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ARTICLE

Stress resilience and subsequent risk of type 2 diabetes in 1.5 million young men

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Abstract
Aims/hypothesis Psychosocial stress in adulthood is associated with a higher risk of type 2 diabetes, possibly mediated by behavioural and physiological factors. However, it is unknown whether low stress resilience earlier in life is related to subsequent development of type 2 diabetes. We examined whether low stress resilience in late adolescence is associated with an increased risk of type 2 diabetes in adulthood.
Methods We conducted a national cohort study of all 1,534,425 military conscripts in Sweden during 1969–1997 (97–98% of all 18-year-old men nationwide each year) without prior diagnosis of diabetes, who underwent standardised psychological assessment for stress resilience (on a scale of 1–9) and were followed up for type 2 diabetes identified from outpatient and inpatient diagnoses during 1987–2012 (maximum attained age 62 years).
Results There were 34,008 men diagnosed with type 2 diabetes in 39.4 million person-years of follow-up. Low stress resilience was associated with an increased risk of developing type 2 diabetes after adjusting for BMI, family history of diabetes, and individual and neighbourhood socioeconomic factors (HR for lowest vs highest quintile: 1.51; 95% CI 1.46, 1.57; $p < 0.0001$), including a strong linear trend across the full range of stress resilience ($p_{\mathrm{trend}} < 0.0001$). This association did not vary by BMI level, family history of diabetes or socioeconomic factors.
Conclusions/interpretation These findings suggest that low stress resilience may play an important long-term role in aetiological pathways for type 2 diabetes. Further elucidation of the underlying causal factors may help inform more effective preventive interventions across the lifespan.

Keywords Psychological resilience · Psychological stress · Type 2 diabetes mellitus

Abbreviations

CDC	Centers for Disease Control and Prevention
HPA	Hypothalamic–pituitary–adrenal
SES	Socioeconomic status

Introduction

Type 2 diabetes prevalence has more than doubled among US adults over the past three decades and now exceeds 8% [1], and may reach one-third by 2050 [2]. Obesity and physical inactivity are well-established risk factors, but less is known about psychosocial effects on disease risk. Some evidence has suggested that traumatic life events [3], job strain [4], general emotional stress [5–7] and anxiety or depression [8, 9] are associated with a higher risk of developing type 2 diabetes. The underlying mechanisms may involve both behavioural and stress-related physiological factors. Psychosocial stress may contribute to unhealthy lifestyle behaviours that are known risk factors for diabetes, such as poor dietary habits, physical inactivity, smoking or alcohol abuse [5, 10]. Chronic stress also activates the hypothalamic–pituitary–adrenal (HPA) axis resulting in increased cortisol levels that may

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contribute to abdominal obesity [11, 12] and cytokine-mediated immunologic responses that may be involved in mediating insulin resistance [13]. As a result, resilience to stress may be expected to be protective against type 2 diabetes. However, to our knowledge, no studies have examined whether stress resilience early in life is related to the long-term risk of developing type 2 diabetes. Such knowledge may provide new insights into psychosocial pathways for diabetes and help inform more effective preventive strategies.

We conducted the first study to examine stress resilience in late adolescence in relation to type 2 diabetes risk in adulthood. Stress resilience was measured using standardised psychological assessments in ~1.5 million 18-year-old male military conscripts in Sweden during 1969–1997, who were subsequently followed up to a maximum age of 62 years. Our aim was to determine whether low stress resilience in late adolescence is associated with a higher risk of developing type 2 diabetes in a large national cohort.

Methods

Study population We identified 1,547,478 men (aged 18 years) who underwent a military conscription examination during 1969–1997. This examination was compulsory for all 18-year-old men nationwide each year except for 2–3% who were either incarcerated or had severe chronic medical conditions or disabilities documented by a physician. We excluded all 13,053 (0.8%) individuals who had a prior diagnosis of diabetes identified from hospital discharge diagnoses. A total of 1,534,425 (99.2% of the original cohort) remained for inclusion in the study. This study was approved by the Regional Ethics Committee of Lund University in Sweden.

