

CONCEPT PAPER TEMPLATE

Provisional Paper Title:	Trajectories of lung function growth and decline
Proposing Author:	Tony (Xian) Zhang
Author's Phone, Fax or E-mail:	xian0727@gmail.com
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P.I. Sponsor (if the proposing author is a student or colleague of an original PI)	Bob Hancox

Objective of the study:

To model the growth of spirometric lung function from age 9 to age 38 years and assess risk factors for failure to achieve peak lung function. To further model decline in lung function from the early adult peak to age 45.

Data analysis methods:

1. Modelling of lung function growth to identify age of peak FEV1 and FVC for men and women and to identify groups with lower than expected peak function and growth trajectories from age 9 years. Analyses will be adjusted for height and done separately for sex (we anticipate sex-interaction in peak function).
2. Trajectory analysis of decline from peak function to age 45. Adjusted for age and sex (separate sex-analyses may be needed).
3. Assess the associations of the "usual suspects" of smoking, asthma, passive smoking, atopy and respiratory symptoms with trajectories of growth and decline.

Variables needed at which ages:

Lung function – all ages
 Basic demographic and height and weight data
 Atopy
 Asthma diagnoses and respiratory symptoms.
 Smoking, cannabis use.

Significance of the study (for theory, research methods or clinical practice):

Impaired lung function and COPD in later life has usually been thought to arise from accelerated lung function decline due to exposure to noxious agents, such as cigarette smoke. It is now believed that it can also originate from a failure to achieve peak lung function in early adulthood.¹⁻³ In other words, not reaching the normal peak level of lung function exposes people to the risk of developing impaired lung function in later life, even though the rate of lung function decline may not be accelerated. Of course, the combination of both a failure of lung growth combined with an early or accelerated decline would greatly increase the risk for early-onset of COPD.

A number of studies have analysed the growth in lung function, but few have the comprehensive data collected by the Dunedin study. Very few have such detailed information on the potential risk factors for impaired lung function growth or on the sequelae of impaired lung growth. The Dunedin study has already assessed a number of risk factors for impaired lung function in early adult life, such as smoking, wheeze and birth weight, but has not modelled trajectories of this. A number of studies have measured lung function decline,¹ but there is little research combining the two phases of growth and decline

This will be a two-stage process. We plan to first model growth trajectories using data up to age 38 (it is expected to peak in the 20s). When phase 45 data become available, we will model lung function decline. Depending on the findings, these two parts may form one or two publications. Of course, the trajectories themselves may lend themselves to further analyses of risk factors and consequences for impaired lung growth and decline.

References:

1. Lange P, Celli B, Agustí A, et al. Lung-Function Trajectories Leading to Chronic Obstructive Pulmonary Disease. *The New England journal of medicine* 2015; **373**(2): 111-22.
2. Global Initiative For Chronic Obstructive Lung Disease. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease (updated 2015): Global Initiative For Chronic Obstructive Lung Disease, 2015.
3. Vestbo J, Lange P, Fletcher and Peto 40 Years On. A Tribute and Reflection. *American Journal of Respiratory and Critical Care Medicine* 2017; **195**(11): 1420-2.