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Vulnerability for Functional Somatic Disorders: A Contemporary Psychodynamic Approach

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Patients with functional somatic disorders (FSD) are markedly heterogeneous with regard to the factors contributing to their illness, their symptoms, and treatment response. In this article, we present a contemporary psychodynamic approach to the conceptualization and treatment of these patients based on attachment and mentalization theory. Extant research is reviewed that suggests a key role for attachment history and mentalization in determining stress and affect regulation, and immune and pain-regulating systems. We focus more specifically on the high interpersonal and metabolic costs associated with the excessive use of insecure secondary attachment strategies in response to stress, and the associated impairments in (embodied) mentalization in patients, both as a cause and consequence of FSD. Finally, a new brief psychodynamic intervention for patients with functional somatic complaints is discussed.

Keywords: functional somatic disorders, attachment, mentalizing, psychodynamic

Patients with functional somatic disorders (FSD) or persistent somatic complaints are highly prevalent in routine clinical practice. They are known to be high utilizers of medical care (Afari & Buchwald, 2003; Annemans, Le Lay, & Taeb, 2009; Annemans et al., 2008; Sicras-Mainar et al., 2009; Spaeth, 2009), and are often considered to be "difficult to treat" (Fischhoff & Wessely, 2003). Clinicians often become entangled in difficult transference–countertransference patterns with these patients; patterns which are often further exacerbated by the use of unhelpful diagnostic labels both by patients and professionals, the neglect of the obvious role of biological factors in these disorders, and negative responses to these patients from their environment (Blom et al., 2012; Kool, Middendorp, Boeije, & Geenen, 2009).

Research over the past decades has clearly shown that different FSD are not isolated disorders. Studies showing high comorbidity among these syndromes and high familial coaggregation (Aggarwal, McBeth, Zakrzewska, Lunt, & Macfarlane, 2006; Anda et al., 2006) suggest that they are part of a spectrum of functional somatic syndromes including Chronic Fatigue Syndrome (CFS), Fibromyalgia (FM), Irritable Bowel Syndrome (IBS), temporomandibulair pain syndrome, chronic pelvic pain, and multiple chemical sensitivity (Ablin, et al., in press; Wessely & White, 2004). Im-

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portantly, there is also high comorbidity among FSD and emotional disorders such as depression and anxiety (Arnold et al., 2006; Pae et al., 2008), leading to the hypothesis that they are also part of a spectrum of affective disorders (Hudson, Arnold, Keck, Auchenbach, & Pope, 2004; Hudson et al., 2003; Hudson & Pope, 1996).

It is clear that these findings call for a developmental, person-centered, and transdiagnostic approach. Traditional disorder-centered approaches in this area have undeniably been overly concerned with the search for the unique causes of each of these disorders, instead of tracing developmental pathways from infancy to adulthood implicated in vulnerability for functional somatic complaints more generally. Such a developmental, person-centered approach is at the heart of contemporary psychodynamic approaches (Luyten, Mayes, Target, & Fonagy, 2012; Westen, 1998). Rather than focusing on vulnerability for different types of FSD, psychodynamic approaches typically take a person-centered approach that attempts to map developmental pathways of individuals that are vulnerable to develop persistent somatic complaints. Studies in this context suggest a complex interplay among both vulnerability and resilience factors (Van Houdenhove & Luyten, 2008), and have generated knowledge that has the clear potential to inform treatment and prevention efforts. Moreover, psychodynamic approaches also focus on the subjective experiences of individuals affected by FSD. We focus on how FSD is typically associated with a severe collapse of subjectivity and its impact on interpersonal relationships and the course of FSD. This focus on the internal world, and particularly the focus on the rooting of individual's internal world in interpersonal relationships, is what differentiates a psychodynamic approach from most other approaches in the field of behavioral medicine, which tend to center on biological, cognitive-perceptual, and behavioral factors. Clearly, however, with the advent of acceptance and commitment therapies as well as mindfulness based approaches, and a growing awareness in the field of the role of attachment experiences and experiential processes more generally (Brooks, Rimes, & Chalder, 2011; Hambrook et al., 2011; Lumley, 2011), there is increasing consensus concerning the importance of an interpersonal/intersubjective approach to FSD. These trends provide interesting opportunities to build bridges between different theoretical approaches, as this special issue attests.

