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Poor structural social support is associated with an increased risk of Type 2 diabetes mellitus: findings from the MONICA/KORA Augsburg cohort study

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What's new?

- Poor structural social support compared with good structural social support in middle-aged men is associated with an increased risk of Type 2 diabetes mellitus.
- The association is independent of known risk factors at baseline and is particularly evident in men with a low level of education.
- The assessment of structural social support may be considered in the risk prediction of Type 2 diabetes mellitus in men.
- Future research should investigate pathophysiological pathways and focus on gender differences as well as examine the association between social support and incidence of Type 2 diabetes mellitus in terms of the functional and structural dimensions in more detail.

Abstract

Aims Several psychosocial factors have been shown to increase the risk of Type 2 diabetes mellitus. This study investigated the association between structural social support and incidence of Type 2 diabetes mellitus in men and women.

Methods Data were derived from three population-based MONICA/KORA surveys conducted in 1984–1995 in the Augsburg region (southern Germany) and followed up by 2009. The study population comprised 8952 participants (4669 men/4283 women) aged 30–74 years without diabetes at baseline. Structural social support was assessed using the Social Network Index. Sex-specific hazard ratios were estimated from Cox proportional hazard models.

Results Within follow-up, 904 incident Type 2 diabetes mellitus cases (558 men, 346 women) were observed. Crude incidence rates for Type 2 diabetes mellitus per 10 000 person-years were substantially higher in poor compared with good structural social support (men: 94 vs. 69, women: 58 vs. 43). After adjustment for age, survey, parental history of diabetes, smoking

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status, alcohol intake, physical activity, hypertension, dyslipidaemia, BMI, education, sleep complaints and depressed mood, risk of Type 2 diabetes mellitus for participants with poor compared with good structural social support was 1.31 [95% confidence interval (CI) = 1.11–1.55] in men and 1.10 (95% CI = 0.88–1.37) in women. Stratified analyses revealed a hazard ratio of 1.50 (95% CI = 1.23–1.83) in men with a low level of education and 0.87 (95% CI = 0.62–1.22) in men with a high level of education (*P* for interaction: 0.0082).

Conclusions Poor structural social support is associated with Type 2 diabetes mellitus in men. This association is independent of risk factors at baseline and is particularly pronounced in men with a low level of education.

Introduction

Beyond genetic predisposition and increasing age as non-modifiable conditions, several somatic and behavioural risk factors have been identified in the development of Type 2 diabetes mellitus, including obesity [1], low physical activity [2], smoking [3] and hypertension [4]. In addition, research has made great efforts to investigate whether psychosocial factors are related to the onset of Type 2 diabetes mellitus. Recent studies found that depression [5,6] and post-traumatic stress disorders [7] as well as burnout [8], high job-strain [9], sleep disorders [10] and perceived mental stress [11] are associated with an increased risk of Type 2 diabetes mellitus.

There is also some preliminary evidence suggesting that social support has an impact on subsequent Type 2 diabetes mellitus development. Studies showed that single indicators of social support such as low emotional support in women [9] and living alone in men [12] are associated with an increased risk of Type 2 diabetes mellitus.

Social support refers to a coping resource provided by relationships with significant others

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including family members, friends, co-workers and club members. Two dimensions of social support should be distinguished, namely functional and structural support. Functional support refers to the aid and encouragement provided to the individual by the social network, whereas structural support describes the characteristics of the network of people surrounding an individual and his/her interactions within this network. This distinction between ‘functional’ and ‘structural’ social support is essential as the term ‘social support’ is commonly used synonymous with functional aspects of social relations [13,14].

In contrast to the aforementioned preliminary evidence for functional and structural social support increasing the risk of Type 2 diabetes mellitus, a recent meta-analysis investigating the association of social support and subsequent coronary heart disease showed that low functional, but not structural, social support had a statistically significant impact on cardiac mortality [13].

Given the preliminary, as well as conflicting evidence, we aimed to examine whether poor structural social support is associated with an increased risk of subsequent Type 2 diabetes mellitus development in a large population-based study of people aged 30–74 years. To investigate whether there are sex-specific particularities, all analyses were performed separately for men and women.

