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COCA-COLA, CANCERS, AND CORONARIES: PERSONALITY AND STRESS AS MEDIATING FACTORS[1]

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Summary.—A theory is presented relating the consumption of stimulant and depressant drugs to cancer and coronary heart disease, with stress/personality acting as an intermediary. The predictions from the theory that large-scale consumption of Coca-Cola would *prevent* cancer and *promote* coronary heart disease was tested and found to be supported by the results of a long-scale prospective study. Results replicate those from an earlier study using *coffee* as a stimulant drug.

There is evidence to suggest that "CNS drugs may effect a new balance resulting in tumour suppression" when animals are injected with certain stimulant drugs (Metzler & Nitsch, 1986, p. 259; Metzler, 1979; Driscoll, Melnick, & Quinn, 1978, Eicke, 1975; Jones, 1985). Metzler and Nitsch (1986) explain their findings on the hypothesis that CNS drugs act on neurotransmitters and on second messengers, and that these, stimulated by CNS drugs, would be in a position to aid the repair mechanism of DNA whose damage was responsible for carcinogenesis (Gebhart, 1977). Neurotransmitters and second messengers are conceived as possible sources or stimuli for the energy required by the cancer cell. Metzler and Nitsch (1986) conclude that "CNS drugs could be used for the treatment of carcinoma patients . . ." (p. 275).

It seemed to Grossarth-Maticek and Eysenck (1991) that the general theory could be tested by making the deduction that stimulant drugs would have a prophylactic function with respect to cancer and that healthy people who drank a great deal of coffee would to some extent be protected from cancer, while users of Diazepam would show the opposite effect. Our original study (Grossarth-Maticek & Eysenck, 1991) was mainly concerned with habitual coffee drinkers consuming an average of 10 cups of coffee per diem. Of 140 heavy drinkers of coffee, 4% died of cancer, 12% of coronary heart disease (CHD). In a matched control group, 16% died of cancer and 6% of CHD, a significant difference in the predicted direction ($p < .001$). We also followed up takers of Diazepam, predicted to have effects opposite to those of coffee. Of 135 Diazepam takers, 28% died of cancer, 1% of CHD, suggesting that our prediction was correct. Imipramine ($N = 139$) acted like coffee; 1% died of cancer, 12% of Imipramine takers died of CHD. All differences are statistically significant. (These figures refer to an 8-year

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follow-up; those for a 13-year follow-up are similar. See the quoted paper for details.) It should be noted that coronary heart disease reacted in the opposite way to cancer; drugs which *prevented* cancer made CHD more likely, suggesting that depressant drugs could prevent CHD, even though they might make cancer more likely.

Along rather different lines, Lynn and Hampson (1975) intercorrelated demographic variables including coffee consumption and CHD over a series of 17 nations and reported that a factor analysis of the demographic variables over countries disclosed two dimensions which they identified as extraversion and neuroticism. Coffee consumption and CHD incidence are close together in the extraverted half of their Fig. 1. These findings, as far as they go, support our theoretical considerations. The theoretical analysis gives a much needed support to epidemiological findings relating disease and demographic variables.

The Coca-Cola Study

Data concerning cigarette consumption, consumption of alcohol and the amount of Coca-Cola consumed were collected in 1973 from 3,684 men and 3,018 women. Data were also collected by interviewers administering personality inventories of the personality and stress experienced by the probands, using the scales presented in full elsewhere (Grossarth-Maticek & Eysenck, 1990). Of these subjects, 203 men and 191 women were found to drink between 2 and 3 litres of Coca-Cola daily for 10 years or more. A control group was formed of 135 men and 159 women who were matched with the Coca-Cola group on age, sex, cigarette and alcohol consumption, as well as on personality type. Another 301 men and 304 women consumed between 1 and 2 litres of Coca-Cola, and a control group was formed of 245 men and 251 women, matched with the Coca-Cola group on the same variables as before. The mean age of all these groups was around 50 years at time of test.

The matching for personality and stress was accomplished by reference to a questionnaire which had been personally administered by interviewers to all the subjects. The questionnaire has been described in detail elsewhere (Grossarth-Maticek & Eysenck, 1990). Scores are used to assign subjects to one of six types (cancer-prone, CHD-prone, alternating personality reaction, healthy autonomous behaviour, rational-antiemotional, and psychopathic) on the basis of the subjects' responses. By making sure that there were no significant differences between groups we attempted to reduce the probability that other factors than drinking Coca-Cola would be responsible for any differences in mortality observed.

All the groups were followed, and mortality and cause of death established in 1986, i.e., after 13 years. Mortality and cause of death were ascertained on the basis of detailed follow-up of all the probands, and by

consulting death certificates. Fortunately there is little movement in a small German town like Heidelberg, so it was possible to follow-up all probands. To discover their status, homes were visited in each case to ascertain whether the proband was alive or dead.

The following predictions were tested: (1) Mortality from coronary heart disease (CHD), i.e., coronary infarct, stroke, and sudden death from circulatory failure, is expected to be higher among Coca-Cola drinkers than among controls. (2) Mortality from cancer is expected to be reduced among Coca-Cola drinkers as compared with controls. (3) There is a dose-response relation between amount of Coca-Cola consumed and mortality from CHD (direct) and cancer (inverse). (The questionnaire used to establish cigarette, alcohol, and Coca-Cola consumption is available from the second author.)

