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The Interplay between Social Factors and Psychotic Symptoms:

Cognitive Vulnerability and Affective Pathway in Focus

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”…the role of evidence is, in the main, to correct our mistakes, our prejudices, our tentative theories—that is, to play a part in the critical discussion, in the elimination of error. By correcting our mistakes, we raise new problems. And in order to solve these problems, we invent conjectures, that is, tentative theories, which we submit to critical discussion, directed towards the elimination of error.”

Karl Raimund Popper
The Myth of the Framework: In Defence of Science and Rationality (1993), 140

Table of Contents

Abstract

Psychotic disorder is a disabling mental disorder listed as the 8th leading cause of living with disability for people between the age of 15 and 44 according to the World Health Report by WHO in 2001. The question of what are the causes of psychotic disorder, however, remains elusive. Drawing from classic twin studies, psychotic disorder has been suggested to be highly heritable, but recent findings from genome wide association studies imply that this may have been overestimated. More recently, evidence has accumulated demonstrating the importance of environmental factors, particularly social factors. However, despite the numerous studies showing associations between various social factors and psychotic disorder, the mechanism that explains this relationship remains unclear. The present dissertation aimed to address these research gaps by testing hypotheses drawn mainly from two theoretical frameworks, the social defeat hypothesis and cognitive model of psychosis.

The social defeat hypothesis argues that the common denominator of various apparently different social risk factors is the negative experience of being excluded from the majority group, and that this social defeat experience has a causal influence on psychosis. The first study in this dissertation tested this hypothesis by investigating whether various social adversities can be summed up into one latent common factor, and whether this factor has an effect on psychotic symptoms while controlling for its effect on depression and anxiety. Competing hypotheses arguing for the reverse effect were also tested. This first study was a cross-sectional survey in a community sample (N = 2350) from Germany (n = 786), Indonesia (n = 844), and the United States (n = 720). Confirmatory factor analysis (CFA) and path analysis with structural equation modeling were used to test the hypotheses. In the CFA two latent factors reflecting current and past experiences of social defeat could be identified with acceptable model fit. Path analysis indicated acceptable fit for both social defeat and reverse models, and both the path from current social defeat to psychotic symptoms and the reverse

path were significant, although the reverse path was weaker. Interestingly, the current but not the past social defeat factor was significantly associated with psychotic symptoms. Overall, the results indicate that postulates derived from the social defeat hypothesis fit the data.

The second study aimed to discover the psychological mechanisms explaining the association between social defeat and psychosis found in the first study. Drawing from cognitive model of psychosis, several putative mediators indicating cognitive vulnerability were tested. Specifically, based on the sample of the first study, low perceived social rank, negative schemas related to self-and other, and loneliness were tested as plausible mediators. Mediation path analysis with structural equation modeling was used. Social rank, negative schemas, and loneliness significantly mediated the relationship between social defeat and negative symptoms and the models explained a large amount of the variance (R^2 = .43 - .44). For positive symptoms, only negative schemas were a significant mediator (R^2 = .27). The results support the assumption that cognitive vulnerability is a relevant translating mechanism as postulated by cognitive models of psychosis.

After discovering that negative-self schemas were the most important mediator, in the third study the postulate from cognitive models of psychosis that negative-self beliefs both cause and maintain psychotic symptoms via negative affect was examined. A longitudinal cohort design in a community sample (N = 962) from Germany, Indonesia, and the United States was used. Negative-self schema, negative affect, and psychotic symptoms were measured repeatedly every four months (baseline, 4-, 8-, and 12-months-follow-up). Cross-lagged panel and longitudinal mediation analyses with structural equation modeling were used. Independent cross-lagged panel models showed a significant unidirectional longitudinal path from negative-self schema to psychotic symptoms and bidirectional longitudinal associations between negative-self schema to negative affect, suggesting a vicious cycle of schema and affect. Furthermore, there was a significant indirect effect pathway of negative

schema (baseline) via negative affect (4-months-follow-up), maintained over the course of eight months (8-months-follow-up) to psychotic symptoms (12-months-follow-up) suggesting mediation. Our findings support the postulated affective pathway from negative-self schema to symptoms. Specifically, negative-self schema and affect formed a vicious cycle building up over the course of several months, before impacting upon psychotic symptoms.

Overall, the results contribute to extending the theoretical framework of social defeat hypothesis and cognitive models of psychosis. Importantly, psychological mechanisms of the relationship between social defeat and psychosis derived from cognitive models of psychosis were empirically found. In this regard, negative schemas were the most important mediator that transmitted its effect onto psychotic symptoms via negative affect. One clinical implication is that breaking the vicious cycle by targeting negative-self schema and negative affect in populations vulnerable to social defeat may be a viable prevention strategy. This approach may be guided by CBT techniques such as cognitive restructuring for negative schema and improving emotion regulation skills for negative affect.

1 Theoretical Background

1.1. Schizophrenia and the continuum of psychosis

Schizophrenia is a mental condition with severe consequences for the individual and the community costing on average 106 USD thousand per person per year (Evensen et al., 2015). According to the DSM-5, schizophrenia is characterized with at least two of the following symptoms occurring notably in a one month period, delusion, hallucination, disorganized speech, grossly disorganized or catatonic behavior, and negative symptoms (American Psychiatric Association, 2013). The diagnostic criteria emphasized that at least one of the symptoms must be delusions, hallucinations, or disorganized speech. However, the most clinically important symptoms are arguably persecutory delusion (Freeman & Garety, 2014) and hallucinations (Beck & Rector, 2003). Persecutory delusion has been reported to be experienced by 70% of first episode psychosis patients (Coid et al., 2013). Similarly, 70% of schizophrenia patients reported to have experienced hallucinations (Sartorius et al., 1986).

However, the view that schizophrenia is a diagnostic category has been contested for some time. Since 50 years ago, the idea that delusions and hallucinations are discrete, discontinuous phenomena with nothing in between has been challenged (Strauss, 1969). This lead to the idea of a continuum of psychosis that has since been elaborated, particularly by van Os and colleagues (Johns & van Os, 2001; Linscott & van Os, 2010).

Because there has been more than one theory of continuum of psychosis, several tenets of the notion of continuum of psychosis adopted in this dissertation are worth elaborating. First, the idea of a psychosis continuum does not imply a continuum of disorder. This is important to be stated because even though around 5.8% of the general population in 18 countries around the globe reports having some kind of psychotic experiences (McGrath et al., 2015), only 0.55% of the population is diagnosed with schizophrenia (McGrath, Saha, Chant,

& Welham, 2008). Secondly, the psychosis continuum is most likely not normally distributed. In particular, the distribution is most likely to be positively skewed (Johns & van Os, 2001). This distribution means that most people have no to very low level of symptoms and there is a significant proportion of the population with non-zero values. Importantly, this hypothesized distribution has been demonstrated exactly as predicted in regard to paranoid thoughts in the general population (Freeman et al., 2005). Third, the continuum view of psychosis holds that psychotic symptoms at the subclinical level, also called psychotic experiences, are on the same continuum with psychotic disorder. Psychotic symptoms experienced by individuals without a diagnosis of psychotic disorder differ only in frequency, severity, and/or distress in comparison to psychotic symptoms experienced by individuals with a diagnosis of psychotic disorder. In other words, they do not differ in kind. This interpretation of the continuum is called the psychosis-proneness-persistence-impairment model (Linscott & van Os, 2013). Specifically, this model postulated that the early expression of psychosis-proneness is transient and relatively common. However, stressors and risk factors may act to prolong and increase the severity of the symptoms thereby increasing the probability of the development of clinically significant psychotic symptoms. This model implies continuity in a phenomenological and temporal sense. Evidence for this model comes from a meta-analysis utilizing odds ratios from various studies that investigated the risk factors of presence of psychotic symptoms and found striking similarity with risk factors of schizophrenia (Linscott & van Os, 2013). They therefore conclude that psychotic symptoms at the subclinical level behave like psychotic disorders, in the sense that the same factors contributing to the development and maintenance of psychotic symptoms at the subclinical level may also contribute to psychotic symptoms at the clinical level. Indeed, this assumption has been strengthened with evidence coming from a twin study showing that psychotic experiences are hereditary to an extent (Zavos et al., 2014). Additionally, they found that both severe and non-

severe psychotic experiences have the same rate of hereditability estimate suggesting consistent etiology as postulated by the psychosis-proneness-impairment model.

1.2. The etiology of schizophrenia: The social defeat hypothesis

Despite years of research, the question of what are the causes of psychotic disorder has not arrive at a conclusive answer. Drawing from classic twin studies, psychotic disorder has been suggested to be highly heritable (Tandon, Keshavan, & Nasrallah, 2008), but recent findings from genome wide association studies suggest that this may have been overestimated (Schizophrenia Working Group of the Psychiatric Genomics Consortium, 2014). More recently, evidence has accumulated suggesting the importance of environmental factors, particularly social factors.

Environmental factors, particularly those of social nature, have been repeatedly shown to be a risk factor of psychosis. Social risk factors of psychosis at a clinical and subclinical level are, for example, migration, experiencing discrimination, low socio-economic status, and childhood trauma (Linscott & van Os, 2013). In response to the accumulating findings of various social risk factors of psychosis, a theoretical framework explaining these findings was proposed called the social defeat hypothesis (SD, Selten, van der Ven, Rutten, & Cantor-Graae, 2013). The SD hypothesis argues that the common denominator of these social risk factors is the negative experience of being excluded from the majority group. The notion stems from animal experimental research, specifically from a well-known phenomenon that rodents exposed to stress induced with SD (e.g. forcing them to live with a dominant rodent) exhibit behavioral markers of psychosis such as fear of other rodents, similar to paranoia (Badowska, Brzózka, Chowdhury, Malzahn, & Rossner, 2015).

The SD hypothesis argues that SD is causally related to schizophrenia. However, establishing causality has always been an elusive endeavor, and particularly so for clinical

psychology and psychiatry (for a discussion, see Kendler, 2012) . To establish SD as a causal factor of schizophrenia, as explained in Selten et al. (2013) two alternative explanations to the association between SD risk factors and schizophrenia have to be examined, 1) the observed association is due to genetic confounding and 2) SD is not a specific risk factor for schizophrenia. These two alternative explanations point out that SD is a consequence, rather than a cause of schizophrenia. First, the genetic confounding explanation assumes that factors that have been found to be associated with schizophrenia such as having a migration background and living in urban areas are the consequence of a schizophrenia genetic predisposition. In other words, this explanation assumes that people who are genetically predisposed to schizophrenia move to urban areas, emigrate due to failing to integrate in home country (i.e. social selection hypothesis (Ødegård, 1932), victimized during childhood due to poor social skills, and use illicit drugs because they are unhappy. However, this genetic confounding explanation rests on too many challenges (see Selten et al., 2013). The most important challenge is the requirement to discover one group of genes that is responsible for many factors at the same time, i.e. causing schizophrenia, moving to urban areas, emigrating, being bullied, and using drugs. In regards to specificity, risk factors are rarely specific in clinical psychology and psychiatry. This also applies in the field of medicine, for example, smoking has been found to cause various forms of cancer, cardiovascular diseases, and respiratory disease (Office of the Surgeon General (US) & Office on Smoking and Health (US), 2004), which makes smoking a non-specific but causal risk factor. Therefore, the notion that the relationship between SD and schizophrenia is causal is more parsimonious and plausible than the alternative explanation of genetic confounding.

However, the notion that various factors of social adversity reflect one common denominator as conceptualized by the SD hypothesis remains a theoretical assumption, yet to

be empirically tested. If this assumption is affirmed, the question is whether this type of experience is a predictor of psychosis and other mental disorders.

1.3. Cognitive models of psychosis: Explaining the relationship between social defeat and psychosis

Pioneering psychological models explaining the formation of various symptoms of psychosis have been developed decades ago (e.g. Mintz & Alpert, 1972). Since then, research findings on psychological factors of psychosis have accumulated and led to advancements and better understanding of psychological models. Cognitive models of psychosis have proved useful and became a basis of a psychological therapy for psychosis, called cognitive behavioral therapy for psychosis (CBTp), that has become widely recommended in the UK (National Institute for Health and Clinical Excellence, 2009).

Cognitive models can focus on different symptoms, so that we differentiate between cognitive model of positive symptoms (Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001), persecutory delusions (Freeman & Garety, 2014; Freeman, Garety, Kuipers, Fowler, & Bebbington, 2002; Kesting & Lincoln, 2013), hallucinations (Beck & Rector, 2003), and negative symptoms (Rector, Beck, & Stolar, 2005a). All of these models were postulated by synthesizing evidence on cognitive, social, and emotional processes from epidemiological, cross-sectional, experimental, and treatment studies. Although the models differ in emphasis, they cohere in emphasizing the importance of cognitive vulnerability factors and an affective pathway to psychosis. In addition, all models focus on providing psychological mechanisms that bridge the connection from vulnerability or risk factors into symptoms of psychosis. Consequently, drawing from cognitive models of psychosis various constructs of cognitive vulnerabilities that may bridge the connection from SD into symptoms of psychosis can be postulated.

1.3.1. Social rank

One appealing construct that may indicate a cognitive vulnerability and mediate the relationship between SD and psychosis is social rank. It is a construct that may represent the perception of SD in humans, as argued by the original authors of the SD hypothesis (Selten et al., 2013). Social rank is formed by comparing oneself to others with similar characteristics (e.g. of age, gender), particularly through the comparison of relative strength, social attractiveness, and relative fit to the group (Allan & Gilbert, 1995). People with relatively low social rank would be characterized by being physically weaker than people around them, not attractive, and left out by the group. These characteristics resemble the situation of male rodents under SD manipulation, which are arbitrarily placed with a dominant male rodent and a female rodent in a cage, thus forcing it to undergo the experience of being physically weaker, less attractive, and left out. On top of that, previous studies have found that social rank is significantly associated with psychotic symptoms (Freeman et al., 2005; Wickham, Shryane, Lyons, Dickins, & Bentall, 2014) and recently, a pioneering virtual reality experimental study found that manipulation of social rank leads to increases of paranoid beliefs (Freeman et al., 2014).

1.3.2. Negative schema

Global negative beliefs about the self and others is another construct that may also mediate the association between social adversity and psychotic symptoms. Numerous studies showed that negative schemas about the self and others are associated with psychosis (e.g. Kesting & Lincoln, 2013; Lincoln, Mehl, Kesting, & Rief, 2011; Taylor et al., 2014). Furthermore, self-esteem has been shown to mediate the effect of social exclusion, an indicator of social adversity, on psychotic symptoms (Kesting, Bredenpohl, Klenke, Westermann, & Lincoln,

2013) and negative beliefs about the self and others to mediate the relationship between childhood adversity and psychotic symptoms (Murphy, Murphy, & Shevlin, 2015).

1.3.3. Loneliness

Another potential mediator that could be linked to some of social adversity factors associated with psychosis (e.g. small social networks, discrimination) is loneliness. Loneliness is defined as the subjective appraisal of social isolation along with feelings of sadness (Hawkley & Cacioppo, 2010). Indeed, it has been shown that being a bully victim in adolescence predicts loneliness in young adulthood (Segrin, Nevarez, Arroyo, & Harwood, 2012). Moreover, loneliness is related to psychotic symptoms in first-episode psychotic patients (Sündermann, Onwumere, Kane, Morgan, & Kuipers, 2014) and in a community sample (Jaya, Hillmann, Reininger, Gollwitzer, & Lincoln, 2016).

Hence, there is an evidence-based rationale to expect social rank, negative beliefs about self and others, and loneliness to arise from an accumulation of adverse social experiences and to mediate the well-known association between social adversities and psychotic symptoms. Critically, however, the abovementioned constructs, namely social adversity (Slavich & Irwin, 2014), social rank (Gilbert & Allan, 1998), negative schemas (Vorontsova, Garety, & Freeman, 2013), and loneliness (Cacioppo, Hughes, Waite, Hawkley, & Thisted, 2006), have also been shown to be predictive of depression. Relatedly, depression is strongly associated with both positive (Hartley, Barrowclough, & Haddock, 2013) and negative (Sax et al., 1996) symptoms of psychosis. Thus, depression has to be taken into account to ascertain the specificity of translator mechanisms to psychosis.

1.4. The affective pathway to psychosis: Negative schemas and negative affect

Among the identified three potential mediators of the relationship between SD and psychosis, negative schemas seem to be one construct that have received most attention in the literature. The importance of negative schemas is best illustrated by a cognitive model by Kesting and Lincoln (2013). They propose that critical interpersonal experiences, such as childhood trauma, lead to the development of negative-self schema. This schema will then be reactivated upon an exposure to a social stressor, followed by an increase in negative affect, and ultimately, lead to the increase in threat beliefs, a precursor of persecutory delusions. These threat beliefs will then trigger a range of behavioral responses such as social withdrawal that further contribute to negative, or the lack of positive, interpersonal experiences creating a vicious circle. Similarly, the model by Garety et al (2001) postulates that general positive symptoms can be traced back to dysfunctional schemas that are followed by negative affect. Furthermore, the model by Beck and Rector (2003) stresses schemas as a crucial causal factor of hallucination. Overall, it can be postulated that negative schemas contribute to the development and maintenance of symptoms of psychosis via negative affect.

The importance of negative schemas in psychosis and the affective pathway to psychosis have been supported by empirical evidence. For example, the postulate that negative-self schema plays an important role in the emergence of symptoms has been supported by the observation that a decrease in self-esteem predicts the emergence of paranoid symptoms in daily life (Thewissen et al., 2011). Additionally, the postulate that negative affect precede psychotic symptoms have also been supported by various empirical evidence, such as an experience sampling study showing that negative affect precede the emergence of paranoia (Kramer et al., 2013) and longitudinal studies showing that anxiety (Oliver, O'Connor, Jose, McLachlan, & Peters, 2012) and depression (Fowler et al., 2012) prospectively predict paranoia. Although evidence has now accumulated in supporting the

assumption that negative-self schema and negative affect play a causal role in psychosis, the complex mechanisms postulated by cognitive models (e.g. Garety et al., 2001) that negative affect longitudinally mediates the relationship between negative schemas and symptoms remains to be tested.

2 Aims of the Dissertation

Although the association between social adversity and psychosis has attracted much attention, the psychological mechanism explaining the association has not been much investigated. Specifically, the psychological processes behind the relationship between social adversity and psychosis are not well understood and have not been much empirically explored. On top of that, negative schemas and its affective pathway to psychosis, a central postulate of cognitive models of psychosis, have not been investigated longitudinally.

Furthermore, most of the abovementioned accumulated evidence on the topic of social adversity, cognitive vulnerability, affective pathway and psychosis is based on a narrow subpopulation, such as research participants from white, educated, industrialized, rich, democratic countries (WEIRD), which may not be generalizable to other subpopulations (Henrich, Heine, & Norenzayan, 2010). Investigating more than one subpopulation is particularly relevant in psychological models where cultures may play a decisive role. The present study thus aimed at clarifying the issues raised above by testing our hypotheses on large community samples from Germany, Indonesia, and the United States.

3 Study 1: Social adversities and psychotic symptoms: A test of predictions derived from the social defeat hypothesis

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3.1. Background

The present study tested several predictions derived from the SD hypothesis using path analysis in a large and heterogeneous population sample from three countries located on different continents. Specifically, reflecting the claims of the SD hypothesis that apparently different negative social experiences may load on a common factor, we hypothesized that negative social experiences that have been shown in previous studies to be associated with psychosis would load on a common factor. As some of these experiences date back to childhood and adolescence whereas others reflect ongoing present adversity, our specific hypothesis was that a) childhood abuse and bullying victim experience in school would indicate a latent past SD factor, whereas present bullying victim experiences, ostracism, social undermining, a low level of social support, small networks, low socioeconomic status, minority status, and perceived discrimination would indicate a latent present SD factor. We also hypothesized that b) there would be specific association of the past and current SD latent factors and psychotic symptomatology (combined positive and negative symptoms) that remain after controlling for their association with depression and anxiety; c) the path that assumes SD to precede psychosis (as predicted by the SD hypothesis) would be stronger than the path that assumes the reverse (as predicted by the competing hypothesis), and d) the pattern of results would be stable across countries.

3.2. Methods

This study used the baseline data of the longitudinal mental health and social situation survey project. Specifically, the study used the baseline sample that consisted of 2350 participants of whom 720 participants completed the English, 786 the German and 844 the Indonesian version of the survey. Furthermore, the study included analyzed data from the following measures, demographic and mental health problem questions, socio-economic status, bullying victim experiences, child abuse, discrimination, social undermining, social network and support, ostracism experiences, anxiety, depression, and psychotic symptoms.

The analyses were conducted with structural equation modeling (SEM). First, confirmatory factor analyses (CFA) were calculated to establish factorial validity of latent variables (past and current SD, psychotic symptoms, depression, and anxiety). Second, path analyses were conducted. Both analyses were conducted using lavaan ver. 0.5-17 (Rosseel, 2012) in R version 3.2.1 and were estimated using maximum likelihood procedure with robust standard errors and Satorra-Bentler scaled statistic to correct for non-normal distribution. We also repeated the path analyses with country as a group variable to investigate whether the results are comparable across the different countries.

3.3. Results

In the CFA two factors reflecting current and past experiences of SD could be identified with acceptable fit. Path analysis indicated acceptable fit for both SD and reverse models, and both the path from current SD to psychotic symptoms and the reverse one were significant, although the former was stronger than the latter. Interestingly, the current but not the past SD factor was significantly associated with psychotic symptoms.

