Pulmonary function changes after 1 h continuous heavy exercise in 0.21 ppm ozone

FOLINSBEE, LAWRENCE J., JOHN F. BEDI, AND STEVEN M. HORVATH. Pulmonary function changes after 1 h continuous heavy exercise in 0.21 ppm ozone. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 57(4): 984–988, 1984.—Prediction equations developed from previous ozone (O₃) exposure studies suggested that athletes exercising at near competitive intensities would be subject to alteration of pulmonary function during exposure to relatively low concentrations of O₃. Accordingly we exercised seven trained athletes for 1 h at 75% of maximal O₂ consumption in both room air and a 0.21 ppm O₃ environment. Pulmonary function tests, including forced expiratory maneuvers and maximum voluntary ventilation (MVV), were performed prior to and immediately following the 1-h test. Significant decreases in forced vital capacity (FVC, −7%), forced expired volume in 1.0 s (−15%), forced expiratory flow over the midhalf of FVC (−18%), and MVV (−17%) were recorded following O₃ exposure. The magnitudes of these changes are similar to those observed in subjects performing moderate intermittent exercise for 2 h in a 0.24 ppm O₃ environment. Symptoms reported following O₃ exposure included laryngeal and/or tracheal irritation and soreness and chest tightness on taking a deep breath. The observed alterations in lung functions in these subjects indicate that individuals performing heavy continuous exercise are more likely to be affected by lower O₃ levels.

THE VOLUME OF AIR INHALED during exposure to air pollutants is an important determinant of the subsequent effect of the pollutant on lung function. Many studies of ozone (O₃) exposure have utilized a low-intensity intermittent exercise protocol in order to simulate active ambient exposure (4, 8, 10). However, the O₃ concentrations have often exceeded even the worst-case levels observed in the ambient environment. More recent studies (1–3, 7, 11) have indicated that heavy exercise during pollutant exposures results in lung function impairment at levels (e.g., 0.20 ppm) that had previously been shown to cause no effects in resting or mildly exercising subjects; such O₃ concentrations have been frequently recorded in the ambient environment. The interaction of exercise ventilation and O₃ concentration in causing decrements in performance on lung function tests has been the subject of several studies that have examined a dose-response relationship (1, 6, 7, 11, 12). Because trained athletes are capable of sustaining heavy work and thus high minute ventilations, we reasoned that they could experience O₃-induced alterations in lung function at lower concentrations than would the less-fit portion of the population.

In the present study, high-intensity continuous exercise was performed during O₃ exposure to determine whether exposure to a low O₃ concentration (at or near the first stage alert level of 0.20 ppm) could induce deterioration of pulmonary function in a relatively brief (1-h) period.

METHODS

Subjects. The subjects were six well-trained men and one well-trained woman; all except one male were competitive distance cyclists and all were nonsmokers. Their physiological characteristics are presented in Table 1. Although questions have been raised regarding the sensitivity of female subjects to O₃ exposure, we (9) have previously been unable to demonstrate a difference in response between males and females working at comparable relative work loads during O₃ exposure. The woman included in the present study was well-trained and had forced vital capacity (FVC) comparable to that of a male of her height, and we therefore saw no reason to exclude her as a subject. Several of the individuals had participated at some time in the last 2 yr in an athletic competition under smoggy conditions. This was not a criterion for selection into the study but was a consequence of their racing schedule, which included many races in the Los Angeles basin. We had no prior knowledge of the subjects’ sensitivity to O₃ except for information they provided about prior ambient exposure. We waited a minimum of 2 wk following any ambient exposure before testing these subjects in the laboratory.

Preliminary evaluation of the subjects included pulmonary function tests [including forced expiratory maneuvers, maximal voluntary ventilation (MVV), and functional residual capacity by helium dilution], electrocardiogram, medical-history questionnaire, and a physical examination. None of the subjects had any history or evidence of cardiopulmonary impairment. Written informed consent was obtained from each subject prior to his or her voluntary participation in the experiment.1

Maximal exercise test. The maximal exercise test was performed on a cycle ergometer. The initial ergometer

1 The nature and purpose of the study and the risks involved were explained verbally and given on a written form to each subject prior to their voluntary consent to participate. The protocol and procedures for this study have been approved by the Committee on Activities Involving Human Subjects of the University of California, Santa Barbara.

