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PREMATURITY AND LUNG FUNCTION IN RELATION TO TELOMERE LENGTH AND INFLAMMATION IN 10-YEAR OLD CHILDREN

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Background and aims: Oxidative stress, as a result of inflammation and oxygen toxicity, may affect lung growth and contribute to the development of bronchopulmonary dysplasia (BPD) in preterm infants. Preterm infants have longer telomeres than term infants at birth. In adults, chronic obstructive lung disease has been related to shorter telomeres. Telomere length in relation to prematurity and lung function therefore was studied.

Methods: Relative telomere length (RTL) was measured using real-time PCR on extracted DNA in children born preterm with a history of BPD at the age of 10 years and a control group of children born healthy at term with a history of asthma. Lung function as dynamic spirometry and inflammation examined as fractional exhaled nitric oxygen (NO) was performed.

Results: Children with BPD (n= 23) compared to children with asthma (n=19) had shorter telomeres (RTL 1,43 vs 1,61, $p< 0,05$) and reduced lung function (Forced Expiratory Fraction (FEF 25-75%), 1,55 vs 1,88, $p< 0,05$) but lower levels of exhaled NO (NO 12,6 ppb vs 22,3 ppb, $p< 0,05$). Lung function and levels of NO were not independently correlated to RTL.

Conclusions: Preterm birth and lung disease in infancy resulted in shorter telomere length and reduced lung function at 10 years of age compared to controls with asthma. This may indicate faster telomere attrition in preterm infants with BPD. The influence of oxidative stress and inflammation in the neonatal period and beyond needs to be further studied in relation to intra-individual telomere shortening rate and long term outcome.