Journal of Abnormal Psychology
2012, Vol. 121, No. 2, 530–543

0021-843X/11/$12.00 DOI: 10.1037/a0026536

Tactile Perceptual Processes and Their Relationship to Somatoform Disorders

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The Somatic Signal Detection Task (SSDT) is a recent paradigm serving to examine perceptual processes likely relevant for somatoform disorders. We tested whether touch illusions are more easily induced in individuals suffering from somatoform disorders (SFD) and whether their perceptual threshold for tactile stimuli is lower compared to healthy controls. Thirty-three participants with SFD and 32 healthy controls reported whether they recognized near-threshold tactile stimuli at their fingertip, which were presented in half of the test trials. With a probability of 0.5, an auxiliary visual stimulus was additionally presented. Tactile detection thresholds, tactile sensitivity, response bias, and the rate of false-positive perceptions of the tactile stimulus were assessed. In both groups, the light stimulus led to an amelioration of tactile sensitivity as well as to a more liberal response style. The SFD group was characterized by a more liberal response bias in the first half of the light-absent condition compared to the healthy controls. Within the SFD group, the report of somatoform (especially pseudoneurological) symptoms correlated positively with illusory tactile perceptions in the SSDT. Tactile thresholds in the SSDT were measured reliably (r_{tt} = .86) and were significantly lower in the SFD group. The notion that general perceptual dispositions influence the formation of symptom perception may thus complement cognitive models of SFD.

Keywords: Somatic Signal Detection Task, somatoform disorders, medically unexplained symptoms, tactile detection threshold, response bias

Somatoform symptoms, also called "medically unexplained symptoms" (MUS), are a common phenomenon in primary health care settings (De Waal, Arnold, Eckhof, & van Hemert, 2004). Afflicted persons suffer from bodily complaints that cannot be explained sufficiently by medical conditions. Various models of somatoform disorders (SFD) have been developed, but the precise etiology of MUS and SFD is still unknown (Brown, 2004; Witthöft & Hiller, 2010). Most of the current models assume an interaction of cognitive and perceptual somatosensory processes that lead to behavioral, affective, and biological consequences. Cognitive–behavioral models emphasize factors like catastrophizing, body-focused attentional styles, and excessive illness behavior, for example, "doctor shopping," leading in combination to vicious circles that amplify symptom perception (Barsky, Wyshak, & Klerman, 1990; Rief, Hiller, & Margraf, 1998).

This article was published Online First December 12, 2011.

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The study was supported by a scholarship from the Psychological Institute of the University of Mainz.

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According to a recently proposed filter model (Rief & Barsky, 2005), the perception of MUS is determined by factors that increase the likelihood of bodily signals (e.g., overarousal, sensitization), on the one hand, and factors that decrease activity of filter systems, on the other hand (e.g., selective attention, lack of distraction). The combination of these two factors could result in the perception of symptoms, which most individuals normally would not experience.

Brown (2004) proposed an integrative, conceptual model of MUS focusing on cognitive psychological principles (Norman & Shallice, 1968). In the tradition of dual-process theories of the human mind (e.g., Barrett, Tugade, & Engle, 2004), Brown proposed two different attentional systems (i.e., a primary and secondary attentional system). These systems select so-called "rogue representations," which refer to information related to physical symptoms. The specific content of these multimodal representations in memory depend on prior experiences (e.g., illness concerning oneself or family members). According to the model, symptom experiences arise from the automatic activation of these symptom representations in the primary attentional system. However, the selection of symptom representations by the primary attentional system can be moderated and facilitated by the secondary attentional system using an extensive body-focused attentional style, negative affect, or a focus on disease-confirming information. In essence, Brown (2004) conceptualizes MUS as illusory somatosensory phenomena involving perceptual and memory processes.

In sum, the different models that have been proposed for MUS agree on the central role of cognitive processes (i.e., attention and memory) in the perception of symptoms. However, one of the crucial unresolved issues is whether individuals suffering from MUS are more or less sensitive in their perception of somatosensory events (e.g., due to conflicting memory representations that cause somatosensory disturbance). We use the term *sensitivity* in the signal detection theory sense (Green & Swets, 1966), denoting the capability of an observer to discriminate between signal and no signal in a detection task (i.e., as indexed by d'), irrespective of his or her willingness to respond that the signal had been presented (response bias, indexed by c). Evidence for the position that patients with MUS are actually less sensitive in their perception of interoceptive processes has been presented by the workgroup of Van den Bergh et al. (Bogaerts et al., 2010) and is in line with the theoretical model proposed by Brown (2004). In contrast, the concept of somatosensory amplification (Barsky et al., 1990) and the filter model (Rief & Barsky, 2005) would rather suggest more sensitive perception of somatosensory events.

To objectively study abnormal somatosensory perceptual processes in the realm of somatoform disorders, an experimental paradigm has recently been proposed and will be described in the following section.

The Somatic Signal Detection Paradigm

The Somatic Signal Detection Task (SSDT; Lloyd, Manson, Brown, & Poliakoff, 2010) aims at operationalizing the idea of "rogue representations" as laboratory analogs of somatoform symptoms in the sense of Brown's cognitive conception of MUS (Brown, 2004). Within the paradigm, cognitively triggered illusory touch experiences are considered to be an analogy of somatoform symptoms. In the SSDT, near-threshold tactile stimuli are delivered to the fingertip with a probability of 0.5, either in combination with or without an auxiliary visual stimulus. Thus, there are four types of trials: vibration only, vibration-plus-light, light-only, and no-stimulus trials. The light stimulus is expected to trigger illusory touch perceptions by activating representations of the tactile stimulus in the light-only condition (Lloyd et al., 2008). Within the paradigm, this effect is attributed to normal multisensory integration, rather than potential conditioning processes between the light and tactile stimulus (McKenzie, Poliakoff, Brown, & Lloyd, 2010). Attending to the body also has an effect on false-alarm rates: for example, illusory touch experiences are significantly more likely in light-present trials when participants have the opportunity to look at their hand (Mirams, Poliakoff, Brown, & Lloyd, 2010). Moreover, independent of the visual stimulus, the number of false alarms, which could be viewed as illusory touch experiences in the SSDT paradigm, was found to be related to the level of somatoform symptoms reported in a nonclinical sample of university students (Katzer, Oberfeld, Hiller, & Witthöft, 2011).

Aims of the Present Study

Until now, studies using the SSDT were carried out in student populations or subclinical samples (e.g., Lloyd et al., 2008; Katzer et al., 2011). In the present study, we examined participants with SFD using the SSDT and compared their behavior to healthy controls. Additionally, we compared tactile thresholds between the two groups to explore their role in the context of SFD. We used an adaptive procedure to measure tactile thresholds (Levitt, 1971) that was demonstrated to be reliable ($r_{tt} = .84$) in a previous study (Katzer et al., 2011).

The first aim of the current study was to examine whether a higher rate of false alarms (illusory tactile perceptions) might be found in individuals with SFD (Brown, Brunt, Poliakoff, & Lloyd, 2010), which should be accompanied by a liberalization of response bias, that is, a pronounced tendency to respond "yes, signal present," irrespective of the actual occurrence of a tactile stimulus.

A second aim was to replicate previous results of SSDT studies in subclinical samples (Lloyd et al., 2008; McKenzie et al., 2010; Mirams et al., 2010; Brown et al., 2010) in order to relate these findings to clinical samples. Whereas for a student sample Katzer et al. (2011) did not find an elevated false-alarm rate in the light-present condition, we expected to find a more pronounced tendency to report false alarms in this cross-modal condition in the clinical sample. Encouragingly, Brown et al. (2010) found in a subclinical sample that experiencing illusory perceptual events was more likely in subjects with a tendency toward somatoform dissociation, that is, pseudoneurological symptoms, despite perceptual abilities comparable to healthy controls.

Tactile sensitivity (as indexed by d') was expected to be slightly augmented in the light-present condition because this was a result of previous cross-modal studies (e.g., Ro, Wallace, Hagedorn, Farnè, & Pienkos, 2004).

Finally, as in our previous study (Katzer et al., 2011), we measured tactile detection thresholds in a two-interval, forced-choice, adaptive procedure. This allowed answering the question whether differences between the two groups in terms of their ability to detect tactile stimuli contributed to observed differences in the SSDT. We also analyzed possible linear relationships between SSDT parameters like tactile sensitivity and response bias, the tactile thresholds, and the level of somatoform symptoms as well as general psychopathology (e.g., trait anxiety and depressive symptoms).

