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# Mortality attributable to smoking in Vietnamese men in 2008

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ABSTRACT

*Objective.* Smoking prevalence among Vietnamese men is among the highest in the world. Our aim was to provide estimates of tobacco attributable mortality to support tobacco control policies.

*Method.* We used the Peto–Lopez method using lung cancer mortality to derive a Smoking Impact Ratio (SIR) as a marker of cumulative exposure to smoking. SIRs were applied to relative risks from the Cancer Prevention Study, Phase II. Prevalence-based and hybrid methods, using the SIR for cancers and chronic obstructive pulmonary disease and smoking prevalence for all other outcomes, were used in sensitivity analyses.

*Results.* When lung cancer was used to measure cumulative smoking exposure, 28% (95% uncertainty interval 24–31%) of all adult male deaths (>35 years) in Vietnam in 2008 were attributable to smoking. Lower estimates resulted from prevalence-based methods [24% (95% uncertainty interval 21–26%)] with the hybrid method yielding intermediate estimates [26% (95% uncertainty interval 23–28%)].

*Conclusion.* Despite uncertainty in these estimates of attributable mortality, tobacco smoking is already a major risk factor for death in Vietnamese men. Given the high current prevalence of smoking, this has important implications not only for preventing the uptake of tobacco but also for immediate action to adopt and enforce stronger tobacco control measures.



## Introduction

Smoking prevalence among Vietnamese men is one of the highest in the world with smoking an integral part of male social behavior (Morrow and Barraclough, 2003; The Tobacco Atlas Online). Reliable smoking prevalence data are not available prior to 1990. A regional study in the 90s reported smoking prevalence of 70% (Jenkins et al., 1997) with the first national estimate of prevalence in excess of 60% for Vietnamese males (General Statistics Office, 1994). Given the very low smoking prevalence among women, per capita annual cigarette consumption implies an annual consumption of about 2600 cigarettes per adult male in the mid-90s (Guindon and Boisclair, 2003). Taken together these data suggest that past consumption has been substantial for men.

Over the last decade, Vietnam has implemented the National Tobacco Control Policy 2000–2010 (Government of Vietnam, 2000) aimed at reducing tobacco-related morbidity and mortality through a number of public interventions including excise tax and advertising bans, but the nation is in a relatively early stage in its tobacco control efforts (Levy et al., 2006). The paradox surrounding the tobacco control policy in Vietnam arises from the contradictory position of a government that benefits from manufacturing of tobacco products, and is also responsible for controlling tobacco consumption. Even with some evidence of success, limited resources and competing interests have impeded the effective implementation of the policy to its full potential. This is reflected in current smoking prevalence which has remained high among adult males at almost 50% (GATS Vietnam Working Group, 2010; General Statistics Office, 2007). Exposure to passive smoking also remains exceedingly high (67.6%) among non-smokers (GATS Vietnam Working Group, 2010). Despite some delays, recent passing of the Tobacco Harm Prevention Law is expected to strengthen implementation of the policy (Ministry of Health (Vietnam), 2012).

Such a policy framework can benefit from a better understanding of the current and future health effects of tobacco. This study applies the method of Peto and Lopez (Peto et al., 1992) to the first ever national estimates of causes of death for Vietnam (Ngo et al., 2010; Nguyen et al., 2011) in order to quantify mortality attributable to smoking in 2008. Given the low smoking prevalence among females the focus is on male smoking attributable mortality. Since smoking risks have not previously been published for Vietnam, the study provides analysis of uncertainty and sensitivity analyses. These estimates

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are provided to emphasize the urgency of strengthening tobacco control initiatives in Vietnam.

## Methods

We used cause-specific mortality estimates from the first burden of disease and injury study for Vietnam in 2008 (Nguyen et al., 2011) based on a nationally representative cause of death survey using verbal autopsy methods (Ngo et al., 2010).

