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Heart 2010 96: 860-864
doi: 10.1136/hrt.2009.190744

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Andreas D Flouris,[1] Giorgos S Metsios,[1] Athanasios Z Jamurtas,[2] Yiannis Koutedakis[2]

[1]FAME Laboratory, Institute of Human Performance and Rehabilitation, Centre for Research and Technology Thessaly, Greece
[2]Department of Exercise Sciences, University of Thessaly, Trikala, Greece

Correspondence to
Dr Andreas D Flouris, FAME Laboratory, Institute of Human Performance and Rehabilitation, Centre for Research and Technology Thessaly, Karies, Trikala 42100, Greece; aflouris@cereteth.gr

All authors contributed equally to this work.

Accepted 25 March 2010

ABSTRACT

Objective Millions of non-smokers suffer daily passive smoking (PS) at home or at work, many of whom then have to walk fast for several minutes or climb a few sets of stairs. We conducted a randomised single-blind crossover experiment to assess the cardiorespiratory and immune response to physical activity following PS.

Design Data were obtained from 17 (eight women) non-smoking adults during and following 30 minutes of moderate cycling administered at baseline and at 0 hour, 1 hour and 3 hours following a 1-hour PS exposure set at bar/restaurant PS levels.

Results We found that PS was associated with a 36% and 38.7% decrease in mean power output in men and women, respectively, and that this effect persisted up to 3 hours ($p<0.05$). Moreover, at 0 hour almost all cardiorespiratory and immune variables measured were markedly reduced ($p<0.05$). For instance, FEV_1 values at 0 hour dropped by 10.2% in men and 10.8% in women, while IL-5 increased by 59.2% in men and 44% in women, respectively ($p<0.05$). At 3-hour mean values of respiratory quotient, mean power, perceived exertion, cotinine, FEV_1, IL-5, IL-6 and INFγ in both sexes, recovery diastolic and mean arterial pressure, IL-4 and TNFα in men, as well as percentage predicted FEV_1 in women remained different compared to baseline ($p<0.05$). Also, some of the PS effects were exacerbated in less fit individuals.

Conclusion It is concluded that 1 hour of PS at bar/restaurant levels adversely affects the response to moderate physical activity in healthy non-smokers for at least 3 hours following PS.

INTRODUCTION

It is beyond any doubt that both active and passive (PS) smoking generate adverse effects on human health.[1–3] And yet, despite adopted measures, the prevalence rates of smoking are increasing.[3 4] At present, more than half of European, American and Chinese adult non-smokers suffer daily PS, while global estimates include 700 million children and 50 million pregnant women.[2] Latest reports show that the global tobacco epidemic is worse today than it has been in past decades.[3 4] Indeed, it may come as a surprise to some that the tobacco industry anticipates a global expansion of the tobacco epidemic in the near future.[4] This expansion is fuelled in part by the scarcity of human experimental studies assessing the acute PS effects, permitting criticism[5] of the tobacco control movement through arguments that only chronic PS increases the risk of cardiovascular disease and that there is no scientific basis for claims that brief, acute, transient PS represents a significant acute cardiovascular health hazard to non-smokers.

Experimental data from our[6–10] and other[11–13] laboratories have shown that even brief PS periods generate marked unfavourable changes in cardiovascular, endocrine and immune mechanisms. Some of these effects are extensive, persisting for at least 3 hours following PS.[6] These findings raise concerns for potentially intensified PS-induced system disruption when additional strains are added such as physical activity and physical exertion, especially in individuals with (or at risk for) cardiovascular disease, chronic lung disease or allergies. Indeed, often, individuals are exposed to PS at home or at work and then have to walk fast for several minutes or climb a few sets of stairs. Understanding the acute and short-term effects of PS on the cardiorespiratory and immune response to physical activity is essential because the physical and metabolic adaptations involved can increase the risk of acute coronary complications and life-threatening myocardial ischaemia even in apparently healthy individuals.[14 15] The literature on PS and physical activity contains only three germane experiments in healthy individuals[16–18] and three additional experiments in patients with coronary artery disease.[19–21] Taken together, these experiments suggest that PS adversely affects physical activity performance in healthy individuals and exacerbates myocardial ischaemia in patients with coronary artery disease. Although valuable, these experiments incorporate limited data and methodological issues that constrain interpretation of the findings and either do not indicate the level of PS exposure,[16] or incorporated too high PS concentrations[17 19 20] or are severely outdated.[18 21] We conducted a randomised single-blind crossover experiment to assess the cardiorespiratory and immune response to moderate physical activity prior to as well as 0, 1, and 3 hours following a 1-hour moderate exposure to PS.

