Burnout and Risk of Type 2 Diabetes: A Prospective Study of Apparently Healthy Employed Persons

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Objective: This prospective study was designed to test the extent to which the onset of type 2 diabetes in apparently healthy individuals was predicted by burnout, a unique affective response to combined exposure to chronic stressors. **Methods:** The study participants were 677 employed men and women who were followed up for 3 to 5 years (mean = 3.6 years) for the onset of diagnosed type 2 diabetes. Burnout was assessed by the Shirom-Melamed Burnout Measure with its three subscales: emotional exhaustion, physical fatigue, and cognitive weariness. **Results:** The burnout symptoms were remarkably consistent over the follow-up period irrespective of changes in place of work and in employment status. During the follow-up period, 17 workers developed type 2 diabetes. Logistic regression results indicated that burnout was associated with a 1.84-fold increased risk of diabetes (95% confidence interval [CI] = 1.19–2.85) even after adjusting for age, sex, body mass index, smoking, alcohol use, leisure time physical activity, initial job category, and follow-up duration. In a subsample of 507 workers, the relative risk of diabetes was found to be much higher after additional control for blood pressure levels (odds ratio = 4.32, 95% CI = 1.75–10.67), available only for this subsample. **Conclusions:** These findings suggest that chronic burnout might be a risk factor for the onset of type 2 diabetes in apparently healthy individuals. **Key words:** type 2 diabetes, stress, burnout.

CI = confidence interval; **CVD** = cardiovascular disease; **SMBM** = Shirom-Melamed Burnout Measure; **HbA1c** = glycosylated hemoglobin A1c; **VE** = vital exhaustion; **MI** = myocardial infarction; **MBI** = Maslach Burnout Inventory; **BMI** = body mass index; **SBP** = systolic blood pressure; **DBP** = diastolic blood pressure; **OR** = odds ratio; **APR** = acute phase response; **CRP** = C-reactive protein; **HDL** = high-density lipoprotein; **HPA** = hypothalamic–pituitary–adrenal.

INTRODUCTION

Type 2 diabetes, a complex disorder characterized by impaired secretion of insulin and increased resistance to insulin, is associated with a two- to four-fold increased risk of coronary heart disease and a four-fold increase in mortality from coronary heart disease (1) as well as increased risk of peripheral vascular disease, renal failure, and blindness (2,3). The past 2 decades have witnessed an explosive increase in the number of people diagnosed with diabetes worldwide, particularly with type 2 diabetes (4–6).

It has been suggested that stress plays a significant role in the etiology of type 2 diabetes (7). In animal studies, stressful situations have been shown to induce hyperglycemia (8), but only a small number of studies have systematically tested the response in human beings (9). Moreover, most of the studies that have examined the proposition were cross-sectional in design (e.g., (10)) focused on stressful life events and the onset of type 1 diabetes and yielded inconclusive results (for a review of these studies, see (11–13)). Even fewer studies have explored the link between work-related stress and the development of type 2 diabetes. Some have shown that the risk of developing type 2 diabetes is higher in certain occupations (14,15). Other studies that focused on working hours have yielded conflicting results (16,17). Only two cross-sectional studies have directly examined the association between job strain (gauged by a combination of high psychological demand and low decision latitude) and clinically diagnosed type 2 diabetes, and they obtained mixed results: negative results were obtained in the first study (18) and low decision latitude was associated with the risk of type 2 diabetes in the second study but not high demands (19). Therefore, the hypothesis that work-related stress is causally implicated in the etiology of diabetes awaits further elucidation.

An inherent problem in exploring the possible link between chronic stress at work and the risk of diabetes is deciding which stressor to focus on given the multitude of chronic stresses to which the individual might be simultaneously exposed. Examples of chronic stresses at work might include overload, within- or cross-role conflicts, injustice, inequity, uncertainty, under-reward, threats of regular physical abuse, ambiguity, job insecurity, job complexity, structural constraints, and sexual harassment (20). Furthermore, the stress literature covers other types of stresses, besides chronic ones, including daily hassles, sudden traumas, and critical life events of the type shown in past research to be related to the onset of diabetes (10,13). It has been suggested that these types may be placed on a continuum that reflects their relative discreteness and time boundaries (21). Most studies designed to predict health outcomes have focused on a specific subset of stresses. We suggest, however, that focusing on burnout, representing as it does the depletion of energetic coping resources as a result of prolonged combined exposure to chronic work and life stresses, offers a promising alternative research strategy that could complement the frequently used strategy of focusing on subsets of stresses.