3.4. Discussion

The study tested some postulates of the SD hypothesis. Specifically, we tested whether various factors of social adversity load on a common factor that is associated with symptoms of mental disorders, including psychotic symptoms. The results of the CFA showed that the conceptualized bi-dimensional SD model had a relatively good fit to the data. In line with the SD hypothesis that postulates SD to be a non-specific risk factor of mental disorders (Selten et al., 2013), we found that current SD factor made an independent significant contribution to symptoms of psychosis, depression, and anxiety, albeit the effect was the largest for symptoms of psychosis. Similar patterns of results were found when the model was examined for each country separately. Furthermore, although we found some support for the social drift and social selection hypotheses, the path from current SD to symptoms of psychosis was almost twice as strong as the reverse path, indicating that SD played a stronger role in influencing rather than in resulting from symptoms of psychosis. Taken together, the findings thus support the notion that SD is a significant risk factor for symptoms of psychosis, as well as for symptoms of depression and anxiety. Thus, SD appears to be a risk factor that can be generalized to different syndromes as well as to different regions and cultures.

4 Study 2: Social Adversity and psychosis: The mediating role of cognitive vulnerability

Jaya, E. S., Ascone, L., & Lincoln, T. M. (2016). Social adversity and psychosis: The mediating role of cognitive vulnerability. *Schizophrenia Bulletin*, sbw104. https://doi.org/10.1093/schbul/sbw104

4.1. Background

In Study 1, we found supporting evidence that the apparently different social adversity factors reflect one common experience of SD and that this factor is related to psychotic symptoms. However, we do not know the mechanism. Here, we tested whether social rank, negative beliefs about self and others, and loneliness mediate the known association of social adversity on both positive and negative symptoms of psychosis. Similar to Study 1, we also include depression as an outcome variable to ascertain the specificity of the postulated mediators on psychosis. In other words, we hypothesize that the mediations are not fully explicable by co-occurring depression.

4.2. Methods

This study used the baseline data of the longitudinal mental health and social situation survey described in the appendix. Similar to Study 1, the study used the baseline sample that consisted of 2350 participants of whom 720 participants completed the English, 786 the German and 844 the Indonesian version of the survey. This study included data from the following measures, demographic and mental health problem questions, socio-economic status, bullying victim experiences, child abuse, discrimination, social undermining, social network and support, ostracism experiences, social rank, negative schemas, loneliness, depression, and psychotic symptoms.

The analyses were conducted with structural equation modeling (SEM). Following a recommended procedure(Hoyle & Smith, 1994), we first tested for a direct relationship between the SA latent variable and the outcome latent variables, and then entered the postulated mediators to test for reduction in the estimates of the direct relationships. By including positive symptoms, negative symptoms, and depression as outcome variables simultaneously in one analysis, which is not possible in traditional regression analysis, it is

possible to estimate the specific effect of the social adversity latent (IV) variable and the mediators (M) on each single outcome while controlling for the effects of IV and M on the other outcomes. For example, if the mediation effect of negative schemas on psychotic symptoms was due to the covariance between psychotic symptoms and depression this would show as zero effect of the paths from social adversity to negative schemas on positive and negative symptoms, but a significant path to depression.

4.3. Results

The pre-conditions of mediation were fulfilled which is indicated by significant bi-directional relationships between the social adversity latent variable, the mediator latent variables, and the outcome latent variables. In the direct effect model, the paths from social adversity to all outcomes were significant (positive symptoms, γ = 0.45, p < .001, R^2 = .20; negative symptoms, γ = 0.61, p < .001, R^2 = .38; depression, γ = 0.67, p < .001, R^2 = .45) with small to medium sized effects. Entering social rank as putative mediator increased the direct effect path from social adversity to positive symptoms while the indirect effect coefficient was negative indicating suppression. Social rank was a significant mediator for negative symptoms and depression, indicated by a reduction of the direct effect path coefficients from social adversity to negative symptoms and depression and by significant indirect effect coefficients (confidence interval not including zero). The social rank mediation model explained a large proportion of variance of negative symptoms (R^2 = .43) and depression (R^2 = .52). Negative schemas reduced the direct effect path coefficients of all outcomes and had significant indirect effects indicating mediation for positive symptoms, negative symptoms, and depression. Notably, the negative schemas mediation model explained a large proportion of variance of all outcomes (positive symptoms, R^2 = .27; negative symptoms, R^2 = .44; depression, R^2 = .49). The path from loneliness to positive symptoms was not significant and the indirect effect coefficient was insignificant, which indicated no mediation. The paths from

loneliness to negative symptoms and depression were significant and the indirect effect coefficients were significant, which indicated mediation. The loneliness mediation model explained a large proportion of variance of negative symptoms (R^2 = .44) and depression (R^2 = .50).

4.4. Discussion

The results emphasize the role of social adversity in psychosis and support the assumption that cognitive vulnerability is a relevant translating mechanism as postulated by the SD hypothesis and cognitive models of psychosis. This underlines the relevance of the clinical practice of targeting beliefs in cognitive interventions for psychosis. It also indicates that targeting cognitive vulnerability in people experiencing social adversity could be a promising approach to prevention.

5 Study 3: Negative schemas and psychotic symptoms: The affective pathway

Jaya, E. S., Ascone, L., & Lincoln, T. M. (submitted). A longitudinal mediation analysis of the effect of dysfunctional self-related schema on psychotic symptoms via negative affect.

5.1. Background

In Study 1 and 2, we found supporting evidence that the seemingly different social adversity factors reflect one common experience of SD and that SD factor is related to psychotic symptoms. The mechanism of this relationship is investigated in Study 2, which shows that negative schemas are a potentially strong mediator. The importance of negative schemas in psychosis has been mentioned in various cognitive models of psychosis (e.g. Garety *et al.*

2001; Beck & Rector 2003; Kesting & Lincoln 2013; Bentall *et al.* 2014; Freeman & Garety 2014). These cognitive models have been immensely important to our understanding of psychosis and are widely used to inform cognitive behavioral interventions of which has been recommended to be routinely offered to patients of psychosis (National Institute for Health and Clinical Excellence, 2009). In one of the first model that focused on positive symptoms of psychosis, symptoms are postulated to be both exacerbated and maintained by negative schemas through negative affect (Garety et al., 2001). However, there is still a dearth of evidence for the postulated longitudinal association between negative schema and positive symptom mediated by affect. Here, we tested the hypotheses that negative schemas longitudinally predict positive symptoms and that this longitudinal association is mediated via negative affect (anxiety and depression).

5.2. Methods

This study used the longitudinal data of the longitudinal mental health and social situation survey described in the appendix. Briefly, The baseline (T0) sample consisted of 2350 participants of whom 720 participants completed the English, 786 the German and 844 the Indonesian version of the survey, 1447 of them responded at 4 months (T1, response rate = 61.6%), 1244 at 8 months (T2, response rate = 52.9%), and 943 at 12 months (T3, response rate = 40.1%) follow up assessments. The participants who responded to the invitation were then subjected to the same inclusion criteria (i.e. complete entry, no longstring, consistent answers). Based on these criteria, there were 682 participants at T1, 409 at T2, and 366 at T3, of which 135 participants completed all follow-up assessments. In this present study, we included participants who participated in at least one follow-up survey (n = 962) of whom 302 participants completed the English, 312 the German and 348 the Indonesian version of the survey. This study included data from the following measures, demographic and mental health problem questions, negative schema, anxiety, depression, and psychotic symptoms.

The analyses were conducted with structural equation modeling (SEM) using cross-lagged panel models. Cross-lagged panel models are ideal for examining temporal association between two variables because various sources of error are taken into account. These include the stability of the variables, cross-sectional associations and prior associations. The longitudinal mediation analysis followed the suggestions of Preacher (2015) in constructing the appropriate cross-lagged panel models. Full information maximum likelihood (FIML) with missing at random (MAR) assumption was used in all analyses.

5.3. Results

First, we found that negative schemas significantly predicted following measures of positive symptom. There were no significant reverse pathways from positive symptoms to negative schemas. Second, we found that negative schema significantly predicted following measures of negative affect. One out of four possible reverse paths from negative affect to negative schema was also significant indicating possible reverse causation. Third, we found that negative affect significantly predicted following measures of positive symptoms. There were no significant reverse pathways from positive symptoms to negative affect.

There were three indirect pathways from negative schemas at T0 to positive symptoms at T3 in the longitudinal mediation model. First, the indirect effect consisting of pathways from negative schema at T0 to negative affect at T1, then from negative affect at T1 to negative affect at T2, and finally from negative affect at T2 to positive symptoms at T3 was significant (0.020, $p < 0.05$, 95% BCa CI, 0.004, 0.035). Second, the indirect effect consisting of pathways from negative schema at T0) to negative schema at T1, then from negative schema at T1 to negative affect at T2, and finally from negative affect at T2 to positive symptoms at T3 was trend-level significant (0.021, $p = 0.06$, 95% BCa CI, -0.001, 0.042). Third, the indirect effect consisting of pathways from negative schema at T0) to negative

affect at T1, then from negative affect at T1 to positive symptoms at T2, and finally from psychotic symptoms at T2 to positive symptoms at T3 was not significant (-0.001, p = 0.145, 95% BCa CI, -0.022, 0.003).

5.4. Discussion

Informed by cognitive models of psychosis (particularly Garety *et al.* 2001) this study set out to investigate the postulated longitudinal mediation hypothesis of negative affect mediating the association between negative schemas and positive symptom of psychosis in a large multi-national sample over the course of one year. We found a vicious cycle of longitudinal association between negative schema and negative affect, and we found uni-directional association from negative schema and negative affect to psychotic symptom. Negative affect was found to be a significant mediator in the association between negative schema and positive symptom, but the mechanism was only observed in the time frame of twelve months and not sooner. Overall, these results suggest that interventions targeting negative schemas and negative affect may reduce and prevent positive symptoms.

6 Discussion

6.1. The social defeat hypothesis

The first study tested predictions made by the SD hypothesis. Specifically, the postulate that various measures of social adversity load on a common factor that is associated with symptoms of mental disorders, including psychotic symptoms, was tested. The results confirmed this postulate and extended the SD theoretical framework further by showing that SD experiences in the past (or, specifically in childhood) are different from SD experiences in the present, particularly in the context of its influence on mental health.

It was unexpected that past SD did not significantly predict symptoms of psychosis in the model. As past SD consists of bullying victim experience at school and child abuse, at first glance this seems to contradict previous research showing a strong link between psychosis and childhood adverse events (van Dam et al., 2015; Wolke, Lereya, Fisher, Lewis, & Zammit, 2014). Additional analysis, however, revealed that past SD had an effect on symptoms of psychosis but was completely mediated by current SD. Thus, people who experienced childhood trauma or were bully victims at school are very likely to also experience current SD which is linked to symptoms. This interpretation is in line with findings from studies on bullying that showed a continuous line of adversity from childhood into adulthood (Arseneault, Bowes, & Shakoor, 2010).

In line with the SD hypothesis that postulates SD to be a non-specific risk factor of mental disorders (Selten et al., 2013), we found that current SD made an independent significant contribution in predicting symptoms of psychosis, depression, and anxiety, albeit the effect was the largest for symptoms of psychosis. Furthermore, although we also found some support for the social drift and social selection hypotheses, the path from current SD to symptoms of psychosis was almost twice as strong as the reverse path. This result suggests that SD played a stronger role in influencing rather than in resulting from symptoms of psychosis. Taken together, the findings thus support the notion that SD is a significant risk factor for symptoms of psychosis, as well as for symptoms of depression and anxiety.

6.2. Psychological mechanisms of the social defeat hypothesis

The second study took the findings from the first study further by investigating the mechanisms that may explain the association between various factors of social adversity and psychosis. Based on previous studies and theoretical frameworks such as cognitive models of psychosis (e.g. Kesting & Lincoln, 2013), we investigated the putative translating role of

cognitive vulnerability factors defined as perceived low social rank, negative schemas and loneliness.

As hypothesized, negative schemas were strongly linked to both social adversity and symptoms and turned out to be the most relevant mediators in regard to all symptomatic outcomes. Unspectacularly, this confirms cognitive conceptualizations of depression (Beck, Rush, Shaw, & Emery, 1979). More interestingly, it also confirms cognitive models of psychosis, in which negative schemas and beliefs are postulated to play a central role in the development of both positive (Bentall, Corcoran, Howard, Blackwood, & Kinderman, 2001; Garety et al., 2001; Kesting & Lincoln, 2013; Morrison, 2001) and negative (Rector, Beck, & Stolar, 2005b) symptoms.

Social rank and loneliness, in contrast, only mediated the pathway from social adversity to negative symptoms and depression. The absence of a commonly found significant relationship between loneliness and positive symptoms is likely to be due to our stringent analysis controlling for depression and negative symptoms, which has not been done in previous studies (e.g. Sündermann et al., 2014). Interpreted in this way, it confirms a previous study by our group (Jaya et al., 2016), in which we found that depression explains the relationship between loneliness and positive symptoms.

Interestingly, we found that taking social rank into account enhanced the strength of the relationship between social adversity and positive symptoms. Nevertheless, and in line with previous work (e.g. Freeman et al., 2014), we found a significant relationship between social rank and positive symptoms when the two constructs were computed in isolation (i.e. not controlling for social adversity). Thus, it could be that previous studies (e.g. Freeman et al., 2014) have found a significant relationship between social rank and positive symptoms because they did not take social adversity into account. This interpretation is supported by the

results of a social rank manipulation experiment (Ascone, Jaya, & Lincoln, 2016), in which only the cognitive aspect of social rank was manipulated and its effect on positive symptoms was not found. In regard to negative symptoms, however, social rank showed the postulated mediating effect. Thus, the idea that the appraisal of being socially inferior that is induced by social adversity fosters deficits in exploration and motivation, one manifestation of negative symptoms, was supported. Moreover, our data show that these mechanisms are not solely explained by depression, a well-known covariate of positive and negative symptoms of psychosis.

6.3. Negative schemas and psychosis: A closer look

In the second study we found that negative schemas are the most important translating mechanism in the relationship between social adversity and psychosis. According to cognitive models of psychosis (particularly Garety et al., 2001) the mechanism by which negative schemas translates into psychosis is an affective pathway. The third study set out to investigate the postulated hypothesis of negative affect longitudinally mediating the association between negative schemas and positive symptoms of psychosis in a large multi-national sample over the course of one year.

Uni-directional pathways from negative schemas and negative affect to positive symptoms were found. This is in line with findings of previous studies that found a uni-directional pathway from negative schema to paranoia and from negative affect to paranoia in a clinical (Fowler et al., 2012) and a healthy sample (Oliver et al., 2012). Together with our findings on general positive symptoms, we can now affirm the importance of negative schema and negative affect in the formation and maintenance of various psychotic symptoms that are often postulated in cognitive models of psychosis with greater confidence (e.g. Beck &

Rector, 2003; Bentall et al., 2014; Freeman & Garety, 2014; Garety et al., 2001; Kesting & Lincoln, 2013).

On top of that, negative affect was found to longitudinally mediate the path from negative schema to positive symptoms. Notably, the original total effect path from negative schema at baseline to positive symptoms at twelve months was no longer significant, which suggests a strong mediating role of negative affect. This underpins previous findings based on a half-longitudinal design that showed anxiety mediated the relationship between negative schema and paranoia (Oliver et al., 2012). It is a significant finding because it is the first systematic demonstration of the longitudinal mediation role of negative affect in the relationship between negative schema and psychosis, a central postulate in cognitive models of psychosis (e.g. Garety et al., 2001; Kesting & Lincoln, 2013).

The findings from the third study may extend current theoretical framework of cognitive models of psychosis by suggesting specific patterns of associations. The results from independent cross-lagged panel models suggest that negative schemas and negative affect may influence each other over time forming a vicious cycle, as indicated by bidirectional pathways between negative schemas and affect. At the same time, their relationship with positive symptoms is mainly uni-directional, suggesting that positive symptoms trigger negative schema and affect to a lesser extent. This may run counter to some previous formulations. Previous cognitive models often postulated a vicious cycle relationship between negative schemas, negative affect and positive symptoms (e.g. Garety et al., 2007; Kesting & Lincoln, 2013), but our study and others (Fowler et al., 2012; Oliver et al., 2012) could not find evidence to support the notion of vicious cycle in which positive symptoms influence negative schema. Although these findings still have to be interpreted with caution, this could indicate that negative schema may be a true vulnerability factor of psychosis and not just an epiphenomenon of the disorder.

Another notable element of the findings is the aspect of time. Psychological theories in general have been criticized to lack consideration of time (Cole & Maxwell, 2003), and this shortcoming has only been recently addressed in the context of psychosis. Putative psychological mechanisms of psychosis may take place at a distal macrolevel and/or at a more proximal microlevel (Reininghaus, Depp, & Myin-Germeys, 2015). The findings suggest that the mechanisms in which negative schemas impacted upon psychosis most likely would need at least around eight months to achieve a meaningful impact, a distal macrolevel mechanism. Speculatively negative affect, as triggered by negative schemas, needs time to build up through a vicious cycle process, until it reaches a certain threshold to impact upon positive symptoms. Such an interpretation is in line with the clinical observation that patients with schizophrenia often present themselves to the clinic with problems related to depressive mood or anxiety first, before they later develop schizophrenia (Häfner, Maurer, & an der Heiden, 2013). While our studies suggest the mechanism to take place at a distal macrolevel, previous studies suggest that it can also take place at a more proximal microlevel. Brief changes of self-esteem and negative emotions have been shown to trigger state symptom measures immediate increase in paranoid beliefs at a micro-level in experimental and experience sampling studies (Kesting et al., 2013; Kramer et al., 2013; Lincoln, Lange, Burau, Exner, & Moritz, 2010; Thewissen et al., 2011). Therefore, the psychological mechanism of negative schemas and negative affect may take place at both macro- and microlevel, and it would be important to pursue this research question further in future studies.

6.4. Stability of findings across samples from Germany, Indonesia, and the United States

Notably, we found that the general pattern of the abovementioned findings were stable across countries indicating global mechanisms. Specifically, the finding that various social adversity measures indicate a common SD factor (i.e. negative experience of being excluded)

and that this SD factor has an adverse impact on mental health including psychotic symptoms seems to apply irrespective of geographical regions and culture. Furthermore, the mechanisms by which social adversities impact upon psychosis, i.e. through negative schema, seem to also apply irrespective of geographical regions and culture. The mediating role of negative affect in the relationship between negative schema and psychosis unfortunately cannot be ascertained to apply irrespective of geographical regions and culture, due to statistical power issues. However, the general pattern of results may also suggest the hypothesized mechanism to be a global mechanism. Together, the findings imply that the SD hypothesis and cognitive models of psychosis are theoretical frameworks that describe a universal human experience that trumps geographical and cultural boundaries.

7 Strengths and Limitations

A major strength of the three studies is that the participants were recruited from the general population from Germany, Indonesia and the United States and covered a significant proportion of the psychosis continuum. Looking at associations across the continuum is advantageous when it comes to investigating putative causal factors, because the findings can be interpreted free of issues that often influence studies that compare clinical to non-clinical populations, such as selective small samples, treatment history, stigma, and medication.

Several limitations should be considered when interpreting the findings from all of the studies. First, the sample was drawn from a population with access to the internet. Similar to other studies that used crowdsourcing websites for recruitment (e.g. Shapiro et al., 2013), participants tended to be highly educated and to have middle to low income in comparison to the national average. For example, 14.7% of the participants from Germany had a university degree, while only 8.3% of the German population have a university degree according to 2013 census (Federal Statistical Office [Destatis], 2014). This slightly reduced heterogeneity may

have led to an attenuation of the relationships´ strengths, particularly any relationships that involve social adversity (see Barrett, 2001). However, an overestimation of the associations cannot be ruled out. Second, the self-reported diagnosis of the participants cannot be externally verified. Nevertheless, false diagnostic status seems unlikely because the reward for completing the long survey was minimal and we used several data verification processes to ensure data validity. Furthermore, although we oversampled participants with schizophrenia spectrum disorders to have sufficient variation in symptoms and increase statistical power for subgroup analyses, the rate of overall psychopathology in our sample was comparable to typical samples reported in epidemiological studies. For example, the average rate of 29% (Germany, 27.2%; Indonesia, 23.2%; US, 33.0%) of participants with a life-time mental diagnosis in the present study is comparable to the reported rate based on nationally representative sample (e.g. 31% from Germany (Jacobi et al., 2004), 30.5% from the United States (Kessler et al., 2005)).

In addition, there are several aspects of strength and limitation that particularly apply to the third study. A notable strength was the careful consideration of the temporal aspect of measurement and assessment points. The specific and uniform reference to the past four weeks in all measurements and assessment points along with the evenly spaced assessment points allow the investigation of temporal pathways between negative schemas, negative affect and positive symptoms repeatedly under uniform conditions. The limitation is related to our choice of analysis in treating the temporal aspect of the data. Cross-lagged panel analysis has been noted to overestimate the stability coefficients of constructs and to underestimate the cross-lagged paths (Cole & Maxwell, 2003). This means that the coefficients of the main results presented are attenuated and conservative estimates, and other methods of analysis may have shown significant temporal paths. On top of that, our analysis of missing data pattern showed a slight tendency of participants with severe symptoms to drop out at follow-

up. This reduces the variation of the dependent variable that contribute to floor effect (Terluin, Boer, & Vet, 2016), and thus attenuating effect sizes. Even though we attempted to correct these shortcomings by estimating the missing data using FIML procedure, these shortcomings nevertheless can lead to an underestimation of all lagged pathways due to a reduction of follow-up data variability.

8 Implication and Outlook

Combined together the results may extend the theoretical framework of SD hypothesis and cognitive models of psychosis. Notably, psychological mechanisms derived from cognitive models of psychosis were empirically found for the SD hypothesis. Specifically, the role of negative schemas was found to be important. In this chapter I discuss theoretical implications of the results and outlook for future studies.

A summary of the results of the three studies is illustrated in Figure 1. In the left side of the figure an ellipse titled SD is shown to be a common denominator of various seemingly different social adversities. SD as a common latent factor of those social adversities is then shown to be associated with negative schema and psychotic symptoms. Specifically, the association between SD and psychotic symptoms is partially mediated by negative schemas. Negative schemas have an indirect influence via negative affect towards psychotic symptoms. Negative affect plays both the role of a mediator that translates the effect of negative schema to psychotic symptoms and a maintenance factor of negative schema, which forms a vicious cycle of negative schema and negative affect reinforcing each other over time.

