A dimensional outlook on clinical high-risk of psychosis

Reframing community data on CHR-P and its transdiagnostic correlates

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Abstract

The clinical high-risk for psychosis (CHR-P) paradigm is an internationally established approach to psychosis prevention and early intervention, developed to address the major public health challenge posed by the severe personal, social, and economic impact of psychosis. Over the last few decades, both clinical and epidemiological research have provided consistent evidence of the psychological and social burden of CHR-P, supporting its association with a highly increased risk of psychosis, general psychopathology, and functional impairment, while identifying concurrent risk factors and etiological mechanisms. In light of the clinical significance of CHR-P, diagnostic and intervention guidelines were developed and specialized programs emerged, showing efficacy in improving general outcomes and delaying psychosis onset. However, CHR-P research faces persistent challenges, including high heterogeneity in symptom presentation and long-term outcomes, declining conversion rates to full-blown psychosis, and a high prevalence of comorbidities. Consequently, it has been proposed that the CHR-P conceptualization be extended beyond its role in psychosis prediction, embracing a broader, more transdiagnostic framing, which has sparked debate in the field. This can be further contextualized within a general shift toward dimensional models in psychopathology, as reflected in recent updates to diagnostic classifications of psychotic and personality disorders in the DSM-5 and ICD-11.

The present thesis illustrates the advantages of a dimensional conceptualization of psychosis, spanning the general population, CHR-P and the full psychosis continuum. Specifically, it explores how integrating CHR-P into the transdiagnostic and dimensional Hierarchical Taxonomy of Psychopathology (HiTOP) may not only address key challenges within the CHR-P paradigm – facilitating phenotyping, risk stratification and consideration of transdiagnostic outcomes – while maintaining a focus on psychosis specificity, but also enrich HiTOP itself by incorporating a stronger developmental and cognitive dimension. To illustrate this, the present work discusses the findings from three publications on CHR-P which, while not originally framed within HiTOP, share its transdiagnostic perspective, lending themselves to a conceptual integration into the model. The three publications are largely based on data from two longitudinal community studies, complemented in one case by clinical data from a specialized early detection service. Specifically, they explore CHR-P symptom trajectories (Publication 1), the interactions of core beliefs and coping strategies with CHR-P symptoms and broader mental health quality (Publication 2), and the associations between personality functioning, cognitive biases, and (non-)perceptive CHR-P symptoms (Publication 3), respectively. Their contextualization within HiTOP informs the proposal of a hypothetical extension to the current HiTOP model, synthesizing and speculatively integrating their findings into its structure while highlighting open questions and future directions for empirical testing. By illustrating the reciprocal benefits of integrating the HiTOP and CHR-P paradigms, this work offers a framework for future efforts to extend the CHR-P conceptualization in alignment with its dimensional, transdiagnostic value, bridging the gap between psychosis risk research and broader psychopathology.

Publications

Publication 1

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1. Introduction

Psychotic disorders represent a critical focus of public health policy due to their profound impact on individuals, their close environments, and society at large (GBD 2019 Mental Disorders Collaborators, 2022; Wittchen et al., 2011). Despite their relatively low lifetime prevalence (0.38% point prevalence, 0.4% 12-month prevalence, and 0.75% lifetime prevalence; Moreno-Küstner et al., 2018), psychotic disorders are a leading cause of disability-adjusted life years in both adults and (pre-)adolescents, with associated direct and indirect costs among the highest of any mental health condition (Collins et al., 2011; GBD 2019 Mental Disorders Collaborators, 2022; Gore et al., 2011; Olesen et al., 2012). A longer duration of untreated psychosis has been consistently associated with worse clinical and functional long-term outcomes, making indicated prevention and early intervention crucial (Charlson et al., 2018; Schmidt et al., 2015; Schultze-Lutter et al., 2015).

Effective prevention and early intervention rely on epidemiological data and accurate etiological conceptualizations (Rose, 2001; Thompson & Broome, 2020). Across diagnoses, the study of risk factors and trajectories is recognized as an intrinsic component of both etiological models and epidemiological research, with great practical implications for treatment, stigma, policy, and research perspectives (Thompson & Broome, 2020). Therefore, over multiple decades, research has extensively explored the prevalence, development and course of psychosis, building explanatory models of the complex biopsychosocial factors and mechanisms involved (Thompson & Broome, 2020).

One of its most fruitful products is the now internationally established clinical high-risk for psychosis (CHR-P) paradigm, which centers on a potentially prodromal state hypothesized to indicate an imminent risk of transition to manifest psychosis (Mei & McGorry, 2020). This paradigm shifted the focus away from the deterministic perspective that initially shaped the scientific exploration of psychosis, wherein its framing as a heritable chronic condition leading to inevitable deterioration and lacking a recovery perspective considerably delayed attempts at indicated prevention, limiting treatment to predominantly palliative care (Kraepelin, 1899; Mei & McGorry, 2020). Although the idea of preventive psychiatry and the description of a psychosis prodrome both date back to the first decades of the 20th century (Bleuler, 1911; Kraepelin, 1919; H. S. Sullivan, 1927), it was not until the 80s and 90s that early psychosis could effectively be reframed as an important window for preventive intervention, leading to the foundation of specialized clinical services for individuals at risk and an exponential growth in research on the early phases of psychosis (Huber & Gross, 1989; McGorry, 1995; Mei & McGorry, 2020).

The CHR-P approach represents the gold standard for early risk detection and indicated prevention of first-episode psychosis, mainly relying on presence, time and severity of two sets of CHR-P symptoms and related criteria (Fusar-Poli, 2017). The benefits of early intervention in CHR-P and psychosis are well-documented, including improved wellbeing, quality of life, and socio-occupational functioning, as well as symptom remission and reduced suicide risk, relapses and inpatient treatment (Correll et al., 2018; Schultze-Lutter & Meisenzahl, 2023; Yung, 2020). However, the CHR-P concept shows some

limitations in its high comorbidity rates with non-psychotic (mainly mood and anxiety) disorders (Solmi et al., 2023) and limited predictive power of its main outcome, i.e., conversion to full-blown psychosis. Specifically, CHR-P symptoms frequently manifest in the general population without ever leading to full-blown psychosis onset, and conversion rates have decreased over the last decades, resulting in debate regarding the specificity of the CHR-P approach for psychosis prediction (McGorry & Hickie, 2019; Tien, 1991; Yung, 2020). On one hand, this does not invalidate its clinical relevance: irrespective of conversion to psychosis or comorbidities, and even after remission of CHR-P symptoms, CHR-P samples are characterized by significant psychological burden and functional impairment (Addington et al., 2019; Lin et al., 2015; Michel, Ruhrmann, et al., 2018; Michel, Schmidt, et al., 2019). Thus, individuals at CHR-P need effective treatment and support, which continue to be effectively informed by ongoing research and clinical advancements within the CHR-P paradigm (Schmidt et al., 2015; Schultze-Lutter & Meisenzahl, 2023). On the other hand, this data points to how its redefinition under a broader, transdiagnostic perspective may be necessary, as its original framework, centering conversion, does not explain the heterogeneity in presentations and outcomes in CHR-P samples (Guloksuz & van Os, 2018).

This proposition can be further contextualized within the ongoing shift towards transdiagnostic, dimensional psychiatric taxonomies (Kotov et al., 2020; Ringwald et al., 2023), as exemplified by the case of personality disorders, for which dimensional – albeit not transdiagnostic – models have been included in both the DSM-5 and the ICD-11 (American Psychiatric Association, 2013; World Health Organization, 2019).

As part of this transformation in our conception of mental illnesses (McGorry et al., 2018), efforts are underway to map CHR-P onto dimensional models (Cowan & Mittal, 2021). In this context, dimensionality also facilitates a better understanding of the heterogeneity in CHR-P manifestations and trajectories, framing CHR-P as a risk state for a psychosis spectrum, rather than specific psychotic disorders (Cowan et al., 2024). This transdiagnostic perspective might additionally help address the current lack of understanding of factors related to different outcomes of CHR-P, which hinders effective sample stratification and the development of tailored treatments and has thus become a central goal to the latest wave of CHR-P research (Yung, 2020). A promising framework unifying these necessities is the Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2021). By conceptualizing psychopathology dimensionally and hierarchically, HiTOP assumes the transdiagnostic value of symptoms while also allowing for the mapping of categorial diagnoses. This makes it particularly well-suited to CHR-P research, addressing challenges related to heterogeneity, specificity, and the clinical significance of subthreshold symptoms (Cowan et al., 2024). Importantly, the definition of the HiTOP structure is largely grounded in epidemiological data, which has been critical to the growing recognition that clinical manifestations often cross the boundaries of discrete diagnostic categories (Jonas et al., 2024; Loch, 2019). Reinforcing the deep connection between etiological modeling and epidemiological research, this effectively illustrates the theorized link between epidemiological questions, e.g.,

"why did this person develop this condition at this time?", and preventive/clinical questions, e.g., "which (preventive) intervention will work for this person at this time?" (Rose, 2001). Taken together, these insights underscore the foundational role of epidemiology in shaping successful etiological and prevention models (Soneson et al., 2020).

Furthermore, in light of the low incidence and long latency of psychosis spectrum disorders, analyzing epidemiological data is an especially important first step to understanding their development within a public health approach (van Os et al., 2021). The present thesis aims at contributing to this first step, presenting epidemiological data analyzed in one longitudinal and two cross-sectional studies, and exploring their framing in the dimensional and transdiagnostic HiTOP model (Kotov et al., 2021).

2. Background

Psychotic disorders are multifaceted psychiatric conditions, characterized by a heterogenous clinical presentation and resulting from complex interactions of genetic and environmental factors that are yet to be fully understood (McCutcheon et al., 2020; Radua et al., 2018). The study of psychosis and the conception of its etiological models have historically centered on schizophrenia, and partly still do. However, this condition only represents a fraction (ca. 30%) of the much broader spectrum of psychotic disorders, which more recent research and etiological models aim to encompass (Guloksuz & van Os, 2018). Both of these tendencies are reflected in the title of the DSM-5 section *Schizophrenia spectrum and other psychotic disorders* (American Psychiatric Association, 2013). Therein, psychotic disorders are defined by presence of one or more of delusions, hallucinations, disorganized thinking/speech, grossly disorganized or abnormal motor behavior (i.e., positive symptoms), and negative symptoms. Thus, across most psychotic disorders, and especially in schizophrenia, DSM-5 diagnostic criteria greatly emphasize positive symptoms. Similarly, the section dedicated to *Schizophrenia or other primary psychotic disorders* in the ICD-11 prioritizes delusions, hallucinations, formal thought disorder, and disorganized behavior, while psychomotor disturbances and negative symptoms are listed as possible accompanying features (World Health Organization, 2019). Key distinctions from the DSM-5 include the possibility of diagnosing psychotic disorders in individuals with relatively preserved or high functioning and some distinctions in diagnostic requirements – e.g., the absence of a required six-month illness duration for a schizophrenia diagnosis – or the specific placement of certain diagnoses – e.g., the placement of mood disorders with psychotic traits under the mood disorders category, rather than the psychotic disorders section as in DSM-5 (Schultze-Lutter et al., 2024). However, like the DSM-5, the ICD-11 moves a step towards dimensional conceptualizations by incorporating a four-level symptom rating system (*none* to *severe*) for positive, negative, and affective symptoms, as well as cognitive impairment and psychomotor disturbances, in the context of (primary) psychotic disorders – as compared to a five-level rating in DSM-5 (*not present* to *present and severe*, including an *equivocal* rating, that allows for the classification of subclinical manifestations and is ab-

sent from the ICD-11; Schultze-Lutter et al., 2024). Further, the ICD-11 states how psychotic symptoms and behaviors indicating reality distortion are present on a continuum throughout the population, and are only indicative of a disorder when showing patterns characterized by such frequency and intensity that they deviate from (sub)cultural norms. This implies the existence of a broader spectrum of psychotic presentation, consistent with meta-analytic epidemiological data showing a prevalence rate of 5-8% and an incidence rate of 2.5% for subclinical psychosis symptoms in the community (van Os et al., 2009). Similarly, the DSM-5 mentions the existence of a psychosis spectrum as the main reason for inclusion of the schizotypal disorder among psychotic conditions.

While these features indicate a shift towards a spectrum-based conceptualization of psychosis, neither of the two classifications further integrates this dimensional model, despite its potential to overcome biases encountered by research in this field, in which severely ill, help-seeking individuals are often selected and the broader psychotic phenotype is underrepresented (Guloksuz & van Os, 2018). Adopting a dimensional perspective, subthreshold positive symptoms (i.e., CHR-P symptoms) could be reconceptualized as non-rare occurrences that can be present along a mental health spectrum, with psychotic disorders as the poorest outcome of general (i.e., non-psychotic) psychopathology (Guloksuz & van Os, 2018; Loch, 2019; van Os et al., 2021). Consistently, recent studies argue for a redefinition of psychosis as a distressing symptom found in many conditions and indicating greater severity of psychopathology, and especially its early expressions, rather than being the defining quality of a distinct category of disorders (Arciniegas, 2015; Franquillo et al., 2021; van Os et al., 2021).

Furthermore, by over-emphasizing positive symptoms, current diagnostic conceptualizations of psychotic disorders overlook the specificity and diagnostic relevance of negative symptoms, which show important associations with premorbid psychosocial functioning and outcome quality (Loch, 2019). This reverberates on psychosis-risk models, limiting early recognition of non-specific psychopathology and investigation of the complex interactions involving different mental health factors, thus ultimately interfering with accurate prediction (Guloksuz & van Os, 2018). In summary, the investigation of specific pathological processes of psychotic disorders proposed by etiological models might be hindered by the use of discrete diagnostic categories and disproportionate focus on positive symptoms and poor-outcome populations, whereas that of symptom dimensions and risk-stratification along a psychopathology continuum might result in new insights and more valid diagnostic classification of psychosis spectrum psychopathology (Guloksuz & van Os, 2018).

2.1. Genetic vulnerability, developmental and environmental correlates of psychosis

Like most of psychosis research, studies investigating its etiology have mainly focused on schizophrenia, with a growing body of evidence in support of its multifactorial origin (Stilo & Murray, 2019). Consistent evidence points at a large genetic component as well as the neurodevelopmental (rather than neurodegenerative) nature of psychotic disorders (McCutcheon et al., 2020). Meta-analytic data

from twin studies estimated the heritability of schizophrenia vulnerability at about 80%, and later genome-wide association studies found significant connections between schizophrenia and >100 specific genetic loci, supporting the view of schizophrenia as a polygenic disorder (Ripke et al., 2014; P. F. Sullivan et al., 2003). Further, some genetic variants resulting from deletion or duplication of DNA sequences – despite being rare in schizophrenia patients – are in and of themselves linked to an increased risk of developing the disorder (e.g., 22q11.2 deletion syndrome, 30-40% lifetime risk; Schneider et al., 2014). While recent scientific advances even allow for the calculation of a polygenic risk score to estimate individual genetic risk (McCutcheon et al., 2020), genome-wide association studies are only able to explain a fraction of the variation in psychosis vulnerability and risk in the general population, which might rather result from complex gene-environment interactions (Stilo & Murray, 2019). The impact of these interactions seems to especially concern certain brain regions and cortical microcircuits, involving excessive pre-synaptic striatal dopamine production, calcium channel regulation, immunity and inflammation processes, GABA- and glutamate neuroreceptors (McCutcheon et al., 2020; Stilo & Murray, 2019). Overall, a reduction in gray and white brain matter, as well as in connectivity and activation concerning frontal, temporal and limbic regions, is consistently reported in psychosis, and traced back to excessive pruning and insufficient myelinization along neurodevelopment (Haller et al., 2014; Schultze-Lutter & Schmidt, 2015).

On the other hand, meta-analytic data also showed a small but significant direct impact (11%) of environmental factors on schizophrenia liability, and evidence suggests their interaction with genetic vulnerability to be crucial to the neurodevelopmental trajectories that predispose to psychosis development (McCutcheon et al., 2020; P. F. Sullivan et al., 2003).

Among earlier environmental factors are pre- and perinatal unfavorable conditions, including maternal infection or malnutrition, preterm birth, preeclampsia, and low birth weight (Abel et al., 2010; Bramon et al., 2005; Xu et al., 2009). A history of obstetric complications has been found to significantly distinguish CHR-P participants from healthy controls (Kotlicka-Antczak et al., 2014) and was associated with a fivefold increase in the risk variance explained by polygenic risk scores, the latter being otherwise unable to reliably differentiate schizophrenia patients from controls (Ursini et al., 2018). Furthermore, schizophrenia in adolescence and early adulthood is frequently preceded in childhood by mild cognitive, hearing and motor deficits, as well as emotional and relational difficulties (Agnew-Blais et al., 2017; Erlenmeyer-Kimling et al., 2000; Reichenberg et al., 2010; Sørensen et al., 2010; van der Werf et al., 2011). Less clear are the effects of late winter/spring birth, whose risk-increasing effect for schizophrenia might be secondary to maternal infection or malnutrition, and older parental age, for which the investigation of association with psychosis risk yielded contrasting results (Haukka et al., 2004; Mortensen et al., 1999; Nosarti et al., 2012; Petersen et al., 2011).

Moreover, trauma and social disadvantage particularly stand out for their correlation to psychosis risk (Stilo & Murray, 2019). In fact, meta-analytic data has shown a strong association of childhood adversity, encompassing abuse, neglect, loss of or long-term separation from a parent, and bullying, with

increased psychosis risk (Varese et al., 2012). Additionally, childhood trauma was associated with higher severity of positive and affective symptoms in adulthood (e.g., Matheson et al., 2013), and life events, both positive and negative, were reported to be three times more likely in the 3 months to 3.6 years before psychosis onset (Beards et al., 2013). Further, cumulative and single-factor social disadvantage has been consistently linked to an increase in psychosis risk, including socio-economic status at birth and proximally to onset (e.g., unemployment, low income, overcrowded living conditions), isolation (e.g., living alone, being single, having no close relationships outside of the immediate family), and migration (especially as a refugee; Bourque et al., 2011; Hollander et al., 2016; O'Donoghue et al., 2014; Seidman et al., 2013; Stilo et al., 2017). An additional contextual factor that evidence has associated with increased odds of developing psychosis is urbanicity, with first findings indicating a protective effect of green space (Engemann et al., 2018; Vassos et al., 2012). Well-established research supports the relevance of substance use for psychosis development: data suggests a role of alcohol misuse, there is reliable evidence on stimulants' (e.g., cocaine, amphetamine) ability to induce psychosis, and, most importantly, on an important etiological role of cannabis use in psychosis, influencing onset age and worsening outcome (Di Forti et al., 2019; Koskinen et al., 2009; Marconi et al., 2016; Sara et al., 2015).

A characteristic shared by these environmental risk factors is their association with different forms and levels of stress, linking them to both neurobiological correlates and psychosocial etiological models of psychosis (Schultze-Lutter & Schmidt, 2015). A significant link in this respect might be the hyperactivity of the hypothalamic-pituitary-adrenal (HPA) axis observed in individuals with psychosis (Walker et al., 2013). Indeed, HPA axis dysregulation might result from chronic stress, trauma, cannabis use, or elevated cytokines in inflammatory diseases – i.e., several of the presented environmental risk factors – and, in turn, lead to increased dopamine production, imbalances in the glutamatergic system, and accelerated neuronal degradation – i.e., key neurobiological correlates of psychosis development (Schultze-Lutter & Schmidt, 2015). Illustrating the complex interplay between different orders of risk factors, this highlights the necessity of integrating psychosocial and neurobiological models into comprehensive etiological frameworks (Howes & Murray, 2014). These integrated frameworks describe how a history of early-life stress, trauma and social adversity might significantly shape cognitive and emotional vulnerabilities that heighten psychosis risk by fostering insecure attachment, negative self-image, external control beliefs, impaired social cognition (e.g., deficits in Theory of Mind), and dysfunctional metacognitive beliefs (Schultze-Lutter & Schmidt, 2015). Specifically, these features might be involved in paranoid ideation, as they are hypothesized to play a role in the development of (i) negative expectations towards abnormally salient stimuli, resulting from dopaminergic dysregulation, and (ii) the tendency to jump to conclusions (Freeman & Garety, 2014). Further, they might contribute to the onset of other delusions or hallucinations by virtue of their role in the development of an impaired sense of agency, which might result, for instance, in the misattribution of own thoughts and actions to an external source (Varese & Bentall, 2011).In summary, several interacting

risk factors for the development of psychotic disorders have been identified, many of which can rely on compelling evidence. No single factor is sufficient, by itself, to determine the onset of a psychotic disorder, nor is any risk factor specific to this diagnostic category, suggesting that the same etiological mechanisms may underlie different mental disorders, and highlighting the necessity of integrative models that account for their interplay (McCutcheon et al., 2020; Stilo & Murray, 2019).

2.2. Evaluating psychosis risk: the clinical high-risk state for psychosis (CHR-P)

As presented above, a wide range of interrelated risk factors are associated with the development of psychotic disorders (McCutcheon et al., 2020), and the specific way risk is evaluated and expressed can vary with different aims and contexts (Soneson et al., 2020). In early detection and prevention of psychotic disorders, the gold-standard approach in risk evaluation is the CHR-P state, with a conspicuous body of evidence supporting its association with a multiple hundred-fold increase in risk of psychosis compared to controls, as well as psychological burden, and functional impairment (Fusar-Poli, 2017; Fusar-Poli et al., 2020; Michel, Ruhrmann, et al., 2018; Schmidt et al., 2015). Definition of the CHR-P state relies on (i) the ultra-high risk (UHR) symptoms and criteria (i.e., subthreshold psychotic symptoms included in the extended psychotic spectrum, see above) and (ii) the basic symptom (BS) criteria, describing subtle changes in subjective experience (Fusar-Poli, 2017; Schultze-Lutter et al., 2015; Schultze-Lutter, Michel, et al., 2020). On the one hand, the UHR approach sets out to identify imminent risk of developing a psychotic disorder, as indicated by attenuated and/or brief intermittent psychotic symptoms, i.e., subthreshold hallucinations and delusions where some insight is retained (attenuated psychotic symptoms; APSs) or the time criteria for a psychotic episode are not met (brief intermittent psychotic symptoms; BIPSs; Fusar-Poli, 2017; Yung, 2020). On the other hand, the BS approach aims to identify psychosis risk as early as possible, relying on disturbances in mental processes, especially cognitive functioning and perception, which, while subtle and usually not detectable by others, are immediately perceived by the subject as deviating from normal experience and the premorbid self (Schultze-Lutter et al., 2015; Schultze-Lutter, Michel, et al., 2020). BSs are conceptualized as the closest psychopathological correlates to the neurobiological processes involved in psychosis development (Schultze-Lutter et al., 2016). A more detailed classification of CHR-P symptoms and criteria is included in Table 2.

In the last few decades, the CHR-P concept has fueled extensive research, shed light on further risk factors and possible etiological mechanisms, and resulted in multiple diagnostic measures, early detection and intervention programs (McGorry et al., 2018; van Os & Guloksuz, 2017). Several diagnostic tools were validated, and early detection and intervention programs developed (e.g., the FETZ Bern; see Publication 2, full text), with data supporting their efficacy in reducing or delaying manifest psychosis and improving overall outcome (e.g., psychological distress, functioning) independent of conversion (Correll et al., 2018; Michel, Ruhrmann, et al., 2018; Nelson et al., 2018; Schmidt et al., 2015;

Stafford et al., 2013; Valmaggia et al., 2015). Based on this research, diagnostic and clinical guidelines were established, notably by the European Psychiatric Association (Schmidt et al., 2015; Schultze-Lutter et al., 2015), with the Attenuated Psychosis Syndrome finding its place in Section III of DSM-5 (American Psychiatric Association, 2013).

While, as mentioned, conversion rates have decreased (Fusar-Poli et al., 2012, 2017), among possible explanations of these figures are actually factors in support of efficacy of early detection and intervention, or even disconfirming the decline itself. For instance, shorter duration of symptoms before referral might be reducing conversion rates (so-called "lead time bias"). Further, earlier treatment might have a greater preventive effect, leading to an increased rate of "false false positives", i.e., individuals at CHR-P who, although they did not develop a psychotic disorder at follow-up, would have done so without treatment (Yung, 2020). Additionally, meta-analytic data indicates that existing figures on conversion might be confounded by widespread baseline exposure to antipsychotics among CHR-P samples, which was consistently associated with a higher conversion rate and should be taken in higher consideration as an indicator of more severe risk (Raballo et al., 2020, 2024).

While these recognitions might help refine sample stratification and prediction (Raballo et al., 2024), current research argues for a broader shift in focus. In fact, the high heterogeneity in clinical presentation and trajectory of CHR-P states remains an issue (Fusar-Poli et al., 2016; Michel, Ruhrmann, et al., 2018; Polari et al., 2018). Specifically, CHR-P shows associations with a wide range of poor psychopathological and functional outcomes, including persistence of CHR-P symptoms, development and maintenance of comorbid psychiatric conditions (predominantly mood and anxiety disorders), and impairments in psychosocial functioning, irrespective of both symptom remission and conversion (Addington et al., 2019; Beck et al., 2019; Lin et al., 2015; Michel, Ruhrmann, et al., 2018; Solmi et al., 2023). Consequently, it became increasingly clear that research, early detection and prevention should prioritize a variety of (unfavorable) outcomes and trajectories of CHR-P, and not just conversion to psychosis (McGorry et al., 2018). Beyond this, a debate began within the field regarding the very nature of CHR-P, with many arguing for its more or less substantial redefinition (McGorry et al., 2018). First, being a symptomatic state linked to psychological burden and functional impairment, the CHR-P state might constitute a self-contained syndrome or disorder (see APS syndrome in DSM-5; American Psychiatric Association, 2013), beyond just indicating an increased risk of developing psychosis (Carpenter et al., 2014; Fusar-Poli et al., 2015). In that case, its associated risk for psychotic (primarily) and non-psychotic disorders (secondarily) would be comparable to the increased risk reported in individuals diagnosed with non-psychotic disorders to develop psychosis within the following three years (77.4-fold higher than in the general population; Lee et al., 2018). Therefore, the non-psychotic outcomes of CHR-P might be partially explained by heterotypic developmental trajectories that CHR-P shares with other mental disorders (McGorry et al., 2018). However, data about non-psychotic comorbidities and outcomes also led to questioning the specificity of the CHR-P state, proposing its recon-

ceptualization in a transdiagnostic or even pluripotential sense, i.e., as a possible marker of risk/severity of psychopathology across diagnostic categories (Cowan & Mittal, 2021; McGorry et al., 2018) or an early risk sign for multiple severe mental disorders (Hartmann et al., 2021).

Overall, the current complex picture of CHR-P shows how its strongest association is indeed with psychosis-related outcomes, but that it also has some relevance for the development and maintenance of non-psychotic disorders. Therefore, a comprehensive approach to the CHR-P concept in research, prevention and clinical work should both center the original goal of optimizing prediction of psychosis specifically and, concurrently, define its transdiagnostic value by broadening its scope (McGorry et al., 2018). Thereby, even before considering its comorbidities, it is worth noting how a certain transdiagnostic value of CHR-P is implied in the fact that the primary outcome it aims at predicting is the broader range of psychotic disorders, i.e., the psychosis spectrum, and not any one psychotic disorder (Cowan et al., 2024). To this end, different solutions have been proposed. Among the most promising are clinical staging models and dimensional, hierarchical models of CHR-P, two approaches presenting points of both contact and conflict (Cowan et al., 2024; McGorry et al., 2018). Clinical staging models aim at tailoring treatments based on symptom severity, specificity, persistence, and resulting impairment (Hartmann et al., 2021). Their underlying assumption is that psychopathology develops on a trajectory along which these parameters increase, from early stages, wherein unspecific, mild symptoms emerge, to more advanced stages, characterized by severe, specific, stable and impairing symptomatology (Hartmann et al., 2021). In other words, clinical staging models represent the practical application of transdiagnostic, pluripotential etiological models of psychopathology, such as the Clinical High At Risk Mental State (CHARMS; Hartmann et al., 2021). In such models, psychopathological outcomes, when they reach higher severity, are still compatible with, and refer back to, discrete diagnostic categories, such as those included in the DSM (McGorry et al., 2018). In contrast, dimensional models aim at reconducting transdiagnostic psychopathology to overarching mechanisms, factors or symptom dimensions, advocating for a non-categorical approach to mental disorder diagnoses along the severity continuum (Cowan & Mittal, 2021). One example is the Research Domain Criteria (RDoC) project, which uses neurobiological and behavioral indicators to identify the mechanisms underlying clinical manifestations, primarily aiming to facilitate translational research and informing nosology revisions (Sanislow, 2016). Thus, the RDoc project focuses on the biological processes involved in psychopathology, rather than the psychopathological manifestations themselves (Kotov et al., 2017). A second example, more relevant to the scope of the present thesis, is the Hierarchical Taxonomy of Psychopathology (HiTOP) model, which conceptualizes symptoms as indicators of latent clinical dimensions, organizing specific syndromes under broader spectra of psychopathology (Kotov et al., 2017).

In summary, clinical staging and dimensional models both imply transdiagnostic views on psychopathology. Clinical staging models still use categorial diagnoses to classify full-blown mental disor-

ders, adopting a conception on their development which centers pluripotency and heterotypy. Conversely, dimensional models aim at integrating or replacing the current psychiatric nosology with a non-categorial classification of mental disorders (Cowan & Mittal, 2021; McGorry et al., 2018). While clinical staging, offering a hands-on way to improve prevention and treatment tailoring, seems particularly suited for clinical applications, dimensional models such as HiTOP appear especially apt to investigate etiology and epidemiology. Specifically, they allow for detailed mapping of subthreshold cases, including CHR-P and other emerging psychopathology in the general population, which can be analyzed at different levels of specificity, including the symptom-level (Cowan et al., 2024; Cowan & Mittal, 2021). These characteristics make dimensional models, and HiTOP specifically, a useful framework for the present thesis.