The purpose of the present study was to explore the possibility of an association between the risk of type 2 diabetes and burnout, a unique affective multidimensional response to stress, the core components of which are emotional exhaustion, physical fatigue, and cognitive weariness (22,23). Burnout, thus defined, does not overlap with related affective dysfunctions such as depression and anxiety (24–27). Moreover, it is conceptually distinct from a temporary state of fatigue that passes after a period of rest. Although there is compelling evidence from longitudinal studies to suggest that

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chronic stressors at work and burnout are reciprocally related over time (e.g., (28–31)), burnout is conceptually and empirically distinct from chronic stress. The empiric support for this argument is based on two meta-analytic studies that have investigated the relations between burnout and chronic stress (32,33) and found low to moderate metacorrelations between overall burnout and the chronic stresses, mostly ranging in the 0.30s.

Attesting to its chronic nature, other studies have demonstrated that the phenomenon of burnout exhibits remarkable stability over time regardless of sample makeup, cultural context, and length of time of the follow-up survey. The cross-time (diachronic) correlations were found to range from 0.50 to 0.60 even with a time interval extending up to 8 years (for review, see (34,35)).

Recently accumulated evidence suggests that burnout has a negative impact on physical health and may be considered a risk factor for physical morbidity and bodily disorders. It has been found to be associated with cardiovascular disease (CVD) risk (36,37) and risk factors for CVD such as atherogenic lipid profile (38,39), sleep disturbances (40,41), impaired fertility (42), musculoskeletal pain, even after controlling for job strain and other possible confounding variables (43), and poor self-rated health (44–46). For further elaboration on the possible mechanisms of the link between burnout and physical morbidity and bodily disorders, particularly CVD, see Melamed et al.'s review (35).

There are some indications that burnout might be associated with the risk of type 2 diabetes. Using the Shirom-Melamed Burnout Measure (SMBM), Melamed et al. (38) found a positive correlation between burnout and adjusted serum glucose levels. Applying the same measure of burnout, Grossi et al. (40) found that it was associated with elevated glycosylated hemoglobin A1c (HbA1c) among women, independent of confounders, including depression. HbA1c measures average glycemic levels over a time scale of weeks, whereas plasma glucose varies greatly on a given day and from day to day and is thus a more accurate and stable measure than fasting blood glucose levels (47). However, HbA1c is recommended not as a diagnostic test for diabetes, but rather to monitor the effectiveness of glycemic therapy (48). In a study of apparently healthy men, Raikkonen et al. (49) found that a cluster of certain characteristics related to yet another measure of burnout, vital exhaustion (VE, see (50)), which has been used to gauge feelings of excessive tiredness, lack of energy, irritability, demoralization, and hostility, were linked to the development of insulin resistance syndrome, a risk factor for type 2 diabetes. All studies were cross-sectional in nature, and none of them examined the association between burnout and diagnosed type 2 diabetes. However, in a case–control cross-sectional study, Wijman and her colleagues (51) found that burnout (as gauged by the Maslach Burnout Inventory [MBI]) did not differentiate between those with diabetes and apparently healthy individuals. This present study is the first attempt to examine the association between burnout and the incidence of type 2 diabetes in a follow-up study of apparently healthy people.