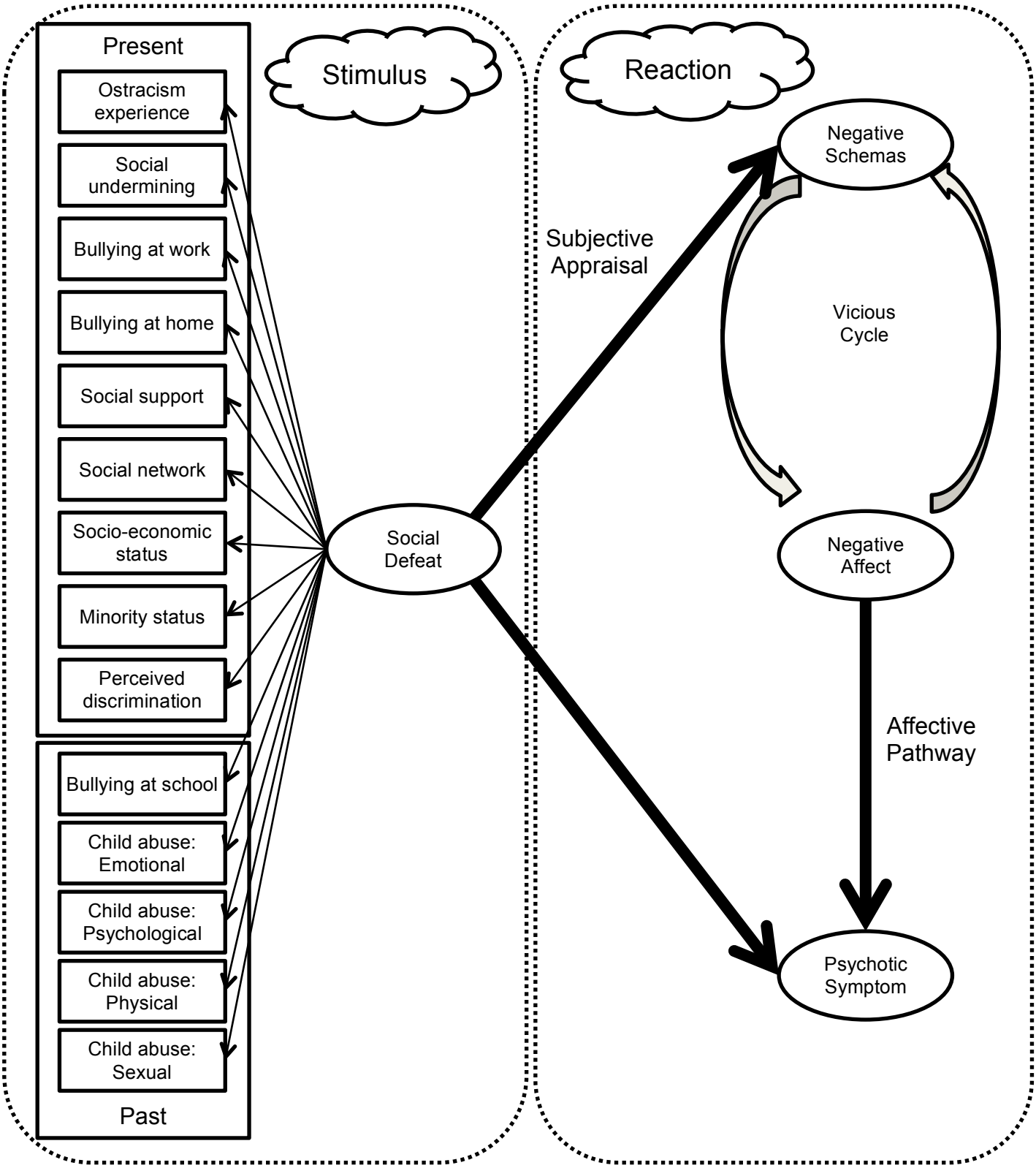

Figure 1. Schematic representation of the results of the dissertation: Psychological model of Social Defeat hypothesis

The results of this dissertation can be formulated as a psychological model of SD hypothesis (see Figure 1). The model can be understood using terminologies of well-known cognitive models of psychosis and cognitive behavioral therapy. For example, SD can be seen as a concrete operationalization of "triggers" (Freeman & Garety, 2014), "stressful events" (Garety et al., 2001) or "adverse life events/interpersonal experience" (Kesting & Lincoln, 2013). This left side of the figure can also be understood as stimulus or antecedents (A) in the terminologies of cognitive behavioral therapy. The constructs on the right hand side of Figure 1 can be understood as a reaction to the stimulus. This reaction may be in the form of a belief or thoughts (B), i.e. represented by negative schemas, emotions or consequences (C), i.e. represented by negative affect, and symptoms of a disorder, i.e. represented by psychotic symptoms.

The direct clinical implications of the results are that they support current practice of CBTp and may provide a stepping stone to further extend some of the techniques in CBTp. First of all, there are similarities of the psychological model of SD hypothesis with the ABC model of CBTp manuals (e.g. Lincoln, 2014). Understood in this way, the present results inform therapists that the phenomenon of social factors that resulted in the feeling of defeat triggering negative thoughts related to the self may be a particularly common theme in the therapeutic context of psychosis. Furthermore, therapists may be more rewarded with success in terms of symptoms reduction of patients if they focus on negative thoughts related to the self, rather than negative thoughts indicative of loneliness or social comparison. Another interesting similarity with techniques in CBTp is the vicious cycle of negative thoughts and negative affect (Lincoln, 2014). As described in a CBTp therapeutic manual (Lincoln, 2014), psychotic symptoms are maintained or increased by increasing negative affect, and that the relationship between negative affect and negative schema is bi-directional. However, the findings reported in this dissertation and other studies (e.g. Fowler et al., 2012; Oliver et al.,

2012) support a uni-directional relationship from negative affect to psychotic symptoms, instead of a bi-directional relationship that is conceptualized in cognitive models of psychosis and described in the therapeutic manual. In practical terms this means that a reduction of psychotic symptoms, for example via the administration of antipsychotics, will not reduce negative affect and negative schema. Mental health professionals who observed a significant symptom reduction that is possibly attributable to antipsychotics have no reason to feel successful because the symptom maintaining cognitive and affective factors are still nevertheless present, and have to be separately addressed. To state it differently, the lack of bi-directional relationship means that reduction of psychotic symptoms through antipsychotics will not automatically improve negative affect and negative schemas.

The proposed psychological model of SD hypothesis is far from complete. Many aspects of the model have to be explored in future studies. First of all, cross-sectional paths in the model should be confirmed longitudinally. Specifically, future studies should confirm longitudinally of the postulated paths from SD construct to negative schema and psychotic symptoms. Secondly, closer looks at negative schema as a translating mechanism of the relationship of SD and psychotic symptoms are needed. Negative schema in the present study was conceptualized very broadly using the Brief Core Schema Scale (Fowler et al., 2006). A more detailed understanding of the role of schema in psychosis, for example through Young Schema Questionnaire (Young, 1998) as suggested by Sundag et al. (2016), may open a new line of treatment research. This is particularly relevant in the context of a recent treatment trial that particularly target negative schema, also measured with the BCSS, but did not manage to induce large improvement of negative schemas and the targeted symptom, persecutory delusion (Freeman, Pugh, et al., 2014). In this context, further understanding of negative schemas and its role in psychotic symptoms is warranted. The results of the third study of the present dissertation suggest that the effect of negative schemas on psychotic symptoms occurs

at the macrolevel, probably in the timeframe of at least around eight months. If this is true, then this may explain the disappointing findings of the abovementioned treatment trial that only has up to 12 weeks follow-up data. Thirdly, a confirmation of the proposed pathways using interventionist-causal model (Kendler & Campbell, 2009) is warranted. While this idea has in part been implemented by a treatment trial that target negative schema (Freeman, Pugh, et al., 2014), they did not implement the trial on a vulnerable population. Specifically, future studies may take the findings of this dissertation further by testing a treatment trial targeting negative schema on populations that are vulnerable to feel socially defeated, for example migrants (Cantor-Graae & Selten, 2005) and people with minority sexual orientations (Gevonden et al., 2014), and test whether the improvement of negative schemas in this population resulted in general psychotic symptoms reduction.

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Appendix A. Longitudinal mental health and social situation survey

1. Participants, design and procedure

Participants from Germany, Indonesia, and the United States were recruited through Crowdflower and other websites (e.g. internet forums and social networking websites) to complete an anonymous 30-minute online survey. Moreover, following the sampling method from the COMED study (Hanssen, Krabbendam, Vollema, Delespaul, & Van Os, 2006), we advertised our study in internet forums focused on mental disorders, and particularly schizophrenia, in order to secure sufficient variation reflecting the continuum of psychosis. Participants recruited from Crowdflower received 0.50 US$ following the median hourly wage in Amazon MTurk (Buhrmester, Kwang, & Gosling, 2011). Participants recruited from other websites were not given compensation for reasons of data security. Previous studies have shown that collecting self-report data on mental health symptoms over the internet is reliable (e.g. Moritz et al., 2013) and that recruiting participants through crowdsourcing websites produces demographically heterogeneous samples (e.g. Shapiro et al., 2013). Only participants who agreed with the consent statements and indicated to be above 18 years of age were allowed to participate.

There were 2501 completed survey entries, of which 151 were excluded due to duplicate entries (n = 98), longstring (i.e. providing the same answer consecutively for 50 items, n = 46, (Johnson, 2005), and inconsistent answers (n = 7). The final sample consisted of 2350 participants of whom 720 participants completed the English, 786 the German and 844 the Indonesian version of the survey. This is the cross-sectional data that is analysed in Study 1 and Study 2.

Participants who completed the baseline survey (T0) were invited via email to complete a follow-up survey after 4 (T1), 8 (T2), and 12 months (T3). Participants recruited

from Crowdflower received increasing compensation to motivate participation in the follow-ups (T1, 0.60 US$; T2, 0.80 US$; T3, 1.00 US$). Participants recruited from other websites could not be given compensation for reasons of data security.

From those who are invited to the follow-ups, 1447 participants responded at T1 (response rate = 61.6%), 1244 at T2 (response rate = 52.9%), and 943 at T3 (response rate = 40.1%). The participants who responded to the invitation were then subjected to the same inclusion criteria (i.e. complete entry, no longstring, consistent answers). Based on these criteria, there were 682 participants at T1, 409 at T2, and 366 at T3, of which 135 participants completed all follow-up assessments. A detailed participant flowchart following the STROBE (STrengthening the Reporting of OBservational studies in Epidemiology) guideline is available in Appendix B. For the analysis in Study 3, we included participants who participated in at least one follow-up survey (n = 962), which consists of 302 participants who completed the English, 312 the German and 348 the Indonesian version of the survey. The study received approval from the ethical committee of the German Psychological Society.

2. Measures

The measurements consist of standardized self-report questionnaires and single questions. The total set of measurements took around 20 to 30 minutes to complete. Back-translation procedure and cultural adaption of measures were conducted by native Germans, British and Indonesians according to guidelines (Schmitt & Eid, 2007).

2.1. Demographic and mental health problem questions

The Demographic questions included age, sex, living arrangement, current relationship status, and frequency of being in a relationship. The mental health problem questions asked whether the participant, and also the participant´s parents and sibling, ever had a diagnosable mental health problem. There were also questions about the type of diagnosis and the current mental

health state of the participants. Participants who reported to have been diagnosed with schizophrenia were asked more specific questions, such as time of diagnosis, types of medication taken, schizophrenia type, previous hospitalizations, and psychological treatment use.

2.2. Socio-economic status

Socio-economic status was measured with a multidimensional index, which was used in the *telefonischen Gesundheitssurveys* 2003-2006 by Robert Koch-Institut (Lampert & Kroll, 2009). This index has also been adapted in online survey format (Klug, 2013).The index (ranges 3 to 21) was construed by summing the score of education (range 1 to 7), household income (range 1 to 7), and job position (range 1 to 7). The respective answer choices for education and household income were created based on the census categories published by statistical offices of Germany, Indonesia, and United States. The index measures participants´ current socio-economic position relative to people from their country. For further information regarding the socio-economic status questions, please see the appendix.

2.3. Bullying victim experiences

Bullying victim experience frequency in childhood in a school context and adulthood in a home and work context were measured with a bullying victimization questionnaire (Wolke & Sapouna, 2008). The questionnaire measured direct and relational bullying victim experience frequency at school, home and work. Behavior examples of direct bullying victim experience were regular experience of being threatened, insulted, subject to ridicule, hit, and/or beaten up. Behavior examples of relational bullying victim experience were getting deliberately left out of get-together or parties, being ignored, and/or having nasty lies or rumors. Each bullying victim experience was measured with a frequency and a duration scale. Frequency was measured with 5-points Likert scale (0: never; 1: once or twice; 2: occasionally; 3: about once

a week; 4: several times a week). Duration was measured with 5-points Likert scale (1: a few days; 2: several weeks; 3: several months; 4: several years; 5: It´s still going on now). Participant who answered "never" in the frequency question was not given the duration question. A score made by averaging the frequency and duration scores ranging from 0 to 5 was created. This score was used in the analyses as an indicator of bullying victim experience at school, home and work.

2.4. Child abuse

Frequency of child abuse experience before the age of 16 was measured by a self-report questionnaire developed based on a semi-structured interview from the NEMESIS study (Janssen et al., 2004). Participants were asked if they ever experienced emotional, psychological, physical, or sexual abuse (yes or no) according to a given definition that was presented (e.g. emotional abuse: "This means for example that people at home didn't listen to you, that your problems were ignored, that you had the feeling of not being able to find any attention or support from the people in your house") and to rate the frequency of the experience on a 6-point Likert scale (0 = never to 5 = very often).

2.5. Discrimination

Experiences of discrimination were assessed with the perceived discrimination measure from the NEMESIS study (Janssen et al., 2003), which includes a section on minority status and a section on perceived discrimination. Minority status was measured with five dichotomous items, which included having a minority sexual orientation, a physical disability, a visible physical condition (e.g. being obese), belonging to an ethnic minority group, and to a minority religion. The perceived discrimination section consisted of seven dichotomous items (age, sex, sexual orientation, physical disability, ethnic minority group, religion, visible physical

condition). The sum score represents the degree of minority status (range 0 to 5) and perceived discrimination (range 0 to 7).

2.6. Social undermining

Social undermining was assessed with the Social Undermining Scale (Vinokur & van Ryn, 1993), a five items scale that measures the frequency of negative interaction with a spouse or significant other over the past four weeks (e.g. How much does the spouse or significant other act in an unpleasant or angry manner toward you?) on a 5-point Likert scale (1 = not at all to 5 = a great deal).

2.7. Social network and support

Social network and support were measured by the six items version of the Social Support Questionnaire (Sarason, Sarason, Shearin, & Pierce, 1987). Social network was measured by asking participants to list people whom they can rely on in relation to six different conditions (e.g. Who accepts you totally, including both your worst and your best points?). The maximum number of people that participants could list for each item was nine. Social support was measured by asking participants´ satisfaction concerning the support they received (How satisfied are you with the overall support?) on a 6-point Likert scale (1 = very dissatisfied to 6 = very satisfied).

2.8. Ostracism experiences

Ostracism experiences was assessed with the Ostracism Experience Scale (Carter-Sowell, 2010). The questionnaire consists of eight items (e.g. *In general, others leave me out of their group*) that measure general ostracism over the past four weeks with a 7-point Likert scale (1 = hardly ever to 7 = almost always). The scale has shown good validity and reliability (Carter-Sowell, 2010).

2.9.Social rank

Social rank was measured with the Social Comparison Scale (Allan & Gilbert, 1995), which consists of eleven bipolar items that ranged from zero to ten (e.g. inferior - superior, left out - accepted) rated over the past four weeks. Higher scores indicated a more positive view of the self in comparison to others. Good validity and reliability have been reported(Allan & Gilbert, 1995).

2.10. Loneliness

Loneliness was measured with the UCLA Loneliness Scale, Version 3(Russell, 1996). The questionnaire consists of twenty items (e.g. I lacked companionship) that are rated on 4-point Likert scale (1 = never to 4 = often) over the past four weeks. Good validity and reliability have been reported (Russell, 1996).

2.11. Negative schemas

Negative schemas were measured with the negative-self and -others subscales from the Brief Core Schema Scales (Fowler et al., 2006). Each of the scales contains six items (e.g. negative-self schemas: I am unloved; negative-others schemas: Other people are hostile). The original format of the scale was slightly modified for technical reasons in the online survey presentation. In the original questionnaire, participants have to answer ‘Yes’ or ‘No’ first for each item, before answering the 4-point Likert scale (1: Believe it slightly, 2: Believe it moderately, 3: Believe it very much, 4: Believe it totally). This was slightly modified into a 5-point Likert scale (1: No, Do not believe it, 2: Yes, Believe it slightly, 3: Yes, Believe it moderately, 4: Yes, Believe it very much, 5: Yes, Believe it totally). Good validity and reliability of the scale have been reported (Fowler et al., 2006).

2.12. Anxiety

Anxiety was measured with the Generalized Anxiety Disorder-7 (GAD-7) scale (Spitzer, Kroenke, Williams, & Löwe, 2006). GAD-7 consisted of seven items based on the DSM-IV diagnostic criteria for generalized anxiety disorder. The items were answered according to the past four weeks with 4-point Likert scale (1: Not at all; 2: Several days; 3: More than half the days; 4: Nearly every day). Good reliability and convergent validity had been reported for the GAD-7 (Kroenke, Spitzer, Williams, & Löwe, 2010). The scale had been used in epidemiological study and normative data is available (Löwe et al., 2008). The published English, German and Indonesian version of the questionnaire was used (available in www.phqscreeners.com).

2.13. Depression

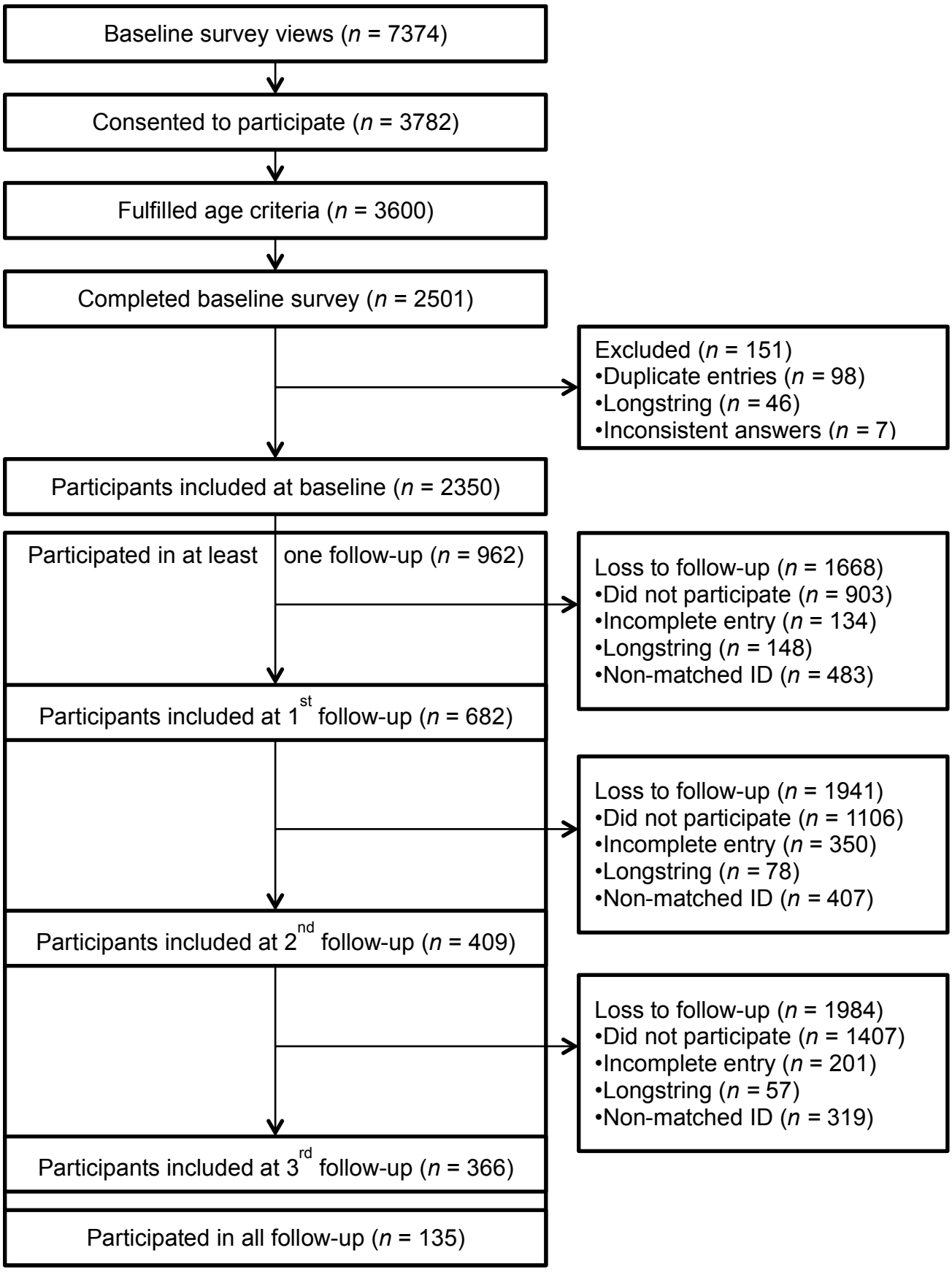
Depression was measured with the Patient Health Questionnaire-9 (PHQ-9) scale (Kroenke, Spitzer, & Williams, 2001). PHQ-9 consisted of nine items based on the DSM-IV diagnostic criteria for depression. Similar with the GAD-7, the PHQ-9 items were answered according to the past four weeks with 4-point Likert scale (1: Not at all; 2: Several days; 3: More than half the days; 4: Nearly every day). PHQ-9 had been shown to have good reliability and validity (Martin, Rief, Klaiberg, & Braehler, 2006). The published English, German and Indonesian version of the questionnaire was used (available in www.phqscreeners.com).

