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Perceiving social pressure not to feel negative predicts depressive symptoms in daily life

Egon Dejonckheere¹, Brock Bastian², Eiko I. Fried³, Sean Murphy² & Peter Kuppens¹

¹KU Leuven – Department of Psychology

²University of New South Wales / University of Melbourne – School of Psychology

³University of Amsterdam – Department of Psychology

Correspondence concerning this article should be addressed to Egon

Dejonckheere, Department of Psychology, KU Leuven, Tiensestraat

102, 3000 Leuven, Belgium. E-mail:

egon.dejonckheere@kuleuven.be_Telephone: +32 16 37 42 46. Fax:

+32 16 32 59 93.

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Abstract

Background – Western societies often overemphasize the pursuit of happiness, and regard negative feelings like sadness or anxiety as maladaptive and unwanted. Despite this emphasis on happiness, the amount of people suffering from depressive complaints is remarkably high. To explain this apparent paradox, we examined whether experiencing social pressure not to feel sad or anxious could in fact contribute to depressive symptoms.

Methods – A sample of individuals ($n = 112$) with elevated depression scores ($\text{PHQ-9} \geq 10$) took part in an online daily diary study in which they rated their depressive symptoms and perceived social pressure not to feel depressed or anxious for 30 consecutive days. Using

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multilevel VAR models we investigated the temporal relation between this perceived social pressure and depressive symptoms in order to determine directionality.

Results – Primary analyses consistently indicated that experiencing social pressure predicts increases in both overall severity scores and most individual symptoms of depression, but not vice versa. A set of secondary analyses, in which we adopted a network perspective on depression, confirmed these findings. Using this approach, centrality analysis revealed that perceived social pressure not to feel negative plays an instigating role in depression, reflected by the high out- and low in-strength centrality of this pressure in the various depression networks.

Conclusions – Together, these findings indicate how perceived societal norms may contribute to depression, hinting at a possible malignant consequence of society's denouncement of negative emotions. Clinical implications are discussed.

Keywords: depression, anxiety, emotions, culture, social norm

Introduction

Prevalence rates of Major Depressive Disorder (MDD) in Western societies are remarkably high. With epidemiological estimates having doubled in the last 3 decades (Compton et al., 2006), today one in six Americans will suffer from depression at some point in their lives (Kessler et al., 2012), making it a leading cause of disability in modern 'first-world' societies (Ferrari et al., 2013). Beside the emotional and psychological distress for patients and their immediate social environment (e.g. reduced quality of life, social dysfunction; Lépine & Briley, 2011), MDD leaves the broader society with an extensive economic burden (Greenberg et al., 2015), pushing researchers to discover the mechanisms underlying this debilitating disorder.

At the same time – and almost paradoxically – society seems to be exceedingly preoccupied with happiness (Bastian et al., 2012; Sheldon & Lyubomirsky, 2006). Particularly in Western countries where MDD prevalence rates are especially high (e.g. Weissman et al., 1996), today's societal norm encourages people to pursue happiness (Bastian et al., 2012; 2015a), ranging from brand commercials emphasizing the hedonic pleasure of consumption (e.g. Lewis & Hill, 1998), to national indexes carefully monitoring citizen's well-being and life satisfaction (e.g. Diener, 2000). Simultaneously, negative emotions like sadness and anxiety commonly receive a maladaptive and dysfunctional connotation (Haslam, 2005), with the adaptive nature of feeling negative at times, such as regulating social interaction (Fischer & Manstead, 2008; McNulty, 2010) and contributing to a meaningful life (Hayes et al., 1999), hardly being mentioned in modern societal discourse (Bastian et al., 2012).

Although emphasizing happiness might seem laudable for people's well-being, recent studies also point to detrimental consequences of the pressure to feel happy and not sad. Lab results indicate that participants who are experimentally induced to value happiness react less positively to happy emotion induction (Mauss et al., 2011). Conversely, perceiving societal pressure not to experience or express negative emotions is associated with higher levels of negative affect and reduced well-being, a finding that was found to be particularly strong in Western societies (Bastian et al., 2012). Such perceived pressure has moreover been related to loneliness (Bastian et al., 2015a) and to biased attention for negative information (Bastian et al., 2015b). The underlying idea is that perceiving high pressure not to experience negative emotion creates a discrepancy between one's *actual* emotional state and the social standard deemed desirable when an individual inevitably feels sad or anxious, leading to negative self-reflections and an ironic amplification of these unwanted emotions (e.g. Carver & Scheier, 1990; Nolen-hoeksema, 1991).

