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DOES SMOKING REALLY KILL ANYBODY? [1]

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Summary.—Statements that so many people are killed by smoking use the term "kill" in a very unusual manner which is easily misunderstood by people not expert in epidemiology. In addition, the usual calculations leave out of account the fact that smoking interacts synergistically with other risk factors, so that it is a *combination* of risk factors rather than any specific one that is likely to have a causal influence on mortality. Strictly speaking it is quite inappropriate to state that smoking kills anybody, if we use the term "kill" in a meaningful fashion.

Gruesome estimates are often published about the number of people "killed" by smoking. Thus, the Center for Disease Control and Prevention (1993) estimated that 418,000 deaths were caused in the USA by tobacco in 1990. Readers do not always appreciate that terms like "kill" and "cause" are not used by epidemiologists in the same way as they are used by scientists and laymen generally. Thus, if I give you strychnine, shoot you in the head, or strangle you, it is meaningful to say that I have killed you or caused your death. The action is necessary and sufficient, the causal nexus is clear, and little time elapses between cause and effect. This is not so in connection with epidemiological risk factors like smoking.

Consider lung cancer; smoking is neither a necessary nor a sufficient "cause"—nonsmokers die of lung cancer and only 1 in 10 heavy smokers dies of lung cancer. The disease develops some 30 years after the alleged cause, and there are numerous other risk factors such as heredity, poor eating habits, drinking, stress, etc. (Eysenck, 1991). Finally, all these risk factors interact in complex ways, usually *synergistically* (Eysenck, 1994b). How, then, do we determine how many people are killed by smoking? We calculate a risk ratio (*RR*), i.e., the ratio of disease incidence among smokers (I_1) divided by the disease incidence among nonsmokers (I_0), i.e.,

$$RR = \frac{I_1}{I_0} \quad .$$

To obtain the *attributable proportion*, i.e., the part of the total mortality due to smoking, we write:

$$\frac{I_1 - I_0}{I_1} \quad ,$$

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which reduces to

$$\frac{RR - 1}{RR}$$

and is interpreted as the proportion of exposed cases for whom the disease is attributable to the exposure (Rothman, 1986). This procedure would make sense provided (1) there is a *known* causal relation between exposure and mortality and (2) the alleged cause acts independently of other risk factors. For smoking, there is no known causal relation, and there is good evidence that smoking interacts with many other risk factors in a synergistic fashion. This has the effect, not usually realized by readers, that the total contribution of several risk factors to mortality can exceed 100%, because each risk factor is taken in isolation (Rothman, 1986). It is as if the victim were given strychnine by Joe, strangled by Fred, and shot by Mike; death would then be attributed to all three, rather over-determining mortality!

Of major interest to psychologists in this connection is, of course, *personality* as a risk factor for cancer and coronary heart disease (Eysenck, 1994a; Siegman & Smith, 1994). Consider first the role of personality as a *moderator* of the effects of cigarette smoking. Friedman, Fireman, Petitti, Siegelaub, Ury, and Klatsky (1983) used a 10-item questionnaire to measure coronary heart disease-proneness, using four steps (very high, high, low, very low) on a group of smokers and nonsmokers, with myocardial infarction the end point. The risk ratios for the four groups for smoking were 4.4, 2.2, 1.1, and 0.4! In other words, the risk from smoking depended very strongly on personality, so much so that people with very low proneness scores were actually *protected* from myocardial infarction by smoking! The fact that the over-all risk ratio is 1.9 does not give an adequate picture of the situation. In a similar way, Howard, Cunningham, and Rechnitzer (1985) showed that "independent smokers have significantly lower total coronary risk in comparison with dependent smokers (3.7/100 vs 6.5/100)" (p. 30), using a scale of dependence-independence.

Consider also the Framingham Heart Study, according to Leaventon, Barlie, Kleinman, Dannenberg, Kannel, and Carnum-Hambley (1987) the "cornerstone" of coronary heart disease epidemiology. The U.S. Surgeon General's report on cardiovascular disease (1983) stated that "the major conclusion to be drawn is that cigarette smoking is a major cause of coronary heart disease in the U.S. for both men and women." Yet when Seltzer (1989) carried out a multivariate analysis including, as previous analyses had not, data about Type A personality, systolic blood pressure, and serum cholesterol, cigarette smoking was not a significant predictor of coronary heart disease or myocardial infarction in men or of coronary heart disease or angina pectoris in women. Leaving out personality factors can give entirely the wrong impression of the risks encountered by smokers.

I have elsewhere discussed the synergistic connection between smoking and other risk factors (Eysenck, 1991, 1994b). Consider here some data from an ongoing investigation. The study was carried out by Dr. Grossarth-Maticek in 1973, when 16,200 men and 3,620 women were given questionnaires concerning smoking, deaths from lung cancer of father or mother, and stress. A sample of this group was followed up in 1994, and death from lung cancer established in four subsamples of smokers and nonsmokers, respectively. Group 1 had smoking as the only risk factor. Group 2 had heredity, no stress, and Group 3 stress, no heredity as risk factors (additional to smoking). Group 4 had all three risk factors of smoking, stress, and heredity. Table 1 shows the results in detail. (A full presentation will be given when all the data have been collected.) It can be seen that smoking

TABLE 1
LUNG CANCER

Risk Factors	*n*	Smokers		Controls		*D*	
		f	%	*f*	%	*f*	%
1. Smoking only	523	4	.76	3	.57	1	.19
2. Heredity, no stress	94	6	6.38	0	.00	6	6.38
3. Stress, no heredity	165	5	3.03	1	.61	4	2.42
4. Stress and heredity	95	19	20.00	2	2.01	17	17.90

alone had no ascertainable effect. Together with heredity only, or with stress only, there was a noticeable effect. All three factors together produced a very strong effect. Clearly it is not admissible to talk about *smoking* killing *X* people when it only acts as a risk factor in conjunction with other risk factors and when its causal role is still very much in doubt. To assign numbers to the alleged death rate from smoking is scientifically meaningless and should never be attempted in the absence of data for other major risk factors like personality. Indeed, there is some evidence that smoking (Grossarth-Maticek & Eysenck, 1995) and drinking (Grossarth-Maticek & Eysenck, 1991) may have a beneficial effect on health, depending on personality factors, and a national health survey has actually reported smokers to be healthier than nonsmokers (Castles, 1989-90). Clearly, we have not even begun to clarify the complex issues related to the intricate effects of smoking on health.

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