

## Risk modification of colorectal cancer susceptibility by interleukin-8 -251T>A polymorphism in Malaysians

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### Abstract

**AIM:** To investigate the allele and genotype frequen-  
cies and associated risk of interleukin (*IL*)-8 -251T>A  
polymorphism on colorectal cancer (CRC) susceptibility  
risk.

**METHODS:** Peripheral blood samples of 255 normal  
controls and 255 clinically and histopathologically con-  
firmed CRC patients were genotyped for *IL*-8 -251T>A  
polymorphism employing allele-specific polymerase chain  
reaction. The relative association of variant allele and  
genotypes with CRC susceptibility risk was determined  
by calculating the odds ratios (ORs). Corresponding  $\chi^2$   
tests on the CRC patients and controls were carried out  
and 95% confidence intervals (CIs) were determined  
using Fisher's exact test. The allele frequencies and its  
risk association were calculated using FAMHAP, haplo-  
type association analysis software.

**RESULTS:** On comparing the frequencies of genotypes

of patients and controls, the homozygous variant AA  
was significantly higher in CRC patients ( $P = 0.002$ )  
compared to controls. Investigation on the association  
of the polymorphic genotypes with CRC susceptibility  
risk, showed that the homozygous variant *IL*-8 -251AA  
had a significantly increased risk with OR 3.600 (95%  
CI: 1.550-8.481,  $P = 0.001$ ). In the case of allele fre-  
quencies, variant allele A of *IL*-8 -251 showed a signifi-  
cantly increased risk of CRC predisposition with OR 1.32  
(95% CI: 1.03-1.69,  $P = 0.003$ ).

**CONCLUSION:** Variant allele and genotype of *IL*-8 (-251  
T>A) was significantly associated with CRC susceptibil-  
ity risk and could be considered as a high-risk variant  
for CRC predisposition.

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**Key words:** Interleukin-8 -251T>A; Polymorphism; Colo-  
rectal cancer; Malaysians

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### INTRODUCTION

Colorectal cancer (CRC), the incidence of which has been  
increasing worldwide for the past few years, represents  
a significant cause of morbidity and mortality. CRC de-  
velops as a result of progressive accumulation of genetic  
and epigenetic alterations that lead to a series of histo-