

LETTERS TO THE EDITOR

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Smoking among Buddhist monks in Phnom Penh, Cambodia

EDITOR,—According to existing studies, Buddhist monks can have an impact on smoking cessation in a given population.^{1,2} It is because of their influence that Buddhist monks in Phnom Penh, Cambodia were selected for a study of their knowledge, attitudes, and practices concerning tobacco, with the long term objective of developing ways of enlisting their support in tobacco control efforts in Cambodia.

The 30 cluster survey method was employed, wherein all of the temples in the city were listed and, according to the number of monks residing at them, 30 sites were randomly selected for interviewing from seven to 11 monks each for a total of 318 interviews. Questions were designed to reflect the potentially sensitive issue of smoking among religious practitioners. There were no cases of interview refusal.

When all 318 respondents were asked, "Do you want to quit smoking?" 44% gave some type of answer other than "not applicable": 37% said "yes", 3% "no", and 4% "not sure". Also, when all respondents were asked, "Why do you want/not want to quit?" a total of 44% gave some reason. Finally, when asked, "What do you do with the tobacco gift packages you receive?" 44% of the 318 respondents mentioned that they smoke the gift tobacco themselves. These figures lead us to believe that the prevalence of current smokers among Buddhist monks is 44%. In comparison, smoking prevalence among the general male population in Phnom Penh is almost 65% (1994) and among Buddhist monks in Thailand 56% (1990).^{3,4}

Of the influences to start smoking 26% of respondents said that an individual friend was the main influence to start smoking; 18% responded group pressure from friends or other monks; 21% complimentary cigarettes; 12% work/stress; 8% father's influence; 3% advertising; and 12% other reasons. As can be seen, these two influences alone—individual friends and group pressure—were responsible for almost half of all influences to start smoking.

When asked what they thought the teachings of Buddha have to say about smoking, 91% of respondents said the teachings of Buddha do not say anything; but when asked if there should be a Buddhist law that recommends monks do not smoke, 71% replied "yes". When asked if the government should require warning messages on all tobacco advertising, 94% agreed; 96% agreed that the government should ban all tobacco advertising.

About one third (34%) of all respondents thought that people should not offer cigarettes to monks, while an equivalent per-

centage (38%) thought people should. Another approximately one third was not sure. These figures can be partially explained by a question in the survey that asked what monks did with the tobacco gift packages. Over 50% "give" the cigarettes away. More commonly, the cigarettes are sold or bartered for extra income, but it would not be appropriate, according to Buddhist principles, to admit this.

Direct assistance for smoking cessation programmes is urgently needed: 84% of smokers want to quit; if a program was available to help people stop smoking, 95% of smokers said they would attend; 86% of all respondents would be willing to teach people about the effects of smoking.

The pattern of responses indicates that, even though the teachings of Buddha do not say anything about smoking directly, there is a stigma tied to smoking that inhibits many monks from admitting their smoking habits directly. The large majority of monks feel that smoking is not an appropriate practice and that there should be a Buddhist law that recommends they do not smoke.

Most monks, however, have little understanding of the specific detrimental effects smoking has on them, as well as the effects of second hand smoke. Health education is needed to raise such awareness, as are cessation programmes to help bring about desired behaviour changes.

The small scale of this research makes it difficult to generalise conclusions for monks throughout the country. However, it does provide useful insights into some trends in tobacco use among monks in Cambodia and highlights a number of important issues for further research. Most importantly, this study reveals the potential that exists for successful cooperation with monks in tobacco control efforts in Cambodia.

