©Journal of Sports Science and Medicine (2008) **7,** 229-234
http://www.jssm.org

Research article

Lung diffusion capacity can predict maximal exercise in apparently healthy heavy smokers

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Abstract
Chronic exposure to tobacco smoking may damage lung and heart function. The aim of this study was to assess maximal exercise capacity and its relationship with lung function in apparently healthy smokers. We recruited 15 heavy smokers (age 47 years ± 7, BMI 25 kg/m^2 ± 3, pack/years 32 ± 9) without any cardiovascular or pulmonary signs and symptoms. Fifteen healthy non smoking subjects were enrolled as a control group. All subjects underwent pulmonary function tests, electrocardiograms at rest and graded cycle exercise tests. In smokers and controls, resting lung and cardiac function parameters were in the normal range, apart from diffusing lung capacity (TLCO) values which were significantly lower in smokers ($p < 0.05$). As compared to controls, smokers presented lower maximal exercise capacity with lower values at peak of exercise of oxygen uptake (peak VO_2), workload, oxygen uptake/watt ratio and oxygen pulse ($p < 0.05$) and higher dyspnoea perception ($p < 0.05$). Moreover, peak VO_2, maximal workload and oxygen pulse at peak exercise were related to and predicted by TLCO ($p < 0.05$). Our study confirms that maximal exercise capacity is reduced in apparently healthy heavy smokers, and shows that TLCO explains some of the variance in maximal exercise.

Key words: Tobacco, lung function, exercise capacity, lung diffusion capacity.

Introduction

Smoking may impair work performance (Unverdorben et al., 2007) and it has been associated with low cardiovascular fitness (Bernaards et al., 2003). The effects of cigarette smoking on exercise capacity have been studied extensively (Bernaards et al., 2003; Bolinder et al., 1997; Pirnay et al., 1971; Horvath et al., 1975; Kobayashi et al., 2004; Morton et al., 1985, Song et al., 1998, Unverdorben et al., 2007). Some reports showed that aerobic capacity is reduced in smokers of various ages (Bernaards et al., 2003; Bolinder et al., 1997; Kobayashi et al., 2004; Unverdorben et al., 2007), whereas other studies reported no difference in maximal oxygen consumption (VO_2 max) between smokers and non smokers in populations of sportsmen (Morton et al., 1985; Song et al., 1998) and of young workers (Maksud and Baron, 1980).

The mechanisms of the impairment of exercise capacity in smokers are complex and multifactorial. Hirsch et al. (1985) studied the immediate effects of cigarette smoking on the cardiorespiratory response to exercise in healthy smokers without and after smoking and reported impaired oxygen delivery to the exercising muscles and lung ventilation-perfusion mismatch. Smoking-induced elevations in the carbon monoxide content of the blood can also reduce exercise tolerance and maximal aerobic capacity (Mc Donough and Moffatt, 1999). In addition, smoking significantly decreases lung diffusion capacity, which is inversely related to smoking history (Frans et al., 1975; Van Ganse et al., 1972). When compared to healthy controls, smoking subjects show lower lung diffusion capacity values both at rest and during exercise (Mahajan et al., 1991).

The purpose of this study was to assess maximal exercise capacity in sedentary, heavy smokers who were not affected by any apparent cardiovascular and lung disease, as compared to sex and age matched healthy non-smoking controls. In addition, we examined the relationship between the maximal exercise capacity and resting lung diffusion capacity both in smokers and in controls. We hypothesised that in apparently healthy heavy smokers, maximal exercise capacity could be related to the resting TLCO values.

Methods

Subjects

We enrolled 15 habitual smoking subjects, who were consecutively referred to our Smoking Cessation Outpatient Clinic from January to June 2007. Inclusion criteria were heavy smoking habit [≥ 20 pack/years; pack years = (number of cigarettes smoked per day x number of years smoked)/20], normal resting lung function, normal ECG findings, normal blood pressure values, absence of cardiovascular and pulmonary signs and symptoms and no regular exercise. Exclusion criteria included obesity (BMI ≥ 30 kg/m^2), anaemia or presence of musculoskeletal disorders which may limit exercise capacity. The smokers were asked to abstain from smoking for at least 2 h before reporting to the laboratory. Fifteen age and gender matched healthy non smoking sedentary subjects were recruited as a control group.