Method

Participants

Participants were recruited in two different manners. Participants with SFD were recruited using a psychotherapeutic outpatient clinic at the University of Mainz. Control subjects were recruited using advertisements and placards in public places and diverse institutions in the city of Mainz. The study was approved by the Ethics Committee of the German Psychological Society. All participants provided written informed consent prior to participation.

Exclusion criteria for participants were: evidence for a single organic cause of bodily symptoms ($n = 1$), inability to comprehend experimental information in the German language ($n = 4$), diagnoses of bipolar disorders, psychotic disorders, or mood disorders of severe psychopathology or psychotic features ($n = 5$), substance-related disorders, and sensibility disturbances of the index finger ($n = 2$). Inclusion criteria implied an age range of 18 to 65 years. Initially, 77 persons were invited to take part in the study; after checking exclusion criteria, 65 participants remained in the sample.

Participants were tested individually in a dimly lit room in front of a console containing a red LED and a 1.4 × 2.3 cm bone conductor surface (Oticon BC461-1) that delivered spike-waveform vibrations with a frequency of 50 Hz to the dominant hand's index fingertip, addressing Pacinian and Meissner corpuscles (Treede, 2007). The intensity of the applied tactile stimuli was adjusted by a second console panel. The experimenter sat at an angle of 90 degrees to the participant in front of a LCD monitor in order to give instructions and to record the participant's responses. The experiment was run with Inquisit software. Circumaural headphones (Sennheiser HD 201) were used to give acoustic signals at a comfortable level of loudness at the beginning and the end of the trials.

Materials and Procedure

Measurement of tactile detection thresholds. The dominant hand was determined using the Edinburgh Handedness Inventory (Oldfield, 1971). The tactile detection thresholds of the dominant hand were measured in a two-alternative forced-choice task combined with an adaptive procedure (Levitt, 1971). Each of the two observation intervals had duration of 1330 ms and was preceded by an auditory signal of 25 ms duration (see Figure 1).

The vibrotactile signal was applied for 20 ms, following 660 ms after the beep, randomly during either the first or the second observation interval, with equal probability. The participant's task was to indicate the interval during which the tactile stimulus occurred. A new trial started after the experimenter had recorded the participant's response. We used a two-interval task to minimize the potential effects of response bias on the threshold estimates (e.g., Ulrich & Miller, 2004; Hiscock, Branham, & Hiscock, 1994; Macmillan & Creelman, 1991; Green & Swets, 1966).

The measurement of the tactile perception threshold involved three blocks, which are outlined in the following paragraphs.

In the practice block, first 10 trials were run at maximum vibration intensity in order to familiarize each subject with the tactile stimulus. The intensity of the vibrotactile stimulus was defined using a scale of arbitrary units that ranged from 0 (*no stimulation*) to 100 (*initial, maximum intensity*). Afterward, whenever registering two consecutive correct responses, the intensity of the vibrating stimulus was decreased by 10 units. If one incorrect response was given, the change in intensity level was reversed and increased by 10. This two-down, one-up adaptive procedure converges at a stimulus intensity corresponding to 70.7% correct responses (Levitt, 1971). The practice block contained 50 trials.

After the practice block, a tactile threshold was determined using the same adaptive procedure as described for the practice block but without presenting the initial stimuli with maximum intensity. In this type of adaptive procedure, a trial on which the direction of the stimulus level sequence changes from up to down or vice-versa is termed a *reversal* (Levitt, 1971). The measurement of the tactile threshold was terminated after eight reversals. The number of trials was not limited. The arithmetic mean of the intensities of the tactile stimulus at the eight reversals was taken as the individual tactile threshold that was used in the subsequent SSDT.

After the SSDT, a second block of the described tactile threshold measurement followed to assess reliability. In this block, the same adaptive procedure as above was used.

Somatic Signal Detection Task. The SSDT (Lloyd et al., 2008) was administered in four consecutive test blocks of 40 trials. Four different trial types were presented: vibration-only, vibration-plus-light, light-only, and no-stimulus. Each trial type was presented 10 times within each block of 40 trials, in random order. Thus, 80 trials were obtained in the light-present condition (vibration-plus-light and light-only trials) and 80 trials in the light-absent condition (vibration-only and no-stimulus trials). Each subject was tested in all conditions. The blocks allowed us to structure the sessions and to offer pauses to the participants. The intensity of the previously measured individual tactile threshold was used when presenting the tactile stimulus.

Figure 2 presents an overview of the temporal structure of the different trial types. In the SSDT, there was only one observation interval. The beginning and the end of each type of trial was signaled using a 25-ms tone. In total, a trial lasted 2300 ms.

In vibration-only trials, the tactile stimulus was presented for 20 ms in the middle of the 2300-ms time period. In vibration-plus-light trials, the light was presented in synchrony simultaneously with the tactile stimulus (cf. Lovelace, Stein, & Wallace, 2003). In light-only trials, only the visual stimulus was presented for 20 ms, in the same temporal position as in the vibration-plus-light trials. In no-stimulus trials, neither a tactile stimulus nor a light were presented.

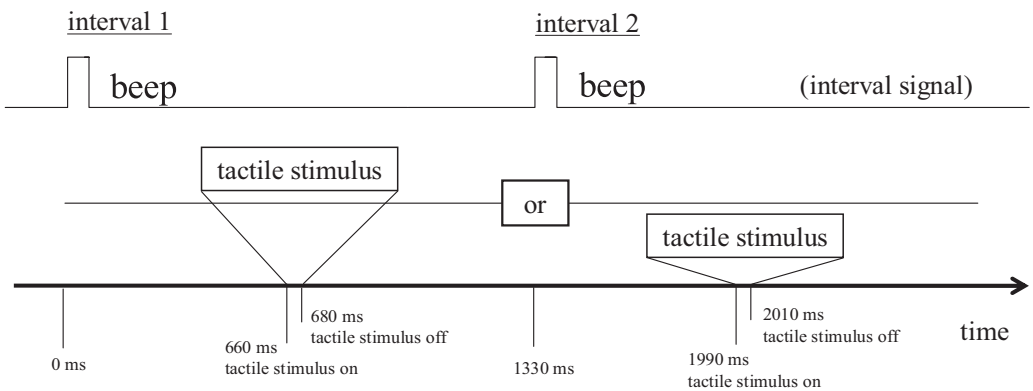

Figure 1. Schematic depiction of the two-interval task used for the threshold measurements.

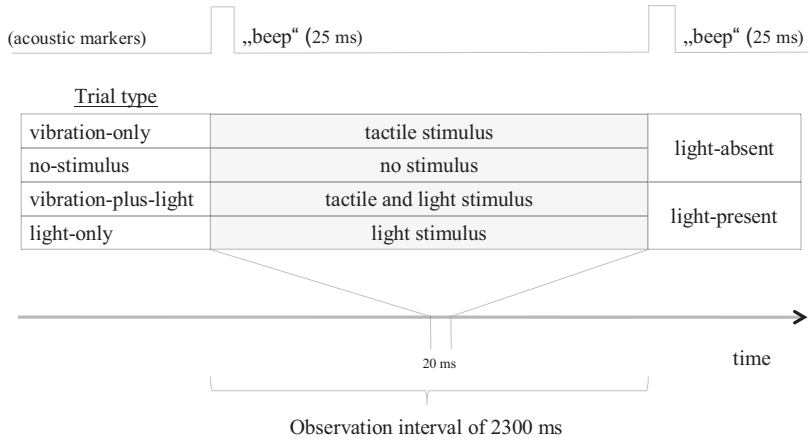

Figure 2. Schematic depiction of a trial in the somatic signal detection task.

Participants rated their confidence with the presence of the tactile stimulus on a 4-point scale using the response categories *definitely yes*, *maybe yes*, *maybe no*, and *definitely no*. The experimenter started the new trial after registering the participant's response.

Diagnostic Procedures

Self-report measures. In the following section, self-report measures that were used to characterize our samples are described.

The Patient Health Questionnaire (PHQ-15; Kroenke, Spitzer, & Williams, 2002) focuses on 15 somatic symptoms relevant to the diagnoses of somatization disorder. It measures somatic symptom severity and comprises 13 items from the German version of the PHQ somatic symptom module. Three response categories, ranging from 0 (*not bothered at all*) to 2 (*bothered a lot*), serve to estimate symptom severity during the last four weeks. Furthermore, two items of the PHQ Depression scale were added; they embody physical symptoms as well (feeling tired; trouble sleeping). The rating scale of these items ranges from 0 (*not at all*), 1 (*several days*), to 2 (*more than half the days* or *nearly every day*). Kroenke et al. (2002) demonstrated good reliability and validity for the PHQ-15, reporting a Cronbach's alpha coefficient of .80. In the current sample, Cronbach's alpha was .86.