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Effect of smokefree bar law on bar revenues in California

EDITOR,—In 1998 a California state smokefree workplace law requiring that bars be smoke free went into effect.^{1,2} Both before passage of this law and shortly after it went into effect, the tobacco industry and its allies predicted that it would hurt the bar business. To test the hypothesis that smoke free bar legislation harms the bar business, we obtained total revenues from eating and

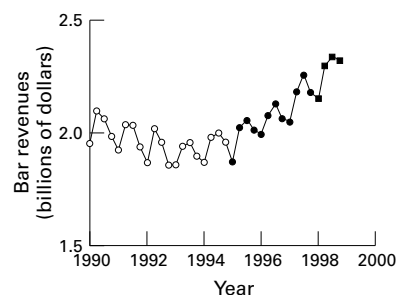


Figure 1 Total revenues from eating and drinking establishments with full liquor licenses in California before a state smokefree workplace law went into effect (open circles), after restaurant provisions went into effect (solid circles), and when bars were required to be smokefree (solid squares). Data from quarterly reports of the California State Board of Equalization.

drinking establishments licenced to serve all forms of alcohol ("bar revenues") from the tax authorities in California (fig 1). We conducted an analysis of these data following a similar approach to earlier analyses of the effects of smokefree restaurant and bar ordinances on communities.^{3,4}

Briefly, we divided bar revenues by total retail sales to account for underlying economic conditions and inflation and conducted a multiple linear regression analysis with time, calendar quarter, a dummy variable to indicate whether the restaurant provisions the law were in force (0 before 1 January 1995, and 1 afterwards), and another dummy variable to indicate if the bar provisions were in force (0 before 1 January 1998, and 1 afterwards). We also examined the fraction of all "eating and drinking establishment" revenues that were going to those with liquor licenses to see if there was any shift in the mix of business associated with either the restaurant or bar provisions of the state smoke free workplace law. (Note that these bar revenues include both revenues of restaurants that include bars as well as free standing bars.)⁵

There was no significant effect of the restaurant provisions of the law on bar revenues as a fraction of total retail sales (coefficient of dummy variable -0.01 (0.04)%, $p = 0.811$); there was a small but significant positive change in bar revenues as a fraction of retail sales associated with the bar provisions going into effect (coefficient 0.09 (0.04)%, $p = 0.029$). Implementation of the smokefree restaurant provisions was associated with an increase in the fraction of all eating and drinking establishment revenues that went to establishments with liquor licenses (0.54 (0.27)%, $p = 0.054$), and a larger increase following implementation of the smokefree bar provisions (0.73 (0.25)%, $p = 0.007$).

As with claims of adverse effects on the restaurant³ and tourist⁶ industries, these data further discredit tobacco industry claims that smokefree bar laws are bad for the bar business. Quite the contrary, these laws appear to be good for business.

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Passive smoking and an increased risk of acute stroke

EDITOR.—Although “passive smoking” may be intuitively harmful, the paper by Bonita and colleagues¹ on the risk of stroke and environmental tobacco smoke (ETS) exposure suffers from two fundamental defects. The first is the enormously disproportionate effect due to a small exposure, and the second is the lack of allowance for confounding variables, especially diet.

Serum cotinine concentrations have recently been determined at the US National Center for Environmental Health using the most sensitive method to date of high resolution gas chromatography with mass spectrometry.² In 10 000 subjects it was shown that the mean serum cotinine concentration in ETS exposed non-smokers was 0.6 ng/ml compared to 300 ng/ml in active smokers. This represents 1/500th of the dose received by the active smoker.

It is difficult to reconcile this degree of exposure with an increased risk of stroke which is one quarter that of the active smoker. A similar disproportionate effect has been claimed for the increased risk of ischaemic heart disease and ETS exposure, but the biological plausibility and mechanisms of effect advanced to support this have been shown to lack credibility.^{3,4}

It is well established that active smokers have other associated risk factors. They are physically less active and have lower intakes of fruit, vegetables, folate, and flavonoids,⁵ which are all linked to a substantial increased risk for stroke,⁶⁻⁸ and many of these characteristics are shared with non-smokers living with smokers.⁹

Although Bonita and colleagues excluded Maori and Pacific islands people from the study, the fact remains that in the residual sample, smoking, and therefore passive smoking, is more prevalent among lower socioeconomic groups, and independent of smoking, these groups have a higher risk of stroke.

