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Coffee-Drinking and Personality as Factors in the Genesis of Cancer and Coronary Heart Disease

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Abstract. A study of the prediction of cancer and coronary heart disease on the basis of coffee consumption and personality shows that in cancer-prone probands (diagnosed on the basis of a personality inventory) coffee consumption was related to *low* incidence of cancer and *high* incidence of coronary heart disease, while diazepam showed the opposite trend. In coronary heart disease-prone probands coffee drinking was also linked with *low* incidence of cancer and *high* incidence of coronary heart disease, with diazepam again showing the opposite trend. In a personality type not prone to either disease, neither coffee consumption nor diazepam was linked with death from cancer or coronary heart disease. It is suggested that personality interacts in a predictable way with coffee and diazepam to determine in part the likelihood of death from cancer or coronary heart disease. Imipramine acts in a fashion similar to coffee, and opposite to diazepam.

Introduction

There is a large literature on the question of whether coffee drinking has an influence on disease [Macmahon and Sugimura, 1984; James and Shirling, 1983; Horowitz, 1981; Rell, 1980]. In particular, the relationship between coffee drinking and cancer, on the one hand, and coronary heart disease (CHD), on the other, has been investigated in considerable detail [Le Grady et al., 1987]. Such studies are of course beset by a number of problems and difficulties, such as the control of cigarette smoking in evaluating the association of coffee drinking and disease [Morrison, 1984]. There is quite a strong relationship between cigarette smoking and caffeine use, for instance [Istvan and Matarazzo, 1984] and the ascertainment and control of this relationship in any particular study present considerable difficulties, as pointed out by Morrison in his critique of a study by Denoix et al. [1958], who attempted to show that coffee drinkers had 1.5 times the chance of developing lung cancer than did non-coffee drinkers, or coffee drinkers drinking less than 10 cups per day.

Accepting this criticism, there seems to be little evidence for a positive relationship between coffee drinking and cancer. Stocks [1970], looking at cancer mortality in relation to national consumption of coffee, found a negative relation. Even cancer of the bladder, which has sometimes been linked with coffee drinking, does not seem to be so related when tobacco smoking is controlled [Carlos et al., 1985]. A very recent study by Le Grady et al. [1987] found an increased mortality from non-coronary causes among non-coffee drinkers, this increased mortality mainly coming from cancer. Similarly, the Lutheran Brotherhood Study [Murray et al., 1981] also found an increased risk of death from non-coronary causes in those consuming little or no coffee. It is thus possible that coffee may even play a prophylactic role as far as cancer is concerned, in the sense that drinking a good deal of coffee may avoid or postpone the onset of cancer. Caffeine of course has a stimulant action, although Mehrabian [1986], in a rather weak study, has suggested that *chronic* effects may be arousal-reducing.

A much more likely relationship is that between heavy coffee drinking and coronary heart disease [Heyden et al., 1978]. Earlier studies were somewhat contradictory [Horowitz, 1981], with a positive association between coffee intake and acute non-fatal myocardial infarction having been found in some case-controlled studies [Jicks et al., 1973], but no prospective investigation had found a significant positive association between coffee intake and any manifestation of CHD that persisted after adjustments for cigarette smoking [La Croix et al., 1986; Klatsky et al., 1973; Murray et al., 1981; Dawker et al., 1974]. It is only quite recently that the work of Le Grady et al. [1987] and La Croix et al. [1986] has definitely established increased mortality from CHD among probands drinking 6 or more cups of coffee.

One possibility investigated in this study is that stress and personality may be important factors, in interaction with other risk factors, in mediating cancer and CHD. There is some evidence for this suggestion in the literature [Eysenck, 1985], and three prospective studies have shown that distinct personality types, reacting differentially to stress, are associated with death from cancer or CHD respectively 10 years after ascertainment of personality type [Eysenck, 1987a, b, 1988; Grossarth-Maticek et al., 1983, 1988a, b]. Essentially, there are 4 personality types, of which type 1 is cancer-prone, type 2 CHD-prone; while types 3 and 4 are relatively healthy. The questionnaires used in these studies are given in detail in the paper by Grossarth-Maticek et al. [1988a].

