

## Introduction

Infection is still an important cause of neonatal morbidity and mortality despite development of broad spectrum antibiotics and clinical advances in life support therapy. Factors which contribute to the high susceptibility of newborn infant to infection include prematurity (influence of very low birth weight and also immune immaturity), maternal genital colonization, transplacental spread following maternal infection, traumatic delivery, invasive procedures such as arterial or umbilical catheterization and underlying problems such as heart disease or hyaline membrane disease (**Eicher and Annibale, 2002**).

For the pediatricians and neonatologists who care for term and preterm infants, the challenge remains to keep these infants free of infection after delivery in special-care nurseries and neonatal intensive care units. Studies of complications associated with term infants at risk due to maternal factors, as well as preterm infants after early delivery, have demonstrated that sepsis is a major cause of neonatal mortality and morbidity (**Venkatesh and Garcia-Prats, 2008**).

Currently initial diagnosis is based upon clinical suspicion accompanied by nonspecific clinical signs and is confirmed upon positive microbiologic culture results several days after institution of empiric therapy. There exists a significant need for rapid, objective, in vitro tests for diagnosis of infection in neonates who are experiencing clinical instability (**Kingsmore et al., 2008**).

Exposure to microorganisms and their derived products triggers a rapid and coordinated sequence of host reactions resulting in recruitment

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of leukocytes into areas of inflammation or sites of microbial invasion (**Cavaillon et al., 2003**).

The production, release and function of neutrophil granulocytes in newborns are suppressed as well as the ability to increase the circulating neutrophil pool in response to an infection (***Källman et al., 1998 and Carr, 2000***).

Reduced neutrophil granule content might contribute to a decreased bactericidal capacity in neonatal neutrophils (**Ambruso et al., 1984, Bektus et al., 1990, and Levy et al., 1999**).

Human neutrophil lipocalin (HNL) is a newly discovered protein from human neutrophil secondary granules. It is regarded as a specific marker of neutrophil activity. It is located in bone marrow cells as well as lung, bronchial and colonic epithelial cells (**Carlson et al., 2002**).

Increased concentrations of HNL have been demonstrated in the sera of patients with acute bacterial infections, and HNL appears to be more specific and sensitive in the distinction between viral and bacterial infections (**Fjaertoft et al., 2005**).

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