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DEATH AND TOBACCO TAXES

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ABSTRACT

This study analyzes the effects of tobacco excise tax changes on mortality due to heart disease, cancer, and asthma. Reduced form regressions of mortality rates on tax data for the years 1954-1988, with controls for state, year, income, and unobserved persistence, indicate that tax increases lead to statistically significant decreases in mortality. A 10% increase in the tax is projected to save approximately 5200 lives a year.

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1. Introduction.

Recent interest in tobacco taxes has centered upon their role as a source of revenue for the financing of health care reform. Underlying this interest is the belief that cigarettes and other tobacco products are inelastically demanded, so that a tax would be an effective source of revenue generation. According to proponents of the tax, smokers have higher health care costs, which are subsidized by nonsmokers in the absence of any direct tax on the behavior leading to the higher costs. Opponents of any tobacco tax increase argue that the tax is unfair to the majority of smokers, and to tobacco industry workers (see the work of Manning et al. (1989, 1991) on the net burden of smoking for a comprehensive analysis).

Ignored in the debate to this point has been any discussion of the direct effects of tobacco taxes on health (Grossman (1993), Warner (1986), and Harris (1987) are important exceptions). This perhaps reflects the widespread belief that smoking is not responsive to price increases, or at least that heavy smoking is not sufficiently responsive to generate any meaningful health benefits. This belief does not, however, have any direct empirical foundation. An extensive literature on the demand for cigarettes reveals a statistically significant, although inelastic, negative relationship between cigarette consumption and price (or, in some formulations, tobacco taxes). An even more extensive literature documents a positive dose-response relationship between smoking and a variety of diseases, including cancer of various forms, cardiovascular disease, and chronic obstructive pulmonary diseases. These relationships are typically observed at all consumption levels, with the disease rates higher at higher smoking levels. Implicit in

these findings is the prediction that the incidence of disease would respond to reductions in smoking short of quitting altogether, so that all that would be necessary for a tax increase to generate health effects would be for smokers to curtail their tobacco consumption somewhat.

Some researchers conclude on the basis of these two distinct effects-that taxes affect smoking and that smoking affects disease rates- that an increase in the tobacco tax would reduce rates of smoking-related mortality and morbidity (Harris (1983), Warner (1984), Grossman (1989)). This is not necessarily the case. The cigarette demand studies describe the behavior of the average smoker, not the heavy smoker, and there is no statistical evidence of the effects of taxes on heavy smoking. If heavy smokers do not respond to tax increases, then it is possible that a tax increase would generate only limited health benefits to light to moderate smokers, where smoking-related disease rates are lower (albeit higher than those for nonsmokers).

In this paper, I address this deficiency in the existing knowledge of the effects of tobacco taxes on smoking and, ultimately, on health, by estimating the effects of such taxes on mortality rates for those disease categories affected by tobacco consumption. Mortality rates for lung cancer, oral cancer, laryngeal cancer, cardiovascular disease in general and, more specifically, ischemic heart disease, act as proxies for heavy smoking. Thus, any effects of taxes on heavy smoking should be reflected in effects on mortality.

The empirical strategy used has a clear antecedent in the work of Cook and Tauchen (1982), who estimated the effects of liquor taxes on cirrhosis mortality. Similar issues arise in connecting the results of alcohol demand studies with clinical evidence on

the relationship between alcohol abuse and cirrhosis, as exist in the smoking/tax debate. Prior to the Cook and Tauchen study, liquor taxes had only been shown to affect average consumption, while cirrhosis mortality is a consequence of heavy consumption.[1] Cook and Tauchen find a significant effect of liquor taxes on cirrhosis mortality in their reduced form equations, providing strong evidence that heavy drinking responds to taxes. (An important difference between tobacco and alcohol consumption is that light to moderate drinkers are much more prevalent than light to moderate smokers.)

