Journal of Psychosomatic Research 79 (2015) 614–619

Contents lists available at ScienceDirect

Journal of Psychosomatic Research

Somatisation as a risk factor for incident depression and anxiety

Sandra M.A. Dijkstra-Kersten [a,*], Kate Sitnikova [a], Harm W.J. van Marwijk [a], Marloes M.J.G. Gerrits [a], Johannes C. van der Wouden [a], Brenda W.J.H. Penninx [b,c,d], Henriëtte E. van der Horst [a], Stephanie S. Leone [e]

[a] Department of General Practice and Elderly Care Medicine, EMGO institute for Health and Care Research, VU University Medical Center, Amsterdam, The Netherlands
[b] Department of Psychiatry, EMGO Institute for Health and Care Research, VU University Medical Center, GGZ inGeest, Amsterdam, The Netherlands
[c] Department of Psychiatry, Leiden University Medical Center, The Netherlands
[d] Department of Psychiatry, University Medical Center Groningen, The Netherlands
[e] Department of Public Mental Health, Netherlands Institute of Mental Health and Addiction, Utrecht, The Netherlands

ARTICLE INFO

Article history:
Received 19 April 2015
Received in revised form 14 July 2015
Accepted 31 July 2015

Keywords:
Anxiety disorder
Depressive disorder
Prospective cohort-study
Somatisation

ABSTRACT

Objective: In this study, we aimed to examine somatisation as a risk factor for the onset of depressive and anxiety disorders.
Methods: 4-year follow-up data from the Netherlands Study of Depression and Anxiety (NESDA), a multisite cohort study of the course of depression and anxiety, was analysed. Participants (18–65 years) without a lifetime depressive or anxiety disorder at baseline were included ($n = 611$). Somatisation was measured at baseline with the somatisation subscale of the 4 Dimensional Symptoms Questionnaire. Onset of depression and anxiety was assessed with the CIDI interview at 2-year and 4-year follow-up.
Results: Somatisation was a risk factor for the incidence of depression [Hazard Ratio per unit increase (HR); 95% Confidence Interval (CI): 1.13; 1.09–1.17] and anxiety [HR; 95% CI: 1.14; 1.09–1.18]. Associations attenuated but remained statistically significant after adjusting for socio-demographic characteristics, chronic somatic disorders, and baseline levels of (subclinical) depressive or anxiety symptoms [adjusted HR for depression; 95% CI: 1.06; 1.00–1.12, adjusted HR for anxiety; 95% CI: 1.13; 1.07–1.20].
Conclusion: Persons who somatise have an increased risk of becoming depressed or anxious in subsequent years, over and above baseline levels of depressive or anxiety symptoms. They may represent a target group for prevention of depressive and anxiety disorders.

Introduction

Somatisation, a common phenomenon in primary care, is defined as "the tendency to experience and communicate somatic distress and symptoms unaccounted for by pathological findings, to attribute them to physical illness, and to seek medical help for them" [1], p. 1359. While experiencing some physical symptoms unaccounted for by pathological findings is a common phenomenon, experiencing multiple unexplained physical symptoms from different organ systems implies somatisation [2]. Physical symptoms, such as fatigue, dizziness and pain, are prevalent, often co-occurring and can range in severity with syndromes such as somatic symptom disorder and functional somatic syndromes (e.g. fibromyalgia) at the severe end of the clinical spectrum to self-limiting symptoms. These are especially prevalent in people who have mental health problems, such as depressive and anxiety disorders [3–5]. A cross-sectional study by Bekhuis et al. (2014) [5], for instance, showed that depressive and anxiety disorders were moderately to strongly associated with all clusters of somatic symptoms. Simon et al. (1999) [4] found that half of the depressed patients in primary care were somatising. Somatisation could also constitute an important risk factor for the occurrence of these disorders and, therefore, persons who somatise may represent a target group for prevention of depressive and anxiety disorders, which continues to be an important public health goal [6–8]. However, as to date most studies that have investigated this association have been cross-sectional, [3,9,10], thus precluding conclusions about temporality, little is known about the actual risk of developing a depressive or anxiety disorder. Examining the relationship between somatisation and the onset of depressive or anxiety disorders is important as the co-occurrence of somatisation with mental health problems affects health outcomes, functioning, and economic costs, thus increasing the burden of disease. The presence of somatisation is associated with less improvement of depressive and anxiety symptoms, and is predictive of a poorer treatment response in depressed patients [11]. In addition, the co-occurrence of symptoms of somatisation and depression is associated with decreased physical, social, and occupational functioning and an increase of health care services use [8].

