

ORIGINAL ARTICLE

## Lung function profiles and aerobic capacity of adult cigarette and hookah smokers after 12 weeks intermittent training

Abdessalem Koubaa<sup>1,2†</sup>, Moez Triki<sup>3\*†</sup>, Hajer Trabelsi<sup>2†</sup>, Liwa Masmoudi<sup>2†</sup>, Khaled N. Zeghal<sup>1†</sup>, Zouhair Sahnoun<sup>1†</sup> and Ahmed Hakim<sup>1†</sup>

<sup>1</sup>Laboratory of Pharmacology, Faculty of Medicine of Sfax, University of Sfax, Sfax, Tunisia; <sup>2</sup>Research Unit (EM2S), Higher Institute of Sport and Physical Education of Sfax, University of Sfax, Sfax, Tunisia; <sup>3</sup>Laboratory of Cardio-Circulatory, Respiratory, and Hormonal Adaptations to Muscular Exercise, Faculty of Medicine Ibn El Jazzar, University of Sousse, Sousse, Tunisia

**Introduction:** Pulmonary function is compromised in most smokers. Yet it is unknown whether exercise training improves pulmonary function and aerobic capacity in cigarette and hookah smokers and whether these smokers respond in a similar way as do non-smokers.

**Aim:** To evaluate the effects of an interval exercise training program on pulmonary function and aerobic capacity in cigarette and hookah smokers.

**Methods:** Twelve cigarette smokers, 10 hookah smokers, and 11 non-smokers participated in our exercise program. All subjects performed 30 min of interval exercise (2 min of work followed by 1 min of rest) three times a week for 12 weeks at an intensity estimated at 70% of the subject's maximum aerobic capacity ( $\dot{V}O_2\max$ ). Pulmonary function was measured using spirometry, and maximum aerobic capacity was assessed by maximal exercise testing on a treadmill before the beginning and at the end of the exercise training program.

**Results:** As expected, prior to the exercise intervention, the cigarette and hookah smokers had significantly lower pulmonary function than the non-smokers. The 12-week exercise training program did not significantly affect lung function as assessed by spirometry in the non-smoker group. However, it significantly increased both forced expiratory volume in 1 second and peak expiratory flow (PEF) in the cigarette smoker group, and PEF in the hookah smoker group. Our training program had its most notable impact on the cardiopulmonary system of smokers. In the non-smoker and cigarette smoker groups, the training program significantly improved  $\dot{V}O_2\max$  (4.4 and 4.7%, respectively),  $v \dot{V}O_2\max$  (6.7 and 5.6%, respectively), and the recovery index (7.9 and 10.5%, respectively).

**Conclusions:** After 12 weeks of interval training program, the increase of  $\dot{V}O_2\max$  and the decrease of recovery index and resting heart rate in the smoking subjects indicated better exercise tolerance. Although the intermittent training program altered pulmonary function only partially, both aerobic capacity and life quality were improved. Intermittent training should be advised in the clinical setting for subjects with adverse health behaviors.

Keywords: cigarette smokers; hookah smokers; pulmonary function; aerobic capacity; interval training

\*Correspondence to: Moez Triki, Clinical Laboratory of Physiology, Physiology and Functional Testing Department, 98/UR08-67, Medical School of Sousse, University of Sousse, Avenue Ibn ElJazzar, Sousse 4000, Tunisia, Email: trikimoez27@yahoo.fr

Received: 19 November 2014; Revised: 17 January 2015; Accepted: 19 January 2015; Published: 17 February 2015

Examining the relationship of smoking habits, respiratory symptoms and lung function to mortality in men from the general population aged 50–60 years, Olofson et al. (1) demonstrated that the mortality rate was significantly related to age, smoking habits, and dyspnea. Furthermore, these authors showed that impaired

lung function is an important factor to be considered in the assessment of mortality risk, besides smoking and dyspnea.

Smoking is one of the most important risk factors for future cardiovascular morbidity and a major cause of cardiovascular disease mortality (2). It has significant

<sup>†</sup>These authors contributed equally to this work.

detrimental effects on both the structure and function of the lung; it is the single most important risk factor for the development of chronic obstructive pulmonary disease (COPD) (3). Studies have documented lower forced expiratory volume in 1 second ( $FEV_1$ ), accelerated loss of ventilatory function, and increased respiratory symptoms among smokers compared with non-smokers (4). Data from other studies have consistently shown increased mortality from COPD, and pneumonia among cigarette smokers compared with non-smokers (5). Smokers had double the mortality rate of non-smokers (1). According to Higgins et al., the prevalence of chronic bronchitis was higher and mean  $FEV_1$  was lower in cigarette smokers than non-smokers, and heavy smokers were affected more than light smokers (6). Likewise, the results reported by Mohammad et al. showed a higher proportion of chronic bronchitis and a quasi-permanent alteration in maximum mid-expiratory flow (MMEF 25–75%) in narguileh smokers compared with cigarette smokers. These authors added that  $FEV_1$  was more altered in cigarette smokers than in narguileh smokers (7).