Relative risk estimates for cause-specific mortality related to tobacco use were derived from a re-analysis of the American Cancer Society Cancer Prevention Study, Phase II (CPS-II) which included adjustment for important covariates (Table 1) (Ezzati et al., 2005a; Ezzati et al., 2005b; Oza et al., 2011).

### *Base analysis*

Following the Peto–Lopez method (Peto et al., 1992), we used estimated lung cancer mortality in Vietnam as an indirect indicator of the accumulated hazards of smoking taking into account the time lag between exposure and outcome. However, lung cancer diagnosis by verbal autopsy is difficult at older ages and with a small number of lung cancer deaths in the sample, the resulting lung cancer mortality age pattern in the first Vietnam burden of disease study was unstable. Consequently, we used the Vietnam burden of disease study to derive the level of lung cancer mortality for 2008 but the age pattern was smoothed by applying the age pattern for males in China 2004–2005 (Chinese Center for Disease Control, personal communication 2011).

**Table 1**
Relative risk estimates of disease specific mortality for CPS-II smokers relative to never-smokers.[a]

| Disease outcome (ICD-10 codes) | Age group (years) | Males CPS-II RRs (95% CI) |
|---|---|---|
| Lung cancer (C33–C34) | ≥30 | 21.3 (17.7–25.6) |
| Upper aerodigestive tract cancer (C00–C14, C15, C30–C32) | ≥30 | 8.1 (5.7–11.7) |
| *Other cancer* | | |
| Stomach cancer (C16) | ≥30 | 2.2 (1.8–2.7) |
| Liver cancer (C22) | ≥30 | 2.3 (1.5–3.8) |
| Pancreatic cancer (C25) | ≥30 | 2.2 (1.7–2.8) |
| Bladder cancer (C67) | ≥30 | 3.0 (2.1–4.3) |
| Myeloid leukemia[b] (C92) | ≥30 | 1.9 (1.3–2.9) |
| Colorectal cancer (C18–C21) | ≥30 | 1.3 (1.2–1.5) |
| Chronic obstructive pulmonary disease (J40–J44) | ≥30 | 10.8 (8.4–13.9) |
| Other respiratory diseases (J00–J22, H65–H66, J30–J39, J45–J47, J60–J80, J82–J89, J91–J98) | ≥30 | 1.9 (1.5–2.4) |
| Tuberculosis[c] (A15,A16,A19) | ≥30 | 1.6 (1.2–2.3) |
| *Cardiovascular diseases* | | |
| Ischemic heart disease (I20–I25) | 30–44 | 5.51 (2.47–12.25) |
| | 45–59 | 3.04 (2.66–3.48) |
| | 60–69 | 1.88 (1.70–2.08) |
| | 70–79 | 1.44 (1.27–1.63) |
| | ≥80 | 1.05 (0.78–1.43) |
| Cerebrovascular disease (I60–I69) | 30–44 | No estimates (insufficient events) |
| | 45–59 | 3.12 (2.10–4.64) |
| | 60–69 | 1.87 (1.43–2.44) |
| | 70–79 | 1.39 (1.09–1.77) |
| | ≥80 | 1.05 (0.63–1.77) |
| Other cardiovascular diseases (I10–I15, I26, I28, I47–I49, I70–I84, I86–I89) | ≥30 | 2.15 (1.94–2.38) |
| Diabetes mellitus (E10–E14) | ≥30 | 1.42 (1.10–1.83) |

The 95% confidence interval for the effect of smoking and cardiovascular disease (CPS-II) in a number of age groups crossed the null. We included all draws of the relative risk distribution including those that show a protective effect in the uncertainty analysis because the overall relationship for the risk factor across all ages for these diseases is statistically significant.
ICD-10 = International Classification of Diseases, 10th revision (World Health Organization, 1992).
[a] Source: Re-analysis of CPS-II data (Ezzati et al., 2005a, 2005b; Oza et al., 2011). RRs were estimated from Cox proportional hazard models with never smokers as the reference group. All risks were adjusted for age, race, education, marital status, "blue collar" occupation, weekly consumption of vegetables and citrus fruit, vitamin use, alcohol use, aspirin use, body mass index, exercise, dietary fat consumption. In addition, cancer RR were also adjusted for additional covariates including family history of cancer (Ezzati et al., 2005a) and cardiovascular RRs were also adjusted for hypertension and diabetes at baseline (Ezzati et al., 2005b).
[b] The relative risk for myeloid leukemia was applied to all leukemias (C91–C95).
[c] Relative risks for tuberculosis are from a separate meta-analysis (Lin et al., 2007) because there were too few cases in CPS-II to make estimates.