The statistical approach used here, like Cook and Tauchen's, uses state level data on smoking, taxes, and mortality rates in a longitudinal design. Fixed effects for state and year control for all unobservable state and period-specific effects, and data on state-level income control for the effects of wealth on smoking and mortality. Autocorrelation corrections likewise control for unobservable effects, such as "persistence," that are not captured by the time dummies. The observed effects of taxes on mortality rates can be interpreted directly as causal effects, and are therefore directly relevant for policy evaluation.[2]

The results indicate a pervasive effect of tobacco taxes on mortality rates. Cancer of the lung, oral cavity, and larynx, cardiovascular disease, and asthma all decrease significantly following tobacco tax increases. No effect is found on ischemic heart disease. The preponderance of the evidence indicates that tobacco taxes directly affect smoking-related mortality. The implication for health care is that smoking-related health care costs might be affected significantly via tax policy, quite independently of any financing considerations, by reducing the adverse health outcomes. The net cost of

reductions in smoking is not clear, since former smokers may utilize more health care resources, due to their extended longevity.

2. Smoking and Health.

A now voluminous literature documents the relationship between cigarette smoking and various types of disease. These findings are summarized in the recent articles by Bartecchi et al. (1994) and MacKenzie et al. (1994), and described in detail in the various *Surgeon General's Reports on the Health Consequences of Smoking*, including reports on Cancer (1982), Cardiovascular Disease (1983), and Chronic Obstructive Pulmonary Disease (1984). The basic message of these reports is that higher disease rates are observed among smokers for cancers of the lung, lip, oral cavity, larynx, and pharynx (and other cancers), ischemic heart disease, stroke, and arteriosclerosis, and for bronchitis, emphysema, and asthma. Furthermore, there is consistent evidence of a dose-response relationship between many of the diseases studied and the quantity of tobacco consumed. This quantity as studied varies by degree of inhalation, number of cigarettes smoked per year, cumulative smoking (i.e., years smoked) and years since quitting.

Delineation of the nature of the dose-response relationship is crucial to structuring the empirical analysis. The plausibility of the model requires that health respond to smoking changes, and smoking to tax changes. A more subtle issue, however, concerns the timing of the relationship between changes in taxes, smoking, and health; interpretability of the results requires that tax and mortality changes be located fairly closely to each other in time.

The major prospective studies of the relationship between smoking and mortality,

which are documented in detail in the Surgeon General's Reports, indicate an effect of smoking on mortality that is both quite general and, in many cases, quite large.[3] Coronary heart disease (CHD) mortality in each of the major studies is about 50% higher in smokers of all types than in nonsmokers. Lung cancer mortality rates are anywhere from 2 to 14 times higher in smokers. Similar ranges are exhibited for other cancers, including those of the mouth, larynx, and esophagus.

Epidemiological evidence also documents a strong dose-response relationship in each of the prospective studies according to number of cigarettes smoked. Again, the figures for all diseases show a systematic effect that increases almost without exception with quantity smoked. The mortality ratios are particularly striking for lung cancer, with rates in excess of 20 times higher for smokers of one or more packs of cigarettes per day.

A third important element of the dose-response relationship is that between excess mortality and the duration of time since quitting. In all of the major prospective studies, mortality ratios are inversely related to years since quitting. In most cases, mortality ratios for former smokers approach those of never smokers after 10-15 years. Significant declines in mortality are also found for shorter durations. In the ACS 25-state study, for example, former smokers of 1-19 cigarettes/day showed no change in lung cancer mortality in the first year of quitting, a 31 percent decline in the next three years, and the same mortality as non-smokers after 5-9 years. Heavier smokers (20+ cigarettes/day) exhibited a similar 30 percent decline in 1-4 years, a 60 percent decline in 5-9 years, and a return to never smokers risk levels 10 years after quitting. The other studies yield similar findings for durations of quitting of 5 years or more.

