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Psychosocial and biobehavioral characteristics of hypertensive men with elevated atherogenic lipids

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Summary

Epidemiologic studies demonstrated an excess risk of ischemic heart disease (IHD) among individuals who simultaneously exhibit hypertension and elevated atherogenic lipids (coronary high risk (CHR)-status). Yet, relatively little is known about factors which contribute to the development of CHR-status during early and middle adulthood. The present study explores the role of selected biobehavioral and psychosocial factors in explaining CHR-status using data from a prospective 6.5 years investigation of a cohort of 416 middle-aged (40.8 ± 9.6 years) male blue-collar workers. Multivariate logistic regression analysis shows that overweight (odds ratio (o.r.) 4.14), smoking (o.r. 2.19), low promotion prospects at work (o.r. 2.71), competitiveness at work (o.r. 2.79) and feelings of sustained anger (o.r. 5.41) independently contribute to the explanation of CHR-status. Furthermore, the operational definition of CHR-status is validated by estimating its power in predicting IHD incidence in the study cohort. In conclusion, co-manifestation of hypertension and elevated lipids is more likely to exist among those blue-collar men who exhibit distinct work-related characteristics in addition to established biobehavioral risks such as overweight and smoking. In view of the high prevalence of CHR-status and of IHD among men in the lower socio-economic strata this finding is also of interest for health policy.

Key words: Hypertension; Atherogenic lipids; Co-manifestation; Occupational stress; Sustained anger; Overweight; Smoking

Introduction

It is now established that elevated lipids are more prevalent in hypertensives as compared to

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normotensives [1,2]. Furthermore, there is a powerful interaction between these two factors to produce ischemic heart disease (IHD) [3]. The co-manifestation of hypertension and of elevated lipids in an individual defines a coronary high risk status (named here CHR-status). The conditions which contribute to the development of CHR-status during early and middle adulthood are still poorly understood. Although epidemiologic studies identified distinct biobehavioral factors such as overweight, cigarette smoking, alcohol consumption, lack of physical exercise and age, these factors explain only part of the variation in CHR-status [4–6]. A different approach towards explaining CHR-status focusses on the role of sustained autonomic nervous system (ANS) activity [7]. Sustained ANS activity has been implicated in the pathogenesis of some forms of hypertension [8], and it has been shown to elevate atherogenic lipoproteins via enhanced endogenous lipid metabolism [9]. Thus, exposure to conditions which induce sustained ANS activity might increase the probability to simultaneously develop high blood pressure and high blood lipids. This association is expected to persist after controlling for the effects of the biobehavioral risk factors mentioned.

The present study analyses the role of distinct work-related and personal characteristics in explaining CHR-status. These characteristics are assumed to provoke and maintain experiences of sustained emotional arousal (distress) which in turn activate the cardiovascular system via enhanced ANS activity. To identify these work-related and personal characteristics, a theoretical model is needed. This model defines the discrepancy experienced between high effort spent at work (high workload) and low control over long-term rewards (low status control) as a crucial distress-provoking and -maintaining condition [10]. Distressing experiences often result from basic threats to the continuity of a crucial social role. In adult life, this usually implies the occupational role. Most clearly, basic threats are experienced when job termination or job instability occur. However, forced occupational change, downward mobility and lack of promotion prospects produce similar effects. In all these instances, it is difficult to adjust to the threats of basic occupational rewards. We maintain that an imbalance between high workload and low status control is critical in promoting distress-related cardiovascular risks [10,11]. This imbalance is further intensified by the presence of distinct personal characteristics of coping with the demands at work, especially so by a high degree of competitiveness and of work commitment [12]. These coping characteristics are likely to provoke longlasting negative emotional responses such as anger and irritation which in turn contribute to ANS activation.

Blue-collar workers are at special risk of suffering from high workload and low status control [11,12]. At the same time, they are at increased risk of cardiovascular morbidity and mortality [13–17]. An analysis which combines these two arguments in explaining CHR-status is of interest for theoretical and for health policy reasons. In its first part, the paper validates the concept of CHR-status by estimating the relative risk of IHD-incidence from a 6.5 years prospective investigation into the blue-collar group under study. The second part relates to the explanation of CHR-status in this blue-collar group by combining the psychosocial and biobehavioral variables mentioned.

