How to Use the New DSM-5 Somatic Symptom Disorder Diagnosis in Research and Practice: A Critical Evaluation and a Proposal for Modifications

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somatic symptom disorder, somatoform disorder, pain disorder, hypochondriasis, illness anxiety disorder, somatization, medically unexplained symptoms

Abstract

The fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) changed the term "somatoform disorders" to "somatic symptom and related disorders" and further modified diagnostic labels and criteria. We review evidence for the validity of the new criteria, specifically of somatic symptom disorder (SSD), and present a critical discussion of unsolved and new problems. We also provide an update of mechanisms and interventions that have been empirically evaluated in somatoform disorders. For many mechanisms, it is unclear whether their role can be easily transposed to SSD. Therefore more research is needed on the similarities and differences between medically unexplained and medically explained conditions. To overcome the obvious shortcomings of the current classification, we offer a modification of this DSM-5 section as well as a crossover system to apply these criteria for somatic symptom and related disorders. This proposal allows working with DSM-5 but also continuing successful lines of research with concepts such as hypochondriasis/illness anxiety, chronic pain, and medically unexplained versus medically explained syndromes.

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INTRODUCTION

Somatic symptoms are a common phenomenon and the most frequent reason for doctor visits. Many somatic symptoms are neither a mere correlate of mental disorders nor medically explained in the context of a general medical condition. Up to 80% of the general population reports somatic symptoms during the last seven days, and about one-fifth of the general population suffers from serious, disabling, and frequently chronic somatic complaints (Hiller et al. 2006). Medically unexplained somatic symptoms produce health care burdens that are comparable to anxiety and depressive disorders (Konnopka et al. 2012). Because of the tremendous relevance for health care systems, a reliable and valid classification for somatoform symptoms is crucial as the basis for improved detection, adequate treatment, and relevant research efforts. However, the classification for somatoform symptoms in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) had been frequently criticized, and the fifth edition of the DSM (DSM-5) suggests a new classification approach, summarized as "Somatic Symptom and Related Disorders." Although the introduction of DSM-5 was celebrated with tremendous enthusiasm from its proponents (Kupfer et al. 2013), others expressed criticisms about missed opportunities and uncontrolled risks (Frances & Widiger 2012). Will DSM-5 offer an improved platform for diagnoses, management, and research in the field of somatic complaints? And what should be done with the research results and traditions that were based on former classification approaches, such as mechanisms of somatization, medically unexplained pain conditions, or treatment approaches for hypochondriasis? Can these results be generalized to the new diagnoses, and can we save some successful traditional research lines using crossovers to the new classification approach? In the following article, we discuss these questions and offer specifications and crossovers that preserve the research evidence from past classification systems while allowing the use of the new DSM-5.

REPLACING SOMATOFORM DISORDERS WITH SOMATIC SYMPTOM DISORDER: THE NEWER, THE BETTER?

The Problems with DSM-IV Somatoform Disorders

SSD: somatic symptom disorder

It was generally agreed by researchers that the concept of somatoform disorders introduced in DSM-III and modified for DSM-IV needed substantial revision. Somatic symptoms that are not attributable to biomedical conditions are a substantial problem. It is estimated that at least every fifth visit to medical doctors is caused by this type of complaint (Steinbrecher et al. 2011). An estimate based on data of 514 million European citizens ranks somatoform disorders third on the list of the most prevalent mental disorders after anxiety disorders and depression (Olesen et al. 2012). In contrast to these large prevalence rates, the prototype of somatoform disorders, somatization disorder, is over-restrictively defined. Moreover, Anglo-American countries, mainly the United States and the United Kingdom, rarely use the somatoform disorders diagnoses. Statistics based on 1.2 million inhabitants of Virginia who sought medical help and received a diagnosis revealed that only 0.02% received a diagnosis from the category of somatoform disorders (Levenson 2011), and a similarly low number received the diagnosis "psychological factors affecting medical conditions." This finding is in sharp contrast to the fact that nearly every American family doctor reports that these patients are seen frequently in primary care (Dimsdale et al. 2011). Many researchers were concerned that the terms somatoform and somatization overemphasized mind-body dualism. Many clinicians interpreted these terms as indicating that "everything is just in the mind." Notably, DSM-III could not be blamed for this interpretation, as it introduced the title just as a descriptive term in the sense of "somatoform = indicating a somatic condition, albeit biomedical disorders do not explain the symptoms."

Many physicians in Anglo-American countries found it unacceptable for their patients to receive these diagnoses (Dimsdale et al. 2011) and were concerned that patients disliked the terms, even though physicians in other countries used these diagnoses without any patients protesting. One deeper problem might be that guidelines of how to explain these types of diagnoses to patients were missing. Underdiagnosis is also evident for the related functional syndromes: General practitioners did not identify 90% of patients with chronic fatigue syndrome, 77% of patients with fibromyalgia, and 69% of patients with irritable bowel syndrome (Warren & Clauw 2012). Patients were identified using self-reports of physicians' diagnoses. Finally, bias in health care systems and legal regulations can further amplify the reluctance to use a diagnosis such as somatoform disorder. Many health care systems tolerate underdiagnosis and misdiagnosis of mental disorders, even though it is unacceptable to miss physical diagnoses, even those that are benign.

The DSM-5 Somatic Symptoms Disorders Work Group tried to address these issues when developing a proposal for a new diagnostic group. They created a new category name, "Somatic Symptom and Related Disorders," introduced its prototype, the somatic symptom disorder (SSD), and modified the criteria substantially (Am. Psychiatr. Assoc. 2013).

Compared to somatoform disorders and especially its prototype somatization disorder, Somatic Symptom and Related Disorders brings substantial changes. Most radical is the abolition of the distinction between medically explained and medically unexplained somatic complaints. As soon as any somatic complaint qualifies as being distressing, criterion A of SSD ("One or more somatic symptoms . . ."; Am. Psychiatr. Assoc. 2013, p. 311) is fulfilled, even if the complaint is only a single symptom and even if it is clearly medically explained.

One critique of somatoform disorders was the lack of psychological criteria that qualified the classification of somatic symptoms as "mental" disorders, albeit the symptoms are physical by nature (Rief & Isaac 2007). A common characteristic of mental disorders according to DSM is the inclusion of psychological features, e.g., body dissatisfaction in eating disorders and fear of

fear in panic disorder. In contrast, somatoform disorders did not have any positive psychological criteria but were based only on somatic symptoms and the "negative" criterion that they should not be (fully) medically explained. The DSM-5 diagnosis of SSD now requires as a B criterion that "excessive thoughts, feelings, or behaviors are related to the somatic symptoms or associated health concerns" (Am. Psychiatr. Assoc. 2013, p. 311) and suggests that at least one of three psychological symptoms needs to be present to fulfill the criterion. Indeed, the only justification for classifying somatic complaints as mental disorders is the inclusion of "positive" psychological criteria (Voigt et al. 2010).

The corresponding DSM-5 Somatic Symptoms Disorders work group obviously attempted to limit the number of diagnoses in this category and to lump as many diagnoses as possible in one single diagnosis (Dimsdale et al. 2013). Therefore, the new diagnosis of SSD is designed not only to cover patients with somatization disorder but also patients with chronic pain conditions, most patients with hypochondriasis, and many patients with medical conditions that are accompanied by psychological features. Although the general attempt to reduce the number of diagnoses in DSM is commended, the lumping of these heterogeneous conditions must be critically discussed. The specificity of diagnoses should follow the same rules for all DSM diagnoses, and therefore the question arises whether SSDs and hypochondriasis should be lumped as suggested, while major depressive disorder, persistent depressive disorder, and other specified depressive disorder are considered different diagnoses. In fact, the DSM-5 Somatic Symptoms Disorders Work Group was not able to transpose this lumping process and had to acknowledge that significant groups of patients were not covered by the diagnosis of SSD, albeit they should be. Therefore, additional diagnoses were added (e.g., illness anxiety disorder). However, this resulted in serious problems. If patients suffer mainly from illness anxiety, they are now disseminated over two diagnoses: If they have an additional somatic complaint, the SSD diagnosis will be given; if they do not have an additional somatic complaint, the diagnosis of illness anxiety disorder has to be used. This example underlines the need to discuss these DSM-5 criteria more critically.

How Valid Are the New Classification Criteria?

