Short communication

All serotypes of dengue virus induce HLA-A2 major histocompatibility complex class I promoter activity in human liver cells

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1. Introduction

Dengue virus (DV) belongs to the \textit{Flaviviridae} family and comprises four antigenically distinct serotypes, DV1 to DV4, which are transmitted to humans primarily by the mosquito bites of \textit{Aedes aegypti}.\textsuperscript{1} Dengue diseases affect over 100 million people worldwide each year, with more than 2.5 billion people at risk for endemic transmission.\textsuperscript{1} While the past decade has witnessed important advances in DV pathology, epidemiology, immunology, structural biology and pharmacology, the pathogenesis of DV infection remains a challenging puzzle. Elucidation of the immunological responses to flaviviruses has clearly shown a complex interplay of viral and host factors, with both innate and adaptive elements playing important roles in reducing viremia and viral clearance.\textsuperscript{2}

To evade the host’s immune response, viruses have evolved several mechanisms that block the major histocompatibility complex (MHC) class I antigen presentation pathway.\textsuperscript{3} Many viruses escape this by down-regulating surface expression of the MHC class I molecules. In contrast, flaviviruses such as West Nile Virus (WNV) and hepatitis C virus (HCV) have been reported to increase the MHC class I antigen presentation.\textsuperscript{3} This led us to investigate the effect of dengue virus on the MHC class I gene, in particular the HLA-A2 gene, in dengue-infected human cells.

2. Materials and Methods

2.1. Construction of Luciferase Reporter plasmids

Genomic DNA was isolated from HepG2 cells using DNA Extraction Kit (Qiagen, Germany) according to manufacturer’s instruction. A DNA fragment of the HLA-A2 promoter was amplified using primer set HLA420F (5’-CCCCCCCCCTCGAGGGACAGAGAT-3’) and HLA420R (5’-CTCTTAAAGCTTCTCGGGCTTGT-3’) to generate a 420 bp