Abstract


The metabolic availability of vitamin A is decreased at the onset of diabetes in BB rats.

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OBJECTIVE: Streptozotocin (STZ)-induced diabetic rats have been associated with an impaired metabolic availability of vitamin A (retinol). This study was undertaken to investigate whether Biobreeding (BB) rats, in which diabetes mellitus resembling human type I diabetes develops spontaneously, respond the same way at the onset of diabetes.

METHODS: Weaning diabetes-prone (BBdp) and normal (BBn) BB rats consumed NIH-07 nonpurified diet ad libitum until 120 d of age.

RESULTS: Plasma and hepatic concentrations of retinol and its carriers, retinol-binding protein (RBP) and transthyretin (TTR) were lower in diabetic BB (BBd) rats than in BBn rats. In parallel with RBP, the abundance of mRNA was lower in the liver of BBd rats. Furthermore, the status of zinc, an important factor for the synthesis of RBP, was also disturbed in BBd rats, as indicated by lower circulatory levels and greater urinary excretion. To determine whether the biochemical evidence of vitamin A deficiency in BBd rats could be reversed, BBdp rats were fed a diet supplemented with vitamin A either alone or in combination with zinc. None of these treatments increased plasma vitamin A concentration. The hepatic abundance of RBP mRNA was significantly greater, whereas circulatory RBP concentrations were unaffected by vitamin A plus zinc supplementation.

CONCLUSION: Overall, these results suggest that impaired metabolic availability of vitamin A, possibly caused by its decreased transport from hepatic stores, is another metabolic derangement associated with type I diabetes.

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