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PERSONALITY, STRESS, AND MOTIVATIONAL FACTORS IN DRINKING AS DETERMINANTS OF RISK FOR CANCER AND CORONARY HEART DISEASE[1]

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Summary.—Alcohol consumption is a well-known risk factor for cancer, coronary heart disease (CHD), and various other diseases. It is here suggested that motivational factors may be important in mediating any effects of drinking on health. In particular, the hypothesis was tested that drinking to drown one's sorrows (S-type) was much more a risk factor than pleasure drinking (P-type). A total of 1,706 men were tested and followed for 13 years, when death and cause of death were established. The hypothesis was supported for all levels of drinking. In addition, subjects were divided into those 900 who were under stress at the beginning of the study and those 806 who were not. Stress and drinking combined in a complex fashion to form a risk factor for disease, and motivation combined with both in the predicted direction.

The consumption of alcohol is usually treated as a risk factor for cancer (Kissin & Haley, 1974; MacSween, 1982), coronary heart disease (except in small quantities, when it may have a prophylactic effect), and other diseases (Kissin & Begleiter, 1974; Klatsky, Friedman, & Siegelaub, 1981; Marmot, 1984; Marmot, Shipley, & Rose, 1981).

As regards cancer, the evidence is much more convincing than in the case of heart disease (US Department of Health and Human Services, 1981). The work of Schottenfeld (1979), Williams and Horn (1977), and Wynder and Stellman (1977) leaves little doubt on the subject. As far as coronary heart disease is concerned, dosage seems to play an important part, there being an inverse relationship between moderate alcohol use and myocardic infarction (Alderman & Colthart, 1982; Blackwelder, Yano, Rhoads, Kagan, Gordon, & Palesch, 1980; Klatsky, Friedman, & Siegelaub, 1974), but with problem drinkers showing a direct effect (US Department of Health and Human Services, 1981). Stroke seems to present a positive relation with drinking (Katsuki, 1971). All of these studies, of course, suffer from the usual epidemiological failure to use *univariate* rather than *multivariate* statistics (Eysenck, 1991). Clearly, where there are many risk factors, some of them correlated with each other to some extent, attribution of causality to one arbitrarily chosen risk factor is scientifically impermissible. One important risk factor clearly is genetic predisposition (Reed & Hanna, 1986); another may be ethnicity (Reed, 1985).

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Our studies of smoking as a risk factor for these diseases have suggested that physical causes interact with psychosocial ones in a synergistic fashion, so that personality and stress may modulate the effects of smoking (Grossarth-Maticek, Eysenck, & Vetter, 1988; Eysenck, 1988a, 1988b, 1991; Grossarth-Maticek & Eysenck, in press). Here we have tested the hypothesis that drinking may interact with personality and stress in a similar complex fashion, so that personality and stress may *modulate* the effects of alcohol consumption.

For coronary heart disease, important factors appear to be anger or hostility, which have emerged as the major causal traits in the "Type A personality" to link it with disease (Booth-Kewley & Friedman, 1987; Eysenck, 1988b, 1990; Chesney & Rosenman, 1985; MacDougall, Dembroski, Dinsdale, & Hackett, 1985; Williams, Haney, Lee, Kung, Blumenthal, & Whalen, 1980; Kaufman, Rosenberg, & Helurrich, 1985). There is some evidence that hostility or aggression, in turn, may be related to alcohol consumption (Williams, 1970; Jones, 1968; Ritson, 1971; Tarter, 1970; Schonwetter & Janisse, in press).

Drinking may be related to (relief from) anxiety (Spielberger, Gorsuch, & Lushene, 1970; Hire, 1978), so patients treated for alcohol detoxification have a high prevalence of anxiety disorders (Weiss & Rosenberg, 1985). Moderate consumers of alcohol, compared with abstainers and heavy drinkers, have fewer symptoms related to stressful events (Neff, 1989), suggesting the possibility that alcohol, like smoking, may be tension reducing (Levenson, Sher, Grossman, Newman, & Nevlin, 1980; Sher & Levenson, 1982; see also Williams, Calhoun, & Ackoff, 1982).

