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Hereditary Cataract of the Nakano Mouse

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The Nakano mouse cataract is inherited as an autosomal recessive trait. It was originally discovered by Dr. Kennji Nakano in 1957. Animals from this strain open their eyes on day 14, the same as normal mice, at which time the lenses of the Nakano mice are still clear. Homozygous animals develop a pin head opacity in the nucleus of the lens on the 24th postnatal day, and over the next 36 days the cataract becomes mature.

This paper will review recent advances in the study of the Nakano mouse system. It will include studies of cation levels, morphology, lens implantation, capsule glycosaminoglycan synthesis, and predicted gap junctional activity in the lenses from Nakano and normal mice.

The Nakano mouse is a hereditary cataract model whose most characteristic change is a deficiency in lens Na^+ , K^+ -ATPase. The amounts of calcium in also change suddenly in the lens, with accumulated levels higher than any other type of cataract. Other biochemical changes coincide with the development of lens opacity, including decreases in the levels of reduced glutathione, ATP, biosynthetic activity of proteoglycans in epithelial cells, and the permeability of gap junction channels in fiber cells. The decrease in the activity of Na^+ , K^+ -ATPase results in changes in a number of key metabolic parameters, resulting in the eventual opacification of the Nakano mouse lens at approximately 30 days of age.

* 本報告は Exp. Eye Res (1990) 50, 671—676 に発表.