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Elevated Burnout Predicts the Onset of Musculoskeletal Pain Among Apparently Healthy Employees

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Musculoskeletal (MS) pain is highly prevalent in the working population, often resulting in chronic disability. Burnout represents accumulated exposure to work-related stresses and therefore could predict the incidence of MS pain. We investigated prospectively the extent to which changes in the levels of burnout over time predict new cases of MS pain. Participants were 1,704 apparently healthy employed men and women who underwent periodic health examination at three points of time (T1, T2, and T3), over a period of about three years. We used the T1 to T2 changes in the levels of burnout, depressive symptoms, and anxiety to predict the onset of new cases of MS pain between T2 and T3, while controlling for possible confounders. Logistic regression results indicated that the T1–T2 change in burnout levels was associated with a 2.09-fold increased risk of MS pain (95% confidence interval = 1.07–4.10). No support was found for the possibility of reverse causation; that is, that MS pain predicts subsequent elevations of burnout levels. It was concluded that burnout might be a risk factor in the development of MS pain in apparently healthy individuals.

Keywords: burnout, psychosocial factors, musculoskeletal pain, incidence

Musculoskeletal (MS) problems are very common among adults and have important consequences for the afflicted individuals and society. They are major causes of the disease burden around the world (Brooks, 2006). Neck/shoulder and low back pains are highly correlated and represent very close comorbidities (Elfering, Grebner, Gerber, & Semmer, 2008) and therefore we refer to them as MS pain. MS pain is highly prevalent among the working population; it is estimated that 60% to 80% of the adult population experience MS pain at some point in their lifetime (Griffith et al., 2007). MS pain has been found to be associated with reduced work ability, functional capacity limitations, frequent sickness absences, chronic disability, early retirement, and impaired quality of life (Cote et al., 2008; Dagenais, Caro, & Haldeman, 2008; Punnett et al., 2005; Waddell, 2006). Back pain constitutes over 50% of all MS problems (Van Poppel, Koes, Deville, Smid, & Bouter, 1998). In the workplace, MS pain represents the single most costly injury in terms of its contribution to total workers' compensation costs (Baldwin, 2004). Following the above epidemiological data, it is apparent that accurate identification of the etiologic factors related to the onset of MS pain has the potential of advancing preventive health practices at workplaces.

Most of the reviews on risk factors for MS pain (e.g., Cote et al., 2008; Malchaire, Cock, & Vergracht, 2001; Manek & MacGregor, 2005) have concluded that its etiology is multifaceted. Manek and MacGregor (2005) noted that approximately 90% of MS pain cases have no identifiable cause and are designated as nonspecific. MS pain is highly prevalent among white-collar employees who do not perform physical work (cf. Sprigg, Stride, Wall, Holman, & Smith, 2007). To illustrate, a recent study of 2,000 office workers revealed that the annual preva-

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lence of self-reported MS pain, most notably in head/neck, lower back, and upper back regions, have reached 63% (Janwantanakul, Pensri, Jiamjarasrangsri, & Sinsongsook, 2008). Psychosocial factors, alone and interacting with physical factors at work, have been increasingly recognized as risk factors for MS pain at work (Bongers, Ijmker, van den Heuvel, & Blatter, 2006; Macfarlane et al., 2009). In summarizing the findings of several reviews of both cross-sectional and longitudinal studies that related psychosocial stress to MS pain, Macfarlane et al. (2009) noted considerable differences in conclusions among individual reviews. Proposed mechanisms that link work-related stress and MS pain were outlined in the comprehensive review by Huang, Feuerstein, and Sauter (2002).

