



# **Are Public Smoking Bans Necessary?**



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## INTRODUCTION<sup>1</sup>

*The great tragedy of Science –  
the slaying of a beautiful  
hypothesis by an ugly fact.*

Thomas H Huxley  
English biologist (1825-1895)

**G**iven that the UK public smoking ban, introduced in July 2007, is due for a review in 2010, it is appropriate to revisit the rationale advanced in favour of this controversial measure.

The champions of public smoking bans, both here in the UK and elsewhere, have argued that such bans are justified on the basis of four facts:

1. Environmental tobacco smoke (ETS) poses fatal health risks for nonsmokers.
2. Such bans encourage smoking cessation and reduced smoking initiation.
3. There are no acceptable alternatives to such bans.
4. There are no perverse and unintended consequences arising from the bans.

This paper examines each of the four reasons cited by proponents of the ban in an effort to determine their validity.

## ENVIRONMENTAL TOBACCO SMOKE & NONSMOKERS' HEALTH

**T**he first and most significant reason advanced as a justification for public smoking bans in the UK is that environmental tobacco smoke (or secondhand, or passive smoke) poses fatal risks to the health of *all* nonsmokers. It should be noted that the health risks to nonsmokers allegedly associated with ETS go beyond lung cancer and heart disease. For example, some of the other

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health risks that are claimed to be linked to ETS exposure are the exacerbation of asthma and respiratory infections in young children. These risks, however, are never cited as providing the decisive reason for public smoking bans. Rather, it is the risks for lung cancer and heart disease associated with ETS exposure that are routinely cited as the definitive justification for smoking bans.

The scientific support for such claims about the risks from ETS comes from a series of epidemiological studies conducted over the last 30 years that have examined the relationship between ETS exposure (in home, social, and occupational settings) in healthy nonsmokers and diseases such as lung cancer and heart disease. In the case of lung cancer, there have been 76 such studies and 42 studies on the association between ETS exposure and heart disease. In addition to these individual studies, there have been at least 20 meta-analyses of the ETS data, as well as numerous reports by public health agencies and governments, all of

which have been also been used to justify public smoking bans.

For the proponents of public smoking bans, the scientific evidence about the risks of ETS exposure for healthy nonsmokers is definitive. In its 2008 consultation paper on the *Future of Tobacco Control*, the UK Department of Health argues that, 'Exposure to second-hand smoke is a serious health hazard, and there is no safe level of exposure'.<sup>1</sup> According to the US Surgeon General, the 'science is clear' and the 'debate is over'.<sup>2</sup>

Dr Richard Carmona, the US Surgeon General report's author, echoes the UK Department of Health by claiming that, 'Breathing secondhand smoke for even a short time can damage cells and set the cancer process in motion'. He says, 'There is no risk-free level of secondhand smoke exposure'. But a careful examination of the methodology used in the relevant studies, the actual scientific evidence, itself, and the findings of various government and

public health reports suggests that this is not the case.

## METHODOLOGY OF ETS STUDIES

**E**nvironmental tobacco smoke research is bedeviled, some would suggest fatally, by two problems. The first of these is what Dr Gio Bata Gori, the leading epidemiologist and toxicologist, has called the 'measurement problem'.<sup>3</sup> Scientific claims about risk depend on accurate, reliable, and replicable measurements about exposure to a risk. In the case of ETS, this means accurate measurements about the exposure of nonsmokers to ETS. Yet, claims about the effects of ETS on nonsmokers provide no actual measurements of exposure but, rather, are based on recall studies, generally at the end of life, in which nonsmokers are asked to recall and estimate their childhood and adult exposure to secondhand smoke. As Gori observes:

Epidemiologists traditionally interpret such recalls as 'measurements'. Yet, by any factual standards – scientific or otherwise – this qualification is not sustainable, because the *sine qua non* of statistical elaborations is that discrete characteristics of individuals must have been physically measured using the same meter, that the measurement error is known from prior testable experience, or that it has been determined in the study at hand by multiple measurements of each characteristic, on a sufficient number of subjects.