METHODS

Participants

Study participants (N = 1183) were all apparently healthy employees pooled together from two samples: one sample consisted of workers (n = 887) who underwent an onsite health checkup at their workplace conducted by the National Institute of Occupational and Environmental Health (NIOEH), Raanana; the second comprised workers (n = 296) who participated in a study, conducted by the same institute, on job strain and burnout. Baseline and follow-up data were collected during the years 1998 to 2003. Three to 5 years (mean = 3.6 years) after the baseline measures were taken, 901 (76.2%) workers were located and asked to complete a follow-up demographic and medical questionnaire. Eight hundred forty-five (93.8%) agreed to take part in the follow-up study. One hundred fifty-one subjects were excluded from the study for having chronic diseases at the baseline (hypertension, coronary heart disease, diabetes, stroke, and cancer), and 26 subjects had missing data on one or more of the study variables. The final sample consisted of 677 subjects (76.5% men). Their mean age was 42.6 years (standard deviation [SD] = 9.56); they had on average 13.3 years of education (SD = 3.19). We systematically checked for nonresponse bias and found that nonparticipants did not differ from participants on any of the sociodemographic or biomedical variables.

Procedure

The study was approved by the local NIOEH ethics committee. At time 1, all participants completed a sociodemographic and medical questionnaire. For those workers who had undergone a medical checkup, the data on blood pressure levels obtained in these checkups was also used in the present study. These levels had been measured three times, in a sitting position, after 10 minutes of rest. The second and third measures were averaged and used as the baseline measure in our study. Only 507 subjects had blood pressure data. The follow-up questionnaire was either handed to study participants at their workplace or mailed to them if they were no longer gainfully employed. Confidentiality was assured, and each participant signed a written informed consent statement.

Measures

Both the baseline and the follow-up questionnaires covered background information, occupational data, the burnout measure, information on health habits and physical morbidity factors. The occupational data included job description, which served as a basis for classification into five job categories: senior management position, middle management or supervisory position, professionals (e.g., engineers, laboratory technicians, teachers, and computer workers), nonprofessional workers, and self-employed persons. In the follow-up questionnaire, the respondents also had to note changes in place of work and changes in employment status. Based on these data, the participants were classified into four categories: working in the same workplace, have changed workplace, retired, and unemployed at the time of follow-up. Health habits included smoking, alcohol use, and leisure time physical activity.

Incidence of Type 2 Diabetes

The occurrence of new cases of type 2 diabetes was based on self-reports of diagnosed and treated diseases. The use of self-reported diabetes (and of other chronic diseases) as an indicator of health status is a common practice in many epidemiologic studies and surveys (52–57). Reliable evidence indicates that there is substantial to excellent agreement between self-reported diabetes and medical record data, in-person interview data, or clinical diagnosis, with kappa coefficient values ranging from 0.76 to 0.85 (58–61) and specificity over 95% (59,62,63). Also included in the follow-up questionnaire were other chronic diseases such as hypertension, myocardial infarction (MI), stroke, and cancer.

Burnout Measure

Burnout was measured by the 14 items of the SMBM, which comprises three subscales: emotional exhaustion (e.g., "I feel like my emotional batteries are dead"), physical fatigue (e.g., "I am physically exhausted"), and cognitive weariness ("My thinking process is slow" (64)). Items were scored on a 7-point frequency scale, ranging from 1 = almost never to 7 = almost always. The mean score across the 14 items was used. The actual range of the initial burnout score was 1.00 to 6.92. The reliability coefficient (Cronbach's alpha) for the SMBM was 0.91. A series of studies confirmed expected relationships between the SMBM and physiological variables thus substantiating its construct validity (35). In these studies, respondents' total score on the SMBM was used to predict risk factors for cardiovascular disease (38,39), quasiinflammatory factors in the blood (64), salivary cortisol levels (41), upper respiratory infections (65), and inflammation biomarkers (66). The psychometric characteristics of the SMBM have been systematically compared with those of the MBI (67) with very favorable results. All participants completed the SMBM at time 1. However, at time 2, only 352 (52.0%) of the participants did so, mainly because certain employers specifically requested that the follow-up questionnaire be shortened.