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**TABLE 1. Anthropometric data of subjects**

<table>
<thead>
<tr>
<th>Subj No.</th>
<th>Sex</th>
<th>Age, yr</th>
<th>Ht, cm</th>
<th>Wt, kg</th>
<th>FVC, ml</th>
<th>FEV₁₋₅ FVC</th>
<th>%Predicted FVC</th>
<th>VO₂ max, ml kg⁻¹ min⁻¹</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>24</td>
<td>169.0</td>
<td>59.0</td>
<td>5120</td>
<td>0.83</td>
<td>140.3</td>
<td>63.5</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>22</td>
<td>181.9</td>
<td>73.0</td>
<td>6851</td>
<td>0.73</td>
<td>120.5</td>
<td>71.3</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>18</td>
<td>194.8</td>
<td>73.4</td>
<td>6368</td>
<td>0.98</td>
<td>107.2</td>
<td>75.7</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>25</td>
<td>172.1</td>
<td>67.2</td>
<td>5060</td>
<td>0.88</td>
<td>106.1</td>
<td>71.3</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>27</td>
<td>185.0</td>
<td>74.6</td>
<td>6142</td>
<td>0.89</td>
<td>106.9</td>
<td>67.0</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>21</td>
<td>183.1</td>
<td>72.2</td>
<td>6065</td>
<td>0.78</td>
<td>105.0</td>
<td>69.6</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>18</td>
<td>178.0</td>
<td>69.2</td>
<td>5655</td>
<td>0.88</td>
<td>101.9</td>
<td>72.9</td>
</tr>
</tbody>
</table>

Mean ±SD: 21.8 ± 3.7 180.8 ± 5.0 71.6 ± 2.8 6024 ± 2014 0.84 ± 20.07 107.9 ± 26.4 71.3 ± 2.9

FVC, forced vital capacity; FEV₁₋₅, forced expired volume at 1.0 s; VO₂ max, maximal O₂ consumption.

Power output was set at zero load (~33 W) and was subsequently increased by 33 W every 2 min until the subject was no longer able to continue. The subjects were free to choose their individual pedaling frequency, typically ~90/min. Measurements were taken continuously, and calculations were made at 1-min intervals. The only criterion for achievement of maximal O₂ consumption was the failure of the subject to continue despite intense encouragement. For the purposes of the present study, the peak O₂ consumption was sufficient to describe the subject’s aerobic capacity.

**Apparatus.** The subjects exercised on an electrically braked cycle ergometer (Quinton 845). CO₂ and O₂ contents of mixed expired air were determined from continuous samples drawn from a 3-liter mixing chamber through a pneumotachograph (Fleisch no. 3) previously calibrated using several constant flows that were validated with a large Tissot spirometer. All appropriate measuring devices were interfaced to a PDP 11/60 laboratory computer (Digital). Ventilation, O₂, and CO₂ were sampled at a frequency of 50 Hz, and gas exchange calculations were made each min after appropriate corrections for analyzer sampling delays. During the O₃ exposures, ventilation was measured with a 300-liter Tissot spirometer, and mixed expired gas aliquots were obtained and analyzed for CO₂ and O₂ with a gas chromatograph (Quinton). Subjects breathed through an Otis-McKerrow valve equipped with a vane (partition). The effective dead space of this valve is less than 100 ml (4), and the inspiratory and expiratory resistances are less than 0.50 kPa·l⁻¹·s⁻¹ at a flow of 2 l/s.

**Exposure chamber.** A double-walled, insulated, acrylic environmental chamber (1.8 m wide, 2.4 m long, and 2.6 m high) was used for the O₃ exposure. Inlet air was passed through potassium permanganate-impregnated aluminum oxide (Purafil) and particulate filters, including a high efficiency particulate air (HEPA) filter at a flow of approximately 2.8 m³/min. Air temperature and humidity were controlled by an air-conditioning system (BEMCO). O₃ was generated from pure O₂ using a corona discharge ozone generator. O₃ concentration was continuously monitored with an ultraviolet O₃ photometer (Dasibi) periodically calibrated against a standard O₃ photometer provided by the California Air Resources Board (El Monte, CA) and, more frequently, against the meter's internal standard O₃ generator. O₃ was sampled near the subject's head, but the inlet was arranged so that the subject's expired air would not contaminate the sample.

**Pulmonary function measurements.** Pulmonary function tests were performed prior to and immediately after the 1-h cycle rides. These tests included spirometry and MVV. The best of at least three satisfactory forced expirations was used. The largest (FV0) and forced expired volume in 1.0 s (FEV₁) were always reported, and the forced expiratory flow over the mid-half of the FVC (FEF₂₅₋₇₅) was taken from the best-effort test, defined as the test with largest sum of FVC and FEV₁. Pulmonary function tests were performed on a water-filled spirometer (Collins) interfaced to a laboratory minicomputer. Backup spiromgrams (volume-time) were recorded on a kymograph.

