratgan.

Mazkur qarashlar tarix falsafasining yagona, o‘zgarmas talqini mavjud emasligini ko‘rsatadi. Tarixiy jarayonlarni tushuntirishda turli nazariy yondashuvlar mavjud bo‘lib, ularning har biri jamiyat rivojlanishining muayyan jihatlarini ochib beradi.

2. Tarixiy taraqqiyot va uning asosiy omillari

Tarixiy taraqqiyot murakkab va ko‘p omilli jarayon bo‘lib, unda iqtisodiy, siyosiy, ijtimoiy, madaniy, ma’naviy va tabiiy omillar o‘zaro ta’sir ko‘rsatadi. Shu sababli tarixiy voqealarni faqat bitta sabab bilan izohlash ko‘pincha biryoqlama xulosalarga olib keladi.

Birinchidan, iqtisodiy omillar jamiyat taraqqiyotining muhim asoslaridan biridir. Ishlab chiqarish usullari, mehnat taqsimoti, savdo aloqalari va mulkiy munosabatlardagi o‘zgarishlar ijtimoiy hayotning boshqa sohalariga ham ta’sir ko‘rsatadi. Masalan, Buyuk ipak yo‘li orqali amalga oshirilgan savdo aloqalari

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Markaziy Osiyoda shaharlar rivoji, hunarmandchilik va madaniy almashinuvning kuchayishiga xizmat qilgan.

Ikkinchidan, siyosiy omillar tarixiy jarayonlarning yo‘nalishini belgilashda muhim ahamiyatga ega. Davlat boshqaruvi, huquqiy tizim, siyosiy qarorlar va xalqaro munosabatlar jamiyat hayotining rivojlanishiga bevosita ta’sir qiladi. Tarixiy tajriba samarali boshqaruv va ijtimoiy barqarorlik o‘rtasidagi bog‘liqlikni o‘rganish imkonini beradi.

Uchinchidan, ma’naviy va madaniy omillar jamiyatning ichki birligi va taraqqiyotida katta rol o‘ynaydi. Til, din, urf-odatlar, ilm-fan, ta’lim va san’at tarixiy xotirani shakllantirib, avlodlar o‘rtasidagi ma’naviy davomiylikni ta’minlaydi.

To‘rtinchidan, inson omili tarixiy taraqqiyotning muhim tarkibiy qismidir. Tarixni faqat obyektiv qonuniyatlar natijasi sifatida talqin qilish insonning ongli faoliyati, tanlovi va mas’uliyatini yetarlicha hisobga olmaslikka olib kelishi mumkin. Aslida, tarixiy jarayonlar ijtimoiy sharoitlar bilan bir qatorda, insonlarning qarorlari, tashabbuslari va faoliyati orqali ham shakllanadi.

Demak, tarixiy taraqqiyotni chuqur anglash uchun turli omillarni o‘zaro bog‘liqlikda o‘rganish va tarixiy hodisalarni muayyan davr sharoitidan kelib chiqib baholash zarur.

3. Tarix falsafasi va tarixiy xotira

Tarixiy xotira xalq va jamiyatning o‘z o‘tmishi haqidagi tasavvurlari, qadriyatlari va tajribalarini saqlash hamda avlodlarga yetkazish jarayonidir. U milliy o‘zlikni anglash, ijtimoiy birdamlikni mustahkamlash va ma’naviy merosni asrashda muhim o‘rin tutadi.

Tarix falsafasi tarixiy xotirani tanqidiy va ilmiy yondashuv asosida o‘rganishga yordam beradi. Chunki tarixiy xotira faqat o‘tmish voqealarining saqlanishidan iborat emas, balki ularning jamiyat tomonidan qanday talqin qilinishi bilan ham bog‘liq. Ayrim tarixiy hodisalar turli davrlarda turlicha izohlanishi mumkin. Shu bois tarixiy manbalarni solishtirish, dalillarni tekshirish va voqealarni o‘z davri sharoitida tahlil qilish muhimdir.

O‘zbekiston tarixiga nazar tashlasak, qadimgi sivilizatsiyalar, Buyuk ipak yo‘li, ilm-fan va madaniyat rivoji, buyuk mutafakkirlar merosi xalqimizning tarixiy tajribasi naqadar boy ekanini ko‘rsatadi. Muhammad al-Xorazmiy, Ahmad al-Farg‘oniy, Abu

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Rayhon Beruniy, Abu Ali ibn Sino, Mirzo Ulug‘bek va Alisher Navoiy kabi allomalar jahon ilm-fani va madaniyati rivojiga katta hissa qo‘shganlar.

Ularning merosini o‘rganish tarixiy faxr tuyg‘usini shakllantirish bilan birga, zamonaviy avlodni ilm olishga, izlanishga va yangilik yaratishga undaydi. Biroq tarixiy merosga munosabat faqat o‘tmish bilan faxrlanishdan iborat bo‘lmasligi kerak. U tarixdan saboq olish, yutuqlar bilan birga xatolarni ham xolis baholash va kelajak uchun asosli xulosalar chiqarishni taqozo etadi.

Shunday ekan, tarixiy xotira ilmiy haqiqat, tanqidiy tafakkur va umuminsoniy qadriyatlar bilan uyg‘unlashgandagina jamiyat taraqqiyotiga samarali xizmat qiladi.

4. Globallashuv sharoitida tarix falsafasining dolzarbligi

XXI asrda globallashuv, axborot texnologiyalarining rivojlanishi va madaniyatlararo aloqalarning kengayishi tarixni anglashga bo‘lgan ehtiyojni kuchaytirmoqda. Bugungi kunda tarixiy ma’lumotlar internet va ijtimoiy tarmoqlar orqali tez tarqalmoqda. Biroq bu jarayon ishonchsiz ma’lumotlar, tarixiy faktlarning buzib talqin qilinishi va turli mafkuraviy qarashlarning tarqalishi xavfini ham yuzaga keltiradi.

Bunday sharoitda tarix falsafasi insonlarda mustaqil fikrlash, dalillarni tahlil qilish va voqealarning sabab-oqibatlarini aniqlash ko‘nikmalarini rivojlantirishga yordam beradi. Tarixiy hodisalarni faqat tashqi ko‘rinishi bilan emas, balki ularning ijtimoiy, iqtisodiy va ma’naviy asoslari bilan birgalikda o‘rganish zarur.

Tarix falsafasi turli xalqlar va sivilizatsiyalar tajribasini qiyosiy o‘rganish uchun ham nazariy asos yaratadi. Bunday yondashuv insoniyat tarixidagi umumiy jihatlar bilan bir qatorda, har bir xalqning o‘ziga xos taraqqiyot yo‘lini ham anglash imkonini beradi.

Shuningdek, tarixiy tajribani o‘rganish tinchlikni saqlash, nizolarning sabablarini tushunish, bag‘rikenglikni rivojlantirish va jamiyatda o‘zaro hurmat muhitini mustahkamlash uchun muhimdir. Tarixni chuqur bilish o‘tmishdagi ziddiyatlarni takrorlamaslikka, inson huquqlari va erkinliklarining qadriga yetishga yordam beradi.

Demak, tarix falsafasi faqat nazariy fan bo‘lib qolmasdan, zamonaviy jamiyatning ma’naviy va intellektual rivojlanishiga xizmat qiladigan muhim bilim sohasi hisoblanadi.

Xulosa

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Xulosa qilib aytganda, tarix falsafasi insoniyatning tarixiy tajribasini chuqur anglash, jamiyat taraqqiyotining asosiy omillarini aniqlash va kelajak uchun ilmiy asoslangan xulosalar chiqarishda muhim ahamiyatga ega. U tarixiy voqealarni oddiy bayon qilish bilan cheklanmay, ularning mohiyati, sabablari, oqibatlari va o‘zaro bog‘liqligini tahlil qiladi.

Tarixiy taraqqiyot iqtisodiy, siyosiy, ijtimoiy va ma’naviy omillarning murakkab o‘zaro ta’siri natijasida yuz beradi. Shu bois tarixiy hodisalarni xolis, tizimli va tanqidiy yondashuv asosida o‘rganish zarur.

Tarixiy xotirani asrash, milliy va umuminsoniy qadriyatlarni anglash, tarixiy merosni ilmiy asosda o‘rganish jamiyatning ma’naviy rivojlanishiga xizmat qiladi. Ayniqsa, globallashuv va axborot oqimi kuchaygan bugungi davrda tarixiy tafakkurni rivojlantirish yosh avlodni mustaqil fikrlaydigan, o‘z tarixini biladigan va kelajak oldidagi mas’uliyatini anglaydigan shaxslar etib tarbiyalashda muhim o‘rin tutadi.

Shunday qilib, tarix falsafasini o‘rganish nafaqat o‘tmishni anglash, balki bugungi voqelikni to‘g‘ri baholash va kelajak taraqqiyotining istiqbollarini belgilash uchun ham zarurdir.

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TYPHOID FEVER: CLINICAL FEATURES, DIAGNOSIS, TREATMENT AND PREVENTION

Mohd Arshad , Medical Student, Samarkand state medical university, Uzbekistan

Rohit Kumar Nirala , Medical Student, Samarkand state medical university, Uzbekistan

Mohd . Aslam , Medical Student, Samarkand state medical university, Uzbekistan

Mohd. Zaid , Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Typhoid fever is a systemic bacterial infection caused by Salmonella enterica serovar Typhi. It remains an important public-health concern where drinking water, sanitation and food safety systems are unreliable. Transmission usually occurs when people ingest food or water contaminated with faeces from an infected person or a chronic carrier. The illness can begin gradually with persistent fever, headache, weakness, abdominal discomfort and altered bowel habits; severe disease may lead to intestinal bleeding, perforation or other complications. Because symptoms overlap with many febrile illnesses, clinical suspicion should be supported by appropriate microbiological testing, preferably blood culture. Antibiotic selection must reflect local resistance patterns and susceptibility results, as drug-resistant Salmonella Typhi increasingly complicates care. Prevention requires safe water, effective sanitation, hand hygiene, food-safety practices and vaccination where recommended. This narrative review summarises the organism, transmission, clinical presentation, diagnosis, management and prevention of typhoid fever, drawing on guidance from the World Health Organization (WHO) and the US Centers for Disease Control and Prevention (CDC).

Keywords: Typhoid fever; Salmonella Typhi; enteric fever; blood culture; antimicrobial resistance; vaccination; sanitation; food safety.

1. Introduction

Typhoid fever, also called enteric fever, is a potentially serious infection that affects the whole body rather than only the intestine. It is caused by Salmonella enterica serovar Typhi, a bacterium adapted to humans. Paratyphoid fever is a related illness caused by Salmonella Paratyphi and may look clinically similar, although the organisms are different. Typhoid continues to occur in parts of the world where access to safe drinking water and adequate sanitation is limited. WHO cited estimates for 2019 of approximately nine million illnesses and 110,000 deaths globally, illustrating the

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continuing scale of the problem [1]. These are modelled estimates for that year, not a count of cases occurring in the current year.

The disease is preventable and treatable, but outcomes depend on early recognition, access to effective antibiotics and measures that interrupt faecal–oral transmission. A key challenge is that early symptoms are not specific: malaria, dengue, other bloodstream infections and gastrointestinal illnesses can present with fever and malaise. For this reason, typhoid should be considered in the appropriate epidemiological context, but not diagnosed from symptoms or a single serological test alone [2,3].

2. Causative organism and transmission

Salmonella Typhi enters the body through the mouth, most often through contaminated water or food. After ingestion, bacteria cross the intestinal lining and can spread through the bloodstream to other organs. An infected person may shed bacteria in stool; some people continue to carry and release the organism after symptoms resolve. These carriers can unknowingly contaminate food or water if hand hygiene and sanitation are inadequate [1,2]. Transmission is therefore closely linked to water treatment, sewage disposal, food handling and access to handwashing facilities.

Risk is higher for people living in or travelling to areas where typhoid is endemic, particularly where water supplies are unsafe or food is handled without adequate hygiene. Crowded living conditions and disrupted water and sanitation services can also increase opportunities for spread. A history of recent travel, unsafe food or water exposure, or contact with a known case can help guide clinical assessment.

3. Clinical features and natural history

The incubation period is commonly about 6–30 days, although it can vary with the dose of bacteria and individual factors [2]. Illness often develops progressively. Early symptoms may include headache, fatigue, loss of appetite, abdominal discomfort and fever that rises over several days. Constipation or diarrhoea may occur, and some patients develop a dry cough or faint, rose-coloured spots on the trunk. None of these findings is present in every patient, and no single symptom reliably confirms typhoid [2,3].

Untreated infection can persist and become severe. Important complications include gastrointestinal bleeding, intestinal perforation, confusion or encephalopathy, and circulatory complications. These are medical emergencies. Prompt evaluation is especially important when a persistent fever is accompanied by severe abdominal pain, blood in stool, confusion, fainting or marked weakness. A person with suspected

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typhoid should seek medical care rather than relying on home treatment or leftover antibiotics.

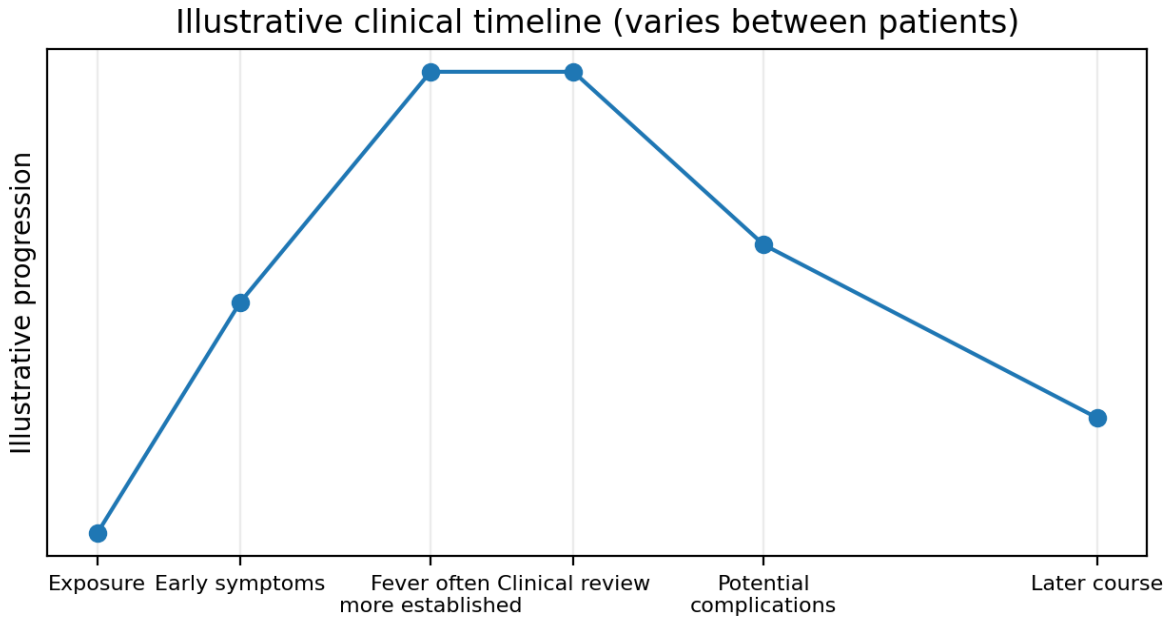


Figure 1. Illustrative timeline only; symptoms and progression differ between individuals. The figure is not a diagnostic tool.

4. Diagnosis

Laboratory confirmation is important because the clinical picture overlaps with other infections and treatment depends on identifying the likely organism and its antibiotic susceptibility. Blood culture is the preferred diagnostic method. A single culture may be negative, so clinicians may request additional cultures when suspicion remains high. Bone-marrow culture is more invasive but can be more sensitive in selected cases; stool culture is less useful early in the illness [2,3]. Where feasible, cultures should be collected before antibiotics are started, without delaying urgent treatment in a seriously ill patient.

The Widal test and other antibody-based rapid tests have limitations. They may produce false-positive results and may not reliably distinguish current infection from previous exposure or other causes of antibodies. Consequently, a Widal result should not be treated as definitive proof of typhoid. Test choice should follow local clinical guidance and laboratory capacity [2,3].

Test	Role	Main limitation

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Blood culture	Preferred test to confirm infection and obtain an isolate for susceptibility testing.	Sensitivity is imperfect; more than one sample may be needed.
Bone-marrow culture	Can be more sensitive in selected cases, including after antibiotics.	Invasive and not routinely used.
Stool culture	May help identify shedding or infection later in the course.	Often less sensitive early in illness.
Widal/serology	Sometimes used where culture access is limited.	False positives and poor ability to distinguish current from past infection.

Table 1. Common diagnostic approaches and their practical limitations. Summary based on CDC clinical guidance [2,3].

5. Treatment and antimicrobial resistance

Typhoid fever requires clinician-directed antibiotic treatment. The choice of medicine should be informed by the patient's severity, travel and exposure history, local resistance data and, whenever available, laboratory susceptibility results. Resistance to several commonly used antibiotics has increased, and extensively drug-resistant strains have been reported. A medicine that was effective in one region or year may not be appropriate elsewhere [2,4]. For this reason, this article does not recommend a single antibiotic regimen for all patients.

Patients should take antibiotics exactly as prescribed and complete the course unless their treating clinician changes the plan. Self-medicating, sharing antibiotics or stopping treatment as soon as fever improves can contribute to treatment failure and resistance. Hydration, appropriate nutrition and monitoring for complications are important supportive measures. People who are severely unwell, unable to drink, confused or showing signs of intestinal complications may need hospital care. If symptoms persist or return, medical reassessment is necessary.

Clinical improvement does not always mean that transmission risk has ended. Some patients may continue to shed the organism, and a small proportion become chronic carriers. Food handlers, healthcare workers and people caring for young children may need specific clearance or follow-up testing under local public-health rules before returning to certain duties [1,4].

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6. Prevention and public-health control

Typhoid prevention works best when household actions are supported by reliable public infrastructure. Safe water, sewage treatment, food-safety regulation and accessible handwashing facilities reduce exposure at community level. At household level, people should wash hands with soap after using the toilet and before preparing or eating food; use treated, boiled or sealed safe drinking water when water quality is uncertain; eat food that is thoroughly cooked and served hot; and wash or peel produce with safe water. Food prepared in unsafe conditions and ice made from unverified water can be sources of exposure [1,5].

Vaccination is an important additional layer of protection, especially in endemic settings and for travellers at risk. WHO recommends typhoid conjugate vaccines for use in relevant public-health programmes, with decisions based on national policy and epidemiology [6]. Vaccines reduce risk but do not provide complete protection, and they do not replace safe water, sanitation or food hygiene. Vaccine product, age eligibility and schedule differ by country; individuals should follow local immunisation guidance and seek advice before travel.

Prevention measure	Practical action
Safe water	Use a reliably treated supply; if safety is uncertain, boil or otherwise treat water according to public-health advice.
Hand hygiene	Wash with soap after toilet use and before handling food or eating.
Food safety	Cook food thoroughly; eat it hot; avoid unsafe raw produce, unpasteurised products and ice from uncertain sources.
Vaccination	Ask a healthcare professional or travel clinic whether typhoid vaccination is indicated and which schedule applies.
Carrier and case management	Follow medical advice, finish prescribed treatment and observe local rules for food-handling or childcare duties.

Table 2. Practical actions to reduce transmission, adapted from WHO and CDC prevention guidance [1,5,6].

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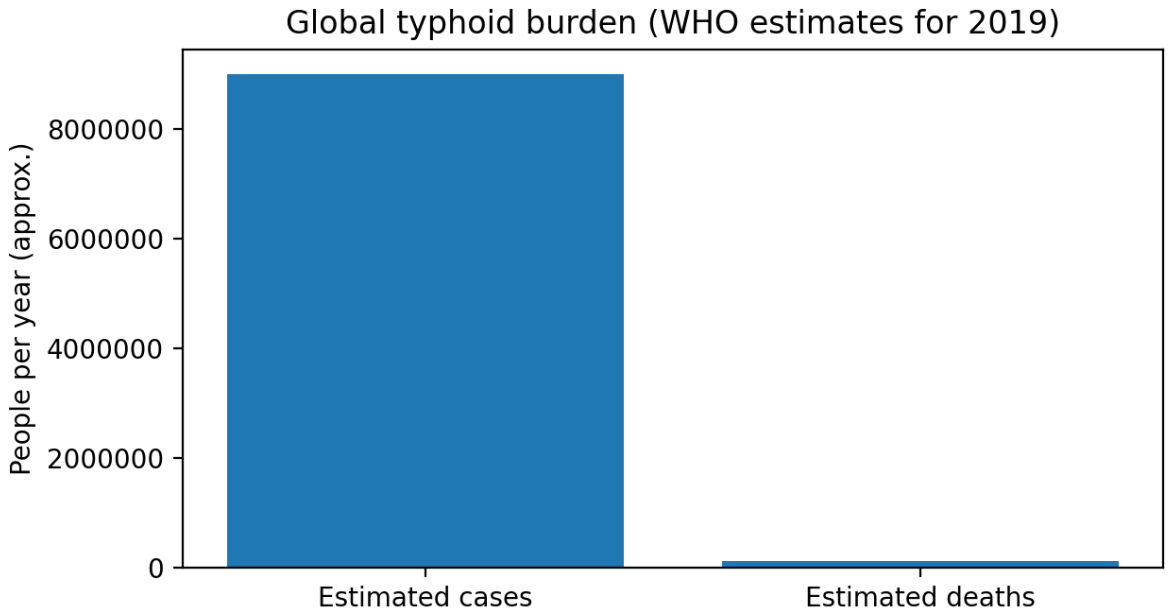


Figure 2. Approximate global burden cited by WHO for 2019. The values are estimates and the large difference in scale means deaths are visually compressed.

7. Discussion

Typhoid fever illustrates how clinical medicine and environmental health are connected. Effective treatment can save an individual patient, but it cannot by itself prevent new infections when contaminated water and unsafe food practices remain common. Likewise, vaccination can reduce disease burden but works best as part of a wider strategy that includes surveillance, laboratory capacity, sanitation and public education. Antimicrobial resistance makes this combined approach increasingly important: preventing infection reduces the need for antibiotics and therefore reduces opportunities for resistant organisms to spread.

Health education should be practical and locally relevant. Messages about handwashing, safe water storage, thorough cooking and early assessment of persistent fever are more useful when people have the resources to act on them. Public-health authorities also need timely reporting, reliable culture and susceptibility testing, and communication between clinicians, laboratories and environmental-health services. Future progress depends not on one intervention alone but on sustained improvements across the prevention and care pathway.

Conclusion

Typhoid fever is a preventable, potentially life-threatening systemic infection transmitted mainly through food and water contaminated by Salmonella Typhi. Persistent fever with constitutional or abdominal symptoms should prompt timely

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clinical assessment, particularly in settings where the disease is common. Blood culture remains the preferred method for confirmation, while serological tests have important limitations. Effective treatment requires appropriate antibiotics guided by resistance patterns and clinical judgement. Long-term control depends on safe water, sanitation, hand and food hygiene, vaccination where recommended, and responsible antibiotic use. Combining these measures offers the strongest route to reducing illness, complications and onward transmission.

