Factors That Affect Normal Lung Function in White Australian Adults*

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**Study objective:** To classify abnormal lung function in epidemiologic studies, we first calculated “normal” values using data from Australian white adults. We then examined the effects of airway hyperresponsiveness (AHR), respiratory symptoms, current and past asthma, and current smoking on spirometric function.

**Methods:** A large random sample of 1,527 adults aged 18 to 73 years was studied. We measured respiratory symptoms and smoking history by questionnaire and AHR by histamine inhalation test.

**Results:** Data from 729 “normal” subjects (asymptomatic nonsmokers without AHR) were used to obtain regression models for FVC, FEV₁, peak expiratory flow rate, and forced expiratory flow between 25% and 75% of FVC. The R² values were 0.76, 0.74, 0.58, and 0.29, respectively. The presence of AHR reduced FVC by 0.1 L and FEV₁ by 0.2 L, on average. Subjects with asthma-related symptoms had a mean reduction in FVC of 0.1 L for both genders and in FEV₁ of 0.08 L for women and 0.2 L for men. Current asthma reduced FVC by 0.3 L, on average, and FEV₁ by 0.5 L for women and 0.6 L for men. The FEV₁ was reduced by 0.002 L per cigarette smoked daily.

**Conclusion:** Recent symptoms, AHR, and current smoking were all important predictors of reduced lung function. (CHEST 1997; 112:1539-46)

**Key words:** airway hyperresponsiveness; asthma related symptoms; current, past asthma; current smoking; lung volume

**Abbreviations:** AHR=airway hyperresponsiveness; ATS=American Thoracic Society; CI=confidence interval; FEF25.75%=forced expiratory flow between 25% and 75% FVC; %FEV₁=percentage of predicted FEV₁; PEFR=peak expiratory flow rate

The factors that cause a reduction in lung function must be established before effective prevention programs can be undertaken. Many studies in Europe and the United States have demonstrated a strong relationship between reduced pulmonary function and airway hyperresponsiveness (AHR), respiratory symptoms (wheeze, shortness of breath, asthma), and cigarette smoking. Cross-sectional studies of adults have consistently shown that lung function is lower in subjects with respiratory symptoms and/or AHR. Furthermore, longitudinal studies have reported a more rapid decline of FEV₁ with age in subjects with symptoms or AHR than in normal subjects. Observations from numerous cross-sectional and follow-up studies have also identified cigarette smoking as an important determinant of the level of lung function. However, the effects of these factors have not been widely studied in Australian adults in whom the prevalence of AHR and respiratory symptoms is high.

To examine the association between lung function and the factors that affect it, it was necessary to obtain accurate prediction models for normal lung function. Woolcock et al have recommended that accurate prediction formulas for measurements of ventilatory lung function should be derived from the population being studied. The American Thoracic Society (ATS) recommends that prediction equations should be obtained from large cross-sectional samples tested by standardized technical methods. However, normal values in Australian adults presented in the literature have used different equipment and/or a population not representative of ours.

The aim of this study was to examine the facts that affect normal lung function in Australian adults. To do this accurately, we first calculated “normal” values for lung function for white Australian adults. These
subjects came from the same population and were totally free from respiratory symptoms, AHR, and a history of asthma, and they were not current smokers. Using these prediction equations for normal lung function, we were then able to explore the effects of AHR, asthma-related symptoms, current and past asthma, and current smoking on spirometric function.

**Materials and Methods**

**Study Population**

The studies were conducted in two rural regions of NSW (Lismore and Wagga Wagga) in 1991 and in 1992. We studied a large random sample of children in winter and, 3 months later, we studied the parents and guardians of these children. We asked all adults living in the same household to complete a questionnaire and then to attend a location in the town center for lung function and allergy tests. Follow-up phone calls were made to arrange appointments. A random selection of refusers and non-attenders was surveyed by telephone to collect information of respiratory symptoms and asthma medication use.