Stress resilience ascertainment The Swedish Military Conscription Registry contains information from a 2 day standardised physical and psychological examination administered annually to all Swedish military conscripts starting in 1969. Stress resilience was assessed using a 20–30 min semi-structured interview administered by a trained psychologist [14]. The overall objective of the interview was to assess the ability to cope with psychological requirements of military service, including stress resilience during armed combat. In the interview, the psychologist asked about adjustment problems and conflicts, as well as successes, responsibilities taken on, and initiatives shown or experienced in school, work, home or in leisure activities [14]. Emotional stability, social maturity and active/passive interests were rated by the psychologist, who then assigned a summary score on a 'standardised nine' (1 to 9) scale, which is constructed to have a normal distribution with a mean of 5 and SD of 2. A validation study in which 30 recorded interviews from 1972 to 1973 were scored by 30 psychologists reported high interrater reliability (correlation 0.86) [15]. Low stress resilience using these data has previously been examined in relation to other outcomes, including coronary heart disease [16], stroke [17] and peptic ulcer disease [18].

Type 2 diabetes ascertainment The study cohort was followed up through 31 December 2012 for type 2 diabetes, which was identified using ICD diagnosis codes in the Swedish Hospital Registry and Swedish Outpatient Registry. The Swedish Hospital Registry contains all primary and secondary hospital discharge diagnoses from six populous counties in southern Sweden starting in 1964, and with nationwide coverage starting in 1987; and the Swedish Outpatient Registry contains outpatient diagnoses nationwide starting in 2001. Earlier ICD versions did not distinguish between type 1 and type 2 diabetes; therefore, we ascertained type 2 diabetes using ICD-9 code 250 (excluding codes 250.X1 and 250.X3) during 1987–1996 (www.icd9data.com/2007/Volume1) and ICD-10 code E11 during 1997–2012 (www.who.int/classifications/icd/en/). A sensitivity analysis was performed that further included all diabetes diagnoses during 1969–1986 using ICD-8 code 250 in hospital discharge records (before outpatient data were available), of which the majority are expected to be type 2 based on inpatient data after this period.

Adjustment variables Other variables that may be associated with the risk of type 2 diabetes were obtained from the Swedish Military Conscription Registry and national census data, which were linked using an anonymous personal identification number. The following were used as adjustment variables: year of the military conscription examination (modelled simultaneously as a continuous and categorical [1969–1979, 1980–1989, 1990–1997] variable); BMI (weight in kg/height in m^2; modelled alternatively as a continuous or categorical variable using Centers for Disease Control and Prevention [CDC] definitions for children and adolescents aged 2 to 19 years to facilitate comparability with US studies: overweight is defined as ≥85th and <95th percentile and obesity as ≥95th percentile on the CDC's 2000 sex-specific BMI-for-age growth charts, which correspond to BMI ≥25.6 and <29.0 and BMI ≥29.0, respectively, for 18-year-old men [19]); family history of diabetes in a parent or sibling (yes or no, identified from medical diagnoses in the Swedish Hospital Registry from 1964 to 2012 and the Swedish Outpatient Registry from 2001 to 2012, not self-reported, thus enabling unbiased ascertainment); highest attained education level during the study period (<12, 12–14, ≥15 years); and neighbourhood socioeconomic status at baseline (SES, included because neighbourhood characteristics have been associated with type 2 diabetes [20] and with psychosocial stress [21]; comprised of an index that includes low education level, low income, unemployment and social welfare receipt, as previously described [22], and categorised as low [>1 SD below the mean], medium [within 1 SD from the mean] or high [>1 SD above the mean]).

A secondary analysis was performed that further adjusted for depression and anxiety, which were ascertained using ICD diagnosis codes in the Swedish Hospital Registry and Swedish Outpatient Registry (depression: ICD-7 301.1, ICD-8/9 296.2–296.3, ICD-10 F32–33; anxiety: ICD-7 310, ICD-8/9 300.0, ICD-10 F40–41), and modelled as time-dependent variables (older ICD codes are available at www.wolfbane.com/icd/).