In analyzing data on mortality and duration since quitting, it is important to note that, in many cases, quitting might result from poor health, so that observed mortality ratios for quitters will understate the effects of quitting on the average smoker. If poor health is a major determinant of quitting, one could even expect observed mortality to worsen shortly after quitting, with the beneficial effects beginning to dominate only after some time had elapsed.

In the ACS CPS-II study of 1.2 million males and females, for example, the effects of endogenous quitting are seen clearly by comparing the results for both healthy and unhealthy smokers who had quit for less than one year. In the healthy subsample, the mortality ratios for males drop by about one-third almost immediately, while in the full male sample mortality increases by 50-100 percent, depending on amount smoked. Identical results are observed for females over the first five years following quitting. For all males and for females who smoke less than one pack per day, the effect of quitting for 1-5 years is largest in year 1. Mortality due to coronary heart disease (CHD) exhibits similar patterns in this study.

More generally, CHD mortality declines almost immediately in five of the seven prospective study subsamples restricted to individuals with no prior conditions, with first-year declines of 20-30% common. In the unrestricted samples, CHD mortality increases initially in two of three cases. In every case, CHD mortality is lower after one year of quitting than for current smokers, and declines more or less steadily thereafter.

These results indicate that an analysis of the effects of recent taxes on mortality rates might be more fruitful than previously believed. Harris (1987) states, for example,

that determination of the health consequences of tobacco tax changes is difficult, because "... some of the health consequences of the resulting changes in smoking could take decades to be manifest. Thus, one's excess risk of lung cancer may not return to that of the non-smoker for more than a decade." The veracity of this statement, which accords closely with the results described above, is not in doubt. However, the return to health is not a discontinuous shift at 10 years, but a continuous improvement that shows results at much earlier stages.

These results are critical for the logic of the empirical models estimated below. An extremely long lagged effect (10 years) of taxes on mortality would be difficult to estimate and interpret. Lags of 1-2 years, however, will capture tax changes that are beneficial to health, and provide the empirical model a more plausible structure.[4] In the empirical analysis, current and last year's cigarette consumption are significant predictors of heart disease and lung cancer mortality.[5] Longer lags affect mouth and throat cancer. Combined with a contemporaneous effect of taxes on smoking, one year lags of taxes should therefore have significant effects on a broad range of diseases.

The presence of a dose-response relationship is important for the argument that tax changes will affect health. Even for light to moderate smokers, the evidence indicates reductions in mortality ratios over a short time horizon as smoking is curtailed. Also, heavy smokers can reduce the likelihood of a smoking-related death by curtailing their tobacco intake somewhere short of quitting altogether. For a tax to affect health, it is not necessary that it induce heavy smokers to quit, nor is it necessary that it affect heavy smokers at all. Of course, the benefits are greatest for heavy smokers who do quit,

and studies show that mortality ratios for former heavy smokers approach those of never smokers over periods ranging from 5 to 20 years.

It is clear from the preceding discussion that mortality responds to changes in amount smoked, and that the response begins to appear quite soon following these changes. There are also important health effects that will not be realized for decades – those due to reduction in initiation rates by youths. The data considered here will not reflect these long-run effects. Rather, any improvement in health will reflect immediate reductions in quantity smoked in response to tax increases. These effects will be centered in middle-aged or older cohorts. Thus, any observed effects of tax increases on mortality will understate the long-term effects, as they ignore the effects on young smokers who quit, or who don't start smoking, due to the tax increase. Long run effects will also be understated due to the addictive nature of tobacco, whereby long run elasticities exceed those observed in the short run (see Becker, Grossman, and Murphy (1994)).