We first discuss our views concerning the nature of FSD from a contemporary psychodynamic perspective rooted in mentalizing and attachment theory. These views reflect our ongoing attempts to integrate empirical findings regarding the neurobiology of FSD into a coherent theoretical model of the nature of FSD. Next, we discuss the treatment format that we have developed based on this theoretical approach.

A Mentalization-Based Approach To FSD

A Working Model of FSD

FSD are quite heterogeneous disorders in terms of their causes, course, and treatment response (Aslakson, Vollmer-Conna, Reeves, & White, 2009; Dadabhoy, 2006). Research findings amply demonstrate that complex interactions among biological and psychosocial factors are involved in the causation as well as maintenance of FSD (Heim et al., 2009; Van Houdenhove & Luyten, 2007, 2008; Yunus, 2008).

For some time, based on contemporary research findings, we have advanced a theoretical model of FSD that essentially proposes that FSD result from negative vicious cycles as a result of person-environment interactions (see Figure 1; Luyten & Van Houdenhove, in press; Van Houdenhove & Luyten, 2009). The model distinguishes predisposing, precipitating, and perpetuating factors. Research has shown that both biological and environmental factors may *predispose* individuals to FSD. For instance, there is increasing evidence for the role of genetic polymorphisms in FSD, as well as early adversity and most probably interactions between both (Buskila, Sarzi-Puttini, & Ablin, 2007; Heim et al., 2009; Rajeevan et al., 2007). *Precipitating factors* that have been identified are both of a psychological (e.g., problems related to work, relationships; Aslakson et al., 2009) and physiological (e.g., chronic infections or whiplash) nature. It is highly likely that an accumulation of these factors disturb allostasis, resulting in what has been termed *allostatic load* (McEwen, 2007), a state that entails a fundamental disturbance of the dynamic equi-

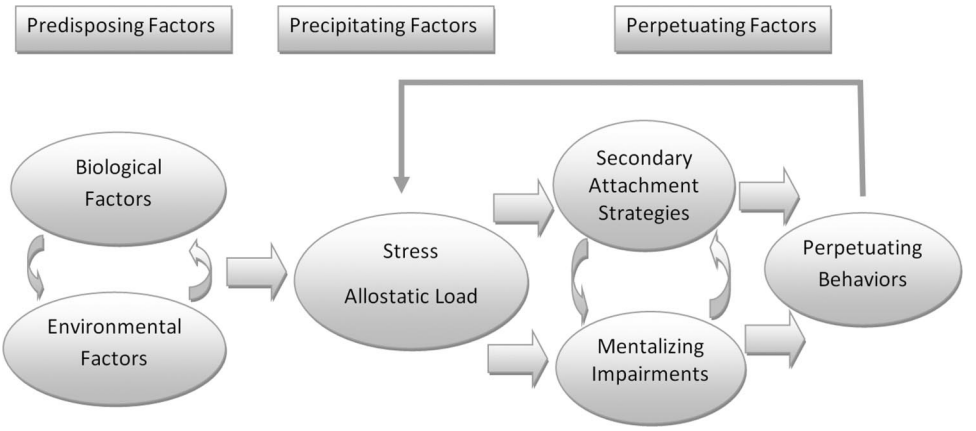

Figure 1. A mentalization-based approach to functional somatic disorders.

librium that normally characterizes stress regulation. Most probably, this is mediated by dysfunctions of the Hypothalamus Pituitary Adrenal (HPA) axis, the main human stress system (Heim et al., 2009; Tak & Rosmalen, 2010). These dysfunctions are furthermore associated with abnormal inflammatory activity, with pro-inflammatory cytokines inducing feelings of lethargia, increased fatigability, concentration loss, light fever, generalized hyperalgesia, and hypersensitivity to stress, and a tendency to withdraw from the outside world (Dantzer, O'Connor, Freund, Johnson, & Kelley, 2008; Watkins & Maier, 2005). In combination with neuroplastic changes in the spinal cord and brain, and cognitive, emotional and behavioral factors (e.g., negative affect, catastrophizing, and sleep problems), this often leads to pain sensitizing (Ablin et al., in press; Van Houdenhove & Luyten, 2008).

