Attentional Bias, Memory Bias, and Symptom Attribution in Idiopathic Environmental Intolerance and Classical Somatoform Disorders

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1 INTRODUCTION

According to the observations of professionals working in the domain of health care, there is an increasing discrepancy in the industrialized countries between the constantly improving health status and the subjective judgments of one's physical health and well-being, which are actually decreasing. This phenomenon termed as the "paradox of health" in an article by Barsky (1988) is partly reflected in the rising of so called medically unexplained symptoms, that is, subjective complaints for which no adequate cause can be found in medical examinations or standard laboratory tests. Although such mostly unfounded, short-living, and fully reversible symptoms like headache, musculoskeletal pain, fatigue, dizziness, or gastrointestinal problems are extremely frequent in the general population (e.g., Erisen & Ursin, 2004) some people suffer constantly and overproportionally from these conditions and display a high usage of the health care systems, often without a satisfactory outcome for both sides, patients and health-care professionals. Since clear-cut biomedical causal explanations for theses conditions are typically lacking, researchers from various disciplines of psychology and medicine have begun to broaden the bandwidth of relevant explanatory constructs by considering both psychological and psychobiological processes. Interdisciplinary evidence is growing that medically un- or under-explained symptoms represent complex conditions for which an old-fashioned dualistic conception of mind and body is no longer appropriate. Research in the domains of behavioral medicine and neuropsychology have demonstrated convincingly that perceptual processes or emotional states all have biological correlates, e.g., in the sense of altered blood-oxygenation levels as proxies for neural activation patterns and biochemical alterations in endocrine processes. Thus the traditional dichotomy of mental and organic disorders and illnesses is blurred by new experimental findings and even one of the most prototypical anxiety disorders like spider phobia is marked by biological correlates (e.g., an increase in neural activation in parts of the limbic system during confrontation with a real or virtual spider). On a general level, one can conclude that the way in which we *perceive* and *interpret* our environment directly manifests in neurobiological changes to the central and peripheral nervous system and that these changes in turn form feed-back loops and thereby influence perceptual and interpretative processes.

Adopting a cognitive-psychological point of view, *perception* is an active, constructive, and interpretative process guided both by objective physical or biological characteristics of the referring stimulus (bottom-up) and by prior formed knowledge, beliefs, and expectancies (top-down). This view holds not only for the perception of external stimuli,

but also for internal perceptual sensations (e.g., Pennebaker, 1982) as changes in skin temperature, heart rate, or nasal congestion (Pennebaker & Skelton, 1981). A perceptual-cognitive approach to symptom perception sharply contrasts with the traditional biomedical model that implies cause and effect relations between symptoms and an underlying biological or medical cause (e.g., Cioffi, 1991; Van den Bergh, 2005). The inadequacy of the traditional medical illness or disease model to account for idiosyncratic consequences of illness is documented in the finding that even in the case of known primarily organic diseases, the correlation between (subjective) symptoms and objective physical parameters ranges from .40 to .60 (e.g., for respiratory diseases; Van den Bergh, 2005). In contrast, cognitive models of symptom perception are suited to explain rather complex patterns of chronic medical states, which are puzzling because they lack adequate biomedical causes. Cognitive construction processes based on subjective cognitive illness representations or schemata (e.g., Leventhal, Diefenbach, & Leventhal, 1992) mediate between objective (minor) physical changes and the subjective impression of a severe symptom. From a cognitive-psychological perspective, symptom perception could therefore be considered as an interaction of bottom-up (physical) and top-down (cognitive) processing (Cioffi, 1991):

> "Somatic interpretation is a multiprocess elaboration upon a real or perceived physiological state. This elaboration is best characterized as an interaction between stimulus-driven and top-down processes […] "(Cioffi, 1991, p. 29).

Notwithstanding either a non-dualistic, psychobiological or biopsychological perspective of somatoform disorders and somatization in general (e.g., Rief & Barsky, 2005), the two empirical studies outlined in this thesis explicitly focus on *cognitive-psychological* abnormalities that coincide with the chronic manifestation of medically unexplained physical symptoms.

Overview

The thesis is organized into three main parts: a theoretical section, an empirical part presenting results of two studies, and a concluding summary.

In the theoretical part (chapter 1-4) we will indtroduce the realm of medically unexplained symptoms and the concept of somatization as a frequent and complex phenomenon. Somatization is defined as the occurrence and persistence of physical-like symptoms for which no organic cause can be identified or for which an existing organ pathology remains insufficient to account for the degree of idiosyncratic suffering. Following

a brief summary on epidemiology of somatoform disorders, current models that attempt to account for the etiology and persistence of medically unexplained symptoms are reviewed. As a comprehensive and complete coverage of the diverse methodological accounts of somatization is beyond the scope of the current thesis the review of models will focus on the most prominent and empirically most supported cognitive and cognitive-behavioral models to date, respectively.

Chapter three introduces Idiopathic Environmental Intolerance (IEI), formerly termed Multiple Chemical Sensitivity (MCS), in detail as a complex condition considered by some researchers as a modern variant of somatoform disorders. The chapter gives a short review of the phenomenology, the epidemiology and the various theoretical accounts of IEI. It ends with formulation of a cognitive-behavioral model of IEI as a starting point for the generation of specific hypotheses regarding information processing abnormalities in IEI, as they are the main focus this thesis.

Chapter four aims at briefly introducing the reader into the field of *selective attention research* in clinical psychology. Biased information processing has been traditionally of interest in anxiety disorders and affective disorders like depression. Recently, abnormalities in selective attention have also been found in patients with somatoform disorders. The chapter will also give an overview of the most common experimental paradigms for the assessment of affect modulated attention and memory processes.

The following two chapters (5 and 6) represent the empirical part of the thesis: In the first study (chapter 5), three experimental groups (participants with IEI, participants with a somatoform disorder but without IEI, and non-IEI and non-somatoform control participants) are compared with respect to selective attention and memory processes associated with disorder related linguistic stimuli. In this study the emotional Stroop and the dot-probe task were used as measures of selective attention, whereas a recognition task subsequent to an incidental learning period served as a measure of explicit memory bias.

The second study (chapter 6) represents a follow-up assessment of the participants of the first study after a one-year period. The aim of the second study was to gain information about the stability of the symptom measures for the clinical groups and to replicate and extend the experimental findings regarding attentional and memory biases associated with IEI and somatoform disorders in general. In this study an innovative experimental paradigm, the extrinsic affective Simon task (EAST; De Houwer, 2003) was used to simultaneously assess emotional intrusion effects of disorder related linguistic stimuli as an index of selective

attention and implicit association (or evaluation) effects as a proxy for specific disorder related cognitive schemata.

In the last chapter seven, the empirical findings are summarized and discussed with regard to the question of classification of IEI, that is similarities and differences between traditional somatoform disorders and IEI. After specifying somatoform symptoms as a cognitive-emotional phenomenon, we briefly summarize possible therapeutical implications of our empirical findings and theoretical considerations for the treatment of people with IEI. The chapter ends with an outline of questions and suggestions regarding the investigation of promising future directions in the study of IEI. In sum, the thesis aims at elucidating cognitive-emotional aspects relevant for the development and maintenance of IEI. This focus on cognitive explanatory constructs is not meant to disregard the contribution of biological or physiological variables (e.g., changes in the endocrine and the immune system) in understanding "somatoform" conditions. As will be outlined in detail later, we consider a psychophysiological or psychobiological multi-level model as most promising in understanding somatoform disorders in general and IEI specifically.

2 MEDICALLY UNEXPLAINED SYMPTOMS AND SOMATOFORM DISORDERS

Currently, many terms coexist that try to account for the phenomenon of bodily symptoms in the absence of objective medical explanation. Popular expressions are "subjective health complaints (SHC)" (e.g., Eriksen, & Ursin, 2004; Ursin, 1997), "functional somatic symptoms or syndromes" (e.g., Barsky & Borus, 1999; Fink, Rosendal, & Toft, 2002), "medically unexplained symptoms (MUS) (e.g., Kirmayer, Groleau, Looper, & Dominicé, 2004; Kisely & Simon, 2006)", "fashionable illnesses" (Ford, 1997), and "somatoform symptoms or disorders" (e.g., Hiller, 2006). Some researchers in the field avoid the term "somatoform" because of its implied psychogenic etiology and propose other, etiologically more neutral and less pejorative terms as for instance "physical symptom disorder (PSD)" (Kroenke, 2006). As the debate on labelling of these medically insufficiently defined symptoms goes on (see the special-mini series on somatoform disorders starting with Kroenke and Sharpe, 2006), we will use the terms above synonymously but will mostly refer to "somatoform" symptoms as the currently valid diagnostic term according to ICD-10 and DSM-IV. Another issue is the distinction between "disease" and "illness" with the former referring to a malfunction in the organic system, which can be diagnosed with existing medical diagnostic procedures and the latter indicating subjective perceptions of physical symptom-like sensations, the cause of which does not have to be a biological disease (e.g., Spurgeon, 2002). From our point of view, the conditions marked by somatoform or medically unexplained symptoms have to be considered "illnesses" rather than "diseases" because little is known about organic causes yet and detailed etiological and pathogenetic models remain to be proven empirically. Since the aim of the current thesis is a better understanding of single aspects of symptom development and maintenance in somatoform disorders, historical theoretical concepts and roots of the term "somatization" will be neglected (the interested reader might refer to Brown, 2004 or Ursin, 1997) and epidemiological data will be reviewed only briefly in the next section. Afterwards (in section 2.2), more emphasis will be put on different contemporary models that try to account for the development of medically unexplained symptoms.

2.1. Phenomenology, classification, and epidemiology

In contrast to single medically unexplained symptoms as a frequent and non-pathological phenomenon in the general population, patients with *somatoform disorders* are marked by subjective distress, dysfunctional illness behavior (like frequent doctor visits), and

psychosocial impairment in the absence of a sufficient organic-medical explanation (e.g., Hiller, 2005). Although MUS overlap considerably with symptoms associated with anxiety and depression, they are empirically distinguishable form traditional *mental* disorders (Henningsen, Zimmermann, & Sattel, 2003) and form a separate nosological entity. Since the definition of a symptom or syndrome as "somatoform" depends not exclusively but in part on negative medical test results, the diagnosis is considerably influenced by the current state of medical testing and examination technology (Hiller, 2005). The field of somatoform disorders represents one of the most controversial sections in current classification systems of mental disorders like the F-section (mental and behavioral disorders) of the ICD-10 and the DSM-IV. Descriptive non-etiological criteria for the diagnosis of different somatoform disorders were first introduced in DSM-III and affected ICD-10 and DSM-IV similarly (Hiller & Janca, 2003). In both classification systems somatization disorder refers to the most severe expression of medically unexplained symptoms starting early, before the age of 30, and affecting multiple organ systems. In contrast, undifferentiated somatoform disorders mostly refer to a milder variant of somatization with a dominance of symptoms in one organ system (e.g., the gastrointestinal system). The diagnosis of (somatoform) pain disorder refers to the perception of pain symptoms for which no sufficient physical pathology can be found. Conversion disorder encompasses pseudoneurological symptoms like sensory loss, cognitive decline, or convulsion that are suggestive of a neurological condition but lack typical medical explanations (e.g., Brown, 2004). Classification systems differ slightly with regard to the diagnosis of body-dysmorphic disorder (BDD), which is a distinct variant of somatoform disorders in DSM-IV and a subtype of hypochondriacal disorder (F45.2) according to ICD-10. BDD and hypochondriasis are the two disorders for which the classification under the realm of the somatoform disorders is most controversial. BDD refers to the unfounded or highly exaggerated conviction that special parts of the body are malformed (e.g., hair, skin, or teeth). Hypochondriasis encompasses the conviction that one suffers from a severe disease (e.g., cancer, AIDS) that has not yet been detected by a physician or the fear that one will develop such a disease in the future. This fear or conviction is typically triggered or maintained by the perception of minor bodily symptoms. Since for none of the two disorders medically unexplained symptoms are part of their diagnosis (Brown, 2004), their classification as somatoform disorders remains controversial. Rief and Hiller (1999) have proposed a taxonomy of somatoform disorders consisting of three subgroups: Firstly, polysymptomatic somatoform disorders that are marked by multiple symptom variants in different organ systems (somatization disorder and undifferentiated somatoform disorder); secondly,

monosymptomatic somatoform disorders that comprise only one symptom category (like pain symptoms in somatoform pain disorder or pseudoneurological symptoms in conversion disorder); and thirdly, hypochondriasis and body dysmorphic disorder (Hiller, 2005).

Single medically unexplained symptoms are a very frequent phenomenon in the general population. According to a study by Hiller, Rief, and Brähler (2006), 81.6 % of persons in a representative sample in Germany reported at least one of the 53 somatoform symptoms listed in the DSM-IV and ICD-10 causing mild impairment (71 % reported at least one pain symptom of the DSM-IV, 35.4 % a gastrointestinal symptom, and 27.1 % a pseudoneurological symptom). About one fifth (22.1 %) even reported severe impairment by one or more somatoform symptoms. Although somatoform symptoms represent an everyday phenomenon, in some people symptoms persist, cause considerable distress, and significantly impact on quality of life. In primary care settings on average about 20 to 35 percent of patients present with medically unexplained symptoms and somatoform disorders are the most frequent mental disorders (Toft, Fink, Oernboel, Christensen, Frostholm, & Olesen, 2005). In secondary care contexts even between 30 and 50 percent of patients report medically unexplained symptoms (Hamilton, Campos, & Creed, 1996; Nimnuan, Hotopf, & Wessely, 2000; Reid, Wessely, Crayford, & Hotopf, 2001). Among the most frequent MUS are gastrointestinal complaints, back pain, and headache (Reid et al., 2001). Regarding the prevalence of the distinct diagnostic categories defined according to ICD-10 and DSM-IV, figures vary considerably with respect to the referring sample: According to some studies, the somatization disorder can be considered as a rare phenomenon in the general population with a median rate of 0.4% (range 0.03% to 0.82%) (Creed & Barsky, 2004). The prevalence of the full picture of hypochondriasis appears equally small: In a population based sample in Montreal (N = 533), Looper & Kirmayer (2001) only found one subject (0.2 %) fulfilling DSM-IV criteria of hypochondriasis whereas 1.3 % (N = 7) met abridged hypochondriasis criteria (disease conviction, distress or interference with functioning, and medical care-seeking; Gureje, Üstün, & Simon, 1997). In a large representative study in the general population in Germany (n = 4181) Jacobi, Wittchen, Hölting, Höfler, Pfister, Müller, and Lieb (2004) found a lifetime prevalence for at least one somatoform disorder or syndrome (including the somatic syntom index, SSI4,6; Escobar, Rubico-Stipec, Canino, & Karno, 1998) of 10.3 % in males and 22.2 % in females.

Gureje et al. (1997) demonstrated convincingly, that using more liberal diagnostic criteria for hypochondriasis (e.g., omitting the ICD-10 C-criterion “refusal to accept medical reassurance”) results in the inclusion of people that are obviously not less impaired than the

people fulfilling the full criteria of hypochondriasis. These observations underline the difficulties connected with the current diagnoses of somatoform disorders. The phenomenon of somatization affects not only the patient but also the health care system. Barsky, Orav, and Bates (2005) reported, that irrespective of mental or physical comorbidity, patients with somatization had twice the medical care utilization and medical care costs of patients without the diagnosis of a somatoform disorder.

2.2. Current models of somatization

The term "somatization" stems originally from the realm of psychoanalysis and goes back to an erroneous translation of the German term "Organsprache" used by Wilhelm Stekel in 1925 (Mai, 2004; Marin & Carron, 2002). Although "Organsprache" originally had a slightly different meaning, the term somatization was later used to describe a process identical to conversion, that is, the somatic or physical expression of a hidden psychological conflict. Despite these psychoanalytic roots of the somatization concept, the currently most influential models to explain this phenomenon stem from the fields of behavioral medicine and cognitive-behavior therapy (CBT). As an example of this class of models, we will briefly outline a model proposed by Kirmayer and Taillefer (1997). Rief and Hiller (1998) proposed a similar theoretical approach for somatization disorder and hypochondriasis. In contrast to these models that stress the importance of cognitive constructs like symptom focused attention and dysfunctional interpretation of bodily symptoms, Van den Bergh and colleagues have proposed a learning account of medically unexplained symptoms. Recently, Brown (2004) has proposed a genuine cognitive-psychological model based on fundamental principles of attentional and perceptual processes.

2.2.1. A cognitive-behavioral model of somatoform disorders

The core feature of cognitive or cognitive-behavioral models of somatoform disorders is the assumption that cognitive processes (e.g., sustained attention toward symptoms, interpretation as harmful, and attribution as sign of a severe illness) mediate the relationship between the perception of (minor) bodily symptoms and behavioral changes (e.g., help-seeking behavior, physical and social inability). Behavioral changes in turn amplify the perception of symptoms by directing attention toward symptoms (Barsky, 1992) and physical de-conditioning resulting from the avoidance of physical activity. Therefore, similar to

prominent models of panic disorder, CBT models of somatization mainly consist of one or more vicious circle(s) of symptom detection, catastrophic interpretation and attribution processes (e.g., symptom as sign of severe illness), and different variants of (physical) avoidance or illness behavior leading to a higher probability of symptom manifestation in future (e.g., via prolonged arousal or because of decreased bodily fitness) (e.g., Hiller, 2005; Looper & Kirmayer, 2002). Additionally, psychosocial and interpersonal factors like characteristics of the compensation system, availability of health care providers and reactions of friends and relatives are proposed as possible reinforcing mechanisms that might contribute to the maintenance of somatoform symptoms (Looper & Kirmayer, 2002). Although such an operant mechanism might play a significant role (as also proposed in the realm of chronic pain), nearly all researchers in the domain of somatoform disorders agree that symptoms are subjectively *real,* though not under volitional control. Somatoform symptoms are therefore clearly distinguishable from the phenomenon of simulation, i.e. the intentional and volitional presentation of symptoms in order to achieve certain goals. Figure 2-1 shows a cognitive behavioral model of somatoform symptoms proposed by Looper and Kirmayer (2002). The model depicts that different conditions, not only emotional stressful experiences, but also primarily organic illnesses (e.g., infectious diseases), can trigger a cascade of dysfunctional attentional, attributional, and behavioral consequences leading to the manifestation and chronification of somatoform disorders. The model therefore corresponds with clinical observations, that sometimes the starting point of a somatoform disorder represents an organic illness such as an Eppstein-Barr virus infection.

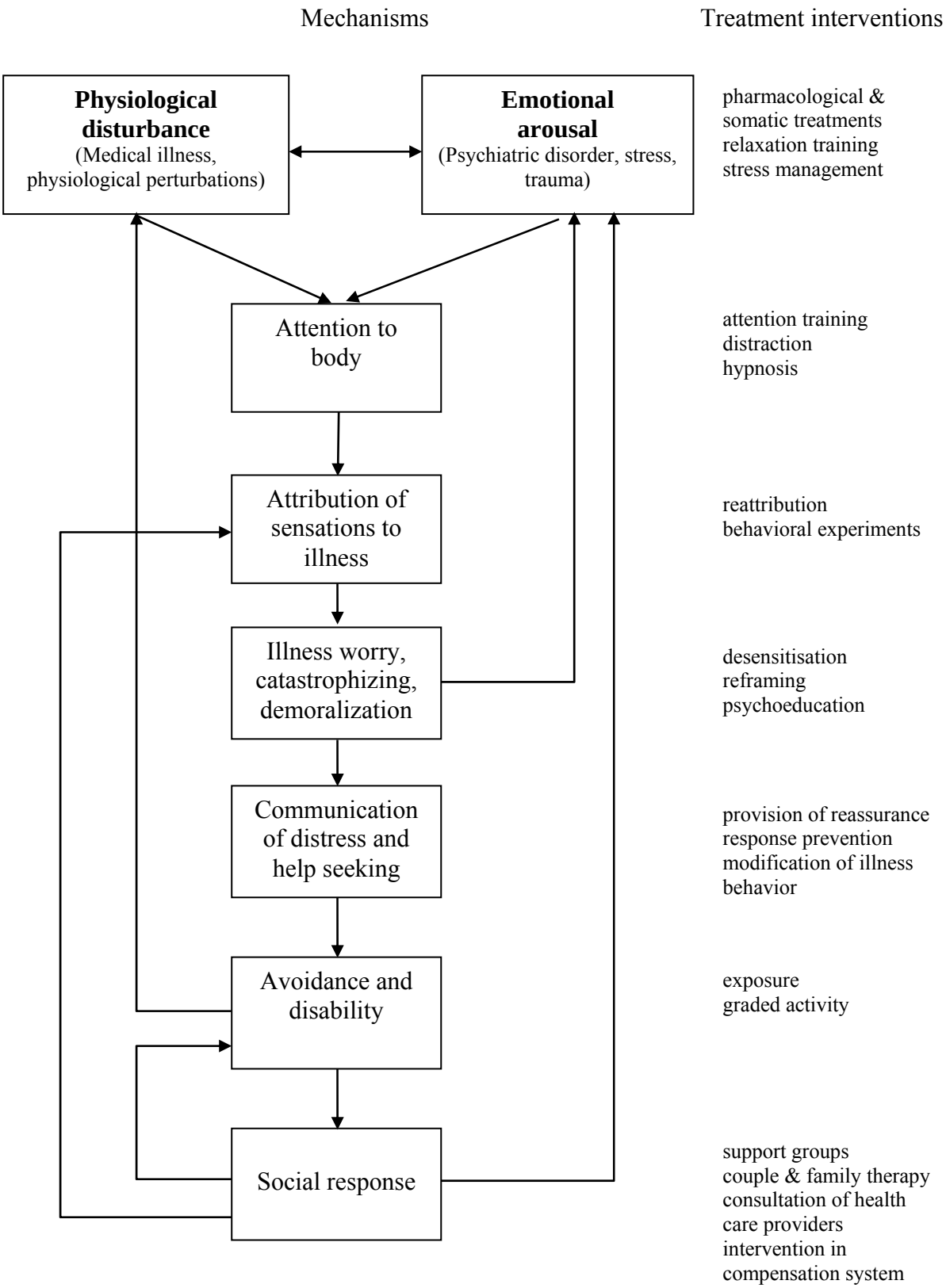

Figure 2-1: A multifactorial model (psychosocial mechanisms, interpersonal interactions, and discursive practices) of somatization and corresponding treatment interventions (Looper & Kirmayer, 2002).

2.2.2. A learning/conditioning perspective on somatic symptoms

Similar to somatoform disorders, panic disorder and agoraphobia are also characterized by diverse bodily symptoms, for instance dyspnea, tachycardia, or light-headedness. These and many other symptoms can easily be provoked even in normal people by hyperventilation instruction (i.e., fast and deep breathing for about 3 minutes). Physiological theories have focused on hypocapnia, which is a reduction of carbon dioxide pressure in the arterial blood, as an explanation for the observed range of reversible symptoms. However, as reviewed by Stegen, De Bruyen, Rasschaert, Woestijne, and Van den Bergh (1999), empirical evidence documenting reduced carbon dioxide pressure in patients with panic attacks have remained weak. The authors therefore hypothesized that symptoms that might have originally resulted from hypocapnia are prone to associative learning or classical conditioning processes. In other words - typical hyperventialion symptoms can be elicited by contextual stimuli that become conditioned stimuli (CS) via Pavlovian conditioning. In a series of experiments in healthy people and psychosomatic patients, Van den Bergh and colleagues demonstrated elegantly that in a differential conditioning account only few pairings of a conditioned stimulus (e.g., an unpleasant odor) with CO_2 enriched air (UCS) are sufficient to produce a conditioned response (e.g., alterations in breathing and subjective symptom reports) (Van den Bergh, Stegen, & Van de Woestijne, 1997; Devriese, Winters, Stegen, Van Diest, Veulemans, Nemery, Eelen, Van de Woestijne, Van den Bergh, 2000). Interestingly, even a mental image as CS (e.g., script of a situation being stuck in an elevator or a sauna) sufficed to elicit symptoms previously provoked by CO_2 enriched air as the UCS (Stegen et al., 1999).

The principal of the learning account of bodily symptoms in humans proposed by Van den Bergh and colleagues supposes, that bodily symptoms that are normally the natural reaction to certain internal (e.g., hypocapnia) or external triggers (e.g., toxic substances) can become associated with previously neutral stimuli (e.g., odors or mental images). The consequence of such an association is that even in the absence of a "natural" trigger unspecific somatic symptoms can be provoked by various conditioned stimuli. As this model proposes that aversive olfactory stimuli are well suited to become CS, the model has been successfully applied to the multiple chemical sensitivity syndrome (MCS) (Van den Bergh, Devriese, Winters, Veulemans, Nemery, Eelen, & Woestijne, 2001) that will be outlined in the following chapter three.

2.2.3. A cognitive-psychological approach to medically unexplained symptoms

Brown (2004) proposed another cognitive psychological model to explain the development and maintenance of medically unexplained symptoms. Brown's model is mainly informed by the theory of attentional control of Norman and Shallice (Norman & Shallice, 1986). In the tradition of dual-process theories in psychology (e.g., Feldmann Barrett, Tugade, & Engle, 2004), Brown distinguishes two attentional control systems, a primary attentional system (PAS) and a secondary attentional system (SAS) (Figure 2-2). The PAS refers to an effortless, intuitive, and automatic mode of information processing; the SAS operates under conditions of self-awareness and the impression of cognitive effort and deliberateness.

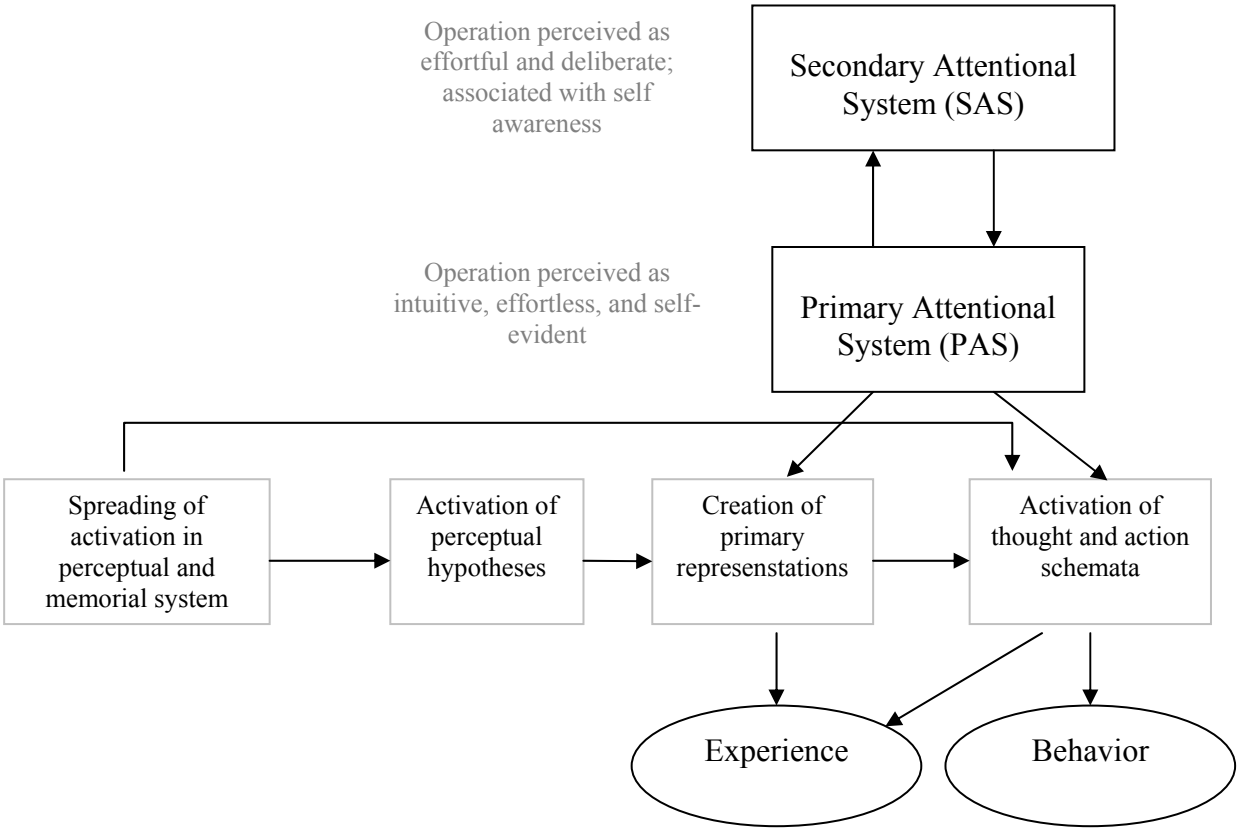

Figure 2-2: The generation of experience and control of action by the cognitive system. Figure taken from Brown (2004; p. 801).

According to Brown (2004), like perception in general, the formation of medically unexplained symptoms is the result of a complex constructive process that relies on both existing knowledge structures and schemata in memory as well as on actual inputs from the perceptual system. False or rogue perceptions of symptoms arise when attentional selection

processes of the PAS are biased by existing and (over-)active knowledge structures in memory. Brown compares the perception of unexplained symptoms to certain visual illusions, that demonstrate convincingly how powerful expectancies or habits can guide and mislead our perception of the "real" sensory world. Whereas the primary locus of dysfunction in patients with MUS is the PAS because it automatically activates or selects (rogue) symptom representations, the chronification and maintenance of MUS is mainly a function of the amount of "high-level" attention consciously allocated to prior formed symptom representations via the secondary attentional system (or Supervisory Attentional System, SAS, in the Norman and Shallice framework). Factors that perpetuate the direction of attentional resources to symptoms are displayed in Figure 2-3 and include dysfunctional attributional strategies (e.g., catastrophizing thoughts about symptoms as signs of severe illness), ongoing rumination about symptoms or illness, illness behavior like checking the body for signs of illness or avoidance of physical activity, personality factors like dispositional negative affectivity or hypnotic susceptibility. The repeated allocation of attention by the SAS to symptoms is supposed to decrease the threshold for the automatic selection of symptom representations by the PAS and thereby again fosters the experience of subjectively "real" but actually "rogue" (symptom) representations. The resulting vicious circle is presented in Figure 2-3.

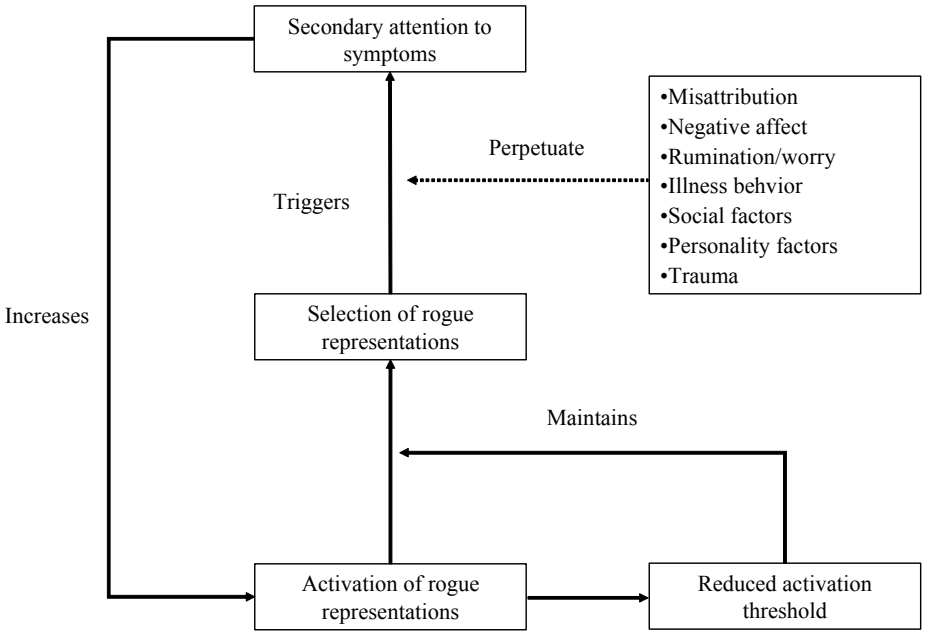

Figure 2-3: The role of secondary attention in the development of unexplained symptoms. Factors perpetuating the allocation of secondary attention to rogue representations are shown in the dotted box (figure and legend from Brown, 2004, p. 804).

According to Brown (2004), memory structures that bias the operation of the PAS (i.e., symptoms representations) can have various origins. They either stem from "real" physiological disorders in the past, they might be the consequence of traumatic experiences, or they might even have developed in the absence of any prior illness experience in the self, simply through information from or observation of others. Once those symptom representations have developed it depends on the operation of the hypothesized perpetuating state and trait variables proposed above (e.g., trait negative affect, rumination or worry; Figure 2-3) if an individual will develop and maintain the experience of MUS.

Several aspects of the entire model proposed by Brown (Figure 2-4) are already included in the model postulated by Kirmayer and Taillefer (1997) (Figure 2-1) or the concept of somatosensory amplification (Barsky, 1992; Barsky, Goodson, Lane, & Cleary, 1988). However, in contrast to the former models Brown precisely elaborates possible cognitive psychological origins of medically unexplained symptoms. The idea that prior episodes of severe organic illnesses are one option leading to the formation of overactive and primary attention guiding symptom representations fits well with the clinical impression that many patients suffering from MUS report episodes of "real" physical diseases. In essence, the Brown model conceptualizes MUS as artificial reactions or false alarms of a highly sensitized information processing system. In this respect it resembles the idea of "somatovisceral illusions" with respect to emotional experiences as proposed by Cacioppo, Berntson, and Klein (1992). Although the final model (Figure 2-4) appears quite complex and many relations between the involved constructs remain hypothetical, Brown finally proposes detailed hypotheses of how parts of his model might be tested empirically. Most relevant for our work is the hypothesis that people with medically unexplained symptoms in contrast to nonsomatoform controls should demonstrate an *attentional bias* towards symptoms (hypothesis 2, p. 807) that should be detectable with cognitive paradigms (e.g., the emotional Stroop task) and that abnormalities in attentional processes should be associated with alterations in high-level postattentive processing rather than low-level preattentive processing (hypothesis 4, p. 807). Brown's conceptualization of altered attentional processes in somatoform disorders fits other models that try to account for biased attentional processes in psychopathology (e.g., in anxiety and depression). We will come back to detailed models and experimental paradigms that try to explain and assess attentional biases toward idiosyncratic relevant and mostly negative laden emotional stimuli in the fourth chapter.

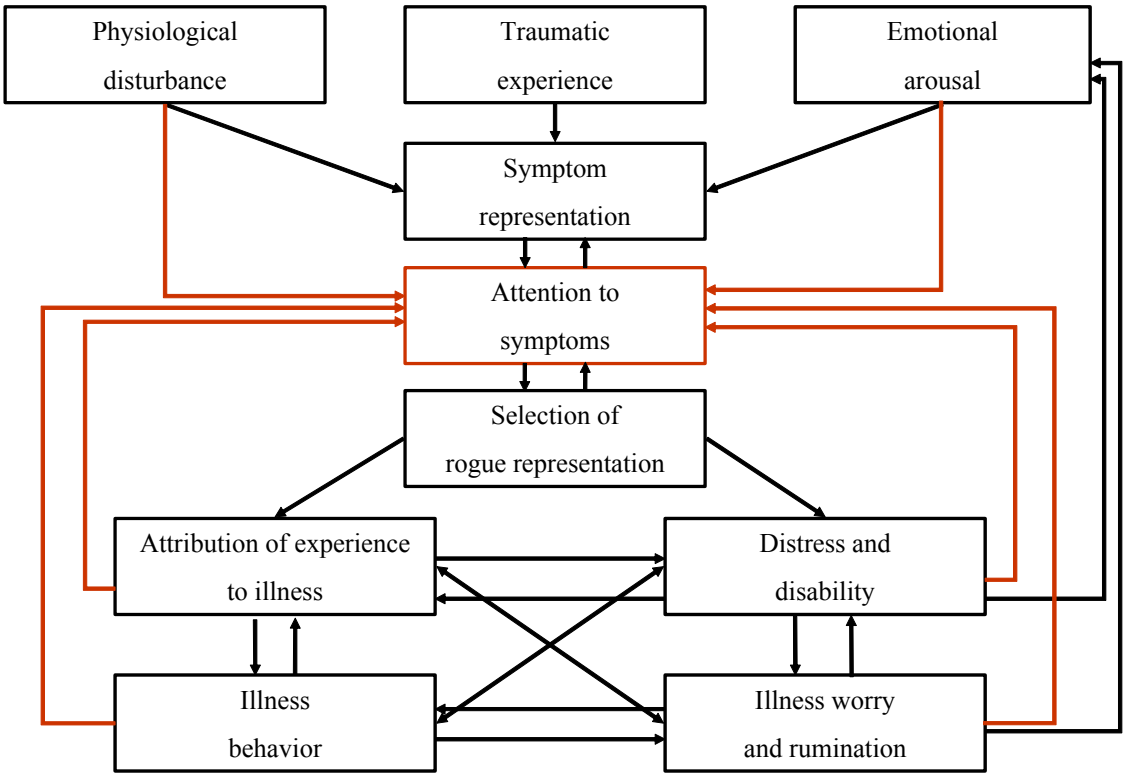

Figure 2-4: Factors involved in the development of symptom chronicity (figure and legend from Brown, 2004; p. 804).

2.2.4. A psychobiological perspective on medically unexplained symptoms

In contrast to the almost pure cognitive-psychological approach proposed by Brown (2004), Rief and Barsky (2005) outline a psychobiological model of unexplained symptoms. The authors support the position that somatoform symptoms are not physiologically unfounded. Rief and Barsky review evidence that the symptom reports in people with somatoform disorders are presumably associated with a heightened autonomic arousal (e.g., a decreased recovery response of heart rate activity after mental distress; Rief & Auer, 2001), changes in the endocrine system (i.e., alterations in the functioning of the hypothalamic-pituitary-adrenal axis leading to hypocortisolism), dysfunctional activation of the immune system, and abnormalities regarding certain monoamino acids and neurotransmitters. Although the role of those biological factors in somatoform disorders remains equivocal (e.g., in case of the postulated hypocortisolism) and empirical evidence in this domain is still weak, it seems plausible that alterations in the immune system are associated with certain aspects of somatoform disorders like illness behavior, and that biochemical changes in the

neurotransmission process contribute to alterations in pain and symptom perception thresholds (Rief & Barsky, 2005). Even if future research confirms the role of these hypothesized biochemical factors the question of causality and possible bi-directionality of biological, cognitive, and behavioral factors remains as a crucial issue.

