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REGULAR ARTICLE

Attention to bodily sensations and symptom perception in individuals with idiopathic environmental intolerance

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Abstract
Introduction Idiopathic environmental intolerance (IEI) is characterized by non-specific symptoms attributed to exposure to environmental odours or chemicals at levels below those known to induce adverse health effects. A clarification of whether psychological processes involved in sensory perceptions are associated with IEI would add to our understanding of this complex disorder.
Purpose To examine if measures of somato-sensory amplification, autonomic perception and absorption are associated with IEI.
Methods The study included individuals with self-reported or physician-diagnosed IEI. Participants ($n = 732$) completed questionnaires that included items on descriptive variables of IEI, the Somato-Sensory Amplification Scale (SSAS), the Autonomic Perception Questionnaire (APQ), the Tellegen Absorption Scale (TAS) and a Negative Affectivity Scale (NAS).
Results Multiple, hierarchical linear regression analyses revealed significant positive associations between SSAS, APQ, and IEI, while small and inverse associations were seen between TAS and IEI.
Conclusions The association with SSAS and APQ suggests that perceptual personality characteristics are important in understanding this disorder.

Keywords Absorption · Autonomic perception · Chemical sensitivity · Idiopathic environmental intolerance · Somato-sensory amplification

Abbreviations

APQ	Autonomic Perception Questionnaire
CHS	Chemical Hypersensitivity Scale
CNSS	CNS Symptom Scale
CSAS	Consequences for Social Activities Scale
CSS-SHR	The Chemical Sensitivity Scale for sensory hyper-reactivity
IEI	Idiopathic environmental intolerance
MUSS	Mucosal Symptom Scale
NAS	Negative Affectivity Scale
SSAS	Somato-Sensory Amplification Scale
TAS	Tellegen Absorption Scale

Introduction

Idiopathic environmental intolerance (IEI) is a condition characterized by individual reports of non-specific symptoms from various organ systems attributed to exposure to environmental odours or chemicals at levels below those known to induce adverse health effects [1]. A typical

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symptom pattern is difficult to establish since the reported symptoms typically vary between individuals, with women generally being more sensitive and reporting more symptoms than men [2–5]. Several sets of diagnostic criteria have been proposed [6–8], but none are currently internationally accepted. In population-based studies, reports of symptoms attributed to environmental odors are common, with prevalence estimates ranging from 9 to 33% [2–4, 9]. In contrast, prevalence rates of physician-diagnosed IEI or reports of disabling consequences in the form of social and occupational disruptions range from 0.5 to 6.3% [3, 4, 10].

Studies incorporating systematic exposure to symptom-inducing agents with the purpose of identifying pathophysiological mechanisms have to date been without consistent findings [11, 12], and the aetiology of IEI has thus been the focus of much controversy [1, 11]. It has been suggested that IEI is best classified as a functional somatic disorder [13, 14], partly because several studies have reported associations between IEI and psychological traits often connected with such disorders [14–16]. Increased rates of psychiatric co-morbidity is frequently reported, often in terms of major depression, somatoform disorders, anxiety or panic disorder [17]. Sensitization and conditioning processes have been suggested as mechanisms involved in the acquisition and maintenance of IEI [18–25], while others have speculated that IEI can be conceptualized and studied as a pain disorder [26]. Persistent pain states may involve central plasticity changes [26] and, as in pain disorders, it may be hypothesized that psychological factors play a role in various phases of IEI, such as in the transition from acute to persistent states [26]. An evaluation of psychological factors reflecting individual differences in sensory perception processes may therefore be relevant for a deeper understanding of this complex disorder.