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BOTULISM AND FOOD POISONING: HYGIENE AND PREVENTIVE MEASURES

Lizzette Joanna Lawrance , Medical Student, Samarkand state medical university, Uzbekistan

Khan Rania Ali , Medical Student, Samarkand state medical university, Uzbekistan

Sipai Aarzoo Manjurhushen , Medical Student, Samarkand state medical university, Uzbekistan

Dabhade Asmita Ganesh , Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Foodborne illness is a continuing public-health concern, ranging from short-lived gastrointestinal upset to severe, life-threatening disease. Food poisoning may result from bacteria, viruses, parasites or toxins entering food during production, storage, preparation or serving. Botulism is a particularly serious form of foodborne intoxication caused by botulinum neurotoxin, most often associated with improperly processed or stored preserved foods. This narrative review discusses the distinction between common food poisoning and botulism, routes of contamination, clinical warning signs, diagnosis and management principles, and practical hygiene measures for households and food-service settings. Prevention depends on clean hands and equipment, separation of raw and cooked foods, thorough cooking, safe temperatures, safe water and raw ingredients, and validated procedures for preserving food. Because botulism can cause progressive paralysis and respiratory failure, suspected cases require emergency medical assessment and prompt treatment. The article synthesises public-health guidance from the World Health Organization, the US Centers for Disease Control and Prevention and food-safety authorities.

Keywords: Botulism; food poisoning; foodborne disease; food hygiene; food safety; Clostridium botulinum; prevention; sanitation; food storage.

1. Introduction

Food is essential for health, growth and daily life, yet unsafe food can become a vehicle for infectious organisms and toxins. Foodborne disease occurs when a person consumes food or drink contaminated by harmful microorganisms, their toxins or certain chemicals. Many cases cause nausea, vomiting, diarrhoea and abdominal cramps, but severe illness can lead to dehydration, kidney complications, neurological

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injury or death. Children, older adults, pregnant people and people with weakened immune systems may be at greater risk of serious outcomes. Food safety is therefore not only a kitchen responsibility: it involves farmers, manufacturers, transporters, retailers, food handlers, regulators and consumers [1,2].

Botulism deserves particular attention because it is uncommon but potentially fatal. It is caused by botulinum neurotoxin, which interferes with nerve signalling to muscles. Foodborne botulism usually follows consumption of food in which the toxin formed before the food was eaten, often because preservation or storage conditions were unsafe. Unlike many common foodborne infections, botulism may cause blurred or double vision, drooping eyelids, difficulty speaking or swallowing, and progressive muscle weakness. Early recognition can be lifesaving [1,3].

2. Understanding food poisoning and botulism

The term food poisoning is commonly used for a range of illnesses caused by contaminated food. Depending on the cause, symptoms may begin within hours or after several days. Common causes include bacteria such as Salmonella, Campylobacter and certain strains of Escherichia coli, viruses such as norovirus, and toxins produced by microorganisms. Contamination can occur when raw foods contact ready-to-eat foods, hands are not washed, cooking is incomplete, food is left at unsafe temperatures, or water and ingredients are contaminated [2,4].

Foodborne botulism is different because the principal danger is a potent neurotoxin produced by Clostridium botulinum under suitable conditions, including low-oxygen environments. The organism forms heat-resistant spores that can survive ordinary cooking. Improper home canning of low-acid foods, unsafe fermentation and unsuitable storage can create conditions that allow toxin formation. A container may look, smell and taste normal even when the toxin is present, so tasting questionable food is never a safe test [1,3,5].

Feature	Common food poisoning	Foodborne botulism
Main cause	Different pathogens or toxins; varies by food and setting.	Botulinum neurotoxin in contaminated food.
Typical symptoms	Nausea, vomiting, diarrhoea, cramps; fever may occur.	Neurological symptoms such as blurred vision, drooping eyelids, swallowing or speaking

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		difficulty; gastrointestinal symptoms may also occur.
Main danger	Dehydration or complications depending on the organism.	Progressive paralysis, including respiratory muscle weakness.
Urgency	Depends on severity, dehydration, blood in stool and patient risk.	Medical emergency if suspected; do not wait for symptoms to worsen.

3. Transmission and risk factors

Food may become contaminated at any point between production and consumption. Poor hand hygiene can transfer microorganisms from people to food; raw meat, poultry, seafood and eggs can contaminate surfaces or cooked foods; and unsafe water can introduce pathogens into produce, drinks or ice. Inadequate refrigeration allows some organisms to multiply, while insufficient cooking may fail to destroy pathogens. Reusing unwashed cutting boards, storing raw meat above ready-to-eat foods and leaving cooked dishes at room temperature for long periods are preventable hazards [2,4].

Botulism risk is especially associated with incorrectly canned, preserved or fermented foods and some foods held in low-oxygen packaging under unsuitable conditions. Low-acid vegetables, meat, poultry and seafood require particular care during home preservation. Honey should never be given to infants younger than 12 months because it may contain spores linked to infant botulism; infant botulism is not the same as typical foodborne intoxication in adults [3,5].

4. Clinical features and when to seek help

Common food poisoning may cause nausea, vomiting, watery diarrhoea, abdominal pain and sometimes fever. Most mild episodes improve with time, but frequent fluid loss can cause dehydration. Warning signs include very little urination, dry mouth, dizziness, persistent vomiting, blood in stool, severe abdominal pain, high fever or symptoms that are worsening. Medical advice is especially important for infants, older adults, pregnant people and those with significant underlying illness [2,4].

Botulism often begins with fatigue, dizziness or gastrointestinal symptoms, followed by visual changes, drooping eyelids, dry mouth, slurred speech and difficulty swallowing. Weakness may progress downwards through the body and affect breathing. Fever is not a defining feature. Any suspicion of botulism warrants immediate emergency assessment, even if the person is still able to breathe normally.

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Do not wait for laboratory confirmation before seeking help; early antitoxin and supportive care can improve outcomes [1,3].

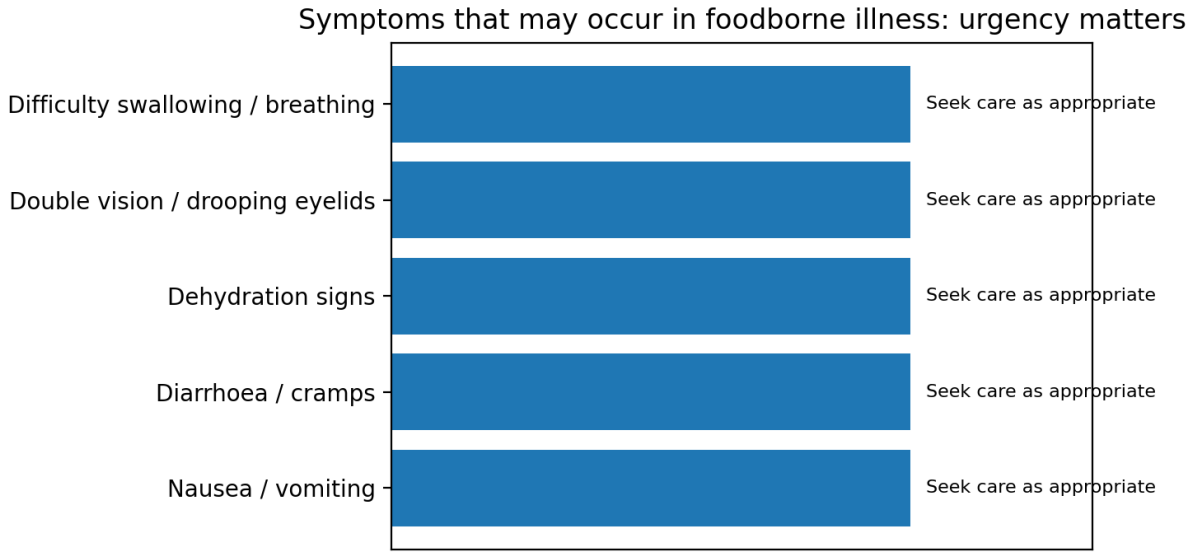


Figure 1. Symptom overview for awareness. This is not a diagnostic scale; neurological symptoms suggestive of botulism require emergency care.

5. Diagnosis and treatment principles

Clinicians assess the timing of symptoms, foods eaten, travel or exposure history, hydration status and signs of complications. Stool, blood or food testing may be selected according to the suspected cause. In a possible outbreak, public-health teams may investigate other people who ate the same food and arrange safe collection of food samples. Patients should not handle or transport suspected contaminated food unless public-health authorities provide instructions.

Treatment depends on the cause and severity. Replacing fluids and electrolytes is central to many cases of gastroenteritis. Antibiotics are useful only for selected bacterial infections and are not appropriate for every episode of diarrhoea; they do not treat toxin-mediated botulism. Suspected botulism requires urgent hospital care, antitoxin as indicated and monitoring or support for breathing. People should not self-prescribe antibiotics or use leftover medication. Severe dehydration, bloody diarrhoea, neurological symptoms or breathing difficulty need prompt professional assessment [1,3,4].

6. Hygiene and preventive measures

Food safety is most effective when it becomes a routine rather than an occasional precaution. The WHO Five Keys to Safer Food provide a practical framework: keep clean; separate raw and cooked; cook thoroughly; keep food at safe temperatures; and

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use safe water and raw materials [2]. Hands should be washed with soap before preparing food, after using the toilet, after handling raw meat or eggs, and after touching rubbish or pets. Worktops, utensils and reusable cloths should be cleaned regularly, and people who are ill with vomiting or diarrhoea should avoid preparing food for others when possible.



Figure 2. Five practical pillars of safer food handling, based on WHO food-safety guidance [2].

Measure	Recommended practice	Why it matters
Personal hygiene	Wash hands with soap; cover cuts; keep nails and food-contact surfaces clean.	Reduces transfer of microorganisms to food.
Prevent cross-contamination	Use separate boards and utensils for raw meat and ready-to-eat food; store raw items below cooked foods.	Stops pathogens moving from raw products to food that will not be cooked again.
Cooking	Cook foods thoroughly and use a food thermometer where appropriate; follow tested recipes for preservation.	Adequate heating destroys many harmful organisms.

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Time and temperature	Refrigerate perishable foods promptly; keep hot foods hot and cold foods cold; follow local temperature guidance.	Limits growth of many disease-causing organisms.
Water and ingredients	Use safe water, pasteurised products and fresh ingredients from reliable sources; wash produce safely.	Prevents contamination before cooking or serving.
Home preservation	Use tested canning instructions and appropriate equipment; pressure-can low-acid foods as directed; never taste suspect jars.	Ordinary boiling may not destroy botulism spores; incorrect canning can allow toxin formation.
Infant protection	Never give honey to a child under 12 months.	Reduces risk of infant botulism.

7. Special precautions for botulism prevention

Home food preservation requires more than clean jars. The method must be suitable for the food's acidity and follow a tested recipe. Low-acid foods such as most vegetables and meats need pressure canning according to authoritative guidance; an improvised water-bath method is not a safe substitute. Do not rely on appearance, smell or taste to judge a preserved food. Discard leaking, bulging, spurting or otherwise suspicious containers without tasting the contents, and follow local public-health advice for safe disposal [3,5].

Refrigeration is important for many foods, but it cannot correct every unsafe preservation method. Keep homemade garlic- or herb-infused oils refrigerated and follow authoritative storage limits; refrigerate opened canned and pickled foods as advised. If botulism is suspected after a meal, seek emergency care and tell clinicians what was eaten and when. Do not serve the same food to anyone else.

8. Discussion

Preventing foodborne disease depends on combining household behaviour with safe systems. A careful cook cannot fully compensate for contaminated water, unsafe processing or a broken cold chain. Similarly, regulations and inspections are less

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effective when food handlers lack training, clean facilities or the equipment needed to follow standards. Schools, universities, healthcare facilities, restaurants and community kitchens can strengthen prevention through staff training, clear cleaning schedules, temperature monitoring, supplier checks and prompt reporting of suspected clusters.

Botulism also shows why food-safety education must explain the reason behind a rule. Because the toxin may not change a food's appearance or smell, sensory checks alone are inadequate. Clear advice about tested canning methods, avoiding improvised preservation and responding quickly to neurological symptoms can prevent severe outcomes. Public-health communication should be accessible, culturally appropriate and consistent with national guidance.

Conclusion

Food poisoning is a broad group of illnesses that can be reduced substantially through good hygiene, safe preparation, proper cooking and careful storage. Botulism is a rarer but especially dangerous foodborne illness because its toxin can cause progressive paralysis and respiratory failure. Prevention requires tested preservation methods, particular caution with low-acid home-canned foods, safe refrigeration, clean hands and equipment, separation of raw and cooked foods, and safe water and ingredients. Honey must not be given to infants under one year. People with possible botulism symptoms need emergency medical assessment, while severe dehydration or other warning signs after food exposure also warrant prompt care. Consistent food-safety practices, public education and effective food-control systems remain essential to protecting individual and community health.

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INSOMNIA AND ITS IMPACT ON PHYSICAL AND MENTAL HEALTH

Mohsina Mistry , Medical Student, Samarkand state medical university, Uzbekistan

Shaik Ariff Hussain , Medical Student, Samarkand state medical university, Uzbekistan

Abdul Azeez , Medical Student, Samarkand state medical university, Uzbekistan

Abu Saharat , Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Insomnia is a common sleep disorder in which a person has difficulty falling asleep, staying asleep, or obtaining restorative sleep despite having adequate opportunity to sleep. It can occur briefly during periods of stress or persist over months, affecting concentration, mood, work, relationships and general wellbeing. This narrative review describes the nature and common causes of insomnia, its effects on physical and mental health, methods of assessment, and evidence-informed approaches to prevention and treatment. Persistent sleep disruption is associated with fatigue, impaired attention, irritability and reduced quality of life; chronic insomnia may coexist with anxiety, depression, pain and cardiometabolic conditions. These relationships can be bidirectional, meaning health problems may worsen sleep while poor sleep may make symptoms harder to manage. Assessment begins with a careful sleep and medical history, often supported by a sleep diary. Cognitive behavioural therapy for insomnia (CBT-I) is recommended as a first-line treatment for chronic insomnia in adults, while regular routines and a suitable sleep environment provide useful support. The review emphasises early help-seeking, avoiding unsupervised use of sleep medicines, and recognising that sleep problems are real health concerns rather than a personal failure.

Keywords: Insomnia; sleep disorder; physical health; mental health; sleep hygiene; cognitive behavioural therapy; CBT-I; fatigue; anxiety; depression.

1. Introduction

Sleep is a basic biological need. During sleep, the brain and body carry out processes involved in learning, memory, emotional regulation, tissue repair and everyday functioning. Yet for many people, bedtime becomes a period of frustration rather than rest. They may lie awake for hours, wake repeatedly, or rise too early and remain tired throughout the day. When these problems occur despite enough time and an appropriate setting for sleep, they may indicate insomnia [1,2].

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Insomnia should not be dismissed as simply having a few bad nights. Short-term sleep difficulty is common during examinations, family changes, illness, grief or work pressure. For some people, however, the difficulty continues and begins to affect health and daily life. Chronic insomnia is generally characterised by sleep difficulty at least three nights per week for three months or longer, with daytime consequences [1]. Understanding its effects matters because sleep and health influence one another: pain, stress and illness can disturb sleep, while ongoing poor sleep can increase distress and make coping with illness more difficult.

2. Types, symptoms and contributing factors

Insomnia may be short-term or chronic. Short-term insomnia can follow a stressful event, travel across time zones, a change in work schedule or an unfamiliar sleeping environment. Chronic insomnia persists over time and may become maintained by a cycle of worry about sleep, irregular sleep schedules, spending long periods awake in bed and trying harder to force sleep. The more a person fears another sleepless night, the more alert and tense they may become at bedtime [1,3].

Common symptoms include difficulty falling asleep, frequent waking, waking too early, feeling unrefreshed, daytime tiredness, irritability and difficulty concentrating. Contributors can include anxiety, depression, chronic pain, menopause, restless legs, thyroid disorders, certain medicines, caffeine, nicotine, alcohol, shift work, noise and bright light. Alcohol may initially make a person feel sleepy but can fragment sleep later in the night. Insomnia may also occur alongside other sleep disorders, such as obstructive sleep apnoea, so a broad assessment is important [2,4].

Factor	How it may affect sleep	Practical consideration
Stress and worry	Increases mental alertness and makes it harder to relax.	Use a wind-down routine; seek support if stress persists.
Caffeine and nicotine	Can delay sleep or increase alertness, especially when used later in the day.	Review timing and amount; individual sensitivity differs.
Alcohol	May bring sleepiness but can lead to lighter, interrupted sleep.	Do not use alcohol as a sleep treatment.
Irregular schedule / shift work	Can disrupt the body's sleep-wake rhythm.	Keep wake time consistent when possible; plan light exposure and rest.

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Pain, mood or medical conditions	Symptoms or treatments may interrupt sleep.	Discuss ongoing symptoms and medicines with a healthcare professional.
Bedroom conditions	Noise, light, heat or an uncomfortable bed may disturb sleep.	Make the room quiet, dark and comfortably cool where possible.

3. Impact on physical health

Insomnia can affect physical wellbeing first through the experience of fatigue. A tired person may feel physically drained, develop headaches, struggle to exercise, or find ordinary tasks more difficult. Attention and reaction time can decline, increasing the risk of mistakes at work, falls and road accidents. People who feel sleepy should not drive or operate machinery when they cannot do so safely [3,5].

Sleep is also connected with the regulation of several body systems. Research links chronic sleep problems with a higher risk of conditions such as high blood pressure, coronary heart disease and diabetes, although the relationship is complex and does not mean that insomnia alone inevitably causes these conditions. Poor sleep can influence stress responses, appetite, activity levels and the management of existing illnesses. In turn, physical illness and pain can make sleep more difficult, creating a cycle that may need attention on both sides [3,5].

Insomnia can also affect how people care for themselves. When energy is low, it may be harder to prepare balanced meals, remain physically active, attend appointments or follow a treatment plan. The impact is not identical for everyone: some people experience marked daytime sleepiness, while others describe exhaustion without easily falling asleep during the day. A clinician should consider the pattern and the individual's health history rather than relying on one symptom alone.

4. Impact on mental and emotional health

Sleep and emotional regulation are closely connected. After a poor night, small problems can feel overwhelming, patience may be reduced and concentration can become less reliable. Repeated sleep loss may affect memory, learning, decision-making and the ability to manage everyday stress. Relationships may also suffer when a person feels irritable, withdrawn or unable to participate fully in family and social activities [2,5].

Insomnia often occurs alongside anxiety and depression. Worry can keep the mind active at night, while repeated sleeplessness can increase negative thinking and reduce

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emotional resilience. The relationship is not one-way: mental health symptoms can cause or worsen insomnia, and persistent insomnia may make emotional difficulties harder to manage. Treating sleep problems can therefore be an important part of broader mental healthcare, but it does not replace appropriate assessment and treatment for a mental health condition.

People with ongoing insomnia sometimes blame themselves or feel embarrassed about needing help. In reality, insomnia is a recognised health problem with psychological, behavioural, environmental and biological contributors. A supportive response is more useful than telling someone simply to relax or try harder to sleep. If sleeplessness is accompanied by severe depression, thoughts of self-harm, or inability to function safely, urgent professional support is important.

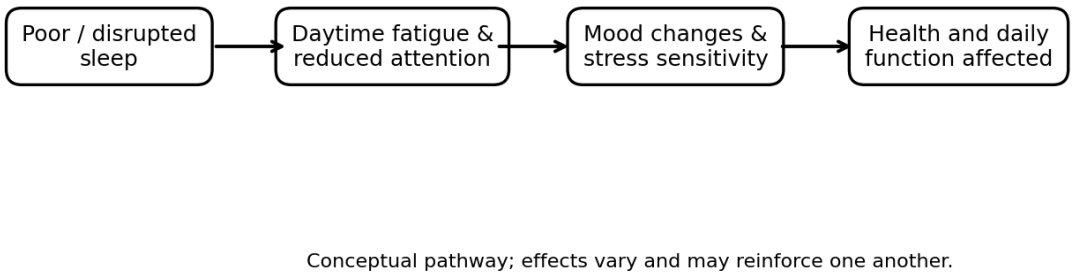


Figure 1. Conceptual pathway showing how poor sleep and daytime difficulties may reinforce one another. It is illustrative, not a quantitative causal model.

5. Assessment and diagnosis

Assessment usually begins with questions about the person's sleep pattern, when the problem started, how often it occurs and how it affects daytime life. Clinicians may ask about work shifts, stress, caffeine and alcohol, medications, pain, mood, snoring, gasping during sleep and restless sensations in the legs. A sleep diary kept for one or two weeks can record bedtime, estimated time to fall asleep, awakenings, final waking time, naps and daytime tiredness [1].

Not every person with insomnia needs a laboratory sleep study. Such testing may be considered when another disorder, such as sleep apnoea or a circadian rhythm problem, is suspected. Blood tests or other examinations may be appropriate if the history

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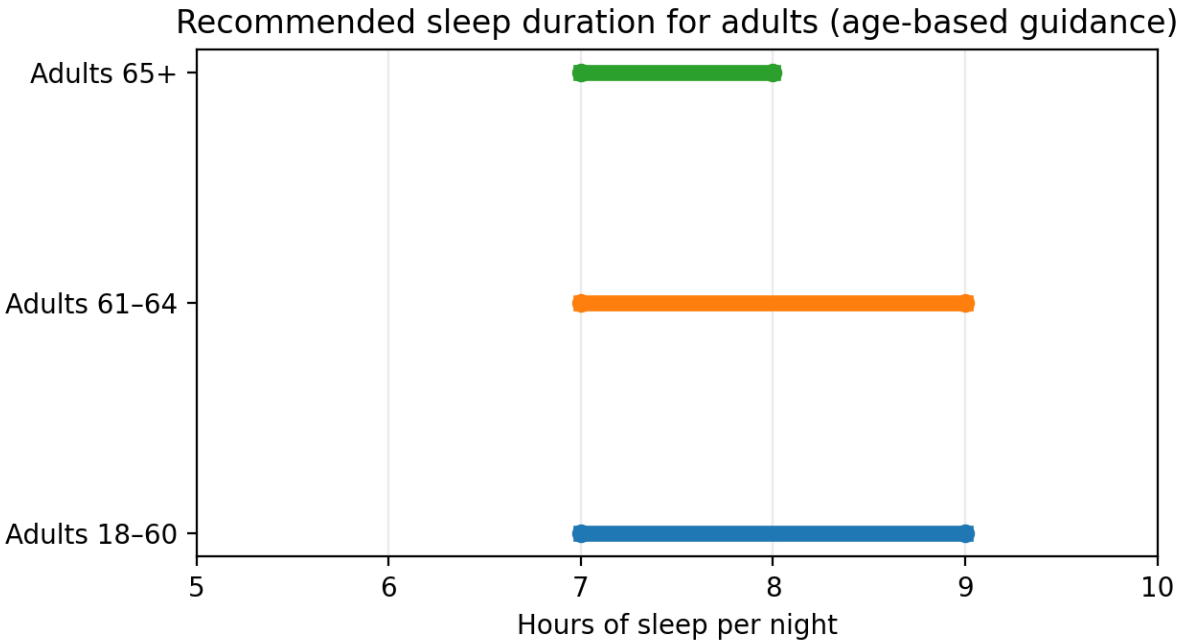
suggests a medical contributor. The aim is to understand the whole picture and identify factors that can be treated, rather than focusing only on the number of hours slept.

6. Prevention and treatment

Healthy sleep habits can support recovery and may help prevent some short-term sleep problems. Useful steps include keeping a reasonably consistent wake-up time, creating a quiet and dark sleeping environment, limiting caffeine later in the day, avoiding nicotine near bedtime, staying physically active during the day and setting aside time to wind down. Bright screens and stimulating work immediately before bed may keep some people alert, so a calmer routine can be helpful. These measures are supportive, but sleep hygiene alone may not be enough for chronic insomnia [3,4].

For chronic insomnia, cognitive behavioural therapy for insomnia (CBT-I) is recommended as a first-line treatment in adults. CBT-I is a structured programme that may include changing unhelpful beliefs about sleep, stimulus control, relaxation methods and carefully planned sleep scheduling. It is more than general advice to practise good sleep hygiene and can be delivered in person or through appropriate digital programmes [6].