2.3. Theoretical framework: a dimensional conceptualization of psychosis

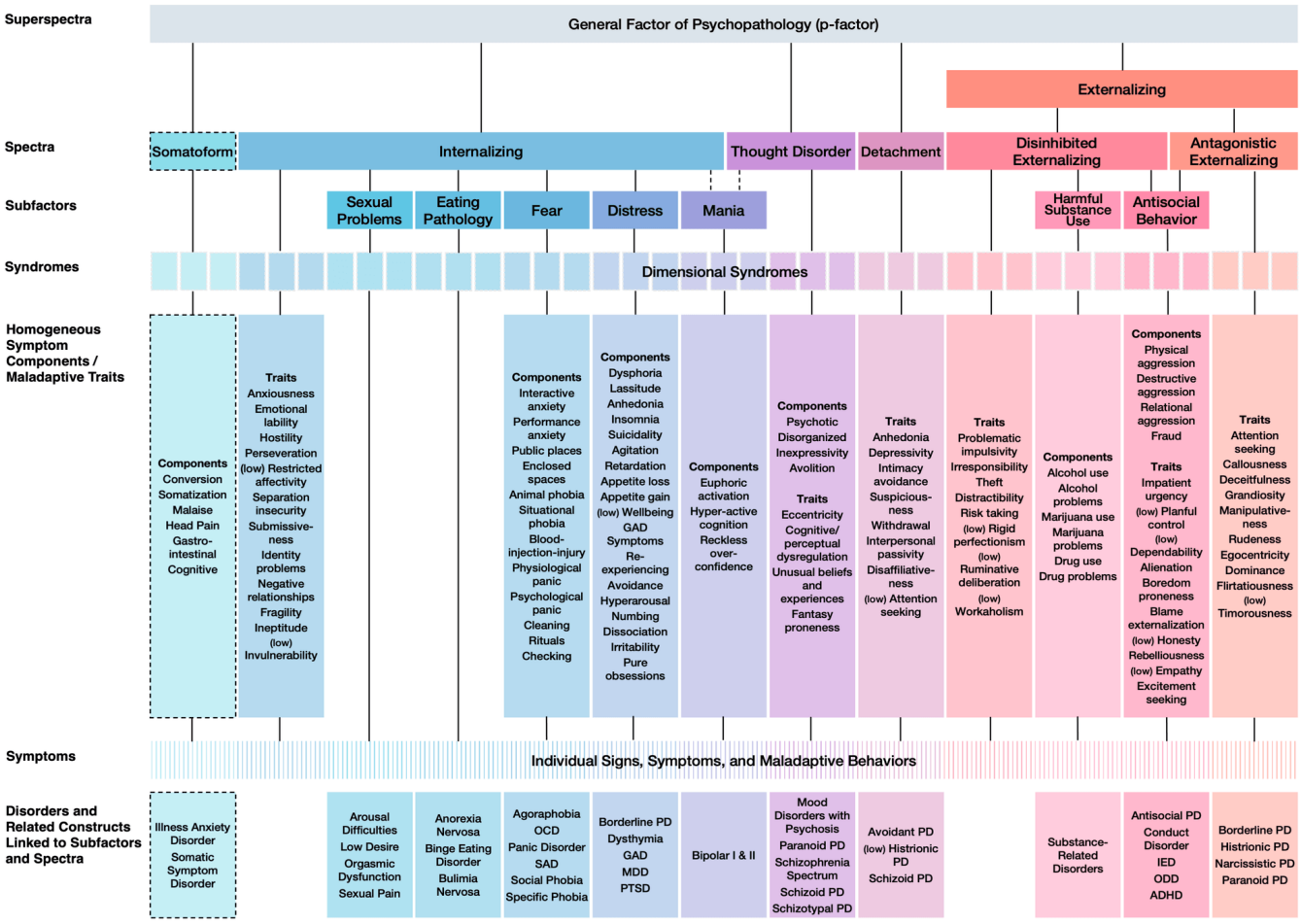
The systematization of psychotic disorders in the main classification systems has been shortly presented above. As Linscott and van Os (2010) argue, while classification implies a categorical distinction between diagnostic entities, it does not necessarily postulate a discontinuous structure of the latent psychopathology, i.e., imply discontinuity in the conceptualization of a disorder as an all-or-nothing phenomenon. Indeed, a clear disclaimer against the drawing of theoretical implications from the classification structure can already be found in the DSM-III, with the classification system explicitly aiming at being atheoretical. Furthermore, regarding psychotic disorders specifically, the latest editions of both the DSM and ICD make explicit mention of a schizophrenia continuum, despite not integrating it fully into the respective classification (American Psychiatric Association, 2013; World Health Organization, 2019). Therefore, dimensional and categorical nosology are not incompatible per se.

Indeed, the dichotomy between categorical and non-categorical classifications seems to be even more superficial in the case of psychosis. Reviewing multiple etiological theories, informed by the research on psychosis factors and correlates presented above, Linscott and van Os (2010) concluded that most authors, while they seldom addressed the question explicitly, seemed to imply a dimensional view of psychosis within the population (i.e., normality – psychosis) and across phenotypes (i.e., psychosis – other psychopathology), upon which a distinction between discrete disorder entities is superimposed for mainly practical reasons. Among these are epidemiological goals, that is, understanding the cause of psychotic disorders by describing their distribution, a task clearly made easier by categorical classifications, where disorders are distinct entities. Yet, in contrast, prevalence rates of subclinical symptoms pertaining to the psychosis phenotype, which are higher than prevalence rates of psychosis, appear consistent with dimensional models of psychosis. At the same time, existing evidence is consistent with the idea of a qualitative difference between individuals at risk and not at risk in the general population, supporting the validity of the early psychosis paradigm (Linscott & van Os, 2010) and still allowing for the identification of a boundary on which to base the dichotomous decision of whether to offer support or not (McGorry et al., 2018). Relatedly, dimensional models are not per se incompatible

with the CHR-P paradigm, as the definition of a CHR-P status can be regarded as "a dichotomization of a continuous variable" (Cowan et al., 2024, p. 13), i.e., the placement of an individual along the psychosis continuum. Dimensional models, then, could help both integrating recent findings on psychosis and psychosis risk in a coherent reconceptualization and solving the dispute between the specificity and the transdiagnostic fronts on CHR-P. As hinted above, the HiTOP model appears particularly suited for this task, especially when the main focus lies on the epidemiology and etiology of psychosis risk. The next sections will be dedicated to its more detailed presentation and the ongoing efforts to map CHR-P on this dimensional and hierarchical taxonomy.

2.3.1. The Hierarchical Taxonomy of Psychopathology (HiTOP) and the psychosis superspectrum

Within the HiTOP model, as opposed to categorical classifications, comorbidity and the transdiagnostic nature of psychopathological manifestations do not represent a drawback, but a fundamental assumption (Kotov et al., 2017). The psychopathological dimensions which make up the model in its current form were created by de-structuring existing categorical diagnoses into their constituting



Official HiTOP Figure. This figure depicts the full current official HiTOP framework. Dashed lines indicate dimensions included as provisional aspects of the framework.

Abbreviations: ADHD, attention-deficit/hyperactivity disorder; GAD, generalized anxiety disorder; IED, intermittent explosive disorder; MDD, major depressive disorder; OCD, obsessive–compulsive disorder; ODD, oppositional defiant disorder; PD, personality disorder; PTSD, posttraumatic stress disorder; SAD, separation anxiety disorder.

Figure 1 *The current HiTOP model. Source: HiTOP Consortium, https://www.hitop-system.org/current-model. Figure reproduced for academic use under the terms of the Creative Commons Attribution Licence (CC BY 4.0).*

symptoms and traits, then statistically testing their latent structure, mainly using factor analysis (Kotov et al., 2011; Kotov et al., 2017). The resulting dimensions or latent components "are psychopathologic continua that reflect individual differences in a maladaptive characteristic across the entire population" (Kotov et al., 2017, p. 3), which were subsequently organized hierarchically from narrowest to broadest. At every level, these dimensions can be analyzed taking their interrelationships into account (Jonas et al., 2022). The HiTOP model is constantly evolving as new data is acquired, and what will be presented here is merely its current form (see https://www.hitop-system.org).

Following the hierarchy from bottom to top (Figure 1), closely related symptom manifestations are organized in symptom components. At the same hierarchical level, they are joined by specific maladaptive traits (i.e., personality characteristics). Further up, both are organized in empirically defined syndromes, representing "composites of related components/traits" (Kotov et al., 2017, p. 3). In turn, broader patterns of related syndromes make up spectra, some of which present with subfactors, which are positioned on an intermediate hierarchy level, between syndromes and spectra. In total, the HiTOP model includes six positively correlated spectra: (i) the somatoform spectrum, encompassing health anxiety and physical symptoms which cannot be explained by medical or other conditions; (ii) the internalizing spectrum, referring to an individual's proclivity to low mood and fear; (iii) the thought disorder (psychoticism) spectrum, including positive psychotic symptoms, oddity, eccentricity of behavior and beliefs, and disorganized behavior; (iv) the detachment spectrum, encompassing the (lack of) interest in interpersonal relationships; (v) the disinhibited externalizing and (vi) the antagonistic externalizing spectra, referring to the (lack of) regulation and control skills over one's behavior and entitled, manipulative, aggressive behavior, respectively. Within each spectrum, evidence shows shared genetic and environmental risk factors, as well as neurocognitive and biological features and similar treatment response (Kotov et al., 2020). Next, superspectra represent the broadest dimension, composed of multiple related spectra, and finally, at the top, the p-factor of general psychopathology reflects the shared vulnerability hypothesized to underlie all psychopathological manifestations (Lahey et al., 2017).

Especially relevant to the scope of the present thesis is the conceptualization of the psychosis superspectrum, which encompasses the two underlying spectra of thought disorder (psychoticism) and detachment (Figure 2). Research on the validity and reliability of the psychosis superspectrum is currently exploring nosology, etiology, developmental trajectories, neurobiology, and clinical utility, and finding extensive evidence in its support (Jonas et al., 2024; Kotov et al., 2024). However, data on the structure of the psychosis superspectrum are still too inconclusive to allow for formal integration into the current HiTOP model, accordingly not depicted in Figure 1. This reflects an ongoing refinement and integration process and does not diminish its scientific and clinical potential (Kotov et al., 2024). As the exploration of the structure of the psychosis superspectrum evolves, its key components have been identified and extensively analyzed.

As mentioned, the HiTOP model and its components are conceptualized as dimensions of psychological function at every hierarchical level, thus virtually encompassing the whole population. When described dimensionally, thought disorder spans from conventional thinking to perceptive and cognitive functioning loosely grounded in reality, encompassing both positive symptoms and the trait of positive schizotypy, otherwise known as psychoticism (Cicero et al., 2019; Lenzenweger, 2018). Meanwhile, detachment includes individual variations ranging from effort to achieve goals to apathy, from high social investment to disinterest in others, and from high to restricted affective expression, including normative introversion as well as negative schizotypy and negative psychotic symptoms (Cicero et al., 2019; Lenzenweger, 2018; Suzuki et al., 2015). While connected, the two dimensions show distinct manifestations, etiologies, and implications for treatment and outcome, reflecting the heterogenous clinical picture observed in psychotic disorders and the related psychopathology (Kotov et al., 2020). Notably, the HiTOP psychosis superspectrum encompasses positive- and negative-symptom manifestations without prioritizing one over the other, doing justice to both the clinical relevance of negative symptoms (Loch, 2019), which is underplayed by current diagnostic criteria, and the long-standing finding that positive symptoms do not universally represent the largest burden in psychosis, since many patients are instead suffering the most from negative symptoms (Andreasen & Olsen, 1982; Kotov et al., 2020). Overall, this conceptualization of a psychosis superspectrum supports the growing agreement on the dimensional nature of psychosis, capturing most of the psychosis spectrum as defined in previous sections of this work, that is, spanning from adaptive functioning to maladaptive traits and subthreshold symptoms (i.e., trait- and state-like risk), and all the way to most categorized psychotic disorders (Guloksuz & van Os, 2018; van Os et al., 2009; van Os & Reininghaus, 2016). Furthermore, once again reflecting clinical heterogeneity, some categorial diagnoses can be mapped onto both thought disorder and detachment spectra (i.e., schizophrenia, schizophreniform and schizoaffective disorder, schizotypal and paranoid personality disorders), while others (i.e., schizoid and avoidant personality disorders) only on detachment (Figure 2; Kotov et al., 2020). At this stage, some symptoms remain just provisionally mapped (e.g., mania and dissociation), or were temporarily left out of the model due to insufficient evidence (e.g., catatonia and cognitive impairments). Nonetheless, the HiTOP psychosis superspectrum can be further integrated to include both empirically defined categorical clinical profiles (Kotov et al., 2013) and information acquired from developmental models such as clinical staging (Hartmann et al., 2021). This shows the promise of using HiTOP for the epidemiological and etiological study of psychosis along a continuum encompassing the general population.

Figure 2 *"Dimensions within the Hierarchical Taxonomy of Psychopathology (HiTOP) psychosis superspectrum. PD - personality disorder." [Not included due to copyright restrictions. For the original figure, see Kotov et al. (2020), World Psychiatry, 19(2), p. 155. https://doi.org/10.1002/wps.20730]*

Regarding psychosis risk specifically, HiTOP shows a remarkably good conceptual fit to the existing evidence on CHR-P that was presented above, offering multiple advantages. First, HiTOP assumes both comorbidity and the transdiagnostic value of the symptoms and traits it centers, while also allowing for the mapping of specific categorial diagnoses. Thus, the model might allow to concurrently con-sider both the value of CHR-P in relation to the specific risk of developing psychotic disorders and the transdiagnostic risk linked to other existing diagnoses. Additionally, the question of whether CHR-P represents a standalone psychiatric syndrome could be explored within the psychosis superspectrum. In that context, individuals presenting with subthreshold symptoms could be offered help in accord-ance with their needs, independently from whether they developed or will develop a categorially de-fined disorder. Indeed, since HiTOP dimensions are only probabilistically related to poor mental health and functioning outcomes, the recent decrease in transition rates to psychosis would not even lead to questioning the whole psychosis risk model, if the latter could be framed in this dimensional, hierarchical conceptualization. Here, the integration of developmental information from risk models, both "traditional" (specific for psychosis) and broader (CHARMS, clinical staging), might help iden-tify cutoffs useful for clinical and policy decisions. Indeed, the focus on both symptoms and traits within HiTOP is coherent with risk paradigms, which include schizotypal traits alongside unspecific symptoms as an indicator of vulnerability in the premorbid phase (Schultze-Lutter & Michel, 2017; see Figure 8). Ultimately, the HiTOP model could contribute to a more accurate and comprehensive

understanding of the nature of CHR-P, without penalizing clinical utility. The next section will explore the ongoing efforts to map CHR-P onto HiTOP.

2.3.2. Mapping CHR-P onto HiTOP

As much as the HiTOP model might improve our understanding of CHR-P, the converse is also true: mapping the heterogenous manifestations of CHR-P, with ties to multiple outcomes and comorbidities, including symptoms and syndromes whose placement in the model is still unclear (e.g., mania, obsessive-compulsive disorder), could greatly contribute to structuring HiTOP (Williams et al., 2024). Consequently, HiTOP-informed research on CHR-P is emerging, leading to interesting preliminary results on both fronts.

In a first study, Williams and colleagues (2024) aimed to recreate the HiTOP structure using self-report and interview data from a longitudinal, US-based study involving youth at CHR-P (n = 710) and healthy controls (n = 96; North American Prodrome Longitudinal Study-3; Addington et al., 2022). Their focus lay on the relationship between HiTOP and disorders with uncertain placement in the model, as well as childhood trauma, functioning impairment, and conversion to psychosis. The HiTOP model showed nearly adequate fit to the data, with findings indicating the necessity of some modifications to its structure. Specifically, the authors found links between bipolar spectrum disorders and the psychosis superspectrum, as well as complex links of obsessive-compulsive disorder and dimensions at multiple hierarchical levels, including the internalizing spectrum as well as the psychosis superspectrum. Furthermore, results supported associations of HiTOP with both childhood trauma and psychosocial functioning, and the model was able to predict conversion to a psychosis-superspectrum disorder (R^2 = .13). The study underscores how data from CHR-P samples can contribute to the evolving structure of HiTOP, while showing its clinical utility and its potential to further CHR-P research.

Even more notably, a study by Cowan and colleagues (2024) set out to analyze the transdiagnostic phenotypic profile of CHR-P symptoms within a hierarchical dimensional framework. Relevant symptom dimensions at multiple levels of analysis were defined via a hierarchical unfolding factor analysis of self-report data from a large US community sample (N = 3,460 young people, aged 16-30), assessing subclinical psychotic symptoms, depression, anxiety, mania, dissociation, and substance use. In the resulting model, a general psychopathology factor split into progressively more specific dimensions at each of ten levels, first separating into an internalizing and a psychosis factor, and then further declining these in their subcomponents, starting at the third and sixth level of analysis, respectively. Next, the authors explored the relationships between the defined dimensions and three psychosis risk-relevant variables using regression models. Measured in a CHR-P-enriched subsample (n = 436), these variables included clinician-rated CHR-P status, APSs, and attenuated negative symptoms. Overall, the results of this complex procedure were in support of the transdiagnostic value and complex comorbidities of psychosis risk, while highlighting specific relationships of dissociation, mania and substance use with its different facets, which might be helpful in identifying different CHR-P profiles. In

particular, APSs showed links to all latent components of psychosis (i.e., reality distortion, disorganization, and detachment), as well as some components of the internalizing factor (i.e., fear and dissociation), highlighting the influence of distress on subthreshold positive symptoms. Regarding attenuated negative symptoms, although they also showed links with psychosis dimensions, they were most strongly associated with components of the internalizing factor. While this finding underscores the challenge of differentiating negative from internalizing symptoms in early stages of psychopathology, the model shows a negative correlation of attenuated negative symptoms with mania and substance use, thus providing a possible strategy to improve differential diagnosis (i.e., if a clinical picture features mania or substance use alongside symptoms impacting on volition and affect, the latter might more likely develop into internalizing, rather than negative, manifestations). Finally, CHR-P status showed weaker effects and was more closely associated with reality distortion in the psychosis factor, as well as somewhat related to distress and fear in the internalizing factor. To interpret this finding, the authors argue that, within a perspective on psychosis as a continuum spanning the whole population, the operationalization of a CHR-P status could represent the dichotomization of a continuous variable, thus providing a lower level of detail and precision. These specific patterns were not evident before testing their links to the latent structure that was defined in the first step, thus supporting the value of dimensional models and showing consistency with previous evidence presented in this work.

Taken together, these findings underscore how HiTOP-informed research can refine our understanding of CHR-P and vice versa, setting the stage for the publications presented in the following section.

3. Three publications on CHR-P

The present dissertation aims to contextualize three publications on CHR-P, mostly involving community participants, in the framework introduced thus far. These publications build on data from research projects conceived before more recent evidence set the reconceptualization of the CHR-P paradigm in motion. However, the specific research questions and interpretations of the findings were oriented by a transdiagnostic perspective, focusing on the interactions of CHR-P with several factors of mental health. Similarly, although the study designs and assessment employed were not directly informed by HiTOP, their joint presentation and discussion in this dissertation will primarily reflect on their contextualization in this dimensional, hierarchical framework. As Kotov and colleagues point out (2020), HiTOP dimensions can be estimated from categorical diagnoses, and the transdiagnostic value of relevant relationships can be tested without necessarily employing HiTOP-conformant measures or latent variable modeling. Thus, in the following paragraphs, this work will summarize the three publications, highlighting their key findings and implications. Then, relevant points for their contextualization in HiTOP will be highlighted and integrated in the Discussion section. All three publications feature data from one or more time-points of the BEAR (*Bern Epidemiological At-Risk*) and BEARS-Kid (*Bi-national Evaluation of At-Risk Symptoms in children and adolescents*) longitudinal community studies. Across the respective baseline and first follow-up assessments, BEAR and BEARS-Kid were conducted separately. The two samples were then joined for a second follow-up assessment, approximately ten years after baseline. In light of their relevance to the present thesis, this section will begin with their brief description.

3.1. The BEAR and BEARS-Kid studies

The *Bern Epidemiological At-Risk* (BEAR; ethics ID: PB_2018-00132) and the *Bi-national Evaluation of At-Risk Symptoms in children and adolescents* (BEARS-Kid; ethics ID: PB_2016-02192) community studies aimed to explore the natural trajectories of psychosis risk, broader mental health outcomes and interactions with neurodevelopment across different life stages (Schultze-Lutter et al., 2018; Schultze-Lutter, Ruhrmann, et al., 2020; Schultze-Lutter et al., 2021). Specifically, two age ranges were selected at baseline, including 16- to 40-year-olds in BEAR and 8- to 16-year-olds in BEARS-Kid, respectively. The first age range was selected due to its documented high frequency of subclinical psychotic symptoms, making it highly relevant for psychosis risk (Kirkbride et al., 2006; McGrath et al., 2016). Meanwhile, the second age range was chosen to investigate the neurodevelopmental aspects of CHR-P symptoms, which show higher prevalence but lower clinical significance during development (Schultze-Lutter, Schimmelmann, et al., 2020). For participation in BEAR and BEARS-Kid, eligible residents were randomly selected from the population registry of Canton Bern, Switzerland, using stratified sampling to ensure representativeness. Baseline exclusion criteria for both studies included insufficient German, French or English language skills and a lifetime diagnosis of psychosis (Schimmelmann et al., 2015). Figure 3 reports a flowchart of participant selection for all

BEAR assessment points as well as the second follow-up (ethics ID: PB_2020-02856), including BEARS-Kid participants.

Validated clinical interviews conducted on the phone constituted the main assessments in BEAR. Prior to administration, the reliability of this method was supported by a feasibility study, showing high concordance (78–100%) with face-to-face interviews (Michel et al., 2014). Each time point included an add-on study. Specifically, the three-year follow-up assessment featured self-report measures of coping and core beliefs, which were analyzed in Publication 2, while the ten-year follow-up assessment included self-report measures of personality functioning, maladaptive personality traits and cognitive biases, which were analyzed in Publication 3 (see the respective sections for details about the instruments). In BEARS-Kid, the baseline and first follow-up assessments were conducted face-to-face, while the second follow-up assessment, after merging with the BEAR sample, was also conducted on the telephone.

In both studies and at all assessment points, first contact occurred via regular mail by means of a short information letter. First telephone contact was attempted within the following four weeks. For all telephone assessments, failing answer after 100 contact attempts was considered to indicate insufficient, outdated contact information and led to exclusion. At every time point, BEAR participants gave their informed consent by participating in the telephone interview. Meanwhile, BEARS-Kid participants and their parents provided written informed assent/consent before the baseline and first-follow up assessments. At second follow-up, participation of BEARS-Kid participants over 18 years of age in the telephone interview was equated to informed consent. Minors, who were all at least 14 years old, were required to provide written consent/assent prior to assessment, as well as written consent of a parent if they were younger than 16 years of age. Additionally, in both studies and at every time point, participants and, whenever necessary, their guardians were asked for permission to be re-contacted for following assessments, and had to provide an additional consent to participation in the add-on studies. Whenever needed, e-mail reminders were sent out to participants in the add-on studies after three weeks, then regularly about once a year.

In both studies, the main outcome measures focused on CHR-P symptoms (i.e., positive ultra-high risk – UHR – and basic symptoms – BSs) and related criteria, non-psychotic psychopathology, and psychosocial functioning. These main outcomes were assessed consistently at all assessments, which, along with the thorough training and supervision of the interviewers – all clinical psychologists – ensured high-quality data collection (Michel, Schimmelmann, et al., 2018). Specifically, positive UHR-symptoms and criteria were evaluated using the Structured Interview for Psychosis-Risk Syndromes (SIPS; McGlashan et al., 2010), while BSs and relative criteria were measured with the Schizophrenia Proneness Instrument, Adult/Child and Youth versions (SPI-A/-CY; Marshall et al., 2012; Schultze-Lutter et al., 2007). Further, general psychopathology was assessed with the Mini-International Neuropsychiatric Interview, adult and child and adolescent versions (M.I.N.I./M.I.N.I-KID; Sheehan et al., 1998, 2010), reflecting DSM-IV Axis-I diagnostic categories. Finally, psychosocial functioning was

assessed with both the Global Assessment of Functioning (GAF; American Psychiatric Association, 1994) and the Social and Occupational Functioning Assessment Scale (SOFAS; American Psychiatric Association, 1994), the former considering symptoms in its evaluation, the latter focusing on social and occupational functioning independently from psychopathology. Alongside the main outcomes, sex, age, education level on the International Standard Classification of Education (ISCED; UNESCO United Nations Educational, 2003), and other sociodemographic variables (e.g., nationality, relationship status) were assessed.

Across both studies, a quarter of participants reported CHR-P symptoms at baseline, with 2.4% meeting CHR-P criteria. At the first three-year follow-up of the BEAR study, five participants (0.5%) converted to psychosis, all of whom had reported CHR-P symptoms at baseline. Similarly, the first two-year follow-up of the BEARS-Kid study involved 195 participants, with one individual converting to psychosis. By the second follow-up, two additional participants had converted to psychosis. Comparisons of participants and non-participants across time points revealed no significant differences in age or sex, suggesting minimal selection bias in sociodemographic variables. Further details on the study procedures and their findings are included in the supplementary materials to Publications 1, 2, and 3. Overall, the BEAR and BEARS-Kid studies provide a robust foundation for examining CHR-P symptoms and their relationship to broader mental health, offering valuable insights into the dynamic trajectories of risk and resilience. Three relevant resulting publications will be presented below.

Table 1 *Summary box: main features of BEAR and BEARS-Kid*

BEAR	**BEARS-Kid**
Goal: Investigating the natural trajectories of CHR-P in the community	***Goal:*** Examining CHR-P symptom development in childhood
Sample: 16–40 years old, randomly selected from the Bern population registry	***Sample:*** 8–16 years old, randomly selected from the Bern population registry
Time points: Baseline, 3-year follow-up, 10-year follow-up	***Time points:*** Baseline, 2-year follow-up, later merged with BEAR for 10-year follow-up
Instruments: SIPS (UHR-symptoms), SPI-A/-CY (Basic Symptoms), M.I.N.I. (general psychopathology)	***Instruments:*** SIPS (UHR-symptoms), SPI-CY (Basic Symptoms), M.I.N.I.-Kid (general psychopathology)
Special features: Telephone interviews, focus on a critical age range for psychosis risk and onset	***Special features:*** In-person interviews (except 10-year follow-up), focus on development

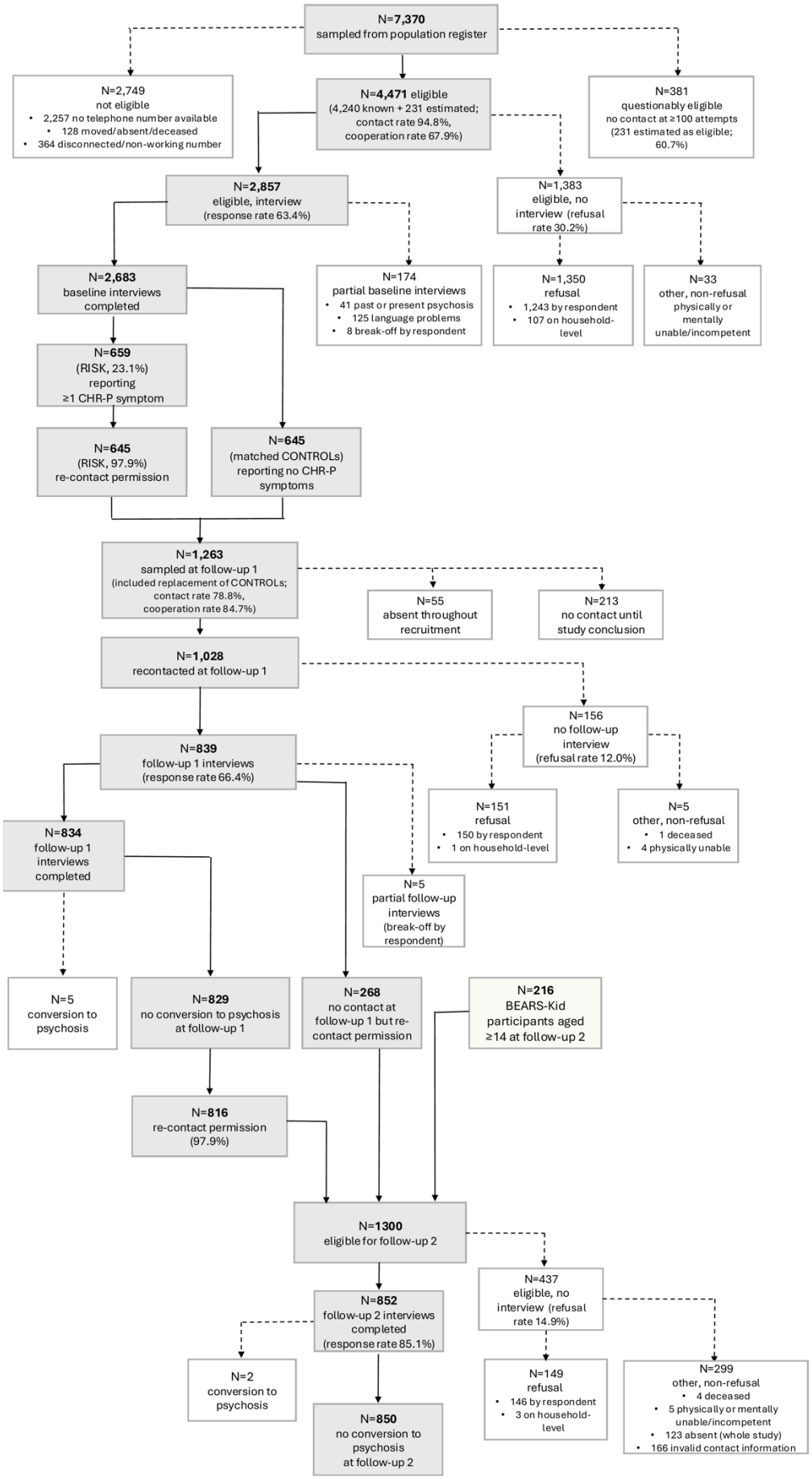


Figure 3 *The BEAR and BEARS-Kid Sample at follow-up 2. Adapted from Michel et al. (2025), Epidemiology and Psychiatric Sciences, under the terms of the Creative Commons Attribution Licence (CC BY 4.0). https://doi.org/10.1017/S2045796024000147*

3.2. Publication 1: Course of CHR-P symptoms in a community sample

Michel, C., Osman, N., Rinaldi, G., Schimmelmann, B. G., Kindler, J., & Schultze-Lutter, F. (2025). Three-year course of clinical high-risk symptoms for psychosis in the community: a latent class analysis. *Epidemiology and Psychiatric Sciences* 34, e3, 1–17. https://doi.org/10.1017/S2045796024000891

Full text on page 64.

Publication 1 aimed to define distinct clinical profiles of CHR-P symptoms and other mental health factors, following their course in 829 participants in the baseline and three-year follow-up assessments of the BEAR study. In light of (i) the relative homogeneity of the BEAR data, characterized by low prevalence of psychopathology and CHR-P symptoms, and (ii) the well-documented heterogeneity of CHR-P presentations and their course, this research goal was pursued by conducting two latent class analyses (LCA). This procedure had been employed successfully in previous literature (Healey et al., 2018; Ryan et al., 2018; Valmaggia et al., 2013; van Tricht et al., 2015). Specifically, Publication 1 aimed to define "hidden" distinct clinical profiles based on the distribution of CHR-P symptoms, at both baseline and follow-up. The profiles would then be compared and described with respect to Axis-I psychopathology, psychosocial functioning, and demographic data (see The BEAR and BEARS-Kid studies for information on the assessments).

The analyses revealed a three-class solution at both baseline and follow-up, as indicated by goodness of fit and class-separation indices (Akaike Information Criterion, Bayesian Information Criterion, relative entropy; see Publication 1, full text). At baseline, Class 1 (n=19, 2.3%) showed a high probability of BSs and was overall the most impaired, showing high rates of deficits in psychosocial functioning and psychopathology, as well as lower education and unemployment or sheltered/temporary employment. Similarly, Class 2 (n=25, 3.0%) exhibited a high rate of APSs/BIPSs, equal functional impairment to Class 1, but comparably lower rates of psychopathology, along with older age and the lowest rates of single people. In contrast, Class 3, the largest group (n=785, 94.7%), exhibited a low probability of CHR-P symptoms and was overall the healthiest profile, with the lowest rates of psychosocial impairment, psychopathology, and divorce, as well as the highest frequency of regular employment. At follow-up, differences in the Baseline Classes were overall not substantial (see Publication 1, full text; Figure 4). In contrast, despite a general resemblance to Baseline Classes, the second LCA conducted on follow-up data revealed relevant changes concerning the main outcomes, and especially the CHR-P symptom profiles. Specifically, Follow-up Class 1 newly had a high rate of perceptual APSs/BIPSs alongside the highest likelihood of most BSs, while Follow-up Class 2, alongside the highest probability of APSs/BIPSs, now showed the highest frequency of four specific BSs (*inability to divide attention*, *disturbance of receptive speech*, *derealization*, and *decreased ability to discriminate between ideas & perception, fantasy & true memories*) and an elevated rate of a fifth BSs (*visual*

perception disturbances). Most notably, perceptive BSs increased substantially at follow-up, thus becoming highly influential for the identification of the symptomatic Follow-up Classes 1 and 2. Conversely, APSs/BIPSs concerning unusual thought content, which had been central to the definition of the symptomatic Baseline Classes 1 and 2, only remained such for Follow-up Class 2. Furthermore, Follow-up Class 2 was the most impaired in terms of psychosocial functioning, and presented the lowest rates of regular employment and married people. There were newly no age differences between Follow-up Classes.