Physical training can enhance health in many ways. It has been shown to improve cardiovascular performance (8, 9), to prevent premature death (10), and to promote longevity (11, 12). In previous studies, regular exercise training has been related to better pulmonary function (13, 14). Also other studies report a positive association between physical activity and physical fitness and lung capacity (15, 16), while others do not (17).

The  $\dot{V}O_2$ max components, heart rate (HR) and systolic blood pressure (SBP), are important indicators of cardiovascular health and fitness. Lower HR at rest and during exercise is associated with improved physical fitness (18–20). Higher values of HR and SBP at rest, as well as their increased variability and response during exercise, are important risk factors and prognostic indicators of cardiovascular disease mortality (21, 22). Smokers usually exhibit elevated HR and reduced exercise capacity and, thus, lower overall cardiovascular fitness (23, 24).

The benefit of training programs appears to be important in the general healthy population for increasing cardiorespiratory performance (25). The practice of physical activity is associated with an increase in  $\dot{V}O_2$ max regardless of age (26, 27). Moreover, several studies examining the effects of physical training on BP, HR, and  $\dot{V}O_2$ max in healthy adults have yielded convincing results (12, 28, 29).

Most studies on the effects of physical activity on respiratory and cardiovascular functions focused on special populations, such as athletes or patients with COPD (30, 31). However, it is not known whether intermittent physical training can retard the deterioration of pulmonary function and improve cardiorespiratory fitness in smokers unwilling or unable to quit.

Investigating the relationship between physical activity and cardiovascular and respiratory functions of smokers

will aid in understanding the mechanisms of how physical activity improves quality of life. The major purpose of this study was to examine the effects of 12 weeks of intermittent training on pulmonary function among sedentary cigarette smokers versus hookah smokers. We also evaluated how physical activity affects exercise tolerance of the participants.

## Methods

### Participants

A total of 35 sedentary smokers and non-smokers with the following average characteristics  $\pm$  standard deviation (SD) participated in this study: age,  $44.7 \pm 4.5$  years; weight,  $71.3 \pm 2.7$  kg; height,  $174.3 \pm 2.3$  cm.

After receiving a complete verbal description of protocol, risks and benefits of the study, each subject signed a written consent. This study was approved by the Faculty of Medicine Ethical Research Committee, University of Sfax, Tunisia.

We excluded people who had any self-reported physician-diagnosed chronic disease (arthritis, diabetes, hypertension, cancer, heart attack, chronic cough, bronchitis, abnormal exercise electrocardiogram, or  $FEV_1/FVC\% < 70\%$ ) (32, 33) at the visit before protocol.

Cigarette and hookah smokers unable or unwilling to quit were recruited according to the number of cigarettes and hookahs per day and how long they have been smoking. We considered cigarette smokers all subjects with consumption greater or equal to 10 pack-years (PY) and an average score of tobacco dependence of  $7.33 \pm 1.67$ , measured by the Fagerström nicotine dependence test (34). In the absence of specific international codification, we quantified hookah consumption, as in the study of Kiter et al. (35), in hookah-years (HY) and kilogram of cumulative tobacco. The tobacco used in a single hookah session weighs between 10 and 25 g (36). Regular hookah smokers are those having tobacco consumption greater or equal to 5 HY (37).

Participants were divided into three groups: a cigarette smoker group (CS) ( $n = 11$ ), a hookah smoker group (HS) ( $n = 12$ ), and a non-smoker group (NS) ( $n = 12$ ).

### Pulmonary function assessment

Spirometry assessments were undertaken in accordance with standards described by the American Thoracic Society (38). Standard procedure requires forced vital capacity (FVC) and  $FEV_1$  to be measured from a series of at least three forced expiratory curves (39). Consequently, this study required participants to perform three correct manoeuvres. Participants completed the spirometry assessment seated, using a portable spirometer (MIR Spirobank G USB Spirometer, Rome, Italy), with a nose clip attached. Pulmonary function variables included FVC,  $FEV_1$ , and  $FEV_1/FVC$  ratio. Results were expressed as percentages

of the predicted value to allow comparison of results across participants.

### Aerobic capacity assessment

Exercise tolerance, achieved during a maximal treadmill exercise test, is a leading indicator of circulatory system capacity as it is strongly related to maximum oxygen uptake ( $\dot{V}O_2\text{max}$ ) (40). All the participants underwent a progressive exercise test performed on a treadmill (Pulmonary Function Equipment, COSMED, Rome, Italy) in the research unit of the Higher Institute of Sport and Physical Education of Sfax. The test began with a warm-up at a speed of 6 km/h for 5 min, after which the speed was increased incrementally by 1 km/h every 2 min until exhaustion. HR was monitored throughout the test using Polar Electro Oy (Kempele, Finland) and was recorded at the conclusion of every 2-min stage. Verbal encouragement was provided throughout the test to ensure that the maximal effort was achieved. During the exercise test, oxygen consumption was continually recorded using an oxygen analyzer (Fitmate, version 1.2 PRO COSMED). Oxygen consumption ( $\dot{V}O_2$ ) during the exercise test was measured in real time by means of a dynamic mixing chamber, and data were recorded every 2 min. At the end of the test, a detailed report was printed.

