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A mentalization-based approach to the understanding and treatment of functional somatic disorders

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Patients with functional somatic disorders (FSD) represent a sizeable group in our health care system. FSD are associated with high health care use and considerable personal and economic costs. Evidence-based treatments for FSD are only modestly effective in a large subgroup of patients, particularly in the long run, which emphasizes the need to develop more effective treatments rooted in extant knowledge about the nature of FSD. This paper presents a contemporary psychodynamic perspective on the conceptualization and treatment of patients with FSD rooted in attachment and mentalization theory. First, we review animal and human research demonstrating the close relationships among attachment, stress regulation, and immune and pain-regulating systems. We highlight research findings concerning the high interpersonal and metabolic costs associated with the use of insecure secondary attachment strategies (i.e. attachment deactivating and hyperactivating strategies) leading to increased vulnerability for stress. Next, we review evidence for the role of impairments in (embodied) mentalization in patients with FSD both as cause and consequence of functional somatic complaints, leading to the re-emergence of so-called non-mentalizing modes, i.e. modes of subjectivity that antedate the capacity for full mentalizing. Based on these views, a novel brief psychodynamic intervention for patients with functional somatic complaints is presented.

Keywords: functional somatic disorders; somatoform disorders; mentalization; attachment; stress

Introduction

Functional somatic syndromes are part of a spectrum of disorders, including chronic fatigue syndrome (CFS), fibromyalgia (FM) and irritable bowel syndrome (IBS), but most probably also other disorders such as temporomandibular pain syndrome, chronic pelvic pain and multiple chemical sensitivity (Ablin et al., 2012; Wessely & White, 2004). This assumption is borne out by findings of high comorbidity among these syndromes and high familial coaggregation (Aggarwal, McBeth, Zakrzewska, Lunt, & Macfarlane, 2006; Anda et al., 2006). Studies suggest that up to 4% of the general population and up to 9% of patients in tertiary

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care suffer from multiple chronic functional somatic disorders (Bass & May, 2002). Functional somatic disorders (FSD) also have high comorbidity with emotional disorders such as depression and anxiety (Arnold et al., 2006; Pae et al., 2008), which has led to the hypothesis that they are part of a spectrum of affective disorders (Hudson, Arnold, Keck, Auchenbach, & Pope, 2004; Hudson et al., 2003; Hudson & Pope, 1996).

Functional somatic disorders or persistent somatic complaints are highly prevalent and are associated with considerable medical, economic and psychosocial costs (Afari & Buchwald, 2003; Annemans, Le Lay, & Taeb, 2009; Annemans et al., 2008; Sicras et al., 2009; Spaeth, 2009). Recent optimism about treatment possibilities of patients with FSD has been tempered by findings that evidence-based treatments such as cognitive behavioural therapy (CBT) and graded exercise treatment (GET) are only modestly effective, particularly in the long term, in a substantial group of patients (Hauser, Bernardy, Arnold, Offenbacher, & Schiltenwolf, 2009; Luyten & Van Houdenhove, 2012; Malouff, Thorsteinsson, Rooke, Bhullar, & Schutte, 2008; NICE, 2007; van Koulil et al., 2007).

This has led to recent calls for broader and more integrative treatment programmes for these patients, fuelled by three related developments that are particularly relevant for psychodynamic approaches. First, there is a growing awareness that many patients with FSD feel misunderstood and stigmatized, and have a history of failed treatments. Indeed, patients with FSD are often considered as 'difficult to treat' (Fischhoff & Wessely, 2003). Unhelpful diagnostic labels, simplistic theories about the causation of these disorders and negative responses from the patients' environment further foster feelings of invalidation and even embitterment (Blom et al., 2012; Kool, Middendorp, Boeije, & Geenen, 2009), leading to often difficult, and at times turbulent, transference–countertransference patterns with health professionals.[1] Second, there is an increasing realization of the role of early adversity and personality/attachment issues more generally in a substantial subsample of patients with FSD (Lumley, 2011; Luyten & Van Houdenhove, 2012). Finally, there is a renewed interest in the role of interpersonal factors in FSD. Indeed, clinicians working with patients with FSD often are struck by these patients' tendency to catastrophize, externalize, rigidly adhere to somatic attributions and to exhibit claiming and clinging behaviour in the process. Likewise, many of these patients are also known for their tendency towards emotional avoidance, distancing and relentless criticism of those who offer help, leading clinicians to experience feelings of irritation, helplessnes and anger (Luyten & Van Houdenhove, 2012; Maunder & Hunter, 2008). As a result, many clinicians become entangled in difficult to resolve transference–counter-transference cycles.

