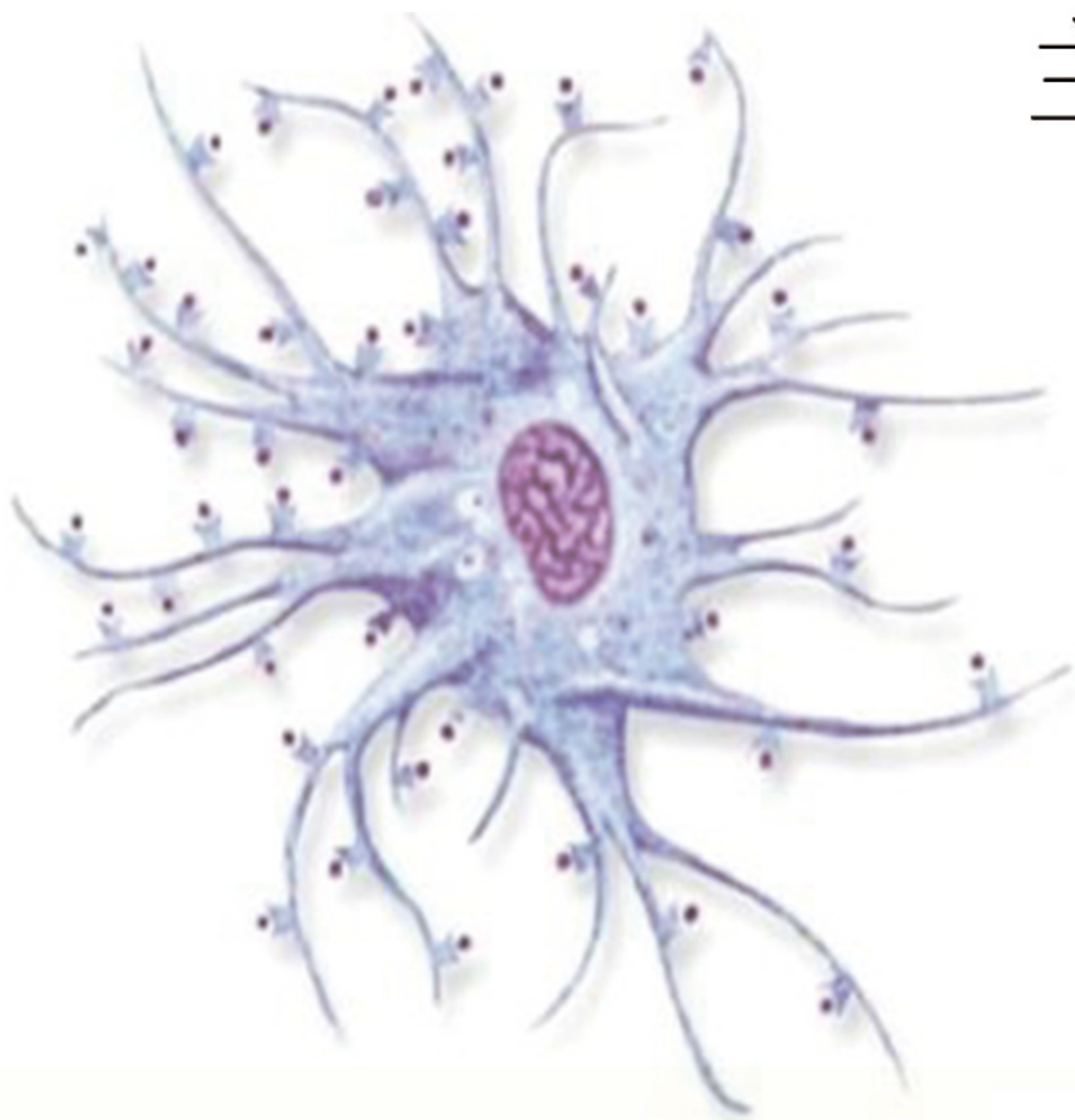


# 学术讲座

## Dendritic cell paralysis induced by antigen non-specific regulatory T cells 与铝盐佐剂相关的细胞水平机制研究



**主讲人** 石彦 教授 (清华大学医学院)

**时间** 2015.11.27 (星期五)

10:00a.m.-11:00 a.m. 报告

11:00a.m.-12:00 a.m. 圆桌讨论

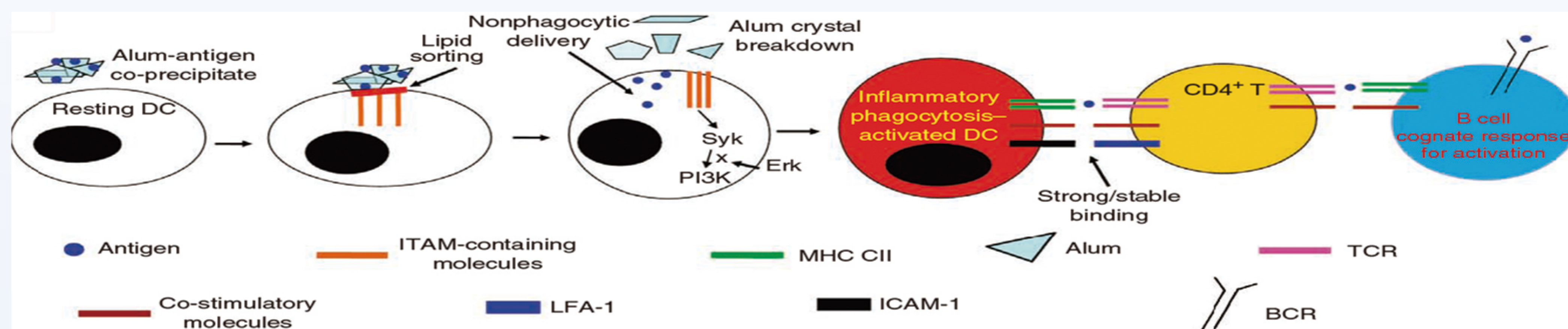
**地点** 报告：曾宪梓楼101报告厅

圆桌讨论：曾宪梓楼302会议室

**邀请人** 赵勤俭 教授

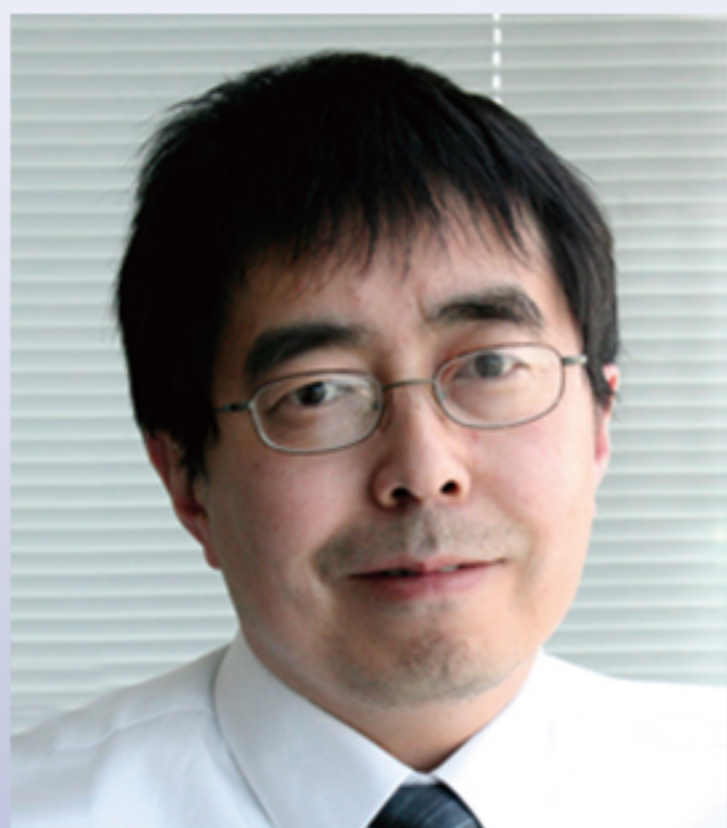
### 讲座内容：

Although regulatory T cells (Treg) target dendritic cells (DCs) for suppression, how this is accomplished remains elusive. We show by intravital microscopy that, during inflammatory immune activation, Treg cells are conditioned for prolonged, antigen non-specific contacts with DCs that reduce the ability of DCs to form stable adhesion with cognate T cells in vivo. In vitro force-spectroscopy and super-resolution imaging demonstrate that Treg cells express reduced levels of calpain, generate strong LFA-1-dependent DC-adhesive forces, and focus Fascin-1-dependent actin polarization of the DCs toward the Treg adhesion zone. While gradually reversible upon Treg disengagement, this polarized sequestration of essential cytoskeletal components paralyzes DCs, disabling contemporaneous antigen-specific T cell adhesion. These results suggest polyclonal Tregs function in part as feedback regulators that suppress DCs in a contact-dependent, antigen non-specific manner.



(Flach et al, *Nature Medicine* 2011, 17:479-487.)

### 主讲人简介：



**石彦**，现任清华大学教授。主要从事免疫学，树突状细胞生理，疫苗，自体免疫调控，以及原子力显微镜基础上的单细胞激活研究。科研成果包括从细胞浆中分离尿酸并证明它是哺乳动物免疫系统感受到的所谓危险因子，为以后这方面的研究打定了基础；找到了晶体和结石引起吞噬细胞炎症反应的机制，阐述了铝盐佐剂直接和树突状细胞表面的胆固醇和鞘脂结合而引起佐剂效应，在阐明应用多年的铝盐佐剂的作用机制方面提供了一些新的分子基础。