Additionally, the Screening for Somatoform Symptoms–2 (SOMS-2; Rief, Hiller, & Heuser, 2008) was used to explore bodily complaints during the last two years that were not explained by organic causes. Its 53 items are based on the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (*DSM–IV*) as well as the *International Classification of Diseases, Tenth Revision*. Rief et al. (2008) reported an internal consistency of Cronbach's alpha = .88. Retest-reliability (72 hr) has been reported between r_{tt} = .85 and r_{tt} = .87. Furthermore, discriminant validity has been shown for the SOMS-2 (Rief et al., 2008). Cronbach's alpha in the current sample was .92. The SOMS-2 subscales showed the following reliabilities in our sample: The SOMS-2 Pain scale had a Cronbach's alpha of .77; SOMS-2 Gastrointestinental scale, a Cronbach's alpha of .84; Pseudoneurological scale, a Cronbach's alpha of .75; and Vegetative scale, a Cronbach's alpha of .73.

The Whitley Index, with 14 dichotomous items, was used to assess health anxiety. Whereas the dimensionality of the 14 items is still under debate (e.g., Conradt, Cavanagh, Franklin, & Rief, 2006; Schwarz, Witthöft, & Bailer, 2007), acceptable reliability and validity of the Whitley Index have been shown. Hinz, Rief, and Brähler (2003) reported a Cronbach's alpha of .83 for the 14 items. In the current sample, Cronbach's alpha was .79.

Trait anxiety was measured by the German version of the State-Trait Anxiety Inventory (STAI; Laux, Glanzmann, Schaffner, & Spielberger, 1981). It consists of 20 items with response categories ranging from 1 (*almost never*) to 4 (*almost always*). The Cronbach's alpha coefficient of the STAI in the current sample was .94.

The Beck Depression Inventory−II (BDI-II; Hautzinger, Kühner, & Keller, 2009) was used to assess severity of depressive symptoms. It consists of 21 items that comprise different depressive symptoms (e.g., loss of interest or pleasure, changes in appetite or weight, decreased energy). The Cronbach's alpha coefficient in the current sample was .95.

Structured Clinical Interview for DSM–IV Axis I Disorders. The German version of the Structured Clinical Interview for DSM–IV Axis I Disorder (SCID-I; Wittchen, Wunderlich, Gruschwitz, & Zaudig, 1997) was administered to all participants in order to diagnose somatoform disorders, to control for the occurrence of comorbid disorders, especially current affective and anxiety disorders, and to describe the characteristics of our sample. Final experimental group membership was assigned after the SCID-I administration. A trained clinical psychologist administered the SCID-I. Validity of the SCID-I diagnosis was affirmed by the previously mentioned questionnaires (see Table 1).

Procedure and Design

All participants were tested individually in a session lasting about 3−4 hr. First, individual tactile detection thresholds were measured. In a second step, participants were examined with the SSDT paradigm, and immediately afterward the threshold procedure was repeated a second time to test its reliability. Then, participants were asked to answer to the described questionnaires and, finally, we administered the SCID-I interview. All partici-

Table 1
Sample Characteristics, Symptoms, and Diagnoses

	SFD group (n = 33)		Control group (n = 32)		Test result
	M (SD)		M (SD)		Group comparison
SOMS-2	14.06 (8.35)		4.69 (4.18)		$t(63) = 5.68$, $p < .001$, $d = 1.38$
PHQ-15	11.57 (6.13)		3.81 (2.88)		$t(63) = 6.50$, $p < .001$, $d = 1.62$
Whiteley Index	3.73 (2.48)		1.28 (1.85)		$t(63) = 4.50$, $p < .001$, $d = 1.12$
STAI[a]	49.76 (11.91)		37.25 (10.37)		$t(63) = 4.51$, $p < .001$, $d = 1.12$
BDI-II	17.09 (11.76)		7.72 (10.72)		$t(63) = 3.36$, $p < .001$, $d = 0.83$
	N	%	N	%	
Any SFD[b]	33	100	0	0	
Somatization disorder	6	18.18	0	0	
Pain	12	36.36	0	0	
Undifferentiated somatoform	15	45.45	0	0	
Hypochondriasis[c]	1	3.03	0	0	
Current depression[b]	13	39.39	2	6.25	$\chi^2(1) = 10.05$, $p = .002$
Current anxiety disorder[b,d]	8	25.00	2	6.25	$\chi^2(1) = 4.04$, $p = .08$

Note. SOMS-2 = Screening for Somatoform Symptoms; PHQ = Patient Health Questionnaire; STAI = State-Trait Anxiety Inventory; BDI-II = Beck Depression Inventory; SFD = somatoform disorder.
[a] One control group participant had a missing STAI score, which was replaced by the mean of the scale. [b] According to the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition.* [c] Hypochcondriasis as comorbidity. [d] Number of persons suffering at least from one anxiety disorder.

pants were paid 20 Euros. In accordance with SCID-I diagnoses, participants were allocated to two groups. One group was comprised of participants with SFD and the other group—healthy controls—participants without SFD. The groups were balanced with respect to age, sex, and educational level. Participants were naive about the purpose of the study before having passed all stages of the study.

Statistical Analysis

A t test and chi-square tests were used to examine group differences in sociodemographic variables. Using signal detection theory (cf. Green & Swets, 1966), responses on the SSDT were classified as hits (participant reported the tactile signal on a vibration-present trial), misses (participant did not report the tactile signal on a vibration-present trial), false alarms (participant reported the tactile signal on a vibration-absent trial), and correct rejections (participant did not report the tactile signal on a vibration-absent trial) in order to calculate the parameters d' (sensitivity) and c (response bias). The responses on the 4-point category scale (*definitely yes*, *maybe yes*, *maybe no*, *definitely no*) were pooled into "yes" and "no" responses because not all participants had used more than two response categories. For this reason, further analyses of the rating responses (e.g., estimation of a receiver operating characteristic [ROC] curve) were impossible. For a subsample that used more than two response categories, the area under the ROC curve (A_z) was estimated. We used a maximum-likelihood procedure (Dorfman & Alf, 1969) for fitting a binormal model (Hanley, 1988). The area under the ROC curve (A_z) is an index for sensitivity requiring less restrictive assumptions than d' (cf. Swets, 1986) and could therefore be used to check the validity of the d' scores. All analyses of tactile thresholds are based on averaged scores of the two threshold assessments before and after administering the SSDT, respectively. Correlational analyses (Pearson correlations, two-tailed) were conducted in order to check for linear relationships between SSDT parameters and dimensional measures of somatoform symptoms, health anxiety, depression, and trait anxiety. Light-induced changes in SSDT parameters were captured by the difference scores of the parameter in question in the light-present versus the light-absent condition.

The log-linear correction (cf. Hautus, 1995) was applied when calculating hit and false-alarm rates, which were used to determine the signal detection theory statistics $d' = z(\text{hits}) - z(\text{false alarms})$ and $c = -0.5 \times [z(\text{hits}) + z(\text{false alarms})]$. The index d' represents the sensitivity to correctly detect the tactile stimulus. The tendency to report a tactile signal is estimated by the response bias parameter c (cf. Macmillan & Creelman, 1990). A value of $c = 0$ corresponds to unbiased responding, while negative values of c represent a tendency toward "yes, signal present" responses. Both estimates were calculated separately for light-present and light-absent trials.

The SSDT took approximately 45 minutes to administer. The four test blocks were aggregated into two test halves (Test Half 1, Test Half 2) in order to assess changes in response behavior during the course of the experiment.

According to our a priori hypotheses, the SSDT parameters (i.e., false-alarm rates, response bias, and sensitivity) were analyzed by a $2 \times 2 \times 2$ repeated-measures analysis of variance (ANOVA). The two within-subjects factors were test half (Test Half 1 vs. Test Half 2) and light stimulus (light present vs. light absent). The group variable (SFD group vs. control group) entered as a between-subjects factor. Post hoc tests (ANOVA and t tests) were conducted in case of significant interaction effects.

