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Prevalence and structure of anxiety-depression in an Australian community sample

Christopher F. Sharpley, Vicki Bitsika, Emmanuel Jesulola, Linda L. Agnew

Summary

Aims: To describe the prevalence and factor structure of anxiety-depression in a community sample and to derive indicators for treatment planning.

Method: A sample of 398 members of the electoral roll for the New England region of Australia were recruited at random and completed the Zung Self-Rating Anxiety Scale and the Zung Self-Rating Depression Scale.

Results: The prevalence of anxiety-depression was 28.1%, over twice that for either anxiety or depression alone. The anxiety-depression construct comprised four underlying factors: cognitive agitation and depressed mood, pessimism, cardiovascular reactivity, pain and sleep disturbance. There were different patterns of these four factors across anxiety-only, depression-only and combined anxiety-depression, with evidence of a unique symptomatological profile in participants with clinically significant levels of anxiety-depression.

Conclusions: Treatment decisions for anxiety and depression need to go beyond consideration of the two disorders separately to include the underlying factor severity of the combined construct of anxiety-depression.

community/mental health/factor analysis/anxiety-depression/anxiety-depression comorbidity

Anxiety and depression are associated with increases in physical disease, relationship problems and cognitive difficulties [1] as well as elevated risk of suicide [2,3]. For example, anxiety can be a precursor of low-level illnesses acting via prolonged arousal of the hypothalamic-pituitary-adrenal (HPA) axis [4] and the cardiovascular, metabolic and immune systems [5]. Chronic anxiety has been demonstrated to precede emotional and behavioral problems such as demoralization, hostility and mistrust [6], impaired concentration, fatigue and confusion [7], and feelings of helplessness and depression [8]. It can also elevate the risk of physical problems such as coronary heart disease [9] and depletion of the immune system [10]. Depression is a major contributor to the total disease burden [11] and has greater adverse effects on personal health [12] and higher costs of care [13] than other chronic diseases. It is also associated with suicide in about 15% of all depressed patients [14] and carries a similar risk for mortality from all causes as does smoking, even when related health factors such as blood pressure, alcohol intake, cholesterol and social status are taken into account [15]. Recent meta-analytic data indicate that people with a mood disorder have a relative risk of mortality from all causes that is 1.86 times that for individuals without depression and that there are 2.74 million deaths annually from depression [16].

Christopher F. Sharpley[1], Vicki Bitsika[2], Emmanuel Jesulola[1], Linda L. Agnew[1]: [1]Brain-Behaviour Research Group, University of New England, Armidale, New South Wales, Australia; [2]Centre for Autism Spectrum Disorders, Bond University, Gold Coast, Queensland, Australia.

Correspondence address: csharpl3@une.edu.au

In addition to these effects from anxiety and depression as separate entities, they are often comorbid, with about half of all people who fulfill the criteria for major depression [14] also meeting the criteria for major anxiety [3,14] . Anxiety-depression comorbidity is associated with more frequent medical symptoms [17], greater risk of heart disease [18], delayed recovery, poorer quality of life, as well as increased suicide risk [19] and suicide attempts [20]. Even though they are most commonly examined as separate disorders in comorbidity studies, it has been suggested that the distinction between anxiety and depression is artificial and solely due to limitations engendered by a categorical model of disorder [21]. Anxiety and depression share the common symptoms of fatigue [14,22] and irritability [14] and may also be causally linked, with elevated anxiety increasing the risk of developing depression [23]. Consequently, several models of mixed anxiety-depression have been proposed, so many that one review described this heterogeneity in definition as 'leading to clinical confusion and inconsistencies in the literature' [24: p. 252]. Although diagnostic procedures have included mixed anxiety-depression for some time [25], the DSM–5 [14] has changed that diagnosis to major depressive disorder (MDD) with anxious distress as a specifier for depressive disorders. However, that diagnosis requires only two of the diagnostic criteria for generalized anxiety disorder (GAD) and is not the same as comorbidity of major anxiety and depression.

An alternative to adopting one of these definitions of mixed anxiety-depression disorder, or depression with anxious features (which uses truncated versions of GAD), is to use a combined metric of anxiety-depression symptomatology in research studies of anxiety-depression. That is, as well as the total scores on separate scales of anxiety and depression, the total score on a combination of the two scales could help provide a metric that encompasses both sets of symptoms. In addition to this, a total score of anxiety-depression is of potential value in understanding the form or structure of the combined metric to examine the underlying factor structure of anxiety-depression, so that a more detailed model of the combined form of anxiety-depression can be developed.

Several studies have been made of anxiety and depression in Australia (e.g. Wilhelm et al. [26],Andrews et al. [27]), with the prevalence of each disorder at about 10% [28]. Although the association between depression and demographic or physical disease factors has been described [26], and comorbid anxiety elevates the risk of depression-related suicide [20], the prevalence and structure of anxiety-depression comorbidity of Australian adults has not been previously reported. Because of the implications that such comorbidity has for the various health-related outcomes mentioned above, the current study examined the prevalence of anxiety-depression comorbidity in an Australian sample the underlying factors in anxiety-depression and total scale scores, and aimed to describe the nature of anxiety-depression and its prevalence.