Patients and methods

Setting, design and study population

The presented data were derived from the population-based Monitoring of Trends and Determinants in Cardiovascular Disease Augsburg (MONICA) studies, conducted in 1984–1995 [15]. Three independent cross-sectional surveys covering the region of Augsburg were carried out in 1984/1985, 1989/1990 and 1994/1995. Altogether 13 426 people aged 25–

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74 years participated in at least one of the surveys. Incident cases of Type 2 diabetes mellitus were assessed within the framework of Cooperative Health Research in the Region of Augsburg (KORA) using follow-up questionnaires in 1987/1988, 1997/1998, 2002/2003 and 2009 [16]. Only participants aged 30–74 years at baseline were included in the current analyses ($n = 12\,027$). Participants with prevalent Type 2 diabetes mellitus or with other types of diabetes at baseline ($n = 572$) were excluded leading to a sample of 11 455 participants. Furthermore, participants without information on diabetes status at follow-up ($n = 624$) or with incomplete data on all covariables required for the main analyses (1879) were excluded from the cohort leading to a final study population of 8952 participants (4669 men and 4283 women). Written informed consent was obtained from each study participant and the study was approved by the local ethics committee.

Assessment of structural social support

Structural social support was assessed using the Social Network Index (SNI) initially designed for the Alameda county study [17] comprising household status, the number and frequency of contacts with relatives or friends one feels close to as well as informal and formal group associations. The SNI comprises not only the number of social ties, but also their relative importance. Intimate contacts are weighed more heavily than church affiliations or group memberships. The SNI is divided into four categories (1 = least connected, 4 = most connected). Because these four categories showed crossing Kaplan–Meier survival curves, they were grouped into two categories (categories 1 and 2 as the category of poor structural social support; categories 3 and 4 as the category of good structural social support). Of 2114 men with poor structural social support, 474 had an SNI score of 1 and 1640 men had an SNI score of 2. The group of 2218 women with poor structural social support was made up of 662 women with an SNI score of 1 and 1556 women with an SNI score of 2.

Ascertainment of Type 2 diabetes mellitus

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Incident cases of Type 2 diabetes mellitus were assessed in the follow-up questionnaires. All incident cases, which had been diagnosed up to 31 December 2009 were included. Self-reported Type 2 diabetes mellitus and the date of diagnosis were validated by hospital records or by contacting the participants' treating physicians. In addition, the hospital records of those deceased during the follow-up period without a diagnosis of Type 2 diabetes mellitus at baseline were examined and/or their last treating physicians were contacted. Thus, only clinically diagnosed Type 2 diabetes mellitus cases were included in the analyses [18].

Assessment of sociodemographic, behavioural, clinical and psychosocial risk factors

Baseline information on sociodemographic variables, smoking habits, alcohol consumption, physical activity level, parental history of diabetes, medication use, sleep complaints and depressed mood was obtained in standardized personal interviews conducted by trained medical staff and a self-administered questionnaire. In addition, all participants underwent an extensive standardized medical examination including the collection of a blood sample. All measurement procedures have been described elsewhere in detail [18,19].

Sociodemographic risk factors

Level of education was estimated by counting years of schooling completed. Participants were classified to have a low educational status if they completed less than 12 years of schooling.

Behavioural risk factors

Information on smoking habits was provided by the participants (never, past only, occasional, or regular). Assessment of alcohol intake was based on questionnaire data regarding weekday and weekend consumption of beer, wine and spirits. Each participant was questioned regarding leisure-time physical activity during winter and summer. Study participants were classified as physically active if they exercised in both, summer and winter, and on average

≥ 1 h/week throughout the year.

Clinical risk factors

Study participants were coded to have a parental history of diabetes if they stated that at least one of their parents had diabetes. BMI was calculated as weight in kg divided by height in m². Actual hypertension was defined as blood pressure values ≥ 140/90 mmHg and/or use of an antihypertensive medication given that the participants were aware of being hypertensive. Dyslipidaemia was defined as a ratio of total cholesterol to high-density lipoprotein cholesterol ≥ 5.0.

Psychosocial risk factors

Sleep complaints were items regarding difficulties initiating sleep and difficulties maintaining sleep and were adopted from the Uppsala Sleep Inventory [20]. Participants were coded as having sleep complaints if they stated to have at least one of these difficulties often. Depressed mood was assessed using a subscale from the von Zerssen affective symptom checklist [21]. The depression and exhaustion subscale (DEEX) combines eight items (fatigue, tiredness, irritability, loss of energy, difficulty concentrating, inner tension, nervousness, anxiety) ranging from 0 to 3, leading to a Likert-like scoring range of 0–24 [22]. Participants in the top tertile of the depressive symptom distribution stratified by sex were considered as suffering from depressed mood.