**Respiratory Symptoms and Interviews**

Each subject completed a self-administered questionnaire that was a shortened version of the International Union Against Tuberculosis questionnaires.\(^{20}\) We collected information about recent and past respiratory symptoms, family history of asthma, diagnosed asthma and asthma medication use, and hospital and physicians attendances. In addition, questions about smoking history and occupation were included. All subjects were divided into five categories of occupation: professional, clerical (white collar), skilled (blue collar), unskilled, and unemployed. The methods used in these studies and occupation characteristics of the sample have been described in detail.\(^{12}\)

**Physical Characteristics**

Subjects were weighed in indoor clothing and without shoes. Height was measured, also without shoes, with heels against a wall on which a height measure was placed. Ethnicity was assessed by visual characteristics and was assigned as white or non-white.

**Lung Function**

Baseline forced expiratory curves were measured with the subject standing and without a nose clip, using dry rolling seal spirometers (Mijnhardt VRS 2000; Mijnhardt BV; Bunnik, Holland). For computerized data collection, the spirometer was connected to a laptop computer, running data acquisition software (Scientific and Medical; S&M Instrument Company Inc; Doylestown, Pa). The spirometers adhered to the standards recommended by ATS. The calibration of each spirometer was checked weekly using a 3-L syringe. Barometric pressure and ambient temperature were measured at the time of the study and all spirometric results were corrected to BTPS by the software (S&M) during real-time data collection. The measurements were repeated until readings for FEV\(_1\) and FVC were reproducible to within 100 mL and readings with the largest value of FEV\(_1\) were used in analysis. Subjects who had taken a \(\beta\)-agonist <6h before the test were asked to make another appointment and to withhold treatment with this medication before testing.

**Asthma Hyperresponsiveness**

AHR was assessed by histamine inhalation test using the rapid method.\(^{21}\) After baseline lung function was recorded, two puffs of saline solution were administered as a control and lung function was recorded again. Histamine diprophosphate was then administered by use of hand-held nebulizers (DeVilbiss No. 45; DeVilbiss Medizinische; Langen, Germany) in doses ranging from 0.06 to 3.9 \(\mu\)mol histamine. The challenge was stopped if the FEV\(_1\) fell by 20% or if the highest dose of histamine had been given. Subjects with a fall in FEV\(_1\) >10% were given salbutamol aerosol to aid recovery. Subjects who presented with baseline FEV\(_1\) <60% of predicted did not undergo histamine challenge but were given 200 \(\mu\)g salbutamol from a metered-dose inhaler and lung function was measured again after 10 min. Response to bronchodilator was measured as the increase in FEV\(_1\) as a percentage of the initial value. An increase of \(\geq 15\%\) was regarded as positive. Subjects with a fall in FEV\(_1\) of \(\geq 20\%\) following histamine inhalation test, or a positive bronchodilator response, were classified as having AHR.

**Definitions**

**Asthma-Related Symptoms:** Subjects who reported respiratory symptoms (wheeze, wheeze following exercise, chest tightness on waking, or shortness of breath coming on at rest) or an asthma attack in the 12 months prior to study were classified as having “asthma-related symptoms.”

**Current Asthma:** Subjects with AHR and asthma-related symptoms as defined above were classified as having “current asthma.”

**Past Asthma:** Subjects without AHR or asthma-related symptoms but with a previous diagnosis of asthma were classified as having “past asthma.”

**Asymptomatic Current Smokers:** Subjects who did not belong to any of the above groups but who smoked at least 10 cigarettes per day were considered to be “asymptomatic current smokers.”

**Normal Group:** Subjects without AHR or asthma-related symptoms, who did not have past asthma and who were not asymptomatic current smokers were classified as “normal.”