If it is true that alcohol in moderation may have positive effects on health, either directly through increasing plasma levels of high density big protein cholesterol (Cantelli, Doyle, & Gordon, 1977; Heiss, Johnson, Reiland, Davis, & Tyroler, 1980), through the production of thrombocytopaenia and a reduction of platelet aggregation (Meade, Chakrabarti, Haines, North, & Stirling, 1979), through dietary habits (Jones, Barrett-Connor, Crique, & Holbrook, 1982), or through tension and anxiety reduction leading to a lowering of anger and hostility (Schonwetter & Janisse, in press), then the often found U-shaped relationship between mortality and alcohol use (Marmot, 1981) may have a biological explanation.

Our own study was designed to test the U-shaped relation between mortality and alcohol use; to establish the influence of stress on mortality; to link together these two risk factors in order to establish their relationship (independence, synergistic, additive); and finally to link both with cigarette-smoking as an additional risk factor. Stress was defined in terms of a subject's report of strain, i.e., his subjective reaction to external stress.

As an additional hypothesis, also originating from our published stud-

ies, we argue that motivational factors only partly related to personality and stress might affect the extent to which alcohol consumption might constitute a risk factor for health. In particular, we wish to suggest the possibility that drinking for *negative* causes, i.e., to drown one's sorrows, to relieve tension and anxiety, or to forget for the moment unfavourable circumstances, would have much more serious consequences for one's health than drinking for *positive* reasons, i.e., for enjoyment, for pleasure, or to celebrate some fortunate event. S-type drinking (sorrowful), on this hypothesis, would lead to death from cancer, coronary heart disease, or other diseases significantly more frequently than P-type drinking (pleasureful).

Method

A sample of 1,706 men, aged between 50 and 60 years, were chosen on a random basis from the population register of Heidelberg, a small West German town, and followed up for a period of 13 years. These men were administered a personality inventory, the Personality-Stress Questionnaire by Grossarth-Maticek and Eysenck (1990) which gives scores on six personality types, depending on the subjects' responses to stressful situations. Type 1 is cancer-prone, Type 2 is coronary heart disease-prone, Type 3 has an hysterical personality, Type 4 is a healthy, autonomous sort of person, Type 5 tries to cope with stress by appeals to reason and logic, suppressing emotional reactions, and Type 6 is frankly psychopathic and egocentric. To arrive at a measure of stress, the sum $\Sigma 1$, 2, and 5 – $\Sigma 3$, 4, and 6 is taken. This formula is based on a factor analysis of score intercorrelations, as well as on prospective studies of healthy probands correlating personality type with mortality (Grossarth-Maticek & Eysenck, 1990; Eysenck, 1991). A detailed drinking questionnaire was also administered by interviewers in addition to the Personality-Stress Questionnaire. The main independent variable for our calculations was the average amount of alcohol consumed per diem over a period of at least 5 years, as indicated on the questionnaire. The questionnaire also contained questions concerning the occasions when alcohol was consumed and the feelings associated with such occasions, i.e., negative or positive. Copies of the questionnaire can be obtained from the first author.

The actual questions and responses used to assign a person to the category of S-drinkers or P-drinkers were as follows:

What are the most usual reasons why you drink alcoholic beverages?

(1) Because of long-lasting trouble and grief, because of unsolved personal problems, e.g., because I am unable to accommodate myself to a separation, or to come to terms with a traumatic life event, or to solve a personal problem, or to relax and overcome tension and anxieties, etc.

(2) As a reward for some accomplishment, work well done, or some task brought to a successful conclusion.

(3) To increase pleasure at certain times, such as social gatherings, meetings with a sexual partner, or simply to enjoy the taste of the drink, or the feeling produced by the alcohol.

Probands who ticked (1) were counted as S-drinkers, those who ticked (2) or (3) were counted as P-drinkers.

Statistical calculations used the Vetter algorithm (Vetter, 1988); we are indebted to Dr. Vetter for advice and help with the estimation of probabilities. Most of the p values are so low as to suggest that alternative approaches would not have given different results.

