

New advances in Cat Mammary Tumours: Comparative Genomic Hybridization profiles in a case of tubulopapillary carcinoma

A. Borges ⁽¹⁾, S. Santos ⁽¹⁾, F. Adegá ⁽¹⁾, F. Gärtner ⁽²⁾, H. Guedes-Pinto, R. Chaves ⁽¹⁾,

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

⁽²⁾ Institute of Molecular Pathology and Immunology of the University of Porto (IPATIMUP)

The advent of Comparative Genomic Hybridization (CGH) has opened a reliable way for detection of all genomic imbalances in each tumor through a single analysis. Its utility is based on the concept that chromosome regions with increased copy-number reveal sites that may contain dominantly acting oncogenes, whereas regions with decreased copy-number may hSpontaneously Cat Mammary Tumours (CMT) offer a unique opportunity as cancer models for human breast oncology. Data regarding CMT cytogenetic characterization is very scarce, most especially at the molecular level. Moreover, the cytogenetic analysis of solid tumours has been a challenge area to work on; some of the technical hitches are difficulties in chromosome preparation and the complexity of the karyotypes displayed by these cells. In the present work, we analyze a CMT case by CGH that allowed the detection of most significant genomic imbalances. As far as we know, this is the first time that a GGH analysis is described in CMTs. Two mammary mass respectively with 1,5 cm and 4,5 cm diameter, were chirurgically removed from an 15 years old female cat. The tumour masses were histopathologically classified as a tubulopapillary carcinoma with identification of some solid pattern and necrosis areas. Simultaneously, gDNA was extracted for a CGH evaluation. The CGH analysis allowed the identification of several gains in all chromosomes with 99,99% of confidence. Chromosomes B2 (p12p15, p12q11, q31, q33, q33q36), C2 (p11, p23p24, q24q25, q26q28), D3 (p11p12, p13p14, p15, q22, q22q23), E1 (p12p14, q11, q13q14), E2 (p12q11, q13q14), E3 (p13, q11q13, q13) and F1 (q11q13, q23, q24) were the ones showing to be more prone to this type of genomic imbalances. In the future, it will be important to extend the analysis to a higher number of cases in order to correlate

the genomic imbalances detected with cancer critical genes. arbor putative suppressor genes.