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To Evaluate the Effects of the Introduction of a Smoke-free Environment on the Lung Function of Bar Workers in Dublin

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To Evaluate The Effects Of

The Introduction Of a Smoke-Free Environment

On The Lung Function of Bar Workers in Dublin

By

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Abstract

Introduction It has long been recognised that exposure to environmental tobacco smoke (ETS) causes respiratory and cardio-vascular disease. A ban, prohibiting smoking in the workplace, was sanctioned by the Irish Government and came into effect on 29th March 2004. Bar staff were an ideal group to study the health effects of the introduction of this ban.

Methods Workers were recruited through their Trade Union, Mandate, and 81 participated in the pre-ban phase of testing between September 2003 and March 2004. They attended the Respiratory Laboratory in St. James's Hospital and underwent lung function tests and measurement of exhaled carbon monoxide (CO). They also completed a questionnaire relating to their respiratory health and personal smoking history. 75 (92.6%) returned one year later (6 – 11 months post ban) and repeated the tests and questionnaire.

Results 73 barmen were included in the analysis. 34 (47%) had never smoked, 31 (42%) were ex-smokers, and 8 (11%) were current smokers. After the introduction of the ban upper and lower airway symptoms were significantly reduced. Worker's exposure to ETS in work was reduced from an average of 40 hours per week to less than half an hour post ban (-99%, p=<0.01). Exhaled carbon monoxide (used as a marker of exposure) was reduced by a mean of 40% in the non-smoking barmen. 57 (88%) of non smokers reported the same or less exposure to ETS outside work. Tests to measure how well the lungs were working showed significant improvement in those barmen who never smoked, but showed deterioration in smokers.

Conclusion Overall, the workplace ban on smoking has shown an immediate effect on the respiratory health in non-smoking bar workers, with reduction in symptoms and CO levels, and better lung function results.

Declaration

I certify that this thesis which I now submit for the award of Master of Philosophy, is entirely my own work and has not been taken from the work of others save and to the extent that such work has been cited and acknowledged within the text of my work.

This thesis was prepared according to the regulations for postgraduate study by research of the Dublin Institute of Technology and has not been submitted in whole or in part for an award in any other Institute or University.

The work reported on in this thesis conforms to the principles and requirements of the Institute's guidelines for ethics in research.

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Signature Michèle Agnew **Date** 12/12/06

Candidate

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PREFACE

Results from this study have been presented to date as follows :

1) Analysis of 56 subjects presented as an oral presentation at the annual meeting of the European Respiratory Society, Copenhagen, September 2005.

2) Analysis of the results presented as an oral presentation at the Annual Scientific meeting of the Irish Institute of Clinical Measurement Science, Gleeson Hall, D.I.T., September 2005.

3) Final results presented in poster format at the annual meeting of the Irish Thoracic Society, Galway, November 2005. Boehringer award (grant) achieved, following review by Senior Respiratory Consultants, for 'Best clinical poster' at this meeting. Copy of poster in Appendix L, and letter of award in Appendix M.

4) Paper including the results of this study submitted to American Journal for Respiratory and Critical Care Medicine (AJRCCM), accepted for publication (awaited). Copy of paper in Appendix N.

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CHAPTER 1

INTRODUCTION

In 1994 the Irish government introduced laws prohibiting smoking in certain public places e.g. airlines, cinemas [1]. This was in an effort to reduce the levels of exposure to cigarette smoke for the non-smoking population and to reduce the risks to their health. However, this law failed to protect some workers who had long hours of exposure to secondhand smoke, and in 1999 and 2001 two all-party parliamentary committee reports recommended a total ban of smoking in all enclosed public places [2, 3].

1.1 AIM OF THE STUDY

The aim of this study is to evaluate the effects of a smoke-free working environment on the lung function and respiratory health of bar workers in Dublin pubs before and after the introduction of legislation outlawing smoking in the workplace [4].

1.2 BACKGROUND TO THE GOVERNMENT PLAN

As far back as 1986 the US Surgeon General proclaimed that passive smoking was a cause of disease, including lung cancer, in healthy non-smokers [5]. Since then, more evidence that environmental tobacco smoke (ETS) is harmful to health has become available to members of the international scientific community.

Increasing public awareness, along with reports of recent court cases in the United States, Australia and The Netherlands – where employees successfully sued their employers – puts pressure on governments to safeguard public health by introducing appropriate legislation to reduce risk of exposure to ETS in the workplace.

Previous Irish law, which prohibited or restricted smoking in most public places, served to protect some workers from passive smoking in the workplace [1]. However there are exemptions – restricted smoking is allowed in restaurants, trains and psychiatric hospitals; unlimited smoking was allowed in prisons, bookmakers, bars and nightclubs and many other workplaces.

Given the increasing concern about the health effects of ETS, the Health and Safety Authority and the Office of Tobacco Control commissioned an independent scientific working group to investigate the health risks posed by ETS in the workplace [6]. This group was to 'identify and report on the degree of consensus that exists amongst leading international scientific authorities on the question of the hazard and risk posed by ETS to human health in the workplace'.

Following this investigation carried out in 2002, the minister for Health and Children drafted legislation banning smoking in the workplace, including public houses, clubs and restaurants [4].

1.3 STUDY PLAN

This change in Irish law offered a unique opportunity to objectively investigate the effects of ETS on workers exposed to long hours of passive cigarette smoke in their workplace. Bar workers in the Dublin area were invited to attend the Respiratory Laboratory in St. James's Hospital for lung function evaluation and to complete a questionnaire on respiratory health and symptoms. These investigations were carried out before the introduction of the ban and again one year later, after the introduction of the ban.

Chapter 2 outlines the health effects of exposure to ETS and the reasons behind the government plan to eliminate ETS from the workplace.

Chapter 3 explains the lung function and exhaled carbon monoxide tests available in the Respiratory Laboratory and the equipment used to carry them out.

Chapter 4 outlines the study protocol used during this project.

Chapter 5 presents results of the tests carried out on the bar workers over the two periods of the study (pre- and post-ban).

Chapters 6 and 7 discuss these results and suggest some conclusions.

1.4 REFERENCES

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CHAPTER 2

ADVERSE HEALTH EFFECTS OF ETS EXPOSURE

2.1 ENVIRONMENTAL TOBACCO SMOKE

ETS is a major source of indoor pollution. It is the complex mixture of chemicals generated during the burning of tobacco products. The principal contributor to ETS is side stream smoke, the material emitted from the smoldering tobacco product between puffs. It is made up of over 4,000 chemicals in the form of particulates and gases, which are known to be harmful and can cause cancer [1, 2, 3, 4]. Many potentially toxic gases are present in higher concentrations in side stream smoke than in mainstream smoke (smoke that has been inhaled and then exhaled by the smoker) and nearly 85% of the smoke in a room results from side stream smoke [5]. The characteristics of ETS change as it ages and combines with other constituents in the ambient air.

The particulate phase includes tar (itself composed of many chemicals), nicotine, and benzene [6]. Nicotine is addictive. It stimulates the central nervous system, and in large quantities it is extremely poisonous. The gas phase includes carbon monoxide, ammonia, formaldehyde, and hydrogen cyanide. Some of these particulates and gases have marked irritant properties and some 60 are known or suspected carcinogens (cancer causing substances) [6].

Whenever a person smokes a cigarette, the chemicals, particularly nicotine and carbon monoxide, damage the cardiovascular system [6]. Nicotine causes both immediate and longer term increases in blood pressure, heart rate, cardiac output

and coronary blood flow. Carbon monoxide binds to the haemaglobin, which is what normally carries oxygen from the lungs via the bloodstream, and therefore reduces the amount of oxygen reaching body tissues [7]. Smoking also makes blood vessels and blood cells sticky, allowing cholesterol and other dangerous fatty material to build up inside them [8]. This is called atherosclerosis. This in turn can lead to raised blood pressure and clot formation.

While barworkers may not be active smokers, they are still at risk of damage caused by exposure to high levels of mainstream and side stream smoke in their working environment. Various factors, including the fact that side stream smoke is produced at lower temperatures than mainstream smoke mean that many carcinogens and other toxicants are generated in greater amounts in side stream smoke than in mainstream smoke [9].

A study from 1992 confirmed that second hand smoke tar stayed longer in the airways, suggesting that it penetrated more deeply into the lung, reaching much smaller airways, and takes longer to disappear from the exhaled breath [10]. Deeper penetration is made possible by the smaller particle size of second hand smoke. This may cause lasting damage to small airways, with nicotine penetrating the alveoli and reducing tissue available for transfer of oxygen, leading to lung disease including Chronic Obstructive Pulmonary Disease (C.O.P.D.) and emphysema. While health risks from passive smoking are less than those from active smoking, because the diseases are common, the overall health impact is large.

In 2002 the World Health Organisation (WHO) declared that passive smoke was a known human carcinogen. On foot of this they have recognised ETS as being equivalent to asbestos exposure and declared it to be a Category 1 carcinogen [6].

2.1.1 The effects of exposure to ETS

Some of the immediate effects of passive smoking include eye irritation, headache, cough, sore throat, dizziness and nausea. Adults with asthma can experience a significant decline in lung function when exposed. Short-term exposure to tobacco smoke also has a measurable effect on the heart in non-smokers – just 30 minutes exposure is enough to reduce coronary blood flow [11].

Passive smokers with long-term exposure suffer an increased risk of smoking related diseases. Non-smokers, exposed in the home, have a 25% increased risk of heart disease and lung cancer [12]. Workers exposed to these chemicals during working hours are at a higher risk of developing heart and lung disease than those working in a smoke-free environment [13]. Children exposed to ETS while in adult company, are at greater risk of developing lower respiratory tract infections e.g. bronchitis and pneumonia [14].

Passive smoking increases the risk of upper and lower respiratory tract illness but a smoke free environment improves all these disorders [15]. Ischaemic heart diseases and lung cancer are the main risks for non smoking adults exposed to cigarette smoke. Tobacco use and exposure is the single most important source of preventable morbidity, disability and premature mortality.

2.2 EARLY EVIDENCE OF DAMAGE CAUSED BY ETS

The respiratory consequences of involuntary exposure to tobacco smoke have not been studied as extensively in adults as in children. However the European Community Respiratory Health Survey, a study carried out between 1990 and 1994, obtained useful information in this respect [16]. A total of 7882, randomly selected never smokers, both male and female, aged 20 – 48 yrs, from 36 centres in 16 countries, had a structured interview concerning passive smoking, respiratory symptoms, asthma and allergic rhinitis. Forced expiratory volume in one second (FEV1) and forced vital capacity (FVC) were recorded, and a bronchial challenge test was performed to assess airway responsiveness. Total and specific serum immunoglobulin (Ig)E were measured.

The proportion of passive smokers among participants was variable, from 75% in Galdakao in Spain, with 53.8% workplace exposure, to 21.5% in Uppsala in Sweden, with 2.5% workplace exposure. In 12 of the 36 centres more than half the population reported that they were passive smokers. There was a positive correlation between the prevalence of current active smoking in the population and the prevalence of passive smoking. Some of the findings included a significant dose-related association with passive smoking for all respiratory symptoms, and a strong positive association between passive smoking in the workplace and asthma. No sex difference was observed in the association between passive smoking and any symptom, bronchial responsiveness, total serum IgE or lung function.

This large study concluded that passive smoking increased the likelihood of experiencing respiratory symptoms and is associated with increased bronchial responsiveness. The authors concluded that all measures limiting passive smoking, particularly in the workplace, could improve respiratory health in the community, and this study was the first to highlight this issue.

2.2.1 Risks of passive smoking

In July 1999, the Health and Safety Commission (UK) issued a draft Approved Code of Practice to clarify the implementation of the Health and Safety at Work Act as it applies to passive smoking in the workplace. The Act states that employers have a duty 'to provide and maintain a safe working environment which is, so far as is reasonably practicable, safe, without risks to health and adequate as regards facilities and arrangements for their welfare at work' Legal opinions argued that this could be interpreted as requiring employers to provide a smoke-free workplace. However, the U.K. has still to introduce a complete ban on workplace smoking.

2.2.2 Passive smoking related deaths

The Wanless report in the U.K. (2004) identifies smoking as the single greatest cause of preventable illness and premature death in the UK, and estimates that it kills 120,000 people per year in the UK (one fifth of all deaths) [17]. In addition, it reports that second hand smoking increases the risk of lung cancer by 20 – 30% for people who live with smokers, equivalent to several hundred deaths per year.

A report from ASH (UK) calculates that each year about 600 lung cancer deaths and up to 12,000 cases of heart disease in non-smokers can be attributed to passive smoking [18].

European Figures : In Norway it is estimated that 50 non-smokers die of lung cancer and 300 – 500 of heart disease each year as a result of long term exposure to environmental tobacco smoke [19].

In Finland, a study to estimate the mortality from exposure to passive smoking at work found that of the 250 fatalities to occur in 1996, 2.8% were from lung cancer, 1.1% from chronic obstructive lung disease, 4.5% from asthma, 3.4% from ischemic heart disease and 9.4% from cerebrovascular stroke [20]. The authors concluded that the magnitude of mortality related to past occupational exposure to passive smoking is considerable, and suggested that preventative measures to reduce environmental tobacco smoke in the workplace would be a powerful means of reducing the high burden of respiratory and cardiovascular disease.

Further Afield : In New Zealand, estimates state that 350 deaths are caused by exposure to second hand smoke each year [21]. These figures included 243 deaths from heart disease, 88 deaths from stroke and 7 deaths from lung cancer. Investigators reported the excess risk for lung cancer to be in the 20% - 30% range, while the risk for heart disease is approximately 20% - 25%. These deaths are seen as potentially avoidable and the authors conclude that second hand smoke exposure should be eliminated.

A study in Hong Kong (published January 2005), investigated the link between passive smoking in the home and increased risk of mortality [22]. Investigators looked at 4838 never smokers, and found significant dose-dependent association

between passive smoking and mortality from lung cancer, chronic obstructive pulmonary disease, stroke, ischemic heart disease, and from all cancers, all respiratory and circulatory diseases, and all causes. The association between mortality and passive smoking did not differ between males and females.

This international research suggests a definite link between increased risk of lung and cardiovascular disease and a high incidence of mortality in never smokers due to exposure to second hand smoke.

2.3 HEART DISEASE CAUSED BY ETS EXPOSURE

Cigarette smoke is harmful to your heart. It causes coronary vasoconstriction, increase in coronary vascular resistance and a decrease in coronary blood flow. The elastic properties of the aorta deteriorate with exposure to ETS, and there is a much higher risk of heart disease in those non-smokers exposed to ETS than in those with no exposure to ETS. Studies conclude that workers are at higher risk of heart disease if exposed to ETS in the workplace.

A possible relationship between passive smoking and coronary heart disease has been widely debated during the past decade. In the United States 37,000 coronary heart disease deaths per year are attributed to ETS exposure, accounting for 70% of all deaths caused by ETS [23], a similar figure to that of New Zealand [21].

A study from China [24] explored the link between passive smoking in work and the incidence of coronary heart disease amongst female workers who had never smoked. 59 patients with coronary heart disease and 126 controls (all Chinese women in full time jobs) were assessed. The investigators considered age, history of hypertension and cholesterol levels. Workplace exposure, and exposure from spouse was taken into consideration. The conclusion of this study was that passive smoking at work is a risk factor for coronary heart disease, and public health measures are needed to reduce smoking and to protect non-smokers from passive smoking.

The Department of Public Health in Helsinki estimated that acute events of coronary heart disease were increased by 25 – 35% by exposure to ETS [25]. Although the number of studies carried out in the workplace is small, they conclude that there is no reason to assume that the cardiovascular effects of ETS differ markedly between home and the workplace. They recommend that workers be protected from exposure to ETS.

These findings are similar to those of a review of heart disease in the workplace carried out in the US in 1997 [26]. Investigators assessed eight studies carried out in workplaces, and compared these with a study carried out on home exposure in 1994. They concluded that relative risks for heart disease from passive smoking at work are roughly equal to those from home-based exposure.

2.3.1 ETS exposure increases risk of heart attack and death in non-smokers

A study, carried out over a 10 year period in the US, investigated the risk of heart attack or death in more than 32,000 non-smoking women who were regularly exposed to passive smoke in the homes or in their workplaces [8]. It found that these women had a 91% higher risk of heart attack or death than those who are not exposed, (for those who only had occasional exposure to smoke, the increase in risk was 58%).

Similar findings were observed in the U.K. [27]. 2105 non-smokers in 18 towns were studied between 1978 and 1980. The men were followed for all-cause and cardiovascular mortality, and information on coronary heart disease events was obtained from General Practitioner (GP) reports and patients' records, through December 2000. Exposure to passive smoke was measured by serum cotinine levels. People who were non smokers but had relatively high levels of cotinine had a heart disease risk of about 50% higher than those people who were exposed to low levels.

The investigators concluded that this evidence should encourage the introduction of legislation to protect non-smokers from the risk of exposure to ETS in the workplace.

2.3.2 Cardiac damage caused by ETS exposure

Numerous clinical studies have demonstrated that cigarette smoking causes coronary vasoconstriction, an increase in coronary vascular resistance, and a decreased coronary blood flow, despite an increase in myocardial oxygen demand [28]. Cigarette smoking also increases diffuse or segmental coronary artery spasm. In habitual smokers, smoking one cigarette increases blood pressure, cardiac index, and myocardial oxygen demand and impairs cardiac performance probably through adrenergic stimulation and catecholamine release. Similar to active smoking, passive smoking has the same adverse effect on the cardiovascular system, with similar changes in haemodynamics and coronary vasomotor tone.

The elastic properties of the aorta deteriorate with exposure to ETS. A study in Greece studied the association between passive smoking and the elastic properties of the human aorta [29]. 16 male non-smokers were assigned to passive smoking study and 32 current, long-term male smokers were assigned randomly to active smoking study (16 subjects) or sham smoking study (16 subjects). In the passive smoking group ETS was vented into an exposure chamber for 5 minutes. Each participant in the active smoking group smoked one filtered cigarette. Each participant in the sham group performed a similar pattern of breathing with an unlit cigarette. Elastic properties were studied by measuring the aortic pressure-diameter relation before and for 20 minutes after passive, active or sham smoking. The results showed that both passive and active smoking were associated with changes in the aortic pressure-diameter relation (decreases of 21% and 27%

respectively). No changes in aortic elasticity were seen in the sham smoking group. The conclusion of this study was that both passive and active smoking are associated with an acute deterioration in the elastic properties of the aorta.

2.4 VASCULAR DISEASE CAUSED BY ETS EXPOSURE

2.4.1 Increased carotid wall thickness

A U.S. study investigated the relationship between active and passive smoking and increased carotid wall thickness [30]. 12,953 black and white women, aged 45 – 65 yrs were examined in the Atherosclerosis Risk in Communities Study. Three groups (current smokers (n=3525), ex smokers (n=4315), and never smokers (n=5113)) reported weekly exposure to ETS. Carotid artery intimal-medial thickness (IMT) was measured by B-mode ultrasound. The results showed that increased IMT was observed in each category order from smallest to greatest increase : never smokers not exposed to ETS, never smokers exposed, past smokers and current smokers. The larger IMT observed in the non-smoking group exposed to ETS compared with the non-smokers not exposed persisted after control for diet, physical activity, body mass index, alcohol intake, education and major cardiovascular risk factors. Among past and current smokers, increased pack years of exposure was associated with increased IMT (1 pack year is where a person smokes 20 cigarettes per day for one year, 40 cigarettes per day for a year would be equivalent to 2 pack years). Among non-smoking men exposed to ETS, there was a significant increase in IMT with increasing number of hours per week of ETS exposure. Investigators concluded that this data confirms a strong relationship

between active smoking and carotid IMT and provides initial evidence that passive smoking exposure is related to greater IMT. Increased exposure to cigarette smoke, either pack years of active smoking or hours of ETS exposure, was significantly related to increased IMT.

2.4.2 Increased risk of acute stroke

A group from Auckland, New Zealand planned to estimate the relative risk of stroke associated with exposure to ETS, and associated with current smoking [31]. They used three study groups – never smokers, ex smokers not exposed to ETS and active smokers. Cases were obtained from the Auckland stroke study, a population-based register of acute stroke. A standard questionnaire was administered to patients and controls by trained nurse interviewers. Information was available for 521 patients with first-ever acute stroke, and 1851 community controls aged 35 – 74 yrs. After adjusting for potential confounders (age, sex, history of hypertension, heart disease and diabetes) using logistical regression, exposure to ETS among non-smokers and long term ex-smokers was associated with a significantly increased risk of stroke. Active smokers had a four-fold risk of stroke compared with people who reported that they had never smoked cigarettes. The risk increased when active smokers were compared with people who had never smoked or had quit smoking more than 10 years earlier and who were not exposed to ETS.

This study confirms the higher risk of stroke in men and women who smoke cigarettes compared with non-smokers. The stroke risk increased further when

those who have been exposed to ETS are excluded from the non-smoking reference group. These findings also suggest that studies investigating the adverse affects of smoking will under-estimate the risk if exposure to ETS is not taken into account.

2.5 LUNG DISEASE CAUSED BY ETS EXPOSURE

The evidence from epidemiological studies, studies of biochemical markers of exposure, and toxicological studies, confirm that there is a causal association between the risk of lung cancer and exposure to environmental tobacco smoke. In epidemiological studies of women who are lifelong non-smokers, there is a 24% higher risk of lung cancer from exposure to environmental tobacco smoke from the spouse, and this increases with the number of cigarettes smoked and duration of marriage [32].

2.5.1 Lung function reduction with ETS exposure / smoking

Studies have been carried out to evaluate the damage caused by smoking and most of these have shown reduced spirometric values, and have concluded that smoking reduces lung function. In China, a study compared spirometry in 180 non smokers and 131 smokers between the ages of 20 and 78 [33]. Investigators found that in current smokers and ex-smokers the Forced Expired Volume in the first second of expiration divided by the Forced Vital Capacity (FEV1/FVC) was significantly lower than that of non-smokers. No significant difference was found in FVC and FEV1. They felt that a low FEV1/FVC appeared to be the earliest discriminatory index in normal asymptomatic smokers, and concluded that cigarette smoking is

associated with a decrease in lung function although the effects take some time to develop. They added that there is an irreversible decrease in FEV1/FVC with cumulative cigarette consumption, but smoking cessation will prevent further deterioration in FEV1 and FVC.