In their final simplified psychobiological model of medically unexplained symptoms (Figure 2-5), Rief and Barsky (2005) conceptualize that somatoform symptoms and disorders are the consequence of two main phenomena: Firstly, an increase in body signals due to numerous (mostly biological) factors as a consequence of frequent distress, a lack of physical condition or a chronically stimulated HPA-axis. Secondly, similar to the gate-control-theory in pain research, a deficient filter system is supposed to amplify bodily signals rather than to inhibit or effectively select them as it would in healthy people. This leads to increased conscious perception of bodily reactions and symptoms. Possible electrophysiological correlates of such a defective filter system comprise increases in the N1-components and decreases of the mismatch negativity in somatoform patients (Gordon, Kraiuhin, Kelly, Meares, & Howson, 1986; James, Gordon, Kraiuhin, Howson, & Meares, 1990; according to Rief & Barsky, 2005). In line with the psychobiological (filter-)model of Rief and Barsky, Thayer and Brosshot (2005) describe how an imbalance between the sympathetic and parasympathetic system and especially the chronic deactivation of the parasympathetic system may lead to malfunctions of the immune system, thereby fostering chronic health problems in general. The core feature of their model is the disinhibition of emotion circuits in the central nervous system leading to a chronic or prolonged stress or fight-and-flight response. One of the reasons for the disinhibition of neural circuits associated with emotion processing and threat detection according to the authors represents a decrease in inhibiting top-down signals from areas within the prefrontal cortex, that is a hypoactivation in the prefrontal cortex. Further research with modern brain imaging techniques (e.g., functional magnetic resonance introspection and positron emission tomography) should address these hypotheses in patients with somatoform disorders.

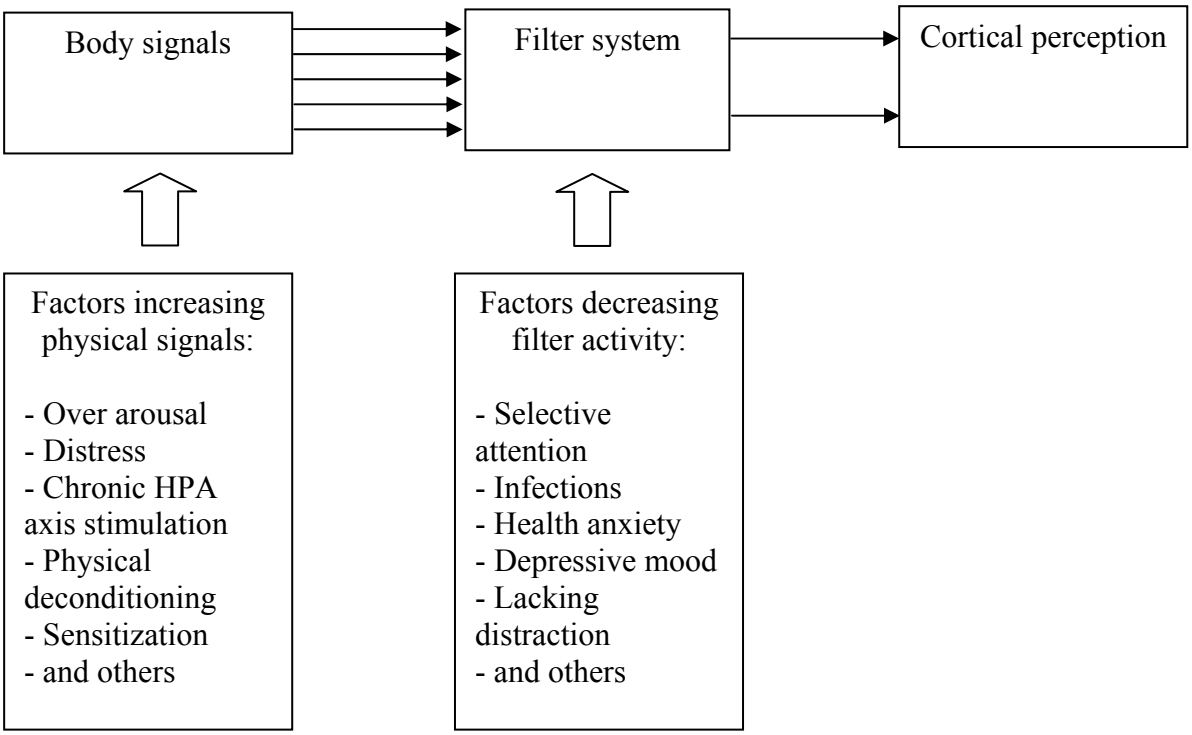

Figure 2-5: The filter model of somatoform symptoms (taken from Rief & Barsky, 2005)

To our impression, there are little sharp contradictions between the theoretical notions proposed by Brown (2004) on the one hand, and Rief & Barsky (2005) on the other. Both theories agree on the position that somatoform disorders rely on abnormalities in the *perception* of bodily signals. The filter system included in the Rief and Barsky model could cognitive-psychologically be conceptualized as the primary attentional system (PAS) included in the Brown model. The most striking difference between the two models might be that, according to the Brown model, medically unexplained or somatoform symptoms can develop in the complete absence of any current biological or physical organic correlate – a position that might be hard to reconcile within the Rief and Barsky model. One of the crucial issues to decide between the two theoretical accounts is therefore the question: Are people with somatoform symptoms (because of heightened emotional arousal or negative affectivity) more sensitive or vigilant toward "real" somatic changes or do elevated symptom reports represent an emotional distress mediated reporting bias? First empirical evidence for the latter position has been presented by Aronson, Feldmann Barrett, and Quigley (2001; 2006), although generalizability of their findings is restricted by their use of a non-clinical sample of university students and the mono-methodological operationalization of somatic sensitivity by

a heartbeat detection task. Similar evidence that self-reported somatosensory amplification scores (Barsky, Wyshak, & Klerman, 1990) are even negatively related to real somatic or physiological sensitivity (measured by the ability to accurately monitor one's own heartbeats) suggests that the subjective impression of amplified bodily sensations might correspond to an inability to detect and discriminate "normal" bodily sensations rather than to hypervigilance to them (Mailloux & Brener, 2002).

However, with the exception of the learning approach presented by Van den Bergh and colleagues, all proposed models of medically unexplained symptoms explicitly agree on the crucial and dysfunctional role of *symptom focused attention* regarding the development, maintenance, and chronification of medically unexplained symptoms. Because little systematic empirical research has addressed the question of in how far patients with different MUS actually show comparable selective attention effects towards bodily symptoms, the area of symptom focused attention represents the main topic of the two empirical studies presented later on. Before introducing some modern experimental paradigms suitable for the experimental assessment of attentional processes a presumed modern variant of somatoform disorders called idiopathic environmental intolerance (IEI), formerly considered as multiple chemical sensitivity (MCS), will be outlined.

3 IDIOPATHIC ENVIRONMENTAL INTOLERANCE (IEI)

In addition to the various forms of somatoform disorders according to DSM-IV and ICD-10 as outlined above, at the end of the 20th century a number of labels have been proposed by different medical subdisciplines to account for medically unexplained symptom clusters with an emphasis on different organ systems. For instance in gastroenterology the irritable bowel syndrome (IBS) became a frequent diagnosis, in rheumatology the fibromyalgia syndrome (FMS), in dentistry the mandibular dysfunction syndrome, and in orthopedics back pain (e.g., Deary, 1999; Escobar, Hoyos-Nervi, & Gara, 2002). Additional modern examples of phenomenological similar conditions are the silicon breast implant illness, the Gulf war syndrome (GWS), the toxic mold syndrome, and the sick-building syndrome (SBS) (for a short review see Binder & Campbell, 2004). All of these syndromes have in common that despite of their fashionable labels implying a simple and clear-cut etiology little causal empirical evidence regarding symptom development could be detected so far. Stewart (1990b) points out the observation that most people with a fashionable illness also fulfill criteria for at least one other unclear syndrome and that patients change their illness label according to special coverage of fashionable illnesses in the media or according to labels offered by physicians. Therefore, a debate continues whether it is reasonable to "split" the different conditions or "lump" them together (Wessely, Nimnuan, & Sharpe, 1999; Wessely & White, 2004). In the domain of clinical ecology (Bell, 1982), Multiple Chemical Sensitivity (MCS) became a famous diagnostic label to account for the subjective complaints of people suffering from a vast array of symptoms like for instance headache, fatigue, light-headedness, and dizziness. Although the symptoms between the different functional somatic symptoms like FMS, CFS, MCS or SBS overlap considerably (e.g., Aaron & Buchwald, 2001), the specificity of MCS or IEI stems from the attribution of symptoms to low-level chemical exposure in everyday life. The existing diagnostic criteria and data on epidemiology as well as current models of the etiology and maintenance of MCS/IEI will be presented in the following sections.

3.1. Terminology, phenomenology and epidemiology of IEI

The following story briefly describes the case of a prototypical patient suffering from IEI:

> "The Story of Eric:
> Eric has been complaining for some time about not feeling well when he is exposed to chemical products. When he enters a room where such products have been used recently, he becomes light-headed, has problems with his balance, and has difficulty breathing. He is about to faint and feels dysphoric, looses his vigour and strength. This condition may last for hours and afterward, he often has a headache for a couple of days and prefers to stay home. He lives in a rural community. He likes to work in the garden and regains his strength this way. "Nature heals itself," he says. The problems started 5 years ago. Eric was working as an employee in a company that produces silicones. Regularly, he had to enter the production units and the warehouses for control and advice. All the employees from the warehouses had a medical check-up regularly, Eric as well. There were never serious problems reported by anyone except by Eric. The complaints developed gradually in Eric's case. First they were tolerable, but they slowly became more disturbing. After a while, he was avoiding any odor that could trigger complaints. He avoided entering the productions units and the warehouses and he called in sick very often. Finally, his problem and avoidance behavior escalated such that he got fired. Now he is unemployed, but his problem has not improved. Meantime, all kinds of products that—according to him—had a chemical odor have been removed from the house: paint, thinner, white spirit, some types of soap, ethyl alcohol, several household cleaning products, and even perfumes; he considered them all poisonous. His avoidance behavior has become so bad that when the house is being cleaned, he has to leave. (Winters et al., 2003; p. 337)."

Over the last two decades there has been an increasing multidisciplinary interest in a controversially disputed disorder called idiopathic environmental intolerance (IEI), or multiple chemical sensitivity (MCS). Recently the WHO has discarded the term MCS because of its implicit and yet unverified etiological implications (Sparks, 2000). Without speculating about etiology, Staudenmayer, Binkley, Leznoff, & Phillips (2003a, p. 235) define IEI as "an acquired disorder with multiple recurrent symptoms, associated with diverse environmental factors tolerated by the majority of people; not explained by any known medical, psychiatric or psychological disorder".

Since no official diagnostic criteria for IEI could be established yet, prevalence rates of the phenomenon vary considerably: between 15-30 % of respondents in population based studies report minor problems with environmental chemicals, while 1-6 % meet more restrictive criteria of a disabling chemical intolerance in the sense of IEI (e.g., Bell & Schwartz, 1993; Kreutzer, Neutra, & Lashuay, 1999; Meggs, Dunn, Bloch, Goldman, & Davidoff, 1996; Reid, Hotopf, Hull, Ismail, Unwin, & Wessely, 2002). IEI is typically associated with various non-specific symptoms like headache, fatigue, muscle pain, arthralgia, sleep disturbance, dizziness, and cognitive impairments (Bornschein, Hausteiner, Zilker, & Först, 2002). Because of this unspecific symptom pattern, IEI phenomenologically overlaps

with other known complex conditions like Chronic Fatigue Syndrome (CFS), Sick Building Syndrome (SBS), Gulf War Syndrome (GWS), all of which are considered functional somatic syndromes (Barsky & Borus, 1999) or modern variants of somatoform disorders. IEI is different from other functional syndromes and traditional somatoform disorders (according to DSM-IV) in specific externalizing attributions of symptoms to diverse chemical and physical environmental triggers (Bailer, Witthöft, Paul, Bayerl, & Rist, 2005). Although a considerable variability and idiosyncracy of such suspected trigger substances is characteristic of IEI, most frequently reported triggers include dental amalgam, lead, metals, organic solvents, wood preservatives, pesticides, and strong odors in general (Bornschein et al., 2002).

Although IEI is a heterogeneous disorder and yet lacks a unitary case definition there is evidence that especially two case definitions (Nethercott, Davidoff, Curbow, & Abbey, 1993; MCS Consensus Definition, 1999) can adequately discriminate between environmental health practice patients and general health patients (McKeown-Eyssen, Baines, Marshall, Jazmaji, & Sokoloff, 2001). The following three criteria are part of both definitions: (1) Symptoms are linked to low-level exposure, (2) symptoms are chronic, and (3) symptoms are provoked by different chemically unrelated substances. Accordingly, the key symptom of most IEI patients represents a hypersensitivity to different chemical odors in concentrations tolerated by the majority of the population (Szarek, Bell, & Schwartz, 1997; Black, 2000; Bailer, Rist, Witthöft, & Paul, 2004a). Theories of etiology and pathogeneses of IEI are still under debate and oscillate between the extremes of psychological and biological standpoints (Labarge & McCaffrey, 2000; Sparks, 2000; Fiedler & Kipen, 1997). In the following paragraphs we will briefly outline the different standpoints and end this chapter with the formulation of a cognitive-behavioral model of IEI. Regarding theories of etiology of IEI, the interested reader might refer to more extensive reviews by Fiedler & Kipen (1997), Labarge & McCaffrey, 2000, and Sparks (2000) (for genetic findings in IEI see Binkley, King, Poonai, Seeman, Ulpian, & Kennedy, 2001; McKeown-Eyssen, Baines, Cole, Riley, Tyndale, Marshall, & Jazmaji, 2004).

3.2. Theoretical approaches to IEI

Theoretical accounts of IEI still try to provide answers for two general questions: Firstly, does a “real or true” association exist between exposure to trigger substances and symptoms or does IEI just represent a bias in reporting and attributing symptoms? Secondly, if we assume a “real or true” association, is it mediated by a toxicological mechanism or by

psychological or psychophysiological factors (e.g., pavlovian conditioning)? In response to these fundamental questions, various theories have proposed different answers: They favor either a toxicological mechanism (implying a direct and causal relationship between a low-dose chemical agent and different symptoms) (e.g., Miller, 2000; Miller, 2001), a primarily psychological mechanism (based on cognitive and conditioning processes; Bolla-Wilson, Wilson, & Bleecker, 1989; Van den Bergh et al., 2001) or a rather complicated psychophysiological interaction of both (e.g., the olfactory-limbic model of multiple chemical sensitivity: Bell, Miller, & Schwartz, 1992). As the current thesis is primarily concerned with the analysis of *cognitive-psychological* factors in IEI, we will only briefly summarize the toxicogenic and psychophysiological class of theories. After a critical comment on these biological approaches we will outline psychological factors relevant for the understanding of IEI and propose a hypothetical cognitive-behavioral model of IEI.

3.2.1. Toxicogenic and biological approaches to IEI

The toxicogenic theories state that hypersensitivity to low-dose chemical exposure results from damages of different organ systems (e.g., the immune system; Levin & Byers, 1987) caused by chemical exposure. Proponents of biogenic or toxicogenic theories assume a causal relationship between chemical exposure and symptoms. The toxicant-induced loss of tolerance approach (TILT) proposed by Miller and colleagues (e.g., Miller, 1997, 2001; 2000; Miller, Ashford, Doty, Lamielle, Otto, Rahill, & Wallace, 1997) states that the TILT syndrome, which underlies not only IEI but also many other medically unclear conditions develops in two phases. In the first phase (initiation phase) individuals lose their natural tolerance either through a single massive toxic exposure or by repeated and long-lasting minor- or low-level exposure (e.g., air contamination in an office building). In the second phase, persons with TILT notice various symptoms triggered by previously tolerated substances (e.g., everyday chemicals like traffic exhaust and fragrances; certain foods and drugs like alcohol or caffeine). From a scientific position, TILT does not refer to a theory or model that is meant to explain the etiology or maintenance of IEI, but it rather represents a purely descriptive term (Ashford & Miller, 1996) that summarizes the impressions of people affected by IEI. The TILT approach is therefore of little scientific value.

Some other biologically oriented researchers consider alterations or dysfunctions of the immune system as fundamental in IEI (Meggs, 1992; according to Labarge & McCaffrey, 2000). However, negative findings with respect to classical parameters in allergic diseases

(such as the IgE) propose that IEI is at least distinct from known classical allergies. Meggs (1993) therefore hypothesizes that the excitation and irritation of olfactory nerves fosters *neurogenic inflammation* processes (i.e., inflammation triggered by the nervous system independent of the immune system) that in turn provoke immune responses that contribute to the clinical picture of IEI. However, similar to the TILT approach, the mechanism of neurogentic inflammation is not restricted to IEI but supposed to play a crucial role in other unclear conditions like migraine, fibromyalgia, and asthma.

A scientifically more elaborated psychobiological approach to IEI comes from Bell and co-workers (e.g., Fernandez, Bell, & Schwartz, 1999; Antelman, 1988; 1994). They propose in their "*limbic kindling hypothesis*" that chemical sensitivity results from a strong and chronic stimulation of the limbic and mesolimbic system by either olfactory stimuli or other strong exogenous substances or events (e.g., traumata). Once sensitization of limbic pathways took place, further weaker stimulation either by substances or psychological stressful events suffice to provoke intense limbic responses. The impact of *olfactory detectable* substances is so strong because the amygdala is directly connected to the olfactory system. The mediating process between (chemical) low-dose stimulation and increased sensitivity of limbic and mesolimbic pathways is considered as "time-dependent sensitization (TDS)" and "kindling", which refers to the amplification of central and peripheral responses to certain stimuli. In line with the sensitization approach, psychophysiological abnormalities (increased EEG resting alpha activity) have been found in IEI individuals and specificity of this finding has been demonstrated compared to individuals with depression only (Bell, Schwartz, Hardin, Baldwin, & Kline, 1998) and people with sexual traumata (Fernandez et al., 1999). Similar to the TILT and the neurogenic inflammation approach, the model of TDS is applied not only to IEI but also to other conditions different from IEI, for instance post-traumatic stress disorder (PTSD) or panic disorder (Antelman, 1988). In their focus on individual differences that contribute to chemical intolerance (CI), Bell and colleagues (Bell, Baldwin, & Schwartz, 2001) propose that genetic and gender related factors contribute to the higher sensitizability of people with CI in different organ systems (central nervous system, autonomous nervous system, and peripheral nervous system). In contrast to the toxicological notions proposed by clinical ecologists, Bell et al. consider not the toxic substance or the exogenous stressor but rather individual abnormalities in different organ systems as crucial for the development and maintenance of CI. Although the psychophysiological model presented by Bell is theoretically more elaborated, empirical evidence for alterations in olfactory-limbic pathways and the operation of sensitization processes are weak and often

indirect at best. The following critique (3.2.2. below) on the biological and psychophysiological notions stems mainly from the work of Staudenmayer and colleagues (Staudenmayer, Binkley, Leznoff, & Phillips, 2003a; 2003b), who have applied Bradford Hill's criteria for the analysis of causality (originally developed for the association between smoking and lung cancer) to both the toxicogenic (Staudenmayer et al. 2003a) and the psychogenic theory of IEI (Staudenmayer et al. 2003b).

3.2.2. Critical evaluation of the "Chemical Hypothesis" and the kindling model

Regarding a purely organic perspective of IEI as favored by clinical ecologists there is little evidence for a simple toxicological notion of IEI, nor an involvement of toxicological factors in more complex psychophysiological models, e.g., limbic kindling or sensitization. As Van den Bergh and colleagues observed (Van den Bergh et al., 2001), evidence for these models is exclusively derived from sensitization studies in animals. Also, doses of chemical substances used in these studies are generally higher than the rather low levels of everyday exposure normally reported by people with IEI. Therefore, the equivalence of models such as time-dependent sensitization (TDS) and limbic kindling to IEI in humans still remains to be proven. Findings that high proportions of people suffering from IEI cannot remember an initial exposition or poisoning event and that chemically intolerant (CI) people reporting such events do not differ significantly in symptomatology from CI people without such crucial events are hard to reconcile with toxicological notions. Furthermore, in contrast to the opinions held by people suffering from IEI, purely biological and toxicological approaches have failed to provide evidence for agent-symptom causality by demonstrating e.g., substance-symptom specificity or dose dependence of symptom strength or frequency (for details about the lack of substance-symptom causality in IEI see Staudenmayer et al., 2003a, b). Additionally, the prevalence of chemical sensitivity is not elevated among high chemical exposure groups, e.g., industrial workers (Kiesswetter, Sietmann, Zupanic, van Thriel, Golka, & Seeber, 1999). Similar evidence against a significant association between chemical sensitivity and long-term chemical exposure (e.g., organic solvents) was presented by Bornschein and colleagues (Bornschein, Hausteiner, Konrad, Förstl, & Zilker, 2006) who found no evidence for an elevated toxic load in urine samples of environmental patients compared to a group of industrial workers with daily exposure to low doses of metals and solvents. Focusing on special parameters (chemosensory event-related potentials) regarding the olfactory information processing, Papo and colleagues (Papo, Eberlein-König,

Berresheim, Huss-Marp, Grimm, Ring, Behrendt, & Winneke, 2006) found no evidence for altered olfactory information processing or lowered olfactory thresholds. Consequently, a simple organic or toxicological conceptualization of IEI as either a dysfunction of the olfactory system or a chronic reaction to environmental poisoning seems unlikely. As reviewed by Staudenmayer et al. (2003b), the most prominent legitimate medical organizations in North America involved in the study of IEI (e.g., the American Academy of Allergy Asthma and Immunology, the American College of Occupational and Environmental Medicine, the American Medical Association, and the American Academy of Toxicology) agree on the position that the toxicogenic theory is unsubstantiated. In contrast, empirical evidence of the involvement of psychological and psychophysiological factors in IEI is accumulating. The psychogenic position proposed by Staudenmayer and colleagues is summarized in the following statement:

> "IEI is a phenomenon best described as a disorder of belief characterized by an overvalued idea infecting the belief systems of individuals and social networks. IEI is another of the fashionable functional somatic syndromes historically described as neurasthenia. Processes of mass psychogenic illness operate to create a contagious effect mediated iatrogenically by clinical ecologists ('evironmental physicians') through support groups, the Internet, and the media. Psychological, psychophysiological, and psychosocial processes, whether compounded by psychopathology or not, explain IEI. We conclude that the psychogenic theory can and should be accepted as the working model of IEI pathogenesis. Further study should be directed toward the mechanisms identified by the psychogenic theory (Staudenmayer et al., 2003b, p. 257)".

Since current empirical evidence for the role of psychological and psychopathological factors in IEI is accumulating, we will focus on psychological aspects of IEI.

3.2.3. Genetic findings in IEI

Investigating the etiology of IEI from a genetic perspective yielded at least two noteworthy findings: Firstly, MCS/IEI-cases have been shown to differ from controls in polymorphisms in drug-metabolizing enzymes which might be related to differences in the ability of the organism to decompose environmental chemicals (McKeown-Eyssen et al., 2004). Secondly, an increased prevalence of a polymorphism associated with panic disorder has been demonstrated in IEI supporting the notion that IEI might share a biological diathesis for panic disorder (Binkley et al., 2001).

3.2.4. Psychological mechanisms in IEI

In their extensive review on IEI/MCS, Labarge and McCaffrey (2000) differentiate three kinds of psychological mechanisms: cognitive influences, conditioning processes, and known psychiatric disorders. With respect to the last aspect (IEI as an atypical form of a known mental disorder) some researchers (e.g., Tarlo, Poonai, Binkley, Antony, & Swinson, 2002) favor a model of IEI in which odors act as conditioned stimuli that elicit panic-like reactions. In line with this hypothesis, a polymorphism associated with panic disorder has been found in connection with IEI (Binkley et al., 2001), and hyperventilation provocation tests have produced stronger reactions in people with IEI compared to control participants (e.g., Binkley & Kutcher, 1997). Additionally, anxiety sensitivity values, as an explanatory construct for the development of panic disorder, were increased in IEI (Caccappolo-van Vliet, Kelly-McNeil, Natelson, Kipen, & Fiedler, 2002). Others highlight parallels between IEI and depression (Schottenfeld, 1987), psychotic disorders (Hausteiner, Mergeay, Bornschein, Zilker, & Förstl, 2006), and traditional somatoform disorders or functional somatic syndromes (e.g., Barsky & Borus, 1999; Stewart, 1990a).

Regarding the involvement of conditioning processes in IEI and somatoform symptoms, Van den Bergh and colleagues have clearly demonstrated in a series of experiments that psychosomatic complaints can easily be associated with and subsequently triggered by unpleasant odors (Van den Bergh et al., 2001; Van den Bergh et al., 1997; Van den Bergh, Winters, Devriese, & Van Diest, 2002). Specific external information (e.g., warnings about environmental pollution; Winters, Devriese, Van Diest, Nemery, Veulemans, Eelen, Van de Woestijne, & Van den Bergh, 2003) and personal characteristics such as a high degree of negative affectivity (Devriese et al., 2000) seem to foster these conditioning processes. This learning mechanism may also underlie the Gulf War Syndrome (Ferguson, Cassaday, & Bibby, 2004). However, learning accounts of IEI that focus on odors as triggers of complaints cannot easily explain (a) why some people suffer from IEI without reporting a hypersensitivity to odorous agents and (b) why symptoms continue even after strictly avoiding supposed triggers (e.g., odors) of complaints.

The first notion of a substantial involvement of cognitive psychological aspects in IEI has often been the subject of elaborate speculations and sound theoretical considerations. The mechanisms discussed include selective attention and hypervigilance to physical symptoms, specific fear-networks and mental representations concerning IEI-trigger substances, and the operation of retrospective self-validations and false attributions (e.g., Barsky & Borus, 1999;

Bock & Birbaumer, 1998; Lange & Fleming, 2005; Staudenmayer et al., 2003b; Williams & Lees-Haley, 1993). Interestingly, these theoretical propositions did not stimulate experimental research in the involved areas of social, clinical, cognitive, and health psychology so far. Only a few studies have experimentally addressed cognitive variables regarding IEI symptoms. In a provocation test study with a non-clinical sample, expectations about the effects of a chemical agent systematically influenced both the report of symptoms and perceived irritation (Dalton, Wysocki, Brody, & Lawley, 1997). Barsky & Borus (1999) theoretically proposed somatosensory amplification (i.e., a self-perpetuating and self-validating circuit of body-focused hypervigilance, symptom perception and catastrophic interpretation) as the central pathogenetic mechanism in functional somatic syndromes e.g., IEI, chronic fatigue syndrome, and sick building syndrome. Although reasoning and indirect evidence presented by Barsky seems intuitively plausible and convincing, empirical and experimental results regarding the involvement of somatosensory amplification in people with IEI are rare so far.

In summary, current empirical and theoretical evidence suggests that IEI represents a complex psychophysiological disorder that might phenomenologically be considered as a new variant of somatoform disorders (Black, 2000; Bornschein et al., 2002; Pennebaker, 1994; Staudenmayer, 2000) or a functional somatic syndrome (Barsky & Borus, 1999). Three arguments are central to this standpoint: Neither dose-symptom dependency nor agent-symptom specificity have been documented so far (Staudenmayer et al., 2003b; Staudenmayer, Selner, & Buhr, 1993). Also, the overlap between IEI and general psychopathology is considerable: People suffering from IEI show elevated levels of anxiety, depression and somatization (Bailer, Rist, Witthöft, Paul, & Bayerl, 2004b; Bornschein et al., 2002; Simon, Daniell, Stockbridge, Claypoole, & Rosenstock, 1993) as well as typical dysfunctional cognitions and attribution styles of bodily symptoms related to panic disorder, somatization disorders and hypochondriasis (Poonai, Antony, Binkley, Stenn, Swinson, Corey, Silverman, & Tarlo, 2001). Moreover, the risk for a current and lifetime comorbid psychological disorder is increased in people with IEI (Bornschein et al., 2002; Fiedler & Kipen, 1997). Although it is likely that the etiology of IEI like other unclear (functional) syndromes will rely on complex multifactorial processes and interactions between psychological and biological factors, the current study will primarily focus on cognitive psychological aspects (e.g., selective attention and attribution processes) and their relation to symptom reporting.

3.2.5. A cognitive psychological / cognitive-behavioral approach to IEI

As outlined above, cognitive theories of medically unexplained symptoms or somatoform disorders stress the importance of constructs like negative affectivity (e.g., Watson & Pennebaker, 1989), dysfunctional cognitive (attribution) styles (Robbins & Kirmayer, 1991), anxiety sensitivity (Zvolensky & Forsyth, 2002), and increased symptom focused attention and amplification of somatic symptoms (Barsky, 1998; Brown, 2004; Hiller, Cuntz, Rief, & Fichter, 2001; Pennebaker, 1994; Rief, Hiller, & Margraf, 1998). The combination of these mechanisms into a vicious circle emphasizes the key role of reciprocal processes of increasing awareness *to* and catastrophizing misinterpretation *of* bodily symptoms. The resulting chronic state of hyperarousal generates and aggravates symptoms (e.g., either by physiologically decreasing the individual symptom perception threshold or by cognitively fostering availability of emotionally congruent symptom episodes in working memory) and may mislead patients into believing that they suffer from a severe illness (Barsky & Borus, 1999). In case of patients with a full-blown IEI those beliefs sometimes appear close to persecutory delusions. Although evidence for these mainly pathogenetic theories stems from traditional somatoform disorders, there is some experimental evidence supporting the importance of cognitive processes for the development and maintenance of IEI. Dalton and colleagues (1997) demonstrated in a provocation-test study with a non-clinical sample that expectations about the effects of a chemical agent systematically influence report of symptoms and perceived irritation. Additionally, Winters and colleagues (Winters et al., 2003) showed in an olfactory differential conditioning paradigm that experimentally presented media warnings regarding chemical pollution facilitated the acquisition and report of symptoms in response to unpleasant and pleasant odors previously paired with a CO2-challenge. Given this evidence for the involvement of psychological mechanisms, we suggest a hypothetical cognitive-behavioral model for the development and maintenance of IEI symptoms (Figure 3-1). The model is mainly informed by cognitive-behavioral approaches of MUS (e.g., Looper & Kirmayer, 2002), by the cognitive-psychological approach of Brown (2004), and the theoretical-cognitive model concerning the formation and maintenance of persecutory delusions (Freeman, Garety, Kuipers, Fowler, & Bebbington, 2002).

In the tradition of models from the realm of cognitive-behavior therapy (CBT; e.g., Brewin, 2006) we distinguish three levels, namely the vulnerability factors (level 0), the onset conditions (level 1), and the maintenance factors (level 2). The vulnerability or risk factors are

divided into unspecific factors that contribute to the development of MUS in general (like proposed in the models of Brown 2004, and Looper & Kirmayer, 2002) and specific vulnerability factors that foster IEI-specific symptoms and beliefs (e.g., chemical odor sensitivity, high suggestibility, and openness to experiences). According to the onset of IEI, the model assumes an interaction of several dysfunctional individual and external conditions: For instance, critical life events might produce medically unexplained symptoms (via the lack of coping strategies). In search of plausible and self-protective externalizing attributions offered by physicians or certain media, a person may develop the hypothesis to suffer from IEI. Under adverse conditions (e.g., maintained negative affect and arousal; inability to adequately perceive and regulate emotions) the initial hypothesis might become stronger and at the end form a threat belief similar to persecutory delusions (Freeman et al., 2002). As outlined by Freeeman et al. (2002), several cognitive factors contribute to the maintenance of the threat belief (i.e., in our case the belief to suffer from IEI). Among these cognitive processes is a general confirmation bias (i.e., selective search for confirming and inhibition of disconfirming evidence) as well as attentional and memory biases. In the case of IEI, specifically attentional processing and evaluation of external perceptions and bodily sensation will be guided by the belief to suffer from IEI. Accessibility or IEI-relevant memory structures should be enhanced, leading to a focusing of confirming evidence for the belief to suffer from IEI. The core feature of the proposed model is a vicious circle of selective attention toward threat related information (e.g., media reports), increased symptom focused attention (symptom perception), catastrophizing cognitions, and repeated attention toward external information as confirming explanations (“false attributions”). At least two attentional processes seem essential in such a model: Firstly, increased selective attention to somatic changes (non-specific bodily symptoms) and to environmental threat related information (e.g., potential IEI-trigger substances); secondly, catastrophizing cognitions about the non-specific symptoms (as signs of IEI) and the harmfulness of everyday environmental chemicals.

According to Karl Popper, a reasonable model or theory should be both bold and falsifiable to promote scientific progress. While boldness remains in part subjective, falsifiability is given by precise hypotheses that can by derived from the model and afterwards empirically disconfirmed. Although a complete test of the proposed model is beyond the scope of the current study, we would like to derive crucial hypotheses concerned with attentional processes during the maintenance of symptoms and beliefs and test them empirically in our two studies presented later. Our specific hypotheses are based on the general idea that IEI is a condition that is closely related *to* but *not* isomorphic with known

somatoform disorders. Consequently, people suffering from IEI should reveal both similarities as well as differences compared to people with classic somatoform disorders or MUS without IEI-specific attributions. Our specific hypotheses derived from the model (Figure 3-1) are:

1. People with IEI should demonstrate similar vulnerability factors as people with traditional somatoform disorders, such as elevated levels of trait anxiety, negative affectivity, and dysfunctional beliefs regarding body and health.

2. People with IEI should specifically be marked by elevated levels of (hypnotic) suggestibility and openness to new and unusual experiences. The personality trait "absorption" postulated in the personality framework of Tellegen represents a potential indicator for this phenomenon.

3a. People with IEI compared to people without IEI or a somatoform disorder should show an attentional bias toward (a) unspecific bodily symptoms and (b) suspected IEI-trigger substances.

3b. In contrast, people with typical somatoform disorder compared to non-somatoform controls should demonstrate and attentional bias toward symptoms, but not IEI triggers.

4a. As evidence of catastrophic interpretation processes people with IEI should show negative explicit and implicit evaluations of IEI-trigger substances and somatic symptoms compared to people without IEI.

4b. People with a somatoform disorder but without IEI should present negative implicit and explicit evaluations of symptoms only.

5. Irrational (or overvalued) beliefs/ideas of IEI participants might be based on altered memory structures: therefore, people with IEI should show elevated memory performance for IEI-trigger words compared to people without IEI.

The hypotheses 3, 4, and 5 can be tested directly with different experimental procedures for the assessment of attentional and evaluative processes. We will outline briefly the most prominent experimental paradigms in the next chapter. In the following fifth chapter the aims and results of the first study will be presented.

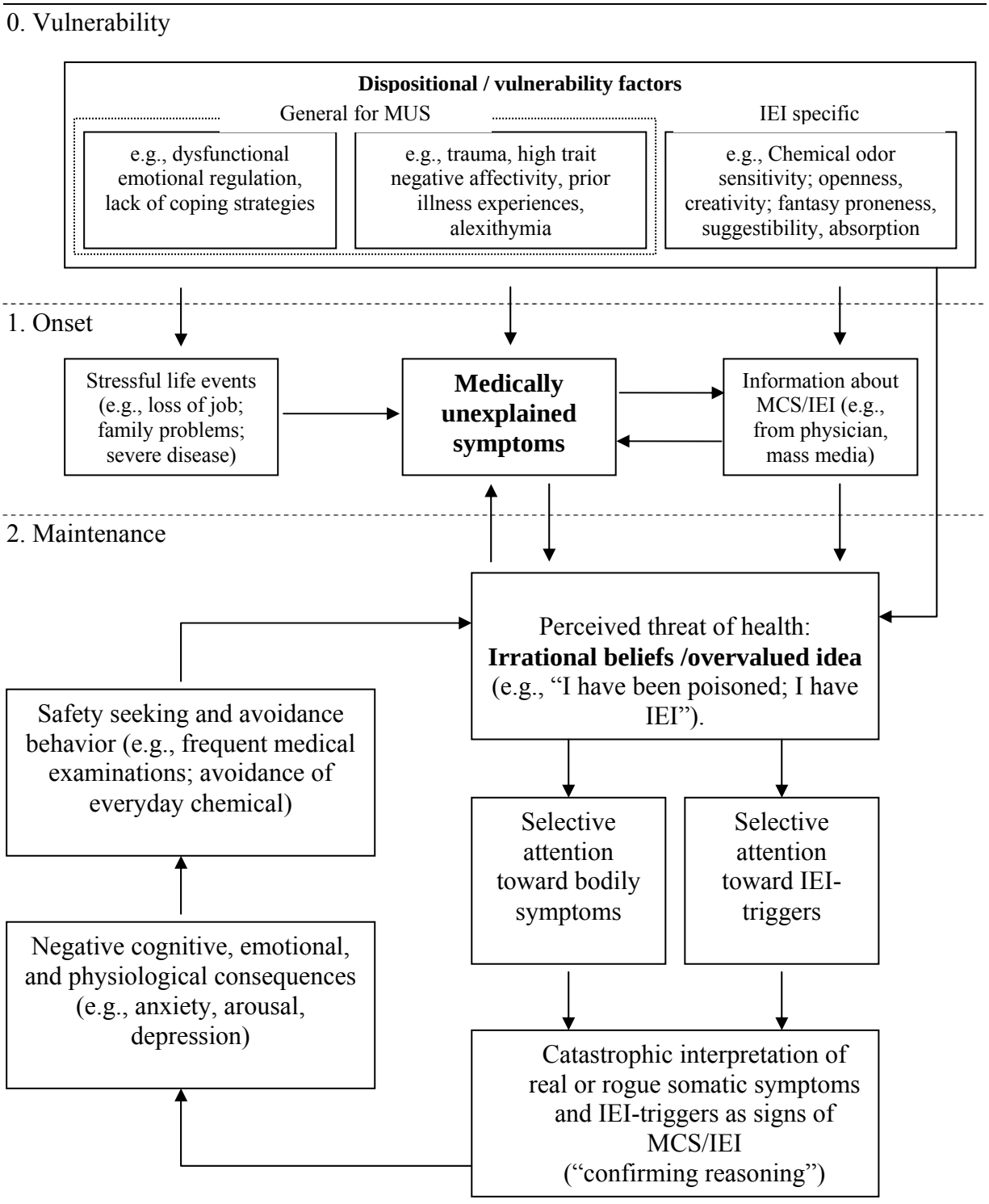

Figure 3-1: Hypothetical cognitive-behavioral model of the development and maintenance of IEI/MCS.

