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SHORT COMMUNICATION

Preliminary findings of down-regulated genes in nasopharyngeal carcinoma

Edmund Ui-Hang Sim[1], Alan Kie-Leong Toh[1], and Thung-Sing Tiong[2]

[1]*Department of Molecular Biology, Faculty of Resource Science and Technology, University Malaysia Sarawak, Kota Samarahan, Sarawak, Malaysia*
[2]*Department of Surgery, Faculty of Medicine and Health Science, Universiti Malaysia Sarawak, Kuching, Sarawak, Malaysia*

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Abstract. The cause and mechanism of nasopharyngeal carcinoma (NPC) progression are multifactorial and multigenic in nature. Despite the increasing number of genes found to be linked with NPC, the comprehensive list of associated genetic factors remains incomplete and the precise molecular pathways to this cancer are largely undefined. Here we show early evidence of possible association between several genes and the tumourigenesis of NPC. By employing the GeneFishingTM DEG Technique that allows the comparative analysis of expression profiles between normal and tumour nasopharyngeal biopsy tissues, we have identified 10 differentially expressed genes. These genes were down-regulated in tumours relative to normal control and have never been brought into the context of NPC tumourigenicity. Our findings represent preliminary yet novel clues of several associative genetic factors to neoplastic malignancy of the nasopharynx.

Keywords. Differentially expressed genes, Nasopharyngeal carcinoma

INTRODUCTION

Nasopharyngeal carcinoma (NPC) is a carcinogenic malignancy of the nasopharynx tissue – the neoplasm usually originating at the high recesses near the opening of the Eustachian tube in the fossa of Rosenmuller. As explained by Carlos *et al.* (1990), it represents epithelial cell cancer that generally arises from tumourigenic development of squamous cell (80% of all nasopharyngeal tumours) at the lateral or posterosuperior walls of the nasopharynx. Based on histopathogical types, the World Health Organisation (WHO) 1991 classifies NPC as Type I – the keratinizing carcinoma, Type IIa – the non-keratinizing carcinoma, and Type IIb – the undifferentiated carcinoma. Type I is characterized by clear histological evidence of squamous differentiation and is the most common type. Types IIa and IIb have similar behaviour and contain undifferentiated squamous cell carcinomas and lymphoepitheliomas (carcinomas with lymphoid infiltration). To date, the molecular aetiology of NPC that correlates with mal-development of the cell/tissue of origin for each type of cancer is poorly understood.

The highest incidence of NPC is in South East Asia, predominantly among the Chinese (Marks *et al.*, 1998). In Malaysia, the population of Chinese descent represents the highest risk group (Prasad and Rampal, 1992). However, in the East Malaysian state of Sarawak, studies by Devi *et al.* (2004) conducted from 1996 to1998 revealed that the native population exhibits the highest age-standardized rates of NPC occurrence in the world. Literature, to date, has no evidence of established findings linking ethnicity and genetic susceptibility to NPC.

The occurrence of NPC can be associated with Epstein-Barr virus (EBV) infection (Raab-Traub *et al.*, 1983), especially for Type IIb (Teng *et al.* 1996). Recently, Lee *et al.* (2007) demonstrated that EBV tends to target and modulate differentially expressed genes in NPC cell lines, implicating an enhancer role of EBV in transforming epithelial cells to NPC. However, to date, the definitive mechanism of tumourigenesis as a consequence of EBV-NPC association is largely unclear.

Like most types of cancer, the occurrence of NPC is probably multifactorial in origin and multigenic in mechanism. Evidence for this can be found in the extensively

*Author for Correspondence.
Mailing address: Department of Molecular Biology, Faculty of Resource Science and Technology, Universiti Malaysia Sarawak, 94300 Kota Samarahan, Sarawak, Malaysia. Tel: 082-583041; Fax: 082-583160; Email: uhsim@frst.unimas.my