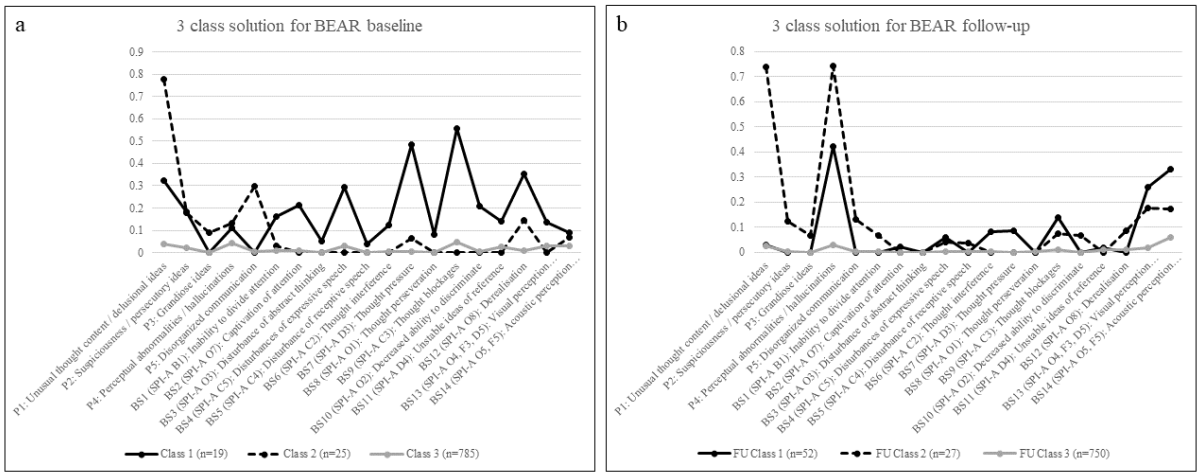


Figure 4 *Latent class profiles of basic symptoms and (attenuated) psychotic symptoms at baseline (a) and follow-up (b). Reproduced from Michel et al. (2025), Epidemiology and Psychiatric Sciences, under the terms of the Creative Commons Attribution Licence (CC BY 4.0). https://doi.org/10.1017/S2045796024000147*

Finally, over the three-year follow-up, most members of the symptomatic Baseline Classes 1 and 2 (73.7% and 88.0%, respectively) moved to the healthiest Follow-up Class 3, where most participants in Baseline Class 3 (91.0%) had remained. However, a minority of members of Baseline Class 3 (9%) moved to the symptomatic Follow-up Classes 1 or 2 (6.1% and 2.9%, respectively; Figure 5). Publication 1 was the first community study to employ an LCA for the longitudinal analysis of profiles of CHR-P symptoms and also include BSs. Overall, its findings of a three-class structure align with existing literature (Healey et al., 2018; Ryan et al., 2018), with the most comparable study, involving healthy controls, also finding a 'mild' class, comparable to Class 3 (Healey et al., 2018). While the separation of APSs/BIPSs and BSs in distinct symptomatic classes is consistent with existing evidence (Jimeno et al., 2020, 2022), the increase in perceptive symptoms at follow-up was not expected, as a their higher frequency had been reported in younger age by previous studies (Schultze-Lutter et al., 2017; Schimmelmann et al., 2015; Schultze-Lutter, Schimmelmann, et al., 2020). Consistent with existing literature (Addington et al., 2022; Solmi et al., 2023), the overall CHR-P symptom load im-proved, and most transitions from symptomatic classes were into Follow-up Class 3. However, Classes 1 and 2 retained high rates of psychopathology and functional impairments, emphasizing the clinical relevance of CHR-P symptoms even in the absence of psychosis conversion (Campion et al., 2012;

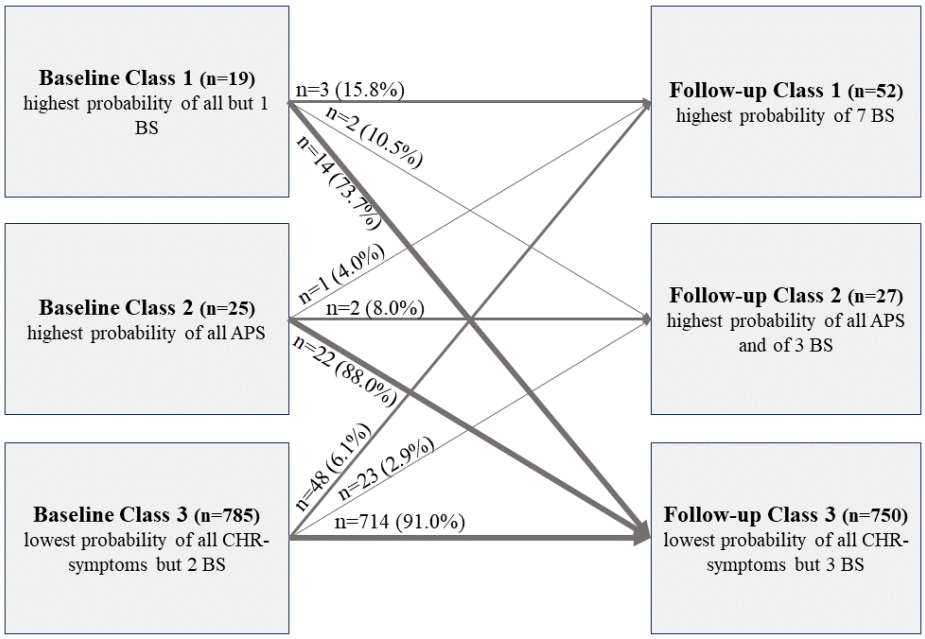


Figure 5 *Changes of class membership over time. Reproduced from Michel et al. (2025), Epidemiology and Psychiatric Sciences, under the terms of the Creative Commons Attribution Licence (CC BY 4.0). https://doi.org/10.1017/S2045796024000147*

Porru et al., 2023). Finally, Class 1 members, showing the highest rates of baseline psychopathology, additionally exhibited the highest rates of functional impairment at follow-up, possibly supporting findings on the combination of CHR-P symptoms and non-psychotic psychopathology being linked to poorer outcome (Hasmi et al., 2021).

As a whole, Publication 1 illustrates the heterogenic and highly dynamic nature of CHR-P symptoms in the community, showing their association with distress and impairment irrespective of transition to psychosis. Consequently, it concludes that preventive strategies should focus on enhancing public knowledge of CHR-P symptoms, improving screening and longitudinal monitoring in primary healthcare settings, and tailoring interventions to individuals' risk profiles, under consideration of psy-chosocial, neurocognitive, and biological risk factors. Moreover, the findings of Publication 1 chal-lenge traditional binary approaches to CHR-P and its outcomes, emphasizing the interactions between risk-specific and transdiagnostic dimensions. At the same time, its results underscore the need to in-vestigate transdiagnostic factors to improve the characterization of CHR-P symptoms and their evolu-tion (de Koning et al., 2022; Trotta et al., 2015). This topic was further investigated in Publications 2 and 3.

3.3. Publication 2: Interactions of core beliefs & coping strategies with CHR-P symptoms

Rinaldi, G., Osman, N., Kaess, M., Schimmelmann, B. G., Kindler, J., Schultze-Lutter, F., & Michel C. (2023). Exploring the complex relationships between coping strategies, locus of control and self-esteem with psychopathology: structural equation modeling with a special focus on clinical high-risk of psychosis. *European Psychiatry* 66(1), e88, 1-11. https://doi.org/10.1192/j.eurpsy.2023.2457

Full text on page 92.

Publication 2 investigated the intricate relationships between core beliefs, coping strategies, and mental health outcomes, with a special focus on their interactions with CHR-P symptoms. The clinical significance of CHR-P symptoms, predicting mental health outcomes even beyond psychosis risk, has been extensively described in the Background section. Similarly, research has provided compelling evidence linking transdiagnostic, interrelated factors such as core beliefs and coping strategies with psychopathology, psychosocial functioning, and one's own perception of health (Groth et al., 2019; Mann et al., 2004; Taylor & Stanton, 2007). Core beliefs encompass both competence and locus of control (LOC) beliefs, the first referring to self-esteem and self-efficacy, the second to the perception of control, or lack thereof, over life events (i.e., internal/adaptive versus external/maladaptive LOC, respectively; Buddelmeyer & Powdthavee, 2016; Mann et al., 2004). Coping strategies, on the other hand, are employed to process stressful situations, and can be adaptive or maladaptive depending on both their nature (e.g., problem-focused, active versus avoidant coping strategies) and their flexibility (Griva & Anagnostopoulos, 2010; Wingenfeld et al., 2009). Further, beyond serving as risk or protective factors for overall mental health quality, core beliefs and coping strategies have shown dysfunctional patterns along the psychosis continuum, and are considered potential predictors of psychosis (Harrow et al., 2009; Schmidt et al., 2014). Since (i) analysis of the interactions between core beliefs, coping strategies and mental health outcomes has obtained mixed results (Groth et al., 2019), and (ii) the role of CHR-P symptoms in this context has been underexplored, Publication 2 set out to investigate both questions. In particular, we tested three alternative placements of CHR-P symptoms, both jointly and divided into ultra-high risk symptoms (i.e., APSs/BIPSs) and BSs, in a total of six structural equation models. Based on the model emerged in a recent meta-analysis (Groth et al., 2019), in all models coping strategies mediated the association between core beliefs and multiple mental health outcomes. Regarding operationalization, adaptive and maladaptive coping strategies were assessed with the German Stress-Coping-Questionnaire scales *Positive* and *Negative Coping Strategies*, respectively (Hampel et al., 2001; Janke et al., 1997). Additionally, core beliefs were operationalized using the German Competence and Control Beliefs Questionnaire scales *Internality* and *Externality* for

adaptive and maladaptive LOC, respectively, and the *Self-Concept* scale for competence beliefs (Krampen, 1991).

In a first step, the latent structure of the selected mental health outcomes was tested in an exploratory factor analysis using Oblimin rotation and a following two-factor confirmatory analysis. The procedure resulted in two latent factors: (i) Psychopathology (PP), encompassing axis-I disorders, global functioning (GAF) and psychosocial functioning (SOFAS), and (ii) Self-Rated Health (SRH), reflecting participants' self-reported health status with the EuroQoL-5D, 3 level version (Brooks & Group, 1996).

Subsequently, the six SEMs were tested in a community sample from the first follow-up of the BEAR study (N=518, after pairwise deletion of five observations with missing data), using the maximum likelihood estimator. Their fit to the data was evaluated based on well-established goodness of fit indices (TLI: Tucker-Lewis index; CFI: comparative fit index; SRMSR: standardized root mean square residual; RMSEA: root-mean-square error of approximation). The models' Akaike Information Criterion and Bayesian Information Criterion were then compared, Model 1.2. (Figure 6) was chosen as best fitting the data, and a mediation analysis was conducted on all relevant paths. In this model, maladaptive coping fully mediated the associations of maladaptive LOC with PP, SRH, and CHR-P symptoms, while adaptive coping mediated the association between competence beliefs and PP, but not between adaptive LOC and PP. Additionally, CHR-P symptoms partially mediated the association between maladaptive coping and both PP and SRH. The three core beliefs variables showed no significant direct links with either PP or SRH.

Next, the chosen model was validated in a clinical sample from the Bern Early Recognition and Intervention Centre for mental crisis (FETZ) Bern[1] (N=327, after pairwise deletion of 51 observations missing >50% data). Overall, goodness of fit decreased, as would be expected with validation in a different sample, but remained adequate. When compared to the community sample, key differences were the absence of direct associations between coping as well as LOC (both adaptive and maladaptive) and the two outcomes PP and SRH, and the newly significant direct association between competence beliefs and SRH. Mediation analyses found no significant role for CHR-P symptoms in linking coping or competence beliefs with SRH and PP, nor for coping in the relationship between competence beliefs, LOC, and CHR-P symptoms.

Overall, findings emphasize the importance of targeting maladaptive coping and core beliefs (e.g., LOC and competence) to promote mental health, particularly in community settings, where the mediating role of coping in the associations between core beliefs and mental health outcomes was supported. While CHR-P symptoms were consistently linked to mental health outcomes, their role as a mediator was only supported in the community sample. Multiple explanations of these differences

[1] The FETZ Bern is a specialized outpatient clinical center for early detection and intervention on psychosis, providing 8-40 years old help-seeking individuals with naturalistic, but scientific-informed monitoring and support for (putative) psychotic/CHR-P symptoms (see supplementary material to Publication 2 for further information).

should be considered, including a potentially different focus of coping efforts in the two samples (e.g., more directly targeting CHR-P symptoms in the clinical sample, where they were more prevalent), as well as the higher burden of overall psychopathology and functional impairment in the clinical sample, possibly influencing the explored associations. Nevertheless, the findings of Publication 2 underline the transdiagnostic value of CHR-P, along with both the necessity and the difficulty of accurately mapping it on broader psychopathology models. This will be further explored in Publication 3, under a more dimensional lens.

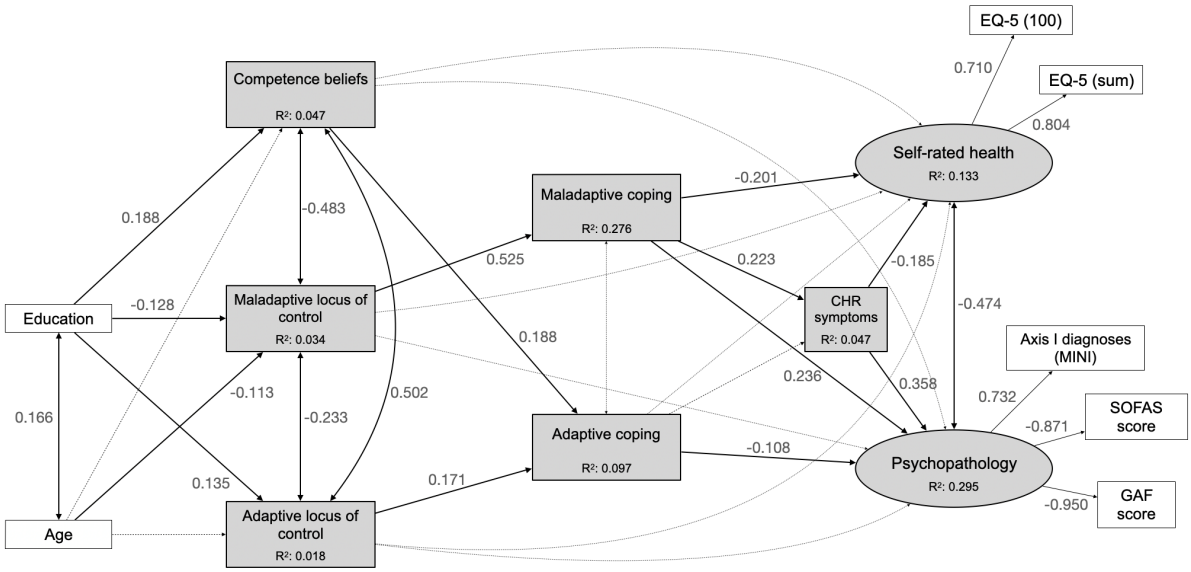


Figure 6 *Model 1.2. in the community sample. Reproduced from Rinaldi et al. (2023), European Psychiatry, under the terms of the Creative Commons Attribution License (CC BY 4.0).*
https://doi.org/10.1192/j.eurpsy.2023.2457

3.4. Publication 3: Interactions of personality functioning & cognitive biases with CHR-P symptoms

Rinaldi, G., Lerch, S., Schultze-Lutter, F., Schmidt, S. J., Cavelti, M., Kaess, M., & Michel, C. (2025). Investigating the associations between personality functioning, cognitive biases, and (non-)perceptive clinical high-risk symptoms of psychosis in the community. *European Psychiatry* 68(1), e13, 1–12. https://doi.org/10.1192/j.eurpsy.2024.1812

Full text on page 104.

Publication 3 delved into specific cognitive and personality-related factors associated with CHR-P symptom presence and severity.

A subsample of 444 participants in the second follow-up of the BEAR and BEARS-Kid studies was included. As part of an add-on study, they had filled out validated self-report instruments assessing personality functioning impairments (Level of Personality Functioning Scale-Brief Form 2.0; Weekers et al., 2019), maladaptive personality traits (Personality Inventory DSM-5; Krueger et al., 2014), and cognitive biases (Cognitive Biases Questionnaire for psychosis; Peters et al., 2014). The operationalization of the two personality factors followed the Alternative Model of Personality Disorders (AMPD) in DSM-5, which posits impairments along two dimensions of personality functioning (i.e., self- and interpersonal functioning) as the central feature of personality pathology (Criterion A), complemented by maladaptive personality traits (Criterion B), also assessed dimensionally (American Psychiatric Association, 2013). This dimensional model was chosen for its consistency with the recent proposal that the core features of personality pathology – rather than the specific traits/disorders considered in research thus far – might underpin the increasingly well-documented associations between psychosis/CHR-P and personality pathology, whose nature and direction is yet unclear (Caretti et al., 2021; Drvaric et al., 2018). From a theoretical perspective, this hypothesis is supported by the centrality of disruptions of the self and interpersonal relationships both in personality pathology and along the psychosis continuum (progressively permeable self-other boundaries, self-disturbances, gradual disruptions of narrative identity, impairment in interpersonal functioning; Cowan et al., 2021; Franquillo et al., 2021; Pionke-Ubych et al., 2022)[2]. Additionally, the analysis focused on a set of specific cognitive biases, i.e., stable and pervasive systematic distortions in information processing which are regarded as a potential link between personality pathology and psychosis/CHR-P, as they are hypothesized to act as the operational component of personality features, actively shaping and sustaining maladaptive

[2] For further details on the intricacies of the association between personality pathology and psychosis/CHR-P and on the conceptualization of personality pathology in the AMPD, please refer to the Introduction of Publication 3).

beliefs implicated in psychopathology development and psychosis vulnerability (Gawęda et al., 2015; Menon et al., 2013; Moritz et al., 2010).

Therefore, Publication 3 investigated the associations of personality functioning impairment and cognitive biases with the presence and expression of CHR-P symptoms in a community sample, controlling for the established ties of CHR-P symptom presentation to general psychopathology (i.e., current number of M.I.N.I. diagnoses) and socio-occupational functioning (i.e., SOFAS score). In order to account for the large number of participants reporting zero CHR-P symptoms, while also taking into account the varying severity of any CHR-P symptoms present, all analyses were conducted using zero-inflated Poisson models (Green, 2021). Following the AMPD, if personality functioning (Criterion A) was significantly associated with CHR-P presence or severity, maladaptive personality traits (Criterion B) were added to the respective model, testing whether their inclusion significantly improved model fit to the data via a likelihood-ratio test. Furthermore, the models examined the associations of personality functioning and cognitive biases with perceptive (e.g., attenuated hallucinations, perceptive BSs) and non-perceptive (e.g., attenuated delusions, cognitive BSs) CHR-P symptoms separately, as previous research highlighted differences in their manifestation, trajectory, and underlying mechanisms (Michel et al., 2023; Waters et al., 2012; Zhang et al., 2018; see Publication 3 for details).

In our findings, the likelihood of presenting with non-perceptive CHR-P symptoms was associated with impairments in personality functioning. When we conducted an exploratory analysis of personality functioning components, we found that these links particularly involved the AMPD elements of identity and self-direction (i.e., the domain of self-functioning). Additionally, more severe cognitive biases showed significant associations with both higher likelihood and severity of CHR-P symptoms, as well as higher likelihood of perceptive CHR-P symptoms and higher severity of non-perceptive CHR-P symptoms. In the following exploratory analysis of individual cognitive biases, dichotomous thinking, emotional reasoning, and catastrophizing showed multifaceted associations with overall CHR-P-symptom expression[3]. Further, the analyses highlighted links between more current M.I.N.I. diagnoses and higher likelihood of CHR-P symptoms across categories, while lower socio-occupational functioning was specifically associated with a higher likelihood of non-perceptive CHR-P symptoms. These findings reflect existing evidence on the comorbidities and general psychological burden tied to CHR-P symptoms, as well as a particular impact of non-perceptive CHR-P symptoms on functioning (Fusar-Poli et al., 2020; Michel, Ruhrmann, et al., 2018).

In conclusion, the results of Publication 3 underscored the complexity of the interplay between cognitive biases, personality functioning, and CHR-P symptoms, possibly contributing to different perceptive CHR-P- and non-perceptive CHR-P-symptom expression patterns. Thereby, first indications of the relevance of personality functioning beyond maladaptive traits or personality disorders emerged,

[3] For further information on the procedures and results of the exploratory analyses, please consult the Methods and Results sections in the Publication 3.

while cognitive biases arose as promising targets for future research on prevention through their association with CHR-P symptom presentation across categories.

4. Discussion

This thesis presented the evolving understanding of psychosis and CHR-P, emphasizing a shift towards an increasingly transdiagnostic, dimensional perspective. Central to this presentation were the HiTOP framework and its potential relevance in solving controversies regarding the CHR-P concept, with a particular focus on the psychosis superspectrum and the ongoing efforts to map CHR-P onto the HiTOP structure. Next, findings from three publications on CHR-P, centering non-psychotic psychopathological outcomes and multiple transdiagnostic correlates, were presented. Mostly using community samples from the BEAR and BEARS-Kid studies, the publications explored (i) the longitudinal course of CHR-P symptoms, identifying distinct "hidden" profiles and their trajectories, (ii) the role of CHR-P symptoms in the interplay of core beliefs and coping strategies with psychopathology and self-rated health, and (iii) the associations of personality functioning and cognitive biases with CHR-P symptom expression. Together, they offer novel insights into the complexity of CHR-P, extending its conceptualization beyond psychosis-specific outcomes. While the publications were not explicitly designed within the HiTOP framework, their findings are compatible with its main principles.

Overall, Publication 1 exemplifies the current shift in CHR-P research, moving away from psychosis transition exclusively to encompass both broader mental health outcomes and the transdiagnostic value of CHR-P (McGorry et al., 2018). In fact, converters were excluded from the analyses (a choice also oriented by statistical reasons, such as their low rate), while the main goal became the data-driven, person-centered definition of "hidden" homogenous clinical profiles. When conducted in a community sample and within the mentioned constraints, this operation can aid in classifying different phenotypes along the psychosis continuum, encompassing adaptive presentations on the psychosis dimension, vulnerability, and especially clinically distressing manifestations closer to its milder end as well as criteria-relevant CHR-P (Guloksuz & van Os, 2018; Linscott & van Os, 2010). As noted in the Background section, this is recognized as one focal point of future CHR-P research.

Specifically, emphasizing outcomes other than transition and transdiagnostic characteristics can help enrich our understanding of CHR-P beyond categorical diagnoses, in line with the HiTOP approach. Furthermore, in Publication 1, presentation profiles (i.e., phenotypes) were built on a CHR-P-symptom level to subsequently explore their associations with higher-level and/or transdiagnostic outcomes (i.e., psychosocial functioning, sociodemographic indicators, general presence of psychopathology). Thus, although without a perfect overlap, the methodology used in Publication 1 shares with HiTOP the use of empirically driven analyses to uncover hidden configurations in symptom-level data, with the primary aim of defining higher-level structure. Importantly, the two differ in their target, with LCA (as in Publication 1) looking to define phenotypic profiles in which to cluster individuals, while the structural analyses employed within HiTOP (e.g., factor analysis) rather focus on identifying the latent structure of psychopathology across individuals (Kotov et al., 2017). Nonetheless, both Publication 1 and HiTOP-informed research share a data-driven approach to describing symptom organization, as

well as a focus on the transdiagnostic associations of the symptoms they center. In this regard, the analysis in Publication 1 underscored the potential transdiagnostic value of thought pressure, derealization, visual perception disturbances, and suspiciousness/persecutory ideas especially, which occurred across classes. This can be understood as preliminary evidence that transposing this symptom-level analysis into a dimensional context might advance efforts at mapping CHR-P on HiTOP. So far, the HiTOP-informed work by Cowan and colleagues (2024) reported that APSs loaded on both a psychosis and an internalizing latent factor. However, like most studies mapping CHR-P on HiTOP, as well as most prominent clinical staging models (e.g., CHARMS; Hartmann et al., 2019, 2021), the authors did not consider BSs. This is despite evidence that BSs have great potential not only as early indicators of psychosis risk and conversion predictors (Michel, Ruhrmann, et al., 2018), but also for the characterization of CHR-P profiles and dimensional phenotyping along the psychosis continuum, including its milder end (Michel, Flückiger, et al., 2019; Schmidt et al., 2015; Schultze-Lutter, Michel, et al., 2020; Stüble et al., 2024). Therefore, the use of BSs in profile characterization in Publication 1 represents an important innovation, and indeed, among the symptoms highlighted by the analyses for their potential high transdiagnostic value (i.e., thought pressure, derealization, visual perception disturbances, and suspiciousness/persecutory ideas), all but one are BSs.

As a whole, given the broad transdiagnostic relevance of BSs across the psychosis continuum and beyond, their contextualization within the HiTOP transdiagnostic framework could enhance our understanding of CHR-P by addressing one of the paradigm's major challenges, advancing not only its mapping within the HiTOP model, but also the overall structural refinement of the model itself – particularly the psychosis superspectrum. Indeed, BSs show strong ties to neurobiology and neurocognitive dysfunction, while also connecting to non-psychotic – especially internalizing – psychopathology, psychological distress, functional impairment, and other CHR-P symptoms, such as APSs (Schultze-Lutter et al., 2016; Schultze-Lutter, Ruhrmann, et al., 2020; Stüble et al., 2024). Given this, including BSs in structural latent analyses – e.g., hierarchical factor/cluster analysis (Cowan et al., 2024; Ruggero et al., 2019) – aiming to map CHR-P symptoms onto HiTOP might simultaneously (i) shed light on the nature of CHR-P and (ii) enhance the overall accuracy of HiTOP, wherein cognitive dysfunction is currently underexplored despite being present across multiple psychopathological spectra (Abramovitch et al., 2021). Furthermore, given their potential to parse out heterogeneity in clinical presentations, mapping BSs onto HiTOP might provide a dimensionally anchored framework for stratifying psychosis risk, addressing another major challenge of the CHR-P paradigm. Meanwhile, it might inform clinical decision-making within HiTOP, addressing criticisms about its current lack of clear intervention thresholds (McGorry et al., 2018).

Building on existing CHR-P and HiTOP literature, it can be speculated that BSs might occupy within HiTOP a similar placement to APSs, positioned at the intersection of the psychosis superspectrum – particularly the thought disorder spectrum – and the internalizing spectrum – notably fear and distress

(Cowan et al., 2024). Crucially, BSs might present close links with cognitive functioning and impairment, thus offering the unique contribution of bridging the current HiTOP structure and (neuro)cognition – an essential, yet currently overlooked domain (Cowan et al., 2024; Jonas et al., 2024; Kotov et al., 2020). Future research should empirically test these structural hypotheses, concurrently examining the reciprocal placement and interactions of APSs and BSs, as literature supports both their distinctive clinical, developmental role and their conjoint organization under CHR-P (Schmidt et al., 2015; Schultze-Lutter et al., 2015; Schultze-Lutter, Schimmelmann, et al., 2020; see Figure 7 and the accompanying text for further considerations on CHR-P structure).

Finally, the transition between LCA classes from baseline to follow-up, particularly from healthier to symptomatic classes, underscores the importance of transdiagnostic mediators and moderators in shaping psychopathology expression and severity. This further supports the dynamic and continuous nature of CHR-P, reinforcing its conceptualization within a dimensional model of psychosis (Cowan et al., 2024). Therefore, mapping CHR-P onto HiTOP also offers the opportunity to adopt a more longitudinal perspective within HiTOP, integrating the static, cross-sectional symptom classification with a developmentally informed element that remains unexplored in current formulations (Kotov et al., 2020).

Like Publication 1, and aligning with both dimensional and clinical staging models, Publication 2 underscores the transdiagnostic relevance of CHR-P, as supported by its associations with core beliefs and coping strategies – i.e., transdiagnostic factors of mental health (Hartmann et al., 2021; Kotov et al., 2020). However, it does not clarify the directionality of these associations, exemplifying the challenges of mapping CHR-P within broader psychopathology. Despite this, a conceptual distinction can be drawn between more stable transdiagnostic elements, such as deeply interiorized beliefs on the self (e.g., self-esteem, self-efficacy, locus of control), which can be organized in personality traits (e.g., core self-evaluation), and more modifiable transdiagnostic factors, which can be targeted during treatment to influence mental health trajectories (Compas et al., 2017; Judge, 1997). Accordingly, it seems reasonable to speculate that stable elements like core beliefs might be more directly included in the HiTOP structure, whereas modifiable ones, like coping strategies, could be better contextualized as external mediators or moderators of severity and overall expression of psychopathology – a hypothesis which should, however, be empirically tested against alternative placements within HiTOP (Bornstein, 2019; Kotov et al., 2017). While the statistical procedure used to define the PP and SRH latent factors in Publication 2 shares some commonalities with the HiTOP approach – both aiming to uncover latent structures overarching narrow-band dimensions (Kerber et al., 2024) – there are key methodological distinctions between Publication 2 and the HiTOP guidelines. First, Publication 2 only explored the latent structure of its outcome variables, resulting in the PP and SRH latent factors – not that of CHR-P symptoms, core beliefs or coping strategies. Second, while core beliefs and coping strategies were largely operationalized dimensionally (i.e., standardized scores on continuous scales from validated instruments; Hampel et al., 2001; Janke et al., 1997; Krampen, 1991), CHR-P symptom scores were

dichotomized before forming sum-scores, resulting in only a partially dimensional operationalization (see Limitations). Thus, an empirically valid inclusion of CHR-P symptoms, core beliefs, and coping strategies in HiTOP would require (i) a fully dimensional operationalization of CHR-P symptoms (i.e., using absolute 1-6 SIPS and SPI-A/-CY scores), and (ii) a latent structure analysis (e.g., hierarchical factor/cluster analysis) integrating all variables within the HiTOP structure. This approach offers a promising path for clarifying the complex interactions explored in Publication 2, whose research question remains otherwise partially unanswered.

Further, while discussing the findings of Publication 2, the need for more group-dependent research – comparing community and clinical samples – was highlighted as a way to elucidate the examined interactions; instead, an even more comprehensive alternative would be adopting a fully dimensional framing – such as HiTOP – which would allow for the simultaneous exploration of these associations across the entire psychosis continuum (Guloksuz & van Os, 2018). This underscores another potential advantage offered by HiTOP in developing a more comprehensive understanding of the interactions explored in Publication 2.

In turn, integrating adaptive coping strategies and core beliefs in HiTOP could be beneficial for the HiTOP framework itself. Indeed, the inclusion of adaptive coping strategies, locus of control, and competence beliefs in HiTOP aligns with recent efforts to relate HiTOP dimensions to positive psychological factors and resources (Oliveira et al., 2021). Expanding the HiTOP structure to encompass not only maladaptive manifestations but also adaptive functioning would bring it closer to a truly dimensional perspective on mental health (HiTOP Research Group, 2023). This is particularly relevant when characterizing the milder end of the psychosis continuum, where protective mechanisms and resilience factors may play a key role, and would therefore have important implications for etiology, early intervention, and clinical treatment – including better stratification of CHR-P samples.