Somato-sensory amplification refers to a tendency to experience somatic and visceral sensations as being unusually intense, noxious, and disturbing; it can be understood both as a trait, i.e. an enduring perceptual style, and as a state influenced by mood and circumstances [27, 28]. Somato-sensory amplification is thought to play a role in the maintenance of symptoms in functional somatic disorders [27, 29] due to a self-perpetuating cycle of increased attention to bodily sensations and a biased symptom perception. Negative affects have been suggested as risk factors in this process [13] by increasing attention on perceptual experiences with a negative quality, such as pain [30]. Somato-sensory amplification has also been suggested to influence some of the variance in somatic symptomatology found among patients with the same underlying medical disorder [31], such as the development of anticipatory nausea in relation to cancer treatment [32]. The role of somato-sensory amplification in IEI has been examined in a longitudinal study of psychological predictors of symptom severity by Bailer and colleagues [13]. Significant zero-order correlations ($P < 0.05$) between symptoms of IEI and somato-sensory amplification at baseline (0.45) and 1-year follow-up (0.43) were reported and, taken together, the psychological predictors explained 41% of the variance in subjective symptom severity [13]. Autonomic perception describes the relationship between an autonomic response, such as to feelings of anxiety or anger, and associations between increased autonomic symptom awareness and anxiety have been reported [33, 34]. The possible role of autonomic perception in relation to reports of IEI needs further examination.

The personality trait of absorption [35] is defined as an openness to experience and a predisposition to experience alterations of cognition and emotion across a broad range of situations [36]. Absorption has been shown to correlate with hypnotizability [37] and to be related to increased reports of distress and somatic symptoms [38]. It has also been associated with increased sympathetic and parasympathetic reactivity during exposure to an experimental stressor [38], suggesting that individuals who have high scores for this trait may be more reactive when exposed to a stressful situation. The reactivity to aversive stimuli in high-absorption individuals points to the possibility that they may be more vulnerable to conditioning [32]. Evidence of a link between absorption and IEI has been reported in a longitudinal study, which led to the conclusion that absorption may be a specific risk factor in IEI [15].

It is well established that IEI can develop into a chronic and disabling condition [14, 15, 39]. A possible avenue to a clearer understanding of the complexities of IEI could be exploring the possible association with psychological factors associated with increased attention to bodily sensations and biased symptom perception. In the study reported here, we hypothesized that somato-sensory amplification, autonomic reactivity and absorption would be associated with more severe self-reported reactions attributed to environmental odours.

Methods

Participants

Three groups were invited to participate in the study: (1) individuals from the general population; (2) individuals who had contacted the Danish Research Centre because of symptoms attributed to environmental, odorous chemicals; (3) patients with physician-diagnosed IEI. Group 1 included 18- to 69-year-old individuals randomly drawn from the Danish Civil Registration System [8] who had responded to a population-based cross-sectional survey

($n = 4260$). These respondents were invited to participate in the study reported here providing they had: (1) reported being bothered by exposure to at least one common chemical (e.g. fragranced products and newly printed magazines); (2) confirmed that exposure to odourous chemicals was associated with symptoms and not perceived as merely unpleasant; (3) given consent to be contacted again ($n = 787$). Group 2 included individuals who had contacted the Danish Research Centre for chemical sensitivities between 1 January 2006 and 1 August 2007 because of reactions consistent with IEI and who had consented to participate in the our study ($n = 101$). Group 3 included individuals who had received a diagnosis of IEI either at the Copenhagen University Hospital, Rigshospitalet, or at the Hamlet Private Hospital, Denmark between 1 January 1990 and 1 January 2007. This group received a letter inviting them to participate in our study ($n = 136$). In total, 1024 women and men were invited to participate in the study. The overall response rate was 71.5% ($n = 732$), with the response rate being 72.5, 77.7 and 60.4% in groups 1, 2 and 3, respectively.

Measurements

The following self-report measures were included in the study:

The Somato-Sensory Amplification Scale (SSAS) measures responses to ten bodily sensations that may be experienced as unpleasant (e.g. "I hate being too hot or too cold"), but which generally do not connote serious disease [27, 28]. Responses are rated on a 5-point Likert scale and summarized into a total score. Evaluation of the psychometric properties of the SSAS suggests adequate internal consistency (Cronbach's Alpha = 0.82) and test–retest reliability (0.79) [28]. The SSAS has been shown to correlate moderately with self-report measures of negative affectivity and psychological distress [40]. The internal consistency (Cronbach's Alpha) of SSAS in our study was 0.88.