Missing data for each variable were imputed using a standard multiple imputation procedure based on the variable's relationship with all other covariates [23]. Missing data were relatively infrequent for stress resilience (7.2%), BMI (7.2%), education level (0.4%) and neighbourhood SES (9.1%). Data were complete for all other variables. As an alternative to multiple imputation, sensitivity analyses were performed after restriction to individuals with complete data for all variables (n=1,327,760; 86.5%).

Statistical analysis Cox proportional hazards regression was used to compute HRs and 95% CIs for the association between stress resilience level and subsequent risk of type 2 diabetes. The Cox model time scale was elapsed time since the military conscription examination (which also corresponds to attained age because baseline age was the same [18 years] for all conscripts). Individuals were censored at emigration (n=86,400; 5.6%) or death (n=58,835; 3.8%). Stress resilience was modelled alternatively as a categorical variable (1 to 9) or an ordinal variable to test for trend. Two different adjusted models were performed: the first was adjusted for year of the military conscription examination, and the second was further adjusted for BMI, family history of diabetes, education level and neighbourhood SES (as defined above). The proportional hazards assumption was assessed by examination of $\log_e$–$\log_e$ plots and was met in all models. Likelihood ratio tests were used to assess for first-order interactions between stress resilience and the model covariates in relation to type 2 diabetes risk. All statistical tests were two-sided and used an α-level of 0.05. All analyses were conducted using Stata version 13.0 (StataCorp, College Station, TX, USA).

Results

Among the 1,534,425 men in this cohort, 34,008 (2.2%) were subsequently diagnosed with type 2 diabetes in 39.4 million person-years of follow-up (average follow-up of 25.7 years). The median age at the end of follow-up was 46.1 years (mean 45.9 years, SD 8.9, range 19.0 to 62.0 years). The median age at diagnosis of type 2 diabetes was 46.8 years (mean 44.7 years, SD 9.9, range 18.0 to 62.0 years).

Stress resilience Figure 1 shows the distribution of stress resilience scores (on a scale of 1–9) in men who did or did not develop type 2 diabetes. Low stress resilience was associated with an increased risk of developing type 2 diabetes (Table 1). In the fully adjusted model, men in the lowest quintile of stress resilience had a 1.5-fold risk of type 2 diabetes relative to those in the highest quintile (adjusted HR, 1.51; 95% CI 1.46, 1.57). There was a highly significant linear trend in the risk of type 2 diabetes across the full range of stress resilience ($p_{trend}<0.0001$). Figure 2 shows the adjusted HRs and 95% CIs for type 2 diabetes across the full range of stress resilience, relative to men with average resilience as the reference group. A steep slope of increasing risk is seen for men with below-average stress resilience, whereas above-average resilience is slightly protective relative to the mean.

Other risk factors High BMI was the strongest risk factor for development of type 2 diabetes. Obese men (≥95th percentile on the CDC's 2000 sex-specific BMI-for-age growth chart) had more than a sixfold risk, and overweight men (≥85th and <95th percentile) had more than a threefold risk, relative to those with normal BMI (Table 1, adjusted model 2). A first-degree family history of diabetes was associated with more than a twofold risk of developing type 2 diabetes (Table 1). High education was associated with lower risk ($p_{trend}<0.0001$). In the fully adjusted model, neighbourhood SES had a backward J-shaped relationship with type 2 diabetes risk, with highest risk corresponding to low neighbourhood SES and a modestly increased risk with high neighbourhood SES, relative to the intermediate group (Table 1).

