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PSYCHOLOGICAL FACTORS IN THE RELATIONSHIP BETWEEN ALCOHOL AND CARDIOVASCULAR MORBIDITY

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Abstract—Most major studies have found a U-shaped relationship between the level of alcohol consumption and all cause mortality, largely as a consequence of lower death rates from coronary heart disease (CHD) amongst moderate drinkers. Previous attempts to unravel the significance of this observation have focused on controlling for possible confounders, such as smoking, social class and the existence of previous ill-health in the group of abstainers. Our analysis of data from the Whitehall II study of British Civil Servants sought to determine whether psychological factors (GHQ, Hostility, Affect Balance, Social Supports) may be influencing the observed relationships between levels of alcohol consumption and some of the established risk factors for CHD. We found evidence of weak confounding only with respect to levels of apolipoprotein B (APoB) and as such have failed to provide compelling evidence that the U-shaped relationship between alcohol and CHD mortality could be easily explained by psychosocial confounding. At the same time we would not claim that the measures we have used are either flawless or exhaust the range of psychological variables that might plausibly influence physiological mediators of cardiovascular disease.

Key words—alcohol morbidity, coronary heart disease, risk factors, blood pressure, psychosocial factors

INTRODUCTION

As is now well established [1, 2] most major studies have found a U-shaped relationship between the level of alcohol consumption and all cause mortality, this being largely a consequence of lower death rates from coronary heart disease (CHD) amongst moderate drinkers. This has generated considerable debate as to whether moderate levels of alcohol confer a protective effect [3, 4]. Previous attempts to unravel the significance of this observation have centred on controlling for possible confounders, such as smoking, social class [5] and the existence of previous ill-health in the group of abstainers [6, 7]. We would like to present data from the Whitehall II study of British Civil Servants [8] which seeks to ascertain whether previously unconsidered psychological factors could play some role in confounding relationships between levels of alcohol consumption and some of the established risk factors for CHD. It is certainly conceivable that how a person deals with stressful life events (for example by recourse to varying levels of social support) may be related both to their current level of alcohol consumption and to future disease outcomes. Were such relationships to be confirmed, then reinterpretation of the significance of the U-shaped curve would be appropriate.

SUBJECTS AND METHODS

We began investigating a cohort of 10,314 (6900 males and 3414 females) civil servants aged from 35 to 55 between 1985 and 1988 [8]. Subjects each completed a self-administered questionnaire which included social and demographic data, a variety of health status measures, details of health behaviours, social networks and social supports and a number of measures of psychological functioning. In addition respondents attended a medical screening examination at their place of work. We obtained a response rate of 73% from our original list, which after allowing for those who had moved and were no longer eligible at the time of entry to the study, is likely to be around 4% higher.

For the current analysis we selected several indicators of physiological functioning and health state measured during the screening examination [blood concentrations of apolipoproteins AI (ApoAI) and B (ApoB), fibrinogen, factor 7, cholesterol and systolic blood pressure]. All of these are potential mediators in cardiovascular disease [8–11]. Psychological and psychosocial measures used, included a Life Events scale, a scale for degree of upset associated with a life event, the GHQ, Satisfaction with Life Scale, Hostility Scale [12], positive and negative subscales of the Affect Balance Scale [13], measures of different types of social support confirmed

from factor analysis [14] (confiding, emotional, practical and negative) and measures of 7 different psycho-social work characteristics (pace, conflicting demands, variety and skill use, level of control, support at work, job importance and job satisfaction). Alcohol intake was assessed from that reported in the week immediately prior to completion of the questionnaire. This was subsequently grouped into five levels (1 = None; 2 = Light; 3 = Moderate; 4 = Moderate to Heavy; and 5 = Heavy) with different cut off points for males and females—for males 1 (no alcohol consumed in previous week), 2 (1–10 units), 3 (11–20), 4 = (21–30) and 5 (31 units and above), for females 1 (no alcohol consumed in previous week), 2 (1–6 units), 3 (7–10), 4 (11–20), and 5 (21 units and above). We are not able to distinguish between lifelong and current abstainers. Smoking status has been coded into 5 levels [none, ex-smoker, light (1–10 cigs/day), medium (11–20/day) and heavy (>21/day)]. Table 1 shows the breakdown of males and females in our sample belonging to each drinking category.