We investigated the hypothesis that an increase in the levels of burnout predicts subsequently assessed occurrences of MS pain. There are several reasons for the current focus on burnout as a risk factor in the etiology of MS pain. A unique affective multidimensional response to stress, of which the core components are emotional exhaustion, physical fatigue and cognitive weariness (Shirom, 2003), burnout reflects the depletion of energetic coping resources resulting from prolonged exposure to chronic work stresses. It can therefore be used as a proxy effect on health-related outcomes, including MS pain (cf. Melamed, Shirom, Toker, Berliner, & Shapira, 2006a). This focus represents a parsimonious and more promising research strategy in comparison with the frequently used research strategy of focusing on subsets of work-related stresses as predictors of MS pain. Nevertheless, while there is compelling evidence from longitudinal studies to suggest that chronic stresses at work and burnout are reciprocally related over time (e.g., de Lange, Taris, Kompier, Houtman, & Bongers, 2004; Demerouti, Bakker, & Bulters, 2004; Shirom, Oliver, & Stein, in press), burnout is conceptually and empirically distinct from chronic stress (Melamed et al., 2006a). The empirical support for this argument is based on two meta-analytic studies that have investigated the relations between burnout and chronic stress (Collins, 1999; Lee & Ashforth, 1996) and found low to moderate metacorrelations between overall burnout and the chronic stresses, mostly ranging in the .30s. Finally, burnout has been found to be relatively stable across time (cf. Melamed et al., 2006a), like other affects at work (Luthans, Avolio, Avey, & Norman, 2007).

With one exception, reviewed below, the possible association between burnout and MS pain has been investigated only by cross-sectional (Honkonen et al., 2006; Langballe, Taris, Kompier, Houtman, & Bongers, 2009; Miranda, Viikari-Juntura, Heistaro, Heliovaara, & Riihimaki, 2005) or case-control (Soares & Jablonska, 2004) studies. In these studies, burnout was independently associated with a 1.2- to 1.7-fold increased risk of experiencing MS pain, even after controlling for a number of potential confounding variables, such as strenuous physical work and depressive symptoms (Honkonen et al., 2006) or job strain (Soares & Jablonska, 2004). A major limitation of the above cross-sectional studies is that they do not permit making causal inferences concerning the directionality of influence. We found only one longitudinal study that did so. In this study (Melamed, 2009), burnout symptoms were associated with a 1.67-fold risk of MS, in 3–5 years of follow-up, even after adjusting for possible confounding variables. Those exhibiting high burnout levels, compared with others, showed higher relative risk of MS pain (odds ratio [OR] = 2.45, 95% confidence interval [CI] = 1.35–4.45). However, baseline depressive symptom was not controlled in this single study. This is an important limitation because depressive mood has been suggested as an ethological factor in the development of MS problems (Manek & MacGregor, 2005; Jones et al., 2009). Burnout is conceptually different from depression, but empirically they have been found to be related (for a review, see Melamed et al., 2006a). Yet, in two cross-sectional studies, in which both depression and burnout were included as predictors of MS pain, burnout was found to be significantly associated with MS problems, even after adjusting for depression (Honkonen et al., 2006; Miranda et al., 2005). There is evidence that indicates that another affective state, namely, anxiety, is also associated with the development or existence of MS pain (Jones et al., 2009; Sprigg et al., 2007). Furthermore, comorbid anxiety and depression are common among pain patients (Bair, Wu, Damush, Sutherland, & Kroenke, 2008; Poleshuck et al., 2009). In addition, burnout, depression, and anxiety often occur together (Glass & McKnight, 1996; Schaufeli & Enzmann, 1998). To our knowledge, neither cross-sectional nor longitudinal past studies of the putative association between burnout and MS pain have simultaneously controlled for the covarying negative affects of anxiety and depression. Following the above rationale and body of evidence, we formulated the following hypothesis:

> *Hypothesis 1:* Elevations of initial levels of burnout will predict subsequent occurrences of

MS pain, even after controlling for anxiety and depression.

Another limitation of the only longitudinal study found (Melamed, 2009) is it did not test the possibility of reverse causation. Based on the Conservation of Resources (COR) theory (Hobfoll, 1988, 1989), we may also hypothesize that baseline occurrences of MS pain predict the intensification of burnout levels across time. The onset of MS pain probably represents an experienced stress that may lead to an escalating spiral of losses of energetic resources, as argued by COR theory. Therefore, initial levels of MS pain, by depleting energetic resources, may exacerbate subsequently experienced burnout. As we indicated, no past study to our knowledge investigated this reverse-causation hypothesis. Our second hypothesis was therefore thus formulated:

Hypothesis 2: Initial occurrences of MS pain will predict subsequent elevations of burnout levels.