**Statistical Analysis**

Statistical analysis was performed using a standard statistical package (SAS; SAS Institute Inc; Cary, NC). To calculate predictive regression models in the normal group, multiple regression analysis was performed using a stepwise procedure. For the current study, FVC, FEV\(_1\), peak expiratory flow rate (PEFR), and forced expiratory flow between 25% and 75% of FVC (FEF\(_{25-75}\%\)) were analyzed separately. For FVC and FEV\(_1\), the control (after saline solution) value was used if it was larger than the baseline value. To test the hypothesis that the mean percentage of predicted FEV\(_1\) values was different between respiratory groups, we performed analysis of variance with Duncan’s multiple range post hoc test. The effects of asthma-related symptoms, AHR, past asthma, and occupation on measurements of lung function were assessed by adding these factors as dummy variables to regression models, in which all subjects were included. The effect of current smoking was examined by adding the number of cigarettes smoked daily as a continuous variable to the model. The effect of all biologically plausible interactions between covariates was examined by adding their interaction terms to the model.
RESULTS

A total of 1,527 subjects aged 18 to 73 years from two rural Australian areas participated in this study. These subjects represented approximately 61% of the selected population; 57.8% were female. Due to the study design, most of the sample (99%) were subjects aged 30 to 50 years. After excluding non-white subjects and subjects with technically unsatisfactory lung function tests or with missing data, 1,499 subjects remained for analysis, of whom 729 comprised the “normal group.” Table 1 shows the anthropometric characteristics of the subjects included in the analyses and Figure 1 illustrates the selection criteria for our normal group. Subjects were divided into six mutually exclusive groups on the basis of their asthma-related symptoms, AHR, current and past asthma, and their smoking status. Only 49% of the sample (729 subjects) comprised the normal group; 17% were asymptomatic former smokers. Table 2 shows that normal subjects were not significantly different from the rest of the sample in terms of the mean age, height, and weight (p>0.05).

The data for the normal subjects were used to obtain regression equations for FVC, FEV₁, PEFR, and FEF₂₅₋₇₅%, with weight, age, gender, and polynomial transformation of height as the main predictors. The models of best fit for each lung function parameter are shown in Table 3. These models explained 76% of the variation in FVC, 74% of the variation in FEV₁, 58% of the variation in PEFR, but only 29% of the variation in FEF₂₅₋₇₅%. Men had a significantly larger reduction in FEV₁ with age (0.032 L/yr, 95% confidence interval [CI], −0.039, −0.025 L/yr) than women (0.02 L/yr, 95% CI, −0.027, −0.013 L/yr). Examination of the residuals for each model showed no violations of the assumption of normality or linearity and there were no outliers or remote points that had a significant effect on the regression coefficients. The lower limits of normal, which represent the limits above which 95% of the normal group falls, were age and gender specific and are listed as percent predicted in Table 4.

Using our prediction equations, we calculated mean percentage of predicted FEV₁ values (%FEV₁) for the whole sample (Fig 2). Both women and men in the current asthma group had significantly lower mean %FEV₁ values (83%) than those of other groups. There was no statistically significant difference in %FEV₁ values between male and female subjects within any group.

We then examined the factors that affect lung function (Table 5). All four lung function parameters were lower in the presence of AHR and/or asthma-related symptoms. For PEFR, the age slope was modified by the symptoms indicators. The presence of symptoms increased the decrement in PEFR with age from 0.021 L/s/yr (95% CI, −0.03, −0.007 L/s/yr) to 0.06 L/s/yr (95% CI, −0.08, −0.04 L/s/yr). Neither a history of past asthma nor occupation had any significant effect on FVC, FEV₁, PEFR, or FEF₂₅₋₇₅%, after adjusting for the effects of AHR, symptoms, and/or current smoking.

