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# Nutrition and Lung Growth

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**Abstract:** Experimental evidence from animal models and epidemiology studies has demonstrated that nutrition affects lung development and may have a lifelong impact on respiratory health. Chronic restriction of nutrients and/or oxygen during pregnancy causes structural changes in the airways and parenchyma that may result in abnormal lung function, which is tracked throughout life. Inadequate nutritional management in very premature infants hampers lung growth and may be a contributing factor in the pathogenesis of bronchopulmonary dysplasia. Recent evidence seems to indicate that infant and childhood malnutrition does not determine lung function impairment even in the presence of reduced lung size due to delayed body growth. This review will focus on the effects of malnutrition occurring at critical time periods such as pregnancy, early life, and childhood, on lung growth and long-term lung function.

**Keywords:** lung development; lung function; intrauterine growth restriction malnutrition; vitamins; omega-3 fatty acids; pediatrics

## 1. Introduction

Lung development is a multistage and multilevel process sustained by biochemical, mechanical and anatomical events spanning all gestational ages, from the end of the third week post-conception onwards, and continuing into post-natal life until around 22 years of age [1,2]. The lung has limited potential for recovery from early-life damage and poor lung function, a possible consequence of prenatal and perinatal insults, tracks throughout life with long-term consequences for respiratory health [3–6].

Nutrition has a key role in prenatal lung development, directly affecting mechanisms of lung growth but also influencing developmental programming through epigenetic changes [7,8]. The influence of nutrition on lung growth also continues in post-natal life, especially in early infancy [9].

Intrauterine growth restriction (IUGR) is the most common effect of chronic impaired prenatal nutrition, mostly (80–90%) due to reduced flow of nutrients and oxygen to the fetus through the placenta because of either placental insufficiency or maternal dietary deficiencies [10]. Placental insufficiency generally occurs in the second half of pregnancy, at the time of acinar and alveolar development, therefore the distal lung is most likely to be affected by IUGR [11–13].

In the neonatal period, adequate nutritional management is particularly important in extremely low birth weight infants (<1000 g), as growth failure in the first weeks of life is associated with an higher rate of bronchopulmonary dysplasia (BPD) in this group [14].

Less is known about the effects of infantile and childhood malnutrition on lung development. Recent evidence seems to indicate that it does not determine functional impairment but may affect lung size [15–18].

Micronutrients influence various aspects of the lung maturation. Retinoids from vitamin A regulate the expression of extracellular matrix proteins that address both airway development and alveolarization [19]. Vitamin D is involved in surfactant metabolism [20] and promotes epithelial–mesenchymal

interactions that are very important for lung maturation [21]. Maternal deficiencies of vitamin E and selenium during pregnancy have been associated with an increased risk of wheezing episodes in offspring [22,23]. Finally, omega-3 fatty acids modulate inflammation and attenuate hyperoxic lung injury [12]; infants born at <30 weeks with low omega-3 plasma levels were found to be at higher risk of BPD [24].

This review will examine the effects of impaired nutrition and micronutrients deficit occurring during pregnancy, early life and childhood, on lung growth and respiratory health. Epidemiology aspects and pathophysiology mechanisms of the relationship between nutrition and the respiratory system will be discussed.

## 2. Fetal Nutrition and Lung Development

Our understanding of the effects of prenatal nutrition on lung growth comes mainly from experimental studies on animal models where conditions of utero-placental vascular insufficiency or maternal nutrient restriction (most commonly protein restriction) were reproduced at different stages of pregnancy. The most widely used animal models of IUGR are lambs, rats and mice. While in sheep the timing of lung development is very similar to that of humans, in rodents alveolarization occurs entirely after term birth and, thus, they represent an optimal model to evaluate the effects of IUGR without the complications of preterm birth [25]. However, the interpretation of these experimental studies is hampered by many factors, including heterogeneous experimental settings with few replication studies and the different animal species selected for the studies; thus, caution is needed when the findings of these studies are applied to humans [26].