The first question is whether a unique diagnosis for these syndromes is justified. Former concepts considered somatic symptoms as a subsyndrome of other disorders (e.g., somatized depression). This has been underlined by a substantial overlap between somatization, depression, and anxiety. However, each of these syndromes has its own characteristics, and somatization is relevant as a comorbid condition with other mental disorders as well as a unique syndrome (Barsky et al. 2005, Löwe et al. 2008). Neuroimaging results and psychobiological properties further underline the validity of a diagnosis for these somatic syndromes (Browning et al. 2011, Rief et al. 2010a). Of note, a statistical clustering of psychopathological features resulted in a best-fitting model that had five factors, and somatoform disorders was one of them (Kotov et al. 2011). Therefore, the DSM-5 had good reasons to continue proposing diagnoses for this field.

The first studies evaluating SSD criteria confirmed aspects of construct and descriptive validity as well as one-year stability of diagnoses (Rief et al. 2011b, Voigt et al. 2012, Wollburg et al. 2013). Moreover, the initial field trials found good interrater agreement for the pilot version "complex somatic symptom disorder" (Regier et al. 2013). However, these general aspects do not indicate that the current proposal is the best solution. Therefore, we discuss the validity of the A and B criteria of SSD in more detail below.

The A criterion of SSD. The A criterion allows the new diagnosis SSD to be considered as soon as a single somatic symptom is present. The justification for this criterion is the fact that

some people can suffer tremendously just from a single symptom whereas others might cope quite well with multiple symptoms. Moreover, the distinction between medically explained symptoms and medically unexplained symptoms (MUS) has been omitted, which seems like an adventurous reformulation. The major reason for this revision is the low interrater reliability and validity of physicians' judgments on whether or not somatic symptoms are medically explained (Fink et al. 2005). An analysis of MUS versus general medical symptoms did not reveal that the former would result in higher specificity, and therefore the authors suggested using the simpler criterion "physical symptoms" without necessarily identifying the cause of it (Tomenson et al. 2010, 2013). Moreover, many symptoms transform back and forth between being considered medically explained or unexplained (Klaus et al. 2013). In many cases, the physicians' personality rather than the clinical picture seems to affect whether symptoms are deemed to be based on a biomedical condition. Some physicians think of symptoms like back pain as almost always medically caused, whereas others consider them mainly psychosomatic. Cultural influences also account for this low interrater reliability of causality ratings.

MUS: medically unexplained symptoms

However, the question arises whether the omission of the criterion "medically unexplained" is the best solution for this problem. In the case examples suggested in **Table 1**, all three patients would receive the diagnosis "Somatic Symptom Disorder," yet it seems unclear whether the mechanisms that are involved in the psychological problems are always the same and whether the patients need the same treatments (which would be reasons to justify the use of a single diagnosis). The lumping of the different conditions shown in **Table 1** would be justified only if it had been shown that the mechanisms were the same. However, we are not aware of studies that sufficiently demonstrate this. Therefore, despite the newly proposed SSD, it might be useful to consider this distinction in research and to use a modified descriptor for "medically unexplained" that was typically used in former classification systems: The symptoms are not better explained by a general medical condition.

Table 1 Is this the same clinical problem? Three case examples of somatic symptom disorders

Case 1: patient diagnosed with the former (DSM-IV) somatization disorder
The 43-year-old male patient reported a long history of multiple somatic complaints, starting with noncardiac chest pain and headache when he was in school, which developed to a broad spectrum of symptoms all over the body (back pain, bloating, abdominal discomfort, joint pains, dizziness, fatigue, general weakness). During the past 20 years, the patient received regular medical examinations. On average, he attended a physician's office every two weeks, and he was also admitted to hospitals at least once or twice a year for thorough medical examinations. Apart from minor descriptive diagnoses without any pathological significance, no biomedical explanation for the symptoms was found.
Case 2: cancer patient
The female patient developed breast cancer when she was 44. She received the typical medical treatments (surgery, radiation, chemotherapy) and started aromatase inhibitors treatment subsequently. Now, three years later, she suffers from somatic symptoms that could be attributed to side effects of the medical treatment (arthralgias) as well as other symptoms, such as headache. Her major problem is that she is concerned about the recurrence risk of breast cancer. The fears of recurrence are always on her mind.
Case 3: diabetes patient
The 64-year-old man received the diagnosis of diabetes several years ago. Although he is aware that diabetes is not life threatening as long as he adheres to the medical regime, his attention is highly focused on his body because he is unsure whether symptoms are caused by hypoglycemia. He suffers from a multitude of somatic complaints such as joint pain, back pain, chest pain, and dizziness, and he is concerned about developing retinopathy and finally blindness. The doctor told him that some of his symptoms could indicate the development of diabetic neuropathy, yet symptoms and the physical conditions must be observed further before drawing clear conclusions about the diagnosis.

Despite good reasons to use this diagnosis for patients with a single somatic complaint, we should be aware that symptom counts still provide relevant information. First, the presence of multiple symptoms reduces the range of medical conditions that could explain the broad variety of somatic complaints. More importantly, the more symptoms patients report, the more likely it is that symptoms will persist and will be associated with serious disability and reductions of quality of life (Chou & Shekelle 2010, Rief & Rojas 2007). Vice versa, if patients report a single symptom (and especially if it is not chronic), the likelihood is substantial that this symptom will vanish whether medical interventions occur or not. Several studies, including latent class analyses, have confirmed the distinction between patients with one or few symptoms and patients with many symptoms (e.g., Rosmalen et al. 2011). Therefore, the differentiation between single- and polysymptomatic syndromes has some validity. Even as a comorbid condition (e.g., in depression), the physical symptom count is a significant predictor of outcome (Huijbregts et al. 2013). The same line of research has confirmed that a differentiation into symptom clusters, such as pain syndromes, gastrointestinal syndromes, or cardiovascular syndromes, might sometimes be found in large data sets but does not provide any added value in classification approaches (Fink & Schröder 2010, Rosmalen et al. 2011). Therefore, the omission of symptom clusters seems to be warranted, although the omission of a unique diagnosis for patients with multiple somatic symptoms is less justified.

The B criterion of SSD. The introduction of psychological and behavioral features is supported by study results showing that it is not the somatic symptoms per se that result in increased health care needs, but rather how patients interpret the symptoms and their illness behaviors (Rief et al. 2010b). Psychological and behavioral features are significant determinants of disability in somatoform disorders (Wollburg et al. 2013). Although some experts suggest other ways to identify this patient group (Fink & Schröder 2010), the inclusion of psychological and behavioral criteria makes sense in the context of a psychiatric classification system. The B criterion of SSD requires that at least one of three psychological criteria should be manifested.

Health anxiety. The first B criterion, health anxiety, seems reasonable, especially if the former diagnosis of hypochondriasis is to be covered in this context. Even for many patients with somatization disorder, health anxieties play a significant role (Rief et al. 2010b, 2011b), and greater health anxiety predicts persistence of somatoform disorders (e.g., McKenzie et al. 2010). High prevalence rates of significant health anxiety in patients seeking care in various medical specialty settings (e.g., neurology, respiratory medicine, gastroenterology, cardiology; mean rate 19.8% in $N = 28{,}991$) suggests that abnormal health anxiety is a considerable problem in a broad range of somatic conditions (Tyrer et al. 2011). Even after controlling for somatic symptom count, health anxiety has an incremental effect on predicting health care use (Tomenson et al. 2012), although there are also conflicting results (Barrett et al. 2012). Another population-based survey (German National Health Interview and Examination Survey–Mental Health Supplement; $N = 4{,}181$) showed that subjects with unrealistic illness worries as compared to subjects without illness worries had higher comorbidities with medical conditions and with mental disorders, higher illness behaviors, and lower health-related quality of life (Martin & Jacobi 2006). The impact of health anxiety in somatic conditions, including pain disorder, and on disability, prognosis, and treatment outcome has been shown (e.g., Aggarwal et al. 2010, Jensen et al. 2010, Kehler & Hadjistavropoulos 2009; for review, see Hadjistavropoulos & Hadjistavropoulos 2003). Despite the overall relevance of health anxiety, it is not necessary for chronic somatic complaints, and many patients suffer from somatic complaints without worrying about undetected serious medical conditions.