* Correspondence to: Sandra Dijkstra-Kersten, VU University Medical Center, Department of General Practice & Elderly Care Medicine, Van der Boechorststraat 7, D-549, 1007 MB Amsterdam, The Netherlands.
E-mail address: s.kersten@vumc.nl (S.M.A. Dijkstra-Kersten).

http://dx.doi.org/10.1016/j.jpsychores.2015.07.007
0022-3999/

Despite its obvious relevance for health care, few studies have investigated somatisation as a predictor for the onset of depressive or anxiety disorders. Data from a primary care study showed that specific unexplained symptoms (e.g. fatigue, pain or dizziness) presented to the GP were not predictive of subsequent depressive or anxiety disorder after three months as recognised and registered by GPs, although the presence of unexplained symptoms increased the concurrent risk of depression or anxiety at least three-fold [12]. However, in a community study somatisation predicted subsequent depressive symptoms at 5-year follow-up in non-depressed women, but was not predictive in men, after correction for depressive symptoms at baseline [13]. There is also some evidence from community and primary care studies focusing on specific medically unexplained physical symptoms (e.g. fatigue) that these symptoms can increase the risk of subsequent psychiatric symptoms and disorders, including depression and anxiety [14–16]. Although some of these studies focused on medically unexplained physical symptoms, it is not clear whether these persons with medically unexplained symptoms somatise.

Besides focusing on a specific symptom, most previous studies either did not take psychiatric history into account, thus could not discriminate between incidence and recurrence, or did not use a clinical interview but rather used self-report questionnaires to determine the presence of a disorder at baseline and follow-up. Therefore, our study aimed to examine whether somatisation is a risk factor for the subsequent onset of an incident depressive or anxiety disorder as determined by a clinical interview in a longitudinal prospective cohort study.

Methods

Design

This study used 4-year prospective data from the Netherlands Study of Depression and Anxiety (NESDA). The overall aim of the NESDA study is to examine the aetiology, course and consequences of depression and anxiety, using biological and psychosocial research paradigms within an epidemiological framework. At baseline (2004–2007), 2981 participants aged 18 through 65 years were included in the study. The sample consisted of healthy controls; persons with a prior history of depression and/or anxiety; persons with a current depressive and/or anxiety disorder and persons at risk because of a family history or subthreshold depressive or anxiety symptoms. Participants were recruited in different settings: the community ($n = 564$), primary care ($n = 1610$), and mental health services ($n = 807$). Exclusion criteria were the presence of a primary psychiatric diagnosis other than depression or anxiety, which might interfere with the course trajectory (i.e. psychotic disorder, obsessive compulsive disorder, bipolar disorder or severe addiction disorder), and lack of fluency in the Dutch language. Baseline and follow-up assessments consisted of a clinical interview, questionnaires, and physical measurements. The baseline, 2-year, and 4-year follow-up measurements were used in this study. Further details on the selection procedure and design of the NESDA study are provided elsewhere [17]. The research protocol was approved by the ethical review boards of the VU University Medical Center and all other participating institutes. All respondents provided written informed consent.

Study population

Participants who never had an episode of a depressive disorder (major depressive disorder or dysthymia) or anxiety disorder (social phobia, panic with agoraphobia, panic without agoraphobia, agoraphobia, generalised anxiety disorder) were included in this study ($n = 652$). Of these participants, 614 (94%) participated in at least one follow-up measurement. Information on the central determinant (i.e. somatisation) was available for 611 of these remaining participants. For these participants, no information on the other variables was missing. These 611 participants represent our study sample.

Measures

Onset of depressive and anxiety disorders

The Composite International Diagnostic Interview (CIDI, lifetime version 2.1) [18] was administered at the 2-year and 4-year follow-up assessments to determine the incidence (yes/no) of a depressive or anxiety disorder during the 4-year follow-up period. The CIDI is a highly reliable and valid assessment tool for depressive and anxiety disorders as defined by the DSM-IV criteria [18]. Incident depression or anxiety was defined as the occurrence of a depressive or anxiety disorder at any point during the follow-up period.