It is also possible that burnout and MS pain may mutually reinforce one another. We considered this theoretical possibility of a reciprocal relationship between burnout and MS pain across time as directly resulting from finding that Hypotheses 1 and 2 are both supported by our data. Therefore, we did not test this hypothesis separately. We used a three-wave repeated measures design to investigate, for the first time, the directionality of the across-time relations between change in burnout levels and MS pain in apparently healthy workers, controlling for changes in depressive symptoms and anxiety levels, and adjusting for other potential confounding variables.

Method

Sample

Study participants (N = 1,704) were apparently healthy employees, attending the Center for Periodic Health Examinations of the Tel-Aviv Sourasky Medical Center, for a routine health examination at Time 1 (T1), Time 2 (T2), and Time 3 (T3), who voluntarily agreed to participate in the study. The average time lags from T1 to T2 and from T2 to T3 were 18 (SD = 7.01) and 17 months (SD = 12.10), respectively. These periodic health examinations were provided to the study participants by their employers as a subsidized fringe benefit: thus, attrition between T1 and T2, as well as between T2 and T3, could be because of change of employer, residence, or work location, and therefore totally unrelated to their participation in the current study. At T1, they represented 92% of the Center's examinees during this period. We systematically checked for nonresponse bias at T1 and found that nonparticipants did not differ from participants on any of the sociodemographic or biomedical variables. Fifty-three percent of T1 examinees did not return for a follow-up at T2 or T3. These were more likely to be male, to be older (near retirement age), to have self-reported a chronic disease at T1, and to have spent less time in regular exercise activity at T1. They had higher burnout and depression scores but lower anxiety scores. These possible sources of attrition bias were controlled for in the data analyses, as explained below.

We excluded from the Study 268 participants who, either at T1 or T3, self-reported diagnosed CVD, cancer and diabetes or who had suffered a stroke or a mental crisis, as well as participants who reported regularly taking antipsychotic medications or antidepressants. This exclusion was based on previous findings suggesting that these disorders and medications could impact levels of burnout and fatigue (Franssen, Bultmann, Kant, & van Amelsvoort, 2003). In addition, we excluded from the Study 98 participants who reported on not working at T3. An additional 270 participants were excluded from the analysis because of missing data for one of the study parameters. Thus, the final sample consisted of 1068 apparently healthy employees (65% men). The mean age at T1 was 46.58 (SD = 9.26). Respondents at T1 had completed a mean of 16 (SD = 2.66) years of education; they worked for an average of 9.6 (SD = 2.25) hours a day.

Procedure

The study's protocol was approved by the ethics committees of the Sourasky Medical Center and the Faculty of Management at Tel Aviv University. The participants were recruited individually by an interviewer while waiting their turn for the clinical examination. They had been promised and subsequently received detailed individual feedback on their results. Confidentiality was assured, and each participant signed a written informed consent form which had been approved by the above ethics committees.

Measures

MS pain was assessed as reported neck pain, pain in the shoulder region, or lower back pain over the

last 12 months and seeking medical care for it, as is customary in studies of psychosocial factors (see, e.g., Elfering et al., 2008; Toomingas et al., 2007). Our decision to focus on neck/shoulder and low back pain, because the outcome variable was based on past studies that have shown this to be stress related and thus customarily used in the studies of psychosocial factors and MS pain (Elfering et al., 2008).

Burnout was assessed by the Shirom-Melamed Burnout Measure (SMBM), whose reliability and validity have been demonstrated in a number of studies (e.g., Melamed, Shirom, Toker, & Shapira, 2006b; Shirom & Melamed, 2006). The SMBM consists of 14 items scored on a 7-point frequency scale, ranging from 1 (*almost never*) to 7 (*almost always*). Its reliability coefficient (Cronbach's alpha) in this study was .92, .93 and .93 for T1, T2, and T3, respectively. Examples of the items are "I feel physically drained" (physical fatigue); "I feel I am not thinking clearly" (cognitive weariness); and "I feel I am unable to be sensitive to the needs of coworkers and customers" (emotional or interpersonal exhaustion). Burnout was used as a continuous measure.

