Secondhand hookah smoke: an occupational hazard for hookah bar employees

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ABSTRACT

Background Despite the increasing popularity of hookah bars, there is a lack of research assessing the health effects of hookah smoke among employees. This study investigated indoor air quality in hookah bars and the health effects of secondhand hookah smoke on hookah bar workers.

Methods Air samples were collected during the work shift of 10 workers in hookah bars in New York City (NYC). Air measurements of fine particulate matter ($PM_{2.5}$), fine black carbon ($BC_{2.5}$), carbon monoxide (CO), and nicotine were collected during each work shift. Blood pressure and heart rate, markers of active smoking and secondhand smoke exposure (exhaled CO and saliva cotinine levels), and selected inflammatory cytokines in blood (ineterleukin (IL)-1b, IL-6, IL-8, interferon γ (IFN-γ), tumour necrosis factor (TNF-α)) were assessed in workers immediately prior to and immediately after their work shift.

Results The $PM_{2.5}$ (gravimetric) and $BC_{2.5}$ concentrations in indoor air varied greatly among the work shifts with mean levels of 363.8 $\mu g/m^3$ and 2.2 $\mu g/m^3$, respectively. The mean CO level was 12.9 ppm with a peak value of 22.5 ppm CO observed in one hookah bar. While heart rate was elevated by 6 bpm after occupational exposure, this change was not statistically significant. Levels of inflammatory cytokines in blood were all increased at postshift compared to preshift testing with IFN-ϒ increasing from 0.85 (0.13) to 1.6 (0.25) (mean (standard error of the mean; SEM)) pg/mL ($p<0.01$). Exhaled CO levels were significantly elevated after the work shift with 2 of 10 workers having values >90 ppm exhaled CO.

Conclusions These results demonstrate that hookah bars have elevated concentrations of indoor air pollutants that appear to cause adverse health effects in employees. These data indicate the need for further research and a marked need for better air quality monitoring and policies in such establishments to improve the indoor air quality for workers and patrons.

INTRODUCTION

The intentional inhalation of tobacco combustion products causes profound respiratory, cardiovascular and numerous other adverse health effects.[1] In addition to the effects of mainstream tobacco smoke, secondhand smoke (SHS) exposure also causes a range of serious health problems in adults, adolescents and children.[1] SHS is the third leading preventable cause of death in the USA, responsible for 3000 lung cancer and 35 000 coronary heart disease deaths annually in never-smokers in the USA.[2] Documented deleterious health effects on workers passively exposed to SHS in various settings played a critical role in implementing the large number of effective public and workplace restrictions on smoking.[3] These efforts, in turn, have resulted in marked decreases worldwide in the involuntary exposure of individuals to SHS.

Accompanying the decline in cigarette use, recent evidence indicates that increasing numbers of US adolescents and adults are turning to alternative tobacco products,[4 5] such as hookahs (aka water pipes). The 2013 National Youth Tobacco Survey reported that 14% of high school students have ever tried hookah and that 5.2% had used it within the past month.[6] A similar alarming statistic from the Monitoring the Future (MTF) Survey found that past year hookah use significantly increased among high school seniors from 18.3% in 2012 to 21.4% in 2013.[7] Both of these surveys used large, nationally representative samples of adolescents in the USA.

Most hookah users erroneously believe that hookah water pipes are safer and less addictive alternatives to cigarettes.[8–20] This belief, unsurprisingly, has led to the social normalisation of smoking hookah as a trendy and acceptable way to socialise with friends.[18 20–24] A number of studies now demonstrate poor indoor air quality of hookah bars,[25–27] and deleterious health effects of secondhand hookah smoke,[28] especially to exposed children,[29] as summarised in a recent review.[30] The perceived lack of concern that hookah bar workers and owners have about the dangers of working in such establishments may be partly due to a lack of awareness of the emerging evidence on the health effects of hookah water pipe mainstream and SHS, but is very likely due to the absence of any data about effects of exposure on the workers themselves.

This study assessed: (1) hookah bar indoor air quality for particulate matter ($PM_{2.5}$), black carbon (BC), carbon monoxide (CO) and nicotine; (2) biological makers of SHS exposure, such as exhaled CO (eCO) and saliva cotinine; (3) cardiac function; and (4) selected markers of systemic inflammation in hookah bar employees before and after their work shifts.