When diagnosed with a depressive or anxiety disorder at either the 2-year or 4-year follow-up measurements, participants were asked to indicate the time of onset of their disorder: less than two weeks ago, two weeks to one month ago, one to six months ago, six to twelve months ago, in the last twelve months, or more than one year ago. Time-at-risk was calculated from baseline until the moment a participant had an event (i.e. onset of a depressive or anxiety disorder), by taking the midpoint of each time interval for onset. For instance, no depressive disorder was diagnosed at 2-year follow-up, while at the 4-year follow-up measurement, a participant reported the time of onset of a depressive disorder between six and twelve months ago. For this participant, time-at-risk was set to 48 months − 9 months = 39 months. Time-at-risk was censored at 48 months when the participant had not developed a depressive or anxiety disorder during the follow-up period. For participants who did not take part in the 4-year follow-up measurement, time-at-risk was calculated over the 2-year follow-up. Participants who did not take part in the 2-year follow-up measurement, but did take part in the 4-year follow-up measurement, were asked about onset and recency of depressive and anxiety disorders over a 4-year period at the 4-year follow-up measurement and time to onset was calculated accordingly.

Somatisation

The somatisation subscale of the Four Dimensional Symptoms Questionnaire (4DSQ) [2] was used to measure somatisation in NESDA. While experiencing one or few physical symptoms is considered normal, experiencing multiple unexplained physical symptoms from different organ systems indicates somatisation [1,2]. The 4DSQ somatisation subscale operationalises somatisation as a high number and frequency of physical symptoms and has been validated against various measures (e.g. somatisation subscale of the SCL-90; GP's diagnoses) and in varying samples, including psychiatric and primary care samples [2]. This subscale consists of 16 symptoms (e.g. during the past week did you suffer from dizziness; painful muscles; headache), which are scored on a 5-point Likert scale ('no', 'sometimes', 'regularly', 'often', and 'very often or constantly'). Responses were recoded ('no' = 0; 'sometimes' = 1; 'regularly' to 'very often or constantly' = 2) and item scores were summated to calculate scale scores (range 0–32) [2]. Missing items were replaced by the mean of the available items, under the condition that no more than 6 out of 16 items were missing. No overall score could be calculated for 3 out of 614 participants (0.5%). These participants were excluded from the analyses. An overall score of 11 or higher is considered to be an indication of an elevated level of somatisation as well as an elevated risk of impaired functioning. A score of 21 or higher indicates a highly elevated score and a very high risk of impaired functioning [2].

Demographic variables

Age, gender, number of years of education, and work status were assessed by self-report questionnaires.

Chronic somatic disorders

We also adjusted for the potential confounding effects of chronic somatic disorders (yes/no) in our analyses as these may be related to both the reporting of somatic symptoms and the occurrence of depression or anxiety [19]. Participants were asked to indicate whether they suffered from any of the chronic somatic disorders listed in the interview and

whether they were receiving any medication or treatment for their disorder. This interview instrument was designed for the Netherlands Study of Depression and Anxiety. Only disorders for which treatment or medication was needed were included. The disorders included: lung disease, heart conditions, diabetes, stroke, arthritis, cancer, ulcers, intestinal disorders, liver disease, epilepsy, thyroid disease, and other chronic diseases. So-called functional somatic disorders (i.e. chronic fatigue syndrome, irritable bowel syndrome and fibromyalgia) were not included as these primarily consist of medically unexplained symptoms, which are the focus of our study.

