

ImmunoTools *special* Award 2016



Aroonroong Suttitheptumrong, MSc

Supervisor: Sa-nga Pattanakitsakul, D.Med.Sc
Associate Professor

Division of Molecular Medicine, 4th Fl. SiMR Bldg.
Faculty of Medicine Siriraj Hospital, Wanglang Rd.
Bangkok 10700, Thailand

Identification of mechanism of endothelial dysfunction induced by association of TNF- α and dengue virus infection

Dengue virus (DENV) infection in human is transmitted by the bites of infected *Aedes aegypti* and *Aedes albopictus* mosquitoes. This virus causes dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS) which are still epidemic diseases in tropical and subtropical regions. DHF is characterized by high fever and hemorrhagic phenomenon that is associated with reduction of the number of platelets, increase in capillary permeability and hepatomegaly. In DSS, the patient is suddenly deteriorated from hypovolemic shock, which occurs as a consequence of significant plasma volume loss by dengue virus infection (*Thein S et al, Am J Trop Med Hyg 1997; 56(5):566-72*).

The hallmarks of severe dengue infection are manifested with thrombocytopenia, vascular leakage and shock. Endothelial cells are the target sites involved in dengue pathogenesis which may be results from dengue infection and/or immune response mediated by cytokines or chemokines of activated lymphocytes. Although the pathogenesis of DENV-related disease remains unclear, viral virulence and immune responses have been proposed to play an essential role in DHF and DSS (*Gubler DJ, Clin Microbiol Rev 1998;11(3):480-96*).

Other factors such as complement activation, platelet activation, and the production of potentially cytotoxic cytokines [including tumor necrosis factor- α (TNF- α), interleukin (IL)-1 and IL-6] by macrophages, lymphocytes and endothelial cells have been suggested to increase the capillary permeability, the hallmark of DHF (*McBride WJ and Bielefeldt-Ohmann F, Microbes Infect 2000;2(9):1041-50; Rothman AL and Ennis FA, Virology 1999;257(1):1-6*).

The present proposal is aimed to observe the alteration of membrane proteomes during induction of TNF- α and dengue virus infection. The isolated membranes from human endothelial cells will be subjected to SDS-PAGE and mass spectrometry analysis. The altered proteins between mock - and treated - cells will be functionally

categorized and analyzed further for the functional effect related to the permeability changes and pathogenesis of DENV infection.

ImmunoTools *special* AWARD for Aroonroong Suttitheptumrong

includes 25 reagents

FITC - conjugated anti-human CD8, CD69, HLA-DR, Control-IgG1, Control-IgG2a, Control-IgG2b

PE - conjugated anti-human CD4, CD61, rh IFNgamma, Control-IgG1, Control-IgG2a, Control-IgG2b

PerCP - conjugated anti-human CD3, CD20, Control-IgG1, Control-IgG2a, Control-IgG2b

APC - conjugated anti-human Control-IgG1, Control-IgG2a, Control-IgG2b

Multicolour combinations anti-human:

CD3 **FITC** / CD4 **PE** / CD45 **PE-Dy647**

CD3 **FITC** / CD8 **PE** / CD45 **PE-Dy647**

CD3 **FITC** / CD4 **PE** / CD19 **APC**

CD4 **FITC** / CD3 **PE** / CD8 **PerCP**

CD4 **FITC** / CD8 **PE** / CD45 **PerCP**

recombinant human cytokines: rh TNF-alpha

[DETAILS](#) more [AWARDS](#)