In the International Agency for Research on Cancer (IRAC) European Multicentre Case-Control Study of Lung Cancer in Nonsmokers, for example, subjects were asked to recall how often their parents or other people living in the home smoke, how many hours they spent in a room with tobacco smoke, and how smoky the room was. Nonsmokers living with smokers were asked to remember how many cigarettes their spouse smoked over the years and how many hours a day they spent exposed to such smoking. Given that most people have considerable

difficulty accurately remembering even recent events, it is highly unlikely that these 30, 40, and 50 year-old recollections are accurate. They certainly fail to provide scientific measurements of ETS exposure. Yet, is these imprecise and scientifically unverifiable and unreplicable memories that provide the measurement basis for the supposedly very precise risks cited as justifying public smoking bans.

The second problem is that these studies are plagued by sampling errors, confounders, biases, and misclassifications of smoking status (when a smoker is in fact classified as a nonsmoker) that can only be subjectively, as opposed to objectively, adjusted for. Consider, for instance, the multiplicity of risk factors for both lung cancer and heart disease – 30 for the former and over 300 for the latter.

Since no ETS study to date has controlled for more than a handful of these, there is no legitimate basis for claiming that the association between the disease and ETS exposure is

genuinely a result of ETS, as opposed to some other risk factor.

## INDIVIDUAL ETS STUDIES

**T**he results of the individual ETS studies fail to establish that ETS exposure is associated with lung cancer and heart disease in healthy nonsmokers. Of the 76 studies that have examined the relationship between ETS and lung cancer in nonsmokers in home, occupational, and social settings, only 15 show a statistically significant association. Of the 42 studies on the relationship between ETS and heart disease in nonsmokers in home, occupational, and social settings, only 18 show a statistically significant association.

Especially relevant in terms of public policy, of the 24 studies that have examined ETS exposure and lung cancer risks among nonsmokers exposed to ETS in the workplace, 19 find no statistically significant association. Moreover, the relative risk reported in the studies

showing a statistically significant association between ETS exposure and heart disease is 1.3. This is a peculiar finding given that the relative risk for heart disease in smokers is 1.7 and nonsmokers ETS exposure is 1/500<sup>th</sup> of that of active smoking.

One of the possible reasons that so few of these studies report positive associations between ETS exposure and lung cancer is that the dose that nonsmokers exposed to ETS receive, even allowing for the lack of accurate measurements, is so minimal. There is considerable evidence, for instance, that smokers who smoke 3-4 cigarettes a day have about the same lung cancer risks as nonsmokers. But nonsmokers exposed to ETS have dramatically lower dose levels than 3-4 cigarettes a day, making their cancer risks dramatically lower. Dr Geoffrey Kabat of Albert Einstein College of Medicine reports that using the results from Roger Jenkins' studies of actual ETS exposure in the United States, a nonsmoker living with a smoker would receive the same

ETS exposure as smoking eight to ten cigarettes a year.<sup>4</sup>

But dose, that is, levels of ETS exposure, also comes into the debate over public smoking bans in a different way. Underlying the use of these studies in justifying public smoking bans is the assumption, stated baldly in the Department of Health consultation, that there is *no* safe level of exposure to ETS. This, however, is simply not true, particularly in light of what is known about the way in which a variety of biological structures work to defend the body against low-level carcinogen exposure.

The scientific understanding of dose-response relationships suggests that there are thresholds below which an exposure to a carcinogen has no significant consequences. This is true, for example, with formaldehyde, arsenic, nitrosamines, and hydrocarbons. It is therefore not necessarily true that there is no threshold of safe ETS exposure.



What is striking about the primary ETS study data is that after three decades of research, the pattern of largely statistically non-significant or marginally significant results has continued unabated. This is telling since studies of genuine, as opposed to phantom, risks are usually weak at first but gain strength and clarity over time. But this has not been the case as ETS studies have continued to produce highly equivocal results. Consider two recent studies:

Enstrom and G Kabat 'Environmental Tobacco Smoke and Tobacco Related Mortality in a Prospective Study of Californians, 1960-98' *British Medical Journal* 2003 326: 1057-1066.

