**Date / Time:**
Tuesday, 6 Sept 2011
12pm – 1pm

**Venue:**
CeLS Auditorium
Centre for Life Sciences,
Level 1,
28 Medical Drive
Singapore 117456

**Convener:**
Dr Zhang Yongliang

**ALL ARE WELCOME**

Visit our website @
www.med.nus.edu.sg/mbio
for more upcoming seminars

---

“**A Complex Interaction: Toll-like Receptors And Autoimmunity.**”

*Dr. Anna-Marie Fairhurst*
Principal Investigator
Singapore Immunology Network, A*STAR

**Abstract**

SLE is a complex chronic autoimmune disease that is classically associated with the production of pathogenic autoantibodies to a wide spectrum of nuclear antigens. This leads to an immune complex deposition of self-reactive material culminating with cellular recruitment, cytokine production and overall inflammation within organs such as the kidney. The etiology of the disease is complex, with both environmental and genetic factors playing a role. Over the last few years, there have been an increasing number of reports demonstrating that exuberant innate mechanisms, particularly TLRs, play a role in the onset and progression of SLE. TLR3, TLR7 and TLR9 are localised intracellularly and recognize nucleic acid components of dsRNA, ssRNA and dsDNA respectively. Initial scientific interest for a role in SLE arose from the presence of anti-RNA and anti-DNA antibodies in patients with lupus. Over the last decade, both mouse and human studies have shown that TLR7 plays an important part in disease pathogenesis. We and others have previously shown that an increased activation of TLR7 is fundamental for disease progression in multiple murine models. Consistent with this, a loss of TLR7 ameliorates disease. However, unexpectedly, a loss in TLR9 drives disease in lupus prone autoimmune murine models. The Fairhurst lab is working to determine the immunological mechanisms of these two receptors in disease progression.

**Selected Publications**


- *Pisetsky DS, Fairhurst AM. The origin of extracellular DNA during the clearance of dead and dying cells. Autoimmunity. 2007 281-4.*