Does physical fitness enhance lung function in children and young adults?

Robert J. Hancox[1] and Finn Rasmussen[2]

Affiliations:
[1]Dept of Preventive and Social Medicine, Dunedin School of Medicine, University of Otago, Dunedin, New Zealand.
[2]Dept of Allergy and Respiratory Medicine, Near East University Hospital, Near East University, Nicosia, Cyprus.

Correspondence:
Robert J. Hancox, Dept of Preventive and Social Medicine, Dunedin School of Medicine, PO Box 913, Dunedin, New Zealand. E-mail: bob.hancox@otago.ac.nz

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ABSTRACT Although physical activity is important for lung health, it is unclear whether physical fitness influences lung function. We investigated associations between lung function and fitness in two population-based cohort studies of children and young adults.

Aerobic fitness was measured using a maximal cycle ergometer test at ages 9, 15, 21 and 29 years in Odense, Denmark and using a submaximal cycle test at ages 15, 26, 32 and 38 years in Dunedin, New Zealand.

Aerobic fitness was positively associated with forced expiratory volume in 1 s (FEV_1) and forced vital capacity (FVC) in cross-sectional analyses at all ages in both cohorts, independently of height, weight, sex, asthma and smoking. Each standard deviation difference in fitness was associated with 2–3% predicted higher values of FEV_1 and FVC. Improvements in fitness during childhood and adolescence were associated with growth in lung volumes in longitudinal analyses. These associations tended to be stronger in males than females. No longitudinal associations were found after peak adult lung function had been attained. Fitness was not significantly associated with FEV_1/FVC ratios.

Aerobic fitness is positively associated with lung volumes. Improving fitness during childhood and adolescence is associated with greater adult lung volumes, but not with airway calibre.

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Introduction

Although physical activity and fitness are believed to be important for lung health, the association between lung function and physical fitness is unclear. Physical training improves exercise capacity and outcomes for patients with asthma or chronic obstructive pulmonary disease, but there is little evidence that it improves lung function [1, 2]. However, these studies do not answer the question of whether improving fitness across the life-course would be associated with better lung function among healthy individuals [3]. Some general population studies have found higher lung volumes among those who are physically active [4–10], but other studies did not find associations after adjusting for confounding [11–13]. Cohort studies suggest that physically active children and adolescents may have greater lung volumes, but these studies also have inconsistent findings [14–17]. Two studies found an association between physical activity and forced vital capacity (FVC) in early adolescence, but only among females [16, 17], while a follow-up of one of these studies in later adolescence found that the association was positive only among males [15]. An intervention study in 7-year-old children found that physical training was associated with greater lung volumes over 5 years of follow-up, but interpretation of this finding is limited by a nonrandom control group [18]. Longitudinal studies in adulthood suggest that physical activity may be associated with a slower decline in lung function [9, 19–22]. Most studies have observed associations between physical activity and dynamic lung volumes (forced expiratory volume in 1 s (FEV_1) and FVC), rather than with measures of airway function such as the FEV_1/FVC ratio.

There is limited research on associations between physical fitness and lung function. Distinguishing physical activity from fitness may be important because the correlations between physical activity and aerobic fitness are modest and there is considerable heterogeneity in individual responses to exercise training [23–25]. Physical activity that does not lead to improved fitness may be less likely to have an impact on lung function. Objective measurements of physical fitness (defined as maximal aerobic exercise capacity) using cycling or running tests have the additional advantage of avoiding the subjective nature and potential misclassification of self-reported activity.

Findings on the association between fitness and lung function have been mixed. A cross-sectional study of 248 children found no association between lung function and performance on a cycle ergometer test [26], and the Amsterdam Growth and Health Study did not find an association between lung function and treadmill running performance in 167 nonasthmatic children followed from age 13 to 27 years [14]. In contrast, a large cross-sectional study of adults aged 18–30 years found that performance on a treadmill test was associated with higher FEV_1 and FVC values [8]. A recent follow-up of this study found that sustained or improved fitness 20 years later was associated with better preservation of lung function [27].

We explored cross-sectional and longitudinal associations between fitness and lung function in two population-based cohorts extending from childhood into mid adulthood. We hypothesised that aerobic fitness would be positively associated with lung function and that improvements in fitness would be associated with improvements in lung volumes.