Additional analyses were conducted to demonstrate the specificity of the findings for SFD and to rule out a potential influence

of anxiety, hypochondriasis, and affective disorders. We used three approaches. First, scale values from questionnaire data (BDI-II, Whitley Index, and STAI) were included as covariates in ANOVAs. Second, the SCID-I diagnoses (anxiety and/or affective disorder absent or present) were included as dichotomous covariates. Third, we reanalyzed data after excluding participants with an anxiety or an affective disorder, according to our SCID interviews, resulting in two smaller subsamples ($N_{SFD} = 16$, $N_{CG} = 28$).

Because our previous study (Katzer et al., 2011) had shown that the response bias (c) and the false-alarm rate aggregated across the light-present condition and the light-absent condition correlated with measures of somatoform symptoms, we also analyzed averaged scores of the SSDT parameters, that is, response bias (c), sensitivity (d'), false alarms, and tactile sensitivity, each in the light-present and in the light-absent conditions. For all analyses, the significance level was set to $p = .05$ (two-tailed). Effect sizes are reported as partial η^2 in case of ANOVA results and as Cohen's d for t test results. The latter measure of effect size is based on means and variances of the d' and c parameters. Following Cohen (1992), $d \geq 0.30$ is considered as a small, $d \geq 0.50$ a medium, and $d \geq 0.80$ a large effect size. Partial η^2 (η_p^2) for ANOVA effects are interpreted according to the following conventions: $\eta_p^2 \geq 0.01$ is a small effect; $\eta_p^2 \geq 0.06$ is a medium effect; and $\eta_p^2 \geq 0.14$ is a large effect.

Results

Demographic and Clinical Data

Participants with SFD. According to the SCID-I, a diagnosis of an SFD could be confirmed in 33 participants (24 women, 9 men; $M_{age} = 43.42$ years, $SD = 9.87$; age range 21−56 years). Sixteen participants had at least 12 years of education, 11 had secondary school levels with at least 10 years of school, and five had at least nine years of education. One participant had no education at all.

We diagnosed a somatization disorder six times (*DSM–IV* code 300.81), an undifferentiated somatoform disorder 15 times (*DSM–IV* code 300.81), and a pain disorder associated with both psychological factors and medical conditions 12 times (*DSM–IV* code 307.89). Exactly 27.27% of this experimental group suffered from at least one comorbid anxiety disorder. Two had posttraumatic stress disorder (*DSM–IV* code 309.81), one had a panic disorder with agoraphobia (*DSM–IV* code 300.21), four had specific phobia (*DSM–IV* code 300.29), and three suffered from social phobia (*DSM–IV* code 300.23). Exactly 36.36% ($n = 12$) reported at least one present mood disorder. Three reported a recurrent but mild major depressive disorder (*DSM–IV* code 296.31); one reported a recurrent major depressive disorder in partial remission (*DSM–IV* code 296.35); four reported symptoms constituting a single episode of a major depressive disorder, among them one unspecified (*DSM–IV* code 296.20), one mild (*DSM–IV* code 296.21), and two moderate (*DSM–IV* code 296.22). A one-time dysthymic disorder (*DSM–IV* code 300.04) was diagnosed. Four reported recurrent major depressive disorder, in full remission (*DSM–IV* code 296.36).

Other comorbid disorders were found in 9.09% of the participants, including hypochondriasis (*DSM–IV* code 300.7), an eating disorder not otherwise specified (binge eating; *DSM–IV* code 307.5), and alcohol dependence in sustained full remission (*DSM–IV* code 303.90).

Control participants. Thirty-two persons without any somatoform diagnosis were matched to the SFD group by age, sex, and educational level. In the control group, age ranged from 22 to 63 years ($M_{age} = 41.72$ years, $SD = 11.53$). Twenty-two were woman (68.75%). Twenty-two had at least 12 years of education, six at least 10 school years, and four had at least nine years of education. Two subjects in this group fulfilled criteria for anxiety disorders (specific phobia, *DSM-IV* code 300.29 and social phobia, *DSM-IV* code 300.23). Two subjects of the control group had current mood disorders: We determined that one had a mild, recurrent major depressive disorder (*DSM–IV* code 296.31) and the other a single episode major depressive disorder in partial remission (*DSM–IV* code 296.25).

Regarding comorbidity, 48.5% of the SFD group did not exhibit any comorbid disorder and 87.5% of the control group did not fulfill *DSM–IV* criteria of any diagnosis. On average, the participants with SFD suffered significantly more often from a comorbid depression, $\chi^2(1) = 6.08$, $p = .01$, and also exhibited a stronger tendency toward anxiety disorders, $\chi^2(1) = 4.04$, $p = .08$, than participants in the control group.

The control group did not differ significantly in age, $t(63) = 0.64$; $p = .52$, $d = 0.16$, educational level, $\chi^2(1) = 2.75$, $p = .10$, or sex, $\chi^2(1) = 0.12$, $p = .72$, from the SFD group. The number of women and men were comparable: women comprised about 70% of both groups.

Table 1 illustrates the results of the diagnostic procedure and symptom measures as well as the results of group comparisons.

As intended, participants in both groups differed with respect to the degree and quantity of somatoform symptoms (SOMS-2, PHQ-15). Additionally, members of the SFD group had significantly higher health anxiety scores on the Whitley Index. Apart from the group-defining diagnose (SFD), current depression and anxiety disorders were more frequent in the SFD group than in the control group. The SFD group also showed higher levels of psychopathology, as measured by trait anxiety (STAI) and depression (BDI-II).

Results of the SSDT

Table 2 shows descriptive statistics for hit and false-alarm rates in light-absent and light-present trials for the 160 test trials, and it lists the signal detection theory statistics d' and c. Scores are displayed separately for the two test halves (each comprising 80 trials) and for the two groups (SFD and control).

Response bias (c). The average response bias c in the different conditions and groups is displayed separately for the two test halves in Figure 3. The visual stimulus had a significant effect on the response bias, $F(1, 63) = 19.85$, $p < .001$, $\eta^2 = .24$. The responses were generally more liberal in the light-present condition than in the light-absent condition. Thus, compatible with expectation, the light stimulus led to a tendency to report tactile sensations.

We found a main effect of test half, $F(1, 63) = 5.56$, $p = .02$, $\eta^2 = 0.81$, with a more liberal response criterion in the first test half. Moreover, we observed a significant three-way interaction of the factors group, test half, and light stimulus, $F(1, 63) = 5.43$, $p < .023$, $\eta^2 = .08$. The other interactions were not significant,

Table 2
Means (and Standard Deviations) for Hit and False-Alarm Rates, Sensitivity, and Response Bias in the Light-Present and Light-Absent Trials for the SFD and Control Groups, for the First and the Second Half of the Experiment

	SFD group (n = 33)				Control group (n = 32)			
	Light-present		Light-absent		Light-present		Light-absent	
SSDT parameter	Half 1	Half 2	Half 1	Half 2	Half 1	Half 2	Half 1	Half 2
Hit rate (%)	68.59 (19.62)	65.04 (24.17)	60.80 (21.51)	52.03 (23.76)	61.59 (21.98)	59.60 (22.36)	48.63 (22.63)	49.09 (25.03)
False-alarm rate (%)	14.60 (12.60)	10.72 (9.08)	13.62 (11.78)	11.75 (10.30)	14.18 (10.50)	11.74 (9.71)	11.43 (10.67)	12.04 (10.25)
Sensitivity (d')	1.83 (0.91)	1.87 (0.83)	1.61 (0.90)	1.41 (0.96)	1.55 (0.87)	1.60 (0.93)	1.34 (1.01)	1.29 (1.01)
Response bias (c)	0.32 (0.45)	0.45 (0.50)	0.46 (0.44)	0.66 (0.41)	0.42 (0.38)	0.53 (0.45)	0.71 (0.37)	0.67 (0.43)

Note. SFD = somatoform disorders.

$ps > .12$. The group variable did not produce a significant difference in response bias, $F(1, 63) = 1.71$, $p = .20$, $\eta^2 = .03$.

Because the analyses demonstrated specificities in the first test half, we conducted a post hoc ANOVA, which showed a significant main effect of the light stimulus in the first test half, $F(1, 63) = 21.63$, $p < .001$, $\eta^2 = .26$. As Figure 3 depicts, the SFD group showed a more liberal response criterion than the control group, indicated by smaller values of c. This group effect just failed to reach significance, $F(1, 63) = 3.82$, $p = .055$, $\eta^2 = .06$. The interaction between light condition and group was not significant, $F(1, 63) = 2.31$, $p = .13$, $\eta^2 = .04$. Nevertheless, post hoc tests showed that the SFD group compared to the control group had a significantly more liberal response style in the first test half in the light-absent condition, $t(63) = 2.48$, $p = .02$, $d = 0.62$.

