The Importance of Perseverative Cognition for Both Mental and Somatic Disorders in a Naturalistic Psychiatric Patient Sample

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Abstract

Comorbidity between mental and somatic disorders is prevalent and is associated with low quality of life and high health care utilization. Research has implicated perseverative cognition (PC) as contributing to the development of both mental and somatic disorders. However, research on the role of PC in the comorbidity of those disorders is largely lacking, especially studies with clinical samples. The current study explored the relative importance of different mental (i.e., depression, anxiety, addiction, autism, ADHD) and somatic disorders (i.e., (cardio-)vascular, immune-/endocrine) for PC in a naturalistic sample of 288 psychiatric patients (60.4% female; M_{age} = 39.91, SD_{age} = 13.92). In a series of multiple regression analyses complemented with relative importance analyses, depression emerged as the most important predictor of overall level of PC, as well as of its different components, followed by addiction. Contrary to our expectations, the results for anxiety were mixed. Also, neither autism or ADHD, nor any of the somatic disorders did show significant contributions to the explained variance in PC. Hence, no evidence for the role of PC in the comorbidity between mental and somatic disorders was found. Rather, in psychiatric samples, PC may be specifically related to stress-related mental disorders.

Keywords: perseverative cognition, repetitive negative thinking, comorbidity, somatic disorders, mental disorders

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Comorbidity among mental disorders is frequent: Nearly half of the individuals presenting with one mental disorder also meet the diagnostic criteria for at least one other mental disorder (Kessler, Chiu, Demler, & Walters, 2005). Besides, mental disorders also show high comorbidity with *somatic* pathology (e.g., Lin et al., 2014; Marrie et al., 2017; Nouwen et al., 2010). This comorbidity is associated with worse outcomes for the individual, such as decreased quality of life (Schram, Baan, & Pouwer, 2009) and increased mortality (van Dooren et al., 2013), as well as with heightened health care utilization (Wagner, Pietrzak, & Petry, 2008). Investigating factors that contribute to the comorbidity between mental and somatic disorders can inform us about possible targets for intervention.

Research has implicated perseverative cognition (PC) – "the repeated or chronic activation of the cognitive representation of one or more psychological stressors" (Brosschot, Gerin, & Thayer, 2006, p. 114) – as contributing to the development of both mental and somatic disorders. PC includes unconscious processes, such as automatic vigilance, as well as conscious processes, such as repetitive negative thinking (Brosschot, Verkuil, & Thayer, 2010). Most often PC is examined by using measures of repetitive negative thinking, which describes a thinking style that is characterized by being repetitive, intrusive, and difficult to disengage from. Its effects are perceived both as being unproductive and as capturing mental capacity (Ehring et al., 2011). According to the perseverative cognition hypothesis (Brosschot et al., 2006), PC prolongs the stress response by upholding the representation of the stressor, which results in longer and hence more negative consequences of psychological and physiological stress. Negative outcomes of this stress response are both mental and somatic health problems (Verkuil, Brosschot, Gebhardt, & Thayer, 2011).

There is ample support for the perseverative cognition hypothesis. Within the field of psychopathology, PC is seen as characteristic of anxiety as well as depressive disorders (American Psychiatric Association, 2013). It has been described as a transdiagnostic risk factor for and predictor of emotional disorders (Arditte, Shaw, & Timpano, 2016; Spinhoven, van Hemert, & Penninx, 2018), and acts as a mediator between anxiety and depressive disorders (Spinhoven, van Hemert, & Penninx, 2019). PC also has been related to addiction and may even predict alcohol abuse (Caselli, Bortolai, Leoni, Rovetto, & Spada, 2008; Caselli et al., 2010; Devynck, Kornacka, Sgard, & Douilliez, 2017; Grynberg et al., 2016). In addition, autism spectrum disorder (ASD) and attention deficit hyperactivity disorder (ADHD) have been associated with higher levels of PC (Crane, Goddard, & Pring, 2013; Burrows, Timpano, & Uddin, 2017; Gotham, Bishop, Brunwasser, & Lord, 2014; Mazefsky, Borue, Day, & Minshew, 2014; Mitchell, Benson, Knouse, Kimbrel, & Anastopoulos, 2013).

There is also evidence for the role of PC in somatic health. PC has been found to impact upon immune, endocrine, and cardiovascular responses to stress (for reviews see Ottaviani et al., 2016; Verkuil, Brosschot, Gebhart, & Thayer, 2010). Research in laboratory as well as naturalistic settings relates PC to elevated levels of inflammatory markers (Zoccola, Figueroa, Rabideau, Woody, & Benecia, 2014), amplified cortisol secretion (Cropley, Rydstedt, Devereux, & Middleton, 2015), and reduced heart-rate variability (Carnevali, Thayer, Brosschot, & Ottaviani, 2018; Chalmers, Heathers, Abbott, Kemp, & Quintana, 2016). Likewise, PC predicts the severity of subjective health complaints, such as fatigue and back pain (Verkuil, Brosschot, Meerman, & Thayer, 2012). Concerning objective health outcomes, PC has solely been linked to physician-diagnosed heart problems so far (Tully, Cosh, & Baune, 2013). Summarizing, there is evidence for the role of PC in several mental disorders, and it has been associated with markers of somatic disorders. Playing a crucial role

in the stress response, PC might help explain the high comorbidity between mental and somatic disorders.