The smoothed lung cancer rate for Vietnam males in 2008 includes smokers and non-smokers and is considerably lower than the US CPS-II smoker lung cancer rates (from the CPS-II reanalysis) but is higher than 1986–88 China smoker lung cancer rates (Jill Boreham, personal communication 2011) at older ages (Fig. 1) as these rates are from an earlier phase of the tobacco epidemic in China. Non-smoker lung cancer rates in China are higher than in the CPS-II population, probably as a result of exposure to other lung cancer risk factors such as coal use for cooking and heating (Ezzati and Lopez, 2003).

The proportion of lung cancer attributable to smoking was estimated as the absolute difference between the smoothed Vietnam lung cancer death rate and the estimated level in non-smokers. In the absence of Vietnam non-smoker lung cancer rates, we used male non-smoker lung cancer mortality rates from the Global Burden of Disease Study 2010 (GBD 2010) where a negative binomial regression of pooled cohort studies was used to generate separate age–sex-specific non-smoker lung cancer mortality rates for 1) China, 2) countries in the high-income Asia-Pacific region, and 3) all other countries (Lim et al., 2012). We used the male non-smoker lung cancer mortality rate for the "all other countries" group as the non-smoker lung cancer rate for Vietnam.

For causes other than lung cancer, we calculated the Smoking Impact Ratio (SIR), defined as the Vietnam lung cancer mortality in excess of non-smokers, relative to excess lung cancer mortality for the reference group of smokers in the CPS-II population (Ezzati and Lopez, 2003):

$$SIR = \frac{C_{LC} - N_{LC}}{S^*_{LC} - N^*_{LC}} \times \frac{N^*_{LC}}{N_{LC}}$$

where $C_{LC}$ is the smoothed age–sex-specific lung cancer mortality rate for 2008 in Vietnam; $N_{LC}$ is the age–sex-specific lung cancer mortality rate of non-smokers for all countries outside China and high-income Asia-Pacific region as estimated in GBD 2010. $S^*_{LC}$ and $N^*_{LC}$ are age–sex-specific lung cancer mortality rates for smokers and non-smokers, respectively, in the reference population CPS-II. The numerator and denominator are normalized with the respective non-smoker lung cancer mortality rates (Ezzati and Lopez, 2003).

Conceptually, the SIR converts the smokers in the Vietnam population with different smoking histories into equivalents of smokers in the CPS-II reference population, where hazards for smoking-related diseases have been measured (Ezzati and Lopez, 2003).

For each disease, the fraction of deaths attributable to smoking was estimated by using the standard population attributable fraction (PAF) formula (Greenland and Robbins, 1988):

$$\text{PAF} = \frac{P(RR-1)}{P(RR-1)+1}$$

with prevalence, *P*, set to SIR for each age group and *RR* the cause-specific relative risks from the CPS-II re-analysis (Table 1).

### *Sensitivity analysis*

#### *Prevalence-based method*

A sensitivity analysis was also carried out using the conventional prevalence-based approach. PAFs were calculated by applying Vietnam current tobacco smoking prevalence data (Table 2) (GATS Vietnam Working Group, 2010) to relative risks from the CPS-II re-analysis. (Table 1).

#### *Hybrid method*

In addition, we followed the approach used in the GBD 2010 study (Lim et al., 2012) and used the Peto–Lopez method, which uses lung cancer mortality as a marker of cumulative population exposure to smoking for conditions where there is a long lag between exposure and outcome such as cancers and