3. The Demand for Cigarettes.

The literature on the regulation of cigarette smoking and, more generally, on the demand for cigarettes has been summarized extensively elsewhere, and another detailed review is not offered here. (Relevant articles include Viscusi (1992), Keeler et al. (1993), Lewit and Coate (1982), Lewit, Coate and Grossman (1981), Wasserman et al. (1991), Baltagi and Levin (1986), and Schneider, Klein and Murphy (1982)). The findings in this literature indicate that, in aggregate time series and cross section studies, smoking is significantly but inelastically related to prices and excise taxes (see Viscusi

(1992) and Wasserman et al. (1991), for recent reviews). In studies utilizing micro data, Grossman and his colleagues find that the decision to begin smoking by youths is sensitive to price and that, conditional on the decision to smoke, quantity demanded is not overly responsive (See Lewit, Coate, and Grossman (1981), and Lewit and Coate (1982). Grossman (1989) reviews these studies). Wasserman, et al., found no tax effect when controls for the stringency of antismoking regulations were added.

Most recently, interest in the rational addiction model of Becker and Murphy (1988) has spawned a number of studies of smoking behavior. Chaloupka (1989), Becker, Grossman, and Murphy (1994), and Keeler et al. (1993), all find significant price elasticities of tobacco demand using the rational addiction framework. Consistent with the predictions of the rational addiction model, the long-run elasticities exceed the immediate effects.

4. The Sample and the Variables.

Data for this study are drawn from a variety of sources. Tobacco tax and consumption data come from *The Tax Burden on Tobacco*, published annually by the Tobacco Institute. Mortality data are drawn from the *Vital Statistics of the United States, Annual Summary*. Income data are from the *Survey of Current Business*. Data cover the period 1954-1988 for 48 states, yielding 1680 observations. Complete mortality series for these years are available for lung cancer, mouth and throat cancer, cerebrovascular disease, and ischemic heart disease. A slightly shorter series (28 years) is also available on asthma.[6]

Table 1 summarizes the principal characteristics of the key variables. The

average tax on a pack of cigarettes in 1983 prices is $0.39. Per capita consumption of cigarettes averaged about 125 packs per person per year. The cigarette consumption data are not distinguished according to tar and nicotine content or tobacco weight, nor do they measure consumption of hand rolled cigarettes or other tobacco mediums.

The disease rates in Table 1 illustrate the relative importance of the various forms of smoking-related morbidity. Lung cancer and cardiovascular disease represent the most prevalent forms of mortality. These rates reflect mortality from all causes. Smoking attributable mortality, as calculated by the Centers for Disease Control, reflects similar relative magnitudes for these diseases. About 30 percent of the overall mortality for heart disease and lung cancer is currently attributed to smoking (U.S. Centers for Disease Control, 1991).

Cardiovascular disease is by far the greatest immediate cause of death in the United States, with an average annual rate of over 400 deaths per 100,000 population. Death rates for cardiovascular disease have declined steadily over the sample period, however, with the 1970 rate equal to 496, and the 1990 average only 368 (*Statistical Abstract of the United States* (1993), p. 91). The same holds true for the subcategory of ischemic heart disease. Lung cancer is the second most prevalent disease considered, with a death rate of 35 per 100,000. Mouth and throat cancers, which include cancers of the lip, oral cavity, gum, larynx, and pharynx, account for a much smaller percentage of mortality. Likewise, asthma death rates are at least two orders of magnitude smaller than those of heart disease.

5. Empirical Results.

Mortality-Tax regressions

Table 2 presents estimates of the effects of tobacco excise taxes on mortality, controlling for heteroskedasticity, income, state, and period-specific effects and, in the lower portion of the table, for first order autocorrelation.[7] The reduced form regressions have a number of interesting features. Unlike structural consumption-mortality regressions, there is no question of measurement error here, as the tobacco tax rates are (at this level of aggregation) accurately measured. Any substitution away from highly taxed forms of tobacco, and changes to low tar or nicotine brands, are implicitly accounted for in the reduced form estimates (see Viscusi (1994) for a discussion of changes in tar levels and their effect on the evaluation of smoking changes). With a few exceptions, there is not a reverse causality problem, as tobacco taxes have most typically been increased as a revenue measure, rather than to improve the public health, with the exception of the period immediately following the publication of the first Surgeon General's Report. Sims-Granger tests showed no evidence of reverse causality in up to 4 lags of the morality and tax variables.[8]

The regressions in Table 2 utilize values of the tax and income variables lagged one year to reflect delays in the effects of smoking changes on disease. As noted in the literature, taxes exert an immediate effect on smoking. The effects of smoking on disease rates range from 0-1 years for heart disease, 0-2 years for lung cancer, and 2 or more years for mouth and throat cancer. These results combine to suggest that the one year lag of the tax variable is appropriate for the major disease categories.