However, until now, the potential role of PC in the comorbidity between mental and somatic disorders has received relatively little attention. The studies that have been conducted indicate that PC might be a linking factor in the development of the mental-somatic comorbidity. In one study, PC mediated the relationship between depressive and perceived somatic symptoms (e.g., nausea, dizziness, or heart rate increase), in a sample of college students (Harding, Murphy, & Mezulis, 2015). Further, PC was found to be a concurrent predictor of college students' post-traumatic stress symptoms, general mental health, as well as somatic symptoms (Zawadzki et al., 2018). These first results on the role of PC in both mental and somatic symptoms are compelling, however, an exploration of the role of PC in the comorbidity between clinical disorders is lacking. Moreover, the studies focused on only a single mental disorder and on subjective (but not objective) health outcomes. As a result, the ecological validity and consequently the generalizability and clinical relevance of the findings is limited, necessitating research that assesses *multiple* mental and somatic disorders in a *naturalistic* clinical sample.

The present study sought to explore the importance of PC for mental and somatic disorders in patients presenting with different mental disorders (i.e., depression, anxiety, addiction, ASD, ADHD) and comorbid somatic disorders (i.e., immune/-endocrine disorders, (cardio-)vascular disorders). Specifically, the primary aim was to examine the unique shared variances of the different disorders with PC, and was tested using multiple regression analyses supplemented with relative importance analyses. Identifying the diagnoses that are most strongly (or equally strong) related to PC will be a first step in localizing the specific disorders for which PC might possess a transdiagnostic function and potentially explain mental-somatic comorbidities. If particular mental and somatic disorders show both

significant and relatively important contributions to the prediction of PC, this can indicate that PC also impacts the development of comorbidity between them. Moreover, the results can speak to the generalizability of the perseverative cognition hypothesis to clinical populations and somatic disorders. Based on previous literature, we expected PC to be associated with all included disorders, but be the most important in explaining anxiety and depressive disorders. However, given the limited evidence in clinical samples using objective outcomes, the study was rather exploratory in nature. The results will support the generation of more specific hypotheses about the role of PC in the mental-somatic comorbidity, and can hence instigate the formation and elaboration of theory (Tonidandel & LeBreton, 2011).

Method

Participants

This study is part of the MIND-Set study, which aims at measuring integrated novel dimensions in a variety of mental disorders. The study is executed at the outpatient unit of the psychiatric department of the Radboud university medical center (Radboudumc), Nijmegen, the Netherlands. The department is specialized in the diagnosis and treatment of comorbidity of neurodevelopmental and stress-related disorders in adults, with a special attention for combined psychiatric and somatic pathology. Patients were eligible if they were 18 years or older and had a diagnosis of a mental disorder (depression, anxiety, addiction, ASD, ADHD). Exclusion criteria were a current psychosis, sensorimotor handicaps, a full scale IQ estimate <70, inadequate command of the Dutch language, or being mentally incompetent to sign an informed consent. Somatic comorbidity was allowed. Data of a total of 288 psychiatric patients (60.4% female; M_{age} = 39.91, SD_{age} = 13.92) was used in the current analyses. About 40% of the sample was highly educated (4.3% no education, 13.2% low education, 41.8% average education, 40.7% high education). The study was approved by the Ethical Review Board of the Radboudumc.

Measures

Socio-demographic information. Patients were asked to indicate their gender, highest level of education, and birth date. Age was calculated as the time from birth date to completion of the socio-demographic questionnaire (in years).

Psychiatric diagnoses. The Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1996) was used to diagnose depressive and anxiety disorders and to exclude psychotic disorders. The Measurements in the Addictions for Triage and Evaluation and criminality (MATE; Schippers & Broekman, 2010) was applied to diagnose addiction disorders according to DSM-IV.

ASD and ADHD were assessed with two instruments each – a self-report instrument for screening purposes, and a clinical interview for the final psychiatric diagnosis. The World Health Organization Adult ADHD Self-Report Scale (ASRS)-short version was administered for ADHD screening (Kessler, Adler, et al., 2005). When the screening was positive (six items, cut-off >3) the Diagnostic Interview for ADHD in adults (DIVA; Kooij & Francken, 2010) was conducted in a next step. For screening on ASD, the Autism-spectrum Quotient (AQ-50; Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001) was used. When a patient scored positive (50 items, cut-off >25) the Dutch interview for the diagnosis of ASD in adults (NIDA; Vuijk, 2014) was used subsequently. Both the DIVA and NIDA were completed in the presence of a partner and/or family member of the patient to be able to retrospectively and collaterally ascertain information on a broad range of symptoms in childhood and adulthood.

