A Developmental Perspective on Functional Somatic Symptoms

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Objective To provide a new approach for conceptualizing and studying functional somatic symptoms (FSS) in children and adolescence. **Methods** A developmental model is proposed based on the synthesis of the extant literature and previous theoretical perspectives of FSS in children and adolescents. **Results** Multiple risk and protective factors from child, familial, social, and environmental domains, the interactions across risk domains, and potential developmental pathways of FSS are identified. **Conclusions** This article underscores the necessity of taking a broader, developmental view of FSS. The tenets of developmental psychopathology emphasize the utility of viewing FSS on a continuum of severity rather than as a discrete entity or diagnosis. This article concludes with directions for future research and treatment implications.

Key words adolescents; children; developmental psychopathology; functional-somatic-symptoms; risk-factors; somatic-complaints, pain.

Functional somatic symptoms (FSS), defined as physical symptoms of unknown pathology (Dhossche, Ferdinand, van der Ende, & Verhulst, 2001; Steinhausen, 2006), affect 10–30% of children and adolescents in the United States (Campo & Fritsch, 1994) and may include headaches, abdominal pain, musculoskeletal pain, vomiting, chest pain, fatigue, and dizziness. FSS have been studied as individual symptoms (e.g., recurrent abdominal pain; Walker et al., 2006b) and as symptom constellations (e.g., the Somatic Complaints subscale on the *Child Behavior Checklist*; Achenbach, Conners, Quay, Verhulst, & Howell, 1989). In addition to their high prevalence, FSS are associated with high levels of functional impairment and comorbid psychiatric symptoms, particularly anxiety and depression (Garber, Zeman, & Walker, 1990; Walker & Greene, 1989). Children with FSS are frequently seen in pediatric settings. Indeed, 2–4% of all pediatric visits are reportedly due to FSS (Campo & Reich, 1999). This high level of medical attention is not only costly to families and society, but may lead to unnecessary and dangerous medical procedures (Campo & Fritsch, 1994; Campo & Reich, 1999).

The traditional Western medical approach to illness is to find a pathological origin for symptoms. However, this approach has limitations when applied to children with FSS. First, this approach emphasizes the presence or absence of a diagnosis. No empirical evidence yet has been found to support FSS as a discrete diagnostic entity. Rather, empirical research shows considerable heterogeneity in the clinical presentation, course, and outcome of FSS in children and adolescents, which leads to the second limitation. The traditional medical approach does not recognize differences in trajectories and outcomes.

Developmental psychopathology (Cicchetti & Sroufe, 2000; Cummings, Davies, & Campbell, 2000) offers an alternative perspective for studying FSS in childhood and adolescence. First, through the developmental lens, childhood behaviors are viewed on a continuum from normal to abnormal (or disordered). Second, developmental psychopathology seeks to identify risk factors that influence the severity, trajectories, and outcomes of maladaptive child behavior. The extant literature does not provide extensive evidence for the contribution of one particular factor to FSS, rather the studies to date offer clues to the multiple factors that may underlie the development and progression of FSS. By applying a developmental psychopathology perspective, issues concerning the antecedents, course, and outcome of FSS can be addressed, drawing upon the extant literature across multiple disciplines (e.g., psychology, psychiatry, nursing, and pediatrics). The purpose of this article is to review

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Journal of Pediatric Psychology 33(5) pp. 547–562, 2008
doi:10.1093/jpepsy/jsm113
Advance Access publication December 3, 2007

the literature regarding individual differences in clinical presentation and pathways of FSS in children and adolescents. Second, this review aims to identify clues in the literature regarding the risk factors from child, social, and environmental domains that directly influence or moderate outcomes of FSS. Third, this article identifies theories that suggest mechanisms for the maintenance and progression of FSS. Finally, future research directions and clinical implications are discussed.

Pathways and Progression of FSS

A prototypical question posed by developmental psychopathologists is whether different risk factors and pathways can distinguish cases of disordered behavior and increase the power of predicting adult outcome. The timing of symptom manifestation and the identification of combinations of risk and protective factors have helped guide developmental psychopathologists in delineating trajectories of childhood disorders (Cicchetti & Sroufe, 2000). Subsequently, the research pertaining to the variability in clinical presentation and continuity of FSS over time is reviewed to illuminate potential pathways of FSS in childhood.

Variability in Clinical Presentation

Cooccurring Maladjustment May Occur for Some

The prognosis can be worse for child-onset problems when they are persistent and/or comorbid, as is the case for antisocial behavior with attention-deficit/hyperactivity disorder (Moffitt, 1990, 1993). Due to the nascent state of the literature in this area, there has been no evidence to demonstrate a similar pattern for FSS in childhood. However, it is documented across disciplines that children with FSS often display elevated levels of psychiatric symptoms, and the frequency of FSS tend to increase with the severity of anxiety and depression symptoms (Bernstein et al., 1997; Dhossche et al., 2001; Garber et al., 1990; Last, 1991; Livingston, Taylor, & Crawford, 1988; McCauley, Carlson, & Calderon, 1991; Walker & Greene, 1989). This is particularly salient given that symptoms such as generalized anxiety, thoughts of death, and those typical of externalizing behaviors, including hyperactivity, oppositionality, and conduct problems, have been found to cooccur with FSS in children between the ages of 3 and 6 (Aromaa, Sillanpaa, Rautava, & Helenius, 2000; Domenech-Llaberia et al., 2004; Stevenson, Simpson, & Bailey, 1988; Zuckerman, Stevenson, & Bailey, 1987). For example, a significantly higher occurrence of conduct problems has been found in boys with headaches and in children with FSS age 6 and younger compared to healthy children (Egger, Angold, & Costello, 1998; Zuckerman et al., 1987).

Significant correlations between children's FSS and self-reported anxiety and depression (with somatic symptoms removed from questionnaires) have been found to be .43 and .37, respectively (Garber, Walker, & Zeman, 1991). In a longitudinal study of adolescents, FSS were significantly correlated with self-reports of anxiety in boys ($rs = .30–.42$) and girls ($rs = .31–.36$) at ages 13, 15, and 18 (Rauste-von Wright & von Wright, 1981). Longitudinal data also demonstrate some support for high levels of FSS in children predicting a later psychiatric disorder (Egger, Costello, Erkanli, & Angold, 1999; Zwaigenbaum, Szatmari, Boyle, & Offord, 1999). For example, Zwaigenbaum and colleagues found that high FSS at baseline was associated with major depressive disorder (MDD) 4 years later, independent of gender, baseline emotional disorder, and sociodemographic factors. In addition, the authors found that having an emotional disorder at baseline moderated the relationship between FSS and later MDD. Specifically, the authors found that the adolescents at the greatest risk for MDD were those with high levels of FSS but without an emotional disorder at baseline compared to peers with low FSS and no emotional disorder. The authors interpret their findings as evidence indicating FSS as an early expression of depressive feelings. In their longitudinal study, Dhossche and colleagues (2001) did not find that youth with high FSS were at a greater risk for a psychiatric disorder at a 6- to 8-year follow-up, despite using similar measurements of FSS and psychiatric disorders to those used in the Zwaigenbaum study. However, Dhossche and colleagues did not investigate the moderating effects of emotional disorders in their study. At this point, the data are mixed concerning whether or not a chronic course of FSS is a risk factor for later psychopathology, specifically clinical depression.

Besides having early psychological problems, another potential moderator of the association between FSS and psychological problems may be gender. In Egger and colleagues' (1998, 1999) longitudinal studies, data suggest that the type of FSS may predict a particular psychiatric diagnosis (as defined by *DSM-III-R* criteria, American Psychiatric Association, 1987), depending on the child's sex. For example, musculoskeletal pains predicted depression in boys and both depression and anxiety disorders in girls. The combination of headaches and abdominal pain also predicted anxiety disorders in

girls, whereas abdominal pain predicted oppositional defiant disorder and attention-deficit/hyperactivity disorder in boys (Egger et al., 1999). Moreover, 30.6% girls with one or more psychiatric diagnosis reported concurrent chronic headaches compared to 9.3% of girls without a psychiatric diagnosis, but this difference was not found in boys.

