Gender Differences in the Across-Time Associations of the Job Demands-Control-Support Model and Depressive Symptoms: A Three-Wave Study

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We used a full-panel longitudinal design to investigate, separately for women and men, the hypotheses that changes in the components of the Job Demands-Control-Support (JDC-S) model predict changes in depression symptoms levels over time and that the reversed prediction would also be found. Our study was conducted on a multi-occupational sample of apparently healthy employees ($N = 692, 68\%$ men) using three waves of data gathering, replicating our tests on two time lags of 18 months and 3 years on average. We controlled for neuroticism and other potential confounding variables. For both time lags, support for our hypotheses was found for the men only. We did not find systematic differences between the time lags, nor did we find a predominance of one of the unidirectional effects examined. We outline the theoretical and practical implications of our findings, including their relevance for efforts to combat depressive symptoms by changing job characteristics.

Keywords: depressive symptoms, gender differences, JDC-S model, longitudinal study, stress

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INTRODUCTION

Depressive symptomatology is recognised as one of the most prevalent and treatable mental disorders (World Health Organization, 2002), and is expected to become the second-ranked cause of disease burden in 2020 (Murray & Lopez, 1997). Symptoms of depression, as examined in this study, were found in past research to be associated with days absent from work, disability, lower productivity, absenteeism, an increase in substance use and accidents, a diminished sense of well-being, utilisation of medical services, visits to medical clinics, morbidity and mortality (Goldberg & Steury, 2001).

It is widely assumed that the etiology of depression is multifactorial, involving genetic, biological, and socioeconomic factors as well as life events (Cole & Dendukuri, 2003; Netterstrom et al., 2008). This mix of risk factors for depression was confirmed in many Israeli studies (for a recent review, see Geulayov, Lipsitz, Sabar, & Gross, 2007). A recent line of research has focused on the notion that psychosocial factors in the workplace are closely associated with depression in both cross-sectional and prospective studies (for recent reviews and meta-analyses of the work-related stress–depression linkage, see Bonde, 2008; Netterstrom et al., 2008; Siegrist, 2008; Stansfeld & Candy, 2006). Most of these reviews defined work-related psychosocial factors based on the Job Demands Control (JDC) model (Karasek, 1979) or its expanded version, the Job Demands-Control-Support (JDC-S) model (Karasek & Theorell, 1990). Job demands usually refer to the psychological job demands, primarily defined as referring to perceived workload, and Job control refers to the freedom permitted the worker in deciding how to meet the demands or how to perform tasks. Work-based social support refers to “overall levels of helpful social interaction available on the job from both co-workers and supervisors” (Karasek & Theorell, 1990, p. 69). Past theory and research on the JDC-S model (de Lange, Taris, Kompier, Bongers, & Houtman, 2003; Van Der Doef & Maes, 1998) indicates that the model leads to two major types of prediction. The first, often referred to as the iso-strain hypothesis (Van Der Doef & Maes, 1998), expects additive (main) deleterious effects of high job demands, low control, and low social support on psychological and physiological strain, including depressive symptoms. The second major type of prediction, referred to as the moderating hypothesis, focuses on the interactive effects of control and social support with job demand. The moderating hypothesis expects that the higher the job control and the higher social support, the less powerful are the effects of demand on the negative outcome (Van der Doef & Maes, 1999).

The major objective of this research is to test both the additive and the moderating hypotheses and also both types of unidirectional effects—from the JDC-S model to depressive symptoms and from depressive symptoms to the JDC-S model—using a longitudinal design. If both effects are found, then
we may conclude that the JDC-S model and depression are reciprocally related across time (cf. de Lange, Taris, Kompier, Houtman, & Bongers, 2004).

**Direct Unidirectional Path: JDC-S Model → Depressive Symptoms**

The direct effect of demand on depressive symptoms is explained in the JDC-S model by the process of using resources to cope with the demand, including the physiological implications of arousal due to the perceived demand (Karasek & Theorell, 1990). How do we explain the role of control and support in the etiology of depression? The process linking the JDC-S model with depressive symptoms can be viewed on the basis of Hobfoll’s Conservation of Resources (COR) theory (Hobfoll, 1989). According to COR theory, people strive to obtain or maintain resources they value (including energetic resources). Job control and social support are examples of such resources. COR stipulates that stress occurs when the rate that work demands consume employees’ resources exceeds the rate at which the resources are replenished. When individuals cannot cope with this stress effectively by allocating or investing new resources, prolonged stress and eventually depressive symptoms may develop. This line of reasoning implies that there is a negative effect of job resources on depression; the less resources one’s job offers, the greater are one’s depressive symptoms.

We were able to identify three recent reviews of longitudinal studies of the JDC-S model and depression (Bonde, 2008, 16 studies; Netterstrom et al., 2008, 14 studies; and Siegrist, 2008, seven studies); their major findings are consistent across the three reviews. The additive hypothesis of the JDC-S model, referred to above as the iso-strain hypothesis, was largely confirmed in most of the studies reviewed in the three reviews, while the moderating effects hypothesis was not confirmed.

According to Zapf, Dormann, and Frese (1996), one’s psychological dysfunction, including depression, may increase with one’s exposure time to chronic stress. There are almost no past studies directly assessing whether an increase in exposure to the job characteristics included in the JDC-S model predicted an increased risk of developing depression (Netterstrom et al., 2008). Most past prospective studies have examined whether the level of exposure at one point in time predicts onset of major depression or depression symptoms level at a later point in time. However, the underlying assumption in most past studies is that work characteristics remain fairly stable over time (at least for the duration of the follow-up) and this allows the making of causal inferences regarding the observed/non-observed differences in psychological and physiological strain over time. In reality, however, longitudinal studies of job strain and health reveal appreciable change in exposure.
histories. In one study, it was found to apply to up to 55 per cent of the sample over three years of follow-up (see de Lange, Taris, Kompier, Houtman, & Bongers, 2002). Indeed, adverse change in work characteristics was found to predict risk of psychiatric disorder (Stensfeld, Fuhrer, Shipley, & Marmot, 1999), but this study did not examine the possibility of intensification of depressive symptoms over time.