For the current analyses, we clustered the specific disorders based on the broader categories of the DSM-IV, namely into depressive disorders (i.e., depressive episode, dysthymia, manic episode), anxiety disorders (i.e., panic disorder, agoraphobia, social anxiety disorder, specific phobia, generalized anxiety disorder, and including obsessive-compulsive

disorder, post-traumatic stress disorder, unspecified anxiety disorder), addiction, ASD, and ADHD. All categories were scored as *present* (1; i.e., one or more diagnosed disorders in that category) or *absent* (0; i.e., no diagnosed disorder in that category).

Somatic comorbidity. Somatic comorbidity was measured using the self-report Statistics Netherlands (Centraal Bureau voor de Stastiek, see www.cbs.nl) questionnaire. For the current study we selected disorders whose development can theoretically be influenced by PC, and excluded disorders that are caused by external events, such as concussion. Included disorders were allergies (hay fever, eczema), lung disease (asthma, chronic bronchitis, pulmonary emphysema), heart disease (cardiac events, heart failure, heart infarction, cardiac arrhythmia, coronary heart disease, angina pectoris), vascular abnormalities, peripheral artery disease, hypertension, stroke, diabetes, arthritis/rheumatism (osteoarthritis, rheumatism), gastro-intestinal disease (gastric ulcer, irritable bowel syndrome, Crohn's disease, colitis ulcerosa, constipation), liver disease, and thyroid disease (Graves' disease, hyperthyroidism, hypothyroidism). The questionnaire is accurate in comparison to general practitioner information, and appears to be independent of cognitive impairment (Kriegsman, Penninx, van Eijk, Boeke, & Deeg, 1996).

Comparable to the psychiatric diagnoses and based on previous research on the effects of PC on endocrine, immune, and cardiovascular responses (Verkuil et al., 2010), we combined the somatic disorders into two clusters, namely 1) (cardio-)vascular disorders (peripheral artery disease, hypertension, heart disease, angina pectoris, stroke, vascular abnormalities), and 2) immune and endocrine disorders (allergies, intestinal disorders, lung disease, arthritis, rheumatism, gastric ulcer, thyroid disease, liver disease, diabetes). As the immune and endocrine system have been shown to cross-talk and to closely interact in the development of disease (such as with diabetes and osteoporosis; see Boutzios & Kaltsas,

2000; Dardenne & Savino, 1996; Taub, 2008), they are often clustered together (Verkuil et al., 2010). Again, clusters were scored as *present* (1) or *absent* (0).

Perseverative cognition. The perseverative thinking questionnaire (PTQ; Ehring et al., 2011) is a 15-item self-report questionnaire used to estimate patients' level of repetitive negative thinking. Items are rated on a 5-point scale ranging from 0 (*never*) to 4 (*almost always*). Three lower-order factors can be distinguished, namely 1) core characteristics of PC (repetitiveness, intrusiveness, difficulties to disengage; e.g., "The same thoughts keep going through my mind again and again"), 2) perceived unproductiveness of PC (e.g., "My thoughts are not much help to me"), and 3) mental capacity captured by PC (e.g., "I can't do anything else while thinking about my problems"). Studies confirm the adequate psychometric characteristics of the PTQ (Ehring, Raes, Weidacker, & Emmelkamp, 2012; Ehring et al., 2011). In the current study we used the total sum score, as well as the sum scores of the three subscales for analyses. Cronbach's alpha was .94 for the total scale, .93 for the core characteristics subscale (9 items), .75 for the perceived unproductiveness subscale (3 items), and .78 for the capturing mental capacity subscale (3 items).

Procedure

The study consisted of multiple phases. In the first phase, patients referred to the outpatient department were asked to fill in an electronic version of a set of questionnaires at home via an online secure questionnaire program, within 14 days before the appointment. Those who preferred a hard copy were sent a paper version of the questionnaires to their home address. Also, patients received the study's information letter at home. During this phase, patients provided socio-demographic information and completed the CBS questionnaire. Moreover, they were screened for autism (AQ-50) and ADHD (ASRS).

The second phase consisted of a face-to-face appointment. This three hour diagnostic examination included a psychiatric, biographical and somatic anamnesis, medication

verification, review of treatment history, structured clinical interviews and a physical examination. At the end of the examination, clinicians decided whether a patient was eligible for inclusion in the MIND-Set study, and if so, the patient was asked for informed consent. When the patient consented for participation, data from the structured interviews were also used for research purposes. After signing the informed consent, an appointment for the third phase was made within the next 14 days. During that third phase, patients were asked to complete another set of questionnaires, including the PTQ. Patients performed computer tasks and a subset of patients also performed tasks in a magnetic resonance imaging scanner; these data are not part of the current study.