"Disproportionate and persistent concerns about the medical seriousness of one's symptoms." The second B criterion, "disproportionate and persistent concerns about the medical seriousness of one's symptoms" (Am. Psychiatr. Assoc. 2013, p. 311), seems to have substantial overlap with the first B criterion, health-related anxiety, and the question arises whether this criterion brings incremental information. Moreover, this criterion is in line with the viewpoint held by many clinicians but is less supported by empirical data. There are patients with a very rigid assumption about a somatic cause of their complaints, and some of them fear serious, maybe even life-threatening, biomedical conditions. Somatic illness attribution was found to identify those patients with somatic symptoms who report increased disability (Rief et al. 2010b), and a few studies have shown that the tendency for somatic causal attributions predicted disability and worse prognosis (Bailer et al. 2008, Henningsen et al. 2005). Many overviews and textbooks describe somatizing patients as patients who do not consider psychological causes for their symptoms. However, in reality many patients with somatoform syndromes and/or SSD are able to consider psychological etiologies for the complaints (for review, see Martin & Rief 2011), and the frequency of psychological symptom attributions is very similar to somatic causality assumptions (Hiller et al. 2010). Surprisingly, some studies have found that more psychological attributions can indicate more disabling conditions in somatoform disorders (Rief et al. 2004). Therefore, both somatic and psychological illness attributions can be associated with increased disability. To summarize this point, although some evidence favors introducing this criterion, several aspects lack clarity. More precise guidelines are needed for the application of this criterion to medically explained symptoms.

"Excessive time and energy devoted to these symptoms or health concerns." Finally, the third B criterion, "excessive time and energy devoted to these symptoms or health concerns," (Am. Psychiatr. Assoc. 2013, p. 311) has some obvious face validity, and in one of our studies, "ruminations about bodily complaints" was found to indicate increased health care needs in somatizing patients (Rief et al. 2010b). But again, it remains unclear whether excessive time itself is a good criterion or whether a criterion such as "inappropriate excessive time" that is devoted to these concerns would be more valid. If a patient has a serious risk of recurrence of life-threatening conditions (see case example 2 in **Table 1**), a substantial amount of time must be devoted to health care issues. Therefore, even if the health concerns and the time devoted to them can be fully explained by the biomedical condition, DSM-5 suggests adding a diagnosis of SSD.

The B criterion of SSD in medically explained conditions. The introduction of "positive" psychological criteria that define this diagnosis of SSD in DSM-5 is well supported by various study results. It moves the focus from a mere description of symptoms to potential mechanisms. However, whereas these criteria can be relevant and valid for MUS, it is unclear what their role is in medically explained symptoms. Although a substantial amount of health-related anxiety and time and energy devoted to health concerns can be considered normal or even necessary in patients who suffer a cancer diagnosis, the same level of these features would be considered inappropriate in patients with MUS, who may suffer from these symptoms for decades without any new findings. Similarly, the interpretation of dizziness as a potential sign of recurrence after suffering from a brain tumor could be well justified, whereas the same interpretation would be considered as overcatastrophizing in case example 1 (**Table 1**) (somatization disorder). The DSM-5 Somatic Symptoms Disorders Work Group decided that the suffering and potentially negative consequences of these mechanisms are the same whether they are appropriate or inappropriate, but this assumption definitely needs further investigation.

The specifiers of somatic symptom disorder. DSM-5 offers three specifiers for the diagnosis of SSD that are determined by the patient's predominant clinical syndromes. The first specifier is "with predominant pain." Specifiers for illness anxiety and the former somatization disorder were under discussion in DSM-5 drafts but did not make it into the final version. When developing the concepts of SSD, it became obvious that about 20% of patients with hypochondriasis would not fulfill the criteria of SSD because of a lack of somatic symptoms despite serious illness anxieties. Therefore an additional diagnosis of "illness anxiety disorder" has been introduced under the same category.

The second specifier of SSD is "persistent," which is defined as severe symptoms, marked impairment, and long duration (more than six months). The duration specification is surprising considering the fact that the C criterion already defines a similar time frame ["the state of being symptomatic is persistent (typically more than 6 months)"] (Am. Psychiatr. Assoc. 2013, p. 311). Finally, the third specifier allows a classification of severity, which is mainly based on the number of psychological symptoms (B criterion). The validity of these specifiers remains unclear.

Shortcomings of the New SSD Diagnosis in DSM-5

As previously mentioned, the concept of somatoform disorders has several problems, and the question arises whether the new formulation of SSDs is able to solve them. The first question is whether this new concept and diagnosis will be used more frequently, especially in those countries that underdiagnose somatoform disorders (i.e., the United States and the United Kingdom). Those experts who have criticized the risk of amplifying mind-body dualism with the old concept of somatoform disorders might be more open to use the new diagnosis. Additionally, those who have been reluctant to use the terms "somatoform" or "somatization" may be in favor of using another term. However, every new term and every new concept bears the risk of being misinterpreted the same way the old one was, and therefore introducing a new term itself does not solve any problems. A successful introduction heavily depends on both the people who engage in disseminating the new diagnosis and concepts and the scientists who use it. Moreover, other reasons for the underdiagnosis of these conditions in many patients must be addressed. The fact that primary care doctors did not use these diagnoses can be only poorly explained by conceptual issues and labeling. A hypothesis is that many health care providers have problems in explaining these types of diagnoses to their patients, and more seriously, they have a greater fear that legal issues may result from the incorrect use of this diagnosis as compared to other diagnoses. If these reasons are relevant, then the solution is not to use a new diagnostic term, but rather to provide training packages so that health care providers can explain these diagnoses to patients in a way that patients can accept. Moreover, stakeholders of health care systems must be convinced that misdiagnosing in the case of mental disorders is as serious as misdiagnosing in general.

The question arises whether the new label somatic symptom disorder is the best option for renaming somatoform disorders (Creed et al. 2010). It is supposed to be merely descriptive (which also was supposed to be the case for "somatoform" when it was introduced in 1980 in DSM-III). On the other hand, this combination of terms is poorly understood by traditional psychopathologists, and the new term still includes a substantial risk of discrimination. The psychopathological purists might argue that classification is a process on three levels: First, the diagnostician collects symptoms (level 1), then he/she combines them to syndromes (level 2), and after the consideration of further criteria, he/she decides whether the criteria for the disorders are fulfilled (level 3). Combining symptoms with disorders in one term confuses the different levels of classification. Although this can be considered to be more theoretical, the practical implications might be more serious. How would patients feel if they had just a symptom disorder instead of having the disorder itself?

How would patients feel if they had anxiety symptom disorder instead of anxiety disorders, or depressive symptom disorders instead of depression? Some patients might feel like they do not have a correct disorder, but just a symptom disorder. An international consensus group discussed which terms should replace the term "somatoform" (Creed et al. 2010), and they also addressed the international transportability of a new term; these aspects did not seem to play a role when suggesting use of the term somatic symptom disorder.

The A criterion of SSD allows a broad variety of clinical conditions to be diagnosed in this category, and the question arises as to whether this is not overliberal and overinclusive. The step from the former somatization disorder to the new SSD is huge. Several studies were able to show that up to 80% of the general population reports some somatic symptoms during the last seven days (Hiller et al. 2006), and therefore the A criterion is easily fulfilled and can hardly be rejected in critical cases. The well-founded validity of the distinction between single somatic symptoms versus polysymptomatic syndromes (Lacourt et al. 2013, White 2013) also is not addressed in DSM-5. The lack of an adequate symptom threshold in SSD was therefore considered to be a threat to clinical utility (Voigt et al. 2010).

Moreover, if the distinction between medically explained and medically unexplained symptoms is so difficult to draw, the omission of this criterion is only one solution. Another option could have been to try to improve this criterion. Other diagnoses of the DSM usually use wordings such as "not better explained by." Without such a specification, some people fear overdiagnosing patients with medical conditions as psychiatric cases, to subject patients to stigma, and to skew patients' self-perception (Frances 2013b). In the same paper, Frances reported false positive rates of 7% for SSD among healthy people of the general population, which must be considered substantial. A criterion such as "If a diagnosed medical condition is present, the thoughts, feelings, and behaviors are grossly in excess of what would be expected, given the nature of the medical condition" (Frances 2013a) could also be an alternative to the abolishment of the medically explained-unexplained distinction. As long as it is unclear whether the mechanisms and treatment consequences are really the same for patients with medically explained versus medically unexplained somatic symptoms, an additional specifier for the diagnosis indicating this distinction might be helpful, at least for research projects. If the similarity of mechanisms and treatment consequences is confirmed, this distinction can be eliminated, but until scientific proof is provided we should try to preserve our knowledge, which is mainly based on MUS and not on psychological factors in medically explained syndromes.

