ImmunoTools *special* Award 2018

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Characterization of the functional role of B cells in macrophages recruitment and polarization in arteriogenesis

Peripheral artery occlusive diseases are greatly associated with increased morbidity and mortality worldwide. Occlusion of arteries resulted in developing ischemic conditions in tissue and further necrosis if the blood supply not resumed. Arteriogenesis *(1)* is a natural bypass event in which pre-existed collateral arteries expands in their diameter to supply the demanded blood flow. Arteriogenesis is triggered by mechanical fluid shear stress but it requires molecular participation.

Finding the molecular signalling which is involved in arteriogenesis in order to develop non-invasive therapeutic applications to treat such occlusive diseases attain interest. Arteriogenesis occurs in an immunoregulatory manner by involvement of macrophages *(2)*. There are two different macrophages classed so far, M1 and M2 macrophages. M1 macrophages are considered as proinflammatory macrophages, whereas M2 macrophages are regulatory macrophages. Molecular signal from the tissue environment can polarize M1 macrophages into M2 macrophages. Different cytokines reported to be involved in this process, i.e. IL-2, IL-4, IL-6, IL-10, IL-13, IL-17, IL-23 etc. We are interested to understand the role of cytokines released by B cells and how they influence Arteriogenesis by recruiting and activating macrophages.

Our preliminary data shows us, reduced perfusion recovery in B cell depleted mice comparing with control mice. This data was reproduced in B cell deficient (JHT) mice. Arteriogenesis is initiated in an inflammatory phase and further continued in an anti-inflammatory phase, where M2 macrophages involve for reconstructing the arteries *(3)*. TNFα is the key cytokine involved in pro-inflammatory phase along with IL-6 from B cells. We would like to use TNFα and IL-6 ELISA kits from ImmmunoTools to check Cytokines levels in readily available serum samples and collateral tissue lysates from mice. Further we would like to analyse blood and tissue collected at different phases of arteriogenesis for B cells, T cells, their subsets and macrophages. We would like to use flow cytometry antibodies CD3, CD4, CD8a, CD45R and CD19 from ImmunoTools.

I would like to take the advantage from ImmunoTools 2018 award and use selected flow cytometry antibodies and cytokine ELISA kits to understand our hypothesis.

References:

(1) *Deindl, E. and Schaper, W. The art of arteriogenesis. Cell Biochem. Biophys. 2005; 43: 1–15*

(2) *O. Chillo, E.C. Kleinert, T. Lautz, M. Lasch, J.I. Pagel, Y. Heun, K. Troidl, S. Fischer, A. Caballero-Martinez, A. Mauer, A.R. Kurz, G. Assmann, M. Rehberg, S.M. Kanse, B. Nieswandt, B. Walzog, C.A. Reichel, H. Mannell, K.T. Preissner, E. Deindl. Perivascular mast cells govern shear stress-induced arteriogenesis by orchestrating leukocyte function. Cell Rep., 16 (2016), pp. 2197-2207*

(3) *Troidl C, Jung G, Troidl K, Hoffmann J, Mollmann H, Nef H, Schaper W, Hamm CW, Schmitz-Rixen T. The temporal and spatial distribution of macrophage subpopulations during arteriogenesis. Curr Vasc Pharmacol. 2013 Jan;11(1):5-12*

ImmunoTools *special* Award for **Kumaraswami Konda** includes 24 reagents

FITC - conjugated anti-mouse CD3e, CD4, CD8a, CD19, CD45, CD45R, NK-cells

PE - conjugated anti-mouse CD4, CD8a, CD19, NK-cells

APC - conjugated anti-mouse CD3e, CD4, CD8a, CD19, NK-cells

mouse ELISA-set (for one 96 plate): mouse IL-6, mouse TNF-a

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