METHODS

Fourteen hookah bars located in Manhattan, New York City (NYC) were selected using the search engine 'Yelp'. Each of these hookah bars was visited for recruitment by directly talking with the workers. From 4 of the 14 targeted hookah bars in the East Village and Greenwich Village in Manhattan, 10 hookah bar workers who met the

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inclusion and exclusion criteria were recruited (table 1). Inclusion was limited to individuals aged 20 or older working in a hookah bar in Manhattan, NYC. Those who were pregnant or current cigarette smokers were excluded from the study. Prior to sample recruitment or testing, approval was obtained from the Institutional Review Board of the New York University School of Medicine and informed consent was obtained from all participants.

The indoor air quality of the selected hookah bars was tested on the nights that workers of that specific venue were being examined (see table 2 for the number of workers studied each night; note that a total of five workers at bar 2 were studied on three separate occasions). Biological testing and air quality sampling were targeted for Thursday, Friday and Saturday nights when hookah bars are most crowded.

Styrene, 2-piece cassettes (SKC, Inc; Eighty Four, Pennsylvania, USA) were used to collect total PM samples on polytetrafluoroethylene (PTFE) filters (low trace element background; Pall Corp, Port Washington, New York, USA) at 4 L/min for gravimetric analyses. Sample flow rates were calibrated before each run using a dry gas meter (BIOS Dry Cal DC-Lite., Brandt Instruments, Prairieville, Louisiana, USA). Nicotine was sampled with an XAD-4 sampling tube (SKC, Inc) at a flow rate of 1.5 L/min. Filters and nicotine sorbent tubes were sampled with personal sampling pumps with battery packs (BGI 400, BGI, Inc, Waltham, Massachusetts, USA). Handbags/backpacks contained a micro-aethalometer (Model AE51, AethLabs, San Francisco, California, USA) and a CO data logger (Lascar EL-USB-CO-300, Erie, Pennsylvania, USA) to measure real time BC with a mass median aerodynamic diameter (MMAD) of 2.5 μm ($BC_{2.5}$) (1 min intervals) and CO concentrations, respectively. A particle size selection device was attached to the inlet of the aethalometer to monitor $BC_{2.5}$. On the night of the testing, a member of the research team visited the corresponding hookah bar unannounced at approximately 22:00 for a 1–2 h sampling session. Sampling equipment was concealed in a bag or backpack with a small portion of the sampling tubing protruding from one corner. Venue characteristics such as the number of hookah water pipes being used at the time of sampling, active smokers, total patrons and the general ventilation status of the bars were recorded every 15 min in the field and are presented in table 2 (mean (SD)).

Participants were tested before the start of their work shift and once after the work shift to detect changes in cardiac function and inflammatory biomarkers. Preshift testing was done 1–2 h prior to the start of each worker's shift. Postshift testing was started 15–30 min after their shift was over. Workers usually started their shifts around 16:00–17:00 in the afternoon and finished around 3:00–4:00 in the morning. Basic demographic information including name, age, sex, height and weight of the participants as well as their work shift length was recorded. They were also asked if they had worked the night before testing took place.

The prework and postwork shift cardiovascular tests included heart rate, which was continuously monitored with a Polar H7 chest strap sensor, iPod Touch, and the Heart Rate Variability Logger app (Marco Altini), and blood pressure, which was measured three times at a minimum of 1 min intervals with a semi-automated monitor (Omron HEM- 705CP, Omron Healthcare, Inc., Japan). Exhaled CO (eCO) levels were measured prework and postwork shift with an eCO monitor (Bedfont Pico Plus Smokerlyzer, coVita, Haddonfield, New Jersey, USA).

Inflammatory biomarkers in blood included ineterleukin (IL)-1b, IL-6, IL-8, IL-10, IL-12 p70, interferon γ (IFN-γ), and tumour necrosis factor α (TNF-α). Blood samples were obtained using a finger stick using a diabetic lancet and dried on a Guthrie card (Whatman) and stored in a dessicator at −80°C until analysis. Blood cytokines were measured using the Meso Scale Discovery System using the methodology of Hejl *et al*.[31] Cotinine levels were assayed by ELISA (Salimetrics) in saliva samples collected prework and postwork shift.[31]

All the data except for blood and saliva samples were recorded on a spreadsheet at the time they were obtained. Data were entered into SPSS 21. First, descriptive characteristics and frequencies were calculated. Pre and post shift measurements were compared using a paired sample t test, although because of a lack of normally distributed values, cytokine comparisons were made with a non-parametric Wilcoxon matched-pairs signed rank test).