### Exercise program

All training sessions were completed at the Higher Institute of Sport and Physical Education of Sfax under supervision of qualified specialist trainers. Subjects in the three groups underwent an intermittent training program that consisted of three sessions per week for approximately 30 min per session, during a 12-week period. The intensity of the exercise was controlled by time and distance travelled. All warm-ups before training were between 50 and 60% of maximum HR for a period of about 10 min.

The intermittent training consisted of 30 min of work/rest. Participants were instructed to run for 2 min at an intensity workload that equated to 70% of their individual  $\dot{V}O_2\text{max}$ , interspersed with recovery periods of 1 min.

This sequence was repeated 10 times during the 30-min period. Exercise intensity was gradually increased every 2 weeks over the course of the training period, based on the ability of each participant.

### Statistical analysis

All statistical tests were processed using STATISTICA Software (StatSoft, France). The data are expressed as mean  $\pm$  SD. Analysis of variance (ANOVA) was carried out to compare the responses of the different groups before and after the training program. Least significant difference (LSD) post-hoc analysis was used to identify significant group differences that were indicated by ANOVA. A probability level of 0.05 was selected as the criterion for statistical significance.

### Results

Before training, we did not observe any significant difference in basal cardiorespiratory values between the non-smoker group and the smoker groups. The ( $\Delta$ ) results of the maximal exercise tests after training are presented in Table 1. At the end of the training program, the participants showed similar improvements. However, no significant differences were found among the three groups. After the training program, a small but significant decrease ( $p < 0.05$ ) in the resting HR was observed in each of the three groups ( $3 \pm 4$ ,  $3 \pm 3$ , and  $2 \pm 4$  for NS, CS, and HS, respectively). Similarly, there was a decrease of  $2 \pm 3$  (mm Hg) in systolic BP for each of the three groups. The decrease in the diastolic BP was significant only in the CS group.

At maximal exercise, there was an increase of 6.7 and 5.6% in  $\dot{V}O_2\text{max}$ , an increase of 4.4 and 4.7% in  $\dot{V}O_2\text{max}$ , and an increase of 7.9 and 10.5% in the Recovery Index for NS and CS groups, respectively. However, for none of these parameters was there a statistically significant change for the HS group (Fig. 1).

We observed significant differences in baseline spirometric values when the three groups were compared (Table 2). Smoking cigarettes or hookah resulted in lower

**Table 1.** Improvement rate ( $\Delta$ ) of cardio-respiratory values in subjects of the three groups (NS, CS, and HS)

Parameters	Mean $\pm$ SD			Pre vs. Post		
	NS	CS	HS	NS	CS	HS
Resting HR (bpm)	$-3 \pm 4$	$-3 \pm 3$	$-2 \pm 4$	†	†	†
Systolic BP (mm Hg)	$-2 \pm 3$	$-2 \pm 3$	$-2 \pm 2$	†	††	†
Diastolic BP (mm Hg)	$-2 \pm 4$	$-3 \pm 5$	$-2 \pm 3$	ns	†	ns
$v \dot{V}O_2\text{max}$ (km/h)	$0.7 \pm 1$	$0.6 \pm 0.9$	$0.4 \pm 0.6$	††	†	ns
$\dot{V}O_2\text{max}$ (ml·min·kg <sup>-1</sup> )	$1.7 \pm 3.3$	$1.8 \pm 2.6$	$1.5 \pm 2.1$	†	†	ns
Recovery index	$1.2 \pm 1.3$	$1.6 \pm 2.5$	$0.9 \pm 1.5$	†	††	ns

NS: non-smokers; CS: cigarettes smokers; HS: hookah smokers;  $\dot{V}O_2\text{max}$ : maximum oxygen uptake;  $v \dot{V}O_2\text{max}$ : velocity at maximum oxygen uptake; HR: heart rate; bpm: beats per minute; BP: blood pressure.

†, ††: significant difference Pre- vs. Post program at  $p < 0.05$ ,  $p < 0.01$ , respectively.

