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## The etiology of functional somatic symptoms in adolescents

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# Chapter 4

**Parental overprotection predicts the development of functional somatic symptoms in young adolescents**

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### ABSTRACT

**Objective:** *To examine whether parental overprotection contributes to the development of functional somatic symptoms (FSS) in young adolescents. In addition, we aimed to study whether this potential effect of parental overprotection is mediated by parenting distress and/or moderated by the adolescent's sex.*

**Study design:** *FSS were measured in 2230 adolescents (ages 10 to 12 years from the Tracking Adolescents' Individual Lives Survey) by the Somatic Complaints subscale of the Youth Self-Report at baseline and at follow-up 2½ years later. Parental overprotection as perceived by the child was assessed by means of the EMBU-C (Swedish acronym for my memories of upbringing–child version). Parents completed the Parenting Stress Index. Linear regression analyses were performed adjusted for FSS at baseline and sex.*

**Results:** *Parental overprotection was a predictor of the development of FSS in young adolescents ( $\beta = 0.055$ ,  $p < .01$ ). Stratified analyses revealed that maternal overprotection was a predictor of the development of FSS in girls ( $\beta = 0.085$ ,  $p < .02$ ), whereas paternal overprotection was a predictor of the development of FSS in boys ( $\beta = 0.072$ ,  $p < .01$ ). A small (5.7%) but significant mediating effect of maternal parenting stress in the relationship between parental overprotection and FSS was found.*

**Conclusions:** *Parental overprotection may play a role in the development of FSS in young adolescents.*

### INTRODUCTION

Functional somatic symptoms (FSS) are commonly experienced by children and adolescents. The most prevalent FSS in children and adolescents are pain, fatigue and gastrointestinal problems (Perquin et al., 2000; Saps et al., 2009; ter Wolbeek et al., 2006). It is clear that FSS are the outcome of a multifactorial process: cognitive, social, and biological factors have been found to play a role. Among the social factors that have been suggested to contribute to the development of FSS in children and adolescents are parental behaviors. Several studies suggested that protecting children too much may ultimately result in worse health outcomes. Parental overprotection has found to be significantly associated with FSS in children and adolescents in cross-sectional studies (Mullick, 2002; Wynick et al., 1997). Retrospective studies in adults also suggested a role of maternal overprotection during childhood in developing FSS in adult life (Fisher and Chalder, 2003; Pogromov et al., 2005).

Most likely, the association between parental overprotection and FSS is not similar to all adolescents but influenced by a wide range of factors, among which parents' own FSS and the duration and nature of the symptoms (Whitehead et al., 1994). In this study, we focused on two potential modifiers: the sex of the child and the parent. Girls often have closer relationships with their parents than boys, especially with their mothers (Shearer et al., 2005). Furthermore, girls have been found to report more parental sympathy and encouragement of their illness behavior than boys and to be allowed more relief from responsibility during illness than boys (Walker and Zeman, 1992). Not only may the sex of the child, but also that of the parent influence associations between parental overprotection and FSS. However, a retrospective study in adults found that both maternal and paternal overprotection during childhood were equally associated with psychological disorders (Overbeek et al., 2007).

The above studies suggest parental overprotection to be associated with FSS (Fisher and Chalder, 2003; Mullick, 2002; Pogromov et al., 2005; Wynick et al., 1997). It is not known whether parental overprotection truly contributes to the development of FSS, nor via which mechanism overprotection may lead to the development of FSS in children. A possible pathway is that parents who have the inclination to overprotect their children experience more stress during parenting. Overprotective parents feel the need to have control over child-rearing situations and may become distressed when they are not able to succeed. Among other things, parental distress may be expressed as depression, anxiety, or parenting stress all of which have been shown to be associated with FSS in children (Domenech-Llaberia et al., 2004; Eccleston et al., 2004; Ramchandani et al., 2006). This study focused on parenting stress in particular, because parenting stress is just as parental overprotection related to the child-rearing situation.

