

**Analysis of Genetic Alterations in Primary Nasopharyngeal Carcinoma by Comparative Genomic Hybridization (CGH).** *L. Sun<sup>1</sup>, Y. Fong<sup>2</sup>, J.S.T. Sham<sup>3</sup>, Y. Guo<sup>2</sup>, M. Deng<sup>2</sup>, Q. Liang<sup>2</sup>, H. Zhang<sup>4</sup>, H. Zhou<sup>4</sup>, H. Tideman<sup>1</sup>, J.M. Trent<sup>4</sup>, X.-Y. Guan<sup>3</sup>.* 1) Oral Maxillofacial Surgery, The University of Hong Kong, Hong Kong, PR China; 2) Cancer Center, Sun Yat-sen University of Medical Sciences, China; 3) Department of Clinical Oncology, The University of Hong Kong, Hong Kong; 4) Cancer Genetics Branch, NHGRI, NIH, Bethesda, MD.

To identify genetic alterations associated with the development and progression of human nasopharyngeal carcinoma (NPC), 57 tumors were analyzed using comparative genomic hybridization (CGH). In 47 cases chromosomal imbalances were found. Several recurrent chromosomal abnormalities were identified in the present study. The most frequently detected chromosomal gains involved chromosomes 12q (24 cases, 51%), 4q (17 cases, 36%), 3q (16 cases, 34%), 1q (15 cases, 32%), and 18q (15 cases, 32%). Common regions of gain involved 12q13-q15, 4q12-q21, and 3q21-q26. High copy number increases of chromosomal materials were detected in 4 chromosomal regions, 3q21-q26.2, 4p12-q21, 8p, and 12q14-q15. The most frequently detected loss of chromosomal materials involved chromosomes 16q (26 cases, 55%), 14q (21 cases, 45%), 1p (20 cases, 43%), 3p (20 cases, 43%), 16p (19 cases, 40%) 11q (17 cases, 36%) and 19p (16 cases, 34%). The most common regions of loss involved 14q24-qter, 1pter-p36.1, 3p22-p21.3, 11q21-qter, and the distal region of 19p respectively. Genomic alterations detected using CGH were compared and found to be largely consistent with those identified using banding analysis and loss of heterozygosity studies. However, several previously unrecognized recurrent alterations were also identified in the present study including: gain of 4q and 18q, and loss of 16q, 14q, and 19p. In addition, gain of 1q, 8q, 18q, and loss of 9q showed statistically significant association with advanced clinical stage ( $p < 0.05$ ). Identification of recurrent sites of chromosomal gain and loss identify regions of the genome that may contain oncogenes or tumor suppressor genes respectively which maybe involved in the tumorigenesis of NPC.