The current study pioneered in testing changes in JDC-S model components as predictors of changes in depressive symptoms. Such testing was possible by measuring both the JSC-S model components and depressive symptoms at three points in time. With one exception (de Lange et al., 2004), no other previous study has done this. Thus, in accordance with the major postulates of the JDC-S model, we expected that changes in the JDC-S model components would predict, additively and interactively, subsequent levels of depressive symptoms. Specifically, we predicted that:

\textit{Hypothesis 1a}: Elevation in workload level is expected to be a positive predictor, while decrease in job control and social support levels is expected to be a negative predictor of subsequent levels of depressive symptoms.

\textit{Hypothesis 1b}: Elevation in job control and social support levels is expected to moderate the potential negative effects of high job demands levels on subsequent levels of depressive symptoms.

**Reversed Unidirectional Path: Depressive Symptoms → JDC-S**

Based on the COR theory, we can suggest that the reversed causal relationship is also possible, in which high levels of depressive symptoms at baseline predict perceived higher levels of demands and lower levels of control and social support with time. The existence of depressive symptoms reflects initial resources loss. COR theory postulates that once loss cycles begin, initial resources loss increasingly results in loss of other resources (Hobfoll & Shirom, 2000). Thus, depressed employees are likely to perceive their jobs as more stressful. Moreover, the capacity of depressed employees to mold their work environment requires an investment of mental resources that are already depleted. For instance, low social support attributions may not only be a result of their negative appraisals, but also a result of having fewer social skills that characterises those with depression (Staw, Sutton, & Pelled, 1994).

Longitudinal studies that specifically used the JDC or JDC-S models to test the “reverse causal” or reciprocal relationship with depression are scarce and their findings are ambivalent. We identified only two relevant studies. One of them (Waldenström, Lundberg, Waldenström, Härenstam, & MOA Research Group, 2003) tested only the unidirectional influence of depression on the JDC model components, and did not find over-reporting of work
demands or under-reporting of work control in depressed subjects. The second study, by de Lange et al. (2004), found a reciprocal association between the JDC-S model and depression. However, the de Lange et al. (2004) study suffers from some limitations, which our study tries to overcome. First, the de Lange et al. (2004) study did not investigate the possibility, suggested by past theory and research described below, that the two unidirectional paths linking the JDC-S model and burnout are gender-determined. Second, de Lange et al. (2004) did not control for personality disposition of neuroticism (a global measure of negative affectivity) as a possible confounding factor, particularly in studies employing self-report measures. Neuroticism explained some of the covariance of work characteristics and common mental disorders (Stansfeld & Candy, 2006). Thus, we formulate the next hypothesis:

**Hypothesis 2**: Changes in depressive symptoms levels with time will predict subsequent changes in the levels of JDC-S model components across time such that elevation of depressive symptoms levels will be a positive predictor of workload level and a negative predictor of job control and social support levels.

### The Appropriate Length of the Between-Waves Time Lag

An important issue neglected in most longitudinal studies of the JDC-S model (the exception being de Lange et al., 2004) is the specific time lag needed to detect the effect of the JDC-S model components on depressive symptoms. This issue is critical in stress research given that the effects of the causal variables on the outcomes will be biased if the time lag in the study is not appropriate (Taris, 2000). There is little information about the appropriate length of time lags in occupational health research (Dormann & Zapf, 2002; Taris & Kompier, 2003; Zapf et al., 1996), and recommendations tend to be inconsistent (de Lange et al., 2003). Most studies have used a time lag of one year, which may not be sufficient to demonstrate the effects of job conditions on employee strain (Dormann & Zapf, 2002). Due to the paucity of relevant evidence, we rely on the theoretical stress reaction model suggested by Zapf et al. (1996), which postulates that depressive symptoms levels may change in response to the change in the stress levels with exposure time. By conducting a three-wave study, the second aim of the present study is to examine which of the time lags yields the strongest lagged effects of the independent variable on the outcome variables (Frese, 1984).

### Are the Two Unidirectional Paths of Influence Gender-Specific?

From an early review of (mainly cross-sectional) studies on the JDC-S model, the authors have concluded that high-strain jobs may influence well-being of
women and men in different ways (van der Doef & Maes, 1999). A meta-analytic review of longitudinal studies on the work environment and mental health, conducted later (Stansfeld & Candy, 2006), have led to a similar conclusion. Many past studies using the JDC-S model to predict the development of depression, including those cited in the above reviews, overlooked the possibility of gender differences and instead only adjusted for gender (Netterstrom et al., 2008). However, a recent systematic review of epidemiological evidence suggests that the impact of job strain on risk of depression is stronger and more consistent in men (Bonde, 2008). In the 14 longitudinal studies reviewed by Netterstrom et al. (2008), there are four studies of job strain in which the analyses were stratified by gender. A closer inspection of their findings uncovers the very same trend noted above. In addition, the same trend emerged in recent population-based studies not included in the above reviews (Blackmore et al., 2007; Wang, Lesage, Schmitz, & Drapeau, 2008). Based on this evidence, we expect that the association between the JDC-S model components and depressive symptoms will be stronger for the men. We maintain that this gender difference will also apply to the reversed relationship from depression to the JDC-S model components. To our knowledge this possibility has never been tested before. Thus, we hypothesise that:

Hypothesis 3: The prospective direct and reversed associations between the JDC-S model components and depressive symptoms will be stronger for men.