Control Variables

The most important risk factor in the onset of type 2 diabetes is obesity (68). Other established risk factors include age and family history of diabetes. Additional factors found to be associated with this condition are alcohol intake, reduced physical activity, and hypertension (69–71). So these variables (age, body mass index [BMI] [kg/m^2], smoking [yes/no], alcohol use [wine ≥3 glasses per week; beer and other alcoholic beverages ≥1 glass per week], engaging in physical activity [yes/no], systolic blood pressure [SBP], and diastolic blood pressure [DBP] as well as gender, educational level, and family history of diabetes) were measured and controlled for in the current study. However, for a variety of reasons, including migrating to Israel alone or being the children of parents who died abroad, over one third of the subjects did not know if their parents had diabetes. So we had to omit this variable from the analysis.

Analyses

Paired *t*-tests were conducted to explore if there was a difference across time in mean burnout scores by employment status. Univariate analysis was used to examine whether those who scored high on burnout (above the mean on the SMBM) differed from their counterparts on other study variables. Multivariate analysis was performed to test the association between burnout and the incidence of diabetes (the onset of new cases of diabetes during the follow-up period) while controlling for all the possible confounding variables listed previously, including job category and follow-up duration.

RESULTS

According to Zapf et al. (72), to confirm the inference of causality in the stressor–strain relationship in longitudinal studies, one needs not only to ensure the stressor existed before ill health developed (as indeed we did by selecting workers not having diabetes at T1), but also to demonstrate that the stressor persisted during the follow-up period. We had the T2 measure of burnout for 358 participants. The cross-time correlation for this outcome was calculated to be 0.54 ($p < .0001$). The mean value of the burnout score was remarkably stable across the follow-up period: 2.51 and 2.52 for T1 and T2, respectively. No significant difference was found between those who had T2 burnout data and the 319 participants not having such data on T1 key parameters of age, gender, education, BMI, burnout, and diabetes incidence during the follow-up period. The former group, however, participated more in leisure time physical activity (42.1% versus 33.6%, respectively), had higher rate of alcohol use (27.9% versus 19.8%, respectively), and lower rate of smoking (21.5% versus 28.7%, respectively).

The characteristics of workers scoring high and low on burnout (using the sample mean as the cutoff point) are shown in Table 1. These groups did not differ in age, sex, education level, BMI, and health habits such as smoking and alcohol use. They did differ, however, on leisure time physical activity engagement. Not surprisingly, those who scored high on burnout reported less engagement in physical activity. In the subsample of workers for whom we had blood pressure measures, the burned-out workers had significantly lower SBP, but not DBP.

During the follow-up period, 17 workers (2.5%) developed type 2 diabetes. The incidence was much higher in the burned-out workers (3.2%) compared with the others (1.8%, odds ratio [OR] = 1.5, 95% confidence interval [CI] = 1.07–2.25).

The association between burnout and risk of diabetes was also tested in multivariate analysis while controlling for possible confounding variables. The logistic regression results

TABLE 1. Characteristics of Workers Scoring Low and High on Burnout

	Low Burnout (n = 329)	High Burnout (n = 348)	p^b
Age (years)	43.34 ± 9.53	41.92 ± 9.25	.055
Sex (percent males)	49.7	50.3	.23
Education (years)	13.7 ± 2.98	13.4 ± 3.07	.21
Body mass index	26.3 ± 4.11	26.2 ± 4.19	.68
Leisure time physical activity (percent yes)	54.3	45.7	.017
Alcohol use (percent)	25.5	22.7	.38
Smoking (percent yes)	24.3	25.6	.70
Systolic blood pressure (mm Hg)[a]	122.69 ± 14.72	119.21 ± 13.36	<.001
Diastolic blood pressure (mm Hg)[a]	75.06 ± 9.64	74.27 ± 9.47	.35
Type 2 diabetes (new cases)	6 (1.8%)	11 (3.2%)	OR = 1.5, 95% CI = 1.07–2.25

[a] Based on n = 261 and n = 247, respectively.
[b] Based on t-test or χ^2 test results.
OR = odds ratio; CI = confidence interval.