Table 1 Characteristics of hookah bar workers (n=10)

Characteristic	Result
Duration of shift in hours, mean (SD)	10.3 (1.4)
Age	24.6 (2.8)
BMI	21.4 (2.8)
Ethnicity, n (%)	
Caucasian	7 (70)
Asian	1 (10)
Hispanic	2 (20)
Sex, n (%)	
Male	2 (20)
Female	8 (80)
Hookah bar, n (%)	
#1	1 (10)
#2	5 (50)
#3	3 (30)
#4	1 (10)
Worked the night before testing, n (%)	
Yes	7 (70)
No	3 (30)

BMI, body mass index.

RESULTS

Among the 10 recruited hookah bar workers, the majority were Caucasian females with an average age of 25 years (table 1). Since the study occurred on weekends when the bars were most busy, many of them worked the night before testing and had shifts as long as 12 h with an average of 10 h.

During the 1–2 h air sampling periods, the $PM_{2.5}$ levels varied among the hookah bars and the mean gravimetric $PM_{2.5}$ was 363.8 μg/m^3 with a range of 62.5–912.5 μg/m^3 (table 2). The mean $BC_{2.5}$ level was 2.2 μg/m^3 with a range of 1.1–3.9 μg/m^3. Mean CO was 12.9 ppm with a range of 9.5–22.5 ppm. In general, the level of indoor air pollutants was directly proportional to the number of active hookah smokers and water pipes in use in the hookah bars, although as seen in bar 1, other factors such as room size and the presence of open windows or doors appeared to play a role in indoor air pollution levels (table 2). Additionally, airborne nicotine was found in all establishments, despite the ban on the use of tobacco-based shisha in water pipes in NYC hookah bars (table 2).

Cardiac functions were measured immediately before and after hookah workers' shifts. Compared to the preshift measurement, postshift heart rate increased from 77.8+9.8 to 83.9

Table 2 Hookah bar air pollution concentrations

	Bar 1	Bar 2-Night 1	Bar 2-Night 2	Bar 2-Night 3	Bar 3	Bar 4
Workers studied	1	1	2	2	3	1
PM ($\mu g/m^3$) Gravimetric	912.5	686.0	246.8	100.0	62.5	175.0
BC ($\mu g/m^3$)	2.0	3.7	3.9	1.2	1.4	1.2
CO (ppm)	10.5	22.5	13.9	9.79	9.5	11.4
Nicotine ($\mu g/m^3$)	10.5	4.3	4.9	0.2	0.6	1.9
Number of active smokers, mean (SD)	10.0 (6.0)	27.4 (2.0)	31.5 (6.0)	16.8 (5.7)	15.2 (3.5)	6.2 (2.4)
Total patrons, mean (SD)	11.6 (5.0)	35.6 (3.3)	39.8 (3.5)	22.5 (9.1)	25.6 (3.6)	7.7 (2.3)
Active water pipes	4.4 (2.1)	13.6 (1.7)	13.3 (2.3)	8.8 (2.2)	6.4 (2.1)	3.5 (0.8)
Open window or door	No	Yes	Yes	No	Yes	No

BC, black carbon; CO, carbon monoxide; PM, particulate matter.

+8.7 bpm (mean+SD, p=0.14), while the eCO significantly increased from 8.3+6.9 to 49.4+32.7 ppm (p=0.001) post shift. Two workers had eCO levels of greater than 90 ppm at postshift testing, though both had worked in the hookah bars the night before testing. Neither systolic nor diastolic blood pressure varied between preshift and postshift. Levels of saliva cotinine were elevated both prework and postwork shift suggesting a carryover effect for secondhand hookah smoke exposure (half life for salivary cotinine is ~ 15 h), the use of hookah or cigarettes despite the exclusion criteria, or exposure to secondhand cigarette smoke outside the work place (table 3).

Five of the assessed inflammatory cytokine markers were increased at postshift testing compared to preshift testing (the majority of IL-10 and IL-12 p70 values were below the limit of detection and are not presented). IFN-γ was the only cytokine that was statistically increased: 0.85+0.13 pg/mL (mean+SE) at preshift to 1.6+0.25 at postshift. Compared to preshift measurements, it is notable that values of IL-6, IL-8, and TNF-α were also approximately doubled at postshift testing, suggesting a broad systemic inflammatory response to hookah smoke (table 4).