Other variables in the analyses include employment grade (ranging from 1 to 6 in order of decreasing salary) and menopausal status (as determined by whether still having periods). The latter has been included in order to provide more accurate adjustment of fibrinogen levels in women. Brunner *et al.* [15] report that fibrinogen levels in women show no relation with age before menopause but rise with age after it.

Our initial analysis examined the nature of the relationships between our chosen variables and different levels of alcohol consumption. Mean values were computed from general linear models using SAS procedure GLM [16] controlling for age, smoking level and social class (as measured by employment grade). Smoking level and grade level were entered as categorical variables.

RESULTS

(i) Physiological and health state measures

Levels of fibrinogen were found to exhibit significant variation with level of alcohol consumption for both males ($P = 0.0008$) and females ($P = 0.0001$) as was the case for ApoAI ($P = 0.0001$ for both genders). For males variation was observed in levels of APoB ($P = 0.0532$), cholesterol ($P = 0.0001$) and systolic blood pressure ($P = 0.0001$). Factor 7 exhibited no significant variation with alcohol consumption for either sex. Examination of the data suggested the presence in males of possible U-shaped relationships with alcohol consumption for systolic blood pressure (bps), and ApoB, with light drinkers tending to have lower levels than either non-drinkers or heavy drinkers.

Table 1. Composition of sample by different levels of reported alcohol consumption in previous week

Alcohol consumption in previous week	Males	Females
None	951	1022
Low	3120	1428
Moderate	862	423
Moderate–heavy	1294	384
Heavy	673	157
Total	6900	3414

(ii) Psychological and psychosocial measures

With the exception of life events, all the psychological variables showed some significant variation with level of alcohol consumption. Though there were gender differences in which variables were significant. For males these were GHQ ($P = 0.005$), Satisfaction with Life ($P = 0.0069$), Hostility ($P = 0.0001$), Positive Affect ($P = 0.0043$), Negative Affect ($P = 0.0013$), Degree of Upset ($P = 0.0020$), Confiding support ($P = 0.0047$), Emotional support ($P = 0.0024$), General support ($P = 0.0098$) and Work satisfaction ($P = 0.0016$). For females the significant variables were Hostility ($P = 0.0004$), Positive Affect ($P = 0.0005$) Negative Affect ($P = 0.0013$), Practical support ($P = 0.0201$) and Work satisfaction ($P = 0.0016$). On some of these indices light drinkers show better psychological well-being [GHQ, Positive Affect, Level of upset (males only) and Hostility (both genders)], and lower levels of social support (confiding, emotional and general—males only) than either the none drinkers or heavy drinkers.

The question thus arises whether any of these psychological factors play a part in producing the observed relationships between alcohol consumption and the various biochemical and physiological endpoints, such that for (a) systolic blood pressure and ApoB the observed U-shaped relationships would disappear or for (b) fibrinogen, ApoAI, factor 7 and cholesterol, consideration of psychological parameters could lead to U-shaped relationships where non presently exist. (In both sexes the prior trend is for Fibrinogen levels to decline with increased consumption and ApoAI levels to increase. Neither sex show any clear pattern in factor 7 levels against consumption prior to adjustment whereas cholesterol levels increased with consumption in males only.)

To investigate these possibilities further new general linear models of the physiological endpoints were constructed separately for each gender; adding to age, grade level and level of smoking those psychological and psychosocial variables which for each sex were found to vary significantly with alcohol consumption.