TABLE 2. Logistic Regression for Predicting Type 2 Diabetes Incidence by Burnout and Several Control Variables[a]

Variable	Odds Ratio	95% Confidence Interval	*p*
Burnout	1.84	1.19–2.85	<.001
Age	1.09	1.02–1.16	<.001
Sex	0.14	0.29–0.92	.04
Body mass index	1.23	1.09–1.39	<.001
Smoking	2.51	0.8–8.02	.12
Leisure time physical activity (percent yes)	0.32	0.08–1.22	.09
Alcohol use (percent)	0.34	0.08–1.72	.20

[a] Including job category and follow-up duration.

presented in Table 2 indicate that burnout (used as a continuous variable) was associated with a 1.84-fold increased risk of type 2 diabetes (95% CI = 1.19–2.85) even after controlling for age, sex, BMI, smoking, alcohol use, leisure time physical activity, job category, and follow-up duration. Finally, we repeated this analysis in the subsample of workers (n = 507) for whom we had blood pressure measures. The incidence of diabetes in this subsample was somewhat lower (2.0%) than in the full sample, but the association between burnout and diabetes risk was much higher than before (relative risk = 4.32, 95% CI = 1.75–10.63); this is while also controlling for blood pressure levels. In this analysis, SBP also turned out to be associated with risk of diabetes, but the magnitude of the association was much lower (OR = 1.09, 95% CI = 1.00–1.20) than for burnout. No significant result was obtained for DBP. No difference was found between this subsample of workers and the other 170 workers who did not have blood pressure measures on T1 key parameters of age, education, BMI, alcohol use, burnout, and diabetes incidence during the follow-up period. The former group, however, had higher percentage of women (27.9 versus 10.3, respectively) and lower rate of participation in leisure time physical activities (33.5% versus 51.1%, respectively).

DISCUSSION

The major finding of the present follow-up study is the association of burnout with a 1.84-fold increase in the risk of type 2 diabetes in apparently healthy workers even after controlling for several potential confounding variables. These include age, sex, BMI, smoking, leisure time physical activity, alcohol use, and job category. On controlling for blood pressure levels, in the subsample of workers for whom this data were available, a much higher risk was discovered (OR = 4.32, 95% CI = 1.75–10.63). This latter finding is particularly meaningful given that the incidence of new cases of diabetes in this subsample was somewhat lower than in the total sample, and the characteristics of the workers, including burnout level, were no different from those of the total sample (data not shown). Adjustment for blood pressure levels is important given the findings (cited previously) of an association between hypertension and risk of diabetes. Consistent with these findings, we also uncovered here that the SBP levels were marginally associated with diabetes risk. It was interesting to note, however, that burned-out persons in the current study had significantly lower SBP levels compared with their nonburned-out counterparts. This perhaps explains the increase in the adjusted risk of diabetes observed in burned-out persons after controlling for blood pressure levels. At any rate, the finding here concerning blood pressure is consistent with a previous report of a lack of association between burnout and blood pressure levels (38). Thus, the current finding suggests that the link between burnout and diabetes is not mediated by hypertension. Alternative mechanisms potentially underlying the effects that we have found are discussed subsequently.

Another noteworthy finding here is the remarkable consistency of the burnout symptoms over the 3 to 5 years of follow up. The mean burnout score of study participants did not change over time, and the cross-time correlation was 0.54. This is consistent with similar findings obtained in other studies, some of them with even longer follow-up periods (see "Introduction"). The important health implication of this finding is that prolonged endurance of the affective state of burnout may set the stage for various pathophysiological processes (briefly outlined subsequently) that may culminate in health impairment such as the onset of type 2 diabetes.

Further studies are needed to crossvalidate this association between burnout and risk of type 2 diabetes in other samples of workers. In parallel, studies may also be initiated to explore the mechanism of the link, including possibilities such as the induction of an acute phase response (APR) followed by a chronic inflammatory process. There is evidence to suggest that stress alone can induce the APR (73,74), which may take the form of dyslipidemia (increased triglycerides and lower high-density lipoprotein [HDL] cholesterol), the sickness response, and the induction in the liver of acute phase proteins (amyloid precursor proteins [APPs], e.g., C-reactive protein [CRP], and fibrinogen). Cytokins, in particular interleukin 6, are the main inducers of the APR. Corticosteroids and catecholamines, the major stress mediators, enhance this induction to a variable extent. Corticoids may simulate the expression of most APPs directly. Moreover, together with cytokines, glucocorticoids evoke a strong synergistic enhancement of most APPs (74). Thus, chronic stress may induce chronic APR, which mediates many of the inflammatory and metabolic events that may culminate in the insulin resistance syndrome (manifested by glucose intolerance with hyperglycemia), type 2 diabetes, and the metabolic syndrome (74).

