

Research paper

Stressful life events and depressive symptoms in mothers and fathers of young children

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ABSTRACT

Background: Parents of young children generally report more depressive symptoms than parents of adult children or people without children, mainly because the presence of young children increases exposure to significant stressors (such as stressful life events). However, most studies on the depressogenic role of stressful life events in parents of young children have focussed on mothers.

Methods: Using data from 1138 families with young children in Norway, we investigated gender differences in the effect of stressful life events after a child's birth on the development of parental depressive symptoms in 3 follow-ups at child's ages 3–6 years. We also explored if gender differences in disposition (personality) may explain any gender differences in the depressogenic effect of life events.

Results: Nesting parents within families, we found a female gender bias for both neuroticism and depressive symptoms but no gender difference in the number of life events reported. Importantly, the number of stressful life events predicted the level and course of depressive symptoms similarly for mothers and fathers. Personality traits did not change the association between stressful life events and depressive symptoms in either mothers or fathers.

Limitations: Given the study design, causality cannot be inferred.

Conclusions: There was no gender difference in the depressogenic effect of stressful life events in our sample. There was no evidence for a female dispositional sensitivity to the depressogenic effect of stressful life events, either. Stressful life events put both mothers and fathers of young children at risk of depression.

1. Introduction

Depression is associated with a number of adverse cognitive, social and physical outcomes in both men and women (Moussavi et al., 2007). Depression in men and women with young children has attracted much interest because early exposure to parental depression is a powerful risk factor of poor child outcomes (Cummings et al., 2005; Goodman et al., 2011) and because the prevalence of depression is, in general, higher in parents of young children than in parents of adult children or people without children (Umberson et al., 2010). The primary explanation is that the presence of young children increases exposure to significant stressors, including the daily demands and time constraints of parenting, increased strain between parents, and work-family conflict (Evenson and Simon, 2005).

However, with the exception of few studies in the postpartum period (e.g., Escribà-Agüir and Artazcoz, 2011; Kamalifard et al., 2014), most of the research into the effect of stressors on depressive symptoms in parents of young children has focussed on mothers. We know very

little about the effect of stressors on depressive symptoms in fathers of young children. We also know little about gender differences in this effect, although it is increasingly recognised that the depressogenic impact of stressors may vary substantially for men and women (Kendler and Gardner, 2014; Kendler et al., 2001). We carried out this study to attempt to fill this gap. In a large cohort of more than 1000 families with young children in Norway, we explored gender differences in the effect of the number of stressful life events after a child's birth on the development of parental depressive symptoms in 3 follow-ups at child's ages 3 to around 6 years.

We also explored gender differences in the effect of the interaction between personality [neuroticism and extraversion, which, according to previous research, are associated with depression (Klein et al., 2011)] and number of stressful life events on this development. Typically, depression is seen as the result of an interaction between psychosocial stressors (such as stressful life events) and diatheses (genetic liability). Diatheses (sometimes simply approximated by personality traits) influence the risk of onset of depression, in part by altering the

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sensitivity of individuals to the depression-inducing effect of stressful life events (Hammen, 2005). Stressors have been shown to interact with both personality and gender to predict depression (Kendler et al., 2004), but, to our knowledge, no study has yet explored this three-way interaction in predicting depression in parents of young children. The findings of such an interaction analysis will have important implications for etiological models of depression as they will suggest a) if the impact of number of stressors on depressive symptoms is independent of the level of neuroticism/extraversion or whether it changes with the level of neuroticism/extraversion, b) if the impact of neuroticism/extraversion on depressive symptoms is independent of the number of stressors or whether it changes with the number of stressors, and c) if there are gender differences in these phenomena.

2. Methods

2.1. Participants and procedure

The data were drawn from the longitudinal Behavior Outlook Norwegian Developmental Study (BONDS), which tracks the development of 1159 children (559 girls) from 6 months. BONDS was approved by the Norwegian Social Science Data Services and the Regional Committee for Medical and Health Research Ethics. Recruitment took place through child health clinics in five municipalities in southeast Norway in 2006–2008. In Norway child health clinics are public and almost universally attended. Compared to the general Norwegian population, the BONDS sample was biased toward mothers with higher education, fewer immigrant parents, more firstborns and fewer single mothers (Nærde et al., 2014 for more details).