Methods

The study sample

A prospective study of a cohort of 416 middle-aged male (25–55 years; mean: 40.8 ± 9.6) blue-collar workers followed over 6.5 years was performed. The study population was recruited from 3 industrial steel and metal plants in West Germany. The 3 plants were comparable in terms of size, type of production and composition of labour force. Sample criteria were age, occupational status (skilled, semi- or unskilled manual workers, foremen), daytime work and native speakers. Workers with documented coronary events as established by ECG were excluded after baseline screening. The sample was representative of the total labour force of the three plants meeting sample criteria with the exception of a slight underrepresentation of unskilled workers. Four screenings took place over a 6.5 years period of observation. Medical data included blood pressure readings at all occasions, height, weight and resting ECG at the beginning and at the end, and

blood samples during second and third screening. During the observation period a sample loss of 15% occurred. Compared to the initial group, the remaining sample was slightly biased with respect to only 2 out of 34 relevant characteristics examined: workers with high blood pressure and workers with high job instability were more likely to withdraw from follow-up. With respect to the first part of the Results section, the sample size is 314 men. This figure represents complete data available over the 6.5 years period of observation (75% of the initial study sample). The second part of the Results section deals with the explanation of CHR-status at second screening (see below). 356 men participated in this second screening. Data from 11 men were excluded due to unusually high levels of high density lipoprotein (HDL)-cholesterol (above 85 mg/dl). These high levels were assumed to indicate chronic alcohol consumption [18]. In addition, data from 35 men had to be excluded from multivariate analysis due to selective missing data. Thus, complete data for the second part of the Results section are available from 310 men.

Biomedical measures

Blood pressure readings were performed by sphygmanometry according to WHO criteria at standardized diurnal time. Total serum cholesterol and triglycerides were determined enzymatically [19]. Lipoproteins were measured by quantitative lipoprotein electrophoresis (for details see Refs. 20, 21) and by available precipitation techniques for quantification of low density lipoprotein (LDL) and of (HDL)-cholesterol. All analyses were performed blind by the Department of Clinical Chemistry. CHR-status was defined as follows, using data from second screening, i.e. 1 year after study onset: (1) systolic blood pressure (SBP) above or equal to 160 mm Hg and/or diastolic blood pressure (DBP) above or equal to 95 mm Hg. 2. LDL-cholesterol above or equal to 180 mg/dl and/or ratio between LDL/HDL-cholesterol > 4.0. These criteria were applied following epidemiologic evidence [22]. As stated earlier, the validity of this definition of CHR-status is examined by exploring its role in the prediction of IHD incidence in the study sample. IHD incidence (definite or probable lethal or non-lethal acute myocardial infarction (AMI) or sudden cardiac death (SCD) (ICD 410–414)) was assessed according to following criteria: (1) ECG (Minnesota Code evaluated independently by two physicians (cardiologists); in case of discordance (5%) a third physician was asked to give his judgement. (2) History taking according to WHO MONICA Protocol [23] (overt cases resulting almost always in hospital admission where positive enzyme signs were an additional criterium to establish the diagnosis AMI). (3) Official cause of death. Definite AMI was coded if clear ECG-signs and unequivocal additional information (criteria 2 or 3) were present. Probable AMI/SCD was coded if clear ECG signs but no definitely reliable additional information was present (in some of these cases silent AMI may have occurred). Fatal and non-fatal cerebrovascular events and non-cardiovascular causes of death were recorded as well but this information is not analyzed here.

Psychosocial data

Based on the theoretical model summarized above, the following variables defining stressful work characteristics (1 and 2) and critical personal coping characteristics (3 and 4) were analyzed:

(1) Job instability. This measure is composed by 4 variables entering factor analysis. The four underlying dichotomous questions relate to the workers' evaluation and personal experience of job instability within their plant, to the experience of cut-down in personnel and of related increase in workload. Workers with scores ≥ 2 (range 0–4) are defined as suffering from job instability.

(2) Low promotion prospects. This is a composed measure which again results from factor analysis. It is based on answers given to 2 questions. They focus on opportunities and personal experiences of further occupational training and promotion. Workers with a score of 2 (range 0–2) are defined as suffering from low promotion prospects.