Could it be that the high premium society places on happiness may paradoxically contribute to the prevalence of depression and its symptoms? Preliminary experimental evidence shows that communicating that public opinion disapproves of the experience of negative emotions leads to a temporary augmentation of negative affect (Bastian et al., 2012). Yet whether these social expectancies play a role in depressive symptoms in the complexity of everyday life remains unexplored.

In the present study, we sought to examine the role of the perceived pressure not to experience negative emotions in the occurrence of depressive symptoms in real life. In particular, we examined whether the perceived pressure not to feel sad or anxious predicts depressive symptoms from one day to another in a group of individuals with elevated depression scores. Participants who exhibited depressive complaints were preselected from a larger initial pool. They next participated in a daily diary study in which they reported their depressive symptoms and perceived social expectancies not to feel depressed or anxious on a daily basis for 30 consecutive days.

In addition to an overall depression score, we also investigated whether this perceived pressure predicted the presence of *individual* depression symptoms. Contemporary theories on psychopathology (Cramer et al., 2010; Fried, 2015; Fried & Nesse, 2015) no longer conceptualize MDD as a homogeneous, demarcated condition, composed by a variety of interchangeable symptoms (e.g. suicidal ideation vs. appetite gain), but rather understand depression as a dynamic system of interacting symptoms, acknowledging the fact that different depressive symptoms may have different risk factors (e.g. Rottenberg et al., 2007; Strange et al., 2016), temporal trajectories (e.g. Iacoviello et al., 2010) or consequences (e.g. Fried & Nesse, 2015). In this respect, these theories advocate to abandon the use of (unweighted) sum-scores in depression research and either wish to focus on (a) different MDD symptoms *individually* or (b) to combine these in depression networks in which they

explore the dynamic interrelations of different symptoms *simultaneously* (Cramer et al., 2010). The present paper thus investigates both the more traditional sum-score and, additionally, takes on a more symptom-based focus in line with the network perspective on psychopathology.

Materials and Method

Participants

We employed Amazon's Mechanical Turk services (MTurk), preselecting potential participants with elevated depression scores. Recent studies investigating characteristics of MTurk samples illustrated that the MTurk community endorses depression and anxiety symptoms to a substantially larger degree than nonclinical samples (Arditte et al., 2015), while resembling the general population in other demographic aspects more closely than traditional convenience samples (e.g. bachelor students; Casler et al., 2013), making the MTurk community a particularly suitable subject pool for our study.

Based on a test for statistical power to detect small to medium between-person effect sizes ($d = .30$, $\alpha = .050$), we intended to recruit a sample of 100 participants. An initial group of 987 MTurkers (56% male) completed the Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001), a scale used as a prescreening instrument for depressive symptomatology (range = 0–27, $M = 6.32$, $SD = 5.90$). Out of the 207 potential participants with a sum-score exceeding the clinical cut-off (PHQ-9 ≥ 10) of Kroenke et al. (2001) and no self-reported diagnosis other than MDD, 194 people were randomly contacted to participate in our daily diary study, expecting about 120 individuals to respond to our request to take part in our daily diary protocol (accounting for an estimated 15% attrition, for a final sample of 100

participants). From this group, 121 individuals responded and began our study (53% male; PHQ-9 prescreening range = 11–27, $M = 14.48$, $SD = 4.10$)¹.

Participants were reimbursed \$1 for completing the survey each day, receiving an additional \$20 at the end of the study if they had completed 25 days or more. Nine participants² were excluded from our analysis due to poor compliance with the daily diary protocol (i.e. <50% response rate), leaving us with a final sample of 112 participants (52% male) ranging in age from 19 to 74 ($M = 34.27$, $SD = 9.78$). In this group, 18 participants (16%) indicated they currently had an MDD diagnosis. In terms of compliance, our final sample completed 28 out of 30 daily surveys on average (range = 18–30, $SD = 2.21$).