We studied the contribution of parental overprotection to the development of FSS in a Dutch general population sample of young adolescents (1132 girls and 1098 boys, ages 10 to 12 years at baseline). We hypothesized that (a) parental overprotection is a predictor of the development of FSS; (b) both maternal overprotection and paternal overprotection are predictors of the development of FSS in young adolescents; (c) girls are more susceptible to develop FSS when perceiving overprotection than boys; and (d) the relation between overprotecting and developing FSS is mediated by parenting stress.

### METHODS

#### Sample and procedure

This study is part of the TRacking Adolescents' Individual Lives Survey (TRAILS), a prospective cohort study of Dutch adolescents. The study was approved by the Dutch Central Committee on Research Involving Human Subjects. The study reported here involves data from the first and second assessment wave of TRAILS, which ran from March 2001 to July 2002 and September 2003 to December 2004, respectively. At both assessment waves, parents' written informed consent was obtained after the procedures had been fully explained. In addition, children gave written informed consent at the second wave.

TRAILS participants were selected from five municipalities in the north of The Netherlands, including both urban and rural areas. Children born between October 1, 1989, and September 30, 1990 (first two municipalities), or October 1, 1990, and September 30, 1991 (last three municipalities), were eligible for inclusion, providing that their schools were willing to cooperate and that they were able to participate in the study. Of all eligible 2935 children, 76.0% ( $N = 2230$ , mean age = 11.09,  $SD = .56$ , 50.8% girls) were enrolled in the study.

Of the 2230 baseline participants, 96.4% ( $N = 2149$ , 51.0% girls) participated in the follow-up, which was held 2 to 3 years after baseline assessment (mean number of months, 30;  $SD = 5$ , range: 17 to 48). Mean age at follow-up was 13.56 ( $SD = 0.53$ ). Of these, 2015 adolescents completed all questions referring to FSS at follow-up. There were no differences in psychopathology scores (including baseline FSS) assessed by teacher reports, sex, or age between responders and nonresponders at follow-up. Detailed information about sample selection and analysis of nonresponse bias has been reported elsewhere (Huisman et al., 2008).

#### Measures

##### *Functional somatic symptoms*

FSS at baseline and follow-up were measured by the Somatic Complaints subscale of the Youth Self-Report (YSR) (Achenbach et al., 2003). The YSR is known to have a good cross-cultural validity (de Groot et al., 1996). The Somatic Complaints subscale contains nine items, which refer to somatic complaints without a known medical cause (aches/pains, headache, nausea, eye problems, skin problems, stomach pain, and vomiting) or without obvious reason (overtiredness and dizziness). The adolescents could indicate whether they

experienced these complaints on a 3-point-scale, with 0 = never, 1 = sometimes or a little bit, and 2 = often or a lot. We performed a factor analysis to examine whether these symptoms could be analyzed as a single trait. The factor analysis indicated that two items (eye problems and skin problems) had low factor loadings at both assessment waves in both girls and boys, suggesting that these items did not represent the underlying construct well in our sample and may better be excluded. The remaining seven items showed good internal consistency (Cronbach's  $\alpha$  at baseline: 0.76, at follow-up: 0.77), and were therefore combined into a scale. The scale score represents the mean item score.

#### *Anxiety and depression*

Symptoms of anxiety or depression at baseline were measured by the Anxiousness and Depressed Subscale of the YSR. This scale contained 13 items referring to symptoms of anxiety and depression, which showed good internal consistency (Cronbach's  $\alpha$ : 0.78). The scale score represents the mean item score.