The cancer-prone personality, as hypothesized in the inventory, portrays the usual traits found in relation to cancer: patterns of behaviour are over-cooperative, appeasing, unassertive, over-patient, unexpressive of negative emotions, avoidance of conflicts, compliant with external authority, and defensive in response to stress [Baltrusch et al., 1988]. Particularly important are the suppression of emotional responses [Kissen and Eysenck, 1962] and the failure to find adequate coping mechanisms for interpersonal stress [Eysenck, 1985].

As regards the CHD-prone type, this is also characterized by failure to cope adequately with interpersonal stress, but the major differentiation from the cancer-prone type lies in the development of strong anger, aggression and hostility responses [Booth-Kewley and Friedman, 1987; Chesney and Rosenman, 1985; Friedman and Booth-Kewley, 1987; Krantz and Manuch, 1984]. Type 3 oscillates between inadequacy and anger/aggression, and seems to be protected from disease through this alternation. Type 4 is a mentally healthy, autonomous sort of person who can cope adequately with stress. More detailed descriptions are given in the sources cited above.

The major question we set out to investigate was formulated to test consequences for human cancer development of some findings by Metzler [1979] and Metzler and Nitsch [1986], to the effect that stimulated drugs prolonged survival times in rats with the 3-MC sarcoma. Thus piracetam combined with cyclophosphamide increased remission rate 6-fold. Prophylactic effects could also be observed. This experiment suggested the possibility that drugs like caffeine might have a prophylactic effect in probands who were cancer-prone according to their personality type. The possible effects of other drugs were also investigated, particularly that of diazepam (Valium), which as a depressant drug should show effects opposite to those of caffeine. Choice of drugs was largely determined by availability of probands taking just one of the drugs included in our study, but none of the others; this was a principle of selection additional to be theoretical one of testing possible generalization of the Metzler and Nitsch research to humans.

The Empirical Study

The subjects of this study were chosen from participants of an 8-year follow-up study begun in 1973, with the results given below obtained in 1981. This 8-year follow-up was supplemented by a second follow-up extending the data base to 13 years. These results constitute our major source of conclusions. The sample was selected on a basis similar to that of the previous Heidelberg samples [Grossarth-Maticek et al., 1988a], i.e. on a semi-random design, with age limit and sex composition determined beforehand; there were 16,250 male and 3,620 female probands in this sample. Individuals were assigned to personality type on the basis of interviewer-administered questionnaires. They were asked about intake of coffee, which constitutes the major point of this study (with particular interest in those consuming 10 cups of coffee or more per day), but in order to have a variety of control groups, and obtain data on other drugs, they were also asked about whether or not they had taken diazepam or imipramine. The definition of a proband taking diazepam was a daily consumption for at least 10 years of over 10 mg of diazepam. For imipramine a dose of 75–150 mg as a maintenance dose was required, over a similar period of time. These additional data are available for type 1 subjects. For type 2 subjects we only have groups taking diazepam, or drinking more than 10 cups of coffee per day; the same data are available for type 3. The number of subjects in each group was 150 for type 1, 122 for types 2 and 3. Subjects were sub-divided into male and female groups, and also into three age groups. For each of the substances analysed in this study, care was taken to ensure that *none* of the other substances was also taken; thus effects are for one single substance only in each comparison. Even in such a large sample, it was sometimes difficult to find sufficient probands for each group.

We have relied on subjective accounts of coffee-drinking, although of course size of cup, type of coffee, and other factors including memory falsification may render this somewhat unreliable. It still seems likely that there is *on average* a difference between groups claiming to have consumed more than 10 cups a day, as compared with those claiming to have consumed less than 10 cups a day.