Depressive and anxiety symptoms

As noted in the Introduction section, symptoms of depression and anxiety are conceptually related to somatisation, and many depression and anxiety measures in their turn tend to cover somatic items. In addition, high but subclinical levels of symptoms of depression and anxiety are among the most important risk factors for the onset of depressive and anxiety disorders [20]. Therefore we adjusted for baseline symptoms of depression and anxiety in our analyses. Depressive symptoms were measured using the validated Quick Inventory of Depressive Symptomatology (QIDS) [21]. The QIDS consists of sixteen items covering mood, cognitive and only few somatic features of a depressive episode. In our study sample, the QIDS correlated less with the 4DSQ somatisation scale than the full IDS ($r = 0.58$ versus $r = 0.67$), the latter including more items on somatic features. The items are scored on a scale of 0 to 3, where zero indicates the absence of the symptom in question. Items are summed to obtain a total score ranging from 0–27. Missing items were replaced by the mean of the available items, under the condition that no more than 3 out of 16 QIDS items were missing. A higher score is indicative of more depressive symptoms. Anxiety symptoms were measured with the subjective scale of the validated Beck Anxiety Inventory (BAI), which consists of seven items covering subjective and cognitive features of anxiety [22,23]. Items are scored (0 to 3) on a 4-point Likert scale and added to obtain a total score, ranging from 0–21 [22]. Again, a higher score is indicative of more anxiety symptoms. The subjective scale of the BAI was used because of its lower correlation ($r = 0.48$, $p < .001$) with the 4DSQ somatisation scale compared to the complete BAI ($r = 0.69$, $p < .001$), which also includes items on somatic features of anxiety. Accordingly, the somatic subscale of the BAI covers only these somatic features of anxiety.

Statistical analyses

Descriptive statistics were used to describe the sample characteristics. Besides, Pearson correlations between baseline symptoms of somatisation, depression and anxiety were calculated.

The associations between somatisation and the occurrence of a subsequent depressive or anxiety disorder were quantified with Hazard Ratios (HRs). Cox regression analyses were used because they account for differences in time-at-risk (i.e. person time), as inevitably there was some loss-to follow-up. In all of the analyses, the somatisation score was entered into the regression model as the central determinant of the incidence of a depressive or anxiety disorder during the follow-up period. The possible confounding effects of sociodemographic (age, gender, education level, and work status), somatic (presence of chronic disease), and psychiatric characteristics (subclinical depressive or anxiety symptoms) were investigated by including these variables in the regression model in a stepwise manner. Analyses were performed with SPSS (version 20) statistical software.

Results

Participants' characteristics

The baseline sample characteristics are presented in Table 1. Twelve percent of the sample of 611 persons scored at or above the cut-off score of 11 on the 4DSQ somatisation scale, indicating an elevated level of somatisation and being at risk for impaired functioning. Only 5 participants (0.8%) scored in the highest range (21–32). Mean somatisation scores at baseline were low [M (SD) = 5.0 (4.6)]. Somatisation was moderately correlated with anxiety symptoms ($r = 0.48$, $p < .001$) and depressive symptoms ($r = 0.58$, $p < .001$) at baseline.

Attrition analyses showed no significant differences between participants ($n = 611$) and non-participants ($n = 41$) for age [M (SD) = 41.0 (14.6) vs. 43.1 (16.2); t (650) = .91, $p = .36$], gender [female: 61% vs. 68%; χ^2 (1) = .89, $p = .35$] and anxiety symptoms at baseline [M (SD) = 1.35 (1.97) vs. 2.18 (3.20); $t(38.8) = 1.58$, $p = .12$.] There was a slight, but statistically significant difference in number of years of education [M (SD) = 12.9 (3.21) vs. 11.2 (2.43); $t(49.9) = -4.20$, $p < .001$], and depressive symptoms at baseline [M (SD) = 3.42 (3.09) vs. 4.53 (3.42); $t(647) = 1.90$, $p = 0.03$].

During the 4-year follow-up, 95 participants (16%) became depressed and/or anxious: 47 participants (8%) developed a depressive disorder, 24 participants (4%) developed an anxiety disorder and 24 participants (4%) developed both a depressive and an anxiety disorder.

Somatisation as a predictor for an incident depressive or anxiety disorder

The Hazard Ratios (HRs) of the association between somatisation and the occurrence of an incident depressive disorder (with or without anxiety), incident anxiety disorder (with or without depression), and depressive and/or anxiety disorder during follow-up are displayed in Table 2. This table shows that somatisation significantly increased the risk of a subsequent incident depressive disorder as one point increase in the somatisation score increased the incidence rate of a depressive disorder by 13% (HR = 1.13; 95% CI = 1.09 to 1.17; $p < .001$). After adjusting for baseline levels of depressive symptoms as measured by the QIDS, the association of somatisation with the onset of depressive disorders was reduced but remained statistically significant (HR = 1.06; 95% CI = 1.01 to 1.12; $p = .032$). Higher levels of somatisation at baseline also significantly increased the incidence rate of an anxiety disorder (HR = 1.14; 95% CI = 1.09 to 1.18; $p < .001$, adjusted HR = 1.13; 95% CI = 1.07 to 1.20; $p < .001$). Similar results were found for the onset of depressive and/or anxiety disorders: having a higher somatisation score at baseline increased the risk of becoming depressed or anxious (HR = 1.14; 95%CI = 1.10 to 1.17; $p < .001$). Again, the Hazard Ratio decreased after adjusting for baseline levels of depressive and anxiety symptoms but remained statistically significant (HR = 1.07; 95%CI = 1.02 to 1.13; $p < .01$). The positive association between somatisation and onset of depressive and/or anxiety disorders is also shown in Fig. 1. The incidence of depressive and/or anxiety disorders was higher in high somatisation groups.