Procedure and Measures

Upon giving their informed consent and completing several other self-report questionnaires in a survey prior to our daily diary study (not relevant to this report), participants received a daily e-mail with a hyperlink to a Qualtrics questionnaire. E-mails were sent out each day at 7:00 PM local time and participants received instructions to complete the survey before 3:00 AM the next day. Questionnaires were filled out around 9:13 PM on average, with a standard deviation of 2 hours 30 minutes. Data from surveys completed after the instructed deadline were excluded from analyses.

Depressive symptoms. Self-reported depressive symptoms were assessed daily using items directly reflecting the DSM-5 diagnostic criteria for MDD (American Psychiatric Association, 2013). Criteria that aggregate two extremes of the same continuum (e.g. insomnia and hypersomnia) were covered by two separate items and suicidal ideation was not assessed. This resulted in a set of 11 items measuring daily depressive symptomatology,

¹ Responders who agreed to participate ($n = 121$) did not differ from the subset of suitable participants that did not take part in our study ($n = 86$) in gender or age, nor in PHQ-scores or self-reported MDD diagnosis (all p 's $\geq .067$).

² Excluded participants did not differ in demographics such as gender or age, nor in PHQ-scores or self-reported MDD diagnosis (all p 's $\geq .122$).

including the two DSM-5 core symptoms of depression: “Today to what extent did you feel sad?” (*sadness*); “Today, to what extent did you experience a diminished interest or pleasure in activities?” (*anhedonia*), complemented with nine secondary DSM-5 MDD criteria which consisted of the cognitive symptoms of depression: “Today, to what extent did you feel worthless?” (*worthlessness*); “Today, to what extent did you experience problems concentrating or decision making?” (*concentration problems*), and the somatic symptoms of depression: “Today, to what extent did you feel tired and fatigued” (*fatigue*); “To what extent did you feel like sleeping today?” (*hypersomnia*); “To what extent did you experience trouble sleeping last night?” (*insomnia*); “Today, to what extent did your body feel slowed down?” (*psychomotor retardation*); “Today, to what extent did your body feel agitated?” (*psychomotor agitation*); “Today, to what extent did you experience a decreased appetite compared to normal?” (*appetite loss*); “Today, to what extent did you experience an increased appetite compared to normal?” (*appetite gain*). Participants rated how much they had experienced each depressive symptom on that particular day using a Likert-scale ranging from 1 (*not at all*) to 7 (*very much*). Total daily depression scores were obtained by averaging all symptom items per day (see Table 1 for descriptive statistics). The average Cronbach’s alpha across days for the daily depressive symptoms composite was .86, ranging from .80 to .89.

Social expectancies. To measure participants’ perceived social expectancies not to experience negative affect, we selected 5 items from the Social Expectancies of Depression and Anxiety Scale (SEDAS; Bastian & Kuppens, *in preparation*) and reformulated them for daily use: “Today, I felt a great deal of pressure from others around me not to feel depressed or anxious.”; “Today, people expected me not to feel depressed or anxious.”; “People valued me today, even though I felt depressed or anxious.” (reversed); “People were disapproving of my feeling depressed or anxious today.”; “Today, people saw me as a failure because I felt

depressed or anxious.”. Participants rated these items on a Likert-scale from 1 (*not at all*) to 7 (*very much*). Data were averaged per day and per participant to reflect participants’ daily social expectancies not to feel anxious or depressed (see Table 1 for descriptive statistics). The average Cronbach’s alpha for daily perceived social expectancies was .78 and ranged from .65 to .86³.

Finally, each daily questionnaire also included two bogus items to control the reliability of participants’ responses (e.g. “This is a control item. Please select ‘2’ for this item.”). Surveys with incorrect answers to these items were excluded from analyses ($n = 13$).

Statistical analysis

To examine the temporal relation between perceived social pressure to avoid feeling negative and depression, we used vector autoregressive (VAR) models with a multilevel extension (Bringmann et al., 2013), meaning that slopes and intercept were allowed to vary across participants to account for possible interindividual differences. All predictors were within-person centered (Hamaker & Grassman, 2015).

In terms of missing data, there was no indication that the missingness was not at random (i.e. compliance was not related to person-characteristics, nor did we observe any time effects). Missing cases were therefore treated as missing (listwise deletion), with no data imputation techniques being carried out. Furthermore, model assumptions (i.e. equally spaced measurement points, stationarity and normally distributed residuals) were checked and observed to be sufficiently met.

