Juan Sanchez-Esteban, Pediatr Therapeut, 1:2
doi: http://dx.doi.org/10.4172/2161-0665.S1.03

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International Conference on
Pediatrics & Gynecology

# Role of the epidermal growth factor receptor (EGFR) in mechanotransduction during fetal lung development

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Mechanical forces generated by constant distension pressure and breathing-like movements are critical for normal fetal lung development. However, little is known about how lung cells sense these mechanical signals and convert them into biochemical responses to promote lung development. A key component of lung development is the differentiation of type II epithelial cells, the major source of pulmonary surfactant that prevents alveolar collapse during expiration. The EGFR is a member of the ErbB family of receptor tyrosine kinase shown to be a critical regulator of fetal lung development. Here, using wild-type and knockout mouse models, we will show that mechanical stretch promotes type II cell differentiation through the EGFR via release of ligands; specifically HB-EGF and TGF-α. In addition, we will demonstrate that stretch-induced release of ligands is mediated via the membrane protease ADAM17/TACE. Furthermore, we will discuss specific integrin subtypes that function as mechanosensors in type II cell differentiation. Finally, we will review organotypic models of mechanical strain in fetal lung development.

## Biography

Juan Sanchez-Esteban completed his M.D. from Autonomous University of Barcelona, Spain and his fellowship in Neonatal-Perinatal Medicine at Women & Infants Hospital of Rhode Island. Currently he is Associate Professor of Pediatrics at Brown University. His research interests are mechanical forces in fetal lung development and lung injury. His research is supported by the National Institute of Health.