The two psychological measures include competitiveness and sustained anger.

(3) Competitiveness. This personal pattern of coping with the demands at work is measured by a subscale of the psychometric scale "need for control" [12]. Degree of individual competitiveness and striving is measured by 6 dichotomous items

defining a unidimensional scale. Workers with scores ≥ 3 (range 0–6) are defined as highly competitive.

(4) Sustained anger. This emotional state is assessed by a 5-point Likert scaled item asking about the intensity of feelings of anger and irritation persisting over the past 12 months. Workers with scores ≥ 3 (range 0–4) are defined as experiencing sustained anger.

Additional variables measuring components of the theoretical model such as work pressure, forced piecework, and status inconsistency were not included in the present analysis as our emphasis is on the search for the most parsimonious model explaining CHR-status. Information on psychosocial and behavioral factors was derived from structured interviews carried out at each screening and from a standardized questionnaire measuring styles of coping with work demands [12]. Information on behavioral risk factors such as smoking, physical exercise, recreation and sleep, and on family history of cardiovascular disease was also obtained from structured interviews. In this analysis, psychosocial data refer to the second screening, as do the biomedical data.

Statistical analysis

Multivariate logistic regression analysis is performed to answer the two questions of this study (predictive role of CHR-status in explaining IHD incidence; prediction of CHR-status by psychosocial and biobehavioral factors). The model fit of the most parsimonious model is tested by the likelihood ratio difference test in a bottom-up procedure [24]. Regression coefficients, standard errors, multivariate odds ratios and the 95% confidence intervals are indicated in respective tables. All calculations were performed on Personal Computers using SPSS-PC [25] and GLIM [26] programs for data analysis.

Results

Validation of coronary high risk (CHR) status

Twenty-one workers (5.9%) suffered from a definite or probable myocardial infarction or cardiac death during the 6.5 years follow-up. Table 1 shows the coronary risk factors (mean and standard deviation (SD) or frequencies (%) in future victims of IHD and in men who remained free from IHD as assessed at baseline (for BP and lipids at second screening). Future IHD victims are significantly older, they have higher systolic and diastolic blood pressure values, and significantly increased total cholesterol and LDL-cholesterol values. Lack of regular physical exercise is more prevalent, but the difference is not significant. Two additional findings are in contrast to our expectation: future IHD victims have a slightly lower body weight (not significant), and

TABLE 1

CORONARY RISK FACTORS (MEAN ± (SD) OR FREQUENCIES (%)) IN FUTURE VICTIMS OF IHD AND IN MEN WHO REMAIN FREE FROM IHD

Variable	Cases ($n = 21$)	Non-cases [a] ($n = 293$)	t-test or χ^2
Age	46.6 ± 7.3	40.7 ± 9.9	2.65 **
BMI (kg/m^2)	26.9 ± 2.5	27.4 ± 3.3	0.57
Syst. blood pressure (mm Hg)	151.1 ± 19.8	140.9 ± 16.6	2.84 **
Diast. blood pressure (mm Hg)	95.3 ± 12.3	89.3 ± 12.1	2.26 *
Total cholesterol (mg/dl)	254.7 ± 25.0	233.0 ± 36.8	2.65 **
LDL-cholesterol (mg/dl)	178.8 ± 24.2	155.7 ± 33.9	3.01 **
HDL-cholesterol (mg/dl)	52.7 ± 9.6	50.6 ± 11.8	0.81
Regular cigarette smoking (≥ 10 cig./day) (%)	28.5	33.7	0.60
Lack of physical exercise (%)	90.0	77.7	1.03

*** $P < 0.001$; ** $P < 0.01$; * $P < 0.05$

[a] Participants with cerebrovascular events ($n = 6$), with non-ischemic heart disease ($n = 7$), with advanced coronary artery disease not meeting criteria of primary endpoint ($n = 21$), and with non-cardiovascular cause of death ($n = 5$) were excluded from analysis.