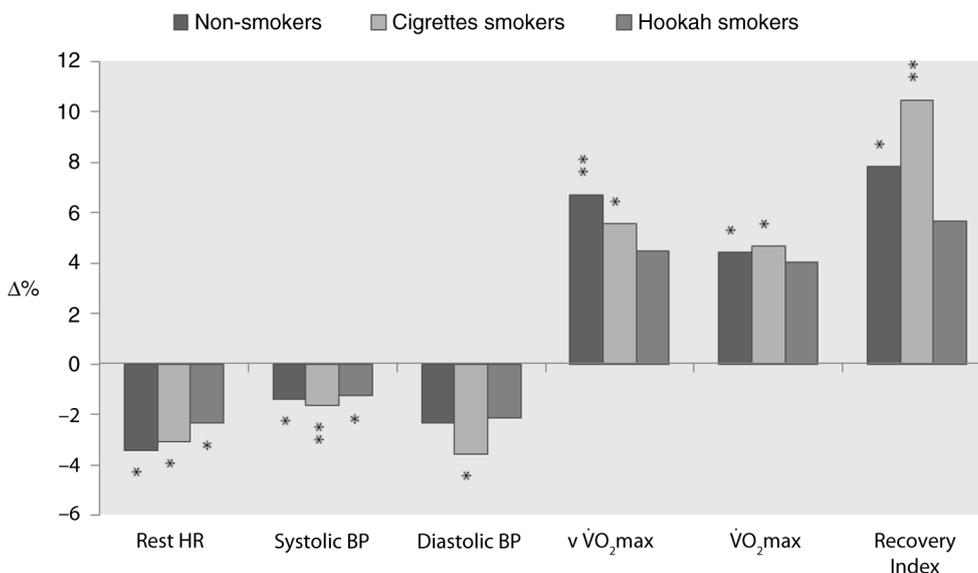


Fig. 1. Intra-group changes in percentage (Δ %) of cardio-respiratory parameters of the three groups.

values of FEV<sub>1</sub> and peak expiratory flow (PEF) ( $p < 0.001$ ) compared with the non-smokers, but smoking did not influence the FEV<sub>1</sub>/FVC ratios ( $p = 0.362$ ). By contrast, forced expiratory flow (FEF<sub>50%</sub>) and FEF<sub>25–75%</sub> values were lower in the smokers compared to the non-smokers ( $p < 0.001$  and  $p = 0.004$ , respectively).

Changes (Δ) in the spirometric values are presented in Table 3. After training, most variables showed a small, positive Δ that did not reach statistical significance. However, significant increases in PEF rate of 3.8 and 3.4% were detected for both CS and HS groups ( $p < 0.01$  and  $p < 0.05$ , respectively), while a significant FEV<sub>1</sub> change was observed in the CS group only.

As shown in Fig. 2, a significant increase in FEV<sub>1</sub> (6.2%) was observed in the SC group ( $p < 0.05$ ). No significant FEV<sub>1</sub> changes were observed for the other two groups.

Furthermore, the training program did not result in any significant changes in any of the three groups with respect to FVC and FEF.

### Discussion

Physical inactivity and low cardiorespiratory fitness are recognized as important causes of morbidity and mortality (41–43). The data presented in this study showed the relation between physical activity in the form of intermittent exercise and cardiorespiratory function for male smokers and non-smokers. In a longitudinal study of Norwegian men (44), the authors concluded that decline in physical fitness and lung function among healthy middle-aged men was considerably greater among smokers than non-smokers. Smoking is the most important modifiable risk factor for decreased respiratory function (16, 45).

This study was designed to measure the effects of an intermittent training program on lung function and aerobic capacity in adult smokers. Initially, the mean spirometric values of smokers were lower than those of non-smokers. After 12 weeks of training, the mean spirometric values at rest were slightly higher, without a significant difference, except for FEV<sub>1</sub> and PEF for the CS

Table 2. Spirometric data of the three groups before the training protocol

Parameters	Mean ± SD			ANOVA
	NS	CS	HS	
FVC (%)	100.5 ± 5.8	95.5 ± 4.5	93.1 ± 7.9**	$p = 0.018$
FEV <sub>1</sub> (%)	103.3 ± 5	94.1 ± 6.5***	95.3 ± 6.6**	$p < 0.001$
PEF (%)	110.3 ± 5.2	102.5 ± 6.7**	101 ± 4.3***	$p < 0.001$
Tiffeneau index (%)	1.03 ± 0.07	0.99 ± 0.06	1.03 ± 0.11	$p = 0.362$
FEF <sub>25–75%</sub> (%)	103.3 ± 10.1	94.9 ± 5**	93.9 ± 4.4**	$p = 0.004$
FEF <sub>50%</sub> (%)	99.8 ± 4.5	94.7 ± 2.6**	93 ± 3.8***	$p < 0.001$

NS: non-smokers; CS: cigarettes smokers; HS: hookah smokers; FVC: forced vital capacity; FEV<sub>1</sub>: forced expiratory volume in 1 second; PEF: peak expiratory flow; FEF<sub>50%</sub>: forced expiratory flow at 50% of FVC; FEF<sub>25–75%</sub>: forced expiratory flow at 25–75% of FVC.

\*\* , \*\*\*: significant difference compared with non-smokers at  $p < 0.01$ ,  $p < 0.001$ , respectively.