One must also question whether the suggested psychological symptoms (the B criterion of SSD) are the most valid, sensitive, and specific empirically founded features. They seem to fit to patients who overlap somatization and hypochondriasis quite well, but the question arises whether the three B criteria are highly correlated. The presented psychological symptoms are mainly cognitive-affective. Unfortunately, behavioral features were not explicitly included in the B criteria list, although in several fields the evidence for diagnostic validity of behavioral characteristics is higher than for psychological characteristics. For chronic pain patients, it was shown that fear avoidance behavior (avoidance of any situations and movements that are expected to induce symptoms) is among the best predictors for translating acute to chronic pain (Chou & Shekelle 2010). For patients with multiple somatic syndromes, avoiding situations that challenge the body is also one of the most powerful variables distinguishing highly disabled patients from those with low health care needs, even if both groups have a similar number of somatic symptoms (Rief et al. 2010b). Behavioral avoidance is also common in some of the functional somatic syndromes, for example in chronic fatigue syndrome, and is related to various clinical characteristics and disability (Cella et al. 2013, Nijs et al. 2013). Excessive reassurance-seeking behavior (presented to physicians as well as to significant others, such as partners), checking behavior (e.g., self-inspection of body functions or for signs of disease), and avoidance of situations or activities that provoke illness anxiety are major

features of hypochondriasis, and they are also often behavioral symptoms in somatization disorder. These behavioral factors are considered crucial for the maintenance of the disorders, but none of these features entered the symptom list of SSD B criteria. Moreover, cognitive symptoms other than "disproportionate and persistent thoughts about the seriousness of one's symptoms" (the first B criterion; Am. Psychiatr. Assoc. 2013, p. 311) are not listed, whereas ruminations about physical complaints, self-concept of bodily weakness, and subjective low symptom tolerance have previously been suggested as cognitive variants (Rief et al. 2010b). Finally, the only affective characteristic required for the B criterion is health anxiety; other affective symptoms such as negative affectivity, desperation, and demoralization related to somatic symptoms are not included.

A clear critique on the DSM-5 formulation concerns splitting hypochondriasis into two diagnoses. The clinical encounter with a patient suffering from full-blown hypochondriasis is very different from patients with somatization disorder or patients with chronic pain conditions. The higher relevance of anxiety processes and the more fluctuating course with ups and downs into crises indicate that hypochondriasis has a strong overlap not only with somatization but also with anxiety disorders. Powerful interventions have been developed for hypochondriasis; in contrast, psychological interventions for somatization disorders and pain conditions suffer from modest effect sizes (Clark et al. 1998, Kleinstäuber et al. 2011, Sørensen et al. 2010). These differences between hypochondriasis and somatization underline the validity of a unique illness anxiety diagnosis for patients with hypochondriasis. The progress in our understanding and treatment options for hypochondriasis should not be hindered by the splitting of classification criteria suggested by DSM-5.

The consideration of pain disorders as just a specifier of SSD must also be questioned. Reasons in favor of this position are the significant overlap of pain symptoms with other somatic symptoms in many patients (Fink et al. 2007, Rief et al. 2001). However, many patients report only pain symptoms without other somatic complaints, and pain conditions are by far the most frequent symptoms in this context. The prevalence rates for pain conditions are estimated to be above 40% of the general population (Raspe et al. 2008). Even if combined with other somatic symptoms, pain symptoms seem to be most stable and chronic, and they predict the most serious disability (Klaus et al. 2013). DSM-5 reduces their tremendous societal impact to a specifier of another diagnosis.

Finally, DSM-5 also includes factitious disorder under the category of somatic symptom and related disorders, which is definitely provoking. The initial goal of the DSM-5 Somatic Symptoms Disorders Work Group was to reduce stigmatization and mind-body dualism of the category of somatoform disorders, but this goal seems to be reversed when mentioning SSD in one group with factitious disorders. We are not aware of any data showing that patients with functional syndromes or somatoform disorders have a higher tendency to show factitious disorders. Is feigning really more frequent in somatic syndromes than in OCD, posttraumatic stress syndrome, depression, or panic disorder? If the inclusion of factitious disorder in this category is not supported by strong evidence, then it is a provocation against every single patient who fears that doctors do not take his complaints seriously, an experience that is unfortunately very common in clinical practice (Dimsdale 2011).

WHAT ARE THE MECHANISMS INVOLVED IN SOMATIC SYMPTOM DISORDER?

Most research on mechanisms in the SSD field has been based on the concept of somatoform disorders, hypochondriasis, and pain disorders. The question arises whether these findings can be transposed to the new concept of SSD. **Figure 1** provides a typical example of current models that have been developed for somatoform disorders (Kirmayer & Taillefer 1997). In the following

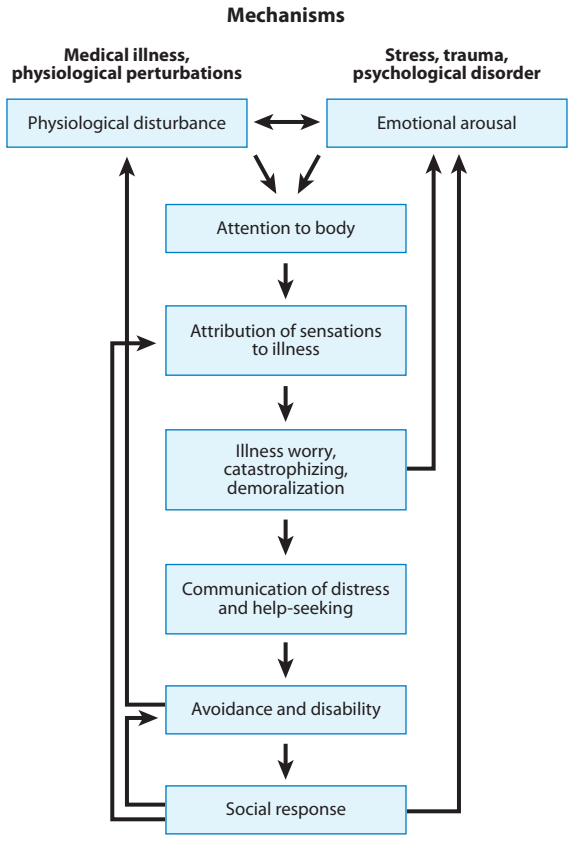

Figure 1

A model of somatoform disorders (Kirmayer & Taillefer 1997).

sections, we introduce several suggested mechanisms and provide an update on empirical evidence for them in reviews of recently published studies. On the basis of previous reviews and our update, we discuss whether these mechanisms might also be suitable for the understanding of SSD. (For a comprehensive overview of previous studies and detailed descriptions of the models for somatoform disorders, see Rief & Broadbent 2007, Witthöft & Hiller 2010.) Although substantial overlap exists between the model presented in **Figure 1** and other models on somatoform disorders, the other models add the role of attention systems and perceptual decisions (Brown 2004) or attachment theory and doctor-patient interactions (Henningsen et al. 2007). In a "perceptual filter model," we propose processes that offer a link to psychobiological mechanisms and to bottom-up as well as top-down regulation processes of symptom development and symptom control (Rief & Barsky 2005). The core features of most models remain the selective attention process, the amplified perception of bodily sensations, and the perpetuation of this process through catastrophizing interpretations and inadequate illness behavior.

Cognitive-Perceptual Processes

Perception of physical sensations. Introduced as somatosensory amplification (Barsky & Wyshak 1990), perceptual and interpretative processes are in the center of mechanisms that contribute to somatoform disorders. According to the concept of somatosensory amplification,

hypochondriasis in particular, but also somatization in a broader sense, results from the amplifying perception of benign sensations and their misattribution to serious diseases. The tendency to experience a somatic sensation as intense, noxious, and disturbing leads to focusing the attention to physical signals and results again in increased awareness of bodily changes and perception of bodily symptoms. In this way a vicious circle of symptom development and maintenance is hypothesized. The empirical evidence for this assumption was mainly based on cross-sectional data and self-rating scales (e.g., using the Somatosensory Amplification Scale; Barsky et al. 1990), showing that the relationship between health anxiety and self-report measures of somatosensory amplification is strong (see meta-analysis of Marcus et al. 2007). That means individuals with high degrees of health anxiety (particularly individuals with hypochondriasis) believe that they are especially aware of and sensitive to their bodily processes. However, it remains unclear whether they are in fact more sensitive and accurate interoceptors. On the basis of their systematic review, Marcus et al. (2007) concluded that there is little evidence of a positive relationship between health anxiety and the ability to perceive autonomic processes or tactile stimuli accurately. Comparably, findings are inconsistent regarding the perception of body signals in somatoform disorders. Although an earlier study demonstrated more precise perceptions of muscle tension (Scholz et al. 2001), two recent studies were unable to show more accurate interoceptive awareness in somatoform disorders and in noncardiac chest pain using two well-established heartbeat perception paradigms (Schaefer et al. 2012; S. Schroeder, A. Gerlach, S. Achenbach, & A. Martin, manuscript submitted). However, both studies revealed that the severity of the disorder may be associated with lower (rather than higher) interoceptive awareness. These findings are in line with two recent models of somatoform disorder that stress the importance of top-down factors influencing the perception of somatic sensations (Rief & Barsky 2005) and more specifically of biased schema-guided processing of interoceptive information (Brown 2004). In recent years, a series of novel experiments has provided accumulating evidence that the experience of MUS is indeed characterized by distorted perceptual processing of (aversive) somatic sensations. Some of these experiments varied sensory input and/or used perceptual illusion techniques such as the somatic signal detection task or the rubber hand illusion (Bogaerts et al. 2010b, 2012; Brown et al. 2010, 2012; Katzer et al. 2011, 2012; Miles et al. 2011).