In a series of primary analyses we first determined temporal directionality between participants’ perceived social expectancies not to experience negative emotion and their total

³ Leaving out the one reversed item led to an increase in internal consistency (average $\alpha = .88$; range $\alpha = .82 - .92$). While all reported analyses are based on the 5-item version, analyses using the 4-item version yielded similar results and support the same conclusions.

depression severity score. In a first multilevel VAR model we tested whether participants' total depression score at day t was predicted by their social expectancies on the previous day ($t - 1$), controlled for their total depression score on the previous day ($t - 1$). Conversely, in a second multilevel VAR model we tested whether participants' social expectancies at day t were predicted by their total depression score on the previous day ($t - 1$), controlling for their social expectancies on the previous day ($t - 1$).

Next to the total depression score, we repeated these analyses for participants' perceived social expectancies and each of the 11 individual depression symptoms *separately*. To reduce the occurrence of Type-I errors, we controlled for multiple testing ($n = 11$) using a False Discovery Rate (FDR) procedure as proposed by Benjamini & Hochberg (1995).

So far, our analyses addressed the role of these perceived social expectancies in relation to either a total depression score or each depressive symptom *individually*. In a secondary and exploratory series of analyses, a network perspective on depressive symptomatology was adopted, allowing us to inspect multiple temporal relations between different variables *simultaneously* (e.g. Bringmann et al., 2015). We examined the role of this perceived social pressure in four different depression networks. A distinction was made between DSM-5 core, cognitive and somatic symptoms (Uher et al., 2008), with the last symptom category being divided in positive and negative somatic symptoms. Each network composed several multilevel VAR models, in which every variable once served as an outcome, regressed on its day-lagged version, as well as on the day-lagged version of each of the other dependent variables in the network. In this way auto- and cross-regressive effects of one variable could be determined, controlled for the predictive value of other variables in the network. Variables were within-person standardized (Schoorman et al., 2016) and in each network we controlled for multiple testing using an FDR procedure (Benjamini et al., 2006), since no explicit hypotheses were specified about the dynamic interplay among individual

depression symptoms⁴. Results were visualized using the *qgraph* R package (Epskamp et al., 2012).

For each symptom network centrality strength plots were calculated, displaying the in- and outstrength centrality of each node in the network. These values refer to the sum of the absolute weights of respectively all incoming or outgoing connections that are present in a node, indicating whether a variable either plays a following or instigating role in the network. Put differently, a node with high outstrength centrality has the capacity to predict other variables in the network. In contrast, a node with high instrength centrality is predicted by the other variables in the network.

Some caution is advised, however, when interpreting the results of these network analyses. In- and outstrength centrality measures are only meaningful within the network they are calculated from and heavily depend on the (amount of) nodes present. Adding or removing nodes will entail different results and might even yield ordinal changes in centrality. Furthermore, estimating meaningful confidence intervals for centrality measures is currently impossible (see Epskamp et al., *under revision*), which makes the results presented next fairly exploratory in nature.

Results

Within-person correlations obtained from multilevel analyses (see Nezlek, 2012) are presented in Table 1. Perceived social expectancies not to feel sad or anxious were significantly and positively correlated with all depression symptoms, except appetite gain.

⁴ A depression network that included all depressive symptoms was not computed, as we deemed this too exploratory, and containing too many variables, creating instability of edges. Instead we constructed four smaller symptom networks, making a trade-off between a bottom-up approach (i.e. looking at the data with the appropriate significance threshold) and a top-down perspective (i.e. making a meaningful distinction between different symptom categories relying on theoretical propositions), allowing us to make relevant claims within appropriate borders.

Primary analyses. First, daily total depression severity scores were significantly predicted by both total depression scores and perceived social expectancies on the previous day. Daily perceived social expectancies on the other hand, were only significantly predicted by daily perceived social expectancies on the day before, with previous-day total depression scores not contributing to this prediction (see Table 2). Next, when investigating the temporal relations between these social expectancies and each of the 11 depression symptoms individually, several findings are noteworthy (see Table 2). First, both DSM-5 core symptoms of depression, sadness and anhedonia, were positively predicted by a person's social expectancies perceived on the previous day, but not vice versa. Second, in terms of cognitive symptoms, daily social expectancies also predicted an increase in feelings of worthlessness and concentration problems on the next day, but again not vice versa. Finally, for the somatic symptoms in depression, participants' perceived social expectancies predicted increases in fatigue, hypersomnia, psychomotor retardation and appetite loss, but not insomnia, psychomotor agitation and appetite gain. Autoregressive effects were significant for all individual symptoms (β 's ≥ 0.07 , p 's $\leq .005$), indicating that each symptom was meaningfully predicted by its score on the previous day⁵.