Statistical analyses

All analyses were performed separately for men and women. Means and proportions of baseline sociodemographic, behavioural, clinical and psychosocial characteristics were computed for participants with poor or good structural social support at baseline. Differences were tested using the chi-square test. Means were compared based on the *t*-test, in case of

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unequal variances the Satterthwaite estimate was used.

Different Cox proportional hazard models were calculated to estimate the effect of structural social support on the risk of Type 2 diabetes mellitus expressed by the hazard ratio (HR). We formed categories for continuous variables and used the reference cell coding as parameterization method to model nonlinear risk relations. The first model included structural social support as exposure (poor structural social support versus good structural social support) as well as the risk factors age (categorical: 30–74 years in 5-year steps) and survey (84/85, 89/90, 94/95). The second model additionally included smoking status (regular, irregular), alcohol intake (categorical men: 0 g/d, 0.1–39.9 g/d, ≥ 40 g/d; categorical women: 0 g/d, 0.1–19.9 g/d, ≥ 20 g/d), physical activity (active, inactive), parental history of diabetes (positive, negative), BMI (categorical: < 25 kg/m², 25–30 kg/m², > 30 kg/m²), actual hypertension (yes, no) and dyslipidaemia (yes, no). The third model included all previous factors plus level of education (low, high), sleep complaints (yes, no) and depressed mood (yes, no). To examine interactions between structural social support and each of the 12 covariates, interaction terms were tested using the Wald chi-square statistics. In case of statistically significant interactions, the Cox regression analyses were additionally stratified by the relevant variable.

The assumption of proportional hazards was assessed graphically by checking the log(–log(survival)) curves for parallelism. No severe deviations from parallelism were evident. The assumption of linear association with continuous variables was tested by plotting each continuous predictor variable against the Martingale residuals from a Cox proportional hazard model that excluded the predictor. Nonlinear risk relations were observed for age, alcohol intake and BMI. Multicollinearity among covariates was examined by computing the variance inflation factors [23]. A variance inflation factor score > 2.5 was considered to indicate multicollinearity, which was not observed among the covariates.

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As sensitivity analyses, all multivariable Cox proportional hazard models were repeated only for participants who had been followed for at least 2 years before being diagnosed with Type 2 diabetes mellitus, in order to exclude possibly undiagnosed cases at baseline.

Two-tailed P -values < 0.05 were considered to be statistically significant except for interaction analyses where $P < 0.15$ was defined as indicating interactions. In the case of multiple testing, the significance level alpha was corrected based on the Bonferroni method. All statistical analyses were performed using SAS (v. 9.3, SAS Institute Inc., Cary, NC, USA).

The analyses and description in this article follow the STROBE guidelines for observational cohort studies [24].

Results

Baseline risk factors

The baseline risk factors of the study sample of 8952 participants (4669 men, 4283 women) varied considerably between participants with poor and good structural social support with $P < 0.004$ (corrected alpha = $0.05/13 = 0.004$), especially in women.

In both sexes, participants with poor structural social support were significantly older, more often physically inactive, consumed less alcohol and reported more likely depressed mood than participants with good structural social support. In addition, women with poor structural social support were more likely regular smokers, hypertensive, had more likely dyslipidaemia, a low level of education and reported more likely sleep complaints than women with good structural social support. By contrast, men with poor structural social support did not differ from men with good structural social support in smoking status, actual hypertension, dyslipidaemia, level of education and sleep complaints.

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In both sexes, no significant differences were seen with regard to parental history of diabetes and BMI between participants with poor and good structural social support (Table 1).