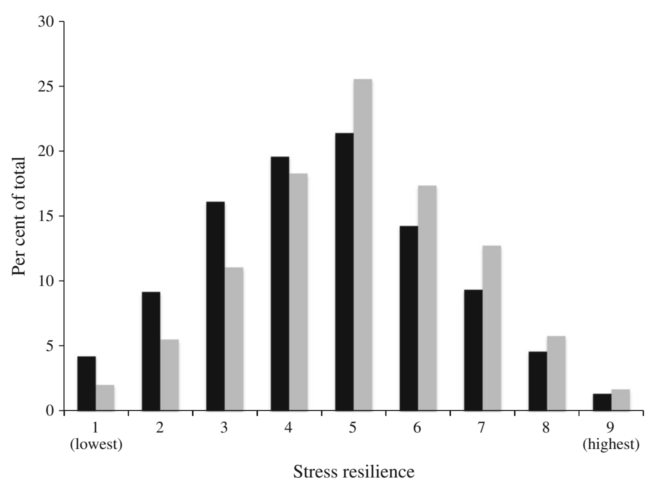

Fig. 1 Stress resilience (on a scale of 1–9) in 18-year-old men who did or did not develop type 2 diabetes in adulthood. Black bars, type 2 diabetes; grey bars, no type 2 diabetes

Table 1 Adjusted HRs for associations between stress resilience or other factors in 18-year-old men and the risk of type 2 diabetes in adulthood

Variable	Type 2 diabetes		Adjusted model 1[a]			Adjusted model 2[b]		
	No (n = 1,500,417)	Yes (n = 34,008)	HR	95% CI	p value	HR	95% CI	p value
Stress resilience								
1 (lowest)	30,183 (2.0)	1,433 (4.2)	2.31	2.18, 2.45	<0.001	1.89	1.78, 2.00	<0.001
2	82,704 (5.5)	3,120 (9.2)	1.90	1.82, 1.98	<0.001	1.54	1.48, 1.61	<0.001
3	166,019 (11.1)	5,482 (16.1)	1.55	1.50, 1.61	<0.001	1.34	1.29, 1.39	<0.001
4	274,475 (18.3)	6,659 (19.6)	1.25	1.21, 1.30	<0.001	1.15	1.11, 1.19	<0.001
5 (reference)	383,401 (25.5)	7,278 (21.4)	1.00			1.00		
6	260,528 (17.4)	4,847 (14.3)	0.95	0.92, 0.99	0.01	0.98	0.95, 1.02	0.39
7	191,159 (12.7)	3,178 (9.3)	0.85	0.81, 0.89	<0.001	0.93	0.89, 0.97	<0.001
8	86,750 (5.8)	1,556 (4.6)	0.84	0.80, 0.89	<0.001	0.97	0.91, 1.02	0.23
9 (highest)	25,198 (1.7)	455 (1.3)	0.78	0.71, 0.86	<0.001	0.91	0.82, 1.00	0.05
Per higher category (trend)			0.86	0.85, 0.86	<0.001	0.91	0.90, 0.91	<0.001
Lowest vs highest quintile			1.98	1.91, 2.05	<0.001	1.51	1.46, 1.57	<0.001
BMI								
Normal	1,388,856 (92.6)	25,918 (76.2)	1.00			1.00		
Overweight	79,952 (5.3)	4,522 (13.3)	3.78	3.66, 3.90	<0.001	3.35	3.25, 3.46	<0.001
Obese	31,609 (2.1)	3,568 (10.5)	8.42	8.12, 8.73	<0.001	6.66	6.43, 6.91	<0.001
Per 1 BMI unit (trend)			1.10	1.09, 1.10	<0.001	1.10	1.09, 1.10	<0.001
Family history of diabetes								
No	1,164,670 (77.6)	18,251 (53.7)	1.00			1.00		
Yes	335,747 (22.4)	15,757 (46.3)	2.52	2.47, 2.58	<0.001	2.20	2.15, 2.25	<0.001
Education (years)								
<12	225,154 (15.0)	8,247 (24.2)	1.14	1.11, 1.17	<0.001	1.04	1.01, 1.06	0.02
12–14	662,094 (44.1)	16,177 (47.6)	1.00			1.00		
≥15	613,169 (40.9)	9,584 (28.2)	0.59	0.57, 0.60	<0.001	0.73	0.71, 0.75	<0.001
Per higher category (trend)			0.71	0.70, 0.72	<0.001	0.84	0.82, 0.85	<0.001
Neighbourhood SES								
Low	231,265 (15.4)	7,864 (23.1)	1.43	1.39, 1.47	<0.001	1.30	1.27, 1.34	<0.001
Medium	988,613 (65.9)	21,166 (62.2)	1.00			1.00		
High	280,539 (18.7)	4,978 (14.6)	0.98	0.94, 1.01	0.12	1.13	1.09, 1.16	<0.001
Per higher category (trend)			0.81	0.79, 0.82	<0.001	0.90	0.88, 0.92	<0.001

