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# MICROPLASTIC CONTAMINATION IN INTRAVENOUS FLUIDS AND ENTERAL FEEDING SYSTEMS

## Implications for Patient Safety in Intensive Care Units

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## Introduction

Picture an intensive care unit at three in the morning. A patient on mechanical ventilation receives a balanced crystalloid through a central line, a vasopressor through a pump, an antibiotic through a piggyback set, and enteral formula through a nasogastric tube. Every one of these lifelines travels in plastic: bags, tubing, connectors, syringes, stopcocks, and feeding sets. Clinicians trust these materials because they are sterile, flexible, light, and inexpensive. Few of us stop to ask what the materials themselves might add to the patient.

That question has become harder to ignore. Over the past decade, microplastics, usually defined as plastic particles smaller than five millimetres, and their smaller relatives, nanoplastics, have been found almost everywhere researchers have looked: oceans, drinking water, food, air, and increasingly the human body. Particles have been reported in blood, placenta, lung tissue, and arterial plaque. If plastic particles can reach human tissues through ordinary life, it is reasonable to ask what happens when plastic devices deliver fluids and nutrition directly into the bloodstream or gut of a patient whose defences are already compromised.

This article takes a narrative approach. It does not claim that microplastics from ICU equipment have been proven to harm patients; that evidence does not yet exist. Instead, it pulls together what is known about particle exposure in hospital infusion and feeding systems, explains why critically ill patients may be uniquely vulnerable, identifies the gaps in our knowledge, and suggests sensible steps that units can take now without waiting for certainty.

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**What we mean by microplastics in a clinical setting**

Microplastics are not a single substance. They are fragments, fibres, and beads of many polymers, including polyvinyl chloride (PVC), polyethylene, polypropylene, polyurethane, and polystyrene, along with the additives that give plastics their properties. Some are manufactured at small size; others arise when larger items degrade through heat, friction, ultraviolet light, or chemical exposure. Nanoplastics, below one micrometre, are the least understood and the hardest to measure, yet they are the most likely to cross biological barriers.

Clinicians already know a related concept under a different name: particulate matter in injectable products. Pharmacopeial standards, such as the United States Pharmacopeia chapter on particulate matter in injections, set limits on the number of visible and subvisible particles allowed in infusion products. For large-volume infusions, the limits are expressed as particles per millilitre at sizes of 10 micrometres and above and 25 micrometres and above. These limits were designed around particles that could occlude capillaries or provoke inflammation, and they rely on methods that count particles without necessarily identifying what they are made of. Plastic fragments may therefore be counted among “particles” without being recognised as polymeric material, and particles below the counting thresholds are not captured at all. In other words, a product can meet its release specification and still carry a population of small plastic particles that nobody was looking for.

**The intravenous pathway: from bag to bloodstream**

An intravenous infusion passes through several plastic interfaces before it reaches the patient. The container is commonly a flexible bag made of polyolefin or PVC. The administration set often contains PVC tubing, a drip chamber, roller clamps, injection ports, and, in pump systems, a tubing segment that is repeatedly compressed. Additional devices such as extension sets, three-way stopcocks, and needleless connectors add more surfaces. Each interface is a potential source of particles, and the cumulative contact area in a complex ICU setup can be large.

Several mechanisms are plausible. Polymer surfaces can shed material when they are flexed, compressed, or abraded, which happens constantly in peristaltic pump segments. Storage, heat, and long contact times can encourage leaching of both particles and chemical additives. PVC tubing is historically softened with plasticisers such as di(2-ethylhexyl) phthalate, and concern about phthalate migration into lipid-containing and drug solutions has already pushed many hospitals toward alternative materials for neonates and for certain medications. Particles may also originate upstream, from rubber vial stoppers, syringe components, and the repeated puncturing of closures during drug preparation, which can cut small fragments, a phenomenon known as coring.

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The critical care environment amplifies the exposure. A patient with septic shock may receive many hours of continuous infusion through multiple lines, along with frequent boluses, blood products, parenteral nutrition, and sedatives. Cumulative exposure over days to weeks is likely to exceed that of any ward patient. The unusual feature of the intravenous route is that it bypasses the natural barriers of the gut and lung. A particle delivered into a vein goes first to the right heart and the pulmonary circulation, where the capillary bed acts as a filter, then to the liver, spleen, and other organs of the reticuloendothelial system.

There is some clinical evidence that removing particles matters. In a randomised controlled trial in a paediatric intensive care unit, in-line filtration of intravenous fluids was associated with fewer severe complications and a shorter stay in intensive care compared with unfiltered infusions (Jack et al., 2012). The trial was not designed to measure plastic specifically, and the particles being filtered included glass, precipitates, and fibres as well as polymers. Still, it supports the biological plausibility that sub-visible particles are not inert for critically ill patients, and it hints that a simple device could reduce the burden.