All the procedures and their risks were explained to the subjects, who gave their informed consent to the study. Approval of the local Human Ethics Committee was obtained.

Pulmonary function tests

Lung function was measured by a flow-sensing spirometer and a body pletismograph connected to a computer for data analysis (Vmax 22 and 6200, Sensor Medics, Yorba Linda, U.S.A.). Flow-volume and volume-time curves as

Received: 17 September 2007 / Accepted: 14 March 2008 / Published (online): 01 June 2008

well as maximal voluntary ventilation (MVV) manoeuvres were performed and forced expiratory volume in 1 sec (FEV_1), slow vital capacity (SVC), and MVV were recorded. Total lung capacity (TLC) and residual volume (RV) were also obtained. Carbon monoxide transfer capacity (TLCO) was measured by the single breath method using a mixture of carbon monoxide and methane. At least three measurements were made for each lung function variable to ensure reproducibility and the highest value was used in subsequent calculations. The flow-sensor was calibrated before each test using a three-liter syringe. Predicted values of lung volumes and expiratory flows as well as carbon monoxide transfer capacity were obtained from regression equations by Quanjer et al. and Cotes et al., respectively (Cotes et al., 1993; Quanjer et al., 1993).

Cardiopulmonary exercise test

A cardiopulmonary exercise test was performed according to a standardized procedure (ATS/ACCP Statement, 2003). After calibration of the oxygen and carbon dioxide sensors, the study subjects were asked to sit on an electromagnetically braked cycle ergometer (Corival PB, Lobe Bv, Groningen, The Netherlands) and the saddle was adjusted properly to avoid the maximal extension of the knee. After a 3-min rest period sitting on the ergometer, exercise began with a 3-min warm-up period at 0 watts, followed by a progressively increasing ramp protocol of 10-25 watts/min, according to anthropometric data of the subjects, in order to perform an exercise time lasting 8-12 min. All subjects had to maintain a pedaling frequency of 60 rpm indicated by a digital display placed on the monitor of the ergometer. Breath-by-breath VO_2 (mL/min), carbon dioxide production (VCO_2, mL/min) and minute ventilation (VE L/min) were collected during the test (Vmax 229, Sensor Medics, Yorba Linda, U.S.A.). Subjects were continuously monitored by a 12-lead electrocardiogram (Corina, GE Medical Systems IT inc, Milwaukee W, U.S.A.) and a pulse oximeter (Pulse Oximeter 8600, Nonin Medical Inc, MPLS, Mn U.S.A.). Blood pressure was measured at 2 min intervals. Test termination criteria consisted of symptoms such as unsustainable dyspnoea or leg fatigue, chest pain, ECG ST-segment depression, a drop in systolic blood pressure or $SaO_2 < 84\%$.

At the end of exercise, dyspnoea and leg fatigue were measured by a 0-100 visual analogue scale. The visual analogue scale (VAS) consisted of a horizontal ruler without any mark on the patient's side with the words "not at all breathless" or "not at all leg fatigue" and "extremely breathless" or "extremely leg fatigue" on the left and right end, respectively. Dyspnoea and leg fatigue perception ratings were expressed in mm from 0 to 100 and corresponded to the distance of the marker from the left end of the VAS. Dyspnoea and leg fatigue perception ratings were then divided by the maximal workload (watts) for analysis.

Statistical analysis

Data are reported as mean ± standard deviation (SD) and 95% confidence interval. Comparisons between variables were determined by the unpaired t test, Chi-square test and Mann-Whitney test, when appropriate. Relationships between variables were assessed by the Pearson's correlation coefficient (r) and linear regression analysis. A p value of less than 0.05 was taken as significant.