Table 1. Death from various causes associated with use of coffee, diazepam, or imipramine: 8-year follow-up in type I (cancer-prone) subjects

Substance used	Age range	M	F	Cause of death					
				cancer		CHD		other	
				M	F	M	F	M	F
10 or more cups of coffee per day (n = 150)	32–43	25	25	0	0	2	2	2	2
	44–55	25	25	2	1	3	2	3	3
	56–66	25	25	1	1	6	2	5	4
Diazepam (n = 150)	32–43	25	24	5	3	0	0	1	0
	44–55	26	25	8	5	0	0	0	1
	56–66	24	26	9	8	1	0	1	1
Imipramine (n = 150)	32–43	24	24	0	0	2	2	1	2
	44–55	20	25	0	0	2	2	3	4
	56–66	25	26	1	0	5	3	5	3
Control group (n = 150)	32–43	25	25	2	3	2	2	1	1
	44–55	25	24	4	4	2	1	0	1
	56–66	26	25	4	5	2	1	2	3
Autonomy training (n = 150)	32–43	25	24	1	0	1	0	1	1
	44–55	25	24	1	1	0	1	2	1
	56–66	26	26	1	2	0	1	1	1

In addition to the coffee, diazepam and imipramine groups which of course are self-selected, we also included two further groups of type I subjects. The first of these acted as a control group similar to the drug groups in age and sex composition, but not taking any of the drugs; those members drinking coffee did not do so in excess, i.e. they drank considerably less than 10 cups per day. A fifth group was selected along the same lines as the control group, but was offered a short 1-hour kind of behaviour therapy [Grossarth-Maticek and Eysenck, in press] which also had the added feature of giving the proband a leaflet to read outlining the aims and methods of the therapy (mainly a change in behaviour from that characteristic of type I to that characteristic of type 4). They were then visited two or three times, and interrogated concerning their understanding of the leaflet, and asked to provide examples from their own experience to illustrate the teachings of the leaflet. Each session lasted approximately 1 h. Smoking for all groups was controlled by only accepting for the study probands smoking between 20 and 40 cigarettes a day. The refusal rate for the therapy group (autonomy training group) was 40%; invitations were issued until the required number of 150 participants was reached.

Results

Table 1 shows the main results for whole groups, i.e. subdivided by age and sex; these two factors do not show statistically significant effects on death rates and are therefore not analysed further. Table 2 shows the combined results. χ^2 and other statistics were corrected, with the original data brought up to a standard (maximum) sample size of 145. The overall significance is beyond 0.001, hence individual comparisons between groups are permissible. These are given in table 3, for cancer and CHD deaths only. Changes in these values are minimal when death from other causes is included. Compared

Table 2. Data from table 1 summed over age and sex groups

	Total	Died of						Could not be traced
		cancer		CHD		other causes		
	n	n	%	n	%	n	%	
Heavy coffee drinkers	140	5	4	17	12	19	14	10
Diazepam takers	135	38	28	1	1	4	3	15
Imipramine users	139	1	1	16	12	18	13	11
Control group	143	23	16	8	6	8	6	7
Autonomy training	145	6	4	3	2	7	5	5

Table 3. χ^2 significance, phi and Goodman-Kruskal values for pairwise comparison of independent variables

Comparison groups	χ^2 p	Phi	Goodman-Kruskal gamma
Coffee-diazepam	0.001	−0.80	−0.99
Coffee-imipramine	0.146	0.23	0.65
Coffee-control	0.001	−0.52	−0.82
Coffee-autonomy	0.016	−0.43	−0.76
Diazepam-imipramine	0.001	0.92	1.00
Diazepam-control	0.003	0.35	0.87
Diazepam-autonomy	0.002	0.44	0.91
Imipramine-control	0.001	−0.66	−0.96
Imipramine-autonomy	0.001	−0.66	−0.94
Control-autonomy	0.656	0.07	0.18