The Table 2 results indicate that tobacco tax increases in the previous year cause a significant decrease in mortality rates. In the regressions without correcting for AR(1) disturbances, this result holds for each disease category considered, typically at very stringent significance levels. In the three equations in which a substantial degree of autocorrelation is found, (lung cancer, cardiovascular disease, and heart disease) the negative relationship persists in two of the cases, with the estimates again quite precise.[9]

These results present the first direct statistical evidence of the effects of tobacco excise tax changes on mortality. According to the AR(1) corrected estimates in Table 2, a 10 percent increase in the tobacco tax will result in a 1 percent decrease in lung cancer mortality. At the 1990 rate of about 60 lung cancer deaths per 100,000 population and given a population of 250 million people, this implies a saving of 1500 lives per year.[10] Similar results hold for cardiovascular disease and asthma in percentage terms, with the actual savings in lives proportionately larger or smaller depending on the overall death rate. Cardiovascular disease, which accounted for 370 deaths per 100,000 in 1990, would fall by 0.4 percent with a 10% tax hike. Although this elasticity is small, when applied to a population of 250 million, which experiences almost 1 million deaths due to cardiovascular disease annually, the savings in lives would equal about 3700 per year. Conversely, although the asthma death rate would decline by about 1 percent for a 10 percent increase in the tax rate, the low death rate implies a relatively minor number of lives saved.

6. Implications and Conclusions.

This study has provided direct evidence of the effects of tobacco taxes on

mortality rates. The benefits in terms of lives saved due to tobacco tax increases can be substantial, due to the effects of taxes on the relatively high incidence diseases of the cardiovascular system and lung cancer. A 10% increase in the tax rate is estimated here to save about 5200 lives per year or, more accurately, to extend them. The actual benefits in terms of life years saved depends on the distribution of effects across age categories. Furthermore, the quality of the life extended will depend upon, among other things, health. The net impact on health care costs is ambiguous, as it depends on resource utilization by those whose lives are extended. The purpose of this paper is not, however, to sort out and evaluate these issues and make a welfare judgment. Issues of whether the existing tax rate on tobacco is too high or too low are covered elsewhere (see Manning, et.al. (1990) and Viscusi (1994)).

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FOOTNOTES

1. Heavy smoking and heavy drinking have been shown to be harmful to health. Light to moderate tobacco consumption also produces adverse health effects. The evidence on light to moderate drinking is mixed.

2. There is some suggestion in the literature that the state tobacco tax increases in the mid-1960s reflected concern about the health effects of smoking, so that these tax increases could not be considered as causal of the mortality effects. To circumvent this, lagged values of the tax variables are used in the mortality regressions. Also, statistical tests of causality indicate that lagged mortality does not "cause" tax changes, in the sense of Granger (1969) and Sims (1972).

3. Details related to the findings reported in this section are available in a longer version of this paper (Moore, 1994) available on request. See also the various Surgeon General's Reports.

4. Unstructured lag models of the effects of tax rate changes did not yield any meaningful insights, due to apparent collinearity of the lagged tax variables.

5. These results are available in Moore (1994).

6. A more comprehensive, disaggregated analysis of smoking related mortality using unpublished Vital Statistics data is the subject of my ongoing research.

7. Standard errors are corrected for heteroskedasticity using White's (1980) correction. In each regression estimated, the fixed effect specification is indicated using Hausman's (1978) specification test of fixed vs. random effect models.

