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The etiology of functional somatic symptoms in adolescents

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Thesis summary

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The aim of this thesis was to examine the etiology of functional somatic symptoms (FSS) in adolescents. FSS are symptoms that cannot be completely explained by underlying pathology. They are common during adolescence, but their etiology is still unknown. FSS are generally assumed to be the result of a complex interplay between biological, psychological and environmental factors. Some researchers take the approach that all different FSS share a common etiology and should be studied together, called the lumping approach. Other researchers take the approach that particular FSS have their own specific background and should be studied separately, known as the splitting approach. We assumed that FSS have both a shared etiology, mainly determined by psychological and environmental factors and a specific etiology, mainly determined by biological factors. Therefore, the etiology of FSS was studied by taking the lumping approach when examining psychological and environmental factors and by following the splitting approach when investigating biological factors.

The first psychological risk factors that were examined were anxiety and depression (Chapter 1). Trait and state models were used to examine whether anxiety and depression were risk factors rather than consequences of FSS. The state component of anxiety and depression had a strong and direct effect on FSS. The state component of anxiety and depression had in turn a weaker and delayed effect on FSS. The trait components of anxiety, depression and FSS were related as well. This suggests that FSS and anxiety and depression do not only have a mutual influence on each other, but also share a common background. In Chapter 3, the role of intelligence was examined. FSS were again divided into a state and trait component. The state component of FSS was associated with intelligence, but only in adolescents of whom parents had high expectation of adolescents' school performance. This suggests that high parental expectations and not low intelligence scores make adolescents with low intelligence scores at risk for FSS. The role of the parents in the etiology of FSS, became also apparent in Chapter 4. Parental overprotection predicted the course of FSS in young adolescents. This effect was partially mediated by parenting stress. Suggesting that overprotective parents experienced more parenting stress and thereby made adolescents at risk for FSS. Not only parental overprotection, but also school absenteeism was found to be a perpetuating factor of FSS (Chapter 5). This perpetuating effect was not found in adolescents who were bullied. For bullied adolescents school absenteeism probably had not only a disadvantageous effect by making

adolescents more focused on their symptoms, but also a beneficial effect by releasing these adolescents from being bullied, when staying home.

In part II, biological risk factors for FSS were studied, when taking a splitting approach. The first biological factor that was examined was pubertal development (Chapter 6). Pubertal development was not only studied in the TRAILS-cohort, but also in an American cohort of adolescents. In both cohorts, pubertal development predicted back pain, overtiredness and dizziness at follow-up, but not headache and stomach pain. This study, thus, suggested that headache and stomach pain result from another biological background than overtiredness, dizziness and musculoskeletal pain. In chapter 7, we examined by performing both a confirmatory and an exploratory factor analysis whether two factors were indeed underlying the investigated FSS. This factor analysis confirmed that FSS could be divided into two symptom clusters. One symptom cluster consisted of overtiredness, dizziness and musculoskeletal pain and the other cluster consisted of headache and gastrointestinal symptoms. These symptom clusters were examined in relation to the stress hormone cortisol (Chapter 7). The cluster of overtiredness, dizziness and musculoskeletal pain was related to low cortisol levels after awakening. The cluster of headache and gastrointestinal pain was related to low cortisol levels during social stress. In the last empirical study, we examined whether autonomic nervous system functioning and psychological arousal were differentially related to these symptom clusters (Chapter 8). The cluster of overtiredness, dizziness and musculoskeletal pain was associated with high heart rate variability prior to a stressful situation, and to long lasting psychological arousal. The cluster of headache and gastrointestinal symptoms was related to high heart rate prior to a stressful situation, and to transient psychological arousal.

In Chapter 9, all findings were discussed in light of our new perspective on lumping and splitting. We found that apart from biological factors, psychological factors could best be studied while taking a splitting approach. Anxiety and depression were two-and-a-half time stronger related to the cluster of overtiredness, dizziness and musculoskeletal pain than to the cluster of headache and gastrointestinal symptoms. So our studies suggest that when studying psychological or biological factors, FSS could be divided into two symptom clusters. When studying environmental factors FSS could be lumped and studied together.