#### *Overprotection*

Parental overprotection at baseline was measured by use of the overprotection subscale of the EMBU-C (Swedish acronym for My memories of upbringing), a questionnaire developed to assess children's perceptions of parental rearing practices, which has been shown to be valid for the Dutch population (Markus et al., 2003). Young adolescents filled out this questionnaire for both their mother and their father, resulting in data of both maternal and paternal overprotection. We were interested in overall parental overprotection in the family as well because paternal and maternal overprotection may partly compensate for each other. We calculated total parental overprotection scores in the family by taking the mean of the maternal and paternal overprotection scores. The EMBU-C contains 12 items referring to children's perception of parental overprotection, which can be rated on a 5-point scale ranging from 0 = never to 4 = always (Cronbach's  $\alpha$  parental overprotection = 0.84; paternal overprotection = 0.70; maternal overprotection = 0.71). Examples of overprotection items are: "Are your parents very concerned about your physical health?"; "Do your parents forbid you to do things that your classmates are allowed to do, because they are afraid of something happening to you?" and "Do you think your parents have high expectations as far as your school results, sports achievements and so on are concerned?" The scale score represents the mean item score.

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### *Parenting stress*

The amount of parenting distress parents experienced at baseline was measured by a Dutch short version of the Parenting Stress Index (PSI) the NOSIK (Nijmegen parental stress index short version) (De Brock et al., 1992). Of all parents 2048 filled out the PSI of which 1951 were mothers (>95%). This version of the PSI contains 25 items that could be rated on a 6-point scale ranging from 1 = disagree very much to 6 = agree very much. It consists of 2 subscales, with 11 items referring to child characteristics and 14 items referring to parent characteristics within the caregiving context. One item (item 24: "I feel confident about the future upbringing of my child") was excluded because of a low factor loading in this sample. A total scale score was computed by taking the mean item score, which showed good internal consistency (Cronbach  $\alpha = 0.94$ ).

### **Data analysis**

To test differences between FSS at baseline and follow-up and between maternal and paternal overprotection scores, we used paired-sample *t*-tests. To test differences between boys' and girls' FSS and overprotection scores, we used independent-samples *t*-tests. To examine the cross-sectional association between parental overprotection and FSS at baseline, we performed regression analyses adjusted for sex. Next, we tested whether parental overprotection was a predictor of the development of FSS at follow-up, using a linear regression model in which the effect of overprotection was adjusted for FSS at baseline and sex. We repeated these analyses while adjusting for baseline anxiety and depression, because anxiety and depression could possibly confound the relationship between FSS and parental overprotection. To test whether parental overprotection only starts to play a role after the emergence of FSS or also contributes to the development of FSS in initially symptom-free adolescents, FSS at follow-up was regressed on parental overprotection in the subgroup without FSS at baseline ( $N = 343$ ; mean age, 11.11;  $SD: 0.55$ ; 43.4% girls). In contrast with FSS in the total sample, FSS at follow-up in this group were not distributed normally, we recoded them into 0 = no complaints and 1 = 1 or more complaints at follow-up, and performed logistic regression analyses, again adjusting for sex. Sex differences were explored by performing the prospective linear regression analyses, adjusted for baseline FSS, for boys and girls separately. To test whether maternal parenting stress at baseline mediated the relationship between maternal overprotection at baseline and the development of FSS at follow-up, we used a bootstrapping procedure developed by Preacher and Hayes (Preacher and Hayes, 2008). The latter analysis was confined to the young adolescents of whom the mother completed the parenting stress questionnaire ( $N = 1951$ ; adolescents in this group

did not differ in age or sex from the total sample). We tested this mediation for maternal overprotection and not for paternal overprotection, because of the lack of availability of paternal parenting stress scores. All analyses were done with SPSS 15.0 for Windows (SPSS Inc, Chicago, Illinois).  $P$ -values  $< .05$  were considered statistically significant.

## RESULTS

### Functional Somatic Symptoms

Girls reported significantly ( $t = 3.87, p < .01$ ) more FSS (mean = 3.45,  $SD = 2.48$ ) at baseline than boys (mean = 3.04,  $SD = 2.40$ ). At follow-up, girls reported again significantly ( $t = 9.84, p < .001$ ) more FSS (mean = 3.23,  $SD = 2.56$ ) than boys (mean = 2.17,  $SD = 2.26$ ). Table 1 shows the percentages of girls and boys who experienced FSS at baseline and follow-up. The prevalence of most complaints declined at wave 2 as compared with wave 1. Exceptions were overtiredness, which increased in boys and girls, and dizziness, which increased in girls at assessment wave 2 (Table 1).