To test the specificity of the effects for somatoform disorders, we repeated the analysis of the response bias c by including individual levels of depression (BDI-II), trait anxiety (STAI), and health anxiety (Whitley Index) as covariates (following the procedure described by Delaney & Maxwell, 1981). A slightly attenuated but still significant three-way interaction of the factors group, test half, and light stimulus was observed, $F(1, 60) = 4.35$, $p = .041$, $\eta^2 = .07$. A post hoc ANOVA on the c scores of the first test half revealed an even stronger difference between the SFD group and control group, $F(1, 60) = 6.77$, $p = .012$, $\eta^2 = .10$, indicating that even after statistically controlling for individual levels of depression, trait anxiety, and health anxiety, the SFD group showed a significantly more liberal response behavior in the SSDT. An analogous pattern of results emerged when using dichotomous SCID-I diagnoses of comorbid affective and anxiety disorders as covariates—the three-way interaction of the factors group, test block, and light stimulus remained constant, $F(1, 61) = 5.02$, $p = .029$, $\eta^2 = .08$, the corresponding post hoc ANOVA of the first test block revealed a significant difference between the SFD group and the control group in terms of more liberal responses in the SFD group, $F(1, 61) = 5.06$, $p = .028$, $\eta^2 = .08$. Finally, excluding participants with a diagnosis of an anxiety or affective disorder (SFD group, $n = 17$; control group, $n = 4$) from the analysis resulted in the same pattern of findings: A comparison of the response bias c for the remaining participants (SFD group $n = 16$; control group $n = 28$) revealed a significantly more liberal response bias in the SFD group compared with the control group, $t(30.95) = 2.26$, $p = .03$, $d = 0.62$ (t test for unequal variances,

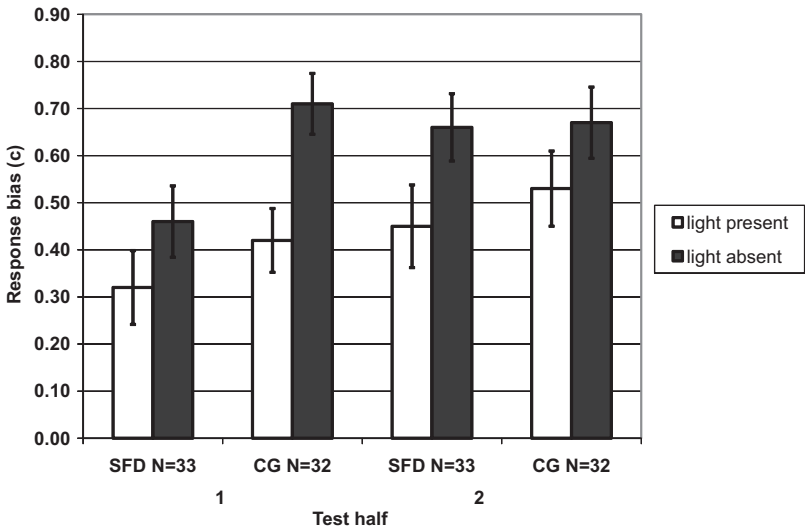

Figure 3. Mean response bias (c) for the participants with SFD and the control participants in the two test halves of the SSDT. Error bars represent ± 1 standard error of the mean. CG = control group.

two-sided). Further t tests of the c scores for each test block revealed that this main effect was moderated by the test block: We observed a significant difference with a large effect size between the SFD and the control groups in the first test block, $t(33{,}88) = 3.34$, $p = .002$, $d = 1.02$, that disappeared in the second block, $t(27.63) = 0.82$, $p = .42$, $d = 0.27$.

Tactile sensitivity (d'). Tactile sensitivity was significantly higher in trials with a light stimulus than in light-absent trials, $F(1, 63) = 24.56$, $p < .01$, $\eta^2 = .28$. There was no significant group effect, $F(1, 63) = 1.45$, $p = .23$, $\eta^2 = 0.02$. This result was expected because both groups were tested with vibration intensities individually selected to correspond to about 71% of correct responses in a two-interval task (Levitt, 1971). Neither the effect of the test half nor any interaction was significant, ps $> .12$.

Originally, we planned to use ROC analyses to examine tactile sensitivity. Because not all participants had used more than two response alternatives, A_Z scores as an index of sensitivity could not be calculated for all participants. Because A_Z scores are considered to be methodologically preferable as compared to d' scores (e.g., Verde, Macmillan, & Rotello, 2006), we determined this index of sensitivity for the light-absent ($M_{AZ} = 0.70$, $SD_{AZ} = 0.15$) and light-present trials ($M_{AZ} = 0.80$, $SD_{AZ} = 0.14$), for all participants who had used at least three response categories ($N = 50$, SFD group $n = 26$; control group $n = 24$). As relates to tactile sensitivity scores d', A_Z scores were affected by a significant light effect, $F(1, 40) = 15.90$, $p < .001$, $\eta^2 = 0.28$. Neither the group variable, $F(1, 40) = 2.93$, $p = .10$, $\eta^2 = 0.07$, nor the interaction effect of the light stimulus and the group variable, $F(1, 40) = 0.84$, $p = .36$, $\eta^2 = 0.02$, led to significant effects on A_Z scores. A_Z scores neither differed in light-present, $t(48) = 1.12$, $p = .27$, nor in light-absent trials, $t(44) = 1.49$, $p = .14$, between the experimental groups. Pearson correlations between the A_Z scores and the d' scores demonstrated good validity of the d' scores (light-present condition: $r = .86$, $p < .01$; $N = 50$, SFD group $n = 24$, control group $n = 26$; light-absent condition: $r = .84$, $p < .01$; $N = 46$, SFD group $n = 24$, control group $n = 22$).

To test the robustness of the reported effects in light of possible influences of anxiety and depression, we repeated the analysis of the sensitivity score d' by including individual levels of depression (BDI-II), trait anxiety (STAI), and health anxiety (Whitley Index) as covariates. None of the main or interaction effects involving the group factor were significant, ps $> .12$. When controlling for the presence of anxiety and affective disorders by including the dichotomous variables as covariates, the same pattern of results emerged and there was no evidence of a significant difference between experimental groups. Excluding 21 participants (SFD group $n = 17$, control group $n = 4$) with a comorbid anxiety or depressive disorder from the analysis did not change the results, that is, no significant group differences between the SFD and control groups were observed for any of the d' values, ps $> .10$.

False alarms. False alarms occurred significantly more frequently in the first test half ($M = 13.47$, $SD = 10.03$) than in the second test half ($M = 11.56$, $SD = 8.92$), $t(64) = 2.15$, $p = .04$, $\eta^2 = 0.07$, $d = 0.20$. The presence of the concurrent light did not change the rate of false alarms, $F(1, 63) = 0.42$, $p = .52$, $\eta^2 = 0.01$. The false alarm rate was only 0.6% higher in the light-present condition. The interaction between test half and light stimulus was not significant, $F(1, 63) = 3.01$, $p = .09$, $\eta^2 = 0.05$, but revealed a tendency toward a higher impact on the number of false alarms in the first test half in the light-present condition. Neither the group variable, $F(1, 63) = 0.02$, $p = .88$, $\eta^2 < 0.01$, nor the interaction between the light stimulus and the group variable, $F(1, 63) = 0.46$, $p = .50$, $\eta^2 < 0.01$, nor the remaining interactions were significant, ps $> .05$.

To control for the possible effects of anxiety, depression, health anxiety, and comorbid mental disorders, we repeated the analysis of false alarms by including dimensional measures of depression (BDI-II), trait anxiety (STAI), and health anxiety (Whitley Index) as covariates. An analog pattern of results emerged and, most importantly, the SFD and control groups did not differ in their false-alarm rates, $F(1, 60) = 0.07$, $p = .79$, $\eta^2 < 0.01$. Similarly, including dichotomous variables of comorbid anxiety and depression diagnoses as covariates, group effect: $F(1, 60) < 0.01$, $p = .99$, $\eta^2 < 0.01$, or excluding participants with a comorbid anxiety or depressive disorder (remaining $n = 44$, SFD group $n = 16$, control group $n = 28$) did not significantly change the pattern of results.