[a] Adjusted for year of military conscription examination

[b] Adjusted for year of military conscription examination, BMI, family history of diabetes, education and neighbourhood SES. The reference category for all variables is indicated by an HR of 1.00

There was no evidence of statistical or biologically meaningful interaction between stress resilience and BMI, family history, education level or neighbourhood SES in relation to type 2 diabetes risk. For example, among men with normal BMI, overweight or obesity, respectively, the adjusted HRs for lowest vs highest quintile of stress resilience were 1.51 (95% CI 1.45, 1.57; $p < 0.001$), 1.50 (95% CI 1.36, 1.66; $p < 0.001$) and 1.52 (95% CI 1.33, 1.73; $p < 0.001$) ($p_{\text{interaction}} = 0.17$). Further adjustment for depression ($n = 54{,}553$; 3.6%) and anxiety ($n = 58{,}926$; 3.8%) also had minimal effect on the results (e.g. adjusted HR for lowest vs highest quintile of stress resilience: 1.49; 95% CI 1.44, 1.54; $p < 0.001$). In sensitivity analyses that included diabetes diagnoses from 1969–1986 (for which type 1 and type 2 could not be distinguished), or that were restricted to individuals without any missing data, all risk estimates were very similar to the main results (data not shown).

Discussion

In this large national cohort study, we found that low stress resilience in 18-year-old men was associated with an increased risk of developing type 2 diabetes during an average follow-up of ~25 years, independently of BMI at baseline,

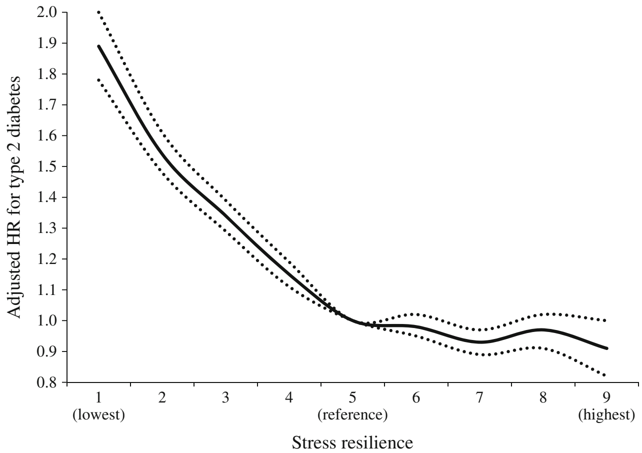

Fig. 2 Adjusted HRs and 95% CIs for the association between stress resilience in 18-year-old men and the risk of type 2 diabetes in adulthood (adjusted for year of military conscription examination, BMI, family history of diabetes, education and neighbourhood SES). Solid line, HR; dotted lines, 95% CI

family history or socioeconomic factors. These findings suggest that psychosocial function and ability to cope with stress may play an important long-term role in aetiological pathways for type 2 diabetes. Additional studies will be needed to elucidate the specific underlying causal factors, which may help inform more effective preventive interventions across the lifespan.

To our knowledge, this is the first study to examine stress resilience in late adolescence in relation to diabetes risk in adulthood. Most previous studies have focused on stressful experiences later in adulthood in relation to diabetes. For example, a cross-sectional study of 2,262 Dutch middle-aged adults reported that major stressful life events (e.g., death of a family member) in the past 5 years was associated with a higher risk of type 2 diabetes and with an increased waist-to-hip ratio [3]. A pooled cohort study of 124,808 European adults reported that job strain was associated with a modestly higher risk of type 2 diabetes among both men and women [4]. Other studies have reported that general emotional stress [5], feelings of anger [6], hostility [7], and anxiety or depression [8, 9] are risk factors for type 2 diabetes. A Swedish study of 237,980 military conscripts (a subset of the present study cohort) found that low stress resilience was associated with a modestly increased risk of coronary heart disease, but did not examine diabetes or other risk factors for heart disease [16].