### Parental overprotection: longitudinal associations

Linear regression analyses, adjusted for baseline FSS and sex, revealed that total parental overprotection scores at baseline significantly predicted FSS at follow-up ( $\beta = 0.055, t = 2.65, p < .01$ ). More specifically, both maternal overprotection at baseline ( $\beta = 0.056, t = 2.68, p < .01$ ) and paternal overprotection at baseline ( $\beta = 0.048, t = 2.32, p = .02$ ) were predictors of FSS at follow-up. When adjusted for adolescents' anxiety/depression at baseline, parental overprotection was still a significant predictor of the development of FSS in young adolescents ( $\beta = 0.057, t = 2.70, p < .01$ ).

To examine whether parental overprotection predicted the development of new-onset FSS as well, we examined all initially symptom-free adolescents. Logistic regression analyses revealed total overprotection to show a trend towards being a significant predictor of the development of new onset FSS (OR: 1.77; 95%-CI: 0.92-3.40). Paternal overprotection was a significant predictor of the development of new onset FSS (OR: 1.98; 95%-CI: 1.03-3.82), maternal overprotection outcomes pointed in the same direction, although results did not reach significance (OR: 1.55; 95%-CI: 0.86-2.81) in this group of initially symptom-free adolescents.

**Table 1. Percentages of adolescents endorsing different levels of functional somatic symptoms at baseline and follow-up**

	Girls T1	Boys T1	Girls T2	Boys T2
<b>Headache</b>				
Sometimes, a bit	56.1	52.8	51.7	40.3
Often, a lot	10.9	8.0	9.7	5.6
<b>Stomach pain</b>				
Sometimes, a bit	58.1	51.3	48.9	34.6
Often, a lot	6.4	3.9	6.4	1.1
<b>Nausea</b>				
Sometimes, a bit	46.5	42.1	40.1	27.6
Often, a lot	3.9	3.1	4.2	1.3
<b>Aches, pains</b>				
Sometimes, a bit	40.5	36.3	26.2	21.7
Often, a lot	2.6	2.8	2.3	1.4
<b>Dizziness</b>				
Sometimes, a bit	28.9	24.7	34.4	22.9
Often, a lot	5.3	3.0	6.4	1.7
<b>Vomiting</b>				
Sometimes, a bit	31.2	30.3	13.9	14.7
Often, a lot	1.5	1.6	0.5	0.5
<b>Overtiredness</b>				
Sometimes, a bit	18.3	21.1	35.0	23.9
Often, a lot	1.7	1.4	8.1	5.1

*T1 = baseline; T2 = follow-up*

### **Parental overprotection: cross-sectional associations**

The mean total overprotection scores was 1.86 ( $SD = 0.39$ ). Maternal overprotection scores (mean = 1.93,  $SD = 0.41$ ) were significantly higher ( $t = -26.2$ ,  $p < .001$ ) than paternal overprotection scores (mean = 1.79,  $SD = 0.39$ ). Linear regression analysis revealed that total parental overprotection was significantly associated with FSS at baseline ( $\beta = 0.22$ ,  $t = 10.18$ ,  $p < 0.001$ ). More specifically, both maternal overprotection ( $\beta = 0.21$ ,  $t = 9.98$ ,  $p < .001$ ) and paternal overprotection ( $\beta = 0.21$ ,  $t = 9.98$ ,  $p < .001$ ) were associated with FSS at baseline.