Bogaerts and collegues (2010b) assessed the experience of dyspnea in relation to respiratory changes due to a laboratory-induced dyspnea in MUS. The results showed that perceived symptoms were less strongly related to relevant physiological parameters in MUS patients than in healthy persons, specifically when afferent physiological input was relatively weak. This suggests a stronger role for top-down psychological processes in symptom perception in patients with MUS. Another experiment (Bogaerts et al. 2012) applied the recency effect paradigm, in which unpleasant somatic experiences are remembered as less aversive when they end gradually rather than abruptly (i.e., the peak-end effect). The authors manipulated the end of laboratory-induced dyspnea in patients with medically unexplained dyspnea. Patients with medically unexplained dyspnea did not show this peak-end effect but rather a deficit recovery of dyspnea that could not be explained by altered respiratory physiology. This experiment very elegantly demonstrates the critical role of distorted perceptual-cognitive processing of aversive somatic sensations in MUS.

Other studies adapted the somatic signal detection task (SSDT) as an experimental paradigm to assess touch illusions as a laboratory analog of MUS. During the SSDT, participants indicate whether they feel vibrations delivered to their fingertip at perceptual threshold. In the experiment, the presence of the vibration and of an additional light signal is varied (Lloyd et al. 2008). Some participants report the presence of the vibration even when no tactile stimulation has been delivered (so-called false alarms or illusory touch experiences), a response pattern that was associated with somatoform dissociation (Brown et al. 2010) and with physical symptom reporting

(Brown et al. 2012). The latter study even demonstrated that no difference existed in the false alarm rate between patients with medically explained and medically unexplained symptoms, suggesting that the tendency to experience somatosensory distortion is linked to physical symptom reporting (Brown et al. 2012) and therefore may also be relevant in SSD. Another research group did not show a higher false alarm rate in patients with somatoform disorder compared to healthy controls, but it did identify a more liberal response bias (in the first half of the light-absent condition of the SSDT) in patients with somatoform disorder (Katzer et al. 2012). Within the somatoform disorder group, the report of somatoform (especially pseudoneurological) symptoms correlated positively with illusory tactile perceptions in the SSDT.

Overperception of symptoms seems to be strongly influenced by top-down processes. This effect can be linked to emotional cues that are able to activate different symptom-related schemas (representations) and has been investigated by comparing high- versus low-habitual symptom reporters (Bogaerts et al. 2010a, Constantinou et al. 2013). Although all participants reported higher levels of negative affect during the negative and symptom-related picture series compared to the positive and neutral picture series, only high-habitual symptom reporters reported more bodily symptoms after viewing the negative and symptom-related pictures (Bogaerts et al. 2010a). A learned association between negative emotional states and symptom reporting in high-habitual symptom reporters could explain this effect.

Symptom perception also seems to be strongly influenced by contextual variables. This has been demonstrated by Pennebaker (1982), who showed that distraction reduces the intensity of somatic symptom perception. The competition of cues model postulates that internal and external cues (internal sensations and symptoms, sensory perception of external stimuli) compete for the attention of the individual, and the larger the power of one of these cues, the lower the perceptual intensity of the others. Neuroimaging techniques further confirmed that distraction not only influences the cognitive interpretation of sensations but is also directly related to reduced amplitudes of sensory stimulation even in primary somatosensory fields (Bantick et al. 2002). Therefore, even in the era of acceptance strategies, distraction plays a role as a potential symptom coping strategy (Kohl et al. 2013).

Because alterations in the perception of body signals are considered essential for the development and maintenance of somatoform disorders, it is important to continue research regarding perceptual accuracy versus perceptual distortions. Some of the recent findings indicate that these processes are related to general symptom reporting independent of their etiology; therefore, these mechanisms may also be important in SSD, especially with polysymptomatic manifestations.

Symptom interpretation and catastrophizing. Catastrophizing cognitions refer to the tendency to overinterpret bodily sensations as signs of a serious disease and/or the tendency to overinterpret the likelihood and/or intensity of potential negative consequences of symptoms. In pain research, catastrophizing often refers more generally to the tendency to excessively focus on pain sensation (rumination), exaggerate its threat (magnification), and to feel helpless about pain (helplessness). Other examples of dysfunctional cognitions about illness-related constructs are the rigid assumption that being healthy is defined by the total absence of any physical sensations, beliefs that one has no control over symptom course or recurrence, or self-concepts of being bodily weak and intolerant of stress. Evidence is robust for these types of dysfunctional body- and health-related beliefs in health-anxious individuals (Marcus et al. 2007), and it also exists in other somatoform disorders and in chronic pain conditions (e.g., Rief & Broadbent 2007). A recent study also provided evidence that in patients with somatoform disorder the explicit self-concept is related to bodily weakness, and the implicit health-related self-concept differs from that of healthy control subjects (Riebel et al. 2013).

The role of cognitive interpretation of perceived sensations has been well confirmed using correlational studies and predictor studies. Catastrophizing somatic perceptions increases the aversive quality of the stimuli and is related to higher pain intensity and higher degrees of disability in a variety of situations. Catastrophizing is negatively associated with the tolerance of pain and other symptoms (e.g., Leeuw et al. 2007, Quartana et al. 2009), pain thresholds (Geisser et al. 2003), and even correlates with brain activation caused by painful stimuli in somatosensory fields of the cortex (Seminovicz & Davis 2006). Pain catastrophizing seems to mediate the relationship between sensation and suffering through unpleasantness, as well as the relationship between pain-related unpleasantness and suffering (Wade et al. 2011). Whereas many studies stress the significance of catastrophizing cognitions in chronic pain and other somatic conditions, other research has shown that preoperative catastrophizing predicts acute, postsurgical pain independent of the presence of anxiety or depression (e.g., Kahn et al. 2012). An up-to-date review also reports empirical support for an association between high catastrophizing and high fatigue in various clinical groups, e.g., patients with cancer, multiple sclerosis, and fibromyalgia (but because the number of studies is small, one cannot yet draw any conclusions for the separate conditions) (Lukkahatai & Saligan 2013). Fatigue catastrophizing is one cognitive factor that possibly contributes to persistent fatigue after cancer treatment (Goedendorp et al. 2013).

A catastrophizing interpretation bias could increase anxiety, which is associated with further physiological changes as well as body-related hypervigilance; both of these reactions promote further symptom perceptions. These cognitive processes could play a causal role in medically unexplained somatic conditions. In SSD related to a known biomedical condition, these cognitive biases may also contribute to increased symptom experiences, maladaptive illness behavior, and disability (a role that needs to be evidenced), but possibly would be of less importance regarding onset of the disorder itself.

Another way to assess the impact of automatic information-processing bias is to experimentally apply the affect misattribution procedure (AMP) by manipulating qualities of prime stimuli. One recent study provided preliminary evidence in a student sample showing that ambiguous stimuli (i.e., Chinese characters) were less often rated as pleasant after presentation of health-threatening prime pictures compared to conditions featuring neutral or no primes and that this AMP effect was associated with a self-report measure of health anxiety (Jasper & Witthöft 2013). However, another recently completed experimental study (S. Schroeder, A. Gerlach, & A. Martin, manuscript submitted) on automatic affective evaluations of somatosensory stimuli (modified AMP) was unable to show a stronger automatic negative interpretation bias concerning somatosensory sensations in patients with noncardiac chest pain when compared to subjects either with cardiac chest pain or without chest pain. Stronger anxiety sensitivity was the only psychological factor related to automatic affective interpretations of somatic sensations. This finding is further confirmed by a recent longitudinal study (Ruchkin & Schwab-Stone 2013).

To summarize, further studies are required to investigate the relevance of automatic interpretative processes to the course of distressing somatic syndromes with or without clinically significant health anxiety. Recent, more experimental approaches extend the knowledge based on correlational designs but offer only initial insights into the complex mechanisms involved.

