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STUDYING THE METABOLIC PROPERTIES OF CREATINE

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Annotation: Creatine is synthesized primarily in the liver as a result of methylation of glycocyamine (guanidinoacetate synthesized in the kidneys from the amino acids arginine and glycine) with S-adenosylmethionine. It is then transported through the blood to other organs, muscles and the brain, where it is converted by phosphorylation into the high-energy compound phosphocreatine

Key words: Creatine, S-adenosylmethionine, guanidinoacetate, synthesized, amino acids.

INTRODUCTION

This is naturally due to the harsh living conditions of animals. First, some data. Different groups of animals shed their antlers at different times. The oldest males are dehorned just before the New Year. Young males carry antlers until the end of February. Non-pregnant females do not shed their horns until mid-May, and pregnant females do not shed their horns until the birth of calves, which are born until the end of June[1-6].

It can be seen that the most vulnerable social groups wear horns longer.

The fact is that horns are needed for protection. To protect their food from other members of the herd.

In winter, the deer obtains food from under the snow, “hoofs”, raking the crust with its hooves to get to the reindeer moss. This takes a lot of energy, and if there is an ice crust or the snow cover is too large, then more energy will be spent on obtaining food than from the food itself. Therefore, in such conditions there is no time for chivalry and stronger animals begin to take away the already dug up food. This is precisely why nature dehorns the largest males before anyone else. You will dig up a strong one, and the horned females will be able to protect their share. Well, then continue along the lines. The longer the winter, the more food pregnant females

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need, so all other groups lose their horns one after another so as not to create competition for the mothers[7-13].

Metod and results

Creatinine is a breakdown product of creatine phosphate as a result of muscle and protein metabolism. It is released by the body at a constant rate (depending on muscle mass [14-20].

Serum creatinine [3] (a blood measurement) is an important indicator of kidney health because it is an easily measured byproduct of muscle metabolism that is excreted unchanged by the kidneys. Creatinine itself is produced through a biological system that includes creatine, phosphocreatine (also known as creatine phosphate), and adenosine triphosphate (ATP, the body's immediate source of energy).

Creatine is synthesized primarily in the liver as a result of methylation of glycocyamine (guanidinoacetate synthesized in the kidneys from the amino acids arginine and glycine) with S-adenosylmethionine. It is then transported through the blood to other organs, muscles and the brain, where it is converted by phosphorylation into the high-energy compound phosphocreatine [4]. The conversion of creatine to phosphocreatine is catalyzed by creatine kinase; During the reaction, spontaneous formation of creatinine occurs [5-20].

Creatinine is removed from the blood primarily by the kidneys, mainly by glomerular filtration, but also by proximal tubular secretion. There is little or no tubular reabsorption of creatinine. If filtration in the kidneys is insufficient, the concentration of creatinine in the blood increases. Therefore, blood and urine creatinine concentrations can be used to calculate creatinine clearance (CC), which roughly correlates with glomerular filtration rate (GFR). Blood creatinine concentrations can also be used separately to calculate estimated GFR [5]. Ketoacids, cimetidine and trimethoprim reduce tubular secretion of creatinine and therefore increase the accuracy of GFR estimates, especially in severe renal failure.

An alternative assessment of renal function can be made by interpreting the plasma creatinine concentration along with the urea concentration. The BUN-to-creatinine ratio (the ratio of blood urea nitrogen to creatinine) may indicate problems other than those specific to the kidneys; for example, a urea concentration that is elevated disproportionately to creatinine may indicate a prerenal problem such as volume depletion. Mechanism syntheses creatinine in vivo:

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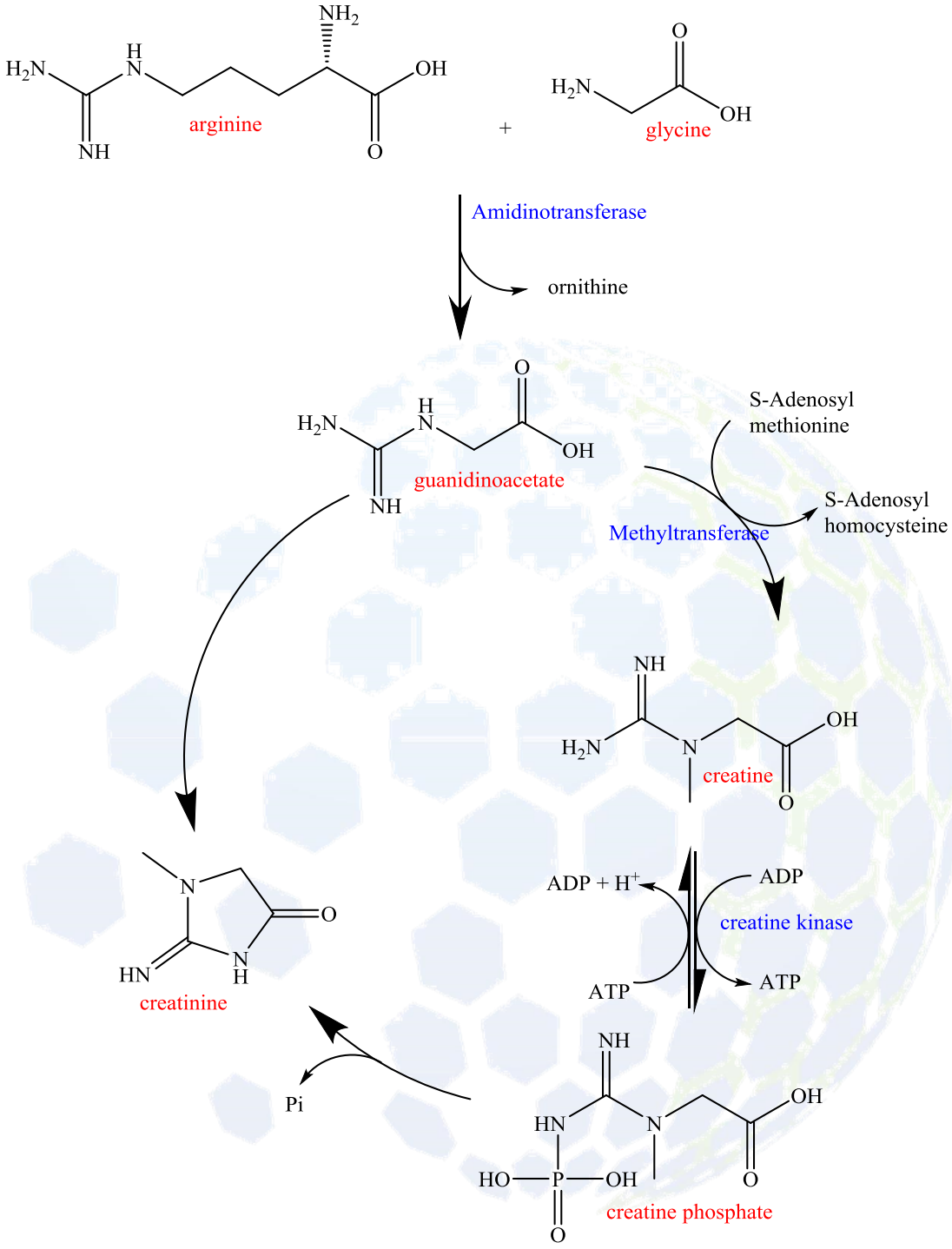

Experimental part

Antibacterial and potential immunosuppressive properties. Research shows that creatinine may be effective in killing many types of bacteria, both gram-positive and gram-negative, as well as various strains of antibiotic-resistant bacteria. Creatinine does not affect the growth of fungi and yeast; this can be used to isolate slow-growing fungi free from the normal bacterial populations found in most environmental samples. The mechanism by which creatinine kills bacteria is currently unknown. A recent report also suggests that creatinine may have

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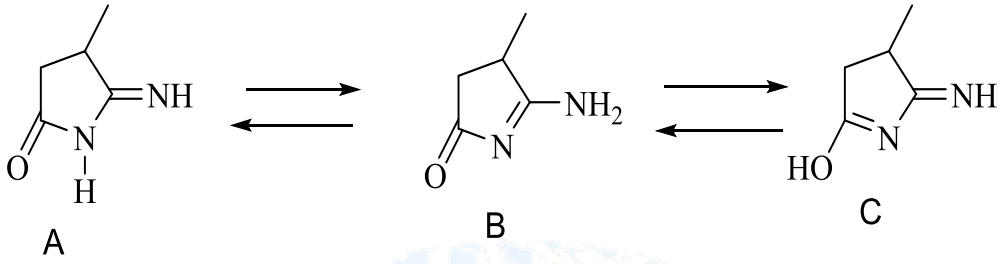
immunosuppressive properties, so we sought to continue our scientific work to study the biochemical properties of creatinine and exists in three different tautomeric states A, B, C:



A B C

Creatine synthesis and amino acid metabolism

How much of a burden does creatine synthesis place on amino acid metabolism? The issue regarding glycine is straightforward, because the entire glycine molecule is incorporated into creatine. Therefore, between d 4 and 11, piglets incorporated ~9.7 mmol of glycine into creatine at a mean rate of 25.4 μmol·kg^{-1}·h^{-1}. The total intake of glycine over the same period (80 mmol) can be estimated from the glycine composition of mature sow milk and a milk consumption of 0.77 L/(piglet·d). Creatine synthesis, therefore, accounts for ~12% of dietary glycine. In addition, it is clear that considerable glycine synthesis occurs in neonatal piglets. Glycine accounts for 11.3% of whole piglet protein. Given that our piglets accumulated 242 g of protein over 7 d, we calculated that they deposited ~360 mmol of glycine in their protein, which is >4 times their glycine intake in milk. The glycine used for creatine synthesis is, therefore, only ~2.7% of the net glycine incorporated into protein.

