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Somatoform disorders and causal attributions in patients with suspected allergies: Do somatic causal attributions matter?

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List of abbreviations

DSM	Diagnostic and Statistical Manual of Mental Disorders
ICD	International Classification of Diseases
IPQ	Illness Perception Questionnaire
IPQ-R	Illness Perception Questionnaire Revised
FSS	Functional Somatic Syndrome
MUS	medically unexplained symptom
NoSFD	non-somatoform disorder
PHQ	Patient Health Questionnaire
SFD	somatoform disorder
VIT	venom immunotherapy

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Abstract

Objective: In view of the forthcoming Diagnostic and Statistical Manual for Mental Disorders (DSM-V), somatic causal illness attributions are being considered as potential positive criteria for somatoform disorders (SFDs). The aim of this study is to investigate whether patients diagnosed with SFDs tend towards a predominantly somatic attribution style.

Methods: The study compares the causal illness attributions of 48 SFD and 149 non-somatoform disorder patients in a sample of patients presenting for an allergy diagnostic work-up, and those of 47 controls hospitalised for allergen-specific venom immunotherapy. The SFD diagnosis was established by means of the Structured Clinical Interview for DSM-IV. Both spontaneous and prompted causal illness attributions were recorded through interview and by means of the causal dimension of the Revised Illness Perception Questionnaire (IPQ-R), respectively. The IPQ-R was submitted to an Exploratory Factor Analysis to identify groups of causal beliefs. In the absence of a clear factor structure, patients' spontaneous and prompted responses were assigned to a psychosocial, somatic or mixed attribution style. Associated mental disorders were assessed both categorically and dimensionally by means of various modules of the Patient Health Questionnaire (PHQ).

Results: Both in the free response task and in their responses to the IPQ-R, SFD patients were no more likely than their non somatoform counterparts to focus on somatic explanations for their symptoms. They were just as likely to advance psychosocial or mixed causes. However, patients with SFDs were considerably more likely to find fault with medical care in the past. SFD patients were significantly more likely than non-somatoform disorder patients to be diagnosed with a psychiatric disorder, and to score higher on depression and

anxiety. In turn, psychiatric comorbidity was positively associated with a mixed attribution style.

Conclusion: Our data do not support the use of somatic causal illness attributions as positive criteria for SFDs. They confirm the dynamic and multidimensional nature of causal illness attributions. Clinical and scientific implications of our findings are discussed.

1 Introduction

In view of the forthcoming Diagnostic and Statistical Manual for Mental Disorders (DSM-V) and the International Classification of Diseases (ICD-11), there is an ongoing debate about the terminology and classification of somatoform disorders (SFDs) (Dimsdale & Creed, 2009; Hiller & Rief, 2005; Kroenke et al., 2007; Mayou et al., 2005; Noyes et al., 2008). There have been calls to move away from the 'negative definition' of SFDs as 'medically unexplained' towards a positive one, considering among other things, somatic causal illness attributions as potential positive criteria .

Since the early 1990ies, and in the light of subjective illness theories, causal illness attributions have been shown to influence the development, maintenance and management of somatoform and functional somatic syndromes (Butler et al., 2001; Cathebras et al., 1992; Kirmayer & Hayton, 1992; Chalder et al., 1996; Dimsdale & Creed, 2009; Henningsen et al., 2005; Korn, 2003; Kroenke et al., 2007; Martin & Crane, 2003; Powell et al., 1990; Stone et al., 2005; Weiss et al., 1992). While the ICD-10 already lists the adherence to somatic causal attributions as one of the main features of SFD patients, empirical evidence of this assumption is relatively rare.

Attribution theory and research have identified various dimensions of causal attribution. In studies on 'Medically Unexplained Symptoms' (MUS) and SFDs, most analyses have focused on the dichotomy of psychosocial versus somatic causal attributions. In addition, supporting the notion that illness attribution is a multidimensional process, factor analytic approaches based on the Illness Perception Questionnaire (IPQ) and its revised version (IPQ-R) have identified a number of attribution categories: psychological, risk factors, immunity and chance factors . While quantitative measures of illness attribution include lists of predetermined causal explanations from which patients can choose the one(s) closest to

their own beliefs, qualitative studies allow patients to use concepts and categories that are relevant and meaningful to them.

The main purpose of the study presented here is to examine the extent of somatic causal illness attribution among SFD patients in order to assess the possible use of this dimension as a positive criterion in the definition of somatoform disorders, with the long-term view to provide the basis for better diagnostic and therapeutic management. Within a naturalistic research setting, I examine the extent to which patients diagnosed with a SFD tend towards a predominantly somatic attribution style, combining and comparing both qualitative and quantitative research measures.

1.1 Somatoform disorders

1.1.1 The current classification

The concept of somatoform disorders (SFD) was first introduced in 1980 in the third Diagnostic and Statistical Manual for Mental Disorders (DSM-III). It stood for a heterogeneous group of disorders characterised by physical symptoms that suggest physical illness or injury and lead to considerable functional impairment, but cannot be fully explained by a general medical condition, substance abuse or be attributed to another mental disorder. It included somatisation disorder, conversion disorder, hypochondriasis, pain disorder, body dysmorphic disorder and atypical somatoform disorder. DSM-IV then introduced the additional diagnosis of ‘undifferentiated somatoform disorder’ to account for a large number of patients presenting with ‘medically unexplained’ symptoms that did not meet the rather exclusive criteria for somatisation disorder (Kroenke et al., 2007; Mayou et al., 2005; p.277 and p.848, respectively). Nevertheless, conceptual problems persisted. DSM-IV criteria for somatisation disorder were considered to be too narrow and those for undifferentiated

somatoform disorder to be too broad for them to be useful in a clinical and research setting (Creed, 2006; Kroenke et al., 1997). Further, both criterion and predictive validity of somatoform disorder diagnoses were found to be low .

In response to these difficulties, Kroenke et al. (1997) introduced a new mid-way category of 'multisomatoform disorder', decribed as a 'moderately severe form of undifferentiated somatoform disorder' (Jackson & Kroenke, 2008, p. 430). Kroenke's criteria for multisomatoform disorder require the presence of at least three currently 'bothersome medically unexplained somatic symptoms', lasting for more than two years, and present on at least half of the days (Jackson & Kroenke, 2008, p.430). The usefulness of the 'multisomatoform disorder' construct was demonstrated in both cross-sectional and longitudinally designed studies (Kroenke et al., 1997 and Jackson & Kroenke, 2008, respectively).

A further challenge to the diagnosis of SFDs is the considerable degree of overlap between somatoform disorder sub-types (Leiknes et al., 2008) – such as for example, somatisation disorder and hypochondriasis -, and between individual functional somatic syndromes (Kroenke, 2003; Leiknes et al., 2008; Wessely et al., 1999). In psychiatry and psychosomatic medicine, 'medically unexplained' symptoms have been categorised under the heading of somatoform disorders; somatic medicine, on the other hand, refers to them as functional somatic syndromes (FSS) (Henningsen et al., 2007; Mayou et al., 2005). Whether a somatoform or functional disorder is diagnosed therefore mainly depends on the treatment setting . This lack of boundaries between the various diagnostic categories lends support to the suggestion of reducing the number of these categories (Leiknes et al., 2008). Further, it suggests that a dimensional classification of these disorders may be more productive and clinically viable (Jablensky, 2005; Voigt et al., 2010; Wessely et al., 1999).

The negative definition. One major problem is that SFDs as opposed to all other medical conditions are defined above all in terms of the lack of a medical explanation of patients' symptoms (Okasha, 2003, p. 163). That is, somatoform disorders are defined 'in terms of what they are not' (Jablensky, 1999, p.6). According to DSM-IV, the common feature of SFDs is 'the presence of physical symptoms that suggest a medical condition and are not fully explained by a general medical condition, …, or by another mental disorder' (APA, 2000, p. 445). Similarly, the ICD-10 regards the main feature of SFDs to be 'repeated presentation of physical symptoms, …, in spite of repeated negative findings and reassurances by doctors that the symptoms have no physical basis' (WHO, 2007). A diagnosis by exclusion contributes to repeat medical testing and delays treatment, which in turn leads to elevated rates of health care utilisation and costs (Barsky et al., 2005). Unable to offer the patient a positive explanation for his/her symptoms, medicine has thus been said to appear to withhold its help and support from the patient (Creed et al., 2010).

A diagnosis by exclusion limits the criterion validity of the classification system . While the term 'medically unexplained somatic symptom' is the most prominent diagnostic feature of somatoform disorders, it cannot be viewed as a specific diagnostic category in itself (Hiller & Janca, 2003; Hiller & Rief, 2005; Kirmayer et al., 2004). Rather, it draws 'attention to a situation in which the meaning of distress is contested' (Kirmayer et al., 2004, p. 663).

What is medically explicable is in a constant flux. As medical knowledge advances, so does our ability to explain certain symptoms or problems. Conversely, previously accepted explanations may fall into disrepute (Avila, 2006; Kirmayer, 1999). Prior to the discovery of the underlying inflammatory basis of atopic disorders, the latter were believed to be purely psychogenic in origin (Chida et al., 2008). Similarly, increasing knowledge of neuroendocrine pathways in somatoform disorders may change our understanding of this group of disorders (Henningsen, 2003).

Mind-body dualism. Finally, various authors have criticised the *mind-body dualism* inherent in the current definition of somatoform disorders (Chaturvedi & Desai, 2006; Creed et al., 2010, De Gucht & Maes, 2006; Jablensky, 1999; Kirmayer, 1999; Kirmayer et al., 2004; Noyes et al., 2008; Okasha, 2003; Sharpe & Mayou, 2004). That is, in the absence of an organic pathology, the cause of somatic symptoms is assumed to be psychological (Sharpe & Mayou, 2004; see also box below). In many instances, however, it is difficult to decide whether a symptom is medically explicable or not (Hausteiner et al., 2009), or what counts as a medical explanation of a symptom (Creed et al., 2010) . Further, it is questionable whether a symptom or symptoms can be explained solely in terms of an organic disease: somatic symptoms in cancer patients have been shown to have both organic and psychological components (Chaturvedi & Maguire, 1998). Conversely, psychiatric disorders such as depression and anxiety are considered as mental disorders despite notable somatic symptoms (Okasha, 2003) . The above authors therefore suggest adopting a positive, multidimensional approach to future diagnostic criteria, including physiological, psychological, and social factors, to improve clinical validity (see also Voigt et al., 2010).

Somatisation: History in a nutshell

The criteria for somatoform disorders are largely based on the concept of somatisation, 'a mental process whereby mental illness manifests as somatic symptoms' p. 850. The current focus on psychological factors to explain somatic symptoms, that are not medically explicable, has predominated only in the past 100 years, since the ascendance of psychoanalysis in the 20th century. Explanations for medically unexplained symptoms have their roots in the notions of hysteria and conversion (hysteria being the earlier word for the more modern term conversion disorder). For some time, a disturbance of bodily organs, in particular the uterus, was seen as the origin of unexplained symptoms. The latter were often referred to as 'hysterical' (the Greek word hystera meaning womb). Hippocrates described hysteria as being caused by the wandering of the uterus through the body, and thought it to symbolise the longing of the body for a child. Similarly, in the Middle Ages, the Latin term conversion described the 'propensity for the suffocation of the womb to evolve into other diseases' (Jablensky, 1999, p.4). At the end of the 19th century, the term hysteria was generally used to describe physical symptoms (such as for example, a paralysed arm or leg with no neurologic cause) that could not be fully explained by a physical disease. In the 17th century, Thomas Willis, the father of neurology, thought 'medically unexplained' symptoms to originate from a disease of the nervous system. Similarly, in the 19th century, Charcot described hysteria as a neurological disorder, and unexplained somatic symptoms as 'functional lesions'. At the end of the 17th century, with the writings of Thomas Sydenham, psychological factors had briefly begun to be seen as relevant. Sydenham emphasised the importance of the clinician's interest in the welfare of his patient. However, it wasn't until the ascendance of psychoanalysis that psychological factors came to be seen as the origin of physically unexplained symptoms. Freud saw the latter as the expression of repressed instincts and described hysteria as 'the incompatible ideas … rendered innocuous by … being transformed into something somatic' (Jablensky, 1999, p. 5). Hence, the term somatisation has come to refer to a process whereby mental problems can show as somatic symptoms.

Current guidelines , however, recommend to use of the term 'somatisation' descriptively rather than pathogenetically. They advocate a dimensional approach, which allows for the consideration of symptom severity and the number of symptoms experienced.

(De Gucht & Fischler, 2002; De Gucht & Maes, 2006; Jablensky, 1999; Noeker, 2002; Sharpe & Carson, 2001)

1.1.2 Estimation of prevalence

Prevalence rates of patients presenting with somatic symptoms that cannot be medically explained and lead to functional impairment range from about 15 to 30% in population-based and primary care studies (De Waal et al., 2004; Hiller et al., 2006; Janca et al., 2006; Kirmayer et al., 2004; Kroenke, 2003) up to about 50% in specialised care clinics (Nimnuan et al., 2001; Reid et al., 2001a). While these figures clearly establish the clinical

importance of medically unexplained symptoms (and thus SFDs), a number of issues needs to be raised.

Classification and conceptual problems (such as for example, regarding the construct validity of categories like ‘somatisation disorder’ or the difficulty to declare a symptom as ‘medically unexplained’) affect the estimation of prevalence of SFDs: The majority of patients presenting with medically unexplained symptoms fall into the (catch basin) categories of undifferentiated somatoform disorders or somatoform disorders not otherwise specified (De Waal et al., 2004; Janca et al., 2006). Prevalence rates for diagnoses such as somatisation disorder and hypochondriasis are very low. In a review of population-based and primary care studies published since 1966, the median prevalence of somatisation disorder and hypochondriasis was found to be 0.4% and 4.2%, respectively (Creed & Barsky, 2004). In general, prevalence rates of SFDs and FSS have been found to vary depending on the diagnostic criteria (Fink et al., 2004; Henningsen et al., 2007), on the assessment instrument used, as well as on the study design (Jacobi et al., 2004).

Numerous epidemiological studies have reported SFDs to be more prevalent among female and younger patients (Barsky et al., 2001, Fink et al., 2004; Jacobi et al., 2004; Kirmayer & Robbins, 1991; Nimnuan et al., 2001), among those who were not married, and those of lower social class (Fink et al., 2004; Jacobi et al., 2004). Reasons put forward to explain sex differences include: a greater willingness of women to admit health problems and to seek medical help, a higher incidence of depressive and anxiety disorders among women which in turn are associated with somatic symptoms, a higher incidence of predisposing factors such as physical and sexual abuse in women, biological differences in responses to pain, a greater bodily awareness of women as compared to men, and gender bias in research and clinical practice (Barsky et al., 2001).

1.1.3 Psychiatric comorbidity or overlapping syndromes: somatisation, depression and anxiety

Comorbidity (literally 'additional morbidity') refers to the presence of more than one disorder in the same individual at the same time. In successive editions of the Diagnostic and Statistical Manual for Mental Disorders and the International Classification of Diseases the trend has been to increase comorbidity, in particular, in the absence of knowledge about pathophysiology. However, comorbidity does not necessarily imply the presence of multiple diseases. Rather, it is a by-product of the current classification systems (Cooper, 2004; First, 2005; Jablensky, 2004, 2005; Pincus et al., 2004), and 'reflects our current inability to apply … a single diagnosis to account for all symptoms' presented by a patient (First, 2005, p. 206).

Somatoform disorders have been found to be strongly associated with various other mental disorders (Cebulla, 2002; Fink et al., 2004; Garcia-Campayo et al., 2007; Noeker, 2002), in particular, with depression and anxiety disorders. At least one third of patients with SFDs (up to about 70%, depending on the study under consideration) are said to be suffering from concurrent depression and/or anxiety (Cebulla, 2002, De Waal et al., 2004; Fink et al., 2004; Hanel et al., 2009; Henningsen et al., 2003; Kroenke, 2003; Löwe et al., 2008b). The degree of association has been found to be particularly high for somatisation disorder, with there being a dose-effect relationship between the number of somatic symptoms (i.e. somatisation) and the number of depression and/or anxiety symptoms (Cree & Barsky, 2004; Henningsen et al., 2003). Further, depression has been found to be a strong predictor of medically unexplained pain symptoms (Leiknes et al., 2007) and SFDs (Leiknes et al., 2008).

Feeding into the discussion about the classification of SFDs, the considerable overlap of SFD, depression and anxiety – partly due to shared diagnostic criteria (Löwe et al., 2008b) - precludes a view of these disorders as discrete nosological entities. The above authors thus

favour a dimensional rather than a categorical description of somatoform disorders, with the former providing a better fit with clinical reality.

1.1.4 Proposals for change

As part of the ongoing debate about the terminology and classification of somatoform disorders (Dimsdale & Creed, 2009; Hiller & Rief, 2005; Kroenke et al., 2007; Mayou et al., 2005; Noyes et al., 2008), there are definite calls to move away from the negative definition of SFDs. The consensus is that positive psychological and behavioural criteria are called for in the definition of SFDs (Kroenke et al., 2007; Löwe et al., 2008a; Voigt et al., 2010). One of the proposed dimensions is that of causal illness attribution (De Gucht & Maes, 2006; Dimsdale & Creed, 2009; Duddu et al., 2006; Henningsen et al., 2002; Kroenke et al., 2007; Löwe et al., 2008a; Rief & Isaac, 2007; Stone et al., 2005b; Wessely et al., 1999). In fact, the ICD-10 lists the adherence to somatic causal attributions as one of the main features of SFD patients (WHO, 2007).

As part of the development work on DSM-V, the Somatic Symptom Disorders Work Group has put forward a set of preliminary recommendations for a new classification of the SFD diagnoses as they are listed in DSM-IV under the chapter of Somatoform Disorders. This work is to be completed by May 2013. Efforts have been made to propose a concept that can be widely accepted, over and above the field of psychosocial medicine, to avoid the mind-body dualism inherent in the notion of 'medically unexplained symptoms' and to circumvent the unreliability of assessing MUS, as well as to establish a diagnostic category with solid construct validity. The major proposed changes include:

- renaming somatoform disorders as 'somatic symptom disorders',

- merging various overlapping disease categories: somatisation disorder, hypochondriasis, undifferentiated somatoform disorder and pain disorder are combined under the heading of ‘complex somatic symptom disorder’, and
- banning ‘medically unexplained symptoms’ as a core defining feature of somatoform disorders.

Instead, psycho-behavioural characteristics, such as for example the ‘belief in the medical seriousness of one’s symptoms despite evidence to the contrary’, are being emphasised. At present, adherence to a particular type of causal attribution is not being called for as one of the defining features of the new somatoform disorder category (APA, 2011).

DSM-IV criteria and proposals for DSM-V criteria are outlined in Table 1 below.

With the classification and definition of SFDs in the process of being thoroughly revised, a clear statement concerning the appropriate terminology is pending. In the study presented here, the term ‘somatoform disorder’ is used to refer to those diagnostic categories characterised by persistent physical symptoms, namely, the current DSM-IV categories of ‘somatisation disorder’, ‘undifferentiated somatoform disorder’ and ‘pain disorder’, as well as Kroenke’s category of ‘multisomatoform disorder’. The rationale for dropping hypochondriasis, conversion disorder and body dysmorphic disorder from the study’s definition of a somatoform disorder will be outlined in the method section below.