On a macroscopic scale, lung growth is proportionally restricted to body growth in lambs that undergo prolonged fetal undernutrition induced by placental insufficiency [27].

Undernutrition during saccular and alveolar stage of lung development mainly impairs alveolarization. In sheep it leads to prenatal altered surfactant protein expression [28], decreased pulmonary vascular growth [29], post-natal permanent reduction of alveolar surface area in relation to lung volume [30,31], increased extracellular matrix, and a thickened blood-gas barrier [31,32], resulting in reduced pulmonary compliance and diffusing capacity [33]. Rodent models of IUGR, at the saccular stage of lung development show thickened distal air space walls [34], and decreased elastin expression and elastic fiber deposition in the extracellular matrix [35].

Evidence from animal models suggests that IUGR also affects the conductive airways. In near-term growth-restricted lambs' fetuses, it was shown that luminal areas, basement membrane perimeters, and airway wall sections of the trachea and larger bronchi have a smaller size [36], while the mucosa showed reduced folding and sparse ciliated epithelial cells [37].

Epigenetic changes induced by chronic fetal undernutrition and IUGR are likely to be responsible for most of these effects [25], impacting signaling pathways that are involved in lung maturation, such as the peroxisome proliferator-activated receptor (PPAR) pathway [38,39] or transforming growth factor (TGF-β) signaling [40]. Of note, undernutrition may not be the only cause of altered developmental programming of the lung. Recent experimental data showed that in mice prenatal exposure to conditions commonly associated with maternal overfeeding in the western world (high-fat diet and hyperglycemia) caused feto-placental inflammation and ultimately delayed fetal lung development, alveolar simplification, and altered nitric oxide metabolism in the airways [41,42], whereas in lambs late gestation maternal overnutrition was associated with impaired surfactant production [43].

## 3. Effects of IUGR on Lung Function and Respiratory Health

There is conflicting evidence as to whether IUGR increases [44–46], has no impact [47–49], or even decreases [50–52] the incidence of neonatal respiratory distress syndrome. Conversely it is well recognized that IUGR is associated with an increased risk of BPD in preterm infants born before 32 weeks of gestational age [53–57].

A few studies investigated the impact of IUGR on subsequent lung function and wheezing or asthma. A population-based prospective cohort study ("Generation R") involving over 5000 children in the Netherlands found that IUGR was associated with higher respiratory resistance in 6-year old children [58], while fetal growth pattern was not associated with wheezing until the age of 3 years [59] or 4 years [60] according to different birth cohort studies. Turner et al. found that low fetal growth during the second and third trimesters was associated with reduced Forced expiratory volume in 1st second ($FEV_1$) and Forced Vital Capacity (FVC) but not altered asthma risk at 10 years of age [61]. Data from the "Generation R" cohort also indicated that restricted fetal weight growth during the second and third trimesters (weight growth percentile change between the time periods $<-0.67$ z-scores) was associated with lower dynamic lung volumes at age 10 years, partly dependent upon infant weight growth patterns, whereas there was no association with asthma or recurrent wheezing [62]. Rapid weight gain in infancy in children with IUGR was associated in some studies with increased risk of wheezing [60,63], asthma [62,63], and abnormal lung function in later life [64]. There is also evidence that IUGR may exacerbate the negative impact of prematurity on lung function in later life [65,66].

Reduced lung function was shown in infants [67–69] and children [66,70] with low birth weight for gestation (below the 10th percentile, a proxy for IUGR) even in the presence of catch-up growth [70]. A metanalysis of 24 birth cohort studies [71], showed that low birth weight and greater infant weight gain, normalized as z-scores, are associated with an increased risk of childhood asthma, lower $FEV_1$ and $FEV_1$/FVC z-score.

These findings could be the consequence of dysynaptic lung growth [72]. After birth, airways increase only in size whereas alveoli continue to increase in both size and number [73], therefore lung parenchyma has higher potential than airways to compensate prenatal impairment, as indicated by evidence of catch-up alveolarization in former preterm children [74,75]. A disproportionate growth between lung parenchyma and airways size may determine airflow limitation [76] and might contribute to the finding of abnormal lung function (mostly impaired $FEV_1$ and $FEV_1$/FVC) in some children with restricted fetal growth [61,62,71].