The metabolic burden on methionine and arginine metabolism may also be calculated in a similar way. Between d 4 and 11, we calculated a methionine intake of 27.8 mmol and an arginine intake of 47.7 mmol. Therefore, creatine synthesis amounts to ~35 and 20%, respectively, of the dietary intake of these amino acids. We therefore estimated that between d 4 and 11, there was a net deposition of 93 mmol of arginine and 31.7 mmol of methionine in piglet protein. That piglets' arginine deposition into protein was twice their arginine intake in milk reflects the substantial endogenous arginine synthesis in these animals. The arginine used for creatine synthesis amounts to ~10.3% of that incorporated into protein. The net deposition of methionine in protein is very similar to our estimates of milk methionine ingestion, which is consistent with the fact that methionine cannot be synthesized in animals. Methionine used for creatine synthesis is ~30.4% of methionine deposited in protein. However, evaluating the burden of creatine synthesis on the metabolism of arginine and methionine is more complex than that

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of glycine, because the entire methionine and arginine molecules are not incorporated into creatine. In the case of methionine, only the methyl group is incorporated from SAM. In the case of arginine, only the amidino group is incorporated. In both cases, methionine and arginine can be regenerated by enzymes in their respective cycles.

Amino acids and creatine synthesis. The production of 1 molecule of creatine requires an entire glycine molecule together with an amidino group (provided by arginine) and a methyl group (provided by SAM). Whether this represents a net utilization of arginine and methionine depends on the occurrence of mechanisms to regenerate arginine from ornithine and methionine from homocysteine. CBS, Cystathionine β-synthase; CGL, cystathionine γ-lyase; CPS 1, carbamoylphosphate synthetase 1; DMG, dimethylglycine; OAT, ornithine aminotransferase; OTC, ornithine carbamoyltransferase; THF, tetrahydrofolate.

The burden imposed by creatine synthesis on arginine metabolism is, also, not clear-cut. AGAT produces ornithine and the key issue lies in the fate of this amino acid. Ornithine may be converted to citrulline by ornithine carbamoyltransferase and, ultimately, to arginine. However, ornithine conversion to citrulline requires a source of carbamoylphosphate. Carbamoylphosphate synthase 1 is absent from the kidney. Alternatively, ornithine may be catabolized via ornithine aminotransferase, which is active in the kidney; however, this results in a loss of its potential to be reconverted to arginine. Clearly, this issue requires more work, but the following may be pertinent: *1*) arginine is a conditionally essential amino acid in neonatal piglets, i.e. endogenous arginine synthesis cannot supply all of the arginine required; and *2*) arginine may become limiting for growth in neonatal piglets. Plasma arginine decreases 20–40% between the ages of 3 and 14 d. The growth of milk-reared piglets between d 7 and 21 can be markedly improved by either provision of supplemental arginine or pharmacological activation of intestinal arginine synthesis. These data suggest that the maximal growth of piglets is limited by arginine availability. It may also be noteworthy that preterm infants exhibit hypoargininemia. The demand for arginine imposed by high rates of creatine synthesis may well be responsible for the neonatal decrease in circulating arginine levels.

Conclusion

Our measurements reveal that neonatal piglets synthesize most of the creatine that is acquired. This, in turn, has substantial metabolic implications. The rate of creatine synthesis is quite small compared with glycine fluxes and can hardly be considered a major drain on glycine pools. On the other hand, creatine synthesis may well make appreciable demands on arginine metabolism. Clarification of this issue requires further work. Finally, creatine synthesis in neonatal piglets is a major user

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of labile methyl groups and, clearly, places a very large burden on the dietary provision of appropriate methyl donors (methionine, choline, and betaine) and on the synthesis of new methyl groups via folate-dependent methylneogenesis. A similar quantity of choline in sow milk would provide a total of ~6.5 mmol to each piglet per week. Milk choline could therefore provide a large portion of the necessary labile methyl groups. However, the degree to which it does so will depend on the extent to which choline is catabolized by piglets compared with its utilization for phospholipid and acetylcholine synthesis. The contribution of different tissues to creatine synthesis also requires further exploration

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