Control Variables Used in the Analyses

Depressive symptomatology (referred to as depression) was controlled in this study because when it is conceptualized as a syndrome, it is often defined to include symptoms of fatigue (Suls & Bunde, 2005). However, we argue that burnout does not overlap conceptually with depression because it is contextualized to occur at work and is determined by the social environment at work, whereas depression is a global state pervading all spheres of the individual's life (Suls & Bunde, 2005). Empirically, depression and burnout have been shown in past quantitative (Glass & McKnight, 1996) and qualitative (Melamed et al., 2006a; Schaufeli & Enzmann, 1998) reviews to be distinct from each other and to be differentially associated. To assess depressive symptomatology, we used the validated measure of the Personal Health Questionnaire (PHQ), the depression section of a patient-oriented self-administered instrument derived from the PRIME-MD (Spitzer, Kroenke, Williams, & the Patient Health Questionnaire Primary Care Study Group, 1999; Spitzer et al., 1994). This measure lists nine potential symptoms of depression, in accordance with each of the nine *DSM–IV* criteria and asks patients to rate the frequency of experiencing each symptom during the past two weeks on a scale ranging from 1 (*never*) to 4 (*almost always*) ($\alpha = .77$ and .75 for T1 and T2, respectively). Its validity as a measure of depressive symptoms in nonclinical settings has been demonstrated in many studies (cf. Kroenke et al., 2009).

We controlled for *anxiety*, defined as a future-oriented discrete affective experience which results from perceptions of threat and inability to predict desired results in upcoming situations (Kubzansky, Kawachi, Weiss, & Sparrow, 1998). The measure of *anxiety* used in this study includes four items (feeling nervous, jittery, fidgety, anxious) adapted from questionnaires used in several large-scale studies conducted by the Institute of Social Research, University of Michigan (e.g., French, Caplan, & Harrison, 1982). All items are scored on a 5-point frequency scale, ranging from 1 (*almost never*) to 5 (*almost always*); $\alpha = .86$ and .86 for T1 and T2, respectively.

We controlled for body mass index (BMI; kg/m^2), measured by a nurse and used as a continuous variable, following the recently proposed association of obesity with the feelings of fatigue and tiredness (e.g., Lean, 2000) characterizing burnout, and given some evidence regarding an association between MS pain and body weight (for a review, see Malchaire, Cock, & Vergracht, 2001). We controlled for *age*, consistently found to be negatively associated with burnout (Schaufeli & Enzmann, 1998) and positively associated with MS pain (for a review see Cote et al., 2008; Malchaire et al., 2001). *Gender* was controlled, following findings indicating that regional MS pain appears to occur more frequently in women (for a review, see Cote et al., 2008; Malchaire et al., 2001). In addition, we controlled for *educational level,* given some indications for a positive association between education and burnout (Schaufeli & Enzmann, 1998) and between educational level and musculoskeletal problems (for a review, see Malchaire et al., 2001). Some evidence suggests that low leisure time physical activity (LTPA) is associated with increased risk for both burnout (Schaufeli & Enzmann, 1998) and MS pain (Malchaire et al., 2001). Thus, on an exploratory basis we tested if LTPA turns out to be a confounding factor, but the result was not significant. Consequently, we did not include this variable in subsequent analyses. *Age, gender* and *educational level* (number of years of education) were taken at T1 from the demographic details as reported by the respondents.

Statistical Analysis

In testing our first hypothesis, we first excluded from the analysis 467 respondents who reported having MS pain at T1 or T2. Thus, our first analysis was

conducted on 601 subjects. Subsequently, we coded respondents reporting MS pain at T3 as = 1, otherwise = 0, on the criterion of T3 incidence of MS pain. We used logistic regression to predict the incidence of MS pain at T3. We predicted the criterion first by T2 burnout (being more proximal to the criterion relative to T1 burnout). We then entered T1 burnout as a control. We subsequently entered the control variables of T2 and T1 depression and anxiety. Then we entered age, gender, education and BMI at T1. We decided on this analytic strategy because it provided a robust and strong test of Hypothesis 1 as formulated (cf. Twisk, 2003).