2.14. Psychotic symptoms

Psychotic symptoms were measured with the 20 positive symptom items and the 14 negative symptom items of the Community Assessment of Psychic Experience (Stefanis et al., 2002). The frequency of symptom experience was rated according to the past four weeks on a 4-point Likert scale (1 = never, 2 = sometimes, 3 = often, 4 = nearly always). Multidimensional conceptualization of the CAPE (bizarre experiences, hallucinations, paranoia, grandiosity, and

magical thinking latent factors load into a second-order positive symptom latent factor, while social withdrawal, affective flattening, and avolition latent factors load into a second-order negative symptom latent factor) was used as it has been shown to have better factorial validity over the original model (Schlier, Jaya, Moritz, & Lincoln, 2015).

Appendix B. Participants flowchart



Appendix C. Study 1

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Social adversities and psychotic symptoms: A test of predictions derived from the social defeat hypothesis

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Prevention

ABSTRACT

Based on the social defeat (SD) hypothesis, this study examines the postulate that various social adversities converge into one common factor, and whether this factor has an effect on psychotic symptoms while controlling for its effect on depression and anxiety. Competing hypotheses arguing for the reverse effect were also tested. The study was a cross-sectional survey in a community sample (N =2350) from Germany (n=786), Indonesia (n=844), and the United States (n=720). Confirmatory factor analysis (CFA) and path analysis with structural equation modeling were used to test the hypotheses. In the CFA two factors reflecting current and past experiences of SD could be identified with acceptable fit. Path analysis indicated acceptable fit for both SD and reverse models, and both the path from current SD to psychotic symptoms and the reverse one were significant, although the former was stronger than the latter. Interestingly, the current but not the past SD factor was significantly associated with psychotic symptoms. Overall, the results indicate that postulates derived from the SD hypothesis fit the data. However, longitudinal research is needed to further confirm the postulated directionality of the associations.

1. Introduction

Recent findings from genome-wide association studies (Schizophrenia Working Group of the Psychiatric Genomics Consortium, 2014) suggest that the role of the environment on psychotic disorders may have been underestimated. Indeed, numerous environmental risk factors including migration, discrimination, trauma and urbanization have now been identified (van Os et al., 2010). To illustrate the possible significance of these factors, Kirkbride et al. (2010) estimated that up to 22% of new psychotic cases could be prevented if all factors associated with migration were identified and removed completely.

One theoretical framework that seems promising to elucidate the socio-environmental risk factors for psychosis is the social defeat (SD) hypothesis. Selten et al. (2013) argue that long-term exposure to experiences of SD leads to a sensitization of the mesolimbic dopamine system and is a common denominator of environmental risk factors for psychosis and other mental disorders. The negative experience of being excluded from the majority group, SD, is argued to be present in major environmental risk factors for psychosis, such as migration (Cantor-Graae and Selten, 2005) and childhood trauma (Read et al., 2005). Feeling socially defeated can also be present in other risk factors, such as having a minority sexual status (Gevonden et al., 2014), being bullied in childhood (Wolke et al., 2014), having a low socio-economic status (Boydell et al., 2013), experiencing discrimination (Janssen et al., 2003), having a small social network and low social support (Gayer-Anderson and Morgan, 2013), and being overly criticized by families or confidants (Cechnicki et al., 2013).

However, so far, the notion that apparently different risk factors reflect one type of experience as conceptualized by the SD hypothesis remains a theoretical assumption, yet to be formally tested. If this assumption is affirmed, the question is whether this type of experience is a predictor of psychosis and other mental disorders and whether the predictive value is generalizable across cultures.

Another issue is that competing hypotheses have argued for the reverse causation stating that social adversity (SA) results from psychosis. One of them is the social selection hypothesis proposed by Ødegård (1932) to explain the increased risk of psychosis among Norwegian-born migrants in the United States. Another is the social drift hypothesis put forward by Dohrenwend et al. (1992) who argued that people with psychosis gradually occupy a lower socio-economic status due to inability to hold employment. Although there is an increasing body of evidence rejecting the competing hypotheses (e.g. van der Ven et al., 2015), these hypotheses may still explain a proportion of the variance and need to

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be taken into account.

Finally, although Selten et al. (2013) did not explicitly differentiate experiences of SD in childhood and adulthood, these experiences may represent separate constructs (Stilo et al., 2013) that differ in their impact on pathology due to differences in the impact they have on brain development and distress (Fox et al., 2010; Taylor, 2010).

The present study tested several predictions derived from the SD hypothesis using path analysis in a large and heterogeneous population sample from three countries located on different continents. Specifically, reflecting the claims of the SD hypothesis that apparently different negative social experiences may load on a common factor, we hypothesized that negative social experiences that have been shown in previous studies to be associated with psychosis would load on a common factor. As some of these experiences date back to childhood and adolescence whereas others reflect ongoing present adversity, our specific hypothesis was that (a) childhood abuse and bullying victim experience in school would indicate a latent past SD factor, whereas present bullying victim experiences, ostracism, social undermining, a low level of social support, small networks, low socioeconomic status, minority status, and perceived discrimination would indicate a latent present SD factor. We also hypothesized that (b) there would be specific association of the past and current SD latent factors and psychotic symptomatology (combined positive and negative symptoms) that remain after controlling for their association with depression and anxiety; (c) the path that assumes SD to precede psychosis (as predicted by the SD hypothesis) would be stronger than the path that assumes the reverse (as predicted by the competing hypothesis), and (d) the pattern of results would be stable across countries.

2. Method

2.1. Participants and procedure

Participants from Germany, Indonesia, and the United States were recruited through Crowdflower and other websites (e.g. internet forums and social networking websites) to complete an anonymous 30-min online survey. Moreover, following the sampling method from the COMED study (Hanssen et al., 2006), we advertised our study in internet forums focused on mental disorders, and particularly schizophrenia, in order to secure sufficient variation reflecting the continuum of psychosis. Participants recruited from Crowdflower received 0.50 US$ following the median hourly wage in Amazon MTurk (Buhrmester et al., 2011). Participants recruited from other websites were not given compensation for reasons of data security. Previous studies have shown that collecting self-report data on mental health symptoms over the internet is reliable (e.g. Moritz et al., 2013) and that recruiting participants through crowdsourcing websites produces demographically heterogeneous samples (e.g. Shapiro et al., 2013). Only participants who agreed with the consent statements and indicated to be above 18 years of age were allowed to participate.

There were 2501 completed survey entries, of which 151 were excluded due to duplicate entries ($n=98$), longstring (i.e. providing the same answer consecutively for 50 items, $n=46$, Johnson, 2005), and inconsistent answers ($n=7$). The final sample consisted of 2350 participants of whom 720 participants completed the English, 786 the German and 844 the Indonesian version of the survey.

2.2. Measures

Back-translation procedure and cultural adaption of measures were conducted by native Germans, British and Indonesians according to guidelines (Schmitt and Eid, 2007). A complete description of the scale and scoring procedure is available from the corresponding author.

2.2.1. Social defeat measures

Bullying victim experience frequency in childhood in a school context and adulthood in a home and work context were measured with a bullying victimization questionnaire (Wolke and Sapouna, 2008). Each experience was measured by its frequency and duration. Total scores for childhood and adulthood bullying victim experiences ranged from zero to five.

Frequency of child abuse experience before the age of 16 was measured by a self-report questionnaire developed based on a semi-structured interview from the NEMESIS study (Janssen et al., 2004). Participants were asked if they ever experienced emotional, psychological, physical, or sexual abuse (yes or no) according to a given definition that was presented (e.g. emotional abuse: "This means for example that people at home didn't listen to you, that your problems were ignored, that you had the feeling of not being able to find any attention or support from the people in your house") and to rate the frequency of the experience on a 6-point Likert scale (0=never to 5=very often).

Experiences of discrimination were assessed with the perceived discrimination measure from the NEMESIS study (Janssen et al., 2003), which includes a section on minority status and a section on perceived discrimination. Minority status was measured with five dichotomous items, which included having a minority sexual orientation, a physical disability, a visible physical condition (e.g. being obese), belonging to an ethnic minority group, and to a minority religion. The perceived discrimination section consisted of seven dichotomous items (age, sex, sexual orientation, physical disability, ethnic minority group, religion, visible physical condition). The sum score represents the degree of minority status (range 0–5) and perceived discrimination (range 0–7).

Frequency of current ostracism experiences was assessed with the Ostracism Experience Scale (Carter-Sowell, 2010). The questionnaire consists of eight items (e.g. In general, others leave me out of their group) that measure general ostracism over the past four weeks with a 7-point Likert scale (1=hardly ever to 7=almost always). The scale has shown good validity and reliability (Carter-Sowell, 2010).

Social network and support were measured by the six items version of the Social Support Questionnaire (Sarason et al., 1987). Social network was measured by asking participants to list people whom they can rely on in relation to six different conditions (e.g. Who accepts you totally, including both your worst and your best points?). The maximum number of people that participants could list for each item was nine. Social support was measured by asking participants' satisfaction concerning the support they received (How satisfied are you with the overall support?) on a 6-point Likert scale (1= very dissatisfied to 6= very satisfied).

Social undermining was assessed with the Social Undermining Scale (Vinokur and van Ryn, 1993), a five items scale that measures the frequency of negative interaction with a spouse or significant other over the past four weeks (e.g. How much does the spouse or significant other act in an unpleasant or angry manner toward you?) on a 5-point Likert scale (1=not at all to 5=a great deal).

Socio-economic status was measured with a multidimensional index developed by Lampert and Kroll (2009). The index (ranges 3–21) is construed by summing the score of education (range 1–7), household income (range 1–7), and job position (range 1–7). The respective answer choices for education and household income were created based on the census categories published by statistical offices of Germany, Indonesia, and United States. The index

measures participants' current socio-economic position relative to people from their country.

2.2.2. Symptom measures

Psychotic symptoms were measured with the 20 positive symptom items and the 14 negative symptom items of the Community Assessment of Psychic Experience (CAPE, Stefanis et al., 2002). Symptom frequency was rated according to the past four weeks on a 4-point Likert scale (1 = never to 4 = nearly always). Multidimensional conceptualization of the CAPE (bizarre experiences, hallucinations, paranoia, grandiosity, magical thinking, social withdrawal, affective flattening, and avolition) was used as it has been shown to have better factorial validity over the original model (Schlier et al., 2015).

Depression was measured with the nine item Patient Health Questionnaire-9 (PHQ-9, Kroenke et al., 2001). Anxiety was measured with the seven item Generalized Anxiety Disorder-7 (GAD-7, Spitzer et al., 2006). Both scales are based on the DSM-IV criteria and rated for presence during the past four weeks on a 4-point Likert scale (1 = not at all to 4 = nearly every day).

2.3. Analyses

The analyses were conducted with structural equation modeling (SEM). First, confirmatory factor analyses (CFA) were calculated to establish factorial validity of latent variables (past and current SD, psychotic symptoms, depression, and anxiety). Second, path analyses were conducted. Both analyses were conducted using lavaan ver. 0.5–17 (Rosseel, 2012) in R version 3.2.1 and were estimated using maximum likelihood procedure with robust standard errors and Satorra-Bentler scaled statistic to correct for non-normal distribution. The following fit indices along with the proposed cut-off criteria were used to assess the fit between hypothesized models and the data: CFI > 0.95, RMSEA < 0.06, and SRMR < 0.08 (Hu and Bentler, 1999). Because the χ^2 statistic is dependent on sample size and tends to reject well-fitting models when the samples they are based on are large (Bentler and Bonett, 1980), we did not use it as a fit criterion. We also repeated the path analyses with country as a group variable to investigate whether the results are comparable across the different countries.

2.3.1. Establishing the factorial validity of the latent variables using CFAs

Current and past latent variables of SD were predicted by various measures (see Fig. 1 for list of variables). Measures referring to the timeframe of childhood and adolescence were specified to load into the past SD latent variable and measures referring to the timeframe of present and past four weeks were specified to load into the current SD latent variable. Because the SD model has not been investigated statistically so far, we expected the initial fit to be non-optimal and sought to improve fit by using the Lagrange-Multiplier test to explore possible dependency between the indicators.

The psychotic symptoms latent variable was specified according to the multidimensional structure of the CAPE (Schlier et al., 2015). The depression and anxiety latent variables were specified to be predicted by the PHQ-9 and GAD-7 items respectively.

2.3.2. Testing the paths in the SD model and the reverse model

Following established procedures (MacCallum et al., 1993) we constructed and tested equivalent models to test the competing hypotheses. Equivalent models have same fit indices but different path directions (MacCallum et al., 1993). First, we constructed an auto-correlational model to confirm that the SD, psychotic symptoms, depression, and anxiety latent variables were correlated (see Supplementary figure, Fig. 3). Second, we constructed a SD model

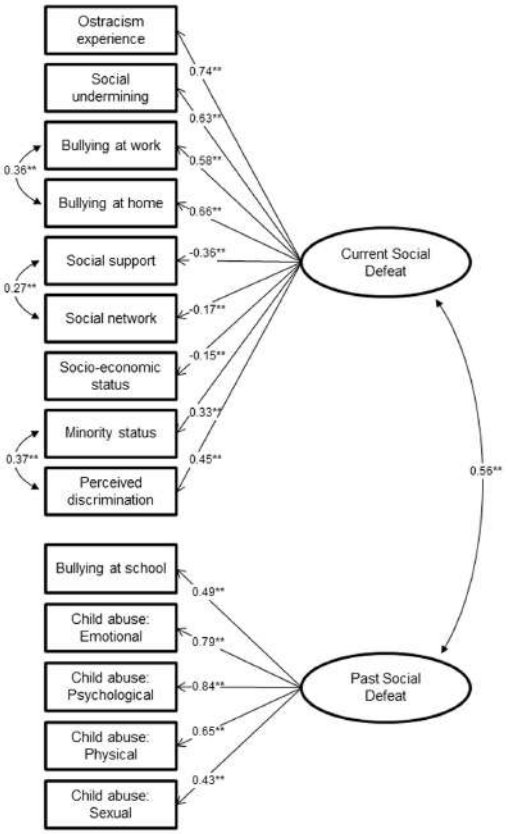

Fig. 1. Factor loadings of the improved final model of the social defeat latent variables, Note. Ellipses indicate latent variables and rectangles indicate observed variables. Curved arrows indicate correlation of residuals. Residuals of the variables are omitted from the figure. Path coefficients are completely standardized. ** p < 0.001.

with paths that reflected our main hypothesis (illustrated as the model on the left in Fig. 2). The SD model was tested under the assumption that there are causal paths leading from current and past SD to psychotic symptoms, depression, and anxiety but no reverse paths (e.g. by fixing the γ from anxiety to current SD to zero). Third, we constructed a reverse model to test for reverse social causation as hypothesized by social selection and social drift hypothesis (illustrated as the model on the right in Fig. 2). Although the reverse model included non-sensical paths (e.g. current psychotic symptoms influencing past SD), these paths had to be included to maintain equivalent degrees of freedom in the model. Analysis with comparable non-sensical paths is recommended to call into question the results of the original analysis (see Campbell and Kenny, 1999).

3. Results

3.1. Participant characteristics

The participants were 32.53 years old on average and 37.8%

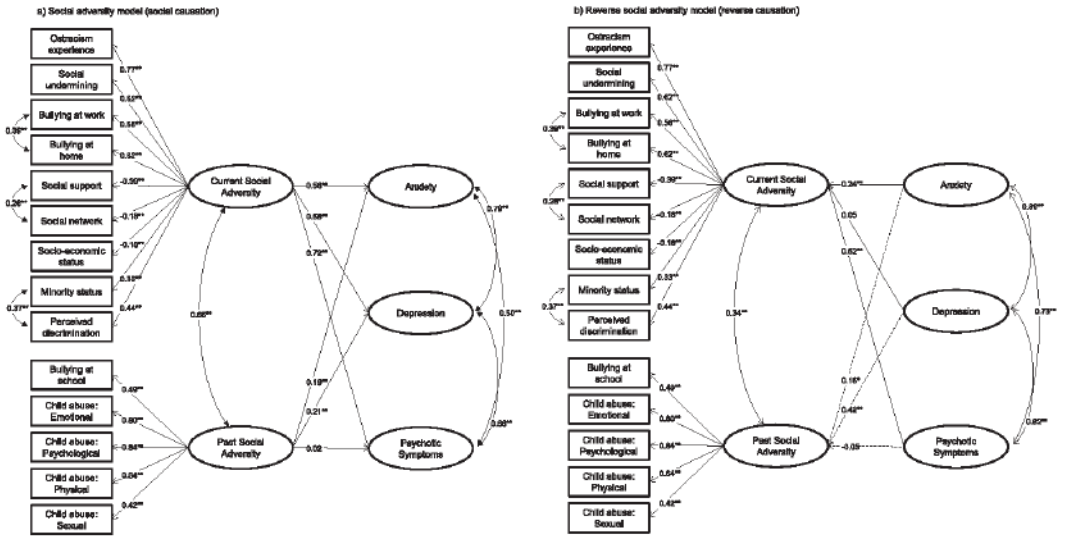

Fig. 2. The social defeat model and the reverse social defeat model. Note. a) The social defeat model (social causation) assumes that symptoms do not influence social defeat (i.e. γ from symptoms to social defeat is fixed to zero). b) The reverse social defeat model (reverse causation) assumes that social defeat does not influence symptoms (i.e. γ from social defeat to symptoms is fixed to zero). Ellipses indicate latent variables and rectangles indicate observed variables. Curved arrows indicate correlation of residuals. Solid arrows indicate factor loadings and hypothesized regression paths. Dashed arrows indicate non-sensical paths. Path coefficients are completely standardized. The factorial structures of the latent variables are not shown for reasons of space (see supplementary figure, Fig. 3, for the complete model). * $p < 0.05$,** $p < 0.001$.

were female. They tended to have high education but a low income in comparison to their national averages (see Supplementary text Section A) and 6.8% were unemployed. Moreover, 29% of the participants reported having a life-time mental diagnosis, and 3.5% participants reported to have a current diagnosis of a psychotic disorder. Detailed participant characteristics are provided in Table 1.

3.2. Factorial validity of the latent variables

The CFA of the initial SD factorial model showed that it met one of the specified cut-off fit indices criteria (SRMR =0.068; SRMR < 0.08, Hu and Bentler, 1999). Then, we improved the model's fit by using the Lagrange-Multiplier Test allowing the residuals of social support with social network, minority status with perceived discrimination, and bullying at home with bullying at work to be correlated. The final and improved SD factorial model now met the RMSEA fit index criteria for acceptable fit (RMSEA =0.074, 90% CI =0.070–0.077; RMSEA < 0.08, MacCallum et al., 1996). The non-optimal fit of the model was probably caused by the violation of the stochastic independence assumption that is central to structural equation modeling. In the context of such violation, SRMR has been shown to reflect the most accurate fit (Hu and Bentler, 1999). As the SRMR criteria were met and the RMSEA was acceptable, we regarded the final SD model fit as acceptable.

The CFA on symptom measures indicated them to have a relatively good fit to the data. The anxiety, depression, and multidimensional psychotic symptoms factorial model met two out of three fit index cut-off criteria. Detailed fit indices are reported in Table 2.

3.3. Path analysis of the SD and reverse model

The equivalent SD model and the reverse model had an acceptable fit to the data meeting criteria for two out of three fit index cut-off criteria (see Table 2 for detailed fit indices of the social and reverse causation models). Before testing the paths in the SD and the reverse model, significant bi-directional relationships between

Table 1
Participant characteristics.

Characteristic	Mean	SD	Range
Age	32.53	11.38	18–74
Socio-economic status	10.06	3.85	3–21
Education	4.55	1.89	1–7
Job position	2.64	1.87	1–7
Income	2.87	1.62	1–7
Perceived discrimination	0.83	1.17	0–5
Minority status	0.87	0.89	0–6
Bullying victim experience at work	1.64	0.85	1–5
Bullying victim experience at home	1.72	0.89	1–5
Bullying victim experience at school	2.20	1.15	1–5
Social support	4.85	1.14	1–6
Social network	2.82	1.82	0–8
Ostracism experience	2.30	1.44	1–7
Social undermining	2.06	1.01	1–5
Emotional child abuse experience	1.51	1.96	0–5
Psychological child abuse experience	1.28	1.87	0–5
Physical child abuse experience	0.97	1.58	0–5
Sexual child abuse experience	0.54	1.24	0–5
Psychotic symptoms			
Bizarre experience	1.54	0.64	1–4
Hallucination	1.39	0.65	1–4
Paranoia	1.77	0.55	1–4
Magical thinking	1.25	0.45	1–4
Grandiosity	2.01	0.80	1–4
Asociality	2.15	0.69	1–4
Anhedonia	1.84	0.65	1–4
Avolition	2.10	0.63	1–4
Anxiety	1.89	0.65	1–4
Depression	1.83	0.71	1–4

Note. The range provided is the range found in the sample, not the possible range of the scale.

Table 2
Fit indices of the factorial and structural models.

Model	Satorra-Bentler χ^2			RMSEA (90%CI)	SRMR	CFI
	χ^{2a}	*df*	*p*			
Factorial models						
Social defeat (initial model)	1508	76	< 0.01	0.090 (0.086–0.093)	0.072	0.761
Social defeat (improved final model)	1005	73	< 0.01	0.074 (0.070–0.077)	0.068	0.844
Psychotic symptoms	2756	518	< 0.01	0.043 (0.042–0.044)	0.062	0.906
Depression	553	27	< 0.01	0.091 (0.086–0.097)	0.044	0.919
Anxiety	242	14	< 0.01	0.083 (0.076–0.090)	0.030	0.960
Structural models						
Social defeat model and reverse model	9761	1621	< 0.01	0.046 (0.045–0.047)	0.091	0.843
(Past) social defeat model	8850	1363	< 0.01	0.048 (0.048–0.049)	0.075	0.829

Note. Factorial models were based on confirmatory factor analysis and structural models were based on path analyses. RMSEA = Root Mean Square Error of Approximation. SRMR = Standardized Root Mean Square Residual. CFI = Comparative Fit Index. 90%CI = 90% Confidence Interval.

the current and past SD, psychotic symptoms, depression, and anxiety latent variables were confirmed in the auto-correlational models (see Supplementary figure, Fig. 3). The factor loadings of the latent variables here were slightly different from the CFA results due to differing variables and assumptions made in the computation.

