THE AMERICAN COUNCIL
ON SCIENCE AND HEALTH

SPECIAL RELEASE

A Critical Assessment of "Lies, Damned Lies, & 400,000 Smoking-Related Deaths"

by Robert Levy and Rosalind Marimont Published in *Regulation*,* Fall 1998

** by Alicia M. Lukachko, M.P.H.
and Elizabeth M. Whelan, Sc.D., M.P.H.

October, 1999

Executive Summary

In the Fall 1998 issue of *Regulation* ("The Cato Review of Business and Government"), the Cato Institute*** published an article by Robert Levy and Rosalind Marimont titled "Lies, Damned Lies, & 400,000 Smoking-Related Deaths." In their article, Levy and Marimont contend that the U.S. government's estimate of approximately 400,000 annual premature deaths due to cigarette smoking is scientifically unsound and substantially inflated. The authors assert that "the war on smoking . . . has grown into a monster of deceit and greed, eroding the credibility of government and subverting the rule of law."

In this report, scientists at the American Council on Science and Health refute Levy and Marimont's key arguments (presented below) as unscientific and inflammatory. ACSH's critique concludes that the estimate of 400,000 annual deaths due to cigarette smoking is indeed reliable and may even be an underestimate.

The authors make four primary assertions leading to their conclusion that the figure of 400,000 is inflated. Those four assertions and a summary of ACSH's critique of each of them follow.

---

* Regulation is a publication of the Cato Institute

** Alicia M. Lukachko is the Assistant Director of Public Health at the American Council on Science And Health
Dr. Elizabeth M. Whelan is President of the American Council on Science And Health

*** The Cato Institute, whose headquarters are in Washington, DC, is a nonpartisan public policy research foundation advocating limited government and free markets

ACSH
1995 BROADWAY
2ND FLOOR
NEW YORK, NY
10023-5860

DR. ELIZABETH M. WHELAN
PRESIDENT

1. *Levy and Marimont discount over one-third of the estimated 400,000 annual deaths caused by smoking with the erroneous claim that "small" increases in the risk of disease or death—marked by relative risks less than 2.0—are "statistically insignificant," and "insuffi - ciently reliable to conclude that a particular agent (e.g., tobacco) caused a particular dis - ease."*

Contrary to the authors' misstatement, relative risks less than 2.0, while small, can indeed be statistically significant and reflect a causal relationship. A relative risk is a measure of the strength of an association between exposure (e.g. smoking) and a disease. Given the pervasiveness of a risk factor, such as smoking, and the prevalence of some of the diseases it causes, small relative risks can, and do, represent serious threats to public health.

Levy and Marimont's assumptions regarding small relative risks violate basic principles of epidemiology. The authors confuse two distinct concepts, that of relative risk and that of statistical significance. The size of a relative risk, alone, *does not* signify its statistical significance. Rather, research findings must undergo statistical tests to assess their "significance." Small relative risks suggest a weak association (or risk factor), not necessarily an insignificant finding.

Moreover, relative risks of *any* value, when considered alone, are insufficient to conclude that an association is causal. Relative risks and several other factors (i.e., the consistency of the finding across studies, the biologic plausibility of the hypothesis, the presence of a dose-response relationship and the time sequence of the cause and effect) must be considered when judging causality.

2. *Levy and Marimont argue that the American Cancer Society's Cancer Prevention Survey (CPS)—a widely used data set for the calcula - tion of public health statistics—is unrepresenta - tive of the general population and is therefore "the wrong sample [to use] as a standard of comparison" when estimating smoking-related deaths in the US.*

It is true that the American Cancer Society's Cancer Prevention Survey includes a greater proportion of white, older, more educated, married and middle-class participants than does the general U.S. population. However, this characteristic alone does not undermine the findings derived from this data set. The CPS, with over one million participants, is the largest study collecting data on smoking and mortality over an extended period of time. It has a uniquely strong study design from which valid estimations have been drawn.

Moreover, the relative risks of dying from smoking-related diseases derived from the CPS are within the range of those from other studies. This consistency lends to the reliability of the CDC's estimate of smoking-related deaths that use relative risks drawn from the CPS.

3. *The authors state that the Centers for Disease Control and Prevention (CDC) fails "to control for obvious confounding variables" in its calculation of smoking-related deaths. They argue that after accounting for other factors that may contribute to deaths among smokers, the CDC's estimate should be greatly reduced.*

According to Levy and Marimont, "if a smoker who is obese; has a family history of high cholesterol, diabetes, and heart problems; and never exercises, dies of a heart attack, the government attributes his death to smoking alone." What the authors are reasonably questioning here is the role of potential confounders—other factors that may explain some of the deaths attributed to smoking—on estimates of smoking-related deaths.

