Journal of Andrology, Vol 21, Issue 5 656-663, Copyright © 2000 by The American Society of Andrology

JOURNAL ARTICLE

Differential zinc transport into testis and brain of cadmium-sensitive and -resistant murine strains

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Recently, we showed that murine strain differences to the testicular toxicity of cadmium (Cd) are the result of variable transport of Cd across the blood-testis barrier. Because Cd is a nonessential trace element, it must be using the transporter for an endogenous substance. The objectives for this study were to determine the natural ligand for the transport system used by Cd to enter testis and brain, and to determine whether the transport of that natural ligand also differs among Cd-sensitive and -resistant murine strains. Because zinc (Zn) and Cd are cations of similar size and charge, and because Cd has been shown to inhibit Zn uptake in a variety of systems, we hypothesized that Cd was using Zn transporters to enter tissues. In this study we characterized Zn transport into the testis and brain of Cd-sensitive and -resistant murine strains. We found that the transport of 65Zn into testis and brain of Cd-resistant A/J mice was significantly reduced compared with that in Cd-sensitive 129/J mice. In 129/J mice, unlabeled CdCl2 significantly reduced 65Zn transport by 56% in testes and by 47% in brain. Pretreatment with Zn had no significant effect on 109Cd transport rates into testes or brain of 129/J or A/J mice, but did reduce the percentage of the injected 109Cd dose in testes of 129/J mice by 44% within 60 minutes. From these results we can conclude that Cd is using transport systems that normally function to regulate Zn levels in testes and brain. Murine strain resistance to the testicular effects of Cd is associated with a concomitant attenuation of the Zn transport system in testis.

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