The Scottish MONICA (from MONItoring CArdiovascular disease) study, was part of a much larger World Health Organisation (WHO) sponsored project which studied 170,000 heart attacks around the world over a 10 year period to get an accurate picture of cardiovascular disease levels and trends [34].

This Scottish limb of the study investigated the relation between lung function in workers and exposure to ETS at work and elsewhere. A total of 301 never smokers attended for administration of a health survey questionnaire (including exposure to ETS), lung function tests and serum cotinine levels.

The results showed that both men and women suffered effects on FEV1 and FVC from exposure to ETS, with higher exposure resulting in lower lung function. Investigators concluded that the exposure-response relation shows a reduction in pulmonary function of workers associated with passive smoking, mainly at work, and state that these findings endorse current policies of strictly limiting smoking in shared areas, particularly in the workplace.

Other studies have shown that stopping smoking can reduce or stop deterioration in lung function [35]. A study from Minnesota carried out a prospective trial at 10 medical centres on 3926 smokers with mild-moderate airway obstruction. They

measured lung function annually for 5 years, and found that participants who stopped smoking experienced an improvement in FEV1 in the year after quitting. The subsequent rate of decline in FEV1 among sustained quitters was half the rate among continuing smokers, comparable to that of never smokers. The authors concluded that smokers with airflow obstruction benefit from quitting despite previous heavy smoking, advanced age, poor baseline function or airway hyper-responsiveness.

A study from Arizona looked at the effect of smoking and smoking cessation on the CO diffusing capacity in asymptomatic subjects [36]. The single breath CO diffusing capacity was measured along with standard spirometry as part of a survey of a randomly selected community population sample. Based on answers to a self-administered questionnaire, subjects free of respiratory symptoms or disease were identified. Data from subjects who had never regularly smoked cigarettes were used to derive reference equations for the test variables, and data from the remaining subjects who had smoked were examined to determine the effect of smoking and smoking cessation on the test. From this analysis investigators found that cigarette smoking is associated with a decrease in diffusion that occurs very soon after beginning the smoking habit. They concluded that there is an irreversible decrease in diffusion with cumulative cigarette consumption, but also a reversible phenomenon that leads to rapid improvement in diffusion on smoking cessation.

Comment

Exposure to ETS, whether at home or in the workplace, causes lung disease. Although some of these study figures are estimates, there can be little argument with the fact that exposure to ETS causes higher risk of mortality and lung cancer, a reduction in FEV1 and FVC in non-smokers, and that smoking causes reduction in gas exchange. This is reason enough to support the elimination of ETS in the workplace to safeguard workers, both non-smokers and smokers.

2.6 BAR WORKERS EXPOSURE AND RISKS

Waiters, and bar workers, a group exposed to high levels of ETS in the workplace, have been shown to have an increased risk of death due to lung cancer [37]. Bars and nightclubs are environments where ETS levels are considerably higher than the norm, and as such, place workers at risk. Jarvis et al. measured salivary cotinine levels in 42 non-smoking subjects from 27 pubs in central London and Birmingham during the summer of 1990 [38]. They found the mean concentration was 9.28ng/ml and the median was 7.95ng/ml. They compared these findings with results previously achieved in adults and children in the UK, and they found that cotinine levels in bar staff are about double those in children with two smoking parents, and are about four times higher than adults reporting recent exposure.

Subjects	Exposure indicator	Median salivary cotinine (ng/ml)
Adults	Working in pubs	7.95
7yr olds	No smokers in household	0.20
	1 smoker in household	1.80
	2+ smokers in household	4.40
11 – 16yr old		Neither parent smoke 0.50
	Father smokes	1.35
	Mother smokes	2.15
	Both parents smoke	3.70
Hosp out-pts		No exp in past 3 days 0.50
	Some exp in past 3 days	1.65

Table 2.1 Cotinine levels in children and adults in the U.K. [from Jarvis et al., ref 38]

In another study, Laranjeira et al. studied 100 non-smoking waiters and compared them with 100 non-smoking medical students during working hours [39]. They measured expired carbon monoxide (CO) levels in both groups before and after a working shift. The pre-exposure CO levels were similar in both groups, but after a mean 9 hours exposure in the workplace, median levels more than doubled (2ppm vs. 5ppm (P=<0.001)) in the waiters. The CO levels correlated with the number of tables available for smokers.

In 1993 Michael Seigel completed a literature search 'to determine the relative exposure to ETS for bar and restaurant employees compared with office employees

and with non-smokers exposed in the home (part 1), and to determine whether this exposure is contributing to an elevated lung cancer risk in these employees (part 2)' [40]. He studied reported levels of CO, nicotine and suspended particles for bars, restaurants, offices and residences, and averaged them in part 1. In part 2 he looked at the relative lung cancer risk for food service workers compared with that for the general population in six identified studies.

His results showed that levels of ETS in restaurants were approximately 1.6 to 2 times higher than in office workplaces, and 1.5 times higher than in residences with at least one smoker. Levels in bars were 3.9 to 6.1 times higher than in offices and 4.4 to 4.5 times higher than in residences. The epidemiological evidence suggested that there may be a 50% increase in lung cancer risk amongst food service workers that is in part attributable to ETS in the workplace.

Seigel concluded that ETS is a significant occupational health hazard for food service workers, and to protect these workers smoking in bars and restaurants should be prohibited.

It has been shown that bar and restaurant workers exposed to ETS in their place of work have a higher incidence of respiratory and irritation symptoms compared to a comparable group who have no workplace exposure [41]. This particular study by Bates et al. published in 2002 examined workers exposure to ETS during a work shift. A questionnaire was also completed by each participating subject. 44 bar

and restaurant workers were compared with 51 government employees (who worked in a smoke free workplace).

The results showed that hospitality workers in bars/restaurants allowing smoking by customers had significantly greater increases in cotinine than workers in smoke-free premises. The investigators concluded that there was a clear association between within-shift cotinine concentration change and smoking policy. They also noted that workers in premises permitting customers to smoke reported a higher incidence of respiratory and irritation symptoms than workers in smoke free workplaces. Concentrations of salivary cotinine found in exposed workers in this study have been associated with substantial involuntary risks for cancer and heart disease.

Fidan et al. reported similar findings in Turkey [42]. Coffee house workers were questioned about their respiratory symptoms and carried out spirometry, and the results were compared with workers from small-scale shops in the same area. In Turkey coffee houses are large rooms with tables and chairs, with a small kitchen area in one corner and are typically located in the basement floor of buildings. Most men spend a considerable part of the day in them, especially in the lower socio-economic neighbourhoods, and they are more densely located where unemployment is higher. Smoking prevalence is high in Turkish men (50 – 60%), and the coffee houses would have very high levels of ETS.

In this study 207 workers were assessed. The results showed that there was a significant increase in respiratory symptoms in these workers, and spirometry values were lower in this group also. Dyspnoea, phlegm, wheezing and coughing,

as well as chronic bronchitis, were all seen more frequently in the coffee house workers compared to the other occupational groups. In addition, chronic bronchitis was seen more among the coffee house workers, especially in the age group over 40 yrs. With regard to spirometry, FEV1 and FVC were reduced in the people exposed to ETS both at home and in work environment. There was a 10% decrease in FEF 25-75 in the coffee house workers, which was significantly lower than the other groups, raising the possibility that ETS exposure has an effect on the small airways. Investigators concluded that working in a coffee house not only results in respiratory symptoms and mild pulmonary function changes, but also significant obstructive airway disease and recommended that banning smoking in these coffee houses should be considered to prevent ETS exposure as a public health priority, both for the customer, but more so for workers.

A study published in 2002 claims that 150 Irish bar workers will die every year from ill health due to passive smoking [43]. James Repace, an American expert on ETS claimed that 'more people died in 2002 from passive smoking at work in the UK than were killed by the Great London smog of 1952'. The study, carried out by the Western Health Board, found that not only were bar ventilation systems unable to maintain ETS at low levels, but levels were dangerously high in two venues. In addition, bar workers were found to be exposed for long periods of passive smoking, with 40% of respondents working in bars for 10 years or more and declaring that they were exposed on average for 40 hours per week to ETS.

A report commissioned by the Cancer Council of New South Wales and conducted by Repace, estimated mortality from second-hand smoke among bar and hospitality workers in New South Wales [44]. This report was based on hours worked, number of smokers in the premises, number of cigarettes smoked, levels of air pollution, and comparison with the number of respiratory deaths from passive smoking in the U.S.A.

Mechanism	**Ave. number per year (rounded)**
Being hit by moving objects	37
Falls	14
Contact with electricity	10
Vehicle accident	12
Drowning	4
Chemicals and other substances	3
ALL	**97**
Secondhand smoke	***73-97 (estimated)***

Table 2.2 Comparison of estimated deaths due to passive smoking in the workplace and the number of recorded workplace deaths in other industries. [from Repace, ref 44]

From this, we can see that the author estimates that ETS related deaths are equal to all other workplace related deaths. As a result of this report, it was concluded that a total smoking ban extending to hotels, clubs, and nightclubs is justified.

All of these studies show strong evidence of the dangers to bar and hospitality workers, employed in establishments that permit smoking by customers, and some

also suggest that the longer workers are exposed to ETS, the worse their respiratory health is. A strong dose-related exposure risk is suggested, and should be kept in mind when considering that our bar workers work an average of 40 hours per week in this dangerous atmosphere, with some having worked in the bar trade for more than 50 years.

There is enough evidence now to encourage governments world-wide to put in place laws which forbid smoking in enclosed workplaces, thus eliminating exposure of non-smoking workers to the dangers of ETS during working hours. All workers are entitled to a clean working environment, and the Irish ban on smoking should improve the long-term respiratory health of our population.

2.7 MECHANISMS TO REDUCE ETS EXPOSURE IN THE WORKPLACE

Following the basic laws of physics, ETS rapidly diffuses throughout a room. At a ventilation rate of one air change per hour it can take more than three hours for 95% of the smoke in a typical room to be removed once smoking has ended. This indicates that using ventilation to eliminate ETS in indoor spaces presents a considerable if not impossible task to ventilation engineers [45]. Air quality may not be the same throughout a ventilated space. What really counts for the occupants of an indoor space is the air quality. All ventilation does is improve the subjective quality of the air (by giving the sense of air movement in a room) and dilute rather than remove pollutants. Ventilation may remove the smell of smoke but not the dangerous toxins.

In most areas ventilation standards are voluntary and designed for comfort, not for safety. Many studies have shown that ventilation systems are usually not well maintained, making them less likely to be effective [46]. Higher ventilation rates, which are noisy and cause discomfort, would be required for them to be effective [47].

There has been some discussion about filtration of air, but air filtration or air ionising equipment can only remove visible particles, it is not effective in removing invisible and highly toxic gases[48]. This type of equipment also clogs up quickly and requires a very high level of maintenance.

2.7.1 Separate smoking areas

In establishments where smokers are segregated from non-smokers, pollution levels may be slightly reduced, but tobacco smoke drifts and staff will still have no choice but to inhale ETS [49]. An extensive study carried out in Sydney in 2003 looked at the efficacy of designated 'no-smoking' areas in the hospitality industry as a means of providing protection from ETS [50]. A total of 17 social and gaming clubs agreed to participate. These clubs were licensed to serve alcohol and, apart from designated areas, smoking was permitted. Measurements were taken of atmospheric nicotine, particulate matter, and carbon dioxide in general use areas and in the designated 'no-smoking' areas during hours of normal operation, and in the open air.

The results showed that by comparison with levels in general use areas, nicotine and particulate matter levels were significantly less in the 'no-smoking' areas, but were still readily detectable at higher than ambient levels.

The authors concluded that provision of designated 'no-smoking' areas in licensed clubs provides only partial protection from ETS – typically about 50% reduction in exposure. The protection afforded is less than users might reasonably have understood and is not comparable with protection afforded by prohibiting smoking on the premises.

The authors stated that the only certain method of eliminating the health risks from second-hand smoke to all workers is a law to ensure completely smoke-free workplaces. This is also the easiest to implement and least expensive method.

2.8 RESPIRATORY DISEASE IN IRELAND

Ireland has a poor record for lung diseases - whether it is the damp weather conditions or the historic poor economic climate, diseases of the respiratory system are one of the main causes of death in this country. The two main lung diseases caused by smoking and exposure to ETS are COPD and lung cancer. Lung cancer is the biggest killer of all the cancer groups, accounting for about 20% of all cancer deaths, and cigarette smoking is estimated to be responsible for approximately 90% of lung cancer cases.

The European Lung White Book (the first comprehensive survey on respiratory health in Europe) published in 2003[51], places Ireland fourth in a list of standardised mortality rates of chronic obstructive lung disease for males in 1990. The four leading nations are Kyrgyzstan, Ukraine, Kazakhstan, and Ireland. It states that mortality rates are 2 – 3 times higher in males than in females. This is not a record we should be proud of.

2.9 GOVERNMENT INTERVENTION TO REDUCE LUNG DISEASE

For Ireland to improve its record of lung disease, something radical had to be done. Following the report commissioned by the government and the Office for Tobacco control [52], the Minister for Health and Children, Micheal Martin, drafted the legislation banning smoking in enclosed workplaces, including pubs, clubs and restaurants. Following some amendments, and much opposition from the hospitality industry, this legislation became law on 29th March 2004.

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CHAPTER 3

LUNG FUNCTION MEASUREMENT AND EQUIPMENT

3.1 ROUTINE LUNG FUNCTION MEASUREMENTS

In the Respiratory Laboratory at St. James's Hospital, routine evaluation of lung function consists of tests to measure the mechanical properties of the lung (spirometry), tests to assess lung volumes (Residual Volume and Total Lung Capacity), and tests to assess how efficiently the lung parenchyma absorbs oxygen from the air and transfers it to the blood (DLCO). These tests are carried out using highly complex equipment and require the full co-operation of the patient/subject. The tests are carried out by fully qualified clinical measurement scientists, and during this study all tests were carried out by a single experienced operator. With good subject co-operation, a complete set of tests takes approximately 30 minutes to carry out.

3.2 THE NORMAL LUNG

The main function of respiration is to provide oxygen to the cells of the body and to remove excess carbon dioxide from them [1]. Oxygen is taken up by the blood in the pulmonary circulation and carbon dioxide released simultaneously from the blood into the alveoli. Respiring tissues need a continuous supply of oxygen and constantly produce carbon dioxide. In humans this gas exchange system has been divided into two subsystems – the lungs and pulmonary circulation, which forms the external respiratory system and the cells which form the internal respiratory system. However, a number of accessory structures are necessary to enable the

primary function of respiration to be achieved. The respiratory system consists of the external nose, internal nose and paranasal sinuses, the pharynx, larynx, trachea, bronchi and the lungs.

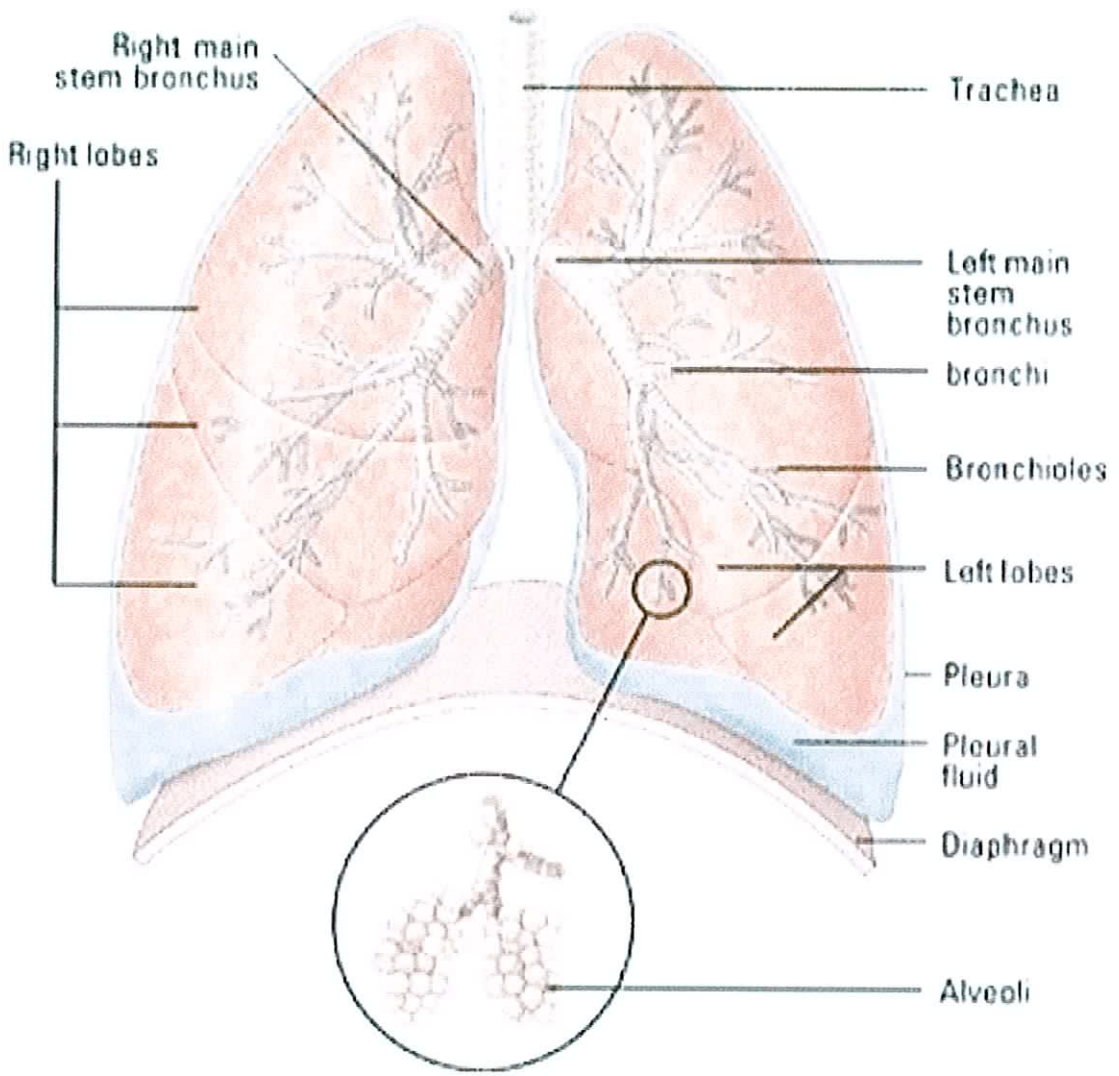

Figure 3.1. The Lungs (***picture taken from www.lungusa.org)***

3.2.1 Topography of the lungs

The lungs are a pair of cone shaped organs lying in the thoracic cavity and separated from each other by the mediastinum – this is a space in the centre of the thoracic cavity extending form the sternum to the vertebrae. The lungs are separated from the abdominal cavity by the diaphragm. The right lung is usually somewhat shorter than the left and is made up of three lobes – upper, middle and lower. The left lung is narrower than the right due to a depression in its surface to accommodate the heart, and is made up of two lobes – upper and lower.

When we breathe in, air enters through either the nose or mouth and travels along the pharynx and larynx and into the trachea. Inspired air is warmed in the nasal cavity and foreign particles are filtered by nasal mucosa. After bifurcation of the trachea into the right and left main bronchi, each of these subsequently branch so that each lobe has its own air supply. Here the airways become collectively known as the conducting airways and comprise of the bronchi and bronchioles. These airways do not take part in gas exchange, but deliver air to the alveoli which do. The tracheobronchial tree is made up of successive generations of air passages from the trachea (generation 0) to the alveolar sacs (generation 24). The number of passages in each generation is double that in previous generation. Alveolar sacs are the last generation of air passages and differ from other air passages in that they are blind ending, and an adult human has about 200 – 600 million alveoli (depending on height).

Alveolar cell walls are extremely thin and have a massive surface area to allow rapid transfer of gas to and from the pulmonary circulation. As the pulmonary circulation is in very close contact with the alveoli, this makes this function easier.

3.2.2 Features of the respiratory system

The respiratory system is such that a) there is a large surface area for gas exchange that separates blood from the air, b) the barrier separating the blood from the air is thin, providing minimal resistance to gas transfer and c) the flow of oxygen across the barrier separating the blood from air occurs by diffusion down a pressure gradient from a point of high pressure to a point of low pressure.

Breathing is spontaneously initiated within the central nervous system. A cycle of inspiration and expiration is automatically generated by neurons located in the brainstem. This system can be modified, altered, or temporarily suppressed by a number of mechanisms. The system controlling breathing regulates a complex series of usually complimentary, but occasionally competitive or even incompatible activities. However, the system must perform three main functions : 1. Maintain, through involuntary controls, a regular rhythmic breathing pattern. 2. Adjust the tidal volume and breathing frequency such that alveolar ventilation is sufficient to meet the demands for gas exchange at cellular level. 3. Adjust the breathing pattern to be consistent with other activities using the same muscles, such as speech. Under most circumstances, breathing is controlled so finely that the oxygen and carbon dioxide are kept within normal limits. The metabolic controller keeps most control, but occasionally a behavioural controller overrides the metabolic controller

to allow for such activities as talking, swallowing and coughing to break through the normal pattern of breathing.

3.2.3 Respiratory Muscles

The primary respiratory muscles are the diaphragm and intercostals accessory muscles of the thoracic wall. Additional muscles of the upper airway are also involved. The muscles are well innervated by the somatic (motor) nervous system and have no inherent rhythm. They generate tension due to a rhythmic pattern of neuron-induced action potentials activating them.

3.2.4 Control of breathing

The motor neurons innervating the muscles of the upper airway are located in the medulla and innervate the muscles of the upper airway and bronchi through the cranial nerves. The muscles are activated just before the major muscles of inspiration are activated resulting in dilatation of these airways before inspiration. Dilatation during inspiration is important as the inspiratory flow rate depends on the force generated by the inspiratory muscles and the resistance and elastance of the system. The overall efficiency of the system is enhanced by the abduction of the vocal folds and the consequent decrease in airway resistance.

In expiration, airflow depends on the recoil and mechanical properties of the system. The duration of expiration is greater that the time required for the passive collapse of the system to functional residual capacity (FRC). In spontaneous, quiet

breathing, expiratory flow is slowed by post inspiratory contraction of the inspiratory muscles and an increase in upper airway resistance due to partial closure of the laryngeal airway. The braking mechanisms are important, as without them the expiratory flow would be higher with possible consequences for both gas exchange and control of breathing.