**The enteral pathway: feeding through plastic**

Enteral nutrition is the preferred route of feeding in most critically ill patients, and its safety profile has tended to be regarded as more forgiving than the intravenous route. Formula is delivered through a tube made of polyurethane, silicone, or PVC, sometimes for weeks, from a container or bag that hangs at the bedside at room temperature. Unlike intravenous infusions, enteral products are not required to meet injectable-grade particulate limits, because the gut is assumed to tolerate particles that the bloodstream would not.

That assumption deserves scrutiny. Laboratory work on infant formula preparation has shown that polypropylene feeding bottles can release very large numbers of microplastic particles when they are exposed to hot liquid and agitation (Li et al., 2020). Although that research concerned household bottles rather than ICU systems, it illustrates a general principle: heat, mechanical stress, and time all increase polymer shedding. In an ICU, formula may be warmed, shaken, decanted, mixed with powder in reusable containers, or administered with powdered medications crushed and flushed through the same tube. Each step creates opportunities for particles to enter the stream.

The gut is not an impenetrable barrier, particularly in critical illness. Sepsis, shock, mechanical ventilation, vasopressors, and prolonged fasting can all increase intestinal permeability. A damaged mucosa may allow uptake of smaller particles, which then enter the portal circulation or lymphatics. In addition, the intestinal microbiome of ICU patients is often profoundly disturbed by antibiotics and illness, and there is early laboratory interest in whether plastic particles can interact with

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microbial communities and the mucus layer. These ideas remain largely hypotheses, but they are biologically coherent and worth testing.

**What happens when particles enter the body**

The human data on microplastics are still emerging, and they are best read with caution. Particles have been detected in human blood samples (Leslie et al., 2022) and in placental tissue (Ragusa et al., 2021). In a notable study published in the New England Journal of Medicine, patients undergoing carotid endarterectomy whose plaques contained detectable microplastics and nanoplastics had a higher combined rate of myocardial infarction, stroke, or death from any cause during follow-up than patients whose plaques did not (Marfella et al., 2024). Such findings are associations, not proof of causation, and they have been debated, particularly with regard to contamination and analytic methods. Even so, they have focused attention on the possibility that particles are not simply passing through.

Experimental work points to several possible mechanisms of injury. Particles can trigger inflammatory responses in immune cells, generate oxidative stress, and disturb the integrity of endothelial and epithelial barriers. Surfaces of particles can adsorb proteins and form a corona that changes how cells recognise them, and they can carry additives, residual monomers, or other chemicals into tissues. Smaller particles may enter cells and accumulate in organelles. Most of these observations come from cell cultures and animal studies, often at concentrations and particle types that may not mirror clinical exposure, so translating them to bedside risk requires humility.

A particularly important question is whether intravenous exposure behaves differently from environmental exposure. Environmental microplastics are mostly ingested or inhaled, and they must cross barriers to reach the circulation. A particle delivered intravenously has no such obstacle. For this reason, the infusion pathway may deserve more scrutiny than its relatively small mass contribution suggests.

**Why critically ill patients are different**

Several features make intensive care patients a vulnerable population. First, they are already in a state of dysregulated inflammation. Sepsis, acute respiratory distress syndrome, and multi-organ failure involve activated endothelium and leaky capillaries, and an additional inflammatory stimulus from particles could in theory tip a fragile balance. Second, they often have impaired clearance. Liver dysfunction, kidney injury, and reduced splenic function may slow the removal of foreign material. Third, their exposure is intense and prolonged: many lines, many drugs, and many days. Fourth, some groups are especially delicate. Preterm infants and neonates receive large volumes of fluid relative

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to body weight, through small-bore tubing, with immature immune and detoxification systems, and they may carry the consequences for decades.

There is also a diagnostic difficulty. If a patient develops a new fever, worsening lung function, or organ dysfunction in the ICU, the differential diagnosis is already crowded with infection, drug reactions, ventilator injury, and the underlying disease. A modest contribution from particulate exposure would be invisible against this background. This is exactly the situation in which a harm might exist without being noticed: it is common, small, non-specific, and attributable to something else.

**The evidence gap**

It is important to be honest about what we do not know. We do not have reliable measurements of the number, size, and polymer type of particles that critically ill patients receive through standard infusion and feeding systems. Methods for detecting nanoplastics in complex fluids such as lipid emulsions, blood products, and enteral formula are technically demanding and not standardised. Studies of human tissue are vulnerable to contamination from laboratory plastics and air. No large clinical study has compared outcomes in ICU patients exposed to different plastic loads, and the existing filtration trials were not designed to separate plastic from other particulate matter.

