



Regulation of Hormones during Fetal Lung Development

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DESCRIPTION

Treatment with corticosteroids and thyroid hormones in utero speeds up the maturation of the foetal lung. The metabolism of pulmonary phospholipids is also influenced by other substances such as catecholamines, thyrotropin-releasing hormone, oestradiol, heroin, and cyclic AMP. In type II cells, glucocorticoids induce early development of the surfactant system and of lung architecture, leading to more stable lungs with bigger air spaces. The characteristics of glucocorticoid action are in line with enzyme induction caused by steroid and cytoplasmic glucocorticoid receptor contact. Both pulmonary fibroblasts and type II cells, as well as the lungs of many species, including the developing human, include receptors. Currently, baby Respiratory Distress Syndrome (RDS) is less frequently seen in prematurely giving birth women who receive corticosteroid medication. When the mother receives 12 mg of betamethasone, the amount of unbound glucocorticoid activity in the foetal plasma increases by a factor of about four, leading to an estimated 80% nuclear steroid complex occupancy. Endogenous corticoids are likely to affect healthy lung development; potential sources of cortisol include the adrenal glands of the mother and the foetus, the amniotic membranes, and lung fibroblasts, which convert cortisone to cortisol. Though they seem to affect several biochemical processes, thyroid hormones have effects that are comparable to those of corticosteroids. Triiodothyronine (T₃) has synthetic counterparts that, in contrast to T₃ and thyroxine, easily pass the placenta and hasten the synthesis and release of surfactants. Thyroid hormones most likely work through nuclear receptors found in both animal and human lung tissue. Additionally, it seems that thyroid medication during pregnancy hastens lung development and shields premature infants from RDS.

The human foetus grows following a certain trajectory that must balance the mother's capacity with the foetus' needs. A difficult delivery is expected if the foetus develops to be too large, endangering the mother, but the foetus faces hazards if it is too small. The clinical care of small infants in the future may benefit

from an understanding of the endocrine mechanisms governing these processes, perhaps enhancing population health. The physiology of human foetal growth and development, as well as the immediate and long-term implications of inadequate foetal growth, has been extensively studied in humans. This review will address the function of the mother, placenta, and foetus in the endocrine regulation of foetal growth while concentrating on the evidence that is currently available from human studies.

The effects of estrogens on lung physiology and development are well known. The mechanism of oestrogen activity in the lung has remained a mystery due to the absence of the traditional Estrogen Receptor (ER α) in this organ. Here, we demonstrate that ER β is widely expressed and physiologically active in the lung, both *in vivo* and *in vitro*. Comparing the lungs of mice with the wild type ER β gene and mice with an inactivated ER β gene (ER β ^{-/-}), researchers found that adult female ER β ^{-/-} mice had fewer alveoli, data that suggested inadequate alveolar development, and signs of surfactant build-up. In the lungs of adult female ER β mice, levels of Platelet-Derived Growth Factor A (PDGF-A) and Granulocyte-Macrophage Colony-Stimulating Factor (GM-CSF), which are crucial regulators of alveolar formation and surfactant homeostasis, respectively, were decreased. Direct transcriptional regulation of these genes by ER β ^{-/-} was also shown to occur. This shows that estrogens affect the expression of GM-CSF and PDGF-A in the lung *via* the ER β . These findings establish ER β as a hitherto unidentified regulator of postnatal lung development and homeostasis and offer a plausible mechanistic explanation for the gender variations in alveolar shape seen in the adult lung. Lung morphogenesis is driven by hormones and growth factors. Organ development is regulated by a few important hormones for metabolic control, including insulin, glucocorticoids, and thyroid hormones. Despite their undeniable relevance, they are not included in our review because there is abundant literature about their involvement in lung formation and organogenesis that the interested reader may easily locate. Instead, it has recently been shown that novel hormones that regulate metabolism play a crucial part in the development of various organs, including

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the lung. Pregnancy under nutrition encourages a number of changes in hormone and metabolic regulation that are necessary to match the energy needs with the available resources. It is simple to connect a calorie deficit to a decrease in the availability of dietary hormone precursors such as retinoid and carotenoids. However, the liver and fat depots may store some of these precursors. In order to reduce the reserves sufficiently to have an impact on baby development during gestation, nutritional shortages of these

hormones must be established, most likely prior to pregnancy. In industrialized nations, monitoring every expectant mother and providing nutritional guidance ought to be sufficient to stop this kind of shortfall. Cholecalciferol levels for a considerable portion of the population are below or outside of normal ranges, which may be particularly important in specific sensitive populations: Poor levels of sun exposure, low dairy and fish consumption, and obesity.