Throughout the dissertation, and despite the various criticisms levelled at the term ’medically unexplained’ disorder or symptom(s), I will use these terms whenever they were used in the original studies reported on.

Table 1. DSM-IV and preliminary DSM-V diagnostic criteria for somatoform disorders

DSM-IV		DSM-V	
Somatoform Disorders	**Diagnostic criteria**	**Somatic Symptom Disorders**	**Diagnostic criteria**
Somatisation disorder	At least 4 pain symptoms, plus 2 gastrointestinal symptoms, plus one sexual symptom, plus one pseudoneurological symptom *Beginning*: before the age of 30 *Duration*: several years	Complex Somatic Symptom Disorder	A. **Somatic symptoms**: One or more somatic symptoms that are distressing and/or result in significant disruption in daily life. B. **Overwhelming concern or preoccupation with symptoms and illness**: At least three of the following: *(1) High level of health-related anxiety.* *(2) A tendency to fear the worst about one's health or bodily symptoms (catastrophising).* *(3) Belief in the medical seriousness of one's symptoms despite evidence to the contrary.* *(4) Health concerns and/or symptoms assume a central role in one's life (ruminative preoccupation).* C. **Chronicity:** Although any one symptom may not be continuously present, the state of being symptomatic is chronic. *Duration:* at least 6 months
Undifferentiated somatoform disorder	One or more physical complaints *Duration:* at least 6 months		
Pain disorder	Pain in one or more anatomical sites Specification: acute (duration of less than 6 months), chronic (duration of 6 months or longer); associated with psychological factors only, or associated with both psychological factors and a general medical condition		
Hypochondriasis	Preoccupation with fears of having, or the idea that one has, a serious disease *Duration*: at least 6 months		
Somatoform Disorder Not Otherwise Specified	Category which includes disorders with somatoform symptoms that do not meet the criteria for any specific somatoform disorder *Duration*: less than 6 months	Simple Somatic Symptom Disorder	One or more somatic symptoms that are distressing and/or result in significant disruption in daily life; at least one B-type criterion *Duration:* at least 1 month
Conversion disorder	One or more symptoms or deficits affecting voluntary motor or sensory function; psychological factors are judged to be associated with the symptom; the symptom or deficit is not intentionally produced or feigned; *Specification*: motor deficit, sensory deficit, seizures, mixed presentation	Functional neurological disorder	The requirements that the clinician has to establish associated psychological stressors, and that the patient is not feigning are to be removed.
Body Dysmorphic Disorder	Excessive preoccupation with an imagined or slight physical defect in appearance		Criteria remain to be determined; moving the disorder to the anxiety disorder group is being considered;

1.2 Causal attributions and somatoform disorders

The occurrence of physical symptoms is an everyday phenomenon, including for healthy individuals. Deciding on what to do about such a symptom – whether to ignore it, worry about it, take some medication, or go and see a doctor – is seen to depend to a large extent on what one believes to be the cause of this symptom (Robbins & Kirmayer, 1991). Current concepts of SFDs emphasize the role of unhelpful causal attributions in the development and maintenance of these disorders.

Causal attributions have been defined as post hoc interpretations or redefinitions of what caused a particular illness and/or the accompanying symptoms (Sensky, 1997). Since the early 1990s, and within the framework of subjective illness theories, they have been shown to influence the development, maintenance and management of somatoform and functional somatic syndromes. In the light of current efforts to develop positive criteria for SFDs, somatic causal attributions have been considered a strong candidate. Understanding the patterns of SFD patients' beliefs about their symptoms has become an important part of investigations into SFDs.

In the following paragraphs, I will trace the origins of causal attributions in social and early clinical psychology and briefly outline their importance in so-called subjective illness theories. I will then review the research instruments used to assess causal attributions, in particular, in relation to SFD patients. The research evidence with regard to an association of somatic causal attribution and SFDs will be examined, including studies on reattribution therapy.

1.2.1 Attribution theory and dimensions of causal attribution

Attribution theory is one of the most important theories in modern psychology. It was developed in the 1960s and 70s by the two influential social psychologists Heider and Kelley, and the cognitive psychologist Bernard Weiner. It is concerned with how individuals interpret events and with the behavioural and emotional consequences of these interpretations. According to Heider (1958), a person can make two types of attribution:

- Internal attribution refers to the inference that a person is behaving in a certain way because of something about the person, such as attitude, character or personality (also called dispositional attribution).
- External attribution refers to the inference that a person's behaviour has something to do with the situation he or she is in (also called situational attribution).

Subsequent developments of Heider's theory introduced further dimensions of attribution (such as, stable vs. unstable, global vs. specific, proximal vs. distant; simple vs. complex, controllable vs. noncontrollable[1]) (Cebulla, 2002; Korn, 2003; Roesch & Weiner, 2001). Very early on, attributions were incorporated into theories trying to explain the development of mental disorders. For example, the attributional revision of the learned helplessness theory developed by Seligman suggests that people become depressed when they attribute negative life events to stable and global causes. Whether self-esteem collapses too is seen to depend on whether they blame themselves for the negative outcome (internal attribution). Further, the depressive-prone individual is thought to show a 'depressive attribution style', that is, a tendency to attribute bad outcomes to personal, global and stable faults of character. Current versions of the theory have come to view attribution

[1] Weiner's attribution theory is mainly about achievement, and he classified attributions along three causal dimensions: locus of control (internal vs. external), stability (do causes change over time or not?), and controllability (causes one can control such as skills vs. causes one cannot control such as luck).

style as one diathesis (among many) in the development of some forms of depression (Cebulla, 2002; Korn, 2003).

Building on the distinction between internal and external attribution, Kelley examined how people decide when to attribute an event to environmental (i.e. external) factors and when to attribute it to internal/dispositional factors such as personal characteristics. According to the so-called discounting principle, Kelley (cited in Robbins & Kirmayer, p. 1030) postulated that an event is attributed to personal characteristics only when it occurs independently of situational factors. Applied to physical illness (Robbins & Kirmayer, 1991), a person will thus first look for some external explanation for their symptom(s), such as temporary fatigue, lack of sleep, changes in the weather etc. If unable to find such a 'normalising' explanation, a person may then attribute their symptom(s) to internal/dispositional factors, involving either psychological causes (such as for example excessive worry) or organic processes (such as for example physical disease). The division of internal/dispositional attributions into psychological and organic ones emanates from research into symptom perception and reflects the biomedical model inherent in Western medicine. In research on illness behaviour, Bishop (1987) found that subjects classified symptoms along four dimensions, two of which corresponded to a physical and a psychological dimension. This has been interpreted as proof that lay perceptions of symptoms are represented along a somatic/psychological axis (Robbins & Kirmayer, 1991).

1.2.2 Subjective illness theories

Causal attributions play a central role in so-called 'subjective illness theories' (Leventhal et al., 1984) or 'lay illness models' (Robbins & Kirmayer, 1991). Theories about health and illness deal with the ideas people use to explain how to maintain a healthy state and why they become ill. The term 'lay beliefs' refers to ideas that are culturally or personally

based rather than attributable to medical understanding (Peters et al., 1998, p. 559). One of the most influential theories in this area is the self-regulation model of illness cognition and behaviour (LSRM) developed by Leventhal and colleagues (Leventhal et al., 1984). According to this model, patients actively develop both cognitive and emotional representations of their illness, which help them make sense of their experience and provide a basis for their coping responses. These representations may draw upon illness information available in people's culture, information obtained in contact with other people, such as medical doctors, and the individual's personal illness experience. Leventhal described five cognitive dimensions around which patients form ideas about their illness :

- Identity is about patients ideas' about possible labels for their symptoms;
- *Cause is concerned with patients' ideas about the likely causes of their condition;*
- Consequence refers to patients' beliefs about illness severity and the personal consequences of the illness (social, psychological, economic, etc.);
- Timeline is about the patient's beliefs about the likely duration of their condition;
- Cure/control reflects the person's belief about the extent to which his or her illness can be cured or controlled.

According to the LSRM, these cognitive dimensions interact with emotional responses, in that for example, a patient's anxiety will influence his or her beliefs about an illness, and the behaviour resulting thereof. Representations reflecting the above dimensions have been shown to influence our decision to seek medical help, to determine compliance with recommended management, coping behaviour, as well as disease outcome (Leventhal et al., 1984; Robbins & Kirmayer, 1991; Roesch & Weiner, 2001).

1.2.3 Assessment of causal attributions in patients with SFDs

Research approaches to the study of causal attributions in patients with SFDs can be divided into quantitative and qualitative approaches. Table 2 provides an overview of instruments developed and/or used to assess causal attributions in patients with somatoform disorders[2]. Most of these instruments were developed to assess causal attributions in relation to a particular illness or group of symptoms. The SIQ (Symptom Interpretation Questionnaire), the KAUSOM (Strukturiertes Interview zur Erfassung von Kausalattributionen bei Patienten mit somatoformen Symptomen) and the CAI (Causal Attribution Interview) assess attributions at the symptom level.

Table 2. Assessment instruments of causal attributions in patients with SFDs

Instrument	Causal attribution dimension	Author (year)
Quantitative – self-rating questionnaire		
SIQ [a]*	psychological, somatic and normalizing attributions	Robbins & Kirmayer (1991)
IPQ/IPQ-R [b]	multidimensional (psychological, risk factor, immunity, and chance attributions)	Weinman et al. (1996) Moss-Morris (2003) Rief et al. (2004)
Inventory of beliefs about symptoms	8 factors including stress, environment, life-style, weak constitution	Salmon et al. (1996)
Itemliste zur subjektiven Krankheits- Theorie	mental, social, interpersonal, and somatic attributions	Faller (1997)
Quantitative - structured interview		
KAUKON [c]	psychosocial and biological-medical attributions	Kröner-Herwig et al. (1993)
KAUSOM [d]*	psychological, social, biological, and medical attributions	Cebulla (2002)

[2] Please note that this list is not all-inclusive.

Qualitative - semi-structured interview		
EMIC [e]	multidimensional	Weiss (1997)
SEMI [f]	multidimensional (don't know, internal, natural, interpersonal/social and supernatural causes)	Lloyd et al. (1998)
CAI [g]	multidimensional (psychological and stress, somatic, environmental)	Hiller et al. (2010)
Qualitative		
open-ended/in-depth interview; content analysis	multidimensional	Martin et al. (2007a) Risør (2009) Salmon et al. (2004, 2009)

Note: * Assessment of causal attributions at the level of individual symptoms.

a. Symptom Interpretation Questionnaire; b. Illness Perception Questionnaire Revised; c. Inventar zur Erfassung von Kausal- und Kontrollattributionen bei chronischen Schmerzpatienten; d. Strukturiertes Interview zur Erfassung von Kausalattributionen bei Patienten mit somatoformen Symptomen; e. Explanatory Model Interview Catalogue; f. Short Explanatory Model Interview; g. Causal Attributions Interview.

Quantitative measures of illness attribution (self-report questionnaires and structured interviews) generally include lists of predetermined causal explanations from which patients can choose the one(s) closest to their own beliefs. With the exception of the KAUSOM, the causal belief items are usually rated on Likert-type scales. The items in the list are based either on clinical observations and/or previous research, taking into account patients' most frequent or typical answers (as in the construction of the IPQ-R), or are theoretically derived (e.g. Robbins & Kirmayer, 1991). Factor analytic techniques tend to be used to identify groups of causal beliefs. Studies using factor analytic approaches (Gaab et al., 2004; Moss-Morris et al., 2002; Moss-Morris & Chalder, 2003; Rief et al., 2004; Van Wilgen et al., 2008; Weinman et al., 1996) support the notion that illness attribution is a multidimensional process, with patients holding coexisting explanations for one and the same symptom or illness. Some quantitative measures (e.g. SIQ, KAUKON) have been criticised for directly assessing a-priori attribution dimensions (e.g. biological-medical vs. psychosocial) instead of allowing respondents to endorse individual causal attribution items (Cebulla, 2002).

Qualitative measures, including open or semi-structured interviews, provide qualitative information that facilitates a deeper understanding of the individual's experience of illness. In line with an *emic* assessment framework (Weiss, 1997), they allow patients to use concepts and categories that are relevant and meaningful to them. Some qualitative studies assess attribution by simply asking patients what they attribute their symptoms to (Martin et al., 2007a). Others apply the more elaborate Explanatory Model Interview (Henningsen et al., 2005; Schroeter et al., 2004). And various authors use the methodology of thematic content analysis of in-depth interviews (Risør, 2009) and of transcripts of audiotaped consultations (Salmon et al., 2004, 2009). The Explanatory Model Interview Catalogue (EMIC) and its shorter version, the Short Explanatory Model Interview (SEMI), were developed to study illness explanatory models in terms of illness-related experience (patterns of distress), meaning (perceived causes) and behaviour (help-seeking history and preferences) (Henningsen et al., 2005; Weiss, 1997). They bridge the gap between qualitative and quantitative methods in that they allow for the collection of qualitative data (prose) which is then analysed quantitatively (Weiss, 1997). While qualitative data may be more clinically relevant (Sensky, 1997), it is more time-consuming to collect and analyse. Further, there is the problem of interviewer bias, i.e. the interviewer may consciously or unconsciously influence the respondent's answers. While interviewer bias can be reduced by using trained interviewers (Cebulla, 2002), to ensure that the analysis is 'grounded in the data rather than reflecting pre-existing ideas' (Peters et al., 1998, p.560) qualitative data should be analysed independently by several different individuals.

Not surprisingly, research outcomes depend on the methods used to assess causal attributions and variations in data handling and analysis (Bhui & Bhugra, 2002; Sensky, 1997). For example, studies assessing causal attributions using both quantitative and qualitative measures found the number of spontaneous mentions to be less than the number of causal attributions endorsed in a questionnaire (Cebulla, 2002; Hiller et al., 2010; Korn, 2003). Further, the dimensions of causal attribution identified seem to vary according to the

research instrument used (see Table 2, above). Also, while some researchers have identified several exclusive attribution *dimensions* - e.g. psychological, somatic and normalising (Robbins & Kirmayer, 1991) -, factor analytic approaches - e.g. based on the IPQ-R (Weinman et al.,1996) -, have yielded a number of attribution *categories*, such as psychological, risk factor, immunity, and chance attributions, with patients endorsing multiple attribution items.

1.2.4 Causal attribution and SFDs – the research evidence

Studies vary largely in terms of the measures used for assessing causal attributions, in terms of data handling and analysis. They differ with regard to the population studied (e.g. chronic pain patients, patients with chronic fatigue syndrome), sample size, setting (e.g. primary vs. tertiary care), the definition and assessment of somatoform disorder (e.g. SFD diagnosed according to SCID, SFD equated with multiple physical symptoms), the comparison group (e.g. SFD vs. NoSFD patients, SFD vs. depressed patients), to name but a few.

Keeping these differences in mind, in the following sections I will look at the types of attribution associated with SFDs, differences in causal attribution according to various socio-demographic variables (such as age and sex), and the association between various causal attribution dimensions and psychopathology. Finally, I will report on the relevance of causal attributions in the treatment and management of SFD patients. In the process, I will describe some of the studies in more detail. The studies selected are to provide an insight into the breadth of research carried out in this area.

1.2.4.1 Causal attributions and SFDs

While some studies support the notion of a tendency towards somatic illness attributions among SFD patients (Kirmayer & Robbins, 1996; MacLeod et al., 1998; Moss-Morris & Petrie, 2001; Nimnuan et al., 2001; Rief et al., 2004), more recent studies and reviews (Aiarzaguena et al., 2008; Goldbeck & Bundschuh, 2007; Hiller et al., 2010; Kirmayer et al., 2004; Martin et al., 2007a; Nikendei et al., 2009; Rief & Broadbent, 2007; Risør, 2009; Schröter et al., 2004) and, in particular, qualitative studies on doctor-patient interaction (Ring et al., 2005; Salmon et al., 2004, 2009) present more of a mixed picture, with SFD patients being open to both somatic and psychosocial explanations of their symptoms.

In a study of 850 patients attending seven outpatient clinics in Southeast London (gastroenterology, gynaecology, neurology, rheumatology, chest, cardiology, and dentistry), Nimnuan and colleagues (Nimnuan et al., 2001) compared the illness attributions of patients with 'medically unexplained' symptoms and those without such symptoms using a self-report questionnaire. Details on scoring were not provided. They found the presence of 'medically unexplained' symptoms to be associated with physical attribution (infectious causes, toxins and allergy), but not psychological attribution (stress, depression, personality and overwork). Patients attributing their symptoms to life-style factors (smoking and drinking) were found to be significantly less likely to have 'medically unexplained' symptoms. They concluded that their results support the notion that patients with MUS attribute their symptoms to physical causes.

Moss-Morris and colleagues (Moss-Morris & Petrie, 2001) compared 53 patients with chronic fatigue syndrome (CFS) with 20 depressed patients on perceptions of their health, illness attributions, and other cognitive factors (self-esteem, cognitive distortions of general and somatic events, symptoms of distress and coping). Two groups of CFS patients

(with or without depression) endorsed significantly more physical and fewer psychological attribution items on the IPQ causal subscale than did depressed patients. In a similar study, comparing illness perceptions and levels of disability in patients with CFS and rheumatoid arthritis (Moss-Morris & Chalder, 2003), CFS patients were more likely to attribute their symptoms to a germ or immune dysfunction.

In a qualitative study carried out in two tertiary care clinics, using a locally adapted version of the EMIC, Henningsen et al. (2005) reported 'pure' SFD patients to predominantly focus on organic causal attributions. This was not the case, however, in patients with anxiety and/or depressive disorders and those in a diagnostic overlap group (SFD and comorbid depressive and anxiety disorders).

Rief et al. (2004) assessed causal illness attributions in a sample of 233 primary care patients, using a 12-item instrument based on the IPQ. Patients diagnosed with a SFD had increased scores on two organic attribution dimensions identified by means of a factor analysis: 'vulnerability to infection and environmental factors' and 'organic causes including genetic and ageing factors'. While SFD patients also considered psychological explanations for their symptoms, scores on 'psychological factors' and 'personal distress' did not differentiate between SFD patients and their non-somatoform counterparts. Furthermore, organic causal beliefs were related to patients' illness behaviour (such as for example, an increased need for medical diagnostic examinations and expression of symptoms). However, given that most patients reported multiple illness attributions, their study also supports the notion of illness attribution as a *multidimensional* process.

In a study comparing the causal attributions of patients at different levels of the health care system, Wessely's team (Euba et al., 1996) examined the causal attributions of patients suffering from Chronic Fatigue Syndrom (CFS) by means of a self-report questionnaire, comparing primary and tertiary care patients with CFS. They found tertiary

care patients to be more likely to attribute their symptoms to organic causes (in addition to presenting with higher levels of fatigue, more somatic symptoms, greater functional impairment, but less overt psychological morbidity). Primary care patients were more likely to make psychosocial attributions. They concluded that physical illness attribution was the result of selection bias and not intrinsic to CFS: the majority of CFS patients in specialist care had been from a higher social class.

