

What is already known on this topic

Drinking wine is associated with lower mortality than drinking beer and spirits

Self reports suggest that wine drinkers have healthier diets than beer or spirits drinkers

What this study adds

An objective measure of alcohol intake and dietary habits found that people who buy and presumably drink wine make more purchases of healthy food items than people who buy beer

metabolic advantages; in contrast, spirits are often consumed at times other than mealtime. In Denmark wine drinkers have a higher level of education, higher income, better psychological functioning, and better subjective health than people who do not drink wine.^{8,9} Similar results have been found in a Californian population: people who prefer wine tend to be educated, healthy, lean, young or middle aged women with a moderate alcohol intake, whereas those who prefer beer tend to be less educated, healthy young men with a higher alcohol intake.¹⁰ Thus, the influence of type of alcoholic drink on mortality could be due to insufficient adjustment for lifestyle factors.⁸

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Chronic stress at work and the metabolic syndrome: prospective study

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Abstract

Objectives To investigate the association between stress at work and the metabolic syndrome.

Design Prospective cohort study investigating the association between work stress and the metabolic syndrome.

Participants 10 308 men and women, aged 35-55, employed in 20 London civil service departments at baseline (the Whitehall II study); follow-up was an average of 14 years.

Main outcome measures Work stress based on the iso-strain model, measured on four occasions (1985-99). Biological measures of the metabolic syndrome, based on the National Cholesterol Education Program definition, measured in 1999.

Results A dose-response relation was found between exposure to work stressors over 14 years and risk of the metabolic syndrome, independent of other relevant risk factors. Employees with chronic work stress (three or more exposures) were more than twice as likely to have the syndrome than those without work stress (odds ratio adjusted for age and employment grade 2.25, 95% confidence interval 1.31 to 3.85).

Conclusions Stress at work is an important risk factor for the metabolic syndrome. The study provides evidence for the biological plausibility of the link between psychosocial stressors from everyday life and heart disease.

Introduction

Stress at work has been linked with coronary heart disease in retrospective and prospective studies.^{1,2} The biological mechanisms remain unclear.³ Plausible pathophysiological mechanisms involve direct neuro-endocrine effects and indirect effects mediated by adverse health behaviours.

The metabolic syndrome is a cluster of risk factors that increases the risk of heart disease and type 2 diabetes (table 1).⁴ Previous studies have found a social gradient in work stress and the metabolic syndrome, and cross sectional studies have linked work stress with components of the syndrome, but this association is not consistent.

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Table 1 Definition of the metabolic syndrome.* Three risk factors need to be present

Risk factor	Defining level
Abdominal obesity (waist circumference)	
Men	>102 cm
Women	>88 cm
Triglycerides	≥1.69 mmol/l
High density lipoprotein cholesterol	
Men	<1.03 mmol/l
Women	<1.29 mmol/l
Blood pressure	≥130/≥85 mm Hg
Fasting glucose	≥6.11 mmol/l

*From the National Cholesterol Education Program expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel III).⁴

Previous studies also lack information on the duration of exposure to work stress. Here, we report the association between work stress (measured in four phases) and the metabolic syndrome over 14 years of follow-up, and we test the hypothesis of a dose-response association.

Methods

The Whitehall II study recruited participants from 20 civil service departments in London from 1985 to 1988 (phase 1). Surveys consisted of postal questionnaires in 1989 (phase 2), 1991-3 (phase 3, which also included a clinical examination), 1995 (phase 4), and 1997-9 (phase 5, which also included a clinical examination). The phase 4 questionnaire collected no information on work stress or health behaviours. Full details are reported elsewhere.⁵

Variables

Data on the components of the metabolic syndrome were collected at phase 5 during the clinical examination. The syndrome was defined by the presence of three or more risk factors. We used obesity (body mass index >30) at baseline as an indicator of risk for the syndrome.

We defined self reported work stress using the job strain questionnaire⁶ as participants with above the median score for the measures of "job demands" and below the median score for the measures of "job decision latitude" (job control). The iso-strain model of work stress hypothesises that socially isolated (no supportive coworkers or supervisors) high job strain carries the highest risk for heart disease. We defined participants in the lowest third of work social support who reported job strain as having work stress and measured the accumulation of exposure to work stress over the four measurement periods (phases 1, 2, 3, and 5) by adding together the number of times the participant was exposed to iso-strain. We defined chronic work stress as experiencing iso-strain three or more times (>75% of the time) over the 14 year study period.

We measured social position using the relative index of inequality, based on employment grade at baseline. The health behaviours (all self reported) were: current smoking, no daily fruit and vegetable consumption, heavy alcohol consumption (men >4 units/day, women >3 units/day), and no exercise. We summed exposure to these health damaging behav-

aviours over the four measurement periods (phases 1, 2, 3, and 5).

Participants who lacked work stress data or data on a specific health behaviour at any of the phases of data collection were given a missing value.

