Preface

Lung development is a methodical and coordinated process that culminates in the creation of an organ that contains airways, alveoli, and blood vessels. Lung development also has multiple innate and acquired host defense systems and processes that weaken the harmful effects of oxygen in the environment. By regulating cellular differentiation and branching morphogenesis, vitamin A, in particular, its active metabolite retinoic acid, plays an essential role during lung development. Retinoic acid is required for normal alveolar development and vitamin A administration increases lung’s elastic fiber contents at birth. Additionally, several experiments showed that retinoic acid restores alveolar architecture to dexamethasone-treated lungs and maintains alveolar development in lungs treated with an angiogenic inhibitor. Clinical trials revealed that vitamin A supplements given to premature infants decreased death and respiratory disorders due to lung immaturity, such as respiratory distress syndrome and bronchopulmonary dysplasia. On the other hand, for vitamin A deficiency, the incidence for diseases of the respiratory tract is considerably increased. Throughout pregnancy, maternal mechanisms of vitamin A homeostasis are mobilized to ensure that adequate levels of retinoids are available and reach the embryo and fetuses through accurate mechanisms of transplacental transfer. Nevertheless, given the fundamental roles of retinoids in embryonic development it is of vital importance that the conceptus should form its autonomous vitamin A reserves and be capable of maintaining normal levels of this compound. The present chapter reviews original and published research on vitamin A signaling pathways during lung maturation, outlining the morphogenetic effects of this compound on the temporal and spatial expression of several molecules involved in lung organogenesis, as well as their possible variation according to sex, since an
References


Baptista, □., et al. (2005). Cited on p. 58