While the above studies tend to support the notion that patients with SFDs/MUS are inclined to use somatic explanations to account for their symptoms, most of the above results do not seem as clear-cut as one may have expected. That is, they do not support the notion of an exclusive organic attribution on the part of SFD patients. The following studies present even more of a mixed picture: the authors promote the idea that SFD patients are open to both somatic and psychosocial explanations for their symptoms und underline the multidimensional nature of causal attributions.

Using the Explanatory Model Interview (EMIC) with in-patients from a pain-therapy ward of an Orthopedic clinic in Heidelberg (Germany), Schröter and colleagues (Schröter et al., 2004) found that patients with a somatoform disorder, compared with non-somatoform pain patients, were more likely to spontaneously attribute their symptoms to somatic causes, despite reportedly high levels of psychological distress. Bodily exhaustion was the most important contributing somatic factor. However, when prompted, the majority of SFD patients (over 80%) also endorsed psychological attribution items. The authors stress the importance of an empathetic and patient-centered communication style to elicit psychological attributions.

Goldbeck and Bundschuh (2007) interviewed children and adolescents with a somatoform disorder (n=25) or bronchial asthma (n=25) and their parents with regard to their illness beliefs (causal attributions and locus of control). The SFD patients were recruited from

psychosomatic outpatient clinics. At the time of interview, they were at different stages of the diagnostic work-up; some were receiving psychotherapy. Answers from the semi-structured interviews were content analysed, leading to seven categories of causal attributions: genetic, mental, somatic, developmental, behavioural, social, and physical/ environmental. Compared with patients in the asthma group, SFD patients significantly more often mentioned psychosocial (mental and social) illness attributions. Furthermore, illness beliefs were found to be multidimensional in that patients (and their parents) held on to various illness attributions at the same time. The latter confirms the findings of Rief et al (2004) in a sample of adult SFD patients presented above. While the predominantly psychosocial attribution of SFD patients may have been influenced by the fact that some had already attended psychotherapy (with the results being due to the effect of psychotherapy or patient selection bias) (Goldbeck & Bundschuh, 2007), other studies confirm the presence of psychosocial causal beliefs at an early stage in the attribution process.

So for example, in a study set in primary care centres in Spain, Aiarzaguena and colleagues (Aiarzaguena et al., 2008) found that among male and female patients who had presented at least four or six medically unexplained somatic symptoms, respectively, over the course of their lives, only one third attributed their symptoms entirely to physical causes. One third attributed them to psychological problems and the remaining third to both organic and psychological issues. Patients' causal attributions had been assessed as part of the somatoform symptoms section of the Composite International Diagnostic Interview (CIDI).

A recent semi-structured interview study by Hiller et al. (2010) further reinforces the view that 'multiple attributions seem to be the rule rather than the exception' (p. 15). A majority of SFD patients admitted as inpatients to the Roseneck Center of Behavioural Medicine in Prien, Germany, attributed their symptoms simultaneously to environmental, somatic, and psychological/stress factors, or a combination of two factors. In addition, their

attributions changed over time from the time of symptom onset, with a significant increase for psychological attributions and a decrease for somatic attributions.

Risør (2009) challenges the biomedical view inherent in the notion that SFD patients tend to be preoccupied with physical illness and attribute their symptoms to physical causes from an anthropological perspective. The latter focuses on the cultural and social context of human behaviour. Risør explored illness explanations in nine patients with 'mild or early MUS' during a period of one and a half years by means of semi-structured qualitative interviews. The study was set in Danish primary care. A thematic content analysis revealed that patients used a variety of explanatory idioms (i.e. context-specific explanation) depending on the situation they found themselves in. 'Symptomatic', 'personal', 'social' and 'moral' idioms were used interchangeably and at times concurrently and 'with different emphasis at different times and in different social situations' (p. 518), thus underlining their dynamic nature. The 'symptomatic' idiom (referring to discourse about the physical symptoms), however, was found to be used mainly in a clinical setting, during consultation with patients' GPs.

1.2.4.2 Causal attribution and sex

The prevalence of SFDs has been reported to be higher among female and younger patients (see section 2.1.2, above). There are very few studies, however, exploring age and sex differences in relation to SFD patients' causal attributions. Nykvist et al. (2002) looked at the causal explanations for common somatic symptoms (neck/shoulder problems and sore/upset stomach) among women and men. In a random survey of 1500 persons, respondents were asked to rate the likelihood of 29 different causes for their symptoms on a 7 point Likert-type scale, and to indicate other important causes in response to an open-ended question. They found women to endorse a larger number of causes than men and to

be significantly more likely to consider psychological explanations for their symptoms. Men were more likely to indicate physical work as an important cause. These results confirm those of Robbins and Kirmayer (1991) who found that women reported more somatic symptoms that were not organically explained and that they scored significantly higher on the psychological attribution scale than men. Reasons put forward to explain these differences include: higher levels of stress experienced by women, women holding on to particular concepts of health (considering psychological factors, family structures and social relationships as being important influences on health) and linking together various life events (Nykvist et al., 2002, p. 298-9).

1.2.4.3 Causal attribution and psychopathology

Numerous studies have reported a high level of comorbidity between somatoform and other mental disorders, in particular, depression and anxiety disorders (see section 1.1.3, above). It is thus important to look at the potential influence of these psychiatric disorders on patients' causal attributions.

MacLeod et al. (1998) presented patients attending a large general practice in London with statements referring to 10 common bodily symptoms taken from the Symptom Interpretation Questionnaire (SIQ) of Robbins & Kirmayer (1991), an anxiety and a hypochondriacal belief scale. Patients were divided into three groups: anxious hypochondriacal, generally anxious and non-anxious. Compared to non-anxious patients, both anxious groups gave more psychological and fewer normalising reasons to explain the symptoms. Hypochondriasis, on the other hand, was related to giving more somatic attributions. Robbins and Kirmayer (1991) had obtained similar results with their sample of family medicine patients.

In their qualitative study, Henningsen et al. (2005) found that psychosocial causal attribution was significantly more prevalent among SFD patients with a comorbid anxiety and/or depressive disorder and those with a pure anxiety and/or depressive disorder than among 'pure' SFD patients. Similarly, from their quantitative study of SFD patients in primary care, Rief et al. (2004) reported comorbidity with depression and/or anxiety disorders to be associated with psychological illness attributions. A recent study by Hiller and colleagues (Hiller et al., 2010) exploring causal attributions by means of semi-structured interviews in SFD and chronic pain patients confirms the above results: they found depression to be positively correlated with psychological/stress and negatively with somatic attributions.

In the study by Moss-Morris and Petrie (2001), mentioned above, in which they examined causal illness attributions among CFS patients and patients with depression, depressed patients attributed their symptoms mainly to psychological factors. Surprisingly, the CFS-depression overlap group were even more likely than the 'pure' CFS patients to mention somatic causal attributions to explain their symptoms.

In sum, the relationship between causal illness attributions and SFDs is complex. Increased scores for both somatic and psychological explanations have been found in SFD patients. Somatic illness attributions have been shown to be related to illness behaviour, in particular, demands for medical treatment. Comorbidity with psychiatric disorders has been reported to be associated with psychological illness attributions. Furthermore, studies support the multi-dimensional nature of causal attributions. An interesting and important contribution to the above discussion comes from studies on treatment outcomes, in particular, on reattribution.

1.2.5 The relevance of causal attributions in the treatment of SFDs

Attributions are part of the cognitive dimension of illness representations. Gaining an adequate understanding of these attributions plays an important role in the treatment of SFD patients, in particular, with regard to cognitive-behavioural approaches. In addition to looking at dysfunctional emotions and behaviours, cognitive-behavioural therapy (CBT) focuses on identifying underlying dysfunctional beliefs, challenges these by reviewing available evidence and considering alternatives (Allen et al., 2006; Martin et al., 2007b; Wright et al., 2009). Evidence exists that CBT is effective for a variety of somatoform disorders (Allen et al., 2006; Kroenke, 2007; Martin et al., 2007b) functional somatic symptoms (such as headache and low back pain) and syndromes (such as irritable bowel syndrome, fibromyalgia and CFS) (Kroenke & Swindle, 2000). In a primary care setting, where somatoform symptoms are a common phenomenon, however, CBT has been found to be suitable and acceptable only to a minority of patients presenting with such symptoms (Arnold et al., 2004).

For use in primary care, Goldberg et al. (1989) developed a so-called *reattribution* treatment model. Based on the assumption that somatoform disorder patients hold on to organic explanations for their symptoms, this model proposes to encourage patients to *reattribute* their symptoms, that is, to relate them to psychosocial problems. Evidence for the effectiveness of reattribution, however, remains equivocal. Morriss and colleagues (Morriss et al., 2007) found that delivering a reattribution training program to GPs improved doctor-patient communication, but did not improve patient outcomes or service use. While patients reported being more satisfied with the help they received, and more patients endorsed an emotional cause for their symptoms, these associations were not significant (Morriss & Gask, 2002). Further reattribution studies report limited, non-significant effects on patients' physical symptoms (Larish et al., 2004), but a significant reduction in health care utilisation (Rief et al., 2006). In sum, 'training GPs to explain how symptoms can relate to psychosocial problems

improves the quality of doctor-patient communication, though not necessarily patient health' (Peters et al., 2008, p. 443).

There is evidence indicating that interpersonal psychodynamic therapy (IPT), a variant of psychodynamic therapy, may have beneficial effects. IPT emphasises the importance of interpersonal processes and relationships as well as emotional issues in the development and maintenance of somatoform symptoms. Here, the exploration of a patient's causal attributions forms part of an appreciation of the patient's subjective illness theories, as the basis for a stable therapeutic relationship. A meta-analytic review of studies in which short-term psychodynamic psychotherapies were delivered to patients with a variety of somatic symptom disorders (including somatoform disorders) revealed positive effects on physical and psychological symptoms as well as on social adjustment (Abbass et al., 2009). In a first randomised controlled study of 211 patients from six German outpatient centres, meeting criteria for multisomatoform disorder, Sattel and colleagues (Sattel et al., 2012) evaluated the long-term effectiveness of a brief IPT intervention consisting of 12 weekly sessions . Treatment significantly improved patients' health related quality of life at nine months follow-up.

2 Aims of the study

In the light of subjective illness theories, causal illness attributions have been shown to influence the development, maintenance and management of somatoform disorders. In view of the forthcoming DSM-V, somatic causal attributions have even been considered as potential positive criteria in the definition of these disorders. While the ICD-10 lists the adherence to somatic causal attributions as one of the main features of SFD patients, empirical evidence of this assumption has been shown to be relatively rare.

Therefore, the overall purpose of the study presented here is to examine the extent of somatic causal illness attribution among SFD patients in order to assess the possible use of this dimension as a positive criterion in the definition of somatoform disorders, with the long-term view to provide the basis for better diagnostic and therapeutic management. In particular, the following research questions are being addressed:

1. Somatic causal attribution and SFDs

According to the literature presented above, the relationship between causal illness attributions and SFDs is complex. While some studies support the notion of an exclusive organic attribution among SFD patients, others have found SFD patients to be open to both somatic and psychological explanations for their symptoms. In the light of such mixed findings, and propositions to use the adherence to somatic causal attributions as a positive criterion for SFDs, I aim to test the hypothesis that SFD patients tend to consider their symptoms as essentially due to somatic factors.

2. Causal attribution and sex

Studies exploring sex differences in causal attributions among SFD patients are rare. In line with the findings of one of these studies presented above (Nykvist et al., 2002), I expect women to be more likely to consider psychological explanations for their symptoms.

3. Causal attribution and psychopathology

Comorbidity with psychiatric disorders has generally been reported to be associated with psychosocial illness attributions among SFD patients. In line with these findings, a positive relationship is expected between the extent of psychosocial causal attribution and the presence of associated psychiatric disorders, in particular, depression and anxiety (assessed both categorically and dimensionally).

4. Comparing qualitative and quantitative research methods

Research outcomes with regard to causal attributions among SFD patients vary largely with regard to the research method used (see section 2.2.3, above). Only a limited number of studies have examined the causal attributions of SFD patients using both qualitative and quantitative research methods (e.g. Cebulla, 2002; Hiller et al., 2010; Korn, 2003). As both research methods have their own strengths and weaknesses, by combining them, the study attempts to offset their weaknesses and to draw on the strengths of both. Therefore, it sets out to assess and compare patients´ spontaneous and prompted causal attributions.

In keeping with previous findings (Cebulla, 2002; Hiller et al., 2010, Korn, 2003), the number of spontaneous mentions is predicted to be less than the number of causal attributions endorsed in a predetermined list of causal attributions (IPQ-R causal scale).

Further, the study intends to examine the potential utility of the IPQ-R causal scale when used for assessing the causal attributions of SFD patients. In particular, and in line with previous research findings (Moss-Morris & Chalder, 2003; Rief et al., 2004; Weinman et al., 1996), it is hypothesised that, the IPQ-R will allow the identification of multiple and coexisting causal attributions among SFD patients. In addition, the relevance of the factor structure identified in physical illness is assessed for our patient group.

3 Method

This study is part of a larger cross-sectional study, the so-called 'SomA study', exploring potential positive criteria for SFDs (Hausteiner et al., 2009). In a sample of patients presenting for an allergy diagnostic work-up, it examines the causal illness attributions of SFD and non-somatoform disorder (NoSFD) patients and those of their controls, hospitalized for allergen-specific immunotherapy (VIT). In particular, the study compares patients´ spontaneous and prompted causal attributions using both qualitative and quantitative research measures.

3.1 Study participants, design and procedure

3.1.1 Participants

300 consecutive patients admitted as inpatients to the TUM allergy department (Klinik und Poliklinik für Dermatologie und Allergologie am Biederstein, Technische Universität München) were invited to participate in the study. 245 of these patients were hospitalised for allergy testing (work-up patients): their symptoms could not be diagnosed with sufficient certainty in an outpatient setting or provocation testing was considered fraught with risk. 55 patients already had an established diagnosis of hymenoptera (bee and wasp) venom allergy, and were admitted for allergen-specific venom immunotherapy (VIT patients). They were included in the study to control for possible effects of the work-up situation. Patients were recruited when they were aged 18-65 and had a good command of the German language. An 11 months study period (January to November 2007) was chosen to account for seasonal variations in the type of allergies presented.

3.1.2 Procedure

At admission to the clinic patients were handed an information sheet about the study "Allergy and bodily symptoms" by the attending physician (see Appendix 1). Physicians had been instructed to emphasise that all eligible patients attending the allergy clinic were being invited to participate in the study. This was to prevent any apprehension on the part of the patient that only a certain subgroup (e.g. those with apparent psychological problems) was being selected to take part. All work-up patients received a thorough clinical examination, including blood and skin testing, as well as double-blind, placebo-controlled provocation testing with foods, additives, drugs, or contact/inhalative substances (such as paint or latex), in line with their presenting symptoms. Within the first two days of their stay in the clinic, all eligible patients were contacted by the research team and informed about the aims and extent of the study. Patients giving informed consent (see Appendix 2) were then interviewed by one of two board certified psychiatrists (both certified SCID-interviewers). Following the interview, patients were asked to fill in a set of self-report questionnaires. Two days following the interview, and most importantly, prior to patients obtaining any medical test results, questionnaires were collected by the research team. At the end of the work-up, allergists rated the organic explicability of patients' presenting symptoms.

3.2 Assessment instruments

The study instruments consisted of a semi-structured interview and a battery of self-rating questionnaires. In addition, at the end of the battery of tests, information about patients' age, sex, marital and socioeconomic status was obtained by means of closed questions.

3.2.1 Interview

The interviewers emphasised that they were not members of staff and that they had no previous knowledge about the interviewee, thus attempting to create an atmosphere in which a discourse about the patients' experiences and thoughts about their health and previous contact with the health care system could freely develop. First, patients' medical history, current symptoms and illnesses and utilisation of health care services in the last 12 months were recorded. Then, patients' *spontaneous causal attributions* were explored. The main question asked was: 'What do you think is or are the causes of your current symptom(s) and/or intolerance(s)?' (in German: 'Man macht sich ja so seine Gedanken: Was glauben Sie selbst, ist die Ursache dieser Beschwerde(n)/ Unverträglichkeit(en)?'). Responses were recorded verbatim.

The Structured Clinical Interview for DSM-IV. The diagnosis of a SFD was ascertained using section "G" (somatoform disorders) of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I, abridged and German version), the current gold standard for the diagnosis of SFDs (Hiller & Janca, 2003; Wittchen et al., 1997). SCID is a semi-structured interview and was originally designed to improve on the limitations of unstructured clinical interviews: SCID-I for assessing Axis I (psychiatric) Disorders and SCID-II for assessing Axis-II (personality) Disorders. SCID-I covers all the major mental disorders and includes a separate section (section G) on somatoform disorders.

Reliability and validity. While there are extensive studies on the reliabilities of SCID for various axis I and axis II mental disorders (Columbia University, 2011), such studies are largely missing for all but a few somatoform disorder diagnoses. A recent assessment of the inter-rater reliability of 12 Axis I disorders (not including somatoform disorders) of SCID I

showed moderate to excellent inter-rater agreements[3] (Lobbestael et al., 2011). Interrater-reliabilities have been reported to be lower for SFDs than for depressive and anxiety disorders, with a Kappa value of 0.7 for somatisation disorder as compared to 1.0 and 0.96 for depressive and anxiety disorders, respectively (Löwe et al., 2003). In a German study reported by Hiller & Janca (2003), test-retest reliability of the SCID for DSM-III-R somatisation disorder was reported to be poor (with a Kappa value of 0.22)[4]. The validity of the SCID is difficult to determine because of the lack of an agreed standard against which to test the interview results. By default, diagnoses based on the SCID have come to be considered a 'gold standard' (Hiller & Janca, 2003, p. 169).

During the interview, DSM-IV criteria for the following somatoform disorders were evaluated by means of semi-structured, open-ended questions: somatisation disorder, undifferentiated somatoform disorder, and pain disorder, (see Table 1, section 1.1.4 for DSM-IV diagnostic criteria). These questions systematically review symptoms pertaining to various organ systems, the impairment in social, occupational, or other areas of functioning resulting thereof, and the extent to which these symptoms can be organically explained. The trained SCID interviewer makes diagnostic decisions based on patients' answers in the interview and all other available information (such as, observations during interview, third-party information, or available medical reports). Patients who fully met criteria for a somatisation disorder, pain disorder, or undifferentiated somatoform disorder were identified as SFD patients.