**Protocol.** Following the preliminary evaluation and maximal exercise test, the subjects performed two 1-h rides. The first was performed in an exercise laboratory with ambient indoor air ([O₃] < 0.01 ppm) so that the work load could be adjusted to obtain a steady-state power output of ~75% of maximal aerobic power. The control exposure was performed in the exercise laboratory because it was part of another study of exercise ventilation that required the on-line analysis of ventilation and metabolism not available in the exposure chamber. This also enabled a more accurate adjustment of the work load to the proposed 75% of maximum. On another day, at least 1 wk later, the subjects worked at the same work load in the environmental chamber that contained 0.21 ppm O₃. The temperature (19–21°C), relative humidity (60–70%), and air movement were similar for the two conditions. Ventilation was measured during the last 3 min of each 15 min of exercise. At the end of the exposure the subjects were interviewed regarding the symptoms experienced during the exposure period.

The experimental design was flawed because the control and O₃ exposures were not assigned randomly. Six of the subjects had completed the room-air portion of the experiment previously, and because of limited availability during their competitive season, a strictly counterbalanced design was not deemed practical. The seventh subject performed the O₃ exposure first. The control experiment of one of the subjects was repeated because of computer data losses, although the observed changes in pulmonary function were not affected. Thus the order was reversed for two of the subjects.

**Data analysis.** The data were analyzed by a two-factor analysis of variance (time × environment) with repeated measures across time and environment. The Neuman-Keuls post hoc test was applied where appropriate. An α level of P < 0.05 was selected for significance.

**RESULTS**

The chamber O₃ concentration averaged 0.21 ± 0.004 ppm. The mean air temperature was 20.8 ± 0.8°C and the mean relative humidity was 69 ± 3.4%.
ing levels for ambient air controls were <0.01 ppm O₃, 20.6 ± 0.5°C, and 66 ± 3.1%.

The results of the pulmonary function testing are shown in Table 2. There were no significant differences in the preexposure values between the two conditions for FVC, FEV₁₀, FEF25-75%, or MVV. Heavy exercise for 1 h in the clean-air condition produced no significant changes in the measured variables. However, under similar exercise conditions with 0.21 ppm ozone present there were significant decreases in FVC (6.8%), FEV₁₀ (14.8%), FEF25-75% (17.6%), and MVV (16.8%). Individual data for FEV₁₀ and MVV changes are plotted in Figs. 1 and 2.

Under clean-air conditions, ventilation during the 1-h period averaged 99.7 (85-108) l/min for the male subjects. The corresponding mean breathing frequency and tidal volume were 43/min and 2.35 liters, respectively. Ventilation measured during the chamber exposure averaged 89 (77-105) l/min at a breathing frequency at 39/min and tidal volume of 2.3 liters. This lower ventilation was attributed to the somewhat greater resistance and inertia of the Tissot spirometer used for these measurements, since the work load and O₂ consumption were the same under both exposure conditions. It is likely, however, that ventilation during free breathing (i.e., no mouthpiece) was similar under both conditions. The female subject had ventilations of 78 and 72 l/min in control and O₃-exposure conditions, respectively. Because of the difference in ventilatory measuring devices, analysis of variance of the ventilatory pattern was not performed. A Wilcoxon sign test revealed that during the O₃ exposure both the ventilation and the respiratory frequency tended to increase. The breathing frequency averaged some 8 breaths/min higher at the end than at the beginning of exposure. Tidal volume was not changed.

**Symptoms.** During O₃ exposure, six of the seven subjects complained of either substernal discomfort or chest tightness. Most of the subjects coughed following the forced expiratory or MVV tests, but only two reported coughing at other times during the O₃ exposure. Other symptoms reported by two or more individuals included shortness of breath, nausea or queasiness, dizziness, or fatigue. Fatigue was reported with similar frequency following room-air or O₃ experiments, as might be expected for work of this intensity and duration.

**DISCUSSION**

These data support our hypothesis that well-trained subjects exercising heavily during O₃ exposure would experience decrements in lung function at lower O₃ concentrations than those at which we had previously reported deleterious responses (7, 8, 12). Recently others (3, 11) have reported modest pulmonary function responses following exposure to even lower (0.15-0.18 ppm) O₃ concentrations.