**Adolescents' sex differences**

Overprotection scores reported by boys (mean = 1.88,  $SD = 0.39$ ) were significantly higher ( $t = -2.81$ ,  $p < 0.01$ ) than those reported by girls (mean = 1.84,  $SD = 0.37$ ), although this is only a small difference. We performed analyses adjusted for baseline FSS for boys and girls separately to explore whether the predictive effects of parental overprotection on the development of FSS showed sex differences. The association between total overprotection scores at baseline and FSS at follow-up was significant for boys ( $\beta = 0.066$ ,  $t = 2.15$ ,  $p = 0.032$ ). For girls the association between total overprotection at baseline and FSS at follow-up approached significance ( $\beta = 0.049$ ,  $t = 1.65$ ,  $p = 0.098$ ). Exploration of mother-daughter, mother-son, father-daughter and father-son dyads revealed a significant relationship between maternal overprotection at baseline and FSS at follow-up in girls (Table 2) and paternal overprotection at baseline and FSS at follow-up in boys (Table 3). No significant relationships were found between paternal overprotection at baseline and FSS at follow-up in girls (Table 2) and between maternal overprotection at baseline and FSS at follow-up in boys (Table 3). These tables also show that baseline FSS is a strong predictor of FSS at follow-up.

**Table 2. Linear regression analyses predicting functional somatic symptoms at follow-up in girls**

Predictor	B	T	R <sup>2</sup>
Maternal overprotection	0.072*	2.44	0.18
FSS T1	0.41**	13.8	
Paternal overprotection	0.021	0.71	0.18
FSS T1	0.42**	14.2	

*FSS T1 = functional somatic symptoms at baseline. \* $p < .05$ , \*\* $p < .001$*

**Table 3. Linear regression analyses predicting functional somatic symptoms at follow-up in boys**

Predictor	B	T	R <sup>2</sup>
Maternal overprotection	0.042	1.35	0.16
FSS T1	0.39**	12.5	
Paternal overprotection	0.085*	2.72	0.16
FSS T1	0.37**	11.9	

*FSS T1 = functional somatic symptoms at baseline. \* $p < .05$ , \*\* $p < .001$*

### **Mediation by maternal parenting stress**

To test the mediation effect of maternal parenting stress in the relation between maternal overprotection and the development of FSS, we checked the two assumptions of mediation. First, the mediator (maternal parenting stress) has to affect the dependent variable (FSS at follow-up). Maternal parenting stress, adjusted for FSS at baseline and sex, significantly predicted FSS at follow-up ( $\beta = 0.086$ ,  $t = 4.02$ ,  $p < .001$ ). Second, the independent variable (maternal overprotection) has to be associated with the mediator (maternal parenting stress). Maternal overprotection was associated with maternal parenting stress ( $\beta = 0.11$ ,  $t = 4.76$ ,  $p < .001$ ). These analyses revealed that the two assumptions to test mediation were met. When maternal parenting stress was included in the regression model of maternal overprotection predicting FSS at follow-up,  $\beta$  fell from 0.071 ( $t = 3.21$ ,  $p < .01$ ) to 0.067 ( $t = 3.02$ ,  $p < .01$ ). This reduction in regression coefficient was modest, 5.7%. Nevertheless, bootstrapping revealed that the indirect effect was statistically significant, with the 95% confidence interval ranging from 0.017 to 0.091.

### **DISCUSSION**

Our study confirms findings from previous cross-sectional and retrospective studies suggesting a relationship between parental overprotection and FSS (Fisher and Chalder, 2003; Mullick, 2002; Pogromov et al., 2005; Wynick et al., 1997). This enlarges the cross-cultural validity of this finding. Moreover, unlike previous studies we were able to ensure that parental overprotection was a contributive factor to the development of FSS. Analyses in a group of initially symptom-free adolescents resulted in essentially the same findings. Another strength of our study is that we measured parental overprotection as perceived by the child instead of parent reports, because the child's perception of his/her parents' rearing behaviors is likely to be more relevant for the development of FSS than parent reports. Our large general population cohort enhances the probability that the findings are generalizable. Another reason to have confidence in the generalizability of our findings is that the prevalence of FSS found in this study was largely comparable with previous population-based studies (1-3). Our study confirms that FSS are common in adolescents. Furthermore, the general notion that girls report more symptoms than boys and that this sex difference increases during adolescence (Eminson et al., 1996; Hetland et al., 2002; Rauste-Von Wright and Von Wright, 1981) is supported by our study. Unlike some recent studies on the development of FSS (Hetland et al., 2002; Steinhausen and Metzke, 2007), we found a decrease of most FSS during adolescence. This may be due to the short