Thus, the present longitudinal full panel design conducted on a multi-occupational sample of apparently healthy employees was specifically designed to investigate if changes in all three components of the JDC-S model predict changes in depressive symptoms levels over time and if the reverse predictions also hold. The analyses were conducted separately for women and men, in three waves of measures, comparing different time lags (18 months and 3 years), controlling for neuroticism and other potential confounding variables, while excluding respondents whose chronic illnesses or habitual use of medication could have influenced their level of depressive symptoms, an exclusion not practiced in any past longitudinal study.

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
mean 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 at two- or three-year intervals as a subsidised fringe benefit: therefore, attrition between T1 and T2 as well as between T2 and T3 could be due to change of employment, residence, or work location, and thus be totally unrelated to their participation in the current study. At T1, the respondents represented 92 per cent of the Center’s examinees during this period. We systematically checked for non-response bias at T1 and found that non-participants did not differ from participants on any of the socio-demographic or biomedical variables. Fifty-three per cent 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 a self-reported chronic disease at T1, to have spent less time in regular exercise activity at T1, and had higher depression 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 at T1, T2, or T3 self-reported being diagnosed with CVD, cancer or diabetes, or who had suffered a stroke or a mental crisis, as well as participants who reported regularly taking antipsychotic medication or antidepressants. This exclusion was based on previous findings suggesting that these disorders and medications could impact levels of depression (see Clarke & Currie, 2009, for a recent review). Additionally, we excluded from the study 98 participants who reported that they were not working at T3. A further 646 participants were excluded from the analysis because of missing data for one of the study parameters. Thus, the final sample consisted of 692, apparently healthy employees (68% men). The mean age at T1 was 46.81 (SD = 9.69) for the women, and 45.90 years, (SD = 9.20) for the men. Respondents at T1 had completed a mean of 15.38 and 16.09 (SD = 2.93 and 3.14) years of education, for women and men, respectively. They worked for an average of 8.5 and 9.9 (SD = 2.20 and 2.50) hours a day (for women and men, respectively).

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 feedback on the results. Confidentiality was assured, and each participant signed a written informed consent form which had been approved by the above ethics committees.
Measures

Depressive symptomatology was assessed by the validated measure of the Personal Health Questionnaire (PHQ), the depression section of a patient-oriented self-administered instrument derived from the PRIME-MD (Kroenke et al., 2009). It lists eight 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 2 weeks on a scale ranging from 0 (never) to 3 (almost always). Its validity as a diagnostic and severity measure for depressive disorders has been confirmed in large clinical and non-clinical studies (cf. Kroenke et al., 2009).

Workload was measured with six items, similar to those of the job demands scale of the Job Contents Questionnaire (JCQ; Karasek & Theorell, 1990, p. 346). Sample items were: “I am required to work too fast”; “I do not have enough time to meet job demands”. Responses were made on a 5-point scale, ranging from 1 (to a very little extent) to 5 (to a very large extent).

Job control was measured on a seven-item scale, similar to the decision authority scale of the JCQ. Sample items are: “My opinions and suggestions influence what happens at work”; “My job enables me to make decisions on my own and to follow through with them”; “I am free to determine how to perform my work”. The response scale is the same as the job demands scale. This measure has been used in prior works (e.g. Shirom, Melamed, & Nir-Dotan, 2000).

Social support was an eight-item measure which covered instrumental and emotional support from significant others at work (peers and superior) based on work by French, Caplan, and Harrison (1982) that has been used in prior work (e.g. Shirom et al., 2000). Respondents were asked to score, on a 5-point Likert scale ranging from 1 (not true at all) to 5 (very true), how much each of the above significant others at work was easy to talk to, could be relied on when things got tough at work, and was willing to listen to the respondent, as sample items.

Control Variables Used in the Analyses

We controlled for neuroticism, one of the basic personality dimensions of the Five-Factor Model of Personality (Suls, 2001), defined as heightened reactivity to the occurrence of major and minor life stressors (Costa & McCrae, 1992). Several cross-sectional and longitudinal investigations conducted in clinical as well as non-clinical samples have found good support for the associations between neuroticism and symptoms of depression (e.g. Muris, Roelofs, Rassin, Franken, & Mayer, 2005). Moreover, a growing
body of empirical evidence reveals that the interactive effects between job characteristics may actually depend on the existence of certain personality characteristics (for an overview, see Brief, Burke, George, Robinson, & Webster, 1988). However, almost none of the studies on the uni-directional (cf. Netterstrom et al., 2008) or reversed or reciprocal association (e.g. de Lange et al., 2004; Waldenström et al., 2003) between the JDC and JDC-S models and depression controlled for neuroticism or other personality measures. To assess neuroticism, we made use of Saucier’s Big Five Mini-Markers (Saucier, 1994), 40 adjectives which measure five aspects of personality (eight for each aspect): neuroticism, extraversion, agreeableness, conscientiousness, and openness to experience. The eight adjectives applying to neuroticism are fretful, relaxed, moody, temperamental, envious, jealous, touchy, and not envious. Subjects are asked to indicate how accurately or inaccurately these adjectives describe them, on a 9-point Likert scale ranging from 1 (extremely inaccurate) to 9 (extremely accurate) (alpha = .72). The neuroticism factor has been validated repeatedly in many cross-cultural studies (e.g. Yoon, Schmidt, & Ilies, 2002) in many languages, including Hebrew (Hendriks & Perugini, 2003), conducted by many different researchers on a wide range of data sources, samples, and assessment instruments (John, 1990). There is evidence that neuroticism is stable (r = .64) across 20 years (Clark & Watson, 1991; Costa & McCrae, 1986).