DISCUSSION

We report here that selected hookah bars in NYC contain elevated concentrations of indoor air pollutants, far exceeding the US federal standards for outdoor air quality,[32] that resulted in indications of systemic inflammation as evidenced by elevations in inflammatory markers in hookah bar workers. Similar increases in levels of pollutants, such as PM2.5, BC, CO, and nicotine, have now been reported in several studies.[25 26 33] This documented poor indoor air quality suggests that hookah bar workers, as well as patrons of hookah bars, inhale airborne particles and gases that can potentially cause adverse health effects.[28–30] The levels of worker exposure to PM and nicotine in the present study were comparable to exposure levels measured in hospitality venues prior to cigarette smoking bans (8–1375 $\mu g/m^3$ PM2.5; 5.8–14.4 $\mu g/m^3$ nicotine).[34 35] Numerous epidemiology studies have previously demonstrated associations between airborne PM and BC levels and adverse cardiopulmonary effects although the ambient concentrations were much lower than what was measured in the hookah bars in this study.[36 37] One study, in particular, found adverse blood pressure changes associated with daily ambient exposures to BC concentrations that were approximately 1/2–1/3 of the indoor levels measured in this study.[38]

Table 3 Comparison between pre shift and postshift cardiac function

	Preshift Mean (SD)	Postshift Mean (SD)	p Value
Heart rate (bpm)	77.8 (9.8)	83.9 (8.7)	0.14
Exhaled CO (ppm)	8.3 (6.9)	49.4 (32.7)	0.001
Systolic blood pressure (mm Hg)	116.7 (9.5)	115.5 (10.18)	NS
Diastolic blood pressure (mm Hg)	69.3 (5.2)	69.6 (8.4)	NS
Salivary cotinine (ng/mL)	23.8	27.9	NS

NS, not significantly different in comparing preshift to postshift.
CO, carbon monoxide.

The present study demonstrates that hookah workers have significantly elevated exhaled CO, a classic biomarker of exposure to secondhand tobacco smoke, after their work shift compared to before. Although no significant changes were detected in blood pressure, there were modest increases in heart rate as well as inflammatory cytokines in blood immediately following their work shifts. Although the increase in heart rate was not statistically significant, it is consistent with previously reported effects of inhaled tobacco smoke on heart rate.[39] The increase in heart rate could have been due to the physical activity of the workers during their shift, although the hookah workers were transported by taxi to the NYU Medical Center where the physiological measurements began approximately 15–30 min after the end of their work shift. This study, therefore, provides evidence for the potential for adverse short-term health outcomes while working in hookah bars, indicating the need for larger studies and immediate attention from health officials and tobacco policymakers.

High levels of eCO were detected in some workers after their work shift. CO has been implicated as a biomarker for many airway diseases, including chronic obstructive pulmonary disease (COPD),[40] respiratory infections,[41] airway obstruction,[42] cystic fibrosis,[43 44] sepsis[45] and asthma.[46 47] A Canadian study assessed levels of eCO in non-smoking field staff before and after visiting hookah lounges and found that the mean eCO level increased from a 2 ppm baseline to 70 ppm after 2 h of SHS exposure. Acute CO poisoning in hookah users has been reported among hookah bar patrons presenting to the hospital with syncope, confusion, and slurred speech.[48–52]

Although not statistically significant, workers' heart rates post shift were increased compared to pre shift values, suggesting that acute hookah SHS exposure has the potential to affect cardiac function. Indeed, Cobb *et al*[53] found that smoking hookah products was associated with reduced heart rate variability, an index of cardiovascular health, suggesting that

Table 4 Comparison of select systemic inflammatory markers preshift and postshift, n=10

	IL-1b		IL-6		IL-8		IFN-γ		TNF-α	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Mean (SE)	4 (1.1)	4.7 (0.7)	0.48 (0.07)	1.1 (0.5)	3.7 (0.3)	6.3 (3.1)	0.85 (0.13)	1.6 (0.25)	0.66 (0.07)	1.6 (0.8)
*	p=0.43		p=0.16		p=0.99		p=0.01		p=0.08	

*Non-parametric Wilcoxon matched-pairs signed rank test.
IL, ineterleukin; IFN-γ, interferon γ; TNF-α, tumour necrosis factor α.

hookah smoking acutely compromises cardiac function. Using a pretest and post-test design, Hawari *et al*[54] found that acute hookah smoking impaired lung function and exercise capacity by increasing CO levels, respiratory rate and systolic blood pressure, as well as decreasing oxygen consumption and forced expiratory flow. In addition, in vitro studies have demonstrated the ability of tobacco-based hookah smoke condensate to induce endothelial cell dysfunction by exerting oxidative stress, inflammation and impaired endothelial vasodilatory function and repair mechanisms.[45 55] No studies to date, however, have explored the potential long term dangers to employees working in hookah bars who are exposed for extended periods of time.