Table 1. Demographic and baseline pulmonary functional data of 15 smokers and 15 non smokers. Values are means (±SD) [range], apart from gender.

	Smokers	Non smokers
Age (yrs)	47 (7) [31–58]	43 (8) [29–59]
Gender (M/F)	10/5	10/5
BMI (kg/m^2)	25 (3) [22–30]	25 (4) [19–30]
FEV_1 (% pred)	103 (10) [87–122]	115 (10) [103–137]
FEV_1/SVC (%)	78 (6) [69–91)	80 (6) [71–91]
TLC (% pred)	105 (9) [93–119]	106 (11) [94–129]
TLCO (% pred)	79 (10) [65– 96]	101 (31) * [76–184]

* $p < 0.05$.

Results

The smokers recruited in the study had a tobacco history of 32 pack/years ± 9 [pack years = (number of cigarettes smoked per day x number of years smoked)/20]. Demographic and baseline pulmonary function data of the study population are shown in Table 1. At the time of the study all smokers (5 females, age range: 31-58 years) did not complain of any cardiopulmonary symptom and their physical examination did not reveal any pathological sign. All subjects completed the study without any complication.

Table 2. Maximal exercise capacity of 15 smokers and 15 non smokers. Values are means (±SD) [range].

	Smokers	Non smokers
Peak VO_2 ($L \cdot min^{-1}$)	1.99 (.54) [.95-2.83]	2.46 (.65) * [1.42-3.41]
Peak VO_2 ($mL \cdot kg^{-1} \cdot min^{-1}$)	26.9 (5.8) [19.1-38.0]	31.5 (5.1) * [21.1-40.5]
VO_2@AT ($L \cdot min^{-1}$)	1.36 (.30) [.70-1.78]	1.62 (.35) * [1.05-2.19]
VO_2@AT (%)	61 (8) [43-71]	69 (13) * [47-95]
Workload (Watts)	163 (41) [98-218]	198 (44) * [117-248]
VE/VCO_2	28 (3) [24-36]	26 (2) [21-31]
O_2 pulse ($mL \cdot bpm^{-1}$)	13.2 (3.6) [6.2-20.2]	15.7 (4.7) * [8.9-23.5]
VO_2/Watt ($mL \cdot min^{-1} \cdot W^{-1}$)	9.0 (1.2) [5.9-10.8]	10.0 (.8) * [8.4-11.3]
HR (bpm)	152 (15) [114-171]	160 (15) [132-181]
VE ($L \cdot min^{-1}$)	70 (19) [48-106]	83 (19) [54-131]
VE (% MVV)	51 (13) [35-81]	51 (9) [34-81]

* $p < 0.05$.

In smokers, mean values of FEV_1 (% of pred), FEV_1/SVC (%), TLC (% of pred) and TLCO (% of pred)

were 103% ± 10, 78% ± 6, 105% ± 9 and 79% ± 10, respectively. In controls, mean values of FEV_1 (% of pred), FEV_1/SVC (%), TLC (% of pred) and TLCO (% of pred) were 115% ± 10, 80% ± 6, 106% ± 11 and 101% ± 31, respectively. There was no difference with respect to functional parameters at rest between smokers and control subjects except for TLCO, which was significantly lower in smokers ($p < 0.05$). Electrocardiogram showed no alterations in both groups and blood pressure values were normal as well.

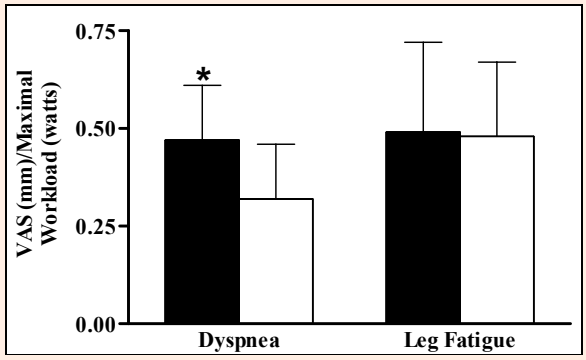

Figure 1. Mean and standard deviation values of dyspnoea and leg fatigue in 15 smokers (filled bar) and 15 non smoking (empty bar) healthy subjects. Dyspnoea and leg fatigue perception ratings were measured in mm by a 0 - 100 visual analogue scale (VAS) and divided by the maximal workload (watts) for analysis.
* $p < 0.05$ by means of Mann Whitney test.