Multiple regression analysis showed that not only were AHR and asthma-related symptoms significant in relation to the reduction in FEV₁, but the interaction term of AHR and recent symptoms was also significant (p<0.01). The combination of AHR and asthma-related symptoms allowed us to estimate the effect of current asthma on lung function. Therefore,

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**Table 1—Anthropometric Characteristics of 639 Men and 860 Women Representing the Australian Rural Population**

<table>
<thead>
<tr>
<th>Age Group, yr</th>
<th>No.</th>
<th>%</th>
<th>Height, cm</th>
<th>Weight, kg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Women</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20</td>
<td>1</td>
<td>0.12</td>
<td>164</td>
<td>—</td>
</tr>
<tr>
<td>20-29</td>
<td>44</td>
<td>5.1</td>
<td>164.2</td>
<td>5.3</td>
</tr>
<tr>
<td>30-39</td>
<td>535</td>
<td>62.2</td>
<td>162.8</td>
<td>5.9</td>
</tr>
<tr>
<td>40-49</td>
<td>263</td>
<td>30</td>
<td>162.5</td>
<td>5.9</td>
</tr>
<tr>
<td>50-59</td>
<td>17</td>
<td>1.98</td>
<td>160.5</td>
<td>8.8</td>
</tr>
<tr>
<td>Total</td>
<td>860</td>
<td>100</td>
<td>162.7</td>
<td>6.0</td>
</tr>
<tr>
<td>Men</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20</td>
<td>5</td>
<td>0.78</td>
<td>182.6</td>
<td>10.5</td>
</tr>
<tr>
<td>20-29</td>
<td>21</td>
<td>3.29</td>
<td>177.4</td>
<td>7.9</td>
</tr>
<tr>
<td>30-39</td>
<td>303</td>
<td>47.4</td>
<td>176.3</td>
<td>6.6</td>
</tr>
<tr>
<td>40-49</td>
<td>269</td>
<td>42.1</td>
<td>176.1</td>
<td>6.6</td>
</tr>
<tr>
<td>50-59</td>
<td>37</td>
<td>5.73</td>
<td>175.2</td>
<td>6.8</td>
</tr>
<tr>
<td>60-69</td>
<td>2</td>
<td>0.31</td>
<td>163.5</td>
<td>0.71</td>
</tr>
<tr>
<td>&gt;70</td>
<td>2</td>
<td>0.31</td>
<td>169.5</td>
<td>3.5</td>
</tr>
<tr>
<td>Total</td>
<td>639</td>
<td>100</td>
<td>176.2</td>
<td>6.7</td>
</tr>
</tbody>
</table>
subjects with current asthma had a reduction in FVC of 0.252 L on average, for both genders, in $FEV_1$ of 0.505 L for women and 0.632 L for men, and in $FEF_{25-75\%}$ of 1.002 L/s for women and of 1.659 L/s for men.

Three lung function measurements ($FEV_1$, PEFR, and $FEF_{25-75\%}$) were found to be significantly lower in current smokers compared with nonsmokers. The reduction was in proportion to the number of cigarettes smoked daily. $FEV_1$ was reduced by 0.002 L ($p=0.053$), PEFR by 0.006 L/s ($p<0.05$) and $FEF_{25-75\%}$ by 0.004 L/s ($p<0.05$) per cigarette smoked daily.

**Discussion**

This study presents the lung function data and prediction equations for several lung function measurements obtained from 729 healthy, nonsmoking white men and women living in an Australian rural environment and quantifies the deficit in lung function that is associated with AHR, asthma-related symptoms, and current smoking. Our spirometric standards were based on measurement techniques that followed the recommendations of the ATS and data were obtained by trained operators using modern equipment that met ATS criteria. The subjects represented a wide range of heights and were drawn from various occupational and social classes. Our relatively strict exclusion criteria for normality meant that 51% of our random sample were not included in the calculation of predicted values. Because we studied a large random sample, we were able to calculate regression coefficients with precision. However, our sample was biased toward individuals aged 30 to 50 years who comprised 99% of our study sample. Thus, our results can be applied only to the white Australian adults in this age range.