Tactile detection threshold. The test-retest correlation between the first measurement of the tactile threshold ($M = 47.99$, $SD = 21.51$) and the second measurement ($M = 53.02$, $SD = 22.17$) was $r_{tt} = .86$ (SFD group $r_{tt} = .82$, control group $r_{tt} = .88$), indicating that the tactile detection threshold was determined reliably in both groups. On average, the detection threshold for the SFD group ($M = 45.06$, $SD = 21.42$) was significantly lower than for the control group ($M = 56.12$, $SD = 19.49$), $t(63) = 2.17$, $p = .03$, $d = 0.54$. To test for a possible difference between the first and second threshold assessment, we conducted a Group (SFD group vs. control group) $\times$ Time (first assessment vs. second assessment) repeated-measures ANOVA. Results revealed a main effect for group, $F(1, 63) = 4.73$, $p = .03$, $\eta^2 = 0.07$, indicating a lower detection threshold in the SFD group compared with the control group, and a significant effect of assessment time, $F(1, 63) = 12.45$, $p = .001$, $\eta^2 = 0.17$. The increase in tactile thresholds over the course of the experimental session (lasting about 60 min including tactile threshold assessment and the SSDT) was unexpected and may indicate that fatigue played a role. The effect of assessment time was not further moderated by the group variable, $F(1, 63) = 0.01$, $p = .92$, $\eta^2 < 0.01$, indicating that the increase in detection thresholds did not differ between the two groups. To control for the possible confounding influences of anxiety and depression on tactile detection thresholds, we repeated the ANOVA and added individual levels of depression (BDI-II), trait anxiety (STAI), and health anxiety (Whitley Index) as covariates. The main effect of group remained significant, $F(1, 60) = 7.73$, $p = .007$, $\eta^2 = 0.11$, suggesting that the lower detection threshold of the SFD group was not attributable to individual levels of depression, anxiety, or health anxiety. Repeating this ANOVA with the corresponding dichotomous variables comorbid anxiety and depressive disorders revealed an even stronger main effect for group, $F(1, 60) = 10.52$, $p = .002$, $\eta^2 = 0.15$. Additionally, in this analysis, the diagnosis of an anxiety disorder was significantly associated with higher perception thresholds, $F(1, 60) = 4.19$, $p = .045$, $\eta^2 = 0.06$, and a trend for an analog association was also revealed for depression, $F(1, 60) = 2.80$, $p = .099$, $\eta^2 = 0.04$, suggesting that anxiety and depression might have opposing effects on detection thresholds compared to somatoform disorders. Finally, excluding participants with a comorbid anxiety or affective disorder still revealed a large difference be-

tween groups for both threshold assessments—assessment 1: $t(31.83) = 3.07$, $p < .01$, $d = 0.95$; assessment 2: $t(29.70) = 2.65$, $p < .01$, $d = 0.84$—suggesting lower detection thresholds in patients with an SFD compared with the subjects in the control group.

Correlational analyses. To explore possible linear associations between the different experimental parameters of the SSDT and the dimensional measures of certain symptom types and symptom severity in greater detail in the total sample as well as in the SFD group and in the control group, we computed zero-order correlations. Table 3 contains the Pearson correlation coefficients between the questionnaire-based measures (SOMS-2 total and subscales scores, PHQ-15, BDI-II, STAI, Whitley Index) with the relevant SSDT parameters (*c*, *d′*, false alarms, detection thresholds) for the two test halves (Test Half 1 vs. Test Half 2). In general, no consistent pattern of associations could be found for most of the somatoform symptom measures (SOMS-2 and PHQ-15) or the SSDT parameters, in the total sample or in the SFD group or the control group. For exploratory reasons, we computed correlations not only for the SOMS-2 total score but also for the different SOMS-2 subscales. Within the SFD group but not the control group, nonsignificant but medium-sized positive associa-

Table 3
Correlations Between SSDT Parameters and Measures of Psychopathological Features of SFD, Depression, and Anxiety

	SOMS-2	SOMS-P	SOMS-N	SOMS-V	SOMS-G	PHQ-15	BDI-2	STAI	WI
Total ($N = 65$)									
FA-1[a]	.18	.18	.27*	.06	.17	.02	.04	.01	.17
FA-2[b]	.14	.10	.22	−.00	.16	.06	.02	.03	.09
ΔFA-1	.01	−.15	.14	.11	−.02	−.06	−.04	−.06	.04
ΔFA-2	.09	−.11	.26*	.12	.04	.08	−.00	−.06	.05
c-1	−.09	−.04	−.17	−.12	−.02	−.02	.10	.14	−.09
c-2	−.13	−.02	−.22	−.17	−.05	−.05	.07	−.07	−.17
Δ*c*-1	−.15	−.29*	.08	.02	−.23	−.17	−.14	−.15	−.11
Δ*c*-2	.12	−.03	.29*	.13	.05	.11	.02	−.05	.04
d′-1	−.04	−.10	−.06	.13	−.12	.06	−.10	−.10	−.06
d′-2	.05	−.01	.03	.24	−.05	.05	−.05	.04	.08
d′-diff-1	−.05	.00	.02	−.05	−.10	.01	.01	.02	−.08
d′-diff-2	−.02	−.01	−.07	−.04	.04	.02	.01	.01	−.03
Tactile threshold	−.04	−.12	.15	.13	−.18	.14	.08	.04	.09
SFD group ($n = 33$)									
FA-1	.28	.25	.36*	.08	.25	.00	.04	.08	.30
FA-2	.31	.30	.28	.02	.33	.16	.11	.20	.23
ΔFA-1	.15	−.10	.21	.22	.15	.01	−.02	−.11	.08
ΔFA-2	.27	−.02	.49**	.23	.13	.11	.02	−.11	.18
c-1	−.05	−.01	−.06	−.05	−.06	.19	.38*	.31	−.06
c-2	−.22	−.23	−.21	−.11	−.18	−.08	.25	.11	−.15
Δ*c*-1	−.03	−.30	.22	.19	−.14	−.13	−.17	−.20	−.09
Δ*c*-2	.09	−.12	.35*	.07	−.03	−.01	−.13	−.22	−.03
d′-1	−.15	−.19	−.22	.12	−.18	−.03	−.24	−.28	−.19
d′-2	.01	.00	−.02	.20	−.09	.03	−.22	−.22	−.02
Δ*d′*-1	−.13	−.11	.06	−.05	−.27	−.08	−.05	.08	−.14
Δ*d′*-2	−.35*	−.27	−.33	−.39*	−.16	−.22	−.27	−.20	−.32
Tactile threshold	.19	.12	.31	.32	−.03	.49**	.39*	.27	.37*
Control group ($n = 32$)									
FA-1	−.04	.04	.06	−.07	−.09	−.13	−.02	−.14	−.06
FA-2	.01	−.09	.34	.01	−.10	.02	−.05	−.14	−.00
ΔFA-1	−.13	−.18	.18	.07	−.24	−.07	.02	.13	.12
ΔFA-2	−.06	−.23	−.03	.06	.02	.24	.01	.01	−.04
c-1	.33	.33	−.15	.10	.38*	.21	.01	.28	.16
c-2	.10	.40*	−.38*	−.26	.24	.11	−.08	−.25	−.17
Δ*c*-1	−.12	−.07	.09	.00	−.22	.14	.10	.14	.10
Δ*c*-2	.12	−.05	.16	.16	.08	.30	.13	.03	.05
d′-1	−.25	−.31	−.04	−.04	−.24	−.13	−.11	−.13	−.13
d′-2	−.07	−.23	−.04	.25	−.11	−.13	.01	.23	.10
Δ*d′*-1	.10	.20	−.11	−.08	.13	.26	.09	−.04	−.02
Δ*d′*-2	.27	.11	.27	.26	.17	.17	.16	.06	.14
Tactile threshold	.07	−.10	.44*	.31	−.18	.28	−.04	.07	.09

Note. SFD = somatoform disorder; SOMS-2 = Screening for Somatoform Symptoms; SOMS-P = Screening for Somatoform Symptoms Pain scale; SOMS-N = Screening for Somatoform Symptoms Pseudoneurological scale; SOMS-V = Screening for Somatoform Symptoms Vegetative scale; SOMS-G = Screening for Somatoform Symptoms Gastrointestinal scale; PHQ = Patient Health Questionnaire; BDI-II = Beck Depression Inventory; STAI = State-Trait Anxiety Inventory; WI = Whiteley Index; FA = false-alarm rate; *c* = response bias; *d′* =tactile sensitivity; Δ = differences of the SSDT parameters between the light-absent and the light-present condition.