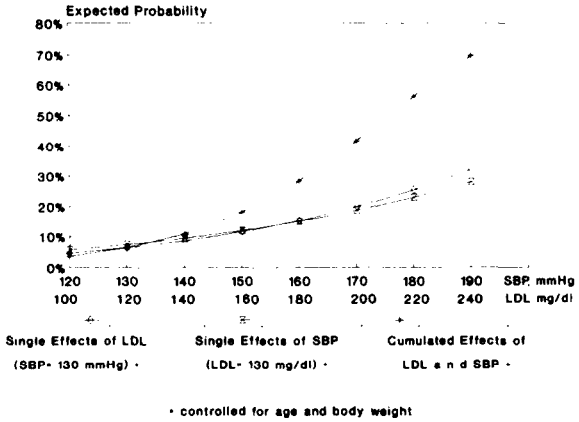

Fig. 1. Single and cumulative effects of systolic blood pressure and LDL-cholesterol on IHD incidence in a logistic regression model ($n = 314$ blue-collar men). Expected probabilities (P) for each point on the curves are calculated as follows:

$$P = \frac{\exp(\text{eta})}{1+\exp(\text{eta})};\ \text{eta} = \beta_0 + \beta_1\chi_1 + \ldots \beta_i\chi_i$$

they do not smoke significantly more cigarettes (for a detailed discussion of these findings see Ref. 11).

To estimate the main effects of high blood pressure and high LDL-cholesterol on IHD-incidence, a multiple logistic regression analysis was performed taking into account "age", and "body weight" as confounding variables. All variables entered the analysis as continuous data. "Cigarette smoking" and "physical exercise", were not included as they were not statistically significant in univariate analysis. In search of the most parsimonious model, the variables "diastolic BP", and "total serum cholesterol" were also excluded. Results are shown in Table 2. As expected, systolic blood pressure and LDL-cholesterol each significantly contribute to the prediction of IHD-incidence after adjusting for the effects of age and body weight.

However, our main argument states that co-manifestation of hypertension and high LDL-cholesterol produces a more powerful effect on IHD-incidence than manifestation of single risk factors. To test this assumption, estimates based on the regression model shown in Table 2 are calculated as follows (see Fig. 1): the lower curve in Fig. 1 estimates IHD probability for those who exhibit normal LDL values, irrespective of their BP values; the mid-curve estimates IHD probability for those who exhibit normal BP values, irrespective of their LDL values whereas the upper curve estimates IHD probability without these restrictions. As demonstrated in Fig. 1, co-manifestation of hypertension and of high lipid levels is associated with a marked increase in the probability of IHD onset.

TABLE 2

LOGISTIC REGRESSION ANALYSIS: FACTORS ASSOCIATED WITH IHD-INCIDENCE ($n = 314$ BLUE-COLLAR MEN)

Variable	Regression coefficient [a]	Multivariate odds ratios [b]	95% CI [b]
Systolic blood pressure	0.03 * (0.01)	2.21	1.06–4.60
LDL-cholesterol	0.02 * (0.01)	2.24	1.17–4.29
Age (years)	0.06 * (0.03)	1.84	1.01–3.35
BMI (kg/m^2)	−0.14 (0.08)	0.72	0.49–1.07
Constant	−8.06 ** (2.75)	LR-$\chi^2 = 133.21$	d.f. = 313

* $P < 0.05$; ** $P < 0.01$

[a] All variables in model; each variable controlled for each other variable. Standard error (SE) of coefficient, is given in parentheses.

[b] Odds ratios and 95% CI refer to the following risk (a) and baseline (b) conditions: systolic blood pressure: (a) 160 mm Hg vs. (b) 130 mm Hg; LDL-cholesterol: (a) 180 mg/dl vs. (b) 130 mg/dl; age: (a) 46 yrs. vs. (b) 36 yrs.; BMI: (a) 27.0 kg/m^2 vs. (b) 24.7 kg/m^2.

The equations applied are as follows:

$$\text{O.R.}\ \exp(\text{eta} * (a-b));\ \text{eta} = \beta_i\chi_i$$

$$95\%\ \text{CI} = \exp(\text{eta} * (a-b))\left[\pm z\sqrt{\text{SE} * (a-b)}\right];\ \text{eta} = \beta_i\chi_i$$

TABLE 3

CORONARY RISK FACTORS (MEAN ± SD OR FREQUENCY (%)) IN THE CORONARY HIGH RISK GROUP (CHR) AND THE REMAINING GROUP (RG)