Functional Impairment

In addition to psychological problems, children with FSS have problems in social and school realms. Research shows that children with headache and other FSS have more problems in daycare, fewer hobbies, and report a greater impact of their symptoms on daily life and leisure activities (Aromaa et al., 2000; Bandell-Hoekstra et al., 2002). FSS are often associated with frequent and prolonged daycare and school absences in pediatric, psychiatric, and community samples (Bernstein et al., 1997; Domenech-Llaberia et al., 2004; Rothner, 1993; Walker, Garber, Van Slyke, & Greene, 2001). The relationship among school attendance, FSS, and psychiatric problems (i.e., anxiety and depressive symptoms) is important to delineate because missing school may have adverse consequences for youth, including strain on or loss of peer relationships, social isolation, and academic difficulties (Bernstein et al., 1997; Vannatta, Gartstein, Short, & Noll, 1998). Moreover, children with frequent school absenteeism (i.e., school refusers) are often referred to a social worker rather than to a psychiatrist, perhaps due to perceived delinquency (Stickney & Miltenberger, 1998). Empirical work from the pediatric and school refusal literature suggests that many children with FSS are not receiving the appropriate treatment for their problems due to the complexity of their presentation (for further reading see Bernstein et al., 1997; Honjo et al., 2001; King & Bernstein, 2001; Last, 1991).

Pain Severity

Pain severity has been noted as an important factor in determining a child's quality of life and may alter the presentation and course of FSS. Research has shown that children with higher levels of pain are more depressed, have a harder time changing their moods or feelings when in pain, and experience more functional impairment, such as lower social competence and more school absences, when compared to children with lower levels of pain or no pain (Cunningham et al., 1987; Gladstein & Holden, 1996; Kashikar-Zuck, Goldschneider, Powers, Vaught, & Hershey, 2001). In a community sample of children, Egger et al. (1998) found that the significant impairment associated with headache pain was specific to depression, but not anxiety, in girls. It follows that having both a psychiatric disorder, particularly depression, and severe FSS may indicate more functional impairment and consequently a worse prognosis, at least in the short-term (Mulvaney, Lambert, Garber, & Walker, 2006). These findings have direct clinical implications. For example, Gladstein and Holden (1996) suggest using the level of impairment or disability associated with symptoms, rather than the classification and diagnosis the presenting problem, for planning treatment.

Trajectories and Outcome

Continuity

Research suggests that adults' FSS have their roots in childhood and may be continuous over time (Campo & Garber, 1998; Fritz, Fritsch, & Hagino, 1997). Walker, Garber, Van Slyke and Greene (1995) found that children with functional abdominal pain demonstrated higher levels of abdominal discomfort, other FSS, and functional disability (such as school or work absences) than healthy controls at a 5- to 6-year follow-up when they were adolescents and young adults. Similarly, in an epidemiological study of adolescents, Dhossche et al. (2001) found that adolescents with a specific FSS tended to report the same symptom along with other FSS at a 6-year follow-up. One longitudinal study of approximately 2,000 community youth found that from late adolescence to early adulthood FSS were stable at a 4-fold higher rate than expected (Steinhausen, 2006). Moreover, Steinhausen found that a high number of FSS in late adolescence predicted phobic disorders and posttraumatic stress disorders in young adult males and somatoform disorders and some anxiety disorders in young adult females. These results provide some support for separate pathways of FSS for males and females.

FSS tend to increase in community and clinical samples over time, but findings do not converge on the stability of symptoms (Aro, Paronen, & Aro, 1987; Dhossche et al., 2001; Walker, Garber, & Greene, 1994), where stability is defined as the number of symptoms and not the particular constellation of symptoms. There is modest support for the stability of the number of FSS over time, particularly in girls (Aro et al., 1987; Dhossche et al., 2001; Rauste-von Wright & von Wright, 1981). However, the continuity of FSS is not universal. For instance, in a pediatric sample of children with recurrent abdominal pain, the correlation between the number of FSS at baseline and 1-year follow-up was .29 ($p < .01$), which, to the

authors, indicated a rather unstable course of FSS (Walker et al., 1994). This lack of stability could be due to differential trajectories of FSS across childhood.

In their 5-year prospective study, Mulvaney and colleagues (2006) delineated three differential pathways of FSS in a sample of 132 pediatric patients with recurrent abdominal pain: A low-risk group (70% of the sample), a short-term risk group (16%), and a long-term risk group (14%). The first two groups showed relatively long-term improvement, whereas, the latter group (the long-term risk) showed elevated levels of FSS across time. In addition, the long-term risk group demonstrated significantly more anxiety and depression, lower perceived self-worth, and more negative life events than the other groups at baseline.

In conclusion, although many studies have investigated the cooccurrence of FSS and internalizing and/or externalizing behaviors, few explicate possible mechanisms for this association. Moreover, not all children with FSS go on to develop anxiety or depression symptoms, let alone a full-blown psychiatric disorder. Indeed, it may be a smaller subset of children with a chronic course of FSS who are at the greatest risk for an emotional disorder, as seen in Mulvaney et al. (2006). At this point, it is not possible to determine whether the comorbidity of FSS, anxiety, and depression represents a single underlying phenomenon or distinct disorders.

Risk Factors of FSS

Since finding a definite and exclusive cause of abnormal child behavior is rare, the purpose of the developmental psychopathology perspective is to define a number of risk and protective factors that contribute to a child's development. Establishing a process or condition as a risk factor is complex by nature. For instance, sometimes one condition may serve as a risk or a protective factor for different outcomes: Being male is a risk factor for conduct disorder but is a protective factor for anorexia nervosa (Cicchetti & Sroufe, 2000). Moreover, a condition normally conceived as an outcome may also serve as both a protective factor and a risk factor for other maladjusted behavior. For example, anxiety is a risk factor for depression in girls but may be a protective factor for conduct disorder in boys. Subsequently, risk from child (i.e., age, gender, puberty, stress reactivity, and coping) and environmental (i.e., family, social, and sociodemographic) domains are reviewed, followed by a review of interactions among risk factors across domains.

Child Factors

The child characteristics age, gender, puberty, stress reactivity, and coping have been shown to affect the prevalence and course of FSS in children and adolescents and are briefly discussed below.

Age and Gender

Age and gender have both proven to be factors directly affecting the type and frequency of FSS. Some developmental trends for the type and frequency of FSS have emerged from the literature. First, the pattern of symptom presentation appears to change as a function of the child's developmental status (Achenbach et al., 1989; Offord et al., 1987). For example, abdominal pain is the most common complaint around 9 years of age, and headache is the most frequent complaint around age 12. Before the age of 6, pseudoseizures are rare; however, they become most apparent during adolescence (Campo & Fritsch, 1994). Second, the incidence of FSS tends to be low in early childhood. Studies have demonstrated that in early childhood, 8–9% of preschoolers have recurrent stomachaches, and 2–3% have recurrent headache (Domenech-Llaberia et al., 2004; Zuckerman et al., 1987). However, the prevalence of FSS, especially polysymptomatic presentation, increases with age (Campo, Jansen-McWilliams, Comer, & Kelleher, 1999; Egger et al., 1998). Regarding gender, epidemiological research has shown that before puberty there is no difference in the prevalence of FSS for boys and girls (Berntsson & Kohler, 2001; Campo et al., 1999). In adolescence, though, girls tend to report more than twice as many FSS as boys (Achenbach et al., 1989; Guidetti & Galli, 2001; Offord et al, 1987; Rauste-von Wright & von-Wright, 1981).

Puberty

The physiological and neurobiological changes associated with puberty may play a role in these sex and age differences (Susman, Reiter, Ford, & Dorn, 2002). In fact, experts on adolescent development have long considered puberty as a precursor of mood and behavior changes (Susman, Dorn, & Schiefelbein, 2003). The timing of puberty is important, for early-onset puberty in girls indicates a longer lifetime of estrogen exposure, which may predispose adolescents to risk for autoimmune diseases, such as chronic fatigue syndrome (Susman et al., 2002). Studies have found that advanced pubertal status in girls is associated with the frequency of FSS (Aro & Taipale, 1987; Rhee, 2005). Other evidence suggests that cluster headaches start earlier in

females than in males (perhaps due to girls' earlier pubertal onset) and have a bimodal age at onset-distribution, with a number of women having their first attacks after menopause (Ekbom, Svensson, Traff, & Waldenlind, 2002).

