Statement of research achievements

Achivement: Distinct Poster Award and Travel award for this Presentation

M2-polarized macrophages attenuate influenza virus-induced acute lung injury. ***European Respiratory Society (ERS) Lung Science Conference- Estoril March 2014.***

Macrophage polarization has been extensively studied in different disease models. However, the functional profile of macrophage subsets of different polarization states in lung injury and resolution after infection has not been convincingly elucidated. We established a FACS protocol to characterize the polarization profiles of different subsets of resident and recruited macrophages within the lung after influenza virus (IV) and *K. pneumoniae* infection. These analyses showed that the resident alveolar and interstitial macrophage pool displays an M2-like phenotype throughout the time course of infection, suggesting low functional plasticity of the tissue-resident pool. In contrast, alveolar and interstitial exudate macrophages (ExMa's) showed an M1 phenotype ($CD40^{high}CD206^{low}$) during the acute phase of injury, and shifted to an M2 phenotype ($CD40^{low}CD206^{high}$) during recovery. This phenotypic switch was confirmed by qRT-PCR analyses in flow-sorted M1 and M2 ExMa´s. Further, when ExMa's sorted from IV-challenged wildtype mice were adoptively transferred into the lungs of IV-infected CCR2-/- mice (lacking ExMa recruitment), only transferred M2- but not M1-polarized ExMa significantly reduced IV-induced lung injury. In summary, our data demonstrate establishment of a FACS protocol to clearly differentiate M1 and M2 ExMa´s in different pneumonia models based on defined polarization markers and relate these polarization states to a functional phenotype, which is barrier-protective with respect to the M2 ExMa population. These findings highlight the broad functional repertoire of ExMa´s in pneumonia and will provide new macrophage-related therapeutic targets to attenuate pneumonia-induced lung injury.