Discussion

In this study, we prospectively investigated somatisation as a risk factor for the onset of incident depressive or anxiety disorders as determined by a clinical interview. We found that somatisation increased the risk of a subsequent incident depressive or anxiety disorder during four years, over and above baseline depressive and anxious symptoms.

Methodological considerations

Since the NESDA cohort study includes detailed information on the history, phenomenology and correlates of these disorders and multiple assessments of depressive and anxiety disorders using clinical interviews, it offers a unique opportunity to prospectively investigate the relationship between somatisation and the onset of depressive and anxiety disorders. However, some methodological considerations should be mentioned. At baseline, we had information on only the presence and extent, but not on the history, duration and course of

Table 1
Baseline characteristics of study sample ($n = 611$).

Baseline characteristics	All participants $n = 611$	Depressive and/or anxiety disorder during follow-up $n = 95$	No depressive or anxiety disorder during follow-up $n = 516$
Age, *M* (*SD*)*	41.0 (14.6)	36.5 (13.8)	41.8 (14.6)
Gender: female, *n* (%)	372 (61%)	65 (68%)	307 (60%)
Education level: years, *M* (*SD*)	12.9 (3.2)	12.7 (3.1)	13.0 (3.2)
Work status, *n* (%)*			
Working	445 (73%)	58 (61%)	387 (75%)
Sickness benefit/occupational disabled	25 (4%)	7 (7%)	18 (4%)
Not working	141 (23%)	30 (32%)	111 (22%)
Chronic condition: yes, *n* (%)	207 (34%)	26 (27%)	181 (35%)
Depressive symptoms: QIDS, *M* (*SD*)*	3.42 (3.09)	6.16 (4.12)	2.91 (2.56)
Anxiety symptoms: BAI subjective scale, *M* (*SD*)*	1.35 (1.97)	2.53 (2.50)	1.14 (1.76)
Somatisation: 4DSQ, *M* (*SD*)*	5.04 (4.65)	8.66 (5.87)	4.37 (4.04)

* Significant difference ($p < .05$) between participants with and participants without incident depressive and/or anxiety disorder during 4-year follow-up.

somatisation. The symptoms reported by the participants were not checked by a doctor to verify whether they could be explained by a somatic disorder. However, we adjusted for the presence of chronic somatic disorders that may explain symptoms in our analyses, and this had no effect on the strength of the hazard ratios. Also, time to onset of depressive and anxiety disorders was roughly determined. We repeated the analyses without including time to onset, but this did not change our results. While there was, unavoidably, some attrition in our study, analyses showed that differences between participants and non-participants were small. Furthermore, inherent to the NESDA sampling methods [17], our sample consisted of persons at high risk of depressive and/or anxiety disorders as well as healthy controls. As a result, this study reported higher incidence rates of depressive and anxiety disorders compared to the general population [24].