In addition, Kroenke's criteria for multisomatoform disorder (Jackson & Kroenke, 2008; Kroenke et al., 1997; Kroenke et al., 2007) were applied. Hypochondriasis, conversion

[3] Kappa values above .75 were considered to reflect excellent agreement; values from .41 to .75, moderate agreement and below .40 poor agreement.
[4] Segal et al. (1993) report inter-rater reliabilities of 1.0 for somatisation and somatoform pain disorder for SCID-I for DSM-III-R. However, their extremely small sample size for somatoform disorders (namely 4), precludes any meaningful interpretation of these results.

disorder (where not congruent with somatisation disorder, pain disorder, or undifferentiated somatoform disorder), and body dysmorphic disorder were excluded from the definition of a somatoform disorder. Hypochondriasis is dominated by health anxiety rather than bodily symptoms, and at the time the study was implemented, it was discussed to be removed from the category of SFD and to be moved to the category of Anxiety Disorders (Kroenke et al., 2007)[5]. The DSM-V Somatic Symptoms Disorder Work Group now regards the DSM-IV category of hypochondriasis as encompassing two separate disorders: 80% of patients previously diagnosed with hypochondriasis are considered to meet criteria for Complex Somatic Symptom Disorder; the remaining patients, characterized by high levels of illness anxiety and minimal somatic complaints would be diagnosed with Illness Anxiety Disorder (APA, 2011). Conversion disorder usually presents with short-term pseudo-neurological symptoms; DSM-IV lists no minimum requirement for their duration. While there have been recommendations to move it to the category of Dissociative Disorders (Kroenke et al., 2007)[6], the Somatic Symptoms Disorder Work Group suggests retaining it in the new Somatic Symptom Disorders section and changing its name to 'functional neurological disorder' (APA, 2011). While section G of the SCID does not explicitly cover conversion disorder, most patients with persistent conversion symptoms do qualify for another SFD, and are therefore captured by the SCID. Body dysmorphic disorder is rarely diagnosed in general medical settings and some experts consider it to be a subtype of Obsessive-Compulsive Disorders (Kroenke et al., 2007; Okasha, 2003; Strassnig et al., 2006). Diagnostic criteria remain to be determined and movement to the category of Anxiety Disorders is being considered (APA, 2011) .

The SCID diagnosis was complemented by the allergists' rating of organic explicability of the patients' presenting 'allergy-suspect' symptoms, at the end of the work-up. This rating was based on a systematic stepped review of all clinical test results. A primary

[5] Answers pertaining to this section of the SCID interview were recorded, but they did not enter analyses as a somatoform disorder.
[6] In the ICD-10 system, conversion disorder is classified as a dissociative disorder.

SFD diagnosis was given to patients whose current and predominant symptom(s) could not be medically explained. A secondary SFD category was used to refer to patients suffering from a SFD (diagnosed according to SCID), but whose *presenting* symptoms were medically explicable, as determined by the allergist's organic explicability rating (e.g. a patient having had an anaphylactic reaction caused by analgesics, and concurrently suffering from a somatoform pain disorder) (see Hausteiner et al., 2009, for details on the organic explicability rating instrument).

3.2.2 Self-report measures

The latter were selected on the basis that they refer to cognitive, affective, behavioural and interactional characteristics previously found to be related to somatoform disorders, and that their psychometric properties have been systematically reviewed. With the focus of the dissertation being on the assessment of patients' causal attributions, I will concentrate on a detailed description of the causal dimension of the revised version of the Illness Perception Questionnaire (IPQ-R). In addition, I will elaborate on the use of the Patient Health Questionnaire (validated German version, PHQ-D) applied to assess patients for various mental disorders.

3.2.2.1 The modified causal attribution dimension of the IPQ-R

Part of the battery of self-report measures (Hausteiner et al., 2009), patients were presented with the IPQ-R causal attribution scale (German version, Gaab et al., 2007). The latter consists of a list of 18 ideas about the likely cause(s) of an illness. It was validated by Moss-Morris *et al.* (2002) and Gaab *et al.* (2004) for eight organically defined illness groups

(asthma, diabetes, rheumatoid arthritis, chronic pain, acute pain, myocardial infarction, multiple sclerosis and HIV) and various somatoform illness groups, respectively.

The causal attribution scale is part of the revised Illness Perception Questionnaire (IPQ-R) which assesses patients' cognitive and emotional representation of illness (Moss-Morris et al., 2002). The latter has demonstrated good construct[7] and discriminant validity, internal consistency[8] and test-retest reliability[9] (Moss-Morris et al., 2002). The items of the causal attribution scale have been subsumed under the following four categories (see Table 4, below): *Psychological* attributions include items such as stress and overwork; *risk* attributions include factors such as diet and heredity; *immunity* attributions include factors such as germs and viruses, and *accident or chance* attributions refer to items such as accident or bad luck.

All items are rated on a five-point Likert-type scale from *strongly disagree* to *strongly agree* (scored 1 to 5). The IPQ-R was designed to be flexible enough to be modified for use with a wide range of illnesses (Moss-Morris et al., 2002). For the purpose of the present study, the wording of instructions was slightly modified, replacing the word 'illness' with 'allergy-suspect symptoms'. Due to the nature of our sample, and after consultation with the author of the German version, J. Gaab, the listing of 18 beliefs was extended by adding the item 'allergy'. Based on feedback from a short pilot study, the original answer code *"neither agree nor disagree"* was replaced by "*partly agree"*.

[7] Construct validity indicates the extent to which the theoretical construct has been successfully operationalised; it refers to the validity of the theory that lies behind the test.
[8] Internal consistency reliability (Cronbach's alpha) assesses the consistency of results across items within a test.
[9] Test-retest reliability assesses the consistency of a measure from one time to another, that is, when administering the same test to the same sample on two different occasions.

Table 4. Causal illness attribution: subscales identified by Moss-Morris (2002) [10]

Subscales	Causal items
Psychological attributions	Stress or worry My mental attitude, e.g. thinking about life negatively Family problems or worries caused my illness Overwork My emotional state, e.g. feeling down, lonely, anxious, empty My personality
Risk factors	Hereditary – it runs in my family Diet or eating habits Poor medical care in my past My own behaviour Ageing Smoking Alcohol
Immunity	Germs or viruses Pollution in the environment Altered immunity
Accident or chance	Chance or bad luck Accident or injury
	*Allergy**

Note: * denotes new item not included in the original IPQ-R.

3.2.2.2 PHQ-D

To screen patients for associated mental disorders, several modules of the widely used and well-established Patient Health Questionnaire (validated German version, PHQ-D) (Löwe et al., 2002) were presented to patients. The PHQ is an internationally used and well-validated measure and allows for both a dimensional (e.g. depressive symptom severity) and a categorical analysis (e.g. major depressive syndrome) of various mental disorders (Kroenke et al., 2001; Spitzer et al., 1999, 2006). The following modules were selected: the PHQ-9 for the categorical and dimensional assessment of depression (Kroenke et al., 2001), one module for the categorical assessment of panic disorder, the GAD-7 for the categorical and dimensional assessment of generalised and other anxiety disorders (Spitzer et al.,

[10] A Principal Component Analysis (Factor Analysis) computed on the 18 causal items produced four factors, accounting for 57% of the total variance. Cronbach alphas ranged from .86 for psychological attributions to .77 for risk attributions, to .67 for immunity and .23 for accident and chance attributions (Moss-Morris et al., 2002).

2006), and modules for the categorical assessment of eating disorders (such as bulimia nervosa and ‘binge eating’).

The PHQ is a self-administered version of the PRIME-MD diagnostic instrument for common mental disorders (Spitzer et al., 1999, 2006). The full version of the PHQ assesses eight mental disorders using the diagnostic criteria from the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV). It distinguishes threshold from subthreshold disorders: The former correspond to specific DSM-IV diagnoses such as major depressive disorder, panic disorder, or other anxiety disorder; the latter refer to disorders whose criteria encompass fewer symptoms than those required for any specific DSM-IV diagnoses such as ‘other depressive disorder’ (Kroenke et al., 2001). *Categorical* assessment is based on diagnostic algorithms (Löwe et al., 2002).

The *dimensional* assessment of depression based on the PHQ-9 asks about the frequency of depressed mood and anhedonia over the past two weeks. For each of nine depressive symptoms, patients indicate whether the symptom has bothered them ‘not at all’, ‘several days’, ‘more than half the days’, or ‘nearly every day’ (scored from 0 to 3, total scale score 0-27) during the previous two weeks (Kroenke et al., 2001). The dimensional assessment of general anxiety by means of the GAD-7 consists of 7 items reflecting the DSM-IV symptom criteria for generalized anxiety disorder (GAD), such as feeling nervous or worrying too much. Similar to the PHQ-9, response options range from ‘not at all’ to ‘nearly everyday’ (scored as 0 to 3, total scale score 0-21) (Spitzer et al., 2006).

3.3 Data analysis

Interview. Similar to Korn (2003) and Martin et al. (2007), the open prose was coded independently by the author (S.G.) and the study supervisor (C.H.) according to five dimensions (psychological, social, medical, health behaviour or 'don't know'). While each answer was placed into one category only, multiple mentions within the same category and across various categories were possible. In a second step, the above dimensions were collapsed into psychosocial (psychological and social), somatic (medical and health behaviour) or mixed attributions. Patients reporting that they had 'no idea' as to what could be causing their symptoms were analysed separately.

IPQ-R causal scale. Analysis of the IPQ-R causal scale does not imply the computation of a scale score. Rather, the items are to be analysed in terms of patients' adherence or non-adherence to the individual causal beliefs. With a sufficient sample size (n=90 or more), factor analysis can be used to identify groups of causal beliefs which can then be used as sub-scales (Gaab et al., 2007).

To investigate the factor structure of the IPQ-R causal scale and to identify groups of causal attributions specific to our patient group, I submitted the 19 causal attribution items to a factor analysis. According to recommendations in the literature (Hagger & Orbell, 2005; Moss-Morris et al., 2002; Wittkowski et al., 2008), I conducted an Exploratory Factor Analysis[11], using a Principal Components Analysis followed by oblimin rotation[12] to rotate the factors to a simple structure. I examined several factor solutions. In the absence of a clear factor structure, I proceeded as follows.

[11] The aim of factor analysis is to simplify an array of data by indicating what the important underlying variables or factors are (Kline, 2002). A factor is a construct or dimension, which accounts for the relationships (correlations) between variables.

[12] The goal of rotation is to simplify and clarify the data structure, that is, to obtain factors that are clearly marked by high loadings for some variables and low loadings for others. As in the social sciences one generally expects some correlation among factors (Costello & Osborne, 2005), I used an oblique rotation method that allows the factors to correlate.

I calculated the percentages of participants endorsing individual causal items on the IPQ-R (i.e. corresponding to 'partly', 'mostly' or 'fully agree'). In line with the analysis of the qualitative data, I subsequently classified the IPQ-R items into psychosocial (items 1, 9-12, 17; see Table 9) and somatic causal attributions (remaining items). I then assigned patients' spontaneous and prompted responses to a psychosocial, somatic or mixed attribution style depending on whether they endorsed purely psychosocial, somatic, or psychosocial *and* somatic attributions.

Further, to assess the relevance of the IPQ-R causal scale for our sample, for each patient holding specific beliefs about the etiology of their symptoms (i.e. excluding 'don't knows'/'no idea') I compared the cause(s) mentioned in the free response task with their answer on the IPQ-R causal scale. That is, where the spontaneously mentioned causal attribution matched one of the 19 causal items of the IPQ-R, I checked whether the corresponding IPQ-R item had been endorsed by a score of 3, 4, or 5 ('partly', 'mostly', or 'fully' agree). Further, I took note of items endorsed on the IPQ-R that had not previously been mentioned in the free response task.

3.4 Statistics

All data were analysed using the Statistical Package for the Social Sciences (SPSS), version 16.0. Interrater-reliability of the allocation of spontaneous causal attributions to the pschosocial-somatic divide was assessed with Cohen's κ coefficient. Continuous variables were summarised using the mean and standard deviation (SD). Absolute numbers and percentages were used to describe categorical variables. In terms of a closed test procedure, comparisons across the three sample groups were followed by pair wise comparisons where significant differences were found. One-way analyses of variance (ANOVA) were applied to

compare means between more than two independent samples, followed by pair wise comparisons using independent t-tests. Where the measurement variable did not meet the normality assumption, Kruskal-Wallis and Mann-Whitney-U tests were used respectively. To compare observed frequencies between patient subgroups I used the χ^2-test. Where sample sizes were small, Fisher's exact test statistics are reported.

With regard to the factor analysis, selection criteria for the best factor structure were: eigenvalues[13] greater than 1.0, item loadings[14] greater than .4, few item cross loadings, and no factors with fewer than two items (Costello & Osborne, 2005). Cronbach's alpha coefficients were calculated to examine the internal consistency of the subscales. Pearson's correlation coefficients were calculated to examine correlations between individual items.

Two-sided tests of significance were carried out at the 0.05 level.

Multiple testing, in particular at the level of the IPQ-R causal scale items, increases the probability of a type 1 error occurring – i.e. deciding that the independent variable had an effect on the dependent variable when it did not have. Here, the overall rate of obtaining significant results by chance may considerably exceed the 0.05 level.

3.5 Ethics

All procedures were performed as approved by the Institutional Review Board, Medical Faculty, TUM. Complete anonymity was assured.

[13] The eigenvalue, corresponding to the sum of squares of the factor loadings, reflects the variance explained by a factor. The larger the eigenvalue, the more variance is explained by the factor.
[14] Factor loadings are the correlations of a variable or item with a factor.

4 Results

4.1 Patient participation, SFD diagnoses and demographics

268 out of 300 patients meeting the inclusion criteria agreed to participate in the study. 89% of work-up patients (218 out of 245; 72% women; mean age 43, SD 13.1) and 91% of VIT patients (50 out of 55; 68% women; mean age 45, SD 10.9) agreed to take part. 14 of the 218 work-up patients participating in the study consented to the interview only. There were no drop-outs during interview. Where applicable, patients only taking part in the interview were included in further analyses. Overall, reasons for non-participation were: lack of interest in the study (n=19), being too busy (n=4), medical (severe allergic reaction and epileptic seizure, n=2), and organisational problems (e.g. very short stay in the clinic, n=7). Reasons for not completing or returning the questionnaire were not recorded. Participants and non-participants did not differ in terms of sex. However, older patients were less likely to participate (p=0.03).

In the work-up group, 69 out of 218 patients (32%) were diagnosed with a SFD; 48 (22%) with a *primary* and 21 (10%) with a *secondary* SFD. None of the 50 VIT patients were diagnosed with a *primary* SFD; 3 (6%) were diagnosed with a *secondary* SFD. Prevalence rates of the various somatoform disorders diagnosed according to SCID are presented in Table 5. The majority of SFD patients were diagnosed with 'undifferentiated somatoform disorder' (n=30, 13.8%). Kroenke's criteria for 'multisomatoform disorder' were applicable to 26 patients (11.9%). 10 patients (4.6%) were diagnosed with a 'pain disorder', and merely 3 patients (1.3%) met the criteria for 'somatisation disorder'. As I was interested foremost in patients' current symptoms and their ideas about the likely causes thereof, I excluded the 24 patients with a secondary SFD from subsequent analyses, ending up with a sample total of 244 patients.

Table 5. Prevalence rates of somatoform disorders (n=268)

Somatoform disorders	***Work-up group*** *(n=218)*			***Control group/VIT*** *(n=50)*		
	Total *n (%)*	*Primary diagnosis* *n*	*Secondary diagnosis* *n*	*Total* *n (%)*	*Primary diagnosis* *n*	*Secondary diagnosis* *n*
Somatisation disorder	3 (1.3)	2	1	-	-	-
Pain disorder	10 (4.6)	7	3	1	-	1
Undifferentiated somatoform disorder	30 (13.8)	21	9	2	-	2
Multisomatoform disorder	26 (11.9)	18	8	-	-	-
Any somatoform disorder	69 (32)	48	21	3 (6)	0	3

SFD patients (n=48), NoSFD patients (n=149) and controls (n=47) were well matched for age, sex, socioeconomic variables (such as education, occupation and marital status), number of concurrent somatic diagnoses and duration of symptoms (see Table 6, below).

4.2 Psychiatric comorbidity

Two sample group comparisons revealed that SFD patients were significantly more likely to be diagnosed with a psychiatric disorder (as assessed by means of the PHQ) than NoSFD ($X2=13.68$, $df=1$, $p<0.001$) and VIT patients ($X2=9.83$, $df=1$, $p=0.002$). No such differences existed between NoSFD and VIT patients ($F= 0.76$, $p= 0.77$). In particular, the PHQ category ‘other depressive syndrome’ was more likely to be diagnosed in SFD patients than in NoSFD or VIT patients ($F=15.02$, $p<0.001$) (see Table 7, below).

Similarly, the results of a Kruskal–Wallis test were significant for both continuous measures of depression (PHQ-9) ($H=41.62$, $df=2$, $p<0.001$) and generalised anxiety (GAD-7) ($H=12.41$, $df=2$, $p=0.002$). The mean ranks of scores were higher for SFD patients than their

Table 6. Sociodemographic variables, concurrent somatic diagnoses and duration of symptoms (n=244)

	Work-up group *(n=197)*		***VIT*** *(n=47)**	
	SFD *(n=48)**	***NoSFD*** *(n=149)**		*p*
Age (in years)	*Mean (SD)*	*Mean (SD)*	*Mean (SD)*	0.99 [a]
	43.0 (12.8)	43.2 (12.9)	43.1 (10.7)	
Sex	*n (%)*	*n (%)*	*n (%)*	0.18 [b]
male	9 (18.8)	47 (31.5)	16 (34.0)	
female	39 (81.2)	102 (68.5)	31 (66.0)	
Education	*n (%)*	*n (%)*	*n (%)*	0.47 [b]
≤ 11 school years	18 (42.9)	49 (36.8)	21 (46.7)	
≥ 12 school years	24 (57.1)	84 (63.2)	24 (53.3)	
Current occupation (incl. training)	*n (%)*	*n (%)*	*n (%)*	0.13 [d]
yes	35 (81.4)	123 (89.1)	42 (95.5)	
no	8 (18.6)	15 (10.9)	2 (4.5)	
Marital status	*n (%)*	*n (%)*	*n (%)*	0.86 [d]
married	23 (54.8)	79 (57.2)	25 (55.6)	
divorced	7 (16.7)	13 (9.4)	4 (8.9)	
widowed	-	3 (2.2)	1 (2.2)	
single	12 (28.6)	43 (31.2)	15 (33.3)	
Living with a partner	*n (%)*	*n (%)*	*n (%)*	1.00 [b]
yes	32 (78.0)	106 (77.4)	33 (76.7)	
no	9 (22.0)	31 (22.6)	10 (23.3)	
Duration of presenting symptoms (in years)	*Mean (SD)*	*Mean (SD)*	*Mean (SD)*	0.25 [c]
	8.8 (10.9)	5.9 (8.4)	6.1 (8.4)	
Number of current somatic diagnoses (other than allergy)	*n (%)*	*n (%)*	*n (%)*	0.24 [d]
0	27 (56.2)	73 (49.0)	32 (68.1)	
1-2	19 (39.6)	64 (43.0)	13 (27.7)	
≥ 3	2 (4.2)	12 (8.1)	2 (4.3)	
History of allergy	*n (%)*	*n (%)*	*n (%)*	0.31 [b]
yes	22 (45.8)	73 (49.0)	17 (36.2)	
no	26 (54.2)	76 (51.0)	30 (63.8)	

Note: SD = Standard deviation

* The number of subjects for each variable varies because of missing data.

a. p-value of the One-way ANOVA

b. p-value of the X^2-test

c. p-value of the Kruskal-Wallis test

d. p-value of the Fisher exact test

non-somatoform counterparts (NoSFD and VIT patients). Two sample group comparisons (Mann-Whitney-U test) revealed that SFD patients tended to be significantly more depressed and anxious than NoSFD patients (z=-5.75, p<0.001; z=-2.82, p=0.005, respectively), than VIT patients (z=-5.66, p<0.001; z=-3.38, p=0.001; respectively). NoSFD and VIT patients did not differ in terms of their depression and anxiety scores (z=-1.82, p=0.07; z=-1.41, p=0.16; respectively).