Stress Reactivity: Contributions of Physiology

Children's limbic hypothalamic–pituitary–adrenocortical (L-HPA) and autonomic nervous systems (ANS) have demonstrated hyperresponsivity to physically aversive events and psychologically stressful situations (Gunnar, Bruce, & Hickman, 2001; Scharff, 1997). Heightened physiological reactivity is associated with internalizing behaviors in early and middle childhood (Bauer, Quas, & Boyce, 2002; Boyce et al., 2001). In the clinical setting, children with FSS tend to be described as conscientious or obsessive (perfectionistic), sensitive, insecure, and anxious (Garralda, 1996; Kowal & Pritchard, 1990). Children with these temperamental vulnerabilities are hypothesized to be at-risk for developing anxiety disorders and are more likely to generate distress responses to potentially threatening or uncertain stimuli (Dorn et al., 2003).

Blood pressure, heart rate, adrenaline, nonadrenaline, oxytocin, and cortisol have been measured as physiological markers of stress reactivity in children with FSS (Alfven, de la Torre, & Uvnas-Moberg, 1994; Borres, Tanaka, & Thulesius, 1998; Dorn et al., 2003; Rauste-von Wright & von Wright, 1981); however, cortisol (a marker of L-HPA) and blood pressure (a marker of ANS) are the only common measures across studies. There have been inconsistent findings for cortisol (Alfven et al., 1994; Dorn et al., 2003; Rauste-von Wright & von Wright, 1981). It should be noted, however, that the measurement of cortisol, the induction of stress, the construction of comparison groups, and sample sizes were all variable across studies. In the study of Alfven and colleagues (1994), children with recurrent abdominal pain (RAP) showed a lower cortisol concentration than controls after a blood draw and demonstrated the same pattern 3 months later. A pilot study of children with RAP found that cortisol levels increased following a combination of social and cognitive stressors, but the results did not reach significance, perhaps due to the small sample size (Dorn et al., 2003). In a community sample of adolescents, Rauste-von Wright and von-Wright (1981), however, found that FSS were negatively correlated with the increased excretion of cortisol after a real-life stressor (a compulsory exam for Finnish students seeking admission to university) in 18-year-old girls only. No conclusions can be drawn for blood pressure, since one of the studies did not find differences (Dorn et al., 2003), and the other study included many nonsomatic symptoms of anxiety and depression in their definition of ''symptoms'' relating to blood pressure, obscuring any meaningful relation to FSS (Borres et al., 1998).

Coping

FSS also may reflect an anxious child's increased focus on bodily sensations and rumination on physical symptoms, as opposed to adaptively coping with the pain or sensation. Empirical evidence suggests that children and adolescents with FSS have fewer adaptive coping strategies and, to some extent, a heightened emotional response to stress compared to children with organic illness and community samples of children (Aromaa et al., 2000; Bandell-Hoekstra et al., 2002; Rauste-von Wright & von Wright, 1981; Rocha, Prkachin, Beaumont, Hardy, & Zumbo, 2003; Ruchkin, Eisemann, & Haeggloef, 2000; Thomsen et al., 2002; Walker, Smith, Garber, & Van Slyke, 1997). In studies of coping in children and adolescents, coping questionnaires defined the stressor as the pain itself (Bandell-Hoekstra et al., 2002; Thomsen et al., 2002; Walker et al., 1997), as general difficulties or problems (Rauste-von Wright & von Wright, 1981; Ruchkin et al., 2000), or as daily hassles (Walker, Smith, Garber, & Claar, 2007). Cumulatively, these studies offer support for the idea that children and adolescents with high levels of FSS use poor coping strategies characterized by disengagement, rumination over pain, avoidance, anger, cognitive interference, or some combination of these processes.

Environmental Factors

Developmental psychopathology promotes an integrative model. It not only observes the contribution of the active individual, but also examines the dynamic processes and complex interplay between the individual and multiple contextual influences in the child's ever-changing environment (Cummings et al., 2000). Developmental psychopathologists describe this dynamic environmental exchange in terms of ''contextualism.'' Each level of the child's ecological context is considered and synthesized as a part of the child's experience. For instance, the child's most proximal context (e.g., parenting), most distal sphere of influence (e.g., cultural environment), and every context in between (e.g., school and neighborhood) are expected to influence the child, her

experiences, and her development. Subsequently, contextual factors from family, social, and other environmental domains are discussed in relation to the development and maintenance of FSS in children and adolescents.

Family Factors

Empirical work has shown that FSS are highly familial, such that children and other family members often share similar symptoms, be these general physical complaints, abdominal pain, or headaches (Aromaa, Rautava, Helenius, & Sillanpaeae, 1998; Campo & Fritsch, 1994; Locke, Zinsmeister, Talley, Fett, & Melton, 2000; Walker, Garber, & Greene, 1991; Walker & Greene, 1989). Family factors also include high rates of health problems and long-term illnesses (e.g., diabetes) and psychological distress (Campo & Fritsch, 1994; Craig, Boardman, Mills, Daly-Jones, & Drake, 1993; Garralda, 1996; Zuckerman et al., 1987). A potential mechanism explaining the familial aggregation of learned illness behaviors is exposure to family adversity during childhood. Craig, Cox, and Klien (2002) found that a mother's exposure to childhood adversity predicted FSS in her child. It is plausible that aspects of family adversity are transmitted across generations. Another study found a moderating effect on familial transmission, such that in families with high levels of negative life events, mothers with high levels of FSS had boys with higher levels of FSS at follow-up than control families (Walker et al., 1994).

Social and Environmental Factors

A number of studies have investigated the association between social and environmental factors and FSS in children and adolescents. The factors that have been studied include: negative or stressful life events (Boey & Goh, 2001; Walker, Garber, & Greene, 1993, 1994), family characteristics, such as marital discord or family cohesion (Terre & Ghiselli, 1997; Zuckerman et al., 1987), daily stressors in children's lives (Torsheim & Wold, 2001; Walker, Garber, Smith, Van Slyke, & Claar, 2001; Walker et al., 2007), social support and social rewards for FSS (Torsheim & Wold, 2001; Walker, Claar, & Garber, 2002), and proxies of social disadvantage, such as neighborhood quality and socioeconomic status (Alfven, 1993; Aromaa et al., 1998; Berntsson & Kohler, 2001; Chapman, 2005; Fearon & Hotopf, 2001; Zuckerman et al., 1987). Overall, empirical evidence supports the notion that negative, adverse events or stressors in the home or at school increase FSS in community and clinic samples of children.

Interactions Among Social, Environmental, and Child Risk Factors

Studies conducted by Walker and her colleagues (1994, 2001, 2002) provide some of the only research investigating interactive effects on FSS in children and adolescents. Walker's research group has found that the relationship of stressors and social rewards to FSS is moderated by poor social and academic skills, high negative affectivity, and low self-worth. Walker's studies describe children who are unhappy and lack competence in at least one area of functioning as being at-risk for FSS in the face of stressful situations. Walker's studies (1994, 2001, 2002) have investigated the complex associations between child and contextual factors, with results supporting a social learning hypothesis. That is, children who may fear failure in social or academic realms are less apt to cope effectively with negative life stressors and consequently experience uncomfortable somatic symptoms. Conveying these symptoms to others may benefit children by allowing them to avoid the feared situation or because they receive attention, thereby reinforcing the expression, and likely the experience, of the somatic distress (Walker et al., 2006a).

Taken together, the literature reviewed above suggests that numerous environmental stressors, ranging from the death of a parent to everyday stressors, are associated with increased FSS in children and adolescents. Longitudinal data suggest that daily stressors in school and family contexts produce greater somatic distress in children with low social competence, and that social rewards maintain FSS, especially when children have low self-esteem. Social disadvantage may compound these effects, particularly in children older than 7 years of age (Alfven, 1993; Berntsson & Kohler, 2001; Fearon & Hotopf, 2001). To clarify the complex relationship among social stressors, social rewards, environmental adversity, age, and gender, the study of additive and interactive effects of these factors must continue.