METHOD

Participants

Participants were residents of the New England region of New South Wales, Australia who were over the age of 18. All were in sufficient good health to be able to complete the questionnaires for anxiety and depression and all were of satisfactory mental capacity to responsibly accept the invitation to participate in this study. Although the study was described as focused on mental health, invitation letters and publicity material emphasized that individuals who had not experienced mental health problems were invited in order to recruit a community sample.

Instruments

Background questionnaire

The questionnaire gathered information regarding participants' age and gender and whether they were currently receiving treatment for anxiety or depression.

Zung Self-Rating Anxiety Scale [29] (SAS)

The 20-item SAS is a measure of state anxiety during the two weeks prior to assessment and includes the current symptoms for GAD [14].

Total raw scores range from 20 to 80, with higher scores indicative of greater anxiety. The SAS correlates 0.75 with the Hamilton Anxiety Scale [29] and significantly discriminates between normal adults and patients with anxiety disorders [29]. Reliability data are 0.71 [29] and 0.79 in an Australian sample [30]. SAS raw scores above 36 indicate the presence of clinically significant anxiety, defined as anxiety severe enough to warrant further assessment and treatment planning [31].

Zung Self-Rating Depression Scale [32] (SDS)

The SDS has 20 items that were identified in factor analytic studies of the syndrome of depression and which underlie the DSM definition of MDD [14]. Raw scores range from 20 to 80, with higher scores indicative of more severe depression. The SDS has split-half reliability of 0.81 [32]. The SDS has been shown to be superior to the MMPI Depression Scale and the Beck Depression Inventory for assessing depression in male psychiatric in-patients [33]. SDS scores of 40 or above indicate the presence of clinically significant depression [32], requiring further clinical investigation. SDS and SAS raw scores were used in this study.

Procedure

A random sample of 398 participants was selected from the New England Electoral Roll (Australian Electoral Commission, 2013). Potential participants were able to respond to initial media publicity or a personal invitation letter by either telephone or email. They then received information about an online portal to access the questionnaires, or a copy of the questionnaire booklet, depending on their preference. Participants' data were de-identified and analysed via SPSS. All tests were two-tailed. Ethical approval for this study was obtained from a relevant human research ethics committee.

RESULTS

The sample had a mean age of 48.2 years (SD=18.6, range 18–101), and included 151 males (37.9%) and 247 females (62.1%). There was no significant difference in age between males (M=50.1 years, SD=19.7) and females (M=47.6 years, SD=17.7; $F(1,391)=2.332$, $MSE=331.621$, $p=0.128$, $\eta^2=0.006$). Participants' scores on the SAS and SDS were significantly correlated ($r=0.727$, $p<0.001$) and there were significant inverse correlations between age and SAS ($r=–0.262$, $p<0.001$) and SDS ($r=–0.170$, $p<0.001$), although these were weak [34].

The sample's mean SAS total score was 37.886 (SD=8.515, range 21–74) and mean SDS total score was 34.249 (SD=8.617, range 20–69). The SAS–SDS combined total mean score was 72.146 (SD=15.915, range 42–140). Females had significantly higher SAS scores (M=35.38, SD=8.55) than males (M=32.36, SD=8.03; $F(1, 296)=8.77$, $MSE=70.16$, $p=0.003$) but the effect size was small ($\eta^2=0.02$). There was no significant difference between the SDS total scores for the males (M=40.96, SD=8.64) and the females (M=42.55, SD=8.54; $F(1, 296)=2.32$, $MSE=73.65$, $p=0.128$, $\eta^2=0.008$). There was a marginal difference between the females' (M=73.37, SD=16.16) and males' (M=70.13, SD=15.35) SAS–SDS total scores ($F(1,396)=3.910$, $p=0.049$) but the effect size was very small ($\eta=0.010$). Tests for normality of the SAS and SDS total scores were significant (Kolgomorov–Smirnov statistics 0.08, $p<0.001$ and 0.00, $p<0.001$ respectively), which is not unusual in large samples [35]. The distributions were slightly skewed to the lower ends of each scale (see Figure 1) but the normal Q-Q plots were almost completely straight lines. Skewness does not make a substantial difference in the parametric analysis of samples of 200 or more [35] and so no normalization of the raw data was undertaken.

Zung Self-Rating Depression Scale.

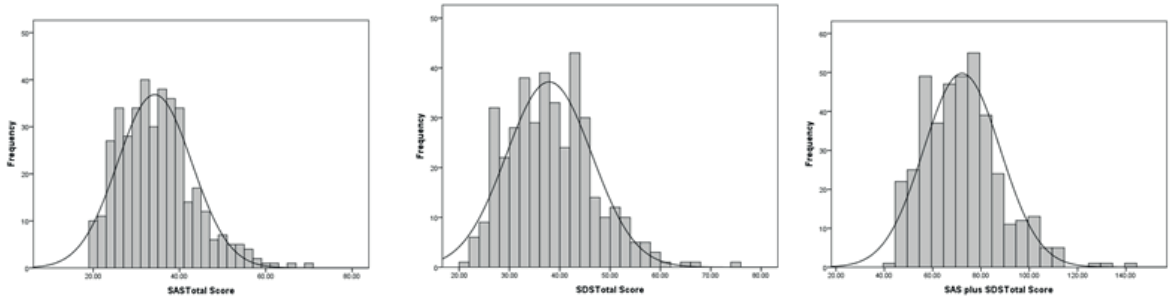

Figure 1. Distributions for (a) SAS (Zung Self-Rating Anxiety Scale), (b) SDS (Zung Self-Rating Depression Scale) and (c) SAS–SDS combined total scores.