In summary, this contextualization of Publication 2 highlights the potential reciprocal advantages of integrating the HiTOP and CHR-P paradigms – along with their transdiagnostic correlates – supporting the main statement of the present thesis.

Although Publication 3 does not feature HiTOP, its framing is based on another dimensional, hierarchical model – the AMPD – and thus explicitly involves a non-categorical perspective on (personality) psychopathology. Notably, emerging literature highlighted similarities between HiTOP and the AMPD, exploring their compatibility and possible integration. Similar to HiTOP, the AMPD posits that the overarching impairments in personality functioning which define personality disorders (Criterion A) are characterized, at a lower hierarchical level, by different maladaptive personality traits (Criterion B; American Psychiatric Association, 2013). Therefore, the AMPD allows for a symptom-level analysis of personality pathology, whose severity can be described dimensionally at both levels of the hierarchy (i.e., personality functioning and maladaptive traits), much like in HiTOP.

Currently, most works contextualizing the AMPD in HiTOP center on Criterion B, as AMPD-traits greatly overlap with HiTOP-traits at the lowest hierarchy level (Kotov et al., 2017). While less straight-forward, integrating personality functioning (Criterion A) with HiTOP might enhance its overall clinical utility by broadening the assessment and treatment framework to include psychopathological processes and not just dimensions (Bornstein, 2019). Even more relevant to the present thesis, it might contribute to a comprehensive understanding of psychopathology within HiTOP by bridging the gap between personality and non-personality manifestations even further (Kerber et al., 2024). Overall, the conceptualization of personality functioning in the AMPD fits well into HiTOP's emphasis on transdiagnostic dimensions of psychopathology, especially at higher hierarchical levels (Bornstein, 2019). Some facets of personality functioning (e.g., emotional regulation, self-esteem - the latter featured in Publication 2) are also recognized as transdiagnostic elements of psychopathology, and employed as treatment constructs across internalizing, externalizing and personality pathology (Sloan et al., 2017). Indeed, emerging evidence supports the relevance of Criterion A across HiTOP spectra, reporting significant associations between facets of personality functioning and all HiTOP domains (Bottesi et al., 2024; Kerber et al., 2024). Consistently, it has been proposed that personality functioning might align with the HiTOP general p-factor, finding indication that personality functioning explains a substantial portion of transdiagnostic variance in psychopathology (Kerber et al., 2024; Widiger et al., 2019). Overall, the exact placement of personality functioning within HiTOP remains debated (Bottesi et al., 2024), with some hypothesizing that it might alternatively represent a distinct factor, or an external mediator/moderator of psychopathology severity and expression, similarly to coping and core beliefs (see Publication 2, full text). Therefore, this should be explored in future research (Bornstein, 2019; Widiger et al., 2019). Nonetheless, evidence on the transdiagnostic valence of personality functioning, even beyond personality pathology, appears promising (Bottesi et al., 2024; Kerber et al., 2024).

Although this question cannot be answered within the scope of the present work, its framing matches our approach to CHR-P across the three publications, centered on understanding its transdiagnostic associations and implications along a dimensional measure of severity and encompassing the general population. Beyond its overall framing, Publication 3 found significant associations of personality functioning with the presence of non-perceptive CHR-P symptoms (see Publication 3, full text). In order to contextualize this specific association within HiTOP, it would be necessary to map perceptive and non-perceptive CHR-P symptoms onto the model, and test whether they can be empirically ascribed to two distinct latent dimensions. While this is plausible, based on both our results and existing literature on their differences (Michel et al., 2023; Waters et al., 2012; Zhang et al., 2018), the few works aiming to map CHR-P onto HiTOP did not explore the distinction between perceptive and non-perceptive manifestations so far (Cowan et al., 2024; Cowan & Mittal, 2021; Williams et al., 2024). In summary, this aspect of Publication 3 cannot be further contextualized in HiTOP than as a research question for future studies.

On the other hand, although they should be interpreted with caution, the exploratory analyses conducted in Publication 3, which featured the empirically derived latent components of AMPD and the individual cognitive biases, offer valuable insights in this respect. In Publication 3 (see full text), we discussed the complex associations between psychosis/CHR-P and personality pathology, hypothesizing a role for self-functioning impairments. This hypothesis was partially supported by findings linking identity and self-direction to the expression of non-perceptive CHR-P symptoms. Given existing evidence associating self-functioning with internalizing symptoms, and identity specifically with psychopathological manifestations across HiTOP spectra (Bottesi et al., 2024; Kerber et al., 2024), the hypothesis can be advanced that self-functioning might be a key factor linking the findings of Publication 3 to HiTOP general psychopathology. Overall, this underscores the importance of exploring personality functioning, and especially self-functioning, within the HiTOP model. Examining personality functioning alongside personality traits, non-personality psychopathology, and CHR-P manifestations could shed light on the long-standing question of the relationship between personality and psychosis/CHR-P.

In contrast, cognitive biases were linked to CHR-P across categories, showing associations with both (i) the presence and severity of overall CHR-P symptoms, and (ii) the presence of perceptive CHR-P symptoms and the severity of non-perceptive CHR-P symptoms. Given their conceptualization as an operational component of personality traits (Gawęda et al., 2015), we might speculate that cognitive biases could be contextualized within HiTOP similarly to coping strategies, that is, as a factor not directly included in the HiTOP structure, yet influencing the expression and severity of (CHR-P) psychopathology (Bornstein, 2019). Exploring the multifaceted associations between individual cognitive biases and CHR-P symptoms within HiTOP could deepen our understanding of lower-level dimensions, perhaps clarifying their impact on specific personality traits. In conclusion, this perspective on the findings of Publication 3 highlights the potential of HiTOP to unify personality and non-personality psychopathology, particularly at the milder end of the psychosis spectrum (i.e., encompassing the general population), while offering a robust framework to investigate key transdiagnostic processes, such as cognitive biases.

4.1. A proposed framework for future CHR-P research: Integrating key findings into HiTOP

In this thesis, the challenges posed by the increasing evidence of heterogeneity in clinical presentation and the transdiagnostic value of CHR-P have been presented, alongside the proposal that integrating CHR-P into the HiTOP model could provide a meaningful solution. Accordingly, the result of three publications on CHR-P, focusing mainly on community data, have been discussed as a foundation for formulating initial hypotheses which might guide future research on this integration.

The purpose of this subsection and its accompanying graphic (Figure 7) is twofold. First, they summarize the discussion of our key findings within the HiTOP framework, offering a hypothetical integrated

depiction of where we might expect them to fit in the model based on both empirical results and conceptual considerations. Second, they highlight key unanswered questions, aiming to orient future

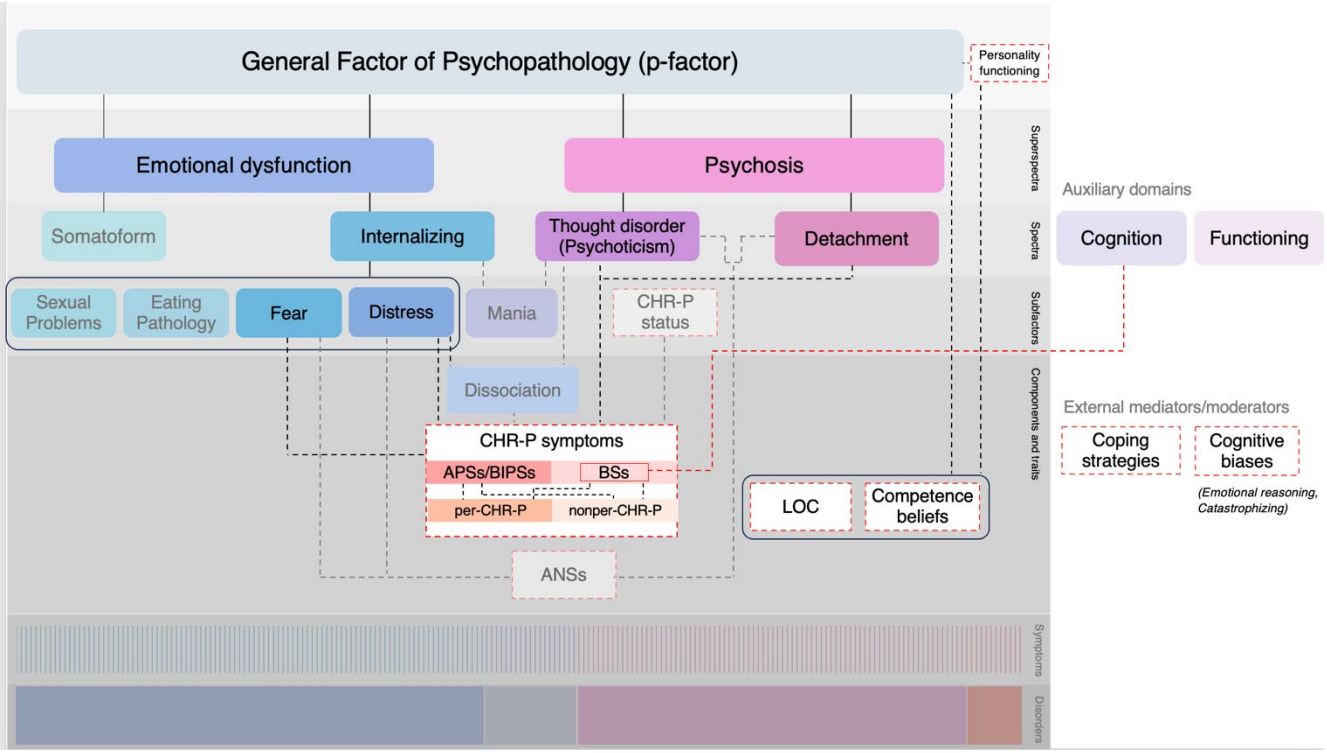


Figure 7 *Proposed model integrating CHR-P into HiTOP, summarizing key findings from the present thesis and highlighting areas for future research – particularly the possible internal structures of CHR-P symptoms and their placement at the intersection of spectra. Adapted from Cowan et al. (2024), Jonas et al. (2024), Kotov et al. (2020), and the official HiTOP model (HiTOP Consortium, https://www.hitop-system.org/current-model), licensed under CC BY 4.0.*
Legend *White rectangles with red dotted borders represent key variables discussed in the present thesis. Dotted lines indicate associations under empirical refinement within HiTOP research and/or hypothesized in this thesis. Red dotted lines indicate hypothesized associations involving auxiliary domains, i.e., domains whose placement within the main model remains unexplored. Greyed out areas denote components and associations not central to the present discussion.* ***Abbreviations*** *CHR-P: clinical high risk of psychosis; APS: attenuated psychotic symptoms; BIPS: brief intermittent psychotic symptoms; BS: basic symptoms; per-CHR-P: perceptive CHR-P symptoms; nonper-CHR-P: non-perceptive CHR-P symptoms; ANS: attenuated negative symptoms; LOC: locus of control.*

HiTOP-informed research on CHR-P by outlining relevant variables and research hypotheses. While this depiction builds on the current HiTOP structure – including the psychosis and emotional dysfunction superspectra, which remain under empirical refinement before their official inclusion – and on ongoing HiTOP research on CHR-P, it is not equivalent to those resulting from the systematical empirical exploration of the latent structure of psychopathology that defines HiTOP research (Kotov et al., 2017). Instead, it serves as an illustrating device, summarizing speculative expectations, open questions, and focal points emerging from the key findings of this thesis, while underscoring future research priorities in the investigation of the placement of CHR-P within the transdiagnostic and dimensional framework of HiTOP.

The model proposed in Figure 7 speculatively conceptualizes CHR-P symptoms as a latent risk component intersecting the psychosis superspectrum and the internalizing spectrum. Structural findings by Cowan et al. (2024) suggest that CHR-P symptoms can be reasonably expected to associate primarily with the thought disorder spectrum of psychosis, as depicted in Figure 7 – particularly with its reality

distortion and disorganization components (omitted for space reasons). Secondary associations might be expected with the detachment spectrum (psychosis superspectrum), followed by the distress and fear subfactors (internalizing spectrum), then by the dissociation component, provisionally modeled as an interface between psychosis and internalizing dimensions. If confirmed, this would align with findings supporting the transdiagnostic value of CHR-P across the three Publications, as well as the growing evidence in recent literature of its particular ties to internalizing psychopathology (Beck et al., 2019; Solmi et al., 2023). Furthermore, given their strong links to cognition, BSs might show specific associations to this auxiliary domain, favoring its integration process into the main HiTOP structure (Jonas et al., 2024; Kotov et al., 2020; Schultze-Lutter & Theodoridou, 2017).

In Figure 7, the placement of CHR-P symptoms at the components and traits level of HiTOP reflects findings by Cowan and colleagues (2024) on APSs, as does the positioning of attenuated negative symptoms at same level and that of CHR-P status at the subfactors level. In contrast, the internal hierarchical structure of CHR-P symptoms is included in the proposed model as an open question. Specifically, while existing literature supports both the distinction between APSs/BIPSs and BSs and that between perceptive (hallucinatory, sensory) and non-perceptive (cognitive, delusional) CHR-P symptoms (Michel et al., 2023; Jimeno et al. 2020, 2022; Schultze-Lutter et al., 2015), their reciprocal structural relationship, especially within the HiTOP framework, was not explored empirically so far. Their hierarchical placement may be key to determining their final placement in the model and refining the classification of CHR-P within HiTOP, and should be investigated in future studies.

Moreover, following both the current literature discussed above, mostly pointing in this direction (Kerber et al., 2024; Widiger et al., 2019), and the hierarchical structure of the AMPD, personality functioning is positioned provisionally at the same level as the general p-factor (Figure 7). However, whether it should be considered a component of p, a parallel but distinct factor, or an external mediator/moderator, referring to psychopathological processes rather than structure, remains an open empirical question. Similarly, the placement of the self- and interpersonal functioning components (also omitted for space reasons) within HiTOP remains to be empirically determined.

Finally, the proposed model incorporates distinct placements for stable versus dynamic transdiagnostic factors, as hypothesized in the discussion above. Therefore, core beliefs (i.e., competence beliefs and locus of control) are mapped within HiTOP, while more dynamic factors describing psychopathological processes, such as coping strategies and cognitive biases, are featured as external mediators/modulators. However, this placement remains speculative, and alternative structures cannot be excluded without conducting an empirical latent structure analysis. Similarly, while core beliefs appear best conceptualized as traits and are consequently mapped at the relative HiTOP level, their exact hierarchical placement and specific associations with other dimensions – beyond their general links to the p-factor and personality functioning – remain to be determined through empirical research.

In summary, the proposed model, aligning with ongoing research while outlining key directions for future studies, highlights the transdiagnostic value of CHR-P and underscores how its integration

within HiTOP could both refine the HiTOP structure and provide a clearer conceptual framework for addressing the challenges of CHR-P research.

4.2. Limitations

Despite the strengths of this work, including the large representative sample(s), the integration of diverse methodologies, and the exploration of CHR-P symptoms through a transdiagnostic, dimensional lens, some limitations should be considered.

One key limitation lies in the variability of CHR-P symptom operationalization across the three publications, which was influenced by data constraints and statistical requirements. Specifically, in Publication 1 CHR-P symptoms were dichotomized (present/absent) based on their clinical relevance to the definition of a CHR-P state, although irrespective of onset, worsening or frequency requirements (see Table 2 for details on criteria). Both subthreshold positive symptoms on the SIPS and basic symptoms (BSs) on the SPI-A/-CY are assigned a score between 1 and 6, indicating severity. Here, BSs were considered present when assigned a score of 1-6 (i.e., independent of their severity) or 8 (indicating definite presence, but unknown severity). On the other hand, subthreshold positive symptoms were only considered present when they qualified as criteria-relevant attenuated positive symptoms (APSs) or brief intermittent psychotic symptoms (BIPSs), i.e., when they received a score of 3-6. This approach flattened milder presentations especially, limiting both the description of CHR-P profiles and the reframing of the findings within the HiTOP dimensional framework. In Publication 2, the same procedure was applied, followed, however, by a second step, in which the dichotomized score for each SIPS-P scale and BS were added up into a UHR-, a BS-, and a comprehensive CHR-P sum-score. Thus, dichotomized UHR and BS criteria were combined into dimensional indices, allowing for a more nuanced perspective on individuals' comprehensive clinical presentations. However, this still flattened the representation of milder presentations on the symptom-level. Finally, Publication 3 adopted the most dimensional operationalization out of the three, using absolute CHR-P symptom severity scores (i.e., 1-6) to obtain a perceptive, a non-perceptive, and an overall CHR-P symptom sum-score. However, the individual symptoms were assigned to the respective categories based on clinical observation and previous evidence, without testing their latent structure, which diverges from the methodology employed in HiTOP. While these different operationalizations reflect the complexity and heterogeneity of CHR-P presentations and were necessary in order to balance statistical constraints and information loss, they limit the comprehensive interpretation of the results, particularly regarding their reframing within the HiTOP model.

Additionally, our analyses did not include non-CHR-P-relevant symptoms, such as negative and affective symptoms, despite their relevance in both predicting psychosis conversion in CHR-P samples and characterizing CHR-P profiles (Ergül et al., 2024; Valmaggia et al., 2013). The exclusion of these symptoms – due to their omission in the BEAR and BEARS-Kid studies – restricts the extent to which

this work can capture the broader transdiagnostic structure of early psychopathology within HiTOP, constituting a second limitation.

Further methodological constraints relate to sample bias and generalizability. Specifically, the reliance of the present findings on predominantly community-based data limits both their applicability to clinical populations and their contextualization within the existing HiTOP structure, which is centered on psychopathological manifestations (HiTOP Research Group, 2023).

Moreover, the joint use of self-report measures and interviews, as well as the fact that most interviews were conducted on the telephone, may introduce a response bias in the analyses. Although this was partially mitigated by the use of validated instruments and the abovementioned feasibility study supporting the reliability of telephone assessment for CHR-P symptoms and other psychopathology (Michel et al., 2014), these remain methodological limitations.

Finally, some potential confounding factors, such as medication use and previous or ongoing psychotherapeutic treatment, were not consistently controlled for and may have influenced the results.

4.3. Future directions

The ongoing efforts to map CHR-P within the HiTOP framework have demonstrated potential to enhance our understanding of CHR-P and general psychopathology, with particular attention to transdiagnostic and dimensional implications. In this context, the present thesis contributes by formulating hypotheses for future exploration of the integration of CHR-P and HiTOP and outlining the resulting reciprocal advantages. However, several open questions and challenges remain.

First, future research should address the integration of BSs within HiTOP with latent structure analysis, not only given their relevance in conversion prediction and CHR-P profile characterization, but also to strengthen the focus on cognitive processes within HiTOP research (Kotov et al., 2021). Concurrently to the exploration of BSs' placement within HiTOP, the internal structure of CHR-P symptoms should be investigated and empirically modeled, with a special focus on (i) the distinction between APSs and BSs, (ii) that between perceptive and non-perceptive CHR-P symptoms, and (iii) their potential interactions and hierarchical relationship. Further, comprehensive structural modeling of CHR-P manifestations within HiTOP should consider non-criteria-relevant symptoms – such as (attenuated) negative and affective symptoms – as these might shed light on the structure of early non-specific psychopathology and its interactions with transdiagnostic factors of mental health (Cowan et al., 2024; Guloksuz & van Os, 2018). Relatedly, future HiTOP-informed research should emphasize transdiagnostic factors directly, whether they are more related to psychopathology structure (e.g., core beliefs) or process (e.g., coping strategies, cognitive biases), to deepen our understanding of their placement and interactions. Such work could advance HiTOP towards becoming a fully dimensional model of psychopathology, including detailed descriptions of adaptive functioning along its different (super)spectra (HiTOP Research Group, 2023). In particular, further exploration of personality function-

ing within HiTOP is essential to bridging the gap between personality and non-personality psychopathology, and might help clarify the complex relationships between personality, psychosis, and CHR-P (Kerber et al., 2024).

Another essential step for future research is the integration of a time-sensitive, longitudinal perspective within HiTOP, both in terms of capturing the fluctuating, heterogenous presentation of CHR-P symptoms and of validating their long-term placement, including their associations with transdiagnostic factors. Regarding short-term symptom fluctuations, technological innovations such as Ecological Momentary Assessment could provide a real-time, ecologically valid assessment of CHR-P symptoms, whose integration into HiTOP could enhance its accuracy and representativeness (Michel et al., 2023).

Overall, integrating a time dimension and a developmental perspective into the HiTOP model would represent a shift from the current static, cross-sectional symptom classification toward a framework that captures the lived experience of psychopathology and its lifespan trajectories – one of the explicit goals of HiTOP research (Kotov et al., 2020). At the same time, this approach could improve the characterization of distinct profiles along the psychosis continuum and clarify individual differences in transdiagnostic correlates (e.g., personality and psychosocial functioning, core beliefs, coping strategies, cognitive biases). First, this might advance phenotyping and risk stratification in CHR-P research. Second, future studies could then explore whether different profiles are associated with different outcomes or benefit from different intervention strategies, with the potential to inform evidence-based policy-making, prevention and treatment.

In summary, integrating a longitudinal perspective, individual variability in transdiagnostic factors, and real-time tracking technologies into HiTOP-informed CHR-P research represents a promising next step toward refining both models.

5. Conclusion

This thesis examined the ongoing paradigm shift towards a dimensional characterization of psychopathology, particularly psychosis-related psychopathology, as reflected in both the growing consensus on the concept of a psychosis spectrum and the introduction of dimensional models and ratings in the main diagnostic manuals (American Psychiatric Association, 2013; Kotov et al., 2020; World Health Organization, 2019). Further, this work analyzed the merits and shortcomings of the CHR-P paradigm, presenting the current debate on its conceptualization and its future in research, prevention, and intervention (McGorry et al., 2018; Schultze-Lutter et al., 2022; van Os & Guloksuz, 2017). Consequently, it explored the potential of the HiTOP framework to effectively address controversies surrounding the CHR-P paradigm, providing a comprehensive perspective that accounts for both its specific relation to psychosis and its transdiagnostic clinical relevance.

By emphasizing the continuity between psychopathology and healthy functioning, HiTOP shows particular promise for the exploration of these questions within community samples. Therefore, the findings from three publications on CHR-P symptoms in the community were presented and contextualized within the HiTOP model. Collectively, these findings illustrate the complexity and heterogeneity of CHR-P symptoms, their longitudinal course, and their associations with multiple transdiagnostic factors, such as core beliefs, coping strategies, personality functioning, and cognitive biases. Their reframing in HiTOP, synthetized in a hypothetical extension to the current HiTOP model, not only shows the potential of dimensional approaches to advance our understanding of CHR-P, but also highlights the unique contribution of CHR-P to refining HiTOP by shedding light on transdiagnostic interactions, developmental aspects, and cognitive processes.

Highlighting open questions and setting the stage for future efforts to extensively map CHR-P-related manifestations onto HiTOP, the present thesis underscores how their empirically driven integration could improve the characterization of CHR-P profiles, elucidate their transdiagnostic implications, and refine models of psychopathology to include both adaptive and maladaptive functioning. In conclusion, this work illustrates how integrating CHR-P into HiTOP holds the potential to bridge early intervention research in psychosis and broader models of psychopathology.

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Appendix

Table 2 *CHR-P symptoms and criteria*

Ultra-high risk (UHR) criteria according to the SIPS
A. 'Brief Intermittent Psychotic Symptoms' (BIPS) ➲ At least any 1 of the following SIPS P-items scored 6 'severe and psychotic' • P1 Unusual Thought Content / Delusional Ideas • P2 Suspiciousness / Persecutory Ideas • P3 Grandiose Ideas • P4 Perceptual Abnormalities / Hallucinations • P5 Disorganized Communication ➲ First appearance in the past three months ➲ Present for at least several minutes per day at a frequency of at least once per month but less than 7 days
B. 'Attenuated Positive Symptoms' (APS) ➲ At least any 1 of the following SIPS P-items scored 3 'moderate' to 5 'severe but not psychotic' • P1 Unusual Thought Content / Delusional Ideas • P2 Suspiciousness / Persecutory Ideas • P3 Grandiose Ideas • P4 Perceptual Abnormalities / Hallucinations • P5 Disorganized Communication ➲ First appearance within the past year or current rating one or more scale points higher compared to 12 months ago ➲ Symptoms have occurred at an average frequency of at least once per week in the past month
C. 'Genetic Risk and Deterioration' Syndrome (1) Patient meets criteria for Schizotypal Personality Disorder according to SIPS (2) Patient has 1st degree relative with a psychotic disorder (3) Patient has experienced >30% drop in global assessment of functioning (GAF) score over the last month compared to 12 months ago ➲ [1 and 3] or [2 and 3] or all are met.
Basic symptom (BS) criteria
Risk criterion 'Cognitive-Perceptive Basic Symptoms' (COPER) ➲ At least any 1 of the following BS with a SPI-A score of ≥3 within the last 3 months: • Thought interference (BS6; SPI-A C2) • Thought pressure (BS7; SPI-A D3) • Disturbance of receptive speech (BS5; SPI-A C4) • Thought perseveration (BS8; SPI-A O1)

- Thought blockages (BS9; SPI-A C3)
- Decreased ability to discriminate between ideas / perception and fantasy / true memories (BS10; SPI-A O2)
- Unstable ideas of reference (BS11; SPI-A D4)
- Derealisation (BS12; SPI-A O8)
- Visual perception disturbances (excluding hypersensitivity to light or blurred vision) (BS13; SPI-A O4, F3, D5)
- Acoustic perception disturbances (excluding hypersensitivity to sounds) (BS14=SPI-A O5, F5)

➲ First occurrence ≥12 months ago

High-risk criterion 'Cognitive Disturbances' (COGDIS)

➲ At least any 2 of the following BS with a SPI-A score of ≥3 within the last 3 months:

- Inability to divide attention (BS1; SPI-A B1)
- Captivation of attention by details of the visual field (BS2; SPI-A O7)
- Disturbances of abstract thinking (BS3; SPI-A O3)
- Disturbance of expressive speech (BS4; SPI-A C5)
- Disturbance of receptive speech (BS5; SPI-A C4)
- Thought interference (BS6; SPI-A C2)
- Thought pressure (BS7; SPI-A D3)
- Thought blockages (BS9; SPI-A C3)
- Unstable ideas of reference (BS11; SPI-A D4)

Figure 8 *Model of early phases of psychosis. [Not included due to copyright restrictions. For the original figure, see Resch & Michel (2021), in: Psychoserisikosyndrome im Kindes- und Jugendalter. In: Fegert et al. (Eds.), Psychiatrie und Psychotherapie des Kindes- und Jugendalters. Springer, Berlin, Heidelberg. https://doi.org/10.1007/978-3-662-49289-5_99-1]*

Publication 1

Full text

Three-year course of clinical high-risk symptoms for psychosis in the community: a latent class analysis.

Chantal Michel, Naweed Osman, Giulia Rinaldi, Benno G. Schimmelmann, Jochen Kindler & Frauke Schultze-Lutter

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Three-year course of clinical high-risk symptoms for psychosis in the community: a latent class analysis

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Abstract

Aims. Clinical high-risk for psychosis (CHR-P) states exhibit diverse clinical presentations, prompting a shift towards broader outcome assessments beyond psychosis manifestation. To elucidate more uniform clinical profiles and their trajectories, we investigated CHR-P profiles in a community sample.

Methods. Participants (N = 829; baseline age: 16–40 years) comprised individuals from a Swiss community sample who were followed up over roughly 3 years. latent class analysis was applied to CHR-P symptom data at baseline and follow-up, and classes were examined for demographic and clinical differences, as well as stability over time.

Results. Similar three-class solutions were yielded for both time points. Class 1 was mainly characterized by subtle, subjectively experienced disturbances in mental processes, including thinking, speech and perception (basic symptoms [BSs]). Class 2 was characterized by subthreshold positive psychotic symptoms (i.e., mild delusions or hallucinations) indicative of an ultra-high risk for psychosis. Class 3, the largest group (comprising over 90% of participants), exhibited the lowest probability of experiencing any psychosis-related symptoms (CHR-P symptoms). Classes 1 and 2 included more participants with functional impairment and psychiatric morbidity. Class 3 participants had a low probability of having functional deficits or mental disorders at both time points, suggesting that Class 3 was the healthiest group and that their mental health and functioning remained stable throughout the study period. While 91% of Baseline Class 3 participants remained in their class over time, most Baseline Classes 1 (74%) and Class 2 (88%) participants moved to Follow-up Class 3.

Conclusions. Despite some temporal fluctuations, CHR-P symptoms within community samples cluster into distinct subgroups, reflecting varying levels of symptom severity and risk profiles. This clustering highlights the largely distinct nature of BSs and attenuated positive symptoms within the community. The association of Classes 1 and 2 with Axis-I disorders and functional deficits emphasizes the clinical significance of CHR-P symptoms. These findings highlight the need for personalized preventive measures targeting specific risk profiles in community-based populations.

Introduction

Early detection and treatment of clinical high-risk for psychosis (CHR-P) states are not only relevant for preventing the onset of the first episode of psychosis, but also for achieving remission of CHR-P symptoms and other comorbidities, and for avoiding impairments in psychosocial functioning (Addington *et al.*, 2019; Caballero *et al.*, 2023; Campion *et al.*, 2012; Schmidt *et al.*, 2015; Schultze-Lutter and Meisenzahl, 2023; Worthington and Cannon, 2021). In clinical samples, many CHR-P patients who do not develop psychosis within follow-up – so-called 'non-converters' – do not experience remission from CHR-P symptoms. Furthermore, they continue to suffer from non-psychotic mental disorders at follow-up – mainly mood and anxiety disorders – (Beck *et al.*, 2019), which are the most frequent comorbid disorders reported for CHR-P states at baseline (Solmi *et al.*, 2023). Irrespective of comorbidities, half of clinical CHR-P samples show a poor psychosocial outcome (Carrión *et al.*, 2013; Lin *et al.*, 2015), even when CHR-P symptoms remit (Addington *et al.*, 2019), with CHR-P state at follow-up (either newly developed or maintained) being associated with significantly lower functioning

(Lin *et al.*, 2015; Michel *et al.*, 2018a; Schmidt *et al.*, 2015). Therefore, regardless of conversion, the CHR-P state itself clearly possesses clinical significance warranting support and care in help-seeking individuals (Fusar-Poli *et al.*, 2020; Ruhrmann *et al.*, 2010; Solmi *et al.*, 2023).

A challenge to the understanding of CHR-P states and their course is the heterogeneous clinical picture. This difficulty has been tackled by various methods, from identifying specific risk profiles linked to neural mechanisms, to building multivariate models that predict heterogeneous outcomes (Caballero *et al.*, 2023; Solmi *et al.*, 2023; Worthington and Cannon, 2021). A common method to parse out heterogeneity by way of clinical profiles is latent class analysis (LCA) (Healey *et al.*, 2018; Ryan *et al.*, 2018; Valmaggia *et al.*, 2013; van Tricht *et al.*, 2015). Considered a 'person-centred' approach to reduce heterogeneity, LCA operates on the notion of finding 'hidden' homogenous groups within heterogeneous populations (Rosato and Baer, 2012). Studies applying LCA to clinical CHR-P samples generally used baseline data only, and characterized groups by transition rates to psychosis, while other relevant outcomes (e.g., non-psychotic mental disorders) were not considered (Healey *et al.*, 2018; Ryan *et al.*, 2018; Valmaggia *et al.*, 2013; van Tricht *et al.*, 2015). They have reported between two and five classes differing in parameters included for class selection (e.g., only positive symptoms or additional negative symptoms, or neurophysiological parameters) (Healey *et al.*, 2018; Ryan *et al.*, 2018; Valmaggia *et al.*, 2013; van Tricht *et al.*, 2015). To date, no study has attempted to determine if and how people might change class membership between baseline and follow-up, or examined stability of classes over time. Moreover, earlier studies were carried out in selected samples of only, or mostly, help-seeking CHR-P patients defined exclusively by ultra-high risk (UHR) criteria, who commonly receive treatment (Healey *et al.*, 2018; Ryan *et al.*, 2018; Valmaggia *et al.*, 2013; van Tricht *et al.*, 2015) and who must therefore be assumed a non-representative minority of the CHR-P population. Consequently, the classes and natural course (i.e., potentially without treatment) of clinician-assessed CHR-P symptoms in the wider community using the whole spectrum of CHR-P criteria and symptoms, i.e., both UHR and basic symptom (BS) criteria (Schultze-Lutter *et al.*, 2015), is largely unknown.