Our findings in context of the literature

Previous research has found a strong concurrent relationship between somatisation and depression [3,5,9]. Bekhuis et al. (2014) [5] for example found that all types of depressive and anxiety disorders, except for dysthymic disorder, were independently associated with all clusters of concurrent somatic symptoms. Somatisation also has negative prognostic value as co-occurrence is predictive of a poorer treatment response in depressed patients [11]. Several underlying mechanisms have been proposed that may explain how these conditions are related: 1) depression and anxiety may be a reaction to somatisation, 2) somatisation may be part of, or a consequence of depression and anxiety, and 3) that all of these conditions are just different expressions and dimensions of a common underlying form of distress [25]. From previous cross-sectional studies no conclusion can be drawn about these mechanisms. Findings pertaining to the first proposition, which can be considered to be the focus of the current study, have thus far been mixed. A study in a community sample found that after correction for depressive symptoms at baseline, somatisation marginally predicted subsequent depressive symptoms at 5-year follow-up in non-depressed women, but were not predictive in non-depressed men [13]. A prospective community cohort study in the UK reported that only a persistently high somatic symptom count was predictive of poor self-reported mental health but a high somatic symptom count at baseline was not predictive [19]. Although each study has its methodological limitations, an important restriction across the board is that, unlike in our study, psychiatric history of depression or anxiety was either not or not adequately taken into consideration. Thus, it was impossible to discriminate between recurrence and incidence, but it can also confound the association between somatisation and subsequent depression or anxiety as psychiatric history is an important predictor of these disorders [20]. In our study, which was restricted to participants without a lifetime history of a depressive or anxiety disorder, we found that somatisation prospectively predicted these disorders above and beyond baseline levels of depressive or anxiety symptoms, and, therefore, provided support for the first mechanism mentioned above. Our results may, however, also be explained by the third mechanism, which postulates that depressive disorders, anxiety disorders and somatisation are different expressions of a common underlying construct.

Somatisation and symptoms of depression and anxiety

Commonly used measures for depressive and anxiety symptoms such as the IDS and BAI also assess somatic symptoms that accompany or are part of depression and anxiety. To minimise difficulties in

Table 2
Hazard Ratios for incidence of depressive disorder, anxiety disorder, and depressive and/or anxiety disorder at follow-up as predicted by baseline somatisation score ($n = 611$).

	Depressive disorder HR (95% CI)[a]	Anxiety disorder HR (95% CI)[a]	Depressive and/or anxiety disorder HR (95% CI)[a]
Somatisation Crude association, per unit increase	1.13 (1.09 to 1.17)*	1.14 (1.09 to 1.18)*	1.14 (1.10 to 1.17)*
Somatisation adjusted for age, gender, education, working status	1.14 (1.10 to 1.19)*	1.17 (1.11 to 1.23)*	1.15 (1.11 to 1.19)*
Somatisation + chronic somatic disorder	1.15 (1.10 to 1.20)*	1.17 (1.11 to 1.23)*	1.16 (1.11 to 1.20)*
Somatisation + depressive and/or anxiety symptoms	1.06 (1.01 to 1.12)*	1.13 (1.07 to 1.20)*	1.07 (1.02 to 1.13)*

HR = Hazard Ratio.
95% CI = 95% confidence interval.
[a] During the 4-year follow-up, 95 participants became depressed and/or anxious: 71 participants developed a depressive disorder and 48 participants developed an anxiety disorder.
* $p < .05$.

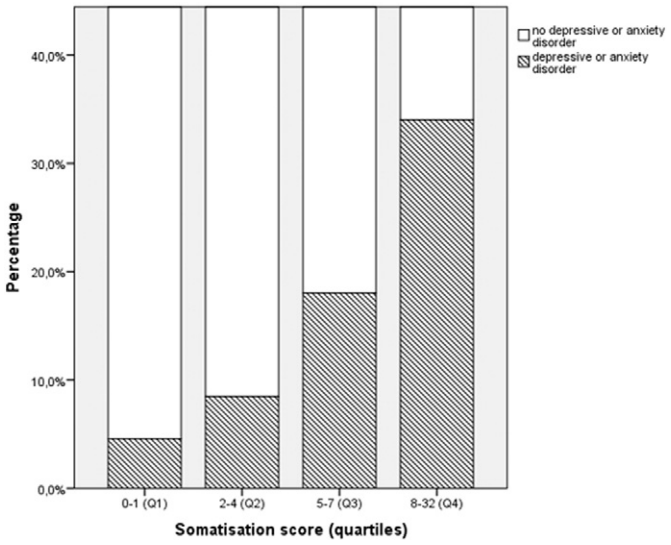

Fig. 1. Incidence of depressive and/or anxiety disorders during 4-year follow-up (%), classified according to somatisation score ($n = 611$).