Methods

Odense study

The Odense schoolchild study is a prospective multidisciplinary study of a community-based cohort of 1369 schoolchildren, first investigated during their third grade (age 8.5–11.0 years) in 1985 [28]. Details of the selection and examination of the random baseline population are published elsewhere [29]. Spirometry and maximal exercise tests were performed at ages 9, 15, 21 and 29 years. Informed consent was obtained for each assessment. The local research ethics committee and the Danish Data Surveillance Authority approved the study.

At ages 9 and 15 years, asthma was identified by the question: "Have you ever had asthma, *i.e.* periods of wheeze and/or cough?" At ages 21 and 29 years, participants were asked: "Is it your doctor's opinion that you have asthma?" Spirometry was measured using a McDermott dry bellows spirometer (Garw Electronic Instruments, Penarth, UK) at ages 9 and 15 years and a pneumotachograph at ages 21 and 29 years (Vitalograph Compact; Vitalograph, Maids Moreton, UK) in accordance with European standardised recommendations, as previously described [30].

Aerobic exercise fitness was measured using a maximal progressive exercise test on an electrically braked cycle ergometer (Medtronic/Siemens, Copenhagen, Denmark), as previously described [28, 31]. The workload was increased every 3 min and participants exercised for approximately five 3-min periods. Heart rates were measured continuously using Polar monitors (Polar Electro OY, Espoo, Finland). Effort was accepted as maximal when the participant exceeded 85% of the expected maximal heart rate calculated as 220 minus age in years. The workload was converted to the maximal oxygen uptake (V'_{O_2max}; $L \cdot min^{-1}$) [28]. Height and weight in light clothing without shoes were measured using an

anthropometric plane and a beam-type balance scale. At age 15 years the degree of sexual maturation was rated on a scale from 1 to 5 [32]. Participants were asked "Do you smoke?"

Dunedin study

The Dunedin Multidisciplinary Health and Development Study is a population-based birth cohort of individuals born in Dunedin (New Zealand) between April 1972 and March 1973 [33]. The cohort has been assessed at multiple ages through childhood and adulthood. Maximal aerobic fitness was first measured at age 15 years (range 14.2–16.0 years) and repeated at 26, 32 and 38 years using a submaximal exercise test and the modified Åstrand–Rhyming protocol as previously described [34]. Participants exercised on a cycle ergometer (Monark, Vansbro, Sweden) for 6–8 min to achieve a stable heart rate of 70% predicted (calculated as 220 minus age). From the workload achieved and the final stable heart rate, maximal aerobic fitness ($V'O_2max$) was calculated [35].

Current asthma was defined as a self-reported diagnosis of asthma with compatible symptoms or medication use in the previous year, as previously described [36]. Current smoking was defined as daily smoking for ⩾1 month in the previous year. Spirometry was measured using a water-sealed spirometer (Godart, Bilthoven, the Netherlands) at age 15 years and a body plethysmograph (Vmax; Sensormedics, Yorba Linda, CA, USA) in adulthood. Height and weight in light clothing without shoes were measured using calibrated stadiometers and scales. The appropriate ethics committees approved all assessments and written informed consent was obtained for each assessment.

Statistical analysis

The cohorts were analysed separately because of differences in the ages of assessment and methods of measuring aerobic fitness. At each age, associations between maximal aerobic fitness and lung function were analysed by linear regression using measures of lung function (FEV_1, FVC or FEV_1/FVC ratio) as the dependent (outcome) variables and aerobic fitness ($V'O_2max$) as the main independent (predictor) variable. To account for age and sex-related differences in lung function and fitness, FEV_1 and FVC were expressed as % predicted values [37], while $V'O_2max$ values at each age were converted to standard deviations scores (z-scores) for each sex. All analyses were adjusted for sex, height, weight and current asthma. Analyses from age 15 years onwards were adjusted for current smoking.

To assess the associations between changes in aerobic fitness and lung function, longitudinal analyses used linear regression to assess whether the changes in $V'O_2max$ between each age predicted FEV_1, FVC or FEV_1/FVC at the older age, with adjustment for the same measure of lung function at the younger age. These analyses also adjusted for sex and height, weight, current asthma and current smoking at each age. Additional sensitivity analyses excluded participants with asthma.