Each of the five detectable cytokines was increased at post-testing compared to pre-testing, with the increase in INF-ϒ being statistically significant. Systemic inflammation is increasingly recognised as a risk factor for cardiovascular and pulmonary diseases, such as atherosclerosis,[56] ischaemic heart diseases,[57 58] stroke and COPD.[59] IL-6 and TNF-α have been specifically implicated in the progression of atherosclerosis[60] and IL-1, TNF-α, and IFN-ϒ are believed to be intimately involved in ischaemic heart disease by promoting angiogenesis and morphological and functional changes in endothelial cells.[61] IL-8 is postulated to play a significant role in the pathogenesis of COPD.[62] A study by Wanamethee *et al* examined the associations between cigarette smoking and inflammatory markers in 2920 men aged 60–79 and found that compared with never smokers, current cigarette smokers had significantly higher levels of inflammatory markers, such as C reactive protein, white cell count, and fibrinogen. It also found that most of these markers improved within 5 years of smoking cessation, though it took over 20 years to revert to the levels of never smokers,[63] suggesting the benefit of smoking cessation on cardiovascular risk. Thus, although the number of inflammatory cytokines that could be measured using the minimally invasive dried blood spot technique was limited, findings from the current study demonstrate the likelihood that hookah smoking poses similar cardiovascular and cardiopulmonary risks to hookah workers through derangements in systemic inflammatory pathways.

A growing number of communities and states are adopting wide-ranging clean indoor air laws to protect visitors and employees from the harmful SHS effects of cigarettes. Thirty-five US states, including New York, have passed laws that require 100% smoke-free workplaces including restaurants and bars.[26 64] However, hookah water pipe use is not subject to the same regulations, and its use is currently unregulated by the FDA.[65] In the past, exposure measurements of cigarette SHS in workers had a marked influence on the development of clean air acts. The profound paucity of health data about secondhand hookah smoke, particularly for workers, warrants more research in this area to create evidence-based regulations on hookah use in bars and other public venues. Moreover, it should be noted that salivary cotinine levels were elevated preshift in the hookah workers, suggesting prolonged exposure to secondhand tobacco smoke, despite the ban on the use of tobacco-based shisha in hookah water pipes in NYC hookah bars. Alternatively, it is impossible to rule out that the hookah workers may have smoked cigarettes (although cigarette smoking was an exclusion criterion in the present study), or that they were exposed to secondhand cigarette smoke in non-occupational settings. With one exception, the workers stated that they did not smoke hookah while working. That one individual worked as a 'hookah boy' who was responsible for lighting and tending the patrons' water pipes, yet surprisingly had a post shift eCO value of 26 ppm, which was below the group mean of 49.4 (table 3).

A number of limitations to this study deserve mention. The selection of hookah bar workers, and thus the bars, was not random. Hookah bar workers were recruited and studied based on the ratings and reviews on two websites regarding the popularity of NYC hookah bars. The relatively small sample size of hookah bar workers, although sufficient to demonstrate both that air quality in hookah bars was dangerous to health and that it caused negative health outcomes in workers limits our ability to generalise these finding to health measures or to other hookah venues. The study also lacked assessment of pulmonary function and provides no insights into potential long-term deleterious effects on the health of hookah bar workers.

In summary, this is the first study that we are aware of to investigate occupational hazards associated with working in hookah bars and it found both dangerous alterations in indoor

What this paper adds

- This is the very first study to investigate the effects of secondhand hookah smoke on workers in hookah bars.
- Prior to this study there were no data on this subject.
- It is not known what the acute effects of such exposure is on pulmonary function and cardiovascular function.
- It is not known if the observed effects would differ in varying sized and ventilated hookah bars; by exposure to different types of shisha; or by characteristics of workers such as age, gender and underlying health conditions.
- The long-term consequences of such exposure to workers in such establishments are not known.
- As a result of this study, we know that there are adverse cardiopulmonary effects in hookah bar workers caused by exposure to secondhand hookah smoke during work shifts. As hookah bars proliferate in the USA and worldwide, these findings suggest that large numbers of workers in these venues will be adversely affected, providing vitally needed information for public health officials, policy makers and clinicians.

air quality and adverse changes in health measures of concern. If corroborated in larger studies in multiple settings, such findings will have marked implications for educational and regulatory actions. Hazards to workers in hookah bars raise the question of whether the use of these tobacco delivery systems should be restricted in public spaces, much as the demonstration of negative effects on the health of workers exposed to secondhand cigarette smoke has done.