This study looked at 35,561 non-smoking Californians with smoking spouses. The participants were part of the American Cancer Society Cancer Prevention Study (CPS) and their lives and deaths were followed in detail from 1960-1998. The relative risk for never-smokers married to smokers was a statistically non-significant 0.94 for coronary heart disease and 0.75 for lung cancer. As the authors note about the

study, 'none of the other cohort studies...has more strengths, and none has presented as many detailed reports'.

Curiously, this study is not cited in the US Surgeon General's report on ETS or in other advocacy reports championing public smoking bans.

Stayner et al 'Lung Cancer Risk and Workplace Exposure to Environmental Tobacco Smoke' *American Journal of Public Health* 2007 97: 545-551.

This meta-analysis looks at ETS exposure in the workplace and lung cancer risk in nonsmokers. It reports a relative risk for lung cancer of 1.24 for occupationally exposed non-smokers. However, of the 25 studies in the analysis, 23 are not statistically significant, including the definitive IARC-World Health Organization workplace study with the largest number of subjects of any workplace research.

But the problem of ETS science in support of public smoking bans is not simply that most of the primary studies report findings that are not statistically

significant, but also that the relative risks, when significant, are extraordinarily weak, falling generally in the range of 1.20-2.00. Relative risks of less than 2 are generally given little weight by epidemiologists and cannot provide robust support for public policy.

As Professor Samuel Shapiro of Columbia University has observed, 'We can only be guardedly confident about relative risk estimates of the order of 2, occasionally; we can hardly ever be confident about estimates of less than 2.0, and when estimates are much below 2.0 we are simply out of business'.<sup>5</sup>

Given that most of the ETS studies of lung cancer and heart disease risk report relative risks below 2.0, the scientific basis for public smoking bans is quite clearly 'out of business'.

## GOVERNMENT AND PUBLIC HEALTH AGENCY ETS REPORTS

Several key government and public health agency reports also severely undermine the claim that ETS represents a major health risk for healthy nonsmokers.

First, the 1992 US Environmental Protection Agency report<sup>6</sup> that has been the fundamental scientific support for public smoking bans was subjected to a devastating empirical analysis in 1998 by the US District Court,<sup>7</sup> which nullified the EPA's risk assessment for ETS. In effect, the EPA's central claim that there was sufficient scientific evidence to classify ETS as a Group A human carcinogen, and as a cause of lung cancer, was rejected. While the court decision was overturned on appeal, due to technical issues, its finding of fact were left untouched.<sup>2</sup>

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<sup>2</sup> The fullest discussion of the EPA report and the court case overturning is found in G B Gori and J C Luik *Passive*

Second, the International Agency for Research on Cancer's Multi-centre Study from Europe failed to find statistically significant associations between ETS and lung cancer in childhood exposures, workplace exposures, home exposures, or social exposures.<sup>8</sup>

Third, after an exhaustive six year investigation into ETS and the demands for workplace smoking bans, the US Occupational Safety and Health Administration (OSHA), which regulates US workplaces, concluded that the 'original risk and exposure estimates [for ETS] are not valid'. Based on this, it withdrew its Notice of Proposed Rulemaking with respect to workplace ETS limits and smoking bans.<sup>9</sup>

Fourth, in 2006, the Economic Affairs Committee of the House of Lords examined at length the issues of ETS, health risks, and public smoking bans. One of the witnesses before the

committee was Sir Richard Peto, the Oxford epidemiologist. Sir Richard told the committee that ETS risks 'are small and difficult to measure directly...The exposure that one would get when breathing other people's smoke obviously depends on the circumstances, but even heavy exposure would be something like one percent of what a smoker gets, maybe in other circumstances 0.1 percent'. Sir Richard noted that he 'did not want to be cast in the role of advocating banning smoking in public places or in private places', because the relative risks from ETS are so very small. As he noted, 'the main way smokers kill people is by smoking themselves, not by killing other people'.