**Table 3.** Improvement rate ( $\Delta$ ) of respiratory parameters in subjects of the three groups (NS, CS, and HS)

Parameters	Mean $\pm$ SD			Pre vs. Post		
	NS	CS	HS	NS	CS	HS
FVC (%)	1.9 $\pm$ 8.8	4.4 $\pm$ 9.6	3.6 $\pm$ 9	ns	ns	ns
FEV <sub>1</sub> (%)	0.5 $\pm$ 7.8	5.8 $\pm$ 9.2	4 $\pm$ 6.4	ns	†	ns
PEF (%)	-1.4 $\pm$ 5.8	3.9 $\pm$ 3.8**	3.4 $\pm$ 3.9*	ns	††	†
Tiffeneau index (%)	-0.01 $\pm$ 0.13	0.02 $\pm$ 0.10	0.001 $\pm$ 0.08	ns	ns	ns
FEF <sub>25-75%</sub> (%)	2.2 $\pm$ 4.4	1.8 $\pm$ 6.6	2.3 $\pm$ 5.8	ns	ns	ns
FEF <sub>50%</sub> (%)	0.5 $\pm$ 4.1	0.8 $\pm$ 3.5	0.9 $\pm$ 5.2	ns	ns	ns

NS: non-smokers; CS: cigarettes smokers; HS: hookah smokers; FVC: forced vital capacity; FEV<sub>1</sub>: forced expiratory volume in 1 second; PEF: peak expiratory flow; FEF<sub>50%</sub>: forced expiratory flow at 50% of FVC; FEF<sub>25-75%</sub>: forced expiratory flow at 25-75% of FVC.

\*, \*\*: significant difference compared with non-smokers at  $p < 0.05$ ,  $p < 0.01$ , respectively; †, ††: significant difference pre- vs. postprogram at  $p < 0.05$ ,  $p < 0.01$ , respectively.

and HS groups. Pulmonary functions parameters were improved to a greater extent in most participants of the smoker groups than the non-smoker group. The most significant improvements occurred in FEV1 and PEF in the two smoker groups. These results are in line with those of Miles (46) and Gass et al. (47). The improvement in PEF was significantly slower among hookah smokers compared to cigarette smokers. In contrast, we did not observe any effect of the 12 weeks of training on FVC or FEF. In the light of these results, we believe that training programs of this duration have only minor effects on spirometric variables.

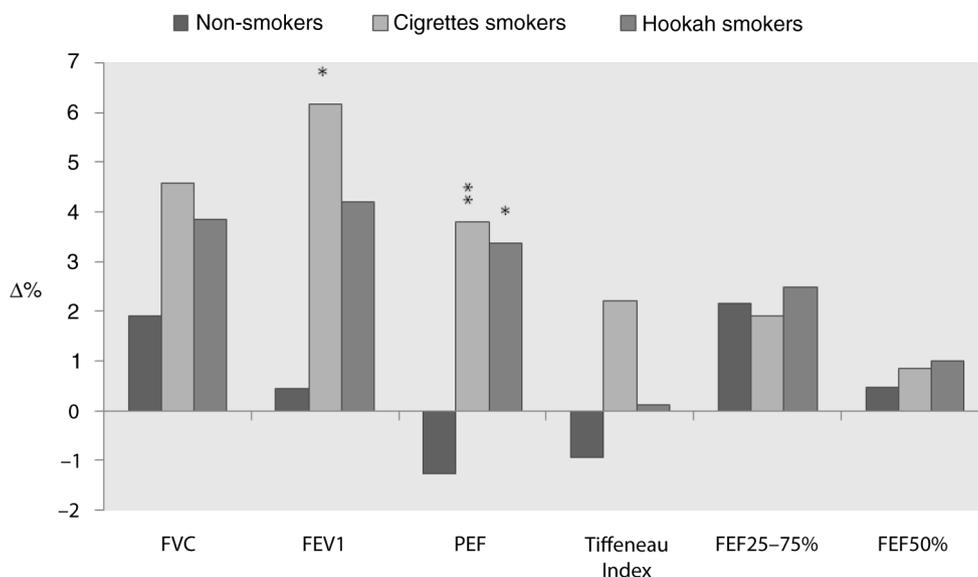
Our study shows that interval training of more than three times per week reduces the decline in pulmonary function. The beneficial effect of physical activity on pulmonary function was independent of smoking and was almost similar for the two smoking methods.

It has also been claimed that physical activity is positively associated with pulmonary function (13, 46, 48)

and can enhance inspiratory muscle endurance (49). Interestingly, using a short-term training protocol, Biersteker et al. (17) did not observe any effect of physical training on physical fitness and lung vital capacity in young adults.

In the present study, the non-smokers group showed no significant change in lung function as a result of the training program. By contrast, the two groups of smokers saw their respiratory ability improve significantly due to training, a result that is consistent with several previous studies (50, 51). Therefore, this study provides objective data supporting the use of a program of intermittent exercise for strengthening the pulmonary functions of smokers.

A number of benefits of intermittent training have been previously demonstrated. This training program slows the decline in pulmonary function and alleviates symptoms and exacerbation of pulmonary disease, resulting in improvement of quality of life. Our study suggests that



**Fig. 2.** Intra-group changes in percentage ( $\Delta$  %) of spirometric values of the three groups.

interval exercise training may be useful in slowing the progression of pulmonary disease due to cigarette smoking.

It is generally accepted that people with higher levels of physical activity tend to have higher levels of fitness and that physical activity can improve cardiorespiratory fitness (52). In our study, intermittent exercises were strongly associated with better exercise tolerance assessed with maximal treadmill test, a finding consistent with other studies (42, 53).