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REFERENCES

1 Samet JM. Tobacco smoking: the leading cause of preventable disease worldwide. *Thorac Surg Clin* 2013;23:103–12.
2 Jones SC, Travers MJ, Hahn EJ, *et al*. Secondhand smoke and indoor public spaces in Paducah, Kentucky. *J Ky Med Assoc* 2006;104:281–8.
3 Office on Smoking and Health (US). The Health Consequences of Involuntary Exposure to Tobacco Smoke: a Report of the Surgeon General. Atlanta, GA: Centers for Disease Control and Prevention (US), 2006. 10, Control of Secondhand Smoke Exposure. http://www.ncbi.nlm.nih.gov/books/NBK44326/
4 Bhattacharyya N. Trends in the use of smokeless tobacco in United States, 2000–2010. *Laryngoscope* 2012;122:2175–8.
5 Saunders C, Geletko K. Adolescent cigarette smokers' and non-cigarette smokers' use of alternative tobacco products. *Nicotine Tob Res* 2011;14:977–85.
6 Arrazola RA, Neff LJ, Kennedy SM, *et al*. Tobacco use among middle and high school students—United States, 2013. *MMWR Morb Mortal Wkly Rep* 2014;63:1021–6.
7 Johnston LD, O'Malley PM, Miech RA, *et al*. *Monitoring the future national results on drug use: 1975–2013: overview, key findings on adolescent drug use*. Ann Arbor: Institute for Social Research, The University of Michigan, 2014. http://www.monitoringthefuture.org/pubs/monographs/mtf-overview2013.pdf
8 Al-Naggar RA, Saghir FS. Water pipe (shisha) smoking and associated factors among Malaysian university students. *Asian Pac J Cancer Prev* 2011;12:3041–7.
9 Eissenberg T, Ward KD, Smith-Simone S, *et al*. Waterpipe tobacco smoking on a U.S. College campus: prevalence and correlates. *J Adolesc Health* 2008;42:526–9.
10 Grekin ER, Ayna D. Waterpipe smoking among college students in the United States: a review of the literature. *J Am Coll Health* 2012;60:244–9.
11 Griffiths MA, Ford EW. Hookah smoking: behaviors and beliefs among young consumers in the United States. *Soc Work Public Health* 2014;29:17–26.
12 Heinz AJ, Giedgowd GE, Crane NA, *et al*. A comprehensive examination of hookah smoking in college students: use patterns and contexts, social norms and attitudes, harm perception, psychological correlates and co-occurring substance use. *Addict Behav* 2013;38:2751–60.
13 Holtzman AL, Babinski D, Merlo LJ. Knowledge and attitudes toward hookah usage among university students. *J Am Coll Health* 2013;61:362–70.
14 Jackson D, Aveyard P. Waterpipe smoking in students: prevalence, risk factors, symptoms of addiction, and smoke intake. Evidence from one British university. *BMC Public Health* 2008;8:174.
15 Jawaid A, Zafar AM, Rehman TU, *et al*. Knowledge, attitudes and practice of university students regarding waterpipe smoking in Pakistan. *Int J Tuberc Lung Dis* 2008;12:1077–84.
16 Klassen BJ, Smith KZ, Grekin ER. Differential relationships between religiosity, cigarette smoking, and waterpipe use: implications for college student health. *J Am Coll Health* 2013;61:381–5.
17 Labib N, Radwan G, Mikhail N, *et al*. Comparison of cigarette and water pipe smoking among female university students in Egypt. *Nicotine Tob Res* 2007;9:591–6.
18 Roskin J, Aveyard P. Canadian and English students' beliefs about waterpipe smoking: a qualitative study. *BMC Public Health* 2009;9:10.
19 Sutfin EL, McCoy TP, Reboussin BA, *et al*. Prevalence and correlates of waterpipe tobacco smoking by college students in North Carolina. *Drug Alcohol Depend* 2011;115:131–6.
20 Primack BA, Sidani J, Agarwal AA, *et al*. Prevalence of and associations with waterpipe tobacco smoking among U.S. university students. *Ann Behav Med* 2008;36:81–6.
21 Cobb CO, Khader Y, Nasim A, *et al*. A multiyear survey of waterpipe and cigarette smoking on a US university campus. *J Am Coll Health* 2012;60:521–7.
22 Ghafouri N, Hirsch JD, Heydari G, *et al*. Waterpipe smoking among health sciences university students in Iran: perceptions, practices and patterns of use. *BMC Res Notes* 2011;4:496.
23 Noonan D, Kulbok PA. Beliefs and norms associated with smoking tobacco using a waterpipe among college students. *J Addict Nurs* 2012;23:123–8.
24 Roohafza H, Sadeghi M, Shahnam M, *et al*. Perceived factors related to cigarette and waterpipe (ghelyan) initiation and maintenance in university students of Iran. *Int J Public Health* 2011;56:175–80.
25 Fiala SC, Morris DS, Pawlak RL. Measuring indoor air quality of hookah lounges. *Am J Public Health* 2012;102:2043–5.
26 Cobb CO, Vansickel AR, Blank MD, *et al*. Indoor air quality in Virginia waterpipe cafes. *Tob Control* 2013;22:338–43.