Summary

This review has illuminated risk and protective factors that contribute to the etiology and maintenance of FSS. Based on the literature reviewed earlier, child, family, social, and environmental factors, and comorbidity with internalizing disorders were associated with FSS. High self-esteem, social competence, and male gender were

identified as possible protective factors. Moreover, female gender, early-onset mood disorder, poor coping skills, and greater pain severity may increase the risk for FSS over time.

Psychological Theories and Possible Mechanisms of FSS

Child researchers from multiple disciplines have investigated many theories in their search to explain the mechanisms influencing the origin and progression of FSS in children and adolescents. As opposed to being limited to one mechanism delineated by a particular theoretical orientation, developmental psychopathology allows researchers to draw from and incorporate multiple theories. Subsequently, some of the more prevalent theories and their corresponding mechanisms are described.

The most influential and long-standing theory pertaining to FSS, somatization disorder (or hysteria), and conversion disorder is Freud's psychodynamic theory (Freud, 1962). This theory highlights the child's repressed needs and emotions as a causal function in the development of FSS. Psychodynamic thinkers view FSS as a psychological defense against repressed or unconscious emotions, thoughts, and impulses, while still allowing the individual to express distress through physical symptoms (Campo, 1995; Lask & Fosson, 1989). A second school of thought, attachment theory, frames FSS as a way for the child to maintain close proximity to the attachment figure (Bowlby, 1973). The child's expression of physical discomfort and distress acts as a care-eliciting function from the attachment figure (Campo & Fritsch, 1994). Neither psychodynamic nor attachment theory, however, has received much empirical attention with respect to FSS in children and adolescents.

Third, the family systems approach posits that children's FSS serve a communicative function for family members to maintain their daily routine functioning as well as a way to avoid conflict and has been supported by empirical studies (Aro, 1987, 1989; Terre & Ghiselli, 1997; Zuckerman et al., 1987). According to Minuchin's family systems theory, children's FSS are conceptualized as a homeostatic mechanism for avoiding conflict in enmeshed, over-protective, and rigid families (Minuchin et al., 1975). FSS also have been viewed as having a communicative function, or being a ''plea for help,'' for children (Campo & Fritsch, 1994), especially in families characterized by poor parent–child relationships (Aro, 1989; Aro et al., 1987; Raust-von Wright & von-Wright, 1981).

Fourth, social learning theorists frame FSS as a learned set of interpersonal or social behaviors that are often reinforced by family members and society (Craig et al., 2002; Walker & Greene, 1989). According to this perspective, the expression of FSS may be reinforced by special attention from parents or by being excused from disagreeable tasks, such as completing chores at home or taking a test at school (Walker et al., 2002). This perspective also asserts that children may learn the importance of health beliefs and display rules from family members. For example, research supports a high positive correlation between parents' and children's FSS in families of children with recurrent abdominal pain but not in families of children with an identified organic disease or in families with healthy children (Walker et al., 1993).

A fifth perspective, the cognitive psychobiological theory, delineates FSS as a consequence of physiological reaction to emotional arousal. Cognitive factors have been hypothesized to play a major role in the reporting of FSS. For example, children with FSS may have a heightened preoccupation with or sensitivity to physical sensations (e.g., attentional bias; Boyer et al., 2006). With distorted information processing and negative cognitions, children with FSS may frame symptoms as a reason for serious concern, and this negatively biased internal monitoring leads to ''amplification'' or misinterpretation of common body sensations and/or normative levels of somatosensory inputs associated with illness or emotional distress (Campo & Reich, 1999). The amplification and misinterpretation of physiological signals may, in turn, be one of the main processes in the development of FSS (Rief, Shaw, & Fichter, 1998).

Sixth, coping and stress response theories delineate that children's cognitive and behavioral responses to chronic pain influences their level of pain and psychological adjustment (Thomsen et al., 2002; Walker et al., 2007). Coping has been defined as a child's voluntary efforts to regulate their emotions, thoughts, behavior, physiology, and environment in response to stressful events (Compas, Conner-Smith, Saltzman, Thomsen, & Wadsworth, 2001). The manner in which children cope with such stressors has a great impact on FSS, pain intensity, and internalizing symptomatology. For example, in studies of children with recurrent abdominal pain, those children who used more accommodating coping strategies (e.g., acceptance or distraction) in response to pain demonstrated fewer FSS and symptoms of anxiety and depression. In contrast, passive coping responses,

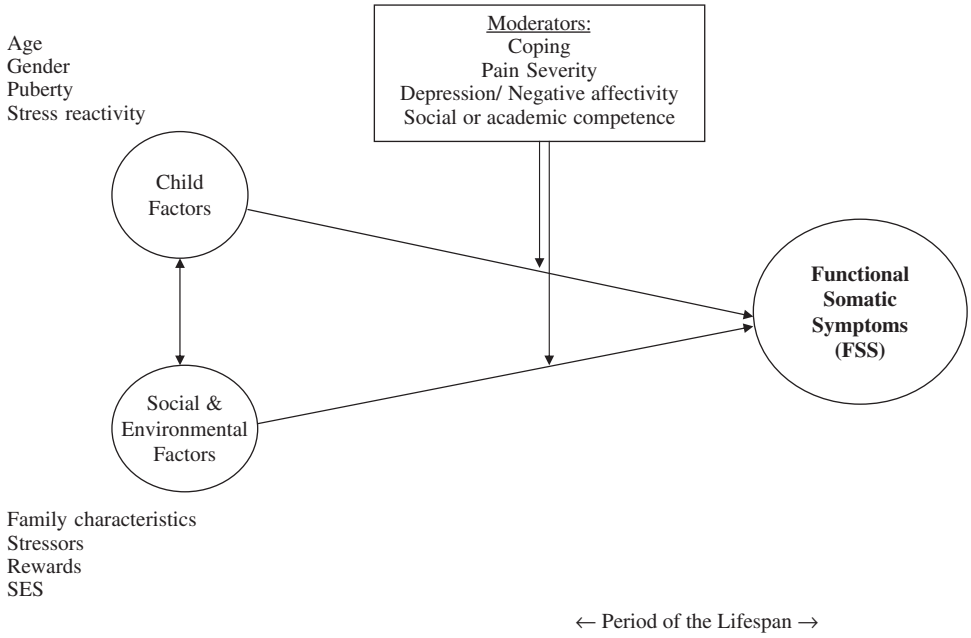

Figure 1. A working model for the study of functional somatic symptoms across childhood and adolescence.

such as disengagement, were found to be strongly associated with increased levels of pain, FSS, and depressive symptoms (Thomsen et al., 2002; Walker et al., 1997).

Lastly, due to the high comorbidity of symptoms and diagnoses, FSS have been hypothesized as a consequence of a psychiatric disorder, such as anxiety or depressive disorders (Campo & Reich, 1999). In the 1970s, FSS were viewed as ''masked depression,'' a condition where children expressed their emotions through physical symptoms (Bschor, 2002; Carlson & Cantwell, 1980). Many studies have investigated the positive relation between internalizing and/or externalizing behaviors and FSS, but few explicate possible mechanisms for this association.

A Developmental Perspective on FSS

The developmental psychopathology approach allows us to integrate the theoretical perspectives described earlier to understand the etiology, pathways, and outcome of FSS. The case below illustrates the combination of multiple theories (in parentheses) and developmental psychopathology constructs (noted in *italics*) with the extant literature reviewed in this article to lend a developmental perspective on FSS throughout childhood and adolescence (Fig. 1). That is, the following integrative summary describes how most of the psychological theories described above can inform our understanding of mechanisms that influence the course of FSS at particular stages in childhood and adolescence. Some theories may be more important in earlier childhood (e.g., attachment theory), while others may be more salient during adolescence (e.g., family systems theory).