A substantial body of evidence links socioeconomic status (SES) to depression. In the current study, we controlled for one of the most important determinants of SES, educational level. Recent studies (for a meta-analysis see Cole & Dendukuri, 2003) show that persons with low educational level are at a higher risk of depression. Furthermore, a recent review (Baum, Garofalo, & Yali, 1999) shows that low socioeconomic status is associated with greater stress. We also controlled for age. There has been little consensus in the literature regarding age differences in depression (cf. Jorm, 2000). Furthermore, there are some indications that as people get older, they experience less occupational stress (Rook, Dooley, & Catalano, 1991). Similarly, marital status has been found to be related to stress (Luecken et al., 1997; Thoits, 2006). The literature consistently reports that married working women with one or more children experience higher stress levels than single men or women, and married men and women without children (Davidson & Fielden, 1999; Luecken et al., 1997). Accordingly, we decided to control for marital status and number of children. Our decision to control job seniority in the analyses is based on recent evidence of a higher order moderating effect of job seniority on the JDC-S model–strain relation (e.g. Bradley, 2007). Age, educational level (number of years of education), seniority, marital status, and number of children were taken at T1 from the demographic details as reported by the respondents.

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Statistical Analysis

To examine changes in depressive symptoms or in each of the JDC-S model components over time, we used ordinary least squares (OLS) regressions. Adding baseline levels of the predictors to the regression equation enables the measurement of change in the criterion (Twisk, 2003). By including baseline levels of our variables in all our analyses, we avoided the well-known artifacts of using change scores (Taris, 2000). Following current standards (Cortina, 1993) in each OLS regression analysis, we tested the possibility of nonlinear relationships between the predictors and the criterion by testing if the quadratic term of each predictor was a significant predictor of the criterion. Furthermore, in predicting T2 and T3 depressive symptoms, we systematically tested the interactive hypothesis of the JDC-S model represented by the two-way interactions of the JDC-S components. We also tested their three-way interaction. In order to test the three-way interaction, all the possibilities of the two-way interactions were forced to enter into the regression first. To reduce the possibility of multicollinearity among the interaction and quadratic terms and their component predictors, all predictors were centered prior to the regression runs (Aiken & West, 1992). Because none of the nonlinear terms was significant, we did not include them or the non-significant interactions in Tables 2–3 (cf. Cortina, 1993).

RESULTS

There are several noteworthy findings regarding the descriptive statistics of the study variables (see Table 1). Women reported higher levels of depressive symptoms and worse work characteristics than men almost consistently in all three measurement waves, which is consistent with the trend found in the literature (Godin et al., 2009).

The across-time correlations for depressive symptoms, workload, job control, and social support were high (rs range = .47–.69) for women and men for both time lags. Furthermore, these correlations are consistent with those reported in the past studies reviewed above and in meta-analytic studies (Thoresen, Kaplan, Barsky, Warren, & de Chermont, 2003). The correlations between the JDC-S model components (workload, job control, and social support) and depressive symptoms are somewhat higher for men than for women in nearly all measurement waves. The correlations between neuroticism and the study variables are in the expected direction (i.e. neuroticism is positively correlated with depressive symptoms and workload, and negatively correlated with job control and social support).