Results

A comparison between 900 stressed and 806 nonstressed probands is given in Table 1; dependent variables are cancer, coronary heart disease, and other causes of death. Mortality was ascertained by visits to the homes of probands, and cause of death by consulting death certificates. Mortality was over 20% higher in the stressed probands than in the nonstressed ($p < .001$), so stress must be regarded as a risk factor for health, very much as our earlier studies, using rather different indices, had suggested (Eysenck, 1991; Grossarth-Maticek, Eysenck, & Vetter, 1988).

TABLE 1
Stress as Related to Mortality

	With Stress		Without Stress	
	f	%	*f*	%
*n*s	900		806	
Cancer	130	14.4	42	5.2
Coronary Heart Disease	93	10.3	26	3.2
Other Causes of Death	139	15.7	52	6.5
Still Living	538	59.8	686	85.1

Scores were analysed by analysis of variance, keeping the effects of drinking and smoking constant. The results were significant at the .001 level for cancer, the .001 level for coronary heart disease, and .01 for "other causes." For those still living, the p value was .001. These results make it clear that stress (as defined) is very significantly associated with mortality when smoking and drinking have been statistically controlled.

Table 2 shows the difference between those ($n = 404$) who were nondrinkers and those ($n = 1{,}302$) who drank alcohol in varying amounts. As suggested by the literature surveyed in the Introduction, alcohol consump-

TABLE 2
Mortality as Related to Alcohol Consumption

	n	Cancer		Coronary Heart Disease		Other Causes		Still Living	
		f	%	*f*	%	*f*	%	*f*	%
No Alconol	404	33	8.1	30	7.4	38	9.4	303	75.0
Alcohol	1302	139	10.6	89	6.8	153	11.7	921	70.7

tion is *positively* related to cancer, *negatively* to coronary heart disease, and positively to other causes of death, but the differences are too slight to assume any great importance; there is no statistical significance here. Clearly, what is needed are dose-response relationships, and interaction effects with stress.

Table 3 shows the *interaction* effects of drinking and stress, with drinkers divided into S- and P-drinkers but without taking into account the amount of alcohol consumed. Nondrinkers with stress are clearly more susceptible to cancer, coronary heart disease, and other diseases than are nonstressed nondrinkers. S-drinkers with stress are more susceptible to these diseases than S-drinkers without stress. P-drinkers with stress, too, follow the same pattern. Thus, stress *in all groups* is an effective risk factor for mortality and reaches statistical significance ($p < .001$).

TABLE 3
MORTALITY AS RELATED TO STRESS AND ALCOHOL CONSUMPTION

	No Alcohol			
	With Stress, $n = 203$		Without Stress, $n = 201$	
	f	%	*f*	%
Cancer	25	12.3	8	3.9
Coronary Heart Disease	23	11.3	7	3.4
Other Causes of Death	26	12.8	12	5.9
Σ	74	36.5	27	13.4
Still Living	129	63.5	174	86.6

	Alcohol Consumers							
	Pleasure Drinkers		Sorrow Drinkers		Pleasure Drinkers		Sorrow Drinkers	
	f	%	*f*	%	*f*	%	*f*	%
Over-all	191	27.4	506	72.6	481	79.5	124	20.5
Cancer	18	9.4	87	17.1	23	4.7	11	8.8
Coronary Heart Disease	11	5.7	59	11.6	12	2.5	7	5.6
Other Causes of Death	20	10.4	93	18.3	23	4.7	17	13.7
Σ	49	25.7	239	47.2	58	12.1	35	28.2
Still Living	142	74.3	267	52.8	423	87.9	89	71.8

As regards the influence of *type of drinker*, P-drinkers are not differentiated from nondrinkers when there is no stress; when there is stress, P-drinkers are slightly worse off (cancer) or slightly better off (coronary heart disease) than corresponding nondrinkers. The S-drinkers, however, with or without stress, have a higher mortality for other causes of death than S-drinkers with stress show for cancer and for coronary heart disease. Clearly stress is more important for coronary heart disease than drinking, while both in combination are important risk factors for cancer, coronary heart disease, and "other causes of death."

This point is brought out more clearly in Table 4, which presents the relevant comparisons. It is, of course, the combination of stress and drinking which is most lethal for cancer and other causes of death, but much less so for coronary heart disease.