The development of FSS begins with a sensitive, anxious, or emotionally reactive child who often perceives more threat and danger, be it real or imagined, in the environment compared to his or her same-age peers. In response to this perceived threat, the child may exhibit more signs of physiological reactivity than other children, due in part to a heightened awareness of bodily sensations (cognitive psychobiological and modeling theories). A sensitive or reactive child may be more likely to use FSS in signaling caregivers to help cope with distress (attachment theory). The quality of the parent–child relationship may shape the way a sensitive, anxious, or emotionally reactive child copes with stress, which in turn, may affect the course of FSS (*pathway/progression*). For example, having a harmonious and open parent–child relationship may minimize impairment associated with FSS, insofar as the responsive parent may be helpful in alleviating the child's fears or in providing alternative and more adaptive coping strategies (*risk/protective factor*), which could then lower the child's physiological reactivity and somatic distress. On the other hand, a sensitive and/or anxious child may feel rejected by the parent or may be more prone to get upset and emotionally overaroused in a parent–child relationship ridden by hostility and conflict. In such an acrimonious climate, or

where emotional repression as opposed to emotional expression is accepted, the child may begin to internalize feelings and express FSS more frequently, as it is the only outlet for an anxious child's feelings (psychodynamic theory). A child's expression of FSS might be exacerbated by vicarious learning through observation of parental illness behaviors (modeling theory), reinforced by special attention or other rewards (social learning theory), or maintained by family conflict (family systems theory). This effect might be stronger in socially disadvantaged homes with few financial and social resources, which could potentially increase stress and conflict in the home (*contextualism*).

A child's coping style may depend on his or her temperament, developmental level, or severity of pain, which in turn, may affect the course of FSS. For example, younger children who turn to their caregiver for help in coping with stressful situations and adolescents who use cognitive restructuring or distraction to cope with their emotional arousal may minimize impairment associated with FSS. However, a child who has learned and practiced poor coping strategies in the home is likely to carry these strategies into school and other social environments. The repeated experience of failing to relate to peers or to cope with academic stress may perpetuate an already anxious child's self-isolation and internalizing, further exacerbating FSS and the intensity of the pain.

If a child lacks social competence (*risk/protective factor*), and has a high level of anxiety (*risk factor*), the child may eventually feel helpless and hopeless, which increases the risk for a later mood disorder and continued FSS (*pathway/progression*). With the onset of puberty, girls may be at greater risk for comorbid internalizing and continued FSS than boys, perhaps due to hormones, interpersonal relationship stressors, or socialization practices. Boys with FSS may follow a much different trajectory than girls, where early concentration difficulties and behavior problems prevent boys from learning appropriate coping styles. Social and academic failure and low self-esteem may lead to anger and acting out. If taken to an extreme, these boys may evidence an externalizing disorder, and to a lesser extent, FSS in later childhood and adolescence.

Directions for Future Research and Clinical Implications

The aim of this review was to demonstrate the utility of a developmental perspective in the study of FSS. Contrary to dualism grounded in the traditional medical model, this review has not established that FSS is a psychological disorder in children and adolescents. Rather, this article has demonstrated that comorbid psychiatric outcomes such as anxiety and depression, while common, do not occur in *all* children presenting with FSS. The findings of this article underscore the need to take a broader approach to the conceptualization of FSS in children and adolescents.

This broader approach, based on the tenets of developmental psychopathology, necessitates viewing FSS as a behavior or set of behaviors occurring on a continuum, as opposed to as a discrete entity or diagnosis. In order to further our understanding of the etiology, course, and outcome of FSS in childhood and adolescence, longitudinal studies must replace cross-sectional designs that use large and varied age ranges. More epidemiological studies are needed to understand the extent to which FSS are normative at certain developmental periods, which would help identify when FSS are ''clinically significant'' and warrant treatment.

The developmental perspective on FSS presented in this article has delineated various ways FSS can emerge and develop through the interaction of child and contextual factors. Both adolescence and female gender have been shown to be risk factors for the development of FSS. In order to establish that puberty directly contributes to an increase of FSS in girls (and perhaps a decrease in boys), prospective designs following prepubescent children through the stages of puberty are needed. Including hormone sampling also would strengthen findings pertaining to the role of puberty and hormones, help clarify the developmental progression of FSS, and illuminate similarities/differences between child and adult forms of FSS. Moreover, investigations involving age, gender, and pubertal status are needed in light of the fact that puberty is a biological marker for depressive disorders. That is, there are dramatic increases in the rates of depression, bipolar disorder, and completed suicide around puberty (Ryan & Dahl, 1993), particularly in girls (Zahn-Waxler, Klimes-Dougan, & Slattery, 2000). Thus, it is important to continue researching the effects of puberty on FSS, especially in girls.

A priority of future research should be to establish baseline levels of physiological functioning of children with FSS and to compare these levels to those of healthy children and children with other disorders. By doing so, research could confirm a hyperresponsive reaction to stress in children and adolescents with FSS. Further, it is recommended that research continue to investigate

patterns of physiological reactivity of multiple concurrent systems (e.g., L-HPA and ANS) in relation to FSS, in order to identify specific measures of physiological reactivity that discriminate children with FSS, internalizing problems, organic disease, and comorbid problems (Bauer et al., 2002; Boyce et al., 2001). While this review briefly covered puberty and physiological reactivity to stressors, work investigating other biological substrates of FSS is beginning to appear in the literature. For example, there is an emerging literature on genetic markers of risk for irritable bowel syndrome and functional dyspepsia (Yeo et al., 2004; Camilleri et al., 2002).

Despite the limited data, attachment theory and physiological studies indicate the importance of parent–child relationship and the caregiving environment on the development of FSS (Bowlby, 1973; Gunnar & Donzella, 2002). Unresponsive and/or rejecting parenting has been a risk factor for a number of child outcomes, especially in the presence of other risk factors (Campbell, Pierce, Moore, & Marakovitz, 1996; Shaw et al., 1998), and future studies of FSS should incorporate observational measures of parenting styles, particularly in young children. In addition, future research could test the *cumulative family adversity hypothesis*, which asserts that the number rather than type of risk factors increase the risk for maladaptive outcomes in children, to further our understanding of how family adversity, such as marital conflict and socioeconomic status (SES), influence the development of FSS. High family adversity, poor parenting, and low SES may interact in such a way to exponentially increase stress and anxiety in children's lives, for which they lack the skills to cope effectively.

The intent of the present developmental perspective is to encourage new directions of research to inform treatment interventions for children and adolescents with FSS. A limitation with the current review is that it assumes there is more commonality than dissimilarities among children who present with FSS across different pediatric specialty clinics, such as gastroenterology, neurology, and rheumatology (Wessely, Nimnuan, & Sharpe, 1999). Nevertheless, as more is learned about FSS, the model may need to be modified to fit certain pediatric specialties. The benefit of studying FSS together is that it can make treatments more generalized and diagnostic schemes may be more descriptive and more valuable to clinicians (Wessely et al., 1999).

A number of issues were not discussed in this review but are important to consider when exploring mechanisms of FSS. First, the potential differences in risk associated with different types of FSS (e.g., pain vs. nonpain symptoms) were not investigated due to the paucity of literature in this area. There are instances where the literature is much more developed for one particular symptom, such as abdominal pain, and it is unclear whether this literature can, in fact, be generalized to other FSS or multiple FSS. Second, functional impairment or disability may vary by symptom type or may differ according to the number of FSS present. For example, within the pediatric chronic pain literature, the prevalence of restrictions in daily activities, health care utilization, and medication use have been found to vary by pain location in children and adolescents (Roth-Isigkeit, Thyen, Stöven, Schwarzenberger, & Schmucker, 2005). Perquin et al. (2000) found that half of children who report having chronic pain have multiple pain (e.g., headache and back pain). This is of importance as multiple pain complaints, compared to single complaints, have been associated with higher levels of pain intensity and pain-related disability (Perquin et al., 2000).

Third, the current article integrated findings from studies that used community and tertiary care populations. It is important to note that tertiary care populations, compared to community populations, are likely to have severer symptoms of longer duration and to have developed secondary consequences of FSS. That is, the association between FSS and emotional symptomatology may be more pronounced in a tertiary care sample compared to a community sample, though, further research is needed to understand this causal link. Nevertheless, this article has demonstrated that the trends for risk and protective factors can be seen in both community and tertiary care samples. Finally, potential differences between parent and child report of FSS must also be considered. Garber, VanSlyke, and Walker (1998) found that mothers of children with recurrent abdominal pain reported more somatic symptoms than their children. In addition, mothers with higher levels of distress reported more child symptoms than did their children.