It was estimated that the 1,527 responders comprised at least 61% of the sample base. A random selection of refusers and nonattenders was surveyed by telephone to collect information of asthma-related symptoms and medication use. The survey showed that subjects with asthma-related symptoms were more likely to attend, although the differences

<table>
<thead>
<tr>
<th>Sample</th>
<th>No.</th>
<th>Height, cm</th>
<th>Weight, kg</th>
<th>Age, yr</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Range</td>
<td>Mean (SD)</td>
<td>Range</td>
</tr>
<tr>
<td>Women</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>860</td>
<td>144-184</td>
<td>162.7 (5.9)</td>
<td>38-130</td>
</tr>
<tr>
<td>Normal subjects</td>
<td>435</td>
<td>146-182</td>
<td>162.2 (5.9)</td>
<td>38-130</td>
</tr>
<tr>
<td>Men</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>639</td>
<td>158-199</td>
<td>176.2 (6.7)</td>
<td>50-150</td>
</tr>
<tr>
<td>Normal subjects</td>
<td>294</td>
<td>158-199</td>
<td>176.4 (7.1)</td>
<td>50-150</td>
</tr>
</tbody>
</table>
in prevalence of symptoms between attenders and nonattenders were not statistically significant.\textsuperscript{12} Furthermore, no bias was introduced by the subsampling procedure for the normal group, since there were only minor differences in age, height, and weight of the total sample and normal group (Table 2).

Depending on the baseline value of FEV\textsubscript{1}, we have used two different criteria (response to histamine challenge and response to bronchodilator) to demonstrate the presence of AHR. Although these two criteria cannot be directly compared, both tests are a measure of airway lability and were classified together, since both are well-documented characteristics of asthma.\textsuperscript{22,23} In our study, only 20 subjects (1% of the population) did not do histamine inhalation test because of low baseline FEV\textsubscript{1} but were given bronchodilator challenge. Inclusion of these subjects would not have significantly altered our findings, since only 12 subjects had a positive bronchodilatory response and were included in the AHR group and the other 8 had asthma-related symptoms and were considered as symptomatic without AHR.

Inclusion of former smokers in the normal group did not influence the findings of the study. In our normal group, 17% of the subjects were asymptomatic former smokers. We examined the effect of ex-smoking on normal lung function by stratifying our normal group according to former smoking status (nonsmoker vs former smoker) and by adding smoking status as a dummy variable to the models. Multiple regression showed that the effect of ex-smoking was not statistically significant after allowing for height, age, gender, and/or weight differences.

In our prediction models, the variation in lung function parameters that could be explained by the model ($R^2$) was comparable to, if not better than, those of other authors.\textsuperscript{10,24-26} The age coefficients were similar to those of other published linear regression equations and showed, on average, a yearly reduction in FVC of 26 mL, in FEV\textsubscript{1} of 32 mL for men and 20 mL for women, in PEFR of 26 mL/s, and in FEF\textsubscript{25-75\%} of 33 mL/s/yr on average. In the models for PEFR and FEF\textsubscript{25-75\%}, weight was a significant predictor, in contrast to the models published previously in which weight was not included.\textsuperscript{19,25,26} Thus, our data demonstrated that PEFR

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**Table 3—Normal Values for Lung Function in 729 White Australian Adults**