The CDC's estimate of annual smoking-related deaths does control for age—the confounding variable that has the greatest impact on the association of smoking with disease and death. Analyses that have controlled for multiple factors (such as exercise and alcohol intake) indicate that the impact of potential confounders on the CDC's age-adjusted risk of death due to smoking would be minimal. Some studies have even found that controlling for certain confounders results in an increase in smoking-attributable mortality. For example, one study (which controlled for risk factors including age, education, alcohol intake, diabetes, and hypertension) found smoking-related mortality estimates to be 2 percent higher than the CDC's age-adjusted estimates.

Furthermore, it is important to note that only a fraction of the deaths from smoking-related diseases are attributed to smoking.

4. *Finally, Levy and Marimont purport that the impact of smoking-related mortality is over - stated, particularly with respect to children, given that the majority of smoking-related deaths occur late in life.*

In fact, it has been estimated that over one-half of all smoking-related deaths occur between ages 35 and 69, which translates into an average loss of roughly 23 years of life. Cigarette smoking also accounts for approximately 30 percent of all deaths among this age group. That the majority of deaths caused by smoking occurs among adults does not mitigate the real risks that cigarettes pose to children.

Levy and Marimont insinuate that the deaths of older adults should not be considered premature or preventable. But many adults remain healthy into their eighties and nineties. It is inappropriate to set an arbitrary age limit on premature death.

In conclusion, Levy and Marimont's arguments do not present a scientifically sound and convincing case that the estimate of 400,000 annual smoking-related deaths is a specious, statistical gimmick. Their essay, however, does illustrate the importance of educating the public about basic epidemiological and biostatistical concepts, including the methods used to determine smoking-related deaths.

Introduction

In the Fall 1998 issue of *Regulation* ("The Cato Review of Business and Government"), the Cato Institute published an article by Robert Levy and Rosalind Marimont entitled "Lies, Damned Lies, & 400,000 Smoking-Related Deaths." In their article, Levy and Marimont contend that the government's estimate of 400,000 annual premature deaths due to cigarette smoking is scientifically unsound and substantially inflated. The authors assert: "The war on smoking . . . has grown into a monster of deceit and greed, eroding the credibility of government and subverting the rule of law."

In May 1999 Levy and Marimont's arguments resurfaced through an article written by *Boston Globe* columnist Jeff Jacoby. Mr. Jacoby's column has been widely circulated and cited in op-ed pages nationwide.

The Levy and Marimont article also served in the defense of American tobacco companies in the recent Florida "Engle case," the largest class action lawsuit filed, and the first won, against the tobacco industry.

For over 20 years, the American Council on Science and Health (ACSH), has relied on sound science to educate the public about real versus hypothetical risks to health. ACSH has paid particular attention to well-established and preventable causes of disease and death, especially cigarette smoking. For these reasons, ACSH is in a unique position to examine the veracity of claims made by Levy and Marimont.

In the following report, ACSH evaluates the plausibility of the estimate that 400,000 premature deaths are attributable to smoking. ACSH reviews the confirmed health problems caused by smoking and explains the scientific methods used to establish these risks. Lastly, ACSH evaluates the key arguments employed by Levy and Marimont to discount the fatalities caused by cigarette smoking each year.

ACSH considers this report a work in progress. As more information becomes available, the report will be updated.

About the Authors

Robert Levy, the lead author of "Lies, Damned Lies, & 400,000 Smoking-Related Deaths," is a senior fellow at the Cato Institute specializing in constitutional studies. With a J.D. degree, and a Ph.D. degree in business, he is also an adjunct professor at the Georgetown University Law Center. Although Levy does not have a background in science, he has written extensively about tobacco from a business perspective.

Rosalind B. Marimont, the article's co-author, is described as a mathematician and scientist. Before her retirement in 1979, Marimont worked for the Bureau of Standards (now the National Institute of Standards and Technology). A prominent contributor to the pro-tobacco group FORCES (Fight Ordinances and Restrictions to Control and Eliminate Smoking), Marimont has written several essays criticizing the focus of public health groups on tobacco. Marimont has also been an active member of the National Smokers' Alliance and has testified against local legislation in Maryland to restrict smoking in public places.

The Health Hazards of Smoking

Cigarette smoking has been recognized as a leading cause of disease and death for at least 40 years. Few subjects have received such thorough and extensive scientific scrutiny by both governmental and independent bodies. Thousands of scientific studies have confirmed that smoking is a major health hazard.[1] Besides the relationship between smoking and disease, many studies have found that the overall death rate among smokers is 2–3 times greater than that of nonsmokers.[2] Cigarettes also contain nicotine, a chemical proven to be highly addictive (which has been acknowledged in internal tobacco-industry documents).