Based on this testimony and other expert evidence, the committee concluded that the risks of ETS are 'uncertain and unlikely to be large' and that 'the decision to ban smoking in public places may represent a disproportionate response to a relatively minor health concern'.<sup>10</sup>

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*Smoke: The EPA's Betrayal of Science and Policy* Fraser Institute Vancouver 1999.

## PUBLIC SMOKING BANS AND REDUCED SMOKING

In addition to the claim that public smoking bans are necessary to protect nonsmokers from the serious risks of lung cancer and heart disease, advocates of such bans also argue that such bans, through reducing the opportunities for smoking as well as denormalising it, serve to reduce smoking through encouraging cessation and preventing smoking initiation.

For example, Dr Thomas Farley, New York City's Health Commissioner, has recently proposed banning smoking at all of the city's parks and beaches.<sup>11</sup> Dr Farley admits that the rationale for the ban has nothing to do with any risks posed by ETS to the health of nonsmokers, but rather with preventing people, particularly children, from having to see anyone smoking in public. Farley says, 'We don't think children should have to watch someone smoking'. Farley also defends the proposed public smoking ban extension

to outdoor areas by arguing that it is 'part of a broader strategy to further curb smoking rates'. Such outdoor bans are already in place in other areas of the US.

The UK government has maintained that the public smoking ban has forced record numbers of smokers to quit. According to a July 2008 report prepared on the first anniversary of the ban by the Chief Medical Officer, Sir Liam Donaldson, 234,000 people quit smoking in the months prior to and after the ban. In the forward to the report, Sir Liam claims that the 'significance of the smoke-free laws cannot be underestimated'.

But Sir Liam's claims about the effect of the smoking ban on smoking cessation are not supported by the *Health Survey for England 2007* produced by the National Centre for Social Research and the Department of Epidemiology and Public Health at University College London. According to the *Health Survey for England*, not only did the smoking ban fail to reduce smoking, its first year

saw an increase in cigarette consumption among males aged 18-34. Reporting on smoking after the ban, the *Health Survey* notes that, 'There was no significant difference in cigarette smoking prevalence after the implementation of the smoke free legislation on 1<sup>st</sup> July. Among smokers the mean number of cigarettes smoked per day did not fall significantly'.

In fact, smoking prevalence amongst male smokers increased from 23 to 24 percent. Even more crucially, amongst smokers from the lowest socioeconomic quintile, who have some of the highest smoking rates in the country, the number of cigarettes consumed by men actually increased. Thirty percent of smokers reported that the ban had encouraged them to stay at home where they were free to smoke.

Further evidence suggesting the failure of public smoking bans to reduce smoking is found in a study by Holliday et al, which found that after Wales' public smoking ban there was a non-

significant decline in mean cotinine concentrations in children.<sup>12</sup>

This should not, of course be surprising, for it echoes the experience elsewhere. The state of Ohio, after passing similar public smoking legislation in 2006 saw its smoking prevalence increase by 3 percent since 2007. In France, tobacco consumption for 2008 equalled that of 2004 despite a smoking ban. A French government spokesman recently commented that, 'Measures to prevent passive smoking have not had any effect whatsoever on active smoking. They have completely failed'.<sup>13</sup> In the three years since Spain banned smoking in bars, restaurants, and most other public places, tobacco consumption has increased. According to data from the Tobacco Market Commission, 3.8 billion packs of cigarettes were sold in 2008 compared with 3.75 billion in 2006.

Why is this the case? Part of the answer is to be found in the fact that, like so much of the UK Government's anti-smoking strategy, public smoking bans are built on a foundation of ignorance

rather than knowledge. There is very little evidence, for example, that shows that public smoking bans motivate smokers over the long run to give up smoking. A major review of why smokers stop found that it was 'health concerns'<sup>14</sup> that drive cessation, not social or legal pressure, or even the cost of cigarettes.