To address this gap in knowledge (van Os *et al.*, 2021), the aims of this study were twofold. First, to ascertain CHR-P symptom-based classes of community participants using the whole spectrum of CHR-P symptoms (i.e., attenuated (APS) and brief intermittent psychotic symptoms (BIPS) and criteria-relevant BS), and to examine their clinical and socio-demographic correlates. Second, to explore the stability of these classes longitudinally; specifically, to determine how class membership itself might change, or how individuals might 'move between' baseline and follow-up classes.

Methods

Participants

The sample included participants from both the baseline and follow-up assessments of the 'Bern Epidemiological At-Risk' (BEAR) study (Schultze-Lutter *et al.*, 2018, 2021; for further details, see eTexts 1, 2). At baseline, we evaluated CHR-P symptoms and criteria in a representative random sample of the 16- to 40-year-old Bernese community ($N = 2{,}683$; response rate: 63.4%), using procedures comparable with clinical assessment (Schultze-Lutter *et al.*, 2015). A selected, CHR-P symptom-enriched sample ($N = 834$; response rate: 66.4%) was followed up approximately 3 years later, and only the $N = 829$ non-converters were included in the present analyses (Schultze-Lutter *et al.*, 2021). For a detailed overview of the participant selection process, including reasons for exclusion, please refer to Fig. 1. Participation was voluntary and required informed consent at each time point. The human research ethics committee of Canton Bern approved the study (ID PB_2018-00132).

Assessments

CHR-P symptoms (eTable 1) were assessed using semi-structured interviews with good interrater reliability (McGlashan *et al.*, 2010; Schultze-Lutter *et al.*, 2007). The Structured Interview for Psychosis-Risk Syndromes (SIPS) (McGlashan *et al.*, 2010) was used for UHR symptoms, i.e., five APS/BIPS, and the Schizophrenia Proneness Instrument, Adult version (Schultze-Lutter *et al.*, 2007) for the 14 BS included in the two BS criteria (Schultze-Lutter *et al.*, 2015). For the present analyses, CHR-P symptoms were defined by the presence of APS or BIPS, and/or criteria-relevant BS at baseline, irrespective of the onset/worsening and/or frequency requirements of related CHR-P criteria. The five positive SIPS-items were recoded into binary items: 1 (presence) was assigned to scores between 3 and 6 (indicating presence of APS or BIPS) and 0 (absence) to scores between 0 and 2 (indicating absence of APS and BIPS). Similarly, BS-scores between 1 and 6, and 8 (indicating presence of BS) were recoded as 1 (presence), while 0 (absence) was assigned to BS-scores of 0, 9 and 7 (respectively indicating absence of BS, their only questionable presence, or that the symptom has always been present in the same frequency, making it a trait feature, not a BS). Present DSM-IV non-substance-related axis-I disorders, including affective, anxiety (including specific phobia), eating, somatoform, obsessive-compulsive and post-traumatic stress disorder were assessed using the Mini-International Neuropsychiatric Interview (Sheehan *et al.*, 1998), which was previously successfully applied in telephone interviews of community samples, demonstrating good reliability, concurrent and predictive validity (Schultze-Lutter *et al.*, 2018; Sheehan *et al.*, 1998; Wang *et al.*, 2006).

Clinician-rated global level of psychosocial functioning, independent of overall symptom severity, was estimated on the Social and Occupational Functioning Assessment Scale (SOFAS; American Psychiatric Association (APA), 1994), which showed good psychometric properties, including good interrater reliability and construct validity (Hilsenroth *et al.*, 2000; Rybarczyk, 2011). Over a 0–100 range, lower SOFAS-scores represent lower functioning, with a score of $\leq$70 indicating presence of a functional deficit (American Psychiatric Association (APA), 1994; Morosini *et al.*, 2000; Schimmelmann *et al.*, 2015; Michel *et al.*, 2018b).

Statistical analyses

Analyses were conducted in R (Version 4.2) and RStudio (Version 2022.07.0). To identify the best fitting LCA model for each assessment point, different models were estimated, and subsequent classes were added using the R package poLCA (Linzer and Lewis, 2011). For each model, the Akaike Information Criterion (AIC), the Bayesian Information Criterion (BIC) and the relative entropy were calculated. Lower AIC and BIC values indicate better fit, and higher entropy values indicate better accuracy with the defined classes (Weller *et al.*, 2020). After identifying the best-fitting LCA

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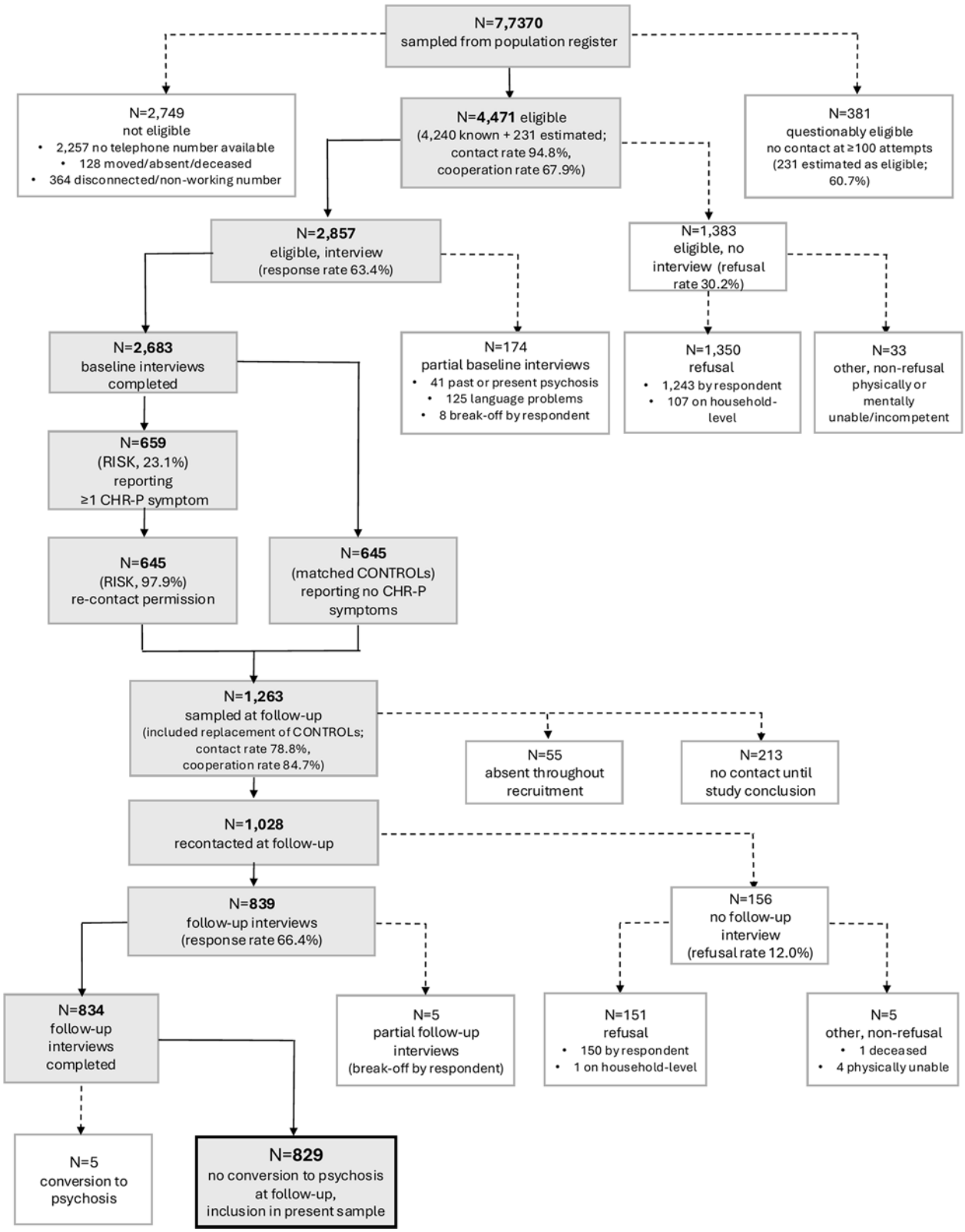

Figure 1. Flowchart of participant recruitment, selection and follow-up in the BEAR study.

model for both baseline and follow-up data, each individual was assigned to a specific class based on the probabilities of class membership obtained from the analysis.

Differences between classes regarding ratio data and categorical variables were tested using ANOVAs and chi-squared tests, respectively. Effect sizes were calculated using eta-square and Cramer's

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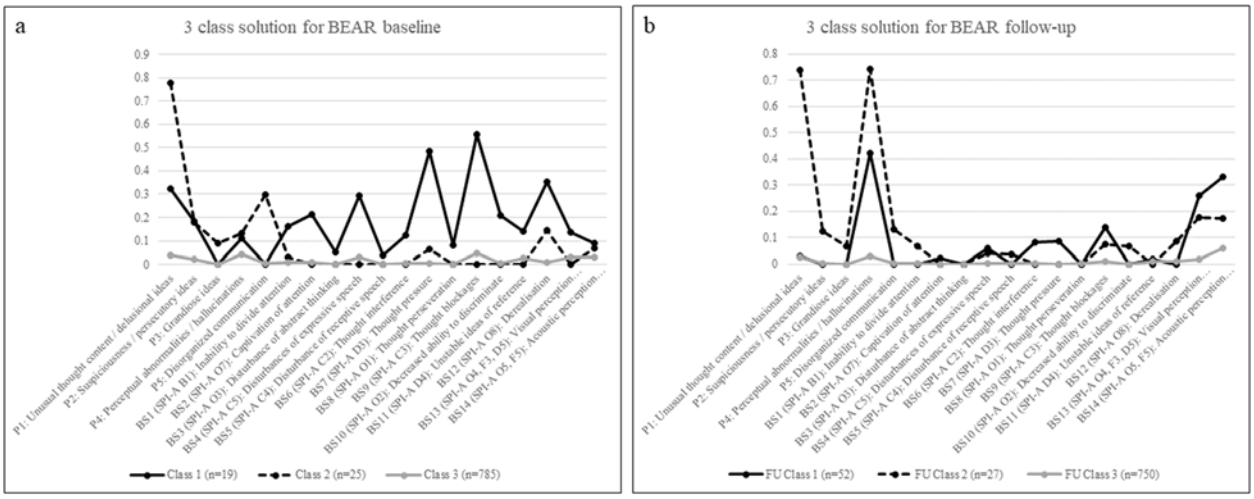

Figure 2. Latent class profiles of basic symptoms and (attenuated) psychotic symptoms at baseline (a) and follow-up (b).

V. Significant ANOVAs were additionally tested using pairwise Bonferroni corrected comparisons. For significant chi-squared tests, the standardized residuals ($\geq|1.96|$) were calculated as a measure of significant cell difference between observed and expected values.

Results

Sample characteristics at baseline and follow-up

The mean follow-up time was 40.60 months (SD = 8.35, Mdn = 39.00, range: 21.00–68.00). Participants were on average 29.8 years old at baseline and 33.3 years old at follow-up (eTable 2). At both time points, the sample was 53.2% female, predominantly Swiss and in regular employment (>95%), with most participants (84.1%) pursuing or holding moderate to high educational qualifications (ISCED $\geq$ 4); roughly half of the sample was single (eTable 2). At both time points, the proportion of participants with a functional deficit remained stable at around 7%, while the rate of axis-I disorders significantly decreased from 17.0% at baseline to 13.3% at follow-up, primarily due to reductions in affective and other disorders (e.g., eating disorders, somatoform disorders; eTable 2). All symptoms decreased in number over time or maintained a low frequency (eTable 2), except for perceptual symptoms, which showed an increase at follow-up (eTable 2).

LCA at baseline

At baseline, three LCA models were tested and compared by goodness of fit.

Although a two-class solution showed the best BIC, which is generally considered the most reliable fit statistic in LCA (Sinha *et al.*, 2021; Weller *et al.*, 2020), its AIC and entropy value were the poorest. Therefore, the two-class solution was discarded.

Overall, the best fitting model was a three-class solution (eTable 3), showing the lowest AIC, the second-lowest BIC and the second-highest entropy (Fig. 2a), indicating clear separation between the classes. Classes 1 and 2 of the three-class solution were mostly characterized by a high probability of BS and of APS/BIPS, respectively. Class 3 was characterized by a low probability of any CHR-P symptom (Fig. 2a).

Baseline characteristics of the Baseline Classes

Baseline Class 1 was the smallest (n = 19, 2.3%), including participants who, compared to the other classes, showed the highest rate of lower education level as well as unemployment or sheltered/temporary employment. Additionally, they showed high rates of functional impairment and were the most affected by axis-I disorders (Table 1). In comparison, Baseline Class 2 was slightly bigger (n = 25, 3.0%), comprising individuals with similar rates of functional impairment, but fewer, although still frequent, axis-I disorders (Table 1). Baseline Class 2 members were also the oldest, and least likely to be single. Finally, Baseline Class 3 was the largest (n = 785, 94.7%), characterized by the highest rate of regular full-time/part-time employment, and the lowest rates of psychosocial deficits, axis-I disorders and divorce (Table 1).

There were no differences between the Baseline Classes in terms of sex, nationality or family history of mental disorders.

Follow-up characteristics of the Baseline Classes

At follow-up, participants in Baseline Class 1 continued to show the highest rates of unemployment or sheltered/temporary employment, lower education level and axis-I disorders. Newly, they showed the highest rates of functional impairment (Table 2). Baseline Class 2 remained the oldest, showing intermediate rates of functional deficits and any axis-I disorder. Among its members, rates of regular full- or part-time employment decreased compared to baseline, while other types of employment were now highly frequent (Table 2). Finally, participants in Baseline Class 3 continued to report the highest levels of education and regular full- or part-time employment, as well as the lowest rates of axis-I disorders and functional impairment (Table 2).

New LCA for the follow-up time point

For the follow-up data, three new LCA models were tested and compared by goodness of fit.

Again, a three-class solution was the best fitting model, showing the lowest AIC and BIC values, yet had relatively low relative entropy (see eTable 4, Fig. 2b), indicating higher within-classes homogeneity at this time point compared to baseline.

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Table 1. Baseline socio-demographic and clinical characteristics of the three Baseline Classes ($N = 829$)

	Baseline Class 1 ($n = 19$)		Baseline Class 2 ($n = 25$)		Baseline Class 3 ($n = 785$)		
	n	% [significant standardized residuals]	*n*	% [significant standardized residuals]	*n*	% [significant standardized residuals]	Statistics
Age (mean ± SD, median, range)	27.53 ± 8.82, 23, 18–39		**35.08 ± 5.09, 37, 19–40**		29.71 ± 7.66, 32. 15–41		$F = 6.877$, df = 2, ***p* = 0.0011**, $\eta^2 = 0.016$
							Bonferroni adjusted:
							Class 1 vs. Class 2:* p = *0.004*, *Class 2 vs. Class 3:* p = *0.002
							Class 1 vs. Class 3: p = *0.651*
Sex (male)	6	31.6	15	60.0	367	46.8	$\chi^2 = 3.5183$, df = 2, $p = 0.172$, Cramer's $V = 0.065$
Nationality (Swiss)	19	100.0	24	96.0	755	96.2	$\chi^2 = 0.75755$, df = 2, $p = 0.685$, Cramer's $V = 0.030$
Highest education							
ISCED level 0–2	1	5.3	0	0.0	14	1.8	$\chi^2 = 11.153$, df = 8, $p = 0.193$, Cramer's $V = 0.082$
ISCED level 3	**6**	**31.6 [2.21]**	2	8.0	109	13.9	
ISCED level 4–5	9	47.4	16	64.0	367	46.8	
ISCED level 7	3	15.8	7	28.0	287	36.6	
ISCED level 8	0	0.0	0	0.0	8	1.0	
Current employment							
Unemployed	**3**	**15.8 [3.72]**	1	4.0	**17**	**2.2 [−2.84]**	$\chi^2 = 119.86$, df = 8, ***p* < 0.001**, Cramer's $V = 0.269$
Sheltered employment	**1**	**5.3 [6.53]**	0	0.0	**0**	**0.0 [−4.23]**	
Temporary employment	**2**	**10.5 [4.31]**	0	0.0	**6**	**0.8 [−2.50]**	
Regular full- and part-time employment	**12**	**63.2 [−7.69]**	24	96.0	**762**	**97.1 [5.19]**	
Other	**1**	**5.3 [6.53]**	0	0.0	**0**	**0.0 [−4.23]**	
Marital status							
Single	12	63.2	**9**	**36.0 [−2.12]**	449	57.2	$\chi^2 = 14.776$, df = 10, $p = 0.141$, Cramer's $V = 0.094$
Married/civil union	7	36.8	13	52.0	315	40.1	
Separated	0	0.0	1	4.0	10	1.3	
Divorced	0	0.0	**2**	**8.0 [3.16]**	**8**	**1.0 [−2.09]**	
Widowed	0	0.0	0	0.0	1	0.1	
Other	0	0.0	0	0.0	2	0.3	
Family history of psychiatric disorders	11	57.9	12	48.0	329	42.1	$\chi^2 = 2.205$, df = 2, $p = 0.332$ Cramer's $V = 0.052$
SOFAS deficit (SOFAS < 70)	**9**	**47.4 [6.69]**	**12**	**48.0 [7.82]**	**41**	**7.5 [−10.43]**	$\chi^2 = 108.79$, df = 2, ***p* < 0.001**, Cramer's $V = 0.362$
Any current axis-I disorder	**11**	**57.9 [4.80]**	**10**	**40.0 [3.11]**	**120**	**15.3 [−5.57]**	$\chi^2 = 33.512$, df = 2, ***p* < 0.001**, Cramer's $V = 0.201$
Any affective disorder	**9**	**47.4 [7.30]**	**7**	**28.0 [4.42]**	**38**	**4.8 [−8.25]**	$\chi^2 = 74.638$, df = 2, ***p* < 0.001**, Cramer's $V = 0.300$

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Table 1. (*Continued.*)

	Baseline Class 1 ($n = 19$)		Baseline Class 2 ($n = 25$)		Baseline Class 3 ($n = 785$)		
	n	% [significant standardized residuals]	*n*	% [significant standardized residuals]	*n*	% [significant standardized residuals]	Statistics
Any anxiety disorder (including specific phobia)	**8**	**42.1 [4.17]**	**8**	**32.0 [3.21]**	**81**	**10.3 [−5.23]**	$\chi^2 = 28.423$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.185$
Other disorder	**6**	**31.6 [5.89]**	**5**	**20.0 [3.90]**	**25**	**3.2 [−6.91]**	$\chi^2 = 51.218$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.249$
CHR-P symptoms							
P1: Unusual thought content/delusional ideas	**7**	**36.8 [4.65]**	**23**	**92.0 [15.63]**	**37**	**4.7 [−15.03]**	$\chi^2 = 270.14$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.571$
P2:Suspiciousness/persecutory ideas	**4**	**21.1 [4.21]**	**7**	**28.0 [6.77]**	**18**	**2.3 [−7.98]**	$\chi^2 = 65.18$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.280$
P3: Grandiose ideas	0	0.0	**3**	**12.0 [8.44]**	**1**	**0.1 [−6.23]**	$\chi^2 = 71.22$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.293$
P4: Perceptual abnormalities/hallucinations	2	10.5	**6**	**24.0 [4.38]**	**34**	**4.3 [−4.08]**	$\chi^2 = 20.693$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.158$
P5: Disorganized communication	0	0.0	**10**	**40.0 [14.55]**	**5**	**0.6 [−10.70]**	$\chi^2 = 211.66$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.505$
BS1: Inability to divide attention (SPI-A B1)	**4**	**21.1 [10.58]**	**1**	**4.0 [1.96]**	**1**	**0.1 [−8.56]**	$\chi^2 = 116.90$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.376$
BS2: Captivation of attention by details of the visual field (SPI-A O7)	**5**	**26.3 [9.63]**	0	0.0	**6**	**0.8 [−5.98]**	$\chi^2 = 92.851$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.335$
BS3: Disturbances of abstract thinking (SPI-A O3)	**1**	**5.3 [3.60]**	0	0.0	**2**	**0.3 [−2.17]**	$\chi^2 = 12.999$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.125$
BS4: Disturbances of expressive speech (SPI-A C5)	**6**	**31.6 [6.35]**	0	0.0	**26**	**3.3 [−3.46]**	$\chi^2 = 40.976$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.222$
BS5: Disturbances of receptive speech (SPI-A C4)	**1**	**5.3 [6.53]**	0	0.0	**0**	**0.0 [−4.23]**	$\chi^2 = 42.683$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.227$
BS6: Thought interference (SPI-A C2)	**3**	**15.8 [6.69]**	0	0.0	**5**	**0.6 [−4.08]**	$\chi^2 = 44.818$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.232$
BS7: Thought pressure (SPI-A D3)	**11**	**57.9 [16.86]**	**3**	**12.0 [3.42]**	**4**	**0.5 [−13.87]**	$\chi^2 = 299.32$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.601$
BS8: Thought perseveration (SPI-A O1)	**2**	**10.5 [9.24]**	0	0.0	**0**	**0.0 [−5.98]**	$\chi^2 = 85.469$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.321$
BS9: Thought blockages (SPI-A C3)	**11**	**57.9 [9.72]**	0	0.0	**38**	**4.8 [−5.52]**	$\chi^2 = 95.51$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.339$
BS10: Decreased ability to discriminate between ideas & perception, fantasy & true memories (SPI-A O2)	**5**	**26.3 [10.74]**	0	0.0	**4**	**0.5 [−6.76]**	$\chi^2 = 115.33$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.373$

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Table 1. (*Continued.*)

	Baseline Class 1 ($n = 19$)		Baseline Class 2 ($n = 25$)		Baseline Class 3 ($n = 785$)		
	n	% [significant standardized residuals]	*n*	% [significant standardized residuals]	*n*	% [significant standardized residuals]	Statistics
BS11: Unstable ideas of reference (SPI-A D4)	**3**	**15.8 [3.29]**	0	0.0	22	2.8	$\chi^2 = 11.499$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.118$
BS12: Derealization (SPI-A O8)	**9**	**47.4 [12.92]**	**5**	**20.0 [5.82]**	**6**	**0.8 [−13.06]**	$\chi^2 = 205.00$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.497$
BS13: Visual perception disturbances (SPI-A O4, F3, D5)	**3**	**15.8 [3.03]**	0	0.0	25	3.2	$\chi^2 = 9.933$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.110$
BS14: Acoustic perception disturbances (SPI-A O5, F5)	2	10.5	2	8.0	25	3.2	$\chi^2 = 4.509$, df = 2, $p = 0.105$, Cramer's $V = 0.074$

Note: SOFAS: Social and Occupational Functioning Assessment Scale.
In **[bold]**, cells with standardized residuals $\geq$|1.96|. This equals significant deviation from the expected cell frequency. An adjusted residual of 1.96 indicates that the number of cases in that cell is significantly larger than would be expected if the null hypothesis were true, with a significance level of 0.05. An adjusted residual that is <-1.96 indicates that the number of cases in that cell is significantly smaller than would be expected if the null hypothesis were true.
P: positive-symptom scale; BS: basic symptom.

Table 2. Follow-up socio-demographic and clinical characteristics of the three Baseline Classes ($N = 829$)

	Baseline Class 1 ($n = 19$)		Baseline Class 2 ($n = 25$)		Baseline Class 3 ($n = 785$)		
	n	% [significant standardized residuals]	*n*	% [significant standardized residuals]	*n*	% [significant standardized residuals]	Statistics
Age (mean ± SD, median, range)	30.95 ± 9.1, 26, 21–45		**38.64 ± 5.15, 39, 22–45**		33.14 ± 7.75, 35, 19–45		$F = 7.029$, df = 2, $\mathbf{p < 0.001}$, $\eta^2 = 0.017$
							Bonferroni adjusted:
							Class 1 vs. Class 2:* p = *0.003*, *Class 2 vs. Class 3:* p = *0.001
							Class 1 vs. Class 3: p = *0.668*
Sex (male)	6	31.6	15	60.0	367	46.8	$\chi^2 = 3.5183$, df = 2, $p = 0.172$, Cramer's $V = 0.065$
Nationality (Swiss)	19	100.0	24	96.0	762	97.1	$\chi^2 = 0.678$, df = 2, $p = 0.712$, Cramer's $V = 0.029$
Highest education							
ISCED level 0–2	1	5.3	0	0.0	14	1.8	$\chi^2 = 11.153$, df = 8, $p = 0.193$, Cramer's $V = 0.082$
ISCED level 3	**6**	**31.6 [2.21]**	2	8.0	109	13.9	
ISCED level 4–5	9	47.4	16	64.0	367	46.8	
ISCED level 7	3	15.8	7	28.0	287	36.6	
ISCED level 8	0	0.0	0	0.0	8	1.0	
Current employment							
Unemployed	**3**	**15.8 [3.98]**	1	4.0	**15**	**1.9 [−3.10]**	$\chi^2 = 67.632$, df = 8, $\mathbf{p < 0.001}$, Cramer's $V = 0.202$
Sheltered employment	**2**	**10.5 [6.39]**	0	0.0	**2**	**0.3 [−4.00]**	
Temporary employment	0	0.0	1	4.0	6	0.8	

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Table 2. (*Continued.*)

	Baseline Class 1 ($n = 19$)		Baseline Class 2 ($n = 25$)		Baseline Class 3 ($n = 785$)		
	n	% [significant standardized residuals]	*n*	% [significant standardized residuals]	*n*	% [significant standardized residuals]	Statistics
Regular full- and part-time employment	**14**	**73.7 [−4.94]**	**22**	**88.0 [−2.02]**	**759**	**96.7 [4.84]**	
Other	0	0.0	**1**	**4.0 [2.58]**	3	0.4	
Marital status							
Single	12	63.2	8	32.0	404	51.5	$\chi^2 = 13.519$, df = 10, $p = 0.109$, Cramer's $V = 0.090$
Married/civil union	7	36.8	13	52.0	348	44.3	
Separated	0	0.0	**2**	**8.0 [2.49]**	12	1.5	
Divorced	0	0.0	2	8.0	17	2.2	
Widowed	0	0.0	0	0.0	2	0.3	
Other	0	0.0	0	0.0	2	0.3	
Family history of psychiatric disorders	12	63.2	12	48.0	388	49.4	$\chi^2 = 1.637$, df = 2, $p = 0.802$, Cramer's $V = 0.031$
SOFAS deficit (SOFAS < 70)	**7**	**36.8 [5.16]**	**5**	**20.0 [2.59]**	**46**	**5.9 [−5.42]**	$\chi^2 = 34.065$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.203$
Any current axis-I disorder	**9**	**47.4 [4.43]**	5	20.0	**96**	**12.2 [−3.73]**	$\chi^2 = 33.512$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.201$
Any affective disorder	**6**	**31.6 [8.38]**	**4**	**16.0 [4.50]**	**10**	**1.3 [−9.02]**	$\chi^2 = 92.579$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.334$
Any anxiety disorder (including specific phobia)	**6**	**31.6 [2.70]**	3	12.0	89	11.3	$\chi^2 = 7.292$, df = 2, $\mathbf{p = 0.026}$, Cramer's $V = 0.094$
Any other disorder	**2**	**10.5 [3.18]**	**2**	**8.0 [2.63]**	**9**	**1.1 [−4.13]**	$\chi^2 = 17.482$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.145$
CHR-P symptoms							
P1: Unusual thought content/delusional ideas	2	10.5	**5**	**20.0 [3.33]**	**37**	**4.7 [−3.22]**	$\chi^2 = 17.482$, df = 2, $\mathbf{p = 0.002}$, Cramer's $V = 0.122$
P2:Suspiciousness/persecutory ideas	0	0.0	0	0.0	6	0.8	$\chi^2 = 0.339$, df = 2, $p = 0.844$, Cramer's $V = 0.020$
P3: Grandiose ideas	0	0.0	**2**	**8.0 [8.03]**	**0**	**0.0 [−5.98]**	$\chi^2 = 64.476$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.279$
P4: Perceptual abnormalities/hallucinations	**5**	**26.3 [2.31]**	**6**	**24.0 [2.27]**	**75**	**9.6 [−3.27]**	$\chi^2 = 10.753$, df = 2, $\mathbf{p = 0.005}$, Cramer's $V = 0.114$
P5: Disorganized communication	0	0.0	**3**	**12.0 [7.47]**	**2**	**0.3 [−5.47]**	$\chi^2 = 55.87$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.260$
BS1: Inability to divide attention (SPI-A B1)	0	0.0	0	0.0	4	0.5	$\chi^2 = 0.225$, df = 2, $p = 0.894$, Cramer's $V = 0.016$
BS2: Captivation of attention by details of the visual field (SPI-A O7)	0	0.0	0	0.0	2	0.3	$\chi^2 = 0.112$, df = 2, $p = 0.945$, Cramer's $V = 0.012$
BS3: Disturbances of abstract thinking (SPI-A O3)	0	0.0	0	0.0	0	0.0	—

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Table 2. (*Continued.*)

	Baseline Class 1 ($n = 19$)		Baseline Class 2 ($n = 25$)		Baseline Class 3 ($n = 785$)		
	n	% [significant standardized residuals]	*n*	% [significant standardized residuals]	*n*	% [significant standardized residuals]	Statistics
BS4: Disturbances of expressive speech (SPI-A C5)	1	5.3	0	0.0	8	1.0	$\chi^2 = 3.395$, df = 2, $p = 0.183$, Cramer's $V = 0.064$
BS5: Disturbances of receptive speech (SPI-A C4)	0	0.0	**1**	**4.0 [2.58]**	3	0.4	$\chi^2 = 6.698$, df = 2, $\mathbf{p = 0.035}$, Cramer's $V = 0.090$
BS6: Thought interference (SPI-A C2)	1	5.3	0	0.0	8	1.0	$\chi^2 = 3.395$, df = 2, $p = 0.183$, Cramer's $V = 0.064$
BS7: Thought pressure (SPI-A D3)	0	0.0	1	4.0	8	1.0	$\chi^2 = 2.218$, df = 2, $p = 0.330$ Cramer's $V = 0.052$
BS8: Thought perseveration (SPI-A O1)	0	0.0	0	0.0	1	0.1	$\chi^2 = 0.056$, df = 2, $p = 0.972$, Cramer's $V = 0.008$
BS9: Thought blockages (SPI-A C3)	**3**	**15.8 [3.39]**	1	4.0	**20**	**2.5 [−2.52]**	$\chi^2 = 11.683$, df = 2, $\mathbf{p = 0.003}$, Cramer's $V = 0.119$
BS10: Decreased ability to discriminate between ideas & perception, fantasy & true memories (SPI-A O2)	0	0.0	0	0.0	2	0.3	$\chi^2 = 0.112$, df = 2, $p = 0.945$, Cramer's $V = 0.012$
BS11: Unstable ideas of reference (SPI-A D4)	0	0.0	0	0.0	9	1.1	$\chi^2 = 0.51$, df = 2, $p = 0.775$, Cramer's $V = 0.025$
BS12: Derealization (SPI-A O8)	**3**	**15.8 [6.26]**	1	4.0	**5**	**0.6 [−5.27]**	$\chi^2 = 41.702$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.224$
BS13: Visual perception disturbances (SPI-A O4, F3, D5)	2	10.5	3	12.0	38	4.5	$\chi^2 = 3.652$, df = 2, $p = 0.161$, Cramer's $V = 0.066$
BS14: Acoustic perception disturbances (SPI-A O5, F5)	2	10.5	2	8.0	75	9.6	$\chi^2 = 0.090$, df = 2, $p = 0.956$, Cramer's $V = 0.010$

Note: SOFAS: Social and Occupational Functioning Assessment Scale.
In **[bold]**, cells with standardized residuals $\geq|1.96|$. This equals significant deviation from the expected cell frequency. An adjusted residual of 1.96 indicates that the number of cases in that cell is significantly larger than would be expected if the null hypothesis were true, with a significance level of 0.05. An adjusted residual that is <-1.96 indicates that the number of cases in that cell is significantly smaller than would be expected if the null hypothesis were true.
P: positive-symptom scale; BS: basic symptom.