Medicines may be considered in selected situations after an individual assessment, but they are not suitable for everyone and can cause side effects or interact with other medicines. Over-the-counter sleep aids and supplements should not automatically be assumed to be safe or effective. People should speak with a healthcare professional before starting or stopping a sleep medicine, particularly if they are pregnant, older, have other medical conditions or take several medicines.



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Figure 2. Adult sleep-duration guidance from the US CDC. Individual sleep needs vary; the ranges are general recommendations, not an insomnia diagnostic test [4].

Approach	Examples	Role and cautions
Sleep routine	Consistent wake time; relaxing pre-sleep routine; comfortable bedroom.	Helpful foundation, but may not resolve chronic insomnia by itself.
Behavioural strategies	Stimulus control and clinician-guided sleep scheduling.	Core elements of CBT-I; individualised guidance is important.
Psychological support	CBT-I; treatment for anxiety, depression or stress when indicated.	Addresses thoughts, behaviours and coexisting concerns.
Medical review	Review medicines, pain, breathing symptoms and other conditions.	Helps identify treatable contributors or another sleep disorder.
Medication when appropriate	A clinician may discuss a short-term or other tailored option.	Consider benefits, risks, interactions and duration; avoid self-medication.

7. Discussion

Insomnia is best understood as a health issue rather than a simple failure of discipline. Advice about routines and screen use can be useful, but it should not imply that every person can solve persistent insomnia alone. Work demands, caregiving, housing conditions, shift work, pain and emotional strain can make consistent sleep difficult. Prevention and treatment are more effective when advice is realistic and adapted to the person's circumstances.

Healthcare providers can improve outcomes by asking about sleep as part of routine care, screening for contributing conditions and offering evidence-based treatment. Public-health education can also help people recognise when sleep problems need assessment. Since sleep affects attention, emotional wellbeing and physical functioning, improving sleep may benefit not only individuals but also families, workplaces and communities.

Conclusion

Insomnia is a common and sometimes persistent disorder that can influence both physical and mental health. Its effects may include fatigue, impaired attention, mood changes, reduced daily functioning and associations with longer-term health

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conditions. Because insomnia can coexist with anxiety, depression, pain and other sleep disorders, assessment should consider the person's whole health and circumstances. Consistent sleep habits and a supportive sleep environment are valuable, while cognitive behavioural therapy for insomnia is a leading evidence-based treatment for chronic insomnia in adults. Medication may be appropriate in selected cases under professional guidance, but unsupervised use is not recommended. Most importantly, persistent sleep difficulty deserves understanding and appropriate care; it should not be dismissed as a personal weakness.

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ATHEROSCLEROSIS: FROM FATTY STREAK TO PLAQUE RUPTURE

Aryan kumar , Medical Student, Samarkand state medical university, Uzbekistan

Ayush raj , Medical Student, Samarkand state medical university, Uzbekistan

Vaishnavi banavath , Medical Student, Samarkand state medical university, Uzbekistan

Dikshant bhanvra, Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Atherosclerosis is a chronic inflammatory disease of medium and large arteries in which lipids, immune cells and connective tissue accumulate within the arterial wall. Its development is gradual, beginning with endothelial dysfunction and retention of cholesterol-containing lipoproteins, followed by the formation of fatty streaks and more complex fibrous plaques. Some plaques remain relatively stable, while others develop a large lipid-rich necrotic core and a weakened fibrous cap. Plaque rupture or surface erosion may expose thrombogenic material to circulating blood, promoting clot formation and potentially causing myocardial infarction or ischemic stroke. This narrative review outlines the biological stages of atherosclerosis, key risk factors, mechanisms of plaque instability, clinical consequences and preventive approaches. It highlights the interaction between lipid metabolism, inflammation, vascular biology and lifestyle-related risks. Early risk assessment, smoking cessation, healthy dietary patterns, physical activity, control of blood pressure and diabetes, and lipid-lowering treatment when indicated can reduce cardiovascular risk. Understanding the pathway from fatty streak to thrombosis helps explain why atherosclerosis can remain silent for years before presenting as an acute medical emergency.

Keywords: Atherosclerosis; fatty streak; endothelial dysfunction; LDL cholesterol; foam cells; inflammation; fibrous cap; plaque rupture; thrombosis; cardiovascular disease.

1. Introduction

Atherosclerosis is one of the major pathological processes underlying coronary artery disease, ischemic stroke and peripheral arterial disease. It is often described as a gradual build-up of fatty material in arteries, but this description captures only part of the story. An atherosclerotic plaque is a living, changing structure containing cholesterol, inflammatory cells, smooth muscle cells, connective tissue and, in

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advanced lesions, areas of cell debris and calcification [1–3]. The condition may develop over decades without obvious symptoms. Problems arise when a plaque significantly restricts blood flow or when its surface is disrupted and a blood clot forms.

The phrase "from fatty streak to plaque rupture" describes a useful sequence, although real disease is not always linear. Some early lesions do not progress substantially, and advanced plaques differ in their composition and stability. The risk of a clinical event depends not only on how much a plaque narrows an artery, but also on its biological features and the response of the blood-clotting system. This article reviews the major stages of atherosclerosis and explains how prevention can interrupt the process.

2. Initiation: endothelial dysfunction and lipid retention

The inner surface of an artery is lined by endothelial cells. Under healthy conditions, these cells help regulate vessel tone, blood clotting and the movement of immune cells. At arterial bends and branch points, disturbed blood flow can make the endothelium more vulnerable to dysfunction. High levels of low-density lipoprotein (LDL) cholesterol, smoking, hypertension, diabetes and other stresses can further impair its protective functions [2,3].

When apoB-containing lipoproteins such as LDL enter and become retained in the inner arterial layer, they may undergo chemical modification. The altered particles activate local inflammatory signalling. Endothelial cells express adhesion molecules and chemical signals that attract circulating monocytes. Once these immune cells enter the vessel wall, they can develop into macrophages and take up modified lipids. Lipid-loaded macrophages are called foam cells, and their accumulation is a defining feature of early atherosclerotic lesions [2,4].

3. Fatty streak formation

A fatty streak is an early lesion formed largely by collections of foam cells within the intima, the innermost layer of the artery. It may appear as a yellowish area when examined directly. Fatty streaks can occur from a young age and do not necessarily mean that a person will develop a dangerous plaque at that exact site. However, they show that lipid retention and an inflammatory response have begun in the arterial wall [3,4].

At this stage, the lesion is often relatively flat and may not significantly obstruct blood flow. The process can nevertheless continue if exposure to atherogenic lipoproteins and other risk factors persists. Some lesions may remain limited, while others develop extracellular lipid pools and recruit smooth muscle cells. This variation is important:

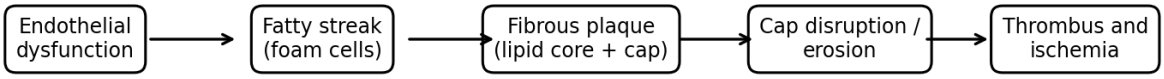
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atherosclerosis is a dynamic process shaped by inherited susceptibility, lifetime exposures and local conditions within the vessel.

4. From fatty streak to fibrous plaque

As a lesion advances, smooth muscle cells migrate from the middle layer of the artery into the intima. They proliferate and produce extracellular matrix, including collagen, which contributes to a fibrous cap over the lipid-rich region. Meanwhile, ongoing inflammation and cell death can create a necrotic core containing cholesterol, cellular debris and other material. Advanced plaques may also contain calcium deposits, small blood vessels and evidence of previous bleeding [2–4].

The fibrous cap matters because it separates the thrombogenic contents of the plaque from circulating blood. A thick, collagen-rich cap with sufficient smooth muscle cells is generally more stable. A plaque with a large necrotic core, intense inflammation and a thin cap may be more vulnerable to disruption. Still, plaque behaviour is complex; not every plaque with a high-risk feature will rupture, and some acute coronary events occur through superficial erosion rather than classic cap rupture.



Simplified pathway; progression is variable and not every plaque ruptures.

Figure 1. Simplified progression from endothelial dysfunction to thrombosis. The stages are conceptual and do not imply that every lesion follows the same course.

Stage	Main biological changes	Possible significance
Endothelial dysfunction	Reduced protective signalling and increased permeability/adhesion signalling.	Creates conditions that favour lipoprotein retention and inflammation.

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Fatty streak	Macrophages take up modified lipids and become foam cells.	Early lesion; may remain limited or progress.
Fibroatheroma	Lipid-rich necrotic core develops beneath a fibrous cap.	May narrow the artery and can develop features of instability.
Plaque rupture or erosion	Plaque surface barrier is disrupted or endothelial lining is lost.	Blood contacts thrombogenic material; clot formation may begin.
Thrombosis	Platelets and clotting proteins form a thrombus at the lesion.	Can suddenly reduce or block blood flow, causing ischemia.

5. Plaque rupture, thrombosis and clinical events

Plaque rupture occurs when the fibrous cap tears, allowing blood to contact highly thrombogenic material within the plaque. Platelets adhere and activate, and the coagulation system generates fibrin, producing a thrombus. If the clot rapidly blocks a coronary artery, the result may be an acute myocardial infarction. In a cerebral artery or an artery supplying the brain, a thrombotic event can contribute to ischemic stroke. Plaque-related disease in leg arteries can cause pain during walking, poor wound healing or, in severe cases, tissue loss [1,2].

Plaque erosion is another route to thrombosis. In erosion, the endothelial surface is lost or damaged without the same type of fibrous-cap tear. Both rupture and erosion can produce acute events, so plaque instability should not be reduced to one mechanism alone [5]. A plaque may also gradually narrow an artery and limit blood flow during increased demand, causing exertional chest discomfort or leg pain even without sudden rupture.

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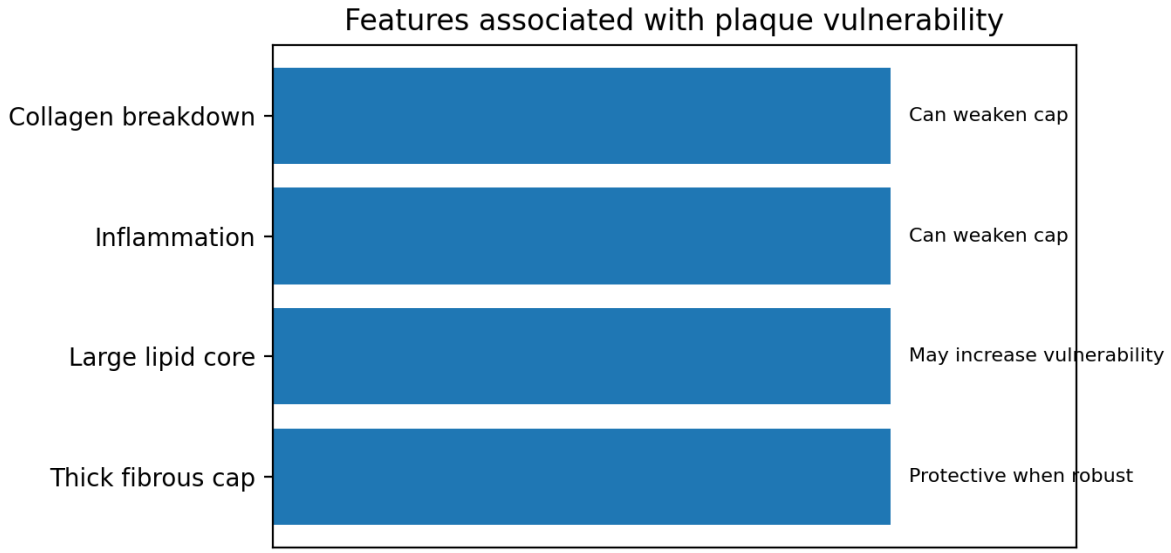


Figure 2. Qualitative illustration of factors relevant to plaque stability. This is explanatory, not a measured risk score.

6. Risk factors

Atherosclerosis develops through interactions among modifiable and non-modifiable risk factors. Age and family history influence baseline risk, while high LDL cholesterol, high blood pressure, diabetes, tobacco exposure, physical inactivity and unhealthy dietary patterns can increase risk. Chronic kidney disease and some inflammatory conditions are also associated with cardiovascular risk. Risk factors often cluster: for example, insulin resistance, elevated blood pressure and abnormal blood lipids may occur together and amplify overall risk [1,6].

LDL cholesterol is particularly important because apoB-containing lipoproteins can enter and become retained in the arterial wall. Lowering LDL exposure over time reduces the opportunity for plaque development and progression. Smoking promotes endothelial dysfunction and inflammation, while high blood pressure increases mechanical stress on arteries. Diabetes can affect lipoproteins, inflammation and vascular function. The contribution of each factor varies between individuals, so prevention is most effective when tailored to overall cardiovascular risk rather than a single measurement.

Risk factor	**Relationship to atherosclerosis**	**Prevention or management approach**
High LDL cholesterol	Increases the pool of atherogenic lipoproteins	Heart-healthy diet; lipid testing; medication when indicated.

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	that may be retained in the arterial wall.	
Tobacco use	Damages vascular function and promotes inflammatory and thrombotic effects.	Avoid smoking and second-hand smoke; seek cessation support.
Hypertension	Adds vascular stress and increases cardiovascular event risk.	Measure blood pressure and follow a clinician-guided plan.
Diabetes	Contributes to vascular injury and often coexists with other risk factors.	Glucose management and comprehensive risk reduction.
Physical inactivity and diet quality	May influence weight, blood pressure, lipid levels and metabolic health.	Regular activity and a balanced dietary pattern.
Age and family history	Non-modifiable factors that affect baseline risk.	Use them to guide earlier assessment and prevention.

7. Prevention and management

Prevention begins before symptoms appear. A dietary pattern rich in vegetables, fruit, legumes, whole grains, nuts and suitable sources of unsaturated fats can support cardiovascular health. Limiting trans fats, excess saturated fat, highly processed foods and excess sodium can help improve risk factors. Regular physical activity, maintaining a healthy weight, sufficient sleep and avoiding tobacco also contribute to risk reduction. These measures are valuable even when medication is required; they complement rather than replace treatment [1,6].

Clinical prevention may include measurement of blood pressure, lipid profile and blood glucose, together with assessment of family history and other conditions. Statins and other lipid-lowering medicines may be recommended according to the person's cardiovascular risk and clinical history. Antiplatelet medicines are appropriate for some patients but are not recommended for everyone because they can cause bleeding. Treatment decisions should be made with a qualified healthcare professional, who can weigh expected benefit against risks and review progress over time.

Urgent symptoms should never be ignored. New chest pressure, breathlessness, cold sweating, sudden weakness or numbness on one side, facial drooping, difficulty speaking or sudden loss of vision may indicate a heart attack or stroke. Emergency

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medical services should be contacted immediately. Atherosclerosis can be silent for years, which is why prevention and early management remain important even when a person feels well.

8. Discussion

The journey from fatty streak to plaque rupture demonstrates that atherosclerosis is both a lipid disorder and a chronic inflammatory disease of the arterial wall. Lipid retention initiates the process, immune activity sustains it, and the balance between tissue repair and breakdown helps determine whether a plaque remains stable. A large plaque is not automatically the most dangerous; composition, cap integrity, inflammation and the local clotting response also matter [2,5].

Public-health strategies should therefore focus on reducing risk across the lifespan. Affordable access to healthy food, opportunities for physical activity, tobacco control, routine screening and effective treatment of hypertension, diabetes and high cholesterol can reduce the burden of cardiovascular disease. Explaining the underlying biology helps people understand why prevention should start early and continue consistently, rather than waiting for symptoms.

Conclusion

Atherosclerosis begins with changes in the arterial lining and retention of cholesterol-containing lipoproteins. The resulting inflammatory response creates fatty streaks, which may develop into complex plaques containing a lipid-rich necrotic core and a fibrous cap. When a plaque ruptures or undergoes erosion, a blood clot may form and abruptly restrict blood flow, leading to serious outcomes such as myocardial infarction or ischemic stroke. Progression is variable, and not every fatty streak becomes a dangerous lesion. Reducing LDL cholesterol exposure, avoiding tobacco, controlling blood pressure and diabetes, maintaining physical activity and following a balanced diet can lower cardiovascular risk. Timely assessment and evidence-based treatment are essential for people at increased risk. Understanding the process from early lesion to thrombosis reinforces the importance of prevention long before symptoms develop.

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SEX-BASED PHARMACOLOGY: Understanding Differences in Drug Response for Safer, More Individualized Treatment

Simran Kaur , Medical Student, Samarkand state medical university, Uzbekistan

Atul narwal , Medical Student, Samarkand state medical university, Uzbekistan

Hitesh sandhu , Medical Student, Samarkand state medical university, Uzbekistan

Anshul bhardwaj , Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Sex-based pharmacology examines how biological sex can influence the way medicines are absorbed, distributed, metabolized, eliminated, and experienced by the body. Differences in body composition, hormones, organ function, gene expression, transport proteins, and immune responses may alter drug exposure, therapeutic benefit, or the risk of adverse effects. These differences are not universal: the direction and clinical importance depend on the medicine, dose, age, health status, and individual patient. Gender-related factors—including access to care, communication, social roles, and patterns of medicine use—can also shape real-world outcomes, but they should not be confused with biological mechanisms. This narrative review summarizes key concepts in sex-based pharmacology, explains the relevance of pharmacokinetics and pharmacodynamics, and discusses examples such as zolpidem, cardiovascular medicines, analgesics, and medicines used during pregnancy. It also highlights limitations in the evidence, including historical underrepresentation of women and insufficient sex-stratified reporting. The central clinical message is that sex should inform research and assessment, but should not replace individualized prescribing. Clinicians should follow current product information, consider organ function and interacting medicines, monitor response, and discuss uncertainty with patients. A more inclusive evidence base can improve both medicine safety and effectiveness.

Keywords: sex-based pharmacology; sex differences; gender; pharmacokinetics; pharmacodynamics; adverse drug reactions; precision medicine; individualized treatment.

1. Introduction

Medicines are often introduced with a standard dose, yet patients do not respond identically. One person may obtain the intended benefit, while another experiences little improvement or develops an adverse reaction. Age, body size, genetics, kidney

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and liver function, other medicines, diet, and adherence are familiar sources of variability. Biological sex is another factor that can matter, although it is frequently oversimplified or overlooked. Research reviews describe sex-related differences in drug exposure and response across several therapeutic areas, while also emphasizing that evidence is incomplete for many individual medicines [1–3].

In this article, "sex" refers mainly to biological characteristics, including chromosomes, reproductive physiology, hormones, body composition, and related organ-level differences. "Gender" refers to social and behavioral factors that can influence healthcare experiences, such as expectations about symptoms, medicine use, caregiving responsibilities, and access to treatment. These concepts interact but are not interchangeable. Neither category can be inferred perfectly from appearance or treated as a complete description of an individual. The purpose of sex-based pharmacology is not to assume that every woman or every man needs a different dose; it is to recognize relevant variation and use evidence to make treatment safer and more effective.

2. Biological foundations of differences in drug response

Sex-linked differences arise through multiple pathways. Sex chromosomes can affect gene expression, while hormones such as estrogen, progesterone, and testosterone influence tissues and metabolic systems. Body composition also matters: on average, women and men may differ in total body water, fat proportion, plasma volume, and muscle mass. These are population-level tendencies, not rules for every individual. The effect on a medicine depends on its chemical properties and the clinical situation. For example, a drug that distributes mainly in body water may behave differently from a highly lipid-soluble drug when body composition differs.

Liver enzymes and drug transporters contribute to metabolism and movement of medicines through the body. Some enzyme activities differ on average by sex, but the pattern is medicine-specific and may be modified by age, pregnancy, menopause, genetics, smoking, diet, and other medicines. Kidney filtration and tubular transport, plasma protein binding, gastrointestinal transit, and immune function may also influence exposure or response. Hormonal changes across the menstrual cycle, pregnancy, postpartum period, and menopause can alter physiology; however, clinically meaningful effects should be established for the particular drug rather than presumed.

3. Pharmacokinetics: what the body does to a drug

Pharmacokinetics is commonly summarized as ADME: absorption, distribution, metabolism, and excretion. A sex-related difference at any stage may change the

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concentration of a medicine over time. The result may be a stronger effect, a longer-lasting effect, a reduced effect, or no clinically important difference.

ADME stage	Potential contributors	Clinical meaning
Absorption	Gastrointestinal motility, pH, transporters, formulation and route	May alter how quickly or how much drug reaches circulation.
Distribution	Body water, fat mass, blood volume, protein binding	May influence apparent distribution volume and concentration.
Metabolism	Liver enzyme activity, hormones, genetics, interacting drugs	May change the rate at which a medicine is converted or inactivated.
Excretion	Kidney function, renal blood flow, transport processes	May change drug or metabolite clearance and duration of exposure.

These mechanisms should not be interpreted as a checklist that automatically leads to different doses. A pharmacokinetic difference matters clinically when it is large enough to alter benefit or harm, and when reliable evidence supports a change in prescribing. For medicines with a narrow therapeutic index, even a modest change in exposure may require closer monitoring. For other medicines, body weight, kidney function, age, or interacting drugs may be more informative than sex alone.

4. Pharmacodynamics: what the drug does to the body

Pharmacodynamics describes the relationship between drug concentration and its effects, including receptor binding, signal pathways, and changes in organ function. Two patients with similar blood concentrations can respond differently if receptor sensitivity, disease mechanisms, or physiological reserves differ. Hormones and sex-linked gene expression may influence these pathways, but the clinical evidence varies substantially between drug classes. Differences in pain perception, immune activity, cardiovascular physiology, and central nervous system function may also contribute to observed response patterns.

Importantly, pharmacodynamic differences cannot always be separated neatly from pharmacokinetic ones. A stronger effect may reflect higher drug exposure, greater sensitivity at the target, or both. Good studies therefore measure outcomes carefully

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and, when possible, examine exposure as well as response. Findings should be replicated and assessed for clinical importance before being translated into routine dosing recommendations.

5. Clinical examples and lessons

5.1 Zolpidem and next-morning impairment

Zolpidem is a well-known example of a sex-related prescribing change. The US Food and Drug Administration required lower recommended doses for women using certain zolpidem products because the medicine may be eliminated more slowly, leaving blood concentrations high enough the next morning to impair alertness and activities such as driving. A lower dose should also be considered for men in relevant circumstances, and patients should follow the specific product label and prescriber's advice [4]. This example demonstrates how pharmacokinetic evidence can lead to a concrete safety action.

5.2 Cardiovascular medicines

Cardiovascular diseases may present differently across populations, and sex can influence body size, hormonal environment, disease mechanisms, drug exposure, and adverse-effect patterns. However, the evidence is not uniform across all beta-blockers, statins, antihypertensives, or antithrombotic medicines. Prescribers should use established indications, measured blood pressure and heart rate, kidney and liver function, comorbidities, and current guidelines rather than applying a broad "female dose" or "male dose." Where evidence supports monitoring for a particular adverse effect, it should be incorporated into follow-up.

5.3 Analgesics and central nervous system medicines

Studies of analgesics, sedatives, and other centrally acting medicines have reported differences in efficacy, metabolism, and adverse reactions. Yet pain is affected by many factors, including the cause of pain, prior treatment, sleep, mental health, expectations, and social context. A sex-based average cannot predict an individual's pain experience. Clinicians should assess the patient's response and adverse effects directly, avoid stereotyping symptoms, and adjust treatment according to evidence and the patient's clinical needs [2,3].

5.4 Pregnancy, postpartum changes, and menopause

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Pregnancy can alter plasma volume, renal filtration, enzyme activity, gastrointestinal function, and protein binding. These changes may affect the exposure of some medicines and can change across pregnancy. At the same time, a medicine may cross the placenta or affect fetal development, so treatment decisions must consider both maternal benefit and fetal risk. Postpartum physiology does not immediately return to a single baseline, and lactation introduces additional considerations. Menopause and hormone therapy may also affect drug metabolism or symptoms that influence treatment. Decisions should be medicine-specific and guided by current prescribing information and specialist advice when needed.