3.2.5 Chemoreceptor Control

Alveolar ventilation is controlled so that the alveolar pressure of carbon dioxide ($PaCO_2$) is maintained within a narrow band of variation and may be regarded as constant. So tight is this control that the $PaCO_2$ does not deviate from its normal value of 5.3 kiloPascals (kPa) by more than 0.3 kPa (5%) for more than a few minutes, even under differing conditions. The central chemoreceptors are involved in the second by second control of ventilation. They are located in the central nervous system or close to the ventrolateral surface of the medulla and are near to, but separate from the brain stem controller. The response of these receptors is simple. An increase in hydrogen ion (H^+) concentration stimulates ventilation and a decrease inhibits it. Thus oxygen from the atmosphere must be delivered to the alveoli and carbon dioxide must be removed. Ventilation maintains the optimal composition of alveolar gas and facilitates gas exchange. Failure of any part of the system, due to either structural or functional failure, results in the system failing. Therefore each part of the system is dependent on the other parts of the system.

The combination of an efficient external gas exchanger, a developed circulatory system and the operational capabilities of the mitochondria in the cells results in a system that can convert oxygen and carbohydrates into energy needed to meet the demands of daily living.

3.2.6 The aging process

As we get older, our lung function declines naturally due to the deterioration in the tissues of the lung, a reduction in muscle strength and a decrease in the compliance of the thoracic cage. With the deterioration in lung tissue the elastic recoil of the lung decreases but the chest wall becomes more rigid. Total lung capacity (TLC) does not alter with increasing age, whilst FRC and residual volume (RV) increase and hence vital capacity (VC) must decrease.

3.2.7 Pulmonary Defence Mechanism

The lungs are in direct contact with the environment. We breathe at about 6 – 8 litres per minute at rest, which increases during exercise. Thus, during a 24 hour period, our lungs are exposed to about 11,500 litres of air. This air may contain infectious micro-organisms, hazardous dusts and/or chemicals.

Exposure to contaminants of ambient air can lead to a diversity of disorders. In the workplace, exposure to hazardous dusts such as asbestos or coal dust may occur. While it is possible to provide protection against these substances, exposure nevertheless occurs. Exposure may also be voluntary, such as cigarette smoking, or involuntarily through breathing contaminated air in the workplace or outdoors.

3.2.8 Deposition of particles

Inhaled particles are deposited within the airways by impaction, sedimentation and diffusion [1]. In addition, turbulence and to a minor degree electrostatic forces also play a part [1]. Airway reflexes influence actual deposition and removal of particles. These mechanisms determine the probability of an inhaled foreign particle touching the surface of the respiratory tract, and being held by it. The actual concentration of the particles is unimportant, as each particle will behave independently of the others.

Where a particle is deposited depends on a variety of factors. The size of the tidal breath and the breathing frequency are important. The rate of airflow will determine the type of deposition. With high flows, impaction is more likely to occur, while at lower flows, sedimentation and diffusion are more likely to occur. At rest, particles larger that 10μm are deposited in the nasopharynx, where the flow rate is high, by inertial impaction. Particles of 2 to 10μm impact in the large intrathoracic airways, with particles of 2μm accounting for about 20% of the removed particles. As gas velocity decreases, sedimentation becomes important for the filtration for particles between 0.2 to 5μm. Overlapping with both impaction and sedimentation is diffusion. About 15% of 0.1μm particles will be deposited by diffusion, with the remainder being suspended in the alveolar air and exhaled from the mouth or nose.

3.3 PREDICTED / REFERENCE VALUES

In order to be able to assess the subject's test results, we have to have some values to compare them with to decide if they are normal or abnormal. Predicted lung function values are those derived from studies of a normal, non-smoking population, i.e. those of similar ethnic background and without lung disease. All respiratory laboratories in Ireland currently use the European Respiratory Society (ERS) 1993 (update) normal values, and these are the predicted values that are used for reference in this study. Rather than using absolute numbers e.g. 2.4 litres, percent of predicted was used e.g. 85% predicted, to compare change in function over the two phases of the study. This was done because it better reflects objective change – the barworkers were one year older in the post-ban phase of the study, and their lung function would be expected to deteriorate slightly over that period due to the ageing process. Their predicted values would therefore be slightly lower in the post-ban study, but the value of e.g. vital capacity as a percent of the predicted may remain the same or even increase.

Parameters used to determine a subject's predicted values are height, age and sex. Race is also taken into account, as some ethnic groups are of smaller stature than Europeans e.g. Asians, and these can have lung volumes that are up to 15% less, but as all the barmen in this study were Irish, no change in predicted values had to be made due to race. Before starting the testing process, the subject is accurately measured on a height/weight scale and details are inputted into the equipment

database along with date of birth and sex, and values relevant to each individual are stored to compare test results to.

3.4 BREATH CARBON MONOXIDE MEASUREMENT

A test of exhaled carbon monoxide (CO) was carried out on the bar workers in the lab. This was done using a Micro Medical Micro CO meter. This is a small, hand-held, battery operated device used to measure the concentration of CO in the expired breath and it calculates the percentage of carboxyhaemaglobin (%COHb) in the blood. It is accurate and easy to use.

3.4.1 What is Carbon Monoxide?

Carbon Monoxide (CO) is a colourless, odourless, tasteless and toxic gas produced by incomplete burning of organic substances [2]. It is easily absorbed into the body when breathed into the lungs. When it is inhaled, it combines with the oxygen-carrying haemaglobin of the blood to form carboxyhaemaglobin (COHb). Once combined with the haemaglobin, that haemaglobin is no longer available for transporting oxygen. The effect of CO exposure is to reduce the amount of oxygen available to the tissues of an exposed person. How quickly the COHb builds up is a factor of the concentration of the gas being inhaled (measured in parts per million or PPM) and the duration of the exposure.

Compounding the effects of the exposure is the long half-life of COHb in the blood. The half-life of COHb is approximately 5 hours. This means that for a given

exposure level, it will take about 5 hours for the level of COHb in the blood to drop to half its current level after exposure is terminated.

% COHb	Symptoms and medical consequences
10%	No symptoms. Heavy smokers can have as much as 12% COHb
15%	Mild headache
25%	Nausea and serious headache. Fairly quick recovery after treatment with oxygen and/or fresh air
30%	Symptoms intensify. Potential for long term effects, especially in the case of children and the elderly
45%	Unconsciousness
50% +	Death

Table 3.1 Symptoms associated with a given concentration of COHb ***(table taken from Micro Medical Micro CO manual)***

3.4.2 Equipment - Micro CO meter

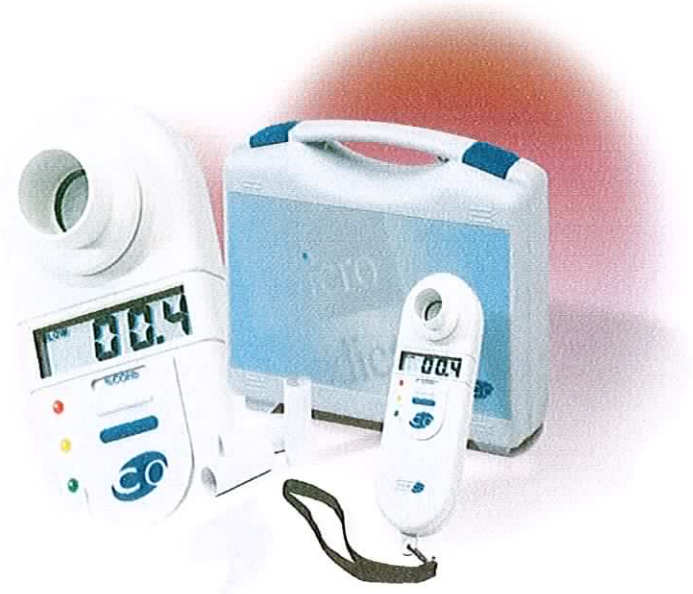

Fig. 3.2. Micro CO meter ***(Picture taken from www.micromedical.co.uk)***

The MicroCO meter is based on an electrochemical fuel cell sensor, which works through the reaction of CO with an electrolyte at one electrode, and oxygen (from ambient air) at the other. This reaction generates an electrical current proportional to CO concentration. Output from the sensor is monitored by a microprocessor, which detects peak expired concentrations of alveolar gas. This is then converted to % carboxyhaemaglobin (%COHb) using the mathematical relationships described by Jarvis et al [3], for concentrations below 90ppm, and by Stewart et al [4] for higher levels. The results are displayed on a clear LCD display. Warning lights are provided to give an instant indication of the smoking level.

3.4.2(1) CO levels and Smoking

Measurement of carboxyhaemaglobin has been well validated as an indirect measure of cigarette consumption and is widely used in smoking cessation programmes [5].

CO (ppm)	%COHb	Cigarette Consumption
0 – 5	0 – 0.8	Non-smoker
6 – 10	1 – 1.6	Light smoker
11 – 72	1.8 – 12	Heavy smoker
> 72	> 12	Suspected poisoning

Table 3.2 Typical values for carboxyhaemaglobin and expired CO (*taken from Micro Medical Micro CO manual)*

3.4.2(2) Operation

The meter is stored and used at room temperature for accurate results. The subject inhales maximally, holds their breath for 20 seconds, and then exhales slowly and completely into the machine. The 20 second breath hold time is to allow time for equilibration of alveolar gas. However if the subject cannot hold their breath for the required 20 seconds, they may blow out earlier. Before repeating a measurement, the unit is turned off, and the mouthpiece and adaptor removed for at least one minute. This is to allow re-equilibration with ambient air and to dry the surface of the sensor.

3.4.2(3) Calibration

The manufacturer states that calibration will remain stable to within 2% over one month and typically to within 10% over 6 months. The machine used during the

study was calibrated on purchase, and again prior to commencing the post-ban testing phase.

Machine specifications are in Appendix A

3.5 EQUIPMENT – SENSORMEDICS VMAX SYSTEM

Fig. 3.3 Sensormedix Vmax system (*Photo taken in St. James's Respiratory Laboratory)*

The Sensormedics Vmax system (Sensormedics, USA) is the system used to measure routine lung function - spirometry, static lung volumes and gas transfer factor for patients attending the respiratory laboratory in St. James's Hospital. It measures both inspired and expired flow directly using a Mass Flow Sensor.

The computer program electronically integrates these flow signals to obtain volume measurements. The sensor uses a pair of heated stainless steel wires to measure gas flow. The rate at which heat is lost from the heated wires when they are exposed to a gas flow (i.e. inspired and expired patient gas) is directly related to the flow rate of gas across the wires. More specifically, the amount of heat extracted from the wires is proportional to the mass of the individual gas molecules flowing across them.

The Mass Flow Sensor is impervious to water vapor. There is also automatic compensation for ambient temperature changes and gas temperature changes over a wide temperature range. The system is very stable. As a maintenance procedure, a self-cleaning function is built into the sensor. There is also an automatic zero flow calibration of the sensor.

The Vmax system uses a Multi-gas Analyser to provide real-time measurement of Carbon Monoxide (CO) and Methane (CH4). In order to measure multiple gases in a single sample, the analyser incorporates an assembly with multiple filters and detectors - the wavelength of each band is carefully chosen to measure only the desired gas.

The Oxygen analyser used in the Vmax system is based on high-sensitivity paramagnetic technology, providing the fast response time necessary for real-time breath-by-breath gas analysis. The Mass Flow Sensor is calibrated daily using a 3

liter syringe, and the gas analysers are calibrated each morning and also between tests using computer software.

Machine specifications are in Appendix B

3.6 LUNG FUNCTION TESTS

3.6.1 Spirometry

Spirometry was carried out on each subject in accordance with European Respiratory Society (ERS) guidelines for spirometry [6]. The volunteer was seated comfortably, and the test was performed using a Sensormedics Microgard single-use patient filter. The subject was instructed for test performance and demonstration was given if required. The subject was encouraged verbally throughout the test and allowed rest briefly between efforts. Parameters measured included Forced Expired Volume in the first second of expiration (FEV1-measured in litres), Forced Vital Capacity (FVC-measured in litres), the ratio between these two measurements (FEV1/FVC%), Peak Expiratory Flow (PEF-measured in litres per minute), and mid section flow rates (FEF25%, FEF75%, FEF 25 – 75%, FEF50%, FIF50%-all measured in litres per second). The test was completed a minimum of three times and the highest values from each effort were reported.

3.6.2 Parameters measured during spirometry testing [2]

1) **Forced Expired Volume in the first second of expiration (FEV1).** This is the volume of air exhaled in a specified time from the start of the forced vital capacity manoeuvre; conventionally the time used is one second, hence FEV1. It is

expressed in litres, at body temperature and ambient pressure saturated with water vapour (BTPS).

2) **Forced Vital Capacity (FVC).** This is the volume of air delivered during an expiration made as forcefully and completely as possible, starting from full inspiration. It is measured in litres, at BTPS.

3) **Peak Expiratory Flow (PEF).** This is the maximum flow during a forced expiratory vital capacity manoeurve, starting from a position of full inspiration, measured in either litres per second, or litres per minute, at BTPS.

4) **Forced Expiratory Flow between 25 – 75% of vital capacity (FEF 25-75).** This is the mean expiry flow between 25% and 75% of the vital capacity, and is also known as the maximum mid-expiratory flow. This index is taken from the blow with the largest sum of FEV1 and FVC. It is highly dependent on the validity of the FVC measurement and the level of expiratory effort.

3.6.3 Lung Volumes

Static lung volumes were performed on all subjects in accordance with ERS guidelines [6], using the Nitrogen Washout method on the Sensormedics Vmax system. The subject was seated comfortably, and a single-use Sensormedics Microgard filter was used. The subject was instructed as to test performance and was 'talked through' the test. Results were evaluated at the end, and if required, a

second test was performed, and the highest value was reported. Parameters measured included Residual Volume (RV), Total Lung Capacity (TLC) and the ratio between the two (RV/TLC%).

3.6.4 Parameters measured during lung volumes [2]

1) **Residual Volume (RV).** This is a measure of the air remaining in the lung at the end of a full expiration, and is measured in litres.

2) **Total Lung Capacity (TLC).** This is the total amount of air in the lungs at the end of a full inspiration, and is measured in litres.

3.6.5 Diffusing Capacity (DLCO)

Gas transfer (diffusing capacity) was carried out on all volunteers in accordance with ERS guidelines [3]. The test was performed twice, with a gap of at least 4 minutes between tests, and the mean of the two values reported. The system gas analysers were calibrated before each test was performed. The volunteer was seated comfortably and a Sensormedics Microgard single-use patient filter was used. The test was explained before being carried out, and the subject was 'talked through' the test.

The four-minute break between tests is to allow for residual gas to be cleared from the lungs prior to the start of the next effort. Parameters measured included the Diffusion in Litres of Carbon Monoxide (DLCO), Diffusion in Litres of Carbon

Monoxide, taking into account the amount of alveolar volume available for diffusion (DLCO/VA), and later these values were corrected for the amount of Carbon Monoxide in the subject's exhaled breath.

3.6.6 Parameters measured during Diffusing Capacity [3]

1) Diffusion in litres of Carbon Monoxide (DLCO). This is the absolute value of gas transfer recorded during the 10 second breath hold. By dividing it by the alveolar volume (DLCO/VA) one can judge how efficiently the available alveolar tissue is functioning.

2) Diffusion in litres of Carbon Monoxide corrected for percent carboxyhaemaglobin (DLCO corrected). Because some of the barmen attending the laboratory had quite high levels of expired CO (smokers), the DLCO was corrected for the % Carboxyhaemaglobin (COHb) – the amount of carbon monoxide combining with oxygen in the blood, derived from the CO measurement taken during the expired CO manoeuvre. By doing this, they were all being leveled off to a similar level of %COHb, and thus eliminating DLCO reduced because of higher levels of CO in the system.

The equation used to do this is:

$$\textbf{Measured DLCO} \times \left(1.0 + \frac{\%COHb}{100}\right) = \textbf{corrected DLCO}$$

3.6.7 Peak flow monitoring

To measure diurnal variability in lung function, the bar workers were given a portable peak flow meter to analyse their peak flow measurements over a one-week period, both in work and at home. They were instructed in the test performance and a baseline value was recorded before leaving the laboratory. A diary card to cover the week (***Appendix C***) was explained to them and they were instructed to complete the test three times per day (three efforts per time, record the highest) – mid morning (approx 11am), mid afternoon (approx 3pm), and in the evening (approx 8pm) and record their peak flow (PEF) value in litres per minute. They were also instructed to record the times of their work shift on that day for correlation. They were given a stamped addressed envelope and asked to post the peak flow meter and diary card back to the laboratory as soon as they had completed the week's recordings.

3.6.7.(1) Piko meter

Fig. 3.4 Piko 1 Peak Flow meter (***Picture taken from www.ferrariscardiorespiratory.com)***

Peak flow was measured using a Piko 1 peak flow meter (Ferraris Medical, UK). This is a battery-operated digital meter, and is designed as an asthma/COPD-management tool and for single patient use. It records FEV1 and PEF and is capable of storing the last 96 tests for download. Reference values can be set on the meter for each individual subject, and warning lights indicate abnormalities or poorly carried out tests, but this facility was not used during this study.

Machine specifications are in Appendix D

3.7 REFERENCES

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CHAPTER 4

STUDY METHODOLOGY

When devising the protocol for the study, the objective was to recruit as many as possible working in bars or nightclubs who were exposed on a regular basis to high levels of ETS. A letter of invitation was written by me and sent to 1100 Mandate members in the Dublin area through the Mandate secretariat, with a cover note from the union President. All those who volunteered by phoning the respiratory laboratory (n=81) were accepted for testing, and subjects were accepted right up until the introduction of the anti-smoking legislation. Bar workers attended the respiratory laboratory in St. James's Hospital to carry out lung function tests and to complete a questionnaire. The hospital is a more controlled environment for performing tests, and a single operator was used to carry out lung function tests, while a separate single operator carried out the questionnaire. Baseline tests were completed between September 2003 and March 2004, before the ban came into effect, and were repeated one year later between September 2004 and February 2005.

Bar workers were first tested while they were still working in an environment with high levels of ETS, and then when the air in the bars was ETS-free. To allow for seasonal variation, tests were repeated at the same time of the year i.e. if the pre-ban tests were carried out in October, then the post-ban tests were carried out in the October of the following year. Also, in as much as possible, tests were carried out

at the same time of day at each visit. However, bar workers were requesting appointments on their days off, so this was not possible in every case.

4.1 STUDY PLAN

Bar workers came to the respiratory laboratory in St. James's Hospital to undergo a series of lung function tests and to complete a questionnaire before the ban was introduced. They were also given a small portable meter to keep a record of peak flow, three times daily, for a one week period, both at home and during working hours. This peak flow analysis was to investigate if there was any fluctuation in peak expiratory flow while the workers were in work (exposed to ETS) or at home (and possibly not exposed to ETS).

The pre-ban tests took place between September 2003 and March 2004. During this time 81 bar workers were studied. The ban was introduced on Monday 29th March 2004.

The post-ban phase of the study commenced in September 2004, and all bar workers were invited back to the respiratory laboratory for repeat testing. Again, bar workers were given suitable appointments to attend for tests, and asked to record peak flow for a one-week period. Two 'prizes' of 500 euro gift tokens were offered by Mandate as an incentive to those completing the study, and a 200 euro gift token for Power City was offered by the respiratory lab to encourage participants to return. The identification numbers of the 75 barmen who completed the study were put into a hat, and a draw for the prizes was made in the Mandate

head office by study/union personnel. The three winners were contacted by the Mandate secretary, and details of the draw was published in the union newsletter. Data collection was completed in February 2005.

4.2 ETHICS SUBMISSION AND APPROVAL

In April 2003 an application form and study protocol was submitted to the St. James's Hospital and Federated Dublin Voluntary Hospitals Joint Research Ethics Committee, based in Tallaght hospital (Adelaide and Meath hospitals incorporating the National Children's Hospital). This covered project supervision arrangements The proposal (***Appendix E***) was discussed by the Committee and ethics approval for the study was granted on 18.7.2003 ***(Appendix F).***

4.3 SUBJECT CONSENT

The subject consent form template from the ethics application was used for the study. Prior to its completion by the subject an explanation sheet detailing the study was given to them to read and further explanation was given if required. Two copies of the consent form were signed by the subject and two witnesses. One was given back to the subject, and the other was kept in their file.

(Appendix E).

4.4 RECRUITMENT OF SUBJECTS

Subjects were recruited through the trade union (Mandate). Letters of invitation, with a covering letter from the Mandate President, were sent to all Dublin-based

union members detailing the study and encouraging them to participate. There was a lot of ill-feeling from the Hospitality industry and the Vintners Association (who initiated a very strong 'anti-ban' lobby), because they felt that there would be a significant number of jobs lost as a result of the ban, and subsequent closure of premises. Irish Times newspaper headlines covering articles about Vintners disapproval are in ***appendices G, H, I.*** Due to the issues involved, some bar workers felt under pressure from their employers not to participate, and initially were slow to come forward. However, letters were continually sent and volunteers were enrolled until the ban came into effect.

In total 81 bar workers volunteered for the study. They were all male. From conversation, most felt that the ban was a positive thing, but some had reservations about job losses and therefore were not entirely in favour of it.

4.5 QUESTIONNAIRE

The questionnaire used in this study was similar to the one used in the Eisner study [1]. Respiratory symptoms were assessed with 5 questions from the International Union Against Tuberculosis and Lung Disease (IUATLD) Bronchial Symptoms Questionnaire [2]. The questions related to wheezing, dyspnoea, morning cough, cough during the rest of the day or night, and phlegm production. The IUATLD questionnaire has been validated against the criterion of bronchial hyper-responsiveness [2,3]. The questionnaire was modified to relate to symptoms during the past 4 weeks, rather than the past 12 months. In addition to the IUATLD

questions, sensory irritation symptoms were also assessed, which can result from ETS-related noxious stimulation of upper respiratory tract and corneal mucous membranes [4]. Personal, active cigarette smoking was measured using questions developed for the National Health Interview Survey [5]. Other questions evaluated ETS exposure duration in work, home, and other settings during the previous 7 days (in hours per week).

Using a question from the National Health and Nutrition Examination Survey (NHANES), subjects were assessed as to whether they had physician-diagnosed asthma and if they were currently taking any medication for this [6].

Finally, demographic information was collected, including age (date of birth), sex, home address and contact telephone number, place of work, number of years in current job, total number of years working in pubs, and a General Practitioner name and address for sending results at end of study.