27 Zhou S, Weitzman M, Vilcassim R, *et al*. Air quality in New York City hookah bars. *Tob Control* 2015;24(e3):e193–8.
28 Zeidan RK, Rachidi S, Awada S, *et al*. Carbon monoxide and respiratory symptoms in young adult passive smokers: a pilot study comparing waterpipe to cigarette. *Int J Occup Med Environ Health* 2014;27:571–82.
29 Tamim H, Musharrafieh U, El Roueiheb Z, *et al*. Exposure of children to environmental tobacco smoke (ETS) and its association with respiratory ailments. *J Asthma* 2003;40:571–6.
30 Kumar SR, Davies S, Weitzman M, *et al*. A review of air quality, biological indicators and health effects of second-hand waterpipe smoke exposure. *Tob Control* 2015;24 (Suppl 1):i54–9.
31 Hejl AM, Adetona O, Diaz-Sanchez D, *et al*. Inflammatory effects of woodsmoke exposure among wildland firefighters working at prescribed burns at the Savannah River Site, SC. *J Occup Environ Hyg* 2013;10:173–80.
32 Lippmann M. Winter air pollution and respiratory function. *Occup Environ Med* 2003;60:81.
33 Zhang B, Haji F, Kaufman P, *et al*. 'Enter at your own risk': a multimethod study of air quality and biological measures in Canadian waterpipe cafes. *Tob Control* 2015;24:175–81.
34 Bohac DL, Hewett MJ, Kapphahn KI, *et al*. Change in indoor particle levels after a smoking ban in Minnesota bars and restaurants. *Am J Prev Med* 2010;39(6 Suppl 1):S3–9.
35 Jenkins RA, Counts RW. Occupational exposure to environmental tobacco smoke: results of two personal exposure studies. *Environ Health Perspect* 1999;107(Suppl 2):341–8.
36 Tsai DH, Amyai N, Marques-Vidal P, *et al*. Effects of particulate matter on inflammatory markers in the general adult population. *Part Fibre Toxicol* 2012;9:24.
37 Weichenthal S, Kulka R, Belisle P, *et al*. Personal exposure to specific volatile organic compounds and acute changes in lung function and heart rate variability among urban cyclists. *Environ Res* 2012;118:118–23.
38 Louwies T, Nawrot T, Cox B, *et al*. Blood pressure changes in association with black carbon exposure in a panel of healthy adults are independent of retinal microcirculation. *Environ Int* 2015;75:81–6.
39 Felber Dietrich D, Schwartz J, Schindler C, *et al*. Effects of passive smoking on heart rate variability, heart rate and blood pressure: an observational study. *Int J Epidemiol* 2007;36:834–40.
40 Montuschi P, Kharitonov SA, Barnes PJ. Exhaled carbon monoxide and nitric oxide in COPD. *Chest* 2001;120:496–501.
41 Yamaya M, Sekizawa K, Ishizuka S, *et al*. Increased carbon monoxide in exhaled air of subjects with upper respiratory tract infections. *Am J Respir Crit Care Med* 1998;158:311–14.
42 Togores B, Bosch M, Agustí AG. The measurement of exhaled carbon monoxide is influenced by airflow obstruction. *Eur Respir J* 2000;15:177–80.
43 Paredi P, Shah PL, Montuschi P, *et al*. Increased carbon monoxide in exhaled air of patients with cystic fibrosis. *Thorax* 1999;54:917–20.
44 Antuni JD, Kharitonov SA, Hughes D, *et al*. Increase in exhaled carbon monoxide during exacerbations of cystic fibrosis. *Thorax* 2000;55:138–42.
45 Zegdi R, Perrin D, Burdin M, *et al*. Increased endogenous carbon monoxide production in severe sepsis. *Intensive Care Med* 2002;28:793–6.
46 Zayasu K, Sekizawa K, Okinaga S, *et al*. Increased carbon monoxide in exhaled air of asthmatic patients. *Am J Respir Crit Care Med* 1997;156(4 Pt 1):1140–3.
47 Yamaya M, Sekizawa K, Ishizuka S, *et al*. Exhaled carbon monoxide levels during treatment of acute asthma. *Eur Respir J* 1999;13:757–60.
48 Bens BW, ter Maaten JC, Ligtenberg JJ. [Carbon monoxide poisoning after smoking from a water pipe]. *Ned Tijdschr Geneeskd* 2013;157:A6201.
49 Clarke SF, Stephens C, Farhan M, *et al*. Multiple patients with carbon monoxide toxicity from water-pipe smoking. *Prehosp Disaster Med* 2012;27:612–14.
50 La Fauci G, Weiser G, Steiner IP, *et al*. Carbon monoxide poisoning in narghile (water pipe) tobacco smokers. *CJEM* 2012;14:57–9.
51 Ozkan S, Ozturk T, Ozmen Y, *et al*. Syncope Associated with Carbon Monoxide Poisoning due to Narghile Smoking. *Case Rep Emerg Med* 2013;2013:796857.
52 Turkmen S, Eryigit U, Sahin A, *et al*. Carbon monoxide poisoning associated with water pipe smoking. *Clin Toxicol (Phila)* 2011;49:697–8.
53 Cobb CO, Sahmarani K, Eissenberg T, *et al*. Acute toxicant exposure and cardiac autonomic dysfunction from smoking a single narghile waterpipe with tobacco and with a "healthy" tobacco-free alternative. *Toxicol Lett* 2012;215:70–5.