In the SD model (see left of Fig. 2) there were significant paths from current SD to psychotic symptoms, depression, and anxiety. While the path from past SD to psychotic symptoms was not significant, the paths to depression and anxiety were significant.

In the reverse model that is depicted in the right hand side of Fig. 2 we found that the path from depression to current SD was not significant. However, the paths from anxiety and psychotic symptoms to current SD were significant. Similar to the SD model, the path from psychotic symptoms to past SD was not significant, but the paths from depression and anxiety to past SD were significant.

3.3.1. *Additional analyses*

To test whether the absence of a significant path from past SD to psychotic symptoms was due to current SD, we recomputed the models including only past SD. As can be seen in Table 2, the fit indices are similar to the original SD model. The (past) SD model revealed significant paths from past SD to psychotic symptoms (γ=0.40, $p < 0.001$), depression (γ=0.52, $p < 0.001$), and anxiety (γ=0.49, $p < 0.001$).

Then, we explored the possibility of mediation of the effect of past SD on symptoms via current SD. We found that current SD completely mediated the path from past SD to psychotic symptoms (indirect effect =0.24; 95% CI =0.20, 0.28), and partially mediated the path from past SD to depression (indirect effect =0.31; 95% CI =0.26, 0.36) and anxiety (indirect effect =0.37; 95% CI =0.31, 0.43).

Furthermore, we also tested the SD model separately in each country. The pattern that current SD suppressed the effect of past SD on psychotic symptoms was found for each country (for details of these analyses see supplementary text section B and C). Moreover, we tested the model adjusting for sex and age and found a similar pattern of results. When analogous analyses were conducted including current disorder status rather than symptoms in the models (presence or absence of schizophrenia spectrum disorder, presence or absence of depression, presence or absence of anxiety disorder) a similar pattern of results was found.

4. Discussion

The study tested predictions made by the SD hypothesis, specifically that various social adverse events that are likely to leave an individual feeling socially defeated load on a common factor that is associated with symptoms of mental disorders, including psychotic symptoms. The factor analysis showed that the conceptualized bi-dimensional SD model had a relatively good fit to the data. In line with the SD hypothesis that postulates SD to be a non-specific risk factor of mental disorders (Selten et al., 2013), we found that current SD made an independent significant contribution to symptoms of psychosis, depression, and anxiety, albeit the effect was the largest for symptoms of psychosis. A similar pattern of findings was shown when the model was examined for each country separately. Furthermore, although we also found some support for the social drift and social selection hypotheses, the path from current SD to symptoms of psychosis was almost twice as strong as the reverse path, indicating that SD played a stronger role in predicting rather than in resulting from symptoms of psychosis. Taken together, the findings thus support the notion that SD is a significant risk factor for symptoms of psychosis, as well as for symptoms of depression and anxiety. Thus, it appears to be a risk factor that can be generalized to different syndromes as well as to different regions and cultures.

Although we focused on symptoms across the continuum rather than on predicting the presence or absence of clinical disorders, we argue that the findings are generalizable to the prediction of clinical disorder status because subclinical symptoms are a reliable predictor of clinical disorders (Werbeloff et al., 2015; Zammit et al., 2013) and are associated with the same risk factors found for clinical disorders (see review, Linscott and van Os, 2013). In support of this, we found a similar pattern of results after substituting symptoms with the presence or absence of psychotic disorders, anxiety disorders or depression.

The pronounced path from SD to symptoms of psychotic symptoms is also in line with cognitive models of psychosis, especially if we assume that the translating mechanisms between SD and psychosis are negative concepts of the self and others along with negative affective states (Freeman and Garety, 2014; Kesting et al., 2013; Kesting and Lincoln, 2013). Moreover, although the somewhat weaker path from symptoms of psychosis to SD is in line with the social drift hypothesis it might also be accounted for by stigma and self-stigma (Penn et al., 2000).

Unexpectedly, past SD did not significantly predict symptoms of psychosis in our model. As past SD consists of bullying victim experience at school and child abuse, this seems to contradict previous research showing a strong link between psychosis and childhood adverse events (van Dam et al., 2015; Wolke et al., 2014). Our additional analysis, however, indicated that current SD mediated the effect of past SD on symptoms of psychosis. Thus, people who experienced childhood trauma or were bully victims at school are very likely to also experience current SD which is

linked to symptoms. In line with this, studies on bullying have shown a continuous line of adversity from childhood into adulthood (Arseneault et al., 2010).

In regard to the practical implications, the findings suggest that interventions that help people to cope with ongoing psychosocial adversity may be effective in preventing or reducing psychotic symptoms. Although reducing SD directly is clearly an important societal aim, from a therapeutic perspective it also appears worthwhile to identify factors that translate SD into symptoms, such as negative views of self and others or perceiving oneself as low in social rank and target these factors in psychological interventions.

4.1. Limitations

The sample was drawn from a population with access to the internet. Similar to other studies that used crowdsourcing websites for recruitment (e.g. Shapiro et al., 2013), participants tended to be highly educated and to have middle to low income in comparison to the national average. For example, 14.7% of the participants from Germany had a university degree, while only 8.3% of the German population have a university degree according to 2013 census (Federal Statistical Office (Destatis), 2014). This slightly reduced heterogeneity may have led to an attenuation of the relationships' strengths (see Barrett, 2001), although an overestimation of the associations can also not be ruled out. Second, the self-reported diagnosis of the participants cannot be externally verified. However, false diagnostic status seems unlikely because the reward for completing the long survey was minimal and we used several data verification processes to ensure data validity. Furthermore, the average rate of 29% (Germany, 27.2%; Indonesia, 23.2%; US, 33.0%) of participants with a life-time mental diagnosis in the present study is comparable to the reported rate based on nationally representative sample (e.g. 31% from Germany (Jacobi et al., 2004), 30.5% from the United States (Kessler et al., 2005)). Third, the cross-sectional design forces us to rely on assumptions in making inferences about the direction of the relationship of SD and psychosis. Relatedly, there is the possibility of a recall bias in regard to past SD that might have led to an overestimation in the strength of current and past SD. Moreover, participants with a history of mental illness may differ from those without a history of mental illness in recalling traumatic experiences. Fourth, despite being acceptable, the model fit of the factorial and structural models was not good. The model fit was not optimal due to the violation of the stochastic independence assumption that is central to structural equation modeling. For example, small social network and low satisfaction of social support often co-occur, and are thus stochastically dependent. Future studies investigating SD may be advised to use stochastically independent measures and – based on our findings – select those with high factor loadings on SD, such as ostracism experiences, and avoid measures with low factor loadings on SD such as social network.

4.2. Conclusion

By showing that various past and present socially adverse experiences load on common factors and that the path leading from these factors to psychosis is stronger than the one in the reverse direction, this study adds to the accumulative evidence supporting the SD hypothesis (e.g. Gevonden et al., 2015; Valmaggia et al., 2015; van der Ven et al., 2015). Future studies could now take the test of the model a step further and attempt to operationalize the subjective perception of SD, for example by using measures of social rank or self-and other related schemas and testing whether these factors mediate the association between SD and symptoms.

Ethical statement

This study received approval from the ethical commission of German Psychological Society and was carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki).

Conflict of interest

The authors declare that they have no conflict of interest.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.psychres.2016.09.002.

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Appendix D. Study 2

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Social Adversity and Psychosis: The Mediating Role of Cognitive Vulnerability

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Background: **Social adversity is a risk factor for psychosis, but the translating mechanisms are not well understood. This study tests whether the relationship between social adversity and psychosis is mediated by cognitive vulnerability in the form of low perceived social rank, negative schemas related to self and other, and loneliness and whether the putative mediations are specific to psychosis or are largely explained by depression.** ***Methods:*** **The study was a survey in a community sample (N = 2350) from Germany (n = 786), Indonesia (n = 844), and the United States (n = 720). Mediation path analysis with structural equation modeling was used to test for the specificity of the hypothesized paths to psychosis controlling for depression.** ***Results:*** **Social adversity had a significant medium to large effect on positive (R^2 = .20) and negative symptoms (R^2 = .38). Social rank, negative schemas, and loneliness significantly mediated the relationship between social adversity and negative symptoms and the models explained a large amount of the variance (R^2 = .43–.44). For positive symptoms, only negative schemas were a significant mediator (R^2 = .27).** ***Discussion:*** **The results emphasize the role of social adversity in psychosis and support the assumption that cognitive vulnerability is a relevant translating mechanism as postulated by the social defeat hypothesis and cognitive models of psychosis. This underlines the relevance of the clinical practice of targeting beliefs in cognitive interventions for psychosis. It also indicates that targeting cognitive vulnerability in people experiencing social adversity could be a promising approach to prevention.**

Key words: social adversity/etiology/childhood trauma/bullying/trauma/social exclusion/prevention

Introduction

Studies with varying designs carried out in different contexts have repeatedly shown social adversity (SA) to be a major risk factor for psychosis. Experiences of SA associated with psychosis and its persistence include childhood trauma,[1,2] migration,[3] having a minority sexual status,[4] being bullied in childhood,[5] having a low socioeconomic status,[6] experiencing discrimination,[7] having a small social network and low social support,[8] and being exposed to high levels of expressed emotion by families or confidants.[9] Moreover, many of these experiences are also predictive of psychotic experiences at a subclinical level[10] and in patients with nonpsychotic disorders.[11] For example, childhood trauma has been shown to be a risk factor for psychotic experiences in children[12] and adults,[13] which again are known to predict psychotic disorders in later life.[14,15]

Although the relationship between SA and psychosis is well established, the translating psychological mechanisms from SA to the emergence of psychotic symptoms are not well understood. Identifying such processes would provide us with targets to develop preventive psychosocial interventions that may reduce the number of psychotic cases traceable to SA (eg, 22% of psychotic cases can be traced to migration[16]).

One possible translating mechanism that has been proposed is social defeat,[17] a concept that originates from animal experimental research. Rodents exposed to chronic social stress (eg., by moving them into a cage with a dominant rodent) exhibit behavioral markers of schizophrenia such as deficits in exploration and motivation, similar to negative symptoms, and fear of other rodents, similar to paranoia.[18,19] Investigating social defeat in humans is more complex as the behavioral signals are less obvious and the appraisal component needs to be taken into account. A construct that closely resembles social defeat in humans and appears to be a good way of operationalizing social defeat is the appraisal of being low in social rank,[20] which has been found to be significantly associated with psychotic symptoms[21,22] and to trigger paranoid

beliefs.[23] Thus, we expect social rank to mediate the association between SA and positive and negative symptoms of psychosis.

Other potential mediators between SA and psychotic symptoms are global negative beliefs about the self and others. Cognitive models of psychosis have emphasized that negative schemas, particularly those related to the self, are relevant to the development of psychotic symptoms.[24–26] In support of these models, numerous studies find both positive and negative symptoms of psychosis to be associated with negative schemas about the self,[27,28] and some have also found negative schemas about other people to be relevant.[29] Furthermore, self-esteem has been shown to mediate the effect of social exclusion on psychotic symptoms[30] and negative beliefs about the self and others to mediate the link between childhood adversity and psychotic symptoms.[31]

A third potential mediator that is intuitively linked to some of the SA associated with psychosis (eg, small networks, discrimination) is loneliness, which is defined as a subjective appraisal of social isolation along with feelings of sadness.[32] Indeed, it has been shown that being a bully victim in adolescence predicts loneliness in young adulthood.[33] Moreover, loneliness was related to psychotic symptoms in first-episode psychotic patients[34] and in a community sample (Jaya et al, unpublished data).

To sum up, there is an evidence-based rationale to expect social rank, negative beliefs about self and others, and loneliness to result from an accumulation of adverse social experiences and to mediate the known association of SA on both positive and negative symptoms of psychosis. However, both SA[35] and the proposed mediators—social rank,[36] negative schemas,[37] and loneliness[38]—are also predictive of depression. Moreover, depression is strongly associated with both positive[39] and negative[40] symptoms of psychosis, which needs to be taken into account to ascertain the specificity of translator mechanisms to psychosis.

Building on previous work from our group showing a strong association between SA and psychosis (Jaya and Lincoln, unpublished data), the present study tests the hypothesis that perceived low social rank, negative schemas, and loneliness mediate the effect of SA on both positive and negative symptoms. We also hypothesize that such associations are not fully explicable by co-occurring depression.

Finally, it is important to consider that findings based on a narrow single subpopulation, such as people from white, educated, industrialized, rich, democratic countries, often do not generalize to other subpopulations.[41] A problem of generalization is particularly likely when the impact of social factors is a core focus of the study. Thus, in order to increase generalizability, the proposed mediation models were tested in a large community sample from 3 countries located in different continents with distinct social and political systems: Germany, Indonesia, and the United States.

Methods

Participants and Procedure

The multinational survey (part of the data set from this multinational survey has been used to study the basic association of SA and psychosis [Jaya and Lincoln, unpublished data]. The present study continues this work by investigating putative mediators of the association) targeted participants by posting the survey in Crowdflower and on other websites (eg, social networking websites and internet forums, particularly forums on the topic of mental health disorders and schizophrenia). Similar to Amazon MTurk, Crowdflower is a crowdsourcing website on which users can participate in a study in exchange for financial compensation. The inclusion criteria were agreement with the consent statements and age above 18 years. Of the initial 2501 survey entries, 151 were excluded due to duplicate entries and inconsistent answers. The final sample included 2350 participants of whom 720 completed the English, 786 the German, and 844 the Indonesian version of the survey. Part of the data set from this multinational survey has been used to study the basic association of SA and psychosis (Jaya and Lincoln, unpublished data). The present study continues this work by investigating putative mediators of the association.

Measures

The translation and back-translation and cultural adaption of measures was conducted by native Germans, British, and Indonesian according to established guidelines.[42]

Positive and negative symptoms were measured with the frequency scales for positive symptoms (20 items) and negative symptoms (18 items) from the Community Assessment of Psychic Experiences (CAPE).[43] Frequency of symptoms experienced during the past 4 weeks was rated by participants on 4-point Likert scales from never to nearly always. The German and English versions demonstrate good validity and reliability.[44,45]

Depression was measured with the 9-item Patient Health Questionnaire-9 (PHQ-9) that is based on the DSM-IV criteria for depression[46] and thus measures all relevant symptoms of depression. It has been shown to have good reliability and validity in large representative population sample[47] and measurement invariance among different ethnic groups.[48] Items were answered according to the past 4 weeks on 4-point Likert scale from not at all to nearly every day.

Bullying victim experiences in childhood and adulthood were measured with a bullying victimization questionnaire.[49] The frequency and duration of bullying were assessed with 6 items. The possible total score for bullying victim experiences ranged from 0 to 5.

Child abuse experiences before the age of 16 years were measured with a self-report questionnaire that was based on a semistructured interview from the NEMESIS study.[50] Participants were asked if they ever experienced

emotional, psychological, physical, or sexual abuse (yes or no) according to a given definition that was presented (eg, emotional abuse: "This means for example that people at home did not listen to you, that your problems were ignored, that you had the feeling of not being able to find any attention or support") and to rate the frequency of the experience on a 6-point Likert scale.

Discrimination experiences were assessed with items modified from the NEMESIS study.[7] This measure contains 2 subscales: minority status and perceived discrimination. Minority status was measured with 5 statements that can be ticked if they are applicable. There was a statement each for having a minority sexual orientation, having a physical disability, belonging to an ethnic minority group, belonging to a minority religion, and having a visible physical condition (eg, being obese). Thus, the total score for minority status ranges from 0 (no minority status) to 5 (minority status in each of the 5 domains). Perceived discrimination was measured with 6 dichotomous (yes/no) items (eg, Have you ever been discriminated due to having a minority sexual orientation or gender identity?) related to age, sex, sexual orientation, physical disability, religion, and visible physical conditions, with the total score accordingly ranging from 0 to 6.

Ostracism was measured with the Ostracism Experience Scale (OES) that consists of 8 items (eg, in general, others leave me out of their group) measuring the frequency of ostracism experiences over the past 4 weeks on a 7-point Likert scale.[51] The OES demonstrates good validity and reliability.

Social network and support were measured by the 6-item version of the Social Support Questionnaire (SSQ-6).[52] Social network was measured by asking participants to list a maximum of 9 people whom they can rely on in relation to 6 different conditions (eg, being accepted, being supported, etc.). The score consisted of the number of people noted and thus ranges from 0 to 9. Social support was measured by asking participants' satisfaction concerning the support they received on a 6-point Likert scale. The validity and reliability of the SSQ-6 are good.[52]

Social undermining was measured with the 5-item Social Undermining Scale[53] that measures the frequency of negative interaction with a spouse or significant other over the past 4 weeks on a 5-point Likert scale and has good validity and reliability.[53]

Socioeconomic status was measured with a multidimensional index[54] construed by summing the score of education, household income, and job position (total index score ranged 3–21). The answer choices for education and household income were created based on the census categories published by the statistical offices of Germany, Indonesia, and United States. The index measures participants' current socioeconomic position relative to people from their country.

Social rank was measured with the Social Comparison Scale (SCS),[20] which consists of 11 bipolar items that ranged from 0 to 10 (eg, inferior-superior, left out-accepted) rated over the past 4 weeks. Higher scores indicate a more positive view of the self in comparison with others. Good validity and reliability have been reported.[20]

Negative schemas were measured with the negative-self and -others subscales from the Brief Core Schema Scales (BCSS).[55] Each of the scales contains 6 items (eg, negative-self schemas: I am unloved; negative-others schemas: Other people are hostile) rated on a 5-point Likert scale (1 = no, do not believe it to 5 = yes, believe it totally). Good validity and reliability of the scale have been reported.[55]

Loneliness was measured with the UCLA Loneliness Scale, Version 3, which consists of 20 items (eg, I lacked companionship), rated on 4-point Likert scale (1 = never to 4 = often) over the past 4 weeks, and has shown good validity and reliability.[56]

Analyses

The analyses were conducted with structural equation modeling (SEM) using the lavaan package version 0.5-18[57] in R version 3.2.2. A maximum likelihood estimation procedure with robust standard errors and Satorra-Bentler scaled statistic was used to correct for non-normal distribution. The Comparative Fit Index (CFI) > 0.95, root mean square error of approximation (RMSEA) < 0.06, and standardized root mean square residual (SRMR) < 0.08[58] along with the proposed cutoff criteria were used to assess the fit between the hypothesized models and the data. Chi-square was reported but not used as a goodness-of-fit criterion because it tends to reject models based on large sample sizes.[59] Moreover, we compared the relative goodness of fit of the different mediation models with the Akaike information criterion, where smaller values indicate a better fit.[60] We also reported the R^2 effect-size measure for each model as it has been shown to be a good measure for mediation analysis with a low bias for samples of $N \geq 100$.[61]

Latent Variable Specification. The SA latent variable was specified to be predicted by bullying at home, bullying at work, minority status, perceived discrimination, ostracism experience, social network, social support, social undermining, socioeconomic status, bullying at school, emotional child abuse, psychological child abuse, physical child abuse, and sexual child abuse. The positive and negative symptoms latent variables were specified according to the multidimensional factorial structure of the CAPE.[45] Depression, social rank, and negative schemas latent variables were specified according to their respective items. The Loneliness latent variable was specified according to the latent factorial structure of the scale.[62]

Mediation Analysis. Following a recommended procedure,[63] we first tested for a direct relationship between the SA latent variable and the outcome latent variables, and then entered the postulated mediators to test for reduction in the estimates of the direct relationships. Furthermore, the bias-corrected bootstrap 95% confidence interval (CI) of the

indirect effect was used as another indicator of mediation as recommended.[64] A variable was considered a mediator if the CI of the indirect effect did not include 0. By including positive symptoms, negative symptoms, and depression as outcome variables simultaneously in one analysis, which is not possible in traditional regression analysis, it is possible to estimate the specific effect of the SA latent variable (independent variable, IV) and the mediators (M) on each single outcome while controlling for the effects of IV and M on the other outcomes. For example, if the mediation effect of negative schemas on psychotic symptoms is due to the covariance between psychotic symptoms and depression this would show as zero effect of the paths from SA to negative schemas on positive and negative symptoms, but a significant path to depression.

SEM also enables to directly estimate and account for measurement error terms, which makes the estimates more accurate than traditional regression approaches that are susceptible to overestimation or underestimation.[65] Reported path coefficients (γ and β) are completely standardized. Reported direct, indirect, and total effect coefficients are unstandardized. Additionally, the proportion of variance explained (R^2) by the model is reported as a measure of effect size that can be interpreted as small (.01), medium (.09), and large (.25) according to Cohen.[66]

Results

Participant Characteristics

Detailed participant characteristics are reported in table 1, and a table outlining the participants' characteristics per country is available in the supplementary section A. As can be seen, the sample included a broad age range and—with very few exceptions—spanned the full range of possible answers concerning SA and the postulated mediators. Furthermore, 29% of the participants reported a lifetime mental diagnosis, and 3.5% a current diagnosis of a psychotic disorder.

Mediation Analyses

The tested models could be identified and fit indices were satisfactory meeting 2 out of 3 fit index criteria, except for the loneliness mediation model that only met one fit index (detailed fit indices are reported in table 2). The preconditions of mediation were fulfilled, which is indicated by significant bidirectional relationships between the SA latent variable, the mediator latent variables, and the outcome latent variables (see supplementary figure 1). Detailed path coefficients of the models are reported in figure 1, and the indirect effect, total effect, and R^2 are reported in table 3. In the direct effect model, the paths from SA to all outcomes were significant (positive symptoms, $\gamma = 0.45$, $P < .001$, $R^2 = .20$; negative symptoms, $\gamma = 0.61$, $P < .001$, $R^2 = .38$; depression, $\gamma = 0.67$, $P < .001$, $R^2 = .45$) with small to medium-sized effects.