In this paper, we provide an update of our ongoing effort to integrate contemporary findings concerning the nature of FSD within a psychodynamic approach towards the conceptualization and treatment of these disorders rooted in mentalizing and attachment theory. More specifically, we focus on the high

metabolic and interpersonal costs associated with so-called secondary attachment strategies, i.e. stress regulation strategies that involve a hyperactivation or deactivation of the attachment system in response to stress, leading to further stress dysregulation and impairments in mentalization, i.e. the capacity to interpret the self and others in terms of intentional mental states (i.e. feelings, wishes, desires, goals, etc.). These impairments, in turn, give rise to (interpersonal) behaviours that perpetuate symptoms and complaints, leading to a typical pattern of stress dysregulation, high symptomatic distress and interpersonal problems in patients with FSD that also complicate treatment. Hence, these views not only attempt to explain the nature and origins of typical symptoms and complaints of patients with FSD, but also their typical interpersonal problems. Moreover, from a mentalizing perspective, interpersonal problems and symptoms and complaints are seen as closely related, which has important implications for treatment.

A mentalization-based approach to FSD

Stress regulation, attachment and mentalizing

FSD are notably heterogeneous disorders with respect to their causes, course and treatment response (Aslakson, Vollmer-Conna, Reeves, & White, 2009; Dadabhoy, 2006). Research findings in this context converge to suggest that complex interactions among biological and psychosocial factors are involved in both the causation and maintenance of FSD (Heim et al., 2009; Van Houdenhove & Luyten, 2007, 2008; Yunus, 2008). Hence, both theoretical and treatment approaches should take these complex interactions into account.

Based on contemporary research findings, we have argued that FSD probably result from negative vicious cycles involving person–environment interactions (see Figure 1; Luyten & Van Houdenhove, 2012; Van Houdenhove & Luyten, 2009). The model we propose distinguishes between predisposing, precipitating and perpetuating factors. With regard to *predisposing factors*, both biological (e.g. genetic, neurophysiological; Buskila, Sarzi-Puttini, & Ablin, 2007; Rajeevan et al., 2007) and environmental (e.g. early and later adversity; Heim et al., 2009) factors have been identified that may render individuals vulnerable to FSD. Studies furthermore suggest that *precipitating factors* typically involve chronic psychological (e.g. work, relationships) (Aslakson et al., 2009) and/or physiological (e.g. chronic infections or whiplash) stress, which disturb allostasis and lead to a state of so-called allostatic load (McEwen, 2007) mediated by dysfunctions of the hypothalamus–pituitary–adrenal (HPA) axis, the main human stress system (Heim et al., 2009; Tak & Rosmalen, 2010). It is not clear whether precipitating factors usually increase already existing stress and exacerbate impairments in stress regulation, or chronic stress as such may lead to stress dysregulation in otherwise not vulnerable individuals (Tak & Rosmalen, 2010). Typically, HPA axis hyperactivity or hypoactivity as a result of a 'switch' of the HPA axis system from a state of 'overdrive' to 'underdrive' is observed in FSD (Fries, Hesse, Hellhammer, & Hellhammer, 2005; Harvey, Wadsworth, Wessely,

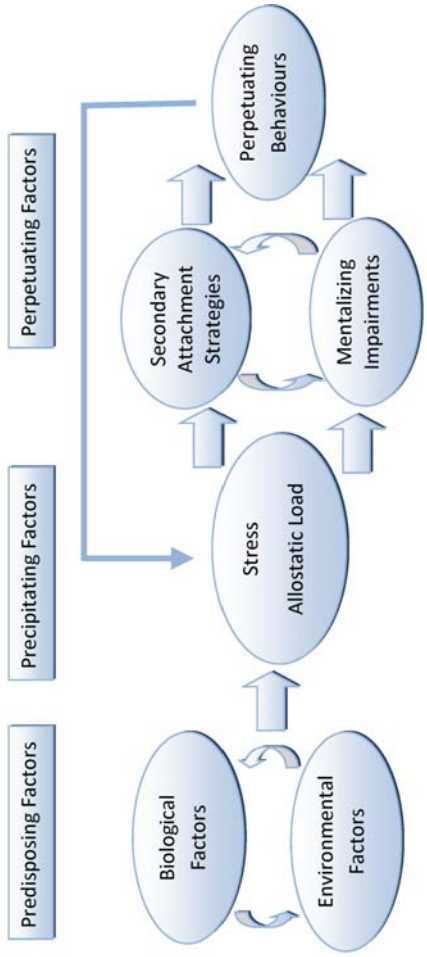

Figure 1. A mentalization-based approach to functional somatic disorders.