As young adults, subjects with low birth weight may reach a poor maximal lung function [77–79], which is associated with an increased risk of developing chronic obstructive pulmonary disease (COPD) in late adult life [80].

A possible link between fetal malnutrition and COPD was also suggested by the increased prevalence of obstructive respiratory disease in people aged about 50, born to mothers who had been exposed to the Dutch famine in the Netherlands (1944–1945). Controls of similar age born before or conceived after the famine were also included. In particular, subjects exposed to famine at mid-gestation showed the greatest effect (odds ratio—OR—adjusted 1.7, 95% CI, 1.1 to 2.6) [81].

## 4. Postnatal Early Nutrition and Lung Growth

After birth, nutrition plays a critical role for respiratory development of premature born infants, especially for very low birth weight (VLBW) infants whose saccular-alveolar stage of lung maturation occurs mostly or entirely in post-natal life [11,25].

The effects of post-natal undernutrition on lung maturation have been characterized in animal models. Rodent models, which are at a saccular stage of lung development when born at term [7], suggest that early post-natal undernutrition disrupts alveolarization [82] and affects the bronchiolar epithelium [83], with a reduction of both epithelial cells division and conversion of Clara cells to ciliated cells. Early post-natal nutritional restriction in rabbits exacerbates the negative impact of hyperoxia on alveolarization and extra-cellular matrix deposition in the lung [84,85], whereas in piglets it affects the oxidative capacity and the expression of myosin heavy chain isoforms in the diaphragm [86].

To what extent these experimental findings can be translated to humans is not known but there is some evidence indicating that nutritional management of prematurely born infants may be involved in the pathogenesis of BPD [87]. A large multicenter study showed that VLBW infants that grew at

the lowest quartile in the first three weeks of life were at higher risk for BPD and neurodevelopmental problems [14]. Excessive fluid intake also might play a role: in a cohort of nearly 1400 extremely low birth weight infants (birth weight 401 to 1000 g), infants who survived without BPD had significantly lower average total fluid intake (parenteral and enteral) from the second to the 10th day of life ($p < 0.001$) and significantly higher average weight loss (expressed as percent of birth weight) between days 6–9 ($p < 0.05$) compared to infants who died or developed BPD [88]. A systematic review of five studies evaluating the impact of restricted versus liberal water intake in preterm infants, pointed to a trend toward reduced risk of BPD in infants undergoing fluid restriction, although the five trials were not homogeneous in terms of study population, duration of the intervention, and targeted volume intakes [89]. Moreover, fluid restriction in preterm infants has to be balanced with the need for adequate caloric intake, as poor nutrition may be a determinant of BPD [90]. A trial comparing early versus delayed sodium supplementation (4 mmol/kg/day started on either the second day of life or when weight loss was 6% of birthweight) in preterm infants of 25–30 weeks of gestational age, did not find significant differences in the rate of continuing oxygen requirement at 28 days of life [91].

In infants with evolving or established BPD, nutritional management is also very important as they often show growth failure [90]. However nutritional management of these infants lies beyond the purpose of this review and has been discussed elsewhere [87], while the influence of micronutrients on post-natal lung growth in early life will be discussed later.