As can be seen in table 3, entering social rank as putative mediator increased the direct effect path from SA to positive symptoms while the indirect effect coefficient was negative indicating suppression (we conducted an additional exploratory analysis and found that taking SA out of the mediation model rendered the association between social rank and positive symptoms significant). However, social rank was a significant mediator for negative symptoms and

Table 1. Participant Characteristics

Characteristic	Mean	SD	Sample Range	Possible Range
Gender, female (%)	888 (37.8%)	—	—	—
Age	32.53	11.38	18–74	18–99
Socioeconomic status	10.06	3.85	3–21	3–21
Perceived discrimination	0.83	1.17	0–5	0–6
Minority status	0.87	0.89	0–5	0–5
Bullying victim experience at work	1.64	0.85	1–5	0–5
Bullying victim experience at home	1.72	0.89	1–5	0–5
Bullying victim experience at school	2.20	1.15	1–5	0–5
Social support	4.85	1.14	1–6	1–6
Social network	2.82	1.82	0–8	0–9
Ostracism experience	2.30	1.44	1–7	1–7
Social undermining	2.06	1.01	1–5	1–5
Emotional child abuse experience	1.51	1.96	0–5	0–5
Psychological child abuse experience	1.28	1.87	0–5	0–5
Physical child abuse experience	0.97	1.58	0–5	0–5
Sexual child abuse experience	0.54	1.24	0–5	0–5
Social rank	5.80	1.78	1–10	1–10
Negative schema	1.93	0.84	1–5	1–5
Loneliness	2.22	0.59	1–4	1–4
Positive psychotic symptoms	1.67	0.54	1–4	1–4
Negative psychotic symptoms	2.04	0.58	1–4	1–4
Depression	1.83	0.71	1–4	1–4

Table 2. Fit Indices of the Mediation Models

Model	Satorra-Bentler χ^2			RMSEA (90% CI)	SRMR	CFI	AIC[a]
	χ^{2a}	df	*P*				
Direct effect	8671	1522	<.01	0.045 (0.044, 0.046)	0.077	0.839	304 713
Mediation models							
Social rank	10 657	2172	<.01	0.041 (0.040, 0.041)	0.075	0.864	398 550
Negative schema	16 584	2256	<.01	0.052 (0.051, 0.053)	0.080	0.773	371 604
Loneliness	14 957	2825	<.01	0.043 (0.042, 0.043)	0.090	0.830	414 118

Note: AIC, Akaike information criterion; CFI, comparative fit index; CI, confidence interval; RMSEA, root mean square error of approximation; SRMR, standardized root mean square residual.
[a]Rounded to the next integer.

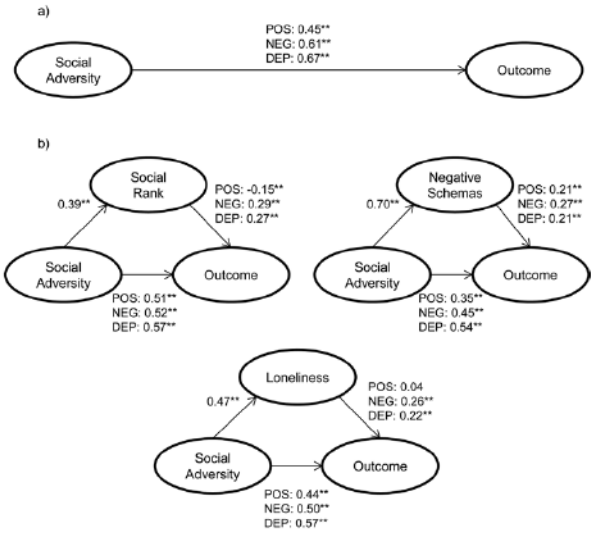

Fig. 1. Mediation analyses. The latent variable *Outcome* represents positive symptoms, negative symptoms, and depression. Each of the models was computed separately. (A) Direct effect model, (B) mediation models. Only latent variables are shown. Path coefficients are completely standardized estimates. ** $P < .01$. DEP, depression; NEG, negative symptoms; POS, positive symptoms.

Table 3. Indirect Effect, Total Effect, and R^2 of the Mediation Models for Positive Symptoms, Negative Symptoms, and Depression

Model	Indirect Effect (95% CI)			Total Effect			R^2		
	POS	NEG	DEP	POS	NEG	DEP	POS	NEG	DEP
Direct effect				0.39	0.51	0.67	.20	.38	.45
Mediation model									
Social rank	−0.05 (−0.07, −0.03)	0.08 (0.06, 0.10)	0.10 (0.08, 0.13)	0.39	0.52	0.67	.22	.43	.52
Negative schemas	0.12 (0.08, 0.17)	0.16 (0.11, 0.20)	0.14 (0.10, 0.19)	0.43	0.53	0.69	.27	.44	.49
Loneliness	0.02 (−0.00, 0.04)	0.10 (0.08, 0.13)	0.11 (0.08, 0.13)	0.40	0.52	0.67	.21	.44	.50

Note: Unstandardized coefficient estimates are reported. CI, confidence interval; DEP, depression; NEG, negative symptoms; POS, positive symptoms.

depression, indicated by a reduction of the direct effect path coefficients from SA to negative symptoms and depression and by significant indirect effect coefficients (confidence interval not including 0). The social rank mediation model explained a large proportion of variance of negative symptoms ($R^2 = .43$) and depression ($R^2 = .52$).

Negative schemas reduced the direct effect path coefficients of all outcomes and had significant indirect effects indicating mediation for positive symptoms, negative symptoms, and depression. Notably, the negative schemas mediation model explained a large proportion of variance of all outcomes (positive symptoms, R^2 = .27; negative symptoms, R^2 = .44; depression, R^2 = .49).

The path from loneliness to positive symptoms was not significant, and the indirect effect coefficient was insignificant, which indicated no mediation. The paths from loneliness to negative symptoms and depression were significant, and the indirect effect coefficients were significant, which indicated mediation. The loneliness mediation model explained a large proportion of variance of negative symptoms (R^2 = .44) and depression (R^2 = .50).

Analogous subgroup mediation analyses were conducted to investigate potential differences among countries, participants with and without a mental disorder, and those with a diagnosis from the schizophrenia spectrum. A similar pattern of results was found within each of these subgroups. Specifically, negative schemas were a significant mediator for the paths from SA to positive symptoms, negative symptoms, and depression. Social rank and loneliness were significant mediators for the paths from SA to negative symptoms and depression. For further details, please see section B in the supplementary text.

Discussion

This study set out to identify relevant mediators of the association between SA and positive and negative symptoms. As such, we investigated the putative translating role of cognitive vulnerability defined as perceived low social rank, negative schemas, and loneliness.

As expected, negative schemas were strongly linked to both SA and symptoms and turned out to be the most relevant mediators in regard to all symptomatic outcomes. Unspectacularly, this confirms cognitive conceptualizations of depression.[67] More interestingly, it also confirms cognitive models of psychosis, in which negative schemas and beliefs are postulated to play a central role in the development of both positive[24,26,28,68] and negative[69] symptoms. The question of how negative schemas translate into psychotic symptoms, however, has been an ongoing subject of debate and speculation. One view is that delusions reduce feelings of inadequacy and the associated negative affect by attributing self-threatening events to others.[24,70] For example, a patient who feels persecuted by the secret service at work could be preserving his or her self-esteem by holding the secret service responsible for own work-related failures. Other researchers[25] propose that persecutory delusions directly reflect impaired self-esteem and the associated emotions. According to this explanation, the delusion that the secret service is observing the patient reflects the patient's concern about his or her incompetence and the anxiety resulting from this view. Similarly, the content of auditory hallucinations has been discussed as a direct reflection of negative views of self and others.[71,72] Another potential mechanism with relevance to negative symptoms is that negative views of self and others will inhibit motivation in general and the natural drive to connect with others, and thus prevent experiences of self-efficacy and positive, corrective social experiences.[73]

Social rank and loneliness, in contrast, only mediated the pathway from SA to negative symptoms and depression. The absence of a significant relationship between loneliness and positive symptoms is likely to be due to our stringent analysis controlling for depression and negative symptoms, which has not been done in previous studies (eg, ref.[34]). Interpreted in this way, it confirms a previous study by our group (Jaya et al, unpublished data), in which we found that depression explains the relationship between loneliness and positive symptoms.

We found that taking social rank into account enhanced the strength of the relationship between SA and positive symptoms. Nevertheless, and in line with previous work (eg, ref.[23]), we found a significant relationship between social rank and positive symptoms when computed in isolation. Thus, previous studies may have found a significant relationship between social rank and positive symptoms because they did not take SA into account. In regard to negative symptoms, however, social rank showed the postulated mediating effect. Thus, the idea that the appraisal of being socially inferior which is induced by SA fosters deficits in exploration and motivation which manifest in negative symptoms was supported. Moreover, our data show that this is not solely explained by depression.

Notably, we found that the mediation results were stable across countries and subgroups, indicating a global mechanism of cognitive vulnerability as a translator of the link between SA and psychosis and SA and depression. This is an important finding because to our knowledge the cognitive models of psychosis have not been investigated outside the European and North American context despite the fact that many psychological constructs are vulnerable to being affected by cultural idiosyncrasies.[41]

Strengths and Limitations

One limitation is that the cross-sectional design forced us to rely on assumptions in making inferences about the direction of the relationship of SA, mediators, and psychosis, which are based on previous experimental[30,74] and longitudinal studies.[75] Longitudinal designs are required to further strengthen these assumptions, and our research group is currently pursuing this endeavor.

A strength of the study is that the participants covered the whole psychosis continuum as indicated by a relatively high prevalence of individuals with a diagnosis of mental disorder including psychotic disorder. Looking at associations across the continuum is advantageous when it comes to investigating putative causal factors because the findings

can be interpreted free of issues that often influence studies comparing clinical with nonclinical populations, such as selective small samples, treatment history, stigma, and medication. A limitation is that the recruiting method limited participants to those with access to the Internet. Similar to other studies that used crowdsourcing websites for recruitment (eg, ref.[76]), participants in this study tended to be somewhat more educated in comparison with their national average and males were slightly overrepresented. The associations might have been even stronger in a sample with more representatives from low socioeconomic backgrounds. However, the sample spanned the full range on almost every variable assessed, and the sizes of the low-income groups were comparable in size with those provided in German and US census data so that we can exclude ground or ceiling effects on any of the variables. Although the sample was biased toward slightly younger participants, this cannot be considered as a fatal flaw when the aim of the study is to investigate pathways to psychosis, which are most relevant to people in their early 20s. Furthermore, although we oversampled participants with schizophrenia spectrum disorders to have sufficient variation in symptoms and increase statistical power for subgroup analyses, the rate of overall psychopathology in our sample was comparable with typical samples reported in epidemiological studies. For example, 29% of the participants in the study reported a lifetime mental disorder diagnosis, which is similar to the rates presented in representative population samples.[77]

Conclusion

In sum, our expectation that cognitive vulnerability would explain a significant part of the psychological black box between SA and psychotic symptoms was confirmed. The strongest translating factor for all 3 types of symptomatic outcome seems to be dysfunctional schemas pertaining to the self and others. The mechanism may operate through a cascade of cognitive and behavioral mechanisms and their interactions, which need further elaboration. In contrast, perceiving oneself as being low in social rank and loneliness appear to be more relevant to negative symptoms and depression than to positive symptoms.

Overall, our results suggest that targeting cognitive vulnerability in people experiencing SA could have a protective effect in regard to psychotic symptoms. Reducing SA is likely to be challenging and costly and goes beyond the scope of the psychological and psychiatric profession. However, targeting cognitive vulnerability in people experiencing SA is well within the scope of the profession and could be a valid strategy for prevention of psychosis and other psychopathologies.

Supplementary Material

Supplementary material is available at http://schizophreniabulletin.oxfordjournals.org.

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Appendix F. Study 3

A Longitudinal Mediation Analysis of the Effect of Negative-Self Schema on Positive Symptoms via Negative Affect

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Abstract

Background. Both cognitively focused and integrated socio-developmental models of psychosis postulate that a negative concept of the self causes and maintains positive symptoms and that this link is mediated by negative affect. However, only few studies have tested the temporal mediation claim systematically using an appropriate design.

Methods. A longitudinal cohort design in an online community sample (N = 962) from Germany, Indonesia, and the United States was used. Negative-self schema (NS), negative affect, and positive symptoms were measured repeatedly over a one-year period (baseline, 4-, 8-, and 12-month follow-up). Cross-lagged panel and longitudinal mediation analyses with structural equation modeling were used to test the hypothesis that NS precede positive symptoms and that this link is mediated by negative affect.

Results. Independent cross-lagged panel models showed a significant unidirectional longitudinal path from NS to positive symptoms and bidirectional longitudinal associations from NS to negative affect. There was also a significant indirect pathway from NS at baseline via negative affect at 4- and 8-month follow-up to positive symptoms at 12-month follow-up, indicating mediation.

Conclusions. Our findings support the postulated affective pathway from NS to symptoms via negative affect. More specifically, our data indicate that negative views about the self and negative affect influence each other and build up over the course of several months before exerting an influence on positive symptoms. We conclude that interrupting this process by targeting NS and negative affect early in the process could be a promising strategy to prevent the exacerbation of positive symptoms.

Keywords: emotion regulation; schizophrenia; depression; anxiety, psychotic experiences; mechanism; mediation; longitudinal

A Longitudinal Mediation Analysis of the Effect of Negative-Self Schema on Positive Symptoms via Negative Affect

1. Introduction

"(…) we are still without an explanation of the essential nature of delusion. The same may be said for the attempt to derive delusion from preceding affects (…)." Jaspers (1963). We have come some way since this sobering evaluation by Jaspers in the 1960s. A number of theoretical models on the emergence of positive symptoms have been proposed synthesizing evidence on cognitive, social, and emotional processes from epidemiological, cross-sectional, experimental, and treatment studies (e.g. Garety *et al.* 2001; Beck & Rector 2003; Kesting & Lincoln 2013; Bentall *et al.* 2014; Howes & Murray 2014). Although these models differ in emphasis, they cohere in stressing the importance of negative affect and extreme negative beliefs about the self (negative-self schema, NS) in explaining the exacerbation of psychosis.

In one of the first and most widely cited of these models, positive symptoms are postulated to be both exacerbated and maintained by NS, resulting from external stressors and social adversity (Garety *et al.* 2001). Specifically, the model postulates a unidirectional pathway from NS to symptoms which is mediated by negative affect. This model has been cited more than five hundred times according to Web of Science since its publication in 2001. It has been immensely important to inform cognitive behavioral therapy for psychosis (CBTp) that lays an emphasis on changing dysfunctional beliefs about the self and now is recommended as a first-line treatment for psychosis (National Institute for Health and Clinical Excellence 2009). In a recent model Howes and Murray (2014) integrate the cognitive perspective with socio-developmental and neural explanations of psychosis. They postulate that childhood adversity both sensitizes the dopamine system, resulting in excessive dopamine synthesis and aberrant assignment of salience to stimuli, and biases the cognitive

schema that the individual uses to interpret experiences in a way that renders paranoid interpretations more likely.

The central postulates related to the cognitive processes in these models are that 1) social stressors result in negative beliefs about self- and others, 2) that NS precede and cause symptoms via affective states. Whereas the first postulate has been backed up by evidence in a rigorous 30 years longitudinal study (Fergusson *et al.* 2013), the second has found some support but lacks evidence from compelling longitudinal studies. Specifically, the postulate that NS play an important role in the emergence of symptoms has been supported by the finding that decreases in self-esteem precede paranoid symptoms within intervals of one to several hours in daily life (Thewissen *et al.* 2011), in experimental studies (e.g. Kesting *et al.* 2013) and over the course of months (Fowler *et al.* 2012). Similarly, experience sampling studies show that negative affect precedes the emergence of paranoia in daily life (Ben-Zeev *et al.* 2011; Thewissen *et al.* 2011; Kramer *et al.* 2013) and two longitudinal studies found anxiety and depression to predict paranoia over the course of several months (Fowler *et al.* 2012; Oliver *et al.* 2012). However, the only study that investigated the role of negative affect as a mediator of the longitudinal association between negative-self schema and positive symptoms (Oliver et al. 2012) used a design with two assessment points (T1, T2) in university students from the UK and New Zealand. They found a unidirectional association between NS (T1) and delusional ideation (T2), which was fully mediated by anxiety (T2), but not by depression. Although the findings are promising, it needs noting that the design was not ideally suited to establish whether changes in negative affect follow NS and precede increases in symptoms, as a rigorous test of this assumption requires at least three assessment points.

Furthermore, most of the accumulated evidence on the association between NS and positive symptoms is based on narrow single subpopulations of, people from white,

educated, industrialized, rich, democratic countries, which may not be generalizable to other subpopulations (Henrich *et al.* 2010). Because culture is likely to play a role in the way psychotic experiences are appraised and maintained (e.g. Luhrmann *et al.* 2014), investigating the antecedents of symptoms in culturally broader samples is necessary to arrive at generalizable findings.

In the face of the existing state of research we agree with Howes and Murray (2014) who concede that further evidence to support the proposal that schemas are biased *before* the onset of psychosis is needed. Clarifying whether NS and negative affect are predictors of positive symptoms or consequences makes a large difference to the way we understand and treat positive symptoms of psychosis. If the postulated causal pathway from NS to symptoms via affect is confirmed, this implies that a therapeutic focus on self-schema and affect in the pre-morbid phase could be an effective way to prevent the development of the full clinical disorder. In contrast, if negative affect and NS temporally follow the experience of positive symptoms, then the more plausible approach would be to continue to focus on reducing symptoms and enhancing patients' coping abilities.

The present study thus tested the following hypotheses using auto-regressive cross-lagged path analysis with four evenly spaced assessment points across 12 months in large community samples from Germany, Indonesia, and the United States: (1) NS longitudinally predict positive symptoms and (2) the association between NS and symptoms is longitudinally mediated by negative affect.

2. Method

2.1. Participants and Procedure

Participants from Germany, Indonesia, and the United States were recruited through Crowdflower, a crowdsourcing website, and other websites (e.g. internet forums and social

networking websites) to complete an anonymous 30-minute online survey. Only participants who agreed with the consent statements and reported to be above 18 years of age were included. After completing the baseline survey (T0), participants were invited via email to complete a follow-up survey after 4 (T1), 8 (T2), and 12 months (T3). Participants who completed the baseline survey received an invitation to the follow-up surveys. Participants recruited from Crowdflower received 0.50 US$ for completing the baseline survey (T0) following the median hourly wage typical of online recruitment platforms (Buhrmester *et al.* 2011). To motivate participation in follow-ups, the compensation was increased subsequently (T1, 0.60 US$; T2, 0.80 US$; T3, 1.00 US$). Participants recruited from other websites could not be given compensation for reasons of data security. The study received ethical approval from the German Psychological Society.

There were 2501 completed baseline survey entries of which 151 were excluded due to duplicate entries (n = 98), longstring (i.e. providing the same answer consecutively for 50 items, n = 46, see Johnson (2005)), and inconsistent answers (n = 7). The baseline (T0) sample consisted of 2350 participants of whom 720 participants completed the English, 786 the German and 844 the Indonesian version of the survey, 1447 of these participants responded at T1 (response rate = 61.6%), 1244 at T2 (response rate = 52.9%), and 943 at T3 (response rate = 40.1%). The participants who responded to the invitation were then subjected to the same inclusion criteria (i.e. complete entry, no longstring, consistent answers). Based on these criteria, there were 682 participants at T1, 409 at T2, and 366 at T3, of which 135 participants completed all follow-up assessments. A detailed participant flowchart following the STROBE (STrengthening the Reporting of OBservational studies in Epidemiology) guideline (Vandenbroucke *et al.* 2007) is available in the supplementary (Supplementary Figure 1). For the current analysis, we included participants who participated

in at least one follow-up survey (n = 962) of whom 302 participants completed the English, 312 the German and 348 the Indonesian version of the survey.[1]

2.2. Measures

Back-translation procedure and cultural adaption of measures were conducted by native Germans, British and Indonesians according to a guideline (Schmitt & Eid 2007). A complete description of the scale and scoring procedure is available from the corresponding author.

NS were measured with the negative-self subscale from the Brief Core Schema Scale (BCSS, Fowler *et al.* 2006). The subscale comprised six items (e.g. I am unloved) rated on a 5-point Likert scale from "no, do not believe it" to "yes, believe it totally". Good validity and reliability of the scale have been reported (Fowler *et al.* 2006).

Anxiety was measured with the seven-item Generalized Anxiety Disorder-7 scale (GAD-7, Spitzer et al., 2006) that includes items such as "Feeling nervous, anxious or on edge". Depression was measured with the nine-item Patient Health Questionnaire-9 (PHQ-9) that is based on the DSM-IV criteria for depression (Kroenke *et al.* 2001) with items such as "Feeling down, depressed, or hopeless". For both scales the instruction was as follows: "Over the last 4 weeks, how often have you been bothered by any of the following problems?" on a 4-point Likert scale from "not at all" to "nearly every day". Both scales have been shown to have good reliability and validity in large representative population samples (Martin *et al.* 2006; Löwe *et al.* 2008). Negative affect was defined as the combined sum-score of the PHQ-9 and GAD-7.

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[1] Part of the data set has been used to study the mediation of emotion regulation in the association between childhood trauma and psychotic experiences (Lincoln *et al.* 2017).