Follow-up characteristics of Follow-up Classes

Overall, Follow-up Class 1 (6.3% of sample) resembled Baseline Class 1, showing the highest rates of lower education and axis-I disorders. However, Follow-up Class 1 members showed only an intermediate rate of functional deficits and had the highest rate of separated persons (Table 3). With the exception of four BS, they showed a high likelihood of perceptual and cognitive BS, and of perceptual abnormalities/hallucinations (P4).

Follow-up Class 2 (3.3% of sample) partially resembled Baseline Class 2, showing an intermediate rate of axis-I disorders and the highest probability of all APS/BIPS at follow-up (Table 3). In contrast with Baseline Class 2, members of Follow-up Class 2 additionally had the highest probability of exhibiting four BS (inability to divide attention, disturbance of receptive speech, derealization and decreased ability to discriminate between ideas & perception, fantasy & true memories), as well as an elevated rate of visual perception disturbances, which was, however, still lower than in Follow-up Class 1. Further, they showed the highest rates of psychosocial deficits among Follow-up Classes, as well as the lowest rate of regular full- and part-time employment, and of married persons (Table 3). Overall, Follow-up Class 2 had a moderate educational level.

Aligning with Baseline Class 3, Follow-up Class 3 was the largest (90.5% of sample), showing a low probability of CHR-P symptoms (Fig. 2b), along with the lowest rates of psychosocial deficits and axis-I disorders among Follow-up Classes. Moreover, Follow-up Class 3 had the highest rate of regular employment, the lowest divorce rate and, newly, the highest educational level (Table 3).

Finally, similarly to Baseline Classes, the Follow-up Classes did not differ in distribution of sex, nationality, or family history of mental disorders, and, additionally, also not in age.

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Table 3. Follow-up socio-demographic and clinical characteristics of the three Follow-up Classes ($N = 829$)

	Follow-up Class 1 ($n = 52$)		Follow-up Class 2 ($n = 27$)		Follow-up Class 3 ($n = 750$)		
	n	% [significant standardized residuals]	*n*	% [significant standardized residuals]	*n*	% [significant standardized residuals]	Statistics
Age (mean ± SD, median, range)	33.38 ± 7.98, 35, 19–44		32.33 ± 8.28, 33, 19–43		33.27 ± 7.75, 35, 19–44		$F = 0.199$, df = 2, $p = 0.820$, $\eta^2 = 0.000$
Sex (male)	18	34.6	12	44.4	358	47.7	$\chi^2 = 3.423$, df = 2, $p = 0.181$, Cramer's $V = 0.064$
Nationality (Swiss)	52	100.0	26	96.3	727	96.9	$\chi^2 = 1.692$, df = 2, $p = 0.429$, Cramer's $V = 0.045$
Highest education							
ISCED level 0–2	**4**	**7.7 [3.29]**	1	3.7	**10**	**1.3 [−3.17]**	$\chi^2 = 24.191$, df = 8, $\mathbf{p = 0.002}$, Cramer's $V = 0.121$
ISCED level 3	7	13.5	3	11.1	107	14.3	
ISCED level 4–5	30	57.7	**19**	**70.4 [2.44]**	**343**	**45.7 [−2.76]**	
ISCED level 7	**11**	**21.2 [−2.28]**	**4**	**14.8 [−2.31]**	**282**	**37.6 [3.28]**	
ISCED level 8	0	0.0	0	0.0	8	1.1	
Current employment							
Unemployed	2	3.8	2	7.4	15	2.0	$\chi^2 = 16.527$, df = 8, $\mathbf{p = 0.035}$, Cramer's $V = 0.121$
Sheltered employment	0	0.0	0	0.0	4	0.5	
Temporary employment	0	0.0	1	3.7	6	0.8	
Regular full- and part-time employment	49	94.2	**23**	**85.2 [−2.85]**	**723**	**95.9 [2.24]**	
Other	1	1.9	**1**	**3.7 [2.46]**	**2**	**0.5 [−2.76]**	
Marital status							
Single	24	46.2	18	66.7	382	50.9	$\chi^2 = 16.408$, df = 10, $p = 0.089$, Cramer's $V = 0.099$
Married/civil union	22	42.3	**7**	**25.9 [−1.96]**	339	45.2	
Separated	**3**	**5.8 [2.36]**	0	0.0	11	1.5	
Divorced	3	5.8	2	7.4	**14**	**1.9 [−2.52]**	
Widowed	0	0.0	0	0.0	2	0.3	
Other	0	0.0	0	0.0	2	0.3	
Family history of psychiatric disorders	28	53.8	14	51.9	370	49.3	$\chi^2 = 0.812$, df = 2, $p = 0.937$, Cramer's $V = 0.022$
SOFAS deficit (SOFAS < 70)	**8**	**15.4 [2.45]**	**8**	**29.6 [4.69]**	**42**	**5.6 [−4.86]**	$\chi^2 = 29.127$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.187$
Any current axis-I disorder	**19**	**36.5 [5.11]**	**8**	**29.6 [2.55]**	**83**	**11.1 [−5.76]**	$\chi^2 = 33.907$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.202$
Any affective disorder	**4**	**7.7 [2.56]**	**5**	**18.5 [5.55]**	**11**	**1.5 [−5.47]**	$\chi^2 = 38.756$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.216$
Any anxiety disorder (including specific phobia)	**17**	**32.7 [4.81]**	**8**	**29.6 [2.91]**	**73**	**9.7 [−5.74]**	$\chi^2 = 33.081$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.200$
Any other disorder	2	3.8	**2**	**7.4 [2.48]**	**9**	**1.2 [−2.63]**	$\chi^2 = 8.371$, df = 2, $\mathbf{p = 0.015}$, Cramer's $V = 0.100$

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Table 3. (*Continued.*)

	Follow-up Class 1 ($n = 52$)		Follow-up Class 2 ($n = 27$)		Follow-up Class 3 ($n = 750$)		
	n	% [significant standardized residuals]	*n*	% [significant standardized residuals]	*n*	% [significant standardized residuals]	Statistics
CHR-P symptoms							
P1: Unusual thought content/delusional ideas	1	1.9	**23**	**85.2 [18.82]**	**20**	**2.7 [−10.45]**	$\chi^2 = 354.36$, df = 2, $\boldsymbol{p < 0.001}$, Cramer's $V = 0.654$
P2:Suspiciousness/persecutory ideas	0	0.0	**4**	**14.8 [8.78]**	**2**	**0.3 [−4.78]**	$\chi^2 = 77.172$, df = 2, $\boldsymbol{p < 0.001}$, Cramer's $V = 0.305$
P3: Grandiose ideas	0	0.0	**2**	**7.4 [7.72]**	**0**	**0.0 [−4.36]**	$\chi^2 = 59.551$, df = 2, $\boldsymbol{p < 0.001}$, Cramer's $V = 0.268$
P4: Perceptual abnormalities/hallucinations	**31**	**59.6 [12.03]**	**22**	**81.5 [12.32]**	**33**	**4.4 [−17.38]**	$\chi^2 = 311.23$, df = 2, $\boldsymbol{p < 0.001}$, Cramer's $V = 0.613$
P5: Disorganized communication	0	0.0	**4**	**14.8 [9.70]**	**1**	**0.1 [−5.38]**	$\chi^2 = 94.04$, df = 2, $\boldsymbol{p < 0.001}$, Cramer's $V = 0.337$
BS1: Inability to divide attention (SPI-A B1)	0	0.0	**2**	**7.4 [5.28]**	**2**	**0.3 [−2.76]**	$\chi^2 = 27.944$, df = 2, $\boldsymbol{p < 0.001}$, Cramer's $V = 0.184$
BS2: Captivation of attention by details of the visual field (SPI-A O7)	**2**	**3.8 [5.47]**	0	0.0	**0**	**0.0 [−4.36]**	$\chi^2 = 29.957$, df = 2, $\boldsymbol{p < 0.001}$, Cramer's $V = 0.190$
BS3: Disturbances of abstract thinking (SPI-A O3)	0	0.0	0	0.0	0	0.0	—
BS4: Disturbances of expressive speech (SPI-A C5)	**5**	**9.6 [6.13]**	1	3.7	**3**	**0.4 [−5.87]**	$\chi^2 = 40.238$, df = 2, $\boldsymbol{p < 0.001}$, Cramer's $V = 0.220$
BS5: Disturbances of receptive speech (SPI-A C4)	0	0.0	**1**	**3.7 [2.46]**	3	0.4	$\chi^2 = 6.193$, df = 2, $\boldsymbol{p = 0.045}$, Cramer's $V = 0.086$
BS6: Thought interference (SPI-A C2)	**9**	**17.3 [11.66]**	0	0.0	**0**	**0.0 [−9.29]**	$\chi^2 = 135.96$, df = 2, $\boldsymbol{p < 0.001}$, Cramer's $V = 0.405$
BS7: Thought pressure (SPI-A D3)	**9**	**17.3 [11.66]**	0	0.0	**0**	**0.0 [−9.29]**	$\chi^2 = 135.96$, df = 2, $\boldsymbol{p < 0.001}$, Cramer's $V = 0.405$
BS8: Thought perseveration (SPI-A O1)	0	0.0	0	0.0	1	0.1	$\chi^2 = 0.105$, df = 2, $p = 0.949$, Cramer's $V = 0.011$
BS9: Thought blockages (SPI-A C3)	**12**	**23.1 [8.97]**	2	7.4	**10**	**1.3 [−8.26]**	$\chi^2 = 83.803$, df = 2, $\boldsymbol{p < 0.001}$, Cramer's $V = 0.318$
BS10: Decreased ability to discriminate between ideas & perception, fantasy & true memories (SPI-A O2)	0	0.0	**2**	**7.4 [7.72]**	**0**	**0.0 [−4.36]**	$\chi^2 = 59.551$, df = 2, $\boldsymbol{p < 0.001}$, Cramer's $V = 0.268$
BS11: Unstable ideas of reference (SPI-A D4)	2	3.8	0	0.0	7	0.9	$\chi^2 = 4.148$, df = 2, $p = 0.126$, Cramer's $V = 0.071$
BS12: Derealization (SPI-A O8)	0	0.0	**3**	**11.1 [5.11]**	**6**	**0.8 [−2.45]**	$\chi^2 = 26.412$, df = 2, $\boldsymbol{p < 0.001}$, Cramer's $V = 0.179$

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Table 3. (*Continued.*)

	Follow-up Class 1 ($n = 52$)		Follow-up Class 2 ($n = 27$)		Follow-up Class 3 ($n = 750$)		
	n	% [significant standardized residuals]	*n*	% [significant standardized residuals]	*n*	% [significant standardized residuals]	Statistics
BS13: Visual perception disturbances (SPI-A O4, F3, D5)	**21**	**40.4 [11.82]**	**5**	**18.5 [3.18]**	**17**	**2.3 [−11.68]**	$\chi^2 = 153.76$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.431$
BS14: Acoustic perception disturbances (SPI-A O5, F5)	**26**	**50.0 [10.27]**	5	18.5	**48**	**6.4 [−9.46]**	$\chi^2 = 109.84$, df = 2, $\mathbf{p < 0.001}$, Cramer's $V = 0.364$

Note: SOFAS: Social and Occupational Functioning Assessment Scale.
In **[bold]**, cells with standardized residuals $\geq|1.96|$. This equals significant deviation from the expected cell frequency. An adjusted residual of 1.96 indicates that the number of cases in that cell is significantly larger than would be expected if the null hypothesis were true, with a significance level of 0.05. An adjusted residual that is <-1.96 indicates that the number of cases in that cell is significantly smaller than would be expected if the null hypothesis were true.
P: positive-symptom scale; BS: basic symptom.

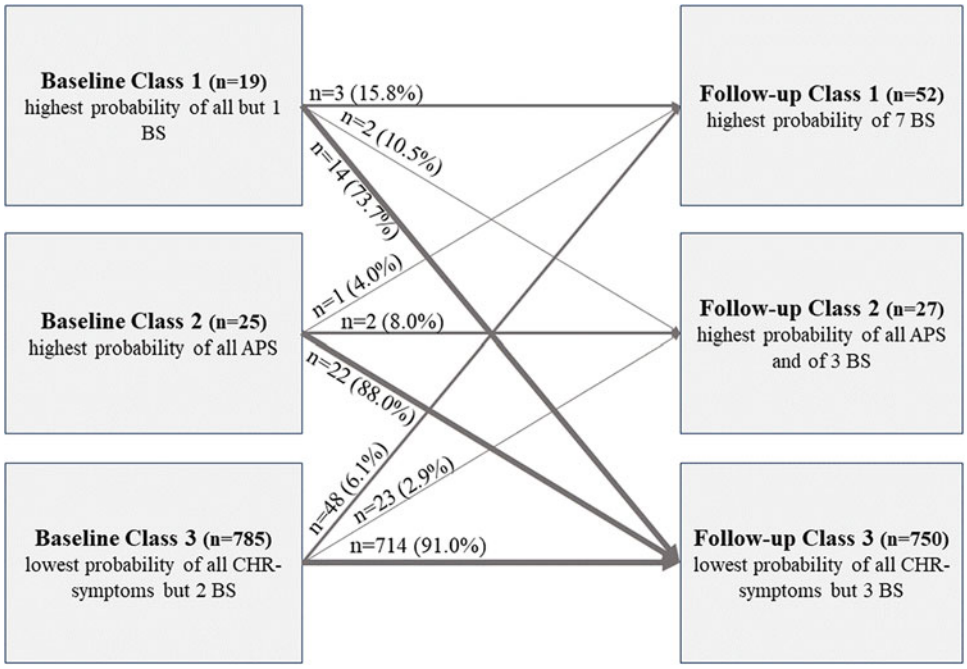

Figure 3. Changes of class membership over time.

Movement between classes from baseline to follow-up

In absolute terms, more participants ($n = 79$) were included in the two more impaired Classes 1 and 2 at follow-up than had been at baseline ($n = 44$). However, less than a quarter ($n = 8$) of Baseline Class 1 or 2 members stayed in, or moved to, the corresponding Follow-up Classes 1 or 2. Instead, the majority of participants in the more impaired Baseline Classes 1 and 2 (73.7% and 88.0%, respectively) moved to the 'healthy' Follow-up Class 3, which still included most (91.0%) members of the 'healthy' Baseline Class 3 (Fig. 3). In contrast, 9.0% of members ($n = 71$) of the least impaired Baseline Class 3 moved to the more impaired Follow-up Classes 1 or 2 (6.1% and 2.9%, respectively; Fig. 3).

Demographic and socio-economic characteristics across CHR-P classes

The classes differed little in distribution of sex, nationality, family history of psychiatric disorders and age, although participants in Baseline Class 2 were the oldest at both baseline and follow-up. Across time points, Class 3 had the highest rate of regular employment.

The distribution of education and marital status showed more variation. While there was less distinction between Baseline Classes at either time point, Follow-up Class 3 showed significantly higher education and lower divorce rates than Follow-up Classes 2 and 1. In turn, Follow-up Class 2 participants were most frequently unmarried, while Follow-up Class 1 members were most often separated. Finally, the education level in Follow-up Class 2 was slightly higher than in Follow-up Class 1.

Changes and class characteristics of CHR-P symptoms

CHR-P symptom profiles showed some relevant changes across classes and time points.

At follow-up, Baseline Classes 1 and 3 showed a more than twofold increase in the rate of (attenuated) hallucinations. As a result, Baseline Class 1, whose members also exhibited increased

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rates of thought blockage and derealization, now showed a similar rate of (attenuated) hallucinations to Baseline Class 2. Despite this, the rate of (attenuated) hallucinations in Baseline Class 3 remained significantly smaller than in both symptomatic classes. Similarly, perceptual BS had more than doubled, with the increase being particularly pronounced in Baseline Class 3, thus leading to a lack of significant class differences in perceptual BS at follow-up. In summary, while perceptual symptoms had little influence on class identification at baseline, their increase at follow-up turned them into highly influential symptoms for the definition of both symptomatic classes.

Conversely, unusual thought content (SIPS P1), which had been highly influential on class separation at baseline, did not maintain this role for Class 1 at follow-up. However, it remained highly influential for Class 2, which continued to show the overall highest rate of any APS/BIPS at this time point. Newly, Follow-up Class 2 also showed the highest prevalence rates of four BS: inability to divide attention, disturbance of receptive speech, decreased ability to discriminate between ideas/perception and fantasy/true memories, and derealization. Additionally, visual perception disturbances occurred more frequently in Follow-up Class 2, although still less frequently than in Follow-up Class 1. This was a notable change compared to the baseline assessment, where no BS had been most frequent in Class 2, although disturbance of receptive speech, derealization and thought pressure had occurred frequently.

Discussion

To the best of our knowledge, this is the first longitudinal study of classes of a comprehensive collection of CHR-P symptoms in the community, and the first that not only examines homogeneous classes of individuals at baseline but also their stability and change in class membership over time.

Symptomatic characteristics of classes over time

Our three-class solution aligns with earlier LCA studies of UHR patients, which predominantly reported three classes (Healey *et al.*, 2018; Ryan *et al.*, 2018), though some found four (Valmaggia *et al.*, 2013) or five (Ryan *et al.*, 2018). Most focused solely on UHR patients, with only one (Healey *et al.*, 2018) including healthy controls, making it most comparable to ours. Healey et al. also identified a three-class solution with a 'mild' class similar to our Class 3. However, unlike our study, APS/BIPS were not highly influential in their results, possibly due to UHR criteria favouring positive symptoms. The influential role of negative symptoms in earlier studies contrasts with our study's emphasis on APS/BIPS, possibly due to the exclusion of negative symptoms in our analysis.

The differentiation of symptomatic classes in our community study into one characterized mainly by APS/BIPS, and one characterized mainly by BS, is in line with previous reports of SIPS positive items and BS mostly clustering in different classes (Jimeno *et al.*, 2020, 2022). In a recent network cluster analysis (Jimeno *et al.*, 2020), only hallucinatory symptoms (SIPS-P4) had joined the cluster of BS; this being broadly in line with Follow-up Class 1 that was characterized by seven BS and (attenuated) hallucinations (SIPS-P4). However, APS/BIPS and BS were best separated at baseline.

Baseline class characteristics remained consistent over time, with notable exceptions, particularly an increase in perceptual symptoms in Class 1 at follow-up. Given earlier findings linking (attenuated) hallucinations and BS to younger age (Schimmelmann *et al.*, 2015; Schultze-Lutter *et al.*, 2017, 2020a, 2020b; Schultze-Lutter and Schmidt, 2016; Walger *et al.*, 2020), this increase was unexpected. Future studies should examine features related to this increase to better understand the course of perceptual symptoms in the community. Further, the cross-class occurrence of thought pressure, derealization and visual perception disturbances, as well as suspiciousness/persecutory ideas, may be attributed to their transdiagnostic nature, not observed in other CHR-P symptoms or criteria (Schultze-Lutter *et al.*, 2022).

Associated features over time and class solutions

Interestingly, despite the sample's generally reduced symptom load at follow-up, which is in line with other studies (Bergé *et al.*, 2024; Salazar de Pablo *et al.*, 2022), the number of members in the two symptomatic classes increased from baseline to follow-up. Comparing the socio-demographic and clinical features between assessment times and LCA solutions, however, revealed some small changes in distribution of axis-I disorders that tended to be most frequent in Class 1, and least frequent in Class 3 over time, and across solutions. This was despite a decline in axis-I disorders over time, in particular in affective and other disorders (i.e., eating disorders, somatoform disorders, etc.), that aligns with reports of a decline of comorbid mental disorders over time from clinical CHR-P samples (Solmi *et al.*, 2023). The combination of CHR-P symptoms and non-psychotic mental disorder is considered a particularly 'risky' form of CHR-P state, with poorer outcome compared to CHR-P symptoms in isolation (Hasmi *et al.*, 2021). This might explain the poor outcome of Baseline Class 1 members who had the highest rate of baseline axis-I disorder and, at follow-up, had the highest rates of axis-I disorders and functional deficits.

Functional deficits demonstrated little change in overall frequency over time, and were generally lowest in Class 3, but differed between the symptomatic classes in distribution over time and solutions. While functional deficits were similarly frequent in Baseline Classes 1 and 2 at baseline, at follow-up, they were most frequent in Baseline Class 1 and in Follow-up Class 2. This lack of a significant improvement in functioning is in contrast to reports from follow-up studies of CHR-P samples that commonly report significant functional improvement over time (Salazar de Pablo *et al.*, 2022). The difference in findings may be related to a difference in samples, with far fewer participants with functional deficits in our community sample and/or to the assessment of functioning – dichotomized data in our study, and continuous raw data in most clinical studies (Salazar de Pablo *et al.*, 2022). Overall, the generally maintained disadvantages of the symptomatic classes over time, despite symptomatic improvements, underscore the importance of preventive approaches not only with regard to mental disorders but also functional deficits and vocational-educational disadvantages (Campion *et al.*, 2012; Porru *et al.*, 2023).

Membership changes between Baseline and Follow-up Classes

In line with the general symptomatic, clinical and socio-demographic stability of Class 3 over time, this class showed the lowest rate of changes into any symptomatic class, indicating that most participants remained 'healthy' over time. Furthermore, the highest rate of class membership changes of the two symptomatic classes were into Follow-up Class 3, indicating health improvement and an attenuation of most CHR-P symptoms over time (Addington *et al.*, 2020). In absolute numbers, however, more

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participants moved from the large Baseline Class 3 into one of much smaller symptomatic Follow-up Classes; with a third of them into Follow-up Class 1. The unexpected transition from 'healthy' to psychopathological symptoms suggests that even those with few or no symptoms may be at risk for later development of CHR-P symptoms. This broadens the focus of early detection efforts beyond solely 'at-risk' individuals with CHR-P symptoms, prompting exploration of hidden factors including (neuro)biological and psychosocial influences, such as inflammatory processes and negative life events (de Koning *et al.*, 2022; Trotta *et al.*, 2015). Understanding these factors beyond genetic predisposition is crucial for comprehensively addressing psychopathology development.

Follow-up Class 1 also showed higher membership stability compared to Follow-up Class 2 (16% vs. 8%). This broadly aligns with reported changes of CHR-P criteria in a clinical sample of an early detection service over 1–10-year follow-up (Michel *et al.*, 2018a), in which most non-converters had remitted from CHR-P status (72%), and more non-converters with the baseline BS criterium 'Cognitive Disturbances' than with baseline UHR criteria maintained their risk status (18% vs. 12%). Furthermore, 91% of CHR-P-negative patients remained CHR-P-negative (Michel *et al.*, 2018a). Overall, our results support the fluctuating nature of CHR-P symptoms.

Practical recommendations

Based on our findings, we propose several practical steps to improve early detection and intervention for CHR-P symptoms. Community-based prevention efforts should prioritize targeted mental health literacy programmes aimed at the public, healthcare providers and educators. These programmes should focus on increasing awareness of early CHR-P symptoms – such as perceptual disturbances, cognitive difficulties and social withdrawal – while addressing stigma to promote timely help-seeking.

In primary care settings, integrating brief and validated CHR-P screening tools, such as the Prodromal Questionnaire-Brief (PQ-B; Loewy *et al.*, 2011) or the Community Assessment of Psychic Experience (CAPE; Mossaheb *et al.*, 2012), into routine clinical practice can facilitate earlier identification of individuals at risk. Training primary care professionals to recognize key indicators of CHR-P, including comorbid mood or anxiety symptoms, is essential for appropriate referral to specialized services.

For individuals with functional impairments or symptomatic profiles, targeted interventions such as cognitive-behavioural therapy, stress management techniques and resilience-building programmes should be offered. Family psycho-education and support can also play a critical role in improving social and functional outcomes.

Given the heterogeneity of CHR-P presentations, personalized preventive strategies are crucial. These should be informed by comprehensive assessments of psychosocial factors (e.g., trauma history, family dynamics), neurocognitive deficits (e.g., executive dysfunction) and biological risk markers (e.g., sleep disturbances or neuroinflammation). Tailoring interventions to individual risk profiles increases their precision and effectiveness.

Lastly, longitudinal monitoring of individuals with mild or subthreshold symptoms is vital to detect emerging risk states. This can be achieved through structured follow-ups and the use of digital tools, such as Ecological Momentary Assessment and telehealth platforms, which allow real-time tracking of symptom trajectories and functional outcomes. Such continuous monitoring enables adaptive and timely interventions that may prevent progression to fully manifest psychosis.

By implementing these strategies, we can enhance the early identification of CHR-P states, provide timely and individualized interventions, and ultimately improve long-term outcomes for at-risk individuals.

Strengths and limitations

Our symptom selection is both a strength and limitation. While our study is the first LCA study to include the full spectrum of CHR-P symptoms (Schultze-Lutter *et al.*, 2015), it did not include non-CHR-P-relevant symptoms, such as negative symptoms, which have been shown to differentiate classes (Healey *et al.*, 2018; Ryan *et al.*, 2018; Valmaggia *et al.*, 2013). However, the shift towards a stepwise psychosis detection approach, assessing CHR-P criteria first (Schultze-Lutter and Meisenzahl, 2023, 2024; Woods *et al.*, 2023), suggests our classes may reflect early diagnostic steps. Strengths of our study include clinical assessments by trained psychologists and a large, well-representative sample (Schultze-Lutter *et al.*, 2018). Still, the small size of symptomatic classes warrants caution in interpretation. Additionally, like earlier studies, we did not account for the impact of treatment, which could have influenced class development. Treatment may be particularly influential, as higher symptom loads often lead to increased help-seeking (Michel *et al.*, 2019).

Conclusion and future directions

Our results suggest that CHR-P symptoms cluster similarly in the community as in clinical samples, despite their fluctuation over time, underpinning the largely distinct and, therefore, complementary nature of the BS and symptomatic UHR approaches (Schultze-Lutter *et al.*, 2020a). In addition, the association of the two symptom classes with axis-I disorders and functional deficits emphasizes the clinical significance of CHR-P symptoms beyond a potential bias towards higher clinical relevance in patient samples (Fusar-Poli *et al.*, 2016; Ruhrmann *et al.*, 2010; Schmidt *et al.*, 2015).

These results emphasize the importance of preventive measures in general, and point to the need to improve mental health literacy in relation to CHR-P states and symptoms in the community (Kelly *et al.*, 2007). All the more so, as compared to other mental disorders, such as depression (Svensson and Hansson, 2016), there is a significant lack of knowledge, misunderstanding and negative stereotyping of psychotic disorders, including their symptoms and risk stages (Doll *et al.*, 2022; Goodwin, 2014; O'Keeffe *et al.*, 2016; Patel, 2004), in the healthcare system, the public and the media. Even those affected often lack a clear understanding of the CHR-P condition, which delays their help-seeking (Haidl *et al.*, 2019). At the clinical level, improved stepwise diagnostic approaches drawing from broad psychopathological, resilience, neurocognitive and biogenetic assessments for improved risk profiling for various outcomes and risk-adapted treatments should enable a more personalized, broader prevention approach that better fits the need of different person classes (Schultze-Lutter and Meisenzahl, 2023, 2024; Worthington and Cannon, 2021).

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Availability of data and materials. Data will not be directly available on a public repository or in the supplements. However, it can be made available on request via the corresponding author (C.M.).

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Ethical standards. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2013.

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Publication 2

Full text

Exploring the complex relationships between coping strategies, locus of control and self-esteem with psychopathology: structural equation modeling with a special focus on clinical high-risk of psychosis.

Giulia Rinaldi, Naweed Osman, Michael Kaess, Benno Graf Schimmelmann, Jochen Kindler, Frauke Schultze-Lutter & Chantal Michel

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Exploring the complex relationships between coping strategies, locus of control and self-esteem with psychopathology: structural equation modeling with a special focus on clinical high-risk of psychosis

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Abstract

Background. Coping strategies, competence, and locus of control (LOC) beliefs are important predictors of mental health (MH). However, research into their complex interactions has produced mixed results. Our study investigated them further in the previously unexplored context of clinical high-risk (CHR) of psychosis.

Methods. We tested six alternative structural equation models in a community sample (N = 523), hypothesizing a mediating role of coping and treating CHR symptoms as (i) an additional mediator or (ii) a specific outcome. Our measurement model included two latent factors of MH: (1) psychopathology (PP), consisting of presence of mental disorders, global and psychosocial functioning, and (2) self-rated health (SRH) status.

Results. In the model with the best Akaike Information Criterion and the latent factors as outcome variables, maladaptive coping completely mediated the impact of maladaptive LOC on PP and SRH. Additionally, CHR symptoms partially mediated the effect of maladaptive coping on PP and SRH in the community sample, as long as sex was not entered into the model. In the clinical sample (N = 371), the model did not support a mediation by CHR symptoms, despite significant pathways with both coping and MH outcomes; further, competence beliefs directly impacted SRH.

Conclusions. Coping strategies are an important intervention target for MH promotion, especially in the community. In clinical populations, interventions focusing on coping strategies may improve CHR symptoms, thus potentially supporting better MH, especially SRH. Additionally, due to their mostly cascading effects on MH, improving competence and LOC beliefs may also promote psychological well-being.

1. Introduction

Psychotic disorders are among the most frequent causes of disability-adjusted life years in adults [1] and adolescents [2] and rate second in resulting costs [3]. Psychotic episodes are mostly preceded by a prodromal phase, in which the onset of clinical high-risk (CHR) symptoms, other mental health (MH) problems, and deficits in psychosocial functioning often leads to help-seeking [4–6]. Longer duration of an inadequately treated prodromal phase is associated with negative outcomes of first-episode psychosis (FEP) [2, 7–9]. Therefore, this phase offers a unique point of intervention for an indicated prevention, aimed at reducing CHR symptoms and distress, thereby postponing or preventing manifest psychosis [10].

Despite direct associations of CHR symptoms with distress and an increased risk for psychosis [10–13], relative declines in transition rates and high rates of onset and persistence of non-psychotic disorders in CHR populations have been observed [11, 14–16]. This has generated debate regarding diagnostic specificity of CHR in predicting psychosis, with suggestions that it might be pluripotential, indicating risk for developing a range of different psychiatric conditions [17, 18]. Consequently, it was proposed that the CHR state be redefined as a transdiagnostic at-risk mental state (e.g., Clinical At-Risk Mental State; CHARMS [19]), allowing for the identification of early signs of multiple severe mental disorders. However, other studies [20–23] support the diagnostic specificity of CHR symptoms, indicating that only emergent psychotic disorders significantly differentiate between CHR patients and non-CHR help-seeking controls

[21], and that the onset and persistence of non-psychotic disorders occur at a similar frequency in both groups, suggesting that a CHR status does not specifically represent a risk factor for non-psychotic disorders [21, 22].