Table 4. Results of coffee and diazepam effects for probands of type 2 and 3

	Total n	Died of			Could not be traced
		cancer	CHD	other causes	
Type 2					
Heavy coffee drinkers	121	1	10	20	1
Diazepam takers	121	9	1	17	1
Type 3					
Heavy coffee drinkers	120	0	1	6	2
Diazepam takers	122	1	1	5	2

Table 5. Data from table 4 summed over age and sex groups

Substance used	Age range	M	F	Cause of death					
				cancer		CHD		other	
				M	F	M	F	M	F
CHD-prone (type 2)									
10 or more cups of coffee per day (n = 122)	32–43	20	20	0	0	1	0	1	0
	44–55	21	21	1	0	3	2	3	3
	56–66	20	20	0	0	2	2	6	7
Diazepam (n = 122)	32–43	20	20	1	1	0	0	1	0
	44–55	20	20	2	1	0	0	4	1
	56–66	21	21	3	2	1	0	5	6
Mixed type (type 3)									
Ten or more cups of coffee per day (n = 120)	32–43	20	20	0	0	0	0	0	0
	44–55	20	20	0	0	0	0	0	0
	56–66	20	20	0	0	1	0	3	2
Diazepam (n = 122)	32–43	20	20	0	0	0	0	0	0
	44–55	21	20	1	0	0	0	0	0
	56–66	20	21	0	0	1	0	2	2

with the control group, Valium is associated with death from cancer, but acts as a prophylactic as far as CHD is concerned. Coffee and imipramine have the opposite effect. Autonomy training, like no drug taking, does not favour either cancer or CHD, but protects from both. Those prophylactic effects of autonomy training for cancer and CHD agree with previous research [Eysenck, 1987a, b; Grossarth-Maticek et al., 1988a].

Table 6. Data for 13-year follow-up, summed over age and sex groups

	Total n	Died of			Could not be traced
		cancer	CHD	other causes	
Heavy coffee drinkers	134	17	27	29	16
Diazepam takers	130	52	7	18	20
Imipramine users	120	30	38	45	30
Control group	140	35	18	17	10
Autonomous training	140	9	5	14	10

It should of course be noted that in table 3 the respective effects of any two treatments, as far as cancer and CHD are concerned, are compared, not their *absolute* effects. Thus the control and autonomy training groups differ profoundly as regards overall death rates, but the relative effects on death from cancer and CHD are not significantly different. Autonomy training has a strong prophylactic effect, but this is not different for cancer and CHD.

Table 4 shows the results for types 2 and 3, i.e. the coronary heart disease-prone type and the mixed (healthy) type, and their reactions to 10 or more cups of coffee per day and diazepam respectively.

Table 5 shows the main results eliminating insignificant sex and age differences. One person in each of the two groups of heavy coffee drinkers and diazepam takers could not be traced. It will be seen that again heavy coffee drinking appears to be a prophylactic against cancer, diazepam is a prophylactic against CHD. χ^2 is significant for this comparison.

Type 3, being a relatively healthy type as far as personality predisposition to disease is concerned, only has 1 death from cancer and 2 deaths from CHD, as well as 11 deaths from other causes. (One person in the coffee drinking group and 2 persons in the diazepam taking group could not be traced.) For this group, there is no difference for coffee drinkers and diazepam takers.

The main results for the 13-year follow-up are given in table 6. They are in principle very similar to the 8-year follow-up, and the statistical significance of the comparisons, by χ^2, give very similar results. It seemed more rewarding to undertake a correspondence analysis of the two sets of follow-up; the results of this analysis are given in figure 1.