TABLE 4
TYPE OF DRINKING, STRESS, AND MORTALITY: PERCENT

Classification	Cancer	Coronary Heart Disease	Other Causes	Still Living
1. P-drinkers without stress	4.7	2.5	4.7	87.9
2. Nondrinkers without stress	3.9	3.4	5.9	86.6
3. P-drinkers with stress	9.4	5.7	10.4	74.3
4. S-drinkers without stress	8.8	5.6	13.7	71.8
5. Nondrinkers with stress	12.3	11.3	12.8	63.5
6. S-drinkers with stress	17.1	11.6	18.3	52.8

Looking at statistical significance, and using again the model of analysis of variance employed in relation to Table 1, we find for cancer that drinking and stress are significant ($p = .01$ and .001); for coronary heart disease similar p values are obtained, while for "other causes" p values are .001 and .01. For all causes of death together p values are .001 and .001. Looking at P- vs S-type drinking, only "other causes of death" gives a significant result ($p = .05$), with all causes of death taken together giving a strong significant difference ($p = .001$). Abstainers are significantly differentiated from P-drinkers for coronary heart disease only ($p = .05$) and from S-drinkers for "other causes" only ($p = .05$).

We must now turn to a consideration of the quantitative aspects of drinking. Table 5 shows the results for cancer mortality. Apart from nondrinkers, there are six groups of drinkers classified by increasing amounts of drinking. In addition, we have divided drinkers again into P- and S-drinkers, and all probands were divided into those with and without stress. This inevitably means that numbers in some categories are quite small, but the over-all trends are clear. (1) For all groups, there is an increase in mortality with increase in alcohol consumption. This is least for P-drinkers without stress. The other three groups show increments which are not clearly differentiated although they are differentiated from the no-stress P-drinking group. (2) There is little if any effect of alcohol consumption below 61 grams; the regression is clearly nonlinear for all groups. The slight dip at the beginning of the table, for low consumption of alcohol as compared with abstinence, may or may not be replicable; it would be safest to regard it as accidental.

Table 6 gives similar figures for coronary heart disease mortality. (1) The results are comparable to those above, with P-drinkers without stress showing practically no increase in mortality, while S-drinkers with stress

TABLE 5

DOSE-RESPONSE RELATIONSHIP AMONG ALCOHOL CONSUMPTION, STRESS, TYPE OF DRINKING, AND MORTALITY FOR CANCER (FREQUENCY AND PERCENT)

Alcohol Consumption Daily	Cancer Mortality										Age (yr.)
	n	With Stress				*n*	Without Stress				
		f		%			*f*		%		
0 grams	203	25		12.3		201	8		3.9		57.3
		P-Drinkers		S-Drinkers			P-Drinkers		S-Drinkers		
		f	%	*f*	%		*f*	%	*f*	%	
1—20 gm	149	60	40.2	89	59.7	132	100	75.7	32	24.3	57.1
Mortality		3	5.0	9	10.1		3	2.7	2	6.5	
21—40 gm	103	25	24.3	78	75.7	97	71	73.2	26	26.8	56.9
Mortality		1	4.0	10	12.8		2	2.8	1	3.8	
41—60 gm	153	24	15.7	129	84.3	126	101	80.2	25	20.8	57.4
Mortality		2	8.3	19	14.7		3	2.9	1	4.0	
61—80 gm	109	31	28.4	78	71.6	76	61	80.3	15	19.7	56.7
Mortality		4	12.9	15	19.2		4	6.4	2	13.3	
81—100 gm	83	20	24.1	63	75.9	79	62	78.5	17	21.5	56.1
Mortality		5	25.0	16	25.3		4	6.4	3	17.6	
101 + gm	100	31	31.0	69	69.0	95	86	90.5	9	9.5	55.8
Mortality		3	9.6	18	26.0		7	8 1	2	22.2	

have much the highest mortality. (2) Again, there is a slight fall from non-drinkers to restrained drinkers, with the rise proper beginning when consumption exceeds 61 to 80 grams.