& Hotopf, 2008; McEwen, 2007; Miller, Chen, & Zhou, 2007; Van Houdenhove & Egle, 2004; Van Houdenhove & Luyten, 2008, 2010; Van Houdenhove, Van Den Eede, & Luyten, 2009b). Resulting HPA axis dysfunctions are associated with abnormal inflammatory activity that may explain these patients' propensity for infections as well as the typical 'sickness behaviour' that is often observed. Pro-inflammatory cytokines induce feelings of lethargia, increased fatigability, concentration loss, light fever, generalized hyperalgesia and hypersensitivity to physical and mental stressors, as well as a tendency to withdraw from the outside world (Dantzer, O'Connor, Freund, Johnson, & Kelley, 2008; Watkins & Maier, 2005). Moreover, this may lead, together with neuroplastic changes in the spinal cord and brain, and cognitive, emotional and behavioural factors (e.g. pain-related negative affect, catastrophizing and sleep problems), to pain sensitizing (Ablin et al., 2012; Van Houdenhove & Luyten, 2008).

In an attempt to deal with increasing levels of stress, patients start to rely excessively on so-called secondary attachment strategies (i.e. attachment deactivating and attachment hyperactivating strategies) in an attempt to regulate stress, which also give rise to mentalizing impairments, particularly with respect to embodied mentalizing (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). As we will explain in more detail below, secondary attachment strategies and mentalizing impairments further perpetuate symptoms and complaints as they increase stress and thus allostatic load (e.g. catastrophizing, excessive somatic attributions; see Figure 1).

It is essential in this context to realize that attachment problems and mentalizing impairments may already exist before the onset of the disorder. Yet, they may also result from or be exacerbated by an inability to regulate increasing levels of stress and allostatic load, and thus reflect desperate attempts to cope with stress. Moreover, the model assumes, congruent with findings concerning gene–environment interactions and correlations, that vulnerable individuals may in part generate their own stressful environment and/or may be particularly vulnerable for the development of FSD when they are in an environment that impinges upon their vulnerability to stress (Luyten et al., 2011). In what follows, we discuss the different features of the model in more detail.

Attachment and stress regulation throughout the life cycle

Developmental research in both animals and humans has demonstrated a close relationship between stress regulation and attachment, with secure attachment experiences playing a key role in the developing stress system and the development of resilience in the face of adversity (Gunnar & Quevedo, 2007). By contrast, insecure attachment experiences are related to increased vulnerability to stress-related disorders (Gunnar & Quevedo, 2007; Luyten & Van Houdenhove, 2012).

When faced with stress, we seek proximity to attachment figures. In securely attached individuals, such proximity seeking is associated with the effective downregulation of stress. From this perspective, normative stress regulation always involves the effective co-regulation of stress using attachment figures, either real or internalized (Diamond & Aspinwall, 2003; Luyten, Mayes, Fonagy, & van Houdenhove, 2010; Sbarra & Hazan, 2008). Neurobiological findings concerning the role of oxytocin, a neuropeptide, provide further support for these assumptions. Oxytocin not only plays a central role in attachment behaviour, but is also a key stress regulator (Neumann, 2008), and fosters mentalization, i.e. the meta-cognitive capacity to interpret oneself and others in terms of mental states (Fonagy & Luyten, 2009). Specifically, activation of the attachment system is associated with (a) the activation of a mesocorticolimbic, dopaminergic system that plays a key role in the brain's reward system (Insel & Young, 2001), (b) neuroendocrine stress regulation systems (HPA axis and sympathetic nervous system), and (c) 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 cingulate cortex (Bartels & Zeki, 2004; Buchheim et al., 2006; Lemche et al., 2006; Lieberman, 2007; Mayes, 2006; Satpute & Lieberman, 2006). Importantly, the capacity for mentalization, particularly under high levels of stress, has been associated with resilience (Fonagy, Steele, Steele, Higgitt, & Target, 1994), primarily through so-called 'broaden and build' (Fredrickson, 2001) cycles of attachment security, which reinforce feelings of secure attachment, personal agency and affect regulation ('build'), and lead to one being pulled into more adaptive environments ('broaden'; Hauser, Allen, & Golden, 2006; Mikulincer & Shaver, 2007). Hence, the attachment system appears to be an evolutionarily developed system that is closely associated with the reward system in the brain, and thereby reinforces affiliative behaviour that in turns reinforces mentalization and thus the regulation of stress, leading to 'broaden and build' cycles associated with attachment security (Luyten et al., 2010).

