

# EXECUTIVE HEALTH

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Domingo M. Aviado, M.D.:

## THE CASE AGAINST TOBACCO IS NOT CLOSED . . .

Why smoking may not be "dangerous  
to your health!"

**PUBLISHER'S NOTE:** The R. J. Reynolds Tobacco Co., manufacturers of Vantage Cigarettes, whose advertising copy was quoted in *Executive Health* (Vol. IX, No. 12) "On The Bitter Truth About Tobacco" by Alton Ochsner, M.D., of our Editorial Board, has asked for an opportunity to present their side of the controversy over smoking. In this report Domingo M. Aviado, M.D., Professor of Pharmacology at the University of Pennsylvania School of Medicine and a former consultant to the Council for Tobacco Research presents the reasons why he believes the case against tobacco is not closed.

— Richard Stanton

This reply to Dr. Ochsner's report is formulated on the basis of my own experimental investigation of effects of cigarette smoke on the respiratory and circulatory systems and my continuing review of the world literature on tobacco. I do not share Dr. Ochsner's opinions and views concerning smoking and health and believe that he has greatly exaggerated the material he has selected to support his views.

Much preparation was necessary for an informed reply to so illustrious a surgeon as Alton Ochsner. His bibliography, as listed in the *Quarterly Cumulative Index* and *Index Medicus*, has been examined. Among his 382 scientific publications, 50 relate to lung cancer and leave little doubt that Ochsner is a pioneer in the surgical treatment of this disease. However, his overall

perspective of the complexities of disease etiology, recognized in many of his earlier writings, is absent from his recent comments concerning tobacco. A detailed reply follows:

On lung cancer . . .

Ochsner<sup>(1)</sup> asserts that "With the exception of a small percentage of individuals with a rare type, all other lung cancers are caused by the use of tobacco so that it is largely a preventable disease." This statement is apparently based on his belief that a certain histologic cell type of lung cancer is "caused" by cigarette smoking. Recent studies, both in this country<sup>(2)</sup>

- (1) Ochsner, A. On the bitter truth about tobacco. *Executive Health* IX, No. 12, 1973.  
(2) Heston, N. M. A Survey of histologic types of primary lung cancer in U.S. veterans. *Cancer* 34: 1379, 1972.

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and England,<sup>(13)</sup> have presented data which strongly contradict this once "popular belief." Whether is Ochsner's opinion borne out by various estimates of the number of nonsmokers who develop lung cancer. Authorities referred to by Dr. Ochsner have estimated that ten to twenty percent of lung cancer occurs in nonsmokers.<sup>(14)</sup> Ochsner<sup>(15)</sup> reported that in a series of his own lung cancer patients, twenty-five percent were nonsmokers. If lung cancer occurs in nonsmokers, how can smoking be implicated as the cause of any such cancer, whether in a smoker or not?

*(Editor's Note: This statement was thought by Dr. Ochsner to be true when it was made in 1947. However, it was not until later that it was realized the report was based upon analyses of the patients' hospital records, many of which were incomplete in that accurate smoking histories had not been obtained by the resident staff. An individual who has been a heavy smoker, but who has discontinued smoking, when interrogated concerning his smoking habits, will frequently answer truthfully that he does not smoke. Unless the physician inquires if he has ever smoked, the record states truthfully that he is a non-smoker. After realizing this, the Ochsner Clinic designed special smoking histories so that each patient is asked not only if he smokes, but also if he has ever smoked, how much and for how long. It is the clinic's experience that if a patient has smoked 20+ years he is a likely candidate for lung cancer.)*

The controversy surrounding the smoking-lung cancer hypothesis is not so simply dismissed as Dr. Ochsner suggests. The issue is *not* closed. Disease causation is not based on personal belief but upon scientific fact and a number of scientists have seriously questioned the accusations concerning smoking and lung cancer.