Breastfeeding seems to have a protective role against viral wheezing, especially in the first two years of life [92]. This effect is probably mediated by immune system-enhancing properties of human breast milk that limit the rate of viral respiratory infections in infancy [93–95]. In contrast, the impact of breastfeeding on later childhood asthma remains unclear [92,96]. The relationship between breastfeeding and subsequent lung function has been also largely investigated: a systematic review of five birth cohorts and three cross-sectional studies [97] found that children and adolescents who had been breastfed for more than 4 months had a 20–100 mL greater FVC than children who received formula feeding. A positive but weaker association between breastfeeding for more than 4 months and $FEV_1$ was also reported in some longitudinal studies [98,99], but not in others [100–102]. More recently, spirometry data recorded at 10 years of age in over 4000 children who had been breastfed showed that breastfeeding shorter than six months was associated with lower $FEV_1$ and FVC, although the impact was limited (z-score change for both $FEV_1$ and FVC, −0.01, 95% CI, −0.02 to 0.00; per month shorter breastfeeding) and probably clinically not relevant [103]. Overall, these findings suggest that breastfeeding might enhance lung growth, possibly through the action of cytokines and growth factors involved in lung maturation, such as TGF-β [104], or inducing epigenetic changes [105].

## 5. Malnutrition and Lung Growth in Childhood

The effects of infant and childhood malnutrition on lung development have been poorly investigated so far. This field of research could be particularly relevant for low- and middle-income countries due to the high burden of malnutrition and respiratory disease in such settings.

Alveolarization continues until at least 2 years of age [2], and therefore infant malnutrition could be expected to have greater long-term consequences on lung development compared to undernutrition occurring later in childhood. A recent study by Lelijveld et al. [106] investigated spirometry outcomes in a cohort ("ChroSAM study") of Malawian children, 7 years after a hospital admission for severe acute malnutrition (SAM). Siblings and healthy children from the same community were also evaluated. Surprisingly, 237 survivors of SAM had lung function comparable to the two control groups (143 siblings and 121 community controls), although it should be noted that 46% of the case group died before follow-up, representing a group which probably included the most severe SAM cases and could have had poorer lung function. Measuring sitting height, a pattern of shorter legs but preserved torso height was found in SAM survivors that might allow lung function to be preserved. Children who had suffered from SAM before two years of age (a period of greater susceptibility of lung development toward external influences) did not differ from the rest of the case group [106]. In a cross-sectional study

including over 1000 school-age children from different parts of sub-Saharan Africa [17], it was shown that undernourished children (Body Mass Index—BMI—z-score < −2) had reduced $FEV_1$ and FVC z-scores compared to normotrophic children but a preserved $FEV_1$/FVC ratio, implying smaller chest diameters without evidence of functional impairment. Also in that study, children with the lowest BMI z-scores had relatively shorter legs compared to torso height (highest sitting/standing height ratio), as previously found in SAM survivors. A secondary analysis of the ChroSAM cohort [18] confirmed that children with a BMI z-score < −2 have proportionally reduced dynamic lung volumes with normal $FEV_1$/FVC ratio z-score. Similar findings were also previously reported by Sonappa et al. in semi-urban and rural Indian children [16], that had significant reductions of ~0.5–0.9 z-scores in both $FEV_1$ and FVC (with preserved $FEV_1$/FVC ratio) and of ~1 z-score in BMI, as compared to Indian urban counterparts and children of Indian ancestry living in the UK. Overall these studies seem to support the hypothesis of a "lung-sparing growth" in children with malnutrition, where lung development is relatively spared compared to other less vital organs [107].

## 6. Influence of Micronutrients on Lung Development

Micronutrient deficiency is common worldwide and vitamins are extensively recognized as being important for the developing fetus and neonate [108]. Pregnant women have higher metabolic demands and are at risk of micronutrient deficiency [109], especially those of low socio-economic status from developing countries [110,111].

Micronutrients with the most relevant effects on lung development are Vitamins A, D, E, selenium and omega-3 docosahexaenoic acid.

### *6.1. Vitamin A*

Biologically active metabolites of Vitamin A (e.g., all-trans retinoic acid) are involved in many aspects of lung development. In mice, retinoic acid contributes to branching morphogenesis and to limiting excess branching and smooth muscle proliferation in the airways [112,113]. Retinoids are also important for adequate formation and maintenance of the alveoli [114,115] and have a role in regulating type II pneumocyte proliferation and synthesis of surfactant-associated proteins B and C (SP-B and SP-C, respectively) [116]. Many of these effects depend on the modulating activity of retinoids on the expression of several extracellular matrix proteins, most importantly elastin [117,118].