Table 7. Psychiatric comorbidity (n=244)

	Work-up group *(n=218)*		***VIT*** *(n=45)*	
	SFD *(n=42)**	***NoSFD*** *(n=141)*		*p*
Any psychiatric diagnosis (PHQ-D)	*n (%)*	*n (%)*	*n (%)*	0.001 [a]
Yes	14 (33.3)	14 (9.9)	3 (6.7)	
No	28 (66.7)	127 (90.1)	42 (93.3)	
PHQ-D diagnoses***	*n (%)*	*n (%)*	*n (%)*	
Major depression	2 (4.8)	1 (0.7)	-	0.12[a]
Other depressive disorder**	11 (26.2)	7 (5.0)	2 (4.4)	<0.001[a]
Panic disorder	3 (7.0)	6 (4.3)	-	0.20[a]
Other anxiety disorder	1 (2.3)	2 (1.4)	1 (2.3)	0.64[a]
Bulimia/binge-eating disorder	1 (2.3)	2 (1.4)	-	0.62[a]
Depression (PHQ-9)	*Mean (SD)*	*Mean (SD)*	*Mean (SD)*	
	8.1 (4.3)	3.9 (2.9)	3.0 (2.8)	<0.001 [b]
Anxiety (GAD-7)	*Mean (SD)*	*Mean (SD)*	*Mean (SD)*	
	5.1 (3.3)	3.5 (2.6)	3.0 (2.6)	0.002 [b]

Note: * The number of subjects for each variable varies slightly because of missing data.
** Patients diagnosed with a major depression also appear in the category 'other depressive disorder'.
*** Multiple diagnoses possible.
a. p-value of the Fisher exact test
b. p-value of the Kruskal-Wallis-Test

4.3 Spontaneous causal attribution

Out of the total sample (n=244), patients holding specific beliefs about the etiology of their symptoms (n=163) cited 234 causes altogether (mean 1.4, SD 0.7). Of these, 53 (mean

1.1, SD 0.3) were psychosocial and 181 (mean 1.3, SD 0.6) were somatic attributions. Examples of responses given and the categories they were assigned to are presented in Table 8.

Table 8. Classification of spontaneous causal attributions

Categories	Spontaneous causal attributions
Psychosocial	
Psychological	Mental; emotional sensitivity; anxiety; my emotional state (e.g. feeling lonely, anxious, empty); anxiety; psychological factors; cursed by my deceased father; psychosomatic;
Social	Stress or worry (as a child, at home, at work, at school, marriage, caring for a relative, bereavement); family drama; lack of work-life balance; burn out; the last straw; feeling overburdened;
Somatic	
Biological/medical	Allergies (to medication, antibiotics, analgesics; dairy products, latex, fish, nickel, pollen, various allergens, wasps, products in the house); too many insect stings; mastocytosis; too many antibiotics (as a child); body can't cope with too much medication; heart tablets; wrong homeopathic remedies; hypersensitivity; the sun; food intolerance; additives; hereditary/runs in the family/disposition/genetic; disease of civilization; poor general condition; COPD; hyperthyroidism; internal organ failure; related to mucosa frailty; pollution in the environment; climate; chemicals/noxa (disinfectant, chlorine, dye; additives, multiple chemical sensitivity); dental filling; amalgam; palladium; nickel; vaccine; altered immunity; acne inversa; hypersensitivity to adrenaline; immune mediated disease; gastro-intestinal problems; stasis dermatitis; polyarthritis; appendectomy; radiotherapy; autoimmune disease; hyperthyroidism; cardiovascular system; thyroidectomy; infection; malfunctioning digestive system; hormones; acupuncture; new apartment; side-effects from operation; germs or viruses; chlamydia; animals/germs are changing; since contracted scabies on a holiday in Costa-Rica; Epstein-Barr virus; climate; wind;
Health behaviour	Diet/eating habits; alcohol; lack of exercise; nicotine;
'No idea'	Don't know; no idea;

The level of agreement between raters was good (Cohen's kappa = 0.89). In the case of discrepancies, the latter were discussed until agreement was reached. While 22 patients (9%) exclusively mentioned (a) psychosocial cause(s) to explain their symptoms (psychosocial attribution style), 116 patients (48%) put forward purely somatic explanations (somatic attribution style). One in ten (n=25) presented more of a mixed picture, making both

psychosocial and somatic causal attributions (mixed attribution style). 81 patients (33%) said that they had ‘no idea’ as to what could be causing their symptoms.

4.3.1 Attribution style: between group differences

Age. A one-way analysis of variance revealed no difference in attribution style according to age ($F(3,240)=1.67$, $p=0.17$).

Sex. Further, men and women did not differ in their attribution style ($X^2=1.74$, $df=3$, $p=0.63$).

SFD. The average number of spontaneously mentioned causes was 1.3 (SD 0.7) for SFD, 1.0 (SD 0.7) for NoSFD, and 1.2 (SD 0.6) for VIT patients, with no significant group differences ($F(2,160)=2.56$, $p=0.08$). The three sample groups (SFD, NoSFD, VIT) significantly differed in their attribution styles ($F=17.66$, $p=0.007$). Two sample group comparisons revealed that these differences existed between both work-up groups (SFD, NoSFD) and VIT patients ($F=10.38$, $p=0.01$; $F=13.78$, $p=0.003$, respectively), but not between SFD and NoSFD patients ($X^2=3.73$, $df=3$, $p=0.29$). SFD patients were no more likely than their non somatoform counterparts (NoSFD) to focus on somatic explanations for their symptoms. They were just as likely to advance psychosocial and/or mixed causes. Nevertheless, the somatic attribution style was the most common among both work-up groups (see Table 9).

Table 9. SFD and attribution style (spontaneous) (n=244)

	Work-up group *(n=197)*		***VIT*** [b,c] *(n=47)*	
	SFD [b] *(n=48)*	***NoSFD*** [c] *(n=149)*		*p*
Attribution style	*n (%)*	*n (%)*	*n (%)*	
Somatic	26 (54.2)	61 (40.9)	29 (61.7)	0.007 [a]
Mixed	7 (14.6)	18 (12.1)	-	
Psychosocial	4 (8.3)	17 (11.4)	1 (2.1)	
„no idea“	11 (22.9)	53 (35.6)	17 (36.2)	

Note: a. p-value of the Fisher exact test.
[b,c] denote pairs of groups different at the 0.05 level.

Psychopathology. With numbers for the various categorical PHQ diagnoses being rather small, I limited my analysis of the relationship between comorbid psychiatric disorders and attribution style to analyses of “any psychiatric disorder according to the PHQ” and the dimensional measures of depression and anxiety. I found no association between overall psychiatric comorbidity and attribution style (F=3.18, p=0.37). Further, no association existed between attribution style and the PHQ-9 depression score (H=1.48, df=3, p=0.69) or the GAD-7 score for generalised anxiety (H=4.64, df=3, p=0.20).

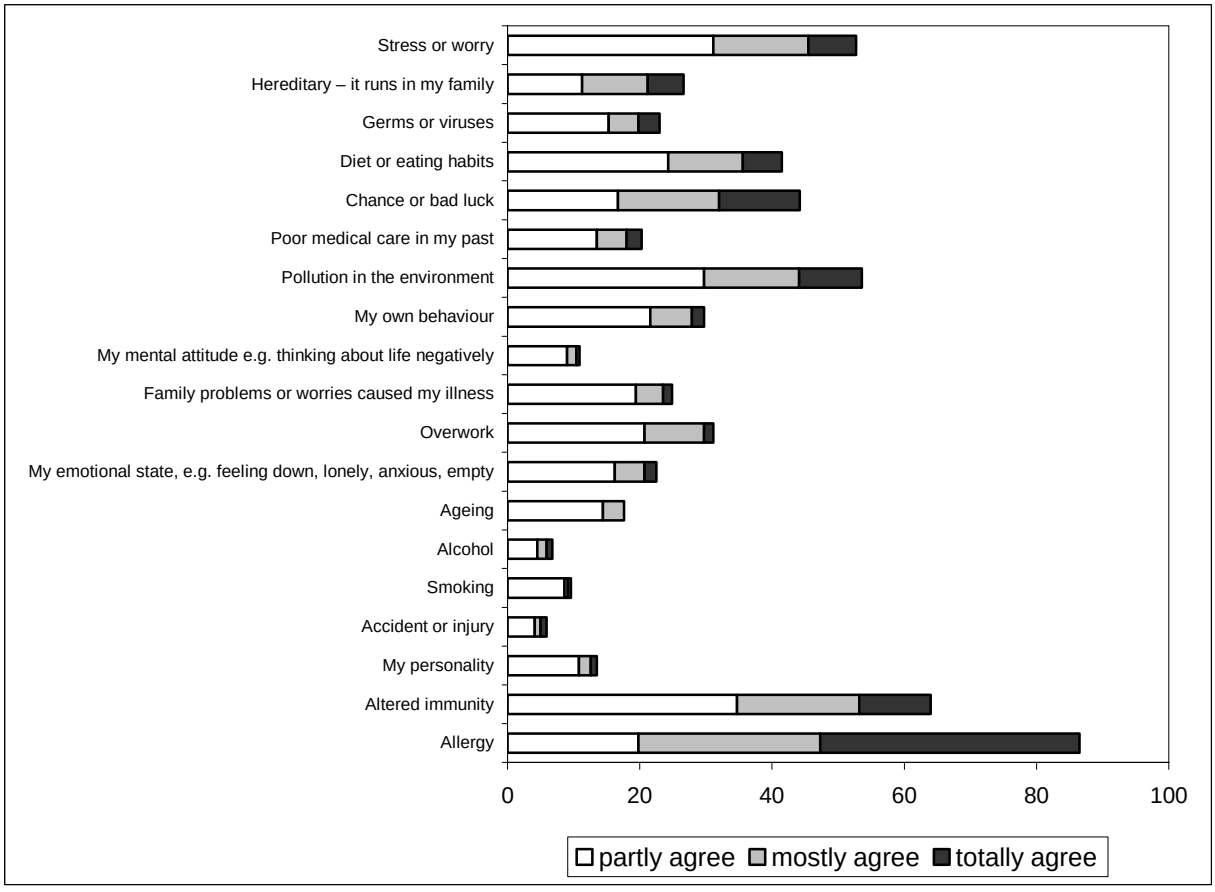
4.4 Causal attribution according to the IPQ-R

4.4.1 Endorsement of the IPQ-R causal items

Patients in our sample fully completing the IPQ-R (n=222) endorsed 1297 causes altogether (mean 5.8, SD 3.2). Of those, a mere 2 patients had not endorsed any of the items of the IPQ-R. Most patients (87%) suspected their symptoms to be due to an allergy.

Subsequently, the most frequently endorsed causal beliefs were ‘altered immunity’ (64%), ‘pollution in the environment’ (54%) and ‘stress or worry’ (53%). ‘Accident or injury’ (6%), ‘alcohol’ (7%), ‘smoking’ (10%), ‘my mental attitude’ (11%), ‘my personality’ (14%) and ‘aging’ (18%) were least likely to be seen as possible causes of patients’ symptoms. Interestingly, these latter causes had not been mentioned at all in the free response task. A breakdown into percentages of participants partly, mostly, and fully endorsing individual causal items (i.e. assigning a score of 3, 4 or 5 on the IPQ-R scale) is presented in Figure 2.

Figure 2. Percentage of patients endorsing individual causal items of the IPQ-R (n=222)*



Note: * Patients not fully completing the IPQ-R causal scale were not included in the analyses.

4.4.2 IPQ-R causal items: between group differences (n=222)

Age. Younger participants were more likely to attribute their symptoms to diet or eating habits (z=-2.42, p=0.02). Older patients, on the other hand, were more likely to blame the pollution in the environment (z=-2.09, p=0.04), ageing (z=-4.52, p<0.001), and an altered immunity (z=-2.39, p=0.02) for their symptoms. No age differences were observed for any of the other IPQ-R items.

Sex. Male participants were more likely than their female counterparts to attribute their symptoms to their own behaviour (X^2=4.17, df=1, p=0.04).

SFD. The average number of endorsed items was 6.2 (SD 3.2) for SFD, 6.1 (SD 3.3) for NoSFD, and 4.8 (SD 2.9) for VIT patients, with no significant group differences (F(2,219)=2.65, p=0.07). Table 10 illustrates the extent to which SFD (n=43), NoSFD (n=136) and VIT patients (n=43) endorsed the individual causal attribution items of the IPQ-R. The three groups differed with regard to their subscription to a number of causal items (items 1, 4, 5, 6, 10). Two sample group comparisons revealed however that a difference between SFD and NoSFD patients existed only for item 6. That is, SFD patients were considerably more likely than NoSFD patients to view 'poor medical care in the past' as a possible cause of their symptoms (X^2=5.39, df=1, p=0.02).

Psychopathology. Patients with significantly higher depressivity and anxiety scores (p< 0.01) attributed their symptoms mainly to psychosocial factors - such as 'stress or worry' (item 1), their 'mental attitude' (item 9), 'family problems or worries' (item 10), 'overwork' (item 11), their 'emotional state' (item 12), and their 'personality' (item 17) - , and their own behaviour pattern - such as 'diet and eating habits' (item 4).

Table 10. Comparison of causal items endorsed on the IPQ-R by SFD, NoSFD and VIT patients (n=222*)

	Work-up group *(n=179)*		***VIT*** *(n=43)*	
	SFD *(n=43)*	***NoSFD*** *(n=136)*		***p***
IPQ-R causal items	*n (%)*	*n (%)*	*n (%)*	
1. Stress or worry	26 (60.5) [a]	80 (58.8) [b]	11 (25.6) [a,b]	<0.001
2. Hereditary – it runs in my family	10 (23.3)	39 (28.7)	10 (23.3)	0.67
3. Germs or viruses	13 (30.2)	33 (24.3)	5 (11.6)	0.10
4. Diet or eating habits	22 (51.2) [a]	63 (46.3) [b]	7 (16.3) [a,b]	0.001
5. Chance or bad luck	16 (37.2) [a]	53 (39.0) [b]	29 (67.4) [a,b]	0.003
6. Poor medical care in my past	**16 (37.2) [a]**	**27 (19.9) [a,b]**	**2 (4.7) [a,b]**	**0.001**
7. Pollution in the environment	22 (51.2)	69 (50.7)	28 (65.1)	0.24
8. My own behaviour	13 (30.2)	45 (33.1)	8 (18.8)	0.19
9. My mental attitude e.g. thinking about life negatively	4 (9.3)	16 (11.8)	4 (9.3)	0.88**
10. Family problems or worries caused my illness	11 (25.6) [a]	40 (29.4) [b]	4 (9.3) [a,b]	0.03
11. Overwork	15 (34.9)	46 (33.8)	8 (18.6)	0.14
12. My emotional state, e.g. feeling down, lonely, anxious,empty	11 (25.6) [a]	35 (25.7) [b]	4 (9.3) [a,b]	0.07
13. Ageing	8 (18.6)	25 (18.4)	6 (14.0)	0.79
14. Alcohol	1 (2.3)	12 (8.8)	2 (4.7)	0.34**
15. Smoking	3 (7.0)	13 (9.6)	5 (11.6)	0.77**
16. Accident or injury	4 (9.3)	8 (5.9)	1 (2.3)	0.43**
17. My personality	6 (14.0)	20 (14.7)	4 (9.3)	0.66
18. Altered immunity	28 (65.1)	85 (62.5)	29 (67.4)	0.83
19. Allergy	37 (86.0)	114 (83.8)	41 (95.3)	0.16

Note: * Analyses only include patients who fully completed the IPQ-R causal scale.
** p-value (two-tailed significance) of the Fisher exact test
[a,b] denote pairs of groups different at the 0.05 level X^2-test

4.4.3 Discovering the underlying dimensions of attribution: Factor analysis of the IPQ-R causal scale

Exploratory Factor Analysis of the IPQ-R causal items, followed by oblique rotation and retaining factors with eigenvalues greater than 1.0, did not yield a structure corresponding to the four subscales proposed by Moss-Morris and colleagues. Therefore, I

subsequently undertook a number of fixed-factor analyses. A five-factor solution, accounting for 56.4% of the variance, best fit the data. That is, it presented the 'cleanest' factor structure, with item loadings above .4, few item cross loadings and no factors with fewer than 2 items. Factor loadings for individual items are presented in Table 11 (see Appendix 4 for further details). Inspection of the factors revealed that the first factor, accounting for 27.4% of the variance, corresponded to the six psychosocial attribution items (items 1, 9, 10, 11, 12 and 17 of the IPQ-R causal scale) identified by Moss-Morris and colleagues. Factor II, accounting for 9.9% of the variance, included items such as 'poor medical care in the past', 'ageing', 'alcohol', 'smoking', and 'accident or injury'. These items had been labeled risk factors by Moss-Morris and colleagues. The third factor accounted for 7% of the variance and included four items, namely, 'germs or viruses', 'diet or eating habits', 'chance or bad luck' and 'my own behaviour'. The fourth factor accounted for 6.1% of the variance and included the items 'hereditary-it runs in my family' and 'altered immunity'. The final factor, accounting for 5.9% of the variance, regrouped the items 'pollution in the environment' and 'allergy'. Cronbach's alpha for the psychological attributions was .871[15]. For the other factors, its value was unsatisfactory, ranging from .590 to .218. Further, correlations between items loading onto these factors were low (ranging from .10 to .24), suggesting that they did not form reliable sets of items. Finally, content exploration of factors III to IV did not reveal a meaningful categorisation of causal attribution items. In line with the analysis of the qualitative data, I thus proceeded to classify the IPQ-R items into psychosocial (Factor I items) and somatic causal attributions (all remaining items) – although factor analysis did not show these latter items to be forming a single construct.

[15] Cronbach's alpha is a measure of internal consistency reliability, that is, of how closely related a set of items are as a group. An alpha of 0.7 is normally considered to indicate a reliable set of items (Kline, 2002).