Variable	CHR ($n = 42$)	RG ($n = 268$)	T-test or χ^2
Age (years)	44.3 ± 7.8	41.6 ± 9.5	1.76
BMI (kg/m^2)	29.7 ± 3.2	27.6 ± 3.3	3.88 ***
Cigarette smoking (≥ 10 cig./day)	38.1%	28.4%	1.21
Job instability	26.2%	17.9%	1.12
Low promotion prospects	83.3%	65.3%	4.61 *
Sustained anger	16.7%	6.0%	4.59 *
Competitiveness (score ≥ 3)	33.3%	15.3%	6.90 **

*** $P < 0.001$; ** $P < 0.01$; * $P < 0.05$.

Prediction of CHR-status by psychosocial and biobehavioral data

Turning to the second part of the Results section, we analyze the role of biobehavioral and psychosocial factors defined above in predicting CHR-status at second screening. 42 men (13.6%) exhibit CHR-status as defined. Table 3 shows mean values and standard deviations or frequency distributions of the biobehavioral and psychosocial factors analyzed among the 2 groups with and without CHR-status. As can be seen, 4 of the 7 variables are significantly more prevalent among CHR individuals in univariate analysis: overweight, low promotion prospects, sustained anger and competitiveness. Two further variables, "smoking" and "job instability", are also more frequent among CHR-members, but the difference is not statistically significant.

No significant correlations are found between biobehavioral variables and the psychosocial variables listed in Table 3 with the exception of "age" and "low promotion prospects". Thus, in a next step, these variables enter logistic regression analysis in search of the most parsimonious model estimating CHR-status. Results of this analysis confirm that the three psychosocial variables "low promotion prospects", "sustained anger", and "competitiveness"predict CHR-status. In addition, "body weight" and "cigarette smoking" significantly contribute to the estimation of CHR-status. As expected, no significant interaction terms are observed between psychosocial and biobehavioral variables. In view of the relatively small sample size the 95% confidence intervals are well acceptable.

Discussion

This paper confirms the powerful role of a co-manifestation of hypertension and of elevated lipids in the prediction of IHD-incidence (see Fig. 1). As coronary high risk status is an important predictor on its own, it is worth while to explore conditions which contribute to its development. Our further results show that CHR-status in the population under study is explained, in addition to established biobehavioral factors, by distinct psychosocial factors derived from a theoretical model. This model predicts effects of enhanced

TABLE 4

LOGISTIC REGRESSION ANALYSIS: FACTORS ASSOCIATED WITH CORONARY HIGH RISK STATUS ($n = 310$ BLUE-COLLAR MEN)

Variable	Regression coefficient [a]	Multivariate odds-ratio	95% CI
Smoking (≥ 10 cig/day)	0.78 * (0.38)	2.19	1.05– 4.59
BMI (≥ 27 kg/m^2)	1.42 ** (0.44)	4.14	1.74– 9.86
Job instability	0.43 (0.42)	1.54	0.68– 3.49
Low promotion prospects	1.00 * (0.46)	2.71	1.11– 6.63
Sustained anger	1.69 ** (0.55)	5.41	1.85–15.83
Competitiveness	1.03 ** (0.39)	2.79	1.29– 6.04
Constant	−4.36 *** (0.61)	LR-$\chi^2 = 211.03$	$d.f. = 303$

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$

[a] All variables in model; each variable controlled for each other variable. SE of coefficient is given in parentheses.

ANS activity on cardiovascular pathology in terms of a discrepancy between high effort spent at work and low job rewards obtained [11]. The data collection in this study did not include a systematic assessment of dietary saturated fat intake. Theoretically, it is possible that fat intake is a confounder of the 4 psychosocial variables measured. Yet, this is highly unlikely for a number of reasons. First, dietary saturated fat intake is usually associated with overweight whereas the psychosocial variables in our study are not. Secondly, all workers lived in a highly homogeneous rural region with low geographical and social mobility, and with a traditional blue-collar lifestyle. Most workers were married, and the majority of them were offered identical meals on the job. Moreover, in a recent study on changes in life expectancy in Japan it was shown that cardiovascular risk factors are explained by socio-economic and work-related factors independent of changes in nutrition [27]. Thus, an interpretation of the findings in terms of stress physiology seems to be justified [7,9] and it is consistent with similar results obtained from independent socio-epidemiologic studies [28,29]. The possible confounding effect of alcohol intake related to lipids and blood pressure is a further point deserving discussion. As indicated, we excluded a subgroup of workers suspected of heavy alcohol consumption from this analysis. But no valid information on moderate alcohol intake was available in the present study. Therefore, we cannot exclude a possible effect of moderate alcohol consumption on the reported association between psychosocial conditions and CHR-status.