In response to the background questionnaire, 33 (8.3%) participants indicated that they were currently receiving treatment for anxiety and 38 (9.5%) were receiving treatment for depression; 25 participants (6.3%) were receiving treatment for both anxiety and depression. As might be expected, there were significant Spearman's correlations between each of these categories of treatment and SAS, SDS and SAS–SDS total scores (all $p<0.001$).

Using Zung's [31,32] suggestions for identification of clinically significant anxiety described above, Figure 2 shows the relative percent prevalence for those participants who had SAS and SDS scores that were in the clinically significant range for each of the six SAS and SDS subgroups (i.e. (1) neither SAS nor SDS at clinically significant levels, (2) SAS at clinically significant levels, (3) SDS at clinically significant levels, (4) both SAS and SDS at clinically significant levels, (5) SAS but not SDS at clinically significant levels, (6) SDS but not SAS at clinically significant levels).

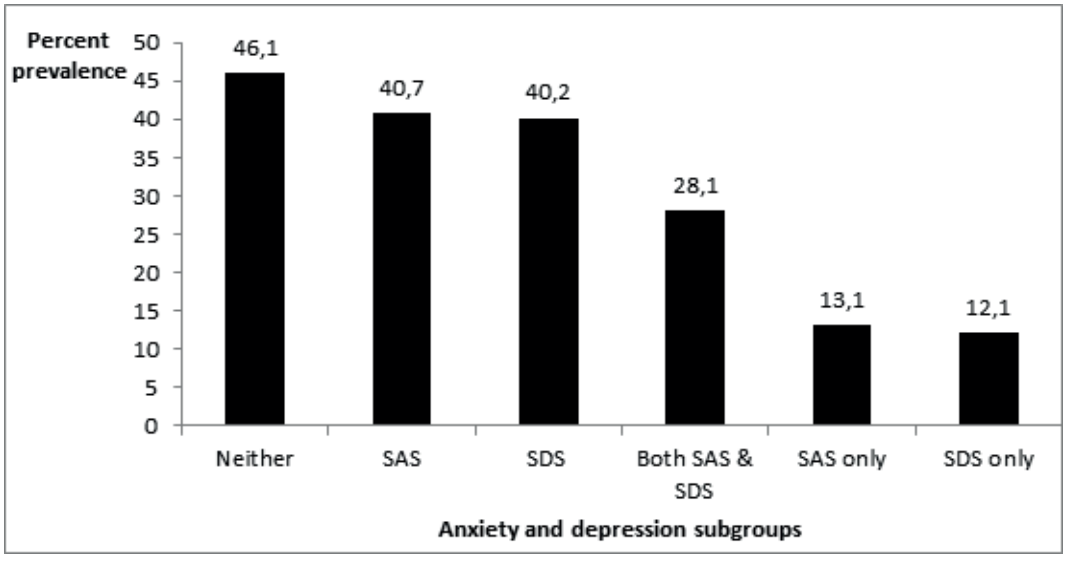

Figure 2. Prevalence of clinically significant anxiety and depression. SAS, clinically significant anxiety (Zung Self-Rating Anxiety Scale); SDS, clinically significant depression (Zung Self-Rating Depression Scale).

Because there are no previous reports of the factor structure of the SAS–SDS within this population and because factor structures may vary across samples [36], exploratory factor analysis was used to identify the underlying structure of the SAS–SDS construct. As well as meeting the sample size requirements of approximately 10 participants per scale item [37], there were many inter-item correlations greater than 0.3, the KMO (Kaiser-Meyer-Olkin) measure of sampling adequacy was 0.89 and Bartlett's test of sphericity was significant ($\chi^2(465)=4949.23$, $p<0.001$), thus justifying factor analysis with these data. Principal components extraction, inspection of the scree plot and parallel analysis revealed four factors, with inter-factor correlations of between

0.125 and 0.331, arguing for a relatively discrete factorial structure. Oblimin rotation confirmed this solution, with all four factors accounting for 46.53% of the variance (factor 1 eigenvalue 9.06, 29.24%; factor 2 eigenvalue 2.07, 6.67%; factor 3 eigenvalue 1.84. 5.94%; factor 4 eigenvalue 1.45, 4.64%). Applying the guidelines for identifying factors by the relative strength of item loadings [38], factor 1 was identified as 'cognitive agitation and depressed mood', factor 2 as 'pessimism', factor 3 as 'cardiovascular reactivity', and factor 4 as 'pain and sleep disturbance'.

