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Molecular characterization of thyroid tumors of dogs – a multicentric Portuguese series

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Abstract

Background: The incidence of thyroid carcinoma (TC) in human population has been increasing worldwide, and it was estimated an incidence of 2.1% of cancer new cases in 2019 [1]. A smaller incidence is reported for canine population (1.1%) in 1995-2005 [2]. Diagnosis, prognosis, and management of human TC rely on mutation screening of *BRAF*, *RAS* genes, and *TERT* promoter. *BRAF*, *NRAS*, *HRAS*, and *KRAS* encode proteins that are key effectors of MAPK signaling pathway, an important kinase pathway, conserved in mammals [3]. **Objective: O**ur goal was to explore the canine TC's oncobiology, and to verify whether natural occurring canine TC could (or not) be set as suitable model to study its human homolog. **Methods:** We collected 57 samples (5 adenomas (9%), and 52 carcinomas (91%)), from which we performed DNA extraction from formalin-fixed paraffin-embedded tissues, PCR, and Sanger sequencing of exon 16 of BRAF (n = 49), exon 2 of NRAS (n = 41), exon 3 of HRAS (n = 41), and exon 3 (n = 31) and 4 (n = 20) of KRAS. **Results:** We detected silent mutations on *HRAS* (p.N47=) (n = 14/41, 34%) and *NRAS* (p.E63=) (n = 1/41, 2.4%), however, no mutations were found in the other genes. **Conclusions:** Our results corroborate those described by Campos et al. (2014) [4]. Nevertheless, both studies only evaluated the homologous regions of the hotspots of human most common TC mutations. We cannot exclude the hypothesis that in dogs, those genes can present activating mutations in other exons, different from human's hotspots.

Keywords: Thyroid; tumours; dogs

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