Incidence of Type 2 diabetes mellitus

During a mean follow-up period of 15.5 years (SD 6.2 years), a total of 904 incident cases of Type 2 diabetes mellitus (558 men, 346 women) were observed between 1984 and 31 December 2009. Incidence of Type 2 diabetes mellitus was higher in participants with poor structural social support than in participants with good structural social support for both sexes. The crude incidence rates of Type 2 diabetes mellitus amounted to 94/10 000 person-years in men with poor structural social support compared with 69/10 000 person-years in men with good structural social support. The corresponding incidence rates for women were 58/10 000 person-years and 43/10 000 person-years. Figures 1 and 2 show the Kaplan–Meier survival curves stratified by poor and good structural social support for men and women. Participants with good structural social support had higher probabilities of living without a diagnosis of Type 2 diabetes mellitus. According to the log-rank tests, these differences were statistically significant in both men ($P < 0.001$) and women ($P = 0.003$).

Multivariable analyses

As shown in Table 2, poor structural social support significantly increased the risk of subsequent Type 2 diabetes mellitus development in both men and women in unadjusted models. After adjustment, structural social support had a significant effect on incidence of Type 2 diabetes mellitus in men but not in women. When adjusting for age and survey (Model 1), the risk of developing Type 2 diabetes mellitus for men with poor structural social support compared with men with good structural social support was 1.29 [95% confidence interval (CI) = 1.09–1.52]. The corresponding HR for women was 1.10 (95% CI = 0.89–1.37). When additionally adjusting for smoking status, alcohol intake, leisure-time physical activity,

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parental history of diabetes, BMI, actual hypertension and dyslipidaemia (Model 2), men with poor structural social support had a relative risk of 1.30 (95% CI = 1.10–1.54) for developing Type 2 diabetes mellitus compared with men with good structural social support. The corresponding HR for women was 1.10 (95% CI = 0.88–1.38). Further adjustment for level of education, sleep complaints and depressed mood (Model 3) slightly altered the effects, which however, remained significant in men but non-significant in women.

The majority of interactions between structural social support and one of the 12 covariates did not reach statistical significance with $P > 0.0125$ (corrected alpha = $0.15/12 = 0.0125$). A statistically significant interaction was only seen with level of education in men ($P = 0.0082$). The stratified survival analyses shown in Table 3 revealed that the effect of structural social support on Type 2 diabetes mellitus risk was stronger and significant only in men with < 12 years of education. In this subpopulation, men with poor structural social support had an unadjusted relative risk of 1.54 (95% CI = 1.27–1.84) for developing Type 2 diabetes mellitus compared with men with good structural social support. When adjusting for age and sex (Model 1), the HR was slightly attenuated to 1.53 (95% CI = 1.26–1.86). Additional adjustment for smoking status, alcohol intake, leisure-time physical activity, parental history of diabetes, BMI, actual hypertension and dyslipidaemia (Model 2), as well as sleep complaints and depressed mood (Model 3) resulted in a HR of 1.50 (95% CI = 1.24–1.83) and 1.50 (95% CI = 1.23–1.83), respectively. The corresponding HR in the subpopulation of men with a high level of education ranged from 0.98 in Model 1 to 0.87 in Model 3, but did not reach statistical significance.

Sensitivity analysis

In sensitivity analysis, the HR and P -values for structural social support were robust when

excluding all participants with < 2 years of follow-up (67 cases, 69 non-cases). No severe deviations from the main analyses were observed in men (Model 3: HR = 1.29, 95% CI = 1.08–1.54) and women (Model 3: HR = 1.13, 95% CI = 0.90–1.43).

Discussion

Overall findings

This study demonstrated that poor structural social support compared with good structural social support is associated with an increased risk of Type 2 diabetes mellitus in middle-aged men from the general population. This association was independent of known risk factors at baseline and was particularly evident in men with < 12 years of education.

Scientific context

Recent studies investigating the effect of structural social support on Type 2 diabetes mellitus confirm these findings. A previous investigation in the same population by Meisinger *et al.* [12] found that men living alone were more likely to develop Type 2 diabetes mellitus than men not living alone, independent of common risk factors at baseline. The reported HR of 1.89 (95% CI = 1.33–2.70) exceeded the HR in this study, possibly indicating that living alone as a single indicator for social support has a strong effect on Type 2 diabetes mellitus risk. However, this effect is slightly attenuated when additionally taking into account other indicators of social support, i.e. the number of friends and relatives one feels close to, frequency of contacts as well as group affiliations.

A recent prospective study carried out in Sweden [25] showed that women with prediabetes, aged 50–64 years who lived alone also had an increased risk to progress to Type 2 diabetes mellitus. However, this association was mainly explained by known factors associated with

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Type 2 diabetes mellitus.