At the time of writing, follow-up assessments were conducted at children's ages 1, 2, 3, 4 and 5 years and at first grade (i.e., at the beginning of primary school, at around age 6 years). Participation rates were very high, at 98%, 95%, 92%, 93%, 84% and 82%, respectively. Data were collected through interviews with parents, except for the 5-year follow-up which was conducted by telephone. Both mothers and fathers were invited to the first interview at 6 months whereas fathers were targeted at 1 and 3 years and mothers at 2 and 4 years. The telephone interview at age 5 was conducted with either parent, and while fathers were targeted for the interview at the beginning of school, mothers were also asked to fill out a questionnaire. If the targeted parent was not able to take part in an interview, the other parent was asked to participate instead. In view of the objectives of the current study, the analytic sample ($N = 1138$) consisted of all BONDS families where both parents (living together or not) had completed at least one interview. Two families were excluded because the mother had died, 17 because the father did not live with the target child and did not participate in the study, and two because they withdrew their consent.

2.2. Measures

All measures described below were collected through self-report questionnaires at the interview assessments, and were available for both mothers and fathers.

2.2.1. Stressful life events

In BONDS, stressful life events were first measured at child's age 2 years. However, we could not measure them for both parents at the same time because of the design of BONDS. Therefore, we measured life events in the past 12 months at child's age 2 for mothers and at child's age 3 for fathers. The events, identical for both parents, were: Problems with friends/family; Serious illness in the home; Death of someone close; Mental/physical/sexual abuse; Caused someone hurt or pain; Something terrible has happened to me but I don't want to talk about it; Problems with housing; Problems with work; Problems with child care; Problems with finances; Problems with physical health; Problems with partner relationship; Alcohol problems with someone in the home;

Health problems in partner; Health problems in children; Problems with raising child; Problems with balancing work and raising child; Pregnancy/birth; Miscarriage. A summative score for each parent was created to indicate the number of stressful life events experienced in the past 12 months.

2.2.2. Parents' personality traits

Personality traits (*extraversion* and *neuroticism*) were measured at the 6-month assessment with the 30-item version of the Eysenck Personality Questionnaire (EPQ; Eysenck and Eysenck, 1975; Eysenck and Tams, 1990). Extraversion and neuroticism were each measured with 10 dichotomous (yes/no) items.

2.2.3. Parents' depressive symptoms

Depressive symptoms were measured (as in Narayanan, and Nærde, 2016) with six items [(on four-point scales ranging from 1 (not at all) to 4 (extremely)] of the 10-item version of the Hopkins Symptom Checklist (HSCL), an abbreviated version of the original HSCL-90 (Derogatis et al., 1974; Tams and Moum, 1993; Winokur et al., 1984). The HSCL asks about depressive symptoms in the last 2 weeks. In this study, we used data from the age 3, age 4 and first grade follow-ups, and we modelled symptoms on a continuum.

2.2.4. Covariates

These were: mother's and father's age (measured at child's age 2 years), mother's and father's education and ethnicity, presence of other young children (< 6 years old) in the family (measured at child's age 1 year), and history of depressive episodes (reported at child's ages 2 and 3 for mothers and fathers, respectively). A history of depressive episodes was defined as having ever experienced 3 or more symptoms (depressed/sad mood, no appetite or eating more than usual, lack of energy, feelings of worthlessness, and problems with attention or making decisions) for at least 2 weeks.

2.3. Analytic strategy

We modelled stressful life events as summative scores in line with the predominant research tradition in the field. To reduce measurement error we modelled depressive symptoms as latent variables loading on the HSCL depressive symptoms. It was not possible to model the personality traits as latent constructs, however, as the large number of parameter estimates caused severe delays and convergence problems. Therefore, we used instead the factor scores for neuroticism and extraversion derived from a confirmatory factor analysis (CFA) of the neuroticism and extraversion items. The factor scores for the two personality constructs were estimated jointly. The two factors were allowed to correlate with each other, but items were only allowed to load on one of the factors, and items loaded on one factor were not allowed to correlate with items loaded on the other.