Then, again, part of the answer why such bans fail to increase quitting or prevent initiation no doubt lies in 'reactance', in which smokers become increasingly resistant to the external efforts of what they perceive as a hostile and judgemental society to force them to change their ways. The more society pushes, as with smoking bans, the more smokers push back by declining to quit, or, in some instances, smoking more.

In a study about motivations to quit smoking, Curry et al found that smokers who had decided to quit voluntarily and were strongly motivated, as opposed to those who had in some respect felt externally pushed, whether through

social pressure or legislation, were far more successful at quitting.<sup>15</sup>

The evidence from the UK and elsewhere, as well as the literature on smoking cessation, suggests that public smoking bans have not resulted in fewer smokers and might well have a perverse, unintended consequence of encouraging smokers not to quit.

In an effort to shift the justification for public smoking bans away from the clearly false claim that such bans reduce smoking, proponents of such bans, both in the UK and elsewhere, have asserted that such bans have other public health benefits, most notably, that they reduce heart attacks. In support of this claim, a number of studies have been produced. A review of all of these is beyond the scope of this paper. Here, we review the report on public smoking bans and heart attacks issued by the US Institute of Medicine and the sensational new claim from the Chief Medical Officer of Wales that the public smoking ban there had reduced heart attacks.

Institute of Medicine *Secondhand Smoke and Cardiovascular Effects: Making Sense of the Evidence* October 2009.

According to the press release from the Institute of Medicine (IOM), 'Smoking bans are effective at reducing the risk of heart attacks and heart disease associated with exposure to secondhand smoke'. Lynn Goldman, professor of environmental health sciences at Johns Hopkins, and chair of the committee which produced the IOM report, noted public smoking bans 'reduce the risks of heart attack in nonsmokers as well as smokers'.

A careful examination of the evidence adduced by the IOM, however, suggests that these claims are false. The report examines 11 published studies of the alleged effect of public smoking bans on myocardial infarction (AMI). However, it omits numerous unpublished studies, including the largest study to date, which looked at the US as a whole, along with studies from Scotland, England, Wales, Denmark, Florida, California, Oregon, and New York. We examine the US study, produced by the

National Bureau of Economic Research, below.

However, the central problem with the IOM report is not in excluding non-confirming evidence; rather, it is the fact that its own evidence simply does not support its conclusion. This is because the report cannot, as with the primary studies on ETS exposure, provide any reliable measurements of the effects that it is supposedly studying. The report itself concedes this in writing that:

The committee was unable to determine the magnitude of effect on the basis of the 11 studies because of the variability among and uncertainties within them. Characteristics of smoking bans vary greatly among the locations studies and must be taken in account in reviewing results of epidemiologic studies. Those characteristics include the venues covered by the bans...and compliance with and enforcement of the bans. Other differences or potential differences among the studies include the length of follow-up after implementation, population characteristics (such as underlying rates of

acute coronary events and prevalence of other risk factors for acute coronary events, including diabetes and obesity) and size, secondhand smoke exposure levels before and after implementation, preexisting smoking bans or restrictions, smoking rates and method of statistical analysis. The time between implementation of a ban and decreases in secondhand smoke and acute cardiovascular events cannot be determined from the studies, because of the variability among the studies and indeed the difficulty of determining the precise time of onset of a ban.

Two factors from this list of limitation render any claims about reduced or, indeed, increased heart attack rates impossible to substantiate. First, the studies did not measure ETS exposure levels before and after implementation in order to determine whether there was an actual change in exposure. Without knowing this, it is impossible to determine whether a reduction in heart attacks was a result of a reduction in exposure. In other words, the studies are nonsensical.

Second, the studies do not measure the time between the onset of a smoking ban and the occurrence of the heart attacks, which again makes it impossible to determine whether the one is at least associated with, if not a cause of, the other. Because of these problems, the committee concludes that it had 'little confidence in the magnitude of the effects and...thought it inappropriate to attempt to estimate an effect size from such disparate designs and measures'.