[a] 1 = averaged scores of the first test half. [b] 2 = averaged scores of the second test half. [c] One control group participant had a missing STAI value for one item, which was replaced by the mean of the scale.

* $p < .05$. ** $p < .01$.

tions between the SOMS-2 total score and the false-alarm rates (first test half $r = .28$, second test half $r = .31$) were observed, that is, patients with more medically unexplained symptoms reported more false alarms in the SSDT. For the SOMS-2 subscale assessing pseudoneurological symptoms (i.e., the subscale that shows the closest relation to functional somatization), we observed a significant positive correlation with the number of false alarms in the SSDT for the total sample, block 1: $r = .30$, $p = .02$; block 2: $r = .32$, $p < .01$. Moreover, the increase in false alarms, $r = .26$, $p = .04$, as well as a more liberal response bias, $r = .29$, $p = .02$, induced by the light stimulus (in the second test block) was positively associated with the number of self-reported pseudoneurological symptoms. It is interesting that these positive associations of pseudoneurological symptoms with SSDT parameters were only observable in the SFD group (false alarms: $r = .36$; light-modulated false alarms, i.e., the difference in the false-alarm rate between the light-present and the light-absent conditions: $r = .49$; light-modulated c: $r = .35$) but not in the control group (false alarms: $r = .06$; light-modulated false alarms: $r = -.03$; light-modulated c: $r = -.38$), suggesting that the positive associations in the total sample were mainly attributable to associations in the SFD group.

With respect to the tactile detection thresholds, for the SFD group we found significant positive correlations with the PHQ-15 score (threshold 1 $r = .51$, threshold 2 $r = .45$) as well as with the BDI-II scores (threshold 1 $r = .36$, threshold 2 $r = .40$), and Whitley Index scores (threshold 1 $r = .29$, threshold 2 $r = .38$), but not with any SOMS-2 scores. In the total sample and in the control group, none of these correlations were significant. This finding suggests that, in the SFD group, the detection threshold increased with increasing levels of somatic symptom distress (PHQ-15), depression (BDI-II), and health anxiety. This finding at first glance seems to contradict the group comparison analysis in which a significant lower detection threshold could be observed in the SFD group compared to the control group.

Regarding the d' parameter, no consistent pattern of results emerged: We only observed significant negative correlations in the second test block between difference scores of sensitivity in the light-present minus light-absent condition and the SOMS-2 total score, $r = -.35$, $p < .05$, as well as SOMS-2 vegetative scores, $r = -.39$, $p = .02$. This suggests that the gain in sensitivity induced by the light stimulus decreases with the number of reported symptoms. This correlation was not affirmed by PHQ-15 scores and could not be found in the other subsample or total sample.

Discussion

Based on cognitive models of symptom formation in individuals with SFD, the primary aims of the current study were to examine whether individuals with SFD are prone to overreport tactile perceptions and whether individuals with SFD differ from healthy controls in terms of their detection threshold for nonpainful tactile stimuli. To our knowledge, this study applied the SSDT (Lloyd et al., 2008) for the first time in individuals suffering from an SFD according to *DSM–IV*. In line with our hypotheses, participants with SFD exhibited more liberal response criteria (i.e., a tendency toward reporting tactile stimulation) in the SSDT. Unexpectedly, this effect was limited to the first half of the experiment. Furthermore, participants with SFD had significantly lower detection thresholds for nonpainful tactile stimuli compared to the control group.

The correlational analyses suggest a positive medium-sized association between the number of self-reported MUS and the number of illusory tactile sensations reported during the SSDT, especially within the sample of patients with SFD. The fact that these associations were most evident in case of the Pseudoneurological scale of the SOMS-2 is in line with previous studies that also found associations between the experience of illusory tactile sensations in the SSDT and somatoform dissociative symptoms (Brown et al., 2010).

However, as revealed by the lack of a stronger pattern of correlations between symptom measures and SSDT parameters, the level of self-reported somatoform symptoms (as measured by the SOMS-2 and the PHQ-15) could not entirely explain the group differences between the participants with SFD and the controls. This suggests that factors associated with the diagnosis of a SFD not included in dimensional somatoform symptom measures might be important to the findings in the SSDT. We can only speculate about the exact nature of these factors. Possibly, they include a clinically significant level of distress and impairment and factors such as symptom chronicity, which are typically not well assessed in the questionnaire-based measures. Additionally, the current diagnosis of a somatoform symptom according to *DSM–IV* involves the clinical evaluation of medical explanations for a given symptom. It appears likely that in questionnaires like the SOMS-2 and the PHQ-15 the symptom score might be confounded by medically explained body symptoms. Specifically, the PHQ-15 does not distinguish between medically explained and unexplained symptoms.

In the following, we will first discuss the findings on the SSDT in more detail and will then comment on the results in terms of the proposed theoretical models of SFD.

SSDT Results

As compared to the control group, the SFD group exhibited a more liberal response style in the first half of the SSDT (especially in trials without an additional light stimulus). In other words, participants with SFD more frequently reported tactile sensations than those in the control group. According to the analyses of covariance, this effect was not influenced by anxiety or depression. Consequently, it seems specific for the functional somatic symptom genesis in SFD and can be distinguished from more general types of somatization that are also prevalent in hypochondriasis or affective and anxiety disorders (Kirmayer & Robbins, 1991).

In the second test half, the members of the SFD group adopted a response style comparable to that of those in the control group. A speculative explanation would be that the behavior of the participants with SFD in the first part of the experiment represents a more typical and spontaneous response behavior to novel tactile stimuli (i.e., overreporting tactile perceptions). This spontaneous behavior may rely on top-down processing due to existing cognitive schemata specific for patients with SFD (Brown, 2004). The normalization of the response behavior in the second test half (e.g., due to habituation to the experimental setting and the type of tactile stimuli applied) might indicate that participants in the SFD group learn to focus more on external somatosensory stimulus

properties and to inhibit possible overly liberal response tendencies that are schema driven. In terms of the filter model of Rief and Barsky (2005), the effect might be interpreted as a more restrictive calibration of the filter on the tactile stimuli applied in the SSDT. The result concerning a more liberal response criterion in participants with SFD corroborates the findings of our previous study (Katzer et al., 2011). In this previous study (Katzer et al., 2011), the level of response bias liberalization was significantly associated with the level of somatoform symptoms in a college student population. The result is also in line with Brown et al. (2010), who reported that individuals with high scores for pseudoneurological symptoms are characterized particularly by a more liberal response bias. Given that pseudoneurological symptoms might represent a special marker of functional somatization (Brown, 2004), the observed correlations between SSDT parameters and the Pseudoneurological scale of the SOMS-2 are worth a closer look. Pseudoneurological symptoms were related to light-induced changes in response bias as well as to changes in the false-alarm rate in the second test half in the SFD group. Furthermore, in the first test half, a relation between false-alarm rates and pseudoneurological symptoms was found. This result is in line with findings by Brown et al. (2010), who concluded that " [. . .] somatoform dissociators appear more likely to experience illusory perceptual events under conditions of sensory ambiguity than nondissociators, despite comparable perceptual abilities more generally" (p. 1).

Concerning tactile thresholds assessed before and after the SSDT, two aspects seem noteworthy. First, even when controlling for anxiety, negative affectivity, and health anxiety, tactile thresholds were lower in patients with SFD than in the control subjects. Second, we observed that tactile thresholds increased in both groups during the course of the experiment. Given the duration of the experiment framed by the tactile threshold measurements, we suppose that signs of fatigue occurred after 60 min of concentration on a monotonous task.

In general, we registered a cross-modal augmentation of tactile sensitivity (d') when comparing light-absent and light-present trials. In light-present trials, participants of both groups detected the tactile stimulus more reliably. This result is also in line with Katzer et al. (2011). Other studies have also found tendencies toward an amelioration of tactile sensitivity in the light-present condition (e.g., Ro et al., 2004). On average, in the first test half more false alarms (i.e., illusory perceptions of the tactile stimuli) were registered than in the second test half, whereas the expected effect of the light stimulus on the false-alarm rate was not found. As in Katzer et al. (2011), a significant increase in false alarms in the light-present condition without an actual tactile stimulation could not be registered, although other studies found such a manipulation of illusory tactile perceptions (e.g., Brown et al., 2010; Lloyd et al., 2008). More important, the rate of false alarms was independent of group membership. Nevertheless, the correlations between the two false-alarm rate variables and the total SOMS-2 score reached a medium-sized association in the group with SFD without being statistically significant. Whereas a shared variance of approximately 9% is certainly not insubstantial, one cannot be certain whether a lack of statistical power, a heterogeneous sample, or the nonexistence of the effect is responsible for the lack of significance. Given these methodological limitations, it is not possible to rule out that high-symptom reporters with SFD show a tendency to report more false alarms, that is, illusory tactile perceptions.