CFAs were used to examine model fit for each of the latent variables before including them in the main analysis. Items (on, as explained, four-point scales ranging from 1 to 4) were included as categorical indicator variables and so the standard weighted least squares mean and variance adjusted (WLSMV) estimator was used for parameter estimation. Since depressive symptoms were measured for mothers and fathers and repeatedly over time we examined measurement invariance to ensure that factor loadings and thresholds in our CFAs were equal across time-points and parent genders. Invariance was tested using chi-square difference ($\Delta\chi^2$) tests. As $\Delta\chi^2$ is sensitive to sample size and is expected to become significant in large samples such as ours, we also examined the differences in comparative fit index (ΔCFI) and root mean square error of approximation ($\Delta RMSEA$). ΔCFI and $\Delta RMSEA$ are assumed to indicate sufficient measurement invariance when $< .01$ (Chen, 2007).

The main analysis focussed on the findings from a linear latent growth curve model (described in Fig. 1) fitted to describe and predict

Group Mothers versus Fathers

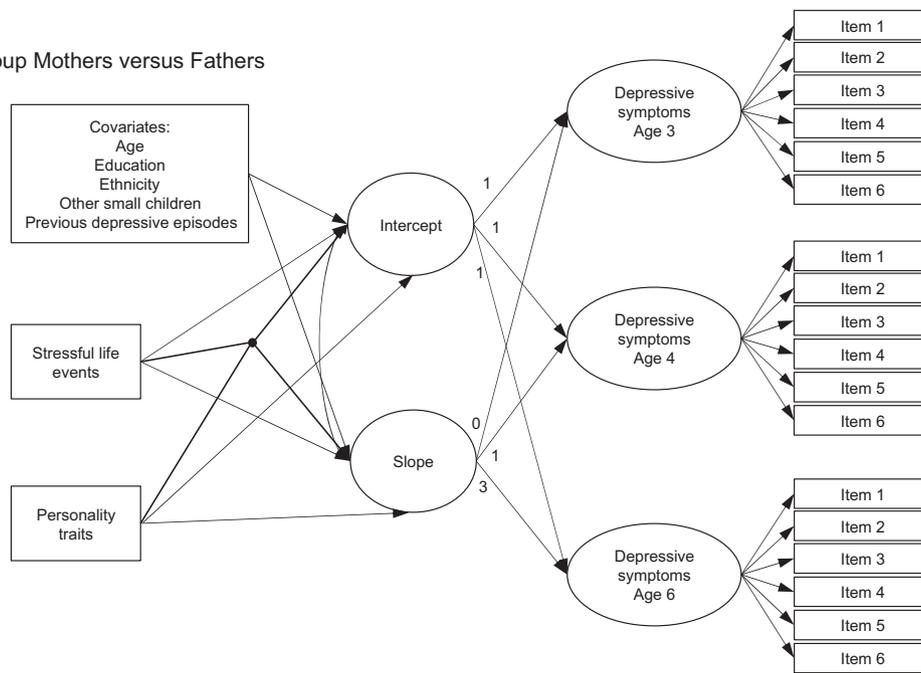


Fig. 1. Simplified illustration of the main analysis model. Note. Since we allowed for the correlation between intercept and slope (as shown), we did not allow the predictor variables to be also inter-correlated.

Table 1
Means (M) and standard deviations (SD) of all main variables, and correlations of key variables with depressive symptoms.