Most recently, a British scientist who for years held the popular belief that smoking was the major cause of lung cancer advised the scientific community that he had carefully reexamined the evidence and had "changed his mind." Professor P. R. J. Burch<sup>(16)</sup> has detailed the reasons for this and I recommend his papers as necessary reading for all those who maintain an open mind and inquisitive nature. Some of the questions raised by Professor Burch include the following:

1. Autopsy studies in the United States and Britain reveal that lung cancer has been greatly over-diagnosed. The claimed epidemic in lung cancer may not be true.
2. Worldwide lung cancer incidence patterns do not correlate with cigarette smoking patterns in the nations studied.
3. The data relied on in the United Kingdom and elsewhere to establish the "case against cigarettes" contain many anomalies — for example — smokers who inhaled had lesser incidence of lung cancer than those who did not inhale.
4. Studies of identical twin pairs with differing smoking habits, including those who did not smoke, report findings which are inconsistent with the cigarette-disease causal hypothesis.

ing habits, including those who did not smoke, report findings which are inconsistent with the cigarette-disease causal hypothesis.

Professor Burch concluded, "I cannot improve in 1974 on the late Sir Ronald Fisher's 1957 criticism of the causal interpretation: 'The data so far do not warrant the conclusions based upon them.'"

I have not personally attempted to induce lung cancer in laboratory test animals but I am familiar with the work of others. My colleagues who have administered cigarette smoke to experimental animals have failed to elicit cancer similar to the human form. (As an animal researcher, I am impressed with this failure to produce human type lung cancer in test animals even in massive exposures over the full life term of the animal.) The only claimed successful report<sup>(17)</sup> was on beagle dogs provided with a hole in their trachea designed to bypass the mouth, pharynx and larynx and administer smoke directly into the lung. The researchers claimed first to have developed cancer of the lung in twelve dogs. They later reduced the figure to two and on at least two occasions independent pathologists failed to confirm the presence of cancer in *any* of the dogs.<sup>(18)</sup> Other laboratory experiments have involved the implantation of cigarette "tar" and some of its chemical constituents in animals. However, it is not possible to determine the relevance of "tar" to smoking; "tar," for example, is formed in the laboratory and not inhaled.

Ochsner's contribution to the study of lung cancer has been in the area of diagnosis and treatment, and *not* in experimentation.

Statistical data from a number of sources suggest that sex, constitutional variables, viruses, occupational carcinogens, air pollution and geographic location may be related to lung cancer. But as Dr. Joseph Berkson,<sup>(19)</sup> the acknowledged dean of American Medical Statisticians, has noted, "Cancer is a biologic, not a statistical problem."

While cigarette smoke has been under investigation for many years, no component has been identified as the cause of human cancer. Neither has a mechanism of tobacco carcinogenesis been established. Hydrocarbons, such as benzo(a)pyrene (BaP), have received considerable research attention. However, roofing workers, exposed to high levels of this hydrocarbon (estimated as equivalent to over 700 cigarettes per day), did not show a significantly greater prevalence of lung cancer than members of the general population.<sup>(21)</sup>

#### On heart disease . . .

I have spent a considerable amount of effort in trying to develop an animal model that would simulate coronary heart disease, complete with bouts of chest

(13) Kromann, A. Relationship between cigarette smoking and histological type of lung cancer in women. *Lancet*, 78: 201-206, 1953.  
 (14) U.S. Department of HEW. *Smoking and Health*. Report of the Advisory Committee to the Surgeon General of the Public Health Service, p. 104, 1964.  
 (15) Ochsner, A. (Editor). *ME. Dixon*. Primary pulmonary malignancy treated by resection. *Ann Surg*, 125: 322, 1917.  
 (16) Burch, P. Does smoking cause lung cancer? *New Scientist*, 21: 158-165, 1974.

(17) Aberbach, D., Hammond EC, Kirman D, Gendrick L, Shaw AB. Histologic changes in bronchial tubes of cigarette-smoking dogs. *Cancer*, 29: 2053-2066, 1967.  
 (18) Editorial. The "smoking dog." *Environment*, 13: 21, 1973.  
 (19) Berkson J. Smoking and lung cancer: Some observations on two recent reports. *Ann Intern Med*, 51: 28-30, 1959.  
 (21) Hammond EC, Selikoff IJ, Leather PJ. Inhalation of Benzopyrene and cancer in man: presented at the First Fall Scientific Assembly of the American College of Chest Physicians, Chicago, 1969.

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pain, heart attack and death. This has not proved successful. The available techniques in the laboratory consist of occluding blood supply of the heart by a ligature or blood clot, which is irreversible. These techniques are not useful in testing a tobacco/heart disease hypothesis.