investigating the relationship between somatisation, depression and anxiety, we used the QIDS, which includes fewer items on somatic features of depression and the subjective scale of the BAI which only focuses on subjective and cognitive aspects of anxiety. Furthermore, the majority of somatisation measures currently available operationalise somatisation as an increased somatic symptom count (e.g. PHQ-15, SCL-90 somatisation subscale, 4DSQ somatisation subscale). Although somatic symptom count is certainly a core feature of somatisation, it is probably not very specific. Increased somatic symptom count as measured with somatisation scales may, in this case, be found to predict depression and anxiety, but questions remain. Is it actually the 'somatisation' (i.e. "the tendency to experience and communicate somatic stress and symptoms unaccounted for by pathological findings, to attribute them to physical illness, and to seek medical help for them" [1], p. 1359) that is predictive. To which condition can we attribute the somatic symptoms? In line with DSM V [26,27], a step forward may be to operationalise and develop more specific measures of somatisation, which include cognitive criteria besides listing somatic symptoms [28,29]. The concept and operationalisation of somatisation as well as its conceptual relationship with depressive and anxiety symptoms remain an important point of consideration and future study.

Conclusions

High physical symptom levels (somatisation) predict incident depressive and anxiety disorders during four years, over and above baseline levels of depressive or anxiety symptoms, in a study sample without a lifetime history of depressive and anxiety disorders. Somatisation scores may, therefore, deserve extra clinical attention in primary care. The 4DSQ offers such a symptom tool and is increasingly used as such in countries around the world. Moreover, persons with high somatisation scores represent a potential target group for prevention of depressive and anxiety disorders as they have a higher risk of becoming depressed or anxious. In primary care, for instance, persons with high somatisation scores could be monitored more closely in shared care models with mental health nurses or start treatment, according to recent primary care guidelines [30].

Competing interests

The authors have no competing interests to report.

Acknowledgments

The infrastructure for the NESDA study (www.nesda.nl) has been funded through the Geestkracht program of the Netherlands Organisation for Health Research and Development (Zon-Mw, grant number 10-000-1002) and participating universities (VU University Medical Center, Leiden University Medical Center, University Medical Center Groningen).