6. Figure: a practical framework for understanding drug response

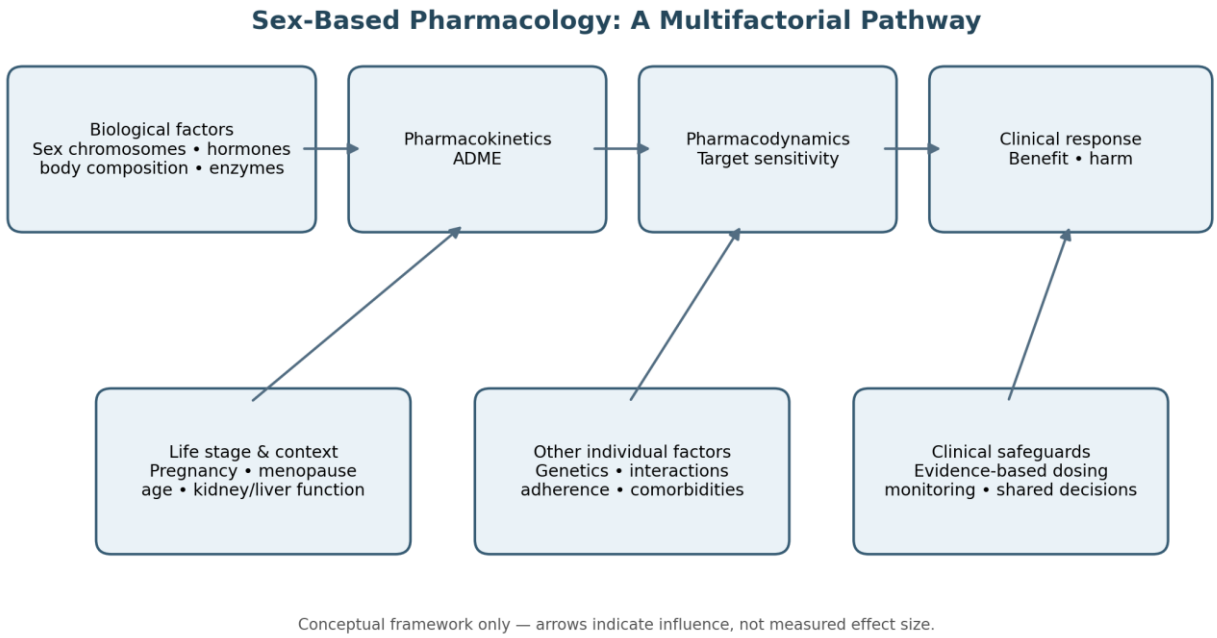


Figure 1. Conceptual framework showing how biological, life-stage, and individual factors can influence pharmacokinetics, pharmacodynamics, and clinical outcomes. The figure is illustrative and does not represent measured effect sizes.

7. Summary table: applying the evidence in practice

Clinical question	Useful action	Avoid
Could sex affect this medicine?	Check current product information and evidence for the specific drug.	Assuming all medicines work differently by sex.

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Could exposure be altered?	Review weight, kidney/liver function, age, pregnancy status where relevant, and interacting medicines.	Using sex as a substitute for clinical assessment.
Is the medicine causing harm or not working?	Ask about response, adverse effects, adherence, and timing; monitor when indicated.	Dismissing symptoms or relying on population averages.
Is evidence incomplete?	Explain uncertainty, document the rationale, and follow the patient.	Treating absence of evidence as proof of no difference.

8. Limitations and research priorities

Sex-based pharmacology is an evolving field. Many studies have not been designed or powered to detect differences between groups, and results may be reported without sex-stratified analyses. Women, pregnant people, older adults, and people with multiple health conditions have not always been adequately represented in drug-development research. Even when a difference is statistically significant, it may be too small to change treatment. Conversely, a meaningful difference can be missed when sample sizes are limited. Differences between studies may reflect population characteristics, dose, disease severity, study design, or measurement methods rather than sex alone.

Future research should recruit participants of different sexes across the life course, prespecify sex-stratified analyses when scientifically justified, and report both absolute and relative effects. Researchers should distinguish biological sex from gender-related influences and include diverse populations rather than treating women and men as internally uniform groups. Post-marketing surveillance and real-world data can help identify adverse effects that were too rare to appear in clinical trials. Ultimately, research should translate into clear clinical guidance rather than adding complexity without a demonstrated benefit.

9. Conclusion

Sex-based pharmacology helps explain why the same medicine and dose may not produce identical outcomes in every patient. Biological sex can influence drug absorption, distribution, metabolism, elimination, and response at the target site. Hormonal life stages and gender-related experiences may add further complexity. The

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strongest practical lesson is not to prescribe according to sex alone, but to recognize where evidence identifies a meaningful difference and combine it with the patient's age, body size, organ function, genetics when relevant, other medicines, and observed response. More inclusive research and careful reporting are essential to close evidence gaps. When clinicians use current guidance, monitor benefits and harms, and make decisions with patients, sex-aware pharmacology can support safer and more equitable care.

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ARTIFICIAL INTELLIGENCE IN DRUG DISCOVERY AND DEVELOPMENT

Rishabh Jasoria , Medical Student , Samarkand state medical university, Uzbekistan

Adil Patel , Medical Student , Samarkand state medical university, Uzbekistan

Patel Mo.Uvesh , Medical Student , Samarkand state medical university, Uzbekistan

Aysha Sama , Medical Student , Samarkand state medical university, Uzbekistan

Bringing a new medicine to patients is slow, expensive and uncertain, and about nine in ten candidates that enter human trials never reach approval. This narrative review looks at how artificial intelligence (AI) is being used to attack that problem in three places: choosing drug targets, predicting toxicity before a compound is ever made, and shortening the time between an idea and a clinical trial. Using published studies and well-documented case examples, including AlphaFold, the antibiotic halicin and the AI-derived drug rentosertib, we describe what these tools do well and where they fall short. Early evidence is encouraging but uneven: molecules discovered with AI appear to pass Phase I safety trials far more often than the historical average, yet their Phase II success rate looks much like that of conventional drugs. AI is therefore speeding up and de-risking the early stages, while proving that a drug works in patients remains the hard part. Data quality, explainability and independent validation will decide how far the progress goes.

Keywords: *artificial intelligence; drug discovery; target identification; toxicity prediction; machine learning; generative chemistry; clinical trials*

1. Introduction

Ask anyone who has worked in pharmaceutical research how a medicine is born and you will hear a story about patience. A biologist spots a protein that seems to drive a disease. Chemists spend years shaping a molecule that can block it. Animal studies follow, then three phases of human trials. The whole journey typically takes 10 to 15 years and costs somewhere around a billion dollars or more, and roughly nine out of ten candidates that reach human testing are never approved (Paul et al., 2010; Sun et al., 2022).

Why do so many fail? Sun and colleagues (2022) analysed clinical trial data from 2010 to 2017 and attributed 40–50% of failures to a lack of clinical efficacy and about 30%

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to unmanageable toxicity, with poor drug-like properties and weak commercial planning making up most of the rest (Figure 1). Those two large slices point to exactly the questions AI is now being asked to help with. Are we aiming at the right biological target? And will the molecule be safe enough to give to people?

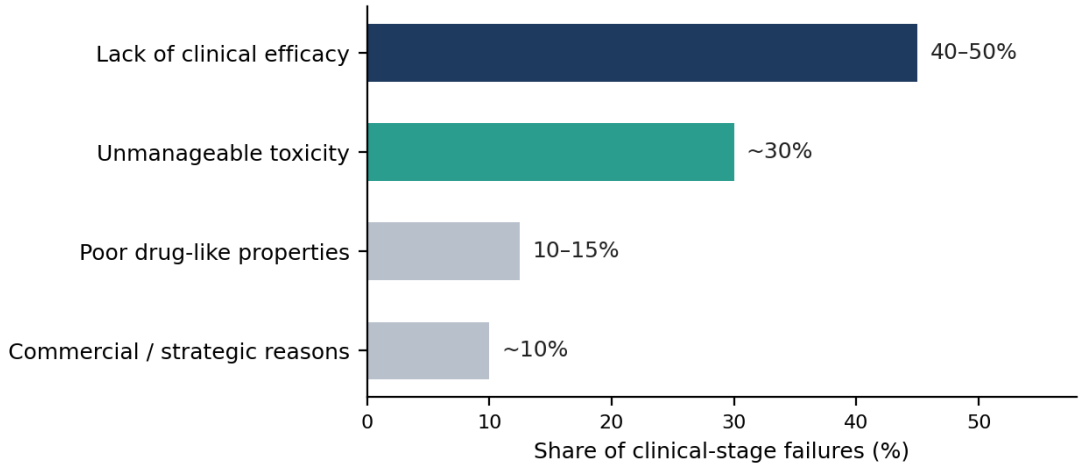


Figure 1. Main reasons for failure in clinical drug development. Bars show the midpoint of each reported range. Data from Sun et al. (2022).

This article follows those questions through the drug pipeline. It is a narrative review rather than a systematic one: sources were chosen for their influence and relevance, not through an exhaustive database search, and the aim is to give a clear, honest picture rather than a count of every publication.

2. Finding the Right Target

A drug target is usually a protein whose activity contributes to a disease. Picking the wrong one is the costliest mistake in the field, because it can take years and many millions before a clinical trial reveals it. Targets used to come from decades of bench biology, human genetics and a certain amount of luck. What has changed is the sheer volume of data: genome sequences, gene-expression profiles, proteomics, patient records and millions of research papers. No team can read all of it, but machine-learning systems can at least rank it.

Several approaches are common. Knowledge graphs link genes, proteins, diseases, drugs and publications into one network, so an algorithm can notice a connection nobody has stated outright. Models trained on omics data compare diseased and healthy tissue and flag genes whose changes look causal rather than incidental. Language models scan the literature at a scale no human reader could manage. Each method

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produces a ranked list, and a scientist still has to decide which entries deserve a laboratory test (Vamathevan et al., 2019).

Structure matters just as much as selection. Once a target is chosen, chemists want to know its three-dimensional shape, because that shape decides which molecules can fit. AlphaFold 2 predicted protein structures with accuracy often close to that of laboratory experiments (Jumper et al., 2021), and its successor, AlphaFold 3, extends the idea to complexes of proteins with DNA, RNA and small molecules (Abramson et al., 2024). For many targets no experimental structure exists, so these predicted models give design teams somewhere to start.

The best-known example of an AI-nominated target is TNIK in idiopathic pulmonary fibrosis (IPF), a progressive scarring disease of the lungs. Insilico Medicine's platform ranked TNIK as a promising anti-fibrotic target and then generated a molecule, rentosertib, to inhibit it (Ren et al., 2025). In a randomised Phase IIa trial of 71 patients, those taking 60 mg daily gained an average of 98.4 mL in forced vital capacity over 12 weeks, while the placebo group lost 20.3 mL (Xu et al., 2025). That is encouraging, but the trial was small and short, more patients on the highest dose stopped treatment early than on placebo, and the authors themselves call for larger studies.

Table 1. Where AI is used along the drug-development pipeline

Stage	Typical AI approach	What it adds
Target identification	Knowledge graphs, omics mining, genetics-based ranking	Prioritises disease-linked proteins from very large datasets
Structure and binding	AlphaFold-type structure prediction; docking with learned scoring	Provides 3D models where no experimental structure exists
Hit finding and design	Virtual screening, graph neural networks, generative models	Explores far more chemical space than a physical library can
Safety and ADMET	QSAR and deep learning on assay data	Flags risky compounds before they are synthesised

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Stage	Typical AI approach	What it adds
Clinical development	Patient stratification, site selection, health-record mining	Aims to reduce delays and mismatched patient groups

3. From Target to Molecule

With a target in hand, the job is to find or build a molecule that fits it. The space of possible drug-like molecules is astronomically large, far beyond anything that could be made and tested, so physical screening only samples a tiny corner of it. AI helps in two ways: it can score existing libraries on a computer, and it can invent new structures.

The halicin story shows the first route. In 2020 a team at MIT trained a graph neural network on a few thousand molecules labelled by whether they stopped E. coli from growing, then used it to screen a library of repurposed compounds. The model picked out halicin, a compound originally studied for diabetes, whose structure looks nothing like known antibiotics. It went on to show activity against a range of resistant bacteria in laboratory and mouse experiments (Stokes et al., 2020).

Generative models go a step further. In 2019, Zhavoronkov and colleagues used a generative approach to design inhibitors of DDR1, a kinase linked to fibrosis, in 21 days and then tested a lead compound in mice (Zhavoronkov et al., 2019). In the rentosertib programme, the same company reported getting from target discovery to a preclinical candidate in about 18 months (Ren et al., 2025), well under the multi-year timelines usually quoted for this stage. Table 2 collects a few of these milestones.

Table 2. Selected milestones in AI-supported drug discovery

Year	Milestone	Why it matters
2019	Generative design of DDR1 kinase inhibitors in 21 days (Zhavoronkov et al.)	Showed the design cycle could shrink from years to weeks
2020	Deep-learning discovery of the antibiotic halicin (Stokes et al.)	Found a structurally novel antibacterial among existing compounds

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Year	Milestone	Why it matters
2021	AlphaFold 2 predicts protein structures at near-experimental accuracy (Jumper et al.)	Opened structure-based design to many previously unsolved targets
2024	Analysis of AI-discovered molecules in trials (Jayatunga et al.)	First systematic look at how such molecules perform in people
2025	Phase IIa results for rentosertib published (Xu et al.)	Early clinical signal for a drug whose target and structure were AI-derived

4. Predicting Toxicity Before It Happens

Toxicity is the second great killer of drug candidates, and it is also where computational methods have the longest track record, because the question can be framed as a plain prediction problem: given a structure, will it harm a liver, a heart or DNA? Early quantitative structure–activity relationship (QSAR) models relied on chemical descriptors chosen by hand. Modern deep-learning models learn their own features directly from the molecular graph. A landmark was DeepTox, which won the Tox21 Data Challenge by learning from roughly 12,000 compounds tested across 12 assays for nuclear-receptor and stress-response signalling (Mayr et al., 2016).

In practice, teams build panels of models, each aimed at one endpoint (Table 3). If a compound is predicted to block the hERG channel, which is a known cardiac risk, chemists can redesign it before anyone makes it. That is where the real saving lies. Failing a computer screen costs almost nothing, while failing in a Phase II trial costs years and tens of millions.

Table 3. Common toxicity endpoints modelled with machine learning

Endpoint	Why it matters	Typical data and models
Cardiotoxicity (hERG block)	Can disturb heart rhythm; a classic reason for late-stage withdrawal	Public hERG inhibition data; random forests, graph neural networks

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Endpoint	Why it matters	Typical data and models
Liver injury (hepatotoxicity)	A leading safety reason for stopped trials and withdrawn drugs	Curated liver-injury labels; datasets are small and noisy
Mutagenicity	Flags compounds that may damage DNA	Large Ames-test datasets; structural alerts combined with ML
Nuclear-receptor and stress-response assays	Signals endocrine disruption and cellular stress	Tox21 library; multitask deep learning such as DeepTox

There are caveats. Toxicity data are patchy and biased towards compounds that were tested because they already looked promising. Many harms are idiosyncratic, striking a small number of people because of their genes or other medicines, and a model trained on cell assays will rarely capture that. Differences between animals and humans remain, too. Finally, deep models are often black boxes, yet regulators and chemists want to know which part of a molecule triggered an alert, so explainability tools matter nearly as much as accuracy.

5. Speeding Up Development

Speed is the headline promise. Faster target ranking, virtual screening and in-silico safety filters shorten the design–make–test cycle, and the DDR1 and TNIK examples suggest that early stages can take weeks to months rather than years. Beyond the laboratory, machine learning is being applied to clinical development itself: mining electronic health records to find eligible patients, choosing trial sites, predicting dropouts, and stratifying patients so a drug is tested in the people most likely to respond (Vamathevan et al., 2019). The evidence for real gains here is thinner than it is for early discovery.

The most informative numbers so far come from Jayatunga et al. (2024), who examined molecules from AI-native biotechnology companies. Of 24 molecules that had completed Phase I, 21 succeeded, a rate of about 80–90% against a historical range of roughly 40–65%. In Phase II, however, success was around 40%, based on only about ten completed trials, which is close to the historical figure of roughly 29% reported by

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Wong et al. (2019) (Figure 2). One sensible reading is that AI is good at producing molecules that behave well in the body, meaning safe and drug-like, but has not yet shown it can choose biology that works in patients more reliably than humans do.

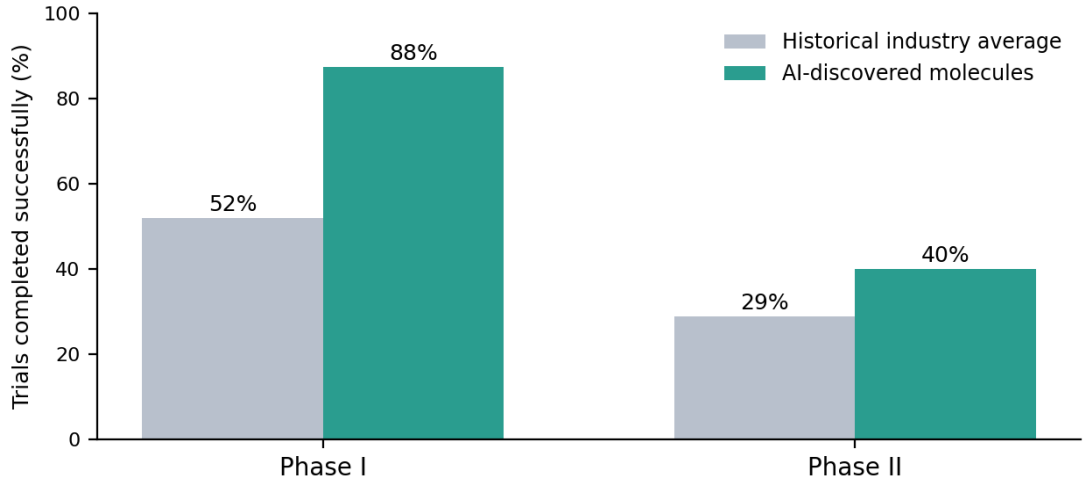


Figure 2. Clinical success rates: AI-discovered molecules versus historical averages. AI figures are 21 of 24 (Phase I) and about 4 of 10 (Phase II) completed trials (Jayatunga et al., 2024); historical figures from Wong et al. (2019).

6. Limits and Cautions

It would be easy to read the success stories as proof that the problem is solved. It is not. Models are only as good as the data behind them, and public datasets lean heavily towards well-studied targets and chemical families, so performance tends to drop on genuinely new biology. Many published results come from the companies that built the tools, and independent replication is still scarce. The Phase II samples are small enough that the numbers could shift as more trials report. Questions about patient-data privacy, regulatory acceptance of AI-derived evidence and the ownership of AI-designed molecules are also unsettled. At the time of writing, industry trackers report that no AI-discovered drug has yet completed the full path to regulatory approval, so the strongest claim anyone can make is that the early stages look faster, not that the whole process is solved.

7. Conclusion

AI is changing drug discovery in ways that are real but specific. It helps researchers sift enormous biological datasets for plausible targets, predict protein structures, design and screen molecules in silico, and screen out likely toxic compounds before the expensive work begins. The best current evidence, high Phase I success but only average Phase II success, suggests that these tools make candidate molecules safer and

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better behaved without yet guaranteeing that they work in patients. The next stage of progress will depend less on cleverer algorithms and more on better data, transparent models and careful clinical validation. If those come together, AI will not replace the scientists, the patience or the clinical trials, but it may make the road to a new medicine noticeably shorter.

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USE OF PESTICIDES AND INSECTICIDES ON CROPS WHICH ARE RESULTING IN CANCER

Mohammed Tahir Pasha, Medical Student, Samarkand state medical university, Uzbekistan
Chandra Uttam , Medical Student, Samarkand state medical university, Uzbekistan
Mohd Kashif , Medical Student, Samarkand state medical university, Uzbekistan
Prethra Rajesh Athulya , Medical Student, Samarkand state medical university, Uzbekistan



Silent Harvester: How Agricultural Pesticides and Insecticides Impact Long-Term Health

- **Abstract / Introduction**

- Modern industrial agriculture relies heavily on synthetic chemicals to boost yield and control pests. However, growing epidemiological research links chronic, low-dose exposure to agricultural insecticides and pesticides with increased cancer risks. This article outlines key pathways of exposure, biological mechanisms, documented cancer links, and actionable policy solutions.

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1. Routes of Exposure

Humans encounter agricultural chemicals primarily through three avenues:

- **Occupational Exposure:** Farmers, pesticide applicators, and field laborers face direct contact via inhalation and skin absorption during mixing, spraying, and harvesting.
- **Environmental Proximity:** Rural populations residing near heavily sprayed agricultural zones absorb drift chemicals through ambient air, topsoil, and local groundwater.
- **Consumer Residues:** The general public ingests trace amounts of systemic insecticides and persistent organochlorines present on unwashed produce and processed goods.

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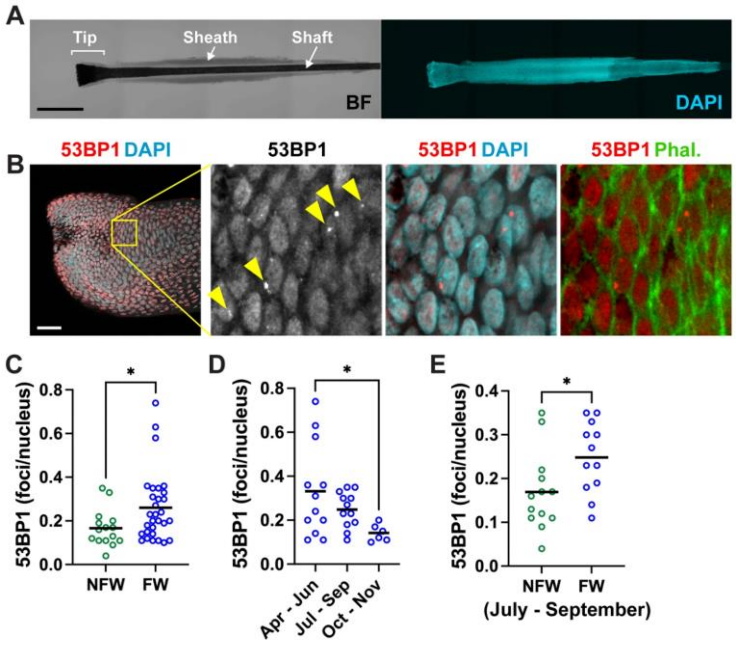




2. Key Biological Mechanisms

How chemical compounds trigger cellular damage leading to oncogenesis:

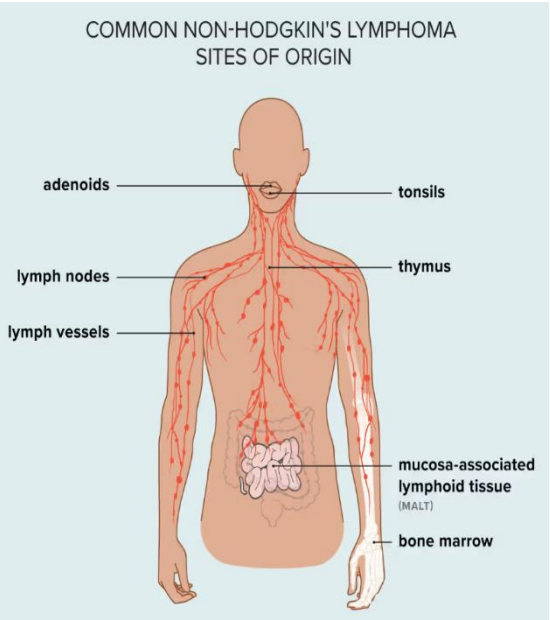
- **DNA Damage & Genotoxicity:** Certain compounds induce single- and double-strand DNA breaks, causing mutations that escape normal cellular repair mechanisms.
- **Endocrine Disruption:** Insecticides (such as organophosphates and synthetic pyrethroids) mimic or block hormones, promoting hormone-receptor-positive cancers.
- **Oxidative Stress:** Chemical toxicity elevates reactive oxygen species (ROS), causing lipid peroxidation, chronic inflammation, and cellular degeneration.