The main effects of adjustment for psychosocial factors are presented in Table 2. For males, variation in levels of ApoB by level of alcohol consumption was reduced to below statistical significance, whilst co-variation with levels of factor 7 was introduced at a low level of statistical significance ($P = 0.049$). Variations in fibrinogen levels were attenuated but still significant. For females, differences in levels of ApoB by drinking category were enhanced after adjustment

Table 2. Least Squares Mean estimates of levels of cardiovascular risk factors by level of alcohol consumption showing factors affected by adjustment for psychosocial variables

1. *Males*

Alcohol consumption	Fibrinogen Before adjustment	Fibrinogen After	ApoB Before adjustment	ApoB After	Factor 7 Before adjustment	Factor 7 After
None	2.93	2.97	107.49	109.01	86.99	86.70
Low	2.85	2.85	106.89	109.10	87.42	87.92
Moderate	2.80	2.84	107.41	108.34	91.26	91.76
Mod–heavy	2.78	2.83	109.81	111.77	88.38	89.03
Heavy	2.73	2.78	108.47	108.79	91.73	93.13
Probability	0.0008	0.013	0.053	NS	NS	0.049

2. *Females*

Alcohol consumption	ApoB Before adjustment	ApoB After	BPS Before adjustment	BPS After
None	99.88	104.56	119.37	119.13
Low	98.63	101.29	119.25	119.26
Moderate	97.16	99.62	118.03	117.19
Mod–heavy	96.56	96.78	119.52	119.75
Heavy	99.31	99.30	122.31	123.04
Probability	NS	0.026	NS	0.059

($P = 0.0256$) with those reporting no alcohol consumption in the previous week having the highest level (an increase of almost 5% on preadjustment levels). Adjustment also introduced variation in systolic blood pressure ($P = 0.059$), with an adjusted decrease 0.84 mmHG for moderate drinkers and an increase of 0.73 mmHG for heavy drinkers.

DISCUSSION

Our results suggest that in males and females, different emotional and psychological factors are associated with variations in levels of alcohol consumption. In itself this should not be considered surprising. In addition some evidence has been presented to suggest that relationships between some of the existing cardiovascular risk factors and levels of consumption may be weakly confounded by some of the psychological and psychosocial factors we have studied. Where such relationships have been found we are not as yet in a position to state the direction of causation; whether the psychological parameters reflect responses to changing health circumstances or whether in some way they mediate the expression of the health parameters we have studied. As these results stand, they do not provide compelling evidence that the U-shaped relationship between alcohol and CHD mortality could be easily explained by psychosocial confounding. At the same time we would not claim that the measures we have used are either flawless or exhaust the range of psychological variables that might plausibly influence physiological mediators of cardiovascular disease. One difficulty with interpreting some of the results pertaining to social supports is that it is uncertain whether lower levels of support indicate lower levels of actual support or a lower need for support. Despite the somewhat negative tone of these results we would still urge that attention be directed to the psychosocial milieu in which drinking occurs. Differences in patterns of alcohol consumption e.g. binge drinking vs slower steady drinking over a prolonged period probably co-vary with different psychosocial stressors. These will need to be understood before the relative advantages or disadvantages of different consumption patterns can be attributed solely to differences in the physiological processing of alcohol.

The importance of studying psychosocial factors with respect to morbidity, independently for males and females is further emphasized by data which point to the differing salience of some of the psychological factors for each gender. We found females more likely than men to report greater numbers of life events in the preceding year (1.17 vs 1.05, $P = 0.0001$), greater levels of upset [after controlling for number of life events (3.14 vs 2.88) $P = 0.0001$] and increased levels of confiding (29.05 vs 26.25, $P = 0.0001$), emotional (14.16 vs 13.69, $P = 0.071$) and general support (41.80 vs 39.45, $P = 0.0019$) after controlling for number of life events and degree of upset. This concurs with work which reports females show greater emotional expressiveness and ability to decode emotions than do males [17].

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