Exercise capacity parameters are shown on Table 2. When compared to the control group, the smokers had lower maximal workload, lower maximal peak VO_2 and lower oxygen pulse values. VO_2 at anaerobic threshold and VO_2/watt ratio ($mL \cdot min^{-1} \cdot watt^{-1}$) values were also significantly lower in smokers than in non smokers. VE/VCO_2 slope and maximum HR values were not significantly different between smokers and healthy controls. Dyspnoea, but not leg fatigue values at peak VO_2 were significantly higher in smokers than in controls (Figure 1).

In smokers, but not in healthy controls TLCO (%) correlated with workload ($r = 0.637$, $p < 0.05$), peak VO_2 ($r = 0.546$, $p < 0.05$) and oxygen pulse at peak exercise ($r = 0.663$, $p < 0.01$) (Figure 2). Regression analysis showed that in smokers maximal workload (watts) = 2.61 (TLCO) – 44.43 ($r^2 = 0.39$), peak VO_2 ($L \cdot min^{-1}$) = 0.03 (TLCO) – 0.37 ($r^2 = 0.28$), and O_2 pulse ($mL \cdot bpm^{-1}$) = 0.25 (TLCO) – 6.34 ($r^2 = 0.43$).

Discussion

In the present study, we assessed the maximal exercise capacity in heavy smokers without any apparent cardiovascular or respiratory disease, as compared to healthy matched control subjects. In smokers, we found that resting pulmonary and cardiac function parameters were in the normal range and did not differ from those of the control group, except for lung diffusion capacity. In addition, when compared with the control group, smokers showed significantly lower maximal oxygen uptake, maximal workload, maximal oxygen pulse, oxygen uptake at anaerobic threshold and VO_2/watt ratio values and higher dyspnoea perception values. Lastly, in smokers, but not in healthy controls, maximal workload, maximal oxygen uptake and maximal oxygen pulse were correlated with lung diffusion capacity at rest.