To test our second hypothesis, we predicted T3 burnout first by T2 burnout, thereby in effect predicting the change from T2 to T3 in burnout (cf. Twisk, 2003), controlling for the aforementioned possible confounders. In this test, the predictor of interest was the dichotomy of reporting MS pain at T2 but not at T1: 406 respondents who reported having MS pain at T1 were excluded from the analysis. Thus, our second analysis was conducted on 662 subjects. Because in testing the second hypothesis the criterion was a continuous variable, we used ordinary least squares (OLS) regressions.

Results

Descriptive Statistics

The prevalence of MS pain at T1 was 37.2% (n = 397). At T2 and T3, 66 (6.2%) and 84 (7.9%) new cases of MS pain, respectively, were identified.

Pearson Product-Moment correlations between all the study's variables are presented in Table 1. MS pain was moderately but significantly correlated with burnout at T2 and T3 (.12, and .11, respectively), and with depression at T1 and T2 (.09, and .08, respectively). Burnout showed stability from T1 to T2 and from T2 to T3 (.70, and .77, respectively). Depression and anxiety showed stability from T1 to T2 (.62, and .59, respectively). The intercorrelations among burnout at T1, T2 and T3 and T1, T2 depression and anxiety were moderate but significant (r values varied between .41 and .55). Age was negatively and significantly correlated with burnout at T1, T2, and T3. BMI was negatively correlated with burnout at T2. Using a paired samples t test for the significance of the T1–T2 mean difference of depression and anxiety (Table 1), we found that anxiety levels decreased significantly from T1 to T2. Using analysis of variance (ANOVA) repeated measures for the significance of the T1–T2 and T2–T3 mean difference

Table 1
Means, SDs, and Pearson Product-Moment Intercorrelations of the Study's Variables (N = 1,068)

Measure	1	2	3	4	5	6	7	8	9	10	11	12
1. MS pain incidence T3[a] (Y/N)		.11*	.12*	.07	.08*	.09*	.05	.06	−.07	−.04	.04	.06
2. Burnout T3	—		.77*	.69*	.52*	.49*	.51*	.43*	−.06	−.06	−.15*	.19*
3. Burnout T2	—	—		.70*	.53*	.46*	.55*	.44*	−.01	−.10*	−.16*	.18*
4. Burnout T1	—	—	—		.41*	.50*	.42*	.49*	−.03	−.06	−.12*	.18*
5. Depression T2	—	—	—	—		.62*	.55*	.38*	−.06	.03	−.07	.19*
6. Depression T1	—	—	—	—	—		.46*	.43*	−.08	.01	−.04	.24*
7. Anxiety T2	—	—	—	—	—	—		.59*	−.03	−.06	−.13	.07
8. Anxiety T1	—	—	—	—	—	—	—		.03	−.03	−.09	.07
9. Education	—	—	—	—	—	—	—	—		.01	.08	−.10*
10. BMI	—	—	—	—	—	—	—	—	—		.20*	−.21*
11. Age	—	—	—	—	—	—	—	—	—	—		.02
12. Gender	—	—	—	—	—	—	—	—	—	—	—	
M	.11	1.92§	1.96	2.11§	1.20	1.21§	1.63	1.90§	16.04	26.46	46.58	.28
SD	.31	.78	.78	.80	.26	.29	.69	.78	2.66	3.71	9.27	.45

Note. BMI = body mass index.
[a] During the T2–T3 period.
* $p < .05$. § Significantly differed from the corresponding T2 values, at $p < .05$.

burnout (Table 1), we found that burnout levels decreased significantly from T1 to T2 and from T2 to T3.

Burnout Predicts the Incidence of MS Pain

The results of the logistic regression analysis testing the prediction that change in burnout levels at baseline predicts the incidence of MS pain at T3 are reported in Table 2. After the aforementioned possible confounders had been controlled, change in burnout levels from T1 to T2 was found to be associated with an increased risk of MS pain from T2 to T3 (OR = 2.09; 95% CI = 1.07–4.10). Although we used the change in burnout levels from T1 to T2, both T2 level alone and after controlling for T1 burnout are significant predictors of the criterion. Therefore, this provided support to our Hypothesis 1.