Since science is based on the ability to measure effect sizes, this means that the IOM report *sans* effects is essentially not science, for without being able to say what the size of an effect is, it cannot claim that there is in fact *any* effect. In plain terms, the IOM committee admits that it cannot really say whether smoking bans have had any effect, positive or negative, on heart attack rates.

As Dr Michael Siegel of Boston University observes:

[W]hat the committee is saying is they have no confidence in



making any estimate of the size of an effect of smoking bans on heart attack rates. Another way to say that is this: the committee has no idea of what the effect of smoking bans on heart attack is.<sup>16</sup>

But the IOM report is also undermined by a recent US National Bureau of Economic Research study.<sup>17</sup> This study covers eight years and over 200,000 AMI admissions and two million AMI deaths in 468 US counties. It is in sharp contrast to the studies cited in the IOM report – and in the press – that generally focus on very short time periods, hundreds as opposed to thousands of AMI admissions, and a single location. The NBER study concludes:

We find no evidence that legislated U.S. smoking ban were associated with short-term reductions in hospital admission for acute myocardial infarction or other diseases...We also show that there is a wide year-to-year variation in myocardial infarction death and admission rates even in large regions such as counties and hospital catchment areas.

Indeed, the study notes that ‘large short-term increases in myocardial infarction incidence following a workplace ban are as common as the large decreases reported in the published literature’. Even more interesting, the report notes that the supposed sudden large decreases in AMI’s following smoking bans are biologically implausible, as ‘the mechanism for these tremendous declines in AMI rates reported in the small studies is unclear’.

Dr Tony Jewell Chief Medical Officer for Wales *Preventing the Preventable* Annual Report 2008.

As we have seen, there is little compelling scientific evidence that public smoking bans reduce heart attacks. Despite this, last week, in his annual report, the Chief Medical Officer for Wales, Dr Tony Jewell, said that there was evidence that they did.

Jewell noted that hospital admissions in Wales for AMI in 2007-2008 declined by 3.7 percent, from 4,324 to 4,164. According to a BBC news report, ‘The

number of people suffering heart attacks has reduced since the smoking ban in Wales began, a report by the chief medical officer has found'.<sup>18</sup> In response to Dr Jewell's report, ASH Wales noted, falsely, that 'bans on smoking in enclosed public places have been demonstrated to effectively reduce heart attack rates so it is not surprising to see Wales following this positive trend'.

However, despite the claims of Dr Jewell and ASH Wales, data on emergency room heart attack admission from Health Solutions Wales fails to support these claims. If one examines the data from 2007 – the year of the smoking ban – to 2008, the decline in heart attack admissions was the same as in previous years, that is, between 5 and 10 percent. In other words, there was no reduction in heart attacks in Wales attributable to the smoking ban. Indeed, as we have seen, it is impossible simply on the basis of the raw numbers to conclude anything about the relationship between smoking bans and heart attack admissions.

It is also interesting to note that, if one looks at the most recent data one finds an *increase* in the number of hospital heart attack admissions. According to Health Solutions Wales, hospital admissions for AMI were 3,999 in 2007-2008 and 4,126 in 2008-2009.

## **ALTERNATIVES TO PUBLIC SMOKING BANS – THE EFFICACY OF VENTILATION**

**T**he third fact supporting the necessity of public smoking bans is that there are no acceptable alternatives, such as ventilation. This, of course, is based on the claim that ETS poses fatal health risks to nonsmokers in even the smallest quantities. Of course, this is a claim that we have seen is not supported by the weight of the scientific evidence.

According to the Canadian Nonsmokers Rights Association, for example, 'Ventilation as a solution to

[secondhand smoke] in bars and restaurants is a propaganda brainchild of the tobacco industry, and is not based on public health protection'.<sup>19</sup> But is this, in fact, true? Is properly designed and maintained ventilation unable to remove ETS from venues such as pubs and restaurants?