What could be the origin of the observed effects of the visual stimulus in the SSDT? As pointed out in the introduction, Lloyd et al. (2008) suggested that the mechanism behind the cross-modal induction of the false perception of touch may arise from the activation of tactile representations in memory by the light. Arguably, this process is similar to that responsible for MUS, according to Brown's model (2004). In more general terms, the SSDT paradigm allows to examine whether a stimulus in one sensory modality influences the perception or the response to a stimulus presented in a different modality (cross-modal processing, cf. Spence, Senkowski, & Röder, 2009). Previous research on cross-modal processing showed that it is important to distinguish between cross-modal effects on sensitivity and on response bias. For example, visual stimulation often results in observers adopting a more liberal response style in an auditory detection task (i.e., respond more frequently than the auditory signal was presented). However, visual stimulation does not improve in all cases the sensitivity for detecting the auditory signal (e.g., Bothe & Marks, 1970). Furthermore, the probability for observing a cross-modal effect is higher if the two stimuli are temporally and spatially coincident, arguably because cross-modal processing is relevant for object detection (cf. Schnupp, Dawe, & Pollack, 2005). Alternatively, a visual stimulus may reduce temporal uncertainty when presented in synchrony with the tactile stimulus (Heron, Whitaker, & McGraw, 2004; Egan, Schulman, & Greenberg, 1961).

It has been argued that cross-modal effects could be due to effects involving neural responses at relatively early processing stages (e.g., Stein, Stanford, Ramachandran, Perrault, & Rowland, 2009). Indeed, there is evidence for direct links between cross-modal effects on behavior (e.g., on the sensitivity in a signal detection task) and the responses of multimodal neurons (e.g., Stein, Huneycutt, & Meredith, 1988). These sensory effects might result in a true increase in sensitivity (when controlling for changes in response bias). The classical example is multisensory stimulation resulting in a neural response that is stronger than the response to the individual modality-specific stimulus (Stein et al., 2009). Alternatively, or in addition, mechanisms acting at later processing stages may also result in a benefit in sensitivity. These mechanisms could, for example, represent cognitive effects of changes in information processing such as the effects of selective attention or the activation of memory traces. The improvement in performance by reducing spatiotemporal uncertainty (Lippert, Logothetis, & Kayser, 2007) is a good example for mechanisms acting at a later processing stage. Note that the frequent effects of multimodal stimulation on response bias rather than on sensitivity are more easily explained by mechanisms involving relatively late decisional processing stages (e.g., Marks, Ben-Artzi, & Lakatos, 2003). The results from the present study demonstrate an effect of the task-irrelevant visual stimulus on both tactile sensitivity, and an even stronger effect on response bias. This pattern is compatible with cross-modal effects involving early sensory, and later attentional and memory selection processes (compare Koelewijn, Bronkhorst, & Theeuwes, 2010).

Compatibility of the Findings With Existing Models of SFD and MUS

Rief and Barsky (2005) have argued that the cortical perception of bodily signals is perpetuated by a decreased activity of filter

systems. A liberal response bias may be one of the constituting elements of such dysfunctional filters besides selective attention, lack of distraction, and other mechanisms. The initial tendency toward a more liberal response bias (in the first test half) suggests that people with SFD might be more likely to expect the occurrence of an attended event. Note that phasic arousal cues are ubiquitous (compare Andor, Gerlach, & Rist, 2008) and may thus serve as the basis for the perception of bodily symptoms, especially in sensitive individuals. In our SSDT experiment, this tendency seemed to adapt to a normal level over the course of the experiment.

In the light of Brown's (2004) integrative model of MUS, it would seem that the generation of symptom perception cannot be reduced to cross-modal perceptual phenomena. There may be certain behavioral dispositions that are characteristic, but it seems difficult to generate specific symptom perceptions in laboratory settings without the additional input of affective material concerning complaints or cognitive contents relevant for the disorder. Moreover, the results of the first test half suggest that the proposed illusory impact of the light stimulus compared to the light-absent condition (in terms of a liberalization of responses reflected in lower *c* scores) was actually stronger in the control group than in the group with SFD. It is tempting to speculate that individuals in the SFD group did not need any further trigger to perceive and overreport weak somatic sensations, because this tendency already reflects their sensory "default" mode, which might be driven by preexisting symptom schemata.

To our knowledge, SSDT studies on participants with SFD have not been published yet. Existing studies using the SSDT were conducted with subclinical high-symptom reporters or healthy samples with low variance in somatoform symptoms (e.g., Brown et al., 2010; Katzer et al., 2011). These studies showed that there might be an interrelation of illusory tactile perceptions, that is, false alarms registered within the paradigm, and the degree of somatoform symptoms (Brown et al., 2010; Katzer et al., 2011). In the clinical SFD sample tested in the present study, this result could not be confirmed.

A higher false-alarm rate in the SSDT induced by the light stimulus would imply that people with SFD are more prone to suggestion. However, Brown, Schrag, Krishnamoorthy, and Trimble (2008) have already shown that high suggestibility is not necessarily a feature of SFD. The present results are in line with these negative findings. Thus, the study does not support the notion of Brown's model (2004) that distorted symptom perception is based on contextual cues that automatically activate emotional memory representations of previous symptom episodes. Nevertheless, the described behavioral disposition of a general liberal response bias beyond emotional processing may constitute an essential condition of the process of symptom formation as described by Brown (2004).

Study Limitations

Further studies are needed to verify that our results based on weak and nonpainful tactile perception are applicable to other contexts that are more symptom-oriented. At present, it is not possible to completely rule out that apart from SFD, depression, anxiety, or associated disorders influence response bias liberalization and the reduction of the tactile threshold. It would be desirable to include additional clinical control groups (i.e., depression, anxiety) and individuals with medical conditions (i.e., bodily illness) in future studies. Another potential problem is the heterogeneity of the SFD sample. By conducting a study that takes into account that SFD are characterized by a diversity of symptoms like pain, gastrointestinal symptoms, dissociations, and other symptoms, and that SFD are differentiable in terms of symptom chronicity, one could increase the specificity of the results in the SSDT task.

Future Directions

Other features such as symptom-related concerns or negative affectivity that likely play a major role in the development of somatoform symptoms should be taken into account in future studies in order to learn more about which interactions between perceptual styles and emotional contents are relevant for symptom formation and perpetuation. Possibly, the observed normalization of the overly liberal response bias only operates in a neutral or positive environment and not when health concerns overwhelm people with SFD. This is why we encourage SSDT studies in different affective contexts, for example, characterized by negative affectivity or health anxiety versus neutral contexts.

Nevertheless, it seems important to note that the described dispositional response style appears as a perceptual mode independent of affective information processing. As such, it could be regarded as a behavioral disposition. Such a response style could act as a relevant factor in the development and maintenance of somatoform symptoms that interact with emotional factors in the sense of Brown's cognitive theory, where for example negative affectivity and disease-confirming information are regarded as moderating and facilitating factors in the development of symptom perception.

We believe that response dispositions, as well as cross-modal effects on the response behavior, are partly responsible for the maintenance and for the triggering of MUS. It seems plausible that, as is the case with altered affective processing, general perceptual processes or dispositions are significantly altered in SFD. These perceptual dispositions involve a liberal response bias concerning symptom perception as well as low perception thresholds. The described processes may be seen in the light of Brown's cognitive model of MUS, but can also be interpreted within the signal filter model of Rief and Barsky (2005). In the latter, the response bias liberalization can be understood as a reduction in the filter function, resulting in an increase in somatoform perceptions. In most cognitive–behavioral models, somatization is primarily understood as the result of a complex interaction of negative affectivity, increased symptom-focused attention (Lim & Kim, 2005; Witthöft, Gerlach, & Bailer, 2006), negative interpretation of body sensations (Witthöft, Basfeld, Steinhoff, & Gerlach, 2011), and symptom amplification. We tentatively add the notion that general perceptual dispositions are also relevant for the development and maintenance of this disabling disorder.

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Received April 21, 2011
Revision received October 17, 2011
Accepted October 18, 2011 ■