The questionnaire took approximately 15 minutes per subject on average, but some took up to 25 minutes to complete it fully. The questionnaire was completed at both visits (pre and post ban).

A copy of the questionnaire is in ***Appendix J***

4.6 LUNG FUNCTION MEASUREMENT

The lung function of the barmen was measured according to ERS guidelines, as described in chapter 3, and reported in the format shown in ***Appendix K***. From previous studies [7], assessment of FEV1 and FVC showed that these measures were lower in smokers than in non-smokers. A study carried out in California [1] showed changes in spirometry post smoking ban and this study may show similar results.

Gas transfer measurement (Diffusing Capacity) is an indicator of damage to lung parenchyma, particularly in smokers and those exposed to ETS. Oxygen absorption is reduced by high levels of carbon monoxide in the system, so this value was important in monitoring change in lung absorption rates. Because of the high levels of CO in some smokers, the DLCO was adjusted for percentage carboxyhaemaglobin (COHb%), and changes were analysed following this adjustment. As the barworkers were all one year older in the post-ban phase of the study, values as 'percentage of predicted' were used to allow for the natural deterioration expected with age.

CO is a good measure to determine exposure to ETS, and so this was used in this study to assess change in the exposure levels of the barworkers. It is a simple and accurate test and yielded important information.

4.7 PEAK FLOW MONITORING OUTSIDE THE RESPIRATORY LABORATORY

The volunteers were instructed how to carry out the peak flow measurement before they left the Respiratory Laboratory at their hospital visit. They were given a diary card to record peak flow values at 11am, 3pm, and 8pm each day for one week, and noting their working hours on each particular day. When they were leaving the laboratory they were given a stamped addressed envelope to return the meter and diary card to the respiratory laboratory at the end of the week of recording. There were problems with these measurements, and this is discussed in a later chapter.

4.8 STATISTICAL ANALYSIS

The traditional approach to reporting a result required you to say whether it is statistically significant or not. To do this one generates a 'p value' from test statistics. 'P' is short for 'probability'. One indicates a significant result with '$p<0.05$'. This means that the probability of getting this result is less than 5% in the general population, and is therefore not just 'by chance'.

When looking at data in this study, three types of analysis were used. The questionnaire data, where subjects reported the absence or presence of a symptom, were analysed using McNemar's non-parametric test for two dichotomous variables for changes in responses using the chi-squared distribution. As the data for exhaled breath Carbon Monoxide exhibited skewed distribution (the non smokers had much lower CO levels than the current smokers), a non-parametric test

(Wilcoxon Signed Rank) was applied to test any significant differences between the pre and post ban levels. Lung function result numbers (using the percentage of predicted) were analysed using the paired T-test procedure comparing the means of the quantitative pairs of variables using SPSS software. The relevance, or 'statistical significance' of all this analysis was recorded using p-values.

Statistical analysis advice was sought from Kathleen Bennett and Zoubeir Kabir from Trinity College on the questionnaire and CO measurement data, and the lung function data I analysed myself using paired-T test method.

4.9 REFERENCES

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CHAPTER 5

RESULTS AND ANALYSIS

5.1 SUBJECT DEMOGRAPHICS

In the pre ban phase of the study, 81 bar workers attended the Respiratory Laboratory and carried out exhaled Carbon Monoxide (CO) assessment, a questionnaire, and lung function tests. All subjects were male, with a mean age of 46.9yrs (range 22 – 68yrs). Between them, they had 2298 yrs of exposure to ETS in their place of work (mean 28.4yrs) (range 6 – 52yrs). Some 10 were current smokers, 34 were ex-smokers and 37 had never smoked. A total of 10 had been told that they had asthma by a physician.

Following the introduction of the workplace smoking ban, 75 subjects completed the study. Two of these were excluded from analysis as their smoking status had changed during the year – one had started smoking again, and one had given up.

For analysis of the final data (pre and post-ban), the breakdown is as follows :

Total : 73 (100%) all male.

Mean age of those for analysis = 48.9yrs (range 22 – 68 yrs).

Mean Body Mass Index (BMI) Pre ban 28.4, post ban 28.5.

9 (12%) had been told that they had asthma by a physician in the past.

5.1.1 Work history

The barworkers work long hours in the public houses, with the mean being 40.78 hrs per week ((range 4* – 75hrs) *just back from holidays, so very short working

week on this occasion). The 73 bar workers for analysis had worked in the bar environment for a total of 2213.5 years, mean = 30yrs (range 7 – 53yrs).

5.1.2 Smoking History

34 (47%) had never smoked.
31 (42%) were ex-smokers (2 – 36 yrs duration).
8 (11%) are current smokers (8 – 42 yrs duration).

Table 5.1 Smoking history of 73 barworkers completing study *(data from questionnaire)*

While in the respiratory laboratory barmen were asked whether they were smoking less as a result of the workplace ban on smoking, and while exact number of cigarettes smoked was not assessed, six of the eight smokers claimed to be smoking less, while two said that their smoking had not changed.

5.1.3 Exposure to ETS

	TOTAL	MEAN	RANGE
PRE BAN	2954	40.5	4.0 - 75
POST BAN	30.75	0.42	0 - 5

Table 5.2 Hours (per week) exposed to ETS in the workplace *(data from questionnaire)*

	TOTAL	MEAN	RANGE
PRE BAN	3420	46.85	4 - 100
POST BAN	345	4.7	0 - 60

Table 5.3 Hours (per week) exposed to ETS in all places (inc. work) *(data from questionnaire)*

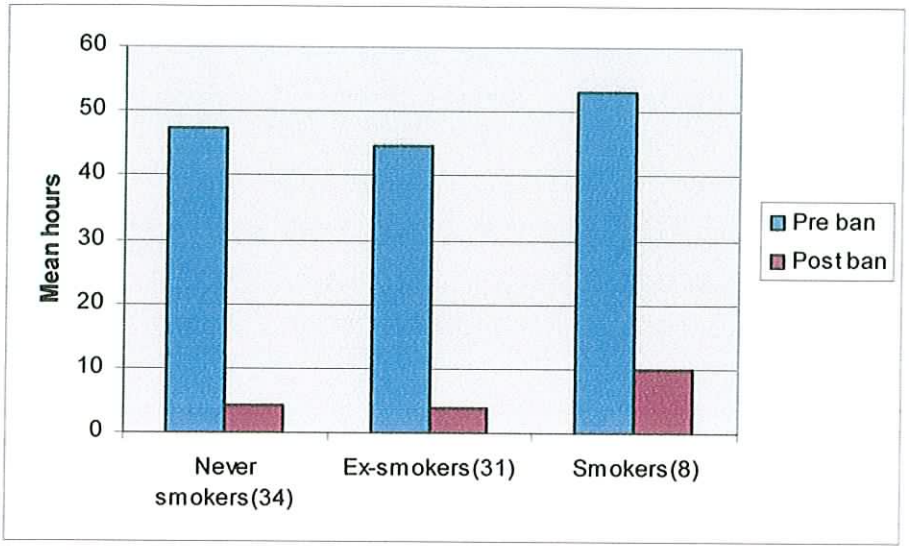

Fig. 5.1 Total hours exposed to ETS – both work/outside work

Outside workplace exposure to ETS

19 (26%) of total (N=73) barworkers reported living with a smoker.

5 (15%) of never-smokers reported living with a smoker.

11 (35%) of ex-smokers reported living with a smoker.

3 (38%) of current smokers reported living with a smoker.

	TOTAL	MEAN	RANGE
PRE BAN	466	6.4	0 - 70
POST BAN	314.25	4.3	0 - 60

Table 5.4 Hours (per week) exposed to ETS outside the workplace ***(data taken from questionnaire)***

Of the 65 non-smoking barworkers, 8 reported increased exposure outside work in the post ban phase, and 57 reported the same or less exposure outside work in the post ban phase.

5.2 MEASUREMENT AND ANALYSIS OF EXHALED CO

Measurement of exhaled CO was carried out at the pre and post-ban phases of this study, as described previously. Barworkers were asked to inhale fully, hold their breath for 20 seconds, and then exhale slowly into a CO meter. Results were recorded as CO in parts per million (ppm), and %COHb was estimated by machine software. Both numbers were recorded for analysis. The results are in table 5.5. Typical values for CO are on page 53 (table 3.2) for comparison.

	Pre ban	Post ban	% change	p value
Never smokers (34)	4.21	2.5	-41	0.0001
Ex-smokers (31)	4.84	2.9	-40	0.0001
Smokers (8)	17.6	16.1	-8.5	0.623

Table 5.5 Mean CO levels in barworkers (measured in ppm)

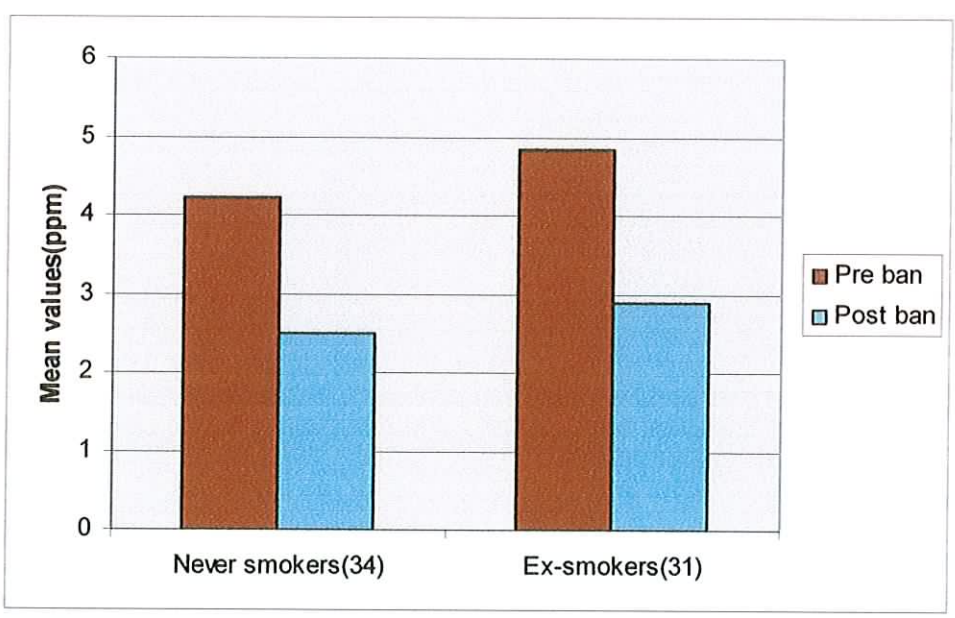

Fig. 5.2 Changes in CO levels in non-smoking barmen

However, analysis was also carried out to compare those living with a smoker, and those not living with a smoker, to assess changes in those with outside workplace exposure to ETS, and those only exposed in the workplace. The results are presented in table 5.6.

	Pre ban	Post ban	% change	p value
Never smokers WITH home exposure(5)	4.4	3	-41	0.08
Never smokers WITHOUT home exposure(29)	4.17	2.48	-40	**<0.001**
Ex smokers WITH home exposure(11)	4.82	3.34	-28	0.268
Ex smokers WITHOUT home exposure(20)	4.85	2.6	-46	**<0.001**

Table 5.6 Comparison in CO levels measured in ppm – those with home exposure v. those without home exposure

Although the reduction in CO is greater than 25% in all groups, the most significant changes are in those with least exposure to ETS, ie. those who have no home exposure.

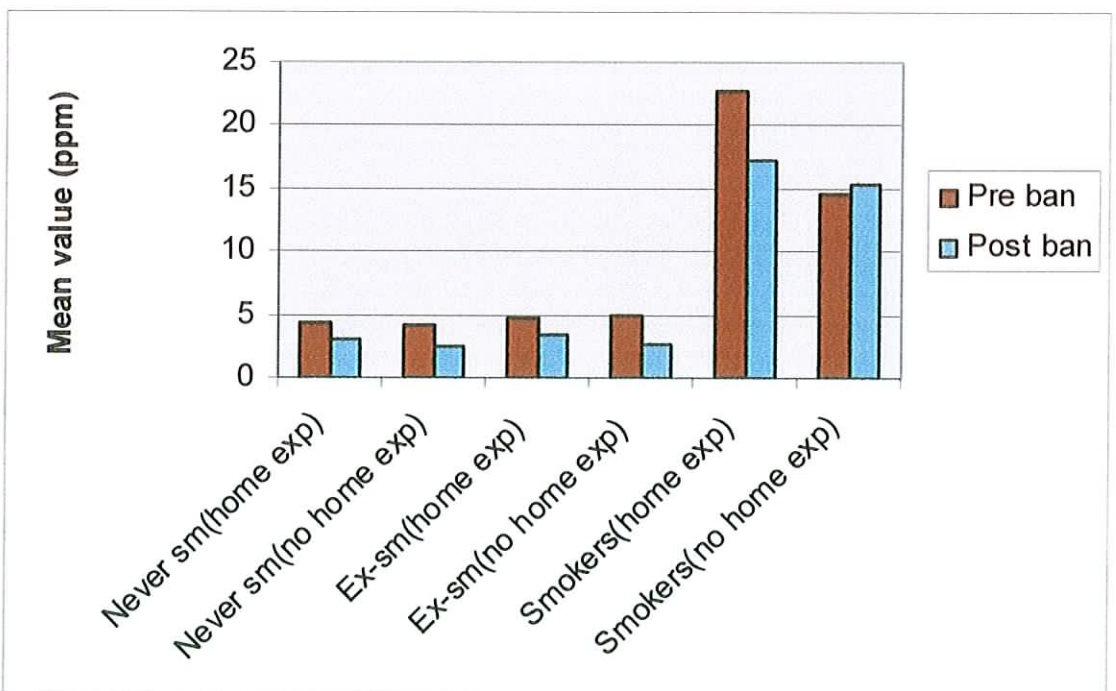

Fig. 5.3 Comparison of CO levels pre and post smoking ban

5.3 QUESTIONNAIRE ANALYSIS

5.3.1 Reporting of respiratory symptoms

The subjects were asked questions relating to breathing symptoms they may have experienced in the four weeks prior to their hospital visit (full questionnaire in *Appendix J)*

Before the ban 63 barmen (86%) reported one or more symptom, with only 10 barmen (14%) reporting no breathing symptoms during the previous 4 weeks.

After the introduction of the ban 45 barmen (62%) reported one or more symptom, with 28 barmen (38%) reporting no breathing symptoms during the previous 4 weeks. This is a decrease of 28% in reporting of respiratory symptoms

RESPIRATORY SYMPTOMS

	Pre ban	Post ban	% Change
Total symptoms	No. reporting	No. reporting	
0	10 (14%)	28 (38%)	180%
1	14 (19%)	13 (18%)	-7%
2	16 (22%)	10 (14%)	-37.5%
3	18 (24%)	13 (18%)	-27.8%
4	8 (11%)	6 (8%)	-25%
5	7 (10%)	3 (4%)	-57%

Table 5.7 Change in reporting of respiratory symptoms

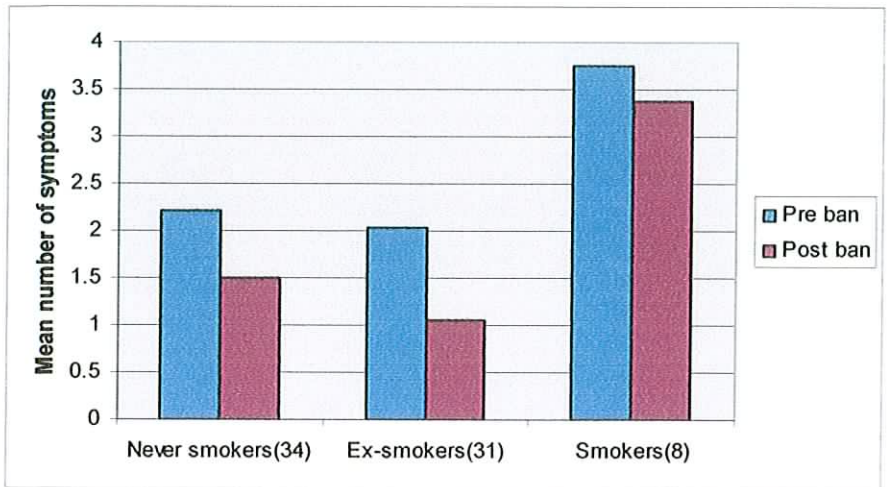

Fig. 5.4 Respiratory symptoms before and after introduction of the ban

Individual question analysis of respiratory symptom questionnaire

	% reporting symptom			
	Pre ban	Post ban	% change	p value
Never smokers (34)	35	21	-42	0.18
Ex-smokers (31)	19	26	33	0.754
Smokers (8)	75	63	-17	1

Table 5.8 Q1. relating to whistling/wheezing in the chest

	% reporting symptom			
	Pre ban	Post ban	% change	p value
Never smokers (34)	26	24	-11	1
Ex-smokers (31)	29	6	-78	0.16
Smokers (8)	50	38	-25	1

Table 5.9 Q2. relating to short of breath

	% reporting symptom			
	Pre ban	Post ban	% change	p value
Never smokers (34)	35	24	-33	0.344
Ex-smokers (31)	29	10	-67	0.109
Smokers (8)	75	75	0	1

Table 5.10 Q3. relating to early morning cough

% reporting symptom				
	Pre ban	Post ban	% change	p value
Never smokers (34)	53	32	-39	0.092
Ex-smokers (31)	58	35	-39	0.118
Smokers (8)	88	88	0	1

Table 5.11 Q4. relating to cough during the rest of the day

% reporting symptom				
	Pre ban	Post ban	% change	p value
Never smokers (34)	71	50	-29	**0.039**
Ex-smokers (31)	65	29	-55	**0.007**
Smokers (8)	88	75	-14	1

Table 5.12 Q5. relating to phlegm production

Analysis was conducted on individual questions to assess whether some symptoms responded better to reduction in ETS exposure than others. In the non-smoking group, early morning cough and production of phlegm responded well, while in the smokers, who had a higher incidence of respiratory symptoms at baseline, there was no significant difference in respiratory symptoms after the introduction of the ban.

5.3.2 Reporting of irritant symptoms

The subjects were asked about eye, nose and throat irritation they may have experienced in the four weeks prior to their visit to the hospital.

Before the ban 64 barmen (88%) reported one or more eye, nose and throat symptom, with only 9 barmen (12%) reporting no symptoms in the 4 weeks prior to the study.

After the introduction of the ban 32 (44%) reported one or more eye, nose or throat symptom, with 41 barmen (56%) reporting no symptoms in the 4 weeks prior to the

study. This was a decrease of 50% in reporting of upper airway irritation following introduction of the ban.

	Pre ban	Post ban	% Change
Total symptoms	No. reporting	No. reporting	
0	9 (12%)	41 (56%)	356%
1	22 (30%)	20 (27%)	-9%
2	27 (37%)	10 (14%)	-63%
3	15 (21%)	2 (3%)	-86.7%

Table 5.13 Change in reporting of irritant symptoms

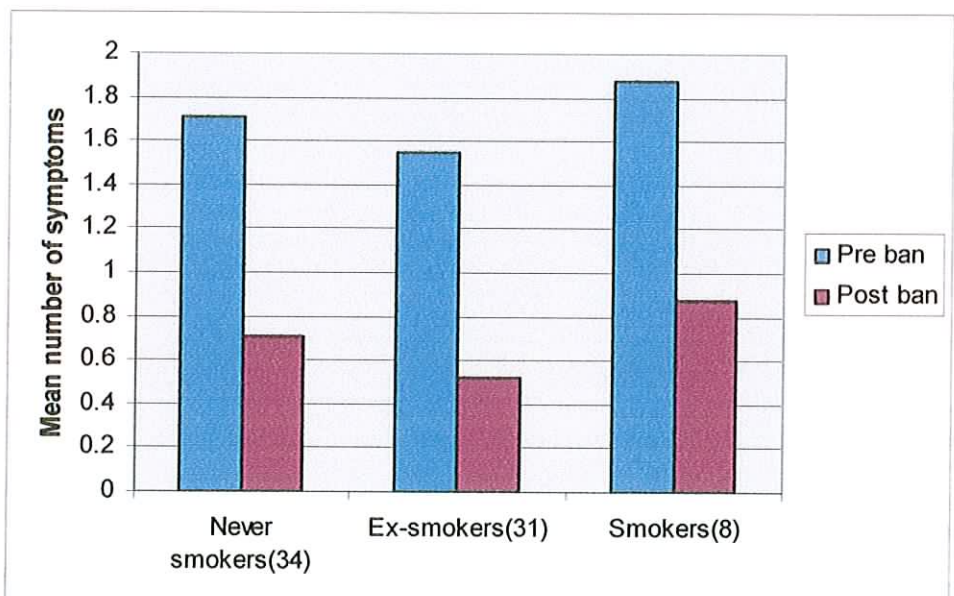

Fig. 5.5 Irritant symptoms before and after introduction of the ban

Questions were individually analysed. The results were as follows :

	% reporting symptom			
	Pre ban	Post ban	% change	p value
Never smokers(34)	59	15	-75	**<0.001**
Ex-smokers(31)	68	6	-90	**<0.001**
Smokers(8)	38	13	-67	0.5

Table 5.14 Q.1 relating to red/irritated eyes

	% reporting symptom			
	Pre ban	Post ban	% change	p value
Never smokers(34)	65	32	-50	**0.001**
Ex-smokers(31)	39	29	-25	0.581
Smokers(8)	100	50	-50	0.5

Table 5.15 Q.2 relating to runny nose, sneezing, or nose irritation

	% reporting symptom			
	Pre ban	Post ban	% change	p value
Never smokers(34)	47	18	-63	**0.006**
Ex-smokers(31)	48	16	-67	**0.013**
Smokers(8)	50	25	-50	0.5

Table 5.16 Q.3 relating to sore or scratchy throat

Eye, nose and throat (irritation) symptoms decreased significantly in almost all groups as you would expect following the introduction of the smoking ban, but the smokers continued to experience some eye irritation and sore/scratchy throat after introduction of the ban, possibly due to exposure to their own cigarette smoke. As the smoker group was small (8) it was difficult to find any statistically significant changes in them.

5.3.3 Assessment of asthmatic subjects

Asthmatic subjects were assessed separately to determine whether they fared better or worse than those who did not suffer from asthma. The 73 subjects were split into a further two groups (1) Asthmatics (n=9), and (2) Non-asthmatics (n=64).