Table 7 shows similar results for other causes of death. Again, the numbers are too small to allow certainty for conclusions drawn, but mortality with stress is clearly much higher than mortality without stress in nearly all groups and in S-drinkers as compared with P-drinkers. Again, there is a non-

TABLE 6

DOSE-RESPONSE RELATIONSHIP AMONG ALCOHOL CONSUMPTION, STRESS, TYPE OF DRINKING, AND MORTALITY FOR CORONARY HEART DISEASE (FREQUENCY AND PERCENT)

Alcohol Consumption Daily	Coronary Heart Disease: Mortality									
	n	With Stress				*n*	Without Stress			
		f		%			*f*		%	
0 grams	203	23		11.3		201	7		3.4	
		P-Drinkers		S-Drinkers			P-Drinkers		S-Drinkers	
		f	%	*f*	%		*f*	%	*f*	%
1—20 gm	149	2	3.3	7	7.8	132	2	2.0	1	3.1
21—40 gm	103	2	8.0	8	10.2	97	1	1.4	1	3.8
41—60 gm	153	1	4.1	10	7.7	126	2	1.9	1	4.0
61—80 gm	109	1	3.2	9	11.5	76	2	3.2	1	6.6
81—100 gm	83	2	10.0	15	23.8	79	3	4.8	2	11.7
101 + gm	100	3	9.6	10	14.4	95	2	2.3	1	11.1

TABLE 7
DOSE-RESPONSE RELATIONSHIP AMONG ALCOHOL CONSUMPTION, STRESS, TYPE OF DRINKING, AND MORTALITY FOR OTHER CAUSES OF DEATH (FREQUENCY AND PERCENT)

Alcohol Consumption Daily	Other Causes of Death								
	n	With Stress				*n*	Without Stress			
		f		%			*f*		%	
0 grams	203	26		12.8		201	12		5.9	
		P-Drinkers		S-Drinkers			P-Drinkers		S-Drinkers	
		f	%	*f*	%		*f*	%	*f*	%
1—20 gm	149	3	5.0	9	10.1	132	2	2.0	2	6.2
21—40 gm	103	3	12.0	12	15.3	97	2	7.0	2	7.6
41—60 gm	153	2	8.3	14	10.8	126	5	4.9	3	12.0
61—80 gm	109	3	9.6	14	17.9	76	3	4.9	3	20.0
81—100 gm	83	3	15.0	20	31.7	79	4	6.4	4	23.5
101 + gm	100	6	19.3	24	34.7	95	7	8.1	3	33.9

significant suggestion of a dip in the mortality curve at the beginning, and a proper rise only beginning at the level of 61 to 80 grams. The uniformity of the doses for all three causes of death suggests but does not prove that possibly ingestion of moderate amounts of alcohol may have an health-giving or health-preserving function (US Department of Health and Human Services, 1981).

The statistical analysis of Tables 5, 6, and 7 was as follows. We first ran an analysis of covariance with slopes for quantity of alcohol, depending on mode of drinking and on stress in order to assess whether the effect of quantity of alcohol consumed was unequal for the drinking and stress group. Results were nonsignificant for cancer and coronary heart disease and barely so ($p = .05$) for "other causes," such that for P-drinkers, equal quantities are associated with less mortality than for S-drinkers. For all causes of death, taken together, the same pattern was observed with p equal to .001. Thus over-all P-drinking is definitely less injurious than S-drinking.

For determining the effects of drinking mode and stress, with quantity controlled, we used an analysis of covariance model with homogeneous slopes for quantity of alcohol consumption. The result were uniformly significant at the .001 level (drinking mode) for cancer, coronary heart disease, and other causes; similarly for stress, all three values were significant at the .001 level. Looking at slopes for alcohol quantity effects, these were significant at the .001 level for cancer and other causes of death and at the .01 level for coronary heart disease.

Interaction of Drinking, Smoking, and Stress

In the analysis presented so far, we have not included smoking as a risk factor possibly correlated with drinking and personality, and possibly interacting synergistically with both. Smoking patterns were ascertained for all

probands at the beginning of the study, and Table 8 shows the results for different groups of drinkers. By chi squared, these results are statistically significant, with $p < .0001$. A *phi* coefficient of 0.364 indicates the strength of that association.