Table 11. Exploratory factor analysis of the IPQ-R causal items (n=222)

	Causal attribution items	*Factor I*	*Factor II*	*Factor III*	*Factor IV*	*Factor V*
		(α=.871)	*(α=.590)*	*(α=.218)*	*(α=.244)*	*(α=.272)*
	Eigenvalue	*5.19*	*1.89*	*1.34*	*1.16*	*1.12*
	% of variance accounted for	*27.4*	*9.9*	*7.0*	*6.1*	*5.9*
Item 1	Stress or worry	**.750**				
Item 9	My mental attitude e.g. thinking about life negatively	**.591**				
Item 10	Family problems or worries caused my illness	**.821**				
Item 11	Overwork	**.885**				
Item 12	My emotional state, e.g. feeling down, lonely, anxious, empty	**.848**				
Item 17	My personality	**.624**				
Item 6	Poor medical care in my past		**.546**			
Item 13	Ageing		**.608**			
Item 14	Alcohol		**.547**			
Item 15	Smoking		**.647**			
Item 16	Accident or injury		**.667**			
Item 3	Germs or viruses			**-.468**		
Item 4	Diet or eating habits			**-.630**		
Item 5	Chance or bad luck			**.664**		
Item 8	My own behaviour			**.426**		
Item 2	Hereditary – it runs in the family				**.633**	
Item 18	Altered immunity				**.499**	
Item 7	Pollution in the environment					**.534**
Item 19	Allergy					**.802**

4.4.4 Attribution style and between group differences

Of the 1297 items endorsed altogether, 349 (mean 1.5, SD 1.8) were psychosocial and 950 (mean 4.3, SD 2.1) were somatic attribution items. The following analyses only include patients who fully completed the IPQ-R and endorsed at least one of the items on the list (n=220). None of the patients displayed a purely psychosocial attribution style, that is, endorsed psychosocial attributions only. 44% (n=97) endorsed only somatic attribution items

(somatic attribution style). The majority (56%, n=123) exhibited a mixed attribution style, endorsing both psychosocial and somatic causal attributions.

Age. As in the case of the spontaneous mentions, there was no association between attribution style and age ($F(45,174)=1.17$, $p=0.23$)

Sex. Further, there were no differences between men and women in their attribution styles based on the IPQ-R ($X^2=0.45$, df=1, $p=0.50$).

SFD. The three sample groups (SFD, NoSFD, VIT) significantly differed in their attribution style ($X^2=14.36$, df=2, $p=0.001$). Two sample group comparisons revealed that these differences existed between work-up group (SFD, NoSFD) and VIT patients, but not between SFD and NoSFD patients (Table 12). VIT patients were more likely to exhibit a somatic attribution style. SFD patients were no more likely than NoSFD patients to focus on somatic explanations for their symptoms (Chi2=0.07, df=1, p=0.79). They were just as likely as NoSFD patients to display a mixed attribution style. In fact, in both work-up groups the mixed attribution style was the most prevalent. Analyses with the item 'allergy' deleted produced similar results (details not shown).

Table 12. Attribution style according to the IPQ-R (n=220*)

	SFD *(n=43)*	***NoSFD*** *(n=134)*	***VIT*** *(n=43)*
IPQ-R attribution style	*n (%)*	*n (%)*	*n (%)*
Somatic	17 (39.5) [a]	50 (37.3) [b]	30 (69.8) [a,b]
Mixed	26 (60.5) [a]	84 (62.7) [b]	13 (30.2) [a,b]
Psychosocial	-	-	-

Note: [a, b] denote pairs of groups different at the 0.05 level X^2-test

* Analysis only includes patients who fully completed the IPQ-R causal scale and endorsed at least one of the items on the list; two patients had not endorsed any of the items and were thus excluded from the analysis.

Psychopathology. Patients with a comorbid psychiatric diagnosis (as assessed by means of the PHQ-D) were significantly more likely to exhibit a mixed attribution style than those without such a diagnosis. The latter were more likely to exhibit a somatic attribution style (X^2=5.25, df=1, p=0.03).

According to their answers to the IPQ-R, patients exhibiting a mixed attribution style scored higher on both depression and generalised anxiety than patients with a somatic attribution style (mean=4.31, SD=2.87; mean=2.99, SD=2.50, respectively). The results of a Kruskal–Wallis test were significant for both depression (H=15.21, df=1, p<0.001) and generalised anxiety scores (H=13.99, df=1, p<0.001). The mean ranks of scores were higher among patients with a mixed as compared to those with a somatic attribution style.

4.4.5 Comparison of causal attributions and attribution style in the free response task and on the IPQ-R

As already outlined above, when asked to spontaneously provide an explanation for their symptoms, 163 (out of 244) patients cited 234 causes altogether (other than 'no idea'). When choosing from a given list of causal attributions, 1297 causes were endorsed by 222 patients fully completing the IPQ-R causal scale. Overall, 62% of spontaneous mentions corresponded to a particular IPQ-R item. The majority of these spontaneous items (85%) were subsequently also endorsed on the IPQ-R causal scale. Details for each of the 19 causal attribution items of the IPQ-R are provided in Table 13.

Table 13. Comparison of causal attribution items in the free response task and on the IPQ-R

IPQ-R causal items	***Spontaneous causal attributions classified according to an IPQ-R item*** (n=244) *n (%)*	***Spontaneous items subsequently endorsed on the IPQ-R*** (n=222) [d] n^{spont}/n^{IPQ-R} *(%)*	***Endorsement of items on the IPQ-R*** (n=222) *n (%)*
Stress or worry	38 (16.2) [a]	30/34	117 (9.0)
Hereditary – it runs in my family	22 (9.4)	18/22	59 (4.6)
Germs or viruses	5 (2.1)	2/4	41 (3.2)
Diet or eating habits	5 (2.1)	4/5	92 (7.1)
Chance or bad luck	-	-	98 (7.6)
Poor medical care in my past	2 (0.9)	2/2	45 (3.5)
Pollution in the environment	17 (7.3) [a]	13/13	119 (9.2)
My own behaviour	2 (0.9)	0/2	66 (5.1)
My mental attitude e.g. thinking about life negatively	-	-	24 (1.9)
Family problems or worries caused my illness	3 (1.3)	2/2	55 (4.2)
Overwork	2 (0.9)	2/2	69 (5.3)
My emotional state, e.g. feeling down, lonely, anxious, empty	5 (2.1)	3/4	50 (3.9)
Ageing	-	-	39 (3.0)
Alcohol	-	-	15 (1.2)
Smoking	-	-	21 (1.6)
Accident or injury	-	-	13 (1.0)
My personality	-	-	30 (2.3)
Altered immunity	11 (4.7)	10/10	142 (11.0)
Allergy	33 (14.1)	26/31	192 (14.8)
Subtotal (classified):	*145 (62.0)* [c]	*112/131 (85)*	*1297 (100)*
Unclassified:			
Medication	28 (11.5)	-	
Chemicals/harmful substances	16 (6.5) [b]	-	
History of organic illness(es)	15 (6.1)	-	
Disposition	12 (4.9)	-	
Hormones	3 (1.2)	-	
Other	9 (3.7)	-	
Subtotal (unclassified):	*83 (35.5)* [c]	-	
Total spontaneous mentions	234 (100)		
Number of patients saying 'No idea'	81	n/a	n/a

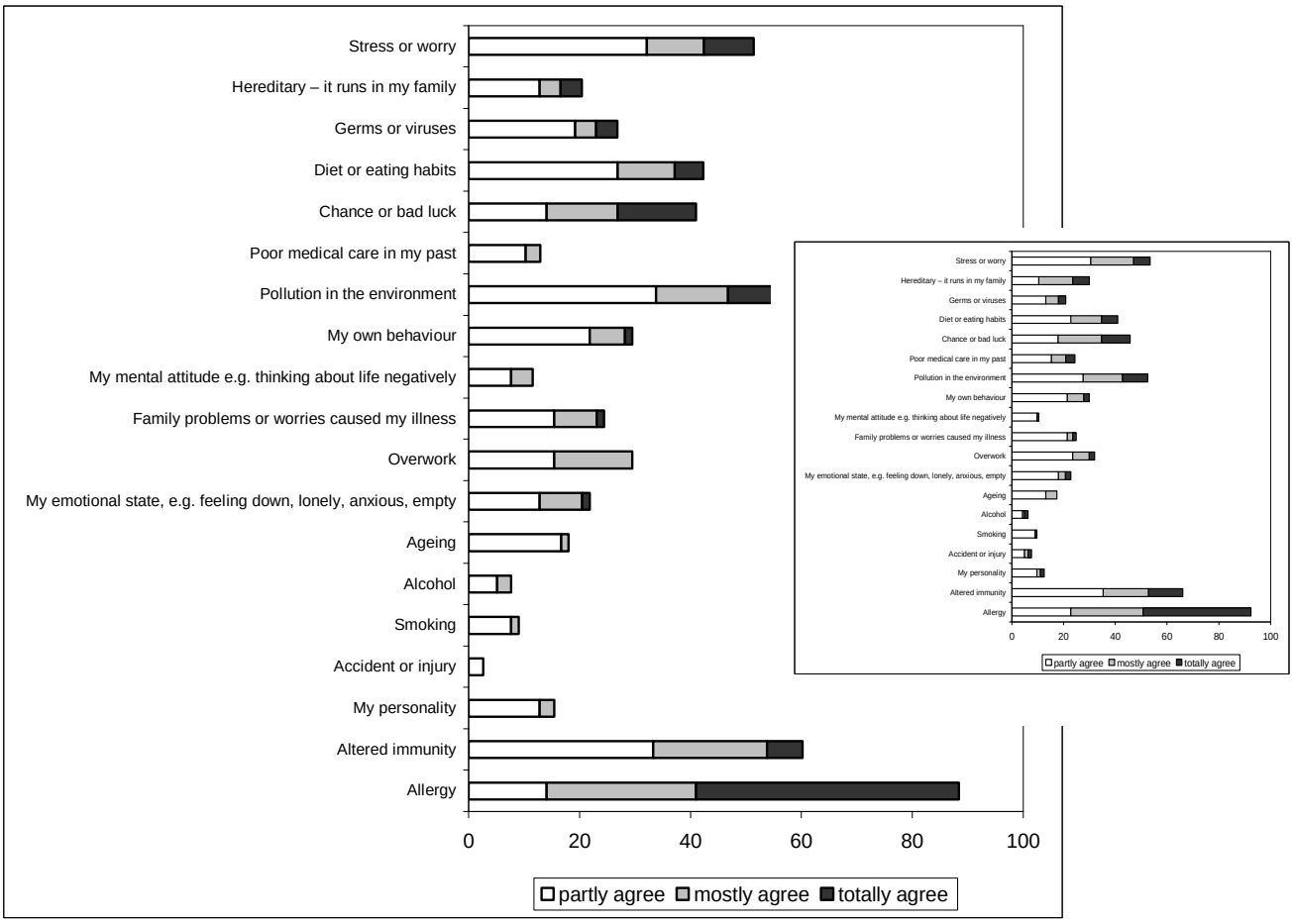
Note: a. One patient made two statements that were examples of the same IPQ-R item (counted only once).
b. Three patients made two open statements that were examples of the same IPQ-R item.
c. These totals do not add up to 100% as for some patients, multiple statements were examples of the same IPQ-R item (see a. and b.).
d. Where one or more causal items of the IPQ-R scale were not rated, the case was not included in the analysis.

About one third of spontaneous mentions (35.5%[16]) were not classifiable under an IPQ-R causal item. Most frequently, these referred to various kinds of medication (11.5%; mainly antibiotics)[17] and chemicals/harmful substances (6.5%). Responses included in the latter category were: 'disinfectant', 'chlorine', 'amalgam', 'palladium', 'additives', 'dye', 'nickel', 'chemicals', 'flat/lodging'. I considered these to be sufficiently different from the IPQ-R item 'pollution in the environment', which refers to outdoor substances, to warrant a separate category. The category 'history of organic illnesses' is self-explanatory. The grouping termed 'disposition' comprises rather vague statements referring to patients' 'reduced general condition', 'hypersensitivity' and 'susceptibility'. Finally, the 'other' category includes a number of disparate causal attributions such as 'acupuncture, 'the sun', 'the climate', 'too much wind', 'curse', and 'too many insect bites'. Further, some causal attribution items of the IPQ-R (namely, 'accident or injury', 'alcohol', 'my mental attitude', 'my personality', and 'aging'), had not been mentioned in the free response task at all. On the IPQ-R, these items were least likely to have been endorsed (see Table 10 and 13, above).

Patients who had reported that they had 'no idea' as to what was causing their symptoms in the free response task (n=81), subsequently exhibited a pattern of individual causal attribution on the IPQ-R (see Figure 2, below) similar to that of the other patients (n=148, inset figure). The majority (55.8%) of the 'no idea' patients displayed a mixed attribution style (Table 14).

[16] The percentages of spontaneous mentions corresponding to an IPQ-R item (62%) and of those not corresponding to an IPQ-R item (36%) do not add up to a total of 100% as some statements made in the free-response task were considered to be examples of the same IPQ-R item.
[17] Vaccination, mentioned by two patients, was also included in this category.

Figure 2. IPQ-R causal attribution items endorsed by 'no idea' patients (n=81)



About half of SFD (52.2%) and NoSFD (44.4%) patients exhibiting a purely somatic attribution style in the free response task, shifted to a multi-dimensional, mixed attribution style, when presented with a predetermined list of causal attribution items (Table 14). Further, half of SFD patients and all NoSFD patients providing purely psychosocial explanations for their symptoms in the free response task, subsequently moved to a mixed attribution style. A minority of patients (14.3% of SFD, 6.3% of NoSFD patients) shifted from a multi-dimensional to a purely somatic attribution style.

Table 14. Attribution style: Free response task versus IPQ-R (n=220)

IPQ-R		Somatic	Mixed
Spontaneous		*n (%)*	*n (%)*
Somatic	*SFD (n=23)*	11 (47.8)	12 (52.2)
	NoSFD (n=54)	30 (55.6)	24 (44.4)
	VIT (n=25)	18 (72.0)	7 (28.0)
	Total (n=102)	59 (57.8)	43 (42.2)
Mixed	*SFD (n=7)*	1 (14.3)	6 (85.7)
	NoSFD (n=16)	1 (6.3)	15 (93.8)
	VIT (n=0)	-	-
	Total (n=23)	2 (8.7)	21 (91.3)
Psychosocial	*SFD (n=4)*	2 (50.0)	2 (50.0)
	NoSFD (n=13)	-	13 (100.0)
	VIT (n=1)	-	1 (100.0)
	Total (n=18)	2 (11.1)	16 (88.9)
'No idea'	*SFD (n=9)*	3 (33.3)	6 (66.7)
	NoSFD (n=51)	19 (37.3)	32 (62.7)
	VIT (n=17)	12 (70.6)	5 (29.4)
	Total (n=77)	34 (42.2)	43 (55.8)

5 Discussion

5.1 Somatic illness attribution revisited

The results of this study (see also, Groben & Hausteiner, 2011) challenge the assumption that SFD patients hold on to a purely somatic explanatory model. In both the qualitative and quantitative measures of causal illness attribution, SFD patients were no more likely than NoSFD patients to focus on somatic explanations for their symptoms. They were just as likely to advance psychosocial or mixed causes. VIT patients, who had an established diagnosis of hymenoptera venom allergy, exhibited a predominantly somatic attribution style. These findings are consistent with recent qualitative research suggesting that SFD patients do incorporate psychosocial factors in their explanations for their symptoms when they are encouraged to do so (Hiller et al., 2010; Peters et al., 2009; Ring et al., 2005; Salmon et al., 2004, 2009). Furthermore, both in the free response task and on the IPQ-R, the majority of work-up patients (SFD, NoSFD) endorsed multiple illness attributions, including psychosocial ones. This confirms the view that symptom attribution is a multidimensional process, with patients holding multifaceted and coexisting explanations for their symptom(s) (Hiller et al., 2010, Rief et al., 2004; Rief & Broadbent, 2007; Ring et al., 2005; Risør, 2009). The similarity in attribution styles and the predominance of mixed attributions among both SFD and NoSFD patients further illustrate that patients are generally open to a bio-psycho-social illness model, in particular in a work-up situation.

In addition, the shift between attribution styles confirms the dynamic nature of the attribution process. That is, a large number of patients shifted from a purely somatic or psychosocial attribution style exhibited in the free response task to a mixed attribution style when presented with the IPQ-R causal scale items. According to Leventhal (1984), the

search for the meaning of an illness is a process characterized by movement and uncertainty: in seeking meaning for the symptoms they experience, patients may hold on to a variety of explanations simultaneously, or may move from one belief to another.

Some people may argue that the above results could be due to a bias in sampling. The study sample consisted of work-up patients hospitalised for allergy testing and whose symptoms could not be diagnosed with sufficient certainty in an outpatient setting. There is wide agreement in social psychology that the search for causes is prompted by negative or unexpected events, or in 'situations with high levels of uncertainty' (Sensky, 1997, p.566). Therefore, the psychological burden of the work-up situation may have been influencing the attributions of both SFD and NoSFD patients (Groben & Hausteiner, 2011; Hausteiner et al., 2009).

In addition, as the IPQ-R was embedded in a batch of self-report instruments, exploring health related cognitive, affective, and behavioral characteristics, this may have contributed to some patients' disclosure of psychosocial causal attributions. On the other hand, the predominance of somatic attribution items in the IPQ-R may have influenced patients first exhibiting a purely psychosocial attribution style to also endorse one or more somatic causal attributions.

Risør (2009) has shown that causal illness attributions are dependent on the social situation, and on whom the 'illness story' is being told to: That is, while in clinical encounters, patients tended to focus on somatic illness attributions, these were by no means the only explanations of significance to them. In fact, where a safe place was provided in which patients felt listened to and understood, they have been shown to be able to disclose psychological factors (Buszewicz et al., 2006; Kirmayer et al., 2004; Martin et al., 2007a; Schroter et al., 2004). The fact that our interviewers had made it very clear that they were not members of hospital staff and that the interview was not to be seen as a medical

consultation, undoubtedly created an atmosphere in which a discourse about the patients' thoughts could more freely develop. Barriers to patient disclosure of emotional issues generally found in clinical consultations (Peters et al., 2009) may thus have been overcome.

Further, time of interview undoubtedly influenced our results. Thoughts about the etiology of an illness have been shown to vary during its course, with patients tending to rely on somatic explanations in the early stages of medically unexplained symptoms (Risør , 2009), the interaction with significant others influencing the development of these thoughts (Helman, 1985). Our patients have had a relatively long history of symptoms (8.8 and 5.9 years for our SFD and NoSFD patients, respectively, see Table 1) and therefore ample opportunity of structuring and re-structuring their etiological thoughts. This may have led them to consider more sophisticated and psychosocial causal attributions (Korn, 2003). On the contrary, other studies have described the somatising effect of the doctor-patient interaction (Ring et al., 2005, Salmon et al., 2005). According to these studies, and given our patients' long history of symptoms, one would have expected our patients to focus on somatic causal attributions. Longitudinal studies looking at the development of causal attributions over time may be able to shed more light on this issue.

Most importantly, given that comorbidity with psychiatric disorders has generally been reported to be associated with psychosocial illness attributions among SFD patients (see section 1.2.4.3, above, see also section 5.3., below), psychiatric comorbidity is likely to have influenced causal attribution among our SFD patients. In fact, in line with previous studies (see section 1.1.3, above) our SFD patients were significantly more likely to be diagnosed with a psychiatric disorder than NoSFD and VIT patients. Further, they scored higher on continuous measures of depression and anxiety. However, given the considerable syndrome overlap of somatisation, depression, and anxiety (Hanel et al., 2009; Henningsen et al., 2003; Löwe et al., 2008) and the view of the latter as essential dimensions of SFDs, the above does not detract from the importance of our results.