Despite the overwhelming evidence to the contrary, Levy and Marimont state that the hazards of smoking remain largely speculative. They allege that the "war on smoking started with a kernel of truth—that cigarettes are a high risk factor for lung cancer." Ironically, it is Levy and Marimont's article that contains only a kernel of truth about the risks of smoking. In fact, while active cigarette smoking has been causally linked to lung cancer, it is also associated with an array of other diseases, listed below.[3]

- Cigarette smoking is a principal cause of cancer of the esophagus, larynx, lip, mouth, pharynx, tongue, kidney, pancreas, urinary bladder, and uterine cervix.

- Cigarette smoking has also been identified as a major cause of cardiovascular disease, including atherosclerosis, coronary heart disease (angina and heart attack), stroke, sudden death, and aortic aneurysm.

- Cigarette smoking causes chronic obstructive lung disease (emphysema, chronic bronchitis, and related conditions). Smokers have been found to suffer more respiratory problems (such as colds, pneumonia, influenza, and bronchitis) and their recovery from these illnesses is slower.

- For men under age 65, smoking has been shown to be an independent risk factor for impotence, including erectile dysfunction. For women, smoking can impair fertility, induce premature menopause and spontaneous abortion, and lead to a host of complications of pregnancy and childbirth.

- Cigarette smoking increases the risk for osteoporosis (a reduction in bone mass) and periodontal (gum) disease.

- Smoking precipitates premature hearing loss, and vision problems, including blindness secondary to cataracts and macular degeneration, and premature hearing loss.

- Smokers face a significantly greater chance than nonsmokers of suffering complications during and after surgery.

Evidence suggests that smoking also increases the risk for other diseases, such as rheumatoid arthritis, and cancers of the prostate and stomach. These relationships, however, have not yet been scientifically established.

Preliminary research also indicates that cigarette smoking may be associated with reduced risk for endometrial cancer and Parkinson's disease. Yet the harmful effects of cigarette smoking dramatically outweigh any of the potential benefits. By identifying and isolating the components of smoking that may have positive effects, potential benefits may be achieved while avoiding exposure to the many toxins contained in cigarette smoke.

Environmental Tobacco Smoke

A mounting body of scientific research reveals that exposure to environmental tobacco smoke (ETS) also poses health risks. The most common and firmly established adverse health effects associated with exposure to ETS are irritation of the eyes, nose, and respiratory tract; exacerbation of asthma and emphysema; and increased susceptibility to respiratory infections.[4] Furthermore, studies have consistently shown that ETS contributes to lung cancer and heart disease.[4]

As the Levy and Marimont article itself illustrates, the concerns over secondhand smoke extend far beyond public health. The political implications of finding a causal association between ETS and disease have fueled long and bitter struggles between interested parties (pro- and anti-tobacco organizations and individuals). To counter the growing trend toward indoor-smoking restrictions and to allay public fears, some parties, including the tobacco industry, have argued that ETS does not pose a "meaningful" lung cancer risk—and therefore does not present a threat to public health.

Similarly, authors Levy and Marimont focus their arguments about secondhand smoke exclusively on lung cancer in an attempt to dismiss all of the health effects associated with ETS. Their argument is simplistic, as it ignores ETS-related health risks other than lung cancer that should also be considered when

developing public health policy—heart disease and respiratory illnesses, for example.

Establishing Cause and Effect

Scientists rely on epidemiology ("the study of the distribution and determinants of disease frequency"[5]) to determine whether a factor, such as cigarette smoking, causes a particular health outcome (e.g., disease or death). They begin by suggesting and then establishing an association. In the case of smoking, the optimal way to evaluate the effect of smoking on health is to compare groups of smokers with groups of nonsmokers to assess the differences (if any) in health outcomes between them. Researchers try to assure that the groups being compared (i.e., smokers and nonsmokers) have similar characteristics in addition to their smoking behavior. By doing so, differences in health outcomes found between these groups are more likely attributable to smoking than to other factors. Dissimilarities between groups can also be accounted for in the analysis of the research data.

Once an association is found between an exposure (e.g., smoking) and a health outcome (e.g., disease), researchers must determine whether the apparent association is *valid*. A valid association is one that is unlikely to be due to chance, bias (on the part of researchers or study participants), or confounders—other factors that caused the disease and are independently associated with smoking.[5]

Statistical tests are routinely applied to research findings to assess the probability that the results are not merely coincidental. A "statistically significant" finding means that the result is unlikely to be due to chance. Factors such as the number of people being examined (sample size) and the strength of the association between the exposure and health outcome influence whether or not a finding will achieve "statistical significance." Generally, the larger the sample size and the stronger the effect of the risk factor, the more likely it is that the results will be found to be significant.