Table 1. Pattern matrix factor structure of the SAS–SDS construct

SAS–SDS items			Factors	
	Factor 1	Factor 2	Factor 3	Factor 4
SAS 1: I feel more nervous and anxious than usual	0.75	0.06	0.05	0.00
SAS 2: I feel afraid for no reason at all	0.74	0.08	0.08	0.02
SAS 3: I get upset easily or feel panicky	0.82	0.02	0.05	0.02
SAS 4: I feel like I'm falling apart and going to pieces	0.77	0.03	0.10	0.02
SAS 5: I feel that everything is alright and nothing bad will happen	0.08	0.43	0.06	0.26
SAS 6: My arms and legs shake and tremble	0.36	0.01	0.44	0.05
SAS 7: I am bothered by headaches, neck aches and back pain	0.14	0.15	0.17	0.59
SAS 8: I feel weak and get tired easily	0.46	0.03	0.17	0.39
SAS 9: I feel calm and can sit still easily	0.11	0.29	0.04	0.42
SAS 10: I can feel my heart beating fast	0.35	0.01	0.52	0.02
SAS 11: I am bothered by dizzy spells	0.21	0.05	0.69	0.06
SAS 12: I have fainting spells or feel like it	0.06	0.05	0.74	0.06
SAS 14: I get feelings of numbness and tingling in my fingers and toes	0.04	0.02	0.61	0.19
SAS 15: I am bothered by stomach aches or indigestion	0.05	0.02	0.29	0.48
SAS 19: I fall asleep and get a good night's rest	0.02	0.29	0.10	0.61
SAS 20: I have nightmares	0.45	0.07	0.06	0.21
SDS 1: I feel downhearted and blue	0.78	0.11	0.07	0.08
SDS 3: I have crying spells or feel like it	0.73	0.02	0.04	0.11
SDS 4: I have trouble sleeping at night	0.25	0.15	0.11	0.59
SDS 5: I eat as much as I used to	0.17	0.07	0.23	0.16
SDS 8: I have trouble with constipation	0.06	0.06	0.34	0.47
SDS 9: My heart beats faster than usual	0.38	0.06	0.39	0.02
SDS 10: I get tired for no reason	0.37	0.09	0.11	0.44
SDS 11: My mind is as clear as it used to be	0.08	0.63	0.18	0.03
SDS 12: I find it easy to do the things I used to do	0.08	0.67	0.18	0.15
SDS 13: I am restless and can't keep still	0.40	0.10	0.13	0.32
SDS 14: I feel hopeful about the future	0.05	0.54	0.03	0.13
SDS 15: I am more irritable than usual	0.62	0.04	0.00	0.13
SDS 17: I feel that I am useful and needed	0.19	0.56	0.08	0.06
SDS 18: My life is pretty full	0.14	0.57	0.00	0.06
SDS 19: I feel that others would be better off if I were dead	0.62	0.22	0.06	0.17

Factor 1: cognitive agitation and depressed mood; factor 2: pessimism; factor 3: cardiovascular reactivity; factor 4: pain and sleep disturbance. SAS, Zung Self-Rating Anxiety Scale; SDS,

The relative loadings of the four SAS–SDS factors across the four most relevant, clinically significant subgroups are presented in Figure 3. The dashed line represents the cut-off score for clinical significance (described above) and indicates that the four SAS–SDS factors were not uniformly severe across the four SAS–SDS subgroups. That is, although (as expected) the 'neither' subgroup did not have mean scores for any of the four SAS–SDS factors at the clinically significant level, each of the remaining three subgroups did, and the distribution of those four SAS–SDS factors across the three remaining subgroups was not uniform. For example, although both the SAS-alone and the SDS-alone subgroups had clinically significant scores for pessimism (factor 2) and pain and sleep disturbance (factor 4), the distribution of those two factors across these two subgroups was in the opposite direction, with the SAS-alone subgroup showing higher scores for pain and sleep disturbance than for pessimism, but the SDS-alone subgroup having lower pain and sleep disturbance than pessimism scores. The 'both' subgroup (i.e. both SAS and SDS at clinically significant levels) was characterized by clinically significant scores for three of the four SAS–SDS factors but not for the cardiovascular reactivity factor. These results suggest that the division of participants according to their clinically significant scores on the SAS and SDS also indicated differences in the underlying structure of their clinically significant symptomatology. That is, the presence of anxiety-depression comorbidity was characterized by a distinct underlying symptom structure that could not be encompassed by reference to anxiety or depression separately.

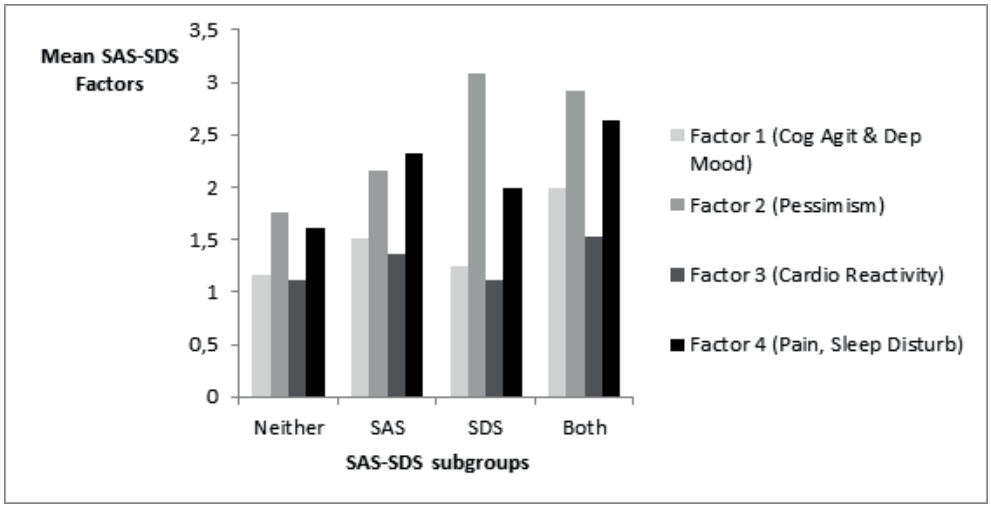

Figure 3. SAS–SDS factors across four subgroups: neither clinically significant anxiety or depression (Neither), clinically significant anxiety only (SAS), clinically significant depression only (SDS), clinically significant anxiety and depression (Both).