Testing the Possibility of Reverse Causation

The reverse-causation hypothesis—that the incidence of MS pain at T2 predicts changes in levels of burnout from T2 to T3—was tested by OLS regressions, again controlling for the potential confounders described above. We found that the effect of the incidence of MS pain at T2 was not a significant predictor ($\beta = -.02$, *ns*) of T3 level of burnout, controlling for T2 burnout (a table describing the results obtained in this regression is available from the first author upon request).

Table 2
Logistic Regression Analysis Predicting the Incidence of Musculoskeletal (MS) Pain at T3 Relative to T2 (N = 601)

Measure	OR	95% CI
Burnout, T2	2.09*	1.07–4.10
Burnout, T1	.64	.34–1.21
Depression, T2	.43	.06–2.91
Depression, T1	3.53	.79–15.66
Anxiety, T2	.74	.37–1.46
Anxiety, T1	.89	.51–1.55
Education, T1	.91	.81–1.02
BMI, T1	.96	.87–1.05
Age, T1	1.02	.98–1.06
Gender	.72	.32–1.58

Note. CI = confidence interval; OR = odds ratio; BMI = body mass index.
* $p < .05$.

Discussion

The major finding of this prospective cohort study, conducted in a large sample of apparently healthy employed persons, is that increased levels of burnout over a period of 18 months is associated with a 2.09-fold increased risk of developing MS pain during the subsequent 18 months of follow-up. Furthermore, such a risk was obtained even after controlling for T1 to T2 changes in anxiety levels and depressive symptoms and for other potential confounding variables.

Consistent with the results of prior studies (cf. Schaufeli & Enzmann, 1998), we found that burnout, depression, and anxiety were moderately correlated at each of the three points of time, T1, T2, and T3. Yet, in the multivariate (logistic regression) analysis only T2 burnout remained a significant predictor, a finding that reinforces the significance of burnout as a risk factor for the onset of MS pain. Another significant finding in this study was that the possibility of reverse causation (the second hypothesis), that MS pain predicts subsequent elevations of burnout levels, was not supported. Thus, we would like to suggest that there is a unidirectional effect of initially assessed changes in the levels of burnout on the subsequent incidence of MS pain. As we noted above, our key finding is congruous with past cross-sectional and case-control studies including those that assessed burnout using the same measure we used.

Differentiating Burnout, Anxiety, and Depression

Our strategy of controlling for depression and anxiety followed the recommendation of Ryff and Singer (2003) that future research should focus on co-occurring discrete emotions as a route to understanding the cumulative effect of the emotional experience on physical health. Ryff and Singer accentuated the importance of studying the co-occurrence of discrete emotions to capture both the subjective experience and map out the physiological mechanisms on the route to health. A major strength of the present study is that we were able to investigate the prospective relationships between MS pain and each of the affective states—anxiety, depression, and burnout—and were therefore able to depict the independent association of each of them with this criterion.

Although some studies have reported a positive association between depression, anxiety and chronic pain (e.g., Bongers et al., 2006; Bongers, Kremer, &

ter Laak, 2002), our results show that a change in depression and anxiety at baseline levels did not predict the incidence of MS pain.

What could be possible account for these findings, obtained even though the within-wave correlations of the three negative affects were moderate to high in magnitude? Theoretically, the three constructs are different from each other: depression signifies a generalized distress encompassing all life domains, whereas burnout is context specific in that it refers to the depletion of individuals' energetic resources at work (as assessed by the SMBM). Like depression, anxiety is also distinct from burnout in represents an activated negative affect which is not contextualized. Thus, the unique core of burnout, depletion of energetic resources, is distinct in its content and nomological network from both depression and anxiety (cf. Suls & Bunde, 2005).

