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Do depressive symptoms predict paranoia or vice versa?

Steffen Moritz [a, *], Anja S. Göritz [b], Benjamin McLean [c], Stefan Westermann [d], Jeannette Brodbeck [d]

[a] Department of Psychiatry and Psychotherapy, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
[b] Occupational and Consumer Psychology, Freiburg University, Freiburg, Germany
[c] School of Psychology, Flinders University, South Australia, Australia
[d] Department of Clinical Psychology and Psychotherapy, University of Bern, Switzerland

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ABSTRACT

Background and objectives: Affective versus nonaffective psychoses are today no longer regarded as mutually exclusive disorders. Theorists have recently highlighted the role of affective symptoms in the formation of paranoid beliefs, particularly negative beliefs about the self, interpersonal sensitivity, sleep disturbances, and worrying, which exist along a continuum in the general population. For the present study, we tested the bidirectional causal relationships between paranoia and affect.
Method: A large population sample ($N = 2,357$) was examined at three time-points (baseline, six months, two years) as to the severity of subclinical paranoid beliefs (Paranoia Checklist, PCL) and depressive symptoms (Patient Health Questionnaire-9, PHQ-9). Worrying and avoidance were measured with items from the Maladaptive and Adaptive Coping Style Questionnaire (MAX).
Results: Depression and paranoid symptoms were strongly cross-sectionally related ($r = 0.69$) and showed high stability ($r > 0.72$). Depressive symptoms at T2 predicted paranoid symptoms at T3 (beta = 0.16; no significant relationship from T1 to T2), whereas paranoid symptoms predicted depressive symptoms from T1 to T2 (beta = 0.09; no significant relationship from T2 to T3).
Limitations: Results should be replicated in a sample of paranoid patients, as risk factors for subclinical versus manifest paranoia may differ. Some constructs were measured with single items derived from a new scale.
Conclusions: The predictive association of depression to subsequent paranoia was small and confined to the long interval from T2 to T3. Treatments should target both paranoia and depression – irrespective of their causal relationship – particularly as patients with psychosis consider treatment of their emotional problems a priority.

1. Introduction

Until the 1950s, the Kraepelian dichotomy of affective (depression, bipolar disorder) versus nonaffective disorders ("dementia praecox", later relabeled schizophrenia) was almost undisputed (Craddock & Owen, 2007). Initially the key difference between the two entities was seen in their course; early cognitive decline to the point of dementia was assumed for dementia praecox only, notwithstanding that Kraepelin himself had seen a number of cases who did not show premature cognitive decline, and had even (partially) recovered (Kraepelin, 1919, p. 184). In addition, the etiologies of both groups of disorders were regarded as distinct. Kraepelin suspected autointoxication as the major cause of dementia praecox/schizophrenia but not of affective disorders (Kraepelin, 1919, p. 244). This dichotomy was adopted by many clinicians and researchers, but as neither prognostic nor etiological aspects qualified as clear discriminators between the two disorders, phenomenological differences were (over-)emphasized in the following years. The strict theoretical separation between the two disorders crumbled with the introduction of the schizoaffective psychosis diagnosis (Kasanin, 1933), but it was still decades before psychiatrists and psychologists were open to the possibility that

* Corresponding author.
E-mail address: moritz@uke.uni-hamburg.de (S. Moritz).

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symptoms of both entities might combine as a rule rather than the exception, and that many patients might suffer from both conditions in the course of their illness.[1] Some theorists, perhaps most prominently Crow (1995), even called for a return to the concept of *Einheitspsychose* (unitary psychosis) that was prevalent before the time of Kraepelin.

Meta-analyses suggest that at least 50% of schizophrenia patients suffer from major depression (Buckley, Miller, Lehrer, & Castle, 2009). The lifetime prevalence of depressive mood (≥2 weeks) at first admission for schizophrenia has been estimated at 83%, and 71% of first episode patients present with clinically relevant depressive symptoms (Häfner et al., 2005). In her review, Upthegrove (2009) reported that the prevalence of depression is 45–83% in the prodrome, 29–75% in the acute phase, and 5–54% post-psychosis.

In addition to depressive symptoms, other affective features such as the elated mood typical of mania/bipolar disorder are often present in schizophrenia, including in those presenting with paranoid delusions, as the mood of many individuals with psychosis is elevated by the importance imparted to them by their paranoid belief; such individuals may believe they are persecuted in envy of their special knowledge or superpowers (Moritz et al., 2015; Sundag, Lincoln, Hartmann, & Moritz, 2014). This phenomenon had already been observed by Kraepelin (1899, p. 194), and has recently been associated with medication nonadherence (Moritz et al., 2013). In addition, there is reason to believe that a number of the so-called negative symptoms of schizophrenia are in fact disguised depressive symptoms (Burckhardt, 2012; Moritz, Jahns et al., 2016a; Moritz, Schröder, et al., 2016b), that are relabeled once a diagnosis of schizophrenia is made (e.g., lack of drive versus avolition).