Analysis

We used logistic regression analysis to assess the odds ratios of the metabolic syndrome for the different explanatory variables and nested logistic regression models to examine the effect of adjusting for different explanatory variables. We excluded 70 patients with non-fatal myocardial infarction at baseline from all the analyses and in some analyses also excluded the 717 obese patients at baseline, to test the causal direction from work stress to the metabolic syndrome and associated obesity. Phase 1 of the Whitehall II study comprised 10 308 civil servants. By phase 5, the participation rate was 75%, taking into account 488 deaths among the 7357 participants at this stage; 5882 had complete clinical data on the indicators of the metabolic syndrome. See bmj.com for details of imputation analysis for missing data.

Participants who were in employment and had never experienced work stress during the 14 years formed the reference group for the work stress measure for both the complete cases and imputed analyses.

Results

In the bivariate analyses of the association between the metabolic syndrome and each of the explanatory variables, age was a risk factor among women but not men (interaction $P < 0.01$). Both men and women from lower employment grades were more likely to have the syndrome. We found a dose-response relation between exposure to job stress and the syndrome (trend $P < 0.05$ for men; $P < 0.01$ for women). Men with chronic work stress (three or more exposures) were nearly twice as likely to develop the metabolic syndrome as those with no exposure to work stress. Women with chronic work stress were over five times more likely to have the metabolic syndrome, but they formed a small group ($n = 18$). The association between the metabolic syndrome and exposure to health damaging behaviours was stronger among men than women (see bmj.com).

In the nested multivariate logistic regression models using imputed data, values for men and women were combined (see bmj.com). When we adjusted for age and employment grade only, greater exposure to work stress was significantly associated with increasing odds of the syndrome. Adjusting for health behaviours did not change the dose-response association (table 2). When we excluded obese men and women at baseline, the linear association between work stress and the metabolic syndrome remained.

The metabolic syndrome showed a social gradient: men and women in the lowest employment grades had more than double the odds of the syndrome than those in the highest grades (odds ratio 2.33, 95% CI 1.38 to 3.93). When we adjusted for work stress, the difference in the log odds between the highest and lowest employment grades was reduced by 11%. Adjusting for health behaviours reduced the social gradient by

Table 2 Odds ratios (95% confidence intervals) of the metabolic syndrome. Multivariate multiple imputation logistic regression models: non-retired men and women in the Whitehall II cohort at phase 5

	Including patients who were obese at baseline			Excluding patients who were obese at baseline	
	No of cases/total	Adjusted for age+employment grade	Adjusted for age+employment grade+health behaviours	No of cases/total	Adjusted for age+employment grade+health behaviours
Men and women:					
No exposures	491/5178	1.00	1.00	388/4881	1.00
1 exposure	134/1253	1.13 (0.70 to 1.82)	1.12 (0.70 to 1.82)	103/1165	1.11 (0.60 to 2.03)
2 exposures	54/383	1.55 (0.85 to 2.85)	1.53 (0.87 to 2.69)	41/356	1.47 (0.74 to 2.92)
≥3 exposures	41/220	2.25 (1.31 to 3.85)	2.39 (1.36 to 4.21)	30/198	2.29 (1.27 to 4.12)
P for linear trend		<0.01	<0.00		0.01
Men:					
No exposures	341/3564	1.00	1.00	281/3407	1.00
1 exposure	95/900	1.11 (0.73 to 1.67)	1.11 (0.73 to 1.69)	77/851	1.12 (0.67 to 1.87)
2 exposures	37/252	1.64 (0.98 to 2.73)	1.57 (0.92 to 2.65)	31/238	1.56 (0.93 to 2.63)
≥3 exposures	32/181	2.01 (0.88 to 4.58)	2.17 (0.92 to 5.09)	24/166	2.04 (0.86 to 4.85)
P for linear trend		0.03	0.03		0.04
Women:					
No exposures	150/1614	1.00	1.00	107/1474	1.00
1 exposure	40/353	1.23 (0.40 to 3.74)	1.27 (0.42 to 3.84)	25/314	1.22 (0.28 to 5.37)
2 exposures	17/131	1.27 (0.34 to 4.83)	1.45 (0.45 to 4.75)	10/118	1.09 (0.15 to 7.94)
≥3 exposures	9/39	3.73 (0.88 to 15.75)	3.72 (0.79 to 17.53)	6/32	4.69 (0.79 to 27.86)
P for linear trend		0.23	0.11		0.26

around 16%. When adjusted for health behaviours and work stress the social gradient was non-significant ($P = 0.07$) (see bmj.com).

Discussion

Greater exposure to job stress over 14 years was linked to greater risk of the metabolic syndrome, in a dose-response manner. The association was robust to adjustment for occupational status and health behaviours. These prospective findings are evidence of the biological plausibility of a causal association between chronic psychosocial stress and the risk of chronic heart disease. Crucially, by excluding participants who were obese at baseline, we showed that pre-existing physiological risk is unlikely to explain the observed association. Furthermore, the social gradient in the metabolic syndrome is partially explained by work stress and a larger proportion is explained by health behaviours.

