Pediatric Rheumatology

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9.1 Innate resistance of endothelial cells to the effects of glucocorticoids

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from 15th Paediatric Rheumatology European Society (PreS) Congress
London, UK. 14–17 September 2008

Published: 15 September 2008

Pediatric Rheumatology 2008, **6**(Suppl 1):S20 doi:10.1186/1546-0096-6-S1-S20

This abstract is available from: http://www.ped-rheum.com/content/6/S1/S20

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Glucocorticoids (GCs) represent a mainstay of treatment in most chronic inflammatory diseases. However, many forms of vasculitis relapse after cessation of GC medication or remain primarily unresponsive. Assuming the vasculature and especially endothelial cells (ECs) as origin of the inflammatory process we investigated the effects of GCs on ECs in comparison to monocytes. We treated human micro- and macrovascular ECs as well as human monocytes for 4 h and 16 h with GC or GC + TNF or pretreated ECs with GC followed by TNF stimulation. Testing several genes by quantitative RT-PCR as well as by microarray analysis considering more than 17,000 genes we found no significant gene expression changes in ECs neither with GC alone nor for GC on TNF-induced genes which was in strict contrast to the impressive GC-response of monocytes. We checked that all known GC receptor (GR) subtypes as well as potential cofactors of GC were readily expressed in untreated and inflammatory stimulated ECs. However, in immunofluorescence stainings we surprisingly found that GC treatment failed to induce a nuclear shift of the GR in ECs in contrast to a substantial shift in steroid-sensitive monocytes.

Summarizing our results indicate that the molecular prerequisites for the implementation of GC effects on gene regulation are not given in ECs in contrast to myeloid cells. The anti-inflammatory effects of GCs in vasculitis might primarily act on infiltrating leucocytes without targeting ECs which may be the molecular basis for symptomatic effects but lack of long-lasting success of GC-treatment in many forms of vasculitis.