Positive symptoms were measured with the 20-item positive symptom subscale from the Community Assessment of Psychic Experiences (CAPE, Stefanis *et al.* 2002), which assesses a range of symptoms, such as paranoia (e.g. "Have you ever felt that you were being persecuted in any way?") and hallucinations (e.g. "Do you ever hear voices when you are alone?"). The items were answered on a 4-point Likert scale from "never" to "nearly always" to measure the frequency of the occurrence of symptoms and from "not distressed" to "very distressed" to measure the appraisal of symptoms. The frequency and distress scales were combined into a weighted score reflecting the severity of each symptom. This was done by multiplying an item´s frequency score with its associated distress score. For example, if an individual reported that they "sometimes" heard voices (frequency score of 2) and were "quite distressed" by this experience (distress score of 3), they would receive a severity score of 6. The final score was the sum of severity scores. The German and English versions of the CAPE demonstrate good validity and reliability (Brenner *et al.* 2007; Schlier *et al.* 2015).

2.3. Analyses

We examined the temporal relationship between NS, negative affect and positive symptoms using cross-lagged panel models. Cross-lagged panel models are ideal for examining temporal associations between two variables, because they take various sources of error, such as the stability of the variables, cross-sectional associations and prior associations into account.

Before computing the cross-lagged panel models, we tested two preconditions: (1) a saturated model that assumes all variables are significantly inter-correlated cross-sectionally and across all assessment points; (2) a within-construct longitudinal model to estimate the extent to which past scores on a variable predict temporally following scores on that variable (e.g. NS at T0 predict NS at T1).

Before computing the longitudinal mediation model, three preconditions for mediation derived from Baron and Kenny's (1986) causal steps approach were established, using cross-lagged panel models: (1) NS longitudinally predict positive symptoms (path c, see Figure 1a); (2) NS longitudinally predict negative affect (path a, see Figure 1b); (3) negative affect longitudinally predicts positive symptoms (path b, see Figure 1c). In each step, we also tested for the possibility of reverse pathways.

The longitudinal mediation analysis followed the suggestions by Preacher (2015) in constructing the appropriate cross-lagged panel models (see Figure 2, for the full statistical model, see Supplementary Figure 2). Moreover, following Mayer *et al.* (2014) we used indirect effect size estimates to confirm whether a variable is a mediator. Because we did not have time-specific hypotheses regarding the temporal mediation pathways (e.g. that the mediation via negative affect only occurs within the first eight months), we computed and reported all possible indirect pathways and their respective effect size coefficients. The indirect effect coefficients were considered significant if the bias-corrected bootstrap 95% confidence interval (BCa CI) did not include zero (Cheung & Lau 2008).

All of the analyses were conducted with structural equation modeling (SEM) using the lavaan package ver. 0.5-22 (Rosseel 2012) in R version 3.2.3. Reported path coefficients (γ *and* β) are completely standardized. Reported overall total effect, overall direct effect, and overall indirect effect coefficients are unstandardized. The following fit indices along with the proposed cut-off criteria were used to assess the fit between hypothesized models and the data: CFI > 0.95, RMSEA < 0.06, and SRMR < 0.08 (Hu & Bentler 1999). The χ^2 is reported but not used as a fit criterion because it tends to reject models that are based on large sample size (Bentler & Bonett 1980). All analyses were estimated using maximum likelihood procedure with standard errors based on the first-order derivatives.

Little's missing completely at random (MCAR) test indicated that NS, negative affect, and positive symptoms across measurement time points was MCAR (χ^2 (45) = 54.18, p = .16). However, the MCAR assumption was not supported when we looked at the correlations of various variables of interest with missing status. Specifically, participation in a follow-up survey was significantly associated with being female, older age, higher socio-economic status, absence of a mental health diagnosis, higher NS (T2), as well as more severe positive symptoms (T0 and T1, see Supplementary Table 1). Consequently, the more conservative full information maximum likelihood (FIML) with missing at random (MAR) assumption was used.

3. Results

3.1. Participant Characteristics

The participants were 34.4 years old on average and 45.6% were female. Generally, the participants tended to have a somewhat higher education, but a lower income in comparison to their national averages (see Supplementary Text). The employment status was diverse with 31.8% workers, 23.1% office employees, 14.9% university students, 6.9% self-employed, 6.0% helping family members (e.g. housewife), 5.4% freelancers, 7.6% were unemployed, and 4.3% other. Moreover, 31.2% of the participants reported a life-time mental diagnosis, and 3.3% participants reported to have a current diagnosis of a psychotic disorder. Detailed participant characteristics are provided in Table 1.

3.2. Preconditions for Cross-Lagged Panel Models

First, saturated models showed that NS, negative affect, and positive symptoms were significantly correlated with each other within and across time points (see Supplementary Table 2). Second, within-construct longitudinal models showed that previous measures significantly predicted subsequent measures for all variables of interest (see Supplementary Table 3) indicating that these constructs are stable over time to a certain extent. In addition,

all fit indices of the cross-lagged panel models tested below (section 3.3, 3.4) met at least two out of three goodness of fit criteria (see Table 2), indicating that all parameter estimates can be interpreted.

3.3. Preconditions for Longitudinal Mediation Analysis

First, NS significantly predicted positive symptoms at the following time-points. In contrast, there were no significant reverse pathways from positive symptoms to NS (for details see Figure 1a). Second, NS significantly predicted negative affect at the following time-points. One of the four possible reverse paths from negative affect to NS was also significant indicating reverse causation (for details see Figure 1b). Third, we found that negative affect significantly predicted positive symptoms at the following time-points. There were no significant reverse pathways from positive symptoms to negative affect (for details see Figure 1c).

3.4. Longitudinal mediation

The longitudinal mediation model is shown in Figure 2. As apparent in Figure 2, there are three possible indirect pathways from NS at T0 via negative affect to positive symptoms at T3, of which one was significant, one was trend-level significant and one was non-significant. First, the indirect effect consisting of the pathway from NS at T0 to negative affect at T1, then from negative affect at T1 to negative affect at T2, and finally from negative affect at T2 to positive symptoms at T3 was significant (0.020, $p < 0.05$, 95% BCa CI, 0.004, 0.035). Second, the indirect effect consisting of pathways from NS at T0 to NS at T1, then from NS at T1 to negative affect at T2, and finally from negative affect at T2 to positive symptoms at T3 was trend-level significant (0.021, $p = 0.06$, 95% BCa CI, -0.001, 0.042). Third, the indirect effect consisting of pathways from NS at T0 to negative affect at T1, then from negative affect at T1 to positive symptoms at T2, and finally from positive symptoms at

T2 to positive symptoms at T3 was not significant (-0.001, $p = 0.145$, 95% BCa CI, -0.022, 0.003).

4. Discussion

Informed by cognitive models of psychosis (particularly Garety *et al.* 2001) this study set out to investigate the postulate that NS precede symptoms and that negative affect mediates this association over the course on one year in a large multi-national sample. As hypothesized, we found a unidirectional, longitudinal pathway from NS to positive symptoms. Together with previous work that provides support for a unidirectional pathway from NS to paranoia (Fowler *et al.* 2012; Oliver *et al.* 2012), we can now more confidently affirm the causal role of NS in the formation and maintenance of positive symptoms as postulated by the cognitive models (e.g. Garety *et al.* 2001) and their integrative extensions (Howes & Murray 2014). Moreover, we could ascertain that this link is uni-directional as we found no significant reverse pathways from positive symptoms to NS. Finally, we confirmed one of the cognitive models' core assumptions, namely that negative affect longitudinally mediates the path from NS to positive symptoms.

Interestingly, the bidirectional pathways in the independent cross-lagged panel models suggest that NS and negative affect enhance each other over time, forming a kind of vicious cycle. Moreover, our findings indicate that it takes a certain amount of time (about eight months in our study) before this build-up of negative affect and NS leads on to psychotic symptoms. This observation is in line with the clinical observation that patients with psychosis often present with problems related to anxiety or depression first, before developing psychotic disorders at a later time-point (Häfner *et al.* 2013). For example, in the Dutch Early Detection and Intervention Evaluation (EDIE-NL) trial more than 50% of the identified prodromal schizophrenia patients had a diagnosis of an anxiety disorder, depressive disorder, or both (van der Gaag *et al.* 2012a).

Moreover, our findings suggest that NS and negative affect precede – and are thus likely to be causal to – positive symptoms rather than vice versa. Although previous cognitive models have often postulated a vicious cycle relationship between NS, negative affect and positive symptoms by claiming that the same factors that precede symptoms are responsible for maintaining them (e.g. Garety *et al.* 2007; Kesting & Lincoln 2013) neither our study nor other studies (Fowler *et al.* 2012; Oliver *et al.* 2012) support the notion that positive symptoms are followed by increased NS or more negative affect. Thus, it appears that NS make people vulnerable to having positive symptoms, but that positive symptoms do not necessarily cause people to feel worse or to see themselves in a more negative way. This does not exclude the possibility that longer-term consequences of positive symptoms (e.g. being hospitalized, stigmatized, socially excluded etc.) will have a detrimental effect on affect and self-schemas, but this would require studies looking at even longer time-periods and selecting a sample that scores on the more severe end of the psychosis continuum. This type of research could also be combined with a PET methodology to investigate the maintenance mechanism as postulated by Howes and Murray (2014), namely that paranoia and hallucinations cause further stress, leading to the repeated dopamine dysregulation which hardwires the psychotic beliefs.

In this context, we would also like to use our findings to spark a discussion on the consideration of time in cognitive models of psychosis. Psychological theories in general have been criticized to lack consideration of time (Cole & Maxwell 2003), and this shortcoming has only been recently addressed in the context of psychosis. Putative psychological mechanisms of psychosis may take place at a distal macrolevel and/or at a more proximal microlevel (Reininghaus *et al.* 2015). For example, brief setbacks in self-esteem have been shown to trigger an immediate increase in paranoid beliefs at a micro-level in experimental (Kesting *et al.* 2013) and in experience sampling studies (Thewissen *et al.*

2011). Similarly, negative emotional states have been shown to play an important role in the development of psychosis at the microlevel, in the sense that increases in negative emotions tend to be followed by proximal increases on state symptom measures (Lincoln *et al.* 2010; Kramer *et al.* 2013). However, these studies do not investigate how NS and negative affect influence symptoms over longer time-periods. Our findings, in contrast, focus on the macro-level and indicate NS and negative affective states to build up over a period of months before they have a meaningful impact on positive symptoms. Thus, it seems that in the case of negative affect and NS both macro-and micro levels mechanisms could be involved and it would be interesting to further pursue this question in future studies.

4.1. Strengths and Limitations

A strength of the study is that the participants covered different regions and cultures. Moreover, they covered the whole psychosis continuum including the severe end as indicated by a relatively high prevalence of individuals with a diagnosis of a psychotic disorder (3.3%). Looking at associations across the continuum is advantageous when it comes to investigating putative mechanisms, because the findings can be interpreted free of issues that often influence studies comparing clinical to non-clinical populations, such as selective small samples, stigma, and medication. Another strength is the careful consideration of the temporal aspect of measurement and assessment points. The specific and uniform reference to the past four weeks in all measurements and assessment points along with the evenly spaced assessment points allowed us to investigate temporal pathways between NS, negative affect and psychotic symptoms repeatedly under uniform conditions.

A limitation is that the recruiting method limited participants to those with access to the internet. Furthermore, although we oversampled participants with schizophrenia spectrum disorders to have sufficient variation in symptoms, the rate of overall psychopathology in our sample was comparable to typical samples reported in epidemiological studies. For example,

29% of the participants in the study reported a life-time mental disorder diagnosis, which is similar to the rates presented in representative population samples (Kessler *et al.* 2005).

Another aspect worthy of discussion is that cross-lagged panel analysis has been noted to overestimate the stability coefficients of constructs and to underestimate the cross-lagged paths (Cole & Maxwell 2003). This means that the coefficients of the main results (e.g. from negative affect to positive symptoms) presented are conservative and might be underestimated. This could also be the reason why not all of the possible mediation paths were significant. Moreover, our analysis of missing data pattern showed a tendency of participants with severe symptoms to drop out at follow-up. We attempted to correct this by estimating the missing data using FIML procedure, but can nevertheless not rule out an underestimation of all lagged pathways due to a reduction of follow-up data variability.

4.2. Conclusion

Our study design allows us to conclude that NS precede symptoms and that this link is mediated by negative affect. This finding confirms a core assumption of cognitive models of psychosis and refines them further by showing that the mechanism applies uni-directionally and in the time-period of months. Our results suggest that interventions that target NS and negative affect could have a buffering effect, reducing and preventing positive symptoms.

The value of intervening at the mediating mechanism, negative affect, as a strategy to reduce positive symptoms has been demonstrated in a couple of pioneering trials that target depression and anxiety in patients with psychotic disorders. The most convincingly designed among these studies compared a brief intervention targeting worry to standard care in a sample of 150 patients with persecutory delusions and was able to demonstrate that focusing solely on worry was successful in reducing paranoid ideation (Freeman *et al.* 2015). In fact, the results of the mediation analysis in Freeman's study showed that 66% of the intervention's effect on delusions was explicable by reductions in worry. Studies focusing on

depression (van der Gaag *et al.* 2012b; Moritz *et al.* 2016) have provided more mixed findings, showing improvements in mood but not in psychotic symptoms. However, this might be due to a lack of study power or the fact that the intervention was delivered online. Further adequately designed research is thus required in order to test which affective processes should be targeted and how to have a sustainable effect on positive symptoms. Another important therapeutic implication could be to target NS in order to prevent or reduce positive symptoms. Again, there is a pioneering work by Freeman et al (2014) investigating the impact of an intervention aimed to improve self-related beliefs in patients with persecutory delusions. However, they did not find a significant effect of the intervention, both on NS and paranoia at a 1 month follow-up assessment, which the authors discussed as being due to the brevity of the intervention (6 sessions in 8 weeks). Thus, developing interventions that are successful in improve self-schemas and testing their impact on positive symptoms remains a challenge for future research.

Given that we found the associations of NS, affect and symptoms across the entire continuum, focusing on NS and negative affect as a strategy to prevent symptom formation before the development of a full psychotic disorder could be particularly promising. This could be done by screening for risk-indicators at the level of schools, universities or other institutions and offering low-threshold interventions that focus on improving self-schemas and reducing anxiety and depression to those who show elevated levels of negative affect or NS. This type of primary prevention might help to prevent progression to an at risk status or from the psychosis prodrome to the full disorder.

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Conflict of interest

None.

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Figures

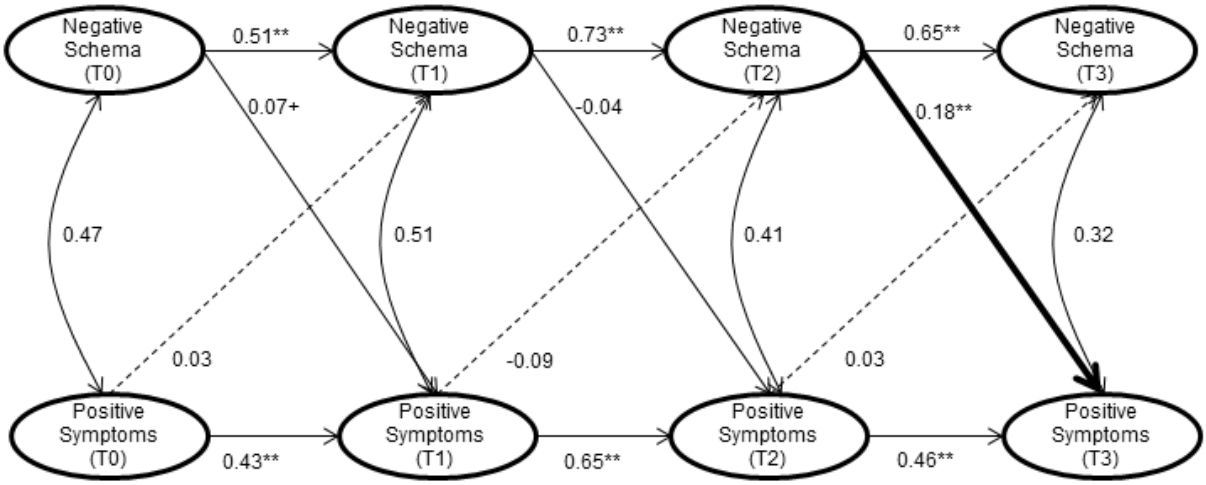
Figure 1. Prospective associations of negative schemas, negative affect, and positive symptoms.

Note. Dotted arrows indicate reverse pathways. Non-dotted arrows indicate hypothesized pathways. Bolded arrows indicate significant cross-lagged pathways. Bi-directional arrows indicate association. Uni-directional arrows indicate prediction. Path coefficients are completely standardized. Negative schema = Negative-self schema. T0 = baseline. T1 = month 4. T2 = month 8. T3 = month 12. $^{+}$ $p < 0.10$, * $p < 0.05$, ** $p < 0.01$.

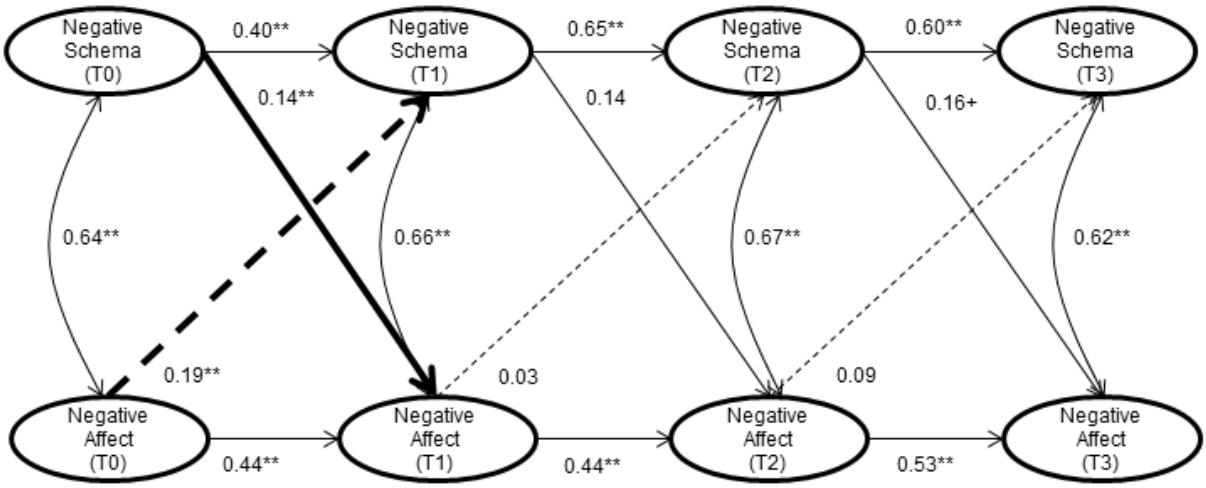
Figure 2. Longitudinal mediation analysis of negative schemas, negative affect, and positive symptoms.

Note. All cross-sectional associations are statistically significant at $p < 0.05$, only significant cross-lagged paths coefficients are reported. Dotted arrows indicate non-significant paths. Path coefficients are completely standardized. Negative schema = Negative-self schema. T0 = baseline. T1 = month 4. T2 = month 8. T3 = month 12. $^{n.s.}$ = not significant. ** $p < 0.01$.