By characterising work stress exposure with multiple measures, we have conducted an innovative and rigorous study of the cumulative effect of psychosocial adversity on the clustering of coronary risk factors. We took account of smoking habit, physical inactivity, poor diet, and excess alcohol intake with repeated measurements over the course of follow-up. Each of these adverse health behaviours predicted the risk of the metabolic syndrome in men and, less consistently, in women, but there was little evidence that these behaviours mediated or confounded the effect of work stress on the risk of developing the syndrome.

Biology of stress

Prolonged exposure to work stress may affect the autonomic nervous system and neuroendocrine activity directly, contributing to the development of the metabolic syndrome. A case-control study showed that participants in the Whitehall II study with the metabolic syndrome had raised cortisol and normetanephrine output, and also had reduced variability in heart rate.⁷ Decrements in cardiac autonomic function

have been linked to the metabolic syndrome in other populations and to low job control and social isolation among men in the Whitehall II study.⁸⁻¹⁰ Psychobiological studies have also shown that heightened stress reactivity and impaired recovery after stress, assessed by blood pressure and inflammatory markers, predict the five year progression of the metabolic syndrome.¹¹ Chronic psychological stress may reduce biological resilience and thus disturb homeostasis. Altered adrenocortical function can influence hepatic lipoprotein metabolism and insulin sensitivity at target organs.^{11 12} Cortisol is an insulin antagonist, and cortisol output is increased in the metabolic syndrome.⁷ Low concentrations of high density lipoprotein cholesterol and glucose intolerance have been linked with high basal secretion of cortisol.¹³

Limitations

We were not able to analyse incidence because of the lack of data on all metabolic syndrome components at study baseline. However, excluding obese participants at baseline did not change our findings. Although the dropout rate between phases 1 and 5 of this study was high, we used analytical procedures that take account of missing data. The results using imputed data were in line with the results from the analysis of the complete cases. Few women had chronic exposure to work stress, necessitating the analysis of men and women together.

Conclusions

A dose-response association exists between exposure to work stress and the metabolic syndrome. Employees with chronic work stress have more than double the odds of the syndrome than those without work stress, after other risk factors are taken into account. The study provides evidence for the biological plausibility of psychosocial stress mechanisms linking stressors from everyday life with heart disease.

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What is already known on this topic

Stress at work is associated with coronary heart disease, but the biological mechanisms underlying this association are unclear

The metabolic syndrome has a social gradient

What this study adds

A dose-response association exists between exposure to work stress and the metabolic syndrome

The study provides evidence for the biological plausibility of psychosocial stress mechanisms linking stressors from everyday life with heart disease

Part of the social gradient in the metabolic syndrome is explained by chronic exposure to work stress

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Are you a globalist or an analyst?

The theory of learning styles says that, to learn most effectively, we should be aware of our own personal learning style. For example, if you are a globalist then you should look at the overall picture when learning and not get too caught up in minutiae. This seems to make sense, and many people do work out their own learning style: at a recent primary care meeting on e-learning, most of the attendees had worked out their learning style according to the commonly used Honey and Mumford classification system.¹

But is this really a worthwhile exercise? And if so, which learning style theory should you follow? There is more than one classification system that you can use, and many are contradictory or overlap. Indeed, according to research by Coffield and colleagues there are 71 different theories out there,^{2,3} and here are just a few of the possible learning styles—activist or theorist, adaptor or innovator, initiator or reasoner, judge or perceiver, holist or serialist. The researchers bemoaned the “proliferation of concepts, instruments and strategies” and “the bedlam of contradictory claims.”⁴

So there is no shortage of learning styles theories, but what they lack is evidence that they are valid, reliable, and consistent and have an impact on learning and teaching.⁴ All theories state that they are just a guide and that we shouldn't treat them as though they were written in stone. But when they are used in practice, how many learners and teachers tend to “oversimplify and label and stereotype” themselves and others?⁴ In his book, Roger Shank claimed that people don't have learning styles at all but simply have different personalities and that we shouldn't confuse the two.⁴

And what do we think? David Tovey, the editor of *Clinical Evidence*, says that we should “wear our learning theory lightly,” and that sums up our approach well. We worry more about content than style. If you want to learn about learning theory then you could start with David Kaufman's article on applying educational theory in practice.⁵ But if you want to learn about recent advances in the use of opioids for patients with cancer pain or about how to predict pharmacokinetics and pharmacodynamics of drugs in people of different ethnic origin then come and have a look at www.bmjlearning.com.

A third option would be to dispense with all the above and establish your own learning styles theory—a bit like having your own religion. You could perhaps ask people whether they are artisans or partisans. You might even get research funding and be able to prove that your theory is valid and that I am just a cynic.

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