There is also a risk of overstatement in the opposite direction. Plastic devices have saved countless lives by enabling safe, sterile, and reliable delivery of therapy. Glass containers have their own hazards, including breakage, glass particles, and the need for air venting, and abandoning plastic without a tested replacement could increase infection and error. The right posture is neither alarm nor complacency, but structured curiosity: measure first, then act in proportion to the findings.

**Practical steps for intensive care units**

While research catches up, units can adopt measures that are low-risk and consistent with good practice already. Many of them reduce particles of all kinds, not only plastic, and several also reduce infection and medication error. The table below summarises key exposure points and pragmatic responses.

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| Exposure point | Likely mechanism | Practical mitigation |
|---|---|---|
| Plastic IV bags and administration sets | Leaching and shedding of particles from polymer surfaces; abrasion at connectors and pump segments; plasticiser migration | Use in-line filters where indicated; prefer glass or non-PVC systems for sensitive drugs; avoid prolonged storage and heat exposure |
| Drug preparation and additives | Particles from vials, rubber stoppers, syringes and coring of closures; repeated needle punctures | Use filter needles, aseptic technique, minimal manipulation, and single-use closed-system devices where available |
| Enteral formula containers and bags | Degradation of polypropylene and other polymers with heat, agitation and time | Avoid heating formula in plastic; limit hang time; use ready-to-hang closed systems per product guidance |
| Feeding tubes and giving sets | Surface wear, flushing and medication crushing; PVC, polyurethane or silicone tubing in contact for days to weeks | Change sets on schedule; avoid unnecessary mechanical stress; flush gently; review tube material and dwell time |
| Water for flushing and dilution | Environmental microplastics in tap or bottled water | Use sterile or filtered water; use dedicated clean containers rather than reused plastic bottles |

Beyond individual measures, organisations can build awareness into governance. Procurement teams can ask manufacturers for data on particulate release and on polymer composition of administration sets, and they can favour products with transparent testing. Pharmacy and nursing leadership can review preparation practices, such as the use of filter needles and the avoidance of unnecessary

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manipulation. Infection prevention and critical care committees can include particulate burden as one dimension of device selection alongside cost, usability, and sterility. In-line filters, which are already standard in some neonatal and paediatric units, deserve evaluation in adult practice, balancing possible benefit against cost, flow limitations, and the fact that lipid emulsions and some drugs cannot pass through standard filters.

Clinicians can also contribute at the bedside. Minimising the total number of infusions and connections, removing lines that are no longer needed, replacing sets on schedule rather than leaving them for extended periods, and avoiding heat or vigorous agitation of plastic containers are small practices that add up. In enteral feeding, using closed ready-to-hang systems when available, avoiding the preparation of formula in reused plastic containers, and flushing tubes gently and with clean water are sensible habits.

**Priorities for research**

A credible research agenda would begin with measurement. Standardised methods are needed to quantify particles from submicrometre to visible size in intravenous fluids, parenteral nutrition, blood products, and enteral formula, with identification of polymer types and with strict contamination controls. The next step would be to characterise exposure across real ICU circuits, comparing different tubing materials, pump types, storage times, and temperatures. Clinical studies could then test whether higher particle exposure is associated with markers of inflammation, organ dysfunction, or outcomes, ideally within randomised evaluations of filtration or alternative materials. Particular attention should be paid to neonates, to patients on long-term parenteral nutrition, and to those receiving extracorporeal therapies such as dialysis and extracorporeal membrane oxygenation, where blood contacts large plastic surfaces for long periods.

Equally valuable would be qualitative and implementation research. How do clinicians perceive the issue? Which interventions are feasible within workflows that are already stretched? How should hospitals communicate with patients and families about a concern that is real but uncertain? Questions of this kind will determine whether any findings translate into safer care.

**Conclusion**

Microplastics in the intensive care unit sit at an uncomfortable intersection: a plausible hazard, a vulnerable population, an incomplete evidence base, and a technology on which modern critical care depends. The available evidence does not justify declaring that ICU plastics are harming patients, nor does it justify dismissing the possibility. What it does justify is attention. Intravenous and enteral

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systems are designed to be clean, but “clean” in the microbiological sense is not the same as free of particles, and the latter has rarely been measured with the tools now available.

The most constructive response is to treat particulate contamination as a patient safety question like any other: define it, measure it, reduce what can be reduced at little cost, and test the rest. The patient in our opening scene, surrounded by plastic that keeps them alive, deserves both the benefits of that technology and the assurance that we have looked carefully at its costs.

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*Note: This article is a narrative overview for educational purposes. References should be verified against the original sources before citation.*