Variable	N	M	SD	Range	Bivariate correlations					
					1.	2.	3.	4.	5.	6.
Maternal depressive symptoms										
1. Age 3	315	1.43	0.52	1–3.8	1	.63***	.49***	.09	–.12	.09
2. Age 4	1024	1.35	0.44	1–4.0		1	.57***	.13**	.10*	.06
3. First grade	765	1.34	0.45	1–4.0			1	.06	.09	.09*
Paternal depressive symptoms										
4. Age 3	762	1.28	0.36	1–3.7				1	.55***	.43***
5. Age 4	550	1.26	0.39	1–3.5					1	.39***
6. First grade	654	1.22	0.32	1–3.0						1
Stressful life events										
Mothers (at child age 1–2)	1022	1.26	1.59	0–9	.43***	.30***	.24***	.17***	.12**	.10*
Fathers (at child age 2–3)	719	1.27	1.47	0–9	.13	.15***	.17***	.47***	.23***	.25***
Maternal personality traits										
Extraversion	1113	7.38	2.18	0–10	–.05	–.08*	–.10**	–.06	–.09*	–.02
Neuroticism	1113	3.70	2.51	0–10	.47***	.40***	.36***	.09*	.03	.07
Paternal personality traits										
Extraversion	674	7.34	2.20	0–10	.15	.004	–.01	–.07	–.07	–.15***
Neuroticism	674	2.29	2.14	0–10	.29**	.10*	.07	.44***	.37***	.35***

* p < .05.
** p < .01.
*** p < .001.

the development of maternal and paternal depressive symptoms over time. This allowed the estimation of the intercept (initial levels of symptoms) and the slope (linear change in symptoms over time). These latent growth factors were modelled to correlate freely. At each age, loadings on the intercept were fixed to 1, and those on the slope were defined as 0, 1 and 3 representing time periods in years between each time-point and the beginning of the study period (3 years), ranging in real time from 3 to 6 years. The role of stressful life events in the level and development of depressive symptoms for mothers and fathers was explored by adding regression paths from stressful life events to the intercept and slope. To test if personality moderated the effect of stressful life events on the level and development of depressive symptoms, interaction terms for stressful life events*neuroticism and stressful life events*extraversion were specified to predict the intercept and slope. To compare whether regression estimates differed for mothers and fathers, we used multiple group analyses with parent gender

as the group variable. Estimates were compared using the Wald chi-square test of parameter equalities. As the majority of the mothers and fathers in the study were nested in families, we used the child's ID number as a cluster variable. In our models throughout, we used paternal covariates in the models for paternal depressive symptoms, and maternal covariates in those for maternal depressive symptoms.

As explained, fathers were targeted for interviews at 3 and 6 years, and mothers at 4 years. Therefore, missingness by design alone was substantial. Missingness in depressive symptoms at 3, 4 and 6 years was 72%, 10% and 33% for mothers and 33%, 52% and 43% for fathers. Missing values for stressful life events were 10% for mothers and 37% for fathers, and missing values for extraversion and neuroticism were 2% for mothers and 41% for fathers. To deal with missingness, we performed multiple imputation with 25 replications in Mplus. All covariates and main variables were used as predictors in the imputation. Descriptive and correlation analyses were conducted in SPSS 22 and all

other analyses in Mplus 7.0 (Muthén and Muthén, 1998–2012). We report estimates for descriptive statistics and measurement models based on the complete data. Estimates for the growth curve models are pooled, based on the imputed data.

3. Results

3.1. Descriptive statistics

Means, standard deviations and bivariate correlations of all main (observed) variables are shown in Table 1, and descriptive statistics for covariates in Table 2. As the correlation coefficients in Table 1 show, depressive symptoms seemed to be more stable over time in mothers compared to fathers. However, there were also important gender similarities. Extraversion was not related to depressive symptoms in either, whereas neuroticism and stressful life events were similarly associated with depressive symptoms in both. As expected, mothers reported more depressive symptoms at all time-points (e.g., for age 3: $t = 5.46$, $df = 1075$, $p < .001$) and higher neuroticism scores ($t = 12.18$, $df = 1785$, $p < .001$), compared to fathers. However, mothers and fathers did not differ significantly either in extraversion ($t = 0.39$, $df = 1785$, $p = .69$) or in the number of stressful life events experienced ($t = 0.05$, $df = 1736$, $p = .96$). [We also attempted to explore gender differences in specific types of events but an exploratory factor analysis in our sample indicated no clear factor solutions (results available on request.)]

