

Introduction

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Respiratory distress syndrome (RDS), also called hyaline membrane disease, is the most common respiratory cause of infant mortality (*Martin et al , 2005*).

The risk of developing RDS increases with decreasing birth weight; the incidence of this disease is estimated at 80% for infants weighing <750 gm at birth and 55% for infants weighing <1000 gm (*Koivisto et al , 2004*).

RDS in premature infants is caused by a structural immaturity of lungs and the insufficient production of surfactant and its incidence is inversely related to gestational age (*Whitsett et al , 2005*).

Preterm infants with lower VEGF suffered prolonged and more severe respiratory distress. These data suggested that VEGF might be a marker of pulmonary maturity (*Lassus et al , 2001*).

An animal study showed that hypoxia-inducible transcription factor-2 α and its downstream target, VEGF, were critical for fetal lung maturation (*Compernelle, et al , 2002*).

Previously, it is demonstrated that infants with severe RDS had less vascular endothelial growth factor (VEGF) in their tracheal aspirate fluid during the early postnatal period than infants with milder RDS (*Lassus et al , 1999*).

Vascular endothelial growth factor (VEGF) is a pluripotent growth and permeability factor that has a broad impact on endothelial cell function. The lung tissue is very rich in this protein; many different lung cells produce VEGF and also respond to VEGF (*Voelkel et al, 2005*).