<table>
<thead>
<tr>
<th></th>
<th>FVC, L</th>
<th>FEV\textsubscript{1}, L</th>
<th>PEFR, L/s</th>
<th>FEF\textsubscript{25-75%}, L/s</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Regression</strong></td>
<td><strong>Coefficient (SE)</strong></td>
<td><strong>P Value</strong></td>
<td><strong>Coefficient (SE)</strong></td>
<td><strong>P Value</strong></td>
</tr>
<tr>
<td>Intercept</td>
<td>1.609 (0.176)</td>
<td>0.0001</td>
<td>1.754 (0.170)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Height\textsuperscript{2}, m\textsuperscript{2}</td>
<td>0.624 (0.028)</td>
<td>0.0001</td>
<td>0.459 (0.024)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Age, yr</td>
<td>-0.026 (0.003)</td>
<td>0.0001</td>
<td>-0.020 (0.003)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>0.592 (0.048)</td>
<td>0.0001</td>
<td>0.991 (0.202)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Interactions:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age and male</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R\textsuperscript{2}</td>
<td>0.7639</td>
<td></td>
<td>0.7402</td>
<td></td>
</tr>
<tr>
<td>MSE</td>
<td>0.17809</td>
<td></td>
<td>0.1293</td>
<td></td>
</tr>
</tbody>
</table>

*R^2= (square of multiple correlation coefficient) = percentage of variance that can be explained by the model; MSE = mean square error of the estimate.

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**Table 4—Lower Limits of Normal: 95th Percentile of Percent Predicted**

<table>
<thead>
<tr>
<th></th>
<th>18-35 yr</th>
<th>36-45 yr</th>
<th>46+ yr</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>95th Percentile</td>
<td>No.</td>
</tr>
<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FVC</td>
<td>48</td>
<td>&gt;84.9</td>
<td>205</td>
</tr>
<tr>
<td>FEV\textsubscript{1}</td>
<td>48</td>
<td>&gt;84.0</td>
<td>205</td>
</tr>
<tr>
<td>PEFR</td>
<td>48</td>
<td>&gt;72.5</td>
<td>205</td>
</tr>
<tr>
<td>FEF\textsubscript{25-75%}</td>
<td>48</td>
<td>&gt;63.9</td>
<td>205</td>
</tr>
<tr>
<td><strong>Women</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FVC</td>
<td>135</td>
<td>&gt;84.6</td>
<td>270</td>
</tr>
<tr>
<td>FEV\textsubscript{1}</td>
<td>135</td>
<td>&gt;84.9</td>
<td>270</td>
</tr>
<tr>
<td>PEFR</td>
<td>135</td>
<td>&gt;73.2</td>
<td>270</td>
</tr>
<tr>
<td>FEF\textsubscript{25-75%}</td>
<td>135</td>
<td>&gt;66.6</td>
<td>270</td>
</tr>
</tbody>
</table>

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CHEST / 112 / 6 / DECEMBER, 1997

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increased by 11 mL/s per kg increase in weight on average and FEF_{25-75} by 8 mL/s per kg increase in weight. In addition, we obtained higher values for the lower limits of normal for FVC, FEV₁, and FEF_{25-75} (Table 4) than those widely used in clinical practice. The inclusion of a larger number of clinically undetected abnormal subjects in the normal groups in other studies might explain the differences in lower 95th percentiles. The differences among the various reference values may result from different selection criteria for the normal subjects studied or from different characteristics of the populations included. The differences emphasize the need to use prediction equations for lung function that are specific for the given population.

In our prediction models, polynomial transformation of height (Ht^3 for FVC, FEV₁, and FEF_{25-75} and Ht^2 for PEFR) was the most important determinant of lung function. Regression models with natural logarithmic transformation and interaction terms, as used by other researchers, were tested but did not increase the adjusted R² of the models when applied to our data. We included gender as an explanatory variable rather than presenting separate models for male and female subjects and tested for interactions between gender and the other explanatory variables. The interaction was only statistically significant in the model for FEV₁ (p<0.05), indicating that the effect of age was significantly different between genders. Men had a significantly larger decrement in FEV₁ with age than women (32 vs 20 mL/yr) which was in agreement with other studies. For example, Morris et al reported a reduction in FEV₁ with age in the two genders similar to ours (32 vs 25 mL/yr for men and women accordingly), while Knudson et al reported values of 29 vs 19 mL/yr.

