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Introduction to Animal Models: Brain Purines in a Mouse Model for Lesch-Nyhan Disease

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Short Communication

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ABOUT THE STUDY

Animal models are an essential tool for researchers hoping to learn more about metabolic disease. In many cases, data cannot be collected from living patients with a metabolic disease, as this sometimes calls for organ dissection or other highly invasive procedures. Model animals can be engineered to express the disease phenotype and can be euthanized in order to collect data. This is the case especially in the following two articles about Lesch-Nyhan and Gaucher's disease model mice. Animal models are an essential tool for researchers hoping to learn more about metabolic disease. In many cases, data cannot be collected from living patients with a metabolic disease, as this sometimes calls for organ dissection or other highly invasive procedures [1]. Model animals can be engineered to express the disease phenotype and can be euthanized in order to collect data. The mice were deficient in an enzyme that breaks down a harmful product in the brain. The first model expressed this deficiency in all brain tissue, and the second had normal expression in the microglia, one particular brain tissue, in order to determine what role that tissue played in the pathology of the disease. Researchers again had to remove the brains of the mice for study.

Researchers created model mice in order to study the effects of phenylalanine in the diet. Although this is not as drastic as removal of the brain, it is still impossible to use human subjects to do a study of this variety. Another reason animal models of metabolic disease are needed is for preliminary testing of therapies. The first and third web site overviews discuss the usefulness of animal models in this case. Using animal models, researchers can identify which tissues might be damaged or aided by the use of a drug. Animal trials with a drug are an important precursor to human clinical trials [2]. Lesch-Nyhan Disease is a human neurogenesis disorder that causes a number of neurobehavioral abnormalities including involuntary or abnormal movement, aggression, and self-injury. The disorder

is caused by a deficiency in the enzyme hypoxanthine-guanine phosphoribosyl transferase (HPRT) which functions in the metabolism of central nervous-system purines. HPRT recycles free purines so that cells do not have to constantly manufacture a new supply. In order to better understand how a lack of HPRT enzyme affects purine levels in the brain, researchers in this study use a mouse model with a deletion mutation in the HPRT gene [3]. The mouse model has been shown to produce undetectable levels of HPRT enzyme or mRNA transcript. Although the model mice are missing functional HPRT enzyme, they do not present with the observable neurobehavioral abnormalities of human Lesch-Nyhan patients [4]. However, the mice still provide a reliable model for studying purine salvage in HPRT deficient brains.

Researchers performed a number of different experiments to isolate HPRT protein from model and control mice and to assay their brains for purine content. The brains of HPRT positive mice were removed and homogenized then subjected to a series of treatments to isolate pure HPRT. The purified HPRT preparation yielded a single major band of approximately 24 kDa on an SDS-PAGE gel [5]. The purified HPRT was used to test a polyclonal rabbit anti-HPRT antiserum by Western analysis. The anti-HPRT antiserum successfully labeled the same 24 kDA band, showing that it effectively binds to the mouse HPRT protein. Using the same purification technique on the brains of HPRT negative mice, the resulting Western analysis showed no binding with the anti-HPRT antiserum, confirming that the mouse model does not in fact produce the HPRT enzyme. HPRT levels in crude protein extracts from HPRT negative mice were also below detectable limits.

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