Project Code : TRG5780207

Project Title : "The polymicrobial sepsis and fungal sepsis severity of Fc gamma receptors IIb decicient induced Systemic Lupus Erythematosus mice with sepsis and monocyte/macrophage signaling"

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Abstract:

Because FcγRIIb de-funntioning polymorphism is the common genetic association with Systemic Lupus erythematosus (SLE) in Thai population, we explored the immune response to infections of FcγRIIb deficient mice, a model mimic FcγRIIb de-funntioning polymorphism, for a more understanding in the infectious immune response of SLE patients. Defunctioning-polymorphisms of FcγRIIb, an inhibitory receptor, results in the hyper immune response and increase the possibility of spontaneous auto-antibody production. Then FcγRIIb deficient mice have spontaneous anti-dsDNA at 8-wk-old and proteinuria, a manifestation of SLE, at 24-wk-old. We test the response to polymicrobial sepsis with cecal ligation and puncture (CLP) and fungal sepsis with *Cryptococcus neoforman* injection.

Surprisingly, FcγRIIb deficient mice were susceptible to Cryptococcosis (a fungal infection) but not to polymicrobial bacterial infection. Then we focus on the immune response to Cryptococcosis. We found that the unique properties of FcγRIIb deficient macrophage, apparent phagocytosis with prominent pro-inflammatory cytokines production, with the intracellular ability of *C. neoforman* leaded to the cryptococcosis susceptibility.

We proposed that the test for FcγRIIb polymorphisms should be done in patients with SLE or patients with the family history of immune-competent cryptococcosis, especially in the endemic area. These patients might be highly susceptible to cryptococcosis and need a special concern and ealier clinical recognition of disseminate cryptococcosis.

Keywords : 3-5 words FcγRIIb deficient, Polymicrobial sepsis, Cryptococcosis

Final report content:

Abstract