The results of the regression analyses conducted for men to test whether changes in the components of the JDC-S model over time predict changes in depressive symptoms levels over time (Hypotheses 1a, 1b) are presented in
| Measure                  | 1   | 2   | 3   | 4   | 5   | 6   | 7   | 8   | 9   | 10  | 11  | 12  | 13  | 14  | 15  | 16  | 17  | 18  | 19  | 20  | α   | M   | SD  |
|-------------------------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|
| 1. Depression, T3       | — .57* | .47* | .12* | .11* | .08 | .16 | .18 | .15* | .21* | .08 | .18 | .27 | .15* | .05 | .10 | .03 | .10 | .03 | .03 | .80 | 1.33 | .35 |
| 2. Depression, T2       | .62* | — .53* | .20* | .15* | .12* | .08 | .11 | .09 | .21* | .15* | .14* | .34* | .11 | .06 | .21* | .08 | .05 | .01 | .10 | .76 | 1.31 | .43 |
| 3. Depression, T1       | .59* | .63* | — .09 | .07 | .18 | — .10 | .13 | .17* | .18 | .13* | .21* | .34* | .05 | .03 | .01 | .04 | .01 | .01 | .08 | 1.38 | .43 |
| 4. Workload, T3         | .20* | .21* | .20* | — .56* | .53* | .03 | .02 | .08 | .24* | .15* | .20* | .10 | .04 | .11* | .05 | .08 | .08 | .11 | .01 | .93 | 2.65 | 1.02 |
| 5. Workload, T2         | .17* | .17* | .14* | .69* | — .63* | .01 | .03 | .08 | .12* | .25* | .10 | .06 | .18* | .05 | .11 | .08 | .01 | .05 | .10 | .89 | 2.72 | 1.06 |
| 6. Workload, T1         | .05 | .10* | .07 | .54* | .62* | — .06 | .01 | .13* | .13* | .20* | .11 | .13* | .06 | .03 | .03 | .12 | .11 | .07 | .04 | .93 | 3.03 | .92 |
| 7. Job control, T3      | — .17* | — .15* | .14* | .02 | .01 | .06 | — .56* | .49* | .45* | .32* | .24* | — .11 | .03 | .11 | .06 | .08 | .13* | .08 | .93 | 3.75 | .87 |
| 8. Job control, T2      | .22* | .21* | .14* | .01 | .02 | .05 | .68* | — .63* | .29* | .38* | .31* | .04 | .09 | .20* | .04 | .03 | .01 | .05 | .08 | .94 | 3.75 | .89 |
| 9. Job control, T1      | .19* | .09 | — .11* | .04 | .04 | .09 | .57* | — .19* | .24* | .18* | .20* | .11 | .18* | .13 | .12 | .11 | .01 | .03 | .93 | 3.73 | .88 |
| 10. Social support, T3  | .32* | .31* | .21* | — .13* | .09 | .38* | .29* | .23* | — .48* | .49* | .24* | — .04 | .01 | .01 | .01 | .01 | .08 | .09 | .88 | 3.92 | .65 |
| 11. Social support, T2  | .27* | .32* | — .19* | — .19* | .14* | .25* | .27* | .20* | — .54* | .11 | .07 | .05 | .02 | .01 | .18* | .24* | .89 | 3.84 | .72 |
| 12. Social support, T1  | .21* | .24* | .19* | .19* | .19* | .56* | .26* | .59* | — .10 | .08 | .04 | .06 | .04 | .02 | .01 | .04 | .08 | 3.80 | .68 |
| 13. Neuroticism         | .24* | .24* | .11* | .10* | .10* | .03 | .02 | .01 | .23* | .21* | .20* | — .08 | .01 | .03 | .03 | .09 | .03 | .04 | .71 | 2.59 | 1.26 |
| 14. Age                 | .02 | .02 | .03 | .21* | .26* | .20* | .17* | .20* | .21* | .03 | .03 | .07 | .05 | — | .14* | .58* | .08 | .46* | .01 | .03 | 46.69 | 9.72 |
| 15. Education           | — .11* | .03 | — .10* | .04 | .01 | .06 | .14* | .17* | .14* | .04 | .05 | .12* | .01 | .04 | — | .15* | .04 | .07 | .03 | .01 | 15.37 | 2.88 |
| 16. Seniority           | .04 | .03 | .09* | .10* | .14* | .04 | .13* | .11* | .11* | .09 | .05 | .08 | .02 | .56* | .08 | — | .04 | .31* | .04 | .06 | 15.81 | 10.42 |
| 17. Marital status      | — .04 | .05 | .07 | .05 | .06 | .14* | .17* | .14* | .04 | — .05 | .12* | .01 | .04 | — | .15* | .04 | .07 | .03 | .01 | 15.37 | 2.88 |
| 18. Children (number)   | — .04 | — .10* | .04 | .01 | .01 | .02 | .23* | .21* | .20* | .12* | .08 | .12* | .04 | .44* | .01 | .23* | .38* | — | .08 | .10 | 2.30 | 1.23 |
| 19. Time lag T1–T3      | .06 | .06 | .05 | .12* | .04 | .02 | .11* | .11* | .06 | .13* | .04 | .02 | .21 | .01 | .01 | — | .03 | .05 | — | .85* | 1267.03 | 431.13 |
| 20. Time lag T1–T2      | .04 | .01 | .03 | .08 | .05 | .01 | .01 | .07 | .12* | .04 | .12* | .02 | .15* | .05 | .04 | .01 | .03 | — | .84 | 639.68 | 245.38 |
| α                       | .78 | .75 | .76 | .93 | .93 | .92 | .94 | .93 | .91 | .90 | .89 | .87 | .74 | — | — | — | — | — | — | — | — | — |
| M                       | 1.20 | 1.19 | 1.20 | 2.52 | 2.60 | 2.94 | 4.10 | 4.05 | 4.08 | 4.00 | 3.92 | 3.77 | 2.55 | 45.78 | 16.12 | 13.42 | .90 | 2.51 | 1172.05 | 592.93 |
| SD                      | .29 | .27 | .30 | 1.03 | 1.02 | .94 | .71 | .73 | .69 | .65 | .61 | 1.20 | 9.21 | 3.07 | 9.55 | .29 | 1.17 | 409.99 | 251.96 |

Note: Entries above the diagonal represent the female employees' parameters, and those below the diagonal represent the male employees' parameters. The symbol α represents Cronbach's alpha; * p < .05.
Table 2. Also included in both regression equations was the neuroticism personality factor, as well as all other control variables listed above. The time lag T1–T2, changes in workload, and job control levels significantly predicted a change in depressive symptoms levels in the expected direction ($\beta = .12$, $-.13$ respectively). Although a change in social support levels did not predict a change in depressive symptoms levels, contrary to the additive hypothesis (Hypothesis 1a), a significant two-way interaction was found. Social support moderated the association between workload and depressive symptoms, such that the higher the social support, the lower the association between workload and depressive symptoms, partly supporting Hypothesis 1b. For the second time frame examined, T1–T3, we found that change in workload positively predicted T3 depressive symptoms levels, whereas, as expected, the opposite was true for both job control and social support. Thus, there was partial support for our Hypothesis 1a. Furthermore, partly supporting Hypothesis 1b, a significant two-way interaction between workload and perceived control was found such that perceived control moderated the

