Genomic analysis of gum disease and hypertrichosis in foxes


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ABSTRACT. Since the 1940s, a proliferative gingival disease called hereditary hyperplastic gingivitis (HHG) has been described in the farmed silver fox, Vulpes vulpes (Dyrendahl and Henricson 1960). HHG displays an autosomal recessive transmission and has a pleiotropic relationship with superior fur quality in terms of length and thickness of guard hairs. An analogous human disease, hereditary gingival fibromatosis (HGF), is characterized by a predominantly autosomal dominant transmission and a complex etiology, occurring either as an isolated condition or as a part of a syndrome. Similar to HHG, the symptom most commonly associated with syndromic HGF is hypertrichosis. Here we explore potential mechanisms involved in HHG by comparison to known genetic information about hypertrichosis co-occurring with HGF, using an Affymetrix canine genome microarray platform, quantitative PCR, and candidate gene sequencing. We conclude that the mitogen-activated protein kinase pathway is involved in HHG, however despite involvement of the mitogen-activated protein kinase kinase 6 gene in congenital hypertrichosis with gingival fibromatosis in humans, this gene did not contain any fixed mutations in exons or exon-intron boundaries in HHG-affected foxes, suggesting that it is not causative of HHG in the farmed silver fox population.
Differential up-regulation of *MAP2K6* gene in HHG-affected foxes does implicate this gene in the HHG phenotype.

**Key words:** Hereditary hyperplastic gingivitis; Hypertrichosis; Mitogen activated protein kinase pathway; MAP2K6; Hereditary gingival fibromatosis