Hypervigilance to somatic sensations and attention to illness-related stimuli. Hypervigilance toward bodily sensations and attentional bias for illness-related cues are central mechanisms in hypochondriasis and other somatoform disorders, according to cognitive-perceptual models. Attentional biases toward threat-relevant stimuli would be expected in health-anxious individuals and may also be present in those with DSM-5 illness anxiety disorder and SSD with health anxiety. A recent study on attentional bias for disorder-related words (emotional Stroop) comparing

patients with hypochondriasis, patients with other somatoform disorders, patients with panic disorder, and controls showed the strongest bias for threat-related words in patients with hypochondriasis. Results also suggest an attentional bias, although less strong, for illness-related words in other somatoform disorders (Gropalis et al. 2012). The attentional bias was reduced after four months of CBT for hypochondriasis and somatoform disorder but not for panic disorder.

CBT: cognitive behavioral therapy

Memory bias is another cognitive bias known to be relevant in other emotional disorders. Some findings suggest that implicit and explicit memory biases occur in patients with somatoform disorder (e.g., Lim & Kim 2005, Martin et al. 2007) and in those with chronic pain (e.g., Pincus & Morley 2001). However, two recent studies did not provide additional support for a disorder-specific memory bias in hypochondriasis and other somatoform disorders based on recognition (Gropalis et al. 2012), free recall, or directed forgetting of illness-related word stimuli (Wingenfeld et al. 2013). Nikendei and colleagues (2009) also failed to find a selective processing of word stimuli related to different symptom attributions when comparing pain patients with a somatic causal attribution style to those with a psychosocial causal attribution style. In a former study, we demonstrated that memory bias is based less on the verbal content per se and more on the memorized likelihood of threatening explanations (Rief et al. 2006b). The direct comparison of subjects with similar somatic symptoms resulting from different diseases produced further interesting results. Evidence for hypervigilance toward gastrointestinal stimuli (word recognition and recall) was stronger in patients with irritable bowel syndrome in comparison to those with an organic gastrointestinal disease (Posserud et al. 2009).

Evidence for cognitive mechanisms comes from investigating their variations in treatment contexts. One of the first evaluation studies using the SSD concept found that reductions in health anxiety, self-concept of bodily weakness, and body scanning significantly predicted improvement of physical functioning (Voigt et al. 2012). Results did not differ for patients with somatoform disorders and patients with SSD, and therefore these cognitive mechanisms seem to play a role in both concepts. However, the samples were highly overlapping and were selected from a psychosomatic hospital, and it remains unclear whether a sample from a more medical setting would produce the same results.

Emotion Regulation and Early Adverse Life Experiences

Emotion and emotion regulation have been thought to be related to somatic symptoms since the introduction of the term "somatization" by Stekel in 1924 (Allen & Woolfolk 2013). The alexithymia concept was developed in the 1970s and hypothesized that people who are unable to detect, name, and express feelings have an increased risk to develop somatic symptoms. The typical assessment instrument for alexithymia, the Toronto Alexithymia Scale (TAS or TAS-R; Taylor et al. 1992), has shown scores to be significantly associated with the number and intensity of somatic symptoms (Bach & Bach 1995). However, these associations between TAS scores and somatoform symptoms could be influenced by other common factors, such as depression, negative affectivity, or traumatic life experiences (Rief et al. 1996). Negative affectivity and neuroticism have been considered as precursors of somatic complaints for decades (Watson & Pennebaker 1989).

Many patients with multiple somatic symptoms do not associate their symptoms with their emotions, and a lack of emotional awareness is associated with difficulties in the differentiation of physical symptoms (Burton et al. 2009, Subic-Wrana et al. 2010). The psychodynamic approach postulates emotion regulation deficits in somatization (Henningsen et al. 2007); the deficits are thought to be caused by early life experience. Modern concepts of pain management emphasize the close association of emotion management and pain control and the symptom-enforcing role of negative emotions (Schweinhardt et al. 2008). A recent investigation indicates that the reduction

of negative emotion predicts reduced pain, especially after a period of high negative emotion (Connelly et al. 2007). Social exclusion can be another powerful trigger for negative emotions, and depending on the intensity and time perspective, the effect of social exclusion on affect and physical sensitivity versus numbing can be different (Bernstein & Claypool 2012). Emotion regulation capacities play a unique role in the prediction of specific facets of adjustment among people with chronic pain (Agar-Wilson & Jackson 2012). However, our understanding of the role of emotion regulation as a risk factor, as a correlate, or as a consequence of somatic symptoms is far from being clear, and more longitudinal and experimental studies are needed in this field. Moreover, whether our limited knowledge on the mechanisms of emotion regulation in somatoform disorders also applies to the new concept of SSD is the subject of vague speculations.

Early lifetime adversities are significantly higher in patients with somatoform disorders, even when assessed using longitudinal approaches (Creed et al. 2012, Gulec et al. 2013, Kuwert et al. 2012, Nelson et al. 2012). Interestingly, an association between traumatic life events and somatic symptoms has been found not only for somatoform disorders and hypochondriasis (Barsky et al. 1994) but also for functional somatic syndromes such as fibromyalgia (Bohn et al. 2013) and irritable bowel syndrome (Bradford et al. 2012). The role of traumatic experiences has been emphasized in typical biomedical conditions such as breast cancer and osteoarthritis (Stein & Barrett-Connor 2000). The current psychobiological model postulates that early lifetime adversities cause abnormalities of the biological stress response (hypothalamic-pituitary-adrenal axis) (Heim et al. 2008a,b) that can last throughout life and lead to vulnerability of the individual to develop somatic symptoms or disorders even decades after the original adverse events (Kuwert et al. 2012).

The experience of traumatic events also seems to cause the development of alexithymic patterns, which is an interesting extension of the traditional alexithymia model. Hereby alexithymia is not considered as a cause of somatic symptoms but rather as a coping strategy after experiencing overwhelming emotional and painful situations. It could be helpful for some patients to downregulate emotional processes after experiencing traumatic events. Increased rates of traumatic experience are considered to be a potential cause of emotion regulation deficits (Cloitre et al. 2005). More research is needed to understand when emotional downregulation is a helpful strategy and when it is associated with emotional avoidance, which functions as a mechanism to prolong symptoms.

Attachment styles have also been connected to the development of somatic symptoms, especially after the experience of adverse early life events. In particular, insecure attachment styles are associated with an increased risk of developing somatic symptoms (Taylor et al. 2000, 2012): a prevalence rate of 28% is reported for insecure attachment in patients with MUS. However, the sensitivity of this criterion is obviously low (with 72% of target patients not showing insecure attachment styles), and the specificity of such a potential risk factor is questionable. Insecure attachment styles have been reported for many other diseases as well (McWilliams & Bailey 2010). The generalization of results on attachment styles from somatoform disorders to SSD is currently not warranted because for many medical conditions, such an association is not shown.

The investigation of all the mechanisms reported above suffers from some serious limitations. Mechanisms that are shown to be able to increase the likelihood that a patient will report chronic somatic symptoms are not identical to the "real" causes for the development of these symptoms in a specific patient. Some of the mechanisms are more relevant for the maintenance and persistence of symptoms than for the original development of them. Some mechanisms are more relevant for understanding symptoms, whereas others are more relevant for understanding associated distress, disability, or lack of coping skills. Despite this critique, our understanding of mechanisms that are involved in the field of somatoform symptoms has greatly increased over recent decades (Rief & Broadbent 2007). The relevance for the new category of SSD is obvious for some of these mechanisms, and the application for the new diagnosis seems justified; in contrast, several other

mechanisms seem to be very specific for somatoform disorders, and their relevance for the new diagnosis is speculative. Notably, the role of the presented mechanisms could differ in SSD in cases with a clear biomedical disease and cases without a sufficient medical explanation. As a potential causal factor in MUS, the same psychological factor could work as a mediator of distress in medically explained symptoms or interact in a different way with reinforcement processes and other mechanisms during the maintenance period. The similarities and differences of these mechanisms in variants of SSD are not well understood, and a premature merging of medically explained and unexplained symptoms would prevent their potential detection.