High BMI, which was measured at age 18 years, was the strongest risk factor for diabetes in this cohort. The risk estimates that we observed (more than sixfold and threefold risk for obese and overweight men, respectively) are consistent with previously reported estimates for BMI measured in adulthood [24]. The more than twofold risk we observed among men with a family history of diabetes, and the inverse relationship between education level and diabetes risk, are also in agreement with previous findings [25, 26]. In addition, we found that low neighbourhood SES was associated with a higher risk of type 2 diabetes, consistent with previously reported associations between neighbourhood characteristics and diabetes risk, including studies of neighbourhood deprivation and built environment in Sweden [20].

The mechanisms by which stress resilience may influence the development of type 2 diabetes are probably complex and involve unhealthy lifestyle behaviours as well as other physiological factors. A Danish cohort study of 7,066 adults with 10 years of follow-up found that self-reported perceived stress was associated with subsequent physical inactivity and unsuccessful smoking cessation or alcohol reduction among men and women, and development of diabetes among men [5]. Anxiety and depression are also associated with physical inactivity, unhealthy diet and smoking [10]. In addition, hormonal and immunological factors that are involved in stress reactions may play a role in the pathogenesis of type 2 diabetes. Many forms of stress can activate the HPA axis, resulting in increased synthesis and release of cortisol, which contributes to lipolysis, body fat redistribution favouring visceral adiposity and insulin resistance [11]. Chronic stress also activates the innate immune system, resulting in increased levels of interleukin 6 and other cytokine mediators of the acute-phase response, which are involved in mediating insulin resistance and are strong predictors of type 2 diabetes [13]. Additional studies with longitudinal behavioural and physiological measurements are needed to delineate the most critical factors and windows of susceptibility across the lifespan.

The strengths of this study include its large national cohort design with prospective ascertainment of stress resilience and type 2 diabetes. This study was the first with sufficient follow-up to examine stress resilience in late adolescence in relation to diabetes in adulthood. Stress resilience was assessed using systematic interviews by trained psychologists with high interrater reliability. The national cohort design prevented selection bias, and the use of prospectively obtained registry data prevented bias that may result from self-reporting. We were able to adjust for other established risk factors for type 2 diabetes, including BMI, family history and socioeconomic factors, which were also prospectively ascertained and not self-reported.

Limitations include a lack of information on certain other diabetes risk factors, such as smoking and diet. The association we observed between low stress resilience and type 2 diabetes is not necessarily causal and potentially may be explained by unmeasured confounders. The study was based on Swedish military conscripts; therefore, the cohort consisted entirely of men, and it is uncertain how the findings would compare in women. A much smaller Danish cohort study reported that perceived stress was associated with unhealthy behaviours among both men and women, but with diabetes only among men, although the sex-specific CIs were wide and overlapped [5]. The present cohort was also relatively young (median attained age 46 years). Additional follow-up to older ages will be needed whenever feasible in this or other cohorts. In addition, outpatient diagnoses in the present study were available starting in 2001, and hence type 2 diabetes prior to this period was under-reported. This under-reporting is expected to be nondifferential with respect to stress resilience level, and therefore to influence results toward the null hypothesis.

In summary, this large national cohort study is the first to examine stress resilience in late adolescence in relation to diabetes risk in adulthood. We found that low stress resilience is associated with a higher risk of developing type 2 diabetes. Further elucidation of the underlying pathways may help improve preventive strategies by addressing important psychosocial factors across the lifespan.

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Duality of interest The authors declare that there is no duality of interest associated with this manuscript.

Contribution statement CC, JS, MAW and KS made substantial contributions to study conception and design; analysis and interpretation of data; critical revision for important intellectual content; and approved the final version. In addition, JS and KS acquired the data, and CC drafted the manuscript. JS is the guarantor of this work.

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