TABLE 8
RELATIONSHIP BETWEEN SMOKING AND DRINKING: FREQUENCY AND PERCENT

Group	Nonsmokers		Smokers		Total
	f	%	*f*	%	
Abstainers	288	71.29	116	28.71	404
Pleasure Drinkers	167	24.85	505	75.15	672
Sorrow Drinkers	247	39.21	383	60.79	630
Total	702		1004		1706

Abstention from alcohol is closely related to nonsmoking. Sorrow-drinkers, rather unexpectedly, are more frequently nonsmokers than pleasure-drinkers, although both types of drinkers are much more likely to be smokers than nonsmokers. Perhaps the pleasure-drinkers smoke for different reasons than the sorrow-drinkers; there are different types of smokers just as there are different types of drinkers, differing in a predictable way with regards to reasons for smoking and occasions when smoking occurs (Eysenck, 1973; Spielberger, 1986).

Table 9 shows the various combinations of smoking (S), stress (Y), and drinking (X) as relating to mortality from all causes. Groups would have been too small to give mortalities separately for cancer, coronary heart disease, and other causes. As an example, consider level of smoking. Nonsmoking is designated 0, smoking is designated 1. The mortality of nonsmokers is 0.256, that of smokers is 0.300. Similarly for stress in this table,

TABLE 9
MORTALITY AS RELATED TO SMOKING, STRESS, AND DRINKING, SINGLY, AND IN COMBINATION

			n	Proportion
Level of Smoking (S)				
		0	702	0.256
		1	1004	0.300
Level of Stress (Y)				
		0	806	0.149
		1	900	0. 402
	S	Y		
	0	0	295	0.159
	0	1	407	0.327
	1	0	511	0.143
	1	1	493	0.464

(continued on next page)

TABLE 9 (CONT'D)
MORTALITY AS RELATED TO SMOKING, STRESS, AND DRINKING, SINGLY, AND IN COMBINATION

			n	Proportion
Level of Drinking (X)				
	0	None	404	0.250
	1	P	672	0.159
	2	S	630	0.435
	S	X		
	0	0	288	0.253
	0	1	167	0.293
	0	2	247	0.235
	1	0	116	0.241
	1	1	505	0.115
	1	2	383	0.564
	Y	X		
	0	0	201	0.134
	0	1	481	0.121
	0	2	124	0.282
	1	0	203	0.365
	1	1	191	0.257
	1	2	506	0.472
	S	Y	X	
	0	0	0	137	0.153
	0	0	1	105	0.190
	0	0	2	53	0.113
	0	1	0	151	0.344
	0	1	1	62	0.468
	0	1	2	194	0.268
	1	0	0	64	0.094
	1	0	1	376	0.101
	1	0	2	71	0.408
	1	1	0	52	0.423
	1	1	1	129	0.155
	1	1	2	312	0.599

mortality for no-stress probands (0) is 0.149, while for stressed probands (1) mortality is 0.402.

Table 10 shows the results of the analysis of variance. All main variables (smoking, stress, drinking) are clearly risk factors for mortality ($p < .001$). Smoking and drinking show equally high interaction. Stress shows up as a synergistic factor in the triple interaction ($p < .006$). This is the most important analysis of our combined data, and it powerfully reinforces the nature of the risk factors studied, in isolation and in combination. *Stress clearly emerges as the most damaging risk factor of the three.*

Confirmatory Study

The analyses reported in the preceding section might be criticized because groups were not equated on such variables as smoking, blood pressure,

TABLE 10
ANALYSIS OF VARIANCE FOR DIFFERENT RISK FACTORS AFFECTING MORTALITY

Source	*df*	Type II *SS*	*MS*	*F*	*p*
S = Smoking	1	2.30	2.30	13.51	<.001
Y = Stress	1	9.58	9.58	56.32	<.001
S × Y	1	0.09	0.09	0 50	.48
X = Drinking	2	8.26	4.13	24 28	<.001
S × X	2	14.82	7.41	43 57	<.001
Y × X	2	0.49	0.25	1.44	.24
S × Y × X	2	1.72	0.86	5.06	.01

blood sugar, cholesterol, and hereditary predisposition. An attempt was made to replicate the original study by extracting from a large group of over 5000 subjects originally studied in 1973, six groups of 61 adult male probands each, who would be equated on all these variables, so that there would be no significant differences between them on the variables mentioned above. Hereditary predisposition was measured by the number of parents and grandparents who suffered or died from cancer and coronary heart disease. Ascertained after 13 years were mortality and incidence; for the latter, the responsible physician was consulted after obtaining written agreement from the patient.

