Background and aims Device-related infections are thought to be initiated by adhesion of the bacteria to a medical device, followed by colonisation and mature biofilm formation. Preterm infants are susceptible to device-related infections caused by *Staphylococcus epidermidis*. Also, preterm infants have lower levels of antimicrobial peptides, including human cathelicidin antimicrobial peptide LL37, a condition that in part may explain their increased vulnerability. Our aim was to evaluate the effect of peptide LL37 on 1) the expression of biofilm-associated genes and 2) biofilm mass, by using an *in vitro* model.

Methods Biofilm formation of *S. epidemidis* was studied on intra vascular catheter pieces and in culture plates, in the absence or presence of LL37. Bacterial biofilm mass was investigated by scanning electron microscopy (SEM). Changes in biofilm-associated gene expression was determined by real-time polymerase chain reaction.

Results Tissue-like concentration of the peptide down-regulated most of the investigated genes after 2 h. A diminished biofilm mass was seen on the catheter surface by SEM after 24 h incubation.

Conclusions Peptide LL37, as part of innate immune defense of the newborn infant is crucial for the regulation of the commensal flora, including *Staphylococcus epidermidis*. A diminished activity of LL37, as found in preterm infants, may contribute to increase their risk of device-related infections.

PS-303

VITAMIN D INCREASES ROI PRODUCTION IN TERM AND PRETERM INFANTS: POSSIBLE MECHANISM OF ENHANCED ANTIBACTERIAL EFFECT

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Introduction Newborn infants are at risk of vitamin D deficiency and various studies implicate vitamin D as having immunomodulatory effect. Adequate generation of reactive oxygen intermediates (ROI) by neutrophils (PMN) during sepsis is bactericidal. However, production of neutrophil oxidase activity in the presence of sepsis is impaired in neonates.

Aim To examine the *in vitro* effect of 1, 25(OH)₂D₃ on whole blood PMN and monocyte ROI, TLR4, CD11b in newborn infants during sepsis *in vitro*.

Methods Whole blood from preterm infants <32 weeksgestation within 24 h of birth, cord blood from term infants and adult controls were analysed for phagocytic expression of Toll-Like Receptor 4 (TLR4; pathogen recognition); CD11b (chemotaxis and adhesion) and ROI production (bacterial kill) using flow cytometry. These were assessed in response to Lipopolysaccharide (LPS; Endotoxin; *in vitro* sepsis) and 1,25(OH)₂D₃.

Results ROI production from preterm and term neonatal neutrophils incubated with LPS alone was not significantly increased in contrast to adults. However pre-incubation with 1,25 $(OH)_2D_3$ before adding LPS demonstrated a significant increase (p=0.001) in ROI production for both preterm and term infants while simultaneous LPS and 1,25 $(OH)_2D_3$ had no effect. Conclusion New born infants were hypo-responsive in the presence of sepsis *in vitro* which recovered on pre-treatment with 1,

25(OH)₂D₃. Pre-treatment with vitamin D may improve term and preterm infants' antibacterial responses.

PS-304

NEONATAL ENTEROVIRUS INFECTIONS REPORTED IN FRANCE, 2012

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Enteroviruses (EV) are among the most common viruses infecting humans. One third of EV infections concern children under 1 year. Neonatal EV infections lead to a wide range of clinical manifestations, from mild febrile illness to severe, potentially fatal sepsislike conditions with multiorgan failure.

EV detections by serotype were reported by the "National Reference Centre for Infections EV" Lyon, on a monthly basis. Demographic, clinical and biological data are also collected in neonates hospitalised in 2012 for EV infection. Two sub-groups were identified according to the beginning of symptoms before or after 8 days of life (D8). There were 120 neonatal EV infections. Before D8, children with severe infection were born more prematurely with a low birth weight. EV most commonly detected in neonates included CVB4 and E11. Risk factors of severe EV infections included liver (73% before D8) and haematological damages (thrombocytopenia 82% and coagulopathy 64% before D8).

This study suggest a systematic serotyping of neonatal EV infections and biological monitoring of liver function to early identification of children at high risk of clinical severity and fatality.

PS-305

HUMAN PARECHOVIRUS 3 AS A CAUSE OF NEONATAL INFECTION

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Background Human parechoviruses (HPeVs) belong to *Parechovirus* genus and have recently been added to the *Picornaviridae* family. Their epidemiology, pathogenicity and virulence is only beginning to be understood. Neonates, infants and young children seem to be the most susceptible subjects. The clinical presentation is similar to that of enterovirus infections. HPeV type 3 has been reported to cause neonatal infection, presenting with central nervous system symptoms or a sepsis-like illness.

Objective As part of a prospective study on neonatal sepsis, we aimed to assess the importance of HPeV as a cause of infection in the neonatal period.

Materials and methods During the period October 2012 – December 2014, term newborns (0–28 days) admitted to the