The Pacific islands people indigenous to New Zealand have a higher incidence of stroke than Europeans indigenous to New Zealand. In this respect it is noteworthy that in the Pacific Melanesian islands where a traditional way of life is followed, but where cigarette smoking is excessive, cardiovascular disease and stroke are apparently absent. An example is the study on the Kitavan islanders, where 80% of people smoke cigarettes rolled from black

imported or home grown tobacco and stroke is absent. Bonita and Beaglehole¹⁰ in their comment on this study noted “. . .this is worrisome in view of the other adverse effects of tobacco”. The staple diet of these people consists of root tubers, fruit, fish and coconuts, low salt, low fat (rather different to the New Zealand diet), they are physically active, and have low body mass index.

High stroke rates in Japan have diminished in recent years, due not to smoking reduction, but largely to salt restriction and a more westernised diet; the high stroke incidence in China is not strongly associated with smoking.

The interaction of diet, ethnicity, socioeconomic, cultural, and behavioural characteristics is complex, but cannot be ignored when considering the effect of smoking on the incidence of stroke. In view of the extremely low exposure and lack of allowance for confounding variables, the increased risk of stroke attributed to passive smoking by Bonita and colleagues¹ is unlikely to be true.

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Response by authors:

Passive smoking and risk of stroke seems a solid connection

EDITOR.—Kenneth Denson refers to results from the US National Center for Environmental Health where the serum cotinine concentration in environmental tobacco smoke (ETS) exposed non-smokers was only 1/500th of the dose received by the active smoker. From this point of view, Denson finds it difficult to reconcile that ETS

exposed non-smokers in our study should have a risk of stroke one quarter that of the active smoker.

Although cotinine is a marker of tobacco smoke exposure, with its own limitations,¹ it has not been proved also to be a valid marker of a person's exposure to all of the toxic compounds in tobacco smoke. There are several possible biological mechanisms by which passive smoking may increase the risk of stroke—for example, increased platelet aggregation² and reduced oxygen carrying capacity.³ Debate continues as to the best biomarker for passive smoking.

While it is true that the National Health and Nutrition Examination Survey (NHANES) study⁴ cited by Denson was based on a large and carefully selected sample, it is noteworthy that the physical examinations and collection of blood sample “usually occurred 2 to 3 weeks after a household interview”, and, furthermore, after the topic of smoking had already been raised. Thus, there was ample opportunity for members of each selected household to change their smoking behaviour well before the blood samples were drawn. Cotinine concentrations would then not have been indicative of usual patterns of exposure to ETS. In addition, NHANES assumed that sharing a home with a smoker equated with passive exposure. This assumption becomes particularly tenuous when 40% of participants in the study were aged less than 12 years; the effects of passive smoking on the health of children were already well known in the community.

While it would have been optimal to have been able to control for differences in diet between non-smokers exposed and not exposed to ETS, confounding is unlikely to explain our findings. There is only limited evidence that the diet of individuals strongly affects their risk of stroke. In general terms, the relative risk associated with a confounding variable needs to be at least double the observed association for that confounder to explain it. Denson is unable to nominate a specific confounder and refers instead to ecological studies which are well known as having many pitfalls. It is highly unlikely that decades of work on the aetiology of stroke, including a number of very large prospective studies, would have failed to uncover a strong dietary risk factor for stroke, if one existed. In the meta-analysis of analytical studies by Law and colleagues⁵ differences in diet were judged likely to account for 6% of the increased risk of coronary heart disease associated with ETS in non-smokers. If those results may be extrapolated to our data on stroke the odds ratio would decrease to 1.72 (1.82/1.06)—which is still a considerable increased risk. Thus, dietary differences are unlikely to explain all of the increased risk in non-smokers exposed to ETS in the present study.⁶

It is always a possibility that one study, by chance, finds a strong association between an exposure and an end point. What accounts to the credibility of our study is that the anti-tobacco campaign in New Zealand has been very successful. In the study of environmental tobacco smoking exposure in the US population, the authors found that 88% of people who were not smokers had detectable concentrations of cotinine, including people who reported not to be exposed either at home or at work.⁴ Thus, the relatively high odds ratios found in our

study, for active as well as passive smoking, could simply reflect a satisfactory allocation of non-smokers not exposed to passive smoking.