In accordance with the literature (54–57), our training program produced a significant increase in  $\dot{V}O_{2\max}$ ,  $\dot{V}O_{2\max}$ , and RI for the two smoker groups. Those results are somewhat similar to those reported by Leon et al. (18), who used the same kind of treadmill test to assess physical capacity of participants. The changes in resting HR, systolic BP, and diastolic BP were also significant. These results demonstrate the efficacy of our intermittent training program in smokers and non-smokers.

In summary, the interval training program used in the present study significantly improved exercise performance and symptoms for both groups of smokers. These conclusions are consistent with the results of several published studies, which indicated that exercise is an important component of pulmonary rehabilitation and may be associated with both physiological and psychological benefits (19).

Although smoking cessation is certainly an important way to reduce the decline in pulmonary function in smokers, this training method appears to be beneficial in both smokers and non-smokers and can be performed by all individuals. Interval training was related to a slower decline in pulmonary function due to smoking.

Because the exercise intensity was adapted to participant capacity, our intermittent training program is a suitable method for improving ventilatory efficiency. Our findings are potentially important from both the public health and the clinical points of view.

Because passive smoking causes lung function decline, and based on previous findings of Mohammad et al. (58), we think that future research should include a group of passive smokers. Other studies using other training methods will be needed to advance our conclusions. We believe that the continuous exercise training programs could improve the aerobic capacity and modify more favorably the lung function of smokers.

## Conclusions

Our intermittent training program improved the aerobic capacity and modified the cardiorespiratory parameters of smokers in a way that approximates those of non-smokers. After 12 weeks of training,  $\dot{V}O_{2\max}$  and recovery index were improved, and there was a significant decrease in resting HR. These results demonstrate the beneficial effects of intermittent training on the cardiopulmonary system and quality of life even in the smoking population.

## Limitations of the study

The lack of a control group may be considered a limitation of the present study (smokers following their usual daily activity during the protocol period). Also, the relatively small sample size could have limited our ability to detect group differences in the chosen parameters. This is indeed a limitation of this work, and should be considered relative to our findings.

## Acknowledgements

We gratefully acknowledge the contributions of Dr. A. Hakim and Dr. Z. Sahnoun. We also wish to thank the participants for their participation and commitment throughout the study.

## Conflict of interest and funding

The authors declare that they have no conflicts of interest concerning this research.

## References

- Olofson J, Skoogh BE, Bake B, Svardsudd K. Mortality related to smoking habits, respiratory symptoms and lung function. *Eur J Respir Dis.* 1987; 71: 69–76.
- Ezzati M, Henley SJ, Thun MJ, Lopez AD. Role of smoking in global and regional cardiovascular mortality. *Circulation.* 2005; 112: 489–97.
- Feenstra TL, van Genugten ML, Hoogenveen RT, Wouters EF, Rutten-van Molken MP. The impact of aging and smoking on the future burden of chronic obstructive pulmonary disease: a model analysis in the Netherlands. *Am J Respir Crit Care Med.* 2001; 164: 590–6.
- Islam SS, Schottenfeld D. Declining FEV<sub>1</sub> and chronic productive cough in cigarette smokers: a 25-year prospective study of lung cancer incidence in Tecumseh, Michigan. *Cancer Epidemiol Biomarkers Prev.* 1994; 3: 289–98.
- Sherman CB. The health consequences of cigarette smoking. *Pulmonary diseases. Med Clin North Am.* 1992; 76: 355–75.
- Higgins MW, Keller JB, Metzner HL. Smoking, socioeconomic status, and chronic respiratory disease. *Am Rev Respir Dis.* 1977; 116: 403–10.
- Mohammad Y, Kakah M, Mohammad Y. Chronic respiratory effect of narguileh smoking compared with cigarette smoking in women from the East Mediterranean region. *Int J Chron Obstruct Pulmon Dis.* 2008; 3: 405–14.
- Spina RJ, Turner MJ, Ehsani AA. Exercise training enhances cardiac function in response to afterload stress in older men. *Am J Physiol.* 1997; 272: H995–H1000.
- Schulman SP, Fleg JL, Goldberg AP, Busby-Whitehead J, Hagberg JM, O'Connor FC, et al. Continuum of cardiovascular performance across a broad range of fitness levels in healthy older men. *Circulation.* 1996; 94: 359–67.
- Pekkanen J, Marti B, Nissinen A, Tuomilehto J, Punsar S, Karvonen M. Reduction of premature mortality by high physical activity: a 20-year follow-up of middle-aged Finnish men. *Lancet.* 1987; 27: 1473–7.
- Paffenbarger RS Jr, Hyde RT, Wing AL, Hsieh C-C. Physical activity, all-cause mortality, and longevity of college alumni. *N Engl J Med.* 1986; 314: 605–13.
- Blair SN, Kohl HW, Barlow CE, Paffenbarger RS, Gibbons LW, Macera CA. Changes in physical fitness and all-cause mortality.