By contrast, both animal (Champagne & Curley, 2009; DeVries, Craft, Glasper, Neigh, & Alexander, 2007; Neumann, 2008) and human (Bakermans-Kranenburg, Van Ijzendoorn, Mesman, Alink, & Juffer, 2008; Gunnar & Quevedo, 2007) research has linked insecure attachment experiences, and early adverse experiences more particularly, to greater vulnerability to stress-related disorders, including FSD, mediated by HPA axis dysfunctions (Heim et al., 2009; Kempke et al., 2011; Van Houdenhove & Luyten, 2008). When attachment figures have been insufficiently available, abusing and/or nonresponsive, attachment deactivating or hyperactivating strategies, or a combination of both, as is typically observed in disorganized attachment, become a habitual response to stress (Dozier & Kobak, 1992; Mikulincer & Shaver, 2007; Roisman et al., 2007).

Studies in this context have found not only high levels of early adversity in patients with FSD (particularly high levels of emotional abuse and neglect;

Kempke et al., 2011; Van Houdenhove, Luyten, & Egle, 2009a), but also high levels of insecure attachment histories more generally (Luyten, Houdenhove, Cosyns, & Van den Broeck, 2006; Maunder & Hunter, 2008; Waller & Scheidt, 2006). Yet, as we will discuss in more detail below, secondary attachment strategies and mentalizing impairments may also be a consequence of FSD, which has important treatment implications.

Patients who primarily use *attachment deactivation* strategies tend to regulate stress by denying attachment needs, asserting their own autonomy, independence and strength in an attempt to downregulate stress (Cassidy & Kobak, 1988; Mikulincer & Shaver, 2007). They often present as strong, autonomous and resilient, but actually are very vulnerable (Van Houdenhove & Luyten, 2008). This is consistent with findings of high levels of self-critical perfectionism and related features such as persistence, overactivity and all-or-nothing behaviour in FSD patients (Luyten et al., 2011), features that are closely associated with attachment deactivating strategies (Luyten & Blatt, 2011) and that represent a defensive attempt to affirm the self and soothe negative introjects (Luyten et al., 2011).

Although attachment deactivating strategies may reduce stress in the short run, they are associated with high interpersonal and metabolic costs, which both further increase stress and foster vicious cycles characterized by symptom exacerbation and interpersonal problems. Interpersonally, attachment deactivating strategies lead to increasing isolation and loneliness (Mikulincer & Shaver, 2007). From a neurophysiological perspective, suppression of distress leads to increasing allostatic load, i.e. a hyperactivity of the stress system, which may lead to increased vulnerability to stress and a switch to HPA axis hypoactivity in the long run because of the 'wear and tear' of prolonged stress (Hill-Soderlund et al., 2008; Miller et al., 2007) (Wirtz, Siegrist, Rimmele, & Ehlert, 2008). Under increasing stress, deactivating strategies furthermore tend to fail, leading to a reactivation of feelings of insecurity, heightened reactivation of negative self-representations, increased levels of stress (Mikulincer, Dolev, & Shaver, 2004) and disturbed immune functioning (Gouin et al., 2009).

Patients with FSD also often rely on *attachment hyperactivating strategies*, expressed in anxious efforts to find support and relief, often through demanding, clinging and claiming behaviour (Waller & Scheidt, 2006). As with deactivating strategies, attachment hyperactivating strategies also have considerable interpersonal and metabolic costs. There is an underlying belief that others cannot provide support and care, and therefore this strategy is associated with much anxiety and demanding behaviour that typically leads to resentment in others. Attachment hyperactivating strategies also hinder 'broaden and build cycles', and inhibit internal security of mental exploration. As a result, interventions that rely on insight are often doomed to fail in these patients. Attachment hyperactivating strategies are also associated with high physiological stress and increased HPA axis activity (Diamond, Hicks, & Otter-Henderson, 2008; Gordon et al., 2008), and thus 'allostatic load' (McEwen, 2007).