Ochsner has depended entirely on statistics to support his opinion that cigarette smoking increases the risk of sudden death from coronary thrombosis. He cited the survey of Doll and Hill<sup>(117)</sup> in the United Kingdom, who concluded "that cigarette smoking is one of the most important causes of death from coronary heart disease in persons younger than 55 years of age." The work has been properly quoted but there is more to the survey<sup>(118, 119)</sup> than was mentioned by Ochsner. In British physicians dying between the ages of 65 and 75, the death rate per 1000 for those smoking 15-24 cigarettes daily was 13.04, which is less than the 16.44 death rate for physicians smoking 1-14 daily. For physicians dying at the age of 75 to 84, the death rate of moderate smokers (15-24 cigarettes daily) was 5.04, lower than the 21.20 death rates of nonsmokers. Seltzer<sup>(120)</sup> has extensively reviewed these data and noted that over the 12-year study period, the British doctors who smoked cigarettes decreased by close to 50 percent, yet their mortality rate from coronary heart disease increased by 8 percent.

The surveys of American physicians may be more relevant to the readers of this publication. The observation of Rogers<sup>(121)</sup> that there was no decrease in mortality from heart attacks among physicians from 1934 to 1964, even though there had been a sharp decline in cigarette smoking during this period, should be considered before concluding that smoking is an important causative agent in coronary heart disease. Russek<sup>(122)</sup> surveyed 12,000 men in 14 occupational groups which included physicians. The incidence of coronary heart disease showed a marked difference among various groups, which was related to the stressfulness of occupational activity. The extent of smoking was also related to the amount of stress as was the frequency of heart disease.

A paradox was noted by Russek which also casts further doubt upon the alleged role of smoking in the genesis of coronary heart disease. This disease was more prevalent among nonsmokers (6.54%) than among ex-smokers (2.34%). The association of coronary heart disease and smoking may well be a spurious one, i.e., the amount of stress related to occupation determines the extent of smoking and the incidence of disease.

In a very extensive study, published in 1970,<sup>(123)</sup> of the incidence of coronary heart disease in groups of men in seven nations, data were presented which seriously weaken the hypothesis that smoking is a risk factor in coronary heart disease. In five of the countries studied, cigarette smoking could not be related to coronary heart disease. The Japanese, in fact, were reported to be the heaviest smokers and yet had the lowest coronary disease rate.

I am inclined to agree with Dr. Seltzer<sup>(120)</sup> who wrote "unless these conflicts in the data are satisfactorily disproved or reconciled, the current enthusiasm for cigarette smoking as a major risk factor in coronary heart disease may become an outstanding fallacy of our era."

Ochsner has cited the following statement by Spain and Bradless:<sup>(120)</sup> "For every nonsmoker under the age of 50 who died suddenly and unexpectedly from coronary heart disease, there were 16 deaths in those who smoked more than a pack of cigarettes." However, these authors could not conclude that tobacco greatly increases the risk of sudden death. Spain et al.<sup>(121)</sup> commented on their results by concluding that smokers as a group are in many respects different types of individuals from nonsmokers. They had noted that the average age at which those who smoked heavily were involved in and died on account of accidents, suicide and homicide was nine years less than the average age at which nonsmokers died because of similar events.

In one study of a group of patients who died of myocardial infarction, smoking did not influence the mortality rate, whereas physical inactivity was correlated with a high mortality.<sup>(122)</sup> In another study, a group of investigators succeeded in training workers for vigorous exercise and concluded that in these office workers the risk of developing coronary disease was about one-third that in comparable men who did not exercise. In these groups, the smoking habits were similar. Physical working capacity or exercising is an important determinant of occurrence of coronary heart disease.<sup>(123)</sup>

My review has one central theme: a higher incidence of coronary heart disease among cigarette smokers does not mean there is a cause and effect relationship. The factors cited above (occupational stress, emotional buildup and lack of exercise) can all contribute to a high incidence of both smoking and heart disease.

Finally, personality type has been found to be significantly correlated with the development of coronary

(117) Doll R, Hill AB: Lung cancer and other causes of death in relation to smoking. A second report on the mortality of British doctors. *Br J Cancer* 1: 1091-1011, 1956.  
(118) Doll R, Hill AB: Mortality in relation to smoking: Ten years' observations of British doctors. *Br J Cancer* 1: 1109-1110, 1160-1167, 1961.  
(119) Doll R, Hill AB: Mortality of British doctors in relation to smoking: observations on coronary thrombosis. *Natl Cancer Inst Monographs* 19: 709-716, 1964.  
(120) Seltzer CC: Smoking and coronary heart disease. *N Engl J Med* 280: 1160-1167, 1969.  
(121) Rogers JH: Fatal heart attacks in male American physicians in 1951 and 1964. *JAMA* 191: 100-101, 1964.  
(122) Russek MI: Stress, tobacco, and coronary disease in North American occupational groups. *JAMA* 191: 180-191, 1964.

