Table 1 Mean FEV$_1$%/FVC and FEV$_1$ values from 28 authors and from the study of Albers and colleagues[1]

	FEV$_1$/FVC (%)			FEV1 (l)		
Age (year)	43	48	Decline	43	48	Decline
Mean	80.2	79.2	1.0	3.486	3.350	0.135
Range	73.8–84.1	72.4–83.5	0.4–1.6	3.10–3.82	2.95–3.66	0.11–0.16
Mean[1]	84.5	79.3	5.2	3.532	3.335	0.197
SD[1]	9.8	8.8	–	0.833	0.806	–

FEV$_1$, forced expiratory volume in 1 s; FVC, forced vital capacity.

without baseline abnormalities. In 5 years, forced expiratory volume in 1 s (FEV$_1$) fell by 200 ml and FEV$_1$%/vital capacity (VC) by 5.2%, remarkably large declines for such subjects. We computed predicted values at ages 43 and 48 years according to 28 authors who had published predicted values for FEV$_1$%/forced vital capacity (FVC) for Caucasians, and 30 who had done so for FEV$_1$.[2] The results are shown in table 1; the values reported by Albers and colleagues[1] are in the bottom two rows.

The decline in FEV$_1$/FVC during the study period was more than five times the expected average drop; the fall in FEV$_1$ was larger than expected. If the non-smokers declined at an expected rate (135 ml in 5 years) and we attribute the excess decline to smokers, the decline in smokers must have been 340 ml; as a minority of smokers exhibit an accelerated decline in FEV$_1$ leading to COPD,[3] a limited number of smokers must have had a decline in FEV$_1$ far in excess of this. In that case, one would expect an increase in the scatter, but the SD of FEV$_1$ did not increase. The decreased scatter in FEV$_1$/VC over the 5 year period suggests that the group became more homogeneous, which makes it unlikely that the excess decline was caused by a subgroup. In any study, the ratio of average FEV$_1$ and average VC is not exactly equal to the average FEV$_1$/VC ratio; however, in 4557 observations from a random sample of a Dutch population, the difference was very small: 0.7623 vs 0.7635. Thus if we reconstruct the VC in the study of Albers and colleagues[1] from FEV$_1$ and FEV$_1$/VC, VC at baseline was about 4.18 l, and 5 years later 4.21 l, so there was at best a trivial change.

One wonders whether these unusual findings are caused by problems with data collection which would invalidate the conclusions of this study. The authors state that variation in spirometer performance was assessed and accounted for; this merits a more detailed account. Measurements were performed according to the American Thoracic Society standards,[4] but the study started prior to that report; were measurements recalculated?

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Competing interests: None declared.

Accepted 26 June 2008

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Authors' response

We thank Quanjer et al for their detailed comments on our study. The decline in lung function is a key aspect of COPD. The DIMCA study has been one of the first that focused on patients in the early stage of COPD and collected data covering a period of 10 years. It is therefore important to review the quality and reliability of our data.

In response to the comments raised by Quanjer et al we would like to stress the following. Our paper[1] reported on the first 5 years of follow-up of our study population. For the baseline and year 10 measurements, the same spirometer (Microspiro HI-298 spirometer; Chest Corporation, Japan) was used. Because a different spirometer was used at year 5 (Fukuda Sangyo spiro analyzer ST-250, Japan), equipment performance was assessed before as well as after lung function measurements in all participants had been completed. As we observed a systematic linear deviation in the lung function indices compared with the original spirometer, we considered it necessary to account for this. Further support for the reliability of our data was found in the follow-up of our study subjects. After 10 years, lung function was reassessed using the same spirometer that was used at baseline, and all assessments at year 10 were performed by the same lung function technician who performed the baseline assessments. We have now analysed the 10 year follow-up data and observed a further lung function decline that was fully in line with the pattern presented at year 5. Although the 10 year data have not been published to date (Mieke Albers thesis: COPD in primary care. Aspects of secondary prevention; chapters 6 and 7), the group of subjects without baseline abnormalities showed a decline in forced expiratory volume in 1 s (FEV$_1$) amounting to 348 ml over the 10 year observation period. Over the 5 year period, this decline amounted to 197 ml. The decline in FEV$_1$/vital capacity (VC) was 10.8% after 10 years of follow-up and 5.2% after 5 years of follow-up.

Given the quality of the measurements and consistency of the pattern over time, we do not think there are reasons to doubt our findings. Quanjer et al point to the use of FEV$_1$/FVC. It is to be expected that our findings would have been arithmetically different had we used this ratio instead of the FEV$_1$/VC ratio. But the systematic difference still leaves the prediction in decline intact. For that reason, we do not believe there were fundamental flaws in our study, although we agree that the decline is relatively high compared with findings in previous population cohorts. We have no explanation for this.

Quanjer et al are correct in that it would have been more appropriate to refer to the 1987 update of the American Thoracic Society statement on the standardisation of spirometry. At the time, this guideline served as the basis of procedures in the lung function laboratory of the University Lung Centre Dekkerswald, where all of our study subjects were measured.

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No role for routine CT scans in paediatric empyemas

In the paper by Jaffe et al, the authors describe the CT findings of 31 patients with thoracic empyema who had three investigations (chest radiography, CT scan and ultrasound scan).[1] They correctly conclude that routine CT scanning has no role for children with empyema treated with urokinase and percutaneous chest drainage. It is interesting to note that CT scanning is becoming popular as nearly half the subjects