Regardless of the cause of this "biopsychosocial crash," in an often desperate attempt to regulate increasing levels of stress and anxiety, patients increasingly rely on so-called secondary attachment strategies (i.e., attachment deactivating and attachment hyperactivating strategies) which in turn lead to impairments in mentalizing, and embodied mentalizing in particular (i.e., the capacity to see the body as the seat of emotions, wishes, and feelings and the capacity to reflect on one's own bodily experiences and sensations and their relationships to intentional mental states in the self and others). Secondary attachment strategies and mentalizing impairments lead to an exacerbation of symptoms, further stress and thus, allostatic load (e.g., as a result of excessive catastrophizing and somatic attributions; see Figure 1).

Hence, individuals that are vulnerable for FSD may in part generate their own stressful environment and/or may be particularly vulnerable for the development of FSD when in an environment that impinges upon their vulnerability for stress, congruent with person-environment transactional models (Hammen, 2005; Luyten et al., 2011; Shahar, 2006).

Attachment, Mentalization, and Stress Regulation: A Developmental Perspective

In support of the model outlined above, burgeoning research indeed suggests inherent relationships among attachment experiences, mentalization, and stress regulation throughout the life cycle. Research in animals as well as humans strongly suggests that secure attachment experiences play a quintessential role in the development of the stress system and the development of resilience when faced with adversity (Gunnar & Quevedo, 2007).

In securely attached individuals, stress typically leads to the seeking of proximity to attachment figures, either real or internalized, resulting in the downregulation of stress. Normative stress regulation thus always involves the effective coregulation of stress in relation to attachment figures (Diamond & Aspinwall, 2003; Luyten, Mayes, Fonagy, & van Houdenhove,

2010; Sbarra & Hazan, 2008). This process appears to be firmly rooted in neurobiology. The neuropeptide oxytocin, for instance, has been shown to play a key role both in fostering attachment, mentalization, as well as in regulating stress (Fonagy & Luyten, 2009; Neumann, 2008). From a neurobiological perspective, activation of the attachment system has been shown to lead to (a) the activation of a mesocorticolimbic, dopaminergic "reward" system (Insel & Young, 2001); (b) downregulation of neuroendocrine stress regulation systems (HPA axis and sympathetic nervous system); and (c) activation of neural systems involved in mentalization, including the lateral prefrontal cortex, medial prefrontal cortex, lateral parietal cortex, medial parietal cortex, medial temporal lobe, and rostral anterior cingulated cortex (Fonagy & Luyten, 2009; Lieberman, 2007). High levels of mentalization, particularly under high levels of stress, have been associated with resilience (Fonagy, Steele, Steele, Higgitt, & Target, 1994) through so-called "broaden and build" (Fredrickson, 2001) cycles of attachment security, which reinforce feelings of secure attachment, agency, and stress and affect regulation ("build"), leading one to being "pulled into" more adaptive environments ("broaden"; Hauser, Allen, & Golden, 2006; Mikulincer & Shaver, 2007).

Secure attachment experiences, through their rewarding nature, reinforce affiliative behavior and mentalization, fostering the effective regulation of stress. Insecure attachment experiences (and early adversity in particular), by contrast, have been associated with greater vulnerability for stress in both animals (Champagne & Curley, 2009; Neumann, 2008) and humans (Bakermans-Kranenburg, Van Ijzendoorn, Mesman, Alink, & Juffer, 2008; Gunnar & Quevedo, 2007). This vulnerability seems to be in large part mediated by HPA axis dysfunctions (Heim et al., 2009; Kempke et al., in press; Van Houdenhove & Luyten, 2008).