Mentalizing and stress regulation

Attachment deactivating and hyperactivating strategies furthermore promote mentalizing impairments that lead to further distress and behaviours that perpetuate symptoms and complaints as well as interpersonal problems (see Figure 1). Traditional psychoanalytic approaches have always emphasized impairments in embodied mentalization, i.e. the capacity to reflect on bodily impulses and sensations, in patients with FSD (Maunder & Hunter, 2008; Sifneos, 1977; Taylor, 1987, 2010). In particular, it has been hypothesized that high levels of alexithymia and low levels of emotional awareness (Pedrosa Gil, Scheidt, Hoeger, & Nickel, 2008a; Pedrosa Gil et al., 2008b; Subic-Wrana, Beutel, Knebel, & Lane, 2010) are implicated in the causation of FSD. However, empirical research clearly suggests that only a limited subgroup of patients with FSD (i.e. those with a history of severe attachment disruptions and/or trauma) show clinically elevated levels of alexithymia and lack of emotional awareness (Pedrosa Gil et al., 2008a, b; Waller & Scheidt, 2006).

From a mentalizing perspective, impairments in (embodied) mentalizing in FSD patients are seen as (a) more *specific* rather than general deficits in emotional awareness (i.e. related to specific experiences and symptoms), (b) often the *consequence* rather than the cause of FSD, and (c) related to (interpersonal) situations and experiences that involve *high arousal or stress*. Indeed, FSD symptoms can be considered as an 'attack' 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 fundamental in our relatedness to others. Driver (2005) has aptly described the 'otherness of the illness' in CFS patients, which leads to regressive fears and fantasies. Shahar and colleagues found that patients with a chronic illness tend to experience their illness as an 'internal object' that threatens the individual from within and that needs to be soothed (Schattner, Shahar, & Abu-Shakra, 2008). High levels of arousal and stress that result from symptoms in FSD, in turn, seem to impair and/or further exacerbate already existing impairments in the ability for (embodied) mentalizing. Indeed, studies have amply shown that there is a negative relationship between stress or arousal and the ability for mentalizing (Fonagy & Luyten, 2009; Luyten, Fonagy, Lowyck, & Vermote, 2012). High levels of stress are associated with a relative deactivation of neural circuits involved in controlled mentalizing and the understanding of social causality in general, leading to impairments in affect and stress regulation.

An important clinical guideline follows from these considerations: regardless of whether impairments in (embodied) mentalization are a cause or consequence of FSD, the focus should be on the recovery of this capacity (Luyten & Van Houdenhove, 2012). Specifically, in patients who primarily use attachment deactivating strategies, there often seems to be a sometimes almost total denial of the importance of inner mental states beyond cognitively sophisticated narratives that lack any affective grounding in subjective experiences. Likewise, patients who primarily use attachment hyperactivating strategy, particularly those with

severe trauma, often show marked distortions in (embodied) mentalization. For example, although many patients with CFS are perfectly aware that they should monitor their energy levels, they typically engage in 'boom-and-bust' patterns of activity (Luyten et al., 2011). Similarly, although these patients often attempt to limit their activities and rest, clinicians learn in the course of treatment – often to their astonishment – that patients run more than 20 miles per week or are involved in otherwise very straining activities. When pointed out that this may explain in part their continuing fatigue and contradicts what they have said before about limiting their activity levels, patients are often as astonished as the clinician that they have never thought about this possible connection. These observations again suggest that general levels of emotional awareness are intact in many patients with FSD, but that they suffer more specific impairments in (embodied) mentalizing, such as the inability to link different emotional and bodily states (Subic-Wrana et al., 2010).

This assumption is further supported by empirical findings finding which demonstrate that emotion recognition as such is not impaired in patients with FSD (Oldershaw et al., 2011), but they are less likely to interpret physical sensations as negative emotional states (Dendy, Cooper, & Sharpe, 2001), have negative beliefs about the experience and expression of emotions (Hambrook et al., 2011), lack cognitive confidence in attention and memory, and have a strong need to control thoughts and feelings (Maher-Edwards, Fernie, Murphy, Nikcevic, & Spada, 2012). In particular, patients with FSD often have a strong belief that they should be able to control emotions, that experiencing negative emotions is a sign of weakness, and that others will negatively react to displays of emotions (Rimes & Chalder, 2010). These results are consistent with general findings concerning FSD patients in that they are less interoceptively accurate in symptom-related contexts (Bogaerts et al., 2008, 2010).