While finding a statistically significant result denotes that *chance* is unlikely to account for the results, *bias* and potential *confounders* must still be addressed to demonstrate a valid association. Furthermore, a statistically significant finding does not alone confirm a causal relationship.

To conclude that a particular disease is *caused* by smoking, five basic criteria must be considered.[5] Researchers must assess:

1. *the strength of the association found between smoking and disease;*

A *relative risk* is one measure of the strength of the association between a risk factor and disease (e.g., between smoking and disease) and is the ratio of disease among smokers to disease among nonsmokers. A relative risk equal to 1 indicates that there is no association between the exposure and the outcome. The closer the relative risk is to 1, the *smaller* or *weaker* the association.

A relative risk of 2.0 for example, indicates that those exposed to a particular agent (e.g., smokers) are twice as likely than those unexposed (e.g., nonsmokers) to develop the health outcome under study (e.g., death from heart disease). The larger the relative risk, the less likely the effect is due solely to bias or confounders. Still, small relative risks do not exclude the possibility that a causal relationship exists, nor do they preclude the possibility that a finding is statistically significant.

2. *the consistency of the finding across studies;*

If several well-designed studies replicate a finding, it is more likely that the relationship being studied is real. As stated previously, the enormous body of research examining the health effects of smoking corroborate the relationship between smoking and disease.[3]

3. *the biologic plausibility of the hypothesis;*

The relationship between exposure and disease must be consistent with what is currently known about biology and the disease process. Much is understood about the biological mechanisms by which smoking causes disease, though more remains to be learned. It is known that cigarette smoke contains approximately 4,000 chemical components, many of which are toxins and some of which are human carcinogens.

4. *the presence of a dose-response relationship; and*

In a dose-response relationship, increases in the degree of exposure are associated with increases in risk. Numerous studies have demonstrated that increases in cigarette use (duration and number of cigarettes smoked) increase the risk for smoking-related disease and death.[3]

5. *the time sequence of the cause and effect.*

The exposure or hypothesized cause must logically precede the effect. Research from multiple study designs affirm that cigarette use among study participants does in fact occur before the health outcomes that are measured.

The basic principles of epidemiology presented above are critical to evaluating the claims made by Levy and Marimont.

Calculating Premature Deaths Due to Cigarette Smoking

The number of deaths that are attributable to cigarette smoking may be thought of as the reduction in deaths that would be achieved if the entire population had never smoked. The basic calculation involves multiplying the death rates for smoking-related diseases among representative nonsmokers by the number of people in the entire population and then subtracting the resulting expected number of deaths from the actual number of these deaths.

Since a significant number of persons who have smoked in the past have quit, and thus have a greater risk of smoking-related disease than those who have never smoked, some formulas, such as that used by the CDC, distinguish between current smokers, former smokers and "never-smokers." The CDC estimates the proportion of deaths from a particular disease (e.g., lung cancer) by multiplying the difference in death rates between smokers and nonsmokers by the proportions of the population that smoke. The formula uses differences in death rates of current smokers compared to never-smokers, and of former smokers compared to never-smokers. These differences are then multiplied by the proportions of the population who currently smoke and used to smoke.

Estimations of this death toll can vary widely depending on the diseases considered to be smoking-related, the data sources used, the control for confounding variables (e.g. age) and variations in formulas.

For over two decades, the U.S. government has been estimating the number of Americans who die prematurely from smoking. According to current government calculations, approximately 430,000 deaths occur each year in the United States as a result of cigarette smoking.[6] Some larger estimates fall in the range of 600,000 to 700,000 annual deaths due to smoking.[7]

In "Lies, Damned Lies, & 400,000 Smoking-Related Deaths," Levy and Marimont challenge the veracity of the associations found between smoking and disease, and ultimately the estimate that 400,000 premature deaths are caused by smoking each year. By employing largely haphazard and unscientific methods, the authors try to minimize smoking's death toll.

The section below assesses the following key arguments maintained by Levy and Marimont, namely that:

- Relative risks less than two are "statistically insignificant" and "insufficiently reliable to conclude that a particular agent (e.g., tobacco) caused a particular disease."
- The Cancer Prevention Survey (CPS) is unrepresentative of the general population and is therefore "the wrong sample [to use] as a standard of comparison" when estimating smoking-related deaths in the US.
- The Centers for Disease Control and Prevention (CDC) fails "to control for obvious confounding variables" in its calculation of smoking-related deaths.
- The impact of smoking-related mortality is overstated, particularly with respect to children, given that the majority of smoking-related deaths occur late in life.

Argument 1: Relative risks less than 2.0 are "statistically insignificant" and "insufficiently reliable to conclude that a particular agent (e.g., tobacco) caused a particular disease." Based on this claim, Levy and Marimont subtract over 150,000 of the 400,000 annual deaths caused by smoking.