The results are in tables 5.17- 5.24.

Respiratory symptoms in asthmatic v. non-asthmatics

	% reporting symptom			
	Pre ban	Post ban	%change	p value
Asthmatics(9)	78	44	-43	0.25
Non-asthmatics(64)	27	25	-6	1

Table 5.17 Q.1 relating to wheezing/whistling in the chest

	% reporting symptom			
	Pre ban	Post b	%change	p value
Asthmatics(9)	56	56	0	1
Non-asthmatics(64)	27	13	-53	0.078

Table 5.18 Q.2 relating to shortness of breath

	% reporting symptom			
	Pre ban	Post ban	%change	p value
Asthmatics(9)	44	22	-50	0.625
Non-asthmatics(64)	36	23	-35	0.115

Table 5.19 Q.3 relating to early morning cough

	% reporting symptom			
	Pre ban	Post ban	%change	p value
Asthmatics(9)	89	67	-25	0.5
Non-asthmatics(64)	55	36	-34	0.036

Table 5.20 Q.4 relating to cough during the rest of the day

	% reporting symptoms			
	Pre ban	Post ban	%change	p value
Asthmatics(9)	89	67	-25	0.5
Non-asthmatics(64)	67	41	-40	0.001

Table 5.21 Q.5 relating to phlegm production

Irritant symptoms in asthmatic v. non-asthmatics

Subjects were also asked about eye, nose and throat irritation during the past four weeks and the results are as follows:

	% reporting symptom			
	Pre ban	Post ban	%change	p value
Asthmatics(9)	78	33	-57	0.125
Non asthmatics(64)	57	8	-86	<0.001

Table 5.22 Q.1 relating to red or irritated eyes

	% reporting symptom			
	Pre ban	Post ban	%change	p value
Asthmatics(9)	89	56	-38	0.375
Non-asthmatics(64)	53	30	-44	0.003

Table 5.23 Q.2 relating to runny nose, sneezing, or nose irritation

	% reporting symptom			
	Pre ban	Post ban	%change	p value
Asthmatics(9)	56	22	-60	0.25
Non-asthmatics(64)	47	19	-60	<0.001

Table 5.24 Q.3 relating to sore or scratchy throat

5.4 LUNG FUNCTION TEST ANALYSIS

All lung function tests were performed in the respiratory laboratory In most cases tests were done in the same month as the pre ban phase to rule out seasonal changes.

5.4.1 Spirometry analysis

The groups were analysed as before – never smokers, ex-smokers, and current smokers. The percentage of predicted was used as the value of choice to analyse absolute change, as this takes into account the fact that the barmen are one year older in the post ban phase.

Some noticeable changes were found.

	PRE BAN	POST BAN	%change	p value
FEV1	3.44	3.49		
%PRED	92	94	2	0.123
FVC	4.17	4.36		
%PRED	91	96	5	0.0001
RATIO	82	80		
PEF	506.6	530	4.6	
%PRED	94	99.5	5.9	0.009
FEF25-75	3.68	3.41		
%PRED	89	83	-6	0.039

Table 5.25 Spirometry results for Never Smokers (N = 34)

	PRE BAN	POST BAN	%change	p value
FEV1	3.38	3.35		
%PRED	93	93	0	0.596
FVC	4.18	4.29		
%PRED	93	96	3	0.012
RATIO	81	78		
PEF	505.7	515	1.8	
%PRED	96	98	2.1	0.148
FEF25-75	3.42	3.11		
%PRED	87	79	-9	0.003

Table 5.26 Spirometry results for Ex-Smokers(N = 31)

	PRE BAN	POST BAN	%change	p value
FEV1	3.51	3.32		
%PRED	88	84	-5	0.276
FVC	4.45	4.31		
%PRED	91	88	-2	0.547
RATIO	79	76		
PEF	489.1	481.3	-1.6	
%PRED	86.4	85	-1.6	0.757
FEF25-75	3.41	3.2		
%PRED	78	73	-5	0.333

Table 5.27 Spirometry results for smokers (N = 8)

To summarise the changes in spirometry across all groups see table 5.28.

Parameter	Never smokers (34)	Ex-smokers (31)	Smokers (8)
FEV1	+2 (p = 0.123)	0 (p = 0.596)	-5 (p = 0.276)
FVC	+5 (p = 0.0001)	+3 (p = 0.012)	-2 (p = 0.547)
PEF	+5.9 (p = 0.009)	+2.1 (p = 0.148)	-1.6 (p = 0.757)
FEF 25-75	-6 (p = 0.039)	-9 (p = 0.003)	-5 (p = 0.333)

Table 5.28 Summary Overall % Change in Spirometry values

As seen from this table, the never smoking group had significant increases in spirometric values, while those who continue to smoke show a reduction in lung function values, suggesting that their personal smoking is continuing to cause damage to their lungs.

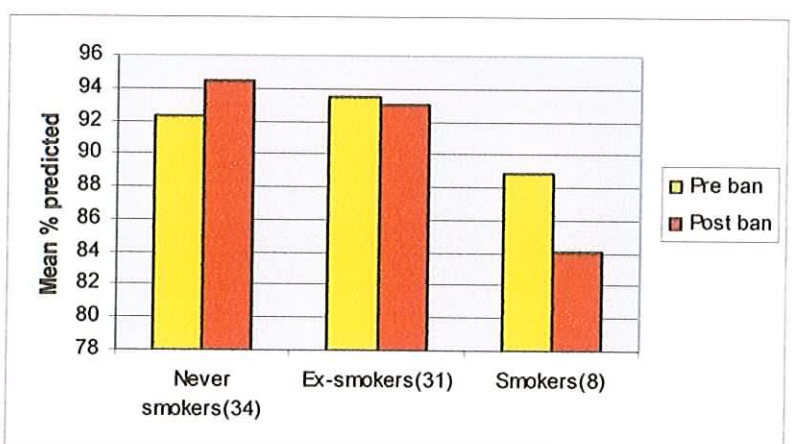

Fig. 5.6 Overall change in FEV1 values before and after the ban

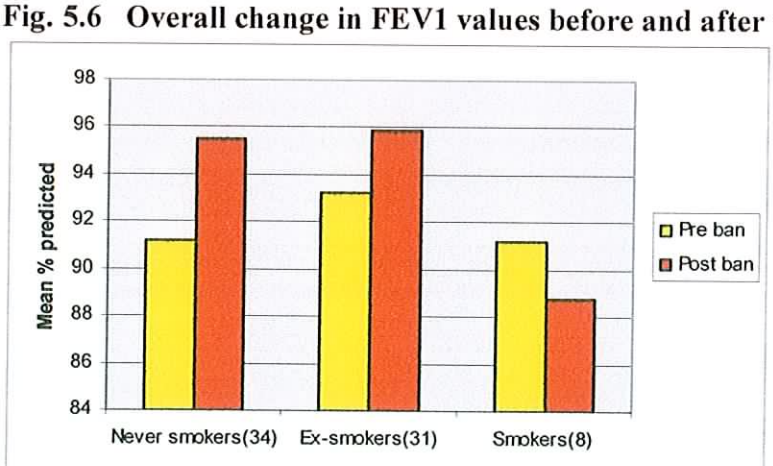

Fig. 5.7 Overall change in FVC values before and after the ban

5.4.2 Lung Volumes and Diffusing Capacity analysis

Looking at the groups individually, there are different changes in each group – see tables 5.29 – 5.31.

	PRE BAN	POST BAN	%change	p value
RV	1.98	1.97		
%PRED	94	93	-1	0.64
TLC	6.24	6.38		
%PRED	90	92	2	**0.02**
CORR DLCO	28.1	29.6		
%PRED	91	96	5	**0.003**

Table 5.29 Lung volume and diffusing capacity in non-smokers (N=34)

	PRE BAN	POST BAN	%change	p value
RV	2.2	2.24		
%PRED	101	101	0	0.815
TLC	6.46	6.58		
%PRED	92	94	2	**0.039**
CORR DLCO	29.2	28.8		
%PRED	96	95	-1	0.75

Table 5.30 Lung volume and diffusing capacity in ex-smokers (N=31)

	PRE BAN	POST BAN	%change	p value
RV	2.54	2.7		
%PRED	115	123	6.8	0.39
TLC	7.03	7.1		
%PRED	95	96	1.84	0.668
CORR DLCO	30	27.8		
%PRED	90	85	-5.6	0.286

Table 5.31 Lung volume and diffusing capacity in smokers (N=8)

:

Parameter	Never smokers (34)	Ex smokers (31)	Smokers (8)
R.V.	-1 (p = 0.64)	0 (p = 0.815)	+6.8(p = 0.39)
T.L.C.	+2 (p = 0.02)	+2(p = 0.039)	+1.84(p = 0.668)
DLCO corrected	+5 (p = 0.003)	-1(p = 0.75)	-5.6(p = 0.286)

Table 5.32 summary % change in Lung Volumes and Diffusing Capacity values

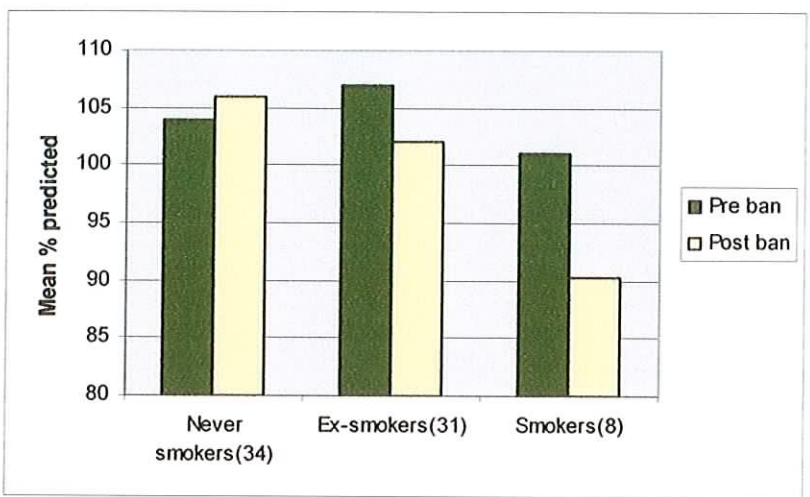

Fig. 5.8 Overall change in DLCO corrected for COHb%

Once more the never smokers fare best, with a reduction in RV (not statistically significant), and a significant increase in TLC and diffusing capacity. The ex-smokers have no change in RV, a small increase in TLC, and a small reduction (not statistically significant) in DLCO, possibly due to damage done previously by smoking. On the other hand the smokers show an increase (not statistically significant) in RV, TLC, and a reduction in diffusing capacity. These changes may be due to the reduction in exposure to ETS in the workplace, but because the smokers are still smoking, their function is deteriorating.

5.4.3 Peak flow monitoring

Not all diary cards and/or peak flow meters were returned. Of those with complete pre and post ban values (N = 51), 6 asthmatics were excluded (including 2 smokers) and a further 4 smokers were also excluded for analysis. 41 completed diary cards were used for final analysis.

The results are as follows :

Pre Ban (41 diary cards assessed for analysis)

15(36%) barmen had higher readings in work than at home

7(17%) barmen had >10% reduction in peak flow while in work (range -10.7% - -30%)

Post Ban (41 diary cards assessed for analysis)

15(36%) barmen had higher readings in work than at home

6(14%) barmen had >10% reduction in peak flow while in work (range -12% - -29%)

The value of this analysis is uncertain, as it is hard to determine the accuracy of the recordings. It would appear that despite the high levels of ETS in the workplace, some workers had higher peak flow readings in work than at home, but this was unchanged after the smoking ban. In the pre ban phase, some workers experienced a greater than 10% reduction in peak flow while in work, and again similar figures

were recorded in the post-ban phase. More accurate recording of peak flow data, including work time, and the presence of a smoker in the home is required to elicit more meaningful results.

5.5 SUMMARY STATISTICALLY SIGNIFICANT RESULTS

	Never smokers 34	Ex-smokers 31	Current smokers 8
Expired CO	↓ -41% p=<0.01	↓ -40% p=<0.01	Not significant (N.S.)
Symptoms of wheeze	N.S.	N.S.	N.S.
Shortness of breath	N.S.	N.S.	N.S.
Morning cough	N.S.	N.S.	N.S.
Daytime cough	N.S.	N.S.	N.S.
Phlegm production	↓ -29% p=0.04	↓ -55% p=<0.01	N.S.
Eye irritation	↓ -75% p=<0.01	↓ -90% p=<0.01	N.S.
Nose irritation	↓ -50% p=<0.01	N.S.	N.S.
Sort throat	↓ -63% p=0.01	↓ -67% p=0.01	N.S.
FEV1	N.S.	N.S.	N.S.
FVC	↑ 5% p=<0.01	↑ 3% p=0.01	N.S.
PEF	↑ 6% p=<0.01	N.S.	N.S.
FEF25%-75%	↓ -6% p=0.04	↓ -9% p=0.03	N.S.
R.V.	N.S.	N.S.	N.S.
T.L.C.	↑ 2% p=0.02	↑ 2% p=0.04	N.S.
DLCO (corrected)	↑ 5% p=<0.01	N.S.	N.S.

Table 5.33 Statistically significant results

CHAPTER 6

DISCUSSION

This study shows that the workplace ban on smoking has achieved what it set out to achieve – a reduction in workplace exposure to ETS (-99%) (table 5.2). Following the introduction of the ban, non-smoking bar workers have had significant improvement in lung function parameters while those of smokers deteriorated (tables 5.25 – 5.32). Less than a year after introduction of the ban, workers previously exposed to high levels of ETS in their workplace reported fewer upper and lower respiratory tract symptoms (figs. 5.4 and 5.5), and measured levels of exhaled carbon monoxide (one of the deadly gases in cigarette smoke) have dramatically dropped in non-smokers (table 5.5), indicating that the air quality in these bars has been greatly improved by this legislation. A study running concurrently with this measured particulate matter in public houses before and after the introduction of the smoking ban (Appendix N). Forty-two public houses were assessed before the ban, and again after the ban, in the same time frame as this study. Investigators found that mean PM 2.5 levels dropped by 83.6%, and Benzene (measured in 26 pubs) dropped by 80%. These particles were measured because it is known that these size particles are responsible for excess mortality, and the benzene was used as a marker for carcinogenic substances in cigarette smoke. Investigators stated that these levels have been reduced as a direct result of the introduction of the smoking ban.

The health of the non-smoking barworkers has improved in terms of lung function, upper and lower respiratory symptoms, while that of smokers has in general continued to decline over the same timeframe.

6.1 SMOKING HABITS AND EXPOSURE TO ETS

Before the introduction of the ban, the barmen reported that they were exposed to ETS for long hours in the workplace (mean 40.5 hours per week) (table 5.2). After the introduction of the ban this was reduced to an average of less than a half an hour per week – a highly significant reduction of 99%. There was still a small amount of exposure for some workers who were assigned to cleaning up outdoor smoking areas – collecting glasses and bottles, and some reported smoking in the toilet areas in their specific place of work. They also reported that their exposure outside work was reduced from a mean 6.4 hours to a mean of 4.3 hours (table 5.4), showing a change of 33%, but data is not specific enough to determine whether this was as a result of barmen going to pubs socially – now smoke-free, or a reduction in the home. However, all reductions in exposure levels were statistically significant.

This change in exposure to cigarette smoke was accompanied by a reduction in CO levels in all barmen (fig 5.2), significant in those who did not smoke, but also a drop of 8.5% in those barmen who smoked. As exhaled breath CO is recognized as a marker for exposure to ETS, these values indicate a statistically significant reduction in CO values in never and ex-smoking bar workers. This reduction in exposure is greatly reducing their respiratory and cardiovascular risks, and by

allowing them to work in a smoke-free environment is improving their longterm health.

A significant finding was that those non-smoking barmen, who did not live with a smoker, or those who had least exposure to ETS, had a more significant reduction in CO levels (table 5.6), thereby suggesting that they were faring better after the introduction of the smoking ban. This would support previous studies claiming that those non-smokers who lived with a smoker had a higher risk of cardiovascular disease [1,2]. This is an accurate objective measurement of exposure and would confirm a significant reduction in ETS exposure in our barworker population.

6.2 QUESTIONNAIRE ANALYSIS

Although this data is subjective, the workers certainly reported fewer symptoms after the introduction of the workplace smoking ban. Comments unrelated to the study questionnaire suggested that they are quite pleased at not smelling of smoke at the end of a work shift, and not having to shower in the middle of the night on returning home from work! In the overall group there was a 28% reduction in reporting of respiratory symptoms, most significantly early morning cough and production of phlegm. Smokers reported a non-significant change in their symptoms, probably due to the fact that they are still exposed to their own cigarette smoke, and this is still causing them problems.

Analysis of the sensory irritant symptoms (eye, nose and throat irritation) showed more significant changes after introduction of the smoking ban. There was an overall reduction of 50% in irritant symptoms reported, with highly significant values in the non-smoking group. While the smokers did have a reduction in symptoms, this was not statistically significant.

The asthmatic barmen (N=9) reported some improvement in respiratory symptoms, but continued to complain of wheeze, shortness of breath and cough, suggesting that symptom improvement was not skewed by larger than average improvement in this group. The asthmatics also had some improvement in irritant symptoms, but again this change was less than that reported by the non-asthmatics, suggesting that the overall improvement was genuine.

6.3 LUNG FUNCTION ANALYSIS

Never-smoking bar workers showed bigger improvements in lung function test results following introduction of the smoking ban than ex-smoking bar workers, while smokers' lung function showed no change or deterioration. Changes in spirometry values were small, with the most significant increase being a 5% improvement in the FVC of never smoking barmen, and the most unusual being a decrease in the FEF25-75% in all groups. This decrease in mid flow rates was also seen by Eisner [3], who stated that mid flow rates are variable, and after correcting for smoking and respiratory tract infection, that this change was not significant. There were not many smokers in the study, so perhaps these changes *are*

significant. In the non-smoking barworkers the FEV1 showed no significant change, while the FVC showed a significant increase. Spirometry parameters decreased in smokers over the pre-ban / post-ban time frame (table 5.28). These values suggest that those with least exposure to ETS have healthier lungs, and those who smoke are continuing to damage their lungs by inhaling the noxious gases of cigarette smoke.

Total lung capacity increased significantly in never and ex-smokers, but not significantly in the smokers (table 5.32). This may support the increase in vital capacity seen on spirometry testing, but while the non-smokers have no significant change in residual volume values, this was increased in the smoker (6.8%) suggesting an increase in air-trapping in this group. The most notable change is that of a reduction of 6% in the diffusing capacity of smokers in the year between the two tests. Although smokers are a small group in this study, this data shows a drop by 6% in the lungs ability to absorb oxygen over a single year in the life of a smoker. To ensure that changes in diffusing capacity were not affected by the presence of higher CO levels in smokers, the DLCO was corrected for the presence of CO, as explained in chapter 3 (page 59). This allowed objective change to be assessed and reported.

6.4 PEAK FLOW ANALYSIS

No definite conclusion can be reached as to whether workers were affected during working hours by the ETS in their place of work. The data was unreliable due to variable effort and poor work time recording. There were problems with the peak flow measurements recorded outside the laboratory – 1) they were not carried out by a trained clinical person and the barmen tended to record very variable efforts. 2) Three specific times were listed to record values – 11am, 3pm, and 8pm, but on some days the barmen listed work time at e.g. 10am to close (midnight), and so no difference could be assessed between work time and home time on those days. 3) Pubs are usually busier at weekends – Friday, Saturday and Sunday nights, and on days when the work reading appeared lower on a Saturday evening, it could not be determined if it was due to higher levels of ETS in a very busy pub, as there was no way of assessing customer numbers. In hindsight, a more stringent diary card should be used listing accurate times of working hours, and numbers of customers in the pub on any given night. Other useful information would have been to know if the barman was in a house with a smoking person on their time off to see if this had any bearing on lower readings while at home.

6.5 STUDY LIMITATIONS

There were some limitations of this study – all the barworkers were volunteers and may not be fully representative of the exposed population. Also, they were all male. On questioning Mandate trade union staff, I was told that approximately 20% of members are female, but most of these work part-time, in cleaning and

kitchen posts. It was hoped that large numbers would participate, but due to the negative attitude of the hospitality industry in general towards the legislation (appendices G, H, and I), workers were reluctant to come forward for the study. As a result of this, the number of smoking bar workers was low, making it difficult to get statistically significant results in this group. Many of them were initially against the change in law, and were reluctant to participate. However, despite these limitations, significant improvements were seen in the working conditions of bar workers, exposure levels and lung function measurements following introduction of the workplace smoking ban.

6.6 COMPARISON WITH PREVIOUS STUDY

A similar study was conducted in California in 1998, when a workplace smoking ban was enforced [3]. There, 53 bar workers were studied pre and post smoking ban and a questionnaire and spirometry were carried out on site.

At baseline, all 53 subjects reported ETS exposure while working in bars during the 7 days prior to interview. Following the introduction of the smoking ban, there was no significant change in weekly work duration from baseline (mean 33.4 hrs to follow-up 32.2 hrs; P = .48). However self-reported workplace ETS sharply declined from a median of 28 to 2 h/wk (P=<.001) after the law went into effect. Despite the ban, 29 subjects (55%) continued to report some ETS exposure while working in bars.

	CALIFORNIA	DUBLIN
No. of subjects	53	73
Mean age	42.5	46.9
Work hours per week	33.4	40.5
Workplace exposure (hrs)	28	40.5
Measurement :		
Baseline FEV1 % pred.	89.2	92
Post FEV1 % pred	89.9	93
Change	1.2%	1%
Baseline FVC % pred.	95.5	92
Post FVC % pred.	99.8	95
Change	4.2%	3%
Baseline FEF25-75% % pred.	81.6	87
Post FEF25-75% % pred.	80.3	80
Change	-5.7%	-8%

Table 6.1 Comparison of study values in the USA v. Dublin (mean values)

Respiratory and Irritant symptoms

39 (74%) of the 53 bar workers reported respiratory symptoms at baseline, while only 17 (32%) were still symptomatic at follow-up. Of the 39 bar workers reporting baseline symptoms, 23 (59%) subjects no longer indicated any respiratory symptoms after introduction of the ban (P=<.001). The majority of bar workers

(77%) also had at least 1 sensory irritation symptom at baseline, with fewer (19%) reporting symptoms at follow-up.

