**Date / Time:**
Wednesday, 3 March 2010
12.00 nn

**Venue:**
Seminar Room @ Level 3
Department of Microbiology
MD4, 5 Science Drive 2
Singapore 117576

**Convener:**
Dr Veronique Angeli

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**“Monocytes: Subsets and Fate During Inflammation”**

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Professor Gwendalyn J Randolph is a graduate of the Temple University at Philadelphia, Pennsylvania (B.S.; specializing in Biology) and State University of New York at Stony Brook (ph.D.; specializing in Immunology and Molecular Pathology). In 1995, she was a Postdoctoral Fellow at The Rockefeller University and from 1997 to 1998, a Postdoctoral Associate at Cornell University Medical College in New York. Currently, Professor Randolph is a Professor at Mount Sinai School of Medicine, New York. The research of Professor Randolph’s group integrates the study of monocyte-derived cells, and dendritic cells with vascular and lymphatic vessel biology. They have pioneered assays to study migration of these immune cells to lymph nodes in the skin and lung of mice, and their work on tracking monocytes has led to advances in understanding the relationship of monocytes to the dendritic cell system in vivo.

**Abstract**

Human and mouse monocyte subsets have been described. To gauge how well mouse monocytes may serve as models for their human counterparts, we compared the gene expression profiles of the major monocyte subsets in each species using an ordered list algorithm. A remarkable degree of conservation between the species was apparent, although important differences in scavenger receptor and lipid metabolic pathways were identified. In the mouse, monocyte subsets can be further divided into 5 subsets based on their expression of MHC II. In the spleen, as in the blood, all five subsets are present. Most, but not all, steady state lymph nodes contain two of these subsets and their possible functions therein will be discussed. Finally, monocyte-derived cells have been shown to leave sites of inflammation by emigrating to lymph nodes through lymphatics. However, the mechanisms underlying their clearance via lymphatics remain obscure. Typically, dendritic cells (DCs) are more capable of entering lymphatics than macrophages. In this lecture, our ongoing studies whether monocyte differentiation to DCs is a key factor in resolution of acute inflammation will be presented, along with an evaluation of whether the mechanisms involved in DC migration are necessary for resolution. Then the possibility that these mechanisms are operative in the removal of monocyte-derived cells from a chronic inflammatory site, atherosclerotic plaques, during plaque stabilization will be discussed.