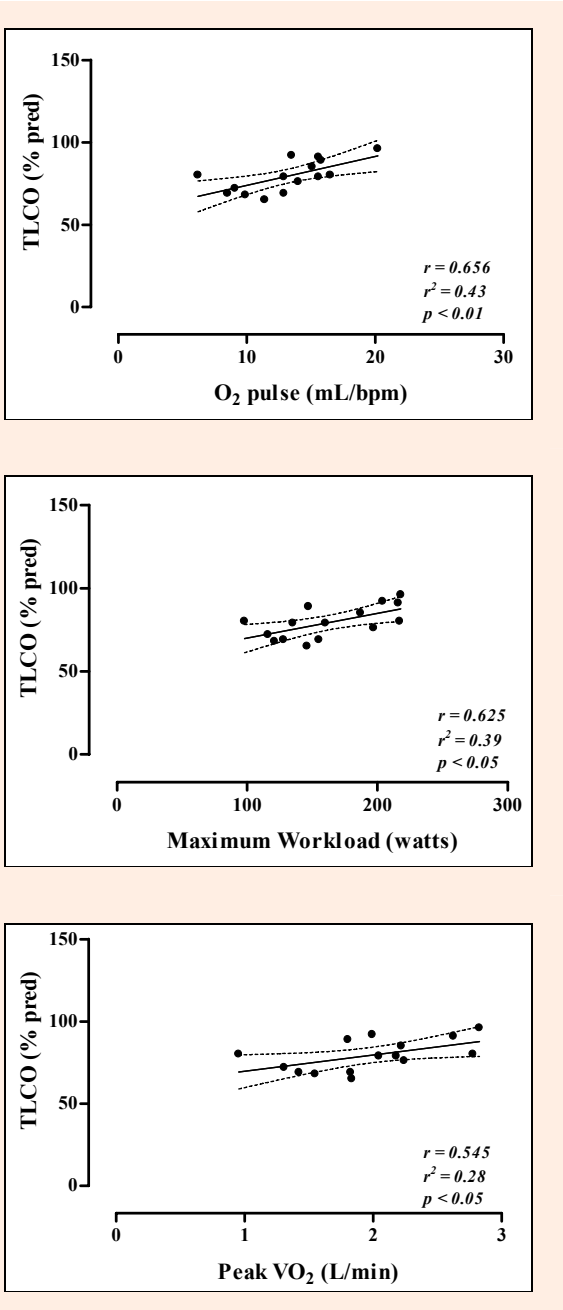

Figure 2. Relationships between TLCO and maximal oxygen pulse (*upper panel*), between TLCO and maximum workload (*middle panel*) and between TLCO and peak VO_2 (*lower panel*) in 15 smokers. Relationships were analyzed by using Pearson's correlation coefficient and linear regression analysis. The continuous line is the regression line; the interrupted lines represent the 95% confidence interval.

Previous reports have already investigated exercise capacity in smokers (Bernaards et al., 2003; Bolinder et al., 1997; Horvath et al., 1975; Kobayashi et al., 2004; Morton et al., 1985; Pirnay et al., 1971; Song et al., 1998;

Unverdorben et al., 2007). However, our study differs from the previous ones in selection criteria of smokers and type of exercise. In some previous studies, the authors recruited either only male patients (Bolinder et al., 1997; Unverdorben et al., 2007) or young people ranging in age between 16 to 36 years (Bernaards et al., 2003; Song et al., 1998), whereas in our study both male and female subjects with a wider age range were included, making our study subject sample more representative of the general population. Bolinder et al. (1997) and Song et al. (1998) studied well-trained subjects, in contrast, we selected only sedentary subjects. Differently from other reports in which pulmonary function tests at rest were not considered (Bernaards et al., 2003; Bolinder et al., 1997; Horvath et al., 1975; Kobayashi et al., 2004; Pirnay et al., 1971; Song et al., 1998; Unverdorben et al., 2007), we included only subjects with a documented normal resting lung function, since even a mild resting ventilatory defect could significantly impair maximal exercise capacity (Ofir et al., 2008; Vrijlandt et al., 2006). Finally, we used a cycle ergometer to assess maximal exercise capacity extending our knowledge on this kind of exercise, whereas in other studies the investigators used a treadmill to assess either maximal (Bernaards et al., 2003; Kobayashi et al., 2004; Morton et al., 1985; Pirnay et al., 1971) or sub-maximal exercise capacity (Kobayashi et al., 2004). It is of note that the quantification of external work during exercise can be more precisely calculated by using a cycle ergometer, rather than a treadmill (Cooper and Storer, 2001).

Previous reports showed that smokers had a reduced peak oxygen consumption (Bernaards et al., 2003; Bolinder et al., 1997; Horvath et al., 1975; Kobayashi et al., 2004; Pirnay et al., 1971; Unverdorben et al., 2007), and a reduced VO_2 at anaerobic threshold (Unverdorben et al., 2007), as well as a lower maximal oxygen pulse (Kobayashi et al., 2004). Consistent with these reports, we found that heavy smokers had lower values of maximal oxygen uptake, maximal workload, maximal oxygen pulse, oxygen uptake at anaerobic threshold and VO_2/watt ratio in comparison with healthy matched controls. Our findings extend the understanding of this matter, by showing that heavy smokers, even without any apparent cardiovascular or respiratory disease, may have a reduction in oxygen delivery and/or extraction.