Initial analyses tested for sex-by-fitness interactions and supplementary analyses were undertaken for each sex separately. Further analyses tested for interactions between smoking and fitness in the cross-sectional analyses. Pregnant women were excluded from all analyses. Data from participants taking β-blocker drugs were excluded from the Dunedin study, because these would impair the heart-rate response and influence the estimation of aerobic fitness. Otherwise all participants with sufficient data were included in each analysis, except for one extreme outlier at age 15 years in the Odense study. Linear regression models were checked by visual inspection of histograms of the residuals, residual *versus* fitted plots, and leverage *versus* squared-residual plots.

To compare the longitudinal analyses between the studies, we estimated age 29 values for the Dunedin study as the mean of the fitness, lung function, weight and height measurements at ages 26 and 32 years. We then analysed associations between changes in fitness and lung function from age 15 years to 29 years in both studies with adjustment for sex and height, weight, current asthma and current smoking at each age (Dunedin study participants who reported smoking or asthma at either age 26 years or 32 years were regarded as having smoking or asthma at age 29 years).

Analyses were performed using Stata 13 (StataCorp, College Station, TX, USA). p-values ⩽0.05 were regarded as statistically significant.

Results

Aerobic fitness increased with age up to early adulthood, but declined after age 21 years in the Odense study and after 26 years in the Dunedin study. FEV_1 also peaked in early adulthood, but FVC continued to increase up to age 29 years in the Odense study and peaked at age 32 years in the Dunedin study (tables 1 and 2 and online supplementary tables S1a and S1b).

In the cross-sectional analyses, FEV_1 and FVC, but not the FEV_1/FVC ratio, were statistically significantly associated with aerobic fitness at all ages in both studies (tables 3 and 4). There were a few statistically

TABLE 1 Aerobic fitness and lung function in the Odense study

	Age years							
	9		15		21		29	
	Mean±SD	95% CI	Mean±SD	95% CI	Mean±SD	95% CI	Mean±SD	95% CI
Female participants n	673		510		427		378	
V'_{O_2max} L·min^{-1}	1.48±0.22	1.46–1.50	2.55±0.45	2.51–2.59	2.38±0.39	2.34–2.42	2.26±0.46	2.22–2.31
FEV_1 L	1.74±0.26	1.72–1.76	3.50±0.49	3.46–3.54	3.43±0.45	3.38–3.47	3.38±0.43	3.34–3.42
FVC L	1.97±0.28	1.95–1.99	3.88±0.60	3.83–3.93	4.11±0.55	4.05–4.16	4.28±0.56	4.22–4.34
FEV_1/FVC %	88.4±4.4	88.1–88.7	90.6±6.0	90.1–91.1	83.7±5.9	83.1–84.2	79.3±6.3	78.7–80.0
Male participants n	696		572		454		420	
V'_{O_2max} L·min^{-1}	1.73±0.25	1.71–1.75	3.21±0.59	3.16–3.26	3.64±0.57	3.59–3.69	3.37±0.64	3.31–3.43
FEV_1 L	1.86±0.27	1.84–1.88	4.04±0.67	3.98–4.09	4.61±0.59	4.56–4.66	4.55±0.65	4.49–4.62
FVC L	2.14±0.31	2.11–2.16	4.59±0.81	4.53–4.66	5.76±0.76	5.69–5.83	5.95±0.84	5.87–6.03
FEV_1/FVC %	87.1±4.7	86.8–87.5	88.3±6.6	87.8–88.9	80.3±6.9	79.7–81.0	76.7±6.5	76.1–77.4

V'_{O_2max}: maximal oxygen uptake; FEV_1: forced expiratory volume in 1 s; FVC: forced vital capacity. Pregnant participants have been excluded.

significant interactions between fitness and sex, but in general there were similar associations in both sexes with the exception that the associations tended to be stronger in male participants at younger ages in the Dunedin study (online supplementary tables S2a and S2b). There were no statistically significant interactions between fitness and smoking at any age.