The present observations are within the range of those reported from other studies of individuals exercising vigorously during O₃ exposure. Folinsbee et al. (7) found a ~7% decrease in FVC and FEV₁₀ in intermittently exercising subjects exposed for 2 h to 0.30 ppm O₃. (Based on the present ultraviolet standard, this concentration...
would be equivalent to ~0.24 ppm.) The ventilation during the above exposures averaged 67 l/min in the exercise periods. McDonnell et al. (11) have reported greater effects (14% decrease in FEV1.0) in subjects exposed to 0.24 ppm O3 using a similar intermittent exercise protocol with similar (65 l/min) exercise ventilation. In a group of trained runners exposed to 0.20 ppm O3 during heavy continuous exercise (ventilation = 80 l/min), Adams and Schlegle (2) demonstrated decreases in FVC and FEV1.0 of ~7 and 6%, respectively. A recent study (3), in which subjects performed moderately heavy (ventilation = 60 l/min) continuous exercise during exposure to 0.24 ppm O3, showed even greater decreases in FEV1.0 (~19%) than did our subjects.

Several efforts have been made to quantitate this interaction of exercise ventilation and O3 concentration by deriving some meaningful expression for the dose of pollutant delivered to the lung. The present observations represent a logical extrapolation of the effective-dose concept as described by Silverman et al. (12), Folinbee et al. (7), and Adams et al. (1). The prediction equations that have been developed (1, 6, 7) each use a dose parameter calculated from the product of ventilation x O3 concentration x time. The derived units differ with each technique, and the reader is referred to individual papers for complete details. In each of the above studies the derived equations would have predicted a pulmonary function decrement in response to lower O3 concentrations in combination with heavy exercise and the consequent higher ventilations.

From our previously developed prediction equations (7), we calculated that the decrement in FEV1.0 should range between 7 and 10% for the present subjects, who actually demonstrated a decrease of almost 15%. The total dose as described by Colucci (6) for the present study is 39.5 µg/min O3, and accordingly we should have expected an ~12% decrease in FEV1.0. The effective dose as described by Adams et al. (1) is ~1,180 ppm-l, and a decrease in FEV1.0 of ~9% would be expected on the basis of their data. Although the decrease in FEV1.0 that we observed is greater than predicted by the various equations, these predictions are based in many cases on data from subjects exposed to O3 while exercising intermittently (6, 7). There is considerable variability in the response of subjects to O3 both within the present study and between this and other studies. Had the unusually sensitive subject who demonstrated a >35% decrease in FEV1.0 not been included in our subject population, the mean decrease in FEV1.0 would have been only 10.5%.

Each of the above predictions was based mainly on data that involved exposure to higher O3 concentrations and subjects who performed less intense exercise. Furthermore, the simple product of O3 concentration, ventilation, and exposure time that has been used to establish dose may not be sufficient to accurately predict responses in subjects performing high-intensity exercise. O3-induced pulmonary function changes may be somewhat greater for continuous exercise exposure compared with intermittent exercise exposures, and prediction equations may have to be adjusted accordingly. Brief resting exposure to O3 at 0.21 ppm has no effect on pulmonary function in humans. Thus at these low O3 levels only exposures that occur during exercise, with the consequent hyperpnea and increased tidal volume, appear to be responsible for pulmonary function alterations (8). As we have previously demonstrated (8), pulmonary function decreases induced by exercise during O3 exposure will begin to return to normal after exercise even if there is continued resting exposure to O3. This observation suggests that there may be a seesaw effect with intermittent exercise exposures which is not present with continuous exercise exposure, and thus the latter may produce greater pulmonary function changes.

Adams and Schlegle (2) tested a group of trained runners who performed a simulated 30-min race while exposed to 0.20 ppm O3. Only half of the runners tested felt that they could have performed maximally if required to compete under such conditions. In our subject group, three of the five male cyclists related instances in which they believed their performance had been adversely affected while competing at a time when O3 levels were elevated. (The O3 concentrations during the races were determined from air-quality records and the time and date of the race. The levels ranged from 0.15 to 0.25 ppm.) The difficulties that they experienced were similar to those reported following chamber O3 exposures, with the major symptom being difficulty or discomfort during deep breathing. The simultaneous exposure to high ambient temperatures (85-95°F) may have influenced their judgement of the severity of the difficulties they experienced. A controlled study of exercise performance during O3 exposure at such low concentrations (0.15-0.20 ppm) is necessary to determine the actual magnitude of the effects on performance.

In summary, it is evident that pulmonary function impairment may be produced in heavily exercising subjects exposed for 1 hr to O3 concentrations of 0.20 ppm. These findings may have important implications for individuals exercising during midday O3 peaks in smoggy areas such as the South Coast Air Basin (Los Angeles).

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