MATERIALS AND METHODS

Participants and procedures

The experimental protocol was approved by the University of Thessaly ethics review board. Seventeen healthy adults (nine men; eight women; 27.1±4.2 years; 172.6±9.42 cm; 66.1±10.59 kg) from the general population volunteered and signed informed consent. Exclusion criteria included smoking, pregnancy, evidence of cardiac or pulmonary disease and previous disease or medications

known to affect lung function. All women participants were premenopausal with regular menstruation and were tested during the late luteal phase of their menstrual cycle.

Familiarisation

Participants were given a detailed verbal description of the protocol, followed by extensive familiarisation with all data collection procedures and instruments during an initial familiarisation visit performed >5 days prior to testing. During this visit, participants underwent a maximal oxygen uptake protocol to determine the appropriate submaximal intensity used in the experiment's main trials. The test involved a 3-minute warm-up period of steady-state cycling (Monark Ergomedic 839E, Vansbro, Sweden) at 60 W, followed by increments of 30 W/min until exhaustion. Pedalling rate was maintained at 60 rpm throughout. An automated gas analyser (Vmax 29, Sensormedics, USA) was used to record respiratory variables every 20 seconds while individuals breathed in room air. The highest oxygen uptake (ml/kg/min) for any 20-second interval was recorded as the individual's maximal oxygen uptake.

Experimental design

To eliminate the effect of cotinine's comparatively long half-life, the study adopted a randomised single-blind crossover design with participants visiting the laboratory on four different occasions, separated by ≥7 days, where they underwent four different trials in a random order through a random allocation algorithm (SPSS 14.0.1, SPSS Inc). Furthermore, in order to eliminate the effect of diurnal variation, all data were collected at the same time of day. Participants arrived in the laboratory at 07.30 for every trial. In the baseline trial participants underwent a physical activity bout that started at 12.00 without any PS exposure. In the remaining trials, participants were exposed to PS for 1 hour inside an environmental chamber either at 08.00, or at 10.00 or at 11.00, while the same physical activity protocol initiated always at 12.00. This design enabled data collection in response to moderate physical activity without PS exposure (T_B) as well as immediately following (T_0), at 1 hour after (T_1) and at 3 hours after PS (T_3), with the data being collected at the same time of day and without the effect of previous or subsequent physical activity since only one assessment was conducted per trial.

Measurements of the targeted variables were conducted using identical pre-calibrated equipment in a quiet room maintained at 22–25°C air temperature. The same room was used for the participants to remain at rest before or after the PS exposures and until the initiation of physical activity. For all trials, caloric intake was restricted while ad libitum water consumption was permitted. All participants arrived at the laboratory following a 10-hour fast, and were instructed to refrain from strenuous physical activity and other excessive stressors for 72 hours prior to each trial. All testing was conducted by the same trained investigators, who were unaware of the specific trial that each participant was undergoing. To ensure that the investigators would not be able to differentiate between trials, all participants were given a shirt and athletic pants to wear upon exiting the chamber following all exposure trials and prior to the physical activity bout in the baseline trial. These clothes had been previously exposed to tobacco smoke inside the chamber and emitted strong tobacco scent. Moreover, since the exercise test was conducted always at 12.00 (with the PS exposure at 08.00, or at 10.00 or at 11.00), it was impossible for the investigators performing the exercise test to know which trial a subject was undergoing. This experimental protocol and all associated methods have been standardised in our laboratory.[6–8]

Passive smoking (PS)

