Why mice don’t get cancer of the retina

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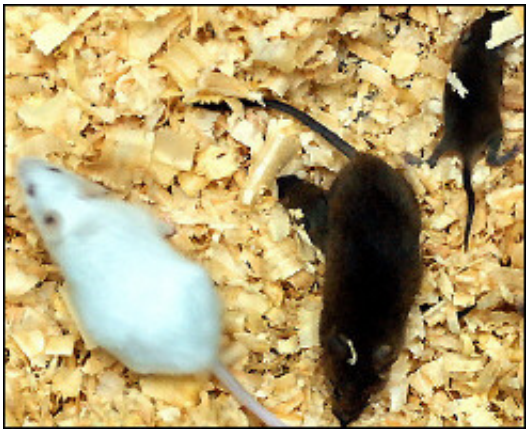

Humans are more susceptible to developing the eye cancer retinoblastoma than mice because, unlike humans, mice can compensate for the loss of activity of a gene critical to normal retinal development , according to results of a study by investigators at St. Jude Children's Research Hospital.

The study, published today in the open access journal BMC Biology, explains why humans with a defective copy of the Retinoblastoma gene RB1 are at high risk of developing cancer of the retina, or retinoblastoma, whereas mice with a similar genetic profile do not develop the cancer.

Stacy Donovan and Brett Schweers from St Jude Children’s Research

Hospital in Memphis, USA and colleagues from St Jude's and from the University of Tennessee Health Science Center in Memphis, studied the expression of the Retinoblastoma proteins Rb (RB1 in humans), p107 and p130 throughout the development of mouse and human retinae, using molecular amplification and immunolabelling techniques.

Donovan et al. find that p107, Rb and p130 are expressed at different stages in the developing mouse retina, with p107 expressed first and Rb and 130 expressed during the late stages of development. The authors show that, in mutant mouse embryos that do not express Rb at all, the levels of p107 are much higher than in wild-type embryos at the same stage in development. The reverse situation is observed in mutant embryos that do not express p107.

This suggests that Rb and p107 compensate for each other in retinal progenitor cells and prevent the deregulated proliferation of the cells that leads to retinoblastoma. By contrast, RB1 is the main protein expressed during retinal development in humans. The protein p107 is only slightly expressed during development and cannot compensate for the lack of RB1, which leads to retinoblastoma.

Source: BioMed Central Limited