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Expression of Selected Inflammatory Cytokine Genes in Bladder Biopsies

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ABSTRACT

Besides the deregulation of oncogene and tumour suppressor gene, bladder carcinoma can also be caused by inflammation. To date, the association of inflammatory cytokines with carcinoma of the bladder (especially the transitional cell carcinomas) is not fully understood. In this study, we report an attempt to examine expression patterns of pro- and anti- inflammatory cytokine genes from normal and tumour tissue biopsies of the human bladder. Our molecular assays involved the use of the GeneXP™ Human Cyto-3 kit and the Reverse Transcription – Polymerase Chain Reaction test. Due to limitation in our experimental process, mainly attributed by inconsistencies in the results obtained between the two assay systems, we cannot reach a conclusion regarding the association of the six selected inflammatory cytokine genes (*IL-8*, *IL-12A*, *IL-18*, *TGF-β1*, TGF-*β2*, and *TGF-β3*) with bladder carcinoma. However, our data provided early novel evidence of expression of four inflammatory cytokine genes, namely *IL-12A*, *TGF-β1*, TGF-*β2*, and *TGF-β3* in tissues derived from the human bladder.

Keywords: Expression analysis, inflammatory cytokines, bladder biopsies

INTRODUCTION

Studies have shown that over-proliferation of cells due to deregulation of oncogenes and tumour suppressor genes, and certain epigenetic mechanism are not the only causative factors of cancer (Coussens & Werb, 2001). In fact, inflammation and infections has been suggested to be amongst the triggers of tumour initiation (Lin & Karin, 2007). Inflammation-mediated carcinogenesis can be explained, in part, by the activation of the inflammatory cells that release mutagenic oxidant-generating enzyme (Michaud, 2007), which may cause irreversible genomic alterations in proliferating epithelial cells (Coussens & Werb, 2007).

According to Michaud (2007), the association between inflammation and carcinogenesis in squamous cell carcinoma of the bladder accounts for the high occurrence of this subtype of bladder cancer in many developing countries where parasitic infection by *Schistosoma haematobium* is common. However, evidence of such association in cases of transitional cell carcinoma (TCC) of the bladder, a predominant subtype in most developed country and also in Malaysia, is less clear. In this short report, we reveal an attempt to investigate inflammation as a potential contributing event in local cases of transitional cell carcinoma of the bladder. We sought the strategy of simultaneous expression analysis of selected pro- and anti- inflammatory cytokine genes in normal and tumour bladder biopsies. Our hypothesis was that differential expression of inflammatory cytokine genes between normal and tumour biopsies, and also between pro- and anti-inflammatory cytokines would form the basis to suspect a link between inflammation and carcinogenesis of TCC.

MATERIALS & METHODS

Sample collection and total RNA isolation

Normal bladder and tumour tissue biopsies were from patients admitted to the Sarawak

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