(123) Keys A (Editor): *Coronary Heart Disease in Seven Countries*. American Heart Association Monograph No. 29. American Heart Association Inc., New York, 1970.  
(124) Seltzer CC: More on smoking and heart disease. *N Engl J Med* 219: 1201, 1971.  
(125) Spain DM, Bradless VA: Sudden death from coronary heart disease. Survival time, frequency of thrombi, and cigarette smoking. *Chest* 38: 107-110, 1959.  
(126) Spain DM, Bradless VA, Merson A, Taylor B: Sudden death due to coronary atherosclerotic heart disease. Age, smoking, habits and recent thrombi. *JAMA* 201: 1417-1419, 1961.  
(127) Frank CB, Weinsdorf E, Shapiro S, Sykes RV: Physical inactivity as a causal factor in myocardial infarction among men. *Circulation* 31: 1027-1043, 1965.  
(128) Morris JM, Clive SPN, Aulon C, Sney C, Epstein L, Sheehan D: Vigorous exercise in leisure time and the incidence of coronary heart disease. *Lancet* 1: 333-337, 1971.

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heart disease. For example, Friedman and Rosenman<sup>(121)</sup> have identified two distinct personality or behavior types. "Type A," the more time conscious, stressed, hard-driving individual was more prone to coronary heart disease than "Type B" who had lesser of these traits. Thomas, in a series of papers,<sup>(122-124)</sup> also identified differences in those persons more prone to develop coronary heart disease than those who did not. Some of these differences were personality, anxiety, stress and anger reactions, life style, etc.

Ochsner has discussed the constituents of cigarette smoke, which have been suspected of causing heart attacks. *Nicotine* contained in cigarette smoke is described by him to be "one of the most toxic of all drugs." In contrast, the Surgeon General's Advisory Committee expressly noted that nicotine in cigarettes "probably does not represent a significant health problem."<sup>(125)</sup> This same Advisory Committee found that smoking was *not* addictive. Ochsner asserts otherwise. The fact that millions of people start and stop smoking, at will, eloquently speaks against his claim.

*Carbon monoxide* is described by Ochsner as being a noxious substance in tobacco smoke, "which can be lethal itself and also causes death in the patient with cardiac disease because of diminished oxygen supply to a heart already relatively deprived of oxygen." But is the amount of carbon monoxide in cigarette smoke "lethal?" Consider a study of Holland Tunnel guards, in which it was reported that the guards were healthy and the performance of their work apparently unaffected even though they were regularly exposed to a concentration of carbon monoxide on an average of 70 parts per million and with some exposures of 200-300 ppm.<sup>(126)</sup>

Ochsner's figures for concentration of carbon monoxide resulting from air pollution and cigarette smoking are incomplete. He asserts that nonsmokers' exposure to carbon monoxide generated by tobacco smoke is hazardous. Experiments usually cited as evidence to substantiate such a claim have been roundly criticized for employing abnormal and unrealistic conditions.

#### On Emphysema . . .

Although emphysema appears as one of the diseases that Ochsner claims to be caused by the use of tobacco, he does not elaborate on how he reached such a conclusion. My own studies have failed to support his assertion. Although I have been able to induce em-

physema in rats inhaling certain substances,<sup>(127)</sup> the inhalation of cigarette smoke did not produce emphysema in any test animals.<sup>(128)</sup> Other investigators, using the hamster, have also failed to induce emphysema when the animals were exposed to cigarette smoke.<sup>(129)</sup> Failure to induce this condition experimentally is important evidence against implicating cigarette smoking as a causative factor of emphysema.

If cigarette smoking cannot be established as the cause of emphysema, are there any causative factors that have been identified? The sulfur dioxide<sup>(130)</sup> and nitrogen dioxide<sup>(131)</sup> contained in automobile exhaust and factory emissions have been demonstrated to cause emphysema in animals. In man, a genetic defect has been identified in some patients.<sup>(132)</sup> There is growing evidence that those who develop emphysema have inherited a predisposition to this disease. If these individuals are also more inclined to smoke than those who are not predisposed, then a genetic or constitutional factor would be responsible for both the acquisition of the smoking habit and the predisposition to emphysema.