4 BIASED INFORMATION PROCESSING IN ANXIETY, DEPRESSION AND SOMATOFORM DISORDERS

The following section is conceptualized as a short introduction to the field of affect modulated cognitive processes in clinical psychology. For detailed reviews on this topic the interested reader might refer to Ehlers and Lüer (1996), Dalgleish and Watts (1990), Ott (1999), Wells and Matthews (1994), Mathews and MacLeod (1994), Becker and Rinck (2000), and Mogg and Bradley (1998), respectively. In general, cognitive and cognitive-behavioral notions of emotional disorders, such as anxiety disorders and depression, highlight the causal role of alterations in cognition-emotion interaction. Two main classes of alterations in processing routines, termed "biases", have been identified as being involved in normal and clinical variations in emotional reactivity, namely *attentional biases* and *memory biases*. Other biases that will not be included in our brief review include *interpretive biases* (e.g., Wilson, MacLeod, Mathews, & Rutherford, 2006) and *covariation biases* (e.g., Tomarken, Mineka, & Cook, 1989). The following paragraphs are by no means exhaustive. Rather, we will selectively focus on experimental paradigms and theoretical accounts that appear suitable for our endeavor to investigate cognitive abnormalities in typical and hypothesized atypical or new variants of somatoform disorders, such as IEI. Therefore we will briefly summarize exemplary findings on *cognitive biases*, mainly from the realm of anxiety and depressive disorders, followed by an introduction of the most popular theoretical accounts and experimental paradigms. The chapter ends with an overview of existing results regarding cognitive abnormalities in somatoform disorders.

4.1. Attentional biases in anxiety and depression

Traditionally, anxiety disorders have been attributed to alterations in early stages of selective attention (attentional bias), whereas depressive states have revealed abnormalities in later, more elaborative stages of processing (memory bias) (e.g., Ehlers & Lüer, 1996; Mineka & Sutton, 1992). In this sense, different phobias and anxiety disorders have been hypothesized to result from a *hypervigilance* (e.g., Eysenck, 1992) toward specific visual or verbal threat cues indicative of the major individual concerns e.g., a spider in spider phobics, an unfriendly or rejecting facial expression in social phobia, the term "heart attack" in panic patients and hypochondriacs, or a traumatic word or picture in patients with post-traumatic stress disorder. In contrast, more characteristic of depressive disorders is a selective and

preferential recollection of negative memory contents for instance during explicit memory tasks like free recall conditions or recognition tasks (Blaney, 1986). Biased recollection or retrieval processes may also underlie the clinical phenomena of rumination and worrying observed in different clinical conditions (Brewin, 2006). However, more fine-grained studies during the last years have demonstrated that the simple dichotomy of '*attentional bias but no explicit memory bias in anxiety'* and *'memory bias but no attentional bias in depression'* is too simple. For instance, in a study with different emotional facial expressions participants with depression but not participants of a control group with generalized anxiety disorder showed a specific attentional bias toward facial expression of sadness in a probe-detection task (Gotlib, Krasnoperova, Neubauer, Yue, & Joormann, 2004; but see also Mogg, Millar, & Bradley, 2000, for discrepant results). In specific phobias or anxiety disorder such an attentional bias is interpreted as a rather involuntary and fast acting phenomenon that occurs in the absence of a feeling of direct volitional control (e.g., a spider phobic person might automatically detect a spider in the corner of a room without a previous voluntary decision to scan the room for spiders). At least in specific phobias, visual or verbal representations of feared objects seem to automatically capture attentional resources, thereby disrupting or freezing current information processing and directing or prioritizing attention and memory resources to the feared object.

Several theories have been proposed to account for the above findings of biased attention. According to the schema theory proposed by Beck (e.g., Beck, 1976), cognitive biases as observed in clinical conditions like depression and anxiety result from the activation of specific cognitive schemata or specific semantic networks in the case of Bower's (1981) theory. Cognitive schemata or semantic networks are partly hypothesized as being the result of early learning episodes in childhood and include dysfunctional beliefs that are concerned with the anticipation of future catastrophes in the case of anxiety or personal failure and hopelessness in the case of depression. Once those schemas or semantic networks become activated by situational conditions they are hypothesized to guide or misguide cognition in a mood-congruent manner. According to these theories, different clinical conditions like anxiety and depression mainly differ in their specific schema contents. Differences in biased processing across anxiety and depression are therefore hard to reconcile with these "early" theories because they tend to make similar predictions for anxiety and depressive disorders with respect to biased information processing: As outlined by Mogg and Bradley (1998), the theoretical notions of Beck and Bower propose emotion congruent biases for both classes of emotional disorders, anxiety *and* depression. In contrast, empirical evidence has revealed

differences between patients with anxiety disorders and depressive states. These discrepant results and phenomenological differences between anxiety and depression have called for theories that make different hypotheses regarding cognitive biases in anxiety and dysphoria. One of the most influential models addressing those differences was proposed by Williams, Watts, Mac Leod, and Mathews (1988). Their model consists of two sequential stages that are both supposed to operate pre-attentively. During the first stage the threat value of a current stimulus is defined by the affective decision mechanism (ADM). In the second step, depending on the individual degree of trait anxiety, processing resources are either allocated to the source of the threat (in case of high trait anxiety) or away from the threat signal (in case of low trait anxiety). As outlined in the review by Mathews and Mackintosh (1998) especially one empirically replicated observation poses a problem to the Williams et al. model - people with clinical or sub-clinical forms of phobia or other anxiety disorders only reveal consistent attentional bias in stimulus competition conditions. Accordingly, anxiety does not seem to be associated with altered detection thresholds for single phobic stimuli (Becker & Rinck, 2004), but rather with a preferential attentional allocation to threat signals in light of conflicting stimuli or stimulus dimensions.

In contrast to the Williams et al. model that is mainly based on automatic processes of stimulus evaluation and resource allocation, Wells and Mathews (1994) stress in their model the importance of voluntarily adopted plans or goals, for instance a threat monitoring plan, which directs attentional resources to threat cues. Accordingly, and in contrast to the model of Williams and colleagues (1988), the phenomenon of biased attention is considered the result of top-down processing rather than a purely stimulus driven bottom-up incidence (Mathews & Mackintosh, 1998). Accordingly, as formulated in a review on dual-process theories of the mind by Feldman Barret, Tugade, and Engle (2004), there is no longer a contradiction between goal-directed attention and automatic allocation of attention to certain stimuli:

> "Moreover, if we accept the idea that controlled processing is not synonymous with conscious experience, then we are free to consider the idea that goal-directed attention may function like a preconscious filter that selects the focus of attention (and potentially what is available to consciousness). This idea is consistent with the emerging view that attention is captured automatically by stimulus features primarily when there is some goal-directed attentional preparation to allow this. As a result, controlled processing may not be merely reversing the effects of automatic processing, but it may also prevent (or allow) the expression of attention on representations that were activated in a stimulus-driven way. As long as one has a processing goal (like an egalitarian goal to prevent stereotyping, for example), as well as the WMC to deploy

> goal-directed attentional effects, that processing goal can be enacted (Feldman Barret, Tugade, & Engle, 2004, p. 564)."

In the above statement, the goal-directed attentional preparation would be a function of the secondary attentional system of the model proposed by Brown as presented in chapter 2 of this paper. Based on this position, a clear and simple distinction between automatic and controlled, and conscious and unconscious processes is blurred.

The model presented by Mathews and Mackintosh (1998) is based on empirical findings that attentional biases mainly occur in situations marked by multiple stimulus attributes (e.g., word color and semantic meaning in case of the emotional Stroop task) that compete for attentional resources. The model is based on the assumption that a threat evaluation system (TES), which is comparable to the affective decision mechanisms (ADM) of the Williams et al. model presented above, rapidly (i.e., prior to awareness) determines the threat level of a given stimulus. According to existing knowledge, the significant threat value that leads to the dominance of the TES over the effortful task demand unit resulting in biased attention may either arise from phylogenetic/biologically determined preparedness and evolutionary relevance (e.g., Le Doux, 1996; Öhmann, Flykt, & Esteves, 2001; Seligman, 1971) or from (individual) learning episodes (Blanchette, 2006; Richards & Blanchette, 2004). Although the debate, in how far these attentional biases are limited to evolutionary-relevant phylogenetic prepared stimuli goes on (e.g., snakes, spiders), experimental evidence suggests, that "modern" threatening stimuli (like guns and syringes) similarly have the potential to bias attention (Blanchette, 2006).

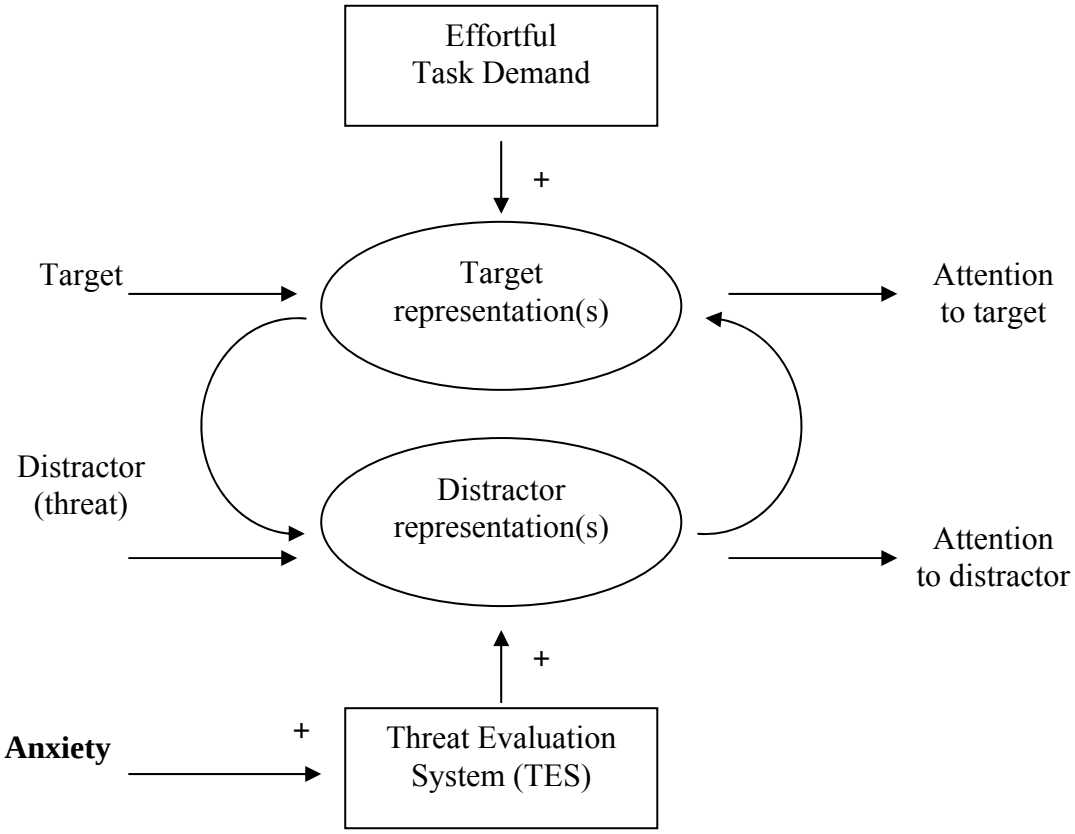

Figure 4-1: Schematic outline of the model proposed by Mathews and Mackintosh (1998; Figure 4, p. 547).

The finding, that alterations in attentional processes are mostly detected in stimulus competition or conflict situations is crucial for the design of experimental paradigms suitable for the detection of individual differences regarding affect modulated attentional processes. Two prominent classes of experimental paradigms exist for the assessment of attentional biases, namely *interference paradigms* like the emotional Stroop task (e.g., Ehlers, Margraf, Davies, & Roth, 1988; Watts, McKenna, Sharrock, & Tresize, 1986; Hope, Rapee, Heimberg, & Dombeck, 1990) and *facilitation paradigms* like the dot probe or probe detection task (MacLeod, Mathews, & Tata, 1986). Although the dot probe paradigm was often considered as a purer measure of attentional bias compared to the emotional Stroop paradigm (e.g., Mineka & Sutton, 1992), the latter seems to produce more robust and reliable results.

4.1.1. The Emotional Stroop Paradigm

The emotional or modified Stroop task represents the most prominent experimental paradigm designed to assess the (automatic) allocation of selective attention towards

individually salient and mostly negative or threatening information (for an extensive review see Williams, Mathews, & MacLeod, 1996). Based on the classical Stroop color-naming task (Stroop, 1935), a prototypical emotional or modified Stroop task uses stimulus words with two varying attributes, namely color and word content. Test participants are required to name or identify the color of a presented stimulus word as fast and accurately as possible by simultaneously ignoring the word content. The emotional Stroop effect now concerns the observation, that vocal and manual[1] answer latencies are slowed by about 10 to 150 ms in the case of emotional or individually relevant compared to neutral or irrelevant words. In this sense, slowed color-naming latencies to emotional or concern related words in comparison to neutral words have been considered as an indicator of an *attentional bias* (Williams et al., 1996). Despite its prominence, the underlying mechanisms responsible for this characteristic slowdown associated with self-relevant, emotionally negative stimulus material are not yet completely understood. Recently, a debate has started whether the slowdown observed with emotion-laden stimuli corresponds at all to the classic color word Stroop effect. Some researchers consider the two phenomena totally distinct (Algom, Chajut, & Lev, 2004; McKenna & Sharma, 2004) and the term "emotional Stroop" as misleading, others highlight parallels between the classical and the emotional Stroop (Dalgleish, 2005). McKenna and Sharma (2004) regarded the term "emotional intrusion effect" as more adequate to describe this phenomenon. Currently it might be reasonable to distinguish between the paradigm itself ("emotional Stroop task") and the resulting indicator of selective attention, which is an emotional intrusion effect in nature. Further, some authors (McKenna & Sharma, 2004) recommend differentiating two distinct components of the emotional Stroop effect: A slow component and a fast component. However, despite some technical and theoretical problems with the EST, there is convincing evidence for the emotional connotation of stimuli to give rise to an emotional intrusion effect: In people with high trait anxiety, formerly neutral stimuli (non-words) elicit interference after a classical conditioning procedure (Richards and Blanchette, 2004). Earlier studies questioned the preconscious nature of the effect (Thorpe & Salkovskis, 1997), but conscious processing of the respective stimuli is not necessary for an emotional intrusion effect, and subliminal presentation may even raise its validity (Putman, Hermans, & van Honk, 2004). With the emotional Stroop task, biases of selective attention have been studied extensively in many clinical populations (e.g., eating disorders, personality disorders, drug dependencies, etc.; Williams et al., 1996). For an extensive review on

[1] Although some researchers hypothesized that emotional Stroop effects might be restricted to vocal responses (Sharma & McKenna, 1998), others could demonstrate the effect also with manual key press responses (Brown & Besner, 2001).

emotional Stroop effects in addiction disorders see Cox, Fadardi, and Pothos, 2006. Although different hypotheses concerning the nature of the emotional intrusion effect have been proposed in the past (e.g., emotionality hypotheses, self-relevance hypotheses, Mathews & Klug, (1993), and threat hypotheses, McKenna & Sharma, 1995), empirical findings have clearly demonstrated that neither the general emotionality of the words nor their status as personal concerns are sufficient to produce the characteristic intrusion effects obtained with threatening stimuli (McKenna & Sharma, 1995). At present, the emotional intrusion effect of linguistic stimuli is best considered as reflecting a cognitive threat response or fear-driven bottom-up interruption of ongoing information processing with evolutionary adaptive value (Algom et al., 2004; Isenberg, Silbersweig, Engelien, Emmerich, Malavade, Beattie, Leon, & Stern, 1999; Kindt & Brosschot, 1997; McKenna & Sharma, 2004; Wyble, Sharma, & Bowman, 2005).

> "Overall, the results clearly favor the threat hypothesis over the emotionality and self-relevance hypotheses. However, one caveat may be worth noting. Although the results rule out self-relevance as a sufficient factor in producing interference, it may remain the case that self-relevance is a necessary factor. Because one of the essential ingredients of threat is self-relevance, yet the reverse is not true, self-relevance may be a necessary but not a sufficient factor." (McKenna & Sharma, 1995; p. 1604)

The emotional Stroop task has also been used in a few studies on somatoform disorders. We will present the corresponding results in more detail at the end of this chapter when summarizing evidence for cognitive biases in somatoform disorders (cf. paragraph 4.3).

4.1.2. The Dot-Probe Paradigm

The dot probe paradigm represents an additional reaction-time task to map processes of selective orientation or more precisely an "attentional shift" toward threatening stimuli. In contrast to the emotional Stroop task, the relevant indicator of selective attention here is a speeded rather than a delayed response to a neutral stimulus (probe) appearing shortly after and in the same location of a preceding threatening stimulus (either word or picture). Although much of the published research with the dot probe task has been conducted in non-clinical student populations with varying levels of state and trait anxiety (e.g., Bradley, Mogg, Falla, & Hamilton, 1998), there is evidence of an attentional bias towards threatening information in clinical anxiety populations like for example generalized anxiety disorder (Bradley, Mogg, White, Groom, White, & de Bono, 1999) and social phobia (Asmundson &

Stein, 1994; Musa, Lépine, Clark, Mansell, & Ehlers, 2003). However, the direction of the effect in the dot probe task (facilitation vs. slowdown) has proven as less clear than in the emotional Stroop task. Studies in different domains e.g., social phobia and social anxiety (Chen, Ehlers, Clark, & Mansell, 2002; Mansell, Clark, Ehlers, & Chen, 1999) yield avoidance reactions (slowed probe detection in former threat stimuli locations) rather than vigilance or facilitation effects. To our knowledge no study has so far employed this paradigm in a group of somatoform patients.

4.2. Memory biases and schemata in anxiety and depression

In contrast to attentional biases, memory biases refer to altered processes in comparatively "later" elaborative stages of information processing, such as retrieval or recollection. In contrast to attentional biases that seem prototypical for anxiety disorders, *memory biases* toward mood-congruent stimuli, as proposed by schema or semantic network theories (Beck, 1976; Bower, 1981), have mainly been observed in the realm of depression and non-clinical dysphoria (Blaney, 1986; Ehlers & Lüer, 1996; Bradley, Mogg, & Williams, 1995). These observations are in line with the notion of Williams et al. (1988; 1997), that people with elevated trait anxiety or an anxiety disorder should be impaired in early, attentional but not later, interpretive or elaborative stages of information processing (for the original distinction between *integrative* and *elaborative* memory processes see Graf & Mandler, 1984). Accordingly, elevated anxiety was hypothesized to coincide with alterations regarding implicit memory processes (e.g., tachistoscopic identification or word stem completion) and normal performance in explicit memory tests like free recall or recognition paradigms. Whereas some empirical results are in accordance with this hypothesis (e.g., Bradley et al., 1995; MacLeod & McLaughlin, 1995; Mathews, Mogg, May, & Eysenck, 1989), other findings blur the proposed distinction. As outlined in the review by Becker and Rinck (2000) it seems likely that different kinds of anxiety disorders are associated with an explicit memory bias (e.g., panic disorder, post-traumatic stress disorder), whereas others are not (e.g., generalized anxiety disorder). In contrast, patients with depressive disorders especially seem to better recall negative information concerning the self (self-referent recall bias; Bradley & Mathews, 1983). In general, Hartlage, Alloy, Vazquez, and Dykman (1993) state in their review that depressive states interfere with *effortful* task demands, whereas automatic processes mostly remain intact. However, the question remains in how far such an explicit memory bias in depression represents an enduring phenomenon (in the sense of a

vulnerability factor) or rather a state phenomenon depending on the current negative mood (e.g., Hedlund & Rude, 1995; Teasdale & Dent, 1987). Next, we will outline some prominent experimental paradigms for the assessment of altered memory processes.

4.2.1. Explicit and implicit memory tasks

Different experimental paradigms have been used to study *memory biases* in anxiety and depression. The most prominent paradigms are word stem completion and tachistoscopic identification tasks as measures of implicit memory processes, free recall, and cued recall paradigms as examples of explicit memory tasks. Both implicit and explicit memory tasks consist of two stages, an encoding stage in which certain stimuli (words or pictures) are sequentially presented for the first time (e.g., in connection with a word color naming task). In a second stage, the effect of the pre-exposure during the encoding phase is tested either implicitly or explicitly. In explicit memory tasks, participants are instructed to consciously recollect the referring stimuli from memory, either via free recall or in a recognition task. In contrast, within implicit memory tasks (e.g., tachistoscopic identification tasks or word stem completion tasks) participants are not instructed to recollect previous items consciously but they are confronted with a seemingly unrelated task. In this context, the indicator of implicit memory processes is the *involuntary* facilitation of processing in the light of a pre-exposed stimulus compared to a not pre-exposed stimulus. This clear-cut distinction between implicit and explicit processes according to the experimental paradigm used (e.g., word stem completion as an implicit procedure versus free recall or recognition as explicit procedures) bears methodological problems. As mentioned by MacLeod and McLaughlin (1995), for instance, especially the word stem completion task has been considered a rather impure measure of implicit memory because it seems substantially confounded with explicit processes. Accordingly, implicit and explicit paradigms are no longer considered pure measures of a single underlying (either implicit or explicit) process. In contrast, the process dissociation framework (e.g., Jacoby, 1991; Ott, 1999) considers implicit and explicit processes related phenomena that simultaneously occur in different tasks. The process dissociation procedure offers a way of dissociating implicit and explicit influences within a single memory task.

Because the assessment of pure implicit memory biases is methodologically rather complex, and available results from the realm of somatoform disorders presented above point to the relevance of *explicit memory processes*, we decided to use an explicit memory test in

ourfirst study. In order to circumvent methodological problems associated with a free-recall procedure like the problem of ceiling effects in case of many briefly presented stimuli and the problem of dissociating individual response criteria (Hock & Egloff, 1998), we have chosen a recognition procedure that will be outlined in detail later on.

Whereas experimental paradigms designed for the assessment of (implicit and explicit) memory biases only indirectly assess schemata or activated semantic networks, recently another experimental paradigm has been introduced as a better proxy to directly assess components (i.e., single semantic associations) of hypothesized schemata.

4.2.2. Experimental assessment of cognitive schemata

Although cognitive schemata, maladaptive beliefs or specific semantic networks in memory are hypothesized as crucial vulnerability factors or exploratory constructs within influential theories in the realm of clinical psychology (e.g., Beck, 1976; Bower, 1981; Clark, 1986), experimental evidence for the existence of those schemata has remained weak. Recently, a prominent experimental paradigm, the implicit association test (IAT), that has been developed in social psychology as an implicit measure for the assessment of individual attitudes, stereotypes and prejudices (Greenwald, McGhee, & Schwartz, 1998), was adapted to clinical psychological research questions. In the clinical context, the IAT is hypothesized as a proxy for implicit schemata in memory that guide perception and evaluation of internal and external stimuli. Teachman (2005), for instance, used the IAT to study dysfunctional and implicit semantic associations in people with high values on anxiety sensitivity. Results confirmed the hypothesis of an implicit panic specific self-schema in people high on anxiety sensitivity (AS) as compared to low AS participants. Similarly, using the IAT, specific implicit fear associations could be demonstrated in spider phobic individuals (Ellwart, Rinck, & Becker, 2006). Interestingly, indicators derived from the IAT significantly and incrementally predicted performance in a spider related behavioral avoidance test after controlling for self-report measures of spider phobia. As outlined by De Houwer (2002) and Teachman (2005), it is worth noting that the IAT *only* measures the strength of associations between concepts in memory. Since schemata or dysfunctional beliefs theoretically constitute more complex structures mostly comprising many different single associations, the IAT does not offer a direct test of maladaptive cognitive schemata. Nevertheless, existing results propose that the IAT and related task variants such as the extrinsic affective Simon tasks (EAST), which we will describe in more detail in the second study, offer interesting insights

into implicit evaluation or interpretive processes beyond explicit self-report instruments. Since cognitive biases have so far mainly been studied in anxiety and depressive disorders, little is known about such processes in somatoform disorders. Existing empirical evidence will be reviewed in the following section.

4.3. The role of cognitive biases in somatoform disorders

Although cognitive phenomena like the attentional bias, memory bias and the (implicit) interpretive bias have genuinely been studied in clinical and sub-clinical forms of anxiety and depression, these processes are hypothesized to play a key role in cognitive-behavioral or behavioral medical approaches to somatoform disorders (e.g., Brown, 2004; Kirmayer & Taillefer, 1997; Looper & Kirmayer, 2002; Rief & Hiller, 1998). Accordingly, a body and symptom focused attentional style in combination with a biased catastrophizing attributional style should maintain and increase illness worries in specific, and negative affectivity in general. However, despite the broad acceptance of cognitive-behavioral theories and their strong impact on treatment programs, comparatively few studies have so far explicitly studied processes of selective attention and cognitive biases in somatoform disorders, with the exception of pain disorder (e.g., Roelofs, Peters, Zeegers, & Vlaeyen, 2002) and elevated health anxiety in university student populations (Lecci & Cohen, 2002; Owens, Asmundson, Hadjistavropoulos, & Owens, 2004).

In another study, Lupke and Ehlert (1998) compared attentional biases towards health threatening words in patients with somatoform disorders and patients with a somatic disorder. The authors could demonstrate that only the somatoform patients but not the somatic control patients took disproportionately longer to color name health threatening words compared to neutral words (attentional bias). This attentional bias could be demonstrated to be independent of the comorbidity with panic disorder. Furthermore, the attentional bias in somatoform patients was found significantly reduced after a cognitive-behavioral or behavioral-medical treatment program. The study, therefore, proposes that attentional biases are specific to psychosomatic or somatoform conditions and that adequate treatment programs are associated with a reduction in biased attention allocation. However, as Lupke and Ehlert (1998) included not only specific physical threat words in their study but also rather non-specific negative words (like blood, death, casket, cancer), the question if somatoform patients specifically show an attentional bias toward physical threat words or just generally direct attention toward negative word stimuli still remains. Another critical issue in their study might represent the

use of a card version of the emotional Stroop task. It remains unclear if their results are replicable with a modern computerized version of the emotional Stroop task.

In another experimental study, Lim and Kim (2005) have compared patients with panic disorder, somatoform disorder, and depressive disorder according to several cognitive biases (memory biases and attentional biases). In line with the theories presented above, interesting differences under certain task conditions were found among the three disorder groups: Patients with panic disorders showed an attentional bias to physical threat words (e.g., injury, seizure, inflammation) and generally negative words (e.g., mistake, fault, hostility) only under the very brief, subliminal stimulus presentation condition in an emotional Stroop task. No evidence for such an attentional bias under supraliminal presentation conditions for panic patients was found. In constrast, performance of somatoform patients revealed the opposite pattern of results with no attentional bias for physical threat words under subliminal conditions and evidence of an attentional bias when physical threat words were presented supraliminally. In line with the content specificity hypothesis, people with depression demonstrated a supraliminal attentional and an explicit memory bias toward negative stimuli, but not physical threat words. A measure of implicit memory bias (tachistoscopic indentification tasks) did not show any group specific effects. This pattern of results nicely fits information processing theories (e.g., Williams et al., 1988, 1997) that propose processing biases in anxiety disorders in early, pre-attentive, and integrative levels of processing and biases in depression in later, post-attentive, and more elaborative phases. With regard to somatoform patients, Lim and Kim's results propose that (a) there is an (supraliminal) attentional bias and an explicit memory bias in somatoform patients, (b) that the two biases are specific for physical threat words and do not generalize to negative words, and (c) that neither patients with panic disorder nor depression show a similar supraliminal attentional bias or explicit memory bias toward physical threat words.

Regarding the existence of a memory bias for disorder related stimuli in somatoform disorders Pauli and Alpers (2002) compared patients with and without somatoform disorders from a private medicine practice on experimental measures of a memory bias (free recall and recognition memory of pain words, neutral words, and positive and negative words). Results revealed a better free recall of pain words and a poorer recall of positive words in patients with hypochondriasis compared to non-hypochondriasis patients. In another study on information processing abnormalities in people with medically unexplained conditions, namely the chronic fatigue syndrome (CFS), Moss-Morris & Petrie (2003) found evidence for altered interpretive processes in a phonological ambiguous word cue task (e.g., week/weak or

vein/vain) for patients with CFS. However, in contrast to the expectations of the authors no attentional bias for somatic words could be observed in this study. Accordingly, there is at least some empirical evidence suggesting the existence of specific attentional and memory biases not only in patients with anxiety or depressive disorders, but also in patients with a somatoform disorder or medically unexplained symptoms. Results of Lim & Kim (2005) and Pauli and Alpers (2002) propose that cognitive biases in somatoform disorders might be differentiated from attentional biases in anxiety disorders (like panic disorder) with regard to their time course. Whereas anxiety disorders (like panic disorder) seem to be marked by very fast, subliminal detectable biases, cognitive abnormalities in somatoform patients likely affect later post-attentive phases of information processing similar to depressive disorders and conditions marked by dysphoria.

Within the following first study we will therefore primarily focus on the question: Do people with IEI show similar abnormalities with regard to cognitive biases (i.e., attentional bias and memory bias toward suspected IEI-trigger substances and somatic symptom words) that have been previously found in people with somatoform disorders. As prior studies have demonstrated those biases differentiate between patients with organic or somatic disorders and patients with somatoform conditions (Lupke & Ehlert, 1988; Pauli & Alpers, 2002), the existence of cognitive biases in IEI would strengthen the hypothesis that IEI might best be understood as a new variant of somatoform disorders. In the second study, we will then refer to the question of specific implicit schemata in memory that might underlie the cognitive biases. As a proxy for those schemata or specific semantic associations in memory, the implicit association test (IAT) that was originally developed in social psychology, has recently been adopted to the realm of clinical psychology. We will use a similar experimental paradigm, the extrinsic affective Simon task, to replicate the findings regarding the attentional bias and to simultaneously test for the assumption of specific implicit evaluation processes both in IEI and SFD. To our knowledge, this is the first study that systematically analyzes cognitive biases in people with IEI by using prominent experimental paradigms.

5 STUDY 1: ATTENTIONAL BIAS AND MEMORY BIAS IN IEI AND SOMATOFORM DISORDERS

Based on the hypothesized importance of biases in information processing not only for the most frequent emotional disorders like anxiety and depression but also for somatoform disorders (e.g., Brown, 2004; Rief & Hiller, 1998) our first study aims at assessing abnormalities in *selective attention processes* and *explicit memory processes* toward disorder related stimuli in participants with traditional somatoform disorders (SFD), people suffering from IEI as a hypothesized new/modern variant of somatoform disorders, and control participants without SFD or IEI (CG). The specific aims and hypotheses are based on our hypothetical cognitive-behavioral model of IEI (chapter 3). In the following paragraphs we will outline the specific aims of the study, the methodology, and the corresponding results in greater detail.

5.1. Aims and hypotheses of study 1

The first study is designed to assess cognitive markers related to medically unexplained symptoms in two samples, namely in people with somatoform disorder (SFD; according to DSM-IV) and in people with IEI. The major aims of the current study are twofold: Firstly, we tried to demonstrate selective attention and explicit memory biases toward IEI-trigger words, non-specific (physical) symptoms and specific emotional evaluations of these stimuli in participants fulfilling criteria for IEI. Secondly, we tested the specificity of these effects by comparing them with a group of patients with a clear defined somatoform disorder (according to DSM-IV criteria) and without an IEI related, environmental symptom attribution style. Both groups were compared with non-somatoform and non-IEI control participants. We expected group differences in two stages of cognitive processing, namely attention allocation and retrieval. Our main hypotheses were that participants with IEI but not the SFD and control participants would show an enhanced emotional intrusion effect for IEI-trigger words (slower reaction times in color-naming IEI-trigger words in comparison to neutral words in the emotional Stroop task) as evidence for a prioritized processing of these stimuli and that IEI and SFD participants but not the control group would show an enhanced emotional intrusion effect for words representing non-specific symptoms. Concomitantly, we hypothesized an attentional bias toward IEI-trigger words in participants with IEI (faster detection of probes replacing IEI-trigger words) but not the other

two groups and a corresponding bias toward symptom words in IEI and SFD but not the control group. Finally, we expected a memory bias (better recognition) for IEI-trigger words exclusively in IEI-participants and a memory bias for symptom words in both IEI and SFD-participants without IEI. As for the explicit emotional evaluations of the verbal stimuli we expected a similar pattern of results regarding the valence and arousal ratings of IEI-trigger words and typical symptom words. In summary, we expected specific (regarding IEI-trigger words) as well as non-specific (regarding symptom words) abnormalities in measures of selective attention, recognition memory, and emotional judgment in IEI compared with a somatoform control group without IEI and a second non-somatoform and non-IEI control group.

Apart from the experimental measures and in accordance with the hypothetical model of IEI presented in chapter 3, we expected similar values on self-report measures of somatoform symptoms, and proposed vulnerability factors like negative affectivity, trait anxiety, and dysfunctional beliefs regarding body and health between participants with IEI and SFD. However, since people with IEI are marked by idiosyncratic very specific and comparatively unusual symptom attributions and beliefs, we expected that people with IEI might show elevated levels of (hypnotic) suggestibility and openness to new and unusual experiences. The personality trait “absorption” postulated in the personality framework of Tellegen represents a potential measure of this domain.

5.2. Methods

This study was approved by the Ethics Committee for Clinical Research of the medical faculty at the University of Heidelberg, Germany.

5.2.1. Participants

Participants were selected for this study by a two-stage procedure. Stage one entailed a cross-sectional questionnaire screening of 970 adults. The screening package included a self-report questionnaire for chemical odor sensitivity (COSS; Bailer, Witthöft, & Rist, 2006b), two somatic symptom questionnaires (Patient Health Questionnaire [PHQ] somatization module; Spitzer, Williams, & Kroenke, 1999; SCL-90R somatization scale; Franke, 1995), and a disease check list. On the basis of their screening results, individuals were invited to take part in a further study if they fulfilled any of the following three criteria: (a)

hypersensitivity to environmental chemicals (defined as COSS scores >34 for women and >27 for men, corresponding to the upper 10% of the gender-specific distribution of COSS scores of the normative population), (b) presence of typical somatoform symptoms (defined as a positive screening result in the somatization module of the PHQ), and (c) neither presence of chemical sensitivity (COSS scores <35 for women and <28 for men) nor of somatoform symptoms (a negative screening result in the PHQ somatization module). Of those screened, 174 participants agreed to take part in the study. Several participants were excluded because of the presence of a psychotic disorder (n = 1), substance-use associated disorders (n = 3), noncompliance (n = 1), or missing inclusion criteria (n = 3). Eight participants were excluded because of the general exclusion criteria (aged < 18 or > 65 years, organic brain disease, present or past psychotic disorder, somatic disease that could account for the bodily complaints, substance-associated disorders, or noncompliance). Those who completed the entire study were paid 60 Euros ($72.00). All participants provided written informed consent. During stage one of the selection procedure participants were recruited from several sources. IEI and somatoform participants were recruited from polyclinics of environmental medicine, psychiatry, and psychosomatic medicine at the University of Heidelberg (Germany), and by advertisements in local newspapers asking for volunteers who were either especially sensitive to environmental chemicals or suffering from medically unexplained physical symptoms. The control subjects were recruited from a polyclinic of dental medicine (patients who attend to the polyclinic for a routine check-up and not for complaints which could be suspected to be somatoform), and by advertisements in local newspapers and in health centers asking for participation in an environmental health study.

5.2.2. Assignment to experimental groups

Final experimental group membership was assigned at Stage 2 of the recruitment procedure. 174 positively or negatively screened participants were evaluated with the Structured Clinical Interview for DSM–IV (SCID I; Wittchen, Wunderlich, Gruschwitz, & Zaudig, 1997). The criteria-based IEI diagnoses were reached following a second structured interview (Bailer, Witthöft, & Rist, 2006a). Participants who met the following three criteria were given the diagnosis of IEI: (a) reporting at least three symptoms that have been experienced during the past 6 months, (b) naming at least three trigger substances that mostly or always provoke symptoms, and (c) avoiding at least three trigger substances mostly or always. The IEI-interview covered 15 characteristic trigger substances (e.g., car exhaust,

perfumes, pesticides) and 15 symptoms potentially linked to environmental chemicals (e.g., dry nose, smell sensitivity, muscle or joint pains). The participants were asked how often (0 = never, 4 = always) exposure to each substance provokes symptoms and how often they avoid that particular substance. Our case definition is similar to those used by Black, Doebbeling, Voelker, Clarke, Woolson, Barrett, & Schwartz (2000) and Nimnuan, Rabe-Hesketh, Wessely, & Hotopf (2001). As proposed by Nethercott et al. (1993) and the 1999 MCS consensus definition (MCS consensus definition, 1999), we included an additional criterion for chronicity (symptoms for more than 6 month) in order to identify more severe IEI cases. Six participants were excluded from the IEI group because they did not meet all interview-based IEI criteria, and 8 participants were excluded from the two control groups (6 SFD and 2 participants of the non-somatoform and non-IEI control group [CG]) who were diagnosed as being IEI. The final three groups consisted of (a) 54 participants with medically unexplained symptoms fulfilling IEI-case criteria, (b) 44 participants with medically unexplained symptoms who met DSM–IV criteria of a SFD but not the interview-based criteria of IEI (SFD-only group), and (c) 54 control participants free of both SFD and IEI diagnosis (nonsomatoform CG). The different steps of the recruitment and selection procedure are graphically summarized in Figure 5-1.

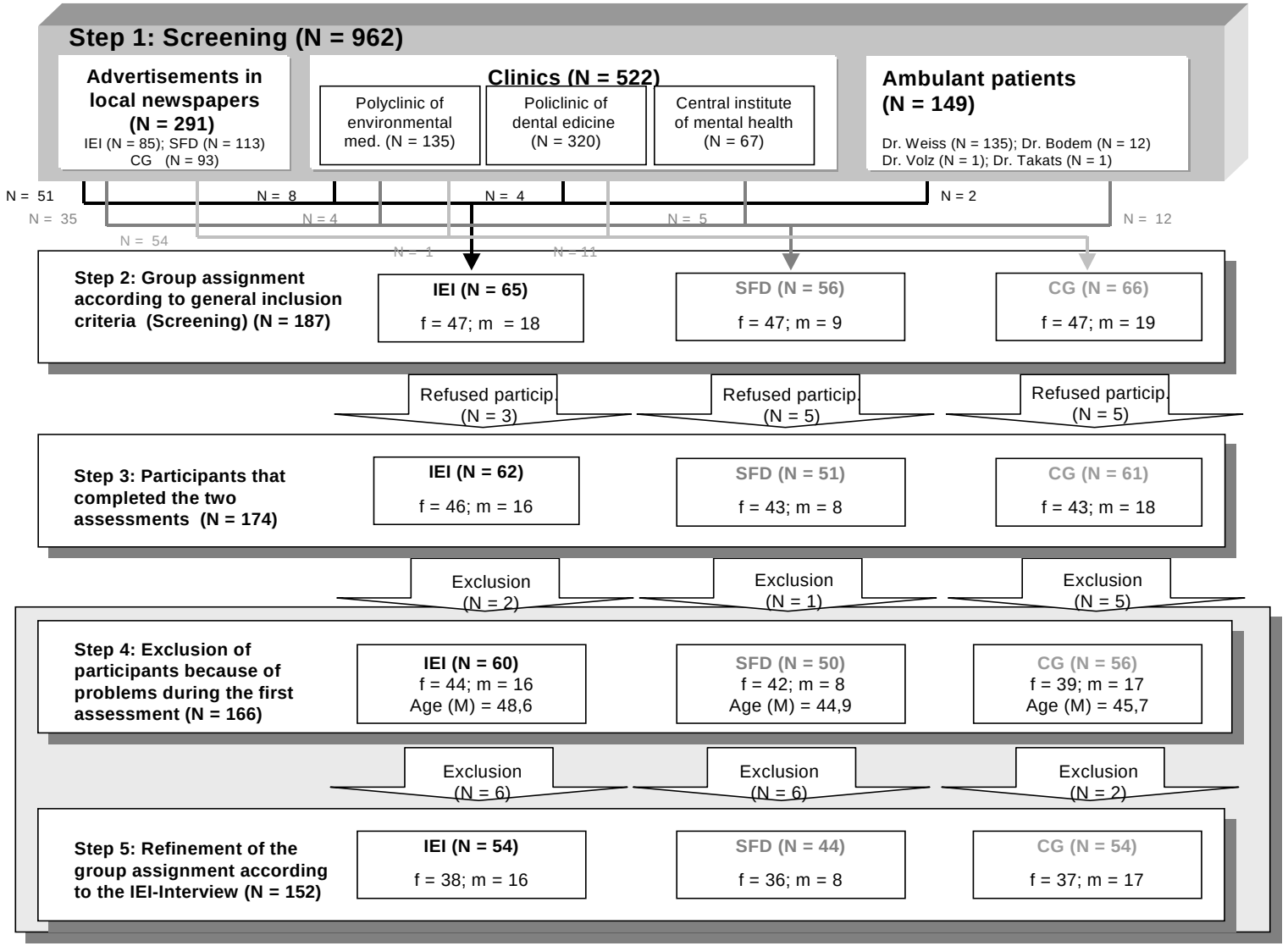

Figure 5-1: Steps of the recruitment and selection procedure (f = female; m = male).