In the longitudinal analyses, increases in aerobic fitness between ages 9, 15, 21 and 29 years were associated with higher FEV_1 and FVC values in the Odense study, although the associations were weaker at older ages. Similarly, increases in aerobic fitness between ages 15 years and 26 years in the Dunedin study were associated with increases in FEV_1 and FVC, but these associations were weaker and not statistically significant after age 26 years (tables 5 and 6). There were statistically significant sex interactions for the younger ages in the Odense study: when analysed separately, the associations between changes in fitness and spirometric volumes were stronger in male participants in both studies, although the direction of association was the same in females (online supplementary tables S3a and S3b). Associations between changes in fitness and changes in FVC between each age are shown in figures 1 and 2. Longitudinal analyses from age 15 years to 29 years showed that the magnitude of association between changes in fitness and lung volumes was similar in both studies, despite the differences in methodology. These associations were stronger in males than females (table 7).

TABLE 2 Aerobic fitness and lung function in the Dunedin study

	Age years							
	15		26		32		38	
	Mean±SD	95% CI	Mean±SD	95% CI	Mean±SD	95% CI	Mean±SD	95% CI
Female participants n	403		342		401		430	
V'_{O_2max} L·min^{-1}	2.46±0.38	2.42–2.49	2.66±0.63	2.59–2.73	1.81±0.22	1.79–1.84	1.67±0.19	1.65–1.69
FEV_1 L	3.33±0.44	3.29–3.37	3.42±0.48	3.37–3.47	3.34±0.47	3.30–3.39	3.13±0.44	3.09–3.17
FVC L	3.70±0.50	3.65–3.75	4.06±0.56	4.00–4.12	4.20±0.57	4.14–4.25	4.06±0.54	4.01–4.11
FEV_1/FVC %	90.2±6.5	89.5– 90.8	83.7±6.3	83.0–84.4	79.8±6.2	79.2–80.4	77.2±6.5	76.6–77.9
Male participants n	434		431		462		460	
V'_{O_2max} L·min^{-1}	2.82±0.57	2.77–2.88	3.75±0.83	3.67–3.83	3.25±0.36	3.22–3.28	2.96±0.33	2.93– 2.99
FEV_1 L	3.69±0.70	3.63–3.76	4.66±0.70	4.60–4.72	4.45±0.64	4.39–4.51	4.17±0.62	4.11–4.23
FVC L	4.31±0.75	4.24–4.38	5.79±0.80	5.71–5.86	5.81±0.78	5.74–5.88	5.56±0.75	5.49–5.63
FEV_1/FVC %	85.7±7.3	85.0–86.4	80.2±7.2	79.5–80.8	76.7±6.8	76.1–77.4	75.1±7.0	74.5–75.8

V'_{O_2max}: maximal oxygen uptake; FEV_1: forced expiratory volume in 1 s; FVC: forced vital capacity. Pregnant participants and those using β-blockers have been excluded.

TABLE 3 Cross-sectional associations between fitness and lung function in the Odense study

	Participants n	Coefficient (95% CI)	p-value
Age 9 years	1293		
FEV_1 % pred		**2.8 (2.2–3.5)**	**<0.001**
FVC % pred		**2.4 (1.8–3.1)**	**<0.001**
FEV_1/FVC %		0.3 (0.0–0.6)	0.074
Age 15 years	752		
FEV_1 % pred		**3.2 (2.0–4.4)**	**<0.001**
FVC % pred		**3.7 (2.6–4.8)**	**<0.001**
FEV_1/FVC %		−0.6 (−1.3–0.0)	0.068
Age 21 years	871		
FEV_1 % pred		**3.1 (2.3–3.8)**	**<0.001**
FVC % pred		**3.0 (2.3–3.7)**	**<0.001**
FEV_1/FVC %		−0.2 (−0.7–0.3)	0.411
Age 29 years	783		
FEV_1 % pred		**2.7 (1.8–3.5)**	**<0.001**
FVC % pred		**2.1 (1.3–2.9)**	**<0.001**
FEV_1/FVC %		0.4 (−0.1–0.9)	0.120

FEV_1: forced expiratory volume in 1 s; FVC: forced vital capacity. The independent (predictor) variable is maximal oxygen uptake (V'_{O_2max}). Coefficients represent the difference in lung function associated with one standard deviation (z-score) difference in V'_{O_2max}. Analyses were adjusted for sex, height, weight, current asthma and current smoking (age 15 years onwards). Pregnant participants have been excluded. Bold type represents statistical significance.

