題名:NMDA 或/和 甘胺酸在鼠胃生理角色之研究

Physiological roles of NMDA and/or glycine on the stomachs

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關鍵字:甘胺酸

胃酸分泌

NMDA

cold-restraint stress

NO

NMDA

glycine

isolated stomach

acid secretion

cold-restraint stress

NO

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摘要:The objective of the study was to measure net AA flux rates across the portal-drained viscera (PDV) and liver in the presence and absence of abomasal glucose infusion. Decreasing the fraction of AA metabolized by the mucosal cells may increase the fraction of AA being released into the blood. A potential mechanism to reduce AA catabolism by mucosal cells is to provide an alternative source of energy. We hypothesized that increasing glucose flow to the small intestine would increase net appearance of AA across the portal-drained viscera. Eighteen mature ewes with sampling catheters were placed on study. The experimental design was a split-plot with a complete randomized design on the whole-plot and a Latin-square subplot with 5 periods and incremental levels of protein infusion. One-half of the ewes received abomasal glucose infusions (3.84 g/h), and all ewes received each of 5 protein abomasal infusion levels over 5 periods (0, 2.6, 5.2, 7.8, and 10.5 g/h). Net PDV release of isoleucine, leucine, methionine,

phenylalanine, aspartate glutamate, glutamine, proline, serine, and tyrosine increased linearly with increased protein infusion, and net PDV release of histidine, lysine, threonine, valine, alanine, and glycine did not differ with protein infusion. Net hepatic glucose release decreased with glucose infusion. With the exception of histidine, phenylalanine, and valine, net hepatic AA uptake increased linearly with increased delivery of protein to the liver. Glucose infusion increased the hepatic lysine and valine uptake and decreased phenylalanine uptake. Based on the observations in the current study, we reject our hypothesis that glucose can spare AA metabolism by PDV tissue. Our findings suggest that hepatic gluconeogenesis can be increased in the presence of increased AA delivery to the liver and that hepatic gluconeogenesis can be decreased with increased absorption of dietary glucose. Our findings support the concept that for most AA, hepatic transport of AA can be described by mass action kinetics; however, the rates of hepatic uptake of specific AA are up-regulated directly or indirectly by elevated glucose.