5.2.3. Structured clinical interviews: SCID I and IEI-interview

The SCID I (Wittchen et al., 1997) was used to assess diagnoses of somatoform and of current affective and anxiety disorders according to the DSM-IV criteria. If criteria were met for both a somatoform disorder and a depressive or an anxiety disorder, both were diagnosed. The SCID interview included an additional section (from the extended German version of the Anxiety Disorders Interview Schedule, DIPS; Margraf, Schneider, & Ehlers, 1994) for conversion disorders. The criteria for all types of somatoform disorders were checked at least once except the unspecified category ”somatoform disorder NOS”. Specifically trained clinical psychologists (two PhD psychology candidates) administered the SCID and also the fully structured IEI interview (Bailer et al., 2006a). All interviewers had received one full week of training for the SCID-interview by a SCID expert. There was no special training procedure for the IEI-interview but both the IEI- as well as the SCID-interview were closely supervised by one of the senior researchers (Josef Bailer) with extensive clinical experience. The interviewers were encouraged to use all available sources of information (patient, laboratory findings, former medical diagnoses, and medical records) in rating the presence or absence of a symptom. In order to calculate interrater reliabilities of the two diagnostic instruments, 30 participants (10 of each experimental group) were evaluated by a rater and a co-rater using a conjoint interview. Intraclass correlation coefficients between raters 1 and 2 were as follows: r = .99 for the IEI trigger substances, r = .99 for the IEI symptoms, and r = .99 for IEI avoidance behavior. Kappa coefficients for the diagnoses of IEI were .92, for the category ”any somatoform disorder” 1.00 (range for single diagnoses: .78-1.00), for ”any current anxiety disorders” .83 (range: .65-1.00), and for ”any current depression” 1.00.

5.2.4. Self-report measures

The Chemical Odor Sensitivity Scale (COSS). The COSS (Bailer et al., 2004a; Bailer et al., 2006b) contains 11 statements describing strong physical responses (e.g., trouble in breathing, nausea, cough, dizziness) to the odor of common environmental chemicals (e.g., sprays, paints, cigarette smoke, cleansing agents, perfumes, exhaust fumes, gasoline). Participants rate on a 6-point Likert-type scale to which extent they show these responses (high scores indicating high chemical sensitivity). Reliability of the COSS has been

established across diverse samples with Cronbach's α between .89 and .93 (Cronbach's α in the current sample = .96). The COSS was factor analytically derived from the Questionnaire of Chemical and General Environmental Sensitivity (QCGS; Kiesswetter et al., 1999; Kiesswetter, Sietmann, Golka, Zupanic, & Seeber, 1997). In validation studies (Bailer et al., 2004a, Bailer et al., 2006b) the COSS was found to be dimensionally independent from respiratory symptoms not related to IEI triggers and from self-reported allergy to pollen and food. Evidence for convergent construct validity was gained with the Environmental Sensitivity Questionnaire (ESQ, see below) as a measure of cognitions of environmental threat.

Environmental Sensitivity Questionnaire (*ESQ).* The ESQ (Bailer, Rist, Rudolf, & Staehle, 2000; Bailer, Rist, Rudolf, Staehle, Eickholz, Triebig, Bader, & Pfeifer, 2001) contains a 10-item list of more or less harmful dental and environmental entities (e.g., electrosmog, radioactivity, harmful substances in air and water and dental filling materials). Participants are asked to judge the damaging effect of these agents on their health. The scale has shown adequate internal consistency (Cronbach's α between .86 and .89; current sample Cronbach's α = .91).

Cognitions About Body and Health Questionnaire (*CABAH).* The CABAH (Rief et al., 1998) assesses cognitive styles, attitudes, and interpretations of body perceptions typically found in patients with somatoform disorders. This 31-item questionnaire consists of five scales, based on factor analyses: Catastrophizing Interpretation of Bodily Complaints (e.g., "Red blotches on the skin are a threatening sign of skin cancer"; Cronbach's α is .75 in the current sample); Autonomic Sensations (e.g., "When I take a bath I often feel how our heart is beating"; Cronbach's α is .65 in the current sample); Bodily Weakness (e.g., "I m physically rather weak and sensitive"; Cronbach's α is .87 in the current sample), Intolerance to Bodily Complaints (e.g., "I consult a doctor as soon as possible when I have bodily complaints"; Cronbach's α is .63 in the current sample), and Health Habits (e.g., "I'm always careful to live really healthy"; Cronbach's α is .64 in the current sample). The CABAH scales reliability and validity have been investigated in earlier studies (Rief et al., 1998; Hiller et al., 2001).

The Tellegen Absorption Scale (TABS). Absorption represents a hypothetical personality dimension (trait) that was originally proposed by Tellegen and Atkinson (1974)

and is part of the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982). Absorption refers to the "readiness for experience of deep involvement, a heightened sense of the reality of the attentional object, an imperviousness to normally distracting events, and an appraisal of information in unconventional and idiosyncratic ways" (Roche & McConkey, 1990; p. 91). The TABS was originally designed to assess individual differences regarding hypnotizability and imaginative capability. Therefore, the TABS asks for synesthetic experiences, day dreaming activity, deep involvement in fantasy, and other variations of perception and altered states of consciousness within a non-clinical range that are marked by the absence of meta-cognitive activity (Ritz & Dahme, 1995). The TABS is the most widely used instrument for the assessment of absorption (Roche & McConkey, 1990) and comprises 34 items (German adaptation by Ritz & Dahme, 1995). Although different subscales within the 34 items have been originally proposed, no satisfactory multi-factor structure could be established. Large to medium sized correlations as evidence of convergent validity of the TABS were found with measures of fantasy proneness, openness to experiences, hypnotic susceptibility, and the ability to recall dreams (Challis & Stam, 1992; Roche & McConkey, 1990). Furthermore, the TABS correlated substantially with measures of spirituality (Hyland, Geraghty, Joy, & Turner, 2006) and is theoretically proposed to measure dissociative tendencies (e.g., Holmes, Brown, Mansesll, Fearon, Hunter, Frasquilho, & Oakley, 2005). Interestingly, absorption was found to be almost uncorrelated with other personality dimensions such as extraversion-introversion and emotional stability-neuroticism (e.g., Radtke & Stam, 1991). The relevance of absorption for somatoform conditions has already been proposed by Kirmayer, Robbins, and Paris (1994): "Absorption may make individuals more liable to focus attention on symptoms and more vulnerable to suggestions that induce illness anxiety (p.125)." Cronbach's α for the 34 items of TABS is .94 in the current sample.

Other psychopathological measures. The *Agoraphobic Cognitions Questionnaire* (ACQ; Chambless, Caputo, Bright, & Gallagher, 1984; German version: Ehlers, Margraf, & Chambless, 1993) was used to assess catastrophizing thoughts related to bodily symptoms in fear situations. According to the authors the 14 items can be subdivided into two subscales with 7 items each: (1) loss of control (Cronbach's α in the current sample = .82) and (2) physical concerns (Cronbach's α in the current sample = .83). *The Screening for Somatoform Symptoms (SOMS)* consists of 53 somatic symptoms relevant for the diagnosis of somatization disorder according to DSM-IV and ICD-10. Participants had to mark all symptoms present during the last two years, which caused suffering but could not be

attributed to a medical cause by a physician. Reported symptoms were added to yield a symptom total score. Cronbach's α in the current sample was .94. Retest reliability and discriminative validity have been shown for the SOMS (Rief, Hiller, & Heuser, 1997). The *Somatic symptom index "PHQ-15"* (Cronbach's α in the current sample = .88) is a measure of somatic symptom severity and comprises 15 somatic symptoms from the *Patient Health Questionnaire* (PHQ; Löwe, Zipfel, & Herzog, 2001). The PHQ-15 has good reliability and validity (Kroenke, Spitzer, & Williams, 2002). The German version of the *State-Trait Anxiety Inventory* (STAI; Laux, Glanzmann, Schaffner, & Spielberger, 1981), consisting of 20 items, was used to assess trait anxiety (Cronbach's α in the current sample = .95).

5.2.5. Experimental measures

Stimulus material. Stimulus words (Table 5-1) consisted of 60 nouns divided into 4 sets of 15 words related to three semantic categories: (1) IEI-trigger substances (e.g., amalgam, solvents, exhaust emissions, cigarette smoke, insecticides), (2) non-specific symptom words (e.g., headache, fatigue, dizziness, nausea) and (3) household related words (e.g., oven, fork, bowl) as neutral stimuli. Since the emotional Stroop effect seems prone to lexical characteristics such as word frequency effects (Larsen, Mercer, & Balota, 2006), this last category consisted of 30 nouns that were matched to the 15 trigger and symptom words according to word length and average frequency in written German language (Belica, Herberger, & al-Wadi, 1992). IEI-trigger stimuli were selected on the basis of the most frequently reported IEI-trigger substances in the scientific literature (e.g., Bornschein et al., 2002; Miller & Prihoda, 1999) as well as according to self-reports of IEI/MCS-patients and information included in IEI-specific information brochures or documents of IEI support groups. In addition to frequency, we tried to ensure heterogeneity of the large spectrum of IEI-triggers by including words of olfactory detectable (e.g., paint smell, cigarette smoke) as well as invisible and inodorous agents (e.g., amalgam, radioactivity). Non-specific symptom words represent highly frequent symptoms included in instruments for the assessment of non-specific / somatoform symptoms (e.g., SCL-90R, SOMS, SCID I). Most of the IEI trigger words and the symptom words used in the experimental paradigms were also included in the IEI interview mentioned above.

Table 5-1: Original (German) Stimulus words used in the experimental tasks

IEI-trigger words	Neutral words (1)	Symptom words	Neutral words (2)
Amalgam	Backofen	Schwindel	Toaster
Wohngifte	Spülbecken	Übelkeit	Kochlöffel
Asbest	Gabel	Kopfschmerzen	Waschbecken
Lackgeruch	Schneebesen	Schwäche	Waage
Zigarettenrauch	Küchenmaschine	Lähmung	Teller
Autoabgase	Küchenmesser	Durchfall	Besteck
Insektizide	Alufolie	Atemnot	Teelöffel
Luftverschmutzung	Geschirrhandtuch	Muskelschmerzen	Kaffeekanne
Radioaktivität	Kaffeemaschine	Hitzewallung	Suppenteller
Lösungsmittel	Kaffeetasse	Müdigkeit	Herdplatte
Strahlung	Schüssel	Nervosität	Eierkocher
Benzindämpfe	Flaschenöffner	Herzrasen	Handfeger
Elektrosmog	Waschmaschine	Erbrechen	Esslöffel
Pestizide	Pfeffermühle	Bauchschmerzen	Topflappen
Formaldehyd	Gefriertruhe	Ohnmacht	Schale

Table 5-2: Translated stimulus words used in the experimental tasks

IEI-trigger words	Neutral words (1)	Symptom words	Neutral words (2)
amalgam	oven	dizziness	toaster
toxins in the house	sink	nausea	wooden spoon
asbestos	fork	headache	basin
paint smell	eggbeater	weakness	scales
cigarette smoke	cuisinart	paralysis	plate
emissions	kitchen knife	diarrhea	canteen
insecticides	tin foil	breathlessness	tea spoon
air pollution	dish towel	muscle pain	coffee pot
radioactivity	coffee machine	hot flash	soup plate
solvents	coffee cup	fatigue	hot plate
radiation	bowl	nervousness	egg boiler
petrol fumes	bottle opener	tachycardia	hand brush
electromagnetic pollution	washing machine	sickness	soup spoon
pesticides	pepper mill	belly ache	oven gloves
formaldehyde	chest freezer	blackout	bowl

Table 5-3: Additional original (German) stimuli used as distractors in the recognition task

IEI-trigger words	Neutral words (1)	Symptom words	Neutral words (2)
Dioxin	Mülleimer	Brennen	Pfanne
Farbstoffe	Nudelholz	Herzklopfen	Auflaufform
Arsen	Messer	Hautausschlag	Backpapier
Sondermüll	Milchkanne	Reizung	Kochtopf
Umweltbelastung	Geflügelschere	Zerrung	Mixer
Nikotin	Eierbecher	Prellung	Schürze
Quecksilber	Warmhalteplatte	Allergie	Eieruhr
Düngemittel	Tortenheber	Asthmaanfall	Suppenlöffel
Wasserverunreinigung	Spaghettizange	Magendrücken	Kuchengabel
Holzschutzmittel	Zuckerdose	Halsschmerzen	Brotmesser
Diesel	Schere	Unbehagen	Dosenöffner
Wasserdampf	Thermoskanne	Aufstoßen	Messbecher
Mikrowellen	Kaffeemühle	Sonnenbrand	Schaumlöffel
Chloroform	Waffeleisen	Blähungen	Einmachglas
Stickoxid	Salatschleuder	Hörsturz	Eimer

Table 5-4: Additional translated stimuli used as distractors in the recognition task

IEI-trigger words	Neutral words (1)	Symptom words	Neutral words (2)
dioxin	trash can	burning	pan
dyes	rolling pin	palpitation	casserole
arsenic	knife	skin rash	baking paper
hazardous waste	milk can	irritation	saucepan
environmental pollution	poultry shears	sprain	mixer
nicotine	egg-cup	bruise	pinafore
mercury	hot plate	allergy	egg timer
fertilizer	cake server	asthma	soupspoon
water pollution	spaghetti tongs	stomach-ache	pastry fork
wood preservative	sugar bowl	sore throat	bread knife
diesel	scissors	discomfort	can opener
steam	thermos flask	belch	measuring cup
microwave	coffee mill	sunburn	skimmer
chloroform	waffle iron	flatulence	preserving glass
nitrogen oxide	salad drainer	hearing loss	bucket

Emotional Stroop task (*EST).* We used a computerized version with a pseudorandom presentation procedure for the disorder related (triggers and symptoms) and neutral words. Throughout the experiment every single word was randomly presented in four colors (red, green, blue, yellow). Before the presentation, a fixation cross appeared in the middle of the screen for 500 ms. Afterwards, one of the words was shown in the middle of the screen until the subject responded to its color. After the offset of the word and a pause of 500 ms the next trial began with the presentation of a fixation cross. Participants were instructed to name aloud the color of the presented word as fast and accurately as possible. Responding reaction times were recorded with an individually calibrated voice key microphone attached to the throat. The task consisted of a first set of 20 practice trials and two test blocks with 120 trials each lasting for about 5 minutes. Trials from the three semantic categories were mixed quasi-randomly so that the same color or the same word could never appear twice in a row. There was a short break of about 2 minutes between the two test blocks.

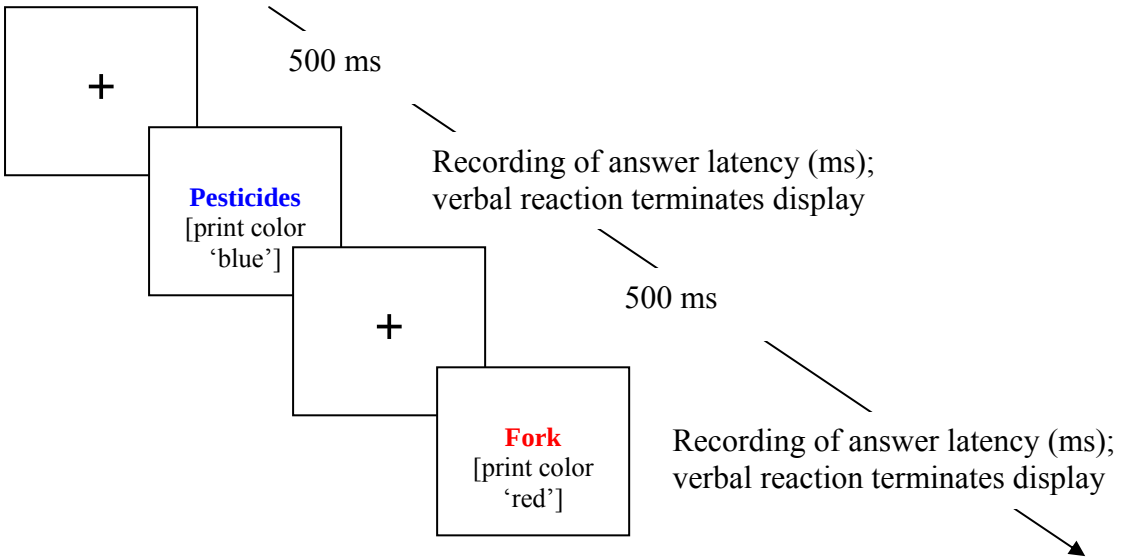

Figure 5-2: Two sample trials of the emotional Stroop task. Critical (symptoms or IEI-triggers) and neutral words (household related) were presented quasi randomly and verbal responses of the word color were recorded with a voice key microphone attached to the throat.

Dot probe task (*DPT).* The task was constructed according to the version used by MacLeod, Rutherford, Campbell, Ebsworthy, and Holker (2002). The same stimulus words (IEI-trigger words, symptom words and neutral words) as in the emotional Stroop task were used. Each trial began with a fixation cross presented for 500 ms followed by the cue display consisting of two stimulus words (one neural and one trigger or symptom word) above and below the fixation cross with a vertical distance between the two words of 3 cm (visual angle

of separation approximately 2°). The cue display remained on the screen for 500 ms and was replaced by a small arrow (target stimulus) pointing to the left or to the right. The arrow remained on screen until the participant responded by pressing the right or the left mouse key corresponding to the pointing direction of the arrow. Trials were separated by a 1000 ms inter trial interval. The task began with 10 practice trials followed by two test blocks with 120 trials each lasting for about 6 minutes. Participants were instructed to respond as fast and accurately to the target arrow as possible. The positions (upper or lower) of the cue word and of the following target stimulus (small arrow) were counterbalanced for each word. Every stimulus words appeared 8 times throughout the task, 4 times in the upper and 4 times in the lower position. The order of the different cue and target displays was randomized.

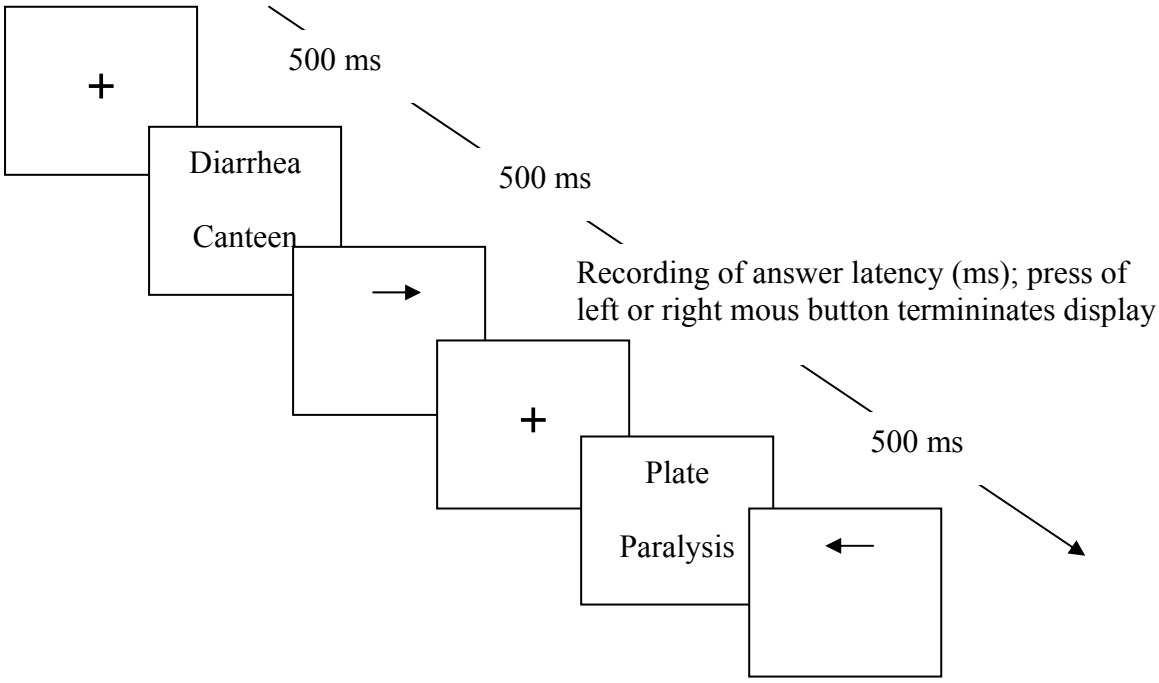

Figure 5-3: Two sample trials of the dot-probe task. Critical (symptoms or IEI-triggers) and neutral words (household related) were presented quasi randomly and manual responses were recorded with the left and right mouse button.

Recognition task. In the recognition task, the original 60 word stimuli from the three categories (IEI-triggers, symptoms and neutral words) were randomly mixed with 60 novel stimuli that (Table 5-3) were matched pairwise to the original stimuli according to word length and category content. The two preceding tasks (emotional Stroop and dot probe) served as an (incidental) encoding phase. During this encoding phase every stimulus word was presented 12 times (4 times during the emotional Stroop task and 8 times during the dot

probe task). After completing both tasks (emotional Stroop and dot probe) and a short break of two minutes the participants were for the first time informed that they would now have to complete a recognition task. Participants were informed about the ratio (50/50) of old (previously presented) and novel stimuli (distractors). During this recognition phase, the 120 words were presented sequentially on the computer screen and the presentation of a single word lasted until participants pressed one of two buttons labeled with "yes" (word was already presented in the tasks before) or "no" (new word) on a standard computer keyboard.

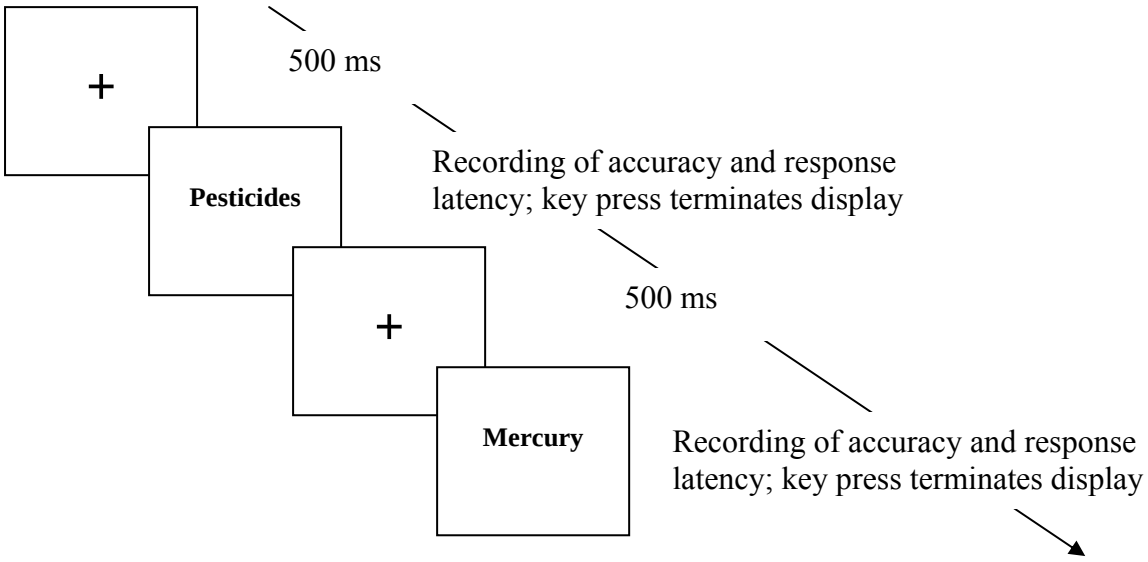

Figure 5-4: Two sample trials of the recognition task. Critical (symptoms or IEI-triggers) and neutral words (household related) and matched distractors (Table 5-3) were presented quasi randomly.

Self-Assessment Manikin (*SAM).* The SAM represents a non-verbal pictorial method for the assessment of self-report emotional evaluation (Bradley & Lang, 1994). We used a modified computerized version of the SAM with the dimensions valence (pleasant vs. unpleasant) and arousal (very arousing vs. not arousing) and a 5-point scale for each dimension (in order to improve usability, we left out the four middle categories used in the original version of the SAM). Participants used the standard computer mouse to click on one of the five buttons representing the original pictorial categories of the SAM valence and arousal dimensions.

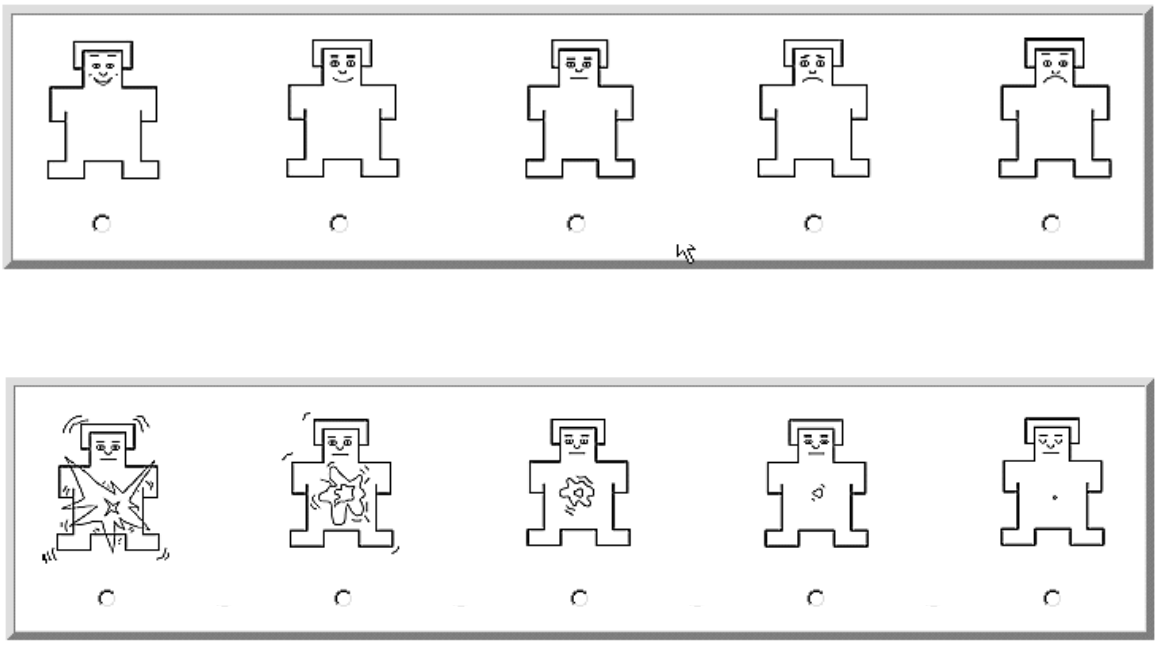

Figure 5-5: Valence (upper part) and arousal (lower part) dimension of the self-assessment manikin (SAM).

5.2.6. Apparatus and Software

In the emotional Stroop and dot probe task the stimuli were presented on a 17'' color monitor, attached to an IBM-compatible PC. Reaction times on the emotional Stroop task were recorded with a voice-key microphone connected to a 16-bit Creative Labs Soundblaster soundcard. The tasks were programmed and run with the ERTS software package (Beringer, 1996).

5.2.7. Procedure

All participants were tested individually in a 2-hr session. The diagnostic information (physical health status and psychopathology) was collected in a preceding session about 1 week earlier. Participants first completed the emotional Stroop task and the dot probe task (or vice versa; order of tasks was counterbalanced). Both tasks served as an incidental learning or encoding phase. Participants then performed the recognition task. After a short break, participants completed a battery of self-report measures and performed the valence and arousal judgments (SAM) at the end of the session.

5.2.8. Parameterization and Statistical Analysis

Response times from the emotional Stroop and dot probe task were corrected for outliers following a two-step procedure: Firstly, all reaction times longer than 2000 ms were eliminated from the analysis (in the emotional Stroop this procedure eliminated 0.59 % of trials in the CG, 0.41 % trials in the IEI and 0.04 % in the SFD group; for the dot probe less than 0.01 % of trials in the three groups were affected). Secondly, each experimental condition reaction time larger than the individual mean plus 3 *SD* units were individually recoded to this boundary value of mean plus 3 *SD* (this procedure affected an additional 0.65 % of trials in the CG, 0.67 % trials in the IEI and 0.63 % in the SFD group; for the dot probe the corresponding rates were 1.2 % in CG, 1.2 % the IEI and 1.0 % in the SFD group). Experimental data of both paradigms were analyzed with mixed 3 × 2 ANCOVA designs with age as a covariate. The 3-level between subjects factor comprised the experimental group membership. In the case of the emotional Stroop task the 2-level between subjects factor referred to the valence of stimuli (threat words vs. neutral words). For the dot probe task the two factor levels referred to the probe location (probe in location of threat words vs. probe in location of neutral word). Age was introduced as a covariate because of a significant main effect of age on group ($F(2, 149) = 3.5$, $p = .03$, $\eta_p^2 = 0.05$). This was due to a slightly higher mean age in the IEI-group (however, post-hoc tests on age between groups did not reach significance).

For all statistical analyses, results of the overall model as well as results of one-sided planned contrasts (Hager, 2002) according to our a priori hypotheses are reported. Contrasts were specified in ANCOVAs with performance on corresponding baseline conditions as additional covariates. Consequently, measures of effect sizes (Cohen's *d*) are based on means and variances of the corresponding residuals. Effect sizes will be reported as partial η^2 (η_p^2) for ANCOVA effects ($\eta_p^2 \geq 0.01$ small effect; $\eta_p^2 \geq 0.06$ medium effect; $\eta_p^2 \geq 0.14$ large effect) and as Cohen's *d* (Cohen, 1992) for planned contrasts between groups ($d \geq 0.30$ small, $d \geq 0.50$ medium, $d \geq 0.80$ large).

5.3. Results

5.3.1. Psychological and symptom measures

Table 5-5 depicts socio-demographic information and the results of the diagnostic ratings and symptom measures. As a result of the selection procedure and group definition criteria, participants in the three groups differed with regard to the degree of chemical odor sensitivity (COSS), environmental sensitivity (ESQ) and number of somatoform symptoms (SOMS, PHQ-15). Apart from the group defining diagnoses (IEI and SFD), the two clinical groups revealed a higher prevalence of concurrent depression. The rate of current anxiety disorders was significantly higher only in the SFD group but not in the IEI group compared to the CG (Table 5-5).

Results of additional psychological self-report measures are described in Table 5-6. Regarding trait anxiety (STAI), the two clinical groups scored higher than the control group. Furthermore, group differences emerged in the two scales of the ACQ: Compared with the control group, participants in the two clinical groups reported higher values in the "physical concern" scale that mainly addresses hypochondriac attitudes, whereas only the IEI participants had higher scores in the "loss of control" scale. With regard to body- and health-related cognitions assessed by the CABAH, the IEI and the SFD group did not differ on any of the five subscales. Compared with the CG, the SFD group scored significantly higher on three (1, 2, 3) and the IEI group on two (2, 3) of the CABAH scales. Both IEI and SFD individuals complained more about autonomic sensations and felt weaker physically than the CG.

In line with our a priori hypothesis, participants in the IEI group had significantly elevated scores on the absorption scale (TABS), indicating higher levels of hypnotic susceptibility and dissociative (normal) experiences compared with the other two groups.

Table 5-5: Sample Characteristics, Symptoms, and Diagnoses (according to DSM-IV)

	1 CG (n = 54)	2 IEI (n = 54)	3 SFD (n = 44)	ANCOVA		Scheffé post hoc test [a]
	$M \pm SD$	$M \pm SD$	$M \pm SD$	F (2,148)	η_p^2 [f]	
Age	44.90 ± 11.40	49.60 ± 9.60	44.30 ± 12.70	3.5 [j]	0.05	*ns*
Chemical Odor Sensitivity Scale (COSS) [b]	9.15 ± 5.94	45.57 ± 6.96	15.56 ± 7.90	397.4 [e, h]	0.84	2>3>1
Environmental Sensitivity (ESQ)	5.98 ± 5.13	14.43 ± 8.35	6.64 ± 5.11	24.6 [f]	0.25	2>1,3
Somatic Symptoms (SOMS)	2.02 ± 2.57	14.48 ± 9.23	16.50 ± 7.44	64.2 [e, h]	0.46	2,3> 1
PHQ-15 [b]	3.28 ± 2.89	12.41 ± 6.20	13.73 ± 3.51	83.1 [e, h]	0.53	2,3> 1
	n (%)	n (%)	n (%)	χ^2 *(2, N = 152)*	φ [g]	Repeated 2 × 2 χ^2 tests
Female	37 (68.5)	38 (70.4)	36 (81.8)	2.5	.13	*ns*
Education (≥ 12 years)	24 (44.4)	15 (27.8)	18 (40.9)	3.5	.17	*ns*
IEI-cases	0 (0)	54 (100)	0 (0)	152	1.00	2>1,3
Any somatoform disorder	0 (0)	31 (57.4)	44 (100)	99.2 [e]	.81	3>2>1
Hypochondriasis (300.7)	0 (0)	1 (1.9)	2 (4.5)	2.6	.13	*ns*
Conversion Dis. (300.11)	0 (0)	1 (1.9)	1 (2.3)	1.2	.09	*ns*
Somatizat. Dis. (300.81)	0 (0)	19 (35.2)	14 (31.8)	23.4 [e]	.39	2,3>1
Undif. Som. Dis. (300.82)	0 (0)	9 (16.7)	19 (43.2)	30.3 [e]	.45	3>2>1
Pain Disorder (307.8)	0 (0)	1 (1.9)	10 (22.7)	22.3 [e]	.38	3>2,1
Concurrent depression	2 (3.7)	9 (16.7)	7 (15.9)	5.3	.19	2,3>1
Concurrent anxiety disorder	6 (11.1)	12 (22.2)	17 (38.6)	10.4 [d]	.26	3>1 [i]

Note. [a] Scheffé post hoc test significant at $p \leq .05$ or repeated 2 × 2 χ^2 tests at $p \leq .05$.
[b] Completed during the Screening procedure.
χ^2 / F- value: [c] $p \leq .05$; [d] $p < .01$; [e] $p < .001$.
[f] measure of effect size for F($\eta_p^2 \geq .01$ small; $\eta_p^2 \geq .06$ medium; $\eta_p^2 \geq .14$ large).
[g] measure of effect size for χ^2 (φ -coefficient: small = .10, medium = .30, large = .50).
[h] F-value (2,148) and effect sizes correspond to an ANCOVA with age as covariate;
[i] Comparisons between group 1 and 2 and group 2 and 3 were not significant;
[j] F(2,149). F and effect size correspond to an analysis of variance.

Table 5-6: Psychological measures (*M* ± *SD*)

	1 CG (n = 54)	2 IEI (n = 54)	3 SFD (n = 44)	ANCOVA F (2,148)	ANCOVA η_p^2 [a]	Scheffé post hoc test [c]
Trait anxiety (STAI) [b]	46.13±8.89	56.72±12.00	61.66±10.08	28.7 [f]	0.28	2,3>1
Absorption (TABS)	42.70±20.39	56.26±23.06	41.02±19.95	6.7	0.08	2>1,3
Loss of Control (ACQ)	1.45 ± 0.46	1.66 ± 0.60	1.62 ± 0.53	3.8 [d]	0.05	2>1
Physical Concerns (ACQ)	1.07 ± 0.12	1.33 ± 0.45	1.32 ± 0.48	7.5 [f]	0.09	2,3>1
Catastroph. cognitions (CABAH 1)	10.00 ± 4.32	11.26 ± 5.75	12.70 ± 4.95	3.8 [d]	0.05	3>1
Autonomic sensations (CABAH 2)	1.87 ± 1.54	4.52 ± 2.40	3.82 ± 2.64	19.1 [f]	0.21	2,3>1
Bodily weakness (CABAH 3)	3.09 ± 2.62	7.48 ± 4.33	7.30 ± 4.17	21.8 [f]	0.23	2,3>1
Intolerance of bodily complaints (CABAH 4)	3.06 ± 1.98	3.80 ± 2.11	4.02 ± 2.16	3.0 °	0.04	*ns*
Health habits (CABAH 5)	5.83 ± 1.83	6.46 ± 1.69	5.59 ± 1.73	2.9 °	0.04	*ns*

Note. [a] measure of effect size for F($\eta_p^2 \geq .01$ small; $\eta_p^2 \geq .06$ medium; $\eta_p^2 \geq .14$ large).
[b] STAI values are t-transformed on the basis of population norms, corrected for age and gender.
[c] Scheffé post-hoc test significant at $p \leq .05$.
F- value (2, 148): [d] $p \leq .05$; [e] $p < .01$; [f] $p < .001$; ° $p < .10$.