Discussion

In these two population-based cohort studies, aerobic fitness was associated with higher values of FEV_1 and FVC among children, adolescents and young adults. Improvements in fitness during childhood and adolescence were associated with increases in lung volumes up to early adulthood. These associations became weaker with age and were not significant in the older age groups (figures 1 and 2). Fitness was not associated with FEV_1/FVC ratios, indicating that aerobic fitness is primarily associated with lung size rather than airway calibre. These findings suggest that improving physical fitness during childhood and adolescence may enhance peak lung function in early adulthood. We found no evidence that physical fitness influenced the early decline in lung function, but the cohorts have not yet been followed into older ages and it is likely to be too early to observe such an association.

TABLE 4 Cross-sectional associations between fitness and lung function in the Dunedin study

	Participants n	Coefficient (95% CI)	p-value
Age 15 years	833		
FEV_1 % pred		**2.9 (1.6–4.2)**	**<0.001**
FVC % pred		**3.0 (1.9–4.2)**	**<0.001**
FEV_1/FVC %		−0.2 (−0.9–0.5)	0.652
Age 26 years	768		
FEV_1 % pred		**2.2 (1.2–3.2)**	**<0.001**
FVC % pred		**2.7 (1.8–3.6)**	**<0.001**
FEV_1/FVC %		−0.3 (−0.8–0.2)	0.230
Age 32 years	863		
FEV_1 % pred		**1.8 (1.0–2.6)**	**<0.001**
FVC % pred		**1.7 (0.9–2.5)**	**<0.001**
FEV_1/FVC %		0.2 (−0.2–0.6)	0.382
Age 38 years	890		
FEV_1 L		**1.8 (1.0–2.6)**	**<0.001**
FVC L		**1.6 (0.8–2.4)**	**<0.001**
FEV_1/FVC %		0.1 (−0.3–0.6)	0.537

FEV_1: forced expiratory volume in 1 s; FVC: forced vital capacity. The independent (predictor) variable is maximal oxygen uptake (V'_{O_2max}). Coefficients represent the difference in lung function associated with one standard deviation (z-score) difference in V'_{O_2max}. Analyses were adjusted for sex, height, weight and current smoking. Pregnant participants and those taking β-blockers at either assessment have been excluded. Bold type represents statistical significance.

TABLE 5 Longitudinal analyses: do changes in fitness predict lung function in the Odense study?

	Participants n	Coefficient (95% CI)	p-value
Age 9–15 years	718		
FEV_1 % pred		**1.8 (1.0–2.4)**	**<0.001**
FVC % pred		**2.0 (1.3–2.6)**	**<0.001**
FEV_1/FVC %		−0.2 (−0.6–0.1)	0.241
Age 15–21 years	539		
FEV_1 % pred		**1.1 (0.5–1.7)**	**<0.001**
FVC % pred		**1.2 (0.6–1.7)**	**<0.001**
FEV_1/FVC %		−0.1 (−0.4–0.3)	0.727
Age 21–29 years	563		
FEV_1 % pred		**0.6 (0.1–1.1)**	**0.031**
FVC % pred		**0.6 (0.1–1.2)**	**0.020**
FEV_1/FVC %		0.0 (−0.4–0.3)	0.761

FEV_1: forced expiratory volume in 1 s; FVC: forced vital capacity. The independent (predictor) variable is the change in maximal oxygen uptake ($V'O_{2max}$) between ages. Coefficients represent the difference in lung function associated with one standard deviation (z-score) difference in change in $V'O_{2max}$. Analyses were adjusted for sex, lung function at the younger age, weight, height, asthma and smoking at each age. Pregnant participants have been excluded. Bold type represents statistical significance.

The strength of the associations between changes in fitness and lung function were modest, but not trivial. Percentage predicted values for FEV_1 and FVC were 2–3% higher for each standard deviation improvement in fitness in early adulthood (tables 3 and 4). Early adulthood is the time of peak lung function, and differences of this magnitude may be clinically important for some individuals. In addition, longitudinal improvements in fitness from childhood to early adulthood were associated with better adult lung function, particularly for males (tables 5–7). These findings may have public health relevance, particularly in view of concerns about declining levels of physical activity and fitness among young people [38].