An obstacle that faces many medical providers is that they must provide an explanation for FSS to children and their families. Utilizing a developmental perspective of FSS can lessen the burden on pediatricians and medical specialists, particularly when psychological and psychiatric treatments are integrated into medical care as opposed to regarding them as separate (Sharpe & Carson, 2001). Medical providers are encouraged to openly discuss psychosocial and environmental factors

with families as this may provide multiple avenues for finding effective treatment interventions for children's FSS. The goal of conceptualizing FSS through a developmental lens is to decrease children's and adolescents' medical health care utilization and provide a quicker resolution of symptoms, distress, and disability with the use of an interdisciplinary treatment (see Bursch, Walco, & Zeltzer, 1998, for a treatment approach to pediatric chronic pain).

As a final note, developmental psychopathologists articulate that, even though the underlying liability to psychopathology may be dimensional, categorical distinctions are often necessary in the real world (Rutter & Sroufe, 2000). For example, clinicians have to decide whether or not to prescribe a psychotropic medication or to admit their patient to the hospital. Beyond that, insurance may not cover rendered services for patients if they do not meet diagnostic criteria. This being said, it is imperative that the diagnostic criteria for somatoform disorders outlined in the *DSM-IV*, particularly somatization disorder and pain disorder with psychological origin, be revised to be appropriate for children and adolescents. We must continue to look at FSS as both a continuous and discrete phenomenon, in order to determine whether the severity of the complaints designates a particular pathway, which could help prescribe treatment to these children and adolescents.

Acknowledgments

The author would like to gratefully acknowledge Dr Lynn S. Walker for her helpful comments on an earlier draft of this article. This work was supported in part by an NIMH training grant (T32-MH18921) and NICHD Grant P30HD15052.

Conflicts of interest: None declared.

Received March 6, 2007; revisions received October 2, 2007; accepted October 29, 2007

References

Achenbach, T. M., Conners, C., Quay, H. C., Verhulst, F. C., & Howell, C. T. (1989). Replication of empirically derived syndromes as a basis for taxonomy of child/adolescent psychopathology. *Journal of Abnormal Child Psychology, 17*(3), 299–323.

Alfven, G. (1993). The covariation of common psychosomatic symptoms among children from socio-economically differing residential areas. An epidemiological study. *Acta Paediatrica, 82*(5), 484–487.

Alfven, G., de la Torre, B., & Uvnas-Moberg, K. (1994). Depressed concentrations of oxytocin and cortisol in children with recurrent abdominal pain of non-organic origin. *Acta Paediatrica, 83*(10), 1076–1080.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders, 3rd edition-revised*. Washington, DC: American Psychiatric Association.

Aro, H. (1987). Life stress and psychosomatic symptoms among 14 to 16-year old Finnish adolescents. *Psychological Medicine, 17*(1), 191–201.

Aro, H. (1989). Stress, development and psychosomatic symptoms in adolescence: A comparison of the sexes. *Psychiatria Fennica, 20*, 101–109.

Aro, H., Paronen, O., & Aro, S. (1987). Psychosomatic symptoms among 14–26 year old Finnish adolescents. *Social Psychiatry, 22*(3), 171–176.

Aro, H., & Taipale, V. (1987). The impact of timing of puberty on psychosomatic symptoms among fourteen- to sixteen-year-old Finnish girls. *Child Development, 58*(1), 261–268.

Aromaa, M., Rautava, P., Helenius, H., & Sillanpaeae, M. (1998). Factors of early life as predictors of headache in children at school entry. *Headache, 38*(1), 23–30.

Aromaa, M., Sillanpaa, M., Rautava, P., & Helenius, H. (2000). Pain experience of children with headache and their families: A controlled study. *Pediatrics, 106*(2 Pt 1), 270–275.

Bandell-Hoekstra, I. E., Abu-Saad, H. H., Passchier, J., Frederiks, C. M., Feron, F. J., & Knipschild, P. (2002). Coping and quality of life in relation to headache in Dutch schoolchildren. *European Journal of Pain, 6*(4), 315–321.

Bauer, A. M., Quas, J. A., & Boyce, W. (2002). Associations between physiological reactivity and children's behavior: Advantages of a multisystem approach. *Journal of Developmental and Behavioral Pediatrics, 23*(2), 102–113.

Bernstein, G. A., Massie, E. D., Thuras, P. D., Perwien, A. R., Borchardt, C. M., & Crosby, R. D. (1997). Somatic symptoms in anxious-depressed school refusers. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*(5), 661–668.

Berntsson, L. T., & Kohler, L. (2001). Long-term illness and psychosomatic complaints in children aged 2–17 years in the five Nordic countries. Comparison between 1984 and 1996. *European Journal of Public Health, 11*(1), 35–42.

Boey, C. C. M., & Goh, K.-L. (2001). The significance of life-events as contributing factors in childhood recurrent abdominal pain in an urban community in Malaysia. *Journal of Psychosomatic Research, 51*(4), 559–562.

Borres, M. P., Tanaka, H., & Thulesius, O. (1998). Psychosomatic and psychosocial symptoms are associated with low blood pressure in Swedish schoolchildren. *Psychotherapy & Psychosomatics, 67*(2), 88–93.

Bowlby, J. (1973). *Attachment and loss: Attachment*. New York: Basic Books.

Boyce, W., Quas, J., Alkon, A., Smider, N. A., Essex, M. J., Kupfer, D. J., et al. (2001). Autonomic reactivity and psychopathology in middle childhood. *British Journal of Psychiatry, 179*, 144–150.

Boyer, M. C., Compas, B. E., Stanger, C., Colletti, R. B., Konik, B. S., Morrow, S. B., et al. (2006). Attentional biases to pain and social threat in children with recurrent abdominal pain. *Journal of Pediatric Psychology, 31*(2), 209–220.

Bschor, T. (2002). Masked depression: The rise and fall of a diagnosis. *Psychiat Prax, 29*, 207–210.

Bursch, B., Walco, G., & Zeltzer, L. (1998). Clinical assessment and management of chronic pain and pain-associated disability syndrome. *Developmental and Behavioral Pediatrics, 19*(1), 45–53.

Camilleri, M., Atanasova, E., Carlson, P. J., Ahmad, U., Kim, H. J., Viramontes, B. E., et al. (2002). Serotonin-transporter polymorphism pharmacogenetics in diarrhea-predominant irritable bowel syndrome. *Gastroenterology, 123*(2), 425–432.

Campbell, S. B., Pierce, E. W., Moore, G., & Marakovitz, S. (1996). Boys' externalizing problems at elementary school age: Pathways from early behavior problems, maternal control, and family stress. *Development & Psychopathology, 8*(4), 701–719.

Campo, J. V. (1995). Somatization disorder. In R. T. Ammerman, & M. Hersen (Eds.), *Handbook of child behavior therapy in the psychiatric setting Wiley series on personality processes*. (pp. 427–451). New York: Wiley.

Campo, J. V., & Fritsch, S. L. (1994). Somatization in children and adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry, 33*(9), 1223–1235.

Campo, J. V., & Garber, J. (1998). Somatization. In R. T. Ammerman, & J. V. Campo (Eds.), *Handbook of pediatric psychology and psychiatry: Psychological and psychiatric issues in the pediatric setting*. (pp. 137–161). Boston: Allyn & Bacon.

Campo, J. V., Jansen-McWilliams, L., Comer, D. M., & Kelleher, K. J. (1999). Somatization in pediatric primary care: Association with psychopathology, functional impairment, and use of services. *Journal of the American Academy of Child & Adolescent Psychiatry, 38*(9), 1093–1101.

Campo, J. V., & Reich, M. D. (1999). Somatoform disorders. In S. D. Netherton, & D. Holmes (Eds.), *Child & adolescent psychological disorders: A comprehensive textbook*. (pp. 320–343). New York: Oxford University Press.

Carlson, G. A., & Cantwell, D. P. (1980). Unmasking masked depression in children and adolescents. *American Journal of Psychiatry, 137*(4), 445–449.

Chapman, M. V. (2005). Neighborhood quality and somatic complaints among American youth. *Journal of Adolescent Health, 36*, 244–252.

Cicchetti, D., & Sroufe, L. (2000). The past as prologue to the future: The times, they've been a-changin'. *Development & Psychopathology, 12*(3), 255–264.