<table>
<thead>
<tr>
<th>Measure</th>
<th>Time lag T1–T2</th>
<th>Time lag T1–T3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B (SEB) $\beta$</td>
<td>B (SEB) $\beta$</td>
</tr>
<tr>
<td>Depressive symptoms, T1</td>
<td>.54 (.04) .56</td>
<td>.47* (.04) .48</td>
</tr>
<tr>
<td>Neuroticism, T1</td>
<td>.02* (.01) .07</td>
<td>.03* (.01) .12</td>
</tr>
<tr>
<td>Age</td>
<td>.01 (.01) .02</td>
<td>.01 (.01) .08</td>
</tr>
<tr>
<td>Education</td>
<td>.01 (.01) .04</td>
<td>-.01 (.01) -.07</td>
</tr>
<tr>
<td>Follow-up durationa</td>
<td>.01 (.01) .03</td>
<td>.01 (.01) .01</td>
</tr>
<tr>
<td>Total $R^2$</td>
<td>Total $R^2$(adjusted) = .43</td>
<td>Total $R^2$(adjusted) = .37</td>
</tr>
<tr>
<td>Workload, T1</td>
<td>-.01 (.01) -.02</td>
<td>-.01 (.01) -.03</td>
</tr>
<tr>
<td>Job control, T1</td>
<td>.02 (.02) .06</td>
<td>-.03 (.02) -.07</td>
</tr>
<tr>
<td>Social support, T1</td>
<td>-.01 (.02) -.01</td>
<td>.02 (.02) .05</td>
</tr>
<tr>
<td>Workload, T2/T3</td>
<td>.03* (.01) .12</td>
<td>.03* (.01) .11</td>
</tr>
<tr>
<td>Job control, T2/T3</td>
<td>-.05* (.02) -.13</td>
<td>-.04* (.02) -.10</td>
</tr>
<tr>
<td>Social support, T2/T3</td>
<td>-.04 (.02) -.09</td>
<td>-.05* (.02) -.11</td>
</tr>
<tr>
<td>Workload $\times$ Social support</td>
<td>-.03* (.01) -.08</td>
<td></td>
</tr>
<tr>
<td>Workload $\times$ Job control</td>
<td></td>
<td>-.05* (.01) -.14</td>
</tr>
<tr>
<td>Total $R^2$</td>
<td>Total $R^2$(adjusted) = .44</td>
<td>Total $R^2$(adjusted) = .39</td>
</tr>
</tbody>
</table>

Note: B and $\beta$ represent the nonstandardised and standardised partial regression coefficients, respectively; SEB stands for the standard error of the former. * $p < .05$; + in months; T2/T3 presents the measure at T2 and T3 (for time lags T1–T2 and T1–T3, respectively).

The control variables seniority, marital status, and number of children (not shown here) were also included in these regression analyses.
### TABLE 3

Multiple Regression of Each of the Components of the JDC-S Model on Depressive Symptoms at T1 and T2 or T3, the Men’s Sample (N = 470)

<table>
<thead>
<tr>
<th>Measure</th>
<th>Workload</th>
<th></th>
<th>Job control</th>
<th></th>
<th>Social support</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Time lag T1–T2</td>
<td>Time lag T1–T3</td>
<td>Time lag T1–T2</td>
<td>Time lag T1–T3</td>
<td>Time lag T1–T2</td>
<td>Time lag T1–T3</td>
</tr>
<tr>
<td>The criterion at T1&lt;sup&gt;b&lt;/sup&gt;</td>
<td>.62*</td>
<td>.04</td>
<td>.58</td>
<td>.56*</td>
<td>.04</td>
<td>.51</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>.02</td>
<td>.03</td>
<td>.02</td>
<td>.01</td>
<td>.03</td>
<td>.01</td>
</tr>
<tr>
<td>Age</td>
<td>−.02*</td>
<td>.05</td>
<td>−.15</td>
<td>−.01*</td>
<td>.01</td>
<td>−.10</td>
</tr>
<tr>
<td>Education</td>
<td>.02</td>
<td>.02</td>
<td>.04</td>
<td>.01</td>
<td>.01</td>
<td>.01</td>
</tr>
<tr>
<td>Follow-up duration&lt;sup&gt;a&lt;/sup&gt;</td>
<td>−.01</td>
<td>.01</td>
<td>−.02</td>
<td>.01</td>
<td>.01</td>
<td>.01</td>
</tr>
<tr>
<td>Total R&lt;sup&gt;c&lt;/sup&gt;</td>
<td>.18</td>
<td>.15</td>
<td>.05</td>
<td>.36*</td>
<td>.16</td>
<td>.10</td>
</tr>
<tr>
<td>Depressive symptoms, T1</td>
<td>.45*</td>
<td>.16</td>
<td>.12</td>
<td>.36*</td>
<td>.16</td>
<td>.10</td>
</tr>
<tr>
<td>Total R&lt;sup&gt;c&lt;/sup&gt;</td>
<td>.42</td>
<td>.33</td>
<td>.44</td>
<td>.37</td>
<td>.39</td>
<td>.36</td>
</tr>
</tbody>
</table>

Note: B and β represent the nonstandardised and standardised partial regression coefficients, respectively; SEB stands for the standard error of the former. * p < .05; <sup>a</sup> in months; <sup>c</sup> adjusted for degrees of freedom; T2/T3 presents the measure at T2 and T3 (for time lags T1–T2 and T1–T3, respectively); <sup>b</sup> Workload, T1; Job control, T1; Social support, T1, respectively; The control variables seniority, marital status, and number of children (not shown here) were also included in these regression analyses.
association between workload and the change in depressive symptoms levels: the higher the job control, the lower the association between workload and depressive symptoms. The JDC-S model components explained 1 per cent and 2 per cent of change in depressive symptoms levels ($p < .05$; for time lags T1–T2 and T1–T3, respectively). In contrast to the significant results observed for men, we found that none of the JDC-S model components predicted change in depressive symptoms level (in both time lags) for women. (In order to save journal space the results for the women’s sample are not shown here, but are available upon request.)

A set of regression analyses was applied to the data in order to examine the reversed causal relationship, whether changes in depressive symptoms levels with time predict T2 or T3 changes in the levels of all components of the JDC-S model while controlling for the respective criteria at T1 and the same variables as before. The results for men (see Table 3) show that changes in depressive symptoms across time are positively associated with changes in workload, and negatively associated with changes in job control and social support in both time lags (T1–T2 and T1–T3). Again none of these results were significant for women. (These results are not shown here, but are available upon request.)

We compared equality CIs of the $b$s of each predictor across the two waves and found that their effect is not significantly different (Cohen, Cohen, West, & Aiken, 2003). These results indicate that there are no systematic differences in the effect sizes of the shorter (T1–T2, 17 months) and the longer (T1–T3, 3 years) time lags.