During T_0, T_1 and T_3, participants remained seated at rest for 1 hour inside a 6×5×4 m environmentally controlled chamber (air temperature: 24°C; air velocity: 0.05 m/s; humidity: 45%). The PS exposure was adjusted at a carbon monoxide (CO) concentration of 23±1 ppm to meet levels previously reported for bar/restaurant environments checked via continuous measurement by a Horiba (MEXA-311GE) CO-CO_2 analyser. The desired CO concentration was achieved by combustion of cigarettes from various popular brands (ie, equal number of Camel, Davidoff Classic, Gauloises Filter, Original Red Lucky Strike, Marlboro Reds, Prince Classic and Silk Cut Purple King Size cigarettes). The cigarettes were lit and placed on ashtrays to burn until reaching the filter at different areas in the chamber. When the desired CO concentration was reached, the participant entered the chamber.

Physical activity protocol and recorded variables

Participants exercised for 30 minutes on a cycle ergometer (Monark Ergomedic 839E, Vansbro, Sweden). Throughout the exercise bout, the aforementioned automated gas analyser was used to record respiratory variables every 20 seconds while individuals breathed in room air. The pedalling rate was maintained at 60 rpm throughout and the ergometer resistance was adjusted to match 60% of the maximal oxygen uptake measured during the familiarisation trial. During exercise, respiratory exchange ratio (RER), mean power output (in kJ) and heart rate (Polar Electro, Kempele, Finland) were continuously recorded, while subjective ratings of perceived exertion (RPE) (ranging from 0 (not hard at all) to 20 (very very hard) with increments of 1)[22] and arterial blood pressure were measured every 5 minutes. Measurements of systolic and diastolic blood pressures were conducted by the same trained investigator using the same mercury sphygmomanometer (AS007, UK). The arterial blood pressure readings were used to calculate mean arterial pressure (ie, diastolic+(0.333·(systolic-diastolic))).[23]

Five minutes following the end of physical activity (passive recovery with participants lying in semi-supine position), heart rate, arterial blood pressure, cotinine, lung function and cytokine levels were measured.

Serum and urine cotinine

Serum and urine cotinine were measured. For serum cotinine analyses, 5 ml of whole blood were used from the total 15 ml collected by a certified phlebotomist from an antecubital vein into plain evacuated test tubes. For urine cotinine analyses, 80 ml urine void was collected in polyethylene specimen jars (Fisher Scientific, Pittsburgh, PA, USA) and was immediately frozen at −20°C, until analysed. The methodology of cotinine analyses in serum and urine have been standardised in our laboratory and are described in detail elsewhere.[6–8]

Lung function

Forced vital capacity (FVC), forced expiratory volume in 1 second (FEV_1), FEV_1/FVC ratio, peak expiratory flow (PEF) as well as maximum expiratory flow when 75%, 50% and 25% of FVC remains in the lungs ($MEF_{75\%}$, $MEF_{50\%}$, $MEF_{25\%}$, respectively) were measured via spirometry using a hand-held spirometer (Spiromed 180, Fukuda Sangyo, Pulmonary Products, Tokyo, Japan) calibrated before each use. The lung function assessment protocol conformed to the American Thoracic Society recommendations.[24] The percentage of predicted FEV_1 ($_{\%}FEV_1$) was also calculated based on sex-specific and age-specific reference values.[25]

Cytokine production

Levels of specific pro-inflammatory (interleukin 6 (IL-6) and tumour necrosis factor α (TNFα)) and allergic-related (interleukins 4 (IL-4), 5 (IL-5) and interferon γ (INFγ)) cytokines, were measured using 10 ml of whole blood via methodology that is described in detail elsewhere.[6–8]

Statistical analysis

The data were divided by sex into groups given the known sexual dimorphism in the acute PS effects.[8] Sample size calculations were conducted based on values from a previous PS experiment in our laboratory.[8] A two-group (men and women) by four times (T_B, T_0, T_1 and T_3) factorial analysis of variance (ANOVA) followed by sex-specific post-hoc t tests incorporating a Bonferroni adjustment was used to assess the effect of sex and time after PS on all the examined variables. Given the statistically significant effect of PS on recovery diastolic blood pressure and recovery mean arterial pressure (see Results), sex-specific stepwise multiple linear regression analyses incorporating backward elimination at the $p<0.05$ level were introduced to model the effect of perceived exertion, heart rate, lung function, cotinine and cytokine levels (independent variables) on recovery diastolic blood pressure and recovery mean arterial pressure (dependent variables). The sample size calculations were conducted with PASS 2000 (Hintze J, Number Cruncher Statistical Systems, Kaysville, UT, USA) software, while all other statistical analyses were carried out with SPSS. The level of significance was set at $p<0.05$ except for post-hoc tests in which a Bonferroni adjustment was applied.