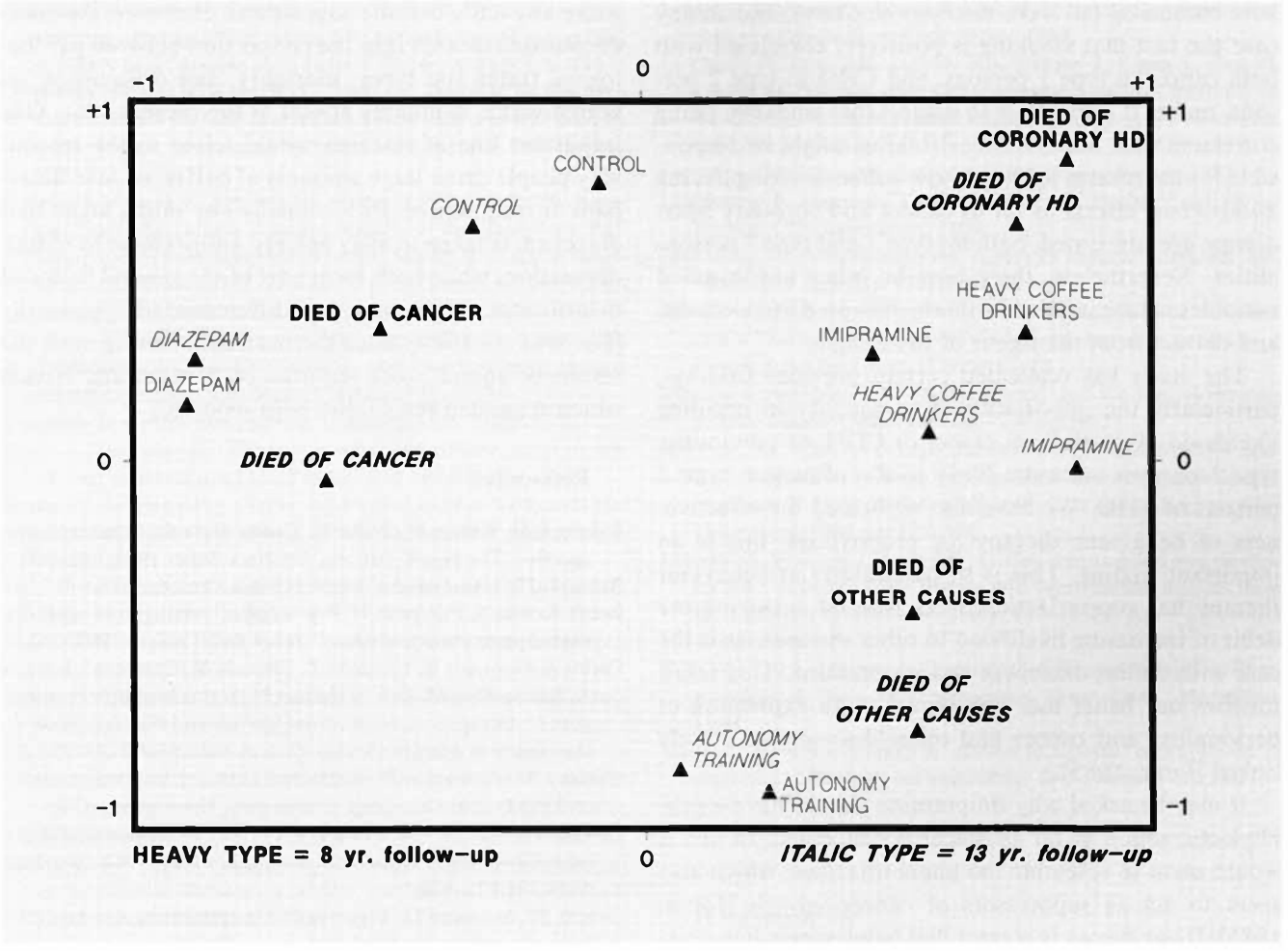

Fig. 1. Correspondence analysis of data from 8- and 13-year follow-up.