Fig. 2 presents symptom trajectories for illustrative cases of parents with different exposures to stressful life events (SLE), as follows: 1 = Mothers with 0 SLE (40.8%); 2 = Mothers with 1–2 SLE (41.5%); 3 = Mothers with $> = 3$ SLE (17.7%); 4 = Fathers with 0 SLE (38.0%); 5 = Fathers with 1–2 SLE (45.3%), and 6 = Fathers with $> = 3$ SLE (16.7%).¹ As can be seen, depressive symptoms seemed to follow similar longitudinal patterns in both parents, although levels of symptoms appeared to be higher in mothers. (Formal tests also showed that the slopes were significant and negative for both parents, and there were no significant differences between slopes.)

3.2. Measurement models

Model fit of parental depressive symptoms at all time-points was good ($\chi^2 = 901.245$, $df = 311$, $p < .001$, $CFI = .96$, $RMSEA = .05$, standardised factor loadings = .56 – .89). We were also able to establish strong (scalar) measurement invariance for maternal and paternal symptoms over time as well as between mothers and fathers ($\Delta\chi^2 = 191.943$, $df = 86$, $p < .001$, $\Delta CFI = .002$, $\Delta RMSEA = .005$). Good model fit was also found for the EPQ traits of neuroticism and extraversion ($\chi^2 = 889.148$, $df = 354$, $p < .001$, $CFI = .92$, $RMSEA = .04$). Standardised factor loadings ranged .26–.85 with 3 factor loadings below .50. As all factor loadings were significant we decided to keep the few items with low loadings, also conforming with the EPQ manual.

3.3. Growth curve models

The unadjusted linear growth curve model showing the development of depressive symptoms fitted the data well ($\chi^2 = 2277.304$, $df = 354$, $CFI = .93$, $RMSEA = .07$).² Intercepts (set by default to 0 for mothers) differed for mothers and fathers (mothers: 0, $SE = 0$; fathers: -0.23 , $SE = 0$; $Wald\ test\ value = 13.45$, $df = 1$, $p < .001$), suggesting that, as also shown by the descriptive analysis above, mothers had more depressive symptoms than fathers at the beginning of our study period

¹ The figure serves only as an illustration because, as we explained in the Analytic Strategy section, we did not use a categorical approach to modelling stressful life events.

² Visual inspection of Fig. 2 suggested the possibility of curvilinear trajectories of depressive symptoms, which we attempted to formally test for. However, we were not able to establish an adequate model fit, especially in fathers.

Table 2
Descriptive statistics for covariates.

Covariate	N	Valid %	M	SD
Age				
Mothers	1137		32.3	4.8
Fathers	1120		34.8	5.3
Ethnicity				
Mothers	1120			
Norwegian		87.0		
Foreign-born		13.0		
Fathers	898			
Norwegian		90.5		
Foreign-born		9.5		
Educational level				
Mothers	1129			
Primary or secondary education/high school		41.6		
College/university		58.3		
Fathers	1114			
Primary or secondary education/high school		52.9		
College/university		47.0		
Other small children	1081			
No		61.1		
Yes		38.9		
Previous depressive episodes				
Mothers	1042			
No		88.7		
Yes		11.3		
Fathers	733			
No		94.4		
Yes		5.6		

Note: Parents' age is reported at child's age 2 years.

(at child's age 3 years). Slopes for mothers and fathers were significant and negative but gentle (mothers: -0.06 , $SE = 0.01$; fathers: -0.06 , $SE = 0.02$), indicating that depressive symptoms in both parents were decreasing over time, although at a very slow rate. Slope estimates did not differ significantly between mothers and fathers.

We then examined whether the number of stressful life events predicted the intercept and slope of depressive symptoms before and after adjustment for covariates. These models showed acceptable fit (Model without covariates: $\chi^2 = 2182.649$, $df = 387$, $CFI = .93$, $RMSEA = .06$; Model with covariates: $\chi^2 = 2847.685$, $df = 611$, $CFI = .89$, $RMSEA = .06$). As can be seen in Table 3 which presents the model results, a larger number of stressful life events predicted higher initial levels of depressive symptoms in both mothers and fathers. Estimates did not differ significantly between mothers and fathers ($Wald\ test\ value = 0.93$, $df = 1$, $p = .34$). Stressful life events were also associated with change in depressive symptoms over time in fathers but not mothers ($p = .07$). Again, slope coefficients for mothers and fathers were not significantly different ($Wald\ test\ value = 0.00$, $df = 1$, $p = .99$).