RESULTS

Factorial ANOVA analysis ($R^2=0.816$; $_{adj}R^2=0.795$) revealed that, compared to T_B, there were significant differences for most variables at T_0 while values at T_1 and—mainly—at T_3 tended towards returning to T_B levels. The majority of the examined variables changed depending on the time of measurement (that is, statistically significant main effect of time: $F_{(75, 114)}=4.011$, $p<0.001$) and the participant's sex (statistically significant main effect of sex: $F_{(25, 36)}=75.546$, $p<0.001$). No interaction ($F_{(75, 114)}=1.309$, $p=0.097$) between time of measurement and sex was detected (ie, the effect of one of the variables being depended on the level of the other variable).

Results from sex-specific post-hoc t tests are presented in tables 1–3 and figure 1 (given the increased volume of results, only the statistically significant results are shown). A general trend was observed whereby marked differences were detected between T_B and T_0 while values in T_1 and—mainly—T_3 showed a tendency towards returning to T_B levels. Specifically, although at T_0 the values of almost all variables were markedly reduced ($p<0.05$), at T_3 some had returned to T_B levels ($p>0.05$). Yet, mean values at T_3 of RER, mean power, perceived exertion, cotinine, FEV_1, IL-5, IL-6 and INFγ in both sexes, recovery diastolic and mean arterial pressure, IL-4 and TNFα in men, as well as $_{\%}FEV_1$ in women remained different than T_B levels ($p<0.05$).

Stepwise multiple linear regression analyses for recovery diastolic blood pressure showed associations with mean power and cotinine levels in both sexes (men ($R^2=0.572$, $F_{(4, 31)}=10.339$, $p<0.001$) and women ($R^2=0.493$, $F_{(2, 29)}=14.081$, $p<0.001$)) as well as associations with $_{\%}FEV_1$ and TNFα only in men ($p<0.05$). Moreover, the regression analysis for recovery mean arterial pressure showed significant associations with mean power, $_{\%}FEV_1$ and IL-5 in men ($R^2=0.441$, $F_{(3, 32)}=8.424$, $p<0.001$) while no associations were detected in women ($R^2<0.001$, $F_{(0, 31)}=0$, $p>0.05$). Interestingly, the associations of recovery diastolic blood pressure and recovery mean arterial pressure with mean power were inverse, suggesting that the PS effects on these independent variables were mostly seen at lower levels of performance (ie, less fit individuals).

Table 1 Mean±SD of cardiorespiratory variables for men and women for the statistically significant post-hoc comparisons

		T_B	T_0	T_1	T_3
Respiratory exchange ratio	M	0.89±0.07	1.01±0.05*	0.97±0.05†	0.98±0.05†
	W	0.88±0.05	0.96±0.05*	0.95±0.03†	0.95±0.02†
Mean power (kJ)	M	36.1±8.7‡	23.1±5.2*,‡	25.2±8.0†,‡	22.4±6.6†,‡
	W	22.5±4.1‡	13.8±4.3*,‡	16.5±3.6†,‡	16.3±3.7†,‡
Perceived exertion	M	11.3±1.0‡	13.4±1.5*	13.4±1.1†	13.6±1.0†
	W	12.5±0.7‡	13.7±0.9*	13.7±0.7†	13.9±0.8†
Recovery HR (beats/min)	M	81.1±3.6‡	89.1±8.5*,‡	89.2±3.9†,‡	82.8±4.1‡
	W	101.9±4.0‡	109.9±5.1*,‡	105.4±4.3‡	103.9±5.0‡
Recovery DBP (mm Hg)	M	69.6±5.4	78.1±3.1*	78.3±1.2†	77.0±4.4†
	W	71.9±5.3	77.5±2.3*	76.4±2.2	73.2±2.8
Recovery MAP (mm Hg)	M	86.4±4.6	94.2±3.2*	93.4±2.5†	93.7±3.9†
	W	88.5±4.9	94.2±2.7*	92.4±3.1	91.1±2.7