Secondary analyses. As Figure 1a illustrates, regarding the DSM-5 core symptoms of depression, social expectancies and sadness both predicted a significant increase in anhedonia, resulting in a relatively high outstrength centrality for social expectancies and *sadness* and a high instrength centrality for anhedonia. Among cognitive depression symptoms social expectancies predicted a significant increase in concentration problems and worthlessness, leaving social expectancies with the highest outstrength centrality and lowest instrength centrality (Fig. 1b). For positive somatic symptoms the only cross-regressive link

⁵ To eliminate the possibility that our findings were merely driven by the subsample that reported having an MDD diagnosis ($n = 18$), we reran all analyses without these cases. With only one minor difference (i.e. social expectancies now did predict an increase in psychomotor agitation, where in the presented analyses it did not), our conclusions remain identical.

that remained significant after applying FDR was hypersomnia predicting an increase in psychomotor agitation. Based on all edges, social expectancies were characterized by the highest outstrength centrality, while psychomotor agitation had the highest instrength centrality (Fig. 1c). Finally, for negative somatic symptoms insomnia positively predicted psychomotor retardation and fatigue, while the latter also predicted an increase psychomotor retardation. Social expectancies predicted an increase in fatigue. Based on the centrality strength plot, fatigue was characterized by the highest outstrength centrality, while psychomotor retardation had the highest instrength centrality (Fig. 1d).

Discussion

Drawing on daily life data of a sample with elevated depression scores, we examined the role of perceived social pressure not to experience negative affect in the prediction of depressive symptoms. We found converging evidence that perceived social expectancies not to feel sad or anxious do not follow from depressive symptoms, but rather themselves predict increases in depressive symptomatology. The more pressure a person perceives from his social environment not to experience negative emotions, the more likely that person is to experience an increase in depressive symptoms. This did not only apply for overall depression severity, but also for the vast majority of individual depressive symptoms. Higher levels of perceived pressure not to feel negative predicted increases in both MDD's core and cognitive symptomatology. For somatic symptoms, only inhibiting, slowed-down symptoms of depression (fatigue, hypersomnia, psychomotor retardation, appetite loss) were predicted by these perceived social expectancies, not their activating counterparts (insomnia, psychomotor agitation, appetite gain). This suggests that perceiving social pressure not to experience negative emotions seems to have a dampening rather than augmenting somatic effect, a finding that a traditional depression sum-score approach could not have captured (e.g. Fried

& Nesse, 2015). Finally, exploratory centrality analyses were in line with the current findings. When multiple relations between depressive symptoms and these perceived social expectancies were assessed simultaneously in various depression networks, experiencing social pressure not to feel anxious or depressed was characterized by a relatively high out- and low in-strength centrality, meaning that this pressure plays a rather instigating than following role in depression.

While traditional depression research generally focusses on the role of person-specific characteristics (e.g. from genes and biomarkers to cognitive and behavioral styles), current findings suggest that (the perception of) larger-scale factors like societal norms are also likely to have implications for people's psychological well-being. When society pressures people to pursue the unattainable state of constant happiness, while marginalizing the natural occurrence of negative emotions like sadness and anxiety, this inevitably creates a discrepancy between people's experienced mood and these salient reference values. Failing to live up to what you perceive as the social standard might lead to negative self-evaluations (Bastian et al., 2012; Carver & Scheier, 1990), resulting in the seemingly paradoxical amplification of these non-normative emotional states.