Finally, we examined the role of personality in moderating the effect of stressful life events on depressive symptoms. After including interaction terms for stressful life events*neuroticism and stressful life events*extraversion, the model fit continued to be acceptable ($\chi^2 = 2753.633$, $df = 675$, $CFI = .89$, $RMSEA = .05$). As can be seen in Table 4, neither interaction term predicted the intercept or slope of depressive symptoms.

4. Discussion

This was the first study to explore gender differences in the effect of stressful life events on depressive symptoms in parents of young children. It was also the first study to test for gender differences in the effect of the interaction between stressful life events and personality traits on depressive symptoms in this population. Using data from a large, population-based cohort of some 1000 families with young children in Norway, we found that, in line with much previous evidence, there was a female gender bias for both neuroticism and depressive symptoms.

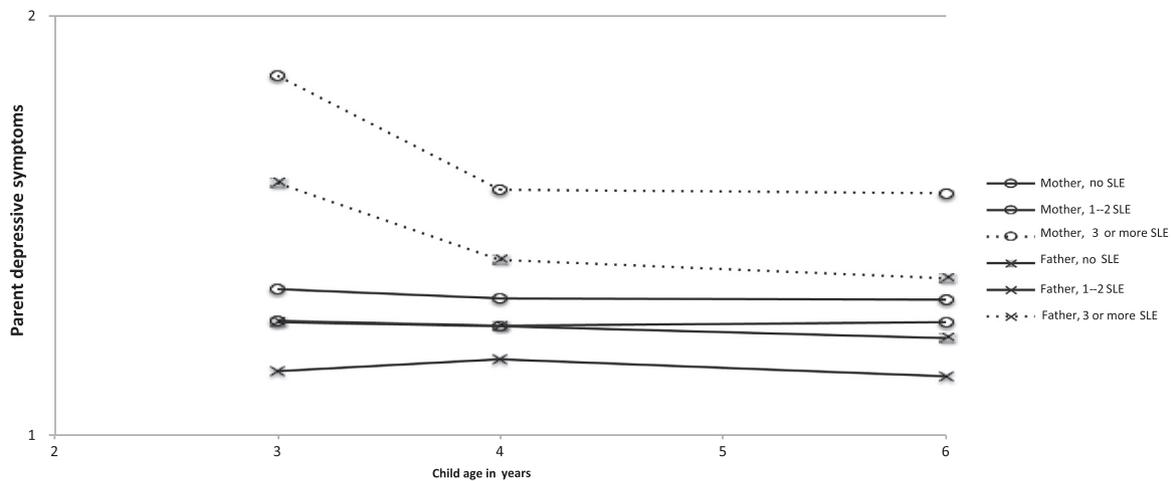


Fig. 2. Depressive symptoms by number of stressful life events for illustrative cases of mothers and fathers Note: SLE = Stressful life events.

Table 3
Regression estimates for stressful life events on the intercept (at child's age 3) and slope of parental depressive symptoms.

	Mothers						Fathers					
	Intercept			Slope			Intercept			Slope		
	B	SE	β	B	SE	B	B	SE	β	B	SE	β
Model without covariates												
Stressful life events	.20***	.02	.40***	-.02*	.01	-.31*	.24***	.03	.44***	-.02*	.01	-.16*
Model with covariates												
Stressful life events	.13***	.03	.24***	-.02	.01	-.22	.16***	.02	.32***	-.02*	.01	-.19*
Neuroticism	.61***	.06	.48***	-.06*	.02	-.28*	.26***	.04	.42***	.00	.02	.02
Extraversion	.02	.08	.01	-.05	.03	-.16	-.18*	.09	-.10*	-.07	.04	-.16
Age	.02*	.01	.10*	-.00	.00	-.07	.00	.01	.01	-.00	.00	-.03
Education	-.05	.10	-.03	-.05	.04	-.17	-.07	.06	-.05	.04	.03	.14
Ethnicity	-.12	.13	-.05	-.05	.05	-.15	-.21*	.11	-.08*	.04	.05	.07
Other small children	-.01	.08	-.01	-.00	.03	.00	.04	.06	.03	.01	.03	.04
Previous depressive episodes	.40***	.12	.15***	-.03	.05	-.08	.26*	.12	.08*	.06	.06	.09