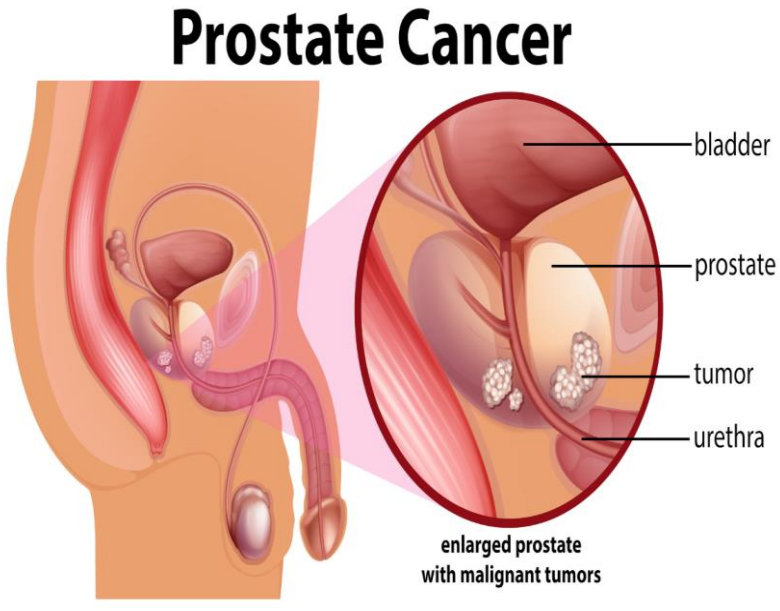


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3. High-Risk Cancers & Associated Compounds

Cancer Type	Primary Chemical Classes / Examples	Primary Exposed Group
Non-Hodgkin Lymphoma (NHL) & Leukemia	Organophosphates (e.g., Diazinon, Malathion), Glyphosate, Lindane	Agricultural workers & children in farming areas
Prostate Cancer	Organochlorines (e.g., DDT derivatives), Fonofos	Male agricultural applicators
Breast & Ovarian Cancers	Endocrine-disrupting pesticides, Organophosphates	Female farmworkers & consumers with high dietary intake
Multiple Myeloma & Bladder Cancer	Permethrin, Captan, Chlorpyrifos	Long-term occupational applicators





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4. Mitigation Strategies & Solutions

1. **Integrated Pest Management (IPM):** Prioritizing biological controls, crop rotation, and targeted non-chemical solutions before applying synthetic sprays.
2. **Regulatory Overhauls:** Enforcing stricter Maximum Residue Limits (MRLs) and phasing out compounds categorized as carcinogenic or endocrine-disrupting by the International Agency for Research on Cancer (IARC).
3. **Safety Gear & Protocol:** Mandating certified Personal Protective Equipment (PPE) and drift-reduction technology during application.
4. **Organic Transition Support:** Providing subsidies and technical assistance for farmers transitioning toward organic farming practices.

Comprehensive Monograph: Agricultural Chemical Exposure, Carcinogenesis, and Public Health Solutions

Abstract

The global expansion of intensive agricultural production relies heavily on synthetic chemicals, including herbicides, insecticides, fungicides, and rodenticides. While these agents have enhanced crop yields and reduced vector-borne disease vectors, extensive epidemiological and molecular research links chronic, low-dose exposure to various malignant neoplasms. This comprehensive study examines the chemical taxonomy of agricultural compounds, exposure vectors, cellular and molecular carcinogenic mechanisms, site-specific cancer etiology, structural barriers to regulation, and sustainable agricultural alternatives.





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Section 1: Chemical Taxonomy and Agricultural Utilization

Modern pesticides encompass diverse chemical classes designed to disrupt physiological processes in target pests. However, many pathways targeted in pests—such as neurotransmission and hormonal signaling—share functional homology with human biochemical systems.

1.1 Insecticides

- **Organochlorines (e.g., DDT, Dieldrin, Lindane, Chlordane):** Highly lipophilic, persistent organic pollutants (POPs) that bioaccumulate across trophic levels. Although banned or restricted in many developed nations under the Stockholm Convention, residual presence in soil and human adipose tissue remains an ongoing exposure risk.
- **Organophosphates (e.g., Chlorpyrifos, Diazinon, Malathion, Parathion):** Esters of phosphoric acid that inhibit acetylcholinesterase ($AChE$), causing accumulation of acetylcholine at neuromuscular junctions. Widely utilized due to lower environmental persistence than organochlorines, yet exhibiting high neurotoxicity and genotoxicity.
- **Carbamates (e.g., Carbaryl, Carbofuran, Methomyl):** Organic compounds derived from carbamic acid. Similar to organophosphates, they inhibit $AChE$ reversibly, but several metabolites demonstrate alkylating properties capable of direct DNA binding.
- **Synthetic Pyrethroids (e.g., Permethrin, Cypermethrin, Deltamethrin):** Synthetic analogs of natural pyrethrins derived from *Chrysanthemum* flowers. Designed to prolong open states of voltage-gated sodium channels in insect nerve membranes. Many act as endocrine-disrupting chemicals (EDCs) in mammalian systems.

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Insecticide	Class	Recommended Use[1]	WHO hazard classification[2]
Bendiocarb	Carbamate	IRS	II
Propoxur	Carbamate	IRS	II
DDT	Organochloride	IRS	II
Fenitrothion	Organophosphate	IRS, Space Spray	II
Malathion	Organophosphate	IRS, Space Spray	III
Pirimiphos-methyl	Organophosphate	IRS, Space Spray	III
α-Cypermethrin	Pyrethroid	IRS, LLIN	II
Bifenthrin	Pyrethroid	IRS	II
Cyfluthrin	Pyrethroid	IRS, LLIN, Space Spray	II
Deltamethrin	Pyrethroid	IRS, LLIN, Space Spray	II
Etofenprox	Pyrethroid	IRS, LLIN, Space Spray	U
λ-Cyhalothrin	Pyrethroid	IRS, LLIN, Space Spray	II
Permethrin	Pyrethroid	LLIN, Space Spray	II
D-Phenothrin	Pyrethroid	Space Spray	U
Resmethrin	Pyrethroid	Space Spray	III

1.2 Herbicides

- **Glyphosate (N-(phosphonomethyl)glycine):** The world's most widely applied broad-spectrum herbicide, operating via inhibition of 5-enolpyruvylshikimate-3-phosphate synthase (EPSPS) in the shikimate pathway. In 2015, the International Agency for Research on Cancer (IARC) classified glyphosate as "probably carcinogenic to humans" (Group 2A).
- **Phenoxy Herbicides (e.g., 2,4-D, MCPA):** Synthetic auxins inducing uncontrolled plant growth. Historically combined with 2,4,5-T (containing 2,3,7,8-tetrachlorodibenzo-p-dioxin [TCDD] contamination) in Agent Orange.
- **Triazines (e.g., Atrazine, Simazine):** Photosynthetic inhibitors that disrupt Electron Transport Chain (ETC) processes in weeds. Atrazine is a potent endocrine disruptor that alters cytochrome P450 aromatase expression.

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Trt no.	Treatment name	*Rate (g a.i. ha^{-1})
1	Hand-weeded check	—
2	Topramazone	12.5
3	Topramazone	25
4	Bentazon	475
5	Bentazon	950
6	Fomesafen	140
7	Fomesafen	280
8	Topramazone+bentazon	12.5 + 475
9	Topramazone+bentazon	25 + 950
10	Cloransulam-methyl	17
11	Cloransulam-methyl	34
12	Cloransulam-methyl+bentazon	17 + 475
13	Cloransulam-methyl+bentazon	34 + 950
14	Fomesafen+bentazon	140 + 840
15	Fomesafen+bentazon	280 + 1683
16	Fluthiacet-methyl	4
17	Fluthiacet-methyl	8
18	Fluthiacet-methyl+bentazon	4 + 475
19	Fluthiacet-methyl+bentazon	8 + 950

*g a.i. ha^{-1}: grams active ingredient per hectare.

Active ingredient	Fungicide	Chemical class
Azoxystrobin (23.1%, w/w)	Amistar	Strobilurine
Bupirimate (27.2%, w/w)	Nimrod	Pyrimidine
Captan (80%)	Captan	Pthalamide
Fenarimol (11.4%, w/w)	Rubigan	Pyrimidine
Fenpropimorph (79.5%, w/w)	Corbel	Morpholine
Fosetyl-aluminium (80%)	Standon Fosetyl-AL	Organophosphorus
Iprodione (25%, w/w)	Rovral Flo	Dicarboximide
Kresoxim-methyl (50%)	Stroby	Strobilurine
Myclobutanil (20%, w/w)	Robut 20	Triazole
Pyrimethanil (37.4%, w/w)	Scala	Pyrimidine
Quinoxyfen (41.32%, w/w)	Fortress	Quinoline
Sulphur (58.8%, w/w)	Sulphur Flowable	Sulphur
Dinocap (33.2%, w/w)	Karathane	Dinitrophenol
Tolylfluanid (50.5%)	Elvaron Multi	Sulphamide
Fenhexamid (50%)	Teldor	Analide

1.3 Fungicides and Other Agents

- **Dithiocarbamates (e.g., Maneb, Zineb, Mancozeb):** Broad-spectrum fungicides that degrade into ethylenethiourea (ETU), a compound demonstrated to induce thyroid carcinomas and hepatic tumors in laboratory animal models.

Section 2: Toxicology and Carcinogenic Mechanisms

Carcinogenesis induced by agrochemicals involves multiple stage-specific biological alterations: tumor initiation, promotion, and progression.

2.1 Genotoxicity and DNA Mutagenesis

Agrochemicals induce structural chromatin alterations via both direct alkylation and indirect metabolic conversion:

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1. **Adduct Formation:** Electrophilic metabolites bind covalently to nucleophilic bases in DNA (e.g., forming $N7$-alkylguanine adducts), causing replication fork arrest and point mutations during repair synthesis.

2. **Chromosomal Aberrations:** Exposure triggers double-strand breaks (DSBs), sister chromatid exchanges (SCE), and micronucleus formation in peripheral blood lymphocytes—established biomarkers of elevated cancer risk.

2.2 Oxidative Stress and Mitochondrial Dysfunction

Pesticides elevate intracellular Reactive Oxygen Species (ROS)—including superoxide anions ($O_2^{\cdot -}$), hydrogen peroxide (H_2O_2), and hydroxyl radicals ($OH^{\cdot}$). Unchecked ROS induce lipid peroxidation of cell membranes and oxidation of DNA bases, primarily yielding 8-hydroxy-2'-deoxyguanosine (8-OHdG), a primary driver of transversions during cell division.

2.3 Endocrine Disruption (EDC Pathways)

Compounds such as Atrazine, Organochlorines, and Pyrethroids interact with nuclear hormone receptors:

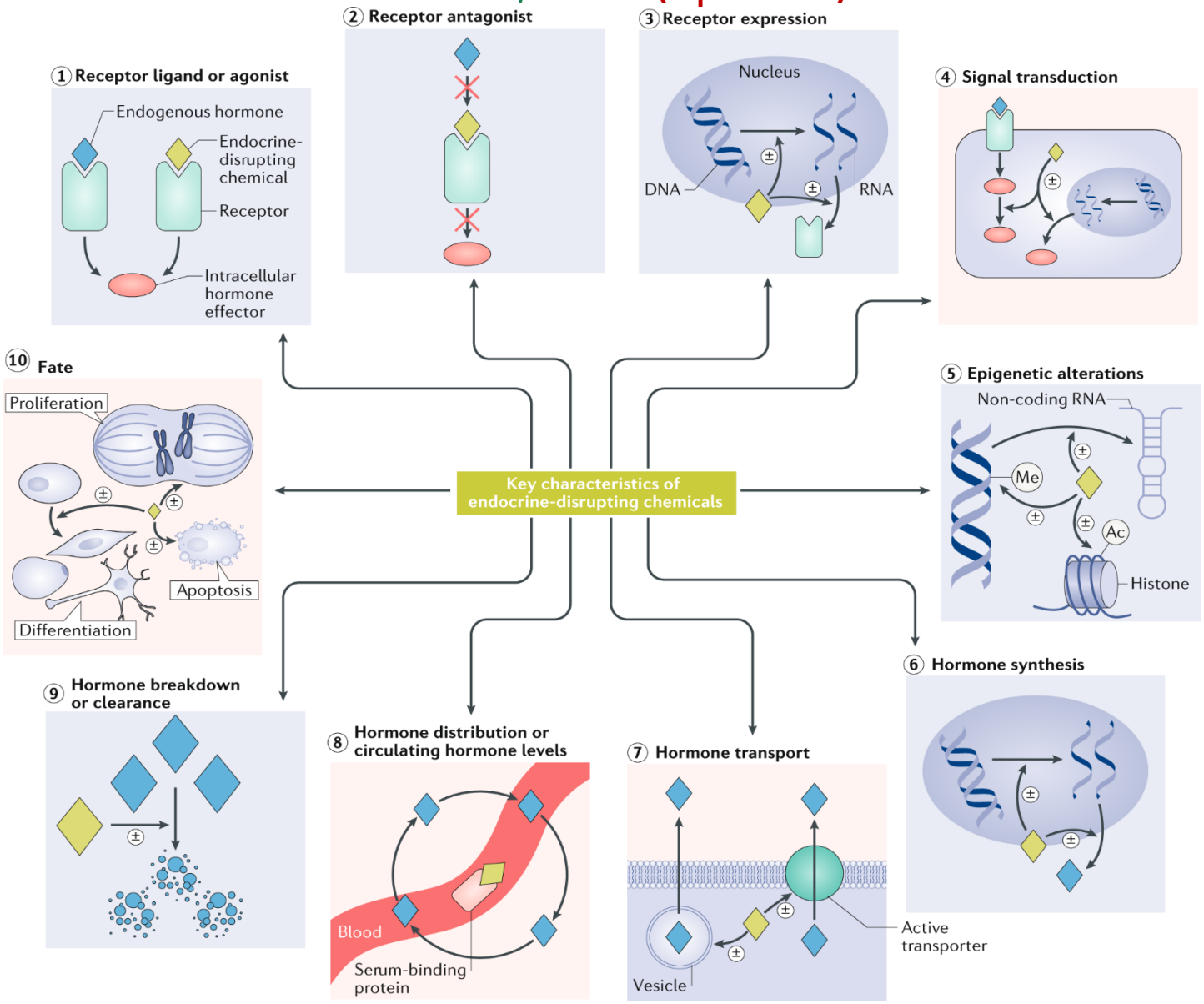
- **Estrogen Receptor ($ER\alpha/ER\beta$) Activation:** Mimicking endogenous 17β-estradiol promotes proliferation of hormone-dependent breast, endometrial, and ovarian cell lines.

- **Androgen Receptor (AR) Antagonism:** Inhibiting endogenous androgen binding induces physiological signaling changes associated with prostate lesions and reproductive dysfunction.

- **Aromatase Induction:** Up-regulating CYP19A1 gene expression accelerates peripheral conversion of androgens to estrogens.

2.4 Epigenetic Dysregulation

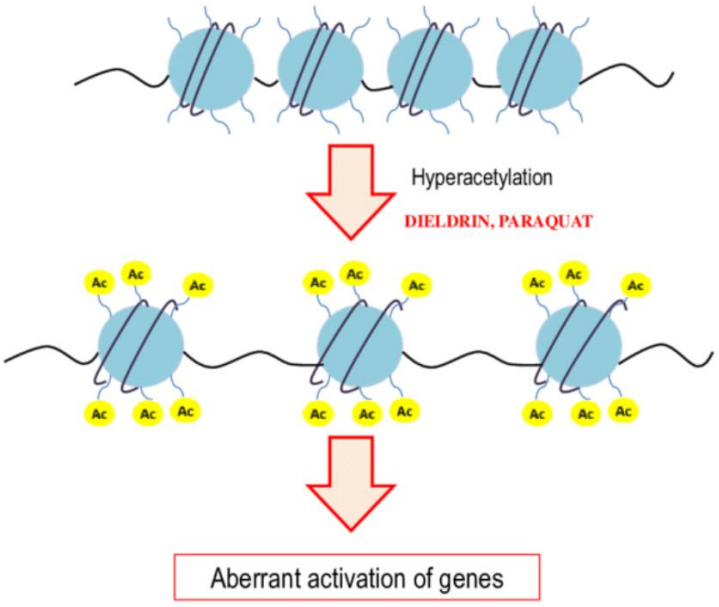
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Chronic low-dose agrochemical exposure induces heritable modifications to gene expression without altering primary DNA sequence:

- **Global DNA Hypomethylation:** Promotes genomic instability and activates proto-oncogenes.
- **Hypermethylation of Tumor Suppressor Genes:** Silences promoter regions of key checkpoint genes such as $p16^{INK4a}$, $p53$, and $BRCA1$.
- **Histone Code Alterations:** Perturbs histone acetylation and methylation patterns, modifying chromatin accessibility to transcription factors.

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Section 3: Routes of Exposure and Human Pharmacokinetics

3.1 Occupational Exposure

- **Dermal Absorption:** Primary exposure route for mixing, loading, and spraying personnel. Skin permeability varies by body site, with the scrotum, forehead, and hands exhibiting high systemic absorption rates.
- **Inhalation:** Inhalation of fine aerosol mists (PM_{10} and $PM_{2.5}$) allows direct pulmonary absorption into bronchial capillaries, bypassing hepatic first-pass metabolism.

3.2 Environmental and Bystander Exposure

- **Off-Target Drift:** Aerial and ground applications disperse active ingredients kilometers beyond targeted boundaries, creating persistent exposure zones for rural residents.
- **Groundwater and Surface Water Contamination:** Soluble compounds (e.g., Nitrate fertilizers, Atrazine, Metolachlor) leach through soil columns into aquifers, resisting degradation in anaerobic subterranean environments.

3.3 Consumer Exposure via Diet

Systemic pesticides absorbed by plants accumulate within edible plant tissues, resisting removal by surface washing. Low-dose daily ingestion yields chronic systemic exposure across urban populations.

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Section 4: Site-Specific Cancer Etiology and Clinical Findings

Epidemiological evaluations—including cohorts from the Agricultural Health Study (AHS)—demonstrate statistical associations between chemical exposure and specific cancer subtypes.

Cancer Subtype	Associated Agrochemical Classes / Agents	Relative Risk / Hazard Ratio Key Observations
Non-Hodgkin Lymphoma (NHL)	Glyphosate, Malathion, Diazinon, Lindane	Strong link to diffuse large B-cell lymphoma (DLBCL) and follicular lymphoma due to chromosomal translocations $t(14;18)$.
Leukemia (Acute & Chronic)	Chlorpyrifos, Permethrin, Benzene derivatives	Higher incidence in pediatric cohorts living near active farming districts (parental exposure connection).
Prostate Cancer	Fonofos, Phorate, Chlorpyrifos, Terbufos	Elevated risk among applicators with family history; synergistic effect with underlying biological vulnerability.
Multiple Myeloma	Permethrin, Dieldrin, Carbaryl	Elevated monoclonal gammopathy of undetermined significance (MGUS)—a premalignant precursor—in exposed applicators.
Thyroid Carcinoma	Mancozeb, Maneb, Ethylenethiourea (ETU)	Thyroid hormone receptor inhibition leading to chronic

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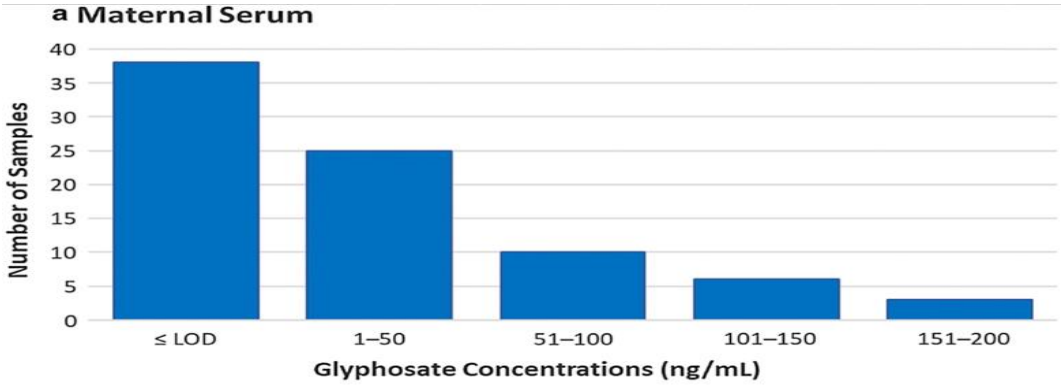
Cancer Subtype	Associated Agrochemical Classes / Agents	Relative Risk / Hazard Ratio Key Observations
		TSH stimulation and follicular hypertrophy/neoplasia.

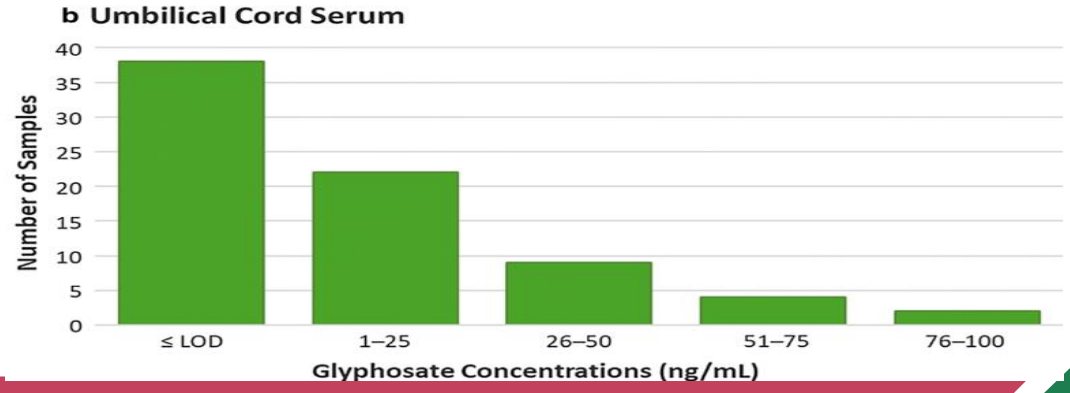
Section 5: Regulatory Frameworks and Methodological Challenges

5.1 Regulatory Gaps and IARC vs. EPA Discordance

Significant discrepancies exist between independent scientific reviews and regulatory approvals:

- **Classification Conflict:** In 2015, IARC designated Glyphosate as Group 2A ("Probably Carcinogenic to Humans"), citing mechanistically sound evidence of genotoxicity and oxidative stress. Conversely, the US EPA and EFSA evaluated industrial dossier data and maintained non-carcinogenic classifications.
- **Chemical-by-Chemical Assessment:** Current risk assessment frameworks test compounds individually. However, real-world exposure involves complex agrochemical mixtures, where synergistic toxicity can exceed predicted additive effects.





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5.2 Epidemiological Research Barriers

Identifying causal links between agrochemical exposure and carcinogenesis presents distinct methodological challenges:

- **Exposure Misclassification:** Reliance on recall surveys introduces recall bias; historical exposure records are often incomplete.
- **Latency Periods:** Malignancies typically manifest 15–40 years post-exposure, obscuring the link to specific chemical compounds applied decades earlier.
- **Confounding Variables:** Occupational co-exposures (e.g., engine exhaust, ultraviolet radiation, zoonotic viruses, silica dust) complicate single-agent attribution.