Clinical implications

Interventions that tackle this perceived social pressure may be implemented both on a micro and macro level, referring to the individual or broader society respectively. From a micro perspective, an individual's *perception* does not necessarily match objective reality (Jussim, 1991). Counselors who communicate the apparent societal acceptability and functionality of negative emotions, may challenge the beliefs their clients hold about their social environment (e.g. cognitive restructuring; Clark, 2014), finding a possible gateway to decrease their depressive complaints (Bastian et al., 2012). Furthermore, it is of note that a balanced view

on positive versus negative emotion is already strongly implied in a lot of the *third wave* cognitive behavioral psychotherapies (e.g. Dialectical Behavioral Therapy, Linehan, 2014; Acceptance and Commitment Therapy, Hayes et al., 1999; Mindfulness-Based Cognitive Therapy, Kabat-Zinn, 2003), which are known to be effective in treating depression (Kahl et al., 2012). These therapeutic approaches all share their origin in Eastern Buddhist culture where a dialectic worldview is emphasized, promoting a balanced embrace of one's emotional repertoire. Finally, creating a receptive and caring social platform (e.g. in the form of therapeutic family sessions) might also be beneficial (Brown & Andrews, 1986).

With respect to possible interventions on a macro level, large-scale (psycho-)educational programs that destigmatize occasionally feeling sad or anxious and tackle people's prejudice towards mood disorders, are likely to be essential in gradually shifting society's conception of negative emotion.

Limitations

Several limitations are noteworthy to our study. For one, the current sample was not assessed in a clinical setting. Although all participants reported considerable depressive complaints, as they all surpassed a clinical threshold on a depression screening instrument, this may have led to a possible underrepresentation of the more severely impaired individuals suffering from depression.

Second, the current investigation only addressed the temporal relationship between perceiving social pressure not to feel negative and depression. Although determining temporal precedence is a first important step and necessary condition, direct causal claims about this relationship cannot be made. However, combined with preliminary experimental evidence showing that manipulating social expectancies leads to increases in negative emotion and decreases in well-being (Bastian et al., 2012), our results are suggestive that

perceiving social pressure to avoid the experience of negative emotions could indeed increase the occurrence of depressive symptoms in daily life. Nevertheless, further experimental work is needed to pinpoint the exact mechanisms and possible boundary conditions.

Third, a logical question following these findings is what factors influence these perceived social expectancies. As the current design is correlational, the apparent relationship between this social pressure and depression may be explained by other unobserved variables. For example, processes like social alienation or lack of social support (e.g. Brown & Andrews, 1986) could possibly be important antecedents in the current chain of events, making this perceived social pressure merely a mediator in predicting depressive symptoms.

Finally, it is currently unclear to what degree the current findings are specific to depression. In fact, it seems plausible to hypothesize that perceiving social pressure not to feel sad or anxious may also predict so-called *secondary disturbances* (i.e. the process of experiencing more negative affect in response to the experience of negative affect; Bastian et al., 2015) in people suffering from, for instance, various anxiety disorders. Whether these perceived expectancies also predict increases in other anxiety-related symptoms (e.g. heart palpitations or shortness of breath in patients with panic disorder or an increase in obstructive thoughts and checking behavior in people with obsessive-compulsive disorder), remains nevertheless to be explored.

Conclusion

In conclusion, our findings indicate that experiencing pressure not to feel negative emotions, paradoxically predicts an increase in depressive symptoms over time. In this way, the current study may reveal a possible malignant consequence of society's one-sided focus on happiness and denouncement of negative emotions. A societal discourse that promotes a more balanced

embracement of the emotional repertoire could possibly be more beneficial for people's well-being.

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Figure legends

A

[Figure 1 here]

Fig. 1. Estimated depression networks for social expectancies and DSM-5 depressive (a) core symptoms, (b) cognitive symptoms, (c) positive somatic symptoms and (d) negative somatic

symptoms. Solid green arrows surpassed the significance threshold after applying FDR (Benjamini et al., 2006) and represent a positive relationship between two nodes. In contrast, dashed gray edges did not survive the correction for multiple testing. Both transparency and thickness of an arrow represent the strength of a connection (i.e. magnitude of the fixed effect), with the scaling being proportional to the strongest edge in the network. Each network is accompanied by its centrality strength plot, displaying the in- and outstrength centrality of each node in the network. These measures are based on all cross-regressive coefficients (i.e. not only the significant ones). EXP = Perceived social expectancies not to feel sad or anxious; SAD = Sadness; ANH = Anhedonia; CON = Concentration problems; WOR = Worthlessness; AGI = Psychomotor agitation; HYP = Hypersomnia; APG = Appetite gain; RET = Psychomotor retardation; INS = Insomnia; FAT = Fatigue; APL = Appetite loss.