Why lung function and fitness are associated is not clear. Unlike cardiac and skeletal muscle function, lung function is not thought to respond to exercise training [39]. However, the studies that led to this conclusion would have been too small to detect effect sizes of the magnitude that we have observed. It is plausible that frequent physical activity could enhance respiratory as well as skeletal muscle function and that this could lead to improvements in lung volumes [39]. It is not possible to be certain of the direction

TABLE 6 Longitudinal analyses: do changes in fitness predict lung function in the Dunedin study?

	Participants n	Coefficient (95% CI)	p-value
Age 15–26 years	650		
FEV_1 % pred		**0.8 (0.1–1.5)**	**0.031**
FVC % pred		**1.4 (0.7–2.1)**	**<0.001**
FEV_1/FVC %		−0.3 (−0.7–0.0)	0.067
Age 26–32 years	699		
FEV_1 % pred		−0.5 (−1.0–0.0)	0.067
FVC % pred		−0.4 (−0.8–0.1)	0.125
FEV_1/FVC %		−0.1 (−0.3–0.2)	0.607
Age 32–38 years	805		
FEV_1 % pred		0.0 (−0.5–0.5)	0.970
FVC % pred		0.1 (−0.3–0.5)	0.641
FEV_1/FVC %		−0.1 (−0.3–0.2)	0.514

FEV_1: forced expiratory volume in 1 s; FVC: forced vital capacity. The independent (predictor) variable is the change in maximal oxygen uptake ($V'O_{2max}$) between ages. Coefficients represent the difference in lung function associated with one standard deviation (z-score) difference in change in $V'O_{2max}$. Analyses were adjusted for sex, lung function at the younger age, weight, height, asthma and smoking at each age. Pregnant participants and those taking β-blockers have been excluded. Bold type represents statistical significance.

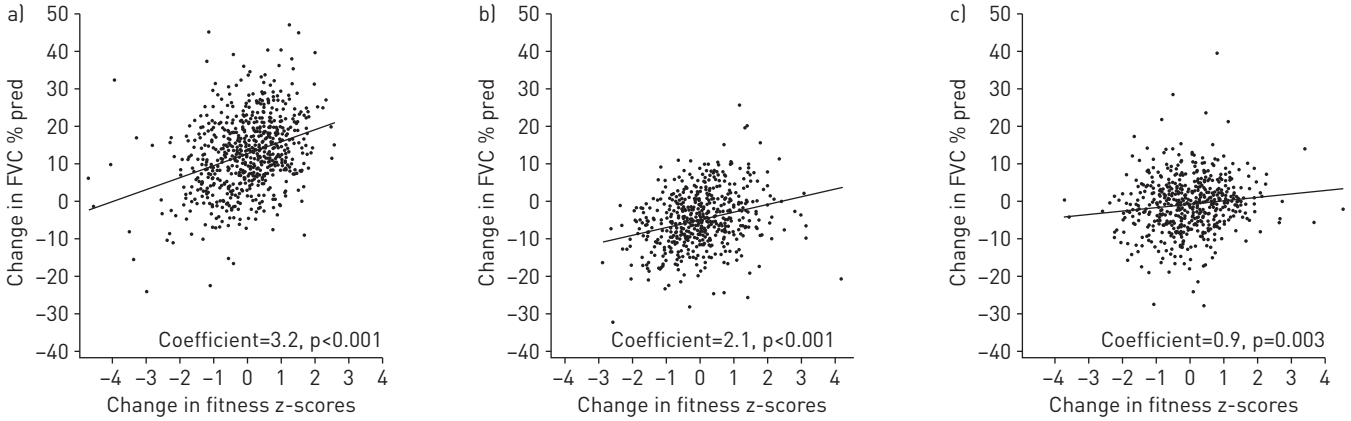

FIGURE 1 Scatterplots of change in forced vital capacity (FVC) % predicted with sex-specific changes in aerobic fitness (maximal oxygen uptake ($V'O_{2max}$) z-scores) between each age in the Odense study. a) 9–15 years; b) 15–21 years; c) 21–29 years. Fitted lines and unadjusted linear regression coefficients are shown. Coefficients represent the increase in FVC associated with each standard deviation (z-score) improvement in $V'O_{2max}$ compared to other participants of the same sex.