Note. Information on mothers' and fathers' intercepts and slopes of depressive symptoms in the main text.

* p < .05.
*** p < .001.

Table 4
Regression estimates for moderation by neuroticism and extraversion.

	Mothers						Fathers					
	Intercept			Slope			Intercept			Slope		
	B	SE	β	B	SE	β	B	SE	β	B	SE	β
Stressful life events (SLE)	.14***	.03	.26***	-.02	.01	-.22	.15***	.03	.29***	-.02	.01	-.20***
Neuroticism (Neuro)	.66***	.07	.51***	-.06*	.03	-.30*	.30***	.05	.49***	.01	.03	.04
Extraversion (Extra)	.02	.10	.01	-.04	.04	-.13	-.22	.11	-.11	-.08	.06	-.19
SLE × Neuro	-.02	.03	-.04	.00	.01	.02	-.02	.02	-.09	-.00	.01	-.06
SLE × Extra	.00	.04	.00	-.01	.02	-.04	.02	.04	.02	.01	.02	.06

Note: Model includes all covariates but estimates for covariates are not shown.

**p < .01.
* p < .05.
*** p < .001.

However, the number of stressful life events predicted the level and course of depressive symptoms similarly for mothers and fathers, and personality traits did not change the association between stressful life events and depressive symptoms in either mothers or fathers.

Why was there no evidence for gender differences in the effect of life events on parental depressive symptoms? Previous research has suggested that the generally higher levels of depressive symptoms in women compared to men are due either to differences in the number

[and/or type (Kendler et al., 2014)] of stressful life events or to differential sensitivity to their pathogenic effect. In our study of couples responding to a long and comprehensive list of, mostly shared, life events (e.g., serious illness in the family, marital problems, financial difficulties) there was no difference in the number of events reported. Our study did not show a female dispositional sensitivity to the effects of events, either.

Thus, our findings seem to contradict much previous evidence for

women's greater sensitivity to the pathogenic effect of stressful life events. Some research, however, suggests that this female sensitivity is not global but, in line with a gender role hypothesis, rather restricted to certain events and experiences, such as problems in social relationships or with children, housing and fertility or reproduction. We were not able to test this hypothesis in this study as we could not find statistical evidence for any categorisation of events by type in our sample. It is therefore possible that we did not find parent gender differences in sensitivity to stressors because we did not consider specific events and experiences. However, there may be another reason for this null finding. An early British study, also on couples' responses to shared life events, showed a greater female sensitivity to events only among couples espousing traditional gender role attitudes (Nazroo et al., 1997). Gender role differences may, on the one hand, enable men to distance themselves from such events and, on the other, result in women being more likely to hold themselves responsible for them. We would expect that in our study of couples in one of the most gender-equal countries in the world, support for traditional gender roles would be low, which would, in turn, explain the null gender effect found. We call for future studies to test our expectation that gender differences in the effect of shared life events on partners' depressive symptoms would be found only in couples supporting traditional gender roles. As far as this study is concerned, however, its findings clearly suggest that, if the association between stressful life events and depressive symptoms is causal, stressful life events put both mothers and fathers of young children at risk of depression. This, in turn, suggests that programmes to prevent and treat depression should target both mothers and fathers in families with high levels of upheaval shortly after a child's birth.

5. Limitations

The study is correlational and therefore causality cannot be inferred. Another, related, limitation is that life events were measured only at one point in time, thus not allowing us to test for reverse causality or reciprocal influences. Finally, the use of more and more varied life events could have enabled us to explore if mothers and fathers of young children are sensitive to specific types of events.

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