*Statistically significant ($p<0.05$) difference from previous time.
†Statistically significant ($p<0.05$) difference of T_1 or T_3 from T_B.
‡Statistically significant ($p<0.05$) difference between sexes for the same measurement.
DBP, diastolic blood pressure; M, men; MAP, mean arterial pressure; W, women.

DISCUSSION

Previous results by our group[7–10] in resting non-smokers have shown that moderate PS for 1 hour at bar/restaurant levels decreases lung function and increases cytokine production. Notably, the latter persists for at least 3 hours following PS. In the present experiment, we found that the cardiorespiratory and immune response to moderate physical activity in healthy non-smokers is adversely affected following PS. More importantly, the effects of PS appear to be intensified when physical activity

Table 2 Mean±SD of cotinine and lung function for men and women for the statistically significant post-hoc comparisons

		T_B	T_0	T_1	T_3
Serum cotinine (ng/ml)	M	6.6±2.5	33.9±25.8*	33.6±19.3†	26.8±12.6†
	W	8.1±2.8	27.0±8.8*	33.6±19.3†	28.8±13.6†
Urine cotinine (ng/ml)	M	61.9±18.8	187.0±76.3*	310.2±126.1*,†	265.6±130.1†
	W	67.5±23.6	185.3±66.1*	309.3±89.0*,†	270.2±77.3†
FVC (l)	M	5.8±1.1‡	5.8±1.1‡	5.8±1.1‡	5.9±1.1‡
	W	4.1±0.5‡	4.1±0.4‡	4.2±0.5‡	4.1±0.5‡
FEV_1 (l)	M	4.9±0.3‡	4.4±0.2*	4.6±0.4‡	4.5±0.3†,‡
	W	3.7±0.4‡	3.3±0.3*	3.3±0.2‡	3.4±0.3‡
$_{\%}FEV_1$ (%)	M	114.0±9.2	101.6±8.4*	105.0±9.2	103.7±9.7
	W	117.8±6.7	103.7±7.8*	106.6±9.1†	107.0±4.8†
FEV_1/FVC	M	0.88±0.08	0.79±0.09*	0.82±0.09	0.80±0.09
	W	0.90±0.06	0.80±0.08*	0.81±0.08	0.83±0.05
PEF (l/s)	M	10.0±1.2‡	10.0±1.2‡	9.8±1.1‡	9.9±1.4‡
	W	7.4±1.4‡	7.7±1.6‡	7.7±1.6‡	7.3±1.4‡
$MEF_{75\%}$ (l/s)	M	8.3±0.2‡	7.7±0.2*,‡	8.1±0.2‡	8.2±0.6‡
	W	7.0±0.5‡	6.3±0.4*,‡	6.8±0.3‡	6.7±0.5‡
$MEF_{50\%}$ (l/s)	M	5.6±0.3‡	5.0±0.4*,‡	5.2±0.3†,‡	5.3±0.3‡
	W	4.8±0.3‡	4.1±0.3*,‡	4.4±0.4‡	4.7±0.4‡
$MEF_{25\%}$ (l/s)	M	2.9±0.3	2.3±0.4*	2.7±0.3*	2.8±0.3
	W	3.1±0.3	2.5±0.2*	2.6±0.4†	2.9±0.2

*Statistically significant ($p<0.05$) difference from previous time.
†Statistically significant ($p<0.05$) difference of T_1 or T_3 from T_B.
‡Statistically significant ($p<0.05$) difference between sexes for the same measurement.
FEV_1, forced expiratory volume in 1 second; $_{\%}FEV_1$, percentage predicted FEV_1; PEF, peak expiratory flow; FVC, forced vital capacity; M, men; $MEF_{75\%}$, $MEF_{50\%}$, $MEF_{25\%}$, maximum expiratory flow when 75%, 50% and 25% of FVC remains in the lungs, respectively; W, women.