Lung Function (spirometry only)

After the smoking ban was introduced, the mean FVC and FEV1 both increased at follow-up. Flow rate at mid lung volumes (FEF 25%-75%) which was highly variable, declined during the study period but when respiratory tract infection and smoking history was taken into account, this reduction was deemed not significant.

Observations from studies

- There were more smokers in the US study (45% USA v. 11% in Dublin).
- The Dublin barmen were slightly older (mean age 46.9 Dublin v. 42.5 USA).
- The Dublin barmen had longer years experience of bar work (30 yrs Dublin v. 7 yrs USA)
- The Dublin barmen had longer hours of workplace exposure (40.8 Dublin v. 28 USA).
- The Dublin lung function was carried out in a controlled, smoke-free, hospital Respiratory department, and not in a bar with other workers coming and going while tests were conducted.
- Both studies show a small improvement in FEV1, a larger increase in FVC, and a decrease in FEF25 – 75% values.

- Both studies show significant changes in symptoms reporting, with significant reduction in respiratory and irritant symptoms in both California and Dublin.

6.7 SUCCESS OF SMOKING BAN IN OTHER COUNTRIES

California (U.S.A.), was one of the first regions to introduce a smoking ban in the workplace in 1998, with a very positive outcome. A study in Los Angeles of customer compliance found that between 1998 and 2002 compliance rose from 46 % to 76% in bars and from 92% to 99% in bar/restaurants [4].

The Smoke-Free Air Act went into effect in New York on March 30, 2003 and one year later New York City issued the following statistics:

- 97% of restaurants and bars are smoke-free
- New Yorkers overwhelmingly support the law
- Air quality in bars and restaurants has improved dramatically
- Salivary cotinine levels decreased by 85% in non-smoking workers in bars and restaurants
- Business tax receipts in restaurants and bars are up 8.7%
- Employment in restaurants and bars has increased

A study in Helsinki, following introduction of a smoke-free workplace law, found that exposure to ETS declined considerably after the legislation was implemented [5]. They also found that tobacco consumption among smokers diminished and nicotine concentrations fell significantly. They concluded that legislation was

more efficient than voluntary workplace-specific smoking restrictions in reducing passive smoking and cigarette consumption.

The European Respiratory Society is actively working to encourage all governments to adopt similar anti-smoking laws to safeguard workers from risk of exposure to ETS. As the WHO has deemed ETS to be a carcinogen, this would seem to be a logical step forward. Many government representatives have visited our country to discuss the ban as implemented here, and to seek advice on how they should manage introduction of legislation in their own country.

This ban is likely to cause significant changes in smoking habits in Ireland. There has been a change in attitude amongst smokers, with most now favouring the ban, while beforehand they were against it or undecided [6]. Information from the OTC has stated that there has been a reduction in smoking prevalence of 1.4% [7] since the introduction of the workplace smoking ban, which is more than three times the average expected rate of decline in the same timeframe [8].

6.8 REFERENCES

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CHAPTER 7

CONCLUSION

From the results of this study it is clear that the introduction, and enforcement of the workplace smoking ban has resulted in an immediate significant reduction in exposure to ETS, a significant reduction in exhaled CO levels, and a reduction in upper and lower respiratory symptoms in bar workers in Dublin. While it was thought that there would be little or no change, there has been an actual improvement in lung function parameters in the non-smoking bar workers since the introduction of the ban, suggesting an immediate health benefit following the reduction in exposure to harmful cigarette smoke.

The introduction of this smoking ban has been the least expensive way to reduce significantly the harmful levels of ETS in the workplace, and with good enforcement and high levels of compliance, means that the workers are experiencing less respiratory symptoms than before. Quantitative measurements have proven that there is an immediate health benefit, and an increase in lung function within one year of the introduction of the ban.

A study by my colleagues (Mc Caffrey et. al) published in the Irish Journal of Medical Science (vol. 175, No. 2) – 'Smoking, occupancy and staffing levels in a selection of Dublin pubs pre and post a national smoking ban, lessons for all' has proven that the outcry by the hospitality industry pending the introduction of the ban, claiming that jobs would be lost, and premises would close, was unnecessary.

The authors state that while there has been a decrease of 8.82% in worker numbers, this is not statistically significant, and there has been an increase of 11% in customer numbers in bars since the introduction of the ban. This study was carried out in 38 pubs around Dublin before and after the introduction of the workplace ban on smoking.

The introduction of this comprehensive smoking ban and good compliance with the regulations, has shown that this type of action can work, and is easier to achieve than was previously thought. It has been a positive move to improve the immediate and longterm health of workers in Ireland.

APPENDIX A

SPECIFICATIONS FOR MICRO CO METER (MICROMEDICAL, UK)

Specifications for Micro CO meter (Micro Medical, UK)

Sensor type	Elector-chemical fuel cell
Range	0 – 500ppm
Resolution	1ppm
Green light indicator	0 to 5ppm(0 to 0.8%COHb)
Amber light indicator	6 to 10ppm(1 to 1.6%COHb)
Red indicator light	11 to 72ppm(1.8 to 12%COHb)
Flashing red light plus alarm	>72ppm(>12%COHb)
Accuracy	+/-5% of full scale or 1ppm whichever is the greater
Sensitivity drift	0.5% / degreeC
Sensor life	2 to 5 years
Response time	< 20 sec(to 90% of reading)
Hydrogen cross sensitivity	< 10%
Operating temperature	15 – 25 C
Operating pressure	Atmospheric +/- 10%
Pressure coefficient	0.02% signal per mBar
Relative humidity	15 – 90% continuous
(Non condensing)	(0 – 99% intermittent)
Baseline drift	0ppm (auto zero)
Long term drift	< 2% signal loss per month
Power source	Single Alkaline 9 volt PP3
Main battery life	> 30 hours of continuous use

Internal battery life	2 years
Weight	160g
Dimensions	170 x 60 x 26 mm
Display	3 ½ digit LCD
Storage temperature	-20 to +70
Storage humidity	30% to 90%

APPENDIX B

SPECIFICATIONS FOR SENSORMEDICS VMAX LUNG FUNCTION SYSTEM

Specifications for Sensormedics Vmax system

Electrical requirements Voltage 100 VAC to 240 VAC

Frequency 50/60 Hz

Phase – single

Electrical safety Leakage current - <100microampere

Dielectric withstand - >2500VAC for 1 minute

Mass Flow Sensor Instantaneous flow range 0 – 16 LPS

Integrated volume range 0 – 350 L/min

Resolution .03 LPS from 0.1 – 16 LPS

Accuracy – larger of +/- 3% of reading or 0.25 LPS, whichever is greater

Flow path resistance - < 1.5 cmH20/LPS at 12 LPS

Integrated volume accuracy - +/- 0.05 L

Multi-gas Analyser Type : Carbon Monoxide (CO) and Methane (CH4).

Non-dispersive infra-red, thermopile.

Sample rate – 500 ml/min

Response time – Overall system nominal < 150msec at 500 ml/min flow

Range (CO) 0 – 3300 ppm

Range (CH4) 0 – 3300 ppm

Resolution (CO) 5ppm

Resolution (CH4) 5ppm

Accuracy (CO) +/- 30ppm*

	Accuracy (CH4) +/- 30ppm*
	*Calibrated within 5% of operating range after 15 minutes warm-up period. Two point calibration.
Oxygen Analyser	Type paramagnetic
	Range 0 – 100% oxygen
	Resolution +/- 0.01% oxygen
	Accuracy +/- 0.02% oxygen*
	Response time – overall system nominal < 130msec (10 – 90%) at 500 ml/min flow
	*Calibrated within 5% of operating range after 15 minutes warm-up period. Two point calibration.
Carbon dioxide Analyser	Type non-dispersive infrared. Thermopile.
	Range 0 – 16% carbon dioxide
	Resolution 0.01% carbon dioxide
	Accuracy +/- 0.02% carbon dioxide*
	Response time – overall system nominal < 130 msec (10 – 90%) at 500 ml/min flow
	*Calibrated within 5% of operating range after 15 minutes warm-up period. Two point calibration.
Direction Pressure Transducer	Range +/- 2cmH20
Mouth Pressure Transducer	Range +/- 300mmHg Accuracy +/- 1%

APPENDIX C

DIARY CARD TO RECORD PEAK EXPIRATORY FLOW OUTSIDE THE LABORATORY

Diary card to record Peak Expiratory Flow outside laboratory

ID :_____	DAY 1	DAY 2	DAY 3	DAY 4	DAY 5	DAY 6	DAY 7
DATE :							
MID MORNING : 11AM							
MID AFTERNOON : 11AM							
EVENING : 8PM							
WORK TIME :							

APPENDIX D

SPECIFICATIONS FOR PIKO – 1 (PEAK FLOW METER), FERRARIS, UK

Specifications for Piko-1 (peak flow meter), Ferraris, UK.

PEF	Range 15 – 999LPM (1LPM resolution)
FEV1	Range 0.15 – 9.99 litre (0.01 L resolution)
Accuracy	PEF +/- 5% or 20 LPM, whichever is greater
	FEV1 +/- 3% or 0.1 L, whichever is greater
Sensor	Pressure/flow sensor technology
Memory	96 tests, containing PEF, FEV1, colour zone, quality,
	time/date stamp (15 min resolution)
Memory type	Non-volatile
Quality factor	Warning and indicator for cough or abnormal blow
Battery	2, type 357 silver oxide button cells
Battery life	2 years (based on average of 6 tests per day)
Dimensions	75 x 35 x 20 mm
Weight	35gr
Back pressure	1.5cmH20 / L/S @ 14 L/S or lower
Operating temperature	10 – 38 C
Humidity	0 – 100% relative humidity
Barometric pressure	550 to 780 mmHg
Standard	ATS 1994 (monitoring devices)

APPENDIX E

PATIENT CONSENT FORM AND PROTOCOL SUBMITTED TO ETHICS COMMITTEE

CONSENT FORM

Title of research study: To evaluate the effects of a smoke-free environment on the lung function of bar staff in Dublin.

This study and this consent form have been explained to me. The technologist has answered all my questions to my satisfaction. I believe I understand what will happen if I agree to be part of this study.

I have read, or had read to me, this consent form. I have had the opportunity to ask questions and all my questions have been answered to my satisfaction. I freely and voluntarily agree to be part of this research study, though without prejudice to my legal and ethical rights. I have received a copy of this agreement.

PARTICIPANT'S NAME:

PARTICIPANT'S SIGNATURE:

Date:
Date on which the participant was first furnished with this form:

Where the participant is incapable of comprehending the nature, significance and scope of the consent required, the form must be signed by a person competent to give consent to his or her participation in the research study (other than a person who applied to undertake or conduct the study). If the subject is a minor (under 18 years old) the signature of parent or guardian must be obtained.

NAME OF CONSENTOR, PARENT or GUARDIAN:
SIGNATURE:
RELATION TO PARTICIPANT:

Where the participant is capable of comprehending the nature, significance and scope of the consent required, but is physically unable to sign written consent, signatures of two witnesses present when consent was given by the participant to a registered medical practitioner treating him or her for the illness.

NAME OF FIRST WITNESS: SIGNATURE:
NAME OF SECOND WITNESS: SIGNATURE:

Statement of investigator's responsibility: I have explained the nature, purpose, procedures, benefits, and risks of this research study. I have offered to answer any questions and fully answered such questions. I believe that the participant understands my explanation and has freely given informed consent.

Technologist's signature:
Date:
(Keep the original of this form in the participant's medical record, give one copy to the participant and keep one copy in the investigator's records)

ST. JAMES'S HOSPITAL AND FEDERATED DUBLIN VOLUNTARY HOSPITALS

JOINT RESEARCH ETHICS COMMITTEE

ADMINISTRATIVE APPLICATION

1. **Title of research project: 'To evaluate the effects of a smoke-free environment on the lung function of bar staff in Dublin.'**

2. **Name of local project supervisor(s) : Professor Luke Clancy.**

3. **Name and address of the person to whom the Committee's decision is to be communicated:**
   Michele Agnew, Chief Respiratory Technician, Respiratory Laboratory, St. James's Hospital, Dublin 8.

4. **For each funded research project a review fee of €634.87 is payable.**
   Payment to the "Federated Dublin Voluntary Hospitals" should accompany the study documentation submitted to the Joint Research Ethics Committee.

   If you believe the review fee should not be charged for the project now being proposed please give the reason(s) here:

   This is not a funded project.

   Please note that, for funded projects, after the initial ethical review is complete, (i. e. after all conditions attached to the approval of the original submission have been responded to and after that response has been approved), any amendment arising attracts a review fee as follows:

   Major Amendment : €126.97 Minor Amendment: €63.49

   Payment should accompany the amendment documentation.

   "Funded research project" generally means a clinical trial sponsored by a Pharmaceutical Company. However, the Committee also expects payment to review other types of research project if the project is financially supported to a degree which makes it reasonable to expect such a payment.

Signed _____ **Date**

_____

Project Supervisor.

ST. JAMES'S HOSPITAL AND FEDERATED DUBLIN VOLUNTARY HOSPITALS

JOINT RESEARCH ETHICS COMMITTEE

Confidential Research Protocol, 2003 Edition.

Please place an "X" or ✓ after the appropriate response in the boxed areas. NA is an abbreviation for Not Applicable.

1. Title of research project: To evaluate the effects of a smoke-free environment on lung function of bar staff in Dublin.

2. Name of local project supervisor(s) : Prof. Luke Clancy.

DECLARATION BY SUPERVISOR

I confirm that the information provided in this protocol is correct. I also undertake to provide an annual report on the anniversary of Research Ethics Committee approval with details of the number of subjects who have been recruited, the number who have completed the study and details of any adverse effects.

Signed: (Project Supervisor)

Date:

Research Ethics Committee comments:

Approved subject to : Approval by Irish Medicines Board

Other conditions:

Approved without conditions.

Signed: (Chair)

Date:

3. What are the objectives of the research project? The objectives of this project are to assess the effects of a smoke-free working environment on lung function of bar staff, previously exposed to large amounts of cigarette smoke generated by clients. New legislation proposed by the Irish Government comes into force on 1st January 2004 and from that date onwards smoking will be prohibited in public places, including bars.

4a. Does the design of the study allow a statistically significant conclusion to be reached?

YES	NO

4b. Has statistical advice been sought?

YES	NO

5. Will the conduct of the project conform to the principles of the Declaration of Helsinki? (Recommendations guiding Medical Doctors in Biomedical Research involving Human Subjects; the text of this Declaration is included on pages 3 to 8).

YES	NO

If not, elucidate:

6. Please itemise here any ethical problems which you perceive to be associated with the research project: I do not perceive any ethical problems associated with this project, as all tests being carried out on subjects are simple tests, routinely carried out at St. James's Hospital, and no medications will be administered during the trial.

SECTION A

Details of project

7. **Background:**

A. What person or organisation devised this project? Project devised by Michele Agnew, Chief Respiratory Technologist, St. James's Hospital, Professor Luke Clancy, Respiratory Consultant, St. James's Hospital, and Dr. Pat Goodman, Department of Physics, D.I.T. Kevin Street, Dublin 8.

B. Has a detailed research protocol been drawn up?

YES	NO

C. Has the investigator who may be asked to present the project to the Committee studied all the documentation drawn up for the project, and will the documentation be studied by all the investigators before the project begins?

YES	NO

D. Briefly describe the scientific rationale for the project: Bar staff, whether current smokers or not, are constantly being exposed to large amounts of cigarette smoke, generated by bar clients. This passive smoking may result in the development of lung disease and other related health risks in these people if continued over a period of time. New no-smoking legislation, prohibiting smoking in public places, including bars, will reduce these risks and should improve lung function status of bar staff.

A similar study carried out in 1998 in San Francisco found an improvement in lung function parameters of bar staff after legislation was introduced there to prohibit smoking in bars and public places.

8. **Planning and organisational structure** (briefly outline the study methods, the various treatment groups, what parameters will be studied, how often and for how long, and what outcome measures or end points will be used to assess the efficacy of the project, for each subject):

a) Subjects will be asked to attend the Respiratory Laboratory in St. James hospital and will have spirometry, static lung volumes, gas transfer measurement and carbon monoxide measurement carried out. A questionnaire will be completed with the subject, with regard to exposure to passive smoke, smoking history, and current respiratory status. The subject will then be given a peak flow meter and diary card to keep reading of peak flow measurement (three times daily) over the next two weeks. They will be instructed how to perform the measurement and the diary card will be explained to them. At the end of this two week period they will return the peak flow meter and diary to the lab. A total of approximately 100 subjects will be studied

before the end of the year, as new legislation comes into effect on the 1st January 2004.

b) In April 2004, subjects will be asked to return to the lab, and spirometry, static lung volumes, gas transfer measurement and carbon monoxide measurement will be repeated. They will again be given the peak flow meter to monitor peak flow measurements over a two week period, and they will then return the meter to the lab.

c) Data collected before and after introduction of legislation will be statistically analysed to see whether there is an improvement in lung function test results in subjects following introduction of smoke-free bars. Peak flow readings will be assessed to see whether subjects fared better while at home (smokey atmosphere / smoke-free), or in work (smokey atmosphere / smoke free).

9. What is the nature and extent of the medical examination that participants and controls are to undergo before participating in this project? No medical examination will be necessary for participants in this study. Subjects are not 'patients', and initial tests will form a baseline for lung function test results carried out while they are still working in a smokey environment. A brief questionnaire will be completed at first visit. This will cover work history, exposure to passive smoke, and current respiratory status.

10. How will the health of the participants and controls be monitored during and after the trial? (list clinical, laboratory and other examinations): Not applicable – no medications will be administered during trial.

11. Will participants or controls undergo independent medical examination, before, during or after the trial?

YES	NO
NA	

12. If a placebo group is to be used, will the group receive the best standard therapy?

YES	NO
NA	

13. If the project involves the use of radioactive substances or of laser therapy has the approval of the Head of Medical Physics been obtained?

YES	NO
NA	

If not, elucidate:

SECTION B

Investigators and Facilities

14. Name, qualification and position of each person associated with this project:

	Name	Qualification	Position
a)	Prof. Luke Clancy	F.R.C.P.	Consultant
b)	Dr. Pat Goodman	PhD.	Senior lecturer in Physics, D.I.T.
c)	Ms. Michele Agnew	Cert. M.P.P.M.	Chief Resp. Tech., St. James Hospital
d)			
e)			
f)			
g)			

15. Is each investigator a registered medical practitioner?

YES ***NO***

If not, elucidate: Michele Agnew is Chief Respiratory Technician in St. James's Hospital, and Dr. Pat Goodman is senior lecturer in the Department of Physics in Dublin Institute of Technology, Kevin Street, Dublin.

16. Is each investigator a member of a major medical defence body?

YES ***NO***

If not, elucidate: only Prof. Luke Clancy, primary investigator.

17. What payments, monetary or otherwise, if any, are to be made to any of the investigators (include payments to any institution or research facility)? None

18. What payments, whether monetary or otherwise, if any, are to be made to any person or institution providing facilities to be used for the purpose of the clinical trial? None

19. **In which hospital or facility will the project take place?** Respiratory Laboratory, St. James's Hospital, Dublin 8.

SECTION C

Participants

20. How many subjects and controls from this centre are expected to participate in this project?

	Number
Subjects: **100** Controls: 0	

21. If this is a multicentre trial please indicate: Single centre only

a) the expected overall number of subjects: Number

b) the number and geographical distribution of the centres involved in the study:

22. What criteria are to be used for the selection of participants? They must be currently working in a bar.

23. Are women of childbearing potential included?

YES NO NA

If so, does the protocol/patient information sheet address the 8 points in the committee's checklist for studies involving women of childbearing potential (1–scientific justification, 2–negative teratogenic studies, 3–warning to subject that fetus may be damaged, 4–initial negative pregnancy test, 5–forms of contraception defined, 6–duration of use to exceed drug metabolism, 7–exclude those unlikely to follow contraceptive advice, 8–notify investigator if pregnancy suspected)?

YES NO
NA

**Women of child-bearing age are not excluded from routine lung function testing at any time.

24. State the exclusion criteria (age, other illness, other medications etc.): None

25. What are the proposed methods by which participants and controls are to be recruited?

"Direct request to suitable patients attending investigator's clinic"

YES ***NO***
NA

If no, elucidate: A formal letter of explanation and invitation will be sent to bar staff through their unions. They will be asked to participate, and if willing, to contact Michele Agnew directly at St. James's Hospital.

26. What inducements or rewards, whether monetary or otherwise, are to be offered participants and controls?

"None, other than minimal expenses to cover taxi fares etc".

YES	*NO*
NA	

If no, elucidate: All subjects completing the study will be entered into a draw for a prize (possibly T.V / video) to the value of maximum of 200 euro.

27. What arrangements exist to provide compensation to each participant who may suffer injury or loss as a result of this research project?

"Participation in this study is covered by an approved policy of insurance in the name of (sponsor). In addition the medical practitioners involved in this study have current medical malpractice insurance cover. The sponsor (name) will comply with the ABPI guidelines and Irish Law (statutory and otherwise) in the unlikely event of your becoming ill or injured as a result of participation in this clinical study."

Is the Ethics Committee's standard compensation statement (above) being adopted ?

YES	NO	*NA*

If "NO" please give alternative wording.

The wording used in answer to question 27 must also appear in the Patient Information Sheet.

28. Have you submitted to the committee, with this form, a patient information leaflet and consent form prepared by a sponsor or other external group, or a patient information leaflet and consent form based on the committee's guidelines (attached to this form) to be given to each participant and control?

YES	NO

If no, elucidate:

29. What criteria are to be used to ensure that the identity of each participant and control remains confidential?

"Only the investigator's group within the institution will know the identity of the subjects; codes will be used to conceal identities in all external communications

(but the sponsor or official regulatory agencies may be given access to the case notes to ensure that the trial has been conducted legitimately)."

YES	NO

If no, indicate the criteria used.

30. Give details of any risks to subjects or to controls from investigative or therapeutic procedures or from withholding of therapy?
NOTE: for the protection of both the investigator and the subject this list must be comprehensive and must also appear in full in the patient information leaflet.

No risks involved, no changes will be made to subjects normal medications and no medications will be administered during the study.

31. Indicate how adverse events are to be notified and evaluated:

There should be no adverse events during visits to hospital. All lung function testing is governed by European Respiratory Society protocols, and these protocols are strictly adhered to by laboratory staff at all times.

SECTION D

Drugs and other Therapeutic Substances

32. Is the object of this project to assess the effect of a drug or therapeutic substance?

YES	***NO***

If NO, skip to the end of the form.