of association between improving lung function and fitness. Reversing the dependent and independent variables in the analyses in tables 5 and 6 provided very similar findings, such that growth in lung function predicted fitness at follow-up (not shown). Of course, it is possible that lung function and fitness were correlated because of their association with other factors, particularly the stage of growth and development among children and adolescents. It is notable that the longitudinal associations tended to be stronger in male than female adolescents in both studies (table 4 and online supplementary table S3a and S3b) and this may reflect differences in physical maturation and the greater increases in both aerobic fitness and lung function among male participants over the follow-up periods (online supplementary table S1). However, the findings were independent of height and weight at each age, indicating that the association is independent of physical growth. In addition, when the Odense study analyses were further adjusted for pubertal developmental stage at the age of 15 years, the findings were substantially unchanged (online supplementary tables S4a and S4b).

As far as we are aware, only one other study has tracked fitness and lung function from childhood to early adulthood in a general population sample. The Amsterdam Growth and Health Study followed spirometry and aerobic fitness on a treadmill running test in 167 individuals aged 13–27 years [14]. No associations were found between fitness and FEV_1, FVC or peak expiratory flow in either univariate or multivariate analyses. The reasons for the differences between our findings and those of the Amsterdam study are not obvious, although the Amsterdam cohort had fewer members than either the Odense or Dunedin cohorts and may have lacked power. However, the Amsterdam study did find that physical activity in childhood and adolescence was associated with higher lung volumes, as did other cohort studies [14–17]. Although

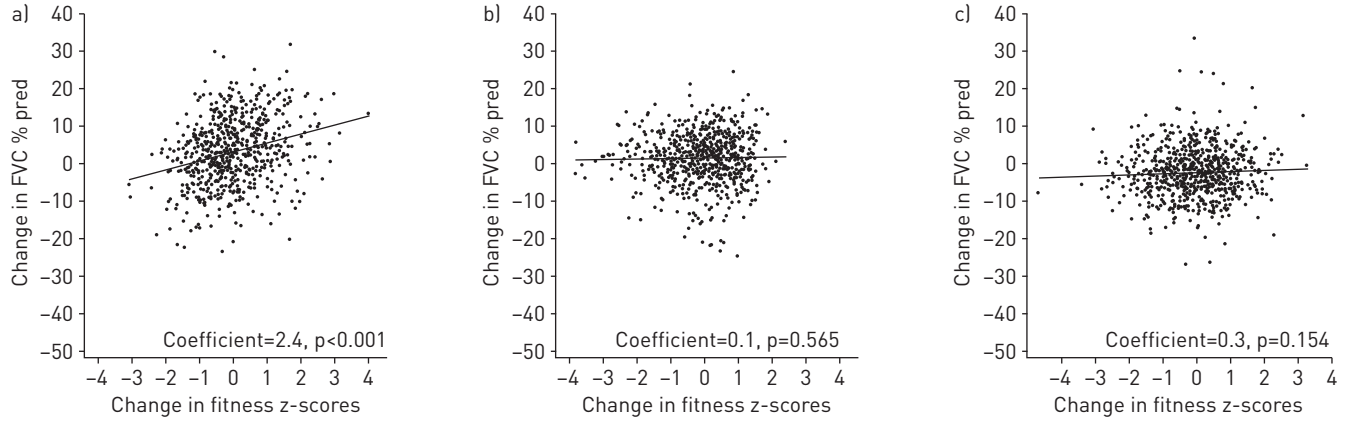

FIGURE 2 Scatterplots of change in forced vital capacity (FVC) % predicted with sex-specific changes in aerobic fitness (maximal oxygen uptake ($V'O_{2max}$) z-scores) between each age in the Dunedin study. a) 15–26 years; b) 26–32 years; c) 32–38 years. Fitted lines and unadjusted linear regression coefficients are shown. Coefficients represent the increase in FVC associated with each standard deviation (z-score) improvement in $V'O_{2max}$ compared to other participants of the same sex.

TABLE 7 Longitudinal analyses: do changes in fitness predict lung function between ages 15 years and 29 years?