Table 3 Mean±SD of cytokine production for men and women for the statistically significant post-hoc comparisons

		T_B	T_0	T_1	T_3
IL-4 (pg/ml)	M	38.1±3.9	47.2±5.9*,‡	45.3±3.4†,‡	45.5±2.2†,‡
	W	36.4±1.9	35.3±3.5‡	36.3±4.1‡	35.4±3.4‡
IL-5 (pg/ml)	M	99.0±6.4	157.6±7.1*	150.9±7.5†	140.2±10.4*,†
	W	103.9±11.8	149.6±14.6*	145.1±16.1†	133.1±16.0†
IL-6 (pg/ml)	M	6.6±1.3‡	12.4±3.0*	14.7±2.5†,‡	14.6±1.7†,‡
	W	8.9±1.5‡	15.1±2.8*	17.5±2.0†,‡	15.5±2.2†,‡
TNFα (pg/ml)	M	14.3±1.7‡	20.9±2.1*,‡	23.6±3.0†,‡	23.7±2.2†,‡
	W	11.4±2.5‡	17.3±2.3*,‡	15.4±1.5†,‡	13.9±2.1‡
INFγ (pg/ml)	M	0.41±0.10	0.61±0.10*	0.55±0.10†	0.55±0.10†
	W	0.41±0.08	0.56±0.13*	0.58±0.10†	0.56±0.10†

*Statistically significant ($p<0.05$) difference from previous time.
†Statistically significant ($p<0.05$) difference of T_1 or T_3 from T_B.
‡Statistically significant ($p<0.05$) difference between sexes for the same measurement.
IL-4, 5 and 6, interleukins 4, 5 and 6, respectively; INFγ, interferon gamma; M, men; TNFα, tumour necrosis factor alpha; W, women.

is involved since the majority of metabolic, vascular, respiratory and immune indices were markedly compromised for at least 3 hours following PS. These results suggest that individuals that are exposed to PS and then have to become physically active may be at an increased risk for allergic, respiratory and cardiovascular symptoms, especially those that are unfit.

The scarce previous experiments investigating responses to physical activity following PS exposure have found increased RER, and perceived exertion[16] increased heart rate[18 21] and prolonged time to heart rate recovery,[17] increased systolic and diastolic blood pressure[21] as well as similar FVC and FEV_1 but lower $MEF_{25\%}$.[18] On the other hand, no changes in submaximal oxygen uptake have been reported,[16] while maximal oxygen uptake has been found both decreased[16 17] and increased.[18] Although valuable, these experiments incorporated limited data and methodological issues that constrain interpretation of the findings. To the best of our knowledge this is the first experiment to use a standardised experimental protocol[6–8] to simultaneously investigate the effects of PS and their duration on the cardiorespiratory and immune response to moderate physical activity in healthy non-smokers . Given that the limited previous studies either do not indicate the level of PS exposure,[16] or incorporated too high PS concentrations,[17 19 20] or are severely outdated,[18 21] the present unique experiment significantly improves our understanding of the PS-induced effects and their duration providing valuable findings for generating/updating public health guidelines.