Tables

Table 1. Means, standard deviations, intra-class correlations and within-person correlations among all measures.

	Me an (SD)	IC C	Correlations											
			1	2	3	4	5	6	7	8	9	10	11	12
1. Social expectancies	3.11 (1.11)	.61												
2. Total depression score	3.45 (0.89)	.54	.48* **											
3. Sadness	3.37 (1.28)	.48	.45* **	.71* **										
4. Anhedonia	3.49	.4	.41* **	.73* **	.57* **									

ia	(1. 21)	3	**	**	**									
5. Worthless ness	3.1 6 (1. 48)	.5 7	.48* **	.70* **	.62* **	.66* **								
6. Concentr ation problems	3.4 1 (1. 18)	.4 3	.40* **	.69* **	.47* **	.50* **	.49* **							
7. Fatigue	4.3 7 (1. 07)	.3 5	.34* **	.77* **	.44* **	.48* **	.45* **	.45* **						
8. Hyperso mnia	4.2 7 (1. 10)	.3 3	.29* *	.70* **	.39* **	.42* **	.39* **	.39* **	.69* **					
9. Insomnia	3.5 3 (1. 35)	.4 4	.19* *	.48* **	.22* *	.21* *	.25* *	.25* *	.36* **	.33* **				
10. Psychom otor retardati on	3.8 2 (1. 25)	.4 4	.34* **	.75* **	.45* **	.50* **	.46* **	.49* **	.66* **	.60* **	.30 **			
11. Psychom otor agitation	3.5 5 (1. 28)	.4 6	.39* **	.65* **	.47* **	.44* **	.46* **	.45* **	.41* **	.32* **	.24 *	.44* **		
12. Appetite loss	2.4 3 (1. 13)	.3 9	.22* *	.44* **	.29* *	.33	.28* *	.28* *	.27* *	.22* *	.18	.29* *	.29 **	
13. Appetite gain	2.5 7 (1.	.4 9	- .007	.25* *	-.14	.20* *	.15	.18	.17	.15	.09	.18	.14	- .49* **

	29)													
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Note. ICC = Intra-class correlation. * $p < .050$. ** $p < .010$. *** $p < .001$.

Table 2. Multilevel vector autoregressive models predicting a total depression score and separate depression symptoms.

	Predicted by social expectancies			Prediction of social expectancies		
	β	SE	95% CI	B	SE	95% CI
1. Total depression score	0.05**	0.02	[0.02, 0.07]	0.02	0.02	[-0.01, 0.05]
2. Core symptoms						
Sadness	0.08*	0.03	[0.03, 0.12]	0.01	0.01	[-0.01, 0.02]
Anhedonia	0.16***	0.04	[0.09, 0.20]	-0.0002	0.01	[-0.02, 0.02]
3. Cognitive symptoms						
Worthlessness	0.12***	0.03	[0.07, 0.17]	-0.02	0.02	[-0.03, 0.01]
Concentration problems	0.09**	0.03	[0.04, 0.14]	0.03	0.01	[0.01, 0.05]
4. Somatic symptoms						
Fatigue	0.09**	0.03	[0.04, 0.14]	0.02	0.01	[0.002, 0.04]
Hypersomnia	0.09*	0.04	[0.04, 0.16]	0.0007	0.01	[-0.02, 0.01]
Insomnia	-0.04	0.03	[-0.09, 0.02]	0.01	0.01	[-0.004, 0.03]
Psychomotor retardation	0.10***	0.03	[0.05, 0.14]	0.02	0.01	[0.001, 0.04]
Psychomotor agitation	0.06	0.04	[0.007, 0.11]	-0.007	0.01	[-0.03, 0.01]
Appetite loss	0.08*	0.03	[0.04, 0.13]	0.006	0.01	[-0.01, 0.02]
Appetite gain	0.01	0.03	[-0.03, 0.05]	-0.02	0.01	[-0.04, -0.0007]

Note. The results presented are non-corrected for familywise error rate. To prevent the occurrence of Type-I errors we applied FDR ($\alpha = .036$; Benjamini & Hochberg, 1995). However, all significant coefficients survived correction for multiple testing. * $p < .050$. ** $p < .010$. *** $p < .001$.

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Author/s:

Dejonckheere, E;Bastian, B;Fried, EI;Murphy, SC;Kuppens, P

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