5.2 Causal attribution and sex

Studies exploring sex differences with regard to SFD patients' causal attributions are rare. Those that do (Korn, 2003; Nykvist et al., 2002; Robbins & Kirmayer, 1991) have found women to be more likely to consider psychosocial explanations for their symptoms than men. The results presented here do not corroborate these findings. Both in the free response task and with regard to the IPQ-R, men and women did not differ in their attribution style. Merely when looking for group differences at the level of individual causal IPQ-R items, were male participants found to be more likely than their female counterparts to attribute their symptoms to their own behaviour ($p<0.05$). There were no sex differences with regard to any other causal attribution item of the IPQ-R.

The abovementioned studies assessed causal attributions and found sex differences with regard to specific individual symptoms – such as headaches and back pains (Korn, 2003), neck/shoulder problems and a sore/upset stomach (Nykvist et al., 2002) - rather than globally for all symptoms experienced by a particular patient. Thus, sex differences may only emerge when separately assessing individual symptoms. On the other hand, as has been mentioned elsewhere (Barsky et al., 2001), 'since positive findings are more likely to find their way into the literature than negative ones, gender differences are more likely to be emphasised and published than the absence of such differences' (p.270).

5.3 Causal attribution and psychopathology

As mentioned above, and in line with previous studies, our SFD patients were considerably more likely to be diagnosed with a psychiatric disorder than NoSFD and VIT patients. Further, they scored higher on continuous measures of depression and anxiety. In

fact, the considerable overlap of SFD, depression and anxiety precludes a view of these disorders as discrete nosological entities (see section 1.1.3., above). Furthermore, the high rate of psychiatric comorbidity among SFD patients seems to preclude a view of SFD patients as generally focusing on somatic causal attributions.

Comorbidity with psychiatric disorders, in particular depression and anxiety, has been reported to be associated with psychological illness attributions. Both studies eliciting causal illness attributions quantitatively, i.e. by means of a list of predefined causes (Hiller et al., 2010; Rief et al., 2004; Robbins & Kirmayer, 1991), and studies based on spontaneously reported causal illness attributions (Henningsen et al., 2005; Hiller et al., 2010; MacLeod et al., 1998; Martin et al., 2007a) have shown a tendency towards psychosocial and/or mixed causal attributions in SFD patients to be associated with concurrent depression and anxiety disorders and/or severity.

As expected, results from the present study confirmed this observation. That is, they revealed a significant positive relationship between a mixed attribution style and depression and anxiety scores, as well as psychiatric comorbidity assessed categorically. While this relationship was observed with regard to causal attribution assessed by means of the IPQ-R, surprisingly, no such relationship was found with regard to spontaneously reported causal attributions. The reason for this discrepancy may be that the rather large group of patients (constituting one third of the total sample) who mentioned that they had ‘no idea’ as to what was causing their symptoms in the free response task did not enter these analyses. These ‘no idea’ patients constituted almost half (45%, n=14) of those patients diagnosed with a comorbid psychiatric disorder (n=31). And the majority of them (56%) subsequently exhibited a mixed attribution style when presented with a predetermined list of causal attributions.

The studies carried out by Rief et al. (2004) and Henningsen et al. (2005) compared the causal illness attributions of SFD patients with concurrent psychiatric diagnoses and

those of so-called ‘pure’ SFD patients. Due to the limited number of SFD patients in our study population, no such analysis was attempted. And as has been mentioned by Hiller et al. (2010), given the large comorbidity rates of SFD and depression, for example, the group of ‘pure’ SFD patients can be expected to be rather small. Consequently, ‘relatively large samples would … be needed to examine whether the attributional pattern of pure SFD differs to that of comorbid SFD’ (p.17).

5.4 Spontaneous and prompted causal attributions

As expected, and in line with previous findings (Cebulla, 2002; Hiller et al., 2010; Korn, 2003), a much higher number of causal attributions was endorsed on the predetermined list of causal attributions of the IPQ-R causal scale than was spontaneously mentioned in response to the open-ended question. It has been argued (Sensky, 1997) that attributions elicited by means of a predetermined list ‘may have little in common with spontaneous attributions, which are much more likely to be clinically relevant’ (p. 570). In fact, while our patients’ spontaneous causal attributions corresponded well to their subsequent answers on the IPQ-R, the relevance of the IPQ-R items to the study population seemed limited, with more than one third of spontaneous mentions not classifiable under an IPQ-R causal item. This suggests that for future use of the IPQ-R with a population in an allergy setting, the item list should be extended so as to include items more specific to this population (see unclassified items in Table 13, above). Merely adding the item ‘allergy’ to the items listed in the IPQ-R did not do justice to our study population. While the causal attribution item ‘allergy’ was the item endorsed most often (namely, by 87% of our patients), this is likely to have been due to the face validity of this item when presented to patients attending an allergy clinic, and asked about the causes of their ‘intolerances’.

Conversely, it is important to note that patients who had spontaneously reported that they had 'no idea' as to what was causing their symptoms, had later on exhibited a pattern of causal attribution similar to that of the other patients. Therefore, the use of a questionnaire could be a useful *complementary* tool in clinical consultations: it may help to engage some patients in further discussions about their symptoms. According to Faller et al. (1991, cited in Korn, 2003, p.111), the higher number of causal attributions elicited by means of a questionnaire may be the result of a thinking process triggered by the predetermined list of attributions.

5.5 Factor analysis of the IPQ-R causal scale

The Exploratory Factor Analysis of the IPQ-R causal items did not confirm the factor structure proposed by Moss-Morris and colleagues and validated for eight organically defined illness groups (Moss-Morris et al., 2002). These authors identified four attributional factors, namely, psychological, risk factor, immune attributions and accident or chance (see table 4, p. 31). Similar to Gaab et al. (2004), Hagger & Orbell (2005) and Wittkowski et al. (2008) I found some support for the psychological attribution and risk factor dimensions. The immune and chance attribution subscales did not appear to be stable. Therefore, apart from the psychological and risk attributions, the causal subscales of the IPQ-R have to be treated with caution as far as our patient group is concerned.

Both the IPQ-R and its predecessor, the IPQ, have been widely used in research involving studies of patients with a range of medical (Hagger & Orbell, 2005; Knibb & Horton, 2008; Wittkowski et al., 2008) and mental health conditions (Fortune et al., 2004), including somatoform disorders (Gaab et al., 2004; Moss-Morris & Chalder, 2003; Rief et al., 2004; Van Wilgen et al., 2008). However, only some studies have examined the psychometric

properties and/or factorial validity of the IPQ/IPQ-R scales. Rief et al. (2004) examined the factorial validity of the IPQ causal scale in a sample of patients with unexplained physical symptoms. Gaab et al. (2004) evaluated and validated the psychometric properties of the IPQ-R in patients with various somatoform illnesses (such as chronic fatigue syndrome, fibromyalgia, chronic whiplash syndrome, and temporomandibular disorder). However, they could not replicate the factor structure of the IPQ-R causal scale as proposed by Moss-Morris and colleagues. Similarly, in a cervical screening and dermatological (atopic dermatitis) context, two notable psychometric studies could provide some confirmatory evidence of the IPQ-R structure (Hagger & Orbell, 2005; Wittkowski et al., 2008). Again, the factor structure of the IPQ-R causal scale could not be replicated.

The failure to replicate the factor structure of the IPQ-R causal scale in the present study is most probably due to differences in sample size and the nature of the population studied (Gaab et al., 2004). In fact, discrepancies between causal attributions mentioned in the free response task and the items included in the IPQ-R (see sections 4.4.5 and 5.5, above) suggest that the relevance of the IPQ-R items to our study population was limited. Indeed, Moss-Morris et al. (2002) suggested adapting the IPQ-R causal scale items according to the condition and population in question. Causal attributions seem to vary between different patient groups (see e.g. Gaab et al., 2004) and have even been shown to differ depending on the symptom under consideration (e.g. Korn, 2003, Hiller et al., 2010).

Further investigations into the psychometric properties of the IPQ-R causal scale when used with an allergy and a SFD population are warranted.

5.6 Illness attributed to poor medical care in the past

In this study, SFD patients were considerably more likely than their NoSFD counterparts to attribute their illness to ‘poor medical care in my past’. In fact, this was the only causal illness attribution differentiating SFD from NoSFD and VIT patients. This finding points to a remarkable discontentment with former medical care among SFD patients. Indeed, assessing the health attitudes of somatising patients, the SomA study (in which this thesis was embedded in) found ‘dissatisfaction with care’ to be an important predictor for the existence of a somatoform disorder (Hausteiner et al., 2009). Earlier studies (Dirkzwager & Verhaak, 2007; Hasler et al., 2004; Noyes et al., 1999) had already pointed towards a major discontentment with medical care among SFD patients. For example, a Dutch study of patients with medically unexplained physical symptoms (MUPS) found that the latter ‘more often felt that they were not taken seriously or not involved in treatment decisions, and more often reported that the GP did not take sufficient time’ as compared to their non-MUPS counterparts (Dirkzwager & Verhaak, 2007, p. 1).

Although the present study has not looked into the reasons for patients’ dissatisfaction with medical care, they may in some cases have involved disagreement over attribution and the legitimacy of patients’ symptoms (Salmon et al., 2004; Sauer & Eich, 2007). According to Kirmayer et al. (2004, p.670), the ‘failure to find a mutually satisfying explanation frequently reflects a breakdown in the doctor-patient relationship’. Elsewhere, impairment in interpersonal functioning (e.g. in terms of SFD patients’ interaction with medical professionals and care-eliciting behaviour) has been proposed as an important dimension to be looked at (Noyes et al., 2008). Further reasons for dissatisfaction with medical care may be rooted in a health-care system poorly equipped to deal with SFD patients (Henningsen et al., 2011). These authors demand an integrated approach that would overcome the dualistic nature of the healthcare system and bridge the gap between

psychiatric and medical services. Furthermore, just as symptom reduction is associated with increased patient satisfaction (Hasler et a., 2004), the persistence of symptoms leads to disappointment, hope- and helplessness, and henceforward, dissatisfaction with the treating physician (Sauer & Eich, 2007). Therefore, proper assessment, management and treatment of SFDs are called for (Henningsen et al., 2007; Rosendal et al., 2005; Stone et al., 2005a, 2005b).

5.7 Clinical implications: diagnosis and treatment

The main defining feature of SFDs is 'the presence of physical symptoms that suggest a medical condition and are not fully explained by a general medical condition, …, or by another mental disorder' (APA, 2000, p. 445). In the study presented here, I aimed to examine the beliefs people hold about the cause(s) of their symptom(s). In particular, I set out to investigate the extent of somatic causal illness attribution among SFD patients and to assess the possible use of this dimension as a positive criterion in the definition of somatoform disorders, with the long-term view to provide the basis for better diagnostic and therapeutic management.

As has been discussed above, this study has found SFD patients to consider psychological causes, alongside somatic ones, as highly relevant explanations for their symptoms. In addition the findings underline the multi-causal perspective of both SFD and non-somatoform disorder patients, as well as the dynamic nature of the causal attribution process. The data presented here thus contradict the assumption that SFD patients tend to focus on somatic explanations for their symptoms. Therefore, they do not support the use of somatic causal illness attributions as positive criteria in the definition of SFDs. On the other hand, exploring SFD patients' explanations of and giving meaning to their symptoms forms

an important part of the assessment of SFDs, of a 'transparent and collaborative' approach (Stone et al., 2005a), as the basis for a stable therapeutic relationship. In addition, the assessment of causal attribution is important in that it has been shown to influence psychological adjustment to illness and coping behaviour (Roesch & Weiner, 2001).

Interventions with SFD patients have generally been based on the assumption that the latter do not recognise the role of psychosocial factors in their problems. However, evidence for the applicability and effectiveness of so-called reattribution therapy, which consists in encouraging patients to relate their symptoms to psychosocial problems (Goldberg et al., 1989), has remained equivocal (Arnold et al., 2004; Larisch et al., 2004; Morriss et al., 2007; Rief et al., 2006). In fact, cognitive behavioural interventions (Kroenke & Swindle, 2000; Kroenke, 2007; Martin et al., 2007b; Stone et al., 2005b), psychodynamic interpersonal therapy (Sattel et al., 2012), and more generally, improved communication between doctors and patients (Aiarzaguena et al., 2007, Henningsen et al., 2007; Kirmayer et al., 2004; Peters et al., 2009; Rosendal et al., 2005; Salmon et al., 2004) have been shown to be most effective in the management of SFDs.

While reattribution focuses on providing psychosocial explanations for SFD patients' symptoms, numerous studies (including the study presented here) have shown that many patients' explanations for their symptoms already include psychosocial factors. Reattribution interventions therefore seem redundant. As far as reattribution training has been shown to improve communication between doctor and patient (Morriss et al., 2007), such interventions may be important in their own right. However, focusing on changing illness beliefs seems inappropriate (Peters et al., 2009). Given that research on SFDs has as yet not been able to provide clear etiological and pathogenetical models, 'causal assumptions cannot be classified as either right or wrong' (Hiller et al., 2010, p.15).

Patients with somatoform disorders mainly want their doctor to understand and accept their problems, and form an alliance (Buszewicz et al., 2006; Salmon et al., 1999). In this vein, IPT (Interpersonal Psychodynamic Therapy), which emphasises the importance of interpersonal processes and relationships as well as emotional issues in the development and maintenance of somatoform symptoms, seems promising (Sattel et al., 2012). IPT legitimates patients' complaints, providing them with the 'sick role' (Weissman et al., 2000). The therapist is a friendly ally, encouraging and supporting the patient (Cutler et al., 2004). In addition, IPT imparts psychoeducation about somatoform disorders and treatment options, thus relieving the patient of the hopelessness of his/her situation.

There is now growing awareness of the role of doctors' responses in generating and shaping medically unexplained symptoms as well as patients' beliefs, and in encouraging somatisation (Henningsen et al., 2011; Salmon et al., 2007). The crux of the matter seems to lie in the interaction between doctor and patient.

In some instances, patients have been found not to be freely communicating important psychological and social issues to their doctors. Peters et al. (2009) have identified a number of barriers preventing patients from addressing such issues. These include: a lack of trust and feeling uncomfortable about discussing emotional aspects of their problems with their GP; the complexity of their problems and the limited time available during consultations; the perception of GPs' explanations as being too dualistic in nature in the face of the complexity of their problems; the fear that attention to psychosocial factors would preclude consideration of physical complaints; as well as, stigma of psychosocial explanations. Using specific communication techniques aimed at encouraging patients to talk openly about sensitive aspects of their lives, taking into account the above concerns, has been shown to have a significant impact on bodily pain (Aiarzaguena et al., 2007). Even where patients do disclose psychosocial difficulties, GPs have been found not to engage with these cues (Salmon et al., 2004). Instead, they have been found to re-assert the somatic agenda or

normalise the problem – thus indicating that the symptoms needed no further treatment. Elsewhere, Ring et al. (2004, 2005) have shown that doctors propose somatic interventions more often than their somatoform disorder patients, and do so in the face of demands for emotional support. Few GPs empathised with their patients about their symptoms. In turn, this may negatively affect patients' satisfaction with the medical care they receive and quite rightfully lead them to attribute their illness to 'poor medical care received in the past'.

GPs generally feel that patients with medically unexplained symptoms are difficult to manage (Reid et al., 2001b), and they feel ill-equipped to deal with and treat these patients (Wileman et al., 2002). SFD patients' demands for emotional support seem to be particularly challenging (Ring et al., 2004; Salmon et al., 2005). It has been suggested that doctors stick to somatic responses to establish authority in a situation in which they feel powerless and insecure (Wileman et al., 2002), to avoid emotional engagement with these patients (Salmon et al., 2005), and to resist demands for emotional support that they consider to be excessive (Salmon et al., 2007). Encounters have been found to be more likely to be rated as 'difficult' by physicians with poor attitudes towards such patients. Therefore, improving physician training and thereby modifying their attitudes towards patients with MUS has been proposed to improve the care of patients with such complaints (Kroenke, 2003).

In sum, exploring SFD patients' explanations of and giving meaning to their symptoms forms an important part of the assessment and management of SFDs. Focusing on changing illness beliefs seems inappropriate (Peters et al., 2009). Understanding and improving the interaction between doctor and patient, and enhancing doctors' communication skills appears to be more promising.

5.8 Methodological issues and scientific implications

Limitations of the current study include the fact that the latter was carried out in an allergology setting which limits the generalisability of the findings. On the other hand, the naturalistic setting in which the study was carried out marks its clinical relevance, all the more given the high participation rate (of 89%) and the absence of attrition. Further, SFD, NoSFD patients, and controls were well matched for age, sex, socioeconomic variables, number of concurrent somatic diagnoses and duration of symptoms.

The cross-sectional study design does not allow inferences to be made with regard to the causal nature of or the influence of certain events or factors on patients' cognitions. However, correlational statistics led to a good description of the issue under consideration. The cross-sectional design allowed for a positive trade-off between cost and number of patients included in the study, and was deemed suitable for studying a disorder such as SFD, which is characterised by high prevalence rates. Notwithstanding, a longitudinal study design would be more suited to investigate the development and dynamic nature of causal attributions over time. It would allow researchers to shed more light on the influence of medical consultations and treatments as well as other factors on patients' causal attributions, and vice versa.

It may be argued that the categorisation of attributions according to the dualistic psychosocial-somatic divide is problematic (Deary et al., 2007), all the more considering current efforts to get away from the mind-body dichotomy inherent in the prevailing SFD classification. However, this categorisation allowed for the comparison of the qualitative and quantitative measures used in this study, the comparison with previous studies, and for addressing our main research questions. Further, while allowing for a mixed attribution category, patients were not confined to two mutually exclusive attribution styles.

One major criticism that may be leveled at the above results is that of multiple testing. That is, as the number of comparisons increases, it becomes more likely that the groups under investigation will appear to differ on at least one attribute. In other words, the probability of making a type 1 error increases. The overall rate of obtaining significant results by chance may considerably exceed the 0.05 level, so that one may decide that an independent variable (such as sex) had an effect on a dependent variable (such as a particular causal attribution item) when it did not have such an effect. Results pertaining to analyses at the IPQ-R item level were therefore interpreted with care. Further, to provide some control of type 1 error at the level of our main independent variables (SFD, NoSFD, VIT), I followed a closed test procedure in that comparisons across the three sample groups were followed by pair wise comparisons only where significant differences were found.

Various methodological strengths of this study include the diagnosis of somatoform disorders by means of the well established SCID, the current gold-standard for SFD diagnosis. Further, through combining both qualitative and quantitative research methods, the study can draw on the strengths of both while offsetting their weaknesses: in the free response task, patients were allowed to use concepts and categories that were relevant and meaningful to them; the use of a pre-determined list of causal attributions such as the IPQ-R causal scale prevented interviewer-bias. Overall, the combination of both research methods permitted triangulation of our findings, i.e. for them to be double-checked and mutually corroborated: in two thirds of our patients spontaneous causal attributions corresponded well to their subsequent answers on the IPQ-R. In addition, through combining the two approaches, I was able to show the potential usefulness of the IPQ-R causal scale as a complementary tool in clinical consultations, and to identify the multidimensional and changing nature of causal illness attribution. On the other hand, qualitative data obtained from the free response task may help to develop a more comprehensive IPQ-R item list for future use with an allergy population.