DISCUSSION

Two caveats need to be kept in mind before discussing the results of this study. First, neither GAD nor MDD were assessed here and therefore generalizations to those more severe disorders are not implied. Second, perhaps because the recruitment process invited participants who had mental health problems as well as those who did not, a larger than might be expected proportion of the sample did appear to require further assessment and potential treatment planning, as recommended on the basis of Zung's criteria for clinically significant anxiety and depression [31,32]. This may have biased the sample away from being truly representative and therefore any implications for the wider community need to be made with caution. That is, although the prevalence of participants who had clinically significant scores on the SAS or the SDS (Table 2, columns 2 and 3) was high, this should not be considered as equal to GAD or MDD. Rather, Zung [31] described 'clinically significant' anxiety and depression scores from the SAS and SDS

as those which required further assessment with a view to provision of services. Participants in this study with these scores were showing not-insignificant levels of anxiety and/or depression symptoms that would benefit from further investigation by a mental healthcare professional. In terms of this study, these are potential patients for assessment and treatment but the final diagnosis of GAD and/or MDD is yet to occur.

However, lest this non-GAD or non-MDD status be considered trivial, it is similar to the criteria for 'subsyndromal' anxiety or depression. Judd et al. [39] described persons with subsyndromal depression (SSD) as having 'no large consistent differences in impairment' to patients with MDD across eight domains of functioning [40]. People who have SSD have a five-and-a-half-fold chance of developing MDD within 1 year compared with people who have none of the symptoms of MDD at all [41], and they also exhibit significantly greater levels of psychological disability, hopelessness and death ideation [42]. Other data suggest that patients with SSD 'are as ill as those with minor or major depression (in terms of) medical burden' [43: p. 214] and that SSD is prevalent, underdiagnosed and undertreated [44,45]. Although it was not an aim of the present study, the data regarding prevalence of clinically significant anxiety and depression call for a further careful consideration of the levels of subsyndromal anxiety and depression in this community sample. As noted earlier, participants were not recruited for their anxious or depressive states and there is little basis for assuming that only anxious or depressed individuals responded to the call for participation. Because the sample was chosen at random, it may be that there is a prevalence of anxiety and depression in this community sample that would benefit from public health attention.

Therefore, bearing in mind that this study was not designed as an epidemiological survey, and that no attempt is made to generalize from these data to the overall mental health of the community from which data were drawn, it is worth noting that anxiety-depression comorbidity was relatively common in this community sample of Australians, with over a quarter of participants meeting the cut-off for clinically significant scores on the combined SAS–SDS construct. That is, as well as needing to describe anxiety and depression as individual issues in this community sample (with implications for further investigation and allocation of assessment and treatment resources that recognize these disorders as individual constructs), these results also highlight the need to consider the presence of clinically significant anxiety-depression as representing a larger proportion of this sample than either anxiety or depression alone. That consideration needs to be made within the framework of the idiosyncratic structure of the anxiety-depression construct identified here. As well as recognizing the likelihood of an increased prevalence of anxiety-depression in community samples compared with anxiety or depression alone, our finding argues for the provision of services that are targeted towards the specific symptomatologies underlying anxiety-depression, such as the factor structures described here.

Typical treatments for anxiety include pharmacological agents and psychological therapies. Pharmacological agents such as benzodiazepines and selective-serotonin reuptake inhibitors (SSRIs) have been prescribed for anxiety for some time [1,46], but recent review data support the use of pregabalin and quetiapine for the long-term treatment of anxiety [47]. Psychological therapies for anxiety are usually cognitively and/or behaviorally based [48], and may involve relaxation [49] or other concentration treatments such as mindfulness therapy [50]. Combinations of pharmacological and psychological therapies have been shown to be effective, as has exercise [51], with recent recommendations that the choice of treatment should be based on patient preferences and possible interactions or contraindications of various treatment options [51]. Treatment of depression also consists of pharmacological [52] and psychological therapies [53,54], with recent recommendations that there is a need to acknowledge patients' needs and treatment sensitivities [55]. There is emerging evidence that computer-based delivery of psychological treatments can be successful for depression [56] and that stimulation of brain activity is also successful for some patients [57]. Calls for personalised medicine approaches to depression have been supported by data from studies of different depression 'subtypes' [58], based on the conceptualisation that depression is heterogeneous rather than unitary [59].

However, these treatment models are based on the conceptualisation of anxiety and depression as relatively independent disorders, whereas results from this study suggest that the anxiety-depression symptom structure is not the simple sum of the symptoms of anxiety and depression but represents a different diagnosis with specific characteristics and structure. That conclusion is drawn from the differences in the distribution of the SAS–SDS factors across the SAS-only, SDS-only and SAS–SDS subgroups shown in Figure 3, when those factors were defined according to their clinically significant status (i.e. above the dashed line). Those results indicate that it was the relatively high scores for factor 1 (cognitive agitation and depressed mood) that distinguished the anxiety-depression subgroup from the anxiety-alone or depression-alone subgroups. That is, factors 2 (pessimism) and 4 (pain, sleep disturbance) were similarly severe across the three subgroups of participants, whereas factor 1 was relatively low (and not at the clinically significant level defined from Zung's criteria) for those participants who reported high scores for anxiety or depression alone, but it was at the clinically significant level for the anxiety-depression subgroup.