Statistical Approach

All analyses were conducted using the R statistical program (R Core Team, 2018). In a first step, sample characteristics, the amount of comorbidity, and Pearson correlations among the study variables were computed, using the stat.desc function of the pastecs package (Grosjean & Ibanez, 2018), the cross.multi.table function of the questionr package (Barnier, Briatte, & Larmarange, 2018), and the rcor.test function of the ltm package (Rizopoulos, 2006), respectively. The main analyses consisted of four separate multiple regression models, with the PTQ total score and its three subscale scores as dependent variables. Predictors were depression, anxiety, addiction, ASD, ADHD, (cardio-)vascular disorders, and immune-/ endocrine disorders, all scored as present or absent. Moreover, we controlled for gender and age in all analyses, due to their known effects on mental and somatic disorders (e.g., Boyd et al., 2015; Dhingra & Vasan, 2012; Solberg et al., 2018).

Those multiple regression models were complemented by relative importance analyses. Relative importance analysis aims at examining how the different predictors in a regression model add up to the proportion of explained variance (i.e., R^2; Tonidandel & LeBreton, 2011). In other words, relative importance analysis overcomes the problem of

correlated predictors' standardized regression weights not adding up to the R^2, by taking into account both the unique and the shared variance of the predictors in explaining the outcome. In the present study, the lmg metric of the package relaimpo (Grömping, 2006) was used to obtain the relative importance (RI) of the different predictors in explaining the four PTQ scores. This metric estimates each predictor's unique R^2 contribution for every possible position in the regression model. A predictor's R^2 contribution averaged over the different orderings of predictors represents its RI. For easier interpretation, those estimates can be adjusted to sum to 100%. Additionally, bootstrapping was applied to calculate confidence intervals (CIs) for the RI estimates, and to determine whether the predictors significantly differ from each other in their RI.

Results

Sample Characteristics

Means and standard deviations of the PTQ scores, as well as the number of reported mental and somatic disorders are presented in Table 1. About two-third of the sample had at least one immune-/endocrine disorder, and about one-third of the sample reported at least one (cardio-)vascular disorder. With regard to mental disorders, nearly half of the sample experienced a depressive disorder, while less than one-fifth of the sample was diagnosed with addiction. About 30% of patients were diagnosed with at least one anxiety disorder (with n = 20 for panic disorder, n = 3 for agoraphobia, n = 26 for social anxiety disorder, n = 8 for specific phobia, n = 12 for obsessive-compulsive disorder, n = 18 for post-traumatic stress disorder, n = 19 for generalized anxiety disorder, and n = 9 for unspecified anxiety disorder).

The number of patients presenting with comorbidity among mental and somatic disorders is presented in Table 2. Overall comorbidity was high. For example, about one-third of the patients diagnosed with depression also had a diagnosis of anxiety, and about one-third of patients diagnosed with anxiety also had a diagnosis of ASD and ADHD. Moreover, half of

the patients diagnosed with addiction also had a diagnosis of ADHD. All mental disorders showed comorbidity with (cardio-)vascular disorders in about 25-35% of the cases, and comorbidity with immune-/endocrine disorders in about 60-70% of the cases.

Bivariate Correlations

Correlations among the mental disorders, somatic disorders, the PTQ and its subscales, as well as the covariates can be found in Table 1. The PTQ total score (continuous) was significantly positive correlated with the presence of depression, anxiety, and addiction (all scored as absent vs. present with 0 vs. 1). Also the PTQ subscales were significantly related to each of those disorders, with the exception of the association between anxiety and the capturing mental capacity subscale. ASD showed a small negative correlation with the perceived unproductiveness subscale, while ADHD did not demonstrate significant associations with the PTQ total or subscale scores. The somatic clusters were positively interrelated, but did not display significant associations with any of the PTQ scores and most of the mental disorders. Solely anxiety was significantly positive correlated with the (cardio-)vascular disorder cluster.

[Insert Table 1]

[Insert Table 2]

Multiple Regression and Relative Importance Analyses

PTQ total score. As presented in Table 3, the first model predicting the PTQ total score was statistically significant ($R^2 = .1560$, $F(9, 278) = 5.708$, $p < .001$), with the predictors collectively explaining 15.60% of the variance in the outcome. Only depression ($b = 5.71$, $p < .001$), anxiety ($b = 3.34$, $p = .008$), and addiction ($b = 5.22$, $p < .001$) emerged as significant positive predictors of the PTQ total score. Regarding the RI estimates (see also Figure 1), depression explained 51.1% of the total explained variance, followed by addiction (26.9%) and anxiety (14.8%). However – and probably due to their rather broad CIs – those three

predictors did not significantly differ from each other in their R^2 contribution (all $p > .05$). Depression showed a significantly higher contribution to the variance in PTQ than all remaining predictors (all $p < .05$). A similar pattern was found for addiction, which had a significantly higher RI contribution than all remaining predictors (all $p < .05$), except from age. Anxiety, on the contrary, did not have a significantly higher RI than any of the remaining predictors (all $p > .05$).

