



Concept Paper Form

Provisional Paper Title: Lung function and cardiovascular disease risk factors at age 45 in the Dunedin Multidisciplinary Health and Development Study
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Objective of the study:

Airflow obstruction and low forced vital capacities are established risk factors for cardiovascular diseases (1), even after adjusting for other standard cardiovascular risk factors such as age and smoking (1, 2, 3). Individuals with Chronic Obstructive Pulmonary Disease (COPD) have more than double the risk of cardiovascular disease (3, 4). Widespread speculation has been made about how these two disease processes relate and interact. These include increased systemic inflammation (1, 2), endothelial dysfunction (1, 5), and early-childhood factors predisposing to lung and cardiac pathology (1). However, the precise mechanism as to the how these clinical findings/pathologies are related to cardiac dysfunction is unknown (1, 2).

Determining an individual's cardiovascular risk is important to prevent cardiovascular disease through the implementation of effective interventions e.g. statins, aspirin, etc. (6). Based on the New Zealand Ministry of Healthy guidelines, Cardiovascular Disease (CVD) Risk Assessment should begin at age 45 in men and 55 in women. However, for Maori, Pasifik and Asian populations, CVD risk assessment is recommended to begin 15 years earlier. (6)

This objective of this project is to assess the relationship between lung function results and cardiovascular disease risk in men and women aged 45 and investigate the potential pathophysiological mechanisms that link lung and cardiac pathology. This will lead to consideration of the potential therapeutic/prognostic benefits of early intervention in individuals with abnormal lung function results at age 45.

Data analysis methods:

Cross-sectional data at age 45 will be statistically analysed using linear regression

using CV risk measures as the dependent variable with measures of percent predicted spirometric lung function (FEV₁, FVC, and FEV₁/FVC) as the main predictor. Analyses will be adjusted for confounders such as sex and smoking. Cardiovascular risk will be calculated using the Framingham (7) or New Zealand PREDICT risk equations (8). However, these equations will be adjusted for smoking due to the shared risk that it plays in both cardiovascular disease and decreasing lung function. The equations will be modified, where necessary, to adjust for the effects of blood pressure, lipid, and anti-platelet treatment because we are interested in the underlying CV risk, rather than the estimated risk on treatment.

For the longitudinal analyses, we will assess whether change in percent predicted lung function since early adulthood (age 18 to 26 depending on the peak lung function (because the spirometric variables peak at different ages)) predicts CV risk at age 45 (using the above equations) in order to assess associations between decline in lung function and CV risk. These analyses will use linear regression adjusting for sex. These analyses will also adjust for smoking and take into account the considerations for the effects of treatment on CV risk.

Variables needed at which ages:

Lung function: All ages
Blood pressure: Age 45
Blood lipids: Age 45
Diabetes/HbA1c: Age 45
Ethnicity
Family history of CVD
Renal function – Creatinine and Albuminuria
CV medication – Aspirin, Lipid-lowering, Anti-hypertensives
SES: All ages
BMI: Age 45
Fitness/Heart rate response to exercise: Age 45
Asthma/Atopy history
Smoking history
Medication history

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

This study hopes to clarify the possible relationship between impaired lung physiology and cardiovascular morbidity/mortality. If there is a positive association between these two disease processes, this can help to guide more targeted interventions to patients suffering from chronic respiratory illnesses such as COPD who often die due to cardiovascular causes. Additionally, by identifying potential early intervention strategies to individuals displaying mild/moderate pulmonary dysfunction in mid-adulthood, this study may also help lead to strategies to prevent cardiovascular deaths.

References:

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