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## Pre-disposition to asthma by epigenetic imprinting of mitochondria activity

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Chronic inflammatory lung diseases (CILD) are increasing worldwide and represent a major issue for daily life performance and public health expenditure. The two major CILD are asthma and chronic obstructive pulmonary diseases (COPD). The World Health Organization estimates that 280 million people suffer from asthma and 40% of all children suffer from asthma symptoms during childhood. According to the World Health Organization, 65 million people developed severe COPD with a death toll of 3 million/year in 2005. The likelihood to develop asthma or COPD was linked to inherited susceptibility factors; however, despite of large genetic cohorts, no specific candidate genes had been identified. Increasing evidence supports the hypothesis that the susceptibility to develop CILD is the consequence of exposure to risk factors during embryogenesis or early childhood. One of these factors is preterm birth which accounts for life-long reduced lung function due to incomplete maturation of the lung structure. Secondly, the exposure to fine dust, ozone, smoke particles, or viral infections early in life increases the risk to develop CILD, but the mechanisms that cause these largely irreversible modifications of lung structure and function are not well understood. Importantly, new studies in humans and animal models suggest that these pre-conditions for CILD susceptibility can be passed on over at least three generations. It is implied that the increased susceptibility for CILD is due to largely irreversible epigenetic modifications which may mimic a genetic trait. The available data points towards an epigenetic mechanism that involves mitochondrial genes rather than the human genome, which may explain why the inheritance of CILD susceptibility was linked to the maternal line. This review summarizes the knowledge supporting the hypothesis of an epigenetic rather than genetic event which underlies the inheritance of asthma and COPD susceptibility.

### Biography

Michael Roth is currently working as Head of Pulmonary Cell Research at University Hospital Basel, Switzerland. He has completed his PhD at University of Basel and worked as a Visiting Professor and Associate Professor at University of Sydney for two years. He has published 148 articles in reputed journals.

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