1.1. Depression as a risk factor for psychosis

The idea that depressive symptoms represent antecedents and perhaps even risk factors for paranoid delusions, the central symptom of schizophrenia,[2] has garnered much empirical support over the years (Upthegrove, 2009). One of the first empirical trials to examine the precursors of schizophrenia, the ABC study (Häfner, Maurer, & An Der Heiden, 2013), demonstrated that depressive symptoms indeed precede schizophrenia years before psychotic breakdown. More recently, Freeman and Garety (2014) proposed a model that ascribes a pivotal role to core depressive and depression-related symptoms (i.e., a worry thinking style, negative beliefs about the self, interpersonal sensitivity, and sleep disturbance), reasoning biases (particularly jumping to conclusions), and anomalous internal experiences in the pathogenesis of paranoid beliefs (the most prevalent form of delusions in schizophrenia). In an update of the model (Freeman, 2016), Freeman added safety-seeking behaviors/avoidance to the list of risk factors, because they hinder the individual from processing evidence inconsistent with the persecutory belief (Garety & Freeman, 2013).

Evidence for the validity of this model stems from two lines of research: 1) Most studies confirm the (predictive) relationship of negative affect with paranoia in both clinical and nonclinical populations (Freeman et al., 2012; Freeman, Dunn, Murray, et al., 2015; Kramer et al., 2014; Morrison et al., 2015; Tone, Goulding, & Compton, 2011), or its mediating role (Freeman, Emsley, et al., 2014); 2) Interventions targeted at affective symptoms (e.g., self-imagery, mindfulness, Yoga, CBT for insomnia) have shown promise in reducing delusions (Bullock, Newman-Taylor, & Stopa, 2016; Ellett, 2013; Freeman, Pugh, et al., 2014; Freeman, Dunn, Startup, et al., 2015; Myers, Startup, & Freeman, 2011; Visceglia & Lewis, 2011). In the discussion we will also highlight some exceptions.

Although the aforementioned studies strongly indicate that core depressive as well as depression-related symptoms are involved in the pathogenesis of paranoia, the mechanisms that translate emotional problems into paranoid but not other symptoms are not yet well understood. Moreover, it is unclear if the set of emotional factors in the model of Garety and Freeman (Freeman & Garety, 2014) is complete, or whether depressive symptoms not considered in the model, such as lack of drive, may carry equal importance.

1.2. The present study

The present study explored to what degree depressive symptoms as well as dysfunctional coping styles (worrying, avoidance) predict later paranoia, and vice versa. We recruited a large population sample, as paranoia and other psychotic features are widely distributed in the general population (Ellett, Lopes, & Chadwick, 2003; Scott, Chant, Andrews, & McGrath, 2006; van Os, Linscott, Myin-Germeys, Delespaul, & Krabbendam, 2009; Verdoux & Van Os, 2002). Our study builds upon the quasi-dimensional model of psychosis advocated by Bleuler (1950) and Meehl (1962). The model posits that subclinical psychopathological expressions exist to different degrees in the general population, whereby only a minority of individuals (i.e., those who surpass a certain threshold of severity and distress) require treatment. This dimensional view is analogous to the relationship between blood pressure and heart attack/stroke; a blood pressure of 140/90 may be tolerated, but higher pressure may require treatment and must be viewed as pathological as it is a risk factor for the development of cardiovascular disorders.

2. Method

2.1. Participants

We conducted the study using WisoPanel, an online service that provides scientists with the opportunity to carry out empirical studies (for the reliability of this and related services, as well as online research in general, see Göritz, 2007; Judge et al., 2006; Piccolo & Colquitt, 2006). WiSoPanel is administered using phpPanelAdmin (Göritz, 2009). As an incentive for participation, a PDF-manual that featured mindfulness and relaxation exercises was offered for download at the end of each survey wave. There was no financial compensation. All participants provided informed consent before participation and the study complied with the Declaration of Helsinki.

At the time of the first assessment, 12,087 individuals from the general population registered with WisoPanel were invited to participate. A total of 2,357 participants completed the web-based survey at T1. Blind to results and before analyses were initiated, 128 participants at T1 were discarded because of unusual response patterns (entering the same value at least 20 out of 21 times) within the Maladaptive and Adaptive Coping Style Questionnaire (Moritz, Jahns et al., 2016a; Moritz, Schröder, et al., 2016b), and 2,229 participants at T1 were considered for inclusion in the final analyses. Of those, 1,124 individuals responded to our follow-up invitation six months later (T2; 50.43% of the initial sample), and 705 individuals took part two years after T2 (T3; 62.56% of T2, 31.63% of the initial sample). A total of 511 participants (22.92%) took part in all three waves.

Part of the collected data exploring the validity and reliability of this new coping questionnaire and its symptomatic correlates has been previously published (Moritz, Jahns et al., 2016a; Moritz, Schröder, et al., 2016b), however the analyses presented here do

[1] Kraepelin (1919) was well aware of a "mingling of morbid symptoms of both psychoses" in at least some cases.

[2] Hallucinations and disturbances of ego-boundaries may also be conceptualized as delusions as it is the presence of a delusional superstructure (e.g. thoughts or voices *being inserted into* one's head) that qualifies these otherwise psychosomatic or sensory phenomena as psychotic.