The anxiety-related version of the Autonomic Perception Questionnaire (APQ) measures attentiveness to bodily responses in anxiety-provoking situations (e.g. "When I feel anxious, I can feel my heart beating faster") [33]. The APQ asks for a brief description of a situation in which the respondent felt anxious followed by 30 specific items. Responses are rated on 9-point Likert-scales, with the initial description serving as reference for rating the responses, and are finally summarized into a total score. Internal consistencies (Cronbach's Alpha) reported for the APQ range from 0.84 to 0.89 [41]. The internal consistency (Cronbach's Alpha) in our study was 0.94.

The Tellegen Absorption Scale (TAS) [35] includes 34 items measuring imaginative involvement and openness to experience (e.g., "Sometimes I feel and experience things as I did when I was a child"). Responses are stated as *true* or *false* with the total score being the number of responses stated as *true*. Test–retest reliability (0.88) and internal consistency (0.82) has been reported as satisfactory in the Danish translation of the TAS [37].

The Negative Affectivity Scale (NAS) includes 15 items measuring the tendency to experience and report negative emotions, including anxiety, guilt, hostility and depression, with low negative affect reflecting a state of calmness [42]. Respondents are asked to respond in relation to experiences of negative emotions within the past week. Responses are rated on a 5-point Likert scale and summarized into a total score. Satisfactory internal consistency (0.87) and moderate test–retest reliability (0.48) have been reported [42]. The internal consistency (Cronbach's Alpha) of the Danish version of NAS used in our study was 0.98.

The Chemical Sensitivity Scale for sensory hyperreactivity (CSS-SHR) comprises 11 statements regarding affective reactions to and behavioural disruptions in daily activities from odorous/pungent environmental chemicals [43]. The metric properties of the CSS-SHR, i.e. test–retest reliability, internal consistency and concurrent validity, have been reported as being favourable [43]. In order to adapt the CSS-SHR for use in Danish studies on IEI, the CSS-SHR was translated into Danish by a professional translation office. The Danish translation was subsequently tailored to Danish usage and then translated back to the original language by a different translator. This translation was then compared with the original version in order to identify potential sense-altering discrepancies. The individual CSS-SHR score is calculated from each point obtained from the 11 statements, with a total score ≤ 55. A score >43 somewhat predicts increased airway sensory reactivity [2, 43]. The internal consistency (Cronbach's Alpha) of CSS-SHR was 0.77 in our study.

The IEI was assessed by: (1) number and severity of 20 symptoms used in a previous Danish population-based study on the prevalence of symptoms attributed to common environmental odours [3], including headache, exhaustion, dizziness, difficulties concentrating, grogginess, sleep difficulties, panic attacks, breathlessness and symptoms from the eyes, nose, sinuses, mouth, throat and lungs; (2) number of symptom-inducing environmental odours, including fragranced products, cleaning agents, nail polish remover, newly printed papers or magazines, new furniture, soft plastic or rubber, cooking fumes, motor vehicle exhaust, tar or wet asphalt, smoke from a wood burner and new electric equipment; (3) social consequences that were assessed by the following question: "Do reactions caused by environmental odours lead you to avoid: (a) social activities (e.g. family- or other private parties), (b) inviting guests, (c) going on holiday, (d) sports activities, (e) using public

transportation, (f) going to the cinema or theatre and (g) going to restaurants"?

The questionnaire was pilot-tested for relevance, comprehension and ease of completion by individual interviews with seven individuals with either self-reported or physician-diagnosed IEI, resulting in minor adjustments.