In this study, we were able to examine factors that affect lung function. We found that asthma-related symptoms, presence of AHR, and current smoking were all associated with reductions in FEV₁, PEFR, and FEF_{25-75} although current smoking did not have a significant effect on FVC. Thus, FVC was affected only by symptoms and AHR. For current smoking, there was a dose-response effect between

![Table 5](image)

**Table 5—Effects of AHR, Respiratory Symptoms, and Current Smoking on Lung Function in 1,499 Australians**

<table>
<thead>
<tr>
<th></th>
<th>FVC, L Regression Coefficient (95% CI)</th>
<th>FEV₁, L Regression Coefficient (95% CI)</th>
<th>PEFR, L/s Regression Coefficient (95% CI)</th>
<th>FEF_{25-75}, L/s Regression Coefficient (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>1.677 (1.42, 1.93)</td>
<td>2.028 (1.80, 2.25)</td>
<td>2.606 (1.55, 3.66)</td>
<td>3.559 (3.05, 4.07)</td>
</tr>
<tr>
<td>Height², m²</td>
<td>0.643 (0.60, 0.68)</td>
<td>0.467 (0.42, 0.50)</td>
<td>-0.021 (-0.03, -0.026)</td>
<td>-0.039 (-0.05, -0.031)</td>
</tr>
<tr>
<td>Age, yr</td>
<td>-0.027 (-0.031, -0.023)</td>
<td>-0.028 (-0.03, -0.026)</td>
<td>-0.021 (-0.03, -0.007)</td>
<td>-0.039 (-0.05, -0.031)</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>0.523 (0.45, 0.59)</td>
<td>0.502 (0.44, 0.57)</td>
<td>1.932 (1.72, 2.14)</td>
<td>0.632 (0.48, 0.78)</td>
</tr>
<tr>
<td>Male</td>
<td>0.136 (-0.22, -0.05)</td>
<td>-0.201 (-0.33, -0.07)</td>
<td>-0.767 (-1.02, -0.51)</td>
<td>-0.785 (-1.00, -0.57)</td>
</tr>
<tr>
<td>Recent symptoms</td>
<td>-0.116 (-0.17, -0.06)</td>
<td>-0.078 (-0.14, -0.01)</td>
<td>-1.31 (-0.32, -2.30)</td>
<td>-0.217 (-0.36, -0.07)</td>
</tr>
<tr>
<td>No. of cigarettes</td>
<td>-0.002 (-0.004, -0.00004)</td>
<td>-0.006 (-0.001, -0.00001)</td>
<td>-0.004 (-0.008, -0.00008)</td>
<td></td>
</tr>
<tr>
<td>Interactions:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AHR and recent</td>
<td>-0.226 (-0.39, -0.06)</td>
<td>-0.226 (-0.39, -0.06)</td>
<td>-0.219 (-0.44, -0.0005)</td>
<td>-0.438 (-0.79, -0.09)</td>
</tr>
<tr>
<td>symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male and recent</td>
<td>-0.127 (-0.22, -0.03)</td>
<td>-0.039 (-0.06, -0.01)</td>
<td>-0.039 (-0.06, -0.01)</td>
<td></td>
</tr>
<tr>
<td>symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age and recent</td>
<td></td>
<td></td>
<td>-0.039 (-0.06, -0.01)</td>
<td>-0.039 (-0.06, -0.01)</td>
</tr>
<tr>
<td>symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AHR and male</td>
<td></td>
<td></td>
<td>-0.438 (-0.79, -0.09)</td>
<td>-0.438 (-0.79, -0.09)</td>
</tr>
<tr>
<td>R²</td>
<td>0.7180</td>
<td>0.6767</td>
<td>0.5488</td>
<td>0.3226</td>
</tr>
<tr>
<td>MSE</td>
<td>0.205</td>
<td>0.1618</td>
<td>1.789</td>
<td>0.803</td>
</tr>
</tbody>
</table>