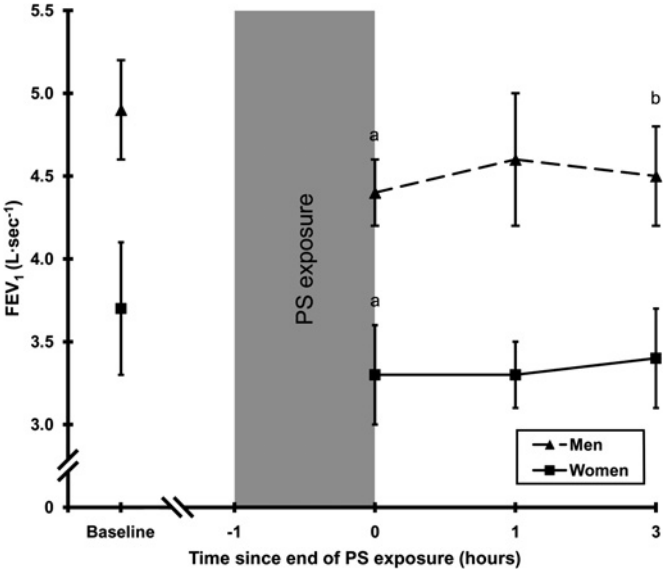

Figure 1 Mean±SD of forced expiratory volume in 1 second (FEV_1) for men and women as a function of time since the end of passive smoking (PS) exposure. Values dropped 10.2% in men and 10.8% in women between T_B and T_0. a=statistically significant ($p<0.05$) difference from previous time point; b=statistically significant ($p<0.05$) difference of T_1 or T_3 from T_B.

We found that PS was associated with a 36% and 38.7% decrease in mean power output in men and women, respectively, and that this effect persisted for at least 3 hours following PS. Therefore, PS results in a dramatic reduction in work output for the same level of oxygen uptake. With regard to energy sources being metabolised, we found a statistically significant increase in RER even 3 hours following PS, suggesting greater reliance on anaerobic metabolism. This mirrors the findings that PS increases resting metabolic processes, possibly through alterations in thyroid hormone secretion[7] which, in turn, occurs because of disruption of extrathyroidial mechanisms or through a down-regulation of thyroid gland hormonogenesis via PS-induced changes in gonadal hormones.[8] On the other hand, the PS-induced increase in RER may also reflect predominance of glucose utilisation.

Recovery heart rate, diastolic blood pressure and mean arterial pressure were significantly increased by PS and, in the latter two, these effects were apparent at least 3 hours following the exposure. Based on the calculated regression models, the effects of PS on vascular variables were emphasised in men where they showed associations with $_{\%}FEV_1$, IL-5 and TNFα. Overall, the cardiorespiratory effect of PS on both sexes was associated with cotinine and was mostly seen at lower levels of performance—that is, in less fit individuals. This is noteworthy, as less fit individuals are at an increased risk for cardiovascular and respiratory events.[26]

Lung function was markedly deteriorated when the physical task was administered immediately following PS and tended to return to baseline values thereafter, confirming the results of our recent experiment in resting non-smokers.[6] Yet, FEV_1 in both sexes, $MEF_{50\%}$ in men as well as $_{\%}FEV_1$ and $MEF_{25\%}$ in women remained decreased even 3 hours following PS, showing that the results of PS on lung function are intensified when physical activity is involved.

The observed PS-induced increase in circulating inflammatory markers extends the findings of a small number of previous human experiments reporting PS-induced increases of interleukin 1 β,[8] white blood cell count, C-reactive protein, homocysteine, fibrinogen[13] as well as leucocyte counts accompanied by an activation of the immune cells.[27] The present increases in IL-4 and TNFα only in men results confirm recent experiments by our group reporting an increased PS-induced inflammatory reaction in men compared to women.[8]

Pro-smoking groups use the reduced sales of tobacco products in selected few countries to argue that smoking bans are not necessary. This delusion is accompanied by an equally pernicious myth—that there is '…no scientific basis for claims that brief, acute, transient exposure to secondhand smoke…represents any other significant acute cardiovascular health hazard in non-smokers'.[5] These arguments are rejected in many studies referenced in the present paper. In fact, to our knowledge, all human exposure studies investigating the acute effects of PS have reported significant detrimental results. With regard to the present paper, it is concluded that 1 hour of PS at levels similar to those of bars/restaurants adversely affects the cardiorespiratory and immune response to physical activity in healthy non-smokers for at least 3 hours following PS.

Acknowledgements The authors thank all participants for their time and enthusiasm, Drs Manolis N Tzatzarakis and Aristeidis M Tsatsakis at the Centre of Toxicology Science and Research, School of Medicine, University of Crete, for conducting the cotinine analyses, and Dr Anastasios Germenis at the School of Health Sciences, Department of Medicine, University of Thessaly, for conducting the analysis on cytokines. The researchers declare full independence from external parties and any other conflicts of interest related to this work.

Competing interests None.

Patient consent Obtained.

Ethics approval This study was conducted with the approval of the University of Thessaly Ethics Review Board.

Provenance and peer review Not commissioned; externally peer reviewed.

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