Strodl and Kenardy [26] investigated the effect of various psychosocial and non-psychosocial variables on subsequent self-reported first diagnosis of diabetes in 10 300 women aged 70–74 years. They found that not having a partner was significantly related to a first diagnosis of diabetes over a 3-year period. After adjustment, however, the effect became insignificant.

The results of this study extend prior knowledge. First, structural social support was measured using an index of various indicators, taking into account that household status, contacts with friends or relatives and group associations need to be considered jointly and that intimate contacts should be weighed more heavily than group affiliations to support conclusions about structural social support [27]. Second, the observations in the current population cohort indicated that the effect of structural social support on subsequent Type 2 diabetes mellitus development in men was significantly modified by level of education. In addition, the index used in this study may also be seen as an indicator of functional social support. Because intimate ties are given more weight than group affiliations, the SNI may indirectly allow for tentative conclusions about the functional dimensions of social support as well.

Potential pathophysiological pathways

This study was not designed to identify the underlying mechanisms that may lead to an independent association of structural social support and Type 2 diabetes mellitus risk. An assessment of potential causal pathways in this association is beyond the scope of this analysis. However, the following potential pathophysiological pathways may be considered and investigated in further studies.

A low level of structural social support may arouse anxiety and lead to augmented stress [28].

It is well known that stress leads to an activation of the hypothalamic-pituitary-adrenal axis,

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which results in elevated secretion of glucocorticoids from the adrenal cortex and increased circulating glucose concentrations [29]. Chronic stress conditions such as poor structural social support may lead to a prolonged activation and a progressive dysfunction of the hypothalamic-pituitary-adrenal axis, which finally could contribute to the development of Type 2 diabetes mellitus [29,30].

In addition, stress has been associated with an activation of the sympathoadrenal system. Chronic release of catecholamines accompanied by inflammatory reactions may alter sensitivity to insulin and also result in Type 2 diabetes mellitus [12].

Strengths and limitations

Our study has several strengths, primarily its population-based prospective design, its large sample size, the long follow-up and its availability of data on health-related behaviour, mental health and additional risk factors for Type 2 diabetes mellitus. Furthermore, all assessments were standardized as well as quality-controlled and all self-reported incident Type 2 diabetes mellitus cases were scrutinized by examination of hospital records or contacting the participant's treating physician.

The study also has some limitations. First, all characteristics including structural social support were measured at baseline only. Consequently, changes in these characteristics throughout the follow-up period were not available and misclassification bias cannot be excluded. Second, information on diabetes status at follow-up was missing for 624 participants who therefore were not included in the analyses, which may lead to a potential bias. Also, it is possible that some of the participants without diabetes did in fact have undetected diabetes. Third, confounding by unmeasured variables such as nutrition parameters or job strain cannot entirely be excluded. These variables may also contribute to

the effect modification by level of education in men.

Implications

The assessment of structural social support may be considered in the risk prediction of Type 2 diabetes mellitus in men. Future studies should investigate possible pathophysiological pathways, especially regarding different effects in terms of sex and level of education and whether these findings are due to different stress reactions or confounding by unmeasured risk factors. Furthermore, future research should examine in more detail differences in the association between social support and incident Type 2 diabetes mellitus in terms of the functional and structural dimension.

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Competing interests

None declared.

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FIGURE 1. Kaplan–Meier survival curves stratified by structural social support for men (log-rank test: $P < 0.001$).

FIGURE 2. Kaplan–Meier survival curves stratified by structural social support for women (log-rank test: $P = 0.003$).

Table 1. Means (\pm SD) and prevalences (%) of baseline risk factors according to baseline structural social support