Placebo and Nocebo Research as a Paradigm for Symptom Development and Symptom Control

Placebo and nocebo mechanism investigations are also shedding light on SSDs. While placebo mechanisms can increase the expected benefit of a treatment, the psychological factors that reduce the expected treatment effects or that induce additional side effects are so-called nocebo mechanisms. Over the past 20 years, it has been impressively shown that placebo and nocebo mechanisms not only influence psychological aspects of disorders but also are able to influence physiological aspects of most biological systems (Enck et al. 2013, Rief et al. 2011a). This line of research is of relevance for SSDs because nocebo mechanisms can be understood as psychological processes in the development of somatic symptoms, whereas placebo mechanisms can be interpreted as mechanisms to control symptom perception. The most frequent mechanisms examined in this field are the roles of expectations, associative learning, and interactional aspects such as doctor-patient relationship. If people expect to develop symptoms after stimulations (e.g., through medication, electromagnetic fields, wind turbines), this expectation increases the risk of developing these symptoms even if no further stimulations are provided. These results have been demonstrated experimentally in a variety of studies (Crichton et al. 2013, Oftedal et al. 2007, Vernia et al. 2013, Witthöft & Rubin 2013). Moreover, people can also learn to develop symptoms in a specific environment through processes of associative learning (Colloca et al. 2008, 2010). Even observational learning can play a role in amplifying the development of somatic symptoms: the more pronounced a pain reaction of an observed person in a specific situation is, the higher the pain perception of the observer when being confronted with the same situation; similarly, the observation of a placebo effect increases subsequent placebo reactions of the observer (Colloca & Benedetti 2009). The development of side effects in clinical trials seems to be another phenomenon that must be interpreted under this perspective. Many patients discontinue drug intake explicitly because of side effects that are attributed to the medication, even though these patients are in the placebo group (Rief et al. 2006a, 2009). The development of side effects seems to be frequently associated with personality factors, sometimes even more than with features of the medication itself. In other words, some people seem to have learned to respond with side effects to any medication that is presented, and they develop an expectation that they will continue to experience side effects in the future ("side effect sensitivity"); therefore, the assessment of past side effect experience is one of the best predictors for the development of future side effects in medical interventions (Horne et al. 2013).

The results of placebo and nocebo research indicate that the corresponding mechanisms are able to act in psychological as well as physiological systems. Therefore, translating these mechanisms from somatoform disorders to SSDs seems to be justified because many studies in the placebo field dealt with somatic symptoms in general (not only somatoform). However, the role of expectation and associative learning can be different for every biological system as well as for the psychological system. Pain perceptions seem to be vulnerable both to expectation

and conditioning. Physiological systems with less perceptual aspects (e.g., immune functioning) seem to be more prone to modifications via learning principles, whereas suggestions/cognitive instructions are less relevant in these conditions.

Placebo and nocebo mechanisms are also relevant to intervention planning. The modification of expectations as well as extinction learning should play a significant role when patients cope with symptoms. Unfortunately, only a few intervention programs for patients with somatoform symptoms focus on these mechanisms. A reformulation of intervention programs, from the perspective of (*a*) how they address expectation optimization and (*b*) how to modify learning processes that were acquired over the many years of symptom persistence, is necessary.

HOW TO USE THE NEW DIAGNOSES OF SOMATIC SYMPTOM DISORDERS IN RESEARCH: A PROPOSAL FOR MODIFYING DSM-5 CRITERIA

The new criteria for SSDs pose several problems in terms of the continuation of research fields and transformation of current knowledge to the new diagnoses. Some diagnoses such as SSD include an extreme heterogeneous cluster of patients with MUS, patients with medically explained symptoms with significant psychological factors, patients suffering from pure pain symptoms, or patients suffering primarily from hypochondriasis. Patients with unique and specific syndromes (e.g., low back pain) are summarized together with patients with a broad variety of multiple symptoms (e.g., somatization disorder). Patients with the diagnosis of SSD can belong to either a group with a very good response to psychological interventions (e.g., hypochondriasis subtype) or a group with low-to-moderate effect sizes of psychological interventions (e.g., chronic pain syndromes). For some of the conditions, anxiety is a prominent psychological feature (e.g., hypochondriasis, fear of progression in cancer), whereas for others, demoralization or physical deconditioning might be more relevant. Moreover, the need to include medical aspects in psychological intervention programs can differ significantly for patients with the same SSD diagnosis. All these aspects challenge the reliability and validity of the diagnostic process and raise questions about the generalizability of research results. Finally, the question arises whether the SSD diagnosis fulfills the most basic requirement of diagnoses, namely to facilitate communication in the health care system. The diagnosis of SSD, based on the current concept of the term, would not be sufficient to steer treatment planning; health care providers would require much additional information. It is impossible to define treatment recommendations for SSD without defining subtypes; thus, attempts to develop empirically founded treatment guidelines would be disabled.

Until the questions raised above are answered, we suggest that the new proposals on how to use the DSM-5 somatic symptom and associated disorders diagnoses be modified. The following proposal enables successful fields of research and treatment recommendations to continue as well as cross-reference to the DSM-5.

At least for research purposes, it would be useful to distinguish the following groups and to define common classification criteria:

- somatic symptom disorder, somatic symptoms not better explained by a biomedical condition, monosymptomatic type
- somatic symptom disorder, somatic symptoms not better explained by a well-known biomedical condition, polysymptomatic type
- somatic symptom disorder in the context of well-known biomedical conditions
- pain disorder (specify: monosymptomatic versus polysymptomatic type; episodic versus chronic type)
- illness anxiety disorder (hypochondriasis)

Table 2 offers an overview of suggested criteria for these five clinical problems as well as a cross-reference to the existing DSM-5 system. The validity for this suggested subclassification comes from different treatment responses (e.g., illness anxiety versus chronic somatization), different treatment needs (medically explained somatic symptoms versus medically unexplained somatic symptoms), different time course of the disorder (e.g., more fluctuation in illness anxiety versus chronic stability in polysymptomatic somatization), different prognoses (poorer prognosis for polysymptomatic versus monosymptomatic disorders), and the different roles of behavioral and psychological factors.

The proposal shown in **Table 2** offers a valid classification approach that allows the changeover to DSM-5 but also makes use of current research knowledge. At present, it is unclear whether SSD in the context of a serious medical condition such as cancer or diabetes can be merged with SSD without any biomedical pathology, and as long as this is unclear, we recommend differentiating it in research. Notably, other diagnostic categories of DSM-5 allow for diagnoses "due to another medical condition" (e.g., depressive disorder; Am. Psychiatr. Assoc. 2013, p. 180) or use criteria such as "not attributable to a concurrent medical condition" (e.g., food intake disorder; Am. Psychiatr. Assoc. 2013, p. 334); the same rationale could apply for SSD. Moreover, the prominent psychological features in different medical conditions can also vary, and a different treatment may be needed for a patient suffering from the fear of suffering from hypoglycemia or long-term consequences in diabetes than for a patient suffering from cancer.

The distinction between monosymptomatic and polysymptomatic syndromes can be considered important because symptom counts and number of pain sites are significant predictors of persistence and disability as well as work absenteeism (Escobar et al. 2010, Haukka et al. 2013, Rosmalen et al. 2011). Moreover, the number of functional somatic syndromes has been evidenced as a strong risk factor for the incidence of another functional somatic syndrome (Warren et al. 2013). If polysymptomatic syndromes are established, the likelihood of the remission of symptoms and a return to normal quality of life will be very low (Keijsers et al. 2010, Rief & Rojas 2007).

Illness anxiety disorder clearly differs from syndromes that were formerly characterized as somatization disorder or chronic pain disorders. In many cases, the course of illness anxiety disorder resembles the course of fluctuating anxiety disorders such as panic disorder, with hours or days of emotional crises being followed by periods of low anxiety. Treatment response to hypochondriasis is high, whereas treatment response to other SSDs can be low to medium. All of these aspects underline the validity of a unique illness anxiety disorder/hypochondriasis diagnosis.

Finally, pain syndromes are by far the most prevalent single somatic symptoms, with special relevance for chronicity and health care use, which underlines the need to establish a distinct pain diagnosis and not just a specifier. Moreover, pain research has its own traditions and concepts, with unique diagnostic approaches (e.g., chronic widespread pain, neuropathic pain), investigation of outcome predictors (e.g., fear avoidance), and treatments (e.g., multidisciplinary treatments, sometimes combining analgesic and psychological interventions). Mechanisms such as the perceptual sensitization process, supported by neural plasticity and activation processes in the so-called pain matrix of the brain, are clearly defined for pain conditions but less so for the variety of (other) syndromes of SSD (Davis & Moayedi 2013).

As mentioned above, the empirical evidence for the selection of the "positive" psychological criteria introduced by DSM-5 is still weak. Therefore we should not wait until the dawn of DSM-6 before evaluating the classification validity of the present, and other, psychological criteria. This has two implications: First, the suggested DSM-5 B criterion for SSD should be continuously evaluated for its validity; and second, new criteria that could be alternatively or additionally used should be investigated (see below).