With regard to the effects of vitamin A supplementation on lung development, in animal models with prenatal vitamin A-deficiency, subsequent treatment with retinoic acid reversed some of the structural alterations that had been induced by missing retinol activity [119,120]. Vitamin A supplementation in very low birthweight infants slightly decreases the incidence of BPD [121,122], but does not affect duration of mechanical ventilation, length of hospital stay, and neurodevelopmental outcomes at 18–22 months of age [123]. Long-term effects of vitamin A supplementation in pregnant women were documented in a region with endemic vitamin A deficiency, where children whose mothers received vitamin A supplementation until 6 months after pregnancy had better lung function at 9 to 11 years of age than children whose mothers had received beta carotene supplementation or placebo [124].

### *6.2. Vitamin D*

The importance of vitamin D for fetal lung development was shown in animal models in several studies. Vitamin D contributes to pneumocyte type II maturation [125], enhances biosynthesis of SP-B [20] and surfactant-related phospholipids [124], and stimulates surfactant release [126]. Moreover, it promotes alveolar epithelial-mesenchymal interactions during lung maturation [127]. Prenatal vitamin D deficiency determines increased airway smooth muscle mass and airway hyper-responsiveness in rats [128], whereas in a mouse model it was associated with increased airway resistance and decreased lung volumes [129]. Genomic analyses in lung tissue from mice and humans have identified several genes involved in lung development that are regulated by vitamin D [130].

Moving to human studies, maternal vitamin D deficiency during pregnancy and low levels of 25-hydroxyvitamin D at birth were associated with decreased lung function in offspring by the age of 6 years [131,132]. A recent metanalysis of birth cohort studies that measured vitamin D blood levels in pregnant women or in cord blood at birth showed a borderline positive association between prenatal circulating 25-hydroxyvitamin D levels and $FEV_1$ of offspring at school age ($FEV_1$ z-score coefficient 0.07, 95% CI, −0.01 to 0.15), but no significant association with wheezing or asthma [133]. The combined analysis of two independent trials [134,135] that randomized pregnant women to receive high-dose vitamin D3 supplementation (4000 IU/d or 2400 IU/d,) versus 400 IU/d (controls), showed that children whose mothers received high-dose vitamin D3 had a 25% reduced risk of recurrent wheezing by the age of 3 years (adjusted OR, 0.74, 95% CI, 0.57 to 0.96, $p$ = 0.02) [136]. These data were recently confirmed by a systematic review indicating that maternal vitamin D supplementation during pregnancy consistently reduces the risk of wheezing in children by the age of 3 years (risk ratio 0.81, 95% CI, 0.67 to 0.98) [137].

### *6.3. Vitamin E*

Vitamin E it is not synthesized by the human body and requires intake from food sources, typically oils. There are eight isoforms of Vitamin E, most studies evaluating α-tocopherol and γ-tocopherol. Maternal plasma level of vitamin E during pregnancy are positively associated with fetal growth [138]. In a birth cohort, body length of human fetuses (crown–rump length) in the first trimester of pregnancy was positively associated with maternal plasma α-tocopherol, and with $FEV_1$ and FVC at 5 years of age [139], leading the authors to hypothesize that vitamin E may enhance fetal lung growth. A metanalysis of seven birth cohort studies found that higher maternal dietary vitamin E intake was associated with a 46% reduction in the odds of wheezing during childhood (pooled OR 0.54, 95% CI, 0.41 to 0.71) while there was no association with asthma [140]. Vitamin E is an antioxidant and has a protective role against oxygen toxicity [141]. In premature infants with respiratory distress syndrome, lower total plasma levels of vitamin E and selenium were associated with increased risk of developing BPD [142,143]. Unfortunately, trials of high-dose vitamin E (α-tocopherol) supplementation in very preterm born infant to prevent BPD showed variable results [144] and an association with increased risk of sepsis [145].