Again, there is ample empirical research to support these claims. Although until recently studies concerning the role of early adversity in FSD where still controversial due to their many methodological limitations, there is now good evidence to suggest that at least a subgroup of patients with FSD are characterized by high levels of early adversity (particularly high levels of emotional abuse and neglect; Kempke et al., in press; Van Houdenhove, Luyten, & Egle, 2009), and insecure attachment (Luyten, Van Houdenhove, Cosyns, & Van den Broeck, 2006; Maunder & Hunter, 2008; Waller & Scheidt, 2006). Typically, individuals characterized by insecure attachment histories rely excessively on so-called secondary attachment strategies in response to stress (Dozier & Kobak, 1992; Mikulincer & Shaver, 2007; Roisman et al., 2007). These entail so-called attachment deactivating or attachment hyperactivating strategies, or a combination of both. Yet, not all patients with FSD are characterized by a history of insecure attachment experiences, let alone that all patients with FSD have a history of serious early adversity and/or premorbid mentalizing impairments. The overreliance on secondary attachment strategies and mentalizing impairments observed in many patients with FSD may also be a consequence of the disorder, or may be further exacerbated by the disorder, which has important treatment implications. Earlier psychoanalytic theories that argued for a close causal association between early adversity and problems with mentalization in FSD were clearly overspecified, mostly based on outdated assumptions concerning mind-body relationships, and failed to account for biological findings in FSD (Luyten et al., 2012; Luyten & Van Houdenhove, in press). Fellow scientists, as well as patients and patient organizations, have rightfully questioned some of these oversimplistic views that often only serve to increase patient's feelings of invalidation, lack of understanding, and embitterment (Blom et al., 2012; Kool et al., 2009).

Both clinical practice and research suggest that many patients with FSD, in an attempt to cope with their illness, begin to excessively rely on *attachment deactivation* strategies, expressed in an often complete denial of attachment needs, assertion of their autonomy, independence, and strength (Cassidy & Kobak, 1988; Mikulincer & Shaver, 2007). Yet, underneath this appearance of autonomy and resilience, there is much vulnerability (Van Houdenhove & Luyten, 2008). The use of attachment deactivation strategies, studies suggest, is also often associated with high levels of self-critical perfectionism and related features such as persistence, overactivity, and so-called "all-or-nothing behavior" (Creed, 2007; Luyten et al., 2011). These represent a defensive attempt to

affirm the self and soothe negative introjects, and there is now increasing evidence that these features are related to FSD in at least a subset of patients (Luyten et al., 2011).

In the long run, these tendencies are associated with considerable interpersonal and metabolic costs. Attachment deactivating strategies (and high levels of self-critical perfectionism in particular) have been shown to lead to increasing isolation and loneliness (Mikulincer & Shaver, 2007), and the suppression of distress has been associated with increasing allostatic load, HPA axis hypoactivity because of the "wear and tear" of prolonged stress (Hill-Soderlund et al., 2008; Miller, Chen, & Zhou, 2007; Wirtz, Siegrist, Rimmele, & Ehlert, 2008), and disturbed immune functioning (Gouin et al., 2009). Further, deactivating strategies progressively fail under increasing stress, expressed in heightened feelings of stress and insecurity (Mikulincer, Dolev, & Shaver, 2004).

Particularly in patients with a history of serious early adversity and/or comorbid dependent and borderline features, *attachment hyperactivating strategies* tend to predominate. This is shown in anxious efforts to find support and relief, often through demanding, clinging, and claiming behavior (Waller & Scheidt, 2006). These strategies are similarly associated with high interpersonal and metabolic costs. Demanding behavior tends to lead to frustration and resentment in others, often confirming these individuals' worst fear that they are misunderstood and rejected by others. Relationships with (mental) health professionals mimic this pattern. Thus, "broaden and build cycles" are inhibited. High levels of physiological stress and increased HPA axis activity (Diamond, Hicks, & Otter-Henderson, 2008; Gordon et al., 2008) further increase "allostatic load" (McEwen, 2007), leading to a vicious cycle because these patients' tendency to respond to increased stress and anxiety with even greater reliance on attachment hyperactivating strategies in an attempt to find relief, support, and understanding (Maunder & Hunter, 2008; Maunder, Lancee, Nolan, Hunter, & Tannenbaum, 2006).