There is evidence to suggest that burnout is associated with many of the key players in the pathway of the link outlined previously among stress, insulin resistance, and type 2 diabetes. Various studies have reported an association between burnout and components of the metabolic syndrome (such as elevated glucose levels and triglycerides and reduced HDL cholesterol levels), an elevated level of HbA1c, dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis (manifested by decreased cortisol response to awakening, hyper-

or hypocortisolism and flattened cortisol curve), increased concentration of proinflammatory cytokines, and inflammation biomarkers such as CRP and fibrinogen (for a review, see (35)).

There is substantial evidence indicating that many of these physiological parameters are associated with increased risk of type 2 diabetes (because a full review of such evidence is beyond the scope of the present article, the interested reader is referred to existing reviews, (e.g., (75,76)). To illustrate, perturbation of the HPA axis was found to be prospectively associated with an increased incidence of type 2 diabetes (77,78). The same applies to increased levels of CRP and interleukin-6 (79–81) and the metabolic syndrome (82).

In addition, burnout has been found to be associated with sleep disturbances, particularly insomnia (41), which have, in turn, been shown to be associated with type 2 diabetes in a number of studies (e.g., (83,84)). Thus, these findings suggest yet another complementary mechanism for the link between burnout and diabetes.

We should, however, note a number of caveats. We did not measure visceral obesity, a better predictor of diabetes risk than BMI (85,86) that has been suggested as a mediator of the link between HPA axis activity and the development of type 2 diabetes (78). Thus, future research should control for both BMI and visceral obesity. The incomplete data on family history of diabetes prevented us from controlling for this possible cofounder of the multivariate analysis. We did, however, examine among those who had such data if family history of diabetes was associated with burnout levels and found no such association. In the total sample, data on family history of diabetes was available for 428 participants. Of those scoring high on burnout ($n = 225$) 22.2% reported having such history. This figure did not significantly differ from the 18.2% reported by those scoring low on burnout ($n = 203$, $p = .28$). A similar trend was observed in the subsample of participants having blood pressure data. The corresponding percentages were 20.5 and 19.5, $p = .41$. Finally, we did not control for depression. A recent meta-analytic study that suggests that depression may be a risk factor for the onset of type 2 diabetes, the pooled relative risk ranged from 1.26 to 1.37, depending on the model applied (87). Empiric evidence shows that burnout measures have positive moderate correlations with depression (24,88–91). However, factor analytic studies of items measuring burnout and depression (26,92) have generally found the two constructs to load on different factors, indicating that they probably tap different domains. Moreover, accumulating evidence indicates that burnout and depression are differentially linked to disease mediators (35). Furthermore, recent studies have shown that the association between burnout and risk indicators such as inflammation biomarkers is not mediated by depression (40,66,93). Thus, we recommend that future studies include a depression measure to enable a test of whether the association between burnout and diabetes risk uncovered here persist even after controlling for depression. Nevertheless, the strength of the present study is its longitudinal design, and the follow up of initially healthy workers, which allows for an interpretation of causality in the link between burnout and risk of diabetes that persists after controlling for possible confounding variables.

CONCLUSION

The findings of the present study increase our knowledge of the potential risk to health of chronic burnout. Previous studies have demonstrated that burnout may pose a risk of CVD and other bodily disorders (see Melamed et al. (35)); the findings here suggest that the risk to health may also be generalized to the development of type 2 diabetes. Altogether, they confirm the need to find effective primary interventions to reduce burnout before it becomes chronic, thereby reducing the potential risk of physical health impairment.

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