### *6.4. Selenium*

Selenium acts together with vitamin E to prevent peroxide formation [9]. In rats selenium deficiency impairs lung development, in particular alveolar septation [146]. A significant negative association was found between maternal plasma selenium concentration during pregnancy and the risk of wheezing episodes in offspring in the first 3 years of life, with no association with asthma [23,147]. In very low birthweight infants, low plasma selenium level is significantly associated with increased respiratory morbidity [148], however selenium supplementation did not have any impact on the incidence of BPD [149].

### *6.5. Zinc*

There is some evidence of abnormal prenatal lung development resulting from maternal zinc deficiency in rats [150]. Maternal zinc intake during pregnancy in the highest quartile was associated with a reduction in the odds of wheezing episodes in preschool children in three birth cohort studies [151–153] (pooled OR, 0.57, 95% CI, 0.40 to 0.81) [153], whereas no association resulted from other studies [22,154,155].

### *6.6. Docosahexaenoic Acid*

Docosahexaenoic acid (DHA) is a n-3 long-chain polyunsaturated fatty acid (LCPUFA) that enhances lung maturation and modulates inflammation [12,156]. In rodent models, maternal supplementation of DHA during late gestation and lactation increases surfactant concentration in amniotic fluid and in the fetal lung [157], enhances alveolarization [158], and ameliorates lung alterations due to IUGR [159].

Moreover, DHA supplementation decreases inflammation and attenuates lung injury induced by hyperoxia [160–164]. Premature infants are typically deficient in DHA [165] and those delivered at the lowest gestational ages are at the highest risk of deficiency [166]. In infants born at <30 weeks, low DHA levels were associated with a 2.5 times increased risk (OR, 2.5, 95% CI, 1.3 to 5.0) of BPD [24]. Based on evidence that DHA modulates inflammation and lung injury due to hyperoxia in animal models, some trials evaluated the utility of DHA supplementation in preterm-born infants for preventing BPD [167–169]. The most recent and best designed study [169] randomized 1205 preterm infants born before 29 weeks to receive an emulsion containing 60 mg/kg of DHA or a control (soy) emulsion, started within 3 days after the first enteral feeding and continued until 36 weeks of postmenstrual age. No significant difference in the incidence of BPD between the two groups (53.2% in the study group vs. 49.7% in controls, $p$ 0.06) was reported.

With regard to the long-term effect of maternal n-3 LCPUFA supplementation in pregnancy, Bisgaard et al. recently reported a 30% relative reduction in the probability of persistent wheezing at 3 years of age and asthma at 5 years in children whose mothers were supplemented with fish oil (2.4 g of n-3 LCPUFA per day) during pregnancy [170].

## 7. Conclusions

Experimental evidence and epidemiology studies show that impaired nutrition may have many possible consequences for lung maturation, depending on the type of nutritional deficit and the stage of lung development at which it occurs. In particular, malnutrition during late fetal life mainly affects the distal lung, exacerbates the negative effects of prematurity, and may determine life-long lung function impairment, probably increasing the susceptibility to COPD in later adult life. Suboptimal nutritional management in preterm infants may contribute to growth failure in the first weeks of life, which has been associated with an increased incidence of BPD. Micronutrients also play an important role in prenatal lung growth and their deficit in critical phases of lung development may have an impact on respiratory morbidity in preterm infants and on the incidence of wheezing in later life. Breastfeeding seems to be associated with lower incidence of preschool wheezing and better lung function at school age. Finally childhood malnutrition might be associated with a "lung-sparing growth" pattern which preserves normal lung function even in the presence of smaller lung size.

Overall many aspects of the effects of malnutrition on lung growth need to be elucidated. The most important are the reproducibility in humans of several physiopathology mechanisms demonstrated in animal models, or the best strategies for micronutrient supplementation during pregnancy and in preterm infants, and, finally, the relationship between IUGR and COPD. The last point could be addressed only by studying the incidence of COPD in people with a certain prenatal diagnosis of IUGR and in relation to the physio-pathological mechanism that induces IUGR. Future studies should address these points.

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