Impairments in (Embodied) Mentalization in FSD

Symptoms and the resulting excessive use of secondary attachment strategies in patients with FSD also has a negative effect on patients' mentalizing abilities, leading to the reemergence of nonmentalizing modes that in turn lead to behaviors that further perpetuate symptoms and exacerbate problems in interpersonal relationships (see Figure 1). As noted, mentalizing impairments are often the consequence of FSD or are further exacerbated by distress and interpersonal problems associated with FSD. FSD symptoms can indeed be seen as an "attack" from within on the capacity to reflect, particularly on the capacity to see the body as a "lived body" that one has ownership of and that is the seat of our relationships with others. Driver (2005), for instance, has vividly described the "otherness of the illness" in patients with Chronic Fatigue Syndrome, leading to regressive fears and fantasies. Shahar and colleagues, in turn, found that patients with a chronic illness typically treat their illness as an "internal object" that constantly threatens the individual and that needs to be negotiated with and soothed (Schattner, Shahar, & Abu-Shakra, 2008). Persistent somatic complaints furthermore increase stress, which further impairs and/or exacerbates already existing impairments in (embodied) mentalizing, congruent with studies showing an inverse relationship between stress and mentalizing (Fonagy & Luyten, 2009; Luyten, Fonagy, Lowyck, & Vermote, 2012).

Psychoanalytic approaches have long hypothesized mentalizing deficits in FSD patients. More specifically, earlier formulations emphasized high levels of alexithymia and problems with emotional awareness in these patients (Pedrosa Gil, Scheidt, Hoeger, & Nickel, 2008; Pedrosa Gil et al., 2008; Subic-Wrana, Beutel, Knebel, & Lane, 2010). Yet, studies suggest that only a minority of patients with FSD (those with substantial early adversity) are characterized by clinically elevated levels of alexithymia and lack of emotional awareness (Pedrosa Gil et al., 2008; Pedrosa Gil et al., 2008; Waller & Scheidt, 2006). Moreover, these features are not specific for FSD, but rather seem to reflect the effects of trauma and emotional neglect.

The mentalization-based approach proposed in this article suggests that rather than considering patients with FSD as generally "alexithymic," impairments in (embodied) mentalizing in these patients are much more *specific* (i.e., related to specific experiences and symptoms) and related to (interpersonal) situations and symp-

toms that involve *high arousal or stress* (Luyten, Van Houdenhove, Lemma, Target, & Fonagy, in press). In fact, both clinical experience and systematic research shows that many of these patients exhibit, interchangeably, excessive or so-called hypermentalization on the one hand (as expressed in apparently highly sophisticated narratives that lack any affective grounding in subjective experiences), as well as an almost total denial of the importance of inner mental states on the other. Rather than general impairments in emotional awareness, many of these patients suffer from an inability to link emotional and bodily states (Subic-Wrana et al., 2010). Studies indeed suggest that patients with FSD are less likely to interpret physical sensations in terms of negative emotional states (Dendy, Cooper, & Sharpe, 2001) and are less interoceptively accurate, particularly in symptom-related contexts (Bogaerts et al., 2008; Bogaerts et al., 2010). They also tend to have negative beliefs about emotions and particularly the expression of emotions (Hambrook et al., 2011), and tend to exhibit a strong need to control thoughts and feelings (Maher-Edwards, Fernie, Murphy, Nikcevic, & Spada, in press; Rimes & Chalder, 2010).

Nonmentalizing Modes of Experiencing Subjectivity and Perpetuating Factors

Context-specific impairments in (embodied) mentalizing lead to the reemergence of so-called nonmentalizing modes that perpetuate symptoms and interpersonal problems (see Figure 1).