**DISCUSSION**

The current longitudinal study improves upon and extends previous longitudinal studies on the JDC-S model and depressive symptoms in several important ways. First, we examined how changes in work characteristics affect changes in depressive symptoms across time. Second, we systematically investigated the reverse causation hypothesis. Third, we tested our hypotheses separately for women and men. Fourth, we provided a constructive replication for our results by comparing two different time frames. Additionally, we tested the four study hypotheses while controlling for theoretically important confounders, including the personality disposition of neuroticism. Thus, to the best of our knowledge, our study pioneered in investigating the directionality of the across-time relations of changes in the levels of workload, job control, and social support at work on changes in depressive symptoms levels, and vice versa, separately for women and men. These features of our study provide support to our inferences of causality.

The results of the OLS regression analyses indicate marked gender differences. A change in the JDC-S model components predicted change in
depressive symptoms levels only for the men. Increase in job demands (workload) and decrease in job control predicted an increase in depressive symptoms levels with time in both time lags, while decrease in social support predicted elevation in depressive symptoms levels only in the longer time lag (T1–T3). Thus, it can be concluded that the male sample results provide strong but not full support for the iso-strain (additive) hypothesis (Hypothesis 1a).

Contrary to our expectation in this regard, we found only partial support for the moderating hypothesis (van der Doef & Maes, 1998) (Hypothesis 1b). Only one of four interactive terms tested in each of the time lags turned out to be significant. While social support moderated the workload–depressive symptoms association in the T1–T2 time lag, job control moderated the workload–depressive symptoms association in the T1–T3 time lag. These ambivalent results are congruent with most longitudinal studies on this issue, which fail to produce the full range of two-way and three-way interaction effects proposed by the JDC-S model (de Lange et al., 2003).

A marked gender difference was uncovered also upon testing the hypothesised reversed causal relationship between the JDC-S model and depressive symptoms. While the reversed causal relationship was not confirmed for the female sample, the results for the male sample support the possibility of a reversed causal relationship between the JDC-S model and depressive symptoms. Increased depressive symptoms levels were found to positively predict changes in workload over time, and to negatively predict changes in job control and social support levels with time for both time lags. Thus, Hypothesis 2 was confirmed only for the male sample.

Theorising on possible underlying mechanisms of the reversed relationship between stressors and health is limited (Zapf et al., 1996). Zapf et al. have suggested that the existence of such a relationship can be explained by several different processes: (a) the depressed worker perceives his or her job more negatively (a perceptual change), what de Lange, Taris, Kompier, Houtman, and Bongers (2005) have termed the gloomy perception mechanism; (b) a “true strain–stressor” hypothesis (i.e. sometimes stressors are in fact influenced by outcomes); (c) the depressed worker “drifts off” to a more negative work environment as a result of job transfer or changes within the same job. We can rule out at least the explanation of job transfer, as we included in our study sample only workers who did not change their job throughout the follow-up period. As we argued above, we maintain that the reversed causal relationship can be understood based on COR theory. People feel depressed when they perceive a continuous net loss of energetic coping resources which cannot be replenished. Depressed individuals may further exacerbate their losses by entering an escalating spiral of losses (Hobfoll & Shirom, 2000) leading to depleted ability to cope with job demands and perceived further loss of important resources such as perceived control and social support at work. Thus, the results of the present study suggest the existence of a dynamic

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vicious circle for the male sample. Job strain and depressive symptoms are reciprocally related and thus reinforce each other across time, and this partly accounts for the stability of the JDC-S model components and of depressive symptoms across time, as noted in the Results section.

We have found no systematic differences in the impact of the work characteristics on depressive symptoms between the 17-month time lag (T1–T2) and the 3-year time lag (T1–T3). This suggests that adverse work characteristics result in worsening of depressive symptoms within one and half years of follow-up and the elevated symptoms are maintained over 3 years of follow-up. It still remains to be tested whether a longer follow-up period would uncover an additional increase in depressive symptoms. Our results are congruent with the conclusion of a recent review (Bonde, 2008) that elevated risk for depression is not dependent on the duration of the follow-up period. In addition, we also uncovered support for the hypothesised reversed casual relationship between the JDC-S components and depression irrespective of the time lag examined. These findings are in contrast to those reported by de Lange et al. (2004) that the strongest reciprocal relationship between work characteristics and a composite measure of mental health was found for a 1-year time lag compared with 2 and 3 years. Thus, further studies that focus on depressive symptoms are needed to examine the long-term reciprocal relationship with adverse work characteristics.

As expected, adverse changes across time in workload, job control, and social support at work were significant predictors of increased depressive symptoms, for men but not for women. This finding replicates and reinforces the finding of the single past study which examined the impact of changes in work characteristics on mental health (Stansfeld et al., 1999). This study found that unfavorable changes in job demands and decision authority resulted in higher risk of psychiatric syndromes in men but not in women. Taken together, these findings suggest that worsening of work characteristics has an impact on the mental health of men but not of women.

One possible explanation for such an outcome is that men’s identity is tied more to their role at work than to their role at home (Griffin, Fuhrera, Stansfeld, & Marmot, 2002), they are expected to be more vulnerable to work characteristics, to perceive them as more threatening, and to be more adversely affected by them. Matthews, Power, and Stansfeld (2001), who studied the link of home and work factors with psychological distress, have found that work factors had a greater impact on social class differences in psychological distress on men. They have suggested that “work may be more important for men than women”. Women’s health and well-being may be more related to beneficial/stressful factors at home. Supporting evidence comes from a recent study of a large cohort of Belgian workers (Godin et al., 2009). It was found that high job strain and work dissatisfaction is predictive of clinically diagnosed depression in men, while in women this outcome was
predicted by private life dissatisfaction. In a similar vein, a prospective study conducted in a Swedish general population has shown that when both occupational and non-occupational factors were included in multivariate analysis, non-occupational factors remained significantly associated with symptoms of sub-clinical depression for women but not for men (Bildt & Michelsen, 2002). Another reason is that the impact of work characteristics on mental health varies for men and women (Stansfeld & Candy, 2006). These authors have also suggested that the perception of emotional support may vary between men and women, and that women tend to seek more social support outside work than men. This perhaps may explain our finding that social support at work is a protective factor and more strongly and negatively associated with depressive symptoms in men than in women (see Table 1). This latter finding is also consistent with the information that outside the workplace, women are more likely to receive and benefit from social support (Hobfoll & Stokes, 1988), whereas inside the workplace women receive and benefit less from social support than men (Geller & Hobfoll, 1994).