These symptom- and context-specific impairments in (embodied) mentalizing are typically associated with the re-emergence of so-called non-mentalizing modes, i.e. modes of subjectivity that antedate the capacity for full mentalizing. These include the psychic equivalence, teleological and pretend modes (Allen, Fonagy, & Bateman, 2008), which seem to play a key role in the perpetuation of symptoms and complaints (see Figure 1).

In a *psychic equivalence mode*, inner and outer reality are equated. What one thinks is real. There is no other interpretation or point of view possible. This is often associated with a lack of desire and/or an inability to explore inner mental states, which hampers treatment efforts. This is particularly the case in patients who 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 also 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 towards acknowledging the role of

psychological factors ('I am exhausted, not angry or depressed'). Psychological pain is experienced as bodily pain, worries feel like a painful weight on one's shoulders; one is literally 'de-pressed', leading to a state of helplessness often characterized by catastrophizing ('I think there is something terribly wrong with me, so there has to be something terribly wrong with me, but nobody can find what is wrong with me, I am beyond help'). Psychic equivalence also negatively influences relationships. Criticism by a colleague, for instance, is felt as an attack on the (bodily) integrity of the self, experienced as physical pain, and threatens 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 didn't feel upset when they fired me, and what I felt doesn't matter, what matters is that I have developed these pains'). In a psychic equivalence mode, one's body starts feeling like an 'alien self-part', a 'machine that is out of control and that doesn't function properly' as one patient put it. 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. Any clinician working with these patients is made to feel their fatigue, pain and often dreadful helplessness, hopelessness, anger and fear. In some patients, this tendency to externalize the alien self is semi-consciously expressed in the desire to have others experience what the patient experiences. As one patient exclaimed: 'It is easy for you to talk like that. I wish that you could feel like I feel just for once. Only then would you know what it is to be me, to be in my shoes. I wish you would come with me, and lie next to me all day, with constant pain, fever, unable to do anything because of the constant "brain fog" in your head, with legs that feel like they each weigh a ton'. In this way, clinicians not only feel what the patient is feeling, but can also begin to imagine the ferocious 'attack' of FSD symptoms on mentalizing capacities, both in the patient and in the therapist.

In a *teleological mode*, there are only observable causes (i.e. observable behaviour reflecting rational, goal-directed behaviour and material causes). For many patients with FSD, only rational, goal-directed behaviours and actions can be effective. This is why so many of these patients are particularly concerned to find 'objective proof' of their illness and only believe in biological causes of their illness. In a teleological mode, clinicians may be drawn into a useless and endless discussion concerning the role of biological versus psychosocial factors.

This may give rise to so-called hypermentalization or 'mentalization on the loose' in *a pretend mode*. Whereas in the psychic equivalence mode, thoughts are felt to be real, in the pretend mode, the relationship between thoughts and feelings and reality is severed. Hence, although clinicians may be impressed with what often seem largely accurate, sensible narrative accounts of the patients' history

and the causes of his or her illness, several features distinguish many of these accounts from genuinely high levels of mentalizing, including (a) the excessive, overly analytical and repetitive length of such narratives, (b) their overly cognitive nature, with little affective grounding, and (c) the inability to switch perspectives (e.g. from the self to others). For instance, one patient, when asked to imagine how others felt about her, responded: 'I'm not interested, we were talking about me, I'm the one who is ill here'.

Needless to say, prementalizing modes further hinder effective stress regulation and increase interpersonal problems. In a psychic equivalence mode, patients start catastrophizing, convinced that they are incurable and/or have some as yet unidentified fatal disease. In a teleological mode, patients seek out alternative (pseudo)biological treatments that offer no relief despite high hopes, or attempt to prove they are not worthless by becoming overactive, which often results in a state of total exhaustion and helplessness. In pretend mode, excessive worry and rumination ensues, leading to anxiety attacks and sleeping problems. Moreover, prementalizing modes further increase interpersonal problems (i.e. increasing feelings that others do not take their illness seriously, that they refuse to offer help, etc.; Van Houdenhove & Luyten, 2008), leading to feelings of invalidation, embitterment and loneliness (Kool et al., 2009).

Dynamic interpersonal therapy for FSD

In the past, it was often believed that psychoanalytic treatment was contraindicated in patients with FSD, primarily because of these patients' so-called alexithymic features. Yet, novel, more structured dynamic treatment approaches have shown considerable therapeutic results in these patients, even with brief treatment formats (Abbass, Kisely, & Kroenke, 2009). Moreover, it is increasingly becoming clear that impairments in mentalizing and 'alexithymic' features are often the result rather than cause of FSD in many patients, or are exacerbated by FSD, and thus that interventions aimed at the recovery of this capacity can be expected to be effective.