Section 6: Policy Intervention and Sustainable Alternatives

Addressing agrochemical health risks requires structural transitions in agricultural management, legislative reform, and public health policy.

6.1 Integrated Pest Management (IPM) and Agroecology

- **Biological Control:** Utilizing natural predators, parasites, and pathogens (e.g., *Bacillus thuringiensis*) to control pest populations naturally.
- **Crop Diversity:** Implementation of complex crop rotations, intercropping, and cover cropping to break pest life cycles without synthetic inputs.
- **Biopesticides:** Transitioning to plant-derived botanical extractives (e.g., Azadirachtin) and microbials that exhibit low environmental persistence and reduced mammalian toxicity.

6.2 Structural Policy Recommendations

1. **Mandated Mixture Toxicity Testing:** Regulatory bodies must require environmental impact assessments that evaluate cumulative toxicity from simultaneous multi-chemical exposures.
2. **Buffer Zone Regulations:** Expanding mandatory non-spray buffer margins around schools, residential developments, and vulnerable aquatic ecosystems.
3. **Occupational Protections:** Universalizing health monitoring protocols, mandatory PPE access, and continuous education programs for farm laborers across developing and developed nations alike.

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1. Advanced Molecular Pathways and Synergistic Toxicity

While individual chemical exposure profiles provide baseline risk assessments, real-world agricultural exposures involve complex mixtures. Understanding the chemical dynamics between distinct compounds reveals accelerated oncogenic pathways.

1.1 Synergistic Toxicity Dynamics

- **Cytochrome P450 Phase I Crosstalk:** Certain fungicides (e.g., triazoles) induce hepatic $CYP1A2$ and $CYP3A4$ enzymes. When co-administered with organophosphate insecticides, accelerated bioactivation converts parent compounds into toxic oxon intermediates faster than endogenous Phase II detoxifying enzymes (GST, UDP-glucuronosyltransferases) can neutralize them.
- **Efflux Pump Inhibition:** Persistent organic pollutants and surfactants in herbicide formulations alter cell membrane permeability by inhibiting P-glycoprotein ($MDR1$) transporters. This blocks multidrug resistance mechanisms, increasing intracellular accumulation of genotoxic compounds.
- **Metabolic Interactions:**
    1. **Potentiation:** Non-toxic adjuvants amplify the cellular uptake of active ingredients.
    2. **Additive Genotoxicity:** Simultaneous exposure to alkylating carbamates and ROS-generating herbicides produces cumulative strand breaks that overwhelm base excision repair (BER) systems.

2. Advanced Diagnostic Biomarkers

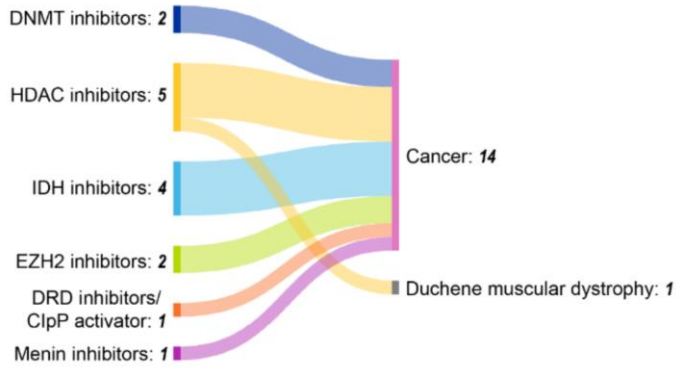
Early identification of chronic agrochemical toxicity relies on specific molecular and physiological biomarkers before clinical tumor manifestation.

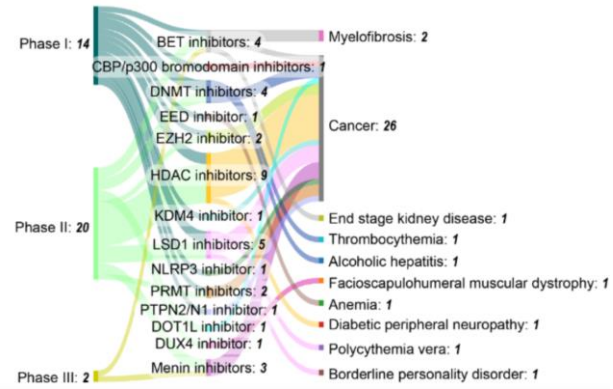
2.1 Genomic and Epigenetic Indicators

- $8-OHdG$ **(8-hydroxy-2'-deoxyguanosine):** Elevated urinary excretion levels reflect systemic oxidative DNA damage.
- **Micronucleus Frequency:** Quantified in buccal mucosa or peripheral blood lymphocytes, serving as an indicator of chromosome breakage or loss.

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- **Telomere Attrition Rates:** Accelerated shortening of leukocyte telomeres observed in long-term applicators, signaling premature cellular senescence and genomic instability.
- **DNA Methylation Profiles:** Specific hypermethylation patterns in promoter regions of $p53$ and $p16^{INK4a}$ genes detectable in blood plasma DNA fragments.





3. Vulnerable Populations and Developmental Susceptibility

3.1 Transplacental and Early Life Vulnerabilities

Fetal and pediatric populations exhibit heightened susceptibility to agrochemical toxicity due to developmental physiology:

1. **Immature Detoxification Pathways:** Lower hepatic expression of Paraoxonase-1 ($PON1$) in neonates reduces the rate of organophosphate hydrolysis.
2. **Placental Transfer:** Lipophilic organochlorines cross the placental barrier, exposing developing embryonic tissues during critical organogenesis windows.
3. **Blood-Brain Barrier Permeability:** Increased permeability in early childhood allows neurotoxic and genotoxic compounds greater access to central nervous system tissue.

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4. Modern Agricultural Precision & Alternatives

Transitioning away from broad-spectrum, high-volume chemical applications involves adopting targeted technology alongside biological management strategies.

4.1 Precision Agriculture and Technological Interventions

- **Targeted Spot-Spraying:** Computer-vision-guided sprayers utilize real-time image recognition to apply micro-doses directly to targeted weeds or pests, reducing overall chemical volume by up to 80%.
- **Drift-Reduction Technologies (DRT):** Air-assisted boom sprayers and specialized drift-reducing nozzles minimize aerosol drift into adjacent ecosystems.
- **Biopesticides and Microbials:** Utilization of natural enemies, entomopathogenic fungi (*Metarhizium anisopliae*), and botanical extracts offers targeted pest management with rapid environmental degradation and minimal human cell toxicity.

5. Comprehensive Policy and Legislative Roadmap

To mitigate public health risks associated with agricultural chemical exposure, regulatory bodies must update policy guidelines:

1. **Mandate Cumulative Exposure Risk Assessments:** Shift from single-chemical evaluations to testing combined real-world exposure mixtures.
2. **Implement Real-Time Worker Monitoring:** Enforce mandatory biological monitoring ($PON1$ activity, urinary $8-OHdG$) for all commercial chemical applicators.
3. **Restrict Known Endocrine Disruptors:** Establish systematic phase-outs for active ingredients meeting IARC Group 1 or 2A classifications.
4. **Expand Community Buffer Zones:** Establish enforceable minimum non-spray boundaries around rural residential zones, public schools, and surface water aquifers.

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CLIMATE CHANGE AND VECTOR-BORNE DISEASES

Sugam Boruah , Medical Student, Samarkand state medical university, Uzbekistan

Neha Bharti , Medical Student, Samarkand state medical university, Uzbekistan

Vakaliya Aish , Medical Student, Samarkand state medical university, Uzbekistan

Khushnuma , Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Climate change is reshaping the environmental conditions in which mosquitoes, ticks, sandflies and other disease vectors survive and transmit pathogens. Changes in temperature, rainfall, humidity and the frequency of extreme events can alter vector development, breeding sites, seasonal activity and geographic distribution. These changes influence risks for diseases such as malaria, dengue, chikungunya, Zika, West Nile fever, tick-borne encephalitis and Lyme disease, but the direction and magnitude of impact differ by pathogen, vector and location. Climate is only one part of the picture: urbanization, land use, water and sanitation systems, housing, human mobility, immunity and public-health capacity can amplify or reduce risk. This narrative review summarizes the principal biological pathways linking climate change with vector-borne disease, highlights selected disease examples, and discusses surveillance, prevention and adaptation strategies. Current evidence supports climate-informed public-health planning, but it also cautions against assuming that warming will increase every vector-borne disease everywhere. Effective responses require local monitoring, reliable healthcare, community engagement and integrated vector management alongside climate mitigation. Research priorities include improved early-warning systems, comparable surveillance data and stronger evaluation of interventions in vulnerable communities.

Keywords: climate change; vector-borne diseases; malaria; dengue; mosquitoes; ticks; public health; climate adaptation; surveillance; vector control

1. Introduction

Vector-borne diseases are infections transmitted to humans or animals by living organisms such as mosquitoes, ticks, sandflies and fleas. They include parasitic diseases, including malaria; viral diseases, such as dengue, chikungunya and West Nile fever; and bacterial infections, such as Lyme disease. The World Health Organization reports that vector-borne diseases account for more than 17% of infectious diseases

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and cause over 700,000 deaths annually [1]. Their burden is unevenly distributed, with many affected communities facing poverty, limited healthcare access and environmental conditions that make prevention difficult.

Climate change matters because vectors and pathogens respond to their surroundings. Temperature can affect how quickly a mosquito develops, how often it bites, how long it survives and how rapidly a pathogen develops inside it. Rainfall can create breeding habitats, while drought may encourage households to store water in containers that become mosquito breeding sites. Humidity influences the survival of adult insects, and warmer conditions may allow some vectors to persist in areas where winters previously limited them.

Yet the relationship is not a simple rule that warmer weather always means more disease. Excessive heat can reduce vector survival, heavy rain can flush larvae from breeding sites, and drought can eliminate some habitats. Disease transmission also requires the pathogen, vector, human host and suitable social conditions to coincide. A careful review therefore needs to consider both climate-related mechanisms and the non-climatic factors that determine whether ecological change becomes a human health problem.

2. Review approach

This narrative review synthesizes selected evidence from World Health Organization resources, the Intergovernmental Panel on Climate Change (IPCC), and peer-reviewed reviews on climate and vector-borne disease. It is not a systematic review or meta-analysis, and no new data were collected. Examples are included to illustrate mechanisms rather than rank countries or predict individual outbreaks. Since effects vary by region and study design, the review avoids treating projections as direct observations or assuming that associations alone prove climate attribution.

3. How climate affects vectors and transmission

Temperature influences several stages of vector and pathogen biology. Within a suitable range, warmer conditions can accelerate insect development and pathogen replication, shorten the time before an infected vector becomes infectious, and extend seasonal activity. However, vectors have thermal limits. Beyond an optimal range, survival and reproduction may fall. These nonlinear responses help explain why warming can increase risk in some cooler areas while reducing suitability in locations that become too hot or dry [2,3].

Rainfall has similarly mixed effects. Moderate rainfall may create pools and containers in which mosquitoes breed. Flooding can disrupt sanitation and create standing water

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after waters recede, but intense rainfall may also wash away larvae. During drought, water storage can unintentionally provide breeding habitats for Aedes mosquitoes. Humidity, vegetation, soil moisture and the availability of animal hosts can further shape vector survival and contact with people.

Climate-related hazards can also affect health systems and behaviour. Floods and storms may damage clinics, interrupt vector-control programmes, displace families and delay diagnosis. Heat can alter outdoor activity and water-use patterns. Human travel and trade may introduce pathogens or vectors into newly suitable areas, while unplanned urban growth can create habitats around homes. The IPCC concludes that higher temperatures combined with land-use change are making more areas suitable for some vector-borne diseases, while emphasizing that outcomes depend on context [3].

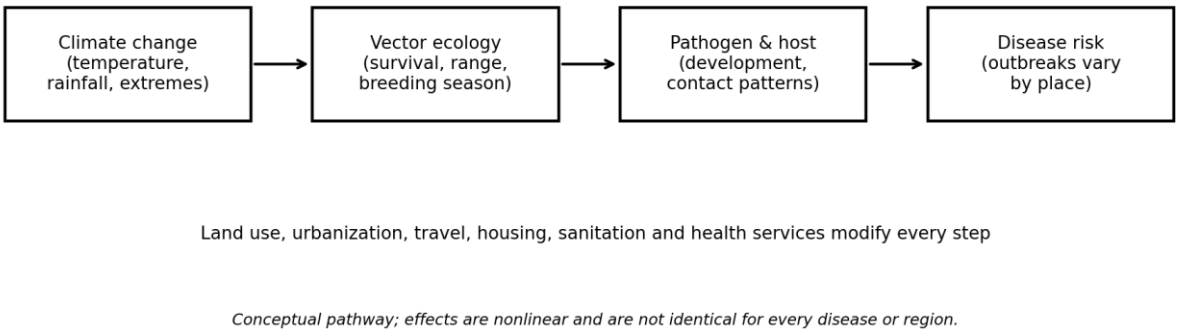


Figure 1. Conceptual pathways linking climate change with vector-borne disease risk. Non-climatic determinants modify each stage; the figure is explanatory rather than quantitative.

4. Selected diseases and climate-related patterns

4.1 Malaria

Malaria is transmitted by Anopheles mosquitoes. Temperature and rainfall affect mosquito survival, breeding habitats and the development of Plasmodium parasites inside the vector. In some highland or fringe regions, warmer conditions may increase seasonal suitability and expose populations with limited acquired immunity to outbreaks. In already hot areas, extreme temperatures or changes in rainfall may reduce suitability or produce less predictable effects. Malaria trends are also strongly shaped by insecticide-treated nets, indoor residual spraying, antimalarial treatment, housing,

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population movement and health-service access; climate alone cannot explain year-to-year changes.

4.2 Dengue and chikungunya

Dengue and chikungunya are primarily transmitted by Aedes mosquitoes, which often breed in small water-holding containers close to people. Warmer conditions can accelerate mosquito development and viral replication within a suitable temperature range. Rainfall, intermittent water supply and household water storage can influence breeding sites. The IPCC reports that climate change and variability have contributed to the spread of chikungunya and dengue suitability in several regions, while stressing interactions with human activities and urban conditions [3]. Dense housing, waste management, reliable water services and community participation remain central to prevention.

4.3 Tick-borne diseases

Ticks are affected by temperature, humidity, vegetation and the distribution of wildlife or other host animals. Milder winters and longer warm seasons can change tick activity and expand suitable habitat in some areas. Lyme disease vectors in North America and Europe, and tick-borne encephalitis in parts of Europe, are among the examples discussed in IPCC assessments [3]. Nonetheless, land management, outdoor behaviour, host populations, diagnostic practices and public awareness also affect observed case numbers. A change in the geographic range of a tick does not automatically mean that every location will experience sustained human transmission.

4.4 Other vector-borne infections

West Nile fever, Zika, yellow fever, leishmaniasis and Rift Valley fever illustrate the diversity of transmission systems. Some depend on mosquitoes and bird or mammal hosts; others involve sandflies, livestock or complex ecological cycles. Climate can alter the distribution and timing of vectors or reservoir hosts, but disease emergence usually requires additional conditions, including pathogen introduction and contact between vectors and susceptible people. These differences make disease-specific and locally grounded surveillance essential [2,4].

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Table 1. Climate-sensitive pathways and examples

Disease / vector	Climate-sensitive mechanism	Possible pattern	Important modifiers
Malaria / Anopheles mosquitoes	Temperature affects mosquito survival and parasite development; rainfall affects breeding sites	Seasonal suitability may rise in some cooler or highland areas; extreme heat can limit transmission	Vector control, treatment access, housing, immunity and land use
Dengue / Aedes mosquitoes	Temperature affects vector and virus development; water availability shapes container habitats	Longer seasons or expansion into suitable areas; drought may increase household storage habitats	Urban density, waste collection, water supply and community action
Chikungunya / Aedes mosquitoes	Similar mosquito ecology to dengue; weather influences seasonal activity	Outbreak risk may shift where vectors and virus overlap	Travel, population susceptibility, surveillance and mosquito control
Lyme disease / Ixodes ticks	Temperature and humidity affect tick activity; vegetation and hosts shape habitat	Activity seasons or suitable ranges may change in some regions	Wildlife hosts, outdoor exposure, land management and awareness
West Nile fever / mosquitoes and birds	Weather affects mosquitoes and bird-vector contact	Local transmission may change with seasonal conditions	Bird ecology, urban design, surveillance and clinical detection

5. What the evidence does—and does not—show

Evidence is strongest for the biological sensitivity of vectors and pathogens to temperature and moisture, and for observed changes in suitability or distribution in particular settings. The IPCC reports that climate change and variability have facilitated the spread or changed the suitability of several vector-borne diseases, with

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confidence levels varying by disease and region [3]. A major review also emphasizes that climate change interacts with land use, human mobility and behaviour rather than acting in isolation [2].

Attribution remains challenging. Surveillance may improve over time, making previously undetected cases visible. Population growth, urbanization, conflict, changes in diagnostic access, insecticide resistance and public-health interventions can all alter disease incidence. Some studies project future suitability rather than actual cases, and a suitable climate is only one condition for transmission. The WHO has noted that the evidence base for some neglected tropical diseases and malaria remains uneven and that further research is needed to clarify impacts and adaptation options [4].

Table 2. Interpreting climate and disease evidence

Evidence type	What it can tell us	Main caution
Laboratory studies	How temperature or humidity affects vector or pathogen biology	Controlled conditions may not reflect field ecology
Field surveillance	Whether cases, vectors or seasons are changing locally	Case detection, reporting and interventions may also change
Climate-suitability models	Where conditions may become more or less suitable	Suitability is not the same as predicted case counts
Outbreak investigations	How weather, ecology and human exposure aligned during an event	Multiple causes often coincide; attribution needs careful analysis

6. Public-health prevention and adaptation

Climate-informed surveillance is a practical starting point. Health agencies can combine local temperature and rainfall information with vector monitoring, case reports, laboratory results and information about population movement. Early-warning systems are most useful when alerts trigger clear actions—for example, targeted vector control, community messaging, diagnostic readiness and supplies for clinics. Forecasts should be evaluated regularly so that warnings do not create unnecessary alarm or divert resources from other health priorities.

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Integrated vector management combines environmental measures, personal protection, community engagement and appropriate insecticide use. Removing standing water, covering storage containers, improving drainage and waste collection, installing screens, using bed nets where appropriate and applying recommended repellents can reduce exposure. Interventions should be tailored to the vector: bed nets are particularly important for many malaria settings, while eliminating small water-holding containers is central to Aedes control. Insecticide resistance monitoring is essential to preserve effectiveness.

Health systems also need resilience. Clinics should maintain access to diagnostic tests, trained staff and effective treatments during floods, heatwaves and other disruptions. Risk communication should be clear, multilingual and locally trusted. Urban planning, reliable water and sanitation, safe housing and environmental management can reduce breeding sites while improving health more broadly. Climate mitigation is also a health intervention: limiting future warming reduces the scale of risks that communities will otherwise have to manage.

7. Equity and vulnerable populations

Climate-related disease risks are not distributed equally. People living in poorly constructed housing, informal settlements, remote communities or areas with unreliable water supply may face greater exposure and have fewer options for protection. Outdoor workers, displaced people and communities affected by conflict can have difficulty avoiding vectors or accessing timely diagnosis. Children, pregnant people, older adults and people with limited access to healthcare may face additional risks from infection or its complications.

Equitable adaptation means involving affected communities in programme design, supporting local health workers and ensuring that prevention tools and treatment are affordable. A technically sophisticated early-warning model will have limited value if the clinic cannot access tests or residents cannot act on the warning. Public-health planning should therefore pair climate information with investment in basic services and community trust.

8. Research priorities

Future research should link long-term climate records with standardized vector surveillance, laboratory-confirmed disease data and information on interventions, land use and human mobility. Studies should distinguish observed changes from projections and report uncertainty transparently. More evidence is needed from underrepresented regions and for diseases with limited research funding. Evaluations should also test

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whether early-warning systems and climate-informed vector-control programmes reduce illness, deaths or response costs—not merely whether they predict environmental suitability.

Researchers and health agencies can strengthen collaboration between meteorology, ecology, entomology, infectious-disease medicine and public health. Shared data standards and accessible local dashboards may help convert climate information into timely decisions. Research should also examine unintended effects, such as the ecological consequences of control measures, and identify adaptation strategies that remain effective under different future climate scenarios.

9. Conclusion

Climate change is altering the environmental conditions that shape vector survival, pathogen development and human exposure. These changes can extend transmission seasons or create new areas of suitability for diseases such as malaria, dengue, chikungunya and some tick-borne infections. However, effects are complex and differ by location; heat can become excessive for vectors, rainfall can either create or remove breeding sites, and social conditions often determine whether ecological suitability leads to disease. Public-health responses should combine climate-informed surveillance, integrated vector management, resilient healthcare, community engagement and improvements in housing, water and sanitation. Reducing greenhouse-gas emissions is essential to limit future risk, while targeted adaptation can protect communities from risks already emerging. The goal is not to predict every outbreak perfectly, but to build systems capable of detecting changing threats and responding quickly and fairly.

Limitations

This is a narrative review of selected sources rather than a systematic review with formal screening and risk-of-bias assessment. Findings vary across diseases, regions and methods; the review should be updated as new evidence becomes available.

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ZAMONAVIY MAKTABLAR: O‘ZBEKISTON VA FILIPPIN TA’LIM TIZIMINING QIYOSIY TAHLILI

Ismoilova Nilufar Muhammadbobir qizi

Toshkent shahridagi Bucheon universiteti, Ta’limda boshqaruv yo‘nalishi magistranti

E-mail: grafiniabarlas@gmail.com

ANNOTATSIYA

Maqolada zamonaviy maktabni rivojlantirishning boshqaruv, o‘quv dasturi, raqamlashtirish, pedagoglar salohiyati va o‘quv natijalarini baholashga oid jihatlari O‘zbekiston hamda Filippin tajribasi asosida qiyosiy tahlil qilinadi. Tadqiqot hujjatlar tahlili, qiyosiy yondashuv va xalqaro baholash ma’lumotlarini sharhlash usullariga tayanadi. O‘zbekistonda “O‘zbekiston — 2030” strategiyasi doirasida maktab infratuzilmasi, elektron doskalar, yangi avlod darsliklari va pedagoglar malakasini oshirishga doir maqsadlar belgilangan. Filippinda esa MATATAG kun tartibi va Basic Education Development Plan 2030 o‘quv dasturining dolzarbligi, o‘quvchi farovonligi, inklyuziv ta’lim hamda o‘qituvchilarni qo‘llab-quvvatlashga urg‘u beradi. OECDning PISA 2022 ma’lumotlari har ikki tizimda ham o‘quvchilarning funksional savodxonligini mustahkamlash va natijalardan boshqaruv qarorlarida foydalanish zarurligini ko‘rsatadi. Maqola yakunida maktab darajasida diagnostik baholash, o‘quvchilarni ehtiyojiga qarab guruhlash, pedagogik hamkorlik, raqamli monitoring va resurslarni dalillarga asoslangan taqsimlash bo‘yicha amaliy tavsiyalar ishlab chiqiladi. Tahlil ikkilamchi manbalarga asoslangan bo‘lib, undagi tavsiyalarni empirik tekshirish uchun alohida maktablarda pilot tadqiqot o‘tkazish lozim.

Kalit so‘zlar: zamonaviy maktab, ta’lim menejmenti, O‘zbekiston, Filippin, PISA, raqamli ta’lim, o‘quv natijalari, maktab rahbarligi.