This relatively elevated level of cognitive agitation and depressed mood for those participants who reported clinically significant anxiety-depression provides a different clinical treatment challenge. That is, if treatment regimes for anxiety-depression were founded on those for anxiety or depression as separate disorders then, based on these data, cognitive agitation and depressed mood (factor 1) would not be considered as important as pessimism (factor 2) and pain and sleep disturbance (factor 4). The increased level of factor 1 in participants with high SAS–SDS scores therefore argues for an added treatment planning target – relief of cognitive agitation and depressed mood when dealing with persons who exhibit elevated scores for anxiety-depression. Reference to Table 1 identifies the specific symptoms comprising factor 1 that might form therapeutic 'targets' for people with anxiety-depression comorbidity. Factor 1 reflects a grouping of SAS and SDS symptoms that include: (a) intense fear (SAS items 1–4, 20), (b) physical agitation and fatigue (SAS item 8, SDS items 13, 15), and (c) sadness (SDS items 1, 3, 20). This combination of fear, exaggerated physiological arousal and depressed mood is recognized to some extent in the diagnostic criteria for MDD [14] and has been partially referred to in clinical and research discussions about anxiety-depression being characterized by sadness and anhedonia with some somatic symptoms [14], but it is different to other models such as those which posit a general factor of psychosocial dysfunction, plus specific depression and somatic symptom factors [60], a tripartite model including negative affect (both anxiety and depressive symptoms), anhedonia and somatic arousal [61], or a more detailed six-factor model of the extended tripartite model [62].

The disguising feature of the factor structure data for anxiety-depression in the current study was the presence of intense and unspecified fear, which does not receive attention in the other models mentioned above, and which holds important implications for provision of treatment. That is, applying the current study's findings to treatment planning would entail attention to intense fear that seems to lack specific source (i.e. SAS item 2: 'I feel afraid for no reason at all'). Assessment processes that acknowledged the possible presence of such intense and unspecified fear, and then incorporated it into treatment planning, would differ from those based on the previous models described above. Treatment of fear is most commonly focused on specific events or objects, such as flying [63], spiders [64] or death [65], and uses extinction or exposure models. Because factor 1 in this study includes feeling afraid 'for no reason at all', treatment of this kind of fear via extinction or exposure models may be unsuccessful simply because there is no clear focus for those extinction or exposure procedures. Some other more generalized treatment used in pharmacological models may hold promise for non-identified fear, for instance citalopram, which has been shown to decrease amygdala responses to masked presentations of threat [66]. The delivery of effective treatment models that include non-specific fear as a target for persons suffering from anxiety-depression requires further attention and development.

Limitations and clinical implications

Some of the limitations of this study have been mentioned above (i.e. absence of a formal diag-

nosis of GAD or MDD, some possible bias in the sample), but others include the use of a single application of the SAS and SDS with no data regarding the consistency of the findings reported here over time. Similarly, the sample was recruited from one specific region of Australia and was voluntary, both of which restrict the generalisability of the findings. That limitation is emphasized by the lack of consistency in factor structures across populations [36]. No attempt was made here to compare rural and urban residents but that is a potential topic for further research.

Notwithstanding those limitations, these results provide an insight into the prevalence and nature of anxiety-depression in a community sample. Clinical implications are for the combined assessment of both anxiety and depression, plus the identification of which attributes of those disorders (i.e. the SAS–SDS factor structure) are present in patients' symptom profiles, particularly acknowledging and focusing on the possible presence of intense fear which appears to be unconnected to specific events or stimuli.