The model outlined in this paper suggests that the treatment should focus on restoring the capacity for stress regulation by a focus on (a) typical attachment strategies in response to stress and (b) recovery of the capacity for (embodied) mentalizing (Luyten & Van Houdenhove, 2012). These formulations are in line with the core tenets of dynamic interpersonal therapy (DIT), a brief 16-session psychodynamic treatment that has been recently developed based on common effective elements across dynamic therapies (Lemma, Target, & Fonagy, 2010). DIT is similarly rooted in attachment and mentalizing approaches. Its effectiveness and efficacy are being investigated in depression (Lemma, Target, & Fonagy, 2011) and an adaptation for patients with FSD (DIT-FSD) is currently being piloted. Although a detailed description of DIT-FSD is beyond the scope of this paper, briefly, it involves the following three phases.

Engagement of the patient and formulation of a treatment focus (the IPAF) (sessions 1–4)

This phase first and foremost involves engaging the patient in the treatment. As noted, this is not always easy, because many of these patients resist psychological explanations and report having had bad experiences with previous psychological therapies and health professionals more generally. Empathically acknowledging feelings of invalidation, lack of understanding and embitterment (Kool et al., 2009) is often crucial in this phase; an understanding, supportive empathic stance that recognizes the reality of their complaints and suffering is needed. Within this model the therapist aims to identify early on the core anxieties mobilized by starting the therapy and hence by the attachment to the therapist. These patients typically focus on narratives about being misunderstood. This can be helpfully explored within the therapeutic relationship along with the attachment activation or deactivation strategies that are deployed to manage the anticipated experience of being misunderstood. This shared understanding early on of what might 'go wrong' in the therapeutic relationship can avert premature ending of the therapy or of other kinds of avoidance behaviour that could undermine the therapy.

In this phase it is important to address either implicitly or explicitly unhelpful illness theories by arriving at a common, acceptable illness theory that recognizes the complexity of the disorder (including the role of biological factors) through consensus rather than conflict (Salmon, 2007). It is our belief that many patients with FSD resist a psychological perspective because professionals try to force (bio)psychosocial theories upon them, rather than slowly and appropriately using the patient's own experiences to illustrate how psychological (and other) factors may be involved in the perpetuation (and perhaps also origin) of their complaints, taking well into account the often profound momentary impairments in mentalizing of patients with FSD when discussing these issues.

In DIT-FSD, this is done by jointly arriving at a so-called interpersonal affective focus (IPAF) during the first four sessions. Briefly, the IPAF entails identifying a recurrent cognitive–affective relational or attachment pattern that is associated with the onset and perpetuation of symptoms and complaints underpinned by a particular, often non-conscious, representation of self-in-relation-to-another and a defensive function of this constellation. For example, in patients who primarily use attachment deactivating strategies, the self is often depicted as autonomous, hard-working and well-meaning in relation to others who are depicted as critical, ambivalent and non-understanding. Feelings of anger, aggression, depression and helplessness typically accompany this configuration. Yet, underlying feelings of dependency are typically defended against, leading to vicious cycles characterized by further attempts to gain approval and love of others. In patients with hyperactivating strategies, the IPAF often involves a representation of the self as caring and concerned for others, often expressed in a pattern of compulsive caregiving, while others are depicted as indifferent and uncaring, leading to feelings of helplessness and hopelessness. Underlying

feelings of frustration and aggression are typically defended against, and allostatic load continues to increase because of this defensive interpersonal pattern.

The IPAF thus helps to formulate a clear and explicit focus of the treatment in collaboration with the patient (e.g. addressing perfectionistic, self-sacrificing features that result from an effort to compensate for underlying feelings of worthlessness).

Working through and consolidation (sessions 5–12)

The middle phase of treatment addresses the patient's typical interpersonal attachment patterns using the IPAF as a focus, often with a specific focus on the patient's ability to reflect on the (bodily) self, others and the self-in-relation-to-others. In patients who primarily rely on attachment deactivating strategies, this often involves addressing hyperrationalized, dis-embodied accounts of the body as a 'machine'. These patients often seemingly adopt an implicitly cartesian, dualistic approach to their subjective experience: that is, the patient defensively maintains a mind–body dualism in which the mind is 'the self' and the body is 'other'. Ostensibly overly invested in the body, the unconscious aim is nevertheless to bypass the implications of realities that are rooted in the body.

