Results: In human cells (fig. 1) the presence of antibody to CB3 enhanced the production of cytokines (IL-6 shown) in response to live virus (inactivated virus did not induce cytokines). Using dendritic cells from knockout mice (fig. 2) we were able to define a role for both specific antibody to CB3 and TLR7 in the induction of a response to CB3.

Conclusions: Some of the inflammatory syndromes that occur post-infection may be related to TLR induction that occurs only in the presence of viral antibodies. A role for TLRs in post-viral "autoimmune" diseases is postulated.

89 Effect of Glucocorticoids on Initial Gene Expression of Innate Host Defense Molecules of Human Monocytes Infected by Candida albicans

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Background: Glucocorticoids are widely used potent anti-inflammatory and immunosuppressive agents, which have long been recognized to predispose patients to invasive fungal infections. However, expression profiles of the multiple genes mediating altered immune responses to glucocorticoids are not well understood.

Objectives: We studied the temporal cascade of gene expression of innate host defense molecules by c-DNA microarray of normal human monocytes (MNCs) in the presence of two dosages of dexamethasone (D).

Methods: MNCs (1×10^7/ml) from 6 normal human volunteers were incubated with D (10μg/ml and 1μg/ml) for 16h. C. albicans (1×10^7/ml) was then added and incubated for 4h. RNA was extracted, labeled and hybridized onto custom printed cDNA human genome microarray chips harboring 42,000 genomic probes. Gene expression profiles of MNCs vs MNCs+D, MNCs+D vs MNCs+D+C, and MNCs+C vs MNCs+D+C were then analyzed. Up- or down-regulation was defined as log2 ratios >1 or <1.

Results: Genes encoding pro-inflammatory cytokines (e.g., TNFα, IL-1, IL-6, IL-18, and LIF), chemokines and chemokine receptors, (e.g., IL-8, Groα, Groβ, MIP1α, MIP1β, MIP3, MIP4, CCR1, and CCR7) showed decreased expression in D-treated MNCs. Expression of genes encoding Th1 and Th2-related molecules (TGF-β, CD86, IL-2R, and IL1RA) also was decreased. However, Candida infection in D-treated monocytes also induced up-regulation of genes expressing pro-inflammatory cytokines and chemokines, albeit not to a normal level. By comparison, genes encoding IL-12, MHC molecules (HLA-DMA, HLA-DOA, and CD86), TLR1, TLR3, and CD14 were down-regulated by D and were not affected by C. albicans, when compared with Candida infection of normal MNCs. No significant difference in gene expression was found between D dosages.

Conclusion: Dexamethasone inhibits both innate immunity and adaptive immunity by suppressing pro-inflammatory molecules and receptors, T cell cytokines and receptors, and MHC molecules, while concomitant infection by C. albicans reverses the D-induced down-regulation of genes encoding pro-inflammatory cytokines and chemokines.

90 Neutrophil and Monocyte Receptor Expression in Uncomplicated and Complicated Influenza A with Pneumonia

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Introduction: Influenza causes significant morbidity and mortality, especially in elderly and those with chronic heart/lung diseases, since they are