Correspondence analysis is a development of multidimensional scaling analysis [Benzecri, 1973; Diday et al., 1986; Greenacre, 1984; Lebart et al., 1984]. It shows in diagrammatic form the relation obtaining between the 5 treatments in our study, and the 3 dependent variables, i.e. death from cancer, CHD, and other causes. The figure incorporates both follow-up sets of data, to demonstrate similarities. The axes have no a priori significance, but in the present case may be roughly interpreted. The ordinate discriminates cancer (left) and CHD (right), with diazepam being linked with cancer, coffee and imipramine with CHD. The abscissa discriminates death from cancer and CHD (top) from avoidance of these diseases (bottom); autonomy training clearly acts as a prophylactic, as compared with the control group; it does not prevent death from other causes (fig. 1).

Discussion

Our results, as far as coffee drinking is concerned, are fairly clear-cut. It appears that for both the cancer-prone and the CHD-prone type, heavy coffee drinking **protects** against cancer, and **increases** the risk of CHD. In this it resembles imipramine, and is at the opposite extreme from diazepam. These results are satisfactory as far as they go, and support the Metzler-Nitsch [1986] results, but of course they should not be over-interpreted. As in all such epidemiological studies, where self-selection plays an important part, there may be many collateral causes which are difficult or impossible to control, other than those selected for analysis. As far as smoking is concerned, we have not found any significant differences in the amount of smoking shown by the different groups

here compared (all were medium smokers), and in any case the fact that smoking is positively correlated with both cancer in type 1 persons, and CHD in type 2 persons, makes it impossible to suggest that smoking, being correlated with heavy coffee drinking, might be responsible for the results, as these show coffee drinking having *antagonistic* effects as far as cancer and coronary heart disease are concerned, both for type 1 and type 2 personalities. Nevertheless, there may be other uncontrolled variables which might facilitate the predicted effects, and detract from the rigour of the design.

The study has replicated certain previous findings, particularly the importance of personality in deciding likelihood of death from cancer or CDH; as previously, type 1 persons are more likely to die of cancer, type 2 persons of CHD. We have also replicated the effectiveness of behaviour therapy for prophylaxis; this is an important finding. This is so particularly as behaviour therapy has prophylactic effects without incurring the debit of increasing likelihood to other diseases, as is the case with coffee, diazepam and imipramine. This result justifies our belief that behaviour, as an expression of personality, and cancer and other diseases are closely linked [Levy, 1985].

It may be asked why imipramine should have a prophylactic action as far as cancer is concerned. In this it would seem to resemble the phenothiazines, which also seem to act as suppressors of cancer growth [Jones, 1985]. If our theory is correct that helplessness-hopelessness depression, as a consequence of a person's inability to cope with interpersonal conflicts, leads to immunosuppression, perhaps through an increase of cortisol in the blood [Eysenck, 1988], then it may be the depression-inhibiting action of imipramine that is responsible for its prophylactic action. Diazepam, on the other hand, is more relevant to the suppression of anxiety, and if such suppression is really basic to cancer proneness [Kissen and Eysenck, 1962; Grossarth-Maticek et al., 1988b], then this very action of diazepam may make matters worse as far as cancer is concerned. These suggestions may be quite premature; the facts are as stated, but possible theories are at present not very firmly based. In particular, the negative effect of imipramine on CHD is difficult to understand. If diazepam does indeed have a prophylactic effect on CHD, this may be due to its sedative action calming down the anger-hostility-aggression behaviour that is so characteristic of the CHD-prone personality.

These possible links are suggested only reluctantly because there is little research which would enable us to make any more definite suggestions. However, they may encourage research into the connection between psychological states and types, mortality, and drug usage, research which is notably absent at the present time. One important line of research would relate to the reasons why people drink large amounts of coffee, or take diazepam or imipramine. Provisionally one might argue that diazepam is taken to allay anxiety, imipramine to reduce depression; while both form part of the general factor of neuroticism, they can be differentiated genetically [Eaves et al., 1989]. Such factors might link up with the results of animal work reported by Metzler and Nitsch which suggested the studies here reported.

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