Proc Soc Exp Biol Med. 1995 Dec;210(3):260-5.

Altered tissue amino acid metabolism in acute T-2 toxicosis

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Abstract

T-2 toxin is a Fusarium trichothecene mycotoxin that has been shown to alter brain neurochemistry and eating behavior in animals eating contaminated diets. Experiments were conducted to determine the role of altered tissue amino acid metabolism in the etiology of acute T-2 toxicosis. Fasted weanling rats were orally dosed with 0 or 2.0 mg T-2 toxin/kg body weight. Blood, brain, liver, and muscle tissue were excised 4 and 8 hr after dosing, and amino acid concentrations were determined. Hepatic enlargement coupled with reduced liver concentrations of free small neutral, large neutral, and basic amino acids were seen 4 hr after dosing. Brain and muscle amino acid concentrations were largely refractory to treatment, while the plasma concentrations of tyrosine and lysine, and the sum of the basic amino acids fell. Hepatic amino acid concentrations returned to control levels 8 hr after dosing at which time aminoacidemia was seen. This was due partially to an increase in plasma concentrations of large neutral amino acids including particularly the branched-chain amino acids. A subsequent experiment was conducted to determine the effect of T-2 toxin on 14C-leucine uptake and incorporation into protein in liver slices 4 hr after dosing. Exposure to T-2 toxin reduced total (free + protein-bound) uptake of leucine due primarily to reduced incorporation of leucine into newly-synthesized hepatic protein. It was concluded that reduced amino acid uptake by liver preceded aminoacidemia in acute T-2 toxicosis, although it is not clear how this might influence subsequent changes in brain neurochemistry and behavior.

PMID: 8539264

Source: https://www.ncbi.nlm.nih.gov/pubmed/?term=8539264