Causal attributions have been shown to differ depending on the level of the health care system looked at. As mentioned above, Wessely's team (Euba et al., 1996) found tertiary care patients to be more likely to attribute their symptoms to organic causes while primary care patients were more likely to make psychosocial attributions. Therefore, further studies at various levels of the health care system (e.g. primary care and outpatient clinic) are needed in order to corroborate the high percentage of psychosocial causal attributions found in our sample group.

Finally, a larger sample of SFD patients would allow for a more graded analysis of patients' attributions, in particular with regard to the IPQ-R items that were rated on five-point Likert-type scales. The small number of subjects in each response category precluded such analyses in the present study. A weighted analysis would provide additional information with regard to the importance attached to some attributions compared to others. Similarly, patients attributing their symptoms to a number of causes in the free response task could be asked to weigh their answers (as in the study by Hiller et al., 2010). Further, given a sufficient sample size the causal attributions of SFD patients with and those without a comorbid psychiatric disorder could be compared. In addition, patients' causal attribution style could be analysed separately in relation to various comorbid psychiatric disorders (e.g. depression, anxiety).

6 Conclusions

Contrary to the widespread assumption that SFD patients do not recognize the role of psychosocial factors in the development of their symptoms, patients frequently attributed their symptoms to psychosocial causes, both in the free response task and when presented with a predetermined list of causal attributions. There were no differences between SFD and NoSFD patients in this respect. SFD patients were no more likely than NoSFD patients to focus on somatic explanations for their symptoms. SFD patients did not predominantly tend towards a somatic attribution style. In addition, the majority of work-up patients endorsed multiple illness attributions. No sex differences were observed with regard to attribution, however psychosocial and mixed attributions were clearly associated with comorbid psychiatric disorders as well as heightened depression and anxiety scores. Discontentment with past medical care appeared to be a prominent feature of SFD patients to the point of these patients attributing their symptom(s) to ‘poor medical care received in my past’.

The above results suggest that due to the lack of specificity somatic causal attribution should not be used as a positive criterion for future classifications of SFDs. In fact, since the implementation of this study, preliminary diagnostic criteria for DSM-V developed through a number of working groups so far do not include somatic causal attribution as a defining feature of the new somatoform disorder categories (APA, 2011). However, this does not preclude the importance of addressing causal attributions in the management of SFDs. Our findings confirm the view that the process of illness attribution is a dynamic and multidimensional process, and point to the importance of appropriate medical care and of the doctor-patient interaction.

Various methodological strengths of the study presented here include the use of the SCID, the current gold standard for diagnosing SFDs, the naturalistic research setting and

the combination and comparison of both quantitative and qualitative measures of causal illness attributions. Combining both spontaneous and prompted techniques, I was able to show the potential usefulness of the IPQ-R scale as a complementary tool in a clinical setting. Further, qualitative data obtained from the free response task can help to develop a more comprehensive IPQ-R item list for future use with an allergy population.

Further studies - longitudinal ones, studies at different levels of the health care system, in different settings and with different SFD populations - are needed to shed more light on the development of causal illness attributions over time, so as to be able to determine associated and influencing factors. In addition, further investigations into the psychometric properties of the IPQ-R causal scale when used with an allergy and a SFD population are warranted.

Appendix 1: Patient Information Sheet

Klinik und Poliklinik
für Dermatologie und Allergologie am Biederstein
des Klinikums rechts der Isar
der Technischen Universität München

Direktor: Univ.-Prof. Dr. med. Dr. phil. Johannes Ring

Biedersteiner Straße 29, 80802 München

Klinik und Poliklinik
für Dermatologie und Allergologie
der TUM
Postfach 401 840, 80718
München

Liebe Patientin, lieber Patient,

Sie befinden sich derzeit bei uns zur Abklärung einer Unverträglichkeit bzw. Allergie. Erfahrungsgemäß können sowohl die damit verbundenen Beschwerden als auch die Unsicherheit über mögliche Erklärungen eine erhebliche Belastung darstellen. Wir möchten Ihre Beschwerden, Erfahrungen im Gesundheitswesen, Gedanken übers Kranksein und einige weitere Aspekte kennen lernen, um Allergien/Unverträglichkeiten besser verstehen, einteilen und behandeln zu können. Deshalb befragen wir in der SomA-Studie „Körperliche Beschwerden in der Allergologie“ ein ganzes Jahr lang *alle* Patienten, die sich in der Dermatologischen Klinik am Biederstein zur stationären Allergieabklärung vorstellen, und zwar unabhängig davon, ob diese Allergie am Ende des Aufenthalts nachgewiesen werden kann oder nicht. Durch Ihre Mitarbeit können Sie uns dabei helfen.

Unsere Befragung besteht aus einem ca. 15-30minütigen Interview und einer Reihe von Fragebögen zum Selbstausfüllen, wozu Sie noch einmal ca. 60 Minuten brauchen. Die gesamte Befragung verläuft standardisiert, d.h. bei jedem Patienten in gleicher Weise. Sie beinhaltet Interviewteile und Fragebögen aus weit verbreiteten Untersuchungsinstrumenten aus der Symptom- und Lebensqualitätsforschung, die schon bei einer großen Anzahl von Patienten und Gesunden angewendet wurden. Eine dafür geschulte Studienmitarbeiterin wird Sie ansprechen und das Interview in den ersten Tagen auf Station durchführen. Ihr stationärer Aufenthalt wird dadurch nicht in seinem Ablauf gestört oder verlängert, alle geplanten Untersuchungen finden selbstverständlich unverändert statt. Ihre Antworten beeinflussen den Ablauf Ihres Aufenthalts nicht, da die Auswertung in aller Regel erst einige Wochen nach Ihrer Entlassung vom SomA-Studien-Team durchgeführt und nicht mit den Stationsärzten oder Ärzten der Allergieabteilung besprochen wird.

Die Ergebnisse werden für wissenschaftliche Zwecke anonymisiert und statistisch ausgewertet, sowie in anonymisierter Form veröffentlicht. Jede(r) Patient(in) hat das Recht, die Ergebnisse der Tests zu erfahren. Alle Patient(inn)en werden darüber aufgeklärt, dass die Zustimmung zu Befragung, Auswertung oder Veröffentlichung der Daten jederzeit zurückgezogen werden kann, ohne dass Nachteile entstehen.

Appendix 2: Patient consent form

Klinik und Poliklinik
für Dermatologie und Allergologie am Biederstein
des Klinikums rechts der Isar
der Technischen Universität München

Direktor: Univ.-Prof. Dr. med. Dr. phil. Johannes Ring

Biedersteiner Straße 29, 80802 München

Klinik und Poliklinik
für Dermatologie und Allergologie
der TUM
Postfach 401 840, 80718
München

Einverständniserklärung zur Teilnahme an der klinischen Studie
„Körperliche Beschwerden in der Allergologie“

Ich bin damit einverstanden, an einer Patientenbefragung im Rahmen der oben genannten Studie der Technischen Universität München teilzunehmen. Die Befragung besteht aus einem kurzen Interview und einer Reihe von Fragebögen zum Selbstausfüllen. Sie setzt sich aus verschiedenen erprobten Instrumenten der Symptom- und Lebensqualitätsforschung zusammen. Der geplante Untersuchungsablauf in der Dermatologie wird durch die Befragung nicht gestört oder verlängert. Die Ergebnisse der Befragung beeinflussen meinen Krankenhausaufenthalt nicht.

Ich stimme zu, dass die Ergebnisse für wissenschaftliche Zwecke anonymisiert und statistisch ausgewertet werden, in anonymisierter Form erlaube ich auch ihre Veröffentlichung. Ich habe das Recht, die Ergebnisse der Befragung zu erfahren. Ich wurde darüber aufgeklärt, dass ich meine Zustimmung zu Befragung, Auswertung oder Veröffentlichung der Daten jederzeit zurückziehen kann, ohne dass mir Nachteile entstehen.

Ort, Datum	Patient(in)	Arzt

Appendix 3: Endorsement of the causal items of the IPQ-R for SFD (n=43), NoSFD (n=136) and VIT patients (n=43)

IPQ-R causal items		Strongly disagree	Disagree	Partly agree	Agree	Strongly agree
		n (%)	*n (%)*	*n (%)*	*n (%)*	*n (%)*
1. Stress or worry	SFD	7 (16.3)	10 (23.3)	16 (37.2)	8 (18.6)	2 (4.7)
	NoSFD	32 (23.5)	24 (17.6)	44 (32.4)	24 (17.6)	12 (8.8)
	VIT	27 (62.8)	5 (11.6)	9 (20.9)	-	2 (4.7)
	Total sample	66 (29.7)	39 (17.6)	69 (31.1)	32 (14.4)	16 (7.2)
2. Hereditary – it runs in my family	SFD	24 (55.8)	9 (20.0)	5 (11.6)	-	5 (11.6)
	NoSFD	72 (52.9)	25 (18.4)	18 (13.2)	15 (11.0)	6 (4.4)
	VIT	26 (60.5)	7 (16.3)	2 (4.7)	7 (16.3)	1 (2.3)
	Total sample	122 (55)	41 (18.5)	25 (11.3)	22 (9.9)	12 (5.4)
3. Germs or viruses	SFD	21 (48.8)	9 (20.9)	7 (16.3)	3 (7.0)	3 (7.0)
	NoSFD	65 (47.8)	38 (27.9)	22 (16.2)	7 (5.1)	4 (2.9)
	VIT	33 (76.7)	5 (11.6)	5 (11.6)	-	-
	Total sample	119 (53.6)	52 (23.4)	34 (15.3)	10 (4.5)	7 (3.2)
4. Diet or eating habits	SFD	11 (25.6)	10 (23.3)	11 (25.6)	6 (14.0)	5 (11.6)
	NoSFD	38 (27.9)	35 (25.7)	36 (26.5)	19 (14.0)	8 (5.9)
	VIT	30 (69.8)	6 14.0)	7 (16.3)	-	-
	Total sample	79 (35.6)	51 (23.0)	54 (24.3)	25 (11.3)	13 (5.9)
5. Chance or bad luck	SFD	22 (51.2)	5 (11.6)	6 (14.0)	7 (16.3)	3 (7.0)
	NoSFD	65 (47.8)	18 (13.2)	24 (17.6)	17 (12.5)	12 (8.8)
	VIT	12 (27.9)	2 (4.7)	7 (16.3)	10 (23.3)	12 (27.9)
	Total sample	99 (44.6)	25 (11.3)	37 (16.7)	34 (15.3)	27 (12.2)
6. Poor medical care in the past	SFD	20 (46.5)	7 (16.3)	10 (23.3)	4 (9.3)	2 (4.7)
	NoSFD	80 (58.8)	29 (21.3)	18 (13.2)	6 (4.4)	3 (2.2)
	VIT	37 (86.0)	4 (9.3)	2 (4.7)	-	-
	Total sample	137 (61.7)	40 (18.0)	30 (13.5)	10 (4.5)	5 (2.3)
7. Pollution in the environment	SFD	12 (27.9)	9 (20.9)	7 (16.3)	7 (16.3)	8 (18.6)
	NoSFD	34 (25.0)	33 (24.3)	43 (31.6)	16 (11.8)	10 (7.4)
	VIT	9 (20.9)	6 (14.0)	16 (37.2)	9 (20.9)	3 (7.0)
	Total sample	55 (24.8)	48 (21.6)	66 (29.7)	32 (14.4)	21 (9.5)
8. My own behaviour	SFD	15 (34.9)	15 (34.9)	7 (16.3)	3 (7.0)	3 (7.0)
	NoSFD	47 (34.6)	44 (32.4)	33 (24.3)	11 (8.1)	1 (0.7)
	VIT	30 (69.8)	5 (11.6)	8 (18.6)	-	-
	Total sample	92 (41.4)	64 (28.8)	48 (21.6)	14 (6.3)	4 (1.8)
9. My mental attitude e.g. thinking about life negatively	SFD	28 (65.1)	11 (25.6)	3 (7.0)	-	1 (2.3)
	NoSFD	87 (64.0)	33 (24.3)	14 (10.3)	2 (1.5)	-
	VIT	34 (79.1)	5 (11.6)	3 (7.0)	1 (2.3)	-
	Total sample	149 (67.1)	49 (22.1)	20 (9.0)	3 (1.4)	1 (0.5)
10.Family problems or worries caused my illness	SFD	17 (39.5)	15 (34.9)	7 (16.3)	2 (4.7)	2 (4.7)
	NoSFD	62 (45.6)	34 (25.0)	32 (23.5)	7 (5.1)	1 (0.7)
	VIT	37 (86.0)	2 (4.7)	4 (9.3)	-	-
	Total sample	116 (52.3)	51 (23.0)	43 (19.4)	9 (4.1)	3 (1.4)
11. Overwork	SFD	16 (37.2)	12 (27.9)	9 (20.9)	5 (11.6)	1 (2.3)
	NoSFD	56 (41.2)	34 (25.0)	29 (21.3)	15 (11.0)	2 (1.5)
	VIT	33 (76.7)	2 (4.7)	8 (18.6)	-	-
	Total sample	105 (47.3)	48 (21.6)	46 (20.7)	20 (9.0)	3 (1.4)
12. My emotional state, e.g. feeling down, lonely, anxious, empty	SFD	19 (44.2)	13 (30.2)	6 (14.0)	3 (7.0)	2 (4.7)
	NoSFD	69 (50.7)	32 (23.5)	26 (19.1)	7 (5.1)	2 (1.5)
	VIT	35 (81.4)	4 (9.3)	4 (9.3)	-	-
	Total sample	123 (55.4)	49 (22.1)	36 (16.2)	10 (4.5)	4 (1.8)
13. Ageing	SFD	25 (58.1)	10 (23.3)	7 (16.3)	1 (2.3)	-
	NoSFD	83 (61.0)	28 (20.6)	20 (14.7)	5 (3.7)	-
	VIT	32 (74.4)	5 (11.6)	5 (11.6)	1 (2.3)	-
	Total sample	140 (63.1)	43 (19.4)	32 (14.4)	32 (14.4)	7 (3.2)

14. Alcohol	SFD	36 (83.7)	6 (14.0)	-	1 (2.3)	-
	NoSFD	102 (75.0)	22 (16.2)	8 (5.9)	2 (1.5)	2 (1.5)
	VIT	35 (81.4)	6 (14.0)	2 (4.7)	-	-
	Total sample	173 (77.9)	34 (15.3)	10 (4.5)	3 (1.4)	2 (0.9)
15. Smoking	SFD	34 (79.1)	6 (14.0)	3 (7.0)	-	-
	NoSFD	109 (80.1)	14 (10.3)	11 (8.1)	1 (0.7)	1 (0.7)
	VIT	35 (81.4)	3 (7.0)	5 (11.6)	-	-
	Total sample	178 (80.2)	23 (10.4)	19 (8.6)	1 (0.5)	1 (0.5)
16. Accident or injury	SFD	36 (83.7)	3 (7.0)	4 (9.3)	-	-
	NoSFD	117 (86.0)	11 (8.1)	4 (2.9)	2 (1.5)	2 (1.5)
	VIT	39 (90.7)	3 (7.0)	1 (2.3)	-	-
	Total sample	192 (86.5)	17 (7.7)	9 (4.1)	2 (0.9)	2 (0.9)
17. My personality	SFD	26 (60.5)	11 (25.6)	3 (7.0)	1 (2.3)	2 (4.7)
	NoSFD	84 (61.8)	32 (23.5)	18 (13.2)	2 (1.5)	-
	VIT	36 (83.7)	3 (7.0)	3 (7.0)	1 (2.3)	-
	Total sample	146 (65.8)	46 (20.7)	24 (10.8)	4 (1.8)	2 (0.9)
18. Altered immunity	SFD	8 18.6)	7 (16.3)	10 (23.3)	12 (27.9)	6 (14.0)
	NoSFD	28 (20.6)	23 (16.9)	51 (37.5)	21 (15.4)	13 (9.6)
	VIT	12 (27.9)	2 (4.7)	16 (37.2)	8 (18.6)	5 (11.6)
	Total sample	48 (21.6)	32 (14.4)	77 (34.7)	41 (18.5)	24 (10.8)
19. Allergy	SFD	2 (4.7)	4 (9.3)	11 (25.6)	13 (30.2)	13 (30.2)
	NoSFD	14 (10.3)	8 (5.9)	31 (22.8)	38 (27.9)	45 (33.1)
	VIT	2 (4.7)	-	2 (4.7)	10 (23.3)	29 (67.4)
	Total sample	18 (8.1)	12 (5.4)	44 (19.8)	61 (27.5)	87 (39.2)

Appendix 4: Factor Analysis: Forced 5-factor structure (IPQ-R causal scale)

Total Variance Explained

Component	Initial Eigenvalues			Extraction Sums of Squared Loadings			Rotation Sums of Squared Loadings[a]
	Total	% of Variance	Cumulative %	Total	% of Variance	Cumulative %	Total
1	5,197	27,354	27,354	5,197	27,354	27,354	4,555
2	1,890	9,948	37,303	1,890	9,948	37,303	2,594
3	1,335	7,026	44,328	1,335	7,026	44,328	1,860
4	1,164	6,128	50,456	1,164	6,128	50,456	2,254
5	1,123	5,912	56,368	1,123	5,912	56,368	1,442
6	1,072	5,641	62,009				
7	,985	5,186	67,195				
8	,897	4,719	71,914				
9	,827	4,351	76,265				
10	,702	3,692	79,958				
11	,637	3,351	83,309				
12	,611	3,216	86,525				
13	,523	2,751	89,276				
14	,456	2,400	91,676				
15	,393	2,069	93,745				
16	,368	1,938	95,682				
17	,331	1,743	97,425				
18	,256	1,346	98,772				
19	,233	1,228	100,000				

Extraction Method: Principal Component Analysis.

Pattern Matrix[a]

	Component				
	1	2	3	4	5
Item 1	,750	,076	-,268	-,250	,116
Item 2	,016	-,008	-,041	,633	-,019
Item 3	-,055	,281	-,468	,373	-,071
Item 4	,284	-,012	-,630	,197	,230
Item 5	-,018	,018	,664	,140	,203
Item 6	-,070	,546	-,082	,142	-,118
Item 7	,139	,267	,196	,089	,534
Item 8	,325	,011	-,426	,342	,059
Item 9	,591	,039	,209	,460	-,178
Item 10	,821	-,026	-,061	-,065	,017
Item 11	,885	,086	,017	-,175	-,030
Item 12	,848	-,080	,053	,135	-,078
Item 13	,162	,608	,254	,247	-,015
Item 14	,036	,547	-,017	,010	,182
Item 15	,049	,647	-,085	-,282	,092
Item 16	-,025	,667	,027	-,058	-,067
Item 17	,624	,017	-,034	,283	-,011
Item 18	,035	,070	-,042	,499	,437
Item 19	-,148	-,122	,015	-,085	,802

Extraction Method: Principal Component Analysis.

Rotation Method: Oblimin with Kaiser Normalization.

a. Rotation converged in 10 iterations.

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