Relative risks less than 2.0, while small, can indeed be statistically significant and reflect a causal relationship. Given the pervasiveness of a risk factor, such as smoking, and the prevalence of some of the diseases it causes, small relative risks can, and do, represent serious threats to public health. For example, cigarette smoking is a much greater risk factor for mortality from lung cancer than from heart disease.[5] But, because heart disease affects many more people than lung cancer, the number of smoking-related deaths from heart disease rivals those from lung cancer.[6]

Levy and Marimont's assumptions regarding small relative risks violate basic principles of epidemiology. The authors confuse two distinct concepts, that of relative risk and that of statistical significance.

The size of a relative risk, alone, does not signify its statistical significance. Rather, as explained in the previ-

ous section, research findings must undergo statistical tests to assess their "significance." Small relative risks suggest a weak association (or risk factor), not necessarily an insignificant finding. Again, small relative risks may have a substantial public health impact if the exposure and the health outcome affect a large proportion of the population.

Moreover, relative risks of *any* value, when considered alone, are insufficient to conclude that an association is causal. As discussed above, relative risks are one of many factors that must be considered when judging causality. An association of small magnitude can be judged as cause and effect after considering the totality of the evidence.

A good illustration of this point is offered by the authors themselves. In their derision of the risks associated with ETS, Levy and Marimont claim that "the relative risk of lung cancer for persons who drink whole milk is 2.4." Even if we accept this highly dubious association, the other criteria necessary to judge causality (i.e., biologic plausibility, consistency of findings, etc.) are not fulfilled. Thus, whole milk cannot be legitimately judged as a cause of lung cancer given the relative risk alone.

The authors mislead readers by misrepresenting a quotation from the National Cancer Institute, which qualifies relative risks, as the agency's "own guideline." In fact, the NCI has no such guideline about relative risks, and the quotation cited is taken from a 1994 NCI press release on abortion and the risk of breast cancer. Given its proper context, this so-called guideline makes a much different point from what the authors suggest.

The authors use the arbitrary and unscientific ceiling of relative risks less than 2.0 as a means of reducing the CDC's estimate of smoking by 163,071 deaths. But, based on the arguments presented above, their logic is fundamentally flawed.

Argument 2: The American Cancer Society's Cancer Prevention Survey (CPS)—a widely used data set for the calculation of public health statistics—is unrepresentative of the general population and is therefore "the wrong sample [to use] as a standard of comparison" when estimating smoking-related deaths in the US.

It is true that the American Cancer Society's CPS includes a greater proportion of white, older, more educated, married, and middle-class participants than the general U.S. population.[2] However, this characteristic alone does not undermine the findings derived from this data set. The CPS has a uniquely strong study design from which valid estimations have been drawn.

Moreover, and perhaps more important, the relative risks of dying from smoking-related diseases derived from the CPS are within the range of those from other studies. This consistency lends to the reliability of the CDC's estimate of smoking-related deaths that use relative risks drawn from the CPS.

The important issue of generalizability (whether the results are applicable to other populations) should be considered only after a study has been determined to be valid. Levy and Marimont overlook the overriding strengths of the CPS: its excellent study design and valid findings. With over one million study participants, the CPS is the largest U.S. study that collects data over an extensive period of time on the relationship between smoking and mortality.

After accepting that the results of the CPS reflect valid cause and effect relationships, the next important question is: How would the results from a mostly white and middle-class population differ, if at all, from those among the general U.S. population? The answer depends on how the data are used. The absolute mortality rates are lower in the CPS than in the general population. But, the CDC's estimation of smoking-related deaths relies on ratios—relative risks comparing smokers with nonsmokers and former smokers within the CPS. These relative risks of smoking-related disease have been found to be within a reasonable range of those from other studies, and therefore, enhance the reliability of the CDC's estimate.[8]

Levy and Marimont advocate substituting data from the National Center for Health Statistics (specifically the National Mortality Followback Survey and the National Health Interview Survey) for data from the CPS—an approach proposed by long-time tobacco industry consultant T.D. Sterling.[9] However, Sterling's approach has been justly criticized for its implausible findings (e.g., previous smoking was found to be protective against coronary heart disease and cerebrovascular disease among males over age 65), and for combining data from two surveys with largely dissimilar, and thus incompatible, study designs.[10,11]

On the other hand, the CPS uses the appropriate study design (a large prospective cohort*) to provide valid relative risk estimates used in the calculation of the number of premature deaths due to smoking.

* A prospective cohort study is one that follows a group of exposed (e.g. smokers) and unexposed (e.g. nonsmokers) subjects over an appropriate time period to observe the health outcome(s) under study.