Results

The main findings are given in Table 1. On analysis of variance there are no significant differences between the control groups. For cancer, there is a significant difference ($p < .001$) between Coca-Cola drinkers and nondrinkers, with drinkers over-all having only about one-fourth of the mortality of nondrinkers. This clearly validates our first prediction. For CHD, there is an equally marked difference between Coca-Cola drinkers and nondrinkers, the drinkers having a much higher mortality than the nondrinkers ($p < .001$) by a factor of a one-fourth death rate among nondrinkers. Separate analysis of death from infarction/stroke and from circulatory failure shows both as significant, with the latter significantly more ($p < .01$). This validates our second prediction.

TABLE 1

Mortality Rates (Percent) From Cancer, Coronary Heart Disease, and Other Causes For Drinkers and Matched Nondrinkers of Coca-Cola

Groups	Death From		
	Cancer	CHD	Other Causes
1. Control Group, 1—2 litres	3.9	4.8	3.7
2. Control Group, 2—3 litres	5.3	4.5	5.3
3. Coca-Cola Group, 1—2 litres	1.0	16.2	6.4
4. Coca-Cola Group, 2—3 litres	1.0	28.0	14.9

For other causes of death, there is also a much higher mortality for Coca-Cola drinkers, their mortality being over twice as high as that of controls. In view of the well-known unreliability of death-certificate diagnoses, it is difficult to comment on this finding (Eysenck, 1991).

Is there a dose-response relationship? The data in Table 1 suggest no such relationship for cancer mortality, and the comparison did not reach the 5% confidence level. For CHD, there is no doubt about the significance of the difference between 1—2 and 2—3 litres drunk daily, with the latter giv-

ing rise to much higher mortality. This holds for both infarct/stroke and circulatory failure, but particularly the latter. Thus our third prediction is falsified for cancer but supported for CHD. For deaths from other causes, the dose-response relationship is also verified at high confidence ($p < .001$).

We may look at sex differences. Over-all mortality is higher for men as far as cancer (3.24% vs 2.14%) and infarct/stroke (5.80% vs 2.77%) are concerned; for circulatory failure there is no difference (8.77% vs 8.43%). How about the control and Coca-Cola groups? The *differences* between drinkers and nondrinkers are preserved faithfully for cancer, as shown in Table 2; so are the differences for coronary heart disease. For deaths from other causes, too, the differences between drinkers and nondrinkers are preserved across the sexes. We also see that there is no dose-response relationship for cancer, for men or women, but a very marked one for coronary heart disease and other causes.

TABLE 2
MORTALITY RATES (PERCENT) FROM CANCER, CORONARY HEART DISEASE, AND OTHER CAUSES FOR MALE AND FEMALE DRINKERS AND MATCHED NONDRINKERS OF COCA-COLA

Groups	Death From					
	Cancer		CHD		Other Causes	
	Men	Women	Men	Women	Men	Women
1. Control: 1—2 litres	4.6	3.3	6.3	3.3	4.2	3.3
2. Control: 2—3 litres	6.2	3.9	8.0	3.3	6.2	4.6
3. Coca-Cola: 1—2 litres	1.3	0.8	17.2	15.2	6.7	6.2
4. Coca-Cola: 2—3 litres	1.5	0.7	31.1	25.3	15.6	14.3

DISCUSSION

The results for the most part bear out the predictions and largely replicate the outcomes of our previous study (Grossarth-Maticek & Eysenck, 1991). Stimulant drugs *reduce* cancer risk and increase risk of coronary heart disease with humans, just as they have been found to do with animals (Metzler, 1979; Metzler & Nitsch, 1986). There is evidence of a dose-response relation as far as coronary heart disease and other causes of death are concerned but none for cancer risk. Sex appears to make no difference as far as these relations are concerned, although the mortality of women is lower over-all than that of men.

Theory suggesting a *causal* relation between drug consumption and mortality is supported by these results, but it should not be assumed that the theorizing is not subject to possible criticism. As with most epidemiological studies, there is a possibility that consumers and nonconsumers of Coca-Cola, as of coffee, differ in other ways which may be causally related to cancer and coronary heart disease, so that the consumption of stimulant drugs is merely a correlate of both mortality and whatever other factor may be causally related to disease. This is an ever-present problem in epidemiology, but

the evidence from strictly *experimental* animal work may reduce its impact in our case. We have only attempted to control for both psychosocial and medical factors by matching, but of course we may not have controlled for other possibly relevant factors, such as occupation, education, etc.

Another possible criticism relates to the quantity of Coca-Cola consumed by our groups; what is true of the people involved may not be true of less extreme consumers. The presence of a dose-response curve for coronary heart disease and death from other causes may reduce the weight of this criticism, but the absence of such a dose-response relationship for cancer leaves this part of our study open to objection.

It would be quite wrong to summarize our study by saying that "Coca-Cola causes coronary heart disease" or that it protects from cancer. The most that can be said is that in excess, Coca-Cola is a risk factor for coronary heart disease, although possibly only a statistical one; the absence of direct evidence for causality must make us careful not to over-interpret our findings. Similarly, we may say that Coca-Cola seems to act as a negative risk factor for cancer, but again we must be careful not to give this conclusion a *causal* interpretation. Epidemiological data are often over-interpreted in a causal direction, as in the case of smoking (Eysenck, 1991); it is important to resist such temptation. We are here concerned only with the testing of a scientific hypothesis linking drug consumption with disease through the mediums of personality and stress.

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