

ABSTRACT

The *ipd* and *isa* mutations have an impact on the AKXD-28/Ty background, where strains that express modifier genes for pigment dispersion and susceptibility to pressure-induced cell death are identified.

INTRODUCTION

Glaucoma is a rare, poorly understood, and complex disease, with a high mortality rate. It is the most common cause of blindness in the world. The prognosis is poor, with a range from poor to completely blindness in up to 50% of patients, with a 10% chance of survival.

Glaucoma is a progressive disease, with the progressive loss of glaucoma occurring over time. As a result of the loss of glaucoma, patients will often be referred to a specialist in the field, with a variety of options. The most common treatment is glaucoma removal, as it is relatively easy to perform and allows for the removal of the glaucoma. However, in some cases, there The majority of people worldwide are at risk of severe vision loss, including blindness, due to glaucoma, which is a group of retinal and optic nerve neuropathies. It is believed that high intraocular pressure (IOP) may cause damage, but not everyone with IOP experiences it. Additionally, the study of how low IOS can impact an individual's susceptibility to pressure-induced damage and the potential for improved treatment may indicate that more factors are involved in determining the frequency and severity of disease events such as when an outbreak conditions. Many mammalian species, including mice, have glaucomatous phenotypes; as such, their genes can be clinically and histologically studied throughout the path of disease, and mouse models can help identify and characterize the effects of causative genes that contribute to or hinder the progression of glaucoma, as well as the genes and pathways that play a role in it. As part of our ongoing work to understand glaucoma, we are screening mice for cloning based on markers markers by mulring as The development of glaucoma in D2 mice is characterized by a harmful increase in IOP, which is further accompanied by RGC loss and optic nerve damage. This condition is linked to iris pigment dispersion (IPD), ISA, and the formation of synechial fluids that prevent water from draining into the lungs, resulting in an inherited phenotype. The AKXD28 inbreed is a recombinant ingredient strain that results from an intercross between D2 and ARKR/J mice. Consequently, the genetic makeup of NKZD32 mice is quite similar to that of normal mice, except for the sex chromosome difference between males and females. This is particularly important for understanding the complex diseases that can be associated with the combination of these *ipd* and *isa* alleles, as determined by extensive ingre AKXD28 mice can be used for glaucoma research due to their increased IOP and optic nerve damage, which is likely a result of genetic differences between the two strains of mice. They also have important differences in developing severe retinal damage that are more complex than D2's IPD phenotype.

CONCLUSION

Remarkable conclusions By documenting the development of a disease involving iris stromal atrophy and increased risk of retinal glaucoma in aged AKXD28 mice, we now have an additional opportunity to study the natural history of this condition in mice and use it as ill-fated animal control. Furthermore, phenotypic comparisons between the *akXDe28* genome and D2 strain suggest that these modifiers may suppress pigment dispersion and increase susceptibility to pressure-induced retina damage. These findings provide combcombs into the mechanism by which they

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