Statistical analysis

Principal components analysis

Initially, 63 items, described in detail in the section on Measurements, covering symptoms attributed to exposure to environmental odours or chemicals, symptom-eliciting chemical agents and consequences in terms of the degree to which reactions had influenced social relations and work were analysed using principal components analysis (PCA) with Varimax rotation. Factors were selected on the basis of an eigenvalue >1, and items were chosen to show loadings ≥0.5 for the central factor. Seven factors were identified, of which four grouped into clusters of five or more items with cross loading of ≤0.25. Factor 1 can be described as responses to symptom-eliciting environmental odours, such as fragranced products, cleaning agents, nail polish remover, newly printed papers or magazines, new furniture, soft plastic or rubber, cooking fumes, motor vehicle exhaust, tar or wet asphalt, smoke from a wood burner and new electric equipment. These 11 items were summarized in the Chemical Hypersensitivity Scale (CHS) which, based on the response format to the questions, yields a total score ranging from 0 to 33. Cronbach's Alpha for the CHS was 0.95. Factor 2 includes seven social or public events that are either usually or always avoided because of reactions attributed to exposure to common environmental odours, such as social activities (e.g. family- or other private parties), inviting guests, going on holiday, sports activities, using public transportation, going to the cinema or theatre or going to restaurants. These items were summarized in the Consequences for Social Activities Scale (CSAS), yielding a total score ranging from 0 to 14. Cronbach's Alpha for the CSAS was 0.92. Factor 3 consists of eight items that describe symptoms from the central nervous system, such as headache, exhaustion, dizziness, difficulties concentrating, grogginess, sleep difficulties, panic attacks and breathlessness. These items were summarized in the CNS Symptoms Scale (CNSS), with total scores ranging from 0 to 8**.** Cronbach's Alpha for the CNSS was 0.68. Factor 4 consists of six items describing mucosal symptoms, including those of the eyes, nose, sinuses, mouth, throat and lungs, summarized in the Mucosal Symptoms Scale (MUSS), with total scores ranging from 0 to 6. Cronbach's Alpha for the MUSS was 0.59.

Groups

Individuals in groups 2 and 3 had contacted either a physician or the Danish Research Centre for chemical sensitivities because of symptoms attributed to common environmental odours, suggesting that these individuals had a more severe state of IEI than those in group 1. We hypothesized that these two former groups would not differ in terms of their scores on the CSS-SHR measuring affective reactions to and behavioural disruptions in daily activities due to odorous environmental chemicals nor would they differ in terms of sex and age. Consequently, they could be pooled in subsequent statistical analysis in order to increase statistical power. The mean score for groups 2 and 3 on the CSS-SHR was 50.5 [standard deviation (SD) 5.4] for group 2, and that for group 3 alone was 49.8 (SD 4.9) ($P = 0.53$). The mean age was 53.3 years (SD 10.4) in group 2 and 51.8 years (SD 11.3) in group 3 ($P = 0.43$). The two groups did not differ in terms of the distribution of sex ($P = 0.51$). Groups 2 and 3 were therefore pooled in the statistical analyses and are referred to as the patient group, whereas group 1 is referred to as the population group.

Multiple, hierarchical linear regression analyses

A number of multiple, hierarchical linear regression analyses were conducted with CHS, MUSS, CNSS and CSAS as the dependent variables and the psychological variables of somato-sensory amplification (SSAS), autonomic perception (APQ) and absorption (TAS) as independent variables, all entered at step 1. Age and sex were entered at step 2, negative affectivity (NAS) at step 3, and patient versus population group at step 4. Finally, interaction terms were added to the regression model to evaluate possible group differences. The data were analysed using SPSS ver. 16.0 and the level of significance was set at $P < 0.05$.

Approval

The study was approved by the Danish Data Protection Agency. According to Danish legislation, questionnaire studies do not need approval by an ethics committee.

Results

Group characteristics

The characteristics of the population and the patient groups are summarized in Table 1. Using independent samples *t* tests, we found a significant difference when we compared

Table 1 Characteristics of the population and patient groups showing means and standard deviations