5.3.2. Experimental measures

Emotional Stroop task (*EST).* Figure 5-6 depicts the interference indices (difference between the latencies for threat words and neutral words) of the emotional Stroop (EST) task subdivided according to the experimental groups. The verbal response latencies were analyzed with 3 × 2 mixed ANCOVAs with age as a covariate, the three groups as a between subjects factor and the two emotional Stroop conditions (emotional vs. neutral words) as a within subjects factor[2]. Since the design was counterbalanced for order of tasks (emotional Stroop task first or the dot probe task first), we first tested whether order had an effect on the emotional Stroop interference indices. This was not the case, neither for the IEI-trigger words ($F(1, 146) < 1$) nor for the symptom words ($F(1, 146) < 1$). Consequently, we combined data of the respective two groups for further analyses. For the IEI-trigger words, a significant main effect for group ($F(2, 144) = 4.1$, $p = .02$, $\eta_p^2 = 0.05$) was found. Post-hoc tests revealed that this main effect was due to slower overall reaction times in the IEI group compared to the SFD group. Neither the word category factor (trigger vs. neutral words) ($F(1, 144) = 0.5$, $p = .46$, $\eta_p^2 < 0.01$) nor the interaction between group and word category ($F(2, 144) = 0.9$, $p = .92$, $\eta_p^2 < 0.01$) were significant. Planned comparisons according to the a priori hypothesis of longer color naming latencies for IEI-trigger words in the IEI group did not reveal significant results ($p > 0.10$, $d < 0.20$). For the symptom words we again found a significant main effect for group ($F(2, 144) = 4.1$, $p = .02$, $\eta_p^2 = 0.05$) and a main effect for word category ($F(1, 144) = 3.9$, $p = .049$, $\eta_p^2 = 0.03$). More importantly there was a marginally significant word category × group interaction effect ($F(2, 144) = 2.9$, $p = .056$, $\eta_p^2 = 0.04$). Planned comparisons indicated that combined the two clinical groups showed a stronger interference effect for symptom words than controls ($F(1, 144) = 4.9$, $p = .03$, $d = 0.39$). This effect can mainly be attributed to larger interference in the IEI-group ($p = .02$, $d = 0.40$) but also to the SFD group ($p = .06$, $d = 0.37$) compared to the controls. The two clinical groups did not differ in their interference effect to symptom words ($p = .65$, $d = 0.07$).

[2] Data of 4 participants were excluded from the analysis because of voice-key problems.

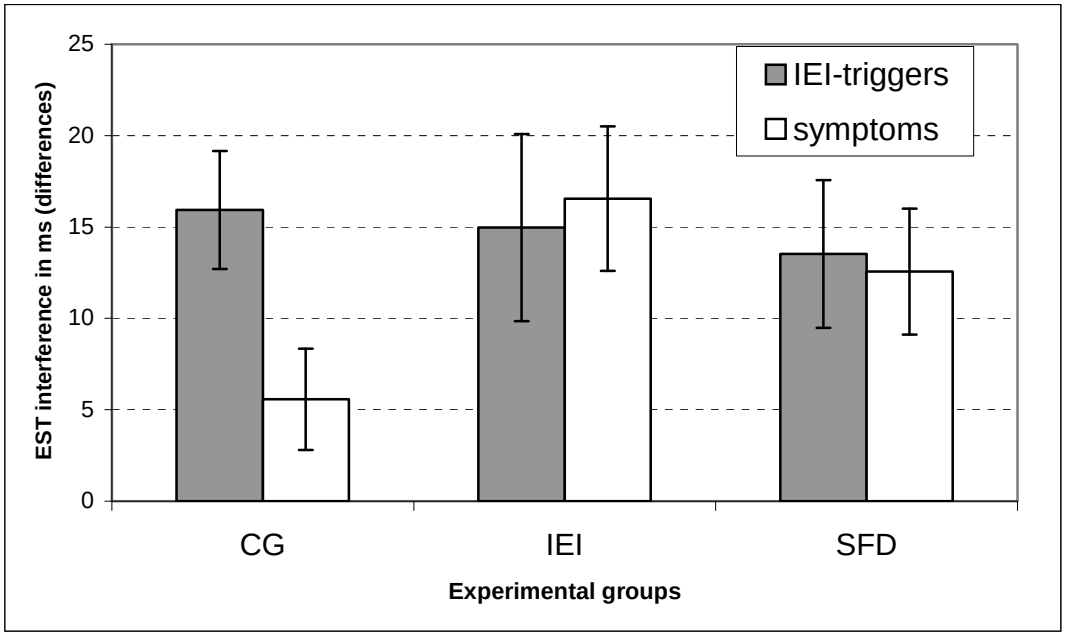

Figure 5-6: Mean interference indices (in ms) and standard errors of the emotional Stroop task for the experimental groups and the two disorder related word categories (IEI-triggers and symptoms). Data represent difference scores between the matched neutral words and the two disorder related categories.

Dot probe task (*DPT).* Figure 5-7 depicts the dot probe indices as differences between conditions with probe after critical words (IEI-triggers or symptoms) and probe after neutral words. As probes in the location of critical words were subtracted from probes in the location of neutral words, positive differences indicate vigilance towards negative stimuli whereas negative differences indicate avoidance of critical stimuli. Again, we first tested whether order of tasks had a significant effect on the dot probe indices. This was not the case, neither for the IEI-trigger words ($F(1, 145) = 1.0$; $p = .31$) nor for the symptom words ($F(1, 145) = 1.3$, $p = .25$). Consequently, we combined data of the respective two groups for further analyses. The response latencies were analyzed with 3 × 2 mixed ANCOVAs with age as a covariate, the three groups as a between subjects factor and the two critical dot probe conditions (probe in the emotional word location vs. probe in the neutral word location) as a within subjects factor[3]. Age as covariate had a large influence on the response latencies in general ($F(1, 143) = 28.6$, $p < .01$, $\eta_p^2 = 0.17$). Additionally, a trend for a main effect of group ($F(2, 143) = 2.4$, $p = .096$, $\eta_p^2 = 0.03$) was found. Post-hoc tests revealed that this trend was attributable to slower overall reaction times in the IEI group compared to the other two groups. Neither the word category factor (trigger vs. neutral words) ($F(1, 143) = 2.2$, $p = .14$,

[3] Data of 4 participants in the IEI group (3 because of extreme values on relevant variables and one because of more than 10 % extreme slow latencies > 2000 ms) and of 1 participant in the CG (more than 5 % errors) were excluded from the analysis.

η_p^2 = 0.02) nor the interaction between group and word category (F(2, 143) = 0.3, p = .72, η_p^2 < 0.01) yielded any evidence for meaningful group differences. Results for the symptom and corresponding neutral words were highly similar. Again, the covariate age had a large influence on the response latencies in general (F(1, 143) = 28.9, p < .01, η_p^2 = 0.17). The main effect of group (F(1, 143) = 2.1, p = .13, η_p^2 = 0.03) did not reach significance. Neither the word category factor (symptoms vs. neutral words) (F(1, 143) = 0.2, p = .64, η_p^2 < 0.01) nor the interaction between group and word category (F(2, 143) = 1.1, p = .32, η_p^2 = 0.02) yielded any evidence for meaningful group differences. Consequently, planned comparisons according to our a priori hypotheses (faster reaction to probes replacing IEI-triggers words in IEI and faster reaction to probes replacing symptoms in IEI and SFD) did not reveal significant results.

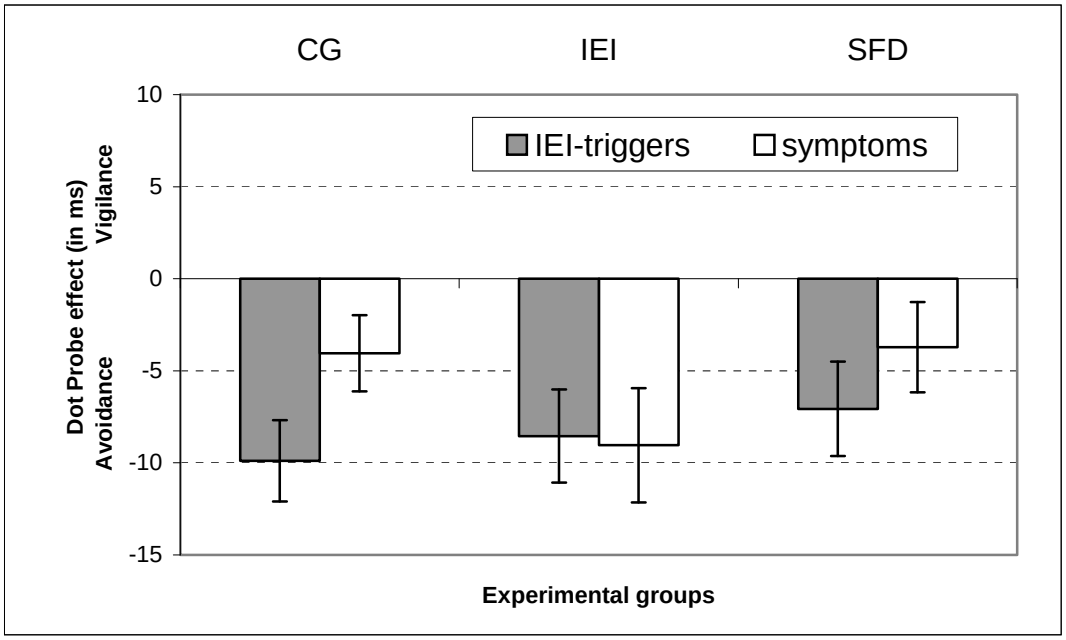

Figure 5-7: Dot probe indicators of vigilance (positive values) and avoidance reactions (negative values) and standard errors for the experimental groups and word categories (IEI-triggers and symptoms). Data represent difference scores between probe in location of neutral word and probe in location of critical word.

Recognition task (RET). Firstly, we tested again if order of the preceding encoding tasks (emotional Stroop and dot probe) had an impact on the recognition performance of critical stimulus classes. This was not the case, neither for IEI-trigger words (F(1, 147) = 0.78, p = .38, η_p^2 < 0.01) nor for symptom words (F(1, 147) = 0.57, p = .45, η_p^2 < 0.01). Consequently, we collapsed the data of the respective two groups for further analyses.

Recognition performance of the three groups as indexed by the discrimination parameter *d*', is presented in Figure 5-8. Analogous to the emotional Stroop data, recognition accuracy data (*d'*-values) were analyzed with 3 × 2 mixed ANCOVAs with age as a covariate, the three groups as a between subjects factor and the word category (emotional vs. neutral words) as a within subjects factor[4]. For the IEI-trigger words, the main effect of group was significant ($F(2, 142) = 6.6$, $p < .01$, $\eta_p^2 = 0.09$). Post-hoc tests revealed a worse overall recognition performance of the SFD group compared to the two other groups. Neither the main effect for word category ($F(1, 142) = 0.4$, $p = .52$, $\eta_p^2 < 0.01$) nor the interaction term group × word category ($F(2, 142) = 2.0$, $p = .14$, $\eta_p^2 = 0.03$) was significant. Planned comparisons according to the a priori hypotheses yielded a trend toward better recognition performance for trigger words in the IEI-group compared to the CG ($p = .06$, $d = 0.27$) and significantly better recognition compared to the SFD group ($p < .01$, $d = 0.69$). There was also a trend for better recognition in the CG compared to the SFD group (p (two-tailed) = .09, $d = 0.34$). Replicating this analysis for recognition of symptom words revealed a main effect for word category ($F(1, 142) = 14.4$, $p < .01$, $\eta_p^2 = 0.09$), indicating better overall recognition of symptom words compared to neutral words. Neither the main effect for group ($F(1, 142) = 1.1$), nor the group × word category interaction ($F(2, 142) < 1$) reached significance. Planned comparisons did not reveal any evidence for differential recognition performance across groups ($p > .10$, $d < 0.20$).

The analysis of individual response criteria (liberal vs. conservative; signal detection parameter *β;* Figure 5-9) did not yield any group specific effects. Overall, participants answered quite conservatively. This is likely the consequence of the relatively high difficulty of the recognition task (large number of words with high degree of similarity and disadvantageous encoding conditions). However, negative difference scores between threat word categories and neutral word categories in all three groups (Figure 5-9) indicate a more liberal response criterion for the two critical word categories over the three experimental groups.

[4] Data of 3 participants were excluded (datasets of 2 CG participants were lost because of computer problems; data of one IEI participant was excluded because of problems with the preceding tasks).

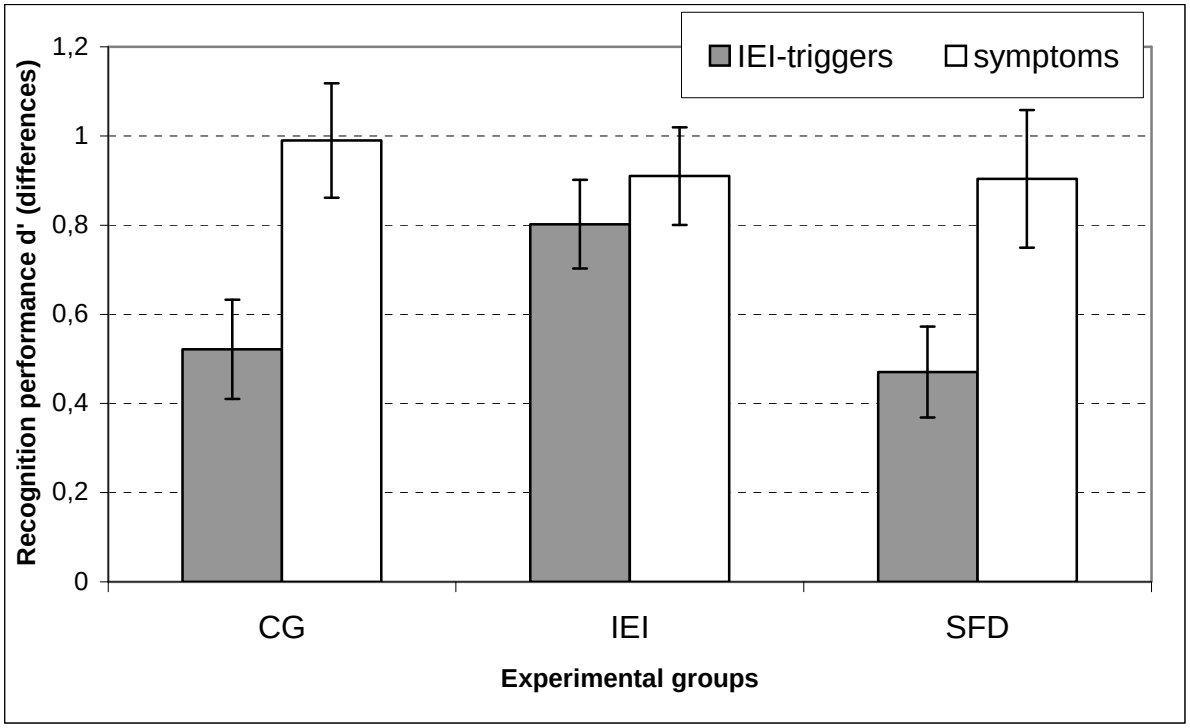

Figure 5-8: Recognition performance (*d'*) and standard errors for the three experimental groups and the different stimulus conditions (*d'* values represent difference scores between threat related word categories and neutral category).

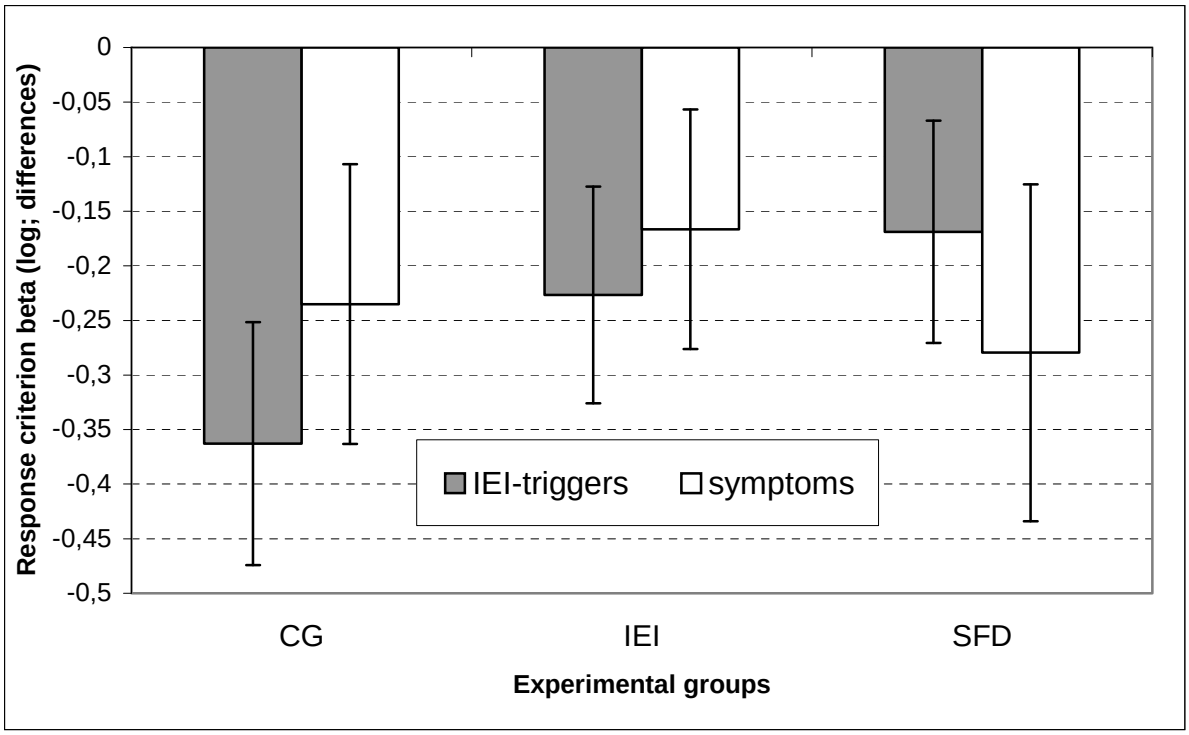

Figure 5-9: Response criterion (*β*) and standard errors for the three experimental groups and the different stimulus conditions (original *β* values are log-transformed; values represent difference scores between threat related word categories and neutral category).

Valence and arousal ratings (*SAM).* Figure 5-10 depicts the valence and arousal ratings for trigger and symptom words for the three groups. Values already represent difference scores between the disorder related categories (trigger and symptoms) and the corresponding neutral words. Analogous to the emotional Stroop data, 3 × 2 mixed ANCOVAs were computed for valence and arousal ratings of trigger and symptom words separately with age as a covariate[5]. For valence ratings of the IEI-trigger words results revealed a significant main effect of group ($F(2, 142) = 15.4$, $p < .01$, $\eta_p^2 = 0.18$), indicating stronger negative ratings in the IEI compared to the other two groups, a significant main effect for word category (trigger vs. neutral words) ($F(1, 142) = 43.6$, $p < .01$, $\eta_p^2 = 0.24$), indicating more negative ratings of the trigger words compared to the neutral words across groups, and most importantly a significant interaction between group and word category ($F(2, 142) = 4.0$, $p = .02$, $\eta_p^2 = 0.05$). A corresponding pattern of results was found for the arousal ratings of the trigger words with significant main effects for group ($F(2, 142) = 10.7$, $p < .01$, $\eta_p^2 = 0.13$) and word category ($F(1, 142) = 18.5$, $p < .01$, $\eta_p^2 = 0.12$) and a significant interaction effect ($F(2, 142) = 9.6$, $p < .01$, $\eta_p^2 = 0.12$). Planned comparisons of the a priori hypotheses revealed that the IEI-group differed significantly in their judgment of valence and arousal from the two other groups (SFD and CG). Thus, the IEI-group rated trigger words as more unpleasant (SFD: $p < .01$, $d = 1.06$; CG: $p < .01$, $d = 1.03$) and more arousing (SFD: $p < .01$; $d = 0.99$; CG: $p < .01$; $d = 0.84$) than the two other groups. The SFD and CG group did not differ significantly regarding their emotional perception of trigger words (valence: $p = .80$, $d = 0.04$, arousal: $p = .92$, $d = 0.02$).

The analysis of the judgments of the symptom words revealed a significant main effect of word category (symptoms vs. neutral words) for valence ($F(1, 142) = 37.0$, $p < .01$, $\eta_p^2 = 0.21$) and arousal ratings ($F(1, 142) = 16.4$, $p < .01$, $\eta_p^2 = 0.10$), indicating that all participants perceived the symptom words as more unpleasant and more arousing than the corresponding neutral words. Only for the arousal ratings a significant group main effect was obtained ($F(2, 142) = 3.3$, $p = .04$, $\eta_p^2 = 0.05$) resulting from generally higher arousal ratings for the IEI and SFD group compared to the CG. The group × word category interaction terms for valence ($F(2, 142) = 0.9$, $p = .42$, $\eta_p^2 = 0.01$) and arousal ($F(2, 142) = 1.4$, $p = .25$, $\eta_p^2 = 0.14$) judgments did not reach significance. Planned comparisons yielded a trend toward more negative valence ratings of the symptom words in the SFD ($p = .09$, $d = 0.30$) but not the IEI group ($p = .24$, $d = 0.14$) in comparison with the CG. Analog contrasts for the symptom

[5] The data sets of 6 participants were excluded from the analysis (4 with extreme-values on relevant variables and 2 with problems in understanding the task).

arousal ratings revealed significantly higher values in the IEI group (p = .01, d = 0.49) and the SFD group (p = .04, d = 0.36) compared to the CG.

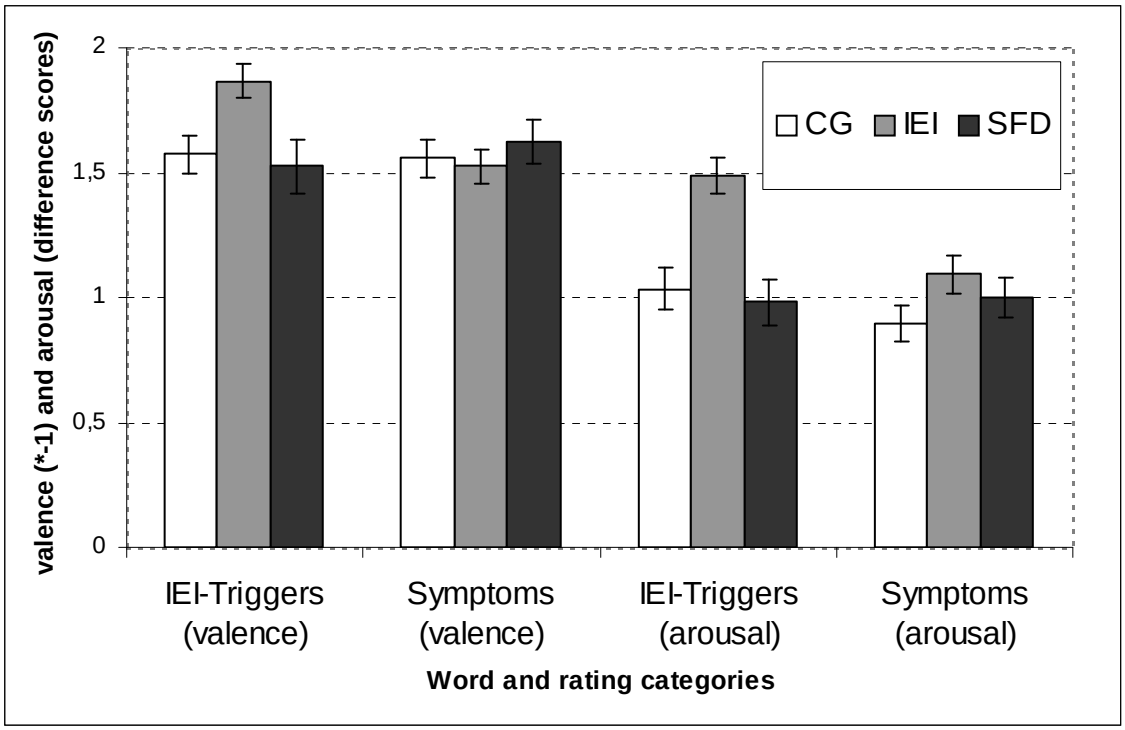

Figure 5-10: Valence and arousal ratings (on a 5-point pictorial scale) of the two word categories (with standard errors). Values represent difference scores of judgments to threat related words (triggers and symptoms) and neutral words. Valence-ratings have been transformed (*-1), so that larger values indicate more negative ratings (compared to the neutral control words).

5.3.3. Reliabilities and correlation analyses

The reliability of measures mathematically limits their maximal possible association (validity coefficients). We therefore computed reliability coefficients (Cronbach's α) for the experimental measures. The measures of emotional judgment (SAM) revealed adequate α coefficients (.86 - .92). In case of the two selective attention paradigms (EST and DPT), we computed difference scores between every single critical word (IEI-triggers and symptoms) and the corresponding neutral word that was matched to the critical words in terms of word length (pair-wise) and word frequency (list-wise) (Table 5-1). Since every word was repeated four times in both paradigms there were a whole of 60 difference scores for computing Cronbach's α of the two critical word classes (IEI-trigger words and symptom words). In general, α coefficients of the EST and DPT scores turned out as very low (EST: trigger words α = .27; CG: α = .17, IEI: α = .35, SFD: α = .24; and symptom words α = .05; CG: α = -.16,

IEI: α = .12, SFD: α = .03; DPT: trigger words α = .12; CG: α = -.01, IEI: α = .22, SFD: α = .06; and symptom words α = .04; CG: α = -.31, IEI: α = .20, SFD: α = -.03).

As depicted in Table 5-7, medically unexplained symptoms (SOMS) were correlated in the total sample with IEI-specific measures like chemical odor sensitivity (COSS) and environmental sensitivity (ESQ) as well as with somatoform risk factors like trait anxiety (STAI), physical concerns (ACQ), loss of control (ACQ), and dysfunctional cognitions about body and health (CABAH). Substantial correlations were also found between symptoms (SOMS) and experimental measures like selective attention toward symptoms (emotional Stroop) and the arousal judgment of triggers and symptoms (SAM). In contrast, indicators of attentional direction toward or away from threat stimuli derived from the dot probe task did not reveal substantial correlations to psychological measures or the other experimental paradigms.

In summary, correlation analyses revealed substantial associations between the emotional intrusion effect toward symptom words and psychological self-report measures on the one hand and between judgments of emotional perception (derived from the SAM ratings) and self-reports measures on the other hand. Table 5-7 summarizes the correlation findings and presents small but significant relations between selective attention toward symptom words in the emotional Stroop tasks and chemical odor sensitivity (COSS), somatic symptoms (SOMS) and the three CABAH subscales “catastrophizing cognitions”, “intolerance of bodily complaints”, and “bodily weakness”. For the interference index of the IEI-trigger words a substantial relationship was only found with the environmental sensitivity questionnaire (ESQ) but not with the COSS. For the valence and arousal ratings of trigger and symptom words weak to medium correlations were found indicating that enhanced levels of symptoms and dysfunctional beliefs of body and health are associated with more negative (unpleasant and arousing) emotional judgments of IEI-triggers and symptom words.

Table 5-7: Correlations between indicators of attentional bias and measures of somatic symptoms and dysfunctional beliefs for the total sample

	SOMS [a]	EST [b] triggers/ symptoms	DPT [c] triggers/ symptoms	SAM [d] valen. / arou. triggers	SAM [e] valen. / arou. symptoms
Psychological and symptom measures					
Somatic Symptoms [a] (SOMS)	-	.07 / **.19** [g]	.08 / .10	-.09 / **.19** [g]	-.09 / **.24** [f]
Chemical Odor Sensitivity Scale (COSS)	**.43** [f]	.02 / **.18** [g]	.05 / -.11	**- .31** [f] / **.40** [f]	.01 / **.20** [g]
Environmental Sensitivity (ESQ)	**.38** [f]	**.20** [g] **/ .19** [g]	.04 / .01	-.15 / **.23** [f]	.09 / .002
Trait anxiety (STAI) [d]	**.64** [f]	.07 / .08	-.06 / .11	-.07 / **.20** [g]	-.11 / **.34** [f]
Absorption (TABS)	**.17** [g]	.01 / .14	-.09 / .01	-.09 / **.20** [g]	-.05 / **.17** [f]
Loss of Control (ACQ)	**.25** [f]	.01 / .13	.07 / .08	.04 / .12	-.01 / **.23** [f]
Physical Concerns (ACQ)	**.43** [f]	.04 / .13	.02 / .16	.02 / **.19** [g]	.02 / **.28** [f]
Cognitions About Body and Health Questionnaire (*CABAH)*					
Catastrophizing cognitions (CABAH 1)	**.24** [f]	-.02 / **.20** [g]	.04 / -.03	-.10 / .09	**.22** [f] / **.22** [f]
Autonomic sensations (CABAH 2)	**.54** [f]	.14 / .15	-.02 / .04	**-.17** [g] / **.26** [f]	-.10 / **.22** [f]
Bodily weakness (CABAH 3)	**.63** [f]	.09 / **.18** [g]	.01 / .05	-.08 / **.23** [f]	-.12 / **.21** [g]
Intolerance of bodily complaints (CABAH 4)	**.32** [f]	.03 / **.26** [f]	-.04 / .09	-.15 / **.19** [g]	**-.18** [g] / **.25** [f]
Health habits (CABAH 5)	.07	.03 / .12	.13 / -.05	-.18 / **.21** [g]	-.16 / .10

Note. Recognition (*d'*) and answer criteria indices yielded no substantial correlations (p>.10).
[a] SOMS = Screening for Somatoform Symptoms total score.
[b] Emotional Stroop interference (EST = ms(threat words) – ms(neutral words); N = 148)
[c] Dot probe (DPT = ms (probe after neutral word) – ms (probe after threat word); N = 147
[d] Valence and arousal ratings of the Self-Assessment-Manikin (SAM; N = 146)
[e] STAI values are t-transformed on the basis of population norms, corrected for age and gender.
Significance levels: [f] $p \leq .01$; [g] = $p \leq .05$

5.4. Discussion

Theoretically based on cognitive-behavioral models (chapter 2) of medically unexplained symptoms, the first study focused on psychological aspects in people with IEI and SFD. Based on a hypothetical cognitive-behavioral model of IEI (chapter 3), we examined whether participants with IEI and SFD show evidence of selective attention and a memory bias as well as differences in the emotional evaluation of threat related words (IEI trigger substances and symptoms). According to the self-report measures (e.g., symptoms, cognitive styles), the IEI and the SFD group reported highly similar symptom patterns and overlapping psychological risk factors for somatization and were clearly distinguishable from non-somatoform and non-IEI controls. The IEI and SFD group were equivalent regarding somatoform and psychological symptom severity. Experimental results support the notion of cognitive psychological abnormalities regarding attention and memory processes both similar and different between classical SFD and IEI. Most striking was the absence of an increased attentional bias towards IEI-trigger words in IEI participants compared to the other two groups. As opposed to non-specific symptom words for which prioritized attentional processes were found in IEI and SFD, differences in the processing of IEI trigger words affected later stages of elaboration of memory contents rather than early fast acting attentional processes. Regarding the unusual character of symptom attributions and beliefs specific of patients with IEI, the elevated levels of absorption compared to the CG and SFD participants suggest that altered attentional styles, a habitual tendency toward dissociative experiences, and a holistic-intuitive rather than a analytic-sequential mode of processing (Kuhl, 1983; Ritz, Maß, & Dahme, 1993) as well as an increased openness to (unusual) experiences might be involved in the etiology and maintenance of IEI-specific beliefs.

5.4.1. Evidence for selective attention and memory bias in IEI and SFD

Results regarding an attentional bias were mixed. In line with our a priori hypotheses enhanced selective attention (emotional Stroop interference) toward symptom words in IEI and (slightly reduced) in SFD were found. Thus, evidence for an attentional bias toward linguistic representations of “internal” threat cues (i.e., bodily complaints) was gained, supporting the theory of somatosensory amplification.

Contrary to our expectation, we could not observe an enhanced selective attention or higher emotional intrusion effect for IEI-trigger words in IEI-participants. This result

contradicts observations of clinicians who report strong automatic and fast phobic-like reactions of patients with IEI when confronted with trigger substances (e.g., fragrances). One could speculate that word stimuli may not be a good and ecologically valid proxy for the actual external fear triggers in IEI. However, the IEI-group evaluated trigger words as much more negative and more arousing in the emotional judgment task (SAM-ratings) than the two comparison groups. These results suggest that the stimuli (especially the IEI-trigger words) are adequately selected. The correlation results offer another potential explanation - although the core feature of IEI is hypersensitivity toward chemical odors, no correlation exists between the severity of chemical odor sensitivity (COSS) and the interference index for IEI-words in the emotional Stroop task. The ESQ that mainly assesses overvalued ideas related to environmental agents, however, correlated with the interference index. Possibly, an individual will only show an attentional bias toward IEI-triggers if olfactory intolerance reactions and additionally overvalued beliefs regarding toxicogenic causation of symptoms act simultaneously. Alternatively, the interpretation of certain IEI-triggers as harmful may rely on a "later" elaborative stage of information processing (according to the model by: Williams, Watts, MacLeod, & Mathews, 1997) without any biologically prepared fast acting and attention capturing process apparent in phobias. Results of the recognition task support this hypothesis. In fact, the IEI-group revealed a better recognition performance of IEI-trigger words compared to the two other groups.

Contrary to our expectations, no evidence for a memory bias toward symptom words was found. Neither the SFD nor the IEI-group showed an enhanced ability to recognize symptom words compared to healthy controls. However, all three groups remembered symptom words and trigger words better than neutral words. Such a memory bias toward symptom words was found in hypochondriac patients (Pauli & Alpers, 2002). Only two participants in the SFD and one in the IEI group had a diagnosis of hypochondriasis. This may explain the absence of such a memory bias in our sample.

Taken together, the results of the emotional Stroop and the recognition task depict a certain asymmetry regarding our two stimulus classes of IEI-triggers and symptom words. While symptom words produced a group specific attentional bias (IEI and SFD) but no explicit memory bias, the IEI-trigger words showed the opposite pattern of results in the IEI-group (better recognition but no specific attentional bias). We suggest that symptom words are a proxy for internal threat cues and elicit processes of selective attention in participants with IEI and SFD. IEI-trigger words probably represent specific external attributions for unexplained symptoms and provoke the activation of specific schemata that allow for better

discrimination and more accurate recognition of IEI-trigger substances. In this respect the group specific emotional evaluation effects (SAM valence and arousal ratings) might also reflect a disorder specific external attribution process in order to reduce uncertainty provoked by medically unexplained symptoms.

Given the findings in the emotional Stroop task, the results of the dot probe paradigm, originally intended as another measure of selective attention, were rather unexpected. Neither for IEI-trigger words nor for symptom words did we find any group specific effect of vigilance or avoidance towards critical word stimuli. Several explanations might account for these negative results. Firstly, evidence exists that the dot probe task might not be as sensitive as the emotional Stroop task in detecting emotion driven attentional processes (Mogg, Bradley, Dixon, Fisher, Twelftree, & McWilliams, 2000; Wenzel & Holt, 1999) and that its low reliability (Schmukle, 2005) limits the use of the paradigm in terms of detecting individual differences. Secondly, recent studies (that were not available during the planning phase of our study) point to the critical influence of cue display durations in mapping either vigilance (facilitation) or avoidance (slowing) processes of a fear-like response (Cooper & Langton, 2006; Mogg & Bradley, 2006; Vassilopoulos, 2005). Specifically a study by Vassilopoulos (2005) comparing socially anxious with non-anxious students yielded specific vigilance reactions with a stimulus display duration of 200 ms and avoidance reactions with a cue display duration of 500 ms for social and physical threat words in the high-anxious group. Similarly, Mogg and Bradley (2006) found the strongest attentional bias of spider-fearful participants towards spider photographs with an exposure duration of 200 ms, whereas no significant bias was detectable at longer exposure durations (500 ms and 2000 ms). Accordingly, our stimulus display duration of 500 ms might have been inappropriate to detect any group specific vigilance reaction. Thirdly, it seems possible that the emotional Stroop and the dot probe paradigm measure different aspects of attentional and emotional processes - a hypothesis supported by the lack of substantial correlations between the two tasks (Mogg et al., 2000). If so, we only found positive evidence for emotional intrusion effects of symptom words already presented in the focus of attention (emotional Stroop) but no evidence for any heightened vigilance towards or facilitated engagement in the processing of disorder related stimuli (dot probe) in IEI and SFD. To determine which of these different explanations holds true further studies with varying cue display durations would be useful.

Apart from altered cognitive-affective processes, it was unexpected to find generally slower reaction times (EST and DPT) in the IEI group. This result of a general mental slowing, that distinguished the IEI participants from the two other groups (SFD without IEI

and controls), cannot be explained by either concurrent anxiety or depression. One could speculate that this slowing is a correlate of central nervous system hypo-activation (lower alertness and / or a decrease of attentional functions) in IEI as documented in previous psychophysiological research (increased resting alpha activity in EEG; Bell, Schwartz, Hardin, Baldwin, & Kline, 1998; Fernandez et al., 1999).

5.4.2. Evidence for psychological mechanisms in IEI and somatization

Assuming that the currently most prominent cognitive formulation of somatization as the result of a complex and vicious circle of increased symptom focused attention, catastrophization, and symptom amplification can at least be partially applied to IEI. According to this notion, differences between IEI and classical SFD without IEI may rely primarily on later elaborative and attributional cognitive processes. Whereas people with a somatization disorder are typically plagued by uncertainty about the causes of their complaints, people with IEI seem to overcome this uncertainty by adopting elaborated beliefs about the specific causes of illness, namely IEI-trigger substances. The elevated levels of absorption observed in the IEI group might explain partly why those idiosyncratic attributions sometimes appear irrational, curious, and exotic. Unfortunately, in the long run, the consideration of frequent trigger substances as harmful causes heightened arousal and increases self-focused attention. According to a recent model of functional somatization (Brown, 2004), these processes might lower individual thresholds for symptom perception.

Our correlation results may provide further evidence for these proposed relations between cognitive processes and symptom perception. Report of multisomatoform symptoms (SOMS) was associated with (a) emotional intrusions of symptom words (emotional Stroop task), (b) the perception of symptoms as more arousing (SAM), (c) enhanced trait anxiety and (d) dysfunctional beliefs and cognitions regarding body and health (CABAH). Cross-sectional data do not allow for a final judgment of causes and consequences. Consequently, the initial influence of these cognitive processes on the etiology of IEI remains speculative. Winters and colleagues (2003) recently demonstrated that information (media warnings) about the danger of environmental pollution moderated the subjective symptom report in a differential olfactory conditioning paradigm. Explicit and implicit cognitive processes of emotional stimulus evaluation (e.g., information about the trigger substances and potentially related symptoms) and selective attention toward such symptoms likely precede and facilitate

the acquisition of false attributions. Furthermore, these processes might manifest substance-symptom-associations (through conditioning) in people suffering from IEI.