PTQ subscale scores. The three models predicting the core characteristics, perceived unproductiveness, and capturing mental capacity subscale of the PTQ showed similar results to the model predicting the PTQ total score (see Table 3). The explained variance, significance and RI of predictors, as well as the significant differences between RI estimates were comparable. However, in the model predicting the core characteristics subscale, ASD emerged as an additional significant predictor ($b = 1.74$, $p = .039$), although it only explained 5.3% of the total explained variance. With regard to the model predicting the perceived unproductiveness subscale, it was notable that depression not only explained 60.7% of the total explained variance, but also was the only predictor showing a significantly higher RI contribution than all other disorders (all $p < .05$), except addiction.

[Insert Table 3]

[Insert Figure 1]

Discussion

The current study explored the importance of PC for both mental and somatic disorders in a naturalistic psychiatric patient sample. In line with our expectations, depression, addiction, and anxiety showed significant associations with PC and its components, as well as the highest relative importance estimates. Unexpectedly, no significant or relatively important associations were found between PC and ASD, ADHD, or the somatic disorders. Taken together, the results question the generalizability of the perseverative cognition hypothesis to

somatic disorders, and consequently do not support the prediction that PC contributes to the comorbidity between mental and somatic disorders.

The finding that depression was predictive of PC is in line with studies highlighting the importance of PC in the onset and maintenance of emotional disorders (e.g., Arditte et al., 2016; Spinhoven et al., 2018) and the predictions of the perseverative cognition hypothesis (Brosschot et al., 2006). Addiction emerged as the second most important predictor of PC. This finding extends the literature on PC in psychopathology, which to date has primarily focused on anxiety and depressive disorders. It has been suggested that alcohol abuse can represent a maladaptive coping strategy to control the perseverative processes themselves, and/or the potentially subsequently developing negative affective states such as depression (Caselli et al., 2010). The independent relationship of both depression and addiction with PC in the current study points to a direct relationship between PC and addiction, which is not necessarily mediated by the development of depression. Our findings thus underscore the relevance of targeting PC in the treatment of depressive disorders, and may indicate that addiction treatment and the treatment of comorbidity between depression and addiction might similarly benefit from a focus on alleviating PC.

Contrary to the perseverative cognition hypothesis and previous empirical findings (Spinhoven et al., 2019), anxiety did not show a significant bivariate association with the capturing mental capacity component of PC. Likewise, its relative contribution to PC was not significantly higher than that of the other disorders. It is possible that the clustering of the separate mental disorders into broader categories has created more heterogeneity within the category of anxiety disorders compared to the other disorder categories examined. PC might be characteristic only for a subgroup of those anxiety disorders, so that their predictive value and relative importance for PC is reduced if analyzed as a single construct. Support for this

explanation comes from a study showing that the magnitude of the relationship between PC and anxiety differs between specific anxiety diagnoses (Spinhoven et al., 2015).

ASD was only significantly related to the core characteristics component of PC, but its relative contribution was low. ADHD was not related to overall PC or any of its components. The absence of important associations between those disorders and PC is contradicting previous studies that report heightened levels of PC in ASD (Crane et al., 2013; Mazefsky et al., 2014) and ADHD (Mitchell et al., 2013). Both ASD and ADHD are neurodevelopmental disorders with an early onset, which possibly reduces the impact of prolonged stress on their development. Hence, PC might not represent a core feature of ASD or ADHD. However, it might exacerbate their negative consequences, for instance by amplifying the negative impact of the heightened amount of stress that individuals with ADHD and ASD experience (Bishop-Fitzpatrick, Mazefsky, Minshew, & Eack, 2015; Yeguez, Hill, Buitron, & Pettit, 2018).

The finding that neither immune-/endocrine disorders nor (cardio-)vascular disorders were related to PC is especially surprising given the wealth of research that relates PC to markers of somatic disease (e.g., Ottaviani et al., 2016). According to the perseverative cognition hypothesis, this pathogenic state should in a next step lead to (diagnosable) clinical disorders (Verkuil et al., 2011). Different explanations for the absence of this relationship in somatic disorders are possible. First, perhaps the pathways to somatic disease are altered in individuals with psychopathology. In support, a study by Huffziger et al. (2013) suggests that in remitted depressed patients the hypothalamic-pituitary-adrenal axis is less responsive to daily emotional experiences than in healthy controls. Second, PC has been described as a proximal risk factor for mental disorders (Spinhoven et al., 2018). In the case of somatic disease it might represent a more distal risk factor, which allows for the influence of other factors, such as diet and exercise (McEwen, 1998; Nolen-Hoeksema & Watkins, 2011). The presence of PC might, however, influence the appraisal of amplified physiological activity,

thereby explaining the link between PC and subjective health complaints (Brosschot, 2002; Eriksen & Ursin, 2004). Third, PC also includes unconscious processes, which are thought to be responsible for a vast amount of the prolonged stress-related physiological activity (Brosschot, 2010). Conscious and unconscious types of PC might be differentially related to mental and somatic disorders, a notion that will need to be addressed in future studies.

