Genetic screening of the canine Connexin 40 and 43 genes in inherited cardiac conduction defects in dogs

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Introduction: The connexin (Cx) genes mutation or mutations that result in human cardiac diseases due to cardiac malformations or conduction defects, are relatively unknown. Because the Connexins are a major component of intercellular channels (gap junctions) that facilitate cell-to-cell adhesion and provide pathways for direct intercellular communication, those genes are important candidates for canine cardiac conduction defects.

Material and methods: We isolated the full length coding exon of Cx40 and Cx43. Those connexins were screened for mutation in dogs with conduction defects (sick sinus syndrome in a miniature Schnauzer, malignant ventricular tachycardia in a German Shepherd and atrial fibrillation in a Boxer)

Results: Analysis of the deduced amino acid sequence suggested that the canine Cx40 and Cx43 are phylogenetically closer to thehuman Cx40 and Cx43 (91% and 98% identity, respectively) than mouse and rat. The highest RNA expression in Cx40 was found in the lung and heart, implicating a critical role for Cx40 role in cardiomorphogenesis and vasculogenesis. However, no significant mutations were observed in this screening.

Clinical relevance: For this screening, we ruled out Cx40 and Cx43 from the candidate for canine conduction defects. Despite the lack of significant sequence changes, the fact remains that these dogs exhibit clinical signs consistent with problems due to Cx40 functionality.

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