Therefore, while the question of the diagnostic specificity of CHR status remains open, the clinical significance of CHR – for example, psychological burden, independent of conversion to a full-blown mental disorder, and negative impact on functioning – is undisputed [10–12, 19, 20, 23], and the inclusion of Attenuated Psychosis Syndrome in Section III of DSM-5 supports its diagnostic and psychopathological relevance [24], highlighting the need to focus on offering CHR patients effective interventions. Moreover, irrespective of the debate regarding pluripotentiality of the CHR state, evidence indicates some transdiagnostic relevance of the CHR state (or symptoms) in terms of (at least) comorbidity with other psychiatric disorders and syndromes [25–27]. This is reflected in new broader transdiagnostic and dimensional psychiatric taxonomies wherein efforts are currently being made to determine the most appropriate way to map CHR for psychosis into these models [28].

Relatedly, other relevant intervention targets for this population include transdiagnostic factors of core beliefs – consisting of locus of control (LOC) and competence beliefs – and coping, demonstrating dysfunctional patterns in CHR [29], FEP [29, 30], and schizophrenia patients alike [31, 32], and are regarded as possible predictors of psychosis [29]. That is, the hypothesis that typical psychotic symptoms, for example, delusions and hallucinations, result from the use of dysfunctional coping and core beliefs in response to basic symptoms (BS; self-experienced subclinical disturbances in thinking, speech, and perception) [33] and stressful stimuli [34].

Beyond their role in CHR, coping and core beliefs are also relevant for general MH quality [35–37], as reflected by multiple outcomes, including psychopathology, psychosocial functioning, and self-assessment of one's own health status [38]. Coping is an especially important predictor of MH quality [35, 39, 40], particularly regarding stress [36] and representing either a risk (maladaptive coping, including avoidant and emotion-oriented strategies [41–43]) or protective factor (adaptive coping, including problem-focused and active strategies [44, 45]). LOC is another predictor for MH [31, 46]: internal LOC (attributing positive events to internal causes and negative ones to external factors such as chance or others) is linked to better MH outcomes and greater resilience [47], while external LOC (the opposite tendency) is associated with psychiatric disorders, including depression and schizophrenia as well as generally poorer functioning [31, 46, 47]. Thus, they can be conceptualized as adaptive and maladaptive, respectively. Finally, competence beliefs, including self-efficacy and self-esteem [48, 49], are strongly associated with MH quality [37, 50], with higher competence beliefs being related to better psychosocial functioning [37, 51].

Investigations into the interactions between coping, core beliefs, and MH, involving mainly community samples but also including a minority of clinical samples, have led to contradictory findings in both populations, indicating a mediating role of coping [52–54] or of core beliefs [49, 55, 56]. A recent meta-analysis [36] – also mostly, but not exclusively, using community samples – supported a mediation by coping on the influence of core beliefs on MH. Specifically, maladaptive coping mediated the relationship between maladaptive LOC and MH problems. Moreover, both adaptive and maladaptive LOC showed a direct influence on MH problems, independent of coping.

In the present study, we extended the meta-analytical and mediation model [36] that had mixed community and clinical samples by first exploring alternative structural equation models (SEM) in a community sample and then examining their validity in a clinical sample. In addition to general psychopathology, we focused on CHR symptoms, in virtue of their association with MH quality [10–12] as well as coping and core beliefs [29]. The aims of the present study were:

1. To explore the association between core beliefs and MH outcomes, in both a community and a clinical sample, assuming a mediation by coping. Specifically, based on the metanalytical model [36], we anticipated that the effect of competence beliefs and adaptive LOC on MH outcomes would be mediated by adaptive coping, and that the effect of maladaptive LOC would be mediated by maladaptive coping.
2. To investigate the specific placement of CHR symptoms in these interactions.

Based on the metanalytical model [36], we did not expect relationships between competence beliefs and adaptive LOC, and maladaptive coping or between maladaptive LOC and adaptive coping, and therefore we did not include these relationships in the models.

2. Methods

2.1. Participants and recruitment procedure

Cross-sectional data from a community and a clinical sample were used in the current study. The former comprised 523 participants in the first follow-up assessment of the Bern Epidemiological At-Risk (BEAR) study [57, 58], whose core beliefs and coping strategies were evaluated in an add-on study (Supplementary eFigure 1, Supplementary eText 1). Inclusion criteria were absence of a psychotic disorder and fluency in German.

The second sample included 378 patients of the Bern Early Recognition and Intervention Centre for mental crisis (FETZ Bern), assessed between November 2009 and July 2022. Inclusion criteria were informed consent to the use of collected data for scientific purposes, age above 13 years (to allow for the assessment of all BS), and sufficient German-language skills. For more information regarding recruitment and assessment procedures in the BEAR study [57] or FETZ Bern [59], see Supplementary eTexts 1–4.

2.2. Assessments

2.2.1. Mental disorders

The Mini-International Neuropsychiatric Interview (MINI) [60] was used to assess current presence of Axis-I mental disorders according to the *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition (DSM-IV) [61]. The presence of each disorder was indicated by a score of 1 in the corresponding scale; their sum score (0–36) was used in analyses.

2.2.2. CHR symptoms

Two approaches are used for the assessment of CHR states: (i) ultra-high-risk (UHR) criteria and (ii) BS criteria (Supplementary eTable 1). The Structured Interview for Psychosis Risk Syndromes (SIPS) [62] was used to assess the presence of UHR symptoms (attenuated (APS) or brief intermittent psychotic symptoms (BIPS)). For each of the positive items (P1–P5; Supplementary eTable1), participants received a score of 1 if they presented symptoms rated between 3 and 5 (APS) or equal to 6 (BIPS), irrespective of whether or not the

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APS/BIPS in question met requirements for onset/worsening and frequency of the UHR criteria that are very infrequent in the general population [57, 62]. Scores were then added in a sum score (0–5).

The presence of the BS criteria, cognitive disturbances (COGDIS), and cognitive-perceptive basic symptoms (COPER) was assessed with the Schizophrenia Proneness Instrument–Adult [63] and Child and Youth [64] versions. Irrespective of the frequency and novelty requirements for BS criteria that are also infrequent in the community [33], the presence of each criterion-relevant BS (Supplementary eTable1) was indicated by a score of 1, and a sum score (0–14) was obtained.

2.2.3. Self-rated health

Self-rated health was evaluated via the EuroQoL-5D, three-level version (EQ-5D-3L) [65], assessing three degrees of severity across five dimensions of health, from which we obtained a sum score (0–100) [66, 67]. Participants' self-rating of their current health state on the EQ-5D-3L analog scale (0–100, "worst" to "best imaginable health state") was also included in our models.

2.2.4. Global, social, and occupational functioning

Functioning was assessed with both the Global Assessment of Functioning (GAF) scale, in which psychiatric symptoms are considered, and the Social and Occupational Functioning Assessment Scale (SOFAS) for the evaluation of functioning independently from symptoms [61].

2.2.5. Core beliefs

The German Competence and Control Beliefs Questionnaire (FKK) [68] was used to evaluate these constructs by means of Self-Concept (FKK-SK; 8 items), Internality (FKK-I; 8 items), and Externality (FKK-PC; 16 items) scales. These were conceptualized in our models as competence beliefs (FKK-SK; as recommended in [68], see also [69]), adaptive (FKK-I), and maladaptive LOC (FKK-PC; "internality" and "externality" are synonyms for internal, that is, adaptive, and external, that is, maladaptive, LOC, respectively [31, 70]). Analyses were conducted with the normative T-values of each scale's sum score, obtained from ratings in their respective items on a bipolar six-level scale.

2.2.6. Coping strategies

Positive and negative coping was assessed via the German Stress Coping Questionnaire, adult (SVF-120) [71] and children/adolescents (SVF-KJ) [72] versions. In each item, the frequency of use of different coping strategies can be rated on a 0–4 Likert scale ("not at all"–"in any case"). In our analyses, we used the relative normative T-values to the sum scores of the global scales Positive and Negative Coping Strategies to represent adaptive and maladaptive coping, respectively.

2.2.7. Sociodemographic variables

Age, level of education, and sex were included in the models as possible confounding variables, the latter only at a later stage during a sensitivity analysis.

Further details regarding instruments can be found in Supplementary eText 5.

2.3. Statistical analyses

Data analyses were performed in RStudio, version 4.1.1, using the lavaan package for preliminary exploratory and confirmatory factor analyses (EFA, CFA) and testing alternative SEMs, and the sempower package for power analysis. The community sample served as the model generation; the clinical sample as model validation sample.

First, an EFA was conducted using variables pertaining to participants' MH (presence of Axis-I mental disorders and self-rated health) based on Spearman correlation matrices and using Oblimin rotation, allowing intercorrelation of factors. Pairwise deletion was applied, excluding one participant who was missing 20% of the data. Based on EFA results, we proceeded with a two-factor CFA.

Finally, six alternative SEMs were computed using the maximum likelihood estimator [73]. After a pairwise deletion of five observations with missing data, the analysis was conducted on 518 participants from the community sample. Along with the EFA/CFA factors, variables included age, education, standard T-values for competence beliefs (FKK-SK), maladaptive LOC (FKK-PC), adaptive LOC (FKK-I), adaptive and maladaptive coping, presence of BS and APS/BIPS, or alternatively presence of either of CHR symptoms. A Tucker-Lewis index (TLI) ≥0.90, a comparative fit index (CFI) ≥0.95, a standardized root mean square residual (SRMR) ≤0.08, a root mean square error of approximation (RMSEA) ≤0.06, as well as a 90% confidence interval (CI) not containing 0.08 indicate excellent model fit [74]. As the Chi-squared test is sensitive to sample size and often results in model rejection when working with large samples [75], we focused on the aforementioned indices in evaluating model fit. After comparing the models' Akaike Information Criterion (AIC) [76] and Bayesian Information Criterion (BIC) [77], one model was selected as fitting the data best; this was validated in the clinical sample.

The clinical sample (N = 371) presented higher amounts of missing data (9.58%). After applying listwise deletion to 51 participants missing >50% [78] of their data, we used a multiple imputation method on data missing from the remaining 327 subjects [79].

To control for sex differences, we conducted a sensitivity analysis by including sex in the chosen model and testing it again in both samples. Here the introduction of a categorical variable in the model required the use of the Weighted least squares and variance-adjusted estimator [73]. We chose this procedure instead of directly including sex in the six alternative SEMs because using this estimator would not have allowed a statistically valid selection of one best-fitting model. Finally, in all samples, we tested all possible mediation pathways indicated in the selected model for significance and calculated their respective 95% bias-corrected bootstrap CIs.

3. Results

3.1. Sample characteristics

The two samples differed in sex (more males in the clinical sample), age, and highest educational level (both lower in the clinical sample), as well as in clinical and functional variables, with lower functioning and more severe psychopathology in the clinical sample (Table 1).

3.2. EFA and CFA in the community sample

Results of the EFA (Supplementary eTable 2) indicate two correlated latent factors (factor correlation 0.34): (i) psychopathology (PP) and (ii) self-rated health (SRH). The model's fit to the community sample data was excellent overall (RMSR = 0.01, TLI = 0.98, RMSEA = 0.059). The CFA (N = 522) confirmed the two-factor

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Table 1. Sample characteristics and group comparison

	Community sample (N = 523)		Clinical sample (N = 371)[a]		
	n	%	n	%	Statistics; effect size
Age (mean ± SD, median, range)	33.4 ± 7.8, 35.00, 19.00–45.00		18.94 ± 4.51, 17.44, 12.98–40.30		U = 186,426, p < 0.001; r = 0.757
Sex (male)	204	39.0	179	47.4	χ^2 = 15.956, p < 0.001; V = 11.166
Highest professional education (ISCED level)					U = 142,062, p < 0.001; r = 0.456
Early childhood education (ISCED 0)	0	0	4	1.1	
Primary school or school for special needs (ISCED 1)	0	0	6	1.6	
Secondary school (ISCED 2)	5	1.0	108	28.6	
Highschool (ISCED 3.4)	8	1.5	10	2.6	
Highschool-level professional education (ISCED 3.5)	36	6.9	38	11.9	
Post-secondary non-tertiary education (ISCED 4)	6	1.1	1	0.3	
Short-cycle tertiary education (ISCED 5)	256	48.9	141	37.3	
Master (ISCED 7)	205	39.2	45	11.9	
Doctoral (ISCED 8)	7	1.3	0	0	
SOFAS score (mean ± SD, median, range)	84.80 ± 6.66, 88, 40.00–100.00		59.35 ± 12.97, 60, 30.00–95.00		U = 174,438, p < 0.001; r = 0.775
GAF score (mean ± SD, median, range)	81.70 ± 9.84, 87.0, 36.00–95.00		51.86 ± 12.51, 53, 21.00–90.00		U = 176,177, p < 0.001; r = 0.770
Current Axis-I disorders, sum score (mean ± SD, median, range)	0.21 ± 0.61, 0, 0.00–6.00		1.06 ± 1.06, 1, 0.00–6.00		U = 37,924, p < 0.001; r = 0.483
Current CHR symptoms, sum score (mean ± SD, median, range)	0.44 ± 0.61, 0, 0.00–5.00		4.28 ± 3.29, 3, 0.00–14.00		U = 17,212, p < 0.001; r = 0.698
Current UHR symptoms, sum score (mean ± SD, median, range)	0.15 ± 0.43, 0, 0.00–3.00		1.74 ± 1.25, 2, 0.00–5.00		U = 25,606, p < 0.001; r = 0.687
Current basic symptoms, sum score (mean ± SD, median, range)	0.29 ± 0.60, 0, 0.00–4.00		2.63 ± 2.51, 2, 0.00–10.00		U = 28,810, p < 0.001; r = 0.608

Abbreviations: CHR, clinical high risk; r, Pearson's r; SOFAS, Social and Occupational Functioning Assessment Scale; UHR, ultra high risk; U, Mann-Whitney U test, V, Cramer's V; χ^2, Chi-squared.
[a]In the FETZ sample, 18 participants (4.8%) were missing data about their education level (ISCED), 30 participants (7.9%) were missing data about their SOFAS score, 26 participants (6.9%) were missing data about their GAF score, 85 participants (6.9%) were missing data about their current Axis-I disorders, 46 participants (12.2%) were missing data about their current CHR symptoms, 26 participants (6.9%) were missing data about their current UHR symptoms, 45 participants (11.9%) were missing data about their current basic symptoms.

structure (Supplementary eTable 3), showing very good model fit (CFI = 0.996, TLI = 0.990, RMSEA = 0.062, SRMR = 0.032).

3.3. SEM models in the community sample

The resulting latent factors were included in six alternative SEM models (Supplementary eText 6). In all models, positive and negative coping strategies mediated the effect of competence beliefs and adaptive and maladaptive LOC on the latent MH factors PP and SRH.

Fit indices and power ranged from acceptable to excellent, except for TLI, which was equally poor for all models (Supplementary eTable 4). Comparison of their AIC and BIC indices, with emphasis on AIC, indicated model 1.2 (Figure 1, Table 2, Supplementary eTable 5) as best fitting the BEAR data (CFI = 0.923, TLI = 0.863, RMSEA = 0.086, 90% CIs = 0.075, 0.098, SRMR = 0.055, power >0.999, AIC = 39,484.669, BIC = 39,684.418), although model 3.2, with CHR symptoms as an outcome of SHR and PP, had lower BIC (BIC = 39,677.074, AIC = 39,485.825). Though the two models had a similarly good fit to the data, AIC was emphasized in model selection, being more relevant to our testing of a complex system of interactions with unknown underlying structure [80], and since BIC can lead to underfitting when working with large samples, non-nested models, and data not following a multivariate normal distribution [81].

In the community sample, maladaptive coping completely mediated the effect of maladaptive LOC on PP, SRH, and CHR symptoms (Table 3), and adaptive coping mediated the impact of competence beliefs, but not of adaptive LOC, on PP. Additionally, CHR symptoms partially mediated the effect of maladaptive coping on PP and SRH. No significant direct effects of competence beliefs and LOC on PP or SRH were detected.

In the sensitivity analysis, introducing sex as an exogenous variable in model 1.2 (Supplementary eFigure 8, Supplementary eTable 6) fit

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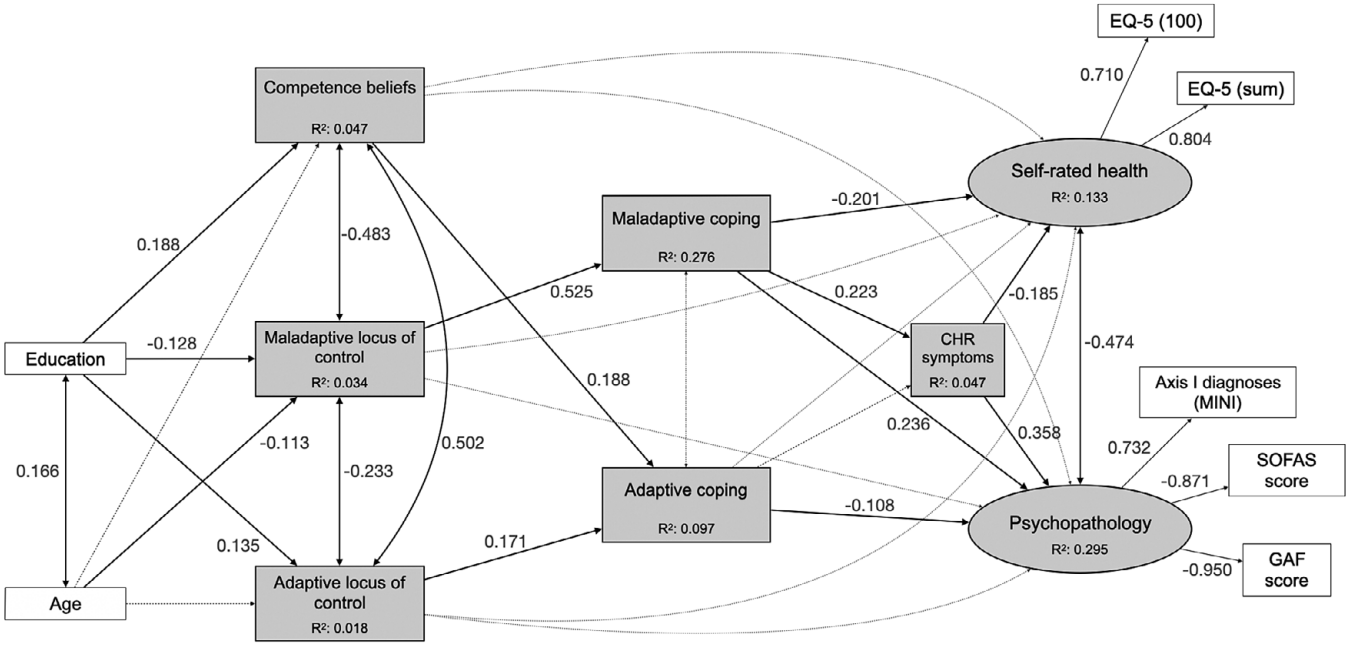

Figure 1. Model 1.2 in the community sample. Rectangles represent observed variables; ovals represent unobserved latent variables; black lines with double-ended arrows represent covariances; black lines with single-ended arrows represent significant paths; dashed gray lines with double- or single-ended arrows represent non-significant covariances or regression paths, respectively; numbers next to the lines indicate coefficients of significant standardized regressions and covariances, or factor loadings; the coefficients of non-significant covariances and regressions are not reported here to facilitate the figure's interpretation; see Table 2 and Supplementary eTable 5 for further details. CHR: clinical high risk; EQ-5 (100): score on the 0–100 analog scale of the EuroQoL-5D, three-level version (EQ-5D-3L); EQ-5 (sum): sum score on EQ-5D-3L – see Supplementary eText 5 for details; GAF, Global Assessment of Functioning; MINI, Mini-International Neuropsychiatric Interview; SOFAS, Social and Occupational Functioning Scale.

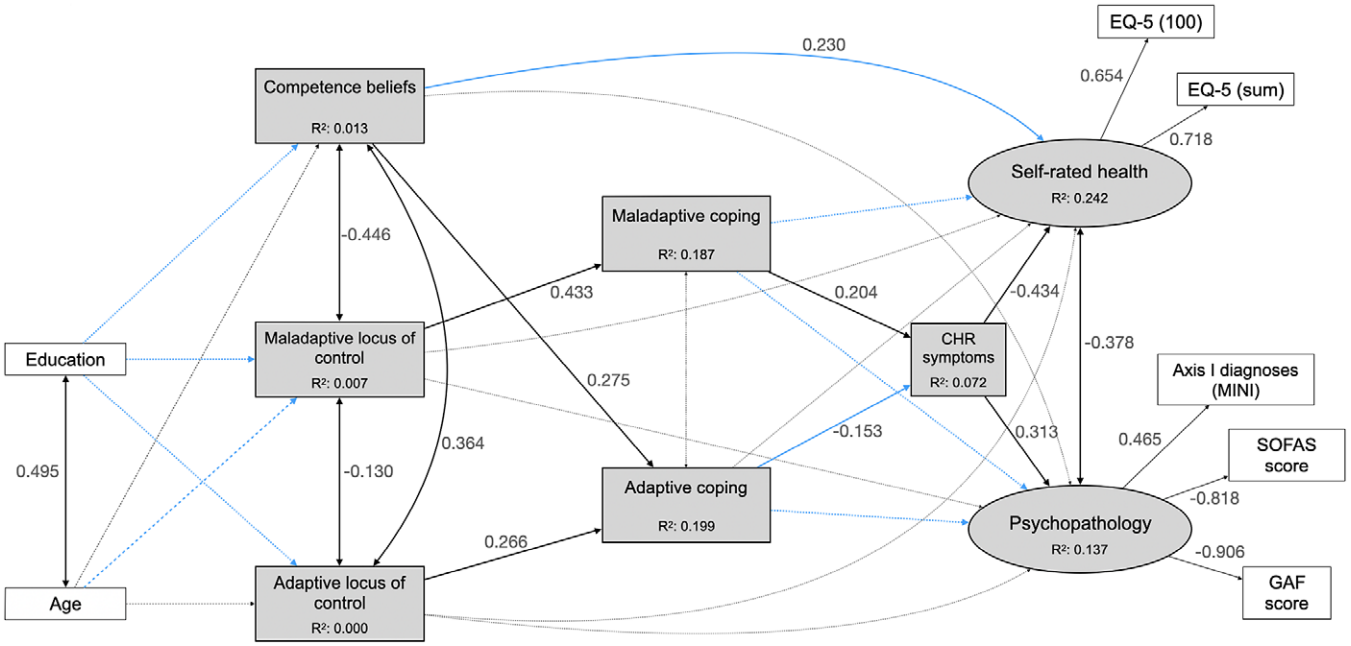

Figure 2. Model 1.2 in the clinical sample. Rectangles represent observed variables; ovals represent unobserved latent variables; black lines with double-ended arrows represent covariances; black lines with single-ended arrows represent significant paths; gray lines with double- or single-ended dashed arrows represent non-significant covariances or regression paths, respectively; numbers next to the lines indicate coefficients of significant standardized regressions and covariances, or factor loadings; the coefficients of non-significant covariances and regressions are not reported here to facilitate the figure's interpretation; see Table 2 and Supplementary eTable 5 for further details. Blue lines indicate differences from results of testing in the community sample. CHR: clinical high risk; EQ-5 (100): score on the 0–100 analog scale of the EuroQoL-5D, three-level version (EQ-5D-3L); EQ-5 (sum): sum score on EQ-5D-3L – see Supplementary eText 5 for details; GAF, Global Assessment of Functioning; MINI, Mini-International Neuropsychiatric Interview; SOFAS, Social and Occupational Functioning Scale.

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Table 2. Standardized regression coefficients (β) and *p* values for relevant paths in model 1.2

	Community sample (*N* = 518)		Clinical sample (*N* = 327)	
	β	p	β	p
Psychopathology(PP)				
Maladaptive coping	0.236	<0.001**	*−0.053*	*0.401*
Adaptive coping	−0.108	0.009*	*−0.080*	*0.212*
CHR symptoms	0.358	<0.001**	0.313	<0.001**
Maladaptive coping				
Maladaptive LOC	0.525	<0.001**	0.433	<0.001**
Adaptive coping				
Competence beliefs	0.188	<0.001**	0.275	<0.001**
Adaptive LOC	0.171	<0.001**	0.266	<0.001**
Self-ratedhealth(SRH)				
Maladaptive coping	−0.201	0.001**	*−0.007*	*0.927*
CHR symptoms	−0.185	<0.001**	−0.434	<0.001**
Competence beliefs	*−0.030*	*0.636*	0.230	0.004*
CHR symptoms				
Adaptive coping	*−0.003*	*0.947*	−0.153	0.005*
Maladaptive coping	0.223	<0.001**	0.204	<0.001**
Competence beliefs				
ISCED level	0.188	<0.001**	*0.101*	*0.113*
Adaptive LOC				
ISCED level	0.135	0.002*	*−0.020*	*0.756*
Maladaptive LOC				
ISCED level	−0.128	0.004*	*−0.092*	*0.150*
Age	−0.133	0.010*	*0.063*	*0.323*

Note: *italics*, not significant, significant in the other sample.***p* < 0.001;
**p* < 0.05.

to the community sample data and power were excellent across all indices (CFI = 0.989, TLI = 0.982, RMSEA = 0.04, 90% CIs = 0.027, 0.045, SRMR = 0.045, power >0.999). Direct paths between the variables remained unaltered, but all mediation effects were insignificant. Competence beliefs newly showed a direct effect on PP.

3.4. SEM model 1.2 in the clinical sample

Next, we tested model 1.2 in the clinical sample (Figure 2). Compared to the community sample, model fit decreased, with CFI (0.865) and TLI (0.761) indicating poor fit, while RMSEA (0.099, 90% CIs = 0.085, 0.114) remained acceptable and SRMR (0.073) and power (0.986) excellent (Table 2, Supplementary eTable 5).

Maladaptive and adaptive coping no longer impacted SRH or PP directly, and neither adaptive nor maladaptive LOC significantly affected the MH outcome variables. Competence beliefs, however, newly directly impacted SRH, which, compared to the community sample model, was more strongly associated with CHR symptoms. Mediation analyses (Table 3), however, revealed no significant mediation by CHR symptoms in the effect of both adaptive and maladaptive coping on SRH and PP. Furthermore, no significant mediation of coping in the relationship of competence beliefs and LOC, and CHR symptoms was found.

The sensitivity analysis (Supplementary eFigure 9, Supplementary eTable 7) led to an increase in goodness of fit and power after including sex in the model. All indices except TLI (0.898) showed values ranging from good to excellent (CFI = 0.942, RMSEA = 0.068, 90% CIs = 0.053, 0.083, SRMR = 0.068, power = 0.994).

Results did not vary except for a newly significant direct effect of competence beliefs on PP and a significant covariation between adaptive and maladaptive coping ($s = -0.136$, $p < 0.001$). No mediation effect was significant.

4. Discussion

4.1. Association between core beliefs and MH outcomes

Our first hypothesis of a mediation by coping in the association between core beliefs and MH was partially supported by findings in the community sample. Aligning with the metanalytical model mostly generated on community samples [36], maladaptive coping completely mediated the effect of maladaptive LOC on CHR symptoms, PP, and SRH, while adaptive coping only mediated the association between competence beliefs and PP. While this suggests that treating maladaptive LOC and coping may promote MH in the

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Table 3. Mediation effect analyses, 95% bias-corrected bootstrap confidence intervals

	Community sample (*N* = 518)			Clinical sample (*N* = 327)		
Mediation pathway	Standardized coefficient	*p*	95% CI	Standardized coefficient	*p*	95% CI
Competence beliefs–adaptive coping–PP						
Indirect effect	−0.020	0.040*	*−0.002, 0.000*			
Total effect	*−0.053*	*0.403*	*−0.009, 0.003*			
Competence beliefs–adaptive coping–CHR symptoms						
Indirect effect				*−0.028*	*0.124*	*−0.024, 0.001*
Total effect				−0.224	0.002*	−0.131, −0.030
Adaptive LOC–adaptive coping–CHR symptoms						
Indirect effect				*−0.027*	*0.107*	*−0.022, 0.001*
Total effect				*0.015*	*0.805*	*−0.037, 0.046*
Adaptive LOC–adaptive coping–PP						
Indirect effect	*−0.018*	*0.071*	*−0.002, 0.000*			
Total effect	*−0.060*	*0.264*	*−0.008, 0.002*			
Maladaptive LOC–maladaptive coping–SRH						
Indirect effect	−0.106	0.026*	−0.200, −0.019			
Total effect	−0.181	0.011*	−0.339, −0.064			
Maladaptive LOC–maladaptive coping–PP						
Indirect effect	0.124	0.003*	0.003, 0.011			
Total effect	0.205	0.001**	0.005, 0.017			
Maladaptive LOC–maladaptive coping–CHR symptoms						
Indirect effect	0.111	<0.001**	0.005, 0.016	*0.027*	*0.302*	*−0.007, 0.030*
Total effect	0.133	0.003*	0.005, 0.020	0.155	0.009*	0.014, 0.097
Maladaptive coping–CHR symptoms–SRH						
Indirect effect	−0.039	0.047*	−0.090, −0.011	*−0.026*	*0.304*	*−0.108, 0.022*
Total effect	−0.240	0.008*	−0.404, −0.061	*−0.033*	*0.704*	*−0.242, 0.162*
Maladaptive coping–CHR symptoms–PP						
Indirect effect	0.076	0.004*	0.001, 0.007	*0.019*	*0.322*	*−0.001, 0.003*
Total effect	0.312	<0.001**	0.008, 0.024	*−0.034*	*0.607*	*−0.007, 0.005*
Adaptive coping–CHR symptoms–SRH						
Indirect effect				*0.043*	*0.101*	*−0.004, 0.125*
Total effect				*0.046*	*0.577*	*−0.131, 0.257*
Adaptive coping–CHR symptoms–PP						
Indirect effect				*−0.031*	*0.101*	*−0.003, 0.000*
Total effect				*−0.110*	*0.102*	*−0.012, 0.000*

Note: *italics*, not significant; value missing, indirect effect was not analyzed in the corresponding sample.
**$p < 0.001$;
*$p < 0.05$.

community, the lack of mediation effects in the sensitivity model, that is, after the inclusion of sex, calls for more research into the role of sex in these associations.

Unexpectedly, but aligning with conflicting results in the two clinical samples of the metanalytical model [36], coping did not mediate the impact of core beliefs on MH in the clinical sample.

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Rather, adaptive and maladaptive beliefs were associated with their coping counterparts. Coping had direct effects on CHR symptoms, which were directly associated with MH outcomes. Newly, the total effects of maladaptive LOC and competence beliefs on CHR symptoms became significant, and competence beliefs were directly linked to SRH. A possible reason is that in clinical populations, both adaptive and maladaptive coping might specifically focus on CHR symptoms, rather than overall MH quality, as our results in the community sample suggest with lower rates of CHR symptoms. Therefore, treatment targeting coping strategies in these populations might help manage and reduce CHR symptoms, preventing maladaptive coping from acting as a trigger for CHR symptoms, exacerbating them, or worsening their outcome [82]. Further, in light of our findings indicating a direct effect of competence beliefs on SRH, and of competence beliefs and LOC on coping, challenging maladaptive core beliefs may also have a positive impact on MH quality. In contrast to the metanalytical model [36], we found no direct effects of LOC on MH outcomes. Possible explanations relate to differences in our study, including added complexity of our model with three MH variables and differing conceptualizations of MH (e.g., including measures of functioning in our study).