It is more difficult to use the above line of reasoning to explain our finding that the reverse causal link between depressive symptoms and the JDC-S model holds only for men. Following the COR theory we might speculate that relative to men, women experiencing depressive symptoms may invest more resources to replenish their lost resources at home and thus gain more resources at home than men, so that their work resources are less affected. Furthermore, women may be interchanging resources more freely across the home–work domain. Given the exploratory nature of our study there is a need for further studies to test these speculations. In these studies, home resources such as social support should be measured to allow testing these possibilities.

As expected, we also found moderate correlations between depressive symptoms and neuroticism, for both women and men in both time lags (rs range = .27–.37). Thus, another noteworthy finding here is that the reciprocal relationship between the JDS-C components and depressive symptoms in the men’s sample remained significant even after adjusting for neuroticism, which is consistent with similar findings in other studies (cf. Netterstrom et al., 2008). This partially overcomes the shortcoming of this study that both measures of the JDC-S model components and depressive symptoms are based on self-reports.

Limitations

Our study has additional limitations. First, our findings could be biased because of the well-known “healthy worker effect”, which refers, inter alia, to the possibility that employees with elevated levels of depressive symptoms decided to change their place of work or stopped working, leaving their healthier colleagues to participate in our research. We assume that this had
probably already occurred before our T1 (Richardson, Wing, Steenland, & McKelvey, 2004). Second, this study is based on self-report measures, which carry the risk of bias, due, for example, to personality traits such as negative affectivity or neuroticism. We believe that we partially corrected for this possibility by adjusting for neuroticism and by using a longitudinal design which focused on changes in our predictors and criteria.

Third, 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 depressive symptoms and linkages with the JDC-S model will be replicated among less resilient respondents.

**Strengths**

The present study applying a full panel design was set to meet the requirements for high-quality studies outlined by de Lange et al. (2003). The components of the JDC-S model and depressive symptoms were all assessed three times, and in two different time lags, thus providing a constructive replication for our results. This allowed us to test the effect of changes in the levels of workload, job control, and social support at work on changes in depressive symptoms levels, and vice versa. We also tested possible interactions between the study variables. Furthermore, we tested and confirmed the possibility that gender could be a mediator of the examined linkages. We tested these relationships in a multi-occupational sample of men and women who stayed in the same job throughout the follow-up period. We excluded respondents afflicted with chronic medical conditions and those using antipsychotic medication or antidepressants which could influence their depressive symptoms levels. Finally, we controlled for neuroticism (a global measure of negative affectivity). Negative affectivity is a potential confounder of the association between job stress and employee outcomes and ought to be controlled for (Judge, Erez, & Thoresen, 2000). As indicated earlier, most of the studies cited above did not control for this personality factor.

**Implications**

The findings of the present full panel design study suggest no predominance of the direct casual relationship between the JDC-S model components and depressive symptoms compared with the reverse relationship. This supports a dynamic view of the bi-directional association between work characteristics
and depressive symptoms in which they mutually influence each other, at least for the men. Thus, the original unidirectional JDC-S model appears too narrow. Karasek and Theorell—the original proponents of the JDC-S model examined here (1990, p. 99)—underscored the importance of using a broader perspective for the relationship between work and health, and proposed a dynamic version of the model that integrates environmental effects with person-based information. Our major findings support this dynamic view in that we found that prolonged exposure to work (and life) stressors may lead to depressive symptoms and that the higher the level of depressive symptomatology among employees, the more likely they are to appraise their job conditions as over-demanding. They may also judge the resources to deal with these demands as lacking, and as proposed in the COR theory this will lead to an escalating spiral of resource losses which cannot be easily replenished, resulting in deepened feelings of emotional distress.

Further longitudinal studies employing a longer time frame with several waves of measurements are needed to explore how the reciprocal relationship between the JDC-S model and depressive symptomatology unfolds with time, separately for women and men. In the present study we included only workers who stayed in the same job during the follow-up period. However, it would be interesting to follow up workers who have changed their jobs or place of work, in order to explore the possibility that depressive symptomatology may become autonomous and chronic even in the face of a change of job conditions or even with a change of job.

Yet another implication of the integrated findings of the present study and similar studies reviewed here is that interventions to combat depressive symptomatology may be more effective if they employ simultaneously both organisational-level interventions (aimed at improving job characteristics and work conditions) and individual-level interventions to ameliorate depressive symptomatology (by using, for example, stress management techniques, such as cognitive behavioral therapy). Focusing on organisational-level interventions may not be sufficient. Depressed workers may not benefit, for example, from the creation of work autonomy groups whose members are provided with higher decision latitude and a higher sense of control. They may still perceive their job control as low. Further studies are needed to explore such a possibility. It is beyond the scope of the present paper to review organisational interventions to combat depression, but there are studies which point to their effectiveness in elevating or even preventing depression (for organisational intervention reviews, see Couser, 2008; Marine, Ruotsalainen, Serra, & Verbeek, 2006). However, it could be that integrative interventions, both individually based (alleviating depressive symptomatology) and job-characteristics focused (reducing job demands, increasing control), would be more effective relative to those that focus on only one of these dimensions.

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REFERENCES


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