Strengths and limitations of the current study should be considered. The representativeness of our naturalistic sample for patients seen in clinical practice ensures that the findings have clinical relevance. It should be noted that this representativeness concerns *psychiatric* samples, and that studying samples with primarily somatic pathology might yield different results. In a similar vein, the educational level of the sample was rather high (cf. van Noorden et al., 2010; Vrijsen et al., 2017). As higher education is related to a healthier lifestyle and better somatic health (Steinvil et al., 2008), this composition could explain the somewhat lower somatic comorbidity in the current sample compared with more diverse psychiatric populations (see Appendix; Sokal et al., 2004). On another note, the results might have been influenced by medication use (Biffi, Scotti, & Corrao, 2017; Maslej et al., 2017). The clustering of the mental and somatic disorders represents a limitation of the measurement approach, as we did not examine the specificity of the disorders in relation to PC and did not take their severity into account. Simultaneously, the heterogeneity within clusters might have caused the broad confidence intervals for some of the disorders, due to which only tentative conclusions can be drawn. Another limitation concerns the cross-sectional design. Studies with longitudinal designs are required to identify whether PC, as proposed by the perseverative cognition hypothesis (Brosschot et al., 2006), plays a causal role in the emergence of comorbidity and to investigate its importance in different stages of disease.

Concluding, the current study is – to the best of our knowledge – the first to explore the role of PC for a variety of objective mental and somatic disorders in a naturalistic patient

sample. Our results provide evidence for the importance of PC in depression and addiction, but not for its contribution to somatic comorbidity in psychiatric samples. The study thereby substantiates the literature on the perseverative cognition hypothesis in clinical disorders and directs future research. This research should further examine the contribution of PC to the mental-somatic comorbidity in samples with varying types and degrees of mental and somatic pathology.

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Table 1

Descriptive Statistics of and Bivariate Pearson Correlations Among the Mental and Somatic Disorders, PTQ Scores, and Covariates

Variable	*M* (*SD*); or % (*n*) present	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Gender	60.4% (174)[1]	-												
2. Age	39.91 (13.92)	-.08	-											
3. Depression	47.6% (137)	.03	.08	-										
4. Anxiety	30.2% (87)	.06	-.01	.06	-									
5. Addiction	17.4% (50)	-.05	.04	.13*	-.02	-								
6. ASD	27.1% (78)	-.03	-.12*	-.16**	.08	-.18**	-							
7. ADHD	36.8% (106)	.07	-.15**	-.18**	-.05	.14*	-.04	-						
8. CV cluster	28.1% (81)	-.08	.25***	.10	.14*	-.04	-.05	-.01	-					
9. IE cluster	66.7% (192)	.12*	.20**	.01	.00	.01	-.08	.07	.16**	-				
10. PTQ-tot	35.66 (10.27)	.03	.07	.30***	.16**	.22***	-.01	-.03	-.00	.05	-			
11. PTQ-CC	22.24 (6.44)	.07	.06	.26***	.17**	.20**	.05	-.03	.00	.08	.97***	-		
12. PTQ-U	6.80 (2.28)	.01	.06	.32***	.12*	.18**	-.12*	-.06	-.05	.03	.84***	.74***	-	
13. PTQ-CM	6.62 (2.44)	-.06	.07	.26***	.11	.23***	-.06	.01	.03	-.02	.86***	.75***	.66***	-

Note. $N = 288$. *M (SD)* = mean and standard deviation of continuous variables; % (*n*) present = percentage (number) present of categorical variables; CV cluster = (cardio-)vascular disorder cluster; IE cluster = immune-/endocrine disorder cluster; PTQ-tot = PTQ total score; PTQ-CC = PTQ core characteristics subscale; PTQ-U = PTQ perceived unproductiveness subscale; PTQ-CM = PTQ capturing mental capacity subscale

[1]percentage (number) of females in the sample

*$p < .05$, **$p < .01$, ***$p < .001$

Table 2

Number of Patients Presenting with Comorbidity Among Mental and Somatic Disorders