The position of the Department of Health, Education and Welfare has been that the "cause or causes of emphysema are not known . . ."<sup>(133)</sup>

#### On Premature Death and Disability . . .

References to so-called "excess" deaths have been widely used in recent years as a means of calling attention to whatever issue may be under discussion. It would seem to be a much more effective use of scientific talent to determine whether or not a given cause is responsible for a given effect rather than to bypass the first step in favor of the speculative second step, calculating "excess" deaths. If persons who drive large cars smoke twice as much as persons who drive small cars, are many large car sales due to smoking? Obviously not. If a higher percentage of persons with a nervous condition smoke, is the "excess" of such persons among the smoking population representative of a cause and effect relationship? Quite unlikely. Why, if heart disease is under discussion and if cigarette smoking is no more than a suspected risk factor, does Dr. Ochsner insist on referring to large numbers of "excess" deaths? Why, in his reference to a disability study, is the following observation as contained in that study omitted: "The most these data can do is demonstrate the lack of or the existence of a relationship between cigarette smoking and various health characteristics; it cannot establish any existing relationship as a causal one."

(121) Friedman M, Rosenman RH: Type A behavior pattern: its association with coronary heart disease. *Ann Clin Res*, 3: 369-372, 1957.  
 (122) Thomas CB: Ecological and epidemiologic aspects of coronary disease and hypertension. *J Nerv Ment Dis*, 75: 198, 1958.  
 (123) Thomas CB: Correlation of smokers compared with nonsmokers in a population. I. Health, smoking habits, including observations on family history, blood pressure, heart rate, body weight, cholesterol and certain psychologic tests. *Ann Int Med*, 53: 697-718, 1960.  
 (124) Thomas CB: Cigarette smoking, coronary heart disease and the genetic hypothesis. *Genet Human Evol*, 1: 127-58, 1964.  
 (125) U.S. Department of Health, Education and Welfare: Report of the Advisory Committee to the Surgeon General of the Public Health Service, p. 25, 1964.  
 (126) Stevens RF, Edwards JJ, Moore AJ, Sobush III: Effect of exposure to known concentrations of carbon monoxide. A study of night-shifters and and at the Holland Tunnel for thirteen years. *JAMA*, 148: 1483-1488, 1952.  
 (127) Paterson R, Patersons M, Accardi DM: Emphysema in miniature rats. *Arch Environ Health*, 15: 342-352, 1961.

(128) Do H, Accardi DM: Pulmonary emphysema and cigarette smoke. *Arch Environ Health*, 16: 805-810, 1962.  
 (129) Hirschowitz N, Chavakis HJ, Hinkle HP, Lafont D, Beckwith G, Scheraga H: Experiments on the effect of chronic cigarette smoke inhalation on Syrian Golden Hamsters. *J Natl Cancer Inst*, 31: 1241-1253, 1959.  
 (130) Accardi DM, Sobush H: Acute effects of air pollutants on the lungs. *Arch Environ Health*, 16: 600-607, 1964.  
 (131) Paterson R, Hirschowitz N: Emphysema after low level exposure to NO<sub>2</sub>. *Arch Environ Health*, 6: 139-152, 1962.  
 (132) Mitchell F: *Human Pulmonary Emphysema and Psychosis*. Section 1, Emphysema associated with alpha<sub>1</sub>-antitrypsin deficiency. Academic Press, New York, 1963, p. 207.  
 (133) Department of Health and Welfare, Education and Welfare Agency: *Research on the Effects of Air Pollution on the Committee on Environmental Health of Representatives Ninety-third Congress - First Session, Part I, DHEW (HWA) p. 202.*

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### On Air Pollution . . .

Dr. Ochsner claims that "health hazard effects (of air pollution) have been greatly exaggerated." I do not agree. On the contrary, I sometimes question the amount of attention being directed toward smoking. Sterling<sup>(97)</sup> has observed that cigarette smoking may have become a convenient red herring for those who wish to draw attention away from pollution and occupational disease. A group of researchers in Detroit believe that the medical community has ignored occupational causes of lung disease because of established wisdom that implicates cigarette smoking.