The first nonmentalizing mode that plays an important role in FSD is the *psychic equivalence mode*. In this mode of experiencing subjectivity, patients equate inner and outer reality. Because of this, there is no room for alternative interpretations. In FSD patients, this is often associated with a lack of desire and/or inability to explore inner mental states, which hampers treatment. This is particularly the case in patients that primarily use attachment deactivating strategies, which also may explain these patients' problems in accepting help and their difficulties believing that professionals can be genuinely concerned about them. Psychic equivalence leads to equating psychological and physical pain, and emotional and physical exhaustion. This may in part explain the high comorbidity between pain, fatigue, and depression (Hudson et al., 2004; Van Houdenhove & Luyten, 2008). It also fosters these patients' resistance toward acknowledging the role of psychological factors ("I am exhausted, not angry or depressed"). They tend to experience psychological pain in terms of bodily pain, worries literally "de-press" the patient, and feel like a painful heavy weight. Helplessness ensues, often in combination with catastrophizing ("I think there is something terribly wrong with me, so there is something terribly wrong with me [= psychic equivalence], but no one notices me [= feeling of invalidation], I am beyond help [= catastrophizing]"). Psychic equivalence also negatively influences relationships. Thinking that others do not care means that others do not care. In case others criticize the patient, this is felt as an attack on the (bodily) integrity of the self which literally is felt as threatening the integrity of the self. Research findings concerning common neural circuits of psychological and physical pain are particularly relevant in this context. Rejection hurts (Eisenberger, Lieberman, & Williams, 2003), but in these patients often only the physical pain associated with rejection seems to be real ("I felt upset when my wife left me, but I can handle that, but it's these pains that are killing me"). In a psychic equivalence mode, one's body starts feeling like an "alien self-part," "a thing that is out of control." As a result, patients are under constant pressure to externalize these alien self parts in a defensive attempt to evacuate pain and feelings of anxiety, helplessness, and depression in an attempt to restore the coherence of the self. The result is that others are made to feel what the patient feels, which often has a destructive influence on relationships, including those with health professionals.

In a *teleological mode*, there is a recognition of mental states as driving behavior, but this is limited to mental states that have clearly observable causes (i.e., observable behavior reflecting rational, goal-directed behavior, and material causes). For many patients with FSD, only rational, goal-directed behaviors and actions can be effective. Hence, their tendency is to be excessively concerned to find "objective proof" of their illness. In a teleological mode, clinicians may be drawn into endless discussions about the purported role of biological versus psychosocial factors involved in the causation of FSD.

This tendency for rumination about the causes of their disorder often leads to what is called hypermentalization or "mentalization on the loose" in *an extreme pretend mode*. Typically for this mode of thinking is that the relationship between thoughts and feelings and reality is severed. Pretend mode functioning may give rise to often extensive narratives that on first impression strike the clinician as sensible accounts of the patients' history and the factors contributing to his or her problems. Yet, on closer consideration, these narratives are often overly analytical and cognitive, lacking any grounding in real affective experiences. They are also often very repetitive and the patient typically has an inability to switch perspectives, and attempts to do this are often met with fierce resistance ("what do you mean by considering what she might feel, we are talking about me").

As noted, prementalizing modes perpetuate both symptoms and problems in interpersonal relationships. Pretend mode thinking leads to excessive worrying and rumination, expressed in symptoms such as excessive anxious concerns about one's health, sleeping problems, and sometimes anxiety attacks, which further impair stress regulation. Associated with this, is the tendency to mull over what patients experience as an unwillingness of others to take their illness seriously (Van Houdenhove & Luyten, 2008), a tendency which studies have shown is related to these patients' feelings of invalidation, loneliness, and even embitterment (Kool et al., 2009). These feelings then are further increased by psychic equivalence thinking ("nobody cares"), often given rise to a paranoid-like distrust in the medical profession ("They want to harm us"). Teleological thinking often leads to desperate attempts to find relief in surgery (e.g., having all fillings of teeth replaced), experimental biological treatments, or alternative medicine. At the same time, many patients attempt to cope with feelings of worthlessness by desperate attempts to prove the contrary, leading to overactivity, often resulting in total agony, exhaustion, and helplessness.