We chose three groups of personality Type 1, i.e., the cancer-prone, and three groups of personality Type 2, i.e., the coronary heart disease-prone. Thus personality or stress typology is held constant, and the results for Types 1 and 2 may be compared. Within each type we have three groups: (1) P-drinkers who habitually drank daily more than 60 grams of alcohol for more than 10 years, (2) S-drinkers who imbibed a similar quantity over the same period, and (3) abstainers. The results are shown in Table 11.

Table 11 was analysed with an analysis of variance model (Vetter, 1988). The independent variables were type, drinking (three levels), and their interaction; drinking was analysed in terms of two contrasts: drinking vs abstinence and sorrow- vs pleasure-drinking. The dependent variables were (1) mortality (cancer, coronary heart disease, other; all causes) and (2) morbidity (cancer, coronary heart disease, either). For *mortality* S- vs P-drinking was significant ($p = .001$) for cancer, coronary heart disease, and all causes, but not for "other causes." Personality type was significant ($p = .001$) for coronary heart disease; other comparisons were insignificant. For *morbidity*, S- vs P-drinking was significant ($p = .001$) for cancer, coronary heart disease, and "either;" personality type was significant ($p = .05$) for cancer and also for coronary heart disease ($p = .001$). The effect for Type 1 personality was always to enhance cancer, for Type 2 to enhance coronary heart disease. The effect of sorrow-drinking always made for higher mortality and morbidity than pleasure-drinking.

TABLE 11

MORTALITY AND TYPE OF DRINKING IN CANCER-PRONE (TYPE 1) AND CORONARY HEART DISEASE-PRONE (TYPE 2) PROBANDS EQUATED ON SMOKING AND OTHER VARIABLES (*n*S = 61)

		Mortality			Incidence		
		Cancer	Coronary Heart Disease	Other Causes	Cancer	Coronary Heart Disease	Not Traced
Type 1							
P-drinker	*f*	4	1	15	7	3	1
	%	6.7	1.7	25.0	11.7	5.0	
S-drinker	*f*	14	6	19	26	12	0
	%	23.0	9.8	31.1	42.6	19.7	
Abstainer	*f*	8	3	13	13	11	1
	%	13.3	5.0	21.7	21.7	18.3	
Type 2							
P-drinker	*f*	2	3	16	4	6	0
	%	3.3	4.9	26.2	6.6	9.8	
S-drinker	*f*	9	18	21	16	27	0
	%	14.8	29.5	34.4	26.2	44.3	
Abstainer	*f*	5	11	13	9	21	1
	%	8.3	18.3	21.7	15.0	35.0	

Two other results are important also. (1) The interaction of type of personality and drinking was never significant. (2) The difference between drinking and abstinence was never significant. In other words, drinking in and of itself had no effect on cancer or coronary heart disease; all depended on the type of drinking.

The data enable us to draw several conclusions: (1) Personality type is related as expected to cancer and coronary heart disease, with the latter showing a stronger relationship statistically. (2) P-drinkers have an over-all mortality of 41, abstainers of 53, and S-drinkers of 87; again, P-drinkers are more like abstainers, and again we have the tantalizing suggestion that P-drinking may have a positive influence on survival, for both cancer and coronary heart disease, but again the difference falls short of significance. (3) For incidence, figures are similar to those for mortality, again with the suggestion that P-drinking might actually prevent cancer and coronary heart disease. (4) Drinking as such is not implicated in the causation of cancer or coronary heart disease; only differences in type of drinking (S- vs P-) give strong statistical evidence of a relationship.