- A prospective study of healthy and unhealthy men. *JAMA*. 1995; 273: 1093–8.
13. Burchfiel CM, Enright PL, Sharp DS, Chyou P-H, Rodriguez BL, Curb JD. Factors associated with variations in pulmonary function among elderly Japanese–American men. *Chest*. 1997; 112: 87–97.
  14. Jonhson BD, Reddan WG, Pegelow DF, Seow KC, Dempsey JA. Flow limitation and regulation of functional residual capacity during exercise in a physically active aging population. *Am Rev Respir Dis*. 1991; 143: 960–7.
  15. Doherty M, Dimitriou L. Comparison of lung volume in Greek swimmers, land based athletes, and sedentary controls using allometric scaling. *Br J Sports Med*. 1997; 31: 337–41.
  16. Twisk JW, Staal BJ, Brinkman MN, Kemper HC, van Mechelen W. Tracking of lung function parameters and the longitudinal relationship with lifestyle. *Eur Respir J*. 1998; 12: 627–34.
  17. Biersteker MW, Biersteker PA. Vital capacity in trained and untrained healthy young adults in the Netherlands. *Eur J Appl Physiol*. 1985; 54: 46–53.
  18. Leon AS, Jacobs DR, DeBacker G, Taylor HL. Relationship of physical characteristics and life habits to treadmill exercise capacity. *Am J Epidemiol*. 1981; 113: 653–60.
  19. Cheng YJ, Macera CA, Addy CL, Wieland D, Blair SN. Effects of physical activity on exercise tests and respiratory function. *Br J Sports Med*. 2003; 37: 521–8.
  20. Lauer MS, Pashkow FJ, Larson MG, Levy D. Association of cigarette smoking with chronotropic incompetence and prognosis in the Framingham Heart Study. *Circulation*. 1997; 96: 897–903.
  21. Tzemos N, Lim PO, MacDonald TM. Is exercise blood pressure a marker of vascular endothelial function? *Q J Med*. 2002; 95: 423–9.
  22. Benowitz NL, Gourlay SG. Cardiovascular toxicity of nicotine: implications for nicotine replacement therapy. *J Am Col Cardiol*. 1997; 29: 1422–31.
  23. McDonough P, Moffatt RJ. Smoking-induced elevations in blood carboxyhaemoglobin levels. *Sports Med*. 1999; 27: 275–83.
  24. Maiorana A, O'Driscoll G, Dembo L, Goodman C, Taylor R, Green D. Exercise training, vascular function, and functional capacity in middle-aged subjects. *Med Sci Sports Exerc*. 2001; 33: 2022–8.
  25. Malbut KE, Dinan S, Young A. Aerobic training in the 'oldest old': the effect of 24 weeks of training. *Age Ageing*. 2002; 31: 255–60.
  26. Evans EM, Racette SB, Peterson LR, Villareal DT, Greiwe JS, Holloszy JO. Aerobic power and insulin action improve in response to endurance exercise training in healthy 77–87 yr olds. *J Appl Physiol*. 2005; 98: 40–5.
  27. Kodama S, Saito K, Tanaka S, Maki M, Yachi Y, Asumi M, et al. Cardiorespiratory fitness as a quantitative predictor of all-cause mortality and cardiovascular events in healthy men and women: a meta-analysis. *JAMA*. 2009; 301: 2024–35.
  28. Tjep BL. Disease management of COPD with pulmonary rehabilitation. *Chest*. 1997; 112: 1630–56.
  29. Lyndon JO, Jacob B, Katzel LI, Andrew PG. Exercise training and cardiovascular health with aging. *Clin Geriatr*. 2005; 13: 40–6.
  30. Murphy M, Nevill A, Neville C, Biddle S, Hardman A. Accumulating brisk walking for fitness, cardiovascular risk, and psychological health. *Med Sci Sports Exerc*. 2002; 34: 1468–74.
  31. Bolinder G, Faire U. Ambulatory 24 h blood pressure monitoring in healthy, middle aged smokeless tobacco users, smokers, and nontobacco users. *Am J Hypertens*. 1998; 11: 1153–63.
  32. Lenfant C, Khaltaev N, (Eds.). *Global initiative for Chronic Obstructive Lung Disease, Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease NHLBI/WHO workshop: 1998*. Available from: <http://www.goldcopd.org/uploads/users/files/GOLDWkshp05Clean.pdf> [cited 15 March 2014].
  33. *Chronic obstructive pulmonary disease: Management of chronic obstructive pulmonary disease in adults in primary and secondary care (partial update)*; 2010. Available from: [NICE.org.uk](http://NICE.org.uk) [cited 25 January 2014].
  34. Heatherton TF, Kozlowski LT, Frecker RC, Fagerström KO. The Fagerström Test for Nicotine Dependence: a revision of the Fagerström Tolerance Questionnaire. *Br J Addict*. 1991; 86: 1119–27.
  35. Kiter G, Ucan ES, Ceylan E, Kilinc O. Water-pipe smoking and pulmonary functions. *Respir Med*. 2000; 94: 891–4.
  36. Knishkowsky B, Amitai Y. Water-pipe (narghile) smoking: an emerging health risk behavior. *Pediatrics*. 2005; 116: E113–19.
  37. Ben Saad H. The narghile and its effects on health. Part I: The narghile, general description and properties. *Rev Pneumol Clin*. 2009; 65: 369–75.
  38. American Thoracic Society. Standardization of spirometry, 1994 update. *Am J Respir Crit Care Med*. 1995; 152: 1107–36.
  39. Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, Coates A, et al. Standardisation of spirometry. *Eur Respir J*. 2005; 26: 319–38.
  40. Bruce RA, Kusumi F, Hosmer D. Maximal oxygen uptake and nomographic assessment of functional aerobic impairment in cardiovascular disease. *Am Heart J*. 1973; 85: 546–62.
  41. Schunemann HJ, Dorn J, Grant BJ, Winkelstein W Jr, Trevisan M. Pulmonary function is a long-term predictor of mortality in the general population: 29-year follow-up of the Buffalo Health Study. *Chest*. 2000; 118: 656–64.
  42. Dunn AL, Marcus BH, Kampert JB, Garcia ME, Kohl HW 3rd, Blair SN. Comparison of lifestyle and structured interventions to increase physical activity and cardiorespiratory fitness: a randomized trial. *JAMA*. 1999; 281: 327–34.
  43. Neas LM, Schwartz J. Pulmonary function levels as predictors of mortality in a national sample of US adults. *Am J Epidemiol*. 1998; 147: 1011–18.
  44. Sandvik L, Erikssen G, Thaulow E. Long term effects of smoking on physical fitness and lung function: a longitudinal study of 1393 middle aged Norwegian men for seven years. *BMJ*. 1995; 311: 715–18.
  45. Kerstjens HA, Rijcken B, Schouten JP, Postma DS. Decline of FEV<sub>1</sub> by age and smoking status: facts, figures, and fallacies. *Thorax*. 1997; 52: 820–7.
  46. Miles DS, Sawka MN, Wilde SW, Durbin RJ, Gotshall RW, Glaser RM. Pulmonary function changes in wheelchair athletes subsequent to exercise training. *Ergonomics*. 1982; 25: 239–46.
  47. Gass GC, Watson J, Camp EM, Court HJ, McPherson LM, Redhead P. The effects of physical training on high level spinal lesion patients. *Scand J Rehabil Med*. 1980; 12: 61–5.
  48. Chen H-I, Kuo C-S. Relationship between respiratory muscle function and age, sex, and other factors. *J Appl Physiol*. 1989; 66: 943–8.
  49. Gimenez M, Cereceda V, Teculescu D, Aug F, Laxenaire MC. Square-wave endurance exercise test (SWEET) for training and assessment in trained and untrained subjects. III. Effect on VO<sub>2</sub>max and maximal ventilation. *Eur J Appl Physiol*. 1982; 49: 379–87.
  50. Le Foll-de Moro D, Tordi N, Lonsdorfer E, Lonsdorfer J. Ventilation efficiency and pulmonary function after a wheelchair interval-training program in subjects with recent spinal cord injury. *Arch Phys Med Rehabil*. 2005; 86: 1582–6.
  51. Tager IB, Hollenberg M, Satariano WA. Association between self-reported leisure-time physical activity and measures of