Variables[a]	Population group			Patient group			*P* value[b]
	Men	Women	Total	Men	Women	Total	
n	194 (34%)	377 (66%)	571	21 (13%)	140 (87%)	161	≤0.001
CSS-SHR	35.0 (9.2)	37.9 (8.4)	36.9 (8.8)	50.1 (4.8)	50.4 (5.4)	50.4 (5.3)	≤0.001
CHS	12.3 (6.9)	13.7 (6.8)	13.2 (6.9)	26.3 (5.6)	25.2 (6.3)	25.33 (6.2)	≤0.001
MUSS	2.2 (1.3)	2.3 (1.4)	2.3 (1.3)	3.5 (1.8)	3.9 (1.7)	3.8 (1.7)	≤0.001
CNSS	1.9 (1.3)	2.0 (1.3)	2.0 (1.3)	5.6 (1.4)	4.8 (1.9)	4.9 (1.9)	≤0.001
CSAS	0.27 (0.82)	0.43 (1.5)	0.38 (1.3)	6.52 (4.0)	6.15 (4.1)	6.20 (4.1)	≤0.001
SSAS	26.1 (6.2)	29.5 (5.8)	28.3 (6.2)	24.9 (5.5)	28.1 (6.9)	27.7 (6.8)	0.27
APQ	95.6 (42.2)	121.0 (50.3)	112.4 (49.2)	85.2 (55.9)	107.3 (48.0)	104.4 (49.5)	0.70
TAS	14.0 (6.6)	15.1 (6.7)	14.7 (6.6)	10.8 (6.3)	12.9 (7.7)	12.7 (7.6)	≤0.001
NAS	12.4 (9.0)	13.6 (9.0)	13.2 (9.0)	11.8 (9.5)	14.4 (9.9)	14.0 (9.9)	0.35
Age	50.1 (11.8)	47.1 (12.6)	48.1 (12.4)	50.9 (11.2)	53.3 (10.6)	53 (10.6)	≤0.01

Number in parentheses is the standard deviation (SD)

[a] See Abbreviations Section at beginning of article for explanation of abbreviations

[b] Independent samples *t* test for equality of means between population and patient group

the total population group and the total patient group in terms of sex, age and mean scores on the CHS, MUSS, CNSS, CSAS and TAS. No significant differences were found for the remaining independent variables (SSAS, APQ and NAS).

Non-respondents

Comparison of respondents and non-respondents in the population group showed that respondents to our questionnaire were significantly older: 46.5 (SD 12.4) versus 42.9 years (SD 12. 8) ($P < 0.001$). They were also more likely to be women [odds ratio (OR) 1.2, $P = 0.08$]. The respondents did not differ from non-respondents in terms of severity of self-reported symptoms attributed to common environmental odours (OR = 0.9, $P = 0.6$). In the patient group, no difference in mean age was seen between respondents and non-respondents: 52.7 (SD 10.9) and 51.8 years (SD 12.1), respectively ($P = 0.56$). Correspondingly, no difference was found for the distribution of sex ($P = 0.54$).

Correlations

Using Pearson's *r*, we found moderate to high correlations between the four dependent variables (CHS, MUSS, CNSS and CSAS) (Table 2). Small to moderate correlations were found between the SSAS and CHS and MUSS, and between the APQ and MUSS and CNSS. A small, negative correlation was found between CSAS and TAS. A number of small to moderate correlations were also found between the independent variables (SSAS, APQ, and TAS) and control variables (NAS, age and sex).

Multiple, hierarchical linear regression analysis

When we entered CHS as the dependent variable in a multiple, hierarchical linear regression model, all steps were found to be statistically significant [*F* 8.8 (step 1) to 66.4 (step 4), $P < 0.001$]. At the first step, which consisted of entering SSAS, APQ, and TAS as independent variables, only SSAS was a significant predictor of CHS. When sex and age were entered at step 2, SSAS continued to be significant together with age. This was also the case when NAS was entered into the equation at step 3, and group (population vs. patient group) at step 4, with the fourth and final model explaining 44% of the variance. The results of the final model are summarized in Table 3.

Likewise, all models were statistically significant when the MUSS [*F* 11.6 (step 1) to 48.9 (step 4), $P < 0.001$] and the CNSS [*F* 4.9 (step 1) to 65.8 (step 4); $P < 0.001$] were entered as the dependent variables. For the MUSS, the SSAS, APQ and TAS were significant predictors at the first step. When sex and age were entered at step 2, and NAS was entered at step 3, all variables continued to be significant. At step 4, when group (population vs. patient group) was entered, only SSAS and APQ remained significant. The final model explained 23% of the variance (Table 3). For the CNSS, only the APQ was significant at step 1, while a trend was seen for the SSAS. At the second step, when sex and age were entered, the APQ remained significant, but this was not the case at step 3. When group (population vs. patient group) was entered at the fourth and final step, SSAS and APQ were significant predictors, with the final model explaining 44% of the variance. Finally, all