Argument 3: The Centers for Disease Control and Prevention (CDC) fails "to control for obvious confounding variables" in its estimation of smoking-related deaths. Levy and Marimont argue that after accounting for other factors that may contribute to deaths among smokers, the CDC's estimate should be greatly reduced.

The CDC's estimate of annual smoking-related deaths does control for age, the confounding variable that has the greatest impact on the association of smoking with disease and death. Analyses that have controlled for multiple factors (such as exercise and alcohol intake) indicate that the impact of potential confounders on the age-adjusted risk of disease/death due to smoking is minimal.[8,12]

According to Levy and Marimont, "if a smoker who is obese; has a family history of high cholesterol, diabetes, and heart problems; and never exercises dies of a heart attack, the government attributes his death to smoking alone." What the authors are reasonably questioning here is the role of potential confounders—other factors that may explain some of the deaths attributed to smoking—on estimates of smoking-related deaths. For some diseases, such as lung cancer, which is almost wholly attributable to smoking (approximately 87 percent of lung cancers are caused by smoking), the influence of confounders is trivial. But, for diseases that have multiple notable risk factors (such as cardiovascular disease), the effect of confounders may indeed be significant.

As Levy and Marimont point out, failing to account for confounders can result in inaccurate estimates of smoking-related deaths. Yet, the authors incorrectly assume that by controlling for potential confounding factors, the CDC's age-adjusted calculation would be substantially diminished. In fact, controlling for some confounders has been shown to result in increases in attributable risk that would suggest that the CDC's estimate may be conservative.[8]

Moreover, only a fraction of the deaths from smoking-related disease are attributed to smoking.

When assessing the impact of controlling for confounding variables on the CDC's estimate, it is important to consider the results of studies that have examined the effects of confounders on smoking risk. The Nurses' Health Study is a well-designed prospective cohort study with 12 years of follow-up on registered nurses in the U.S. Results of this study reveal that after controlling for potential confounders (including hypertension, diabetes, high serum cholesterol, weight, parental history of heart attack before age 60, past use of oral contraceptives, postmenopausal estrogen use, and age at starting smoking), the multivariate relative risk of 1.87 for *total mortality* comparing current smokers with "never-smokers" was basically the same as the age-adjusted estimate of 1.86.[12] This study also showed a slight strengthening of the association between current smoking and mortality due to cardiovascular disease after adjusting for alcohol and exercise.[12]

Another analysis of the CPS data (used by the CDC)—which controlled for risk factors including age, education, alcohol intake, diabetes and hypertension—found smoking-related mortality estimates for the combined disease categories of lung cancer, ischemic heart disease, bronchitis/emphysema, chronic airway obstruction, and cerebrovascular disease to be 2 percent higher than the CDC's age-adjusted estimates.[8]

Contrary to Levy and Marimont's claim, the available data (presented above) strongly suggest that further adjustment for potential confounders (other than age) would have little impact on the CDC's estimate of roughly 400,000 smoking-related deaths.

Argument 4: The impact of smoking-related mortality is overstated, particularly with respect to children, given that the majority of smoking-related deaths occur late in life.

In fact, it has been estimated that over one half of all smoking-related deaths occur between ages 35 and 69, which translates into an average loss of roughly 23 years of life. Cigarette smoking also accounts for approximately 30 percent of all deaths among this age group.[13] That the majority of deaths due to smoking occur among adults does not mitigate the real risks that cigarettes pose to children.

Levy and Marimont aver that smoking "kill[s] people at an average age of roughly seventy-two—far closer to ninety-nine than to childhood or even young adulthood." This unreferenced assertion is inconsistent with studies suggesting that the average age of death among smokers is well below age 72.[13,14]

It is important to consider that what the authors are reporting is an average age of death. Cigarette smoking kills people at ages much younger than 72, as well as at ages older than 72. Long-term follow-up studies of smokers versus nonsmokers have found that smokers are three times more likely to die between the ages of 45 and 64, and two times more likely to die between the

ages of 65 and 84, than are nonsmokers.[2] Thirty-three percent of nonsmokers live to age 85, while only 12 percent of smokers live that long.[2]

Levy and Marimont insinuate that the deaths of older adults should not be considered premature or preventable. But many adults remain healthy into their eighties and nineties. It is inappropriate to set an arbitrary age limit on premature death. A premature, preventable death is a premature, preventable death at any age. The authors' underlying assumption is that deaths among the "old" are less consequential than deaths among the young. This "modest proposal" controverts the fundamental, humanitarian principle of medicine and public health: All human lives are valuable.

Levy and Marimont present smoking-related deaths in terms of years of potential life lost (YPLL) in an attempt to minimize its impact. The authors, however, rely on an outdated approach to calculating YPLL by considering only those years under age 65. YPLL is more accurately calculated from life expectancy, which extends well beyond age 65.