*Abbreviations as in Table 3.*
number of cigarettes smoked daily and the reduction in lung function. In Figure 3, all lines were plotted for a given average height of 169 cm. The line, representing asymptomatic current smokers, shows the effect of 20 cigarettes smoked daily. The mean reduction in FEV\textsubscript{1} associated with the presence of AHR was 200 mL with a further reduction if asthma-related symptoms were experienced (80 mL in women and >200 mL in men). The cumulative effect of AHR and asthma-related symptoms provided the estimation of reduction in lung function due to current asthma. Women with current asthma had a mean reduction in FEV\textsubscript{1} of 500 mL, while the reduction in FEV\textsubscript{1} for men was >630 mL on average. Other authors have reported similar findings. Marcus et al\textsuperscript{28} found that the presence of pulmonary symptoms was associated with higher prevalence rates of airflow obstruction. Sorlie et al\textsuperscript{29} reported that men with symptoms had mean values of FEV\textsubscript{1} that were approximately 0.5 L lower than those without symptoms.\textsuperscript{29} Boezen et al\textsuperscript{2} also showed that symptoms were related to impaired lung function and to increased variability of peak flow, and Rijcken et al\textsuperscript{3} reported that AHR was associated with level of lung function in a community-based sample of adults.

We examined all plausible interactions among AHR, symptoms, number of cigarettes smoked daily, gender, and age (Table 5). We found that the age slope was not modified by presence of AHR, symp-
toms, or current smoking for any lung function parameters except for PEFR, where the additional reduction in the symptoms groups was 0.039 L/s/yr. This is in contrast to the results of longitudinal studies that have reported a more rapid decline of FEV\textsubscript{1} with age in subjects with symptoms and AHR than in normal subjects.\textsuperscript{4-8} The differences in findings may be due to the effect of cross-sectional analysis, which summarizes the actual differences between individuals and does not reflect the longitudinal aspect of the effect of aging.

We found that smoking reduced three lung function parameters: FEV\textsubscript{1}, FEFR, and FEF\textsubscript{25-75%}. The adverse effect of smoking on lung function has been reported in numerous cross-sectional and longitudinal studies. One of the most recent cross-sectional studies in China showed that the adverse smoking effect was greater in women than in men.\textsuperscript{9} However, in our models, there were no interactions between smoking and gender and the reductions in lung function were the same for both genders. Tager et al\textsuperscript{30} also found that the magnitude of FEV\textsubscript{1} decline was similar for both men and women. Burchfiel et al\textsuperscript{10} in their 6-year follow-up study of Japanese-American men, showed that FEV\textsubscript{1} decline was significantly associated with duration of smoking, whereas association with intensity and pack-years was of borderline significance. The results were similar to our study in that there was a borderline significant effect of smoking on FEV\textsubscript{1} (p=0.053).

![Figure 3: Regression lines of FEV\textsubscript{1} on age for men and women in groups classified according to current smoking, asthma-related symptoms, AHR, and current asthma on FEV\textsubscript{1}. Effect of current smoking was calculated for asymptomatic subjects who smoked at least 20 cigarettes per day. All lines were plotted for a given average height of 169 cm.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21755/)
Marcus et al.\textsuperscript{28} also reported a significant dose-response relationship between FEV\textsubscript{1} and cigarette smoking measured as pack-years, number of cigarettes, or numbers of years smoked. Terho et al.\textsuperscript{31} showed that both FVC and FEV\textsubscript{1} were lower in smokers than in nonsmokers, but we were unable to find any significant effect of smoking on FVC.

It is important that reference values that are appropriate for both the equipment being used and the population being tested are used when screening populations or individuals in a clinical setting. Studies that select the most appropriate reference values will provide a more precise measure of the amount of abnormal lung function in the community. Our study shows that in making judgments about abnormalities in lung function, it is important to take AHR, asthma-related symptoms, and current smoking into account, while a history of asthma appears not to be important.

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REFERENCES