8. As shown by Nickell (1981), fixed effect estimates of a model with a lagged dependent variable, such as are used in the causality tests, are biased. However, for a panel with a time series dimension T = 35, the bias is very small. Holtz-Eakin, Newey, and Rosen (1988) develop instrumental variables approaches to testing causality in panel data sets, but these are not implemented here, given the length of the panel.

9. According to Monte Carlo results in Griliches and Rao (1969), the uncorrected least squares estimates are at least as good in terms of small sample performance as the GLS estimates when ρ is less than 0.30. Thus, the uncorrected results from the mouth and throat cancer and asthma regressions are preferred to the corrected regressions, while the corrected results are preferred in the remaining cases.

10. A death rate of 60 per 100,000 and a population of 250 million implies 150,000 deaths annually. A 1% reduction yields 1500 lives saved.

Table 1
Descriptive Statistics and Variable Definitions

Variable	(Std. Deviation)	Definition
Cigarettes	124.79 (29.67)	Per capita packs of tobacco consumption in state s, year t (Source: *Tax Burden on Tobacco*, Tobacco Institute, 1991).
Real Tax	38.68 (10.57)	Real state and Federal excise tax in cents per pack of cigarettes in state s, year t (Source: *Tax Burden on Tobacco*, Tobacco Institute, 1991). Deflated to 1983 prices using Consumer Price Index.
Real Income	8519.4 (2121.8)	Real per capita income in state s, period t (Source: Survey of Current Business, various issues). Deflated to 1983 prices using Consumer Price Index.
Lung Cancer	34.75 (14.55)	Death rate (per 100,000 population) from lung cancer in state s, year t (Source: Vital Statistics Annual Summary (VSA), various years).
Heart Disease	264.12 (68.08)	Death rate (per 100,000 population) from ischemic heart disease in state s, year t (Source:VSA).
Mouth and Throat Cancer	3.28 (.95)	Death rate (per 100,000 population) due to mouth and throat cancers in state s, year t (Source: VSA).
Cardiovascular Disease	461.33 (88.79)	Death rate (per 100,000 population) due to cardiovascular disease in state s, year t (Source: VSA).
Asthma	1.76 (1.01)	Death rate (per 100,000 population) due to asthma in state s, year t (Source: VSA).

Table 2
Mortality-Tobacco Tax Regressions[a]
Estimated Coefficients and Standard Errors

	Lung	*Mouth & Throat*	*Cardio-vascular*	*Heart*	*Asthma*
No AR(1) Correction					
ln(Real Tax)[b]	-.186***	-.065*	-.076***	-.090***	-.127***
	(.024)	(.038)	(.024)	(.018)	(.056)
ln(Real Income)[b]	.548***	.167*	.257***	.545***	.412***
	(.067)	(.119)	(.042)	(.053)	(.160)
Adjusted R^2	.955	.689	.855	.909	.782
ρ	.465***	.073***	.425***	.845***	.182***
	(.022)	(.025)	(.022)	(.013)	(.026)
Sample Size	1632	1632	1632	1632	1488
AR(1) Corrected					
ln(Real Tax)[b]	-.094***	-.053	-.043**	.001	-.087*
	(.020)	(.042)	(.024)	(.008)	(.056)
ln(Real Income)[b]	.221***	.140*	.111***	.006	.348**
	(.052)	(.101)	(.037)	(.023)	(.166)
Adjusted R^2	.880	.546	.713	.699	.703
Sample Size	1584	1584	1584	1584	1440

[a] Each regression also included as control variables 47 state dummy variables and time dummy variables for each year represented in the data. Excluded states are Alaska and Hawaii. All regressions cover the period 1955-88, except for the asthma regression, which includes the years 1961-88 only. All estimates are corrected for heteroskedasticity, using White's (1980) correction.

[b] Variable lagged one period.

*** Statistically significant, 0.01 level, one-tailed test.
** Statistically significant, 0.05 level, one-tailed test.
* Statistically significant, 0.10 level, one-tailed test.