54 Hawari FI, Obeidat NA, Ayub H, *et al*. The acute effects of waterpipe smoking on lung function and exercise capacity in a pilot study of healthy participants. *Inhal Toxicol* 2013;25:492–7.
55 Rammah M, Dandachi F, Salman R, *et al*. In vitro effects of waterpipe smoke condensate on endothelial cell function: a potential risk factor for vascular disease. *Toxicol Lett* 2013;219:133–42.
56 Ross R. Atherosclerosis--an inflammatory disease. *N Engl J Med* 1999; 340:115–26.
57 Danesh J, Collins R, Appleby P, *et al*. Association of fibrinogen, C-reactive protein, albumin, or leukocyte count with coronary heart disease: meta-analyses of prospective studies. *JAMA* 1998;279:1477–82.
58 Pradhan AD, Manson JE, Rossouw JE, *et al*. Inflammatory biomarkers, hormone replacement therapy, and incident coronary heart disease: prospective analysis from the Women's Health Initiative observational study. *JAMA* 2002;288:980–7.
59 Gan WQ, Man SF, Senthilselvan A, *et al*. Association between chronic obstructive pulmonary disease and systemic inflammation: a systematic review and a meta-analysis. *Thorax* 2004;59:574–80.
60 Yudkin JS, Kumari M, Humphries SE, *et al*. Inflammation, obesity, stress and coronary heart disease: is interleukin-6 the link?. *Atherosclerosis* 2000;148:209–14.
61 Vaddi K, Nicolini FA, Mehta P, *et al*. Increased secretion of tumor necrosis factor-alpha and interferon-gamma by mononuclear leukocytes in patients with ischemic heart disease. Relevance in superoxide anion generation. *Circulation* 1994;90:694–9.
62 Barnes PJ, Shapiro SD, Pauwels RA. Chronic obstructive pulmonary disease: molecular and cellular mechanisms. *Eur Respir J* 2003;22:672–88.
63 Wannamethee SG, Lowe GD, Shaper AG, *et al*. Associations between cigarette smoking, pipe/cigar smoking, and smoking cessation, and haemostatic and inflammatory markers for cardiovascular disease. *Eur Heart J* 2005;26:1765–73.
64 Cobb C, Ward K, Maziak W, *et al*. Waterpipe tobacco smoking: an emerging health crisis in the United States. *Am J Health Behav* 2010;34:275–85.
65 Martinasek MP, McDermott RJ, Martini L. Waterpipe (hookah) tobacco smoking among youth. *Curr Probl Pediatr Adolesc Health Care* 2011;41:34–57.