After inappropriately comparing smoking-attributed mortality with immediate deaths from motor vehicle accidents, suicide, and homicide, the authors state that "measured by YPLL, tobacco was . . . not 'the number one killer in America' as alarmists have exclaimed." Some causes of premature, preventable deaths do occur at a much younger age than those due to cigarette smoking. Still, considering the vast number of deaths caused by cigarette use, smoking remains the leading cause of preventable death.

It is important to note that YPLL is just one of many measures representing the public health impact of a risk factor. Aside from the mortality due to smoking, the authors fail to take into account smoking-related morbidity and the poor quality of life that often accompanies the chronic illnesses caused by cigarette smoking.

The authors assert that the concern about youth cigarette smoking is unfounded because the majority of cigarette-related deaths occur later in life. They suggest that alcohol and drug abuse are more legitimate threats to youth. However, the dangers from alcohol and drug abuse do not preclude those from cigarette smoking.

Cigarettes and cigarette smoke contain nicotine, a powerfully addictive drug. People who begin smoking as children are more likely to become lifetime smokers, and, therefore, to die from smoking-caused disease.[12] Smoking at a young age (or any age) causes irreversible genetic and cellular damage that may take years to surface as disease.[15,16] Furthermore, studies have found that cigarette smoking is associated with, and tends to precede, alcohol and illicit drug use—the very behaviors Levy and Marimont deem most threatening to children.[17,18]

Levy and Marimont's arguments obscure the real risks associated with cigarette smoking—effects that may not be immediately observed, but are harmful nonetheless.

Conclusion

Levy and Marimont fail to present a scientifically sound and convincing argument that the estimate of 400,000 annual smoking-related deaths is a specious, statistical gimmick. Their assumptions about the effect of potential confounders and their dismissal of relative risks less than 2.0 as a means to minimize smoking's death toll are unsupported. Moreover, their criticisms of the CPS data and their disregard for the long-term impact of cigarette smoking are misguided. Our assessment concludes that the estimate that 400,000 people die from cigarette smoking each year is indeed reliable and may even be an underestimate.

"Lies, Damned Lies, & 400,000 Smoking-Related Deaths" does, however, bring to light some reasonable questions that the public may share about the methods used to determine smoking-related deaths. The article clearly illustrates the importance of educating nonscientists about basic epidemiological and biostatistical concepts.

In their conclusion the authors make further misleading and unscientific claims, stating, for example, that "the actual damage from smoking is neither known nor knowable with precision." As stated previously, smoking and tobacco use is the single most-studied health risk factor in the history of human health research. In fact, the first report of diminished life span among smokers appeared in 1938. The pathological effects of chronic tobacco use in individuals are well documented. Using rigorous study designs and analytical methods, scientists have established with a high degree of certainty the causal role of tobacco in disease and death.

Levy and Marimont suggest that the "correctly calculated number of smoking-related deaths" nears 100,000 per year. Even if one were to accept this gross miscalculation, is not the premature, debilitating, often painful death of "only" 100,000 Americans (of any age) worthy of being addressed as a significant public health problem?

The authors might well heed their own advice when they criticize federal officials for "tainting science to

advance predetermined ends." By straying from the most basic epidemiological principles in their arguments, and by touting opinions that masquerade as facts, the authors have themselves strayed far from science.

References

1. American Council on Science and Health. *Smoking or Health. . . It's Your Choice.* New York: American Council on Science and Health, 1994.

2. National Institutes of Health, National Cancer Institute. *Changes in Cigarette-Related Disease Risks and Their Implication for Prevention and Control. Monograph 8*. Washington DC: National Institutes of Health, 1997.

3. American Council on Science and Health. *Cigarettes: What the Warning Label Doesn't Tell You*. New York: Prometheus Books, 1997.

4. American Council on Science and Health. *Environmental Tobacco Smoke: Health Risk or Health Hype?* New York: American Council on Science and Health, 1999.

5. Hennekens C, Buring J. *Epidemiology in Medicine*. Boston: Little, Brown and Company, 1987.

6. Centers for Disease Control and Prevention. Cigarette smoking-attributable mortality and years of potential life lost—United States, 1990. *Morbidity and Mortality Weekly Report*. May 23, 1997;46:444-451.

7. Orleans CT. *Nicotine Addiction: Principles and Management*. Oxford: Oxford University Press, 1993.

8. Schulman J, Epstein L, Mowery PD, Pierce B, Euskirchen E, Abed J. *Smoking Attributable Mortality: Control for Confounding*. Atlanta, Georgia: Battelle, Centers for Public Health Research and Evaluation, 1997.