Table 2 Correlations between the CHS, MUSS, CNSS, CSAS, SSAS, APQ-ANX, TAS, NAS, age and sex

Variables	CHS	MUSS	CNSS	CSAS	SSAS	APQ	TAS	NAS	Age	Sex
CHS	–	0.47**	0.60**	0.56**	0.21**	0.08*	0.03	0.16**	0.26**	0.18**
MUSS	–	–	0.60**	0.41**	0.17**	0.21**	0.03	0.14**	0.09*	0.14**
CNSS	–	–	–	0.56**	0.12**	0.14**	0.02	0.18**	0.06	0.15**
CSAS	–	–	–	–	0.08*	0.03	−0.12**	0.11**	0.15**	0.13**
SSAS	–	–	–	–	–	0.41**	0.25**	0.29**	0.03	0.23**
APQ	–	–	–	–	–	–	0.30**	0.37**	−0.13**	0.21**
TAS	–	–	–	–	–	–	–	0.16**	0.05	0.05
NAS	–	–	–	–	–	–	–	–	−0.16**	0.08
Age	–	–	–	–	–	–	–	–	–	−0.05
Sex	–	–	–	–	–	–	–	–	–	–

* Correlation is significant at $P < 0.05$ (two-tailed)

** Correlation is significant at $P < 0.01$ (two-tailed)

Table 3 Final models (fourth step) of multiple, hierarchical linear regression analysis with the CHS, MUSS, CNSS and CSAS as the dependent variables

Independent variables	Dependent variables							
	CHS		MUSS		CNSS		CSAS	
	Beta	*P* value	Beta	*P* value	Beta	*P* value	Beta	*P* value
SSAS	0.18	≤0.001	0.10	<0.05	0.07	<0.05	0.05	0.08
APQ	0.02	0.54	0.20	≤0.001	0.12	≤0.001	0.07	<0.05
TAS	0.01	0.67	−0.02	0.64	0.03	0.32	−0.10	≤0.001
Sex	0.03	0.45	−0.01	0.80	−0.03	0.38	−0.01	0.67
Age	0.18	≤0.001	0.05	0.22	−0.02	0.52	0.04	0.11
NAS	0.10	≤0.01	0.03	0.45	0.08	<0.05	0.04	0.16
Group[a]	0.57	≤0.001	0.42	≤0.001	0.65	≤0.001	0.74	≤0.001
Total adjusted R^2	0.44		0.23		0.44		0.42	

Due to missing values the multiple, hierarchical linear regression analysis includes the following number of participants; CHS, $n = 573$; MUSS, $n = 575$; CNSS, $n = 553$; CCAS, $n = 575$

[a] Group population (1) versus patient (2) group

steps were significant when CSAS [F: 5.6 (step 1) to 116.0 (step 4), $P < 0.001$] was entered as the dependent variable. At the first step, only TAS was a significant predictor. TAS remained significant when sex and age were entered at step 2 and when NAS was entered into the equation at step 3. At step 4, the TAS and APQ were significant, while a trend was seen for the SSAS. The fourth and final model explained 42% of the variance. The results are summarized in Table 3.

As seen in Table 3, the beta-values found when the group variable (population vs. patient group) was entered indicate that this variable has the strongest relationship with the dependent variables. Exclusion of the group variable reduced the estimates of the adjusted R^2 to 15 (CHS), 7 (MUSS), 5 (CNSS) and 8% (CSAS), respectively.

Differences between patient and population sample

To evaluate whether the effects of the independent variables differed between the two study groups, we added an interaction term between group and each of the psychological variables to the regression model: SSAS × group, APQ × group, TAS × group, NAS × group. For CHS and MUSS, none of the interactions reached statistical significance. For the dependent variables CNSS and CSAS, we found significant interactions between TAS and group ($P = 0.01$ and 0.03, respectively). This was due to (1) TAS scores being significantly, positively correlated with CNSS in the population group, while no correlation was seen for the patient group, and (2) TAS scores being more negatively correlated with CSAS in the patient group than in the population group.

