Study of glioma susceptibility in dogs may yield insights for humans

May 12 2016

Boxers are the dog-breed with the highest risk of developing glioma. Credit: Katarina Sundberg

A new study of the genetic factors underlying glioma formation in dogs may hold clues to how these common and often untreatable tumors form in humans. The genome study, which was conducted across 25 dog breeds, identified three genes associated with the tumor. The results from this research, led by Katarina Truvé of the Swedish University of Agricultural Sciences and Kerstin Lindblad-Toh of Uppsala University, were published on May 12 in *PLOS Genetics*.

Gliomas are the most common form of malignant primary brain tumors in humans and the second most common in dogs. Several dog breeds such as Boxer, Bulldog and Boston Terrier have an elevated risk of developing glioma, while certain related breeds do not, suggesting that a mix of genes may impact glioma formation. Dr Truvé says: "Researchers in the consortium are now continuing the analysis of the genes identified, and their functional roles in development and progression of glioma in both dogs and humans."

To identify genetic variations that contribute to the tumor's development, scientists performed a genome-wide association study (GWAS) using blood samples from 39 dogs diagnosed with glioma and 141 control dogs. By screening for variations commonly found in dogs that developed gliomas, they pinpointed three genes highly associated with susceptibility to the tumor: CAMKK2, P2RX7 and DENR.

Two of these genes have additional links to cancer. Further experiments by the scientists showed that both human and canine gliomas express

CAMKK2 at lower levels than normal brain tissue, and previous studies have shown that a variation of P2RX7 reduces protein function in dogs while other variations have been identified in cancer patients. Future investigation of all three genes may yield a better understanding of the causes and potential treatments of glioma in both dogs and humans.

More information: *PLOS Genetics*, DOI: 10.1371/journal.pgen.1006000