	Participants n	Coefficient (95% CI)	p-value
Odense			
Female participants	210		
FEV_1 % pred		0.5 (−0.5–1.51)	0.356
FVC % pred		0.5 (−0.4–1.4)	0.303
FEV_1/FVC %		−0.1 (−0.7–0.5)	0.779
Male participants	259		
FEV_1 % pred		**2.2 (1.2–3.3)**	**<0.001**
FVC % pred		**1.8 (0.7–2.9)**	**0.002**
FEV_1/FVC %		0.4 (−0.2–1.0)	0.222
Dunedin			
Female participants	256		
FEV_1 % pred		0.05 (−0.4–1.4)	0.283
FVC % pred		**0.9 (0.0–1.8)**	**0.048**
FEV_1/FVC %		−0.3 (−0.8–0.2)	0.195
Male participants	348		
FEV_1 % pred		**1.7 (0.5–2.8)**	**0.004**
FVC % pred		**2.1 (1.1–3.2)**	**<0.001**
FEV_1/FVC %		−0.2 (−0.7–0.4)	0.585

FEV_1: forced expiratory volume in 1 s; FVC: forced vital capacity. For the Dunedin study, age 29 years maximal oxygen uptake (V'_{O_2max}), FEV_1, FVC, FEV_1/FVC ratio, height and weight values are calculated as the mean of the measurements at ages 26 years and 32 years. The independent (predictor) variable is change in V'_{O_2max} in sex-specific standard deviation scores. Coefficients represent the difference in lung function associated with each standard deviation difference in change in V'_{O_2max}. Analyses were adjusted for sex and asthma at age 15 years, height, weight, smoking and asthma at both ages. Bold type represents statistical significance.

fitness and physical activity are only moderately correlated among children and adolescents [24], these observations appear to be consistent with our findings for physical fitness. A 20-year follow-up of adults in the Coronary Artery Risk Development in Young Adults (CARDIA) study suggests that maintaining or improving aerobic fitness through mid-adult life also helps to slow lung function decline [27]. Taken together, these findings suggest that enhancing physical fitness throughout the life-course may play an important role in maximising and maintaining adult lung function.

Smoking is associated with other health risk behaviours including physical inactivity [40], and previous studies have suggested that there may be an interaction between the effects of smoking and physical inactivity on lung function in adults [10, 19]. We adjusted for smoking and found no significant interactions at any age in either study indicating that smoking does not explain or modify the associations between fitness and lung function during young adulthood.

Strengths of this study include the use of complementary data from two population-based cohorts of children and young people from Denmark and New Zealand. Both studies followed standard exercise test protocols and were administered by trained personnel at each age, but estimating peak V'_{O_2max} from work rate is inexact and we also cannot exclude the possibility of small changes in protocols over the 20 years of the studies. However, such errors would be most likely to bias any associations between fitness and lung function towards the null, and would not explain the pattern of associations that we have observed. Although the ages and methods of assessing maximal aerobic fitness were different, the findings were similar in both the cross-sectional analyses at the common assessment age of 15 years (tables 3 and 4) and in the longitudinal analysis of changes from the ages of 15 years to 29 years (table 7). Both studies used standard techniques to measure lung function and directly measured the main confounders of height and weight. We also adjusted for asthma, because it is possible that exercise-induced bronchospasm could confound the findings. Excluding participants with asthma from the analyses did not materially change the results (online supplementary tables S5a, S5b, S6a and S6b). Limitations include the fact that only the Odense study measured exercise fitness in prepubertal children and recorded pubertal development at the age of 15 years and that only the Dunedin study extended beyond the late twenties. Neither study has yet been extended to older adults. There was some loss to follow-up in the cohorts, most notably in the Odense study, and it is possible that those who didn't attend were reluctant to undertake exercise tests. Some participants have missing data for confounding variables, such as asthma diagnoses, which reduced

the number of participants in the adjusted analyses. While these missing data may reduce the representativeness of the samples, it is unlikely that this would substantially influence the observed association between fitness and lung function.

Further research is needed to identify plausible mechanisms by which fitness and lung function may be related. In addition, we need to know whether maintaining fitness will help to preserve lung function in old age. Greater lung volumes and fitness are both associated with lower all-cause mortality. The findings of this study give further reason to encourage exercise and the development of fitness in young people.

In summary, aerobic fitness is associated with greater lung volumes in children and young adults. Improvements in fitness during childhood and adolescence are associated with greater growth in lung volumes at follow-up.

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