Abstract: The corneal endothelium is primarily responsible for maintenance of corneal dehydration and transparency, which is critical for normal vision. Corneal endothelial dystrophy (CED) is a late-onset disease in dogs whereby the endothelial cells prematurely degenerate resulting in progressive corneal swelling, vision loss and ocular pain due to corneal ulceration. Secondary corneal infection and perforation can occur necessitating eye removal. Currently, the only definitive treatment for CED and a similar disease in human patients, termed Fuchs’ endothelial corneal dystrophy (FECD), is corneal transplantation. However, corneal transplants are rarely performed in dogs due to the risk for graft rejection, lack of appropriate donor tissue, and expense. Alternative treatments for canine CED are urgently needed. Preliminary work demonstrated that a rho-associated kinase coiled-coil containing protein kinase (ROCK) inhibitor accelerated corneal endothelial regeneration. Netarsudil 0.02% ophthalmic solution (Rhopressa®) is a topical ROCK inhibitor and norepinephrine transport inhibitor recently approved by the Food and Drug Administration (FDA) for use in patients with glaucoma. Preliminary data suggest that netarsudil accelerates corneal endothelial recovery. This study will investigate the efficacy and safety of netarsudil for the treatment of early canine CED. If successful in dogs, netarsudil findings may translate to use for treatment of FECD in human patients.

Objective: This study is using the Affymetrix canine single nucleotide polymorphism (SNP) chip to identify regions that are linked to LCPD in the West Highland White Terrier, Yorkshire Terrier, and Miniature Pinscher breeds.