References

[1] ZJ Lipowski, Somatization: the concept and its clinical application, Am. J. Psychiatry 145 (1988) 1358–1368.
[2] B Terluin, HW van Marwijk, HJ Ader, HC de Vet, BW Penninx, ML Hermens, et al., The Four-Dimensional Symptom Questionnaire (4DSQ): a validation study of a multidimensional self-report questionnaire to assess distress, depression, anxiety and somatization, BMC Psychiatry 6 (2006) 34.
[3] K Kroenke, Patients presenting with somatic complaints: epidemiology, psychiatric comorbidity and management, Int. J. Methods Psychiatr. Res. 12 (2003) 34–43.
[4] GE Simon, M VonKorff, M Piccinelli, C Fullerton, J Ormel, An international study of the relation between somatic symptoms and depression, N. Engl. J. Med. 341 (1999) 1329–1335.
[5] E Bekhuis, L Boschloo, JGM Rosmalen, RA Schoevers, Differential associations of specific depressive and anxiety disorders with somatic symptoms, J. Psychosom. Res. 78 (2015) 116–122.
[6] EX Lau, RM Rapee, Prevention of anxiety disorders, Curr. Psychiatry Rep. 13 (2011) 258–266.
[7] RF Munoz, P Cuijpers, F Smit, AZ Barrera, Y Leykin, Prevention of major depression, Annu. Rev. Clin. Psychol. 6 (2010) 181–212.
[8] A Tylee, P Gandhi, The importance of somatic symptoms in depression in primary care, Prim. Care Companion J. Clin. Psychiatry 7 (2005) 167–176.
[9] MJ Bair, RL Robinson, W Katon, K Kroenke, Depression and pain comorbidity: a literature review, Arch. Intern. Med. 163 (2003) 2433–2445.
[10] R Lieb, G Meinlschmidt, R Araya, Epidemiology of the association between somatoform disorders and anxiety and depressive disorders: an update, Psychosom. Med. 69 (2007) 860–863.
[11] KM Huijbregts, HW van Marwijk, FJ de Jong, B Schreuders, AT Beekman, CM van der Feltz-Cornelis, Adverse effects of multiple physical symptoms on the course of depressive and anxiety symptoms in primary care, Psychother. Psychosom. 79 (2010) 389–391.
[12] K van Boven, P Lucassen, H van Ravesteijn, T Olde Hartman, H Bor, E van Weel-Baumgarten, et al., Do unexplained symptoms predict anxiety or depression? Ten-year data from a practice-based research network, Br J Gen Pract 61(587) (2011) e316–e325.
[13] L Terre, WS Poston, J Foreyt, ST St Jeor, Do somatic complaints predict subsequent symptoms of depression? Psychother. Psychosom. 72 (2003) 261–267.
[14] P Skapinakis, G Lewis, V Mavreas, Temporal relations between unexplained fatigue and depression: longitudinal data from an international study in primary care, Psychosom. Med. 66 (2004) 330–335.
[15] G Van Der Linden, T Chalder, I Hickie, A Koschera, P Sham, S Wessely, Fatigue and psychiatric disorder: different or the same? Psychol. Med. 29 (1999) 863–868.
[16] AM Addington, JJ Gallo, DE Ford, WW Eaton, Epidemiology of unexplained fatigue and major depression in the community: the Baltimore ECA follow-up, 1981–1994, Psychol. Med. 31 (2001) 1037–1044.
[17] BW Penninx, AT Beekman, JH Smit, FG Zitman, WA Nolen, P Spinhoven, et al., The Netherlands Study of Depression and Anxiety (NESDA): rationale, objectives and methods, Int. J. Methods Psychiatr. Res. 17 (2008) 121–140.
[18] HU Wittchen, Reliability and validity studies of the WHO — Composite International Diagnostic Interview (CIDI): a critical review, J. Psychiatr. Res. 28 (1994) 57–84.
[19] FH Creed, I Davies, J Jackson, A Littlewood, C Chew-Graham, B Tomenson, et al., The epidemiology of multiple somatic symptoms, J. Psychosom. Res. 72 (2012) 311–317.
[20] J Karsten, CA Hartman, JH Smit, FG Zitman, AT Beekman, P Cuijpers, et al., Psychiatric history and subthreshold symptoms as predictors of the occurrence of depressive or anxiety disorder within 2 years, Br. J. Psychiatry 198 (2011) 206–212.
[21] AJ Rush, MH Trivedi, HM Ibrahim, TJ Carmody, B Arnow, DN Klein, et al., The 16-Item Quick Inventory of Depressive Symptomatology (QIDS), clinician rating (QIDS-C), and self-report (QIDS-SR): a psychometric evaluation in patients with chronic major depression, Biol. Psychiatry 54 (2003) 573–583.
[22] AT Beck, N Epstein, G Brown, RA Steer, An inventory for measuring clinical anxiety: psychometric properties, J. Consult. Clin. Psychol. 56 (1988) 893–897.
[23] RI Kabacoff, DL Segal, M Hersen, VB Van Hasselt, Psychometric properties and diagnostic utility of the Beck Anxiety Inventory and the State-Trait Anxiety Inventory with older adult psychiatric outpatients, J. Anxiety Disord. 11 (1997) 33–47.
[24] R de Graaf, M ten Have, M Tuithof, S van Dorsselaer, First-incidence of DSM-IV mood, anxiety and substance use disorders and its determinants: results from the Netherlands Mental Health Survey and Incidence Study-2, J. Affect. Disord. 149 (2013) 100–107.
[25] P Henningsen, T Zimmermann, H Sattel, Medically unexplained physical symptoms, anxiety, and depression: a meta-analytic review, Psychosom. Med. 65 (2003) 528–533.
[26] J Dimsdale, F Creed, The proposed diagnosis of somatic symptom disorders in DSM-V to replace somatoform disorders in DSM-IV — a preliminary report, J. Psychosom. Res. 66 (2009) 473–476.

[27] American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders: DSM-5, Fifth ed. American Psychiatric Association, Washington, DC, 2013.
[28] W Rief, E Broadbent, Explaining medically unexplained symptoms-models and mechanisms, Clin. Psychol. Rev. 27 (2007) 821–841.
[29] W Rief, R Mewes, A Martin, H Glaesmer, E Braehler, Are psychological features useful in classifying patients with somatic symptoms? Psychosom. Med. 72 (2010) 648–655.
[30] TC Olde Hartman, AH Blankenstein, AO Molenaar, D Bentz van den Berg, HE Van der Horst, IA Arnold, et al., NHG-Standaard Somatisch Onvoldoende verklaarde Lichamelijk Klachten (SOLK) [NHG guideline on medically unexplained symptoms (MUS)], Huisarts Wet 56(5) (2013) 222-30.