While it might be true that some forms of conventional ventilation may be unable to deal with ETS in hospitality venues, the scientific evidence does not suggest that this is true for all forms of ventilation. For example, a 2001 study by Jenkins et al found that a directional airflow and heat-recovery ventilation system used in a Toronto, Canada, pub with both smoking and nonsmoking areas maintained the ETS components in the nonsmoking section of the pub at the same level as in the control nonsmoking facilities.<sup>20</sup> As the authors observe:

Based on the data collected in this study, mean ETS component concentrations in the nonsmoking section of the Black Dog Pub were not statistically different...from

those determined in the control nonsmoking facilities...This...study clearly shows that a suitably designed ventilation system installed in a restaurant/bar with both smoking and nonsmoking sections can produce ETS levels in the nonsmoking section that are not statistically different from those found in venues where smoking is prohibited...This small study provides important evidence to the regulator, the hospitality industry and the nonsmoking public that there are cost-effective alternatives to a prohibition of smoking in hospitality establishments, alternatives that can satisfy the concerns and interests of both nonsmoking and smoking customers.

The policy implications, as the authors note, are significant: 'If the hospitality venue that provides both smoking and nonsmoking areas can assure its nonsmoking customers that the ETS level in their area is comparable to that which they would find in a completely nonsmoking facility, then there would seem to be no rational reason for a prohibition of smoking in the controlled areas'.

In addition to the Jenkins et al study, a 2007 study commissioned by Imperial Tobacco, found similar results.<sup>21</sup> The study looked at ventilation and indoor air quality in two UK pubs and a restaurant where smoking was allowed in 2006. The study measured respirable suspended particles, carbon monoxide, and nitrogen oxides levels and found that levels inside, with smoking permitted, were comparable with the outside air quality. Equally important, the levels of particles and gases measured in the three venues with smoking were similar to the levels measured in two other studies, one US and the other Irish, where smoking was *not* allowed.<sup>22</sup>

As the authors conclude:

The results of this study which includes three well-ventilated hospitality venues which are typical of similar venues located throughout the UK demonstrates that ventilation systems when operated effectively can achieve levels of particles and gases in an indoor environment where smoking occurs that are comparable to levels of particles and gasses

present in the outdoor environment.

This suggests that even *if* ETS represented a fatal health risk to nonsmokers, which we have seen the scientific evidence does *not* suggest, properly installed, functioning, and maintained ventilation is able to remove it from smoking environments so it does not affect nonsmokers.

## **PERVERSE AND UNINTENDED CONSEQUENCES**

The final claim made by supporters of public smoking bans in the UK is that such bans do not have any perverse and unintended consequences. Again, the evidence does not support this claim. Two sorts of perverse and unintended consequences are particularly important.

First, there have been significant economic consequences in terms of

decreased trade in the hospitality industry. For example, a 2006 study that examined the economic consequences of the Scottish public smoking ban found that it resulted in a 10 percent decrease in food and drink sales in hospitality venues, as well as a 14 percent decrease in custom in Scottish pubs during the first three months following the ban's introduction.<sup>23</sup>

A similar pattern of significant economic consequences has been found in England, with pubs suffering the most damage. According to 2008 data from the British Beer and Pub Association, pub closures have averaged almost four per day, that is, 27 per week since 2007. This rate is significantly different from that in 2006 before the public smoking ban and 14 times more than in 2005. According to the British Beer and Pub Association, some 1,409 pubs closed during 2007.

Second, in addition to the economic and, indeed, social and cultural consequences given the importance of pubs to neighbourhood and community

life, there are also perverse and unintended health consequences of the public smoking ban.

In a just-published paper, Dr Jerome Adda from University College London and Dr Francesca Cornaglia of the London School of Economics examine the unintended effects of public smoking bans on nonsmokers, particularly children, in the US.<sup>24</sup> They report that while smoking bans decrease ETS exposure in workplaces and other public places, they 'can lead to a perverse increase in exposure by displacing smoking towards private areas'. Though smoking bans do not have a causal effect on either smoking prevalence, smoking cessation, or quit attempts, they do have a displacement effect in that they lead individuals to change the way in which they spend their time in public and private places.