Disorder 1 (*n*)	Disorder 2 (*n*)	Disorder 1 and 2 (*n*)	Relative percentage	Absolute percentage
Depression	Anxiety (87)	45	32.8	15.6
(137)	Addiction (50)	31	22.6	10.8
	ASD (78)	27	19.7	9.4
	ADHD (106)	38	27.7	13.2
	CV cluster (81)	45	32.8	15.6
	IE cluster (192)	92	67.2	31.9
Anxiety	Addiction (50)	14	16.1	4.9
(87)	ASD (78)	28	32.2	9.7
	ADHD (106)	29	33.3	10.1
	CV cluster (81)	33	37.9	11.5
	IE cluster (192)	58	66.7	20.1
Addiction	ASD (78)	5	10.0	1.7
(50)	ADHD (106)	26	52.0	9.0
	CV cluster (81)	12	24.0	4.2
	IE cluster (192)	34	68.0	11.8
ASD	ADHD (106)	26	33.3	9.0
(78)	CV cluster (81)	19	24.4	6.6
	IE cluster (192)	47	60.3	16.3
ADHD	CV cluster (81)	29	27.4	10.1
(106)	IE cluster (192)	75	70.8	26.0
CV cluster	IE cluster (192)	64	78.0	22.2
(81)				

Note. $N = 288$. CV cluster = (cardio-)vascular disorder cluster; IE cluster = immune-/ endocrine disorder cluster; relative percentage = percentage of patients with disorder 1 also diagnosed with disorder 2 (disorder 1 and 2 (*n*)/ disorder 1 (*n*) *100); absolute percentage =

percentage of *total* sample diagnosed with the given comorbidity (disorder 1 and 2 (*n*) / 288 * 100)

Table 3

Multiple Regression and Relative Importance Estimates Predicting PTQ and its Subscales

	PTQ Total Score						
	b	*SE b*	*β*	*t*	*p*	RI	RI adj.
(intercept)	28.36	2.19		12.966	<.001***		
Depression	5.71	1.19	.28	4.807	<.001***	.080	.511
Anxiety	3.34	1.25	.15	2.659	.008**	.023	.148
Addiction	5.22	1.56	.19	3.358	<.001***	.042	.269
ASD	1.50	1.32	.07	1.138	.256	.002	.012
ADHD	0.12	1.24	.01	0.099	.921	.001	.006
CV cluster	-1.37	1.34	-.06	-1.025	.306	.001	.010
IE cluster	1.08	1.25	.05	0.864	.389	.002	.016
Gender	0.52	1.18	.02	0.435	.664	.001	.006
Age	0.04	0.04	.06	0.968	.334	.003	.022
R^2		0.1560					
F for R^2		5.708					

	Core Characteristics Subscale						
	b	*SE b*	*β*	*t*	*p*	RI	RI adj.
(intercept)	17.25	1.38		12.520	<.001***		
Depression	3.19	0.75	.25	4.263	<.001***	.063	.422
Anxiety	2.12	0.79	.15	2.682	.008**	.025	.168
Addiction	3.23	0.98	.19	3.293	.001**	.037	.251
ASD	1.74	0.83	.12	2.084	.039*	.008	.053
ADHD	-0.04	0.78	-.00	-0.047	.963	.001	.006
CV cluster	-0.70	0.84	-.05	-0.830	.407	.001	.007
IE cluster	1.07	0.79	.08	1.363	.174	.006	.042
Gender	0.84	0.75	.06	1.124	.262	.005	.033
Age	0.02	0.03	.05	0.883	.378	.003	.019
R^2		0.1484					
F for R^2		5.383					

	Perceived Unproductiveness Subscale						
	b	*SE b*	*β*	*t*	*p*	RI	RI adj.
(intercept)	5.72	0.49		11.752	<.001***		
Depression	1.35	0.26	.30	5.097	<.001***	.092	.607
Anxiety	0.62	0.28	.12	2.204	.028*	.014	.094
Addiction	0.78	0.35	.13	2.249	.025*	.024	.160
ASD	-0.29	0.29	-.06	-0.994	.321	.008	.053
ADHD	-0.08	0.28	-.02	-0.306	.760	.002	.016
CV cluster	-0.57	0.30	-.11	-1.922	.056	.007	.046
IE cluster	0.13	0.28	.03	0.480	.632	.001	.004
Gender	-0.04	0.26	-.01	-0.145	.885	.000	.001
Age	0.01	0.01	.05	0.814	.416	.003	.018
R^2		0.1510					
F for R^2		5.496					
	Capturing Mental Capacity Subscale						
	b	*SE b*	*β*	*t*	*p*	RI	RI adj.
(intercept)	5.39	0.53		10.180	<.001***		
Depression	1.17	0.29	.24	4.081	<.001***	.060	.476
Anxiety	0.60	0.30	.11	1.980	.049*	.013	.101
Addiction	1.22	0.38	.19	3.235	.001**	.043	.342
ASD	0.06	0.32	.01	0.191	.849	.001	.010
ADHD	0.24	0.30	.05	0.813	.417	.002	.012
CV cluster	-0.01	0.32	-.02	-0.308	.758	.000	.003
IE cluster	-0.13	0.30	-.02	-0.420	.675	.001	.004
Gender	-0.28	0.29	-.06	-0.994	.321	.003	.026
Age	0.01	0.01	.06	0.950	.343	.003	.027
R^2		0.1262					
F for R^2		4.460					