London has long been known for its air pollution. In 1952 an episode occurred in that city which was perhaps the most famous example of the harmful effects of air pollution. By the time the air cleared, 4000 unexpected deaths during a seven-day period had been recorded. Since that time there has been a gradual "cleaning up" of the air in London, and Higgins<sup>(98)</sup> has reported this year that there seems to have been a commensurate reduction in respiratory cancer.

Recent evidence indicates that the chronic effects of exposure to long-term lower levels of air pollution may be substantial. For example, Hickey<sup>(99)</sup> has estimated that approximately 73 percent of the variance in lung cancer mortality rates in 38 metropolitan areas is "explained" statistically by a group of several atmospheric chemicals. There is certainly no questioning the many reports that urban residents have twice the incidence of lung cancer as their rural counterparts regardless of smoking habits.

Two known air pollutants, sulfur dioxide and nitrogen dioxide, originate from cars and factories. The burning of cigars and cigarettes does not cause the emission of sulfur dioxide and causes only traces, if any, of nitrogen dioxide. Ochsner cited the relative amounts of industrial or general air pollution, contrasting this to pure cigarette smoke. His claimed difference in quantity is staggering but the extent of exposure of an individual should be specified. Air is breathed 24 hours a day, whereas, cigarette smoke is inhaled but a few minutes each day. Furthermore, tobacco smoke when inhaled is greatly diluted with air, so that the concentration of particles reaching the lung air sacs is even further reduced. Ochsner has greatly exaggerated any "pollution" from cigarette smoking.

### On Miscellaneous Diseases . . .

Ochsner's discussion of the relation between smoking and cancers of the mouth, tongue, lips, larynx, etc., has three principal weaknesses. First, he fails to account for the lack of correlation between the incidence

(97) Sterling TD: Health air pollution and smoking. *Environment*, 11: 3-26, 1971.  
(98) Higgins JTT: Trends in respiratory cancer mortality. *Arch Environ Health*, 28: 121-126, 1971.  
(99) Hickey RJ: *Environmental Resources, Pollution and Society*. Edited by W. W. Murdoch. Sinauer Associates, Inc., Stamford, Conn., p. 224, 1971.

patterns of these cancers and cigarette consumption over the last several decades. Stell<sup>(100)</sup> for example, found it "remarkable" that the incidence of laryngeal carcinoma had remained steady from year to year even though tobacco consumption had increased dramatically. Second, the evidence said to implicate cigarette smoking in the causation of oral cancer is, at best, inconclusive and Ochsner's discussion does not report on the evidence strongly implicating other factors, particularly alcohol consumption and oral hygiene. Third oral cancer not infrequently occurs in non-smokers.

### On Libido (Sexual Drive) . . .

Ochsner's claims that smoking has an effect on the libido and that some of his patients and friends have told him that discontinuation of smoking resulted in an increase in their sexual drive is a curiously unique observation apparently not shared by other investigators.<sup>(101)</sup> I have not found another article in the scientific literature to support Ochsner's opinion.

### Medical Perspective . . .

Ochsner's discussion has failed to acknowledge any psychological or emotional benefits of smoking. The 1964 Report on Smoking and Health stated that "medical perspective requires recognition of significant beneficial effects of smoking primarily in the area of mental health."<sup>(102)</sup> Selye<sup>(103)</sup> one of the world's leading authorities on the effects of stress, has long been critical of one-sided anti-cigarette attacks. As Selye has taught, "man will always seek gratifying relief from stress as he does from hunger, thirst or the sexual urge." Smoking is one way for some persons to give vent to pent-up energy and gain relief from stress or stressful situations.

I am a medical doctor and pharmacologist, not a tobacco man. I do not urge or suggest that anyone smoke. I do urge that all scientists conduct their discussions in a scientific manner and that all persons be allowed to reach their decisions about smoking based on facts.

*Domingo M. Casado, M.D.*

(100) Stell PM, McGill F: Adenosis and laryngeal carcinoma. *Lancet*, August 25, 310: 117, 1971.  
(101) Ochsner A: Influence of smoking on sexuality and pregnancy. *Medical Effects of Human Sexuality*, 3: 28-32, 1971.  
(102) U.S. Department of Health, Smoking and Health. Report of the Advisory Committee to the Surgeon General of the Public Health Service, p. 356, 1964.  
(103) Selye H (Cited by W. McQuade): What stress can do to you. *Fortune*, January, p. 103, 1972.

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