Smoking can affect oxygen kinetics and uptake at different levels. The particulate substances released during tobacco burning increase airway resistance and decrease diffusion capacity for oxygen through the alveolar-capillary membrane (Nadel and Comroe, 1961). CO binds to haemoglobin 225 times more avidly than oxygen, and causes a left shift in the oxyhaemoglobin dissociation curve (decreased P_{50}). Thus, oxygen release to the tissues may be diminished by elevated CO. Importantly, in smokers lower VO_2 max values may be attributed not only to CO binding with haemoglobin, but also to a reduction in oxygen carrying capacity (Mc Donough and Moffatt, 1999). Moreover, increased mismatch of perfusion distribution to working muscles could result in the reduced O_2 extraction (Kobayashi et al., 2004). Smoking also increases the reliance upon glycolytic metabolism during exercise (Mc Donough and Moffatt, 1999). This phenomenon appears to be directly related to arterial O_2 content reduction observed in smokers (Mc Donough and Moffatt, 1999). Smokers could partially compensate for this reduction by increasing O_2 extraction at the muscle and/or by increasing glycolytic metabolism (Mc Donough and Moffatt, 1999). Lastly, cigarette smoking can damage the mitochondrial respiratory chain leading to increased intracellular oxidant levels (Cardellach et al., 2003; Smith et al., 1993). Taken together these factors contribute to dyspnoea and leg fatigue at a lower workload in smokers compared with non smokers.

In this study, we showed that smokers, even without cardiopulmonary disorders, had lower resting TLCO values than healthy controls. In a large general population sample, Viegi et al (1990) previously found significantly lower TLCO values in smokers than in non smokers. Interestingly, nicotine levels were found to be negatively related to TLCO in smokers (Clark et al., 1998). Watson et al (1993) also found that the reduction in TLCO due to tobacco smoking was reversible in subjects who gave up smoking.

The increased carboxyhaemoglobin seems to contribute to the reversible decrease in TLCO. The effect of carboxyhaemoglobin in reducing TLCO is greater than it would be predicted by the back CO capillary pressure effect alone. Frans et al (1975) suggested that as carboxyhaemoglobin increases, the effective haemoglobin mass decreases, thereby decreasing TLCO in what they call an "anemia" effect. They reported that TLCO decreased about 1.2% for each percent increase in carboxyhaemoglobin; about 60% of the decrease was due to the back pressure effect and 40% to the "anemia" effect. Moreover, lung diffusion capacity relies on capillary blood volume and membrane diffusivity. A previous study (Mahajan et al., 1991) showed that smokers, when compared to healthy non smoking subjects, had lower resting TLCO values, due to a significant decrease in capillary blood volume. In smoking subjects, local bronchoconstriction might induce regional hypoxia and pulmonary vasospasm, which in turn can contribute to the capillary blood volume reduction (Krumholz, 1966).

In the present study, we found that in smokers, TLCO values were directly related to and can predict maximal exercise capacity in terms of workload, oxygen uptake and oxygen pulse, and, accordingly, we provided the prediction equations. As far as we know, our study was the first study to report prediction equations for maximal exercise parameters in smokers based on TLCO. However, resting TLCO value does not explain all the variance of the outcome variables of exercise capacity. Other factors, such as tachycardia, increased pulse-pressure product and impaired oxygen delivery, might be involved in exercise capacity impairment in smokers (Hirsch et al., 1985).

Conclusion

In conclusion, our study confirms that heavy smokers have a reduction in maximal exercise capacity. In addition, we showed that a simple and inexpensive test, the lung diffusion capacity for CO, can predict oxygen pulse, workload and oxygen uptake at peak of exercise in smok-

ers without any cardiovascular and respiratory disease. Further studies are required to confirm our results in larger populations, including one with a milder history of smoking.

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Key points

- Chronic exposure to tobacco smoking may damage lung and heart function.
- Smokers present lower diffusion capacity and maximal exercise capacity.
- In smokers maximal exercise capacity can be predicted by resting diffusion lung capacity.

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