The validity of reported findings is explored by a series of additional analyses. First, we repeated all analyses by using information on the psychosocial and biobehavioral predictors from first screening. This is important for 2 reasons, (a) to check the temporal stability of the predictors, and (b) to minimize a possible confounding effect between predictors and outcome measures due to simultaneous assessment (i.e., second screening). Results are not shown in detail but they indicate that the variables exert comparable, although less strong effects on CHR-status. A second test of validity relates to a re-analysis of data based on a slightly different definition of CHR-status. In this definition, low HDL-cholesterol level (< 50 mg/dl) was included in addition to the two criteria mentioned. Resulting in a slightly different composition of the CHR group, the findings of this analysis nevertheless very closely resemble those reported in Tables 3 and 4. Third, we explored the possible confounding effect of antihypertensive and antihyperlipidemic treatment as one might expect an influence of treatment status on the association reported between psychosocial factors and CHR-status. Based on interview data on type of drug therapy and compliance with regimen we excluded all workers who were on regular antihypertensive or antihyperlipidemic medication from a re-analysis. On the basis of a considerably smaller sample size of untreated workers ($n = 230$) multivariate logistic regression analysis was repeated. Interestingly the model was again highly significant, and the beta coefficients of the statistically relevant factors shown in Table 4 were again significant. Thus, treatment status does not seem to affect reported results. Fourth, the role of psychosocial factors in explaining separately the prevalence of hypertension and elevated lipids was explored in contrast to the combined analysis of the two risk factors. Single psychosocial factors were found to be significantly related to hypertension and elevated lipids respectively, but it was only with respect to CHR-status that at least three of the four theoretically meaningful psychosocial variables exerted significant effects. Finally, we have now evidence that 4 psychosocial factors derived from our theoretical model independently and significantly contribute to the estimation of IHD onset in this cohort: "job insecurity", "status inconsistency" (two indicators of high status control), "high work pressure" and "high need for control" (two indicators of high workload) [11]. These findings strengthen the validity of the psychosocial measurements of this study.

In conclusion, CHR-status defines an elevated risk of future IHD incidence among middle-aged blue-collar men, and its prevalence is closely linked to indicators of sustained ANS activity related to the working life. These latter effects are independent of the effects of established biobehavioral variables such as overweight, age, cigarette smoking or physical exercise. If replicated by independent studies, the health policy implications of

these findings for primary prevention in cardiovascular health are clearly of interest.

References

1 MacMahon, S.W., Mac Donald, G.J. and Blacket, R.B., Plasma lipoprotein levels in treated and untreated hypertensive men and women, Atherosclerosis, 5 (1985) 391.

2 Management Committee of the Australian National Blood Pressure Study. Prognostic factors in the treatment of mild hypertension, Circulation, 69 (1984) 668.

3 Castelli, W.P. and Anderson K.A., A Population at risk, Am. J. Med., 80 (Suppl. 2A) (1986) 23.

4 Pooling Project Research Group, Relationship of blood pressure, serum cholesterol, smoking habit, relative weight and ECG-abnormalities to incidence of major coronary events: Final Report of The Pooling Project, J. Chron. Dis., 31 (1978) 201.

5 Tyroler, H.A., Heyden, S. and Hames C.G., Weight and hypertension: Evans County studies of Blacks and Whites. In: Paul (Ed.), Epidemiology and Control of Hypertension, Stratton, New York and London, 1975, p. 176.

6 Heyden, S., Borhani, N.O., Tyroler, H.A., Schneider, K.A., Langford, H.G., Hames, C.G., Hutchinson, R. and Overman, A., The relationship of weight change to changes in blood pressure, serum uric acid, cholesterol and glucose in the treatment of hypertension, J. Chron. Dis., 38 (1985) 281.