Using data from 2003-2006, Adda and Cornaglia find that smoking bans in bars and restaurants decrease the time spent in these venues by smokers by roughly 20 minutes per day. More crucially,

smokers increase the amount of time that they spend at home under smoking bans in bars and restaurants by an average of 57 minutes. Adda and Cornaglia next examine the effects of these sorts of displacements, including workplace smoking bans, on the ETS exposure of nonsmokers, including children, through an analysis of cotinine levels in 42,009 nonsmokers.

The results are striking. Workplace smoking bans lead to an 'increase in cotinine in children aged 8 to 12', an increase that is statistically significant. Smoking bans in bars and restaurants also results in a statistically significant cotinine increase in teens aged 13-19. As the authors note, 'Smokers spend less time in bars and restaurants following the introduction of smoking bans...which would tend to increase the exposure of other family members in private places'. Hence, the American experience has been that 'smoking regulations can have perverse effects on non-smokers. By displacing smoking, and to some extent smokers, bans can contribute to an increase in exposure to

tobacco smoke. This effect is particularly strong for young children and those living with smokers'.

Adda and Cornaglia's research challenges a study by Holliday et al that claims to have found no statistically significant changes in mean salivary cotinine concentrations following the Welsh public smoking ban in 2007.<sup>25</sup> Curiously, Holliday et al claim that concerns about the 'potential displacement of smoking from public places into the home, affecting non-smokers and, in particular, children...[have] found little support to date', and indeed, make no reference to Adda and Cornaglia's study. Instead, Holliday et al claim that 'increasing number of successful smoking cessation efforts amongst adults and an increase in the proportion of smoke-free homes have been observed'.

As we have seen, there is substantial evidence both from the US and England that public smoking bans have *not* led to either more cessation attempts or greater success at quitting, and the

increase in the proportion of smoke-free homes is irrelevant to the problem at hand, which is the unintended and perverse consequence of *greater* ETS exposure for the children of smokers displaced from smoking at work and in bars and restaurants. Indeed, the claimed reductions in children's ETS exposure in Scotland were not true for children with two parents who smoked or with a mother who smoked.

The Holliday et al study suffers from several problems when compared with Adda and Cornaglia.

1. It is a very small sample of less than 2,000 children, compared with the enormous numbers in the Adda and Cornaglia study from across the US.
2. The Holliday et al data and conclusions are compromised by the fact that it relies on imputation for 47 percent of the subjects.
3. The study does not report cotinine concentration levels for children by household smoking status, as do Adda and Cornaglia. Instead, Table 2 reports cotinine concentration levels for all children.
4. The study focuses only on primary school children, whereas Adda and Cornaglia look at both younger children (8-12) and teenagers aged 13-19.
5. Adda and Cornaglia examine displacement both from the workplace and social settings, such as bars and restaurants.

This suggests that the reason that Holliday et al did not find any evidence of displacement is that they failed to look in the right place. It also suggests that this research is not a reliable guide to policymakers of the unintended consequences of public smoking bans.

## CONCLUSION

costs, has been slain by very many ugly facts.

**W**e conclude that none of the reasons offered in defense of public smoking bans provides unequivocal support for such bans.

The scientific evidence is not compelling that ETS poses fatal health risks for nonsmokers and, even if it were, there are viable alternatives to blanket public smoking bans that address not only the health but also the irritant and annoyance concerns of nonsmokers. Moreover, such bans have not significantly reduced smoking in the UK. Finally, such bans have had dramatic and perverse health and economic consequences.

Despite the claims of the champions of public smoking bans, it is clear that the beautiful hypothesis that ETS is a major killer and, hence, a major public health problem that defies solution – except through total prohibition of public smoking – and which leads to reductions in smoking, all without any



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