Dynamic Interpersonal Therapy For FSD: Basic Principles

Based on these views, we have advocated the need for a broad, multidisciplinary treatment approach of patients with FSD, which attends to both biological and psychosocial factors (Luyten, Kempke, & Van Houdenhove, 2008; Luyten & Van Houdenhove, in press; Van Houdenhove & Luyten, 2009; Van Houdenhove & Luyten, 2010). Rather than disorder-specific interventions, congruent with the person-centered perspective taken in this article, we have proposed that broad spectrum interventions that can be tailored to the specific patient and his or her needs are perhaps more fruitful than developing specific treatments for separate FSD. This is not to deny the heterogeneity of these disorders, but this perspective emphasizes the commonalities in terms of dysfunctions in the stress response as well as the view that symptoms reflect responses to threats to the attachment system and mentalizing impairments.

The central overarching principle of treatment for FSD therefore entails a focus on restoring the capacity for stress regulation by fostering (a) the use of more adaptive attachment strategies in response to stress, and (b) recovery of the capacity for (embodied) mentalizing (Luyten & Van Houdenhove, in press). More recently (Luyten et al., in press), we have also developed a brief psychodynamic treatment for these patients based on principles outlined by Dynamic Interpersonal Treatment, an integrative treatment approach initially developed for depression (Lemma, Target, & Fonagy, 2011).

Dynamic Interpersonal Therapy for Functional Somatic Disorders (DIT-FSD) consists of three phases. The first phase focuses on the engagement of the patient, given the obvious problems many of these patients have to form a treatment alliance, and the collaborative formulation of a treatment focus (Sessions 1–4). In this phase, the acknowledgment of feelings of invalidation and experiences of a lack of understanding (Kool et al., 2009) are central. This typically also includes a discussion of the anxieties that starting the therapy activate, which is often crucially important to prevent pseudo engagement and/or early treatment drop out.

Further, in this phase, a discussion of unhelpful illness theories, and particularly the high "costs" that are associated with insecure attachment strategies, is often indicated. This may lead to the formulation of a shared and acceptable illness theory that recognizes the complexity of the disorder through consensus rather conflict (Salmon, 2007).

A final central aspect of the early phase of treatment is the joint formulation of what is called an Interpersonal Affective Focus (IPAF), which becomes the central focus in the treatment. The IPAF refers to a recurrent cognitive-affective relational or attachment pattern that is associated with the onset and perpetuation of functional somatic symptoms and interpersonal problems. It consists of an (nonconscious) representation of self-in-relation-to-others and the defensive function of this constellation. Patients that typically use attachment deactivating strategies, for instance, often depict the self as highly autonomous and self-reliant, while others are described as critical, ambivalent, and nonunderstanding. Affects accompanying this IPAF often include aggression, depression, and helplessness. However, this constellation typically defends against feelings of dependency and longings for approval and love. The IPAF in patients that primarily rely on attachment hyperactivating patients may consist of a representation of the self as caring and concerned about others (sometimes leading to compulsive caregiving). Others, by contrast, are described as indifferent and uncaring, which gives rise to feelings of helplessness and hopelessness. Yet, this constellation defends against feelings of frustration, resentment, and aggression toward others that are seen as unresponsive and uncaring.

The second phase of DIT-FSD consists of the working through of the IPAF and consolidation of treatment gains (Session 5–12). The IPAF is used as a guide to explore the high allostatic and interpersonal costs of typical interpersonal patterns with the aim to foster patients' capacities to reflect on the (bodily) self, others, and the self-in-relation-to-others. This entails in most patients exploring impairments in embodied mentalizing, which may lead to a reinvestment of the body with positive affective meaning through decoupling bodily from relational experiences. Experiencing the interpersonal world in terms of mental states, as this is intended, in and of itself brings about relief from physical stress which the collapse of mentalizing brings with it for these individuals. Further, the intrinsic relationship between symptoms and complaints and feelings as well as interpersonal relationships is explored. This entails both affect differentiation (e.g., recognizing that feeling "not good" actually means that one feels sad, rejected, as well as depressed), affect amplification (e.g., recognizing the influence of emotional states on the self), and linking these to interpersonal experiences (e.g., that one feels so tired because one feels rejected and misunderstood).