	Men (<i>N</i> = 4669)			Women (<i>N</i> = 4283)		
	Poor structural social support (<i>N</i> = 2114)	Good structural social support (<i>N</i> = 2555)	<i>P</i>	Poor structural social support (<i>N</i> = 2218)	Good structural social support (<i>N</i> = 2065)	<i>P</i>
Age (years)	50.3 (11.9)	48.6 (11.8)	< 0.001*	50.3 (11.6)	45.7 (10.6)	< 0.001*
Education < 12 years (<i>N</i> , %)	1374 (65.0)	1592 (62.3)	0.058	1824 (82.2)	1597 (77.3)	< 0.001*
Positive parental history of diabetes (<i>N</i> , %)	407 (19.3)	468 (18.3)	0.415	482 (21.7)	467 (22.6)	0.487
Regular smoking (<i>N</i> , %)	626 (29.6)	673 (26.3)	0.013	453 (20.4)	315 (15.3)	< 0.001*
Alcohol intake (<i>N</i> , %):						
0 g/d	395 (18.7)	374 (14.6)	< 0.001*	965 (43.5)	755 (36.6)	< 0.001*
0.1–19.9 g/d (women)	1049 (49.2)	1274 (49.9)		817 (36.8)	859 (41.6)	
0.1–39.9 g/d (men)						
\geq 20 g/d (women)	670 (31.7)	907 (35.5)		463 (19.7)	451 (21.8)	
\geq 40 g/d (men)						
Active during leisure time (<i>N</i> , %)	838 (39.6)	1262 (49.4)	< 0.001*	691 (31.2)	1028 (49.8)	< 0.001*
BMI (kg/m ²)	27.1 (3.6)	27.3 (3.4)	0.034	26.2 (4.7)	25.9 (4.5)	0.025
Actual hypertension (<i>N</i> , %)	974 (46.1)	1155 (45.2)	0.553	749 (33.8)	562 (27.2)	< 0.001*
Dyslipidaemia (<i>N</i> , %)	1012 (47.9)	1161 (45.4)	0.097	416 (18.8)	289 (14.0)	< 0.001*
Sleep complaints (<i>N</i> , %)	424 (20.1)	440 (17.2)	0.013	627 (28.3)	441 (21.4)	< 0.001*
Depressed mood (<i>N</i> , %)	878 (41.5)	887 (34.7)	< 0.001*	930 (41.9)	689 (33.4)	< 0.001*

*Statistically significant (corrected according to Bonferroni method *P*-values < 0.004 were defined as statistically significant).

Table 2. Sex-specific HR, 95% CI and *P*-values for developing Type 2 diabetes mellitus according to baseline structural social support

	Men			Women		
	HR	95% CI	<i>P</i>	HR	95% CI	<i>P</i>
Model 0*	1.37	1.16–1.62	< 0.001 ¶	1.37	1.11–1.70	0.004 ^e
Model 1†	1.29	1.09–1.52	0.003¶	1.10	0.89–1.37	0.379
Model 2‡	1.30	1.10–1.54	0.002¶	1.10	0.88–1.38	0.399
Model 3§	1.31	1.11–1.55	0.002¶	1.10	0.88–1.37	0.416

*Model 0: unadjusted.

†Model 1: adjusted for age and survey.

‡Model 2: adjusted for age, survey, parental history of diabetes, smoking status, alcohol intake, leisure time physical activity, BMI, actual hypertension and dyslipidaemia.

§Model 3: adjusted for age, survey, parental history of diabetes, smoking status, alcohol intake, leisure time physical activity, BMI, actual hypertension, dyslipidaemia, level of education, sleep complaints and depressed mood.

¶ Statistically significant.

Table 3. HR, 95% CI and *P*-values for developing Type 2 diabetes mellitus according to baseline structural social support in men stratified by level of education

	Men < 12 years education			Men ≥ 12 years education		
	HR	95% CI	<i>P</i>	HR	95% CI	<i>P</i>
Model 0*	1.54	1.2P–1.86	< 0.001 ¶	0.98	0.71–1.37	0.921
Model 1†	1.53	1.26–1.86	< 0.001 ¶	0.98	0.71–1.37	0.923
Model 2‡	1.50	1.24–1.83	< 0.001 ¶	0.88	0.63–1.24	0.463
Model 3§	1.50	1.23–1.83	< 0.001 ¶	0.87	0.62–1.22	0.418

*Model 0: unadjusted.

†Model 1: adjusted for age and survey.

‡Model 2: adjusted for age, survey, parental history of diabetes, smoking status, alcohol intake, leisure-time physical activity, BMI, actual hypertension and dyslipidaemia.

§Model 3: adjusted for age, survey, parental history of diabetes, smoking status, alcohol intake, leisure-time physical activity, BMI, actual hypertension, dyslipidaemia, sleep complaints and depressed mood.

¶ Statistically significant.

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