- cardiorespiratory fitness in an elderly population. *Am J Epidemiol.* 1998; 147: 921–31.
52. Kohl HW, Blair SN, Paffenbarger RS, Macera CA, Kronenfeld JJ. A mail survey of physical activity habits as related to measured physical fitness. *Am J Epidemiol.* 1988; 127: 1228–39.
  53. Tjønnå AE, Lee SJ, Rognmo Ø, Stølen TO, Bye A, Haram PM, et al. Aerobic interval training versus continuous moderate exercise as a treatment for the metabolic syndrome: a pilot study. *Circulation.* 2008; 118: 346–54.
  54. Frederic ND, Joffrey Z, Stephane PD, Elodie P, Evely LL, Stephane D, et al. Effect of interval versus continuous training on cardiorespiratory and mitochondrial functions: relationship to aerobic performance improvements in sedentary subjects. *Am J Physiol Regul Integr Comp Physiol.* 2008; 295: R264–R72.
  55. DeBusk RF, Stenestrand U, Sheehan M, Haskell WL. Training effects of long versus short bouts of exercise in healthy subjects. *Am J Cardiol.* 1990; 65: 101–3.
  56. Jakicic JM, Winters C, Lang W, Wing RR. Effects of intermittent exercise and use of home exercise equipment on adherence, weight loss, and fitness in overweight women: a randomized trial. *JAMA.* 1999; 282: 1554–60.
  57. Schmidt WD, Biwer CJ, Kalscheuer LK. Effects of long versus short bout exercise on fitness and weight loss in overweight females. *J Am Coll Nutr.* 2001; 20: 494–501.
  58. Mohammad Y, Shaaban R, Abou Al-Zahab B, Khaltaev N, Bousquet J, Dubaybo B. Impact of active and passive smoking as risk factors for asthma and COPD in women presenting to primary care in Syria: first report by the WHO-GARD survey group. *Int J Chron Obstruct Pulmon Dis.* 2013; 8: 473–82.