

## **Introduction**

Despite improved neonatal care over the past decades, infections remain common and sometimes life-threatening in neonates admitted to the neonatal intensive care unit (NICU) (*Stoll et al., 2005*).

\* The diagnosis of infection in a neonate remains challenging. Signs and symptoms can be subtle, yet infections can evolve rapidly to sepsis with a potentially fatal outcome (*Fanoroff, 2007*).

For many years, a search has been ongoing to find predictors for neonatal sepsis that identify effectively patients who are at risk of infection (*Ahrens et al., 2004*).

Low levels of certain proteins of the innate immune system at birth were recently identified as being associated with neonatal sepsis. Of these proteins, mannose-binding lectin (MBL) (*Swierko 2009*)

MBL activates the lectin pathway of the complement system by binding to various micro-organisms. This leads to opsonization and enhanced phagocytosis (*Dzwonek, 2008*).

Three structural mutations in exon-1 of the MBL2 gene interfere with the assembly of the protein and cause decreased functional MBL plasma levels (*Garre et al., 2003*).

MBL deficiency has been associated with an increased susceptibility to infections (*Neth, 2004*).

The detection of MBL deficiency at birth should be based on actual MBL plasma levels rather than on MBL2 genotype (*Frakking et al., 2006*).