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A tentative illustration of the smoking initiation and cessation cycles

EDITOR.—In many former papers,^{1–4} the smoker's career is described separately for the processes of initiation and cessation. Furthermore, the recently advocated issues of smoking reduction, sometimes followed by a secondary cessation^{5–7} are not always considered. We have tried to summarise the complete smoker's career in one single schema (fig 1) in a way that could be useful for teaching purposes in the preventive and curative fields.

The non-smoker (A), after a preparatory stage, becomes an occasional smoker (B) (trying and experimentation stages) and afterwards, exceptionally abandons smoking. In most cases, however, experimental smokers progress toward regular, daily use (C). The stage labelled "happy smoker" (D) usually lasts for many years, after which smokers perceive more acutely the "pros" and "cons" of their tobacco use, thus becoming "ambivalent smokers" (E). Later on, some prepare to stop (F), and start to take action (primary cessation) (G), which is sometimes followed by perseverance (H). In most cases, because of withdrawal symptoms, cessation is followed by a relapse (I) and the smoker progresses further, often several times, into the cessation cycle through the

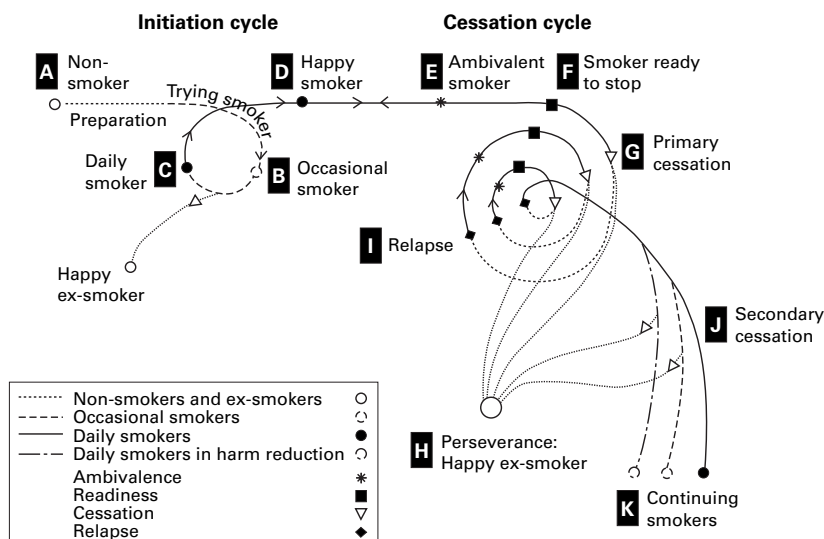


Figure 1 Diagram summarising the complete smoker's career from initiation to cessation.

stages of "ambivalence" and "readiness to stop" before finally succeeding with cessation and becoming a persistent "happy ex-smoker" (H). Some smokers are unable to quit completely but can space their smoking, again becoming occasional smokers (sometimes by using pipes, cigars or cigarillos instead of cigarettes), while others reduce their daily cigarette consumption, often nowadays with the help of concomitant nicotine substitution, in a process of "harm reduction". Some of these smokers finally quit (secondary cessation) (J) to also become "happy ex-smokers". However some remain continuing smokers (K) until their death.

In most cases, the process evolves in the described direction, but, as recently stressed by Butler and colleagues,⁸ unfortunate interventions, especially at the stage of ambivalence, can induce a regression in the cessation cycle and delay quitting by reinforcing the smoker's resistance to change.

Personal variables largely influence the speed of movement through both the initiation and cessation cycles, while external interventions as well as emerging anti-smoking social norms are conducive to change.

A universally applicable quantitative assessment of the mean durations of the various stages is not feasible, since they differ according to different settings. Similarly, the distribution of the population of smokers in

the various stages also differs according to national, ethnic, and socioeconomic parameters.

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