

## **Analysis of a Cat Mammary Tumour (multinodular tubulopapillary carcinoma) by Comparative Genomic Hybridization: a case report**

S. Santos <sup>(1)</sup> A. Borges <sup>(1)</sup> F. Adegas <sup>(1)</sup> F. Gärtner <sup>(2)</sup>, H. Guedes-Pinto <sup>(1)</sup>, R. Chaves <sup>(1)</sup>

<sup>(1)</sup> Institute for Biotechnology and Bioengineering, Centre of Genetics and Biotechnology, University of Trás-os-Montes and Alto Douro (IBB/CGB-UTAD)

<sup>(2)</sup> Institute of Molecular Pathology and Immunology of the University of Porto (IPATIMUP)

An interesting opportunity for comparative oncology has been recognized in spontaneously occurring tumours in domestic animals and, more specifically, cat mammary tumours (CMT) are considered good models for their human counterpart. However, data regarding CMT cytogenetic characterization is very scarce, most especially at the molecular level. Genetic aberrations, such as gene amplification, deletions, and loss of heterozygosity are hallmarks of cancer and are thought to be major contributors for the neoplastic process. The comparison of total genomic DNA extracted from a tumour with total genomic DNA obtained from normal cells – Comparative genomic hybridization technique (CGH) allows the detection of individual genomic changes of high value for differential diagnosis and prognosis. On this respect the CGH technique demonstrates an evident clinical impact in oncology. In the present work, we present the study of a spontaneously occurring CMT by Comparative Genomic Hybridization analysis. As far as we know, this is the first time that a CGH analysis is described in a CMT. The CMT sample (comprising four mammary glands) presented a multinodular growth mass that was surgically removed from a 14 years old female cat and was histologically classified as being a tubulopapillary carcinoma. Simultaneously, genomic DNA was extracted for a CGH evaluation. The CGH analysis, with 95% of confidence, allowed the detection of deletion regions in chromosome X (p11p12, q31q33). We also identified gains in the telomere regions of chromosomes B1 (p13) and D3 (p15), and at the interstitial region of chromosome E1 (p14).