АННОТАЦИЯ

В статье проводится сравнительный анализ подходов к развитию современной школы в Узбекистане и на Филиппинах с акцентом на управление, учебные программы, цифровизацию, профессиональный потенциал педагогов и оценивание результатов обучения. Использованы анализ нормативных документов, сравнительный подход и интерпретация данных международных исследований. В Узбекистане стратегия «Узбекистан — 2030» предусматривает развитие школьной инфраструктуры, внедрение электронных досок, обновление учебников и повышение квалификации педагогов. На Филиппинах программа

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MATATAG и План развития базового образования до 2030 года ориентированы на актуальность учебной программы, благополучие учащихся, инклюзивность и поддержку учителей. Данные PISA 2022 указывают на необходимость укрепления функциональной грамотности и использования результатов оценивания для управленческих решений. Предлагаются рекомендации по диагностическому оцениванию, гибкой организации учебных групп, цифровому мониторингу и распределению ресурсов на основе данных.

Ключевые слова: современная школа, управление образованием, Узбекистан, Филиппины, PISA, цифровое образование, результаты обучения, школьное лидерство.

ABSTRACT

This article compares approaches to modern school development in Uzbekistan and the Philippines, focusing on governance, curriculum, digitalisation, teacher capacity and learning-outcome assessment. The study uses document analysis, a comparative approach and interpretation of international assessment data. Uzbekistan's Uzbekistan–2030 Strategy includes goals related to school infrastructure, electronic boards, updated learning materials and teacher professional development. In the Philippines, the MATATAG Agenda and the Basic Education Development Plan 2030 emphasise curriculum relevance, learner well-being, inclusive education and teacher support. OECD PISA 2022 data indicate that strengthening foundational literacy and numeracy and using assessment evidence for school-level decisions remain important priorities. The article proposes practical measures for diagnostic assessment, flexible grouping, collaborative teacher development, digital monitoring and evidence-informed resource allocation. As the study is based on secondary sources, the proposed measures should be tested through pilot studies in individual schools.

Keywords: modern school, education management, Uzbekistan, Philippines, PISA, digital learning, learning outcomes, school leadership.

KIRISH

Zamonaviy maktab tushunchasi faqat yangi bino yoki raqamli qurilmalar bilan cheklanmaydi. U o‘quvchi ehtiyojiga mos ta’lim, malakali pedagog, xavfsiz va inklyuziv muhit, natijalarni muntazam tahlil qilish hamda maktab jamoasining birgalikda qaror qabul qilishini birlashtiradigan boshqaruv tizimidir. Shu sababli maktabni modernizatsiya qilishda infratuzilma bilan bir qatorda o‘qitish sifati va boshqaruv amaliyotini ham takomillashtirish talab etiladi.

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O‘zbekistonda ta’limni rivojlantirishning strategik yo‘nalishlari “O‘zbekiston — 2030” strategiyasi va maktab ta’limini rivojlantirishga oid normativ hujjatlarda belgilangan. Strategik ko‘rsatkichlar qatorida maktab muhitini yaxshilash, elektron doskalar ulushini oshirish, yangi avlod o‘quv materiallarini yaratish hamda pedagoglarning kasbiy salohiyatini rivojlantirish ko‘zda tutiladi [1; 2]. Filippinda esa MATATAG kun tartibi va Basic Education Development Plan 2030 ta’lim mazmunini dolzarblashtirish, o‘quvchilarning farovonligi, inklyuziv ta’lim va o‘qituvchilarni qo‘llab-quvvatlashni ustuvor vazifa sifatida belgilaydi [3].

Tadqiqotning maqsadi — O‘zbekiston va Filippin ta’lim tizimlaridagi zamonaviy maktabni rivojlantirish yondashuvlarini qiyoslash va O‘zbekiston maktablari boshqaruvi uchun moslashtirilishi mumkin bo‘lgan amaliy yo‘nalishlarni aniqlash. Tadqiqot savoli: xalqaro tajriba maktab rahbarlariga o‘quv natijalarini yaxshilash va resurslardan samarali foydalanishda qanday boshqaruv yechimlarini taklif etadi?

TADQIQOT METODOLOGIYASI

Tadqiqot sifat jihatidan qiyosiy tahlil shaklida tashkil etildi. Manbalar uch guruhga ajratildi: (1) O‘zbekistonning strategik va normativ hujjatlari; (2) Filippin Ta’lim departamentining MATATAG va BEDP 2030 bo‘yicha rasmiy materiallari; (3) OECDning PISA natijalari. Tahlil mezonlari sifatida ta’lim siyosatining ustuvorliklari, o‘quv dasturi, pedagoglarni qo‘llab-quvvatlash, maktab infratuzilmasi, o‘quv natijalarini baholash va maktab boshqaruvida ma’lumotlardan foydalanish olindi.

Metodologik cheklov shundan iboratki, maqolada maktablarda birlamchi so‘rovnoma yoki tajriba-sinov o‘tkazilmagan. Shu bois taqqoslash tizimlarning barcha xususiyatlarini qamrab olmaydi va tavsiyalar sabab-oqibat munosabati isbotlangan natijalar sifatida emas, balki keyingi amaliy tekshiruv uchun takliflar sifatida talqin qilinishi kerak.

NATIJALAR

O‘zbekiston tajribasi. “O‘zbekiston — 2030” strategiyasida umumiy o‘rta ta’lim muassasalarida qulay muhit yaratish, elektron doskalar bilan ta’minlash, zamonaviy darslik va multimedia materiallarini ishlab chiqish hamda pedagoglar malakasini oshirish bo‘yicha bosqichma-bosqich ko‘rsatkichlar belgilangan [1]. Bu maqsadlar maktab rahbari oldiga xarid va infratuzilma masalalaridan tashqari, texnologiyalarning dars jarayonida haqiqiy pedagogik qiymat yaratishini kuzatish vazifasini ham qo‘yadi. Raqamli vositaning mavjudligi o‘z-o‘zidan o‘quv natijasini yaxshilamaydi; undan foydalanish metodikasi, o‘qituvchining tayyorgarligi va muntazam tahlil muhim hisoblanadi.

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Filippin tajribasi. Filippin Ta'lim departamenti MATATAG kun tartibini va Basic Education Development Plan 2030ni milliy siyosat sifatida ilgari surgan. Rejada o'quv dasturining hayotga mosligi, ta'lim xizmatlarini yetkazib berish, o'quvchilar farovonligi va inklyuzivligi hamda o'qituvchilarni qo'llab-quvvatlash alohida yo'nalishlar sifatida ko'rsatiladi [3]. MATATAG o'quv dasturi K–10 bosqichlari uchun joriy etish ko'rsatmalariga ega; yuqori sinflar uchun tegishli alohida tartiblar qo'llanishi qayd etilgan [4]. Bu tajriba o'quv mazmunini soddalashtirish va asosiy kompetensiyalarga e'tibor qaratish bilan bir qatorda, amalga oshirish bosqichlarini aniq belgilash zarurligini ko'rsatadi.

Xalqaro baholash dalillari. OECDning PISA 2022 ma'lumotlariga ko'ra, Filippinda 15 yoshli o'quvchilarning o'rtacha natijalari matematika bo'yicha 355, o'qish bo'yicha 347 va tabiiy fanlar bo'yicha 356 ballni tashkil etgan. Matematikada kamida 2-darajaga erishgan o'quvchilar ulushi 16%, o'qishda 24%, tabiiy fanlarda 23% bo'lgan [5]. Ushbu ko'rsatkichlar o'quvchilarning asosiy savodxonligini tizimli ravishda mustahkamlash muhimligini ko'rsatadi. PISA natijalarini mamlakatlar bo'yicha to'g'ridan-to'g'ri reyting sifatida talqin qilishdan ko'ra, ta'limdagi muammolar va tengsizliklarni aniqlash vositasi sifatida ishlatish maqsadga muvofiq.

Taqqoslash natijalari quyidagi jadvalda umumlashtirilgan.

Mezon	**O'zbekiston**	**Filippin**
Strategik yo'nalish	"O'zbekiston — 2030": maktab muhiti, raqamli jihozlar, darsliklar va pedagoglar salohiyati.	MATATAG va BEDP 2030: dolzarb o'quv dasturi, farovonlik, inklyuzivlik va o'qituvchini qo'llash.
O'quv dasturi	Milliy dasturlarni yangilash, yangi avlod darsliklari va multimedia resurslarini rivojlantirish.	MATATAG orqali K–10 o'quv dasturini qayta ko'rib chiqish va asosiy kompetensiyalarga e'tibor.
Boshqaruv uchun saboq	Raqamli vositalarni dars sifati va o'quvchi taraqqiyoti ko'rsatkichlari bilan bog'lash.	Islohotni bosqichma-bosqich joriy etish, o'quvchi farovonligi va o'qituvchi ko'magini kuzatish.

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Asosiy ehtiyoj	Maktablar kesimida sifat, resurs va pedagogik natijalar bo‘yicha monitoringni kuchaytirish.	Asosiy savodxonlik, kadrlar bilan ta’minlash va infratuzilmadagi hududiy tafovutlarni kamaytirish.

1-jadval. O‘zbekiston va Filippinda zamonaviy maktabni rivojlantirish yondashuvlarining umumlashtirilgan qiyosi.

MUHOKAMA

Qiyosiy tahlil zamonaviy maktab siyosatida uchta umumiy yo‘nalish muhimligini ko‘rsatadi. Birinchidan, o‘quv dasturi o‘quvchilarga faktlarni eslab qolish bilangina cheklanmay, bilimni real vaziyatlarda qo‘llash imkonini berishi lozim. Ikkinchidan, maktab rahbarining vazifasi resurslarni taqsimlash bilan bir qatorda o‘qituvchilarning kasbiy hamkorligi va dars sifati bo‘yicha qayta aloqani tashkil etishdan iborat. Uchinchidan, xalqaro baholash va maktab ichki ma’lumotlari muammoni aniqlash, maqsad belgilash va keyingi choralarni baholash uchun birgalikda qo‘llanishi zarur.

Filippin tajribasidan O‘zbekiston maktablari uchun tayyor modelni to‘liq ko‘chirib olish to‘g‘ri bo‘lmaydi: mamlakatlarning til muhiti, hududiy sharoiti, boshqaruv tuzilmasi va resurslari farq qiladi. Biroq o‘quv dasturidagi ustuvor kompetensiyalarni aniqlashtirish, o‘quvchi farovonligini boshqaruv ko‘rsatkichlariga kiritish va islohotlarni bosqichma-bosqich joriy etish tamoyillari moslashtirilishi mumkin.

Maktab darajasida quyidagi amaliy mexanizm taklif etiladi: har chorak boshida o‘quvchilar bilimini qisqa diagnostika orqali aniqlash; natijaga qarab vaqtinchalik moslashuvchan guruhlar tashkil etish; pedagoglar jamoasida o‘quvchilarning rivojlanishini muhokama qilish; elektron jurnal yoki maktab axborot tizimida davomat, baholar va topshiriqlar bajarilishini kuzatish; chorak yakunida boshlang‘ich ko‘rsatkich bilan taqqoslash. Guruhlar o‘quvchiga doimiy yorliq qo‘yish vositasi emas, balki ehtiyojga qarab o‘zgaradigan qo‘llab-quvvatlash mexanizmi bo‘lishi kerak.

Tavsiya etiladigan boshqaruv indikatorlari: (1) diagnostika va yakuniy baholash orasidagi o‘sish; (2) minimal kompetensiyaga erishgan o‘quvchilar ulushi; (3) dars qoldirish va topshiriq bajarmaslik dinamikasi; (4) pedagoglarning hamkorlikdagi rejalashtirishda ishtiroki; (5) ota-onalar bilan o‘quv natijalari bo‘yicha muloqot; (6) raqamli resurslardan pedagogik maqsadda foydalanish. Indikatorlar maktab sharoitiga mos, tekshiriladigan va jamoa tomonidan tushunarli bo‘lishi zarur.

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XULOSA

O‘zbekiston va Filippin tajribasi zamonaviy maktabni rivojlantirishda strategik maqsadlar, o‘quv dasturining dolzarbligi, pedagoglarni qo‘llab-quvvatlash, inklyuziv muhit va natijalarni tahlil qilish o‘zaro bog‘liq ekanini ko‘rsatadi. O‘zbekistonda belgilangan raqamlashtirish va o‘quv materiallarini yangilash maqsadlari maktab ichidagi pedagogik jarayon hamda o‘quvchi natijalari bilan bog‘langandagina kutilgan samarani berishi mumkin. Filippinning MATATAG va BEDP 2030 yondashuvlari esa asosiy kompetensiyalar, o‘quvchi farovonligi va o‘qituvchini qo‘llash masalalarini boshqaruvning markaziga qo‘yadi.

Amaliy jihatdan maktab rahbarlariga diagnostik baholashni muntazam yo‘lga qo‘yish, moslashuvchan o‘quv guruhlaridan foydalanish, pedagogik hamkorlikni kuchaytirish va raqamli ma’lumotlar asosida qaror qabul qilish tavsiya etiladi. Keyingi tadqiqotlarda ushbu mexanizmni bir nechta maktabda sinovdan o‘tkazib, o‘quvchilar natijalaridagi o‘zgarish, o‘qituvchi yuklamasi va ota-onalar bilan hamkorlik ko‘rsatkichlarini taqqoslash lozim.

SNOSKALAR / MANBALARGA IZOH

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Tahririy eslatma: UDK kodi mavzu mazmuniga mos dastlabki taklif sifatida berildi; yakuniy kodni jurnal kutubxonachisi yoki UDK mutaxassisi bilan tekshirish lozim. OAK talablari jurnalga qarab farqlanadi. Yuborishdan oldin jurnalning so‘nggi mualliflar yo‘riqnomasi, hajm, shrift, havola uslubi, plagiat va mualliflik deklaratsiyasini tekshiring.

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BUDGETING AS A CORE MANAGEMENT FUNCTION IN CONTEMPORARY ORGANIZATIONAL SYSTEMS

O‘rinboyeva O‘g‘iljon Sardorbek qizi
Student at Tashkent State University of Economics
E-mail: ogiloyorinboyeva54@gmail.com
Scientific supervisor: Yusupov Ulug‘bek Shukrullayevich
PhD, Associate Professor Department of Innovative Management
E-mail: Ulug’bekyusupov84@gmail.com

ANNOTATION

The present academic article examines the role of budgeting as a central and integrated management function within modern organisational systems. In the contemporary era of globalisation, macroeconomic instability, and digital transformation, the sustainable operation of enterprises and organisations is contingent upon the implementation of effective mechanisms for planning, allocating, and controlling financial resources. The primary objective of the research is to analyse, from scientific and practical perspectives, the interrelationship of budgeting systems with strategic planning, operational management, and internal control processes in modern enterprises. In addition, the development trends of national enterprises and the financial sector will be assessed based on data from the Statistics Agency of the Republic of Uzbekistan (Stat.uz).

The study employed a range of analytical techniques, including comparative analysis, logical generalisation, and the methods of statistical and systematic analysis of financial indicators. During the research, official data from Stat.uz and the open budget and financial indicators published by the Ministry of Economy and Finance of the Republic of Uzbekistan were analysed. The analyses demonstrated that the transition from conventional rigid approaches to flexible (Rolling Forecast and Beyond Budgeting) and digitised modelling methods in the budgeting process enhances enterprises' resilience to market risks. The dynamic growth observed in recent years in the volume of financial and commercial services for entities in Uzbekistan (for example, growth rates in financial services of over 20 per cent) necessitates the deep implementation of modern management accounting and budget control.

The research findings demonstrate that in modern enterprises and organisations, budgeting should be viewed not merely as a financial plan, but as an integral instrument for optimally allocating resources, motivating employees, and assessing corporate performance. The suggestions and recommendations presented in the article can serve

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as a practical resource for business leaders, financiers, and analysts in optimising the budgeting system.

Key words: Budgeting, management function, management control systems, strategic planning, financial management, rolling forecasts, beyond budgeting, corporate governance, resource allocation, stat.uz data.

INTRODUCTION:

In contemporary organisational systems, the efficacy of management is contingent upon the judicious utilisation of resources and the precise anticipation of future risks. In a market economy, enterprises are confronted with macroeconomic instability, inflationary pressures, supply chain disruptions, and technological changes, which necessitate the constant improvement of financial planning and control tools. In this context, budgeting can be regarded not merely as a technical task for the accounting department within the management system, but rather as a central management function that translates an organisation's strategic objectives into practical operations.

In classical management theory, as developed by Henri Fayol and his contemporaries, the functions of management are delineated, with particular emphasis on planning. In contrast, in modern corporate management, the functions of planning, organising, leading and controlling are distinguished. However, in contemporary practice, budgeting serves as an integrative platform that unites all four of these functions. In the planning stage, it delineates the organisation's goals and objectives in quantitative (monetary) terms; in the organising stage, it allocates resources across departments and projects; in the leadership and coordination stage, it aligns the efforts of staff around a single corporate objective; and in the control phase, it enables actual performance to be compared with planned norms, identifying variances and allowing for corrective action.

Recent years have seen the implementation of large-scale economic reforms in the Republic of Uzbekistan. These reforms, which include the transformation of state-owned enterprises and the encouragement of private sector activity, have necessitated a review of financial management and management accounting in enterprises. This review must be based on modern standards. According to data from the Statistics Agency under the President of the Republic of Uzbekistan (Stat.uz), there has been a rapid growth in the volume of financial turnover in the country's service and industrial production sectors. The expansion of managed resources and financial flows necessitates a scientific and practical budgetary model for the management of enterprises, necessitating the passing of every expenditure and investment decision through this model.

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However, the budgeting process is beset by a number of challenges in the practice of many local and international organisations. Conventional (static) budgeting methods are slow to adapt to rapid market changes, requiring a great deal of time and resources and sometimes encouraging employees to simply "spend" the budget, leading to objections from many academics and practitioners. Consequently, contemporary academic literature and professional practice are witnessing the emergence of novel approaches such as "Beyond Budgeting" and "Rolling Forecasts".

The objective of this article is twofold: firstly, to analyse the role of budgeting in the system of modern organisations from both theoretical and practical standpoints; secondly, to define its scope as a management function; and thirdly, to examine the macro- and microeconomic statistical indicators in Uzbekistan. The objective of this study is to provide scientific conclusions on the optimisation of the financial planning system (Stat.uz data). The following research questions are posited in the article:

1. The present study sets out to explore the manner in which budgeting in modern enterprises integrates strategic and operational management.
2. A critical evaluation of the merits and drawbacks of both traditional and modern budgeting models is therefore essential.
3. In the context of the development dynamics of financial and economic entities in Uzbekistan, what practical opportunities exist for enhancing the effectiveness of budgeting?

LITERATURE REVIEW

The subject of budgeting and its role in corporate governance has been the focus of extensive research in the disciplines of economics, management accounting and corporate finance for a considerable number of years. An analysis of the perspectives of classical and modern researchers has been shown to facilitate a more profound comprehension of the evolution of budgeting.

The fundamental principles of budgeting were initially developed in the first half of the 20th century within large industrial enterprises (e.g., DuPont, General Motors) as a means of controlling costs and allocating resources. In his seminal work, Anthony (1965) distinguished three levels of organisational control systems: strategic planning, management control, and operational control. According to Anthony, budgeting constitutes the central pillar of management control, as it links strategic objectives to day-to-day operational tasks.

In their research on management control systems, Merchant and Van der Stede (2017) identify four key functions of budgeting:

- **Planning and resource allocation**: setting objectives and the means to achieve them for the coming period.

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- **Coordination and communication:** coordinating activities between the organisation's various departments (production, sales, marketing, HR).
- **Motivation and evaluation:** setting Key Performance Indicators (KPIs) for managers and employees and incentivising their achievement.
- **Control and monitoring:** comparing actual results with the budget and making corrective decisions.

However, towards the close of the 20th century and the advent of the 21st, criticism of the conventional budgeting system increased considerably. In their seminal "Beyond Budgeting" model, Hope and Fraser (2003) castigated traditional annual budgets, denoting them as a "bureaucratic, slow, inflexible, and manipulative" mechanism. It is contended by the aforementioned parties that, within the contemporary context of a rapidly evolving market environment, a budget that is static and set 12 months in advance rapidly loses its value. The authors have proposed rolling forecasts and decentralised management structures as an alternative to traditional budgeting.

As demonstrated by Libby and Lindsay (2010), empirical research conducted in North American companies has shown that, despite the valid criticisms levelled at Beyond Budgeting, the majority of companies have not completely abandoned the traditional budget, but have instead hybridised it. It is evident that contemporary companies are implementing Rolling Forecasts mechanisms for the purpose of forecasting, while concurrently maintaining the budget for control and resource allocation.

Otley (1999) and Drury (2018) posit that the efficacy of budgeting is contingent upon the environmental uncertainty in which an organisation operates. Alternative approaches, of which Zero-Based Budgeting (ZBB) is an example (Pyhrr, 1970), require that each expense item be justified anew on an annual basis. ZBB has been demonstrated to be a highly efficacious instrument in the optimisation of expenditure and the reduction of wasteful costs. However, it should be noted that its implementation does necessitate a significant investment of both labour and time.

The organisational and economic characteristics of budgeting have also been extensively studied in the scientific works of scholars from the Commonwealth of Independent States (CIS) and local scholars. In their research on improving management accounting and financial planning in enterprises, Uzbek economists (for example, A.V. Vahobov, A.A. Isadjanov, M.Q. Pardaev) have highlighted the necessity of automating budget control and integrating it with International Financial Reporting

Standards (IFRS) and modern Enterprise Resource Planning (ERP) systems to ensure the financial stability of enterprises in the national context.

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A review of the literature indicates that contemporary research is evolving towards transforming budgeting from a static financial document into a dynamic and digitised management platform.

METHODS AND MATERIALS

In this study, a combination of hypothetical-deductive and empirical analysis methods was employed to analyse the management function of budgeting within modern organisational systems. The research methodology comprises several stages:

- **Theoretical and methodological analysis:** A comparative analysis was conducted on monographs, scientific articles and concepts by international and local scholars in the fields of modern management accounting, corporate finance and strategic management.
- **Collection of empirical and statistical data:** The official database of the Agency for Statistics under the President of the Republic of Uzbekistan (Stat.uz), together with the Official statistical bulletins and data from the 'Budget Message', published by the Institute of Macroeconomic and Regional Studies and the Ministry of Economy and Finance of the Republic of Uzbekistan, formed the practical basis of the research.
- **Statistical and comparative analysis:** The present study draws upon a comprehensive set of statistical data in order to analyse the dynamics of financial and commercial services in Uzbekistan. In addition, the macroeconomic and corporate indicators of the country have been examined, and the results of this analysis are summarised in tables. Finally, percentage growth rates and structural shifts have been calculated.

Analysis of statistical data and tables

The effectiveness of budgeting in enterprises and organisations is directly related to the macroeconomic and sectoral financial environment. The expansion of the service and financial sectors in Uzbekistan has led to an increased need for financial flow planning and budget control within enterprises.

The following table (1) presents an overview of the volume of financial services in the Republic of Uzbekistan, along with an analysis of its structural dynamics. The data presented has been collated from official sources at Stat.uz.

Table 1. The following report details the volume and structure of financial services in the Republic of Uzbekistan from January to August 2024 to 2026.

Ko‘rsatkichlar	2024-yil	2025-yil	2026-yil (yanvar-avgust)	O‘sish sur’ati (2026/2025 y.y. %da

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Jami moliyaviy xizmatlar hajmi (trln so‘m)	**95,4**	**118,1**	**144,7**	
Tarkibi (%da):				
Sug‘urtalash va pensiyadan tashqari moliyaviy xizmatlar	86,2%	85,9%	85,7%	-
Sug‘urtalash xizmatlari	6,8%	6,9%	7,0%	-
Yordamchi moliyaviy xizmatlar	7,0%	7,2%	7,3%	-

**Source: The author's own work is based on official data from the National Statistics Committee of the Republic of Uzbekistan (Stat.uz).*

The following analysis is drawn from the data presented in Table 1. Stat.uz data indicates that the volume of financial services in Uzbekistan amounted to 144.7 trillion soums during the January–August period of 2026, representing a rapid increase of 22.5 per cent compared to the same period last year. Despite the preeminence of banking and core credit-financial operations within the financial services sector, accounting for 85.7% of the total, the proportion of auxiliary financial and insurance services is concurrently exhibiting a steady rise. The substantial increase in financial transactions and credit investment resources underscores the imperative for enterprises to manage these resources effectively, particularly by enhancing their budgeting systems.