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REFERENCES

1. Nutt D. Anxiety and depression: individual entities or two sides of the same coin? Int J Psychiatry Clin Pract. 2004; 8: 19–24.
2. Malone K, Haas G, Sweeney J, Mann J. Major depression and the risk of attempted suicide. J Affect Disord. 1995; 34(3): 173–85.
3. Zimmerman M, McDermut W, Mattia J. Frequency of anxiety disorders in psychiatric patients with major depressive disorder. Am J Psychiatry. 2000; 157: 1337–40.
4. Fries E, Hesse J, Hellhammer J, Hellhammer D. A new view on hypocortisolism. Psychoneuroendocrinology. 2005; 30: 1010–6.
5. Lane J, Adcock R, Burnett R. Respiratory sinus arrhythmia and cardiovascular responses to stress. Psychophysiology. 1992; 29: 461–70.
6. Langewitz W, Ruddell H. Spectral analysis of heart rate variability under mental stress. J Hypertension. 1989; 7: 32–3.
7. Sloan RP, Shapiro PA, Bagiella E, Boni SM, Paik M, Bigger J, et al. Effect of mental stress throughout the day on cardiac autonomic control. Biol Psychol. 1994; 37: 89–99.
8. Sharpley C. Heart rate reactivity and variability as psychophysiological links between stress, anxiety, depression and cardiovascular disease: Implications for Health Psychology interventions. Austral Psychologist. 2002; 37: 56–62.
9. Bosma H, Marmot M, Hemingway H, Nicholson A, Brunner E, Stansfeld S. Low job control and risk of coronary heart disease in Whitehall II (Prospective cohort) study. BMJ. 1997; 317: 558–65.
10. Cohen S, Tyrrell D, Smith A. Psychological stress and susceptibility to the common cold. New Engl J Med. 1991; 325: 606–12.
11. Ustun TB, Ayuso-Mateos JL, Chatterji S, Mathers C, Murray CJL. Global burden of depressive disorders in the year 2000. Br J Psychiatry. 2004; 184: 386–92.
12. Moussavi S, Chatterji S, Verdes E, Tandon A, Patel V, Ustun B. Depression, chronic diseases, and decrements in health; results from the World Health Surveys. Lancet. 2007; 370: 851–8.
13. Langa KM, Valenstein MA, Fendrick AM, Kabeto MU, Vijan S. Extent cost of informal caregiving for older Americans with symptoms of depression. Am J Psychiatry. 2004; 161: 857–63.
14. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM–5). Washington, DC: APA; 2013.
15. Mykletun A, Bjerkeset O, Øverland S, Prince M, Dewey M, Stewart R. Levels of anxiety and depression as predictors of mortality: the HUNT study. Br J Psychiatry. 2009; 195:118–25.
16. Walker E, McGee R, Druss B. Mortality in mental disorders and global disease burden implications: A systematic review and meta-analysis. JAMA Psychiatry. 2015; 72: 334–41.
17. Katon W, Lin E, Kroenke K. The association of depression and anxiety with medical symtpkm burden in patients with chronic medical illness. Gen Hosp Psychiatry. 2007; 29: 147–55.
18. Berecki-Gisolf J, McKenzie S, Dobson A, McFarlane A, McLaughlin D. A history of comorbid depression and anxiety predicts new onset of heart disease. J Behav Med. 2013; 36: 347–53.
19. Brown C, Schulberg H, Madonia M, Shear M, Houck P. Treatment outcomes for primary care patients with major depression and lifetime anxiety disorders. Am J Psychiatry. 1996; 153(10): 1293–300.
20. Handley T, Inder K, Kay-Lambkin F, Stain H, Fitzgerald M, Lewin T, et al. Contributors to suicidality in rural commu-

nities: beyond the effects of depression. BMC Psychiatry. 2012; 12: 105.

21. Widiger T, Samuel D. Diagnostic categories or dimensions? A question for the Diagnostic and Statistical Manual of Mental Disorders– Fifth Edition. J Abnorm Psychol. 2005; 114: 494–504.
22. Zinbarg RE, Barlow DH, Liebowitz M, Street L, Broadhead E, Katon W, et al. The DSM-IV field trial for mixed anxiety-depression. Am J Psychiatry. 1994; 151(8): 1153–62.
23. Muris P, Merckelbach H, Schmidt H, Gadet B, Bogie N. Anxiety and depression as correlates of self-reported behavioural inhibition in normal adolescents. Behav Res Ther. 2001; 39(9): 1051–61.
24. Ionescu D, Niciu M, Henter I, Zarate C. Defining anxious depression: a review of the literature. CNS Spectrums. 2013; 18: 151–60.
25. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders (4th ed, Text Revision). Washington, DC: APA; 2000.
26. Wilhelm K, Mitchell P, Slade T, Brownhill S, Andrews G. Prevalence and correlates of DSM-IV major depression in an Australian national survey. J Affect Disord. 2003; 75(2): 155–62.
27. Andrews G, Henderson S, Hall W. Prevalence, comorbidity, disability and service utilisation. Br J Psychiatry. 2001; 178: 145–53.
28. Kilkkinen A, Kao-Philpot A, O'Neil A, Philpot B, Reddy P, Bunker S, et al. Prevalence of psychological distress, anxiety and depression in rural communities in Australia. Aust J Rural Health. 2007; 15(2): 114–9.
29. Zung W. A rating instrument for anxiety disorders. Psychosomatics. 1971; 12: 371–9.
30. Sharpley C, Rogers H. Naïve versus sophisticated item-writers for the assessment of anxiety. J Clin Psychol. 1985; 41: 58–62.
31. Zung W. How Normal is Anxiety? Durham, NC: Upjohn; 1980.
32. Zung W. From art to science: The diagnosis and treatment of depression. Arch Gen Psychiatry. 1973; 29: 328–37.
33. Schaefer A, Brown J, Watson C, Plenel D, DeMotts J, Howard M, et al. Comparison of the validities of the Beck, Zung and MMPI depression scales. J Consult Clin Psychol. 1985; 53: 415–8.
34. Cohen J. Statistical Power for the Behavioral Sciences. Hillsdale, NJ: Erlbaun; 1988.
35. Tabachnick B, Fidell L. Using Multivariate Statistics (5th ed.). NY: Pearson; 2007.
36. Tabachnik B, Fidell L. Using Multivariate Statistics (6th ed.). Boston: Pearson Education; 2013.
37. Nunnally J. Psychometric Theory. New York: McGraw-Hill; 1978.
38. Comfrey A, Lee H. A First Course in Factor Analysis. Hillsdale, NJ: Lawrence Erlbaum Associates; 1992.
39. Judd L, Rapaport M, Paulus M, Brown J. Subsyndromal symptomatic depression: A new mood disorder? J Clin Psychiatry. 1994; 55(suppl. 4): 18–28.
40. Judd L, Paulus M, Wells K, Rapaport M. Socioeconomic burden of subsyndromal depressive symptoms and major depression in a sample of the general population. Am J Psychiatry. 1996; 163: 1411–7.
41. Lyness J, Heo M, Datto C, Ten Have T, Katz I, Drayer R, et al. Outcomes of minor and subsyndromal depression among elderly patients in primary care settings. Ann Intern Med. 2006; 144(7): 496–504.
42. Chopra M, Zubritsky C, Knott K, Have T, Hadley T, Coyne J, et al. Importance of subsyndromal symptoms of depression in elderly patients. Am J Geriatr Psychiatry. 2005; 13: 597–606.
43. Lyness J, Kim J, Tang W, Tu X, Conwell Y, King D, et al. The clinical significance of subsyndromal depression in older primary care patients. Am J Geriatr Psychiatry. 2007; 15: 214–23.
44. Vanitallie T. Subsyndromal depression in the elderly: underdiagnosed and undertreated. Metabolism. 2005; 54: 39–44.
45. Goldney R, Fisher L, Dal Grande E, Taylor A. Subsyndromal depression: prevalence, use of health services and quality of life in an Australian population. Soc Psychiatry Psychiatr Epidemiol. 2004; 39: 293–8.
46. Nutt D. Overview of diagnosis and drug treatments of anxiety disorders. CNS Spectrums. 2005; 10: 49–56.
47. Perna G, Alciatti A, Riva A, Miceilli W, Caldirola D. Long-term pharmacological treatments of anxiety disorders: an updated systematic review. Curr Psychiatry Rep. 2016; 18: 23.
48. Deacon B, Abramowitz J. Cognitive and behavioral treatments for anxiety disorders: a review of meta-analytic findings. J Clin Psychol. 2004; 60(4): 429–41.
49. Hayes-Skelton S, Roemer L, Orsillo S. A randomized clinical trial comparing an acceptance-based behavior therapy to applied relaxation for generalized anxiety disorder. J Consult Clin Psychol. 2013; 81: 761–73.
50. Zeidan F, Martucci K, Kraft R, McHaffie J, Coghill R. Neural correlates of mindfulness meditation-related anxiety relief. Soc Cogn Affect Neurosci. 2014; 9: 751–9.
51. Bandelow B, Reitt M, Rover C, Michaelis S, Gorlich Y, Dirk W. Efficacy of treatments of anxiety disorders: a meta-analysis. Int Clin Pharmacology. 2015; 30: 183–92.
52. Dale E, Bang-Anderson B, Sanchez C. Emerging mechanisms and treatments for depression beyond SSRIs and SNRIs. Biochem Pharmacology. 2015; 95: 81–97.
53. Cuijpers P, van Straten A, Warmerdam L, Andersson G. Psychological treatment of depression: a meta-analytic database of randomized studies. BMC Psychiatry. 2008; 8: 36.
54. Cuijpers P, van Straten A, Warmerdam L. Behavioral activation treatments of depression: a meta-analysis. Clin Psychol Rev. 2007; 27: 318–26.