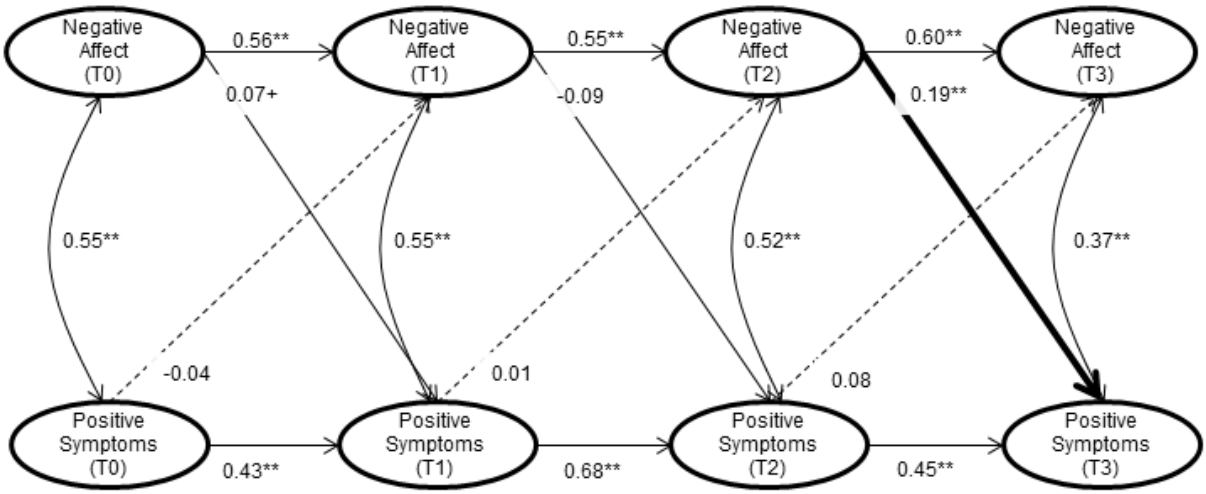
a) Cross-Lagged Panel Model of Negative-Self Schema and Positive Symptoms: Test of Path *c*



b) Cross-Lagged Panel Model of Negative-Self Schema and Negative Affect: Test of Path *a*



c) Cross-Lagged Panel Model of Negative Affect and Positive Symptoms: Test of Path *b*



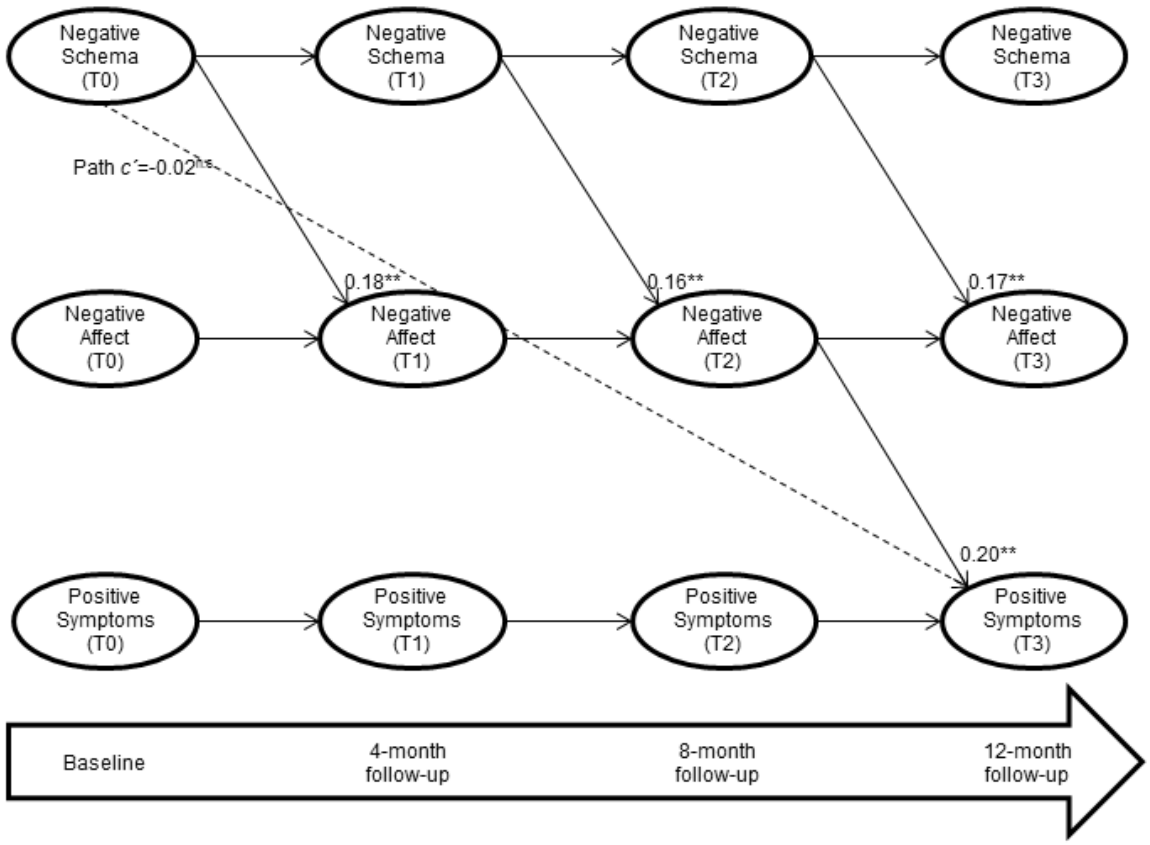

Negative Schema (T0)
Negative Schema (T1)
Negative Schema (T2)
Negative Schema (T3)
Path c´=-0.02ns
0.18**
0.16**
0.17**
Negative Affect (T0)
Negative Affect (T1)
Negative Affect (T2)
Negative Affect (T3)
0.20**
Positive Symptoms (T0)
Positive Symptoms (T1)
Positive Symptoms (T2)
Positive Symptoms (T3)
Baseline
4-month follow-up
8-month follow-up
12-month follow-up

Table 1

Participant characteristics (N = 962)

Characteristic	Mean	SD	Sample Range	Possible Range
Gender, Female (%)	439 (45.6%)	-	-	-
Age	34.41	11.85	18-74	18 - 99
T0 Negative-self schema	1.80	0.90	1.00-5.00	1-5
T0 Negative affect	1.85	0.64	1.00-4.00	1-4
T0 Positive symptoms	2.23	1.47	1.00-16.00	1-16
T1 Negative-self schema	1.81	0.93	1.00-5.00	1-5
T1 Negative affect	1.78	0.67	1.00-4.00	1-4
T1 Positive symptoms	2.11	1.27	1.00-9.35	1-16
T2 Negative-self schema	1.82	0.63	1.00-5.00	1-5
T2 Negative affect	1.78	0.62	1.00-3.89	1-4
T2 Positive symptoms	1.91	1.06	1.00-8.80	1-16
T3 Negative-self schema	1.81	0.95	1.00-5.00	1-5
T3 Negative affect	1.80	0.62	1.00-3.86	1-4
T3 Positive symptoms	1.93	1.23	1.00-14.55	1-16

Note. T0 n = 962, T1 n = 682, T2 n = 409, T3 n = 366.

Table 2

Fit indices of the tested models

Model	χ^2	χ^2 *df*	*p*	RMSEA [90% CI]	SRMR	CFI	AIC[a]
Cross-lagged panel model of negative-self schema on positive symptoms	88	12	< 0.01	0.081 [0.066, 0.097]	0.060	0.951	13 130
Cross-lagged panel model of negative-self schema on negative affect	50	12	< 0.01	0.057 [0.041, 0.074]	0.052	0.984	8 723
Cross-lagged panel model of negative affect on positive symptoms	91	12	< 0.01	0.083 [0.067, 0.099]	0.069	0.953	11 171
Longitudinal mediation model of negative-self schema, negative affect, and positive symptoms	170	38	< 0.01	0.060 [0.051, 0.069]	0.075	0.965	15 661

Note. RMSEA = Root Mean Square Error of Approximation. SRMR = Standardized Root Mean Square Residual. CFI = Comparative Fit Index. AIC = Akaike Information Criterion. 90%CI = 90% Confidence Interval. [a] rounded to the next integer.

Appendix F. Curriculum Vitae

Curriculum Vitae

PERSONAL INFORMATION

Jaya, Edo Sebastian
ORCID http://orcid.org/0000-0002-7232-6312
URL: https://www.psy.uni-hamburg.de/arbeitsbereiche/klinische-psychologie-und-psychotherapie/personen/jaya-edo.html
BLOG: https://edojaya.com/
TWITTER: https://twitter.com/edoyap
RESEARCH GATE: https://www.researchgate.net/profile/Edo_Jaya

ACADEMIC RECORD

EDUCATION

2013 - current	PhD (Dr. phil) on 'The Interplay between Social Factors and Psychotic Symptoms: Cognitive Vulnerability and Affective Pathway in Focus' under the supervision of Prof. Tania M. Lincoln, Clinical Psychology and Psychotherapy, Institute of Psychology, University of Hamburg, Germany
2010-2012	Master in Clinical Psychology (M.Psi., Psikolog) on ´Group Multi-component Cognitive Behavior Therapy (CBT) for Older Adults with Insomnia in Depok´ under the supervision of Dr. Dharmayati B. Utoyo, Faculty of Psychology, University of Indonesia, Indonesia
2006-2010	Bachelor in Psychology (S.Psi.) on ´The relationship between Leader-Member Exchange and Organizational Citizenship Behavior´ under the supervision of Dr. Wustari L. Mangundjaya, Faculty of Psychology, University of Indonesia, Indonesia
2005-2006	High school Bunda Hati Kudus, Jakarta, Indonesia
2004-2005	High school Homebush Boys High School, Sydney, Australia
2000-2004	High school De La Salle College Ashfield, Sydney, Australia

CURRENT AND PREVIOUS POSITION(S)

2013 - current	PhD student, Clinical Psychology and Psychotherapy, Institute of Psychology, University of Hamburg, Hamburg, Germany
2012 - 2013	Associate researcher, Centre of Ageing Studies, University of Indonesia, Depok, Indonesia
	Adjunct lecturer, Faculty of Psychology, Tarumanagara University, Jakarta, Indonesia
	Associate researcher, Society, Technology, and Science Research Group, Tarumanagara University, Jakarta, Indonesia

THESIS SUPERVISION

2013 - current	1 master thesis and 1 bachelor thesis,

Clinical Psychology and Psychotherapy, Institute of Psychology, University of Hamburg, Hamburg, Germany

2012 - 2013 Co-supervision of 2 bachelor theses, Tarumanagara University, Jakarta, Indonesia

TEACHING ACTIVITIES

2012 - 2013 Adjunct lecturer – Advanced Statistics (Master class, 3 credits at 150 minutes/week), SPSS (Bachelor class, 2 credits at 100 minutes/week), Psychological Measurement (Bachelor class, 3 credits at 150 minutes/week)
Faculty of Psychology, Tarumanagara University, Indonesia

2012 - 2013 Adjunct lecturer – Advanced Statistics (Bachelor class, 3 credits at 150 minutes/week), Experimental Psychology (Bachelor class, 2 credits at 100 minutes/week)
Faculty of Psychology, Atma Jaya Catholic University, Indonesia

REVIEW ACTIVITIES

2016 Referee/Reviewer for:
European Psychiatry (IF = 3.91),
Psychiatry Research (IF = 2.47),
Schizophrenia Bulletin (IF = 7.76),
Schizophrenia Research (IF = 4.45)

Note. I have so far reviewed six articles.

RESEARCH RECORD

FUNDING OBTAINED

2013 - current The influence of social factors on psychosis.
Research Grants for Doctoral Candidates and Young Academics and Scientists. Deutscher Akademischer Austauschdienst (DAAD, German Academic Exchange Service), Bonn, Germany. Grant number: 91540971

2012 - 2013

1. Developing Indigenous Stress-Management Intervention for Indonesian Older Adults
Intermediate Research Grant from Kementerian Riset Teknologi dan Pendidikan Tinggi RI (Hibah Riset Madya DIKTI-UI, Indonesian Ministry for Research, Technology, and Higher Education), Jakarta, Indonesia. Grant of Rp 100.000.000 (equivalent to US $10,000). Principal Investigators: Dini P. Daengsari, Dharmayati B. Utoyo, **Edo S. Jaya**, and Retha Arjadi.

2. Developing Psychosocial Intervention for Mild Cognitive Impairment in Indonesian Older Adults
Intermediate Research Grant from the Directorate of Research and Community Engagement of University of Indonesia (Hibah Riset Madya DRPM-UI), Depok, Indonesia. Grant of Rp 100.000.000 (equivalent to US $10,000). Principal Investigators: Dharmayati B. Utoyo, **Edo S. Jaya**, Retha Arjadi, Agnes Utari.

3. Developing Thinking Skills Training through Digital Technology
Research Grant from Tarumanagara University, Jakarta, Indonesia.

Grant of Rp 25.000.000 (equivalent to US $2,500). Principal Investigators: Sri Tiatri, Jap Tji Beng, **Edo S. Jaya**, Retha Arjadi.

4. Conducting Public Mental Health Awareness Campaign to Older Adults
   Community Engagement Grant from the Directorate of Research and Community Engagement of University of Indonesia (Hibah IPTEKS bagi Masyarakat DRPM-UI), Depok, Indonesia. Grant of Rp 50.000.000 (equivalent to US $5,000). Principal Investigators: Dharmayati B. Utoyo, **Edo S. Jaya**, Retha Arjadi, Agnes Utari.

2011 - 2012 Applying Group Multi-Component Cognitive Behavioral Therapy to Increase the Well-Being of Older Adults in Depok
Community Engagement Grant from The Directorate of Research and Community Engagement of University of Indonesia (Hibah IPTEKS bagi Masyarakat DRPM-UI), Depok, Indonesia. Grant of Rp 50.000.000 (equivalent to US $5,000). Principal Investigators: Dharmayati B. Utoyo, **Edo S. Jaya**, Retha Arjadi, Lathifah Hanum, Maha D. D. Putri, Kresna Astri.

PUBLICATIONS

In progress (*corresponding author)

1. **Jaya, E. S.***, Ascone, L., & Lincoln, T. M. (submitted). A longitudinal mediation analysis of the effect of negative-self schema on positive symptoms via negative affect.
2. Gollwitzer, A., Marshall, J., Wilczynska, M., & **Jaya, E. S.** (submitted). Starving psychosis: Applying the interventionist-causal model to reducing the effect of loneliness on psychosis.
3. Pillny, M., **Jaya, E. S.,** Riehle, M., & Lincoln, T. M. (in progress). Negative symptom network in schizophrenia and healthy individuals.
4. Riehle, M.$^{+}$, **Jaya, E. S.**$^{+}$**,** & Lincoln, T. M. (in progress). The vicious cycle of social exclusion and negative symptom. ($^{+}$equal first author)

Refereed journals (*corresponding author)

1. Lincoln, T. M., Marin, N., & **Jaya, E. S.*** (2017). Childhood trauma and psychotic experiences in a general population sample: A prospective study on the mediating role of emotion regulation. *European Psychiatry* (IF = 3.91).
2. Hennig, T., **Jaya, E. S**., & Lincoln, T. M. (2016). Bullying mediates between attention-deficit/hyperactivity disorder in childhood and psychotic experiences in early adolescence. *Schizophrenia Bulletin* (IF = 7.76, 2015). https://doi.org/10.1093/schbul/sbw139
3. Hennig, T., **Jaya, E. S.,** Koglin, U., & Lincoln, T. M. (2016). Associations of attention-deficit/hyperactivity and other childhood disorders with psychotic experiences and disorders in adolescence. *European Child & Adolescent Psychiatry* (IF = 3.34, 2015). doi: 10.1007/s00787-016-0904-8
4. **Jaya, E. S.*** & Lincoln, T. M. (2016). Social adversities and psychotic symptoms: A test of predictions derived from the social defeat hypothesis. *Psychiatry Research* (IF = 2.47, 2015), 245, 466-472. doi: 10.1016/j.psychres.2016.09.002
5. **Jaya, E. S.*,** Hillmann, T., Klaus, M. R., Gollwitzer, A., & Lincoln, T. M. (2016). Loneliness and psychotic symptoms: The mediating role of depression. *Cognitive Therapy and Research*

(IF = 2.03, 2015). doi: 10.1007/s10608-016-9799-4
6. L. Ascone, **Jaya, E. S.***, & Lincoln, T. M. (2016). The effect of unfavourable and favourable social comparisons on paranoid ideation: An experimental study. *Journal of Behavior Therapy and Experimental Psychiatry* (IF = 2.19, 2015). doi: 10.1016/j.jbtep.2016.08.002.
7. **Jaya, E. S.***, Ascone, L., & Lincoln, T. M. (2016). Social adversity and psychosis: The mediating role of cognitive vulnerability. *Schizophrenia Bulletin* (IF = 7.76, 2015). doi: 10.1093/schbul/sbw104
8. Schlier, B.,[+] **Jaya, E. S.[+],** Moritz, S., & Lincoln, T. M. (2015). The Community Assessment of Psychic Experiences measures nine clusters of psychosis-like experiences: A validation of the German version of the CAPE. *Schizophrenia Research* (IF = 4.45, 2015), 169(1–3), 274–279. doi: 10.1016/j.schres.2015.10.034 ([+]equal first author)
9. Jap, T., Tiatri, S., **Jaya, E. S.***, & Suteja, M. S. (2013). The development of Indonesian Online Game Addiction Questionnaire, *PLoS One* (IF = 3.23, 2015), 8(4): e61098. doi:10.1371/journal.pone.0061098
10. Utoyo, D. B., **Jaya, E. S.***, Arjadi, R., Hanum, L., Astri, K., Putri, M. D. D. (2013). Preliminary study on the effectiveness of short group Cognitive Behavioral Therapy (GCBT) on Indonesian older adults, *PLoS One* (IF = 3.23, 2015)*,* 8(2): e57198. doi:10.1371/journal.pone.0057198
11. Oktorina, K., **Jaya, E. S.**, Jap, T., & Tiatri, S. (2010). The relationship of school protective factor, especially 'high expectation', to school students' online game habit. Jurnal Penelitian dan Evaluasi Pendidikan, 3. [in Bahasa Indonesia]
12. **Jaya, E. S.**, Hartana, G. T. B., & Mangundjaya, W. G. (2010). Detecting Social Desirability (SD) in behavioral research variable. Jurnal Psikologi Indonesia, 7 (1). [in Bahasa Indonesia]

Proceedings

1. **Jaya, E. S.,** & Mangundjaya, W. L. (2013). Relationship between Leader-Member Exchanges with Organizational Citizenship Behaviour. In Y. Kashima, E. Kashima, & R. Beatson (Eds.), Steering the cultural dynamics: Selected papers from the 2010 Congress of the International Association for Cross-Cultural Psychology. Melbourne, Australia: International Association for Cross-Cultural Psychology. Retrieved from http://www.iaccp.org/drupal/sites/default/files/melbourne_pdf/Jaya.pdf
2. **Jaya, E. S.** (2012). Relationship of education level to psychological well-being in Indonesian elderly. *Proceedings of Seminar Nasional Universitas Paramadina*, 6 September 2012, Jakarta. [in Bahasa Indonesia]
3. **Jaya, E. S.**, Hanum, L., & Lubis, D. U. (2011). Indigenous psychological well-being for the elderly measurement. In U. Kim, Supriyadi, D. H. Tobing, L. K. P. A. Susilowati, A. A. S. S. Dewi, & I P. G. Darma (Eds.) *Proceedings of the 2nd Indigenous and Cultural Psychology Conference* (pp. 187-200). Denpasar, Indonesia: Udayana University Press.

Selected presentations and posters in conferences

1. **Jaya, E. S.,** Ascone, L., & Lincoln, T. M. (2016). Sozial benachteiligt und psychisch krank? Nicht unbedingt! Die vermittelnde Rolle der kognitiven Vulnerabilität bei psychotischen Symptomen. Poster Presentation in 34. Symposium der Fachgruppe Klinische Psychologie und Psychotherapie der DGPs at Bielefeld, Germany. doi: 10.13140/RG.2.1.3725.7201
2. **Jaya, E. S.,** & Lincoln, T. M. (2015). The relationship between loneliness and positive symptoms in schizophrenia is mediated by depression: insights from a network analysis. Poster Presentation in 33. Symposium der Fachgruppe Klinische Psychologie und Psychotherapie der DGPs at Dresden, Germany. doi: 10.13140/RG.2.1.3985.5449 *Nominated for best poster
3. **Jaya, E. S.,** & Lincoln, T. M. (2015). Testing the social defeat hypothesis. Poster Presentation in 5th European Conference on Schizophrenia Research at Berlin, Germany. doi: 10.13140/RG.2.1.4771.9766

4. **Jaya, E. S.** (2014). Investigation in the validity and reliability of the Indonesian UCLA Loneliness scale. Poster Presentation in 22nd Congress of International Association of Cross-Cultural Psychology (IACCP), Reims, France.
5. **Jaya, E. S.** (2012). Multicomponent cognitive behavior therapy on insomnia for Indonesian elderly. Oral Presentation in 4th Asian Psychological Association, 5-7 July 2012, Jakarta.
6. **Jaya, E. S.**, Tiatri, S., Jap, T., & Halim, W. (2012). Online game effect on perception of violence, sexuality, and delinquency behaviors: study from Salatiga and Jakarta school students. Oral Presentation in 4th Asian Psychological Association, 5-7 July 2012, Jakarta.
7. **Jaya, E. S.** & Mangundjaya, W. L. (2010). Relationship between Leader-Member Exchange (LMX) with Organizational Citizenship Behavior (OCB). Oral Presentation in XXth Congress of International Association of Cross-Cultural Psychology (IACCP), Melbourne, Australia.

PROFESSIONAL ASSOCIATIONS

2015 – current	Member of Komunitas Peduli Skizofrenia Indonesia (KPSI, the Indonesian Community Care for Schizophrenia)
2012 - current	Licensed Clinical Psychologist in Indonesia
2011 – current	Member of International Association of Cross-Cultural Psychology (IACCP)
2011 - current	Member of Himpunan Psikologi Indonesia (HIMPSI, Indonesian Psychological Association)

OTHER SKILLS

Languages	Indonesian: Native language English: Fluent (speaking, reading, writing) German: Basic (speaking, reading, writing)
Advance statistics	Structural equation modelling, confirmatory factor analysis, multilevel analysis, mediation analysis, longitudinal analysis, network analysis

Declarations

Universität Hamburg
DER FORSCHUNG | DER LEHRE | DER BILDUNG

Fakultät für
Psychologie und
Bewegungswissenschaft

Institut für Bewegungswissenschaft
Institut für Psychologie

Erklärung gemäß *(bitte Zutreffendes ankreuzen)*

☐ **§ 4 (1c) der Promotionsordnung**
des Instituts für Bewegungswissenschaft der Universität Hamburg vom 18.08.2010

☐ **§ 5 (4d) der Promotionsordnung**
des Instituts für Psychologie der Universität Hamburg vom 20.08.2003

Hiermit erkläre ich,

_____ (Vorname, Nachname),

dass ich mich an einer anderen Universität oder Fakultät noch keiner Doktorprüfung unterzogen oder mich um Zulassung zu einer Doktorprüfung bemüht habe.

_____ _____
Ort, Datum Unterschrift

Studien- und Prüfungsbüro Bewegungswissenschaft • Fakultät PB • Universität Hamburg • Mollerstraße 10 • 20148 Hamburg
Studien- und Prüfungsbüro Psychologie • Fakultät PB • Universität Hamburg • Von-Melle-Park 5 • 20146 Hamburg
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Psychologie und
Bewegungswissenschaft

Institut für Bewegungswissenschaft
Institut für Psychologie

Eidesstattliche Erklärung nach *(bitte Zutreffendes ankreuzen)*

☐ **§ 7 (4) der Promotionsordnung des Instituts für Bewegungswissenschaft der Universität Hamburg vom 18.08.2010**

☐ **§ 9 (1c und 1d) der Promotionsordnung des Instituts für Psychologie der Universität Hamburg vom 20.08.2003**

Hiermit erkläre ich an Eides statt,

1. dass die von mir vorgelegte Dissertation nicht Gegenstand eines anderen Prüfungsverfahrens gewesen oder in einem solchen Verfahren als ungenügend beurteilt worden ist.
2. dass ich die von mir vorgelegte Dissertation selbst verfasst, keine anderen als die angegebenen Quellen und Hilfsmittel benutzt und keine kommerzielle Promotionsberatung in Anspruch genommen habe. Die wörtlich oder inhaltlich übernommenen Stellen habe ich als solche kenntlich gemacht.

Ort, Datum

Unterschrift