Note. All models were significant with $p < .001$. RI = relative importance estimate; RI adj. = RI metrics are normalized to sum to 100%; CV cluster = (cardio-)vascular disorder cluster; IE cluster = immune-/endocrine disorder cluster

*$p < .05$, **$p < .01$, ***$p < .001$

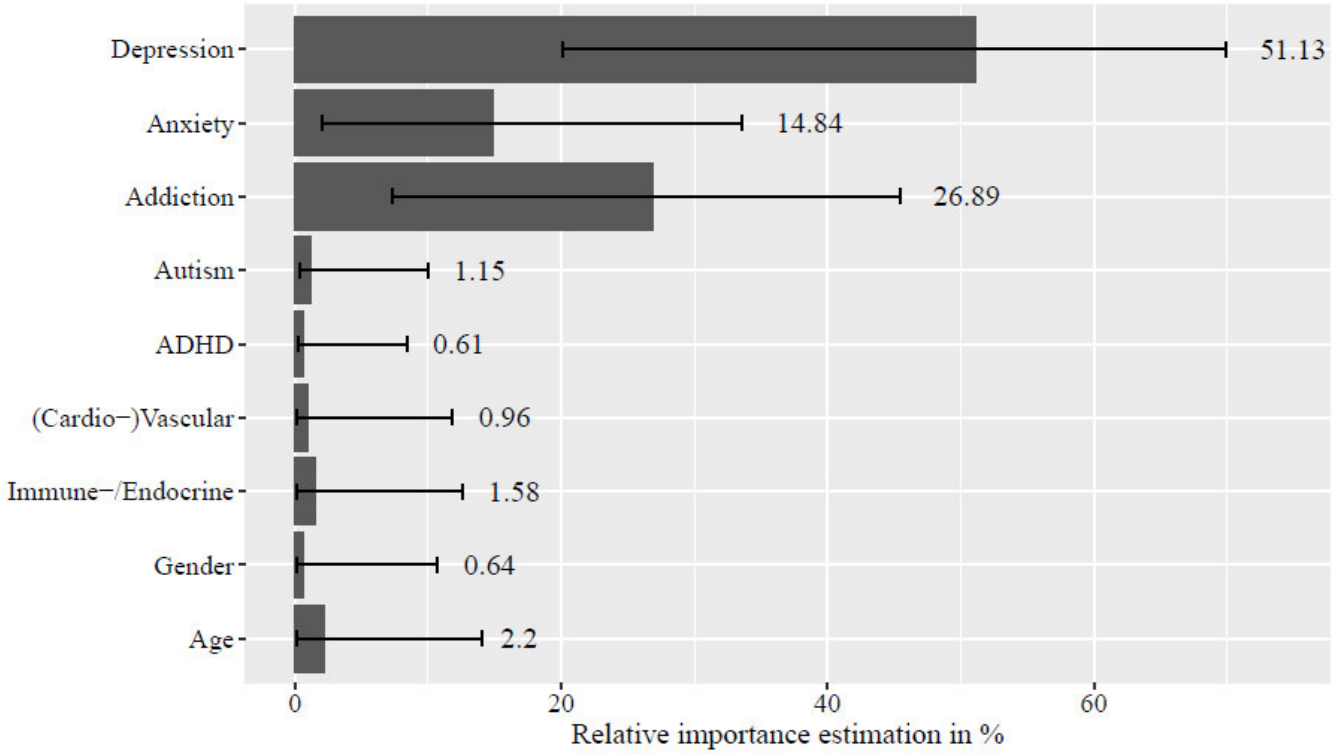

Figure 1. Relative importance coefficients of the different mental and somatic disorder clusters on the PTQ total score, including bootstrapped 95% confidence intervals. Each value represents the unique shared variance between the disorder and the PTQ total score, controlling for gender and age. Estimates are adjusted to sum to 100%.

Appendix

Table A1

Amount of Reported Somatic Disorders Used for Clustering

Somatic cluster	Somatic disorder	*n* present	% present
(Cardio-)Vascular disorders	Peripheral artery disease	34	11.8
	Hypertension	27	9.4
	Heart disease	20	6.9
	Angina pectoris	25	8.7
	Stroke	7	2.4
	Vascular abnormalities	5	1.7
Immune-/Endocrine disorders	Allergies	139	48.3
	Intestinal disorders	52	18.1
	Lung disease	44	15.3
	Arthritis	38	13.2
	Rheumatism	18	6.3
	Gastric ulcer	13	4.5
	Thyroid disease	11	3.8
	Liver disease	2	0.7
	Diabetes	10	3.5

Note. $N = 288$. *n* present = number of patients reporting the given disorder; % present = percentage of patients reporting the given disorder (*n* present / 288 * 100)