Discussion

The objective of our study was to examine the possible influence of individual differences in perceptual style on the severity of IEI as estimated by four scales describing number of symptom-inducing odours (CHS), number of mucosal symptoms (MUSS), number of symptoms from the central nervous system (CNSS) and degree of social consequences (CSAS). The relatively moderate, positive correlations found between the dependent variables (CHS, MUSS, CNSS and CSAS) support the conclusion that, although correlated, they reflect different aspects of intolerance to common environmental odours without substantial overlap. Somato-sensory amplification and autonomic perception as measured by the SSAS and the APQ were found to be significant predictors of multiple mucosal and CNS symptoms in both study groups—after negative affectivity, age, sex and group had been controlled for. Some evidence of a role of these variables in reports of a high number of symptom-inducing odours and social consequences attributed to intolerance reactions was also seen. Evidence of a link between absorption as measured by the TAS and IEI has been reported in a longitudinal study by Witthöft and colleagues [15], which led to the conclusion that absorption may be a specific risk factor in IEI. The association between IEI and absorption in our study, however, was weak and does not immediately support the conclusion made by Witthöft and colleagues [15]. The latter used a different response format for the TAS and, consequently, the scores from the two studies can not be directly compared. Witthöft and colleagues [15] also included a healthy control group, and the differences in designs may offer some explanation of the different conclusions.

According to Barsky et al. [27], the tendency to amplify somatic stimuli involves amplification of normal physiologic sensations, benign symptoms, somatic concomitants of affect as well as symptoms of serious disease. Somato-sensory amplification is proposed to be a predisposing factor in functional somatic disorders [28, 31], and the construct was developed to assess the perceptual style of such patients [31]. In a recent review, Duddu and colleagues [44] suggest that somato-sensory amplification is neither sensitive nor specific to somatizing states and that other psychological traits, such as anxiety, depression and negative affectivity, also contribute. SSAS has also been applied to patients receiving chemotherapy for cancer, where SSAS was found not to predict anticipatory nausea [32, 45]. Furthermore, a study testing the psychometric properties of the SSAS as a measure of somatic sensitivity among healthy volunteers revealed no relationship between SSAS and interoceptive sensitivity as measured by a heartbeat detection task [40]. The authors of the latter study concluded that SSAS is merely a measure of negative emotionality and general distress rather than of somatic sensitivity [40]. However, the relatively moderate correlation found between SSAS and negative affectivity (NAS) in our study, together with the association between SSAS and the dependent variables, independent of NAS, do not support this conclusion. In addition, authors of a study examining the relationship between somato-sensory amplification and different types of evoked potentials in healthy volunteers reported a significant association between SSAS and long-latency potentials of auditory event-related potentials (P300) [46], leading them to suggest that somato-sensory amplification reflects aspects of cognitive processing that include, for example, attention allocation and activation of immediate memory [46]. Taken together, somato-sensory amplification may be a cognitive factor of potential relevance for the understanding of IEI, rather than merely a proxy for negative affectivity.

Standardized regression coefficients for the extended German version of the SSAS of 0.18 at baseline and 0.12 at the 1-year follow-up have been reported, indicating some stability of SSAS scores in individuals with IEI [13]. Our results suggest that individuals who score high on the SSAS and the APQ are likely to report more mucosal and CNS symptoms, report intolerance to more environmental odours and limit their social activities. No significant difference in mean scores on the SSAS and the APQ was seen between the population- and the patient group, and when the group variable was removed from the regression analysis, the models explained between 5 and 15% of the variance in severity scores. The exact role of individual differences in sensory perception in IEI cannot be determined in a cross-sectional study design, but it does warrant further examination in future prospective studies. To estimate the relationship between the psychological variables and the self-reported impact of IEI on social activities, we analysed the consequences for social activities (CSAS) and found a significant relationship between CSAS and the APQ, a borderline significant relationship with the SSAS and a significant inverse relationship with the TAS. These results suggest that somato-sensory amplification and autonomic reactivity also affect the degree of social consequences attributed to IEI.