33. Name of the substance or preparation which is the subject of the proposed project:

34. Name of the company or organisation which produces this substance or preparation:

35. Code number used by the company or organisation for this trial:

36. Does the organisation and performance of this trial conform to the International Conference on Harmonisation guidelines on Good Clinical Practice?

YES	NO

37. Give details of the pharmacology, dosage, toxicity, and side effects of the substance or preparation:
NOTE: for the protection of both the investigator and the subject the list of side effects must be comprehensive and must also appear in full in the patient information leaflet.

Product Authorisation

38. Is there a Product Authorisation ?

YES	***NO***

39. If there is a Product Authorisation, does the study involve a new use not included in the authorisation, or a dose in excess of the maximum authorised, or otherwise exceed authorisation)?

YES	NO
NA	

40. Irish Medicines Board:

Application for approval of the study has been made.

NA	YES	NO
PENDING		

DATE of Application:

Approval has been received.

NA	YES	NO
PENDING		

DATE of Approval:

Conditions attached ?

YES	NO

41. Is the preparation or substance given with therapeutic intent? (Is the principal purpose of its administration to prevent disease in or to save the life, restore the health, alleviate the condition or relieve the suffering of the patient – in contrast to testing a drug in normal controls or volunteers or giving a drug purely to study pharmacokinetics?).

YES	NO

note that if the answer is NO patients who are unable to physically sign consent or unable to comprehend the nature, significance, and scope of the consent required, may not participate.

42. Will the trial begin within 6 days of recruitment? (In order to allow for mature consideration by the participant, a period of 6 days must elapse from the time a subject is invited to participate in a drug trial (and been given appropriate information) and the

beginning of the study. If such a delay is not possible the 6 day rule may be waived by the IMB

YES	NO

42a If YES, has a request for a waiver of the 6 day rule been made to the IMB?

YES	NO
NA	

43. Indicate what phase (IMB phase 1-4) of drug testing the trial represents.
Phase 1 Pharmacokinetics in healthy volunteers
Phase 2: Early studies (kinetics and dose ranging)
Phase 3: Large safety and efficacy studies in population to be treated
Phase 4: Post-marketing/monitoring

Phase		
1	2	3
4		

---00000OOOOOOO00000---

CONSENT FORM

CONSENT FORM FOR PARTICIPATION IN GENETIC RESEARCH

Protocol Number: .....

Participant Identification Number: .....

Title of Protocol: .....

Name of Institution leading the Research : .....

Research Director: .....

Phone Number and Contact Details: .....

Please initial boxes

1. I have read the attached information sheet on the above project dated..... and have been given a copy to keep. The information has been fully explained to me and I have had an opportunity to ask questions about the project and understand why the research is being done and any foreseeable risks or consequences involved. I also understand that no guarantee can be given about the possible results. ☐

2. I agree to give a sample(s) of blood / other bodily sample / DNA for research in the above project. I understand how the sample will be collected, that giving a sample for this research is voluntary and that I am free to withdraw my approval for use of the sample at any ☐

time without giving a reason. If I withdraw my consent I understand that my sample will be destroyed unless I otherwise authorise. I understand that I may ask for my samples to be destroyed and that this will be without my medical treatment or legal rights being affected. I agree that the samples I have given and the information gathered by me can be stored and looked after by the (name of institution).
I understand that any genetic information obtained will / will not be made available to me.

3. I give permission for my medical records to be looked at and information taken from them to be analysed in the strictest confidence by the relevant and responsible people from the (*name of study team* .....) or from organisations supervising the research. I have been told that all medical information / data pertaining to me will be protected by the principles of confidentiality and both national and E U data protection legislation. I have further been told of / shown assurances that this also applies to all medical information / data pertaining to me that are utilised in any non-E U state. ☐

4. I understand that the confidentiality of the sample(s) I donate and information derived therefrom will be protected. I have been told that all medical information / data pertaining to me and derived from the sample(s) will be protected by the principles of confidentiality and both national and E U data protection legislation. I have further been told of / shown assurances that this also applies to all medical information / data pertaining to me and derived from the sample(s) that are utilised in any non-E U state. ☐

FOR OTHER GENETIC RESEARCH :

(Note : New research should be submitted for approval by the Research Ethics Committee before proceeding)

5. I understand that future research using the sample I give may include genetic research aimed at understanding the genetic influences in disease but that such test will not be of predictive / clinical value and that the results of these investigations are unlikely to have any implications for me personally. ☐

6. I understand that I will not benefit financially in any way if this research leads to the development of a new treatment or medical test. ☐

7. I know how to contact the research team if I need to. ☐

.....	.....	
.....		
Name of participant (BLOCK CAPITALS)	Date	Signature
.....	.....	
.....		
Name of researcher	Date	Signature
.....	.....	
.....		
Name of witness (or) GP for patient without capacity	Date	Signature

APPENDIX F

LETTER OF APPROVAL FROM ETHICS COMMITTEE

Dan Lynch, Joint Research Ethics Committee Secretariat
Telephone : 4142861 Fax : 4142171 Email: dan.lynch@amnch.ie

THE ADELAIDE & MEATH
HOSPITAL, DUBLIN
INCORPORATING
THE NATIONAL CHILDREN'S HOSPITAL

TALLAGHT, DUBLIN 24, IRELAND

Michele Agnew,
Chief Respiratory Technician,
Respiratory Laboratory,
St. James's Hospital,
James's Street,
Dublin 8.

18th July, 2003

RE : 'To evaluate the effects of a smoke-free environment on the lung function of bar staff in Dublin'.

Please quote this reference in all communications regarding this study : Chairman's Action, July 2003.

Dear Ms. Agnew,

Dr. Michael Barry, Chairman, has, on behalf of the Joint Research Ethics Committee, given ethical approval for the above study.

Yours sincerely,

Daniel R Lynch

Daniel R. Lynch,
Senior Executive Officer

APPENDIX G

HEADLINES FROM IRISH TIMES NEWSPAPER (29.10.2003)

Irish Times Wed 29/10/2003

Government leaves all the publicans fuming

CARL O'BRIEN

They huffed and they puffed in Portlaoise, but, in the end, they couldn't blow Minister Martin's smoking ban down.

The 1,200 publicans arrived to vent their anger at the Minister's ban. In two and a half-hours of roaring and cheering in a smoke-filled hotel conference centre in Portlaoise, they did just that.

Once the popular image of the Minister for Health was that sepia photograph of him as an angelic youngster wearing his communion suit. Sitting through this meeting you got the image of a demonic monster hell-bent on the destruction of the pub trade.

Mr Paul Stephenson, a pub owner from Ballymoate, Co Sligo, got things going after accusing the Minister and the Government of embarking on a totalitarian campaign. "We seem to have Franco Fahey in Galway, Mussolini Martin, our Health Minister, and Adolf Ahern, ably-supported by the Fianna Fag party," he said.

Adopting a Churchillian mode, the rhetoric soared upwards as Mr Stephenson warned of the dark forces that could be unleashed if the ban is implemented.

"It's our freedom that's at stake, our right to choose one thing in preference to another. Our democracy is at stake . . . if he's failed to listen to the people, and failed to listen to the members of his own party, then maybe he'll listen to members of the judiciary."

Others used a less florid delivery style, choosing instead to ask the Minister for Health how would he get on if trying to eject a drunken, cigarette-smoking customer from his premises.

"I'd like to see Minister Martin, with his long smiling face, trying to do that," thundered Mr Willie Daly, a publican from Ennistymon, Co Clare.

Another said the Minister of skulduggery was ignoring publicans at every possible turn. "Micheál Martin has one hell of a neck," said Mr Gerry Rafter, chairman of the Kilkenny City Vintners. "At this stage he wants to discuss the implementation with us. We've been trying to discuss it with him for the past 11 months and he's ducked and dived with us."

After two and a half-hours, however, the show was over. The mass of people and traffic left just as abruptly as it arrived, with no firm idea on how to halt the ban itself.

APPENDIX H

HEADLINES FROM IRISH TIMES NEWSPAPER (06.01.2004)

New legal challenge threatens introduction of smoking ban

Times 6/1/04

CARL O'BRIEN AND LIAM REID

Publicans, hotel and restaurant owners are to mount a legal challenge against the Government's smoking ban shortly before it is due to come into force.

They will jointly seek a judicial review of the legislation in the High Court which, if obtained, could further delay implementation of the ban for up to a year.

The Vintners' Federation of Ireland, the Licenced Vintners' Association, the Irish Hospitality Industry Alliance and the Irish Hotels Federation are to hold a private meeting later this month to discuss the legal challenge.

The groups will be briefed by a legal expert and will discuss how to share the financial burden of mounting a High Court challenge.

Meanwhile, a study commissioned by the Office for Tobacco Control (OTC) has been unable to draw definitive conclusions as to the economic effect of the ban on the hospitality industry.

The OTC has yet to publish the report, completed by UCD economists Mr Moore McDowell and Mr Joe Durkan last October, which found there was insufficient data from other regions where a ban was implemented to predict the outcome here.

Mr Martin: due to announce date of implementation within fortnight

Officials from the OTC have previously claimed that independent studies of smoking bans in other countries have indicated "no negative economic impact from smoking bans in restaurants and bars".

The Department of Finance is estimating that the smoking ban will contribute to a drop of 2 per cent in beer sales this year.

Last month the Minister for Finance warned that it was difficult to predict the economic impact of the ban, and that this could be "wide of the mark".

The Minister for Health, Mr Martin, is due to announce within the next fortnight the implementation date for the ban, which is now expected in March or April.

The ban cannot be implemented before the end of February, when a 90-day EU approval process ends.

It is likely that a legal challenge could focus on a number of areas, including the wide-ranging effects of the ban. Vintners say it will stop smoking in some publicans' private homes.

The Irish Hospitality Industry Alliance, meanwhile, has sent a letter to Brussels calling for the ban to be delayed for a year until a number of key issues have been addressed.

APPENDIX I

HEADLINES FROM IRISH TIMES NEWSPAPER- (Wednesday 29.10.2003)

Vintners threaten to ignore smoking ban

APPENDIX J

QUESTIONNAIRE

Respiratory Health of Bar Workers

Interview/Questionnaire

Today's Date __ / __ / __

Name of Bar _____

Subject Name/ I.D. no _____

What is your date of birth? __ / __ / __

Gender : Male Female

How long have you worked at this bar? (YEARS) _____

How many years in total have you worked in the bar trade? _____

On average, how many hours per week do you work at this bar? ____

The next questions ask about breathing symptoms that you might have had during the past 4 weeks.

Have you had wheezing or whistling in your chest at any time during the last 4 weeks?
No / Yes

Have you felt short of breath? No / Yes

In the last 4 weeks, do you usually cough first thing in the morning?
No / Yes

Do you cough at all during the rest of the day or night? No / Yes

Do you bring up any phlegm? No / Yes

The next few questions ask you about eye, nose, or throat irritation during the past 4 weeks.

In the past 4 weeks, have your eyes been red or irritated? No/Yes

Have you had a runny nose, sneezing, or nose irritation? No / Yes

Have you had a sore or scratchy throat? No / Yes

The next few questions ask you about your personal smoking habits.

Have you ever smoked a cigarette? No / Yes

[IF YES] Do you currently smoke cigarettes regularly? (By "regularly" I mean on most days or nights). No / Yes

[IF YES] How many packs do you smoke per day? __

The next questions ask about your exposure to other people's tobacco smoke.

Do you live in the same household with someone who smokes tobacco?
No / Yes

During the past 7 days, how many hours per week were you exposed to other people's smoke at work? _____

Including home, work, and other regular activities, how many total hours were you exposed to other people's tobacco smoke during the past 7 days? _____

The next two questions ask you about whether you have asthma.

Has a doctor ever told you that you have asthma? No / Yes

[IF YES] Do you currently take medicine for asthma? No / Yes

[IF YES] Are they prescription or over-the-counter medicines? No / Yes
Name (if known): _____

Please provide the following contact information, so that we can mail you your breathing test results after the study is completed.

Contact Telephone Number _____

Home Address _____

_____

THANK YOU VERY MUCH FOR PARTICIPATING IN THIS STUDY. WE APPPRECIATE YOUR TIME VERY MUCH.

APPENDIX K

LUNG FUNCTION TEST REPORT

Respiratory Laboratory,
St. James's Hospital, Dublin 8
Phone : 4162794

Pulmonary Function Analysis

ID: [redacted]
Last Name: [redacted]
First Name: [redacted]
Street: [redacted]
City: [redacted]
Physician:
Diagnosis:

Birth Date: 02/11/57
Height(cm): 174
Weight(kg): 96.0
Armspan(cm): 0
Room: STUDY
Medication:
Medication:

Smoker: No
How Long:
Quit: No
Stopped:
Technician: M AGNEW

Date: 15/11/04

Spirometry

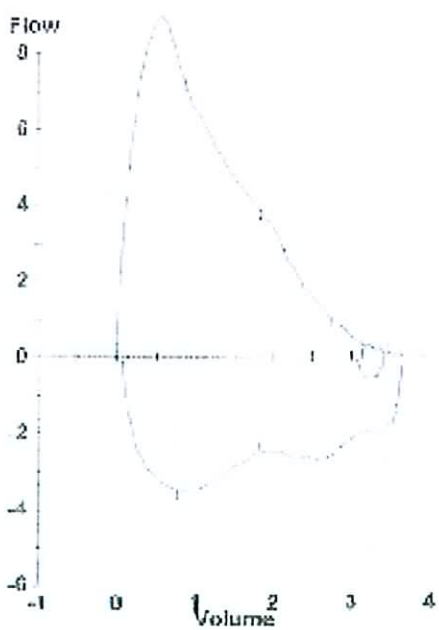
		Ref	Pre Meas	Pre % Ref	Post Meas	Post % Ref	Post % Chg
FEV1	Liters	3.63	2.96	82			
FVC	Liters	4.46	3.66	82			
FEV1/FVC	%	79	81				
PEF	L/sec	8.81	9.37	106			
FEF25-75%	L/sec	4.05	2.98	73			
FEF50%	L/sec	4.79	3.84	80			
FIF50%	L/sec		2.47				

Lung Volumes

		Ref	Pre Meas	Pre % Ref
RV	Liters	2.08	1.85	89
TLC	Liters	6.82	5.64	83
RV/TLC	%	32	33	
FRC N2	Liters	3.40	2.25	66
ERV	Liters		0.40	

Diffusion

		Ref	Pre Meas	Pre % Ref
DLCO	mL/mmHg/min	30.5	29.9	98
DLCO/VA	mL/mHg/min/L	4.47	5.52	123
IVC	Liters		3.70	
VA	Liters	6.82	5.42	79
BHT	Sec		11.38	



APPENDIX L

POSTER THAT WON 'BEST CLINICAL POSTER' AT IRISH THORACIC SOCIETY MEETING, GALWAY, NOVEMBER 2005.

Study poster that won 'Best Clinical Poster' award at the Irish Thoracic Society Meeting, November 2005.

The effects of the workplace ban on smoking on the lung function of bar workers in Dublin.

Agnew, M[1], Goodman, P[2], Clancy, Luke[1,3]. 1-Dept. Resp. Med. St. James's Hospital, Dublin. 2-Dept. Physics, D.I.T., Kevin Street, Dublin 3-Research Institute for a Tobacco Free Society.

Background

It has long been recognised that exposure to environmental tobacco smoke(ETS) causes respiratory and cardio-vascular disease in those exposed to it. Bar staff are a group of workers with long hours of exposure at work, and as such, were an ideal group to study the effects of the government ban on smoking in the workplace.

Methods

Bar workers were recruited through their Trade Union, Mandate, and 81 participated in the pre-ban phase of testing between September 2003 and March 2004. They attended the Respiratory Laboratory in St. James's Hospital for lung function tests and measurement of exhaled carbon monoxide (CO). They also completed a questionnaire relating to their respiratory health, smoking history, and ETS exposure.

75 (93%) returned between September 2004 and March 2005 for repeat testing and again completed the questionnaire.

As 2 had changed their smoking status on return, they were excluded from analysis.

Subject demographics

34(47%) were never smokers
31(42%) were ex-smokers
8(11%) were current smokers

Results - CO

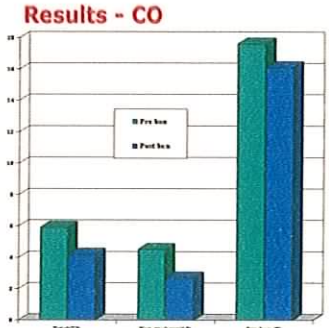
Mean Exhaled (CO)	Pre ban	Post ban	% Change	P value
Total group (n=73)	5.9ppm	4.2ppm	-29%	0.000
Never smokers (n=34)	4.21ppm	2.5ppm	-41%	0.0001
Ex smokers (n=31)	4.84ppm	2.9ppm	-40%	0.0001
Smokers (n=8)	17.6ppm	16.1ppm	-8.5%	0.623

Results - lung function

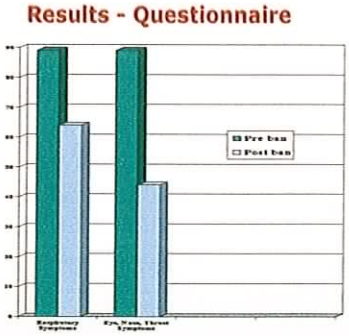
% Change	Total (n=73)	Never Smokers(n =34)	Ex smokers(n =31)	Smokers(n =8)
FEV1	1%	2%	-1%	-5%
FVC	3%	5%	3%	-3%
PEF	3%	6%	2%	-2%
TLC	2%	3%	2%	2%
DLCO (corr. %COHb)	1%	5%	-1%	-6%

Underlined = significant

Results - CO



Results - Questionnaire



Conclusion The workplace ban on smoking has resulted in a reduction in exhaled CO and an increase in lung function parameters in bar workers as well as a reduction in upper and lower respiratory symptoms . It is a positive move to improve the longterm health of workers.

APPENDIX M

LETTER OF AWARD FROM BOEHRINGER INGELHEIM

Boehringer Ingelheim

Boehringer Ingelheim Ireland Limited

13 November 2005

Boehringer Ingelheim/Irish Thoracic Society Travelling Fellowship 2005

Dear Ms. Agnew

On behalf of Boehringer Ingelheim and the Irish Thoracic Society, I wish to congratulate you on winning the prize for the best clinical poster in the competition for the Boehringer Ingelheim/Irish Thoracic Society Travelling Fellowship 2005.

A payment of €2,000 is enclosed. I would be grateful if you could confirm receipt of this payment.

Yours sincerely,

Colm Edwards

J Colin Edwards, PhD
Head of Medical Affairs and Clinical Research, Ireland
Encl.

Our reference
ITS prize

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APPENDIX N

PAPER : 'WORKPLACE SMOKING BAN INTERVENTION: EFFECTS ON BARWORKERS' RESPIRATORY HEALTH AND AIR QUALITY IN DUBLIN PUBS'.

WORKPLACE SMOKING BAN INTERVENTION: EFFECTS ON BARWORKERS' RESPIRATORY HEALTH AND AIR QUALITY IN DUBLIN PUBS.

Goodman P[1], Agnew M[2], McCaffrey M[3], Paul G[4], Clancy Luke[5],

1. **Dr Patrick Goodman PhD, DIT, Kevin Street, Dublin 8.**
2. **Michele Agnew, Chief Technologist, Respiratory Laboratory, St James's Hospital, James's Street, Dublin 8**
3. **Marie McCaffrey, EHO, HSE, Dublin**
4. **Gillian Paul, M.Sc, SRN, Trinity College, Dublin.**
5. **Professor Luke Clancy, M.D.FRCPI, Research Institute for a Tobacco Free Society, Digital Depot, Thomas Street, Dublin 8.**

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Abstract

Background

Environmental tobacco smoke (ETS) causes disease in non-smokers. Workplace bans on smoking are advocated as important interventions to reduce exposure to ETS in an effort to prevent harmful health effects. In pursuance of a policy to create a Tobacco Free Society the Irish Government on the 29th March 2004 introduced the first national comprehensive legislation banning smoking in all workplaces including bars and restaurants. This study examines the impact of this legislation on air quality in pubs and on respiratory health effects in barworkers in Dublin.

Methods

Exposure study

Concentrations of PM2.5 and PM10 particulate matter in 42 pubs were measured and compared before and after the ban. Benzene concentrations were also measured in 26 of the pubs.

Health effects study

Eighty one (81) barmen volunteered to have full pulmonary function studies, exhaled breath carbon monoxide (CO) and salivary cotinine levels performed before the ban and repeated one year later after the ban. They also completed questionnaires on exposure to environmental tobacco smoke (ETS) and respiratory symptoms on both occasions.

Findings

Pub air study

There was an 83% reduction in PM2.5 and an 80.2% reduction in Benzene concentration in the bars.

Barmen study

There was a 79% reduction in exhaled breath CO and an 81% reduction in salivary cotinine. There were statistically significant improvements in measured pulmonary function tests (PFTs) and significant reductions in self reported symptoms and exposure levels in volunteer non-smoking barmen after the ban.

Conclusions

A total workplace smoking ban results in a significant reduction in air pollution in pubs and an improvement in respiratory health in barmen.

INTRODUCTION

On 29th March 2004 the Irish Government introduced the world's first comprehensive national ban on workplace smoking (1). Ten years of partial and voluntary controls on workplace exposure to secondhand smoke had failed to protect all workers (2). Two all-party parliamentary committees reporting in 1999 (3) and 2001 (4) had recommended a total ban. The Public Health (Tobacco) Act 2002 and the Public Health (Tobacco) (Amendment) Act 2004 which followed (1), prohibits smoking in indoor workplaces, including bars and restaurants in order to reduce the risks to workers' health. A number of other European countries Norway, Italy, Sweden and Scotland have subsequently introduced similar bans. Northern Ireland, England and Wales plan to introduce bans in 2007 and France in 2008.

Interventions, which aim to reduce exposure to known air pollutants, can be expected to result in risk reduction (5, 6). Nevertheless there are few studies that have assessed health benefits associated with a workplace smoking ban (7-11).

The benefits that accrue depend on the extent to which the intervention succeeds in reducing exposure and on the response of those exposed. The national smoking ban afforded a unique opportunity to assess the effects of the ban, both on the exposure to ETS in bars, and also to evaluate any health benefits in a group of volunteer barmen.