As illustrated in Table 2, the dynamics of budget revenues have a significant impact on the activities of the state, consolidated and corporate sectors.

Table 2. The following essay will examine the dynamics of the Republic of Uzbekistan's state budget revenues for the 2023–2024 year-end results.

Ko‘rsatkich ko‘rsatkichlari	**2023-yil**	**2024-yil**	**Farq (absolyut, trln so‘m)**	**O‘sish sur’ati (%da)**
Davlat byudjeti daromadlari (trln so‘m)	**231,72**	**274,42**	**+42,70**	**+18,4%**

**Source: The author's own research is based on data from Stat.uz and the Budget message of the Ministry of Economy and Finance of the Republic of Uzbekistan.*

The following analysis is drawn from the second table. As of the close of 2024, state budget revenues totalled 274.42 trillion soums, marking an increase of 42.7 trillion soums, or 18.4 per cent, in comparison with 2023. This indicator is indicative of the country's overall economic activity and the expansion of the corporate tax base. In the context of economic dynamics at the enterprise level, the implementation of "Tax and

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Cost Budgeting" is imperative for the accurate planning of tax liabilities and production costs.

CONCLUSION

The present study has theoretically and practically demonstrated that, in modern organisations and enterprises, budgeting is not merely a calculation tool, but a central management function for the effective operation of the organisation. The company's management employs a budgetary process that translates strategic objectives into quantifiable operational indicators, while also orchestrating the allocation of resources across departments. The staff incentive system is then linked to actual results, and internal controls are established to mitigate potential risks.

Analyses conducted based on data from the National Statistics Committee of the Republic of Uzbekistan (Stat.uz) demonstrate that the country's financial services sector is expanding rapidly (reaching 144.7 trillion soums in 2026, grew by 22.5%) and the turnover of total economic resources is increasing significantly. In such a dynamic environment, traditional, rigid, and static budgeting models that rely solely on historical data are no longer adequate to meet the organic demands of the market. Enterprises and organisations must transition to modern budgeting technologies that are flexible in response to market changes.

In light of the research findings, the following practical recommendations are proposed for enhancing the efficacy of budgeting within contemporary organisational systems:

1. The following essay will explore the concept of hybrid and flexible budgeting models, with a particular focus on rolling forecasts. Enterprises are advised to implement rolling forecasts in conjunction with a fixed annual budget. This enables the expeditious realignment of financial plans in the face of market volatility.
2. Automating Management Accounting (ERP and AI Integration): In order to reduce human error and optimise the time taken for the budgeting process, it is essential that modern ERP and cloud-based automated systems such as 1C, SAP, and Oracle are adopted more widely.
3. The utilisation of zero-based budgeting (ZBB) is a proven method of effective management. In order to enhance cost efficiency and reduce redundant, ineffective expenditure items, it is advisable to periodically conduct a zero-based budgeting (ZBB) exercise for operational costs.
4. Strategy and KPI Integration: It is imperative that budget metrics are organically linked to the organisation's Balanced Scorecard (BSC) and the staff's Key Performance Indicator (KPI) system.

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In conclusion, the modernisation of the budgeting system is imperative for enterprises and organisations to enhance their competitiveness, ensure their financial stability, and achieve long-term strategic development.

In the future, the widespread application of artificial intelligence (AI) and Big Data technologies in our national economy will take budgeting processes to a more precise and automated stage. This, in turn, will enable managers to simulate risks in advance (scenario modelling) and adapt instantly to market fluctuations.

Moreover, the integration of ESG (Environmental, Social, and Governance) principles into corporate governance will signify a shift in the future, wherein the budgeting function will no longer be confined exclusively to financial performance metrics. Instead, it will evolve into a more comprehensive, sustainable management tool that encompasses the costs associated with environmental sustainability and social responsibility.

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THE IMPORTANCE OF COMMUNICATION IN MANAGEMENT AN INTEGRATIVE CONCEPTUAL FRAMEWORK FOR COMMUNICATION EFFECTIVENESS IN MULTINATIONAL ORGANISATIONS

Yusupov Ulug‘bek Shukrullayevich
TSUE, PhD, Associate Professor Department of Innovative Management.
E-mail: ulug’bekyusupov84@gmailcom.
Saydullaxonova Zebinisobegim Shukurullaxon qizi
3rd-year student, Tax and taxation Tashkent State university of Economics(TSUE)
E-mail: saydullaxonovazebinisobegim@gmail.com.

Abstract: Communication is a core managerial process through which goals are interpreted, knowledge is shared, decisions are coordinated, and working relationships are maintained. These functions become more complex in multinational organisations, where employees may differ in language proficiency, cultural expectations, professional norms, time zones, and access to communication technologies. This conceptual article develops an integrative framework that explains how these contextual conditions interact with managerial communication practices to shape team outcomes. Drawing on organisational sensemaking, intercultural communication, media richness theory, social exchange and organisational trust, psychological safety, and leadership communication, the article argues that communication effectiveness is not a direct consequence of cultural similarity or technology choice alone. Rather, it emerges when messages are understandable, channels fit the task and the participants, leaders invite contribution, and team members can clarify meaning without fear of interpersonal penalty. Trust, psychological safety, and shared sensemaking are proposed as related but distinct mechanisms linking communication practices to coordination, knowledge sharing, learning, and performance. The article also identifies limits in national-culture frameworks and fixed media hierarchies, and draws on recent research on global virtual teams and digital communication. Six propositions are presented for future empirical testing. Because the manuscript is a conceptual synthesis rather than a systematic review or an empirical study, it does not claim exhaustive coverage or report original data. Its contribution is a clearly bounded, testable framework for international management research and practice.

Keywords: managerial communication; multinational organisations; intercultural communication; global virtual teams; trust; psychological safety; media choice; leadership

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Communication is more than the transmission of information from one organisational member to another. It is also how people interpret priorities, negotiate responsibilities, build shared understandings, and judge whether it is safe to raise concerns. Barnard (1938)[1] treated communication as a central function of executive coordination, while Weick (1979)[2] emphasised that organising depends on continuing processes of interpretation and sensemaking. On this view, the quality of communication affects not only whether information is received but also whether people give it sufficiently compatible meanings to act together.

These issues carry particular weight in multinational enterprises, where cross-border teams often coordinate work across languages, time zones, professional systems, and cultural expectations. A manager's concise message may strike one employee as efficient and another as abrupt, and silence may signal agreement, deference, uncertainty, or disagreement depending on the situation. Digital channels add further choices. A written message creates a record and gives recipients time to formulate a response, whereas a live meeting may allow faster clarification but can disadvantage participants who are less fluent in the working language or who have limited opportunities to speak.

Research on global virtual teams has consistently identified communication, trust, leadership, culture, and technology as interconnected themes rather than isolated variables. Lynden's (2024)[3] meta-review of 35 reviews describes a shift toward viewing virtuality as a continuum and points to the continuing relevance of trust, leadership, culture, and communication technology. A communication-focused review by Liu (2025),[4] based on 51 empirical studies, similarly examines the interplay between communication, culture, tools, and human factors in global virtual teams. Empirical work also suggests that the alignment of communication channels within a team can matter for sociability and coordination, and not merely whether a team works virtually (Wong et al., 2024).[5]

Despite this accumulated knowledge, a practical and theoretical challenge remains. Cultural frameworks, channel-selection theories, leadership research, trust models, and psychological-safety research often operate at different levels of analysis, and they can

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2. [2] Weick, K. E. (1979). The social psychology of organizing (2nd ed.). Addison-Wesley.
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be combined too loosely, as if national culture, an individual's language skill, a team's psychological safety, and an organisation's technology policy were interchangeable explanations. This article addresses the problem by specifying how the constructs relate and by distinguishing contextual conditions from communication practices, mechanisms, and outcomes.

1.1. Aim and research questions

The aim is to develop a conceptually integrated framework of managerial communication effectiveness in multinational organisations, with particular attention to multicultural and global virtual teams. The article addresses three questions:

Through which mechanisms does managerial communication influence coordination, knowledge sharing, learning, and team performance?

How do cultural expectations, language, channel design, and leadership practices interact to shape communication effectiveness?

What testable propositions and practical implications follow when foundational theories are combined critically rather than treated as independent explanations?

1.2. Scope and contribution

The term “multinational organisations” refers here to multinational enterprises and their cross-border units and teams, not to intergovernmental bodies. The article is conceptual. It synthesises selected foundational theories and recent publications to propose relationships that can be tested in future research, and it does not claim to be a systematic review. Such a review would require a reproducible database search, explicit eligibility criteria, screening records, and a documented synthesis procedure; those steps have not been carried out for this draft.

The contribution is threefold. First, the article distinguishes the contextual conditions of communication from the practices that managers can influence. Second, it specifies trust, psychological safety, and shared sensemaking as related but non-identical mechanisms. Third, it proposes boundary conditions, including task ambiguity, language asymmetry, status differences, and the degree of virtuality, instead of assuming that any one communication style or channel is universally effective.

2. Conceptual foundations

2.1. Organisational communication and shared sensemaking

Organisational communication is constitutive of coordination: it helps establish goals, clarify roles, make decisions actionable, and maintain feedback loops. Weick's (1979)[6] sensemaking perspective is useful for international management because employees may receive the same message yet interpret it through different professional, linguistic, or situational frames. Communication effectiveness should

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therefore be judged partly by whether teams can surface and resolve divergent interpretations, and not only by the speed or volume of messages.

Shared sensemaking does not require identical viewpoints, only sufficient alignment about the task, the rationale for decisions, and the next actions. Managers can support it by stating assumptions explicitly, checking understanding, summarising decisions, and inviting team members to identify alternative readings. These practices matter most when a message concerns change, conflict, risk, or interdependent work.

2.2. Culture: useful context, not a fixed explanation

Hofstede's cultural-dimensions framework (Hofstede, 1980)[7] and Hall's distinction between high- and low-context communication (Hall, 1976)[8] remain influential starting points for thinking about differences in hierarchy, directness, uncertainty, and the use of contextual cues. For example, employees may differ in how comfortable they feel challenging a senior manager in a group meeting or how explicitly they expect a request to be worded. Such frameworks can help managers ask about expectations that might otherwise remain unspoken.

However, national categories should not be treated as reliable descriptions of every individual. McSweeney (2002)[9] challenged the assumptions and inferences used in applications of Hofstede's model, while later discussion of Hall's high-/low-context distinction has highlighted the need to examine how the concept is used and operationalised (Kittler et al., 2011).[10] Within-country differences, organisational cultures, occupational identities, generation, personality, migration experience, and the immediate situation can all shape communication. National-culture scores are aggregate-level indicators and should not be assigned to individual employees as if they were personal traits.

This framework therefore treats culture as a contextual moderator: it may influence how communication behaviours are interpreted, but it does not determine those interpretations. A manager should use cultural frameworks to develop hypotheses and ask respectful questions rather than to stereotype team members or predict their behaviour without evidence.

2.3. Language and communication accommodation

Language proficiency affects how easily employees can understand instructions, express uncertainty, interpret humour, and participate spontaneously. In multinational

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firms, a shared corporate language can reduce some coordination costs but may also create unequal participation when employees differ in fluency. Communication Accommodation Theory proposes that speakers may adjust their communication toward or away from their interlocutors, with consequences for comprehension, identity, and relationships (Giles et al., 1991; Giles, 2016).[11]

In practice, accommodation can include avoiding unnecessary idioms, explaining acronyms, slowing speech without being patronising, checking comprehension, and offering more than one way to contribute. It should not, however, mean expecting only less-fluent employees to adapt: managers and fluent speakers also share responsibility for making communication accessible. Nor is convergence always beneficial, since over-simplification can feel disrespectful and imitation of an accent may undermine trust. The relevant goal is mutual intelligibility and inclusion, not uniformity.

2.4. Choosing and configuring communication channels

Media Richness Theory argues that communication media differ in their capacity to convey cues, provide rapid feedback, and support clarification; richer media may be useful when a task is ambiguous or open to multiple interpretations (Daft & Lengel, 1986).[12] This offers a starting point for deciding whether a manager should use a live conversation, video meeting, email, or collaborative document. Sensitive feedback or conflict may call for dialogue and immediate clarification, whereas a routine update or formal decision may be better served by concise written documentation.

Even so, no channel is universally best. Live video can provide facial and vocal cues but can increase fatigue, expose differences in bandwidth, and make turn-taking difficult. Asynchronous text gives participants time to reflect and supports traceability, but it can delay clarification and strip away tone. Research on virtual-team communication indicates that the alignment of communication configurations among members can shape team sociability and coordination (Wong et al., 2024).[13] The relevant design question is therefore not simply whether a medium is rich, but whether the team uses it consistently and appropriately for the task, the language needs of participants, and the desired record of the decision.

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Recent reviews stress the need to consider technology together with relational and organisational factors. Swart et al. (2022) [14] identify trust, cultural diversity, collaboration tools, leadership, psychological safety, communication guidelines, and resource planning among recurring themes in virtual project teams. This suggests that technology policy belongs to the team's communication system and is not a stand-alone technical decision.

2.5. Trust as a relational mechanism

Trust is the willingness to accept vulnerability based on positive expectations about another party. Mayer, Davis, and Schoorman (1995)[15] distinguish perceived ability, benevolence, and integrity as important bases of trustworthiness. Social exchange theory also emphasises reciprocity and the development of relationships through repeated interactions (Blau, 1964; Cropanzano & Mitchell, 2005).[16] In managerial communication, trust may be strengthened when leaders explain decisions, follow through on commitments, acknowledge uncertainty honestly, and respond consistently to questions.

Trust should not be collapsed into communication quality itself. Communication is a set of practices and interactions; trust is a relational expectation that may be shaped by those interactions, prior experience, formal systems, and the wider institutional context. Trust may also influence later communication, since employees who trust a manager may be more willing to share incomplete information or raise a problem early. The relationship is therefore likely to be reciprocal over time rather than a simple one-way causal chain. Recent reviews continue to treat trust as an evolving research domain with multiple explanatory approaches (Dirks & de Jong, 2022).[17]

2.6. Psychological safety and speaking up

Psychological safety is a shared belief that a team is safe for interpersonal risk-taking, such as asking a question, admitting an error, or disagreeing with a proposed course of action (Edmondson, 1999).[18] It is not the same as comfort, agreement, or trust in a single individual. A team may have positive interpersonal relationships yet still

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discourage dissent, or an employee may trust a manager's competence but fear being embarrassed in front of colleagues.

Managerial communication can support psychological safety when leaders invite dissent, respond to mistakes with curiosity before blame, explain how feedback will be used, and distribute speaking opportunities. In multinational teams, these behaviours matter where language asymmetry or status differences make speaking up costly. Evidence from global virtual teams also links early relational communication to the emergence of a psychologically safe communication climate and team performance (Glikson & Erez, 2020).[19]

Psychological safety does not mean lowering standards or avoiding accountability. Its value lies in enabling people to disclose relevant information early enough for the team to learn and correct course. Managers should pair openness with clear responsibilities, evidence-based decisions, and follow-through.

2.7. Leadership communication as an integrating practice

Transformational leadership emphasises articulating a meaningful direction, encouraging new ways of thinking, and attending to individual development (Bass, 1985; Burns, 1978).[20] These ideas are relevant to cross-border management because a shared purpose can help employees coordinate despite differences in local routines. However, inspirational language alone is not enough: leaders also need to translate broad goals into clear priorities, explain the reasons for decisions, invite questions, and make sure that their commitments match their actions.

Inclusive leadership communication links the other elements of the framework. It can improve channel selection by asking which format works for the task, support accommodation by making language expectations explicit, strengthen trust through consistency, and promote psychological safety by legitimising questions and disagreement. Leadership is thus treated here not as a separate personality trait that automatically produces good communication, but as a set of observable practices that shape the communication environment.

3. An integrative framework

The proposed framework contains four analytically distinct layers. The first is the context in which communication takes place: cultural and professional expectations, language proficiency, time-zone distribution, task ambiguity, status relations, and the degree of virtuality. Managerial communication practices form the second layer, namely clarity, timely feedback, channel–task fit, explicit checking of understanding,

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accommodation, transparency, and inclusive participation. The third layer consists of the mechanisms through which communication may influence team behaviour: shared sensemaking, trust, and psychological safety. The fourth is the outcome layer, which covers coordination, knowledge sharing, learning, error detection, and team performance.

Layer	Core elements	Function in the framework
Context	Culture; language; status; task ambiguity; time zones; degree of virtuality	Shapes how messages are produced, received, and interpreted
Communication practices	Clarity; feedback; channel fit; accommodation; transparency; inclusive turn-taking	Managerially influenceable behaviours
Mechanisms	Shared sensemaking; trust; psychological safety	Explain how communication can affect willingness and ability to coordinate
Outcomes	Coordination; knowledge sharing; learning; error detection; performance	Near-term and downstream consequences

The framework does not assume that each context factor has an independent, additive effect. For example, language asymmetry may be especially consequential when a high-stakes task is discussed in a fast-moving video meeting without written follow-up. By contrast, an asynchronous document with clear definitions and a structured comment period may give less-fluent participants more time to formulate questions. Similarly, a manager's invitation to speak may not create psychological safety if earlier contributions were punished. Context, practice, and history therefore interact.

The three mechanisms must also remain distinct. Shared sensemaking is about whether team members develop sufficiently compatible interpretations of tasks and decisions. Trust involves expectations about another party's competence, intentions, and reliability, while psychological safety concerns whether interpersonal risk-taking is perceived as safe in the team. The mechanisms may reinforce one another, but none can serve as a proxy for the others.

4. Propositions for future research

Proposition 1 – Communication and sensemaking. Greater message clarity, explicit confirmation of understanding, and timely feedback will be positively associated with

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shared sensemaking in multinational teams, particularly when tasks are ambiguous or interdependent.

Proposition 2 - Channel–task–participant fit. The relationship between communication-channel choice and team coordination will depend on the fit among task ambiguity, channel affordances, language needs, and team members' shared communication norms; no single channel will be optimal across all tasks.

Proposition 3 - Trust as a mechanism. Transparent, consistent, and responsive managerial communication will be positively associated with team coordination and knowledge sharing partly through its association with trust.

Proposition 4 - Psychological safety and voice. Inclusive leader communication will be positively associated with employee voice and error reporting partly through psychological safety; this relationship will be weaker where status penalties for speaking up remain high.

Proposition 5 – Contextual moderation. The effects of communication practices on sensemaking, trust, and psychological safety will vary with language asymmetry, cultural expectations, time-zone dispersion, and degree of virtuality, but these factors will not deterministically predict individual behaviour.

Proposition 6 – Alignment in digital communication. Teams with shared and explicit norms for selecting, using, and documenting digital channels will demonstrate better coordination than otherwise comparable teams whose channel practices are fragmented or inconsistent.

These propositions are conceptual deductions, not findings from new data. They can be tested through multi-level survey designs, longitudinal studies of team formation, experiments on channel configuration, or mixed-method research that combines communication logs with interviews and team outcomes. Research should avoid inferring individual behaviour directly from national averages and should distinguish individual, dyadic, team, and organisational levels of analysis.

5. Implications for international management

5.1. Make meaning explicit without over-standardising

Managers should state the purpose of a message, the action required, the responsible person, the deadline, and any uncertainty that remains. After consequential meetings, a concise written summary can record decisions and unresolved questions. These practices reduce avoidable ambiguity while leaving room for local teams to adapt implementation to their context.

5.2. Establish channel norms

Teams should agree which channels are used for urgent issues, routine updates, sensitive feedback, brainstorming, and formal decisions. Norms should cover expected

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response times, meeting participation, documentation, and how to handle time-zone constraints. The aim is not to maximise synchronous meetings but to make channel choice understandable and consistent for all members.

5.3. Design for language inclusion

Use plain professional language, explain acronyms, share agendas in advance, and allow participants to contribute in writing before or after meetings. Leaders should monitor whether the same fluent or senior members dominate discussion. Written and asynchronous options can complement live discussion, especially when the issue is complex or participants need time to process information.

5.4. Build trust through reliability

Trust is more likely to develop when managers keep commitments, explain changes, acknowledge mistakes, and apply standards consistently. Where face-to-face contact is limited, predictable communication matters even more. Managers should avoid interpreting delayed responses automatically as disinterest; time zones, workload, access, and differing norms may provide alternative explanations.

5.5. Make speaking up safe and consequential

Leaders can invite dissent by asking what may be missing, requesting a risk review, and thanking employees who identify problems. They should also close the feedback loop by explaining what was changed, what was not changed, and why. An invitation to speak is not credible if contributions are ignored or punished.

5.6. Assess communication outcomes, not message volume

The number of emails, meetings, or messages is a poor proxy for communication effectiveness. Organisations can instead assess decision clarity, rework caused by misunderstanding, response reliability, participation balance, time taken to resolve ambiguity, knowledge-sharing behaviour, and employees' perceived ability to raise concerns. Measures should be adapted to the task and interpreted alongside qualitative evidence.

6. Limitations and future research

This article has several limitations. First, it is a conceptual synthesis of selected foundational and recent sources, not a systematic or exhaustive review. A formal review should search databases such as Scopus and Web of Science using a preregistered or otherwise documented protocol, specify date and language limits, screen records against eligibility criteria, and maintain a search log. The current draft must not be described as having followed such a procedure.

Second, the proposed framework has not been empirically validated. The propositions are intended to guide research, not to imply that causal effects have already been demonstrated. Much of the foundational work was developed in particular

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historical and national settings. New studies should examine whether the relationships hold across industries, regions, employment arrangements, and levels of organisational hierarchy.

Third, the constructs can overlap if they are not carefully operationalised. Communication clarity, shared sensemaking, trust, and psychological safety may be correlated, but each requires a distinct definition and measure. Researchers should test alternative models, consider reciprocal relationships over time, and account for possible confounds such as task interdependence, team tenure, power distribution, and prior performance.

Fourth, digital communication technologies continue to change. The relevant unit of analysis may not be a single medium but the team's configuration of synchronous and asynchronous tools, documentation practices, and expectations about availability. Recent work on virtuality as a continuum (Lynden, 2024)[21] and on communication configurations (Wong et al., 2024)[22] supports examining how channels are combined rather than ranking them in a fixed hierarchy.

Future research could use longitudinal, multi-level designs to examine how communication practices shape trust and psychological safety during team formation and change. Comparative research could test whether language asymmetry or status differences moderate the effects of inclusive leadership. Field experiments might compare different combinations of meeting discussion, written summaries, and asynchronous input. Such designs would provide a stronger basis for practical recommendations than broad assumptions about national communication styles.

7. Conclusion

Effective communication is a central managerial capability in multinational organisations because it enables people to interpret goals, coordinate interdependent work, share knowledge, and raise concerns. Yet communication effectiveness cannot be reduced to a single cultural dimension, a preferred leadership style, or the richness of a technology. It emerges from the fit between context, communication practices, and the mechanisms through which people make sense of one another and their work.

The framework developed here places shared sensemaking, trust, and psychological safety at the centre of the relationship between managerial communication and team outcomes. It treats culture and language as important conditions without using them as deterministic labels, and it views digital channels as configurations that must fit tasks

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and participants. The six propositions provide a basis for empirical testing and for a more specific discussion of communication practices in international management. Before journal submission, the co-authors should complete a documented literature search, verify every bibliographic record against publisher or DOI metadata, and adapt the manuscript to the target journal's scope and requirements.

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