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Inflammation at the Interface of Innate and Acquired Immunity

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HRP technique to identify α-smooth muscle actin-α-SMA, vimentin, myosin heavy chains-MHC, desmin, CD3, CD45, CD68, S100 protein and PCNA (DAKO specification). Sections were also stained for electron microscopy.

Results: The results of this study have shown that aortic atherosclerosis is characterized by the presence of a huge number of CD68-immunoreactive cells with lipid inclusions in the cytoplasm. This finding indicates the process of monocytes transition into foam cells. The finding of vimentin-immunoreactive foam cells (which points to their smooth muscle origin), suggests that these cells express scavenger receptors and competitively take part with macrophages in the accumulation of lipids and creation of foam cells. In the atherosclerotic lesion, there is also a huge number of cells which are immunoreactive to S-100 protein, which is generally characteristic of vascular dendritic cells.

Conclusions: Foam cells originate from macrophages (express CD68) and smooth muscle cells (express vimentin). At the earliest stage of atherosclerosis, monocytes and macrophages represent the main precursors of foam cells. From the stage of fatty streak, in parallel with synthetic activity, smooth muscle cells start to accumulate lipids. Antigen presenting dendritic cells in atherosclerotic aorta could play an important role in immune mechanisms during atherosclerotic lesion formation.

REGULATORY MECHANISMS IN LOW DOSE STREPTOZOTOCIN DIABETES INDUCTION

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Experimentally induced insulin dependent diabetes mellitus in rodents with multiple low dose of streptozotocin (MLD-STZ) has clinical and immunohistological features similar to those of human disease with T cells and macrophages playing a major pathological role. In bred strains of mice differ in their susceptibility to MLD-STZ diabetes induction, C_{57} BL/6 male mice being susceptible while BABL/C mice are partially resistant. It had been demonstrated recently that low dose cyclophosphamide sensitive $CD4^+$ $CD25^+$ $Foxp3^+$ cells downregulate spontaneous onset of diabetes in non-obese diabetic mice. There is also ample evidence that Th1/Th2 balance affect diabetes development.

We investigated whether the cyclophosphamide regimen enhance diabetes in susceptible C_{57}BL/6 mice and abrogate the resistance to disease induction in BALB/C mice and, we analyzed whether the effect of the lack of ST2 dependent signaling in Th-2 cells my affect disease induction.

Two injections of 20mg/kg body weight at the time of disease induction led to significant enhancement of diabetes induction in C_{57}BL/6 mice with significant increase of TNF production as evaluated by serum TNF level. However, this stimulatory effect of low doses of cyclophosphamide was not seen in BALB/C mice. In addition disease was enhanced in $ST2^{-/-}$ BALB/C mice in comparison with wild type BALB/C mice but cyclophosphamide did not have an additional disease enhancing effect as seen in C_{57}BL/6 mice.

Thus it appears that both Th1-Th17/Th2 balance and Treg cells maybe involved in susceptibility to diabetes depending on genetic back ground of the mice.