Self reporting of changes in symptoms is interesting and important but it was felt that it was necessary to validate these observations with quantitative measurements of changes in markers of exposure and in pulmonary function. Changes in pulmonary function, exhaled breath carbon monoxide and salivary cotinine, as markers of exposure, as well as self reported respiratory symptoms and self reported exposure level changes were measured in 81 barmen pre and post the workplace-smoking ban.

It was also important to know that the banning of smoking had the expected effect on air pollution in pubs and to quantify these changes.This study measures the changes in exposure to environmental tobacco smoke (ETS) in 42 pubs.

Some of the results obtained have been published in abstract form (12, 13).

Methods

Exposure levels were measured in Dublin pubs (n=42) prior to the introduction of the smoking ban, and repeated in the same venues one year later.

Volunteer bar staff (n=81) were recruited through their trade union Mandate to partake in the health effects aspect of the study.

Exposure Assessment

In the greater metropolitan area of Dublin, a group of 42 public houses, licensed to serve alcohol, were studied. The venues were selected to encompass a wide variety of building structures, clientele and were a selection of central, north and south city locations. Size, demographics and socio-economic factors were considered in the selection as well as geographic location and size. This approach was pursued to ensure that a representative sample of the different types of public houses found in Dublin city was obtained.

Based on these criteria the sample consisted of 21 pubs with capacity greater than 50 customers and 21 with capacity less than 50 customers, 14 were located in the city centre, 15 were on the north city suburbs and 13 were on the south city suburbs.

Concentrations of PM2.5 and PM10 particulate mater in 42 pubs were measured for a minimum period of 3 hours inside each venue, using a real time optical based light scattering instrument (Aerocet 531) (14), with readings being taken every two minutes throughout the monitoring period. Concurrent measurements of ambient benzene levels were also recorded, using a passive absorption diffusion tube, identical to those used in the People project (15). The benzene samplers were available only for the last 26 pubs monitored, they were analysed by the joint research centre (JRC) laboratory of the EU at Ispra.

The monitoring protocols adopted, involved the locating of the monitoring instruments at the centre of the room, at table height. The dimensions of each venue were noted, as well the number of doors, and whether any ventilation system was in operation. In addition, the number of people present was recorded each hour, and also the number of people who were smoking. The levels of

PM10 and PM2.5 were also recorded outside the premises both before and after the indoor monitoring for both pre and post ban parts of the study.

The 42 pubs were visited between October 2003 and March 2004 when the pre ban exposure measurements were recorded, and re-visited one year later to measure the post ban exposure levels. The follow-up measurements were made on the same day of the week, and at the same time of day, and in the same month, one year on from the original measurements. This controlled for the day of the week, the month (seasonal pattern) and the time of the day effects for each venue. The outside measurements were also repeated post ban as in the preban period for comparability of prevailing ambient air pollution levels.

Health effects methodology

Eighty one (81) volunteer bar staff were recruited through their trade union Mandate to participate in the health effects study, having responded to a request by letter from us, which was circulated by Mandate to its membership. We accepted every worker who volunteered in time to allow us to complete the tests before the introduction of the ban but would have enlarged the study if there had been more volunteers. No financial inducements were offered. The volunteers were all male. Mandate has approximately 1100 members of whom some 80% are male. Most of the female members are temporary and or part time workers. We do not know why there were no female volunteers but suspect that their status as described may have influenced their decisions as the employers were vehemently anti the ban and warned of job losses (16).

It was decided for reliability and quality control considerations that all subjects would be assessed in a recognised Pulmonary Function Laboratory rather than performing limited breathing tests in the workplace or at home. This allowed us to measure a

wider range of Pulmonary Function Tests (PFTs) than would have been possible off site but may have limited the numbers of volunteers. On the other hand it allowed the barmen participate without the involvement of their employers.

We measured the following parameters: Forced expiratory flow at one second (FEV1,) Forced vital capacity (FVC), Forced expiratory flow (FEF) 25-75 Peak expiratory flow rate (PEF), Residual volume (RV), Total lung capacity (TLC) and Diffusion capacity for carbon monoxide_(DLCO) using a Sensormedics Vmax machine. In addition, PEF was also measured using a Piko 1 peak flow meter. Exhaled breath Carbon Monoxide (CO) was measured using a Micro Medical Micro CO meter and carboxyhaemaglobin (COHB %) was calculated. All of the PFTs, pre and post ban were conducted by a single experienced respiratory technologist (M.A.) and were done in accordance with ERS guidelines (17, 18).

The volunteers attended St James's Hospital, between September 2003 and March 2004 for the pre-ban measurements, the follow-up measurements were conducted one year later, between September 2004 and March 2005.

While at the hospital laboratory they were administered the IUATLD and CEPA (20) questionnaires relating to their respiratory and sensory symptoms, similar to that used by Eisner et al (7). Non-stimulated salivary samples for cotinine analysis were also obtained at the laboratory visits pre and post the ban by a single investigator (GP) and processed as described by Allwright et al (9).

Analytical methods

Air measurements

The mean mass concentrations of PM2.5 and PM10 particulate matter for each venue were analysed using the paired –sample T test procedure comparing the means of the quantitative pairs of variables using SPSS software (v 11.0).

Health effects

For the purpose of analysis, the volunteer bar staff were categorised as "never-smokers" (n=34), "ex-smokers" (n=31) and "current smokers" (n=8). The pulmonary function test results were also analysed for each parameter by comparing the predicted score at the pre and post ban periods using the paired – sample T test procedure.

McNemar's Nonparametric Test for two related dichotomous variables for changes in responses using the chi-square distribution was used for the questionnaire data, where a volunteer reported the absence or presence of a symptom.

Markers of exposure

As the data for Carbon monoxide (CO) and Cotinine exhibited skewed distributions a non-parametric test (Wilcoxon Signed Rank) was applied to test any significant differences between the pre and post-ban CO and cotinine levels.

Exposure Results

The exposure results as measured inside the 42 bars show a statistically significant decrease following the introduction of the ban (Table 1). Complete pre and post ban benzene measurements were available for 26 pubs and also show a statistically significant decrease following the introduction of the ban (Table 1).

The ambient outdoor PM levels as measured outside each venue do not show any significant change between the pre and post ban periods (Table 1). The reduction in PM10 inside the bars was not statistically significant. These results indicate that tobacco smoke was the major contributor to both PM2.5 and benzene levels in pubs prior to the introduction of the workplace smoking ban.

There was no smoking observed inside any of the 42 bars visited in the post ban period confirming full compliance.

Health effects results

The 81 volunteers, 10 current smokers, 34 ex-smokers and 37 never smokers completed a full set of Pulmonary Function Tests (PFTs) pre ban, with 75 completing the post ban measurements. Two subjects had changed their smoking status during the course of the study and were excluded from the analysis leaving 73 bar staff (90%) that completed the study and were suitable for analysis. All of the volunteers were males, working full time in pubs as their main form of employment. They had a mean age of 47.9 (22-68) years at the pre ban assessment. Between them, they had 2298 yrs of exposure to ETS in their place of work (mean 28.4yrs) (range 6 – 52yrs). The mean self reported workplace exposure to ETS was 40.5 hrs pre ban and 0.42 hrs post ban showing a 99% reported decrease in exposure at work. The total ETS exposure was 46.9 hrs pre ban and 4.2 hrs post ban showing a 90% decrease in total exposure. The exposure to ETS outside of work decreased from 6.4 hrs pre ban to 3.7 hrs post ban (% change -42%, p =<0.01). This is of interest as some feared that the ban could lead to increased exposure outside of work (21). FVC increased significantly in never-smokers and ex-smokers, while it declined in current smokers. While FEV1 did not change significantly in any group it tended to

increase in nonsmokers.The TLC increased in never- smokers and ex-smokers but not in smokers. Peak Flow increased significantly in never-smokers, while the increase in ex-smokers was not significant and it tended to decline in current smokers (Table 2). The FEF25-75 decreased in never-smokers and ex-smokers and was unchanged in smokers. There was no statistically significant change in RVs in any group although the RVs of smokers tended to increase (Table 2). The mean DLCO and the DLCO corrected for %COHB show a statistically significant improvement of 5% for the never-smokers group, while the reduction in ex-smokers and the smokers was not statistically significant. (Table 2)

Exhaled breath carbon monoxide (CO) median values, with inter-quartile ranges (IQR) were ppm: 4.0(3, 5) and 2.0(2, 3) in pre and post-ban respectively, difference (-4.8) and is statistically significant ($p<0.001$) Fig1.

Salivary cotinine ng/ml median values, with inter-quartile ranges (IQR) were 5.1 (3.4, 7.6) in pre-ban and 0.6 (0.3, 1.3) in post-ban, difference (-6.1) is also statistically significant ($p<0.001$) Fig1.

Median exhaled breath CO and salivary cotinine levels decreased by 79% and 81% respectively in never and ex-smokers but did not change significantly in current smokers

Questionnaire results

The questionnaire results obtained in this study (Table3, Table4) showed significant improvements in cough and phlegm production in non- smokers (never and ex-smokers combined) but not in smokers whereas sensory irritant symptoms were improved in all subgroups but smokers benefited less.

Discussion

This study shows that the workplace smoking ban in Ireland has significantly reduced the levels of both particulate matter and benzene in the air in pubs. There was a dramatic reduction in exhaled carbon monoxide levels and in salivary cotinine in barmen. The health of non-smoking bar staff has improved in terms of pulmonary function, respiratory and irritant symptoms while in smokers only irritant symptoms have improved with other measured parameters showing a decline in the same period.

The rationale for using particles as markers of air pollution by secondhand smoke is that it is known that particles in this size range are responsible for excess mortality. We had previously shown that reduction of particle levels in ambient air resulted in marked health benefits in terms of respiratory and cardiovascular mortality (5, 6). It has been reported (22) that ETS particles are in the size range 0.01 to 0.67 μgm^{-3}. The pre ban concentrations of PM2.5 are comparable with the findings of Levy et al (23), Lung et al (24) and to those reported by Repace (25). Repace however reported values for PM3.5 and the exposures relate to 8 venues, all sampled during the same evening where the sampling period used was significantly shorter than that used in Dublin. These results confirm that the approach of a total ban on smoking in the workplace is successful in reducing the exposure of workers to particles. Previous studies (26, 27) have shown that partial bans do not work in this regard.

The volatile hydrocarbon benzene was used as a marker for carcinogenic substances as cigarette smoke is a well known source and we had already established ambient outdoor levels for benzene in Dublin. The post ban levels were similar to ambient air levels suggesting that the external contribution to

indoor pub air benzene was not the source of the high levels seen preban. The reduction in benzene levels after the ban is similar to the drop in Poly Aromatic Hydrocarbons (PAH) reported by Repace (25).

The duration of monitoring was considered important as the particle levels vary with the number of customers smoking at any time and with the variation in air movement (Fig. 2) and short sampling times may therefore be unreliable as an indicator of overall exposure. Repace (25) reports on the change in particulate levels in hospitality venues in Delaware pre and post a smoking ban, where he observed a 90% drop in PM3.5 levels, which he attributed to ETS. The findings in this study for PM2.5 are similar and consistent with those reported from Delaware. They are also consistent with the results presented by Mulcahy et al (28) who reported a drop in PM2.5 values for the pre and post ban exposures as measured at 9 public houses in Galway, Ireland, measuring for 4 minutes in each venue. Mulcahy et al (29) also reported on cotinine and nicotine levels pre and post the Irish ban. There is as yet no agreed gold standard for the most appropriate markers or protocols for measurement of ETS (30) exposure but these recent studies show encouraging agreement.

This study has also served to show that a workplace ban on smoking can have immediate beneficial effects on respiratory health. The acute improvements in self reported respiratory and irrative upper airway symptoms are supported by the measurements of pulmonary function. A significant improvement in forced vital capacity (FVC) and in gas diffusion (DLCO) suggests a real health gain. The somewhat counterintuitive findings of an apparent decline in small airway function as reflected in the subdivisions of flow volume loops may have to do with altered mechanics in small airways as suggested by the increase in FVC

and TLC in non-smokers and ex-smokers (Table 2) resulting in changed volume history. A similar finding seems to have occurred in the California study (7). It may also represent the reopening of small airways previously closed contributing air at a lower flow rate. The results including an increase in DLCO seem however more in favour of an improvement in a mild restrictive effect of ETS than any change in an obstructive component.

The dramatic drop in exhaled breath CO may be of significance in terms of the short term reduction in acute myocardial infarction seen in other studies but we do not have information of that in our study (8, 11).

The longer-term health benefits such as in COPD, asthma, and cardiovascular disease need more prolonged studies but can be expected to occur given the known harmful effects of secondhand smoke (31). The reduction of benzene may be an indication of a reduction in the many other known carcinogens in secondhand smoke and may contribute to a reduction in lung cancer.

The cultural and social effects of this workplace ban on smoking are likely to be profound. Earlier incomplete bans such as the Finnish ban (32) have shown significant changes however the Irish ban implemented to protect workers including all service workers recognises the need for a change of mindset as regards all indoor spaces. Early results already show a significant change in attitude in smokers with a majority of smokers now favouring the ban (33). Smoking prevalence estimates show a decline in smoking of 1.4% (34) which is more than three times the average OECD expected rate of decline in the same timeframe (35). Results from data routinely collected by the Central Statistics Office show that employment in the hospitality sector has increased again following an initial drop and that tourism has also increased despite the

predictions preban (36). Although smoking outside pubs is a new noticeable occurrence post ban, limited data suggests that smoking outside pubs by customers visiting pubs is only a fraction of the numbers who smoked inside pubs preban (37).

The health effects results of this study are weakened by the fact that the bar workers were all volunteers and may not be fully representative of the exposed population. They were also all male. The sample size represents only some 10% of the male membership of the Mandate Dublin Trade Union. In addition it was not possible to match the bar staff to the various pubs used as part of the exposure assessment as the pubs were selected as a representative sample of Dublin pubs to show how the levels of exposure changed over a whole series of venues and the overlap with the volunteers was uncontrolled and only partial. The close correlation of the self reported improvements in symptoms and reduction in exposure with the measured improvements in pulmonary function and markers of exposure is reassuring and extends our experience of the beneficial effects of workplace bans.

We conclude that a properly implemented comprehensive workplace ban on smoking as introduced in Ireland can achieve its primary aim. It can protect workers and others from exposure to the harmful particles, chemicals and gases in secondhand smoke and result in immediate and significant health gain.

Acknowledgements

The study was supported by grants from the Dept of Health and Children, OTC, ENSP, and RCD Hospital Trust.

We wish to thank the volunteers who took part in the study and their trade union Mandate who facilitated this process.

The questionnaire data and salivary cotinine samples were collected as a joint project with Shane Allwright and colleagues.

We would like also to thank Kathleen Bennett, Zubair Kabir and Laura Currie for statistical advice and for help with the manuscript.

We thank Dr Emile DeSager from the Joint Research Centre of the European Union (JRC) (ISPRA) for facilitating the benzene measurements, and Mr Richard Connolly also of the JRC who conducted the laboratory analysis of the benzene samplers.

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Table 1: $PM_{2.5}$, PM_{10} & benzene levels in public houses and the outdoor environment pre and post the introduction of the workplace smoking ban (µg/m-3).

	Pre-ban (sd)	Post-ban (sd)	%Change	P-value
Public Houses (n=42)				
Ave $PM_{2.5}$	35.5 (17.8)	5.8 (2.2)	-83.6%	<0.01
Ave PM_{10}	72.1 (27.8)	45.5 (17.1)	-36.9%	NS
Benzene (n=26)	18.8 (14)	3.72 (1.6)	-80.2%	<0.01
Outdoor (n=42)				
Ave $PM_{2.5}$	6 (0.8)	5.2 (0.1)	-13.6%	NS
Ave PM_{10}	24.1(9.3)	20 (5)	-17.4%	NS
Benzene (Peoples project)		3.5		

Table 2: Respiratory parameters and the change by smoking status pre and post introduction of the workplace smoking ban.

	Total (n=73)			Never-Smokers (n=34)			Ex-Smokers (n=31)			Current-Smokers (n=8)		
Parameters (Units)	Pre	Post	P-value	Pre	Post	P-value	Pre	Post	P-value	Pre	Post	P-value
FEV1 (l/sec)	3.42	3.41	--	3.44	3.49	--	3.38	3.35	--	3.51	3.32	--
% PRED	92.0	93.0	NS	92.0	94.0	NS	93.0	93.0	NS	88.0	84.0	NS
FVC (l)	4.21	4.32	--	4.17	4.36		4.18	4.29	--	4.45	4.31	--
% PRED	92.0	95.0	<0.01	91.0	96.0	<0.01	93.0	96.0	0.01	91.0	88.0	NS
FEV1/FVC % PRED	81.0	78.0	<0.01	82.0	80.0	<0.01	81.0	78.0	<0.05	79.0	76.0	0.03
PEF (l/min)	500.7	508.8	--	506.6	530.0	--	505.7	515.0	--	489.1	481.3	--
% PRED	94.0	97.0	<0.01	94.0	99.5	<0.01	96.0	98.0	NS	86.4	85.0	NS
FEF25-75 (l/sec)	3.50	3.24	--	3.68	3.41	--	3.42	3.11	--	3.41	3.20	--
% PRED	87.0	80.0	<0.01	89.0	83.0	0.04	87.0	79.0	<0.01	78.0	73.0	NS
RV (l)	2.14	2.17	--	1.98	1.97	--	2.20	2.24	--	2.54	2.70	--
% PRED	99.0	100.0	NS	94.0	93.0	NS	101.0	101.0	NS	115	123	NS
TLC (l)	6.42	6.55	--	6.24	6.38	--	6.46	6.58		7.03	7.10	--
% PRED	91.0	93.0	<0.01	90.0	92.0	0.02	92.0	94.0	0.04	95.0	96.0	NS
DLCO (ml/min/mm Hg)	28.7	28.5	--	27.9	29.5	--	28.9	28.7	--	29.2	27.2	--
CORR DLCO	29.1	28.7	--	28.1	29.6	--	29.2	28.8	--	30	27.8	--
CORR DLCO % PRED	93.0	94.0	NS	90.0	96.0	<0.01	95.0	95.0	NS	88.0	83.0	NS

Table 3: Respiratory symptoms questionnaire data pre and post workplace smoking ban by smoking status.

	Number Reporting Symptom			
Q1. Have you had whistling/wheezing in your chest?	**Pre-ban**	**Post-ban**	**% Change**	**P-value**
Total non-smokers (65)	18 (28%)	15 (23%)	-17%	NS
Smokers (8)	6 (75%)	5 (63%)	-17%	NS
Q2. Have you felt short of breath?				
Total non-smokers (65)	18 (28%)	10 (15%)	-45%	NS
Smokers (8)	4 (50%)	3 (38%)	-25%	NS
Q3. Do you usually cough first thing in the morning?				
Total non-smokers (65)	21 (32%)	11 (17%)	-48%	0.04
Smokers (8)	6 (75%)	6 (75%)	0	NS
Q4. Do you cough at all during the rest of the day?				
Total non-smokers (65)	36 (55%)	22 (34%)	-39%	<0.01
Smokers (8)	7 (88%)	7 (88%)	0	NS
Q5. Do you bring up phlegm?				
Total non-smokers (65)	44 (68%)	26 (40%)	-41%	<0.01
Smokers (8)	7 (88%)	6 (75%)	-14%	NS
Total reporting any respiratory symptom?	63 (86%)	45 (61%)	-28%	<0.01

Table 4: Irritant symptoms questionnaire data pre and post workplace smoking ban by smoking status.

	Number Reporting Symptom			
	Pre-ban	Post-ban	% Change	P-value
Q1. In the past 4 weeks have your eyes been red/ irritated?				
Never smokers (34)	20 (59%)	5 (15%)	-75%	<0.01
Ex-smokers (31)	21 (68%)	2 (6%)	-90%	<0.01
Smokers (8)	3 (38%)	1 (13%)	-67%	NS
Q2. Have you had a runny nose, sneezing, or nose irritation?				
Never smokers (34)	22 (65%)	11 (32%)	-50%	<0.01
Ex-smokers (31)	12 (39%)	9 (29%)	-25%	NS
Smokers (8)	8 (100%)	4 (50%)	-50%	0.03
Q3. Have you had a sore or scratchy throat?				
Never smokers (34)	16 (47%)	7 (21%)	-56%	<0.01
Ex-smokers (31)	15 (48%)	5 (16%)	-67%	<0.01
Smokers (8)	4 (50%)	2 (25%)	-50%	NS
Total reporting any irritant symptom?	64 (87%)	32 (43%)	-50%	<0.01

Figure 1: Whisker plots showing medians and interquartile ranges (IQR) of CO levels (ppm) and cotinine levels (ng/ml) before and after the workplace smoking ban (n=73).

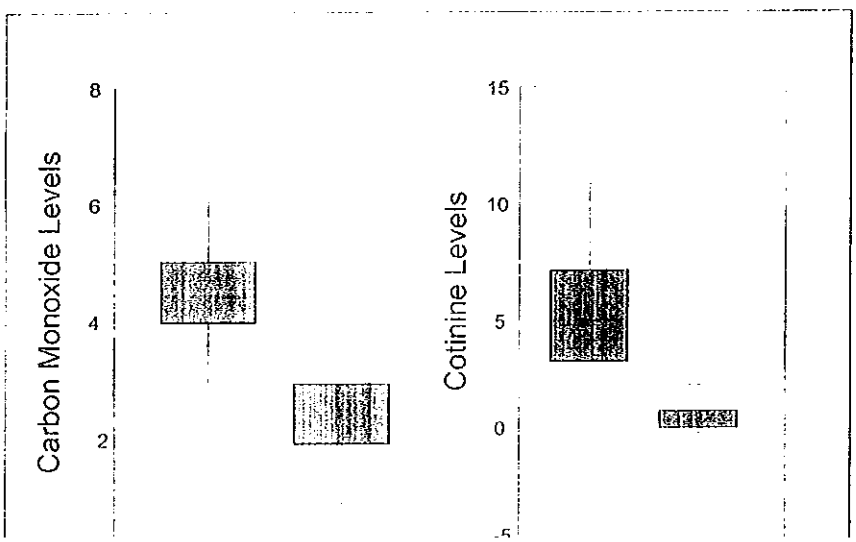

Figure 2: Example showing variation of $PM_{2.5}$ levels during an evening pre and post introduction of the workplace smoking ban in a Dublin pub.