Compas, B. E., Conner-Smith, H. S., Salzman, H., Thomsen, A. H., & Wadsworth, M. E. (2001). Coping with stress during childhood and adolescence: Problems, progress, and potential in theory and research. *Psychological Bulletin, 127*(1), 87–127.

Craig, T., Boardman, A., Mills, K., Daly-Jones, O., & Drake, H. (1993). The South London Somatisation Study: I. Longitudinal course and the influence of early life experiences. *British Journal of Psychiatry, 163*, 579–588.

Craig, T., Cox, A., & Klein, K. (2002). Intergenerational transmission of somatization behaviour: A study of chronic somatizers and their children. *Psychological Medicine, 32*(5), 805–816.

Cummings, E. M., Davies, P. T., & Campbell, S. B. (2000). *Developmental psychopathology and family process: Theory, research, and clinical implications*. New York: Guilford Press.

Cunningham, S., McGrath, P., Ferguson, H., Humphreys, P., D'Astous, J., Latter, J., et al. (1987).

Personality and behavioural characteristics in pediatric migraine. *Headache, 27*(1), 16–20.

Dhossche, D., Ferdinand, R., van der Ende, J., & Verhulst, F. (2001). Outcome of self-reported functional-somatic symptoms in a community sample of adolescents. *Annals of Clinical Psychiatry, 13*(4), 191–199.

Domenech-Llaberia, E., Jane, M. C., Canals, J., Ballespi, S., Esparo, G., & Garralda, E. (2004). Parental reports of somatic symptoms in preschool children: Prevalence and associations in a Spanish sample. *Journal of the American Academy of Child & Adolescent Psychiatry, 43*(5), 598–604.

Dorn, L. D., Campo, J. C., Thato, S., Dahl, R. E., Lewin, D., Chandra, R., et al. (2003). Psychological comorbidity and stress reactivity in children and adolescents with recurrent abdominal pain and anxiety disorders. *Journal of the American Academy of Child & Adolescent Psychiatry, 42*(1), 66–75.

Egger, H. L., Angold, A., & Costello, E. (1998). Headaches and psychopathology in children and adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry, 37*(9), 951–958.

Egger, H. L., Costello, E., Erkanli, A., & Angold, A. (1999). Somatic complaints and psychopathology in children and adolescents: Stomach aches, musculoskeletal pains, and headaches. *Journal of the American Academy of Child & Adolescent Psychiatry, 38*(7), 852–860.

Ekbom, K., Svensson, D. A., Traff, H., & Waldenlind, E. (2002). Age at onset and sex ratio in cluster headache: Observations over three decades. *Cephalalgia, 22*(2), 94–100.

Fearon, P., & Hotopf, M. (2001). Relation between headache in childhood and physical and psychiatric symptoms in adulthood: National birth cohort study. *British Medical Journal, 322*(7295), 1145.

Freud, S. (1962). The aetiology of hysteria. In J. Strachey (Ed.), *The standard edition of the complete psychological works of Sigmund Freud*. (pp. 191–221). London: Hogarth Press.

Fritz, G. K., Fritsch, S., & Hagino, O. (1997). Somatoform disorders in children and adolescents: A review of the past 10 years. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*(10), 1329–1338.

Garber, J., VanSlyke, D. A., & Walker, L. S. (1998). Concordance between mothers' and children's reports of somatic and emotional symptoms in patients with recurrent abdominal pain or emotional disorders. *Journal of Abnormal Child Psychology, 26*(5), 381–391.

Garber, J., Walker, L. S., & Zeman, J. (1991). Somatization symptoms in a community sample of children and adolescents: Further validation of the Children's Somatization Inventory. *Psychological Assessment, 3*(4), 588–595.

Garber, J., Zeman, J., & Walker, L. S. (1990). Recurrent abdominal pain in children: Psychiatric diagnoses and parental psychopathology. *Journal of the American Academy of Child & Adolescent Psychiatry, 29*(4), 648–656.

Garralda, M. (1996). Somatisation in children. *Journal of Child Psychology and Psychiatry & Allied Disciplines, 37*(1), 13–33.

Gladstein, J., & Holden, E. (1996). Chronic daily headache in children and adolescents: A 2-year prospective study. *Headache, 36*(6), 349–351.

Guidetti, V., & Galli, F. (2001). Recent development in pediatric headache. *Current Opinion in Neurology, 14*(3), 335–340.

Gunnar, M. R., Bruce, J., & Hickman, S. E. (2001). Salivary cortisol response to stress in children. *Advances in Psychosomatic Medicine, 22*, 52–60.

Gunnar, M. R., & Donzella, B. (2002). Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology, 27*(1–2), 199–220.

Honjo, S., Nishide, T., Niwa, S., Sasaki, Y., Kaneko, H., Inoko, K., et al. (2001). School refusal and depression with school attendance in children and adolescents: Comparative assessment between the Children's Depression Inventory and somatic complaints. *Psychiatry & Clinical Neurosciences, 55*(6), 629–634.

Kashikar-Zuck, S., Goldschneider, K. R., Powers, S. W., Vaught, M. H., & Hershey, A. D. (2001). Depression and functional disability in chronic pediatric pain. *Clinical Journal of Pain, 17*(4), 341–349.

King, N. J., & Bernstein, G. A. (2001). School refusal in children and adolescents: A review of the past 10 years. *Journal of the American Academy of Child & Adolescent Psychiatry, 40*(2), 197–205.

Kowal, A., & Pritchard, D. (1990). Psychological characteristics of children who suffer from headache: A reasearch note. *Journal of Child Psychology and Psychiatry, 31*(4), 637–649.

Last, C. G. (1991). Somatic complaints in anxiety disordered children. *Journal of Anxiety Disorders, 5*(2), 125–138.

Lask, B., & Fosson, A. (1989). *Childhood illness: The psychosomatic approach: Children talking with their bodies*. New York: Wiley.

Livingston, R., Taylor, J., & Crawford, S. (1988). A study of somatic complaints and psychiatric diagnosis in children. *Journal of the American Academy of Child & Adolescent Psychiatry, 27*(2), 185–187.

Locke, G. R., Zinsmeister, A. R., Talley, N. J., Fett, S. L., & Melton, L. J. (2000). Familial association in adults with functional gastrointestinal disorders. *Mayo Clinic Proceedings, 75*(9), 907–912.

McCauley, E., Carlson, G. A., & Calderon, R. (1991). The role of somatic complaints in the diagnosis of depression in children and adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry, 30*(4), 631–635.

Minuchin, S., Baker, L., Rosman, B. L., Liebman, R., Milman, L., & Todd, T. C. (1975). A conceptual model of psychosomatic illness in children: Family organization and family therapy. *Archives of General Psychiatry, 32*(8), 1031–1038.

Moffitt, T. E. (1990). Juvenile delinquency and attention-deficit disorder: Developmental trajectories from age 3 to 15. *Child Development, 61*, 893–910.

Moffitt, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychological Review, 100*(4), 674–701.

Mulvaney, S., Lambert, E. W., Garber, J., & Walker, L. S. (2006). Trajectories of symptoms and impairment for pediatric patients with functional abdominal pain: A 5-year longitudinal study. *Journal of the American Academy of Child & Adolescent Psychiatry, 45*(6), 737–744.

Offord, D. R., Boyle, M. H., Szatmari, P., Rae-Grant, N. I., Links, P. S., Cadman, D. T., et al. (1987). Ontario Child Health Study: II. Six-month prevalence of disorder and rates of service utilization. *Archives of General Psychiatry, 44*(9), 832–836.

Perquin, C. W., Hazebroek-Kampschreur, A. A., Hunfeld, J. A., Bohnen, A. M., van Suijlekom-Smit, L. W., Passchier, J., et al. (2000). Pain in children and adolescents: A common experience. *Pain, 87*(1), 51–58.

Rauste-von Wright, M., & von Wright, J. (1981). A longitudinal study of psychosomatic symptoms in healthy 11–28 year old girls and boys. *Journal of Psychosomatic Research, 25*(6), 525–534.

Rhee, H. (2005). Relationships between physical symptoms and pubertal development. *Journal of Pediatric Health Care, 19*(2), 95–103.