55. Cuijpers P, Reynolds C, Donker T, Li JZ, Andersson G, Beekman ATF. Personalized treatment of adult depression: medication, psychotherapy, or both? A systematic review. Depression Anxiety. 2012; 29: 855–64.
56. Richards D, Richardson T. Computer-based psychological treatments for depression: a systematic review and meta-analysis. Clin Psychol Rev. 2012; 32: 329–42.
57. Richieri R, Guedj E, Michel P, Loundou A, Auquier P, Lancon C, et al. Maintenance transcranial magnetic stimulation reduces depression relapse: a propensity-adjusted analysis. J Affect Disord. 2013; 151(1): 129–35.
58. Sutin A, Terracciano A, Milaneschi Y, An Y, Ferrucci L, Zonderman A. The trajectory of depressive symptoms across the adult life span. JAMA Psychiatry. 2013; 70: 803–11.
59. Ostergaard S, Jensen S, Bech P. The heterogeneity of the depressive syndrome: when numbers get serious. Acta Psychiatr Scand. 2011; 124: 495–6.
60. Simms L, Prisciandaro J, Krueger R, Goldberg D. The structure of depression, anxiety and somatic symptoms in primary care. Psychol Med. 2012; 42(01): 15–28.
61. Clark L, Watson D. Tripartite model of anxiety and depression: psychometric evidence and taxonomic implications. J Abnorm Psychol. 1991; 100: 316–35.
62. den Hollander-Gijsman M, Wardenaar K, de Beurs E, van der Wee N, Mooijaart A, van Buuren S, et al. Distinguishing symptom dimensions of depression and anxiety: An integrative approach. J Affect Disord. 2012; 136: 693–701.
63. Rus-Calafell M, Gutiérrez-Maldonado J, Botella C, Baños R. Virtual reality exposure and imaginal exposure in the treatment of fear of flying: a pilot study. Behav Modif. 2013; 37(4): 568–90.
64. Matthews A, Scanlan J, Kirkby K. Online exposure treatment for spider fear: the effects of moving versus static images on treatment adherence, fear elicitation and habituation. Behav Change. 2012; 29(01): 15–24.
65. Iverach L, Menzies R, Menzies R. Death anxiety and its role in psychopathology: reviewing the status of a transdiagnostic construct. Clin Psychol Rev. 2014; 34(7): 580–93.
66. Harmer C, Mackay C, Reid C, Cowen P, Goodwin G. Antidepressant drug treatment modifies the neural processing of nonconscious threat cues. Biol Psychiatry. 2006; 59(9): 816–20.