Table 2 Proposed ways to use the DSM-5 diagnoses "somatic symptom and related disorders" in research

Somatic symptom disorder, type A (MUS; monosymptomatic type)
Description: somatic symptoms are not better explained by a general medical condition; monosymptomatic
How to classify?
■ DSM-5 criteria for somatic symptom disorder apply
■ One somatic symptom or well-circumscribed somatic syndrome is the focus of somatic distress (e.g., nausea, abdominal pain and bloating, noncardiac chest pain, dizziness)
■ The most significant somatic symptoms causing distress for the patient are not better explained by a well-known biomedical condition
Specify:
■ Gastrointestinal type (e.g., irritable bowel syndrome, nonulcer dyspepsia)
■ Cardiac type (e.g., noncardiac chest pain)
■ Conversion type (e.g., pseudoseizures)
■ Fatigue type (e.g., chronic fatigue syndrome)
■ Environment attribution type (e.g., multiple chemical sensitivities)
Somatic symptom disorder, type B (MUS; polysymptomatic type)
Description: somatic symptoms are not better explained by a general medical condition; polysymptomatic
How to classify?
■ DSM-5 criteria for somatic symptom disorder apply
■ Somatic symptoms that are the focus of somatic distress cover more than one body site/biological system (e.g., former "somatization disorder" and other patients with multiple somatoform symptoms)
■ The most significant somatic symptoms causing distress for the patients are not better explained by a well-known biomedical condition
Somatic symptom disorder, type C (with biomedical condition)
Description: somatic symptoms in the context of a well-known biomedical condition
How to classify?
■ DSM-5 criteria for somatic symptom disorder apply
■ Somatic symptoms that are the focus of somatic distress are mainly caused by a well-known biomedical condition
Specify: biomedical condition (e.g., diabetes, cancer, Crohn's disease, myocardial infarction, neurodermatitis, epilepsy, migraine)
Pain disorder
Description: Pain disorder
How to classify?
■ Pain symptoms are the focus of somatic distress
■ DSM-5 criteria for somatic symptom disorder, specifier "with predominant pain," apply
■ If pain symptoms are in the context of further somatic complaints, a diagnosis of somatic symptom disorder type B might be more appropriate
Specify:
■ Pain mainly caused by a well-known biomedical condition
■ Pain characterized by physical and psychological factors
■ Monosymptomatic versus polysymptomatic pain conditions

(*Continued*)

Table 2 (*Continued*)

Illness anxiety disorder
Description: illness anxiety, hypochondriasis
How to classify?
■ Preoccupation with having or acquiring a serious illness. If somatic complaints are present, or if illness anxieties or preoccupation are clearly excessive or disproportionate and are a significant reason for distress
One of the following DSM-5 criteria applies:
■ Criteria for somatic symptom disorder
■ Illness anxiety disorder

Note: Although somatic symptom disorder types A and B are exclusive, type C can be combined with both of the other two types. Pain disorder could be combined with the other somatic symptom disorder diagnoses if pain disorder itself does not fully cover the reported symptomatology but is prominent enough to need its own diagnosis. Illness anxiety disorder can be combined with any other disorder reported here. Abbreviations: DSM-5, *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition; MUS, medically unexplained symptom.

Although cognitive, affective, and behavioral characteristics are often interrelated, they are not necessarily present at the same time and within the same individual. Rather, these features seem to vary considerably between affected subjects. We therefore suggest further operationalizing the B criterion of SSD by providing a range of cognitive, affective, and behavioral characteristics for further evaluation of validity aspects.

- Cognitive symptoms: disproportionate and persistent thoughts about the seriousness of one's symptoms (e.g., somatic causal symptom attribution despite contradicting medical information), catastrophizing of bodily sensations, self-concept of bodily weakness, beliefs about low symptom tolerance, fear avoidance beliefs, rumination about physical complaints.
- Affective symptoms: health anxiety, fear of movement, demoralization because of somatic symptoms, negative affectivity.
- Behavioral symptoms: fear avoidance behavior, avoidance of physical effort, avoidance of situations or activities that provoke illness anxiety, excessive reassurance-seeking behavior (presented to physicians as well as to significant others, such as partners), checking behavior (e.g., self-inspection of body functions or for signs of disease).

THE NEW PROPOSAL OF SOMATIC SYMPTOM DISORDER AND ITS RELEVANCE FOR INTERVENTION RESEARCH

The DSM-5 proposal for somatic symptom and related disorders does not seem to offer a good starting point for intervention research, whereas the modifications suggested in this review offer a better platform for treatment studies. From a scientific point of view, treatments are most successful if they follow the results of mechanism research. A good example is hypochondriasis/illness anxiety disorder. Some psychological interventions follow the conceptualization that hypochondriasis is mainly driven by anxiety and therefore interventions focus on managing anxieties: These interventions have been shown to be highly successful, with effect sizes of Cohen's d above 2.0 (Salkovskis 1995). Even short (six-hour) interventions succeed in reducing the syndrome significantly (Barsky & Ahern 2004), and Internet-based applications confirmed very large effect sizes (e.g., $d = 2.09$ for health anxiety inventory, pre-treatment versus follow-up scores) (Hedman 2011).

Several approaches have been tested for various pain disorders but with only moderate effect sizes so far (Glombiewski et al. 2010a,b; Morley 2011). Two strategies can be helpful to further

improve treatment efficiency: First, we can try to better understand the mechanisms involved in chronic pain development and address them more specifically; second, we can try to tailor treatments more specifically to individual problem profiles. Psychological pain management programs have been shown to be useful, but they offer a broad and less theory-driven approach. A substantial subgroup of patients with chronic pain shows fear avoidance (Vlaeyen & Linton 2000), which is a major predictor for symptom persistence. For those patients, exposure-based interventions may be successful (de Jong et al. 2012). Moreover, third-wave interventions such as acceptance and commitment therapy or mindfulness-based cognitive therapy have been also shown to be effective, but on a global level they do not seem to be more effective than standard CBT procedures (Wetherell et al. 2011). An increase in success rates might be achieved by developing decision rules to identify which patient needs standard CBT, exposure-based interventions, and/or more acceptance-based treatments.

For somatization disorder and multiple somatoform symptoms, the effect sizes of interventions are low yet still significant (Kleinstäuber et al. 2011, Sattel et al. 2012, Schroder et al. 2012). The question arises whether current treatment approaches adequately incorporate the knowledge on involved mechanisms. Some well-evaluated mechanisms are not sufficiently addressed in current treatment programs (e.g., the role of expectation). The role of emotion regulation, a concept that has been found to be of relevance for all of these syndromes, also might not be sufficiently addressed in current intervention proposals. After reviewing the neuroimaging literature, Landa and colleagues (2012) concluded that psychotherapeutic and/or pharmacological interventions that foster the development of affect regulation capacities will serve to modulate aberrantly activated brain circuits and thus will be of particular benefit in the treatment of somatoform pain.

Psychological interventions assigned to help patients with medically explained versus medically unexplained somatic symptoms might show some overlap, but some differences also must be considered. Frequently, patients in both groups initially have a more biomedical concept about the cause of their complaints, and therapists need to offer examples of behavioral experiments to demonstrate how psychological factors can influence the intensity and annoyance of somatic complaints. However, if the somatic symptoms derive from a serious medical condition, this must be also considered both in explaining illness models and in deriving intervention rationales with patients. A patient suffering from diabetes and SSD needs a treatment plan that integrates not only coping strategies for somatic symptoms, but also strategies for interpreting somatic sensations correctly (e.g., differentiating hypoglycemia from general dizziness), for adhering to medication, and for reducing fears of developing threatening conditions (e.g., blindness). In contrast, a patient with somatization syndrome might need interventions such as defocusing self-attention from bodily perception, increasing awareness for environmental events and their influence on bodily well-being (competition of cues), reducing physical avoidance behavior, and establishing a useful level of physical activity. Although catastrophizing physical sensations might be fully inappropriate in patients with somatic symptoms not caused by a biomedical condition, it is much more appropriate in a patient who received cancer treatment just a few months ago and is still at increased risk for recurrence of the disease. Thus, the role of specific treatment programs, techniques, and target processes may vary considerably between the possible "phenotypes" of SSD.

CONCLUSIONS

The revision of somatoform disorders to somatic symptom and related disorders in DSM-5 addresses a serious need for reformulation. However, it leaves several problems unsolved, and it must be considered a challenge for researchers specialized in this field. Therefore, we suggest modifications and specifications that are based on DSM-5 criteria, that allow crossovers with past

lines of research, and that are less subject to unclear assumptions. As long as the similarity between mechanisms involved in medically explained and medically unexplained symptoms is not shown, researchers should test this hypothesis, which implies continuing to use this distinction, at least as a covariate. Pain research and research on hypochondriasis/illness anxiety disorder have revealed very specific results; these lines of research merit continuation and should not be sacrificed to DSM-5. The suggested modifications, which are based on DSM-5 and on empirically validated research, offer parallel developments.

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