These data support our major conclusions from the larger study and enable us to be more certain of our findings because care was taken to eliminate from consideration a number of alternative risk factors. Replication is the most acceptable form of validation of findings, and we may suggest that *psychological factors determining the cause of drinking and the emotions and motivations involved play important parts in deciding the health consequences of alcohol consumption.*

Conclusions

It is important not to overinterpret our findings, particularly as the results are correlational only and do not readily permit causal interpretations. Nevertheless, we have attempted to rule out certain alternative hypotheses by controlling for personality or stress in relation to type of drinking, by equating samples for smoking and other physical variables, etc. In spite of such precautions, there must remain many variables which might have influenced results one way or the other and which remain uncontrolled; that is the universal weakness of epidemiological studies, even when a prospective paradigm is employed.

Given this obvious precaution, it seems that there are certain conclusions which may justifiably be drawn from the data. Some of these replicate previous findings, e.g., that personality and stress are clearly risk factors in cancer and coronary heart disease and that typology is related to cause of death, Type 1 being more closely connected with cancer, Type 2 with coronary heart disease (Eysenck, 1984a, 1984b, 1985, 1987a, 1987b, 1990a, 1990b; Grossarth-Maticek, Bastiaans, & Kanazir, 1985; Grossarth-Maticek, Eysenck, Vetter, & Schmidt, 1988; Grossarth-Maticek, Kanazir, Schmidt, & Vetter, 1982, 1985; Grossarth-Maticek, Kanazir, Vetter, & Jankovic, 1983).

In a similar way, our findings that drinking, particularly for consumption in excess of 60 grams per diem, acts as a risk factor for cancer, coronary heart disease, and other diseases is well in line with earlier work already referenced. So is our finding that *small* amounts of alcohol do not constitute a risk and may in fact have a positive rather than a negative effect on health. This effect is not very strong, but it appears in both our studies and in most of the subgroups investigated.

Our major novel finding is the support given by the data for the hypothesis that type of drinking, whether for pleasure or to drown sorrow, is of vital importance for the health consequences of drinking. When stress, amount of drinking, age, and sex are all controlled experimentally or statistically, it is still noted that P-drinkers are as well off as abstainers, while S-drinkers have a significantly higher mortality and morbidity than both. It is of course possible to argue that it is the events *causing* the sorrow of the S-drinkers which are responsible for the negative health consequences, so that essentially drinking has no effect at all. This seems unlikely in view of the dose-response relationship and the fact that the relationship obtains both for stressed and nonstressed probands. However, it is inherently difficult to separate cause and effect in this case, and we can only argue on the basis of probability that the effects noted are at least in part due to the effects of alcohol, with type of drinking modulating the effects on the organism.

Less novel, and in line with previous work, is the finding that small amounts of alcohol do not have a deleterious effect on health and may in-

deed have a beneficial effect. It would be irresponsible to regard the point of positive effects of small doses of alcohol as proven, but taken together with previous work our data do seem to point in that direction. As far as coronary heart disease is concerned, the well-established physical effects of alcohol on the circulatory system may mediate the beneficial effects but with regards to cancer and other causes of death there is no obvious explanation. The reference is to the high density lipoprotein-cholesterol hypothesis, which is based on the observation that alcohol raises high-density lipoprotein cholesterol (HDL-C) levels in blood (see Cantelli, Gordon, Hjortland, Kagan, Doyle, Hames, & Zukel, 1977; Hulley, Cohen, & Widdowson, 1977; Wallerstedt, Gustafson, & Olsson, 1977). Other studies have shown that elevated HDL-C is inversely related to coronary atherosclerotic disease and may play a protective role by aiding in the removal of cholesterol from the body by retarding the formation of atherosclerotic plaques (Rhoads, Gulbrandsen, & Kagan, 1976).

For further studies, our results carry some important suggestions. Prospective studies, in spite of their difficulty and cost, are essential if worthwhile results are to be obtained. These must be on a large scale, in view of the many subgroups requiring examination if multivariate analyses are to be made. The necessity of holding constant, or partialling out, experimentally or statistically, risk factors other than the one under investigation will be obvious, although the point has been neglected in much published work. All such studies should include prominently *psychosocial* variables, such as stress and personality type, in view of the number of times these have been noted as important risk factors for cancer, coronary heart disease, and other causes of death. In addition, it is to be hoped that other researchers would be more informed about their designs by the need to answer theoretical questions raised by previous work and the equally pressing need to solve the problems raised by anomalies so frequently found in empirical investigations.

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