The focus is on reinvestment of the body with positive affective meanings, and linking interpersonal experiences with bodily experiences and vice versa (e.g. recognizing that feeling 'tense' may actually mean that one is angry at someone). In patients using hyperactivating strategies, this phase often entails finding words to express feelings and to differentiate between different sensations, feelings and experiences. For many patients, this implies changes in terms of a life narrative characterized by incoherence and inconsistence toward a more coherent view of one's own past and present, and the relationship between both. In general, during this phase, a different way of relating to one's bodily self and to others emerges, fostering broaden-and-build cycles associated with more effective regulation of distress. Interventions in this phase include (a) supportive interventions (i.e. reassurance, support, empathy), (b) basic mentalizing interventions aimed at affect recognition, differentiation and amplification as well as more advanced expressive techniques such as interpretation, and addressing the transference relationship when appropriate, and (c) directive techniques (e.g. encouraging the patient to try out new ways of relating).

Empowerment (sessions 13–16)

The process of ending is initiated by sharing a draft 'goodbye' letter written by the therapist. The letter provides a record of the therapeutic work that the patient and therapist then work on together in the final four sessions and that the patient can refer to and reconsider once the therapy is over. Our clinical experience across many DIT cases is that these letters provoke strong affects, sometimes an intensification of the sense of loss about ending, and are often experienced by the

patient as supportive and challenging in equal measure. This typically involves an exploration of the conscious and unconscious meanings of ending the therapy.

The letter squarely focuses the patient on a realistic appraisal of the therapy, what they have gained but also on what it has *not* been possible to achieve, that is, it does not sidestep the reality of disappointment. This is especially pertinent when working with patients with FSD, many of whom will have experienced chronic difficulties and for whom the therapy may mark the beginning of a process of change. The letter may be helpful as a reminder of vulnerabilities in the future and may therefore assist in relapse prevention. This final treatment phase aims at fostering autonomy and resilience after the end of treatment. Directive techniques are particularly central in this stage of the treatment to empower patients as they are used to encourage patients to actively explore and try out new ways of thinking, feeling and relating to others and themselves.

Congruent with the approach taken in this paper, throughout treatment, careful titration of interventions is needed as mentalizing capacities in many patients with FSD may easily dwindle, particularly when salient issues are addressed. It is easy for therapists to overestimate these patients' reflective capacities, particularly when interpersonal issues are addressed. Interventions that strongly rely on insight and reflective capacities are therefore likely to be more harmful than helpful.

Conclusions

There is clearly a resurgence of interest in psychodynamic issues in patients with FSD, fuelled by findings of relatively limited treatment effects of treatments that insufficiently take these issues into account (Lumley, 2011; Luyten et al., 2011). This paper presents an update of our view concerning the nature and treatment of FSD rooted in attachment and mentalizing approaches. A novel brief treatment protocol, DIT-FSD, is presented based on these views. Currently, a pilot study addressing the effectiveness of DIT-FSD is ongoing and a controlled trial is planned. Although it is our belief that DIT-FSD, like many other brief, structured approaches, may be effective for a considerable number of patients with FSD, clearly treatment effects may be more limited in patients with significant biological and/or environmental vulnerability and psychiatric comorbidity. Particularly in these patients, a maintenance DIT phase might be indicated, the aim of which is to reinforce the IPAF as it becomes activated in interpersonally stressful situations. Moreover, for some patients, a more extended and broader treatment approach is needed that involves pain control and possible physical deconditioning, psychiatric issues and sometimes also family and social issues (Van Houdenhove & Luyten, 2007). Further research is therefore needed that addresses the heterogeneity of patients with FSD and that clarifies the role of both biological and psychosocial factors in their causation. Only then will we be in a better position to develop more effective treatments to alleviate the suffering of these patients and those close to them.

Note

1. This is also the reason why we prefer the label of 'functional somatic syndromes' or 'persistent somatic complaints' over other diagnostic labels such as 'psychosomatic', 'somatoform' or 'medically unexplained' syndromes. As we shall see, although the causation of these syndromes remains poorly understood, they are far from 'medically unexplained', as a variety of biological factors have been implicated in their etiology and pathophysiology. Similarly, notions such as 'psychosomatic', 'somatization' and 'somatoform' disorders unduly emphasize the primacy of psychological factors or attributions in the causation of these disorders.

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