In order to examine performance on tasks assessing cognitive variables, Witthöft et al. [21] compared a group of individuals with IEI with both a somatoform and a healthy control group. Attentional bias was reported for the IEI group in terms of enhanced attention allocation to symptom words, such as headache and fatigue, in response to an emotional stroop task. While no evidence was reported regarding attentional bias towards words describing symptom-eliciting agents in IEI, such as perfume and paint smell, the IEI group produced more highly negative emotional

ratings of these words than did the other two groups. Enhanced attention to "internal information" supports the theory of somato-sensory amplification as a mechanism involved in symptom maintenance in IEI. Personality traits, such as negative affectivity, may influence both conditioning and sensitization processes as individuals who score high on this trait may be more vulnerable to learning symptoms [19, 20, 47]. Based on the results of their longitudinal study, Bailer et al. [14] concluded that IEI is a chronic condition in which trait anxiety contributes to its maintenance via somatic attributions. Trait anxiety has also been suggested as a predisposing factor in the acquisition of chemical intolerance based on findings of elevated scores on psychometric measures of anxiety and anxiety-related personality traits in a non-patient population sample [48, 49]. These findings were proposed to be possible indications of an increased vulnerability to conditioning [49], which has also been discussed as an explanatory model for symptom generation in IEI [20, 50].

Whether psychopathology and individual susceptibility to sensitivity reactions are part of the aetiology in IEI or merely act as amplifying factors can only be speculative. It is likely that the aetiology of IEI is multi-factorial and, as for somatoform disorders [51, 52], it can be argued that the complexity of IEI is best studied from a bio–psycho–social perspective. This involves the influence of biological factors, such as central sensitization processes [25]. psychopathological processes, such as the role of anxiety and depression [17]. processes involved in symptom perception and amplification [13, 14, 18, 21] and socioeconomic factors [3, 53].

Methodological considerations

Apart from the cross-sectional design used in this study, some additional methodological weaknesses should be noted. The lack of consensus on the IEI criteria raises some uncertainty regarding prevalence estimates across studies and how to approach the reports of multiple and disabling symptoms from a clinical perspective. To classify individuals with self-reported IEI, Berg and colleagues used the level of self-reported adjustments in social life and/or occupational conditions as indicators of severity [3]. Individuals who reported social or occupational consequences tended to report significantly more symptoms and symptom-inducing environmental odours than did individuals who did not report such consequences [3]. Based on the results from the PCA, we used a similar approach by entering symptoms, symptom-inducing environmental odours and social consequences in multiple, hierarchical linear regression models as proxies for estimating subjectively reported severity. Although this severity classification may be inadequate in terms of defining pathophysiological mechanisms, it represents a pragmatic approach by describing the subjectively experienced manifestations of IEI. Due to the broad definition of IEI and the possibility that some respondents may interpret the symptoms and reactions as indicative of other health problems, such as allergy, asthma or other disorders, we cannot rule out selection bias, especially in the population group. The inclusion of a control group in our study design could have strengthened our design by adding information regarding the influence of psychological features on the presence of IEI independent of severity. This research question, however, was not the objective of our study.

Our results provide evidence of a significant association between somato-sensory amplification and autonomic perception and individual reports of multiple mucosal and CNS symptoms attributed to IEI. There was some evidence suggesting that these factors are also associated with higher numbers of symptom-inducing odours and social consequences. In contrast, we found no evidence of a role of the personality trait of absorption. Due to the limitations of the cross-sectional design, we were unable to determine whether somato-sensory amplification and autonomic reactivity play a causal role in the acquisition and maintenance of IEI. Longitudinal studies are needed to establish a possible causal relationship and to provide further evidence of the stability and role of these psychological variables in the course of the disorder. Whether individuals who report IEI are also sensitive to other perceptual experiences, such as pain, and whether this sensitivity may play a role in the pathophysiology and course of IEI needs to be clarified. In conclusion, our study supports existing evidence suggesting that emotional and perceptual personality characteristics are important in understanding the complexities of this disorder.

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