Future Research Directions

A major task for future research is to elucidate the pathophysiological pathways linking burnout and the development MS pain. In linking burnout with the incidence of MS pain, we rely on mediating mechanisms linking psychosocial stress and MS pain as proposed in recent reviews (Bongers et al., 2002; Elfering et al., 2008). First, high mental load and job demands may increase muscle tension and decrease micropauses in muscle activity. These stress-related bodily changes may lead to muscle fatigue, even in cases of low loads, because of the continuous firing of low threshold motor units, possibly triggered by mental loading. Indeed, burnout has been found to be associated with chronic tension and restlessness (Melamed, Kushnir, & Shirom, 1992). Second, work-related exposure to stresses may impede one's ability to unwind during breaks at work and after work. By "unwind," we mean one's ability to reduce one's physiological activation. Analogously, burnout has been found to be associated with postwork irritability and insomnia, which reflects an inability to unwind (Melamed et al., 1999). The third possible path leading from burnout to MS pain relates to stress-induced adverse changes in the immune/inflammatory system. Likewise, burnout has been found to be associated with hypocortisolism (Melamed et al., 2006a). Lack of cortisol availability may promote increased vulnerability to bodily disorders, such as chronic pain (Heim, Ehlert, & Hellhammer, 2000; Raison & Miller, 2003). A fourth path of influence of burnout on MS pain concerns the activation of the sympathetic-adrenal medullary system in response to stress, which leads to secretion of norepineprine, which in turn heightens muscle activity. Indeed, an association between norepineprine levels and regional musculoskeletal pain was found by Elfering et al. (2008). There is indirect evidence for the association between burnout and increased catecholamine (especially norepineprine) levels (Melamed et al., 2006a). Direct studies are needed to test these putative pathways indicating a link between burnout and MS pain.

Limitations

The results of this study should be interpreted with caution because of some limitations. First, our sample of participants undergoing a periodic health examination may not be representative of the general population. Most of the individuals were highly educated white-collar workers who exhibited generally good health behavior patterns: they smoked little and exercised regularly. Owing to their superior health habits, our participants may have been more resilient to the effects of stress. However, it is even more likely that the significant findings obtained here with regard to the link between burnout and MS pain will be replicated among less resilient respondents. The possibility of obtaining stronger associations in the general working population is further reinforced by the finding that those who were lost to follow-up had higher burnout scores than the attendees, which leads to a restriction in the range of burnout scores. In addition, we controlled in our study for a major component of socioeconomic status, education. Third, we did not control for a potential confounding factor of job satisfaction which was found in past studies to be associated with both burnout (Schaufeli & Enzmann, 1998) and MS pain (Bongers et al., 2006; Macfarlane et al., 2008). Fourth, as we noted above, several possible mechanisms may explain the effects of burnout on MS pain. In the current study, we did not investigate the possible mediating mechanism. Testing such mechanisms would have helped to explain, on the physiological level, why burnout turned out to be predictive of MS pain incidence while depression and anxiety did not. It has been previously suggested that burnout and depression are differentially associated with disease mediators (Melamed et al., 2006a). Testing these mechanisms appears to be a promising avenue for future research.

Strengths

Along with the limitations noted above we would to point out several strengths of the current research.

First, we based our study on a large and heterogeneous sample. Second, we carefully excluded individuals who self-reported being chronically ill or taking medications that could influence the major study variables. Third, our design was based on three waves of repeated measures. This has obvious advantages over testing cross-sectional relations because it controls for the confounding influence of time-invariant common causes (Dormann, 2001). Such a design, which also permitted testing and refuting the possibility of reverse causation, allows the interpretation of causality in the link between increased burnout over time and the development of MS pain (Zapf, Dorman, & Frese, 1996).

Implications

The findings of the present study increase our knowledge of the potential risk to health of chronic burnout, thus extending a recent review (Melamed et al., 2006a) which found burnout to be associated with cardiovascular disease (CVD) risk. Burnout has also been found to be associated with risk factors for CVD, such as atherogenic lipid profile (Melamed et al., 1992; Shirom, Westman, Shamai, & Carel, 1997), sleep disturbances (Grossi, Perski, Evengard, Blomkvist, & Orth-Gomer, 2003; Melamed et al., 1999), impaired fertility (Sheiner et al., 2002), and Type 2 diabetes (Melamed et al., 2006b). The findings here suggest that health risks might also be generalized to the development of MS pain. Given the stability of burnout over time, it seems that measures taken to either prevent or alleviate burnout before it becomes chronic, may reduce the risk of debilitating, costly and highly prevalent MS pain and other bodily disorders. Studies have shown that both personal and organizational interventions have proved effective in reducing burnout in a variety of target populations (cf. LeBlanc et al., 2007) and could well be applied as preventive measures to reduce the incidence of MS pain.

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