in invasive infections. The decreased expression of lectin pathway proteins in neonates may contribute to their extraordinary vulnerability for bacterial infections. Low MBL concentrations appear to be an important susceptibility factor for gram-negative sepsis, and low H-ficolin for gram-positive sepsis. In contrast, M-ficolin reflects phagocytic activity and was elevated in early-onset sepsis.

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MANNOSSE BINDING LECTIN (MBL) YY GENOTYPE IS ASSOCIATED WITH AN INCREASED RISK OF NECROTIZING ENTEROCOLITIS(NEC) IN PRETERM NEONATES IN NICU

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Background: MBL initiates complement activation and could have a role in the pathogenesis of ischemia-reperfusion pathologies, such as NEC. We investigated the association between MBL2 genotype, the development of NEC and the MBL expression on specimens of bowel tissue in a cohort of preterm neonates in NICU.

Material and methods: We determined the MBL2 genotype in 118 preterm neonates admitted in NICU. The exon 1 SNPs 52,54, 57 and SNPs -550 and - 221 of the promoter region of the MBL2 gene were also determined. Immunoistochemical analysis was performed on 16 bowel bioptic paraffin-embedded specimens, collected during surgery in neonates with NEC.Clinical cases and summary results:21/118 neonates developed NEC. The analysis of the polymorphisms in the promoter region at -221(variant X/Y) showed that the frequency of the YY genotype was 85.7% in the neonates with NEC and 53.6% in the controls(p=0.013). Adjusting for gestational age, the association between YY genotype and the development of NEC was still significant(OR=5.02;p=0.015). MBL was highly expressed in anastomotic healthy tissue, in

enterocytes and istiocytes. No MBL staining was detectable in the necrotic bowel tissue. Neonates MBL low and deficient producer showed negligible or weak staining for MBL.**Conclusions:** YY genotype of the promoter of the MBL2 gene appeared to increase the risk of developing NEC. The epithelial localization of MBL in the bowel is consistent with a role for MBL in developing full-thickness necrosis during NEC.

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MICROARRAY INVESTIGATION OF HOST RNA EXPRESSION PROFILES IN NEONATAL INFECTION

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Background and aims: Infection causes significant neonatal morbidity and mortality. Currently available methods for diagnosing infection are unreliable. We aimed to examine differences in host RNA expression profiles between infants with confirmed infection and control infants using microarray technology.

Methods: RNA was extracted from neonatal whole blood taken from infants with confirmed infection and from controls using a modified PAXgene™ Blood RNA system protocol. High quality RNA was run on Illumina® Human Whole-Genome Expression BeadChip microarrays. Normalised, validated microarray data was analysed to examine differences between control and infected samples. Functional annotation according to gene ontology and pathway analysis was performed.

Results: 28 infected and 35 control samples were examined. Differential gene expression between infected and control groups was analysed: 448 features had >2-fold up-regulation and 341 features >2-fold down-regulation (p< 0.001) in infected compared to control infants. There was significant immune-related differential gene expression. Up-regulated genes in the infected group included genes involved in cytokine, complement, interferon and Toll Like Receptor related processes. Down-regulated genes included genes involved in antigen processing, MHC II activity and T cell activation and signalling.