follow-up period and the small age range of children studied: other studies who followed up children about the same age closely in time, also indicated a decrease of symptoms after early puberty (Oster, 1972; Rauste-Von Wright and Von Wright, 1981). The exact developmental pattern of FSS during puberty needs further investigation.

It has never been studied whether both maternal and paternal overprotection contribute to the development of FSS in children and adolescents. We do know that both paternal and maternal overprotection predict other mental health outcomes (Overbeek et al., 2007). Consistent with that, we found maternal as well as paternal overprotection to be significant predictors of the development of FSS. This is an important finding with regard to the design of future studies, because mostly only maternal rearing behavior is examined and hence important information is lacking.

We were the first who studied the role of sex differences in the relationship between parental overprotection and FSS. Our exploratory analyses suggest that girls are more susceptible to maternal overprotection and boys to paternal overprotection. Our findings are in line with a previous study on child characteristics and parental overprotection, which found that maternal overprotection influences harm avoidance and self-directedness in girls, whereas paternal overprotection influences harm avoidance and self-directedness in boys (Oshino et al., 2007). The putative stronger relationship between overprotection and FSS in same-sex dyads may be caused by the parents, the children or both. Overprotective parents may pay more attention to the same-sex complaining children because they find it easier to identify with them. For the child the same sex-parent may function as a role model when it comes to dealing with FSS. Indeed, children have been found to mirror the FSS of their parents (van de Putte et al., 2006). Further research should be done to clarify whether overprotective parents are experiencing more FSS themselves.

An additional aim of our study was to examine how parental overprotection contributes to the development of FSS. We found maternal parenting stress to only partly mediate the relationship between maternal overprotection and FSS. We want to address that, although we studied the effect of parental overprotection on parenting stress, this relationship is probably more complex as parenting stress may lead to parental overprotection as well. That parenting stress only partly mediated the relationship between parental overprotection and FSS hints at additional mediators, which explain other parts of the relationship between

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parental overprotection and FSS. Possibly, for instance, overprotective parents pay more attention to their children's complaints. An experimental study showed that children reported significantly more complaints when their parents gave attention to their complaints, than when parents distracted them from their complaints (Walker et al., 2006). Furthermore, parental overprotection may prevent adolescents from developing active coping strategies for their FSS. Active coping strategies have been found to be important for dealing with FSS (Sanders et al., 1994).

We acknowledge two limitations to our study. We cannot know for sure that the FSS reported are FSS in the sense that there is truly no conventional disease accounting for the complaints reported. However, we consider it quite likely that we actually measured FSS. First, the Somatic Subscale of the YSR states that they have to occur without obvious reason or without a known medical cause. Second, all complaints included loaded on the same factor at both assessment waves in both girls and boys, which strongly suggests that they reflect an underlying general trait. Finally, symptoms which are the result of a known medical condition do also have a subjective component, which can be influenced by parental behavior, although probably to a lesser degree (Gidron et al., 1995). Therefore, if we were (accidentally) partly measuring medically explained symptoms, the current findings are probably underestimations of the actual effect of parental overprotection on functional symptoms. Another limitation is that although we found that parental overprotection was a predictor of FSS, we do not know whether this is a causal association. It requires an intervention study to examine whether a reduction in parental overprotection truly leads to a reduction in adolescents' FSS. A family intervention study found that children who received cognitive behavioral family therapy had a higher rate of elimination of pain, lower levels of relapse at follow-up, and lower levels of interference with their activities than children receiving standard pediatric care (Sanders et al., 1994). However, this family therapy was not restricted to parental overprotection.