This fosters a move from narrative incoherence and inconsistence both with regard to the past and the present, toward a more consistent narrative about one's life history, present feelings and relationships. It also opens up future possibilities to live one's life differently. Indeed, more effective stress regulation and ways of relating to others fosters broaden and build cycles. Besides more basic mentalizing (e.g., affect recognition, differentiation, and amplification) and supportive interventions (i.e., reassurance, support, empathy), more advanced expressive interventions (e.g., interpretations of interpersonal patterns, including the transference when appropriate) as well as directive (e.g., encouraging new ways of relating) techniques, are used in this phase.

The final phase of treatment (Session 13–16) focuses on the end of the treatment and aims to transfer what one has "learned" in the treatment to the everyday context of the patient to prevent future relapses. This process is initiated by the sharing of a draft "goodbye" letter that is written by the therapist. The goodbye letter contains a summary of what has been achieved in the treatment, but also what has not been achieved. Its aim is to foster mentalizing with regard to what has and what has not been gained from the treatment. Particularly with FSD patients whom often have a long illness history, DIT-FSD only marks the beginning of a change process. The letter also serves as a "reminder" of the treatment after it has ended; a physical remnant of the treatment that patients can fall back upon in times of difficulty for instance. This letter typically evokes strong reactions: It is often experienced as supportive and challenging at the same time, and typically leads to an intensification of fears about the end of treatment and issues related to the IPAF. This provides another opportunity to explore the link between symptoms, subjective responses, and relationships. Hence, the aim of this phase is to foster autonomy and resilience in the long run. The use of directive techniques is quite pertinent in this phase of the treatment, as patients are actively encouraged to think about and try-out

news ways of thinking, feeling, and relating to others and themselves. Besides DIT-FSD, psychiatric consultation and rehabilitation may be indicated, as well as couple/family treatment in some cases, as a substantial proportion of these patients are trapped in negative vicious cycles that are not easy to change.

Conclusions

Past efforts to conceptualize and treat patients with FSD have mostly taken a disorder-centered perspective, thereby neglecting not only similarities between different types of FSD, but also insufficiently taking into account the subjective experience of patients suffering from these highly disabling disorders, and their impact on the course of these disorders. Psychodynamic approaches, by contrast, are more person-centered and have a strong focus on subjectivity—how individuals respond to both internal and external adverse experiences (such as having chronic, persistent somatic complaints that seriously challenge affect regulation strategies and interpersonal relationships) and employ a host of both adaptive and maladaptive strategies to deal with these experiences. Although this approach does not neglect differences among disorders, on the whole, it points to important commonalities, both in terms of the excessive use of secondary attachment strategies and impairments in (embodied) mentalization, both as a cause and as a consequence, in FSD.

These findings and assumptions have important implications for treatment, as currently many treatments take these factors insufficiently into account (Lumley, 2011; Luyten et al., 2011). This may explain, in part, the limited effectiveness of most current treatments. And although specific psychodynamic treatment modalities have received empirical support in the treatment of FSD (Abbass, Kisely, & Kroenke, 2009; Francis et al., 2003; Sattel et al., 2012), there is much room for improvement for treatment outcome.

In response, we have developed a brief treatment for patients with DIT-FSD, which is currently being piloted. Yet, congruent with the views expressed in this article, maintenance treatment and/or long-term treatment may be indicated from the outset in a substantial number of patients, as restoring allostasis—both at the psychological and biological level—may take considerable time in many patients. Moreover, a multidisciplinary approach may be needed, particularly in chronically ill patients as they suffer from serious biological dysregulations, as well as personal, interpersonal, social, and work-related issues (Van Houdenhove & Luyten, 2007). Many of these patients, because of the impact of FSD on their lives, often in combination with serious premorbid psychosocial problems, need considerable time before they are able to "catch up" with normal developmental and psychosocial tasks and issues, and are able to find a new equilibrium. As our insights into the complex nature of FSD increases, the hope is that more effective treatments may emerge as well.

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