7 Beamish, R.E., Singall, P.K. and Dhalla, N.S. (Eds.), Stress and Heart Disease, M. Nijhoff, Boston, Dordrecht, 1985.

8 Henry, J.P. and Meehan, J.P., Psychosocial stimuli, physiological specificity, and cardiovascular disease. In: H. Weiner, M.A. Hofer and A.J. Stunkard (Eds.), Brain, Behavior and Bodily Disease, Raven Press, New York, 1981, p. 305.

9 Snyder, F. (Ed.), Lipid Metabolism in Mammals, Vol. 1, Plenum Press, New York, NY, 1977.

10 Siegrist, J. and Matschinger, H., Restricted status control and cardiovascular risk. In: A. Steptoe and A. Appels (Eds.), Stress, Personal Control and Health, Wiley, Chichester, 1989, p. 65.

11 Siegrist, J., Peter, R., Junge, A., Cremer, P. and Seidel, D., Low status control, high effort at work and ischemic heart disease: prospective evidence from blue-collar men. Soc. Sci. Med., 31 (1990) 1127.

12 Matschinger, H., Siegrist, J., Siegrist, K., and Dittmann, K.H., Type A as a coping career. In: Th. Schmidt, T.M. Dembroski and G. Bluemchen (Eds.), Biological and Psychological Factors in Cardiovascular Disease, Springer, Berlin, Heidelberg, New York, 1986, p. 104.

13 Karasek, R.A. and Theorell, T., Healthy Work. Basic Books, New York, 1990.

14 Marmot, M.G., Shipley, M. and Rose, G., Inequalities in death – specific explanation of a general pattern? Lancet, 1 (1984) 1003.

15 Holme, I., Helgeland, A., Hjermann, J. and Seren, P., Socio-economic status as a coronary risk factor: The Oslo Study, Acta Med. Scand., Suppl. 660 (1982) 147.

16 Buring, J.E., Evans, D.A., Fiore, M., Rosner, B. and Henneken, C.H., Occupation and risk of death from coronary heart disease, J. Am. Med. Assoc., 258 (1987) 791.

17 Tyroler, H.A. and Haynes, S.G., Task force 1: Environmental risk factors in coronary heart disease, Circulation, 76 (Suppl. 1) (1987) 1139.

18 Seidel, D. and Cremer, P., Guidelines for the clinical evaluation of lipoprotein profiles. In: A.M. Gotto and R.Paoletti, (Eds.), Atherosclerosis Reviews, Raven Press, New York, 1987, Vol. 14, p. 61.

19 Cremer, P., Seidel, D. and Wieland, H., Quantitative Lipoproteinelektrophorese, Lab. Med., 9 (1985) 39.

20 Wieland, H. and Seidel, D., Quantitative lipoprotein electrophoresis. In: Lewis, L.A. (Ed.), Handbook of Electrophoresis III, CRC Press, Boca Raton, FL, 1983, p. 83.

21 Wieland, H. and Seidel, D., A simple and specific technique for precipitation of low-density lipoproteins, J. Lip. Res., 24 (1983) 904.

22 Kannel, W.B., Doyle, J.B., Ostfeld, A.M., Jenkins, C.D., Culler, L., Podell, R.N. and Stamler, J., Optimal resources for primary prevention of atherosclerotic diseases, Circulation, 29 (1984) 157A.

23 World Health Organization (Ed), MONICA Manual, Geneva, 1986.

24 Efron, B., The efficiency of logistic regression compared to normal discriminant analysis, J. Am. Stat. Assoc., 70 (1975) 892.

25 Norusis, M.J., SPSS-X Advanced Statistics Guide. McGraw Hill, New York, 1985.

26 Nelder, J.A. and Wedderburn, R.W.M., Generalized linear models, J. Nat. Statist. Soc. Am., 135 (1972) 370.

27 Marmot, M. and Smith, G., Why are the Japanese living longer? Br. Med. J., 299 (1989) 1547.

28 Siegrist, J., Bernhardt, R., Feng, Z. and Schettler, G., Socio-economic differences in cardiovascular risk factors in China, Int. J. Epidem., 19 (1990) 905.

29 Siegrist, J., Matschinger, H., Cremer, P. and Seidel, D., Atherogenic risk in men suffering from occupational stress, Atherosclerosis, 69 (1988) 211.