5.4.3. Limitations

Small effect sizes. Although we detected significant differences in the experimental paradigms between groups according to our a priori hypotheses, the reported differences from the emotional Stroop and the recognition task are small in size. Technical as well as content related aspects of our tasks might have contributed to this fact. First, emotional intrusion effects obtained with the emotional Stroop task seem to produce larger effects in a block presentation format in which the different stimulus categories are put, sequenced into content homogenous blocks of trials (Holle, Neely, & Heimberg, 1997). However, as long as it is unclear what mechanisms cause these larger effects (e.g., carry-over effects; Waters, Sayette, Franken, & Schwartz, 2005; Waters, Sayette, & Wertz, 2003) we consider the intrusion effects obtained in randomized presentation formats as "purer" indicators of immediate emotional intrusion or disruption. Furthermore, the category of threatening, self-relevant and disorder related stimuli for somatoform people are much more heterogeneous than for specific phobias like spider phobia. An individualized selection procedure (as proposed by Andersson & Haldrup, 2003) of the most relevant stimuli might have contributed to larger group differences. As for the recognition task, the use of an incidental encoding phase during the two other tasks (emotional Stroop and dot probe) likely has introduced sources of unwanted variance. Although we did not ask participants for their expectations regarding the memory task, some reported, informally, after the task that they had anticipated the recognition demand, whereas others, obviously, were totally surprised by it. Generally, effects of the attentional and memory bias group differences are small (e.g., about 10 ms in case of the EST). However, neuropsychological research has shown that extremely short time intervals (e.g., the detections threshold of about 10 ms) suffice to produce neural responses associated with fear (Williams, Liddel, Rathjen, Brown, Gray, Phillips, Young, & Gordon, 2004). Furthermore, MacLeod and colleagues (MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002) have shown that a comparatively small attentional bias (of about 20-30 ms) can have meaningful causal effects on emotional vulnerability. In this respect, even small effects reveal relevant cognitive and emotional processes of somatoform disorders.

Word stimuli. One could argue that the neutral stimuli (household related words) might not have acted as neutral words because of associations between these words and food intolerances that are indeed relevant to IEI and SFD. Yet, the neutral words were predominantly related to kitchen equipment (e.g., bowl, plate, toaster) rather than food per se. As shown empirically, explicit emotional ratings of valence and arousal (SAM) for these neutral words did not differ significantly among the three groups (Valence: $F = 1.0$; Arousal: $F = 0.3$). Theoretically, it would still be possible that on an implicit level the associations to food might have produced an exaggerated emotional response (greater interference in the emotional Stroop task). However, this influence would have led to smaller or underestimated emotional interference effects for IEI-trigger words and symptom words. Furthermore, using a subscale of the QCGS to assess pollen and food allergy (Bailer et al., 2004a), neither the SAM ratings of the neutral words nor the emotional Stroop indicators (or the baseline reactions times) were significantly correlated with self-reported pollen and food allergy ($r < .10$).

Sources of the emotional intrusion effect and memory bias. Another question left unanswered refers to the source of the interference effect demonstrated for symptom words in people with IEI and SFD. At least two processes may explain the development of such an emotional intrusion effect: (a) a facilitated engagement with and prioritized processing of threat stimuli, which slows color naming or (b) a delayed disengagement from threat stimuli ("emotional lingering") on threat information (McKenna & Sharma, 2004). Further research with the Posner cued target paradigm (Posner, Cohen, & Rafal, 1982; Fox, Russo, Bowles, & Dutton, 2001) could dissociate these two mechanisms and shed light on the exact nature of the interference effect found in this study. Also, we cannot exclude, that the depressive psychopathology might have influenced our findings. we therefore tested whether the memory bias can be attributed exclusively to depressive symptoms by including the SCL-90-R depression scale as an additional covariate. The depression scale had no substantial influence on the recognition performance and the corresponding effect sizes remained unaltered. Furthermore, we consider it theoretically unlikely that the recognition results are mainly attributable to mood congruency effects. Two negative word categories (symptoms and IEI-triggers) were used. According to a mood congruency hypothesis we would have expected a memory bias for both categories. Instead, results revealed differential effects only for the IEI-trigger words.

Etiology of IEI and SFD. The current study focused on cognitive psychological processes as well as self-reported symptoms in IEI and somatoform disorders. Assessing biological and genetic factors was beyond the scope of this study. Therefore little can be said about potential biological risk factors or a neurogenetic basis of IEI. Although self-report data and cognitive experimental data presented above point to psychological abnormalities in IEI these variables only partially explain the clinical phenomenon (Bell, Schwartz, Peterson, & Ahmed, 1993). Most likely, psychological and biological factors interact in a complex way as demonstrated for other diseases like coronary artery disease (Zellweger, Osterwalder, Langewitz, & Pfisterer, 2004).

Specificity of the reported effects. The current design is limited in regard to clinical control groups (non-somatoform psychological disorders as well as chronic organic disorders). Further research is needed to determine whether the effects of selective attention towards bodily symptoms found for IEI and SFD are specific. However, the recent study by Lim and Kim (2005) used a set of similar physical threat words and found an attentional bias (emotional Stroop effect) in somatoform participants but not in depressive patients. Our own correlation results point in the same direction that depression and (trait) anxiety alone cannot account for the observed attentional bias to symptom words. We assume that in diverse chronic conditions (psychological as well as physical) in which processes of somatosensory amplification are involved, these processes are also associated with an emotional intrusion effect. Leaving a simple dualistic "biological/organic versus psychological perspective" behind, we would not consider the existence of an attentional bias as evidence for a psychogenic etiology but rather as evidence of a specific cognitive illness representation. This could be caused either primarily organic, psychogenic or by a complex interaction of both.

Sample composition and selection biases. The asymmetrical inclusion criteria for the IEI and SFD group regarding the degree of chemical odor sensitivity presented above were chosen in order to maximize differences between the two clinical groups. Moreover, the case criteria for IEI (at least three symptoms attributed to low levels of environmental chemicals for at least 6 month) necessarily overlap with criteria for the diagnosis of somatoform disorders, therefore the observed co-prevalence of IEI and somatoform disorders was rather expected and design imminent. The reason that not all IEI participants met criteria for a somatoform disorder was the more liberal character of the IEI case definition regarding the

impairment criterion as part of the DSM-IV somatoform disorders section. The focus of the current study was to question whether participants with an IEI-specific attribution style differ from traditional somatoform patients without such specific attributions. This overlap causes problems for the interpretation of the results. Therefore, we tried to disentangle this overlap statistically by repeating the analyses for the three experimental paradigms (emotional Stroop, dot probe, and recognition task) with the definition of two between subject factors namely "diagnosis of a somatoform disorder (SFD)" and "IEI diagnosis". Results reveal a significant interaction between the emotional interference effect (EST) for symptom words and the factor "SFD diagnosis" ($F(1, 143) = 5.3$, $p = .02$, $\eta_p^2 = 0.04$). Simultaneously, for the second between subjects factor "IEI diagnosis", there was a marginally significant interaction with symptom word interference ($F(1, 143) = 3.1$, $p = .08$, $\eta_p^2 = 0.02$). Moreover there was a main effect for IEI-diagnosis ($F(1, 143) = 6.4$, $p = .01$, $\eta_p^2 = 0.04$) indicating generalized slowing of responses independent of word valence in participants with IEI. A trend toward a similar main effect for the factor "SFD diagnosis" ($F(1, 143) = 2.7$, $p = .10$, $\eta_p^2 = 0.02$) was also apparent. The absence of a significant two-way interaction between the two between subjects factors "IEI diagnosis" and "SFD diagnosis" and the emotional interference effect to symptom words shows that the attentional bias is neither uniquely related to IEI (without SFD) nor to SFD (without IEI).

We repeated the same analysis with the specification of two between subject factors ("SFD diagnosis" and "IEI diagnosis") for the recognition data of the IEI trigger words. The interaction between recognition performance for trigger words and the factor "IEI diagnosis" was marginally significant ($F(1, 144) = 3.4$, $p = .07$, $\eta_p^2 = 0.02$), indicating better recognition of IEI-trigger words in participants meeting the IEI case criteria. No such trend was found for the factor "SFD diagnosis" ($F(1, 144) = 0.30$, $p = .58$, $\eta_p^2 < 0.01$). Analog analysis for the dot probe paradigm did not reveal any significant effect of any of the two group factors. Although results of these post-hoc analyses are also prone to a priori group specifications (namely the overlap between IEI and SFD), we interpret the results as evidence that both fulfilling IEI criteria and the diagnosis of SFD contribute to an attentional bias toward non-specific symptom words – a finding that is supported also by the correlation analysis (Table 5-7). As for the better recognition of IEI trigger words, fulfilling the IEI criteria seems most important, irrespective of an additional SFD diagnosis.

Generalization of the results is limited by the sampling procedure, the inclusion and exclusion criteria, and the case criteria used to define IEI. Exclusion of severe organic diseases that might have accounted for the symptoms reported was mainly based on self-

report data. Therefore, we cannot rule out completely that some of our participants may suffer from a current organic disorder. The majority of the subjects assigned to the diagnostic groups were recruited by advertisements (CG: 76 %; IEI: 78 %; SFD: 66 %), the remaining subjects stem from various polyclinics and primary care practices. The participants were given neither a treatment nor a detailed diagnostic feedback from the research staff; therefore neither the IEI nor the SFD subjects are completely comparable to typical IEI or somatoform patients. Together with the screening procedure and inclusion criteria, this selection process might have created a selection bias leading to an unusually high rate of polysymptomatic SFD, but minimized additional depressive disorders in both somatoform groups. We would therefore assume that our prevalence of psychological disorders found in the two groups with increased somatization were less associated with or biased by medical care seeking behavior as reported e.g., in the context of fibromyalgia (Aaron, Bradley, Alarcón, Alexander, Triana-Alexander, Martin, & Alberts, 1996). Nonetheless, both the IEI and the SFD subjects showed demographic and psychopathological features similar to those found in patients with help-seeking behavior (e.g., Bornschein et al., 2002; Hausteiner, Bornschein, Bickel, Zilker, & Förstl, 2003; Simon et al., 1993).

5.4.4. Conclusion

Results presented above reveal altered cognitive psychological processes in IEI and SFD. Five aspects are noteworthy: Firstly, the IEI-group showed an emotional interference effect toward non-specific symptoms comparable to people with a somatoform diagnosis only (emotional Stroop task). Secondly, no evidence for a specific attentional shift toward or away from symptoms or IEI trigger words was found in IEI or SFD (dot probe task). Thirdly, recognition memory for IEI-trigger words was enhanced in participants with IEI compared to the other two groups. Fourth, the emotional evaluation of IEI-triggers as unpleasant and arousing differentiated the IEI-group from the other two groups, and fifth, the emotional evaluations of trigger words were associated with elevated levels of self-reported chemical odor sensitivity, unexplained somatic symptoms and dysfunctional beliefs about body and health. The results suggest that processes of selective attention or emotional intrusion (EST) characteristic for somatoform disorder (without IEI) in general and selective recognition, as well as evaluative abnormalities (SAM) specific for IEI, might contribute to a multi-factorial pathogenesis and psychological maladjustment of IEI. The results of the first study indicate that implicit and explicit cognitive processes are involved in IEI. Patients with symptoms of

IEI will likely profit from specially tailored cognitive-behavioral interventions that focus on reattribution of bodily symptoms and a re-evaluation of the effects of exposure to minimal levels of IEI-trigger substances.

6 STUDY 2: EMOTIONAL INTRUSIONS AND IMPLICIT ASSOCIATIONS IN IDIOPATHIC ENVIRONMENTAL INTOLERANCE AND SOMATOFORM DISORDERS: A REPLICATION AND EXTENSION OF PREVIOUS FINDINGS

As outlined above, we hypothesize that abnormalities in information processing play a crucial role in the maintenance of IEI. Based on the cognitive-psychological model of MUS presented by Brown (2004) and our own hypothetical model of IEI (chapter 3), we consider the "repetitive allocation of high-level attention onto symptoms" (Brown, 2004, p. 807) as a central aspect to account for symptom chronicity in SFD and IEI. Additionally, we suppose that IEI-specific *cognitive schemata* (irrational beliefs or overvalued ideas) exist and can be activated by both external and internal triggers, such as (conditioned) olfactory stimuli (as proposed in the model of Van den Bergh and colleagues), abstract information units (e.g., media reports of environmental threat) or simply by noting bodily sensations. These activated schemata initiate or guide (unintentionally or without volitional control) the allocation of cognitive resources (high-level attention in the sense of Brown, 2004) to unspecific bodily symptoms. In turn, these symptoms are interpreted as confirming evidence for an IEI-specific illness prototype (Williams & Lees-Haley, 1993), implying severe chemically or environmentally caused personal harm. Because the first study mainly looked at the existence of attentional and explicit memory biases toward IEI-trigger words and unspecific symptoms, the second study was designed to test for the existence of specific implicit association effects as evidence of dysfunctional and disorder specific cognitive schemata.

Before presenting the detailed hypotheses and results of the second study, we will briefly outline new methodological aspects that we consider as helpful in testing crucial assumptions of our cognitive behavioral model of IEI.

6.1. New operationalization of selective attention and implicit association processes

In order to test the assumptions of *selective attention toward symptoms* and the existence of IEI-specific *cognitive schemata*, we will use experimental indicators of selective attention analogous to the emotional Stroop task and measures of implicit associative strengths between concepts in memory (e.g., Greenwald et al., 1998). The extrinsic affective Simon task (EAST, De Houwer, 2003) is an innovative variant of the Implicit Association Task (IAT) that was constructed to measure implicit attitudes or associations. As noted by De Jong, Van den Hout, Rietbroek, & Huijding (2003) the affective Simon task belongs to the

class of *irrelevant feature paradigms* consisting of three main components: Firstly, target stimuli whose valence is irrelevant for the task execution and which should be ignored (in our case e.g., IEI-trigger words and physical symptom words). Secondly, attributes to which the associative strength of the target words are be determined (e.g., adjectives representing the two concepts "good" and "bad"). Thirdly, two answer keys that are simultaneously matched to both attributes (e.g., right key "good" and left key "bad") and a second task *relevant* feature (e.g., right key "blue" and left key "green"). This arrangement allows for the manipulation of the compatibility between the target stimuli and the chosen attributes. Once a participant has learned the attribute to answer key mapping, he or she is instructed to respond as fast and accurately as possible to the color of the presented words (e.g., either "green" or "blue"). The central dependent variable represents the reaction time difference between compatible (e.g., headache in green, involving the *extrinsic* "bad" response) and incompatible (e.g., headache in blue, involving the *extrinsic* "good" response) trials.

The EAST has several advantages over the IAT (for details see De Houwer, 2003; Schmukle & Egloff, 2006). The EAST for instance allows the evaluation of the absolute associative strengths of single concepts, whereas the IAT needs complementary pairs of concepts and only reflects relative associative strengths with regard to certain target attributes. Dysfunctional associations or implicit threat associations have recently been demonstrated with the EAST in different areas of clinical psychology and psychopathology, for instance spider phobia (Ellwart, Becker, & Rinck, 2005; Huijding & De Jong, 2006), childhood obesity (Craeynest, Crombez, De Houwer, Deforche, Tanghe, & Bourdeaudhuij, 2005), and alcoholism (De Houwer, Crombez, Koster, & De Beul, 2004).

Because we attempt to measure not only *implicit association effects* with the EAST, but also *emotional intrusion effects* (analogously to an emotional Stroop task), we included household related words as a neutral reference category in addition to the critical target word categories - IEI-trigger words and physical symptom words. The possibility of assessing emotional intrusion effects (i.e., slower responses to critical words compared to neutral words) within an affective Simon paradigm was already briefly discussed in De Jong et al. (2003, p. 532). Before specifying our final hypotheses we will introduce a comparatively new approach to the study of reaction time (RT) data in these experimental paradigms. Until now, this approach has been used only in experiments in general cognitive psychology, but should be useful also for the study of affect modulated cognitive processing.

6.2. Dissociation of components of reaction time distributions

For most of the experimental paradigms used to measure implicit cognitive phenomena or processes of selective attention (e.g., the dot-probe or probe detection paradigm, the Posner cueing task, the emotional Stroop, the implicit association task and its modifications such as the EAST used in this study), individual reaction time is the most popular and widely used dependent variable. Generally, only measures of central tendency of individual response time distributions (the mean or median) are retained. In order to take into account further parameters of individual RT distributions, the ex-Gaussian distribution, a convolution of a Gaussian and an exponentional distribution, and its three parameters (μ, σ, and τ) have been proposed and evaluated in different domains. As Spieler, Balota, and Faust (2000) outline in detail, the ex-Gaussian distribution provides a good fit to individual RT distributions and helps to distinguish components of individual RT distributions that are differentially sensitive to experimental manipulations. Although it would be too simplistic to map the different parameters of the ex-Gaussian distribution to unique cognitive processes (Spieler et al., 2000), the μ parameter (reflecting the mean of the Gaussian part) has been associated with peripheral or automatic processing, whereas the τ parameter (reflecting the mean and standard deviation of the exponential part) is thought to mirror more strongly central or controlled attention demanding mental operations (Hohle, 1965), such as efficient inhibition (Spieler, Balota, & Faust, 1996). In this context, Schmiedeck, Oberauer, Wilhelm, Süß, & Wittmann (in press), recently demonstrated, that individual differences in the τ parameter of choice reaction time distributions were predictive of individual differences in working memory capacity and fluid intelligence. The decomposition of individual response time distributions allows to directly test the hypothesis derived from a recent cognitive model of somatization (Brown, 2004). Accordingly, processes of selective attention characteristic for functional somatic syndromes should affect "later" (i.e., more controlled) stages of information processing. Group differences in reaction time task performance should therefore especially affect the τ parameter.

6.3. Aims and hypotheses of study 2

Although cognitive mechanisms like symptom focused attention and somatosensory amplification have previously been hypothesized to play a role in IEI, the empirical basis for these cognitive hypotheses has remained weak. Without disregarding a possible involvement

of biological (e.g., endocrinological) processes in IEI, the aim of the second study is to gain further evidence for the relevance of *cognitive processes* in IEI and SFD. According to the assumption that both IEI and SFD are chronic conditions, we hypothesize that the emotional intrusion effect toward bodily symptom words found in IEI and SFD participants compared to non-somatoform controls should be replicable one year later, using a different experimental paradigm, namely the EAST. Furthermore, in line with our previous results (study 1), we did not expect emotional intrusion effects for IEI-trigger words in participants with IEI. With regard to the implicit association effect measured by the EAST, we expected stronger negative implicit association effects for IEI-trigger words and symptom words in participants with IEI and for symptom words only in participants with SFD compared to the CG.

Methodologically, we seek to demonstrate that a modified extrinsic affective Simon task (EAST) allows for the simultaneous assessment of two measures of (implicit) psychological phenomena, namely (1) the implicit emotional evaluation of critical stimuli and (2) emotional intrusion as traditionally measured by the emotional Stroop paradigm. By fitting the ex-Gaussian distributional model to the individual RT data we will try to elucidate the sources of potential effects in measures of RT distributions beyond conventional measures of central tendency (such as mean or median). Specifically, we hypothesize that enhanced selective attention and implicit association effects should be reflected in a larger τ parameter for critical emotionally salient word categories.

6.4. Methods

This study was approved by the Ethics Committee for Clinical Research of the medical faculty at the University of Heidelberg, Germany.

6.4.1. Participants

Participants that were originally recruited for the first study (cf. study 1) took part in a prospective follow-up study (study 2) of the specificity and the course of IEI. The majority of the participants (74 %) were recruited from the community by advertisements in local newspapers; the remaining participants were patients from polyclinics of environmental medicine, psychiatry, psychosomatic, and dental medicine at the University of Heidelberg (Germany). Those who completed the follow-up assessment were paid 60 Euros ($72.00). All

participants provided written informed consent. At baseline (t1), all participants (N = 152) underwent a medical examination, a psychiatric interview (SCID I; German version by Wittchen et al., 1997), and the IEI interview (SI-IEI; Bailer et al., 2006a). According to the interview results at t1 (cf. study 1), participants were assigned to three groups: participants with IEI (N = 54), participants with a somatoform disorder (SFD) according to DSM-IV (N = 44) but without IEI, and participants with neither IEI nor SFD (N = 54).

Final sample composition (t2). We re-examined nearly all participants (N = 146; 96 %) of the original sample (N = 152) one year later (t2), with only 6 participants lost (5 IEI, 1 SFD). At baseline, all participants in the SFD group fulfilled the full DSM-IV criteria for any somatoform disorder. Most prevalent were somatization disorder (IEI: 62.1 %; SFD: 32.6 %) and undifferentiated somatoform disorder (IEI: 31 %; SFD: 41.9 %), followed by pain disorder (IEI: 3.2 %; SFD: 23.3 %), conversion disorder (IEI: 3.4 %; SFD: 2.3 %), and hypochondriasis (only SFD: 4.7 %). The final follow-up sample at t2 comprised 49 participants with IEI, 43 participants with a SFD, and 54 participants (CG) with neither IEI nor a SFD (see Table 6-1 for sample characteristics).

6.4.2. Self-report measures

The Chemical Odor Sensitivity Scale (COSS). The COSS (Bailer et al., 2006b) contains 11 statements describing strong physical responses (e.g., trouble breathing, nausea, cough, dizziness) to the odor of common environmental chemicals (e.g., sprays, paints, cigarette smoke, cleansing agents, perfumes, exhaust fumes, gasoline). Reliability of the COSS has been established across diverse samples (Cronbach's α in the current sample t1 and t2 = .96; r_{tt} (t1,t2) = .90). The COSS was found to be dimensionally independent from respiratory symptoms not related to IEI triggers and from self-reported allergy to pollen and food (Bailer et al., 2004a).

Environmental Sensitivity Questionnaire (ESQ). The ESQ (Bailer et al., 2000; Bailer, Rist, Rudolf, Staehle, Eickholz, Triebig, Bader, & Pfeifer, 2001) contains a 10-item list of more or less harmful dental and environmental agents (e.g., electrosmog, radioactivity, harmful substances in air and water and dental filling materials). Participants are asked to judge the damaging effect of these agents on their health. The scale has adequate internal

consistency (current sample Cronbach's α (t1 and t2) = .91) and high temporal (1-year) stability (r_{tt} (t1, t2) = .80).

Other psychopathological measures. The Screening for Somatoform Symptoms (SOMS) consists of a list of 53 somatic symptoms relevant for the diagnosis of somatization disorder. Reported symptoms are added to yield a symptom total score. Cronbach's α in the current sample was .94 (t1) and .93 (t2). Retest reliability (r_{tt}) from t1 to t2 was .71. Good retest reliability and discriminative validity have also been shown previously for the SOMS (Rief et al., 1997). The Somatic Symptom Index (PHQ-15) from the Patient Health Questionnaire (Cronbach's α in the current sample = .88 (t1) and .87 (t2); r_{tt} (t1,t2) = .84) is a measure of somatic symptom severity and comprises 15 somatic symptoms. The PHQ-15 has good reliability and validity (Kroenke et al., 2002). The PHQ-9 is the depressive symptom severity scale from the PHQ (Kroenke et al., 2002), consisting of 9 items (Cronbach's α in the current sample = .88 (t1) and .91 (t2); r_{tt} (t1,t2) = .81). The State-Trait Anxiety Inventory (STAI; Laux et al., 1981) was used to assess trait anxiety (Cronbach's α in the current sample = .95 (t1 and t2); r_{tt} (t1,t2) = .83).

6.4.3. Experimental measures

Stimulus material in the EAST. Ten positive and ten negative adjectives, presented in white were chosen to represent the concepts "good" and "bad". The target stimulus words (presented in green and blue) were identical to those used at t1 (see Table 5-1) and consisted of 4 sets of 15 words, belonging to one of three semantic categories: (1) IEI-trigger substances (e.g., amalgam, solvents, exhaust emissions), (2) non-specific symptom words (e.g., headache, fatigue, dizziness), and (3) household items (e.g., oven, fork, bowl) as neutral stimuli. The neutral words were matched to the 15 trigger words and the 15 symptom words according to word length and word frequency (Belica et al., 1992). IEI-trigger stimuli were drawn from publications (e.g., Miller & Prihoda, 1999), self-reports of IEI/MCS-patients, and information disseminated by IEI-support groups. We tried to adequately represent the large spectrum of potential IEI-triggers by including odorous (e.g., paint smell, cigarette smoke) as well as invisible and inodorous agents (e.g., amalgam, radioactivity). The non-specific symptom words represent symptoms of high prevalence included in instruments for the assessment of somatoform symptoms (e.g., SCL-90R, SOMS). Most of the IEI trigger words and the symptom words used in the experimental paradigms were also included in the IEI

interview described above. Explicit emotional ratings (valence and arousal) of all stimulus words were obtained with the Self-Assessment Manikin (SAM; Bradley & Lang, 1994) during the first study (t1) one year ago (cf. results section study 1).

The extrinsic affective Simon paradigm (EAST; DeHouwer, 2003). As outlined above, the EAST is a variant of the implicit association task (IAT) originally proposed by Greenwald et al. (1998). In this study, the EAST consisted of three practice blocks and four test blocks. In the first practice block, participants were shown 10 unambiguous positive (e.g., nice, honest, friendly) and 10 negative (e.g., dangerous, bad, hostile) adjectives printed in white (on a black background) to which they should react as fast as possible by pressing one of two keys (a left key labeled "negative" and right key labeled "positive") on a computer keyboard. During the second practice block five words of each category (IEI-trigger words, neutral words I and II, and symptom words) were presented in pseudo random order. Each word was presented in "blue" and "green" for a total of 40 trials. Participants were instructed to respond to the color of the words by pressing a corresponding key. In the third practice block, participants were confronted with the actual EAST task demand, i.e., a block of mixed trials with white positive or negative adjectives and colored disorder related or neutral words (printed in blue or green). Participants were instructed to respond to the meaning of the word in case of white words and to the color of the word in case of words printed in green or blue. After this practice procedure, four test blocks with fixed pseudo randomized words (i.e., the same random order for all participants) followed with the restriction that the same word did not appear twice in a row and that the same response button was never required more than three times in a row. Each block included 85 stimuli in a different randomized order. In order to improve the accuracy of responses, visual feedback was provided during the practice blocks, indicating after each trial whether or not the given answer was correct. Feedback was not provided during the following test blocks. In the instructions given prior to the practice and test blocks, speed and accuracy were equally emphasized. To allow the detection of time course effects, block 1 and 2 (half 1 of the EAST) and block 3 and 4 (half 2) were constructed as equivalent with regard to the stimulus frequencies with only the order of stimuli varying across halves 1 and 2. In each half, the 60 stimulus words were presented twice (once in blue and once in green). Every adjective in white color was also presented twice comprising a total number of 160 trials in each test half. Each of the four test blocks was preceded by five warm-up trials, which were not included in the final data analysis.

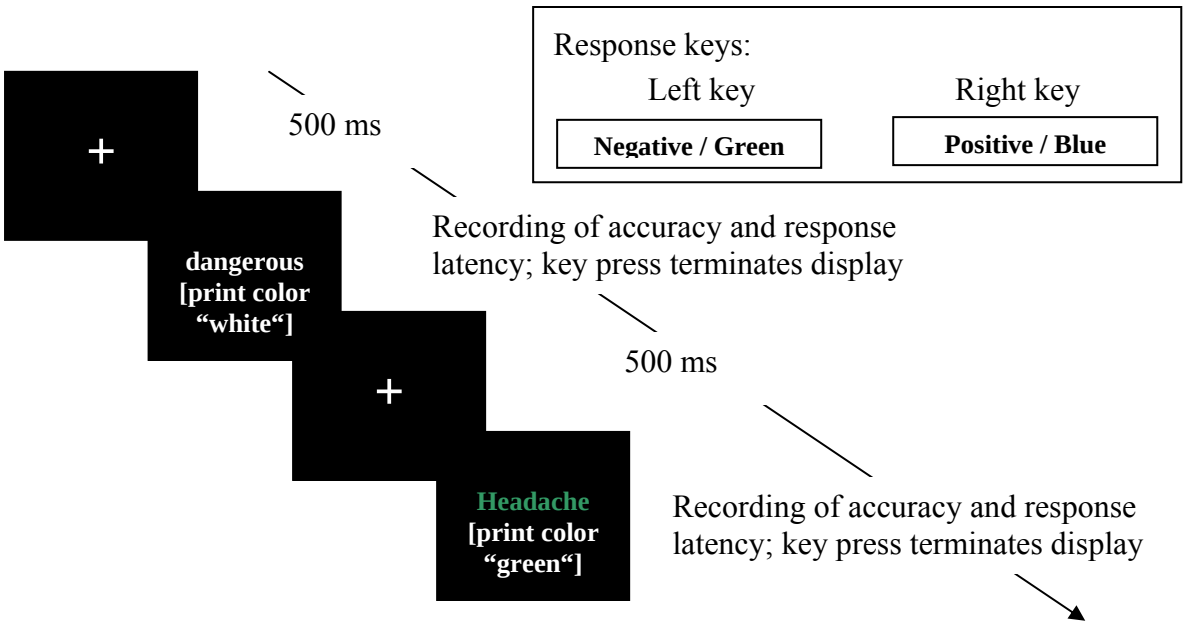

Figure 6-1: Two sample trials of the EAST task. Critical (symptoms or IEI-triggers) words, neutral words, and adjective trials were presented quasi randomly (see text for further details).

6.4.4. Apparatus and Software

In the EAST the stimuli were presented on a 17'' color monitor, connected to an IBM-compatible PC. The tasks were programmed and run with the ERTS software package (Beringer, 1996).

6.4.5. Procedure

All participants were tested individually in a session lasting about 1.5 hours. Participants were first interviewed with the SI-IEI. After a short break they performed the EAST and finally completed a number of psychological self-report instruments that are described above.

6.4.6. Parameterization of response times

Prior to any analysis of the response time (RT) data false reactions were recoded to missing values and thereby eliminated from any further analysis (1.88 % of trials in the CG,

2.37 % trials in the IEI and 1.87 % in the SFD group). The remaining RTs were corrected for outliers following a two-step procedure: (1) reaction times shorter than 200 ms and longer than 2000 ms were eliminated (0.81 % of trials in the CG, 2.92 % trials in the IEI and 1.08 % in the SFD group). (2) Separately for each experimental condition, response times larger than the individual mean plus 3 *SD*s were set to the individual mean value plus 3 *SD*s (0.78 % of trials in the CG, 0.64 % trials in the IEI and 0.68 % in the SFD group).

6.4.7. Statistical Analysis

Response time parameters were analyzed with mixed ANOVA designs with group (IEI, SFD, CG) as between-subjects factor and the different conditions (word valence and compatibility) of the EAST task as within-subjects factors. For all statistical analyses, results of the overall model and results of one-sided planned contrasts (Hager, 2002) according to our a priori hypotheses will be reported. Effect sizes will be reported as partial η^2 (η_p^2) for ANOVA effects and as Cohen's *d* (Cohen, 1992) for planned contrasts between groups.

6.5. Results

6.5.1. Psychological and symptom measures

Table **6-1** depicts sociodemographic information and the results of the diagnostic ratings and symptom measures. Gender was equally distributed across the three groups. The IEI group had a slightly higher mean age compared to the other two groups. As a result of the group definition criteria at t1, participants in the three groups still differ highly significantly with regard to the degree of chemical odor sensitivity (COSS), environmental sensitivity (ESQ) and the number of somatoform symptoms (SOMS, PHQ-15). As originally intended by the experimental design, the group with IEI is marked by a higher degree of chemical odor sensitivity and IEI-specific convictions concerning the harmful effects of environmental agents on their personal health (ESQ) compared to the other two groups (SFD and CG). Additionally, the level of somatization was found to be elevated in the IEI group compared to the CG, and comparable to the SFD group. The two clinical groups (IEI and SFD) report significantly higher levels of depression (PHQ-9) and trait anxiety (STAI) than the CG.

Table 6-1: Sample characteristics and symptoms at one-year follow up

	1 CG (n = 54)	2 IEI (n = 49)	3 SFD (n = 43)	ANOVA		Scheffé post hoc test [a]
	$M \pm SD$	$M \pm SD$	$M \pm SD$	F (2,143)	η_p^2 [f]	
Age	44.9 ± 11.4	50.0 ± 8.8	44.2 ± 12.8	4.0	.05	3>2
Somatoform symptoms (SOMS-2)	2.1 ± 5.1	14.2 ± 10.4	14.3 ± 8.1	38.3	.35	2,3>1
Chemical Odor Sensitivity (COSS)	11.6 ± 9.8	44.5 ± 9.7	19.8 ± 9.5	157.1	.69	2>3>1
Environmental Sensitivity (ESQ)	6.2 ± 5.5	16.0 ± 8.7	9.9 ± 6.2	25.9	.27	2>3>1
PHQ-15	3.4 ± 2.7	11.3 ± 6.4	12.0 ± 4.5	51.8	.42	2,3>1
PHQ-9 (depression)	2.2 ± 2.4	7.8 ± 6.2	9.1 ± 5.0	29.5	.29	2,3>1
Trait anxiety (STAI) [d]	45.9 ± 10.5	56.8 ± 12.3	61.1 ± 9.8	25.5	.26	2,3>1
	n (%)	n (%)	n (%)	$\chi^2(2, N = 146)$	φ	Repeated $2 \times 2\ \chi^2$ tests [b]
Female	37 (68.5)	35 (71.4)	35 (81.4)	2.3	.12 [c]	*ns*
Education (≥ 12 years)	24 (44.4)	13 (26.5)	18 (41.9)	4.0	.17 [c]	*ns*

Note. [a] Scheffé post-hoc test significant at $p \leq .05$ or [b] repeated $2 \times 2\ \chi^2$ tests at $p \leq .05$.
[c] measure of effect size for χ^2 (φ -coefficient: small = .10, medium = .30, large = .50).
[d] STAI values are t-transformed on the basis of population norms, corrected for age and gender.

6.5.2. The Extrinsic affective Simon task (EAST)

Data of 3 participants (2 IEI and 1 SFD participant) were excluded from further analysis because of more than 10 % error responses in the EAST. Mean reaction time values (*M*), *SD*s and the values of the three parameters of the ex-Gaussian distribution are depicted in Table 6-2. Prior to each analysis, box-plots of relevant dependent variables were inspected and extreme values and outliers were removed from further analysis.

Table 6-2: Mean RT values (*M*) and *SD*s for experimental groups and individually estimated parameters of the ex-Gaussian distribution μ, σ and τ for the different conditions of the EAST (*N* = 143)

	CG (*n* = 54)				IEI (*n* = 47)				SFD (*n* = 42)			
	M(SD)	μ	σ	τ	*M(SD)*	μ	σ	τ	*M(SD)*	μ	σ	τ
Word type												
1 IEI-trig. (com.)	695(123)	540	62	163	767(173)	569	60	222	728(124)	561	67	172
2 IEI-trig. (inco.)	684(109)	548	65	145	779(162)	586	72	216	719(111)	560	65	165
3 control 1 (com.)	687(106)	545	65	148	770(149)	585	63	203	704(105)	563	76	146
4 control 1 (inco.)	677(109)	519	59	165	760(172)	556	59	220	705(100)	539	52	176
5 Sympt. (comp.)	678(108)	521	53	166	768(168)	555	64	234	705(104)	546	68	168
6 Sympt. (inco.)	709(113)	557	78	162	806(156)	575	72	261	734(118)	557	74	187
7 control 2 (com.)	671(100)	542	66	137	735(141)	575	64	175	682(97)	555	69	136
8 control 2 (inco.)	672(109)	509	45	169	736(150)	547	58	208	684(105)	540	60	155

Note. Emotional Stroop (ES): IEI-triggers = *M*(1, 2) – *M*(3, 4); symptoms = *M*(5, 6) – *M*(7, 8). Implicit association effect (IA) IEI-triggers = *M*1 – *M*2; symptoms = *M*5 – *M*6.

Emotional intrusion effects. Figure 6-2 depicts the interference indices (difference between the latencies for symptom and IEI-trigger words and corresponding neutral words) separately for the experimental groups. In order to get pure indicators of emotional intrusion effects (analogously to the emotional Stroop effect) we aggregated raw latency data for the compatible and incompatible condition within neutral and critical word categories. We then analyzed mean latencies for critical words (IEI-triggers and symptoms separately) and neutral words with 2 × 2 mixed ANOVAs with experimental group as a between subjects factor and the two word valence conditions (emotional vs. neutral words) as a within subjects factor.

Analysis revealed for the symptom words a significant main effect for group ($F(2, 140) = 5.79$, $p < .01$, $\eta_p^2 = 0.08$), and a main effect for word category ($F(1, 140) = 155.82$, $p <$

.01, η_p^2 = 0.53). Simple main effect analysis revealed that the group main effect was due to slower over all reactions in the IEI group compared to the CG (p < .01) and SFD group (p < .02). The main effect for word category indicated that answer latencies were generally longer to symptom words compared to neutral words. Most importantly, there was a significant word category × group interaction effect (F(2, 140) = 8.93, p < .01, η_p^2 = 0.11). Planned comparisons according to our a priori hypotheses indicated that IEI participants responded disproportionately slower to symptom words than the CG (p < .01, d = 0.79). Similarly, SFD participants had significantly slower reactions to symptom words than the CG (p = .02, d = .54). Also the difference between the IEI and SFD participants was marginally significant indicating stronger emotional intrusion effects to symptom words in IEI compared to SFD ($p_{two\text{-}sided}$ = .05, d = .37). Replicating the analysis for IEI-trigger words (5 participants, 4 IEI and 1 CG, were excluded from this analysis because of outlier values) again revealed main effects for group (F(1, 135) = 4.33, p = .02, η_p^2 = 0.06) due to longer latencies in the IEI group, and word valence (F(1, 135) = 18.95, p < .01, η_p^2 = 0.12) due to slower responses to the IEI-trigger words. In contrast to the symptom words, there was no significant word category × group interaction effect for the IEI-trigger words (F(2, 135) = 1.33, p = .27, η_p^2 = 0.02). To allow for a direct comparison of the strength of the two emotional intrusion effects (for IEI-triggers and physical symptom words) we subjected the intrusion effects to a 3 × 2 mixed ANOVA. Simple main effect analyses indicated that the significant main effect for type of intrusion effect (F(1, 140) = 38.13, p < .01, η_p^2 = 0.02) was due to generally stronger emotional intrusion effects for physical symptom words compared to IEI-trigger words in all experimental groups.