9. Sterling TD, Rosenbaum WL, Weinkam JJ. Risk attribution and tobacco-related deaths. *American Journal of Epidemiology*. 1993;138:128-139.

10. Siegel M, Arday DR, Merritt RK, Giovino GA. Letter to the Editor. *American Journal of Epidemiology*. 1994;140:1051-1052.

11. Choi BCK, Pak AWP. Letter to the Editor. *American Journal of Epidemiology*. 1994;140:1051-1052.

12. Kawachi I, Colditz GA, Stampfer MJ, Willett WC, Manson JE, Rosner B, Hunter DJ, Hennekens CH, Speizer FE. Smoking cessation in relation to total mortality rates in women. *Annals of Internal Medicine*. 1993;119:992-1000.

13. Peto R, Lopez AD, Boreham J, Thun M, Heath C Jr. Mortality from tobacco in developed countries: indirect estimation from national vital statistics. *Lancet*. 1992;339:1268-78.

14. Peto R, Lopez AD, Boreham J, Thun M, Heath C Jr, Doll R. Mortality from smoking worldwide. *British Medical Bulletin*. 1996;52:12-21.

15. American Council on Science and Health. *The Irreversible Health Effects of Cigarette Smoking*. New York: American Council on Science and Health, 1998.

16. Wiencke JK, Thurston SW, Kelsey KT, Varkonyi A, Wain JC, Mark EJ, Christiani DC. Early age at smoking initiation and tobacco carcinogen DNA damage in the lung. *Journal of the National Cancer Institute*. 1999;91:614-619.

17. Li-Tzy W, Anthony, JC. Tobacco Smoking and Other Suspected Antecedents of Nonmedical Psychostimulant Use in the United States, 1995. *Substance Use & Misuse*. 1999;34:1243-1259.

18. Kandel DB, Yamaguchi K, Chen K. Stages of progression in drug involvement from adolescence to adulthood: further evidence for the gateway theory. *Journal of Studies on Alcohol*. 1992;53:447-57.

ACSH gratefully acknowledges the comments and contributions of the following:

Paul Brodish, M.P.H.
University of North Carolina School of Public Health

Gregory N. Connolly, D.M.D., M.P.H
Massachusetts Department of Public Health

Richard Daynard, J.D., Ph.D.
Northeastern University School of Law

Clifford E. Douglas, J.D.
Tobacco Control Law & Policy Consulting Ann Arbor, Michigan

Joseph C. Elia, M.D.
Medical Page Reno, New York

Jeffrey L. Fellows, Ph.D.
Centers for Disease Control and Prevention

Gary Giovino, Ph.D.
Roswell Park Cancer Institute

Victor Herbert M.D., J.D., F.A.C.P.
Mount Sinai School of Medicine Bronx Veteran Affairs Medical Center

Thomas Houston, M.D.
American Medical Association

Corrine G. Husten, M.D., M.P.H.
Centers for Disease Control and Prevention

Ruth Kava, Ph.D., R.D.
American Council on Science and Health

R.T. Raventholt, M.D., M.P.H.
Population Health Imperatives Seattle, Washington

Gilbert Ross, M.D.
American Council on Science and Health

Donald R. Shopland, Ph.D.
National Cancer Institute National Institutes of Health

Robert Sklaroff, M.D.
Medical Oncology/Hemotology Elkins Park, Pennsylvania

John Slade, M.D.
School of Public Health of New Jersey

Frederick Steinberg, M.D., M.B.A.
Hertfordshire, England

Michael Thun, M.D., M.S.
American Cancer Society

Ken Warner, Ph.D.
University of Michigan School of Public Health

Joel E. White, M.D., F.A.C.R.
Radiation Oncology Center Walnut Creek, California

ACSH accepts unrestricted grants on the condition that it is solely responsible for the conduct of its research and the dissemination of its work to the public. The organization does not perform proprietary research, nor does it accept support from individual corporations for specific research projects. All contributions to ACSH—a publicly funded organization under Section 501(c)(3) of the Internal Revenue Code—are tax deductible.

Individual copies of this report are available at a cost of $3.00. Reduced prices for 10 or more copies are available upon request.

SPECIAL RELEASE

A Critical Assessment of

"Lies, Damned Lies, & 400,000 Smoking-Related Deaths"

by Robert Levy and Rosalind Marimont Published in *Regulation*, Fall 1998

AMERICAN COUNCIL
ON SCIENCE AND HEALTH
1995 BROADWAY, 2ND FLOOR
NEW YORK, NY 10023-5860
TEL 212.362.7044 • FAX 212.362.4919
E-MAIL: acsh@acsh.org

Non-Profit Org.
U.S. Postage
Paid
Permit No. 9513
New York, NY