The Role of ASB3 Gene Single Nucleotide Polymorphism in the Progression to Bronchopulmonary Dysplasia in Extremely Low Birth Weight Infants

Anna Zylak
New York Medical College

Leewen Hsu
New York Medical College

Asma Amin
New York Medical College

Shaili Amatya
New York Medical College

Sharina Rajbhandari
New York Medical College

See next page for additional authors

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Authors
Anna Zylak, Leewen Hsu, Asma Amin, Shaili Amatya, Sharina Rajbhandari, Alexander Feldman, Umesh Paudel, and Lance A. Parton

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Bronchopulmonary dysplasia (BPD) is a major cause of neonatal morbidity in Extremely Low Birth Weight (ELBW) infants. The pathogenesis of BPD is very complex and has a number of mechanisms: it is associated with smooth muscle proliferation; there is airway epithelial and vascular endothelial cell dysregulation; these changes occur under the influence of hyperoxia, barotrauma and inflammation; and, there is a strong genetic foundation.

Mechanical stress (i.e. mechanical ventilation) and hyperoxia (i.e. free radicals) may promote bronchial myogenesis, causing both hypertrophy and hyperplasia of the myocytes. The changes in the muscular layer of the airways place many infants at higher risk for subsequent wheezing disorders in infancy, childhood and adulthood. Among the candidate genes that may influence smooth muscle cell changes is the ASB3 gene, located on chromosome 2. It is a member of the ankyrin repeat and SOCS box-containing family of proteins. A recent study reported that genetic variants of the ASB-3 gene are associated with asthma and bronchodilator responsiveness to inhaled β2-agonists. ASB proteins may also play an important role at various levels of myogenesis (figure 1). The changes in the muscular layer of the airways place many infants at higher risk for subsequent wheezing disorders in infancy, childhood and adulthood.

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