Time course of the emotional intrusion effect. To focus on the time course of the emotional intrusion effect (Figure 6-3), we extended the analyses by adding test half (first test half vs. second test half) as another two-level within subjects factor. For the symptom words (after excluding 6 participants, 4 CG, 1 IEI, 1 SFD because of outlying values), analysis yielded significant main effects for group (F(1, 134) = 5.87, p < .01, η_p^2 = 0.08), word valence (F(1, 134) = 156.58, p < .01, η_p^2 = 0.54), and test half (F(1, 134) = 215.85, p < .01, η_p^2 = 0.62). Simple main effect analysis revealed that IEI participants responded generally slower than CG (p < .01) and SFD participants (p = .04). The two other main effects for valence and test half were attributable to symptom words being answered more slowly than neutral words and reactions in the first half being slower than in the second half. In addition to the previously seen word category × group interaction effect (F(2, 134) = 10.52, p < .01,

η_p^2 = 0.14), results yielded a significant word category × test half interaction (F(1, 134) = 115.11, p < .01, η_p^2 = 0.46), a significant test half × group interaction effect (F(2, 134) = 3.95, p = .02, η_p^2 = 0.06), and a marginally significant word valence × test half × group interaction effect (F(2, 134) = 2.64, p = .08, η_p^2 = 0.04). Simple main effect analysis revealed that in all groups increases in performance from test half one to half two were disproportional larger for symptom words compared to neutral words. There is a trend for this effect being marginally stronger in the IEI group compared to the CG (Scheffé post-hoc test: p = .09). Finally, reaction times for IEI participants yielded significantly stronger overall decreases compared to the CG group (Scheffé post-hoc test: p = .02). For the IEI trigger words (after excluding 2 IEI participants because of outlying values), analysis yielded main effects for group (F(2, 138) = 5.38, p < .01, η_p^2 = 0.07), word category (F(1, 138) = 21.60, p < .01, η_p^2 = 0.14), and test half (F(1, 138) = 59.39, p < .01, η_p^2 = 0.30). Only the word valence × test half interaction effect turned out as significant (F(1, 138) = 30.69, p < .01, η_p^2 = 0.18), replicating the finding of a disproportional larger increase in performance for the critical word category compared to neutral words.

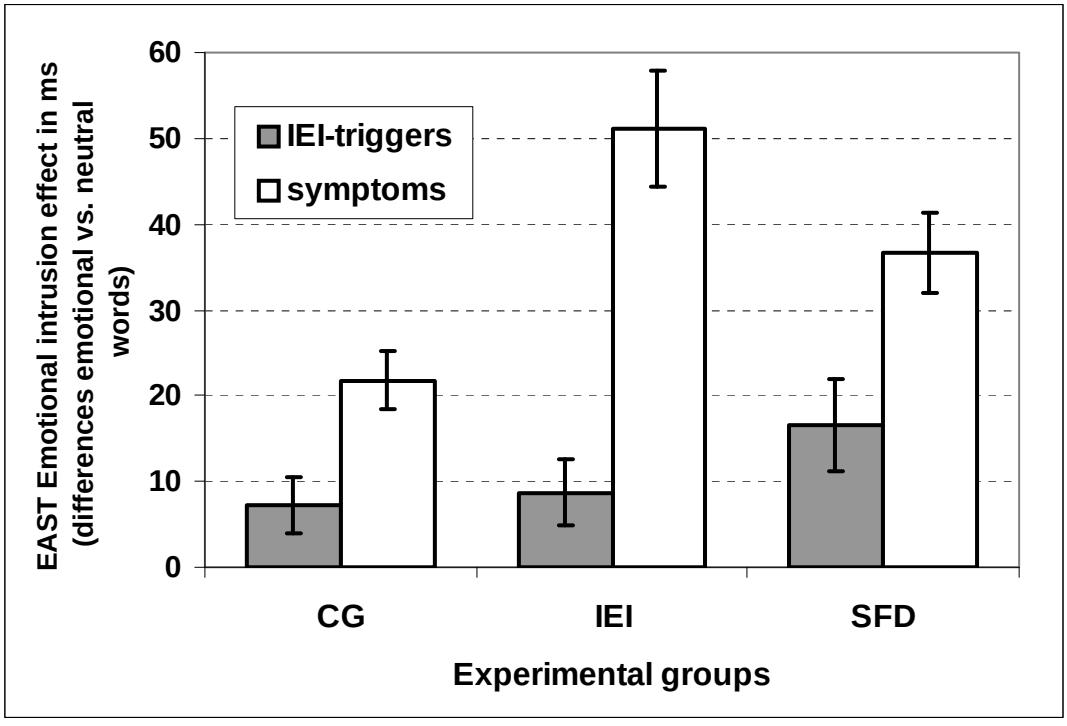

Figure 6-2: Mean indices (in ms) and standard errors of the emotional intrusion effect derived from the extrinsic affective Simon tasks (EAST) for the experimental groups and the two disorder related word categories (IEI-triggers and symptoms). Data represent difference scores between the matched neutral words and the two disorder related word categories.

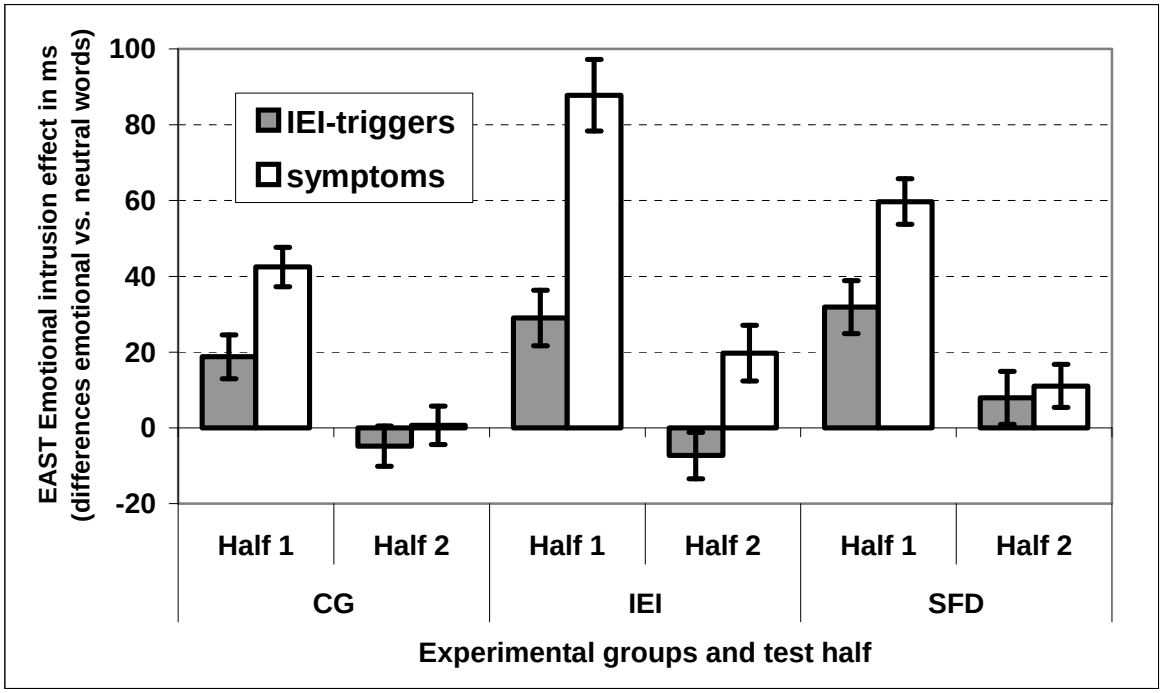

Figure 6-3: Time course of the emotional intrusion effect derived from the extrinsic affective Simon tasks (EAST) for the experimental groups and the two disorder related word categories (IEI-triggers and symptoms).

Implicit association effects. Figure 6-4 depicts the indicators of implicit attitudes toward IEI-trigger and symptom words separately for the experimental groups. As we subtracted latencies for extrinsically negative responses ("compatible" condition) from extrinsically positive responses ("incompatible" condition) for critical words (IEI-triggers and symptoms), positive difference scores indicate a negative attitude (i.e., a stronger implicit association with the concept "negative"). As in case of the emotional intrusion effect, we analyzed data for the two critical word categories (IEI-trigger words and symptom words) separately.

After excluding 7 participants (6 IEI, 1 CG) from the analysis because of outlier values, we computed a 3 × 2 ANOVA for the symptom and corresponding neutral word latencies with experimental group as a between subjects factor, and word valence-answer compatibility as a two-level within subjects factor. Results yielded main effects for group ($F(2, 133) = 5.43$, $p < .01$, $\eta_p^2 = 0.08$) and compatibility ($F(2, 133) = 70.13$, $p < .01$, $\eta_p^2 = 0.35$). Simple main effect analysis indicated that the IEI participants reacted significantly slower than the CG ($p < .01$) and marginally slower than the SFD group ($p = .05$). Furthermore, all groups were significantly faster when a (compatible) negative answer was required for symptom words compared to a (incompatible) positive answer, suggesting

implicit negative attitudes toward symptom words in every group. Additionally, there was a significant group × compatibility interaction effect ($F(2, 133) = 3.89$, $p = .02$, $\eta_p^2 = 0.06$). Planned contrasts according to our a priori hypotheses indicated that this interaction was based on more negative attitudes toward symptom words in IEI-participants compared to the CG ($p = .01$, $d = 0.58$), but not for the SFD group compared to the CG ($p = .84$, $d = 0.04$). Unexpectedly, attitudes toward symptoms of the IEI and SFD group did also differ significantly ($p = .03$, $d = 0.54$).

After excluding 3 IEI and 2 CG participants because of outlier values, we submitted mean latencies for IEI-trigger words to a 3 × 2 ANOVA. Results revealed a main effect for group ($F(2, 133) = 3.68$, $p = .03$, $\eta_p^2 = 0.05$), but no main effect for compatibility ($F < 1$). Additionally, there was a significant group × compatibility interaction effect ($F(2, 135) = 4.03$, $p = .02$, $\eta_p^2 = 0.06$). Planned contrasts according to our a priori hypotheses indicated that this interaction was based on more negative attitudes toward IEI-trigger words in IEI-participants compared to the CG ($p = .01$, $d = 0.58$) and SFD group ($p = .02$, $d = 0.48$). The two non-IEI groups did not differ significantly ($p = .89$, $d = 0.03$).

Replicating these analyses with the two corresponding neutral (household related) word categories revealed neither main effects for compatibility (neutral 1: $F < 0.5$, neutral 2: $F = 1.80$), nor group × compatibility interaction effects (neutral 1: $F < 0.01$, neutral 2: $F = 0.82$).

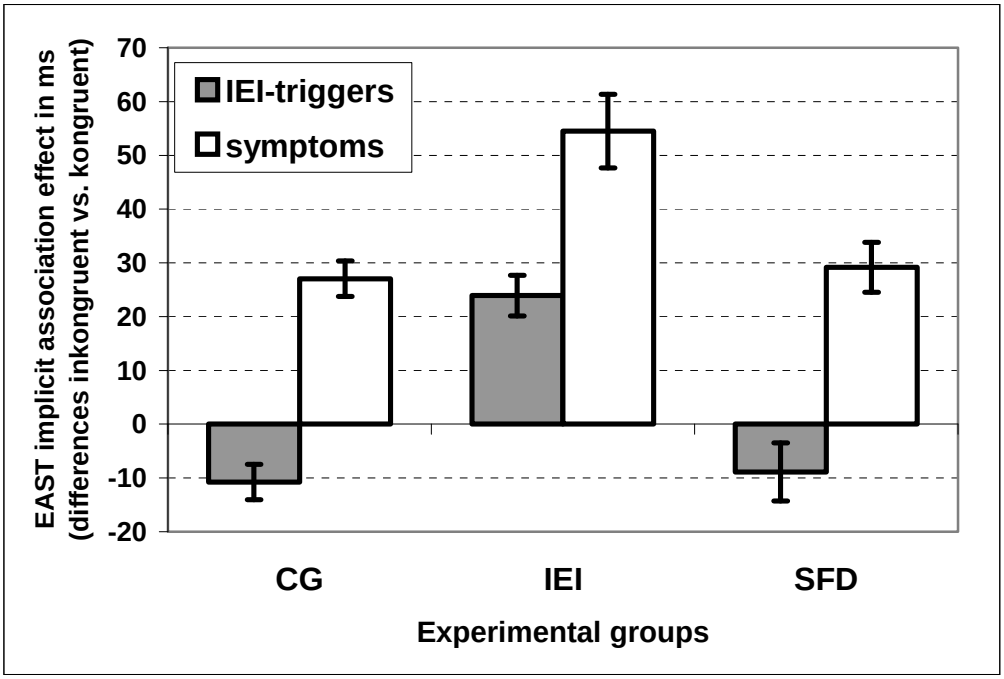

Figure 6-4: Mean indices (in ms) and standard errors of the implicit association effect derived from the extrinsic affective Simon tasks (EAST) for the experimental groups and the two disorder related word categories (IEI-triggers and symptoms).

Time course of the implicit association effect. In case of the emotional intrusion indicators, we also extended analysis for the implicit association scores by adding "test half" as another 2-level within-subjects factor. For the symptom words (after the exclusion of the most severe outlier cases; 1 SFD and 3 CG), results revealed main effects for group ($F(2, 136) = 7.82$, $p < .01$, $\eta_p^2 = 0.10$), compatibility ($F(1, 136) = 42.21$, $p < .01$, $\eta_p^2 = 0.24$), and test half ($F(1, 136) = 316.33$, $p < .01$, $\eta_p^2 = 0.70$). Accordingly, IEI participants reacted generally slower than the other two groups and all participants performed faster on compatible trials (compared to incompatible ones) and in the first half (compared to the second). A significant compatibility × test half interaction ($F(1, 136) = 88.15$, $p < .01$, $\eta_p^2 = 0.39$) indicated that the association effect was limited to the first half and absent in the second half. A significant compatibility × test half × group interaction ($F(2, 136) = 3.22$, $p = .04$, $\eta_p^2 = 0.05$) indicated that the size of the compatibility effect in half 1 was moderated by group membership (the IEI-group had significantly stronger compatibility effects than the CG).

For the IEI trigger words (after the exclusion of the 5 most severe outlier cases, 1 IEI and 4 SFD), analysis revealed main effects for group ($F(2, 135) = 4.89$, $p < .01$, $\eta_p^2 = 0.07$) and test half ($F(1, 135) = 96.93$, $p < .01$, $\eta_p^2 = 0.42$). Only the compatibility × group interaction turned out marginally significant ($F(2, 135) = 2.49$, $p = .09$, $\eta_p^2 = 0.04$). None of the other interaction effects reached significance ($F < 1$).

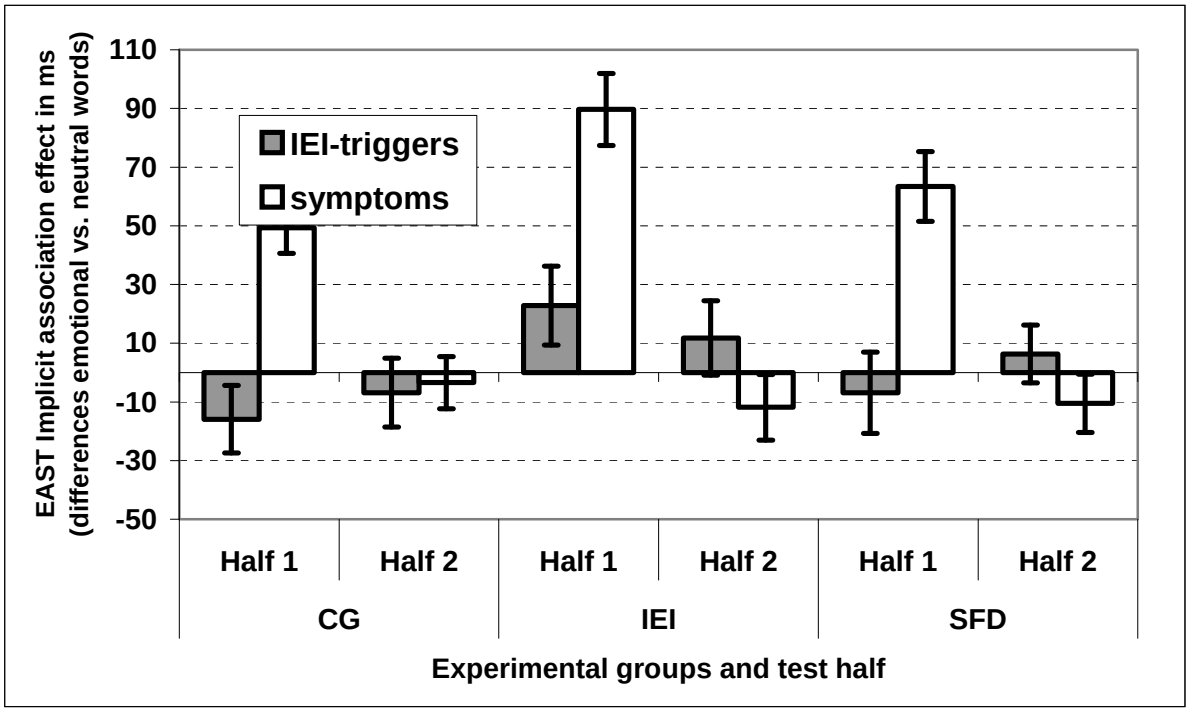

Figure 6-5: Time course of the implicit association effect derived from the extrinsic affective Simon tasks (EAST) for the experimental groups and the two disorder related word categories (IEI-triggers and symptoms).

Replication of analysis with log-transformed RTs. In order to account more accurately for the differences in baseline speed performance documented in the analysis of raw RT data above, we repeated the analysis of the emotional intrusion effects with log-transformed reaction time data. Difference scores computed on the basis of log-transformed RT data represent ratio scores that are less dependent on differences in baseline (speed) performance (e.g., Salthouse & Hedden, 2002). Condition × group interaction effects based on log-transformed RT data therefore indicate disproportional or over-additive effects that are not attributable to differences in general response speed.

Regarding the emotional Stroop indicator for symptom words and IEI-trigger words, the pattern of results was mostly replicated (group × word valence interaction for symptom words: $F(2, 140) = 7.14$, $p < .01$, $\eta_p^2 = 0.09$; planned comparisons: CG vs. SFD $p < .05$, $d = .72$; CG vs. IEI $p < .01$, $d = .47$). Only the trend toward a stronger emotional intrusion effect for symptom words in the IEI compared to the SFD group ($p = .11$, $d = .31$) was no longer apparent with log-transformed data.

In the case of the implicit association results for IEI-trigger words and symptom words, the most important group × compatibility interaction effects were marginally reduced in size but remained significant for the trigger words ($F(2, 135) = 3.69$, $p = .03$, $\eta_p^2 = 0.05$) and marginally significant for the symptom words ($F(2, 133) = 2.84$, $p = .06$, $\eta_p^2 = 0.04$), respectively. Between groups contrasts indicated that the groups still differed in the expected direction regarding the implicit association effect for IEI-trigger words, with the IEI group showing stronger association (i.e., compatibility) effects than the other two groups (IEI > CG: $p = .02$, $d = 0.53$; IEI > SFD, $p = .03$, $d = 0.49$; CG = SFD: $p = .95$, $d = 0.01$). Similarly, the pattern of results remained constant for the symptom words (IEI > CG: $p = .04$, $d = 0.45$; IEI > SFD, $p = .04$, $d = 0.50$; CG = SFD: $p = .97$, $d = 0.01$). Regarding the time course effects, results of corresponding analysis with log-transformed data closely mirrored the above results with raw latencies for the emotional intrusion and implicit association effect, that is a generally stronger effect in the first test block compared to the second test block.

RT distribution analysis. In order to elucidate the origin of the slowing effect observed mainly for symptom words in the IEI and SFD participants, we fitted the ex-Gaussian distribution (characterized by the three parameters μ, σ, and τ as described above) to the individual response time data using the program QMPE 2.18 (Cousineau, Brown, & Heathcote, 2004; Heathcote, Brown, & Mewhort, 2002). The quantile maximum likelihood estimation (QMLE) algorithm allows for the estimation of parameters with a comparably low

number of single trials (of about 40 trials). However, we have to acknowledge that our number of 30 trials in each experimental condition is still at the lower end even for the QMLE algorithm to yield robust estimates. Individual RTs shorter than 200 ms and longer than 3000 ms were excluded before parameter estimation. In order to circumvent statistical problems of parameter dependency of the ex-Gaussian parameters (e.g., Schmiedeck, Oberauer, Wilhelm, Süß, & Wittmann, in press), we first computed differences between the neutral word condition and the critical word condition for each parameter. These differences were subjected to a MANOVA with group as independent variable and the three differences scores for μ, σ, and τ as dependent variables (for absolute parameter estimates see Table 6-2). In such a model, the intercept indicates general effects of word valence on the three parameters, whereas the group effect codes group × valence interaction effects. We computed this analysis separately for symptom words and IEI-trigger words, the implicit association effect, and the emotional intrusion effect, respectively. Because the Box-M statistic in some cases indicated a significant violation of the homogeneity of variance assumption, *F*-values for multivariate tests based on the more robust Pillai's trace statistic are reported.

Emotional intrusion effect. Prior to the analysis of the symptom-neutral word differences we excluded the data of four members of the IEI group and one participant of the SFD group because of an extreme outlying value on the μ, σ, and τ difference score. The MANOVA results revealed a significant main effect of the intercept (Pillai's trace: $F(3, 133) = 39.02$, $p < .01$, $\eta_p^2 = 0.47$) and group (Pillai's trace: $F(6, 268) = 2.54$, $p = .02$, $\eta_p^2 = 0.05$). Between subjects effects yielded no significant intercept for μ ($F(1, 135) = 2.33$, $p = .13$, $\eta_p^2 = 0.02$), but significant intercepts for σ ($F(1, 135) = 7.61$, $p = .01$, $\eta_p^2 = 0.05$) and τ ($F(1, 135) = 40.26$, $p < .01$, $\eta_p^2 = 0.23$), respectively. More importantly, there was a highly significant interaction effect between group and the τ parameter ($F(2, 135) = 6.45$, $p < .01$, $\eta_p^2 = 0.09$). No such interaction was observed for either the μ or the σ parameter ($F(2, 135) < 1.40$, $p > .20$, $\eta_p^2 < 0.02$). One-sided planned contrasts regarding the effect of the τ parameter according to our a priori hypotheses revealed a highly significant difference between the CG and the IEI group ($p < .01$; $d = .73$) and a significant difference between the CG and the SFD group ($p < .05$; $d = .36$).

After excluding six participants (1 IEI, 2 SFD, 3 CG) because of outlying values on at least one of the three parameters, we replicated the analysis for the IEI-trigger word differences. In accordance with the results of the raw reaction time data, results revealed only a significant overall effect of the intercept ($F(3, 132) = 6.16$, $p < .01$, $\eta_p^2 = 0.12$) but not for

group ($F(6, 266) = 1.31$, $p = .25$, $\eta_p^2 = 0.03$). The significant effect of the intercept was mainly attributable to the significant effect of μ ($F(1, 134) = 11.03$, $p < .01$, $\eta_p^2 = 0.08$) and σ ($F(1, 134) = 4.40$, $p = .04$, $\eta_p^2 = 0.03$). The intercept of τ did not reach significance ($F(1, 134) < 1$, $p > .90$).

Implicit association effect (IAT). Prior to the analysis of the IAT effect (reflected in the difference between compatible and incompatible trials) for the symptom words, we excluded data of one IEI and two SFD participants because of extreme outlying values on one of the difference scores for μ, σ, or τ. The MANOVA results revealed a significant main effect of the overall intercept ($F(3, 135) = 14.98$, $p < .01$, $\eta_p^2 = 0.25$) but not for group ($F(6, 272) = 1.47$, $p = .19$, $\eta_p^2 = 0.03$). The inspection of the between subjects effects for the intercept revealed that the significant overall effect was due to larger values of all three parameters in the incompatible compared to the compatible condition (μ: $F(1, 137) = 11.66$, $p < .01$, $\eta_p^2 = 0.08$; σ: $F(1, 137) = 7.81$, $p < .01$, $\eta_p^2 = 0.05$; τ: $F(1, 137) = 4.28$, $p = .04$, $\eta_p^2 = 0.03$).

After excluding two participants (one IEI and one CG) because of extreme difference scores for one of the three parameters, we replicated the analysis for the IEI-trigger word estimations. Neither the overall effect for the intercept nor the factor group reached significance (Fs < 1). Inspection of the between-subjects effects was therefore unnecessary. In line with our raw data analysis, significant overall negative implicit association effects could only be found for symptom words, but not for the IEI-trigger words. However, the finding of significant and stronger negative associations of IEI-trigger words in the IEI group could not be replicated with the ex-Gaussian parameter estimations – a finding that might in part be attributable to the comparatively small number of trials in each condition as mentioned above. As this problem of an insufficient amount of single trials would become even worse when re-analyzing habituation effects (i.e., dividing the trials in two halves), we did not replicate the estimation of ex-Gaussian parameters for the single test blocks as demonstrated above.

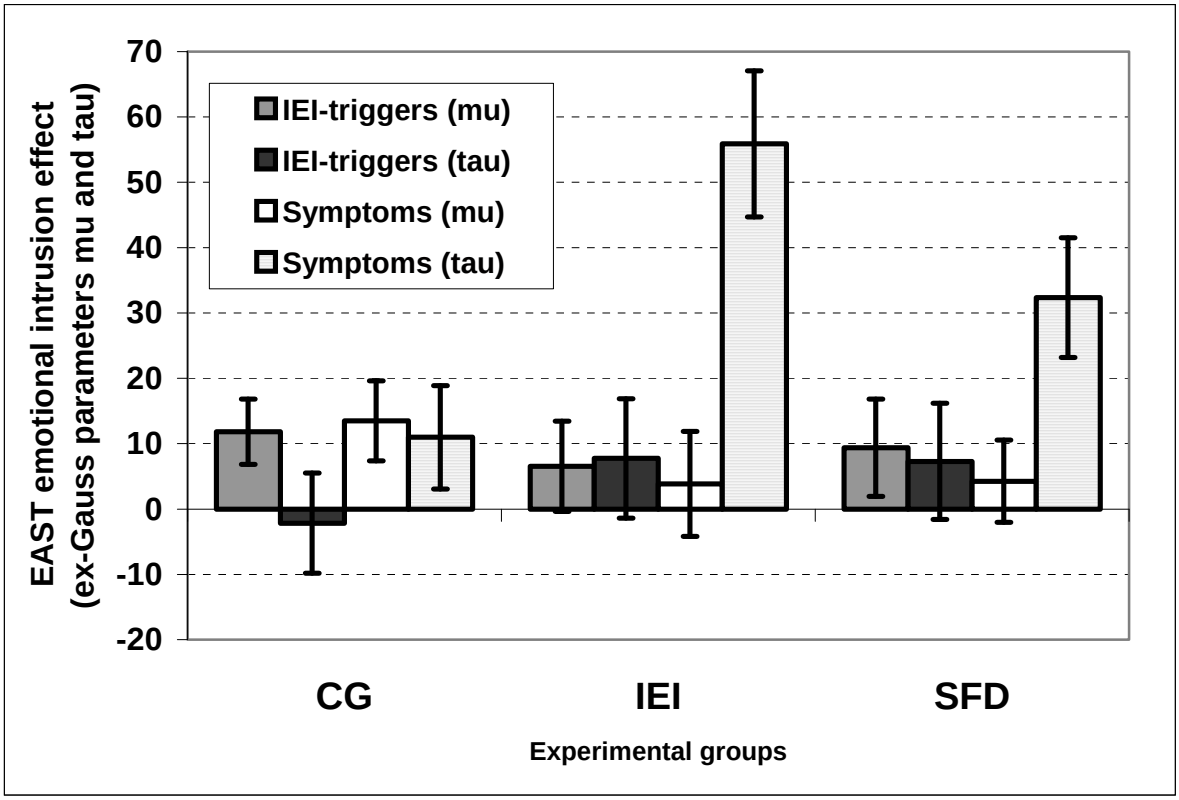

Figure 6-6: Parameters of the ex-Gaussian distribution (μ, and τ) reflecting emotional intrusion effects for the experimental groups and the two disorder related word categories (IEI-triggers and symptoms).

Reliability analysis of experimental effects. In the realm of experimental psychology, measurement *reliability* represents a frequently disregarded issue (e.g., Sander, 2005). In many articles, an effect is considered as "reliable" if it reaches significance. This equation of the terms significance and reliability implies a dichotomous conceptualization of reliability that blurs a more accurate and dimensional consideration of measurement accuracy. As we have no exact replication of experimental effects, we will focus on reliability in terms of the internal consistency between single trials of the referring experimental condition. According to the standard procedure to compute Cronbach's α coefficients for IAT-scores (e.g., Bosson, Swann, & Pennebaker, 2000; Egloff & Schmukle, 2002), we first computed difference scores for every single word referring to the two critical word categories (IEI-trigger words and symptoms) by subtracting the congruent condition (critical word paired with the "bad" response key) from the incongruent condition (critical word paired with the "good" response key). This procedure controls for individual differences in baseline reaction speed (Bosson et al., 2000). As every word (15 IEI-trigger words and 15 symptom words) was repeated twice in each condition (congruent vs. incongruent), there were 30 difference scores in each word category for the computation of the Cronbach's α coefficient. With regard to the internal

consistency of the emotional intrusion effect (analogous to the emotional Stroop effect reported in the first study), we computed difference scores between the critical words (IEI-triggers and symptoms) and the neutral words that were matched to the critical words in terms of word length (pair-wise) and word frequency (list-wise) (Table 5-1). Since every word was repeated four times we have a whole of 60 difference scores for computing Cronbach's α of the emotional intrusion effect. The results of the Cronbach's α computation for the different experimental indicators are summarized in Table 6-3.

Table 6-3: Internal consistency for the experimental indicator of the Extrinsic Affective Simon Task (EAST) for the entire sample (N = 143) and the three experimental groups separately.

	Implicit association effect Cronbach's α (30 trials)		Emotional intrusion effect Cronbach's α (60 trials)	
	IEI-triggers	Symptoms	IEI-triggers	Symptoms
Entire sample (N = 143)	**.46**	**.47**	**.07**	**.31**
CG (N = 54)	.36	.37	-.21	-.28
IEI (N = 47)	.44	.54	-.03	.46
SFD (N = 42)	.57	.36	.35	.10

Table 6-3 indicates that reliability coefficients were generally at the lower end. In case of the implicit association effects, Cronbach's α ranged from .36 to .57, which can be considered as acceptable for experimental measures. In contrast, α-coefficients of the emotional intrusion effect were close to zero or even negative. Only in the IEI group was an acceptable α coefficient (.46) detactable.

6.5.3. Correlation analyses

Table 6-4 depicts the correlations between the different experimental indicators of the EAST (i.e., the emotional intrusion effects for IEI-trigger and symptom words and the implicit association effects for IEI-trigger and symptom words of the EAST) with self-report instruments. For the EAST, correlations are presented for the classical indicator based on raw reaction time differences and for differences based on the τ parameter of the ex-Gaussian distribution, because this parameter has been shown to differ across groups in our previous

analysis. The correlation pattern reveals that only the emotional intrusion effect for symptom words based on raw reaction time means is consistently associated with somatic symptom measures (SOMS, PHQ-15) and chemical sensitivity (as measured by the COSS). Weaker associations of the symptom word intrusion effect are identifiable with trait anxiety and depression. The τ parameter of the symptom word intrusion effect correlates exclusively with chemical sensitivity, whereas the μ parameter (not shown in Table 6-4) is at least weakly (r = .17) associated with the somatic symptoms score (PHQ-15). Regarding the three other experimental indicators (i.e., the emotional intrusion effect for IEI-triggers and the implicit association effects for symptom words and trigger words), none of their associations to the self-report measures, either for the traditional score nor the score based on the τ parameter, reached significance.

Regarding the emotional Stroop effect for symptom words of the first assessment (t1) one year ago, in line with our expectations there was a small but significant correlation (r = .21) with the emotional intrusion effect for symptoms derived from the EAST. In contrast, the negative association of the t1-symptom word intrusion effect with the EAST implicit association effect for symptoms was rather unexpected and remains difficult to interpret.

Table 6-5 depicts the correlations between the traditional measures of experimental effects (RTs difference score) and the corresponding parameters of the ex-Gaussian distribution. Regarding the emotional intrusion effect, the τ parameter correlated strongest with the traditional intrusion score for IEI-trigger and symptom words. For the traditional implicit association effect, medium sized correlations were observable with the μ, and τ parameter.

Table 6-4: Cross-sectional and longitudinal correlations between experimental indicators of attentional bias and implicit associations (EAST) and psychological (symptom) measures for the total sample (*N* = 143).

	Emotional intrusion effect (EAST)				Implicit association effect (EAST)			
	IEI-triggers		Symptoms		IEI-triggers		Symptoms	
	M [a]	τ [b]	M [a]	τ [b]	M [a]	τ [b]	M [a]	τ [b]
Somatic Symptoms (SOMS)	.15	.09	**.27** [c]	.14	.02	-.05	.01	-.14
Chemic. Odor Sens. (COSS)	.01	.03	**.33** [c]	**.25** [c]	.04	-.02	.11	-.10
Environm. Sensitivity (ESQ)	-.04	.06	**.18** [d]	.06	.06	-.05	.15	-.10
Trait anxiety (STAI)	.08	-.01	**.20** [d]	.08	-.08	-.04	.01	-.10
PHQ-15 (somatic symptoms)	.08	.07	**.34** [c]	.05	.04	-.05	.12	-.11
PHQ-9 (depression)	.09	.01	**.27** [c]	.06	-.01	-.06	.07	-.13
Longitudinal correlations with t1-Emotional Stroop Effect (one year before)								
Emotional Stroop t1 (IEI-triggers)	.09	.03	-.05	-.06	-.09	.01	.12	**-.21** [d]
Emotional Stroop t1 (symptoms)	.04	.01	**.21** [d]	.10	-.07	.03	**-.19** [d]	-.11

Note. [a] Score based on mean values of raw RTs
[b] Score based on the τ parameter of the ex-Gaussian distribution
Significance levels: [c] $p \leq .01$; [d] = $p \leq .05$

Table 6-5: Correlations between experimental indicators (Mean RTs *M* and the ex-Gaussian parameters: μ, σ, τ) of attentional bias and implicit associations (EAST) for the total sample (*N* = 143).

	Emotional intrusion effect (EI)						Implicit association effect (IA)					
	IEI-triggers			Symptoms			IEI-triggers			Symptoms		
M	μ	σ	τ	μ	σ	τ	μ	σ	τ	μ	σ	τ
EI (IEI-trig.)	**.22** [a]	**.26** [a]	**.46** [a]	-.13	-.04	.09	-.02	-.12	-.12	-.10	-.05	-.04
EI (sympt.)	-.06	.02	.02	**.18** [b]	.14	**.47** [a]	.01	-.01	.06	.02	-.12	.13
IA (IEI-trig.)	-.02	-.03	-.10	-.13	-.01	.14	**.48** [a]	.14	**.46** [a]	**.29** [a]	**.20** [b]	-.06
IA (sympt.)	.01	.07	-.05	.12	.11	-.03	**.20** [b]	-.04	.16	**.31** [a]	.12	**.33** [a]

Note. Significance levels: [a] $p \leq .01$; [b] = $p \leq .05$.

6.6. Discussion

In the second study, we examined whether participants with IEI or SFD show evidence of selective attention and differential implicit emotional associations of critical word stimuli (IEI trigger substances and symptom words) in the extrinsic affective Simon task (EAST). The experimental group membership that was initially determined during the first assessment one year ago could be validated by the psychological self-report measures of somatoform symptoms and chemical sensitivity. As a consequence of the high one-year symptom stability, the IEI group was marked by elevated levels of chemical sensitivity and medically unexplained somatic symptoms, whereas the SFD group reported only elevated levels of somatoform symptoms in the absence of extreme chemical sensitivity values. Both clinical groups still reported similar symptom patterns and overlapping psychological risk factors (e.g., trait anxiety or negative affectivity) for somatization, and were clearly distinguishable from non-somatoform and non-IEI participants (CG).

The experimental findings replicated the results of our first assessment with the emotional Stroop paradigm. Participants with IEI and SFD had a stronger emotional intrusion effect (as evidence for symptom focused attention) toward symptom words compared to the CG. In line with previous findings, no such intrusion effect was found for IEI-trigger words.

The findings of study 2 therefore support our hypothesis that IEI shares cognitive abnormalities with traditional SFD (as defined in DSM-IV), namely elevated symptom-focused attention. When we dissociated different components of individual response time distributions with the ex-Gaussian distribution, effects were evident for the τ parameter. This result is quite in line with the prediction of Brown's (2004) model of medically unexplained symptoms: the abnormalities were strongest related to later, probably more controlled stages of attentional processing.

Furthermore, in line with the results of study 1, study 2 supports the suggestion that people with IEI process IEI-trigger related information differently compared to symptom words: Although we found a negative implicit association effect for the trigger words in people with IEI, this effect was smaller than the corresponding effect of the symptom words and there was no evidence for an emotional intrusion effect (in the sense of an attentional bias) as found for the symptom words. Probably the implicit negative emotional connotations of the IEI-trigger words were not strong enough to produce emotional intrusion effects. Alternatively, one might speculate that negative implicit associations are a necessary but not sufficient component of danger schemata in order to produce threat driven emotional intrusion effects (as is obvious for the symptom words). Perhaps other conditions (e.g., evolutionary adaptive importance, proximity in semantic networks to representations of personal threat or harm) are additionally necessary for certain stimuli to momentarily interrupt ongoing information processing (i.e., produce emotional intrusion effects). In this way, negative implicit associations might represent "milder" negative schematic representations, whereas the emotional intrusion effect (as traditionally measured with the emotional Stroop) rather represents a combination of (a) active negative schematic representations and (b) a failure to inhibit those active schemata. However, given these experimental results it seems unlikely or at least premature to consider IEI mainly as an environmental *anxiety* disorder.

6.6.1. Emotional intrusion effects in the EST and the EAST

General slowing effects toward words of negative valence analogously to the emotional Stroop effect have been recognized previously in a variant of the EAST (De Jong, Van den Hout, Rietbroek, & Huijding, 2003)[6]. If we compare the emotional intrusion effect

[6] De Jong et al. (2003) interpret this finding as a "negativity bias" (p. 532). Our actual understanding of the emotional Stroop phenomenon casts heavy doubt on the view that "(negative) word valence" is the crucial and

derived from the emotional Stroop task (at t1) with the results of the EAST (one year later at t2) we find several hints that both experimental paradigms measure parts of a common construct, namely selective attention or emotional intrusion effects. Firstly, the pattern of results of the emotional intrusion effects for the different words categories (IEI-trigger words and symptom words) and the three experimental groups seems equivalent for the two paradigms: Compared to the CG, intrusion effects for the symptom words, but not for the trigger words are elevated in both the SFD and the IEI group. Additionally, the pattern of correlations between the emotional intrusion effect for symptom words in the EST and the EAST appear similar. As reported for the EST at t1 (Table 5-7), the EAST symptom word intrusion effect is significantly correlated with somatic symptoms (SOMS), chemical odor sensitivity (COSS), and environmental sensitivity (Table 6-4).

