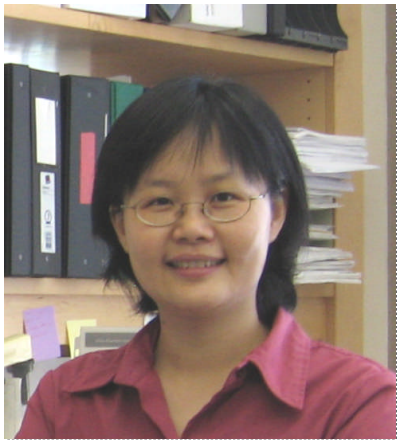

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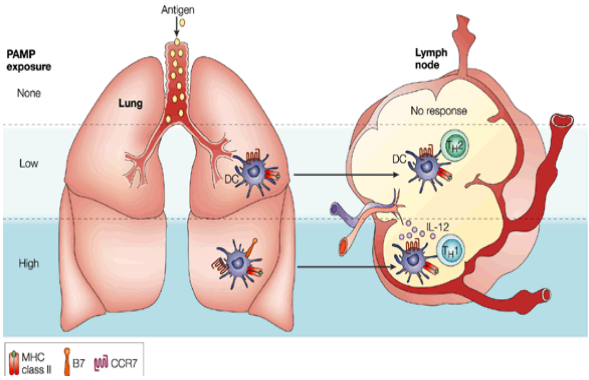
Cellular and Molecular Mechanisms in the initiation of airway inflammation

用氣喘小鼠模型來從細胞及分子的角度研究Toll-like receptor (TLR)是如何調控肺部樹突細胞(pulmonary dendritic cells) 的成熟與功能及樹突細胞被活化之後如何影響T helper cell的分化，並藉此來了解氣喘一開始是如何被prime。為期能達到這目的，研究將分成兩大部分：

(1)研究在肺部的上皮細胞中TLR4/MyD 88-dependent的訊號傳遞是如何影響樹突細胞的功能成熟。

(2)找出調控LPShi /LPSlo引起產生具對T細胞分化成Th1/Th2有誘導能力的肺部樹突細胞的因子及機制。



The response to inhaled antigen is only seen in the presence of a pathogen-associated molecular pattern (PAMP). However, the dose (level) of PAMP exposure determines the type (TH1 versus TH2) of T-cell response that is generated. CCR7, CC-chemokine receptor 7; DC, dendritic cell; IL-12, interleukin-12; TH, T helper.

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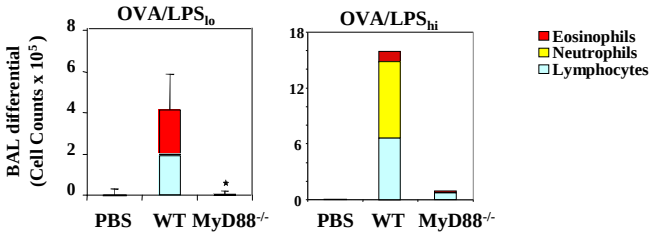
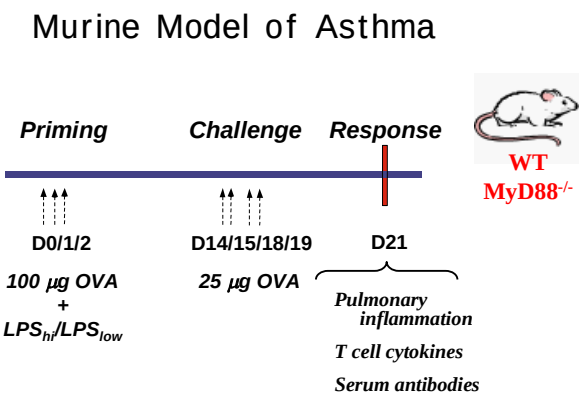


Fig.1: Immune responses to inhaled antigen are defective in MyD88 deficient mice. BAL: bronchoalveolar lavage fluid



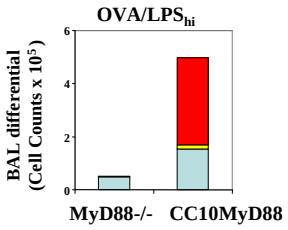


Fig 2: MyD88 signaling in epithelial cells is sufficient for the induction of Th2 immune responses to inhaled antigen with high dose LPS. In CC10MyD88 mice, MyD88 gene is only expressed in lung Clara cells, which comprise about 50-60% of murine respiratory tract epithelium.