Tropical Biomedicine 23(1): 53–59 (2006)

Quantitative analysis of the expression of *p53* gene in colorectal carcinoma by using real-time PCR

Ivyna Bong Pau Ni[1], Patricia Lim[2], Pauline Balraj[2], Edmund Sim Ui Hang[3] and Zubaidah Zakaria[1]
[1] Haematology Unit, Cancer Research Centre, Institute for Medical Research, Jalan Pahang, Kuala Lumpur, Malaysia
[2] Molecular Pathology Unit, Cancer Research Centre, Institute for Medical Research, Jalan Pahang, Kuala Lumpur, Malaysia
[3] Faculty of Resource Science and Technology, Universiti Malaysia Sarawak, Kota Samarahan, Sarawak, Malaysia

Abstract. Colorectal carcinoma ranks third among ten leading causes of cancer in Malaysia. The colorectal carcinoma tumourigenesis involves the inactivation of tumour suppressor genes, and activation of proto-oncogenes. The *p53* is one of the tumour suppressor genes that is involved in the colorectal carcinogenesis. The *p53* gene is located on human chromosome 17p13.1 and comprises of 11 exons. Deficiencies in the *p53* gene can cause the cancerous cells to spread to distant organs such as liver, lungs, lymph nodes, spine and bone. The most common *p53* abnormalities that can lead to the metastasis of colorectal tumours are mutation and deregulation of the gene. In this study, nine colorectal carcinoma samples were used to establish a simple and sensitive strategy in the study on *in vivo p53* expression by using real-time LightCycler SYBR Green I technology.

INTRODUCTION

In Malaysia, colorectal carcinoma is the third leading cause of cancer in the population (Lim & Halimah, 2003). Colorectal carcinoma develops through a cascade of histopathological and genetic defects, which is known as adenoma to carcinoma sequence (Kinzler & Vogelstein, 1996). The *p53* genetic aberration is involved in the late stage of colorectal tumourigenesis (Vogelstein & Kinzler, 1996). This tumour suppressor gene has been mapped to human chromosome 17p13.1 and it plays an important role in cellular growth control (McBride *et al.*, 1986). Under stress condition, the wild type *p53* binds to DNA, stimulates transcription of several genes and mediates cell cycle arrest in the G1 phase for DNA repair or initiates apoptosis when the damaged DNA cannot be successfully repaired (Kumudini *et al.*, 2002). If the *p53* gene is mutated, the damaged DNA remains unrepaired and mutations become fixed in the dividing cells. Subsequently, malignant transformation of the cancerous cells occurs and starts to accumulate the mutated *p53* gene (Kumudini *et al.*, 2002).

Consequently, mutations and deregulation of the *p53* gene are commonly found in cancers (Wunderlich *et al.*, 2000). More than 75% of colorectal carcinomas are due to mutation in the *p53* gene (Erhan *et al.*, 2002), while over-expression of the gene is associated with advanced malignancies (Auvinen *et al.*, 1994). Recent preliminary reports revealed that over-expression of *p53* are an indicator of poor prognosis and survival in the patient (Remvikos *et al.*, 1992; Sun *et al.*, 1992; Zhao *et al.*, 2005). However, other reports correlated low expression of the *p53* gene and adverse outcome (Kumudini *et al.*, 2002). Whether *p53* expression level is associated with patient survival and stages of the diseases in colorectal tumour is still uncertain and remains to be scrutinized.