Rief, W., Shaw, R., & Fichter, M. M. (1998). Elevated levels of psychophysiological arousal and cortisol in patients with somatization syndrome. *Psychosomatic Medicine, 60*(2), 198–203.

Rocha, E. M., Prkachin, K. M., Beaumont, S. L., Hardy, C. L., & Zumbo, B. D. (2003). Pain reactivity and somatization in kindergarten-age children. *Journal of Pediatric Psychology, 28*(1), 47–57.

Rothner, A. D. (1993). Diagnosis and management of headaches in children and adolescents. In N. L. Schechter, C. B. Berde, & M. Yaster (Eds.), *Pain in infants, children, and adolescents*. (pp. 547–554). Baltimore: Williams & Wilkins.

Roth-Isigkeit, A., Thyen, U., Stöven, H., Schwarzenberger, J., & Schmucker, P. (2005). Pain among children and adolescents: Restrictions in daily living and triggering factors. *Pediatrics, 115*(2), 152–162.

Rutter, M., & Sroufe, L. (2000). Developmental psychopathology: Concepts and challenges. *Development & Psychopathology, 12*(3), 265–296.

Ruchkin, V., Eisemann, M., & Haeggloef, B. (2000). Coping styles and psychosomatic problems: Are they related? *Psychopathology, 33*(5), 235–239.

Ryan, N. D., & Dahl, R. E. (1993). The biology of depression in children and adolescents. In D. J. Kupfer (Ed.), *Biology of depressive disorders, part B: Subtypes of depression and comorbid disorders*. New York: Plenum Press.

Scharff, L. (1997). Recurrent abdominal pain in children: A review of psychological factors and treatment. *Clinical Psychology Review, 17*(2), 145–166.

Sharpe, M., & Carson, A. (2001). ''Unexplained'' somatic symptoms, functional syndromes, and somatization: do we need a paradigm shift? *Annals of Internal Medicine, 134*(9 Pt 2), 926–30.

Shaw, D. S., Winslow, E. B., Owens, E. B., Vondra, J. I., Cohn, J. F., & Bell, R. Q. (1998). The development of early externalizing problems among children from low-income families: A transformational perspective. *Journal of Abnormal Child Psychology, 26*(2), 95–107.

Steinhausen, H.-C. (2006). Developmental psychopathology in adolescence: Findings from a Swiss study – the NAPE Lecture 2005. *Acta Psychiatrica Scandinavica, 113*, 6–12.

Stevenson, J., Simpson, J., & Bailey, V. (1988). Research note: recurrent headaches and stomachaches in preschool children. *Journal of Child Psychology and Psychiatry & Allied Disciplines, 29*(6), 897–900.

Stickney, M. I., & Miltenberger, R. G. (1998). School refusal behavior: Prevalence, characteristics, and the schools' response. *Education & Treatment of Children, 21*(2), 160–170.

Susman, E. J., Dorn, L. D., & Schiefelbein, V. L. (2003). Puberty, sexuality, and health. In R. M. Lerner, M. A. Easterbrooks, & J. Mistry (Eds.), *Handbook of psychology: Developmental psychology*. (Vol. 6, pp. 295–324). New York, NY: John Wiley & Sons, Inc.

Susman, E. J., Reiter, E. O., Ford, C., & Dorn, L. D. (2002). Work Group I: Developing models of healthy adolescent physical development. *Journal of Adolescent Health, 31*(Suppl 6), 171–174.

Terre, L., & Ghiselli, W. (1997). A developmental perspective on family risk factors in somatization. *Journal of Psychosomatic Research, 42*(2), 197–208.

Thomsen, A. H., Compas, B. E., Colletti, R. B., Stanger, C., Boyer, M. C., & Konik, B. S. (2002). Parent reports of coping and stress responses in children with recurrent abdominal pain. *Journal of Pediatric Psychology, 27*(3), 215–226.

Torsheim, T., & Wold, B. (2001). School-related stress, school support, and somatic complaints: A general population study. *Journal of Adolescent Research, 16*(3), 293–303.

Vannatta, K., Gartstein, M. A., Short, A., & Noll, R. B. (1998). A controlled study of peer relationships of children surviving brain tumors: Teacher, peer, and self ratings. *Journal of Pediatric Psychology, 23*(5), 279–287.

Walker, L. S., Claar, R. L., & Garber, J. (2002). Social consequences of children's pain: When do they encourage symptom maintenance? *Journal of Pediatric Psychology, 27*(8), 689–698.

Walker, L. S., Garber, J., & Greene, J. W. (1991). Somatization symptoms in pediatric abdominal pain patients: Relation to chronicity of abdominal pain and parent somatization. *Journal of Abnormal Child Psychology, 19*(4), 379–394.

Walker, L. S., Garber, J., & Greene, J. W. (1993). Psychosocial correlates of recurrent childhood pain: A comparison of pediatric patients with recurrent abdominal pain, organic illness, and psychiatric disorders. *Journal of Abnormal Psychology, 102*(2), 248–258.

Walker, L. S., Garber, J., & Greene, J. W. (1994). Somatic complaints in pediatric patients: A prospective study of the role of negative life events, child social and academic competence, and parental somatic symptoms. *Journal of Consulting & Clinical Psychology, 62*(6), 1213–1221.

Walker, L. S., Garber, J., Smith, C. A., Van Slyke, D. A., & Claar, R. L. (2001). The relation of daily stressors to somatic and emotional symptoms in children with and without recurrent abdominal pain. *Journal of Consulting & Clinical Psychology, 69*(1), 85–91.

Walker, L. S., Garber, J., Van Slyke, D. A., & Greene, J. W. (1995). Long-term health outcomes in patients with recurrent abdominal pain. *Journal of Pediatric Psychology, 20*(2), 233–245.

Walker, L. S., & Greene, J. W. (1989). Children with recurrent abdominal pain and their parents: More somatic complaints, anxiety, and depression than other patient families? *Journal of Pediatric Psychology, 14*(2), 231–243.

Walker, L. S., Smith, C. A., Garber, J., & Van Slyke, D. A. (1997). Development and validation of the Pain Response Inventory for children. *Psychological Assessment, 9*(4), 392–405.

Walker, L. S., Smith, C. A., Garber, J., & Claar, R. L. (2005). Testing a model of pain appraisal and coping in children with chronic abdominal pain. *Health Psychology, 24*(4), 364–374.

Walker, L. S., Smith, C. A., Garber, J., & Claar, R. L. (2007). Appraisal and coping with daily stressors by pediatric patients with chronic abdominal pain. *Journal of Pediatric Psychology, 32*(2), 206–216.

Walker, L. S., Williams, S. E., Smith, C. A., Garber, J., Van Slyke, D. A., & Lipani, T. A. (2006a). Parent attention versus distraction: Impact on symptom complaints by children with and without chronic functional abdominal pain. *Pain, 122*, 43–52.

Walker, L. S., Williams, S. E., Smith, C. A., Garber, J., Van Slyke, D. A., Lipani, T. A., et al. (2006b).

Validation of a symptom provocation test for laboratory studies of abdominal pain and discomfort in children and adolescents. *Journal of Pediatric Psychology, 31*(7), 703–713.

Wessely, S., Nimnuan, C., & Sharpe, M. (1999). Functional somatic syndromes: One or many? *Lancet, 354*(9182), 936–939.

Yeo, A., Boyd, P., Lumsden, S., Saunders, T., Handley, A., Stubbins, M., et al. (2004). Association between a functional polymorphism in the serotonin transporter gene and diarrhea predominant irritable bowel syndrome in women. *Gut, 53*(10), 1452–1458.

Zahn-Waxler, C., Klimes-Dougan, B., & Slattery, M. J. (2000). Internalizing problems of childhood and adolescence: Prospects, pitfalls, and progress in understanding the development of anxiety and depression. *Development & Psychopathology, 12*(3), 443–466.

Zuckerman, B., Stevenson, J., & Bailey, V. (1987). Stomachaches and headaches in a community sample of preschool children. *Pediatrics, 79*(5), 677–682.

Zwaigenbaum, L., Szatmari, P., Boyle, M. H., & Offord, D. R. (1999). Highly somatizing young adolescents and the risk of depression. *Pediatrics, 103*(6 Pt 1), 1203–1209.