If both indicators of emotional intrusions for the symptom words indicate a common underlying construct, why do they correlate only weakly (r = .21)? Three possible reasons may limit the overlap between the indicators derived from the two tasks: Firstly, both paradigms (EAST and EST) differ considerably in their special task demands, which may increase task specific method variance. In this respect the higher task complexity of the EAST might partly be responsible for the stronger emotional intrusion effects compared to the easier EST. Secondly, although less is known about the temporal stability of emotional intrusion effects, it seems likely that individual changes might have occurred during the one-year period that separates our two experiments. Thirdly and perhaps most importantly, experimental measures like the emotional Stroop effect suffer from rather low reliability (i.e., internal consistency) that mathematically limits their validity. In our case the corresponding Cronbach's α indices of the emotional intrusion effect for symptom words in the EST at t1 and the EAST at t2 were .05 and .31, respectively. The true validity after correction for attenuation in both measures would therefore rise from r = .21 to a perfect association of about $r = 1$[7].

We conclude that emotional intrusion effects are observable in paradigms different to the original EST (e.g., the EAST), but that these effects, irrespective of the paradigm, are contaminated with large proportions of error variance.

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sufficient factor in producing such slowing effects. Relatedness to "personal concerns" seems more important than "negative valence".

[7] Note that because of the very low internal consistency of the t1-score and the "attenuation paradox" the exact value of r after a double correction of attenuation would be 1.69.

6.6.2. Time course of the emotional intrusion and implicit association effect

The length of the EAST provided us with the possibility of looking at the development of the implicit association and emotional intrusion effect over time. Across the three groups both effects were almost limited to the first half of the task or at least declined heavily in the second half. Regarding the emotional intrusion effect, similar observations have been documented with the emotional Stroop task and were interpreted as a habituation-like effect (McKenna & Sharma, 1995; McNally, Riemann, & Kim, 1990; Witthöft, Rist, & Bailer, under review). Generally, two mechanisms seem plausible to account for the decline of the effects in the second test half. Firstly, the repeated presentation of the critical word stimuli might have tempered their negative emotional connotation implying a kind of (passive) habituation effect. Secondly, the increased task familiarity in half two might have provided additional cognitive resources for (actively) inhibiting irrelevant task features such as the semantic meaning or emotional connotation of the word stimuli. Although the current study does not allow a decision between these two mechanisms or a quantification of their relative contributions, both mechanisms might be highly relevant for interventions that try to directly modify the symptom focused attentional style.

6.6.3. The nature and consequences of emotional intrusion and implicit association effect

What does implicit evaluation mean in our context? Focusing on the nature of the EAST we can summarize that this task indirectly (i.e., without the participants knowing the exact mechanisms of the task) assesses associations of negative and positive concepts (i.e., implicit evaluations or attitudes). If those evaluative connotations are strong enough to automatically (i.e., without volitional cognitive effort) influence the response behavior of participants in the EAST task, as seen for the IEI-trigger words in the IEI group and for symptom words across all three groups, we may infer that these associations or connotations similarly (implicitly) affect information processing outside the experimental context and might contribute to the initiation of defense strategies (e.g., avoidance behavior). As empirical evidence for this hypothesis there are significant associations ($p < .05$) of the implicit association effect for IEI-trigger words with the avoidance behavior assessed in the IEI-interview ($r = .23$ for avoidance behavior at t1 and $r = .21$ for avoidance behavior at t2).

Regarding the emotional intrusion effect, many studies have employed the emotional Stroop paradigm in clinical and normal settings. Still the question remains as to what

processes the effect (i.e., a slowing in the light of negative and individually relevant information) actually reflects. Is it the exact meaning of the word in terms of its semantic content or rather an (implicit) emotional connotation (attached via classical conditioning or associative learning) associated with the word stimulus? Evidence for the latter view is growing (e.g., Richards & Blanchette, 2004). However, even if the emotional Stroop task assesses the strength of (negative) emotional connotations, intrusion effects result from at least two sources: firstly, a strong emotional association or connotation and secondly a poor ability to overcome or override the activation of the emotional association in order to perform the actual task. Thus, strong emotional intrusion effects remain ambiguous, either demonstrating easy activation of emotional connotation, or poor inhibition of such associations or a combination of both. Consequently, the interpretation of our experimental findings is limited by the current knowledge regarding the nature of implicit association (study 2) and emotional intrusion effects (study 1 and 2).

6.6.4. RT distribution analysis

Our findings with regard to the RT distribution analysis with the ex-Gaussian distribution are mixed. The decomposition of different parts of individual response time distributions as reflected in the three parameters μ, σ, and τ revealed further interesting information about the nature of the emotional intrusion effect on a mean level. In this respect our data confirm our hypotheses that the enhanced intrusion effect for symptom words in our two clinical groups is mainly a function of an increase in the τ parameter. Following former interpretations of the τ parameter as an index of failures in controlled attention (e.g., Spieler et al., 1996) it seems reasonable to conclude that slowing effects to symptom words arise from a failure to maintain controlled attention to the primary task or to inhibit the direction of attention to emotional connotations of symptom words. Such an interpretation would be in line with Brown's (2004) hypotheses that "modalities affected by unexplained symptoms will be associated with deficits in high-level postattentive processing but not low-level preattentive processing (p. 807)". In the same direction point the results of Lim and Kim (2005) that patient with somatoform disorder show an attentional bias toward physical threat words only under conditions of supraliminal presentation, but not during subliminal word presentation. Additionally, the ex-Gaussian parameters reveal interesting differences between the emotional intrusion and implicit evaluation effect: whereas the emotional intrusion effect (for symptom words) is reflected in elevated σ and τ parameters, the implicit association

effect (for symptom words) is also marked by an increase in the μ parameter that is supposed to reflect more automatic processing. However, we have to acknowledge several problems and limitations with the parameters derived from the ex-Gaussian distribution. Firstly, results of the raw reaction time data could only be replicated in part. Secondly, no substantial correlations between the three parameters and the symptom reports emerged. Therefore we have to conclude that traditional RT measures in our study seem more reliable and robust for individual differences analysis, which might partly be attributable to the comparatively low number of data points that were available for the parameter estimation.

6.6.5. Limitations

A limitation of the current study refers to the fact that we were not able to repeat the diagnostic interview (SCID I) to assess the stability of the clinical diagnoses. However, the stability data of the self-report measures (from t1 to t2) indicate a rather high-stability of symptoms.

As De Houwer (2002) pointed out, measures derived from the IAT and related paradigms like the EAST only quantify the strength of associations between concepts. As psychopathological relevant "beliefs" are marked by qualified, directional, and often very complex associative structures (De Houwer, 2002), results on the strength of the association of single concepts as presented above can only elucidate small pieces of memory structures involved in more complex pathological networks (e.g., Foa & Kozak, 1986). Additionally, we have to acknowledge that the comparatively low number of trials in each experimental condition might have limited the robustness of the three parameter estimations for the ex-Gaussian distribution. Finally, although our study yielded altered *cognitive* processes in SFD and IEI these phenomena are by no means sufficient to fully explain the complex symptomatology.

Although results regarding an involvement of endocrinological and immunological processes in somatoform disorders are currently mixed, it seems reasonable to consider somatization as a complex psychophysiological phenomenon (Rief & Barsky, 2005).

6.6.6. Conclusion

In conclusion, the results of our second study replicate and support prior findings about the involvement of attentional and implicit evaluative processes in IEI. Conceptually, in line with psychological theories of medically unexplained symptoms, evidence for a symptom focused attentional style was found in both IEI and SFD. Methodologically, we have determined the EAST as a valid measure not only for implicit associations but also for the assessment of robust emotional intrusion effects analogously to indicators derived from the emotional Stroop task. We also consider the decomposition of individual response time distributions with the ex-Gaussian distribution as fruitful to improve our understanding of emotion modulated attentional processes in psychological disorders.

After dealing with cognitive aspects of IEI and typical somatoform disorders, we want to emphasize that the nature of symptom etiology or chronification implied in this work is not meant to disregard the impairment and suffering of participants in the two clinical groups (as documented in the self-report and clinical interview data). Nor can we finally exclude the possibility of severe organic etiological conditions in single cases. However, recent data confirm the notions that both medically explained and unexplained symptoms, irrespective of their (supposed) etiology, are accompanied by severe physical and psychosocial disability (Kisely & Simon, 2006). In line with Pennebaker and Brown, we finally consider medically unexplained complaints as subjectively real and individually distressing.

6.6.7. Future directions

It is tempting to speculate that in line with Pennebaker's competition of cues model the involvement in externalizing attributional reasoning or rumination (as reflected in IEI-specific associations and memory bias - which we consider as the main specific component differentiating IEI from traditional somatoform disorders), represents an adaptive and complex cognitive coping method (rather than a simple phobic reaction). Its implicit function is, to distract attention from threatening internal sensations, thereby reducing negative emotional states resulting from a symptom focused perceptual style. Paradoxically, environmental fear motivated avoidance of external stimulation (a treatment recommended by clinical ecologists) in turn preserves catastrophic expectations and results in increased attentional resources for the perception of physical sensations that might be characterized as a nocebo phenomenon and simultaneously serve as convincing emotional evidence for

idiosyncratic illness schemata (i.e., suffering from IEI). Future research might focus on these three aspects which are "short-term beneficial" consequences of IEI-specific cognitions in terms of emotion regulation, vulnerability of IEI-patients to nocebo reactions (e.g., regarding antidepressive medication), and a tendency towards emotional reasoning in IEI as promising mechanisms for better understanding cognitive abnormalities in IEI.

7 INTEGRATION OF FINDINGS AND GENERAL DISCUSSION

The two studies presented above aim at investigating cognitive abnormalities with regard to attention and memory processes involved in the pathogenesis of IEI. The experimental design, i.e., a longitudinal study with two control groups (people with a somatoform disorder and non-somatoform controls), was chosen to prove specificity and temporal stability of results and to gain further evidence for the question if IEI should be considered and treated as a modern variant of somatoform disorders. In the following paragraphs we will sum up and discuss the major findings and outline implications for therapy of IEI. Finally, we will suggest promising topics for future research.

7.1. Summary of findings

In the first study we used different experimental paradigms to assess attentional biases (emotional Stroop and dot-probe task) toward IEI-trigger words and unspecific bodily symptom words, as well as explicit memory biases toward these stimuli. Results only partly confirmed our hypotheses: In line with our expectations, people with somatoform disorders (SFD) and people with IEI showed an elevated attentional bias toward symptom words in the emotional Stroop but not the dot-probe task compared to people without SFD and IEI. Most surprisingly and in contrast to our expectations, no such attentional bias could be observed for the IEI-trigger words in people with IEI. Since the dot-probe task was designed as an alternative measure of selective attention, it was also unexpected that the attentional bias toward symptom words in the emotional Stroop task could not be replicated in the dot-probe task. We attribute this finding primarily to two reasons - firstly, a lack of reliability of the dot-probe task and secondly, conceptual differences between the processes measured by the emotional Stroop (emotional intrusion effects) and the dot-probe task (attention shift effects). In the case of the recognition task as a measure of explicit memory bias, we found evidence for a better recognition of IEI-trigger words in people with IEI compared to the other two groups, whereas no differences with regard to symptom word recognition could be observed among the three groups. Thus, our first study revealed similarities as well as differences regarding biased information processing in people with IEI and SFD. However, since we obtained discrepant results between the two measures of selective attention (DPT and EST), the question remains as to how reliable and valid the attentional bias toward symptom words would be. In our second study one year later, we therefore aimed at replicating the emotional

intrusion effect in the IEI and SFD group. As we were also interested in another component of our hypothesized model of IEI, namely *implicit* or *schematic* representations of the harmfulness of IEI-triggers in memory, we chose the extrinsic affective Simon task (EAST) as an innovative experimental paradigm that allowed for the assessment of attentional bias and implicit association effects simultaneously. Although the EAST is more complex and demanding, it shares many similarities with the emotional Stroop task used in our first study (e.g., words are presented in different colors and the task is to respond to the color and ignore the word meaning). Interestingly, results of the EAST concerning the attentional bias effect closely replicated the findings of the emotional Stroop task in study 1 - people with SFD and IEI, but not the CG revealed robust selective attention effects toward bodily symptom words but not IEI-triggers words. This attentional bias toward symptom words was even significantly stronger among people with IEI compared to members of the SFD group. We take the findings of this replication study as strong evidence for the hypothesis that people with IEI like patients with SFD in general reveal cognitive abnormalities in attentional processes that mirror habitual body and symptom focused attentional styles. From a methodological perspective it seems notable, that attentional biases, in the sense of emotional intrusion effects, do not seem restricted to verbal/oral responses (study 1) but also manifest in a manual response mode (key press reactions used in study 2). Apart from replicating our major findings of the first study, we additionally observed an interesting pattern of results regarding implicit association effects in the second study: In line with our hypothesis, only the IEI group showed significant implicit negative associations with IEI-trigger words. We interpret this finding as evidence for implicit disorder specific cognitive schemata. Our expectations concerning elevated negative association effects of bodily symptom words in the IEI and SFD groups were only partially supported: Only the IEI group revealed stronger negative associations compared to the CG. No such effect was found for the SFD group. One reason for this result might be that many participants in the SFD groups fulfilled diagnostic criteria of a rather mono-symptomatic variant of somatoform disorders, e.g., gastrointestinal problems considered as irritable bowel syndrome (IBS). As the stimulus words addressing bodily symptom are much more heterogeneous they might be more relevant to people with polysymptomatic variants of somatoform disorders.

Additionally, there are similarities as well as differences between participants with a traditional somatoform disorder (SFD) according to DSM-IV and participants fulfilling our case criteria for IEI. Focusing on attentional processes towards symptom words and self-report data, both clinical groups revealed very similar experimental biases, similar degrees of

psychopathological symptoms (e.g., SCL-90R), and parallel psychological risk factors such as negative affectivity or dysfunctional attitudes toward body and health. In this way, on a group level, the similarities regarding symptom patterns and attentional bias scores between SFD and IEI participants outweigh the differences. In contrast, an explicit memory bias toward IEI triggers, elevated levels of habitual imaginative involvement on the absorption scale, and implicit negative evaluations of IEI-trigger words turned out as specific for IEI. Based on these findings we encourage the consideration of IEI as a variant of somatoform disorders marked by the co-occurrence of two important features: multiple somatoform symptoms *and* specific externalizing environmental symptom attributions, often triggered by low-level olfactory stimuli. Since it is the second part, namely the external symptom attribution style that fosters the chronification of the disorder (either via radical avoidance of many daily activities or via counterproductive medical interventions proposed by representatives of clinical ecology and environmental medicine), perhaps the most important question requiring further research is: Why do some people with somatoform symptoms develop IEI, whereas others do not? Although the results of studies 1 and 2 provide no comprehensive answer to this question we will refer to this question later on and derive some hypotheses that deserve further experimental investigation.

7.2. Re-examining the cognitive-behavioral model of IEI

Since a comprehensive test of our cognitive-behavioral model of IEI presented in chapter 3 was beyond the scope the current work, we mainly focused on cognitive factors hypothesized in the maintenance of IEI, such as *attentional biases* (toward bodily symptoms and IEI-triggers) and specific memory processes (explicit memory biases and implicit association effects) indicating the existence of IEI-specific danger schemata. Whereas people with IEI produced a stronger emotional intrusion effect in the light of somatic symptom words compared to the CG (study 1 and 2), they did not react to IEI-trigger words in the hypothesized manner (i.e., with a slowing of responses). This latter finding casts doubt on parts of the previously proposed hypothetical cognitive-behavioral model of IEI (Figure 3-1). According to this model, participants with IEI should not only direct their attention selectively toward unspecific bodily complaints but also toward suspected IEI-trigger substances in the environment. However, such an early, fast-acting attentional bias toward IEI-trigger words was not detectable, neither in study 1 nor in study 2. Since those findings leave us with a variety of possible alternative explanations, we can only speculate about the

causes. It might be an artifact of the methodology used in our study. Some authors suggest, that emotional Stroop tasks with supraliminally presented stimuli represent "impure" measures of selective attention because those tasks allow for strategic defense reactions that blur emotional intrusion effects (e.g., Putman et al., 2004). Therefore, the use of subliminal task versions in further studies that eliminate the use of conscious performance strategies would clarify this issue. Additionally, with the help of functional magnetic resonance imaging, Van den Heuvel and colleagues (Van den Heuvel, Veltman, Groenewegen, Witter, Merkelbach, Cath, van Balkom, van Oppen, & van Dyk, 2005) could demonstrate in patients with obsessive-compulsive disorder that even in the absence of effects in the behavioral data (i.e., emotional intrusion effects based on response time differences), correlates of selective attention processes can be found in neural activation patterns. Given these results, it might be premature to discard the existence of an attentional bias toward IEI-trigger words in patients with IEI. However, the data of studies 1 and 2 suggest that people with IEI compared to the other two experimental groups are not impaired in their ability to effectively disengage their attention from word stimuli representing common IEI-triggers. In contrast, this ability (disengagement of attention from critical stimuli in order to efficiently perform on the primary task) seems to be impaired in the light of bodily symptom words. Therefore, the paradox remains, that although IEI-trigger words are rated as very unpleasant and highly arousing by the IEI participants (cf. results of the SAM-ratings) and although these words produce negative implicit association effects in the EAST, their threat value does not seem sufficient to elicit a spontaneous interruption to current processing in the emotional Stroop task. In line with the results of the explicit memory task in study 1 (better recognition of IEI-trigger words in patients with IEI) we suggest that IEI-triggers and bodily symptoms are processed in different ways and that information concerning IEI-triggers might be motivationally ambivalent as they represent both danger and reduce uncertainty at the same time: For people with a traditional somatoform disorder as well as people suffering from IEI, bodily symptoms without known origin represent the primary matter of concern (thus automatically capturing attentional resources and producing emotional intrusion effects). The attribution of bodily perceptions as signs of IEI represents a secondary interpretive or evaluative process that takes place later in information processing. In this sense, IEI-related information is ambivalent in that it signals danger ("poison") but at the same time provides an idiosyncratic acceptable explanation that externalizes symptoms, and thereby reduces uncertainty and responsibility.

I would therefore postulate that beliefs about the harmfulness of suspected IEI-triggers serve as an idiosyncratic causal explanation for the otherwise unexplainable somatic symptoms. From a learning perspective, this kind of IEI-specific reasoning is negatively reinforced by the reduction of uncertainty about the causes of bodily reactions, the distraction of attention from the body and negative internal emotional states to the environment, and once IEI-beliefs are established, positive reinforcement results from the confirmation of existing beliefs (i.e., to suffer from IEI) and the (external) attribution of responsibility to the environment. On a more global level, these two mechanisms of negative and positive reinforcement serve as affect or mood regulation strategies. Focusing on short-term consequences, the strategy of externalizing the cause of bodily complaints might be beneficial. In the long run, the repeated interpretation of bodily reactions as signs of IEI represents a catastrophizing appraisal process that directs high-level attention (according to the model presented by Brown, 2004) to the symptom itself and thereby lowers the symptom perception threshold and / or increases and prolongs the conscious representation of bodily symptoms. Accordingly, IEI specific beliefs do not provoke a specific attentional bias toward IEI-triggers itself but rather enhance the emotional intrusion effect for symptom words.

7.3. Symptom attributions in IEI – a delusion-like phenomenon?

The interested reader might have noticed that the formation and maintenance of IEI-specific beliefs that we propose in the cognitive-behavioral model above is already informed by a certain hypothesis – namely that IEI-specific beliefs resemble delusional phenomena like seen in schizotypy (milder forms) and in schizophrenia (more severe forms). As already mentioned in the theoretical section above, some authors (e.g., Staudenmayer et al., 2003b) have hypothesized and reasoned from their clinical experience with IEI patients that their attributions seem to be comparable to overvalued ideas and sometimes appear similar to milder forms of delusional phenomena in psychotic disorders. Our findings with regard to the high absorption values in the TABS for the IEI group (study 1) point in the same direction. Evidence for a common genetic ground for high absorption values and positive (psychotic) symptoms have recently been proposed (Ott, Reuter, Henning, & Vaitl, 2005). It therefore appears possible that the adoption and maintenance of IEI specific attributions is based on a neurobiological diathesis. Since the construct of absorption is also related to schizotypy, considered as a risk factor for schizophrenia, it would be interesting to prove whether people with IEI suffer from elevated levels of schizotypy. In this context, it would be especially

interesting to investigate the existence of general reasoning biases, such as *cognitive bias against disconfirmatory evidence* that seems not only related to chronic schizophrenia (e.g., Woodward, Moritz, & Chen, 2006) but also to schizotypy (Buchy, 2006). This bias also plays a key role in the cognitive model of persecutory delusions as proposed by Freeman and colleagues (2002). The existence of this mechanism in IEI would explain why some patients stick to their idiosyncratic explanations in spite of disconfirmatory information. The model by Freeman et al. (2002) postulates that the generation of delusions is the consequence of a vulnerability-stress interaction: Based on biological (e.g., genetic) and psychological (chronic levels of anxiety and negative affectivity) vulnerability factors and critical life events provoke elevated levels of arousal that lead to inner-outer confusions and anomalous experiences (e.g., "experience of thoughts as voices, actions experienced as unintended," [Freeman et al., 2002; p. 334]). Once those anomalous experiences have been noticed, the individual searches for meaning based on existing knowledge and beliefs. Certain cognitive biases (e.g., "jumping to conclusions", "dysfunctions in theory of mind", Freeman, et al. 2002; "externalizing bias in the sense of non-self attributions", Bell, Halligan, & Ellis, 2006) now lead to erroneous results of the search process and the maintenance of (persecutory) delusions (see Figure 7-1). A potential benefit of delusion-like cognitive processes regarding self-esteem and affect regulation is outlined in the following paragraph taken from Freeman et al. (2002; p. 335):

> "The explanation chosen will be mediated by at least three other factors. The first mediator is beliefs about mental illness and 'madness' (Birchwood, 1995). Simply put, many patients have had to make a choice between something being wrong with them and something being wrong in the world. Believing that something is wrong with them (for instance, that they are becoming mad) may be a more distressing belief then that they are being persecuted, and hence a persecutory belief is more likely to be chosen in such circumstances. In this respect, there is an external attribution that limits the distress caused to individuals in terms of cost to self-esteem; this could be viewed as a defensive attribution."

I propose, that similar defensive mechanisms, marked by externalizing attributions for bodily symptoms, might explain why environmental attributions (or external attributions in general) are preferred over internal (i.e., psychological) attributions in people suffering from IEI.

Another highly interesting construct, probably relevant for our understanding of IEI, refers to *latent inhibition*. This process that is defined as "a decrement in learning performance which results from the non-reinforced preexposure of the to-be-conditioned

stimulus" (Lubow, 1993; p. 398), is hypothesized as an indicator of basal attentional filter processes that have been demonstrated to be impaired in schizotypic individuals (Bell et al., 2006). If one assumes connections between IEI and schizotypy or psychosis-proneness in general, diminished latent inhibition effects (as vulnerability factors) might explain why, especially, people with IEI tend to discover and experience diverse associations between bodily symptoms and various aspects of their environment (e.g., odors, sounds, places). Diminished latent inhibition as an explanatory construct for chemical intolerance has previously been proposed by Otto and Giardino (2001): The authors suggest that diminished latent inhibition might serve as a risk factor for developing conditioned aversive reactions to odors. Furthermore, they point to interesting research that suggests a connection between reduced levels of latent inhibition in individuals that are marked by high levels of psychosis-proneness and the personality construct of openness to experiences (Peterson & Carson, 2000; Peterson, Smith, & Carson, 2002). However, complicating the issue further, low levels of latent inhibition might be beneficial in terms of creative thinking when associated with high levels of intelligence and working memory capacity (WMC), whereas low latent inhibition scores in connection with reduced intelligence and WMC have negative effects on the cognitive system (Carson, Peterson, & Higgins, 2003).

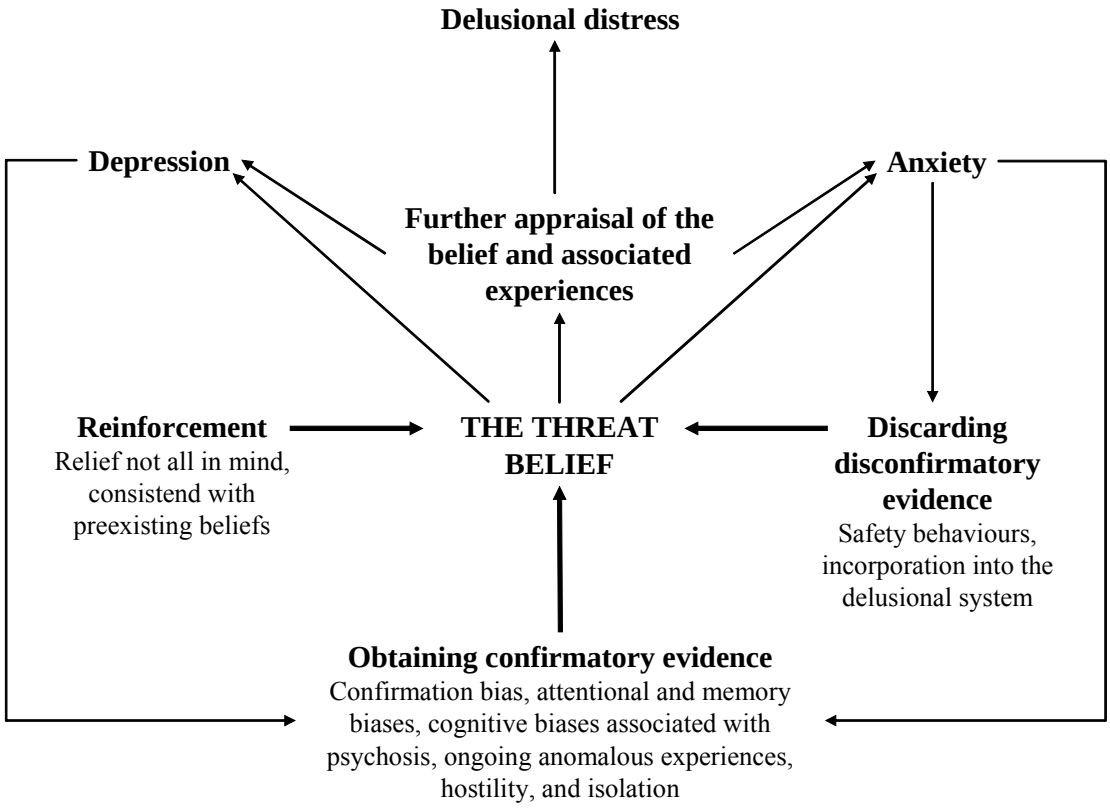

Figure 7-1: Model of maintenance of persecutory delusions (from Freeman et al., 2002; p. 338, Figure 2).

7.4. Implications for Therapy of IEI

Little is yet known about successful intervention strategies that produce long-lasting and profound positive effect in patients with IEI. Available treatment suggestions are mostly derived from case reports. Interesting suggestions for therapy of IEI have been proposed by Guglielmi, Cox, and Spyker (1993). Although we would not generally share the opinion of the authors that MCS/IEI primarily represent an anxiety disorder for which Mowrer's two factor theory can be successfully applied, Guglielmi and colleagues describe in their case report the effective use of a comprehensive desensitization program, including biofeedback-assisted relaxation training, in vivo exposure to chemicals (e.g., cigarette smoke), and cognitive restructuring procedures. The authors convincingly argue that exposure with response prevention (i.e., prevention of avoidance) should be effective to decrease sensitivity to certain *olfactory* stimuli, since the olfactory system seems especially prone to processes of adaptation (i.e., decrease of sensitivity) and cross adaptation.

Based on the findings of studies 1 and 2 that IEI resembles traditional somatoform disorders with regard to experimental indicators of symptom focused attention and self-

reports measures of medically unexplained symptoms and current psychopathology, we suggest treatment elements that have been successfully proposed and applied to the realm of somatoform disorders and hypochondriasis (e.g., Rief & Hiller, 1998). The hallmarks of those programs are: the formulation of a psychobiological model of bodily symptoms (in contrast to a purely organic model), a reduction of avoidance and safety-seeking strategies (e.g., frequent doctor visits) as they are one of the main factors in the maintenance of anxiety and hypochondriacal worries, a decrease in body and symptom focused attention (e.g., via attention trainings), and the use of stress reducing and relaxing strategies (e.g., progressive muscle relaxation). As medically unexplained symptoms (MUS) foster avoidance behavior and physical inactivity (which in turn amplifies MUS and increases depressive mood), one of the main objectives in therapy is to increase healthy physical activity. Cognitive techniques such as Socratic dialogue are proposed to restructure irrational and catastrophizing beliefs prominent in hypochondriasis (e.g., headache is mostly a sign of a brain tumor). As according to the model presented in chapter 3 strong dysfunctional and irrational beliefs are genuine parts of IEI (e.g., "Bodily symptoms indicate that I have been poisoned."), specially tailored cognitive interventions that have been proved effective in treating delusional phenomena in schizophrenia (e.g., Lincoln, 2006) might be helpful in the work with IEI patients. Apart form these cognitive techniques, we consider the reduction of safety-seeking and avoidance behavior as most promising for the therapeutic endeavor to modify IEI-specific dysfunctional beliefs regarding the harmfulness of everyday chemicals.

7.5. Promising Future Directions in the Study of IEI

Psychosis-proneness and latent inhibition as explanatory constructs. As already mentioned above, following a continuum approach of delusions (Bell et al., 2006) we consider the conceptualization of IEI-specific attributions as *overvalued ideas* and *delusion-like processes* (e.g., the belief that bodily symptoms are caused by low-dose chemicals) as fruitful for the deduction of further hypotheses. Given our results of elevated levels of absorption in participants with IEI, we expect that people with IEI reveal elevated levels of schizotypy and decreased levels of *latent inhibition*. Both hypotheses can easily be tested by using self-report measures in the case of schizotypy and cognitive-experimental paradigms in the case of latent inhibition. As a consequence, we would expect in differential aversive conditioning paradigms that people with, or at risk for the development of, IEI should show slower extinctions and faster reinstatement effects as evidence of a less efficient formation of

new associations that have the potential to inhibit the learned fear response. Although this effect might be most pronounced with olfactory stimuli it should generalize to other stimulus modalities (e.g., visual, linguistic, or auditory stimuli).

Functional magnetic resonance introspection (fMRI) and subliminal presentation mode – a final chance for the attentional bias hypothesis of IEI-triggers. The results of studies 1 and 2 propose that counter-intuitively, verbal representations of IEI-triggers do not automatically capture the attention of patients with IEI as symptom words do. However, in order to abandon the notion that IEI-triggers elicit a fear like attentional response we would suggest to re-investigate this issue by introducing additional experimental conditions, for instance pictorial stimuli, subliminal exposure conditions, and individually selected triggers of symptoms. To test for the assumption that attentional biases, though not observable in the behavioral data, might be detectable in altered neural activation patterns we would suggest to combine further attentional bias studies with fMRI technology.

Do people with IEI (and SFD) show general reasoning biases that might be explained by temporal or stable decreases in working memory capacity? The two studies aimed at investigating specific cognitive biases toward illness relevant information. To date, little is known about general cognitive biases in people with IEI (and SFD). Studies of participants with sub-clinical variants of IEI could elucidate if general cognitive biases might partly explain the vulnerability toward dysfunctional beliefs or attributions. A similar study with phobic participants was conducted by de Jong, Weertman, Horselenberg, and van den Hout (1997). The authors demonstrated a general confirming reasoning bias (i.e., a tendency to confirm rather than to falsify prior beliefs) in spider-fearful individuals.

Linking cognitive biases to physiological impairments or – how does psychological/emotional stress get under the skin? As outlined in the previous paragraph, we take the empirical results of studies 1 and 2 as evidence for the significance of cognitive and emotional processes relevant for the etiology and maintenance of somatoform disorders in general and IEI in specific. However, limitations of the two studies to cognitive measures leave the question of associations between cognitive and physiological processes. Brosschot, Gerin, and Thayer (2006) have convincingly argued in their perseverative cognition hypothesis that prolonged *mental representations of stressors* (i.e., worry and rumination) are crucial in causing somatic symptoms (via changes in diverse organic systems like the

cardiovascular, the immune, the endocrine, and the neurovisceral system). Along this line, it is tempting to speculate that our attentional bias effects toward symptom words might partially represent the inability of patients (with IEI and SFD) to effectively deactivate negative emotional contents in working memory. Thus, it would be interesting to investigate if ruminative tendencies associated with anticipated or past stressors are associated with experimental indicators derived from the emotional Stroop task or related paradigms and if performance in these tasks is related to measures of immunology or endocrinology. Such a link between attentional processes and immediate endocrionological responses has recently been proposed by Ellenbogen, Schwartzman, Stewart, and Walker (2006). The authors found interesting correlations between a cognitive measure of (subliminal) attentional disengagement from threat pictures (in a spatial cueing task) and cortisol levels. Similar associations between cognitive and endocrinological processes have been observered by others (e.g., van Honk, Tuiten, van den Hout, Koppeschaar, Thijssen, de Haan, & Verbaten, 2000). We therefore consider the issue of endocrinological and immunological changes associated with cognitive biases as fruitful for further studies in patients with SDF and IEI.

8 SUMMARY

Idiopathic Environmental Intolerance (IEI) refers to a polysymptomatic condition of unknown etiology, poorly understood pathogeneses, and somatoform-like phenomenology. Two studies were designed to assess cognitive biases in people with IEI (n = 54). Specificity of cognitive biases were tested in two control groups, that is, people with a traditional somatoform disorder according to DSM-IV (SFD; n = 44), and people without IEI and SFD (CG; n = 54).

The first study was designed to focus on psychological mechanisms and to detect and compare selective attention, memory bias, and abnormalities in explicit evaluative processes toward threat related words in IEI and SFD. Attentional biases toward somatic symptoms and IEI-trigger words were assessed with the emotional Stroop and the dot-probe paradigm. Memory bias was assessed with a recognition task. Ratings of explicit emotional evaluation were measured with the self-assessment manikin (SAM). The IEI and SFD group showed increased interference in naming the color of symptom words in the emotional Stroop task, whereas no differential interference effect was found for IEI-trigger words. The dot-probe task did not reveal evidence for group specific vigilance or avoidance reactions to critical stimuli. The IEI group recognized IEI-trigger words that they had previously seen slightly better than the other groups. Participants with IEI rated trigger words as more unpleasant and more arousing than the two comparison groups. Indices of attentional bias and explicit emotional evaluation were correlated with somatoform symptoms, dysfunctional beliefs about body and health, and other psychological self-report measures. Results revealed implicit and explicit cognitive abnormalities in IEI similar to SFD that may trigger and maintain processes of somatosensory amplification.

The second study provided data from a 1-year follow-up investigation using an innovative cognitive experimental paradigm - the extrinsic affective Simon task (EAST). In the EAST we dissociated indicators of attentional bias and implicit attitudes toward bodily symptoms and IEI-trigger words. Attentional bias scores mirrored results of the first study, that is, elevated attentional bias toward physical symptom words but not IEI-trigger words in IEI and SFD compared to the CG. As indirect evidence for the existence of dysfunctional specific schemata in IEI, negative implicit attitudes toward IEI-trigger words were found only in IEI-participants. Whereas implicit negative attitudes seem specific for IEI, increased attentional biases toward symptom words in IEI and SFD replicate previous findings and are compatible with the notion of symptom focused attention contributing to somatosensory

amplification and chronicity of medically unexplained symptoms in typical and atypical somatoform disorders.

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10 GLOSSARY OF ACRONYMS

ACQ = Agoraphobic Cognitions Questionnaire
ADM = Affective Decision Mechanism
ANCOVA = Analysis of Covariance
ANOVA = Analysis of Variance
AS = Anxiety Sensitivity
BDD = Body-Dysmorphic Disorder
CABAH = Cognitions About Body And Health Qestionnaire
CBT = Cognitive Behavior Therapy
CFS = Chronic Fatigue Syndrome
CG = Control Group
CI = Chemical Intolerance
COSS = Chemical Odor Sensitivity Scale
CS = Conditioned Stimulus
DPT = Dot Probe Task
DSM-IV = Diagnostic and Statistical Manual of Mental Disorders (edition 4)
EAST = Extrinsic Affective Simon Task
EEG = Electroencephalography
EI = Emotional intrusion effect
ESQ = Environmental Sensitiviy Questionnaire
EST = Emotional Stroop Task
fMRI = Functional Magnetic Resonance Introspection
FMS = Fibromyalgia Syndrome
GWS = Gulf War Syndrome
IA = Implicit association effect
IAT = Implicit Association Test
IBS = Irritable Bowel Syndrome
ICD-10 = International Classification of Diseases (edition 10)
IEI = Idiopathic Environmental Intolerance
MANOVA = Multivariate Analysis of Variance
MCS = Multiple Chemical Sensitivity
MUS = Medically Unexplained Symptoms
PAS = Primary Attentional System

PHQ = Patient Health Questionnaire
PHQ-15 = Patient Health Questionnaire: Somatic symptom severity scale
PHQ-9 = Patient Health Questionnaire: Depression scale
PSD = Physical Symptom Disorder
PTSD = Post-Traumatic Stress Disorder
QCGS = Questionnaire of Chemical and General Environmental Sensitivity
QMLE = Quantile Maximum Likelihood Estimation
RT = Response Time
SAM = Self-Assessment Manikin
SAS = Secondary Attentional System
SBS = Sick-Building Syndrome
SCID I = Structured Clinical Interview for DSM-IV (achsis I)
SCL-90R = Symptom Checklist 90 (revised)
SFD = Somatoform Disorder
SHC = Subjective Health Complaints
SI-IEI = Structured Interview for Idiopathic Environmental Intolerance
SOMS = Screening for Somatoform Symptoms
SSI4,6 = Somatic Syntom Index
STAI = State-Trait Anxiety Inventory
TABS = Tellegen Absorption Scale
TDS = Time-Dependent Sensitization
TES = Threat Evaluation System
TILT = Toxicant-Induced Loss of Tolerance
UCS = Unconditioned Stimulus

11 LIST OF FIGURES

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13 CURRICULUM VITAE

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10/2001 bis 5/2002	Lehrauftrag an der Universität Basel: *Experimentalpsych. Projektseminar (WS 2001/2002 und SoSe 2002)* Abteilung Allgemeine Psychologie und Methodologie (Leitung: Prof. Dr. K. Opwis)

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Erklärung

Hiermit versichere ich, dass ich die vorliegende Dissertation

"Attentional Bias, Memory Bias, and Symptom Attribution in Idiopathic Environmental Intolerance and Classical Somatoform Disorders"

ohne unzulässige Hilfe Dritter und ohne Benutzung anderer als der angegebenen Quellen und Hilfsmittel angefertigt und die benutzten Quellen wörtlich oder inhaltlich entnommenen Stellen als solche kenntlich gemacht habe. Die Arbeit wurde bislang keiner anderen Prüfungsbehörde vorgelegt.

Mannheim, 17.11.2006

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Michael Witthöft