Results indicate the need for more group-dependent research on the impact of the severity of psychopathology – and possibly type and operationalization of psychopathology – on the association and potential mediation effects of core beliefs and coping strategies with MH, as different levels of engagement with the mental healthcare system might act as an additional mediator or moderator. Such future studies will shed light on the most relevant targets for promoting MH, that is, core beliefs, coping, or both.

4.2. Role of CHR symptoms

To our knowledge, the present study was the first to explore CHR symptoms in the context of the interactions between core beliefs, coping, and MH, in both community and clinical samples. In the model selected as the best fit for the data, CHR symptoms were included as a contributor of MH outcome. However, the alternative model with CHR symptoms as an outcome of PP and SRH performed similarly well, indicating a strong association (albeit with unclear direction/placement) between MH variables and CHR in both samples, even after controlling for sex differences. Significant mediation effects of CHR symptoms in the relationship between coping and PP and SHR were found only in the community sample model disregarding sex but in no other model, possibly related to the cross-sectional nature of our study, preventing the drawing of definitive causal conclusions. Further factors that might help explain the differences between the community and clinical samples are (i) the differences in prevalence of CHR symptoms in the two samples, which may influence their role in relation to the other variables in our model as well as the results of our analyses; (ii) the impact of the additional burden of higher psychopathology and more severe functioning deficits in the clinical sample, which is generally more unwell compared to the community sample. Regardless, findings support some transdiagnostic relevance of CHR (regarding broader psychopathology and in relation to transdiagnostic factors) while simultaneously highlighting the challenge of accurately mapping CHR into broader psychopathological systems.

Aligning with earlier research on patients meeting UHR criteria [82, 83], maladaptive coping was more strongly and frequently significantly associated with CHR symptoms compared to adaptive coping. Whereas adaptive coping styles were stable in UHR patients, maladaptive coping more likely changed over time and was related to corresponding changes in UHR symptoms in a UHR sample [82] and, in a community sample, was bidirectionally related over time to psychotic-like experiences [84], which, however, may be a poor estimate of clinician-assessed CHR symptoms [85]. With maladaptive coping also negatively impacting functioning and likely other clinical factors such as severity of symptomatology, including depression or personality traits, interventions that challenge coping strategies – and core beliefs – might be most appropriate for populations in early stages of mental disorders or with subclinical MH problems [83].

4.3. Strengths and limitations

The large size of both the community and clinical samples in this study and their separate analysis provide a comprehensive view of CHR symptoms and their associations with important transdiagnostic factors related to MH and some important first insights into the potential differences between community and clinical samples. Further, the assessment of MH variables in clinical interviews conducted by highly trained psychologists, and the comprehensive definition of CHR symptoms not only by UHR but also BS, adds to data validity.

The lack of control for ongoing psychotherapeutic treatment, which might have affected several variables, may be regarded as a limitation that our study shares with most comparable studies [36]. Moreover, despite growing evidence regarding their impact on CHR outcomes, especially on psychosocial functioning [86–88], we did not include negative CHR symptoms in our models, as they were only assessed in the clinical sample and, therefore, a meaningful comparison with the community sample would not have been possible. The role of psychotherapy and negative symptoms should be explored in future research.

Additionally, for reasons of sample size and power, we opted against recommendations [89] to only impute on variables missing <5% of data but applied multiple imputation to the missing data to the SVF 120/KJ and EQ-5D-3L in the clinical sample as well, potentially constituting a statistical limitation. Furthermore, especially for the low number of participants meeting CHR criteria in the community sample (4.97%), we could not perform sensitivity analyses in CHR persons, limiting comparability with studies on CHR samples [82, 83]. Lastly, as only the model with the lowest AIC – an index that penalizes models less for free parameters and favors more saturated models compared to BIC – was further processed; other possible relevant mediations, in particular PP and SRH in model 3.2 with the lowest BIC, remained unexplored.

4.4. Future directions and conclusion

Our findings support evidence of community studies of a mediation role of coping in the relationship of MH variables with core beliefs, although this role might differ between sexes and may decrease with increasing MH problems. Results in the clinical sample suggest a more complex interplay of the examined variables compared to the community sample, thus indicating the need for more group-specific analyses in future studies. Considering this and the higher severity of psychopathology and functioning deficits, treatment in this population may need to be more comprehensive and tailored to target multiple factors influencing MH outcomes, including coping strategies and core beliefs, to address the specific challenges faced by help-seeking individuals. Regarding CHR symptoms, a clear association with PP and, especially, SRH became evident in all models, with inconclusive results about their constellation. Future

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prospective studies should further examine the transdiagnostic factors coping and core beliefs, their relationship with CHR symptoms, and their emergence of manifest mental disorders. Overall, our results contribute to existing evidence that coping strategies, competence beliefs, and LOC represent worthwhile targets for the promotion of MH and shed further light on their complex interactions.

Supplementary material. The supplementary material for this article can be found at http://doi.org/10.1192/j.eurpsy.2023.2457.

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Competing interest. Drs Kaess, Kindler, Michel, and Schultze-Lutter, Ms Rinaldi (MSc), and Mr Osman (MSc) have declared that they have no conflict of interest in relation to the subject of this study. Dr Schimmelmann has been a consultant and/or advisor to, or has received honoraria from, AstraZeneca, Bristol-Myers Squibb, Eli Lilly, Janssen, Novartis, and Shire.

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Publication 3

Full text

Investigating the associations between personality functioning, cognitive biases, and (non-)perceptive clinical high-risk symptoms of psychosis in the community.

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Investigating the associations between personality functioning, cognitive biases, and (non-)perceptive clinical high-risk symptoms of psychosis in the community

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Abstract

Background. Beyond psychosis prediction, clinical high-risk (CHR-P) symptoms show clinical relevance by their association with functional impairments and psychopathology, including personality pathology. Impaired personality functioning is prioritized in recent dimensional personality disorder models (DSM-5, ICD-11), yet underexplored in CHR-P, as are associations with cognitive biases, which early studies indicate as possibly linking CHR-P-symptoms and personality pathology.

Methods. A community sample (N = 444, 17–60 years, 61.8% female) was assessed via clinical telephone interview and online questionnaires. Using zero-inflated Poisson models, we explored associations of personality functioning, cognitive biases, current psychopathology, and psychosocial functioning with likelihood and severity of overall CHR-P, as well as perceptive (per-) and non-perceptive (nonper-)CHR-P-symptoms distinctly.

Results. Higher nonper-CHR-P-symptom likelihood was associated with more impaired personality functioning and psychosocial functioning, while more severe cognitive biases were associated with higher CHR-P- and per-CHR-P-symptom likelihood, alongside higher CHR-P- and nonper-CHR-P-symptom severity. Further, more axis-I diagnoses were linked to higher CHR-P-, per-CHR-P-, and nonper-CHR-P-symptom likelihood, and younger age to higher CHR-P- and per-CHR-P-symptom severity, with CHR-P-symptom severity appearing higher in females. In an exploratory analysis, personality functioning elements identity and self-direction, and cognitive biases dichotomous thinking, emotional reasoning, and catastrophizing, respectively, showed multifaceted associations with nonper-CHR-P-symptom likelihood and overall CHR-P-symptom expression.

Conclusions. Our study supports the association of CHR-P-symptoms with multiple mental health factors. Findings suggest intricate associations between personality functioning impairments and cognitive biases with CHR-P-symptom expression in non-help-seeking populations, possibly contributing to different per-CHR-P- and nonper-CHR-P-symptom expression patterns. Therefore, they should be targeted in future longitudinal studies, aiming at better understanding CHR-P-manifestations to inform preventive intervention.

Introduction

Within the internationally established clinical high risk for psychosis (CHR-P) approach for early risk detection and indicated prevention of first-episode psychosis, risk criteria are primarily identified by presence, time, and severity of CHR-P-symptoms [1]. To define a CHR-P state, two sets of criteria are mainly used: ultra-high risk (UHR) and basic symptoms criteria [2, 3]. Basic symptoms are self-experienced subclinical disturbances in thinking, speech, and perception that patients immediately recognize as disturbances of their own mental processes and are therefore distinct from both UHR-relevant symptoms (i.e., attenuated or brief intermittent psychotic symptoms) and more persistent frank psychotic symptoms [4]. Further highlighting the complexity of these manifestations, perceptive (per; e.g., perceptual basic symptoms, hallucinations) and non-perceptive (nonper; e.g., cognitive basic symptoms, delusions) CHR-P-symptoms exhibit meaningful differences in prevalence, expression, outcome, and clinical significance [5–8]. Specifically, per-CHR-P-symptoms are more common, but less clinically relevant, in

children and adolescents, with related psychological and functional burden increasing as they stabilize by age 18 [6, 9]. In contrast, prevalence of nonper-CHR-P-symptoms is more consistent across age groups, and they show earlier clinical significance, particularly in late adolescence, due to their stronger link to functional impairments and psychiatric comorbidities [6, 10]. These differences suggest that per-CHR-P-symptoms reflect earlier-stage maturation, while nonper-CHR-P-symptoms align with later-stage processes [10]. While the CHR-P state remains associated with an increased risk of psychotic disorders, recent declines in conversion rates to psychosis, alongside high rates of comorbidity with nonpsychotic psychopathology, have raised questions regarding its specificity [11–14]. Simultaneously, this evidence, coupled with associations between CHR-P-symptoms and impairments in neurocognitive and psychosocial functioning, underscores the burden associated with the CHR-P state, criteria, and symptoms, independently of conversion to manifest psychosis [15–19]. As psychotic disorders are increasingly conceptualized as existing along a continuum, from normativity to more severe psychopathology (DSM-5; [20]), and this hypothesis is gaining empirical support [16], focus is shifting toward the role of the CHR-P state, criteria, and symptoms in broader mental health contexts, and their mapping onto dimensional, symptom-driven models of psychopathology [16, 21, 22–25]. These efforts include investigation of the associations between CHR-P-symptoms and other severe mental disorders or symptom dimensions and may ultimately contribute to a better understanding of the full spectrum of mental health, with potential applications in both clinical and community settings [10, 24, 26, 27]. Specifically, understanding CHR-P-symptoms within the community can provide valuable insights into the psychosis continuum, where UHR- and basic symptoms occur at varying frequencies and levels of severity [10, 28]. In this context, personality pathology emerges as a factor of particular interest, as evidence has consistently linked it to psychosis development and the psychosis continuum [29–31]. Both clinically significant personality traits (e.g., borderline, schizoid, schizotypal, avoidant) [31, 32] and expression patterns of personality domains [29] have been associated with psychotic disorders and CHR-P. Among several models of personality structure, research predominantly features the Big Five Model [29, 30, 33]. Studies have found that high neuroticism and low extraversion predict schizophrenia onset [29, 34, 35], and patients with first-episode psychosis additionally show higher openness and agreeableness, but lower extraversion and conscientiousness than controls [36]. Further, openness has been associated particularly with subclinical psychotic symptoms and psychotic proneness [30, 37]. Moreover, in patients with psychosis, frequent comorbidity with avoidant, schizoid, paranoid and schizotypal personality disorders has been reported [30], and studies involving CHR-P samples have consistently found a high prevalence (on average 39.4%) of personality disorders, most frequently schizotypal and borderline [38]. Yet, despite growing evidence of associations between psychosis (risk) and personality pathology, the direction of any causal associations remains unclear, and evidence on the role of specific personality disorders and traits in CHR-P and conversion to psychosis is inconclusive [38–40]. Therefore, recent literature suggests that, rather than specific traits or personality disorders, the essential and most impairing features of personality pathology – that is, disturbances in the self and interpersonal domains [36] – might underpin its association with psychosis and the CHR-P state [39, 40]. This proposition aligns with the Alternative Model of Personality Disorders in DSM-5 (AMPD; [20]), where moderate or greater (≥Level 2 on a 0–4 scale) impairments along two dimensions of overarching personality functioning, that is, self- and interpersonal functioning, constitute the essential diagnostic feature (Criterion A), complemented by maladaptive personality traits (Criterion B). Self-functioning captures identity and self-direction, encompassing a stable, coherent experience of the self as well as effective emotional regulation, self-reflection, and directed behavior [20]. In contrast, interpersonal functioning refers to interactive aspects of personality functioning, including empathy toward others as well as desire and capacity for intimacy [20]. These features are central to AMPD-personality pathology as they effectively distinguish personality disorders from both normative personality and other psychopathology (e.g., [41]). Further highlighting their relevance, research indicated that personality functioning impairment predicts important negative outcomes such as impaired psychosocial functioning, for example, more accurately than categorial personality disorder (PD) diagnoses, and might address some well-known shortcomings of categorial conceptualizations, including accounting for comorbidity among personality disorders [41, 42]. Thus, in recognition of their clinical utility, dimensional approaches are being embraced more broadly [43], as further exemplified by the new ICD-11, also prioritizing personality functioning impairments in personality disorder diagnoses [44].

This conceptualization is relevant to the associations between personality pathology and psychosis risk because disruptions affecting the self and interpersonal relationships have also been observed along the psychosis continuum [40, 45–47]: moving on from its milder end toward manifest psychosis, progressively permeable self-other boundaries, self-disturbances, and gradual disruptions of narrative identity emerge, as well as impairment in interpersonal functioning [40, 45, 46, 48, 49]. However, research on personality functioning, especially within CHR-P, is still limited [30, 40].

Among factors proposed in literature as potentially underlying the association between psychotic/CHR-P-symptoms and personality pathology, cognitive biases often associated with psychosis emerge as an interesting candidate [47, 50–52]. Indeed, these particular cognitive biases, that is, stable and pervasive systematic distortions in information processing which were initially conceptualized as psychosis-specific, were later also associated with borderline personality disorder, independently from psychiatric comorbidity or a history of psychotic symptoms [51, 53–55]. Moreover, cognitive biases originally linked to psychosis were associated with greater frequency and severity of CHR-P-symptoms in community samples, as well as personality traits and disorders implicated in CHR-P-symptom development [50, 52, 56]. One possible explanation for these associations is that cognitive biases function as the operational component of personality features, actively shaping and sustaining maladaptive beliefs which predispose individuals to psychopathology and psychosis risk [52]. Yet, despite growing evidence suggesting an association of cognitive biases with both personality pathology and CHR-P, existing research has not yet, to our knowledge, explored them together with either CHR-P or a specific focus on personality functioning [47, 50]. Therefore, we explored the associations of personality functioning impairment and cognitive biases with the presence and expression of CHR-P-symptoms in the community. More precisely, our primary research question investigated whether overall personality functioning impairment and cognitive biases were associated with the occurrence and severity of CHR-P-symptoms, controlling for associations with current psychopathology and socio-occupational functioning, as these factors are known to relate to CHR-P-symptom presentation

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[17, 57]. In a second step, consistent with the AMPD framework (Supplementary Materials, eTable 2), whenever personality functioning impairment (i.e., Criterion A) was significantly associated with CHR-P-symptom occurrence or severity, we further examined maladaptive personality traits (i.e., Criterion B) for associations with CHR-P-symptom occurrence and severity. Finally, to address possible differences between CHR-P-symptom subtypes, we additionally tested these associations on per-CHR-P- and nonper-CHR-P-symptoms separately, drawing on the evidence of differences in their manifestation, trajectory, and underlying mechanisms [5, 6, 8].

Methods

Recruitment and procedures

Our analyses involved cross-sectional data from an initial sample of 450 participants (age 17–60 years) who had completed the add-on questionnaires to the second follow-up (ethics ID: 2020–02856) of the "Bern Epidemiological At-Risk" ($N = 418$) and the "Bi-national Evaluation of At-Risk Symptoms in Children and Adolescents" ($N = 32$) community studies by November 2023 (see Supplementary Materials, eFigure 1 for details on the current sample; [5, 58, 59]). Requirements for participation in the add-on study were provision of *ad hoc* informed consent, fluency in German, and no history of psychosis. Data were collected via a main clinical interview conducted via telephone (duration: 45–90 minutes) and add-on self-report questionnaires, filled out online (unless participants expressly requested a paper copy, which they sent back via mail after completion). All data were recorded on REDCap electronic data capture tools (https://projectredcap.org) hosted at the University of Bern [60]. Results of a previous feasibility study supported the reliability of the telephone assessment, showing 78–100% concordance rates with face-to-face interviews [61]. Further information on study procedures and recruitment can be found in eText 1.

Assessments

CHR-P-symptoms

Presence of CHR-P-symptoms was evaluated during the telephone assessment with (i) the Structured Interview for Psychosis-Risk Syndromes (SIPS; [62]), assessing positive UHR-symptoms, and (ii) the Schizophrenia Proneness Instrument, in its Adult (SPI-A; [63]) and Child and Youth (SPI-CY; [64]) versions, assessing basic symptoms. Evidence indicated excellent median inter-rater reliability ($k = 0.89$), as well as strong predictive, convergent, and discriminant validity for the SIPS [65], good inter-rater reliability and discriminant validity for SPI-A [66] and SPI-CY [67].

SIPS-positive scales and SPI-A/CY-items are rated on a 0–6 scale according to their severity and frequency, respectively. We did not consider CHR-P criteria (Supplementary Materials, eTable 1), both because conversion was not our focus, and to increase power, as, consistently with data from earlier assessment times (e.g., [68]), an absolute minority of our sample met the criteria (0.22% for UHR, 2.67% for COPER, and 0.67% for COGDIS).

Next, we created three composite sum-scores by summing individual item scores (range: 0–6). First, we calculated: (i) a per-CHR-P-sum-score (0–18), by adding scores from the SIPS-P4 item and the two SPI-A/-CY items assessing perceptual abnormalities/hallucinations; and (ii) a nonper-CHR-P-sum-score (0–96), by summing scores from all remaining items (Supplementary Materials, eTable 1). These two scores were then added to obtain (iii) an overall CHR-P-sum-score (0–114).

Personality pathology

We evaluated severity of personality functioning impairment (Criterion A, AMPD) on the Level of Personality Functioning Scale-Brief Form 2.0 (LPFS-BF 2.0; [69]), which showed good reliability and construct validity [70]. Each item measures impaired functioning (0–3) in one of 12 facets, capturing impairments in identity, self-direction, empathy, and intimacy (i.e., personality functioning-elements), and providing an overall sum-score.

Further, we assessed maladaptive personality traits (Criterion B, AMPD) with the Personality Inventory DSM-5 (PID-5-BF; [71]), wherein scores (0–3) in 25 items are clustered in five higher-order personality trait domains (i.e., negative affect, detachment, antagonism, disinhibition, and psychoticism), and used to calculate an average total score. Evidence on this instrument showed medium to good convergence and discriminant validity [72].

Both instruments were filled out online.

Cognitive biases

Cognitive biases were evaluated with the Cognitive Biases Questionnaire for psychosis (CBQp; [73]), also administered online. The questionnaire assesses five cognitive distortions (i.e., jumping to conclusions, intentionalizing, catastrophizing, emotional reasoning, dichotomous thinking) of clinical relevance and high frequency in psychosis, using five subscales. For each of 30 vignettes describing everyday events, respondents choose the most likely between three alternative cognitive responses, illustrating absence (scored 1), possible (2), or likely presence (3) of interpretation bias. Then, summing item-scores resulted in an overall sum-score (30–90). The CBQp showed good internal consistency and excellent test–retest reliability, with its use of indirect questioning of cognitive biases, rather than their direct assessment and labeling, effectively countering the risk of report bias [73].

Psychopathology

We assessed current Axis I-psychopathology during the telephone interview with the Mini-International Neuropsychiatric Interview [74], based on DSM-IV psychiatric diagnoses and demonstrating acceptable to high accuracy as well as overall good psychometric properties [75–77]. A score of 1 on the scale assessing each disorder indicated its presence and contributed to the psychopathology sum-score (0–22) reflecting the number of current psychiatric diagnoses.

Socio-occupational functioning and sociodemographic variables

Functioning was assessed with the Social and Occupational Functioning Assessment Scale (SOFAS; 0–100; [75]), a widespread measure of functioning often chosen for its simplicity [78]. Further, we included sex, age, and education level (International Standard Classification of Education or ISCED [79]) as covariates in our models. This data was obtained during the main telephone assessment.

Statistical analyses

Data analysis was conducted in RStudio version 4.3.2., using the *stats* and *pscl* packages.

After listwise deletion of six observations with missing values, we z-standardized the sum-scores evaluating personality functioning impairment, cognitive biases, PID-5, current psychopathology, and socio-occupational functioning, as well as each subscale of the first three. Next, in order to account for overrepresentation of zeros in our outcome variables (i.e., CHR-P, per-CHR-P, and nonper-CHR-P-symptoms), we built three zero-inflated Poisson

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(ZIP) models [80–82]. ZIP models are particularly well-suited to modeling outcomes that are infrequent yet potentially of substantial relevance, making them appropriate for exploring factors contributing to CHR-P symptomatology in the community [81]. While traditional count models (e.g., Poisson regression) would likely lead to biased interpretation of this highly skewed data, ZIP models account for the existence of two distinct underlying processes suggested by the skewed distribution: one determining the likelihood of zero instances of the outcome and the other modeling the count of non-zero instances [81]. In our study, each ZIP model comprised (i) a zero-inflation model, describing how predictors and covariates influenced the likelihood of the outcome variable being zero on a binary distribution, (ii) a count model, describing how predictors and covariates influenced the actual value of the outcome variable on a Poisson distribution. Moreover, each model included: (i) the sum-scores for the two main predictors – personality functioning impairment and cognitive biases – and the control variables including current psychopathology and socio-occupational functioning; (ii) the covariates age, sex, and education level; (iii) the per-CHR-P-, nonper-CHR-P-, and CHR-P-sum-scores as the respective outcome. Then, in the final sample (N = 444), we tested each ZIP model against an equivalent Poisson model, wherein a lower Akaike Information Criterion indicated better data fit [83]. In models where personality functioning was a significant predictor ($p < .05$), we included the PID-5-sum-score (maladaptive personality traits) as an additional predictor, and ran a Likelihood Ratio test with the *lmtest* R-package, wherein significance ($p < .05$) indicated improved model fit. In models where personality functioning or cognitive biases were significant predictors ($p < .05$), we reiteratively replaced them with each of their subscales to analyze their individual contribution, thus testing 19 additional models. Our choice of this procedure, and against simultaneous inclusion of all subscales in one model, was made to avoid multicollinearity, which can arise from high correlations between subscales of an instrument or between instruments measuring related constructs (e.g., LPFS and PID-5, both measuring features of personality). Results of this explorative analysis should be interpreted with caution.

We did not correct for multiple testing in light of (i) the limited number of statistical tests involving the two main predictors (personality functioning and cognitive biases) across three models (six in total), (ii) the correlation between our three outcomes (CHR-P-, per-, and nonper-CHR-P-symptoms), and (iii) the overall exploratory nature of our calculations, which did not involve exact hypotheses on associations between the main variables. All together, these factors determined a limited risk of Type I error, which should most critically be controlled for via multiple testing correction when conducting several comparisons between independent data or in confirmatory designs [84, 85]. In our design, this was weighed against the greater risk of obtaining excessively conservative effect estimates by adjusting p-values [86, 87], and the procedure was considered inappropriate.

Results

Sample characteristics

Our sample comprised a majority of adult (99.77%), female, highly educated, functionally unimpaired (SOFAS > 70; 94.4%) participants (Table 1). As expected in a community sample, most participants showed no current axis-I disorders, personality functioning impairment was below clinical levels, maladaptive personality traits were below reported elevation cut-offs (Table 1, Supplementary Materials, eTable 2; e.g., [88]), and for most participants the CHR-P- (76.44%), per-CHR-P- (83.56%), and nonper-CHR-P-sum-scores (85.33%) were zero (Figure 1).

ZIP models

When compared by data fit, each ZIP model outperformed its equivalent Poisson model (Supplementary Materials, eTable 3) and was therefore retained for further analyses.

CHR-P-symptoms

In the zero-inflation model, more current axis-I diagnoses ($\gamma = -0.69 \pm 0.19$, $p < .001$) and more severe cognitive biases ($\gamma = -0.41 \pm 0.15$, $p = 0.006$) were associated with a lower likelihood of the CHR-P-sum-score being 0 (Figure 2a). Additionally, younger age ($\beta = -0.03 \pm 0.01$, $p < .001$), female sex ($\beta = 0.32 \pm 0.16$, $p = 0.045$), and more severe cognitive biases ($\beta = 0.20 \pm 0.07$, $p = 0.005$) were associated with higher CHR-P-sum-scores in the count model (Figure 2b and c). personality functioning was not a significant predictor of either CHR-P-symptom likelihood or severity (Supplementary Materials, eTable 4).

In the exploratory analyses examining individual cognitive biases, more severe catastrophizing ($\gamma = -0.37 \pm 0.15$, $p = .01$), dichotomous thinking ($\gamma = -0.27 \pm 0.13$, $p = .04$), and emotional reasoning ($\gamma = -0.30 \pm 0.13$, $p = .02$) were associated, in their respective zero-inflation models, with lower likelihood of CHR-P-sum-scores being 0. Additionally, in the corresponding count models, more severe dichotomous thinking ($\beta = 0.11 \pm 0.05$, $p = .03$) and emotional reasoning ($\beta = 0.21 \pm 0.06$, $p < .001$) were associated with higher CHR-P-sum-scores.

Perceptive CHR-P-symptoms

In the zero-inflation model considering only per-CHR-P-symptoms, more current axis-I diagnoses ($\gamma = -0.76 \pm 0.18$, $p < .001$) and more severe cognitive biases ($\gamma = -0.52 \pm 0.18$, $p = .003$) were associated with lower likelihood of the outcome value being 0 (Figure 3a). In the count model, only younger age was associated with higher per-CHR-P-sum-scores ($\beta = -0.02 \pm 0.01$, $p = .03$) (Figure 3b). Personality functioning did not significantly predict either per-CHR-P-symptom likelihood or severity (Supplementary Materials, eTable 5).

As for individual cognitive biases, more severe dichotomous thinking ($\gamma = -0.31 \pm 0.14$, $p = .03$) and emotional reasoning ($\gamma = -0.40 \pm 0.15$, $p = .008$) were associated – in their respective zero-inflation models – with lower likelihood of per-CHR-P-sum-scores being 0. In the count model, intentionalizing and per-CHR-P-sum-scores were negatively correlated ($\beta = -0.20 \pm 0.10$, $p < .04$).

Non-perceptive, delusional, or cognitive CHR-P-symptoms

In the zero-inflation model of nonper-CHR-P-symptoms, more impaired personality functioning ($\gamma = -0.64 \pm 0.26$, $p = .02$) and more current axis-I diagnoses ($\gamma = -0.76 \pm 0.28$, $p = .007$) were associated with lower, while higher socio-occupational functioning ($\gamma = 0.61 \pm 0.31$, $p = .48$) and education level ($\gamma = 0.85 \pm 0.40$, $p = .03$) with higher likelihood of having an outcome score of 0 (Figure 4a). Moreover, in the count model, more severe cognitive biases were associated with higher nonper-CHR-P-sum-scores ($\beta = 0.43 \pm 0.11$, $p < .001$) (Figure 4b; see Supplementary Materials, eTable 6 for results including non-significant predictors).

Since personality functioning impairment was a significant predictor in this model, we included maladaptive personality traits as an additional predictor and compared the two models via a Likelihood Ratio test, which was non-significant ($p = .13$; Supplementary Materials,

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Table 1. Sample characteristics (N = 450)

	n	%
Age (mean ± SD, median, range)	39.38 ± 8.56, 42, 17–60	
Sex (female)	278	61.78
Highest professional education (ISCED level)[a]		
Early childhood education (ISCED 0)	0	0
Primary school or school for special needs (ISCED 1)	0	0
Secondary school (ISCED 2)	6	1.33
High school (ISCED 3.4)	6	1.33
High school-level professional education (ISCED 3.5)	13	2.89
Post-secondary non-tertiary education (ISCED 4)	6	1.33
Short cycle tertiary education, bachelor or master (ISCED 5)	405	90.00
Doctoral (ISCED 6)	12	2.67
SOFAS score (mean ± SD, median, range)	84.6 ± 7.81, 88, 47–95	
Current axis-I disorders, sum-score (mean ± SD, median, range)	0.1 ± 0.38, 0, 0–3	
Current CHR-P-symptoms, sum-score (mean ± SD, median, range)	0.67 ± 1.56, 0, 0–13	
Current per-CHR-P-symptoms, sum-score (mean ± SD, median, range)	0.38 ± 0.99, 0, 0–6	
Current nonper-CHR-P-symptoms, sum-score (mean ± SD, median, range)	0.28 ± 0.86, 0, 0–7	
LPFS 2.0-BF, sum-score (mean ± SD, median, range)[b]	0.68 ± 0.42, 0.67, 0–2.08	
Identity (mean ± SD, median, range)[a]	0.73 ± 0.62, 0.67, 0–2.67	
Self-direction (mean ± SD, median, range)[c]	0.74 ± 0.58, 0.67, 0–3	
Empathy (mean ± SD, median, range)[c]	0.70 ± 0.48, 0.67, 0–2.33	
Intimacy (mean ± SD, median, range)	0.54 ± 0.52, 0.33, 0–2.33	
CBQp sum-score (mean ± SD, median, range)[a]	37.65 ± 3.68, 37, 31–55	
Intentionalizing (mean ± SD, median, range)[c]	7.08 ± 0.91, 7, 6–11	
Catastrophizing (mean ± SD, median, range)[a]	7.43 ± 1.18, 7, 6–13	
Dichotomous thinking (mean ± SD, median, range)	6.73 ± 0.97, 6, 6–13	
Jumping to conclusions (mean ± SD, median, range)	8.73 ± 1.34, 9, 6–15	
Emotional reasoning (mean ± SD, median, range)[a]	7.68 ± 1.43, 7, 6–14	
PID–5 BF (mean ± SD, median, range)	0.43 ± 0.26, 0.40, 0–1.24	
Negative affectivity (mean ± SD, median, range)	0.84 ± 0.54, 0.80, 0–2.60	
Detachment (mean ± SD, median, range)	0.51 ± 0.49, 0.40, 0–2.40	
Antagonism (mean ± SD, median, range)	0.28 ± 0.33, 0.20, 0–1.80	
Disinhibition (mean ± SD, median, range)	0.53 ± 0.46, 0.40, 0–2.60	
Psychoticism (mean ± SD, median, range)	0.50 ± 0.46, 0.40, 0–2.20	

Abbreviations: SOFAS, Social and Occupational Functioning Assessment Scale; CHR-P, clinical high-risk of psychosis; per-CHR-P, perceptive CHR-P; nonper-CHR-P, non-perceptive CHR-P; LPFS-BF 2.0, Level of Personality Functioning Scale-Brief Form 2.0; CBQp, Cognitive Biases Questionnaire; PID-5-BF: Personality Inventory DSM-5 Brief Form.
[a]Data from two participants (0.44%) were missing.
[b]Data from three participants (0.67%) were missing.
[c]Data from one participant (0.22%) were missing.

eTable 7), indicating that the new model did not improve fit to our data. Thus, it was discarded.

Finally, examining the impact of individual cognitive biases and personality functioning elements, we found that more pronounced catastrophizing ($\gamma = -0.48 \pm 0.21$, $p = .02$), identity impairments ($\gamma = -0.76 \pm 0.26$, $p = .003$), and self-direction impairments ($\gamma = -0.56 \pm 0.22$, $p = .009$) were associated with lower likelihood of nonper-CHR-P-sum-scores being 0 in the relevant zero-inflation models. In the corresponding count models, more severe intentionalizing ($\beta = 0.34 \pm 0.10$, $p < .001$), dichotomous thinking ($\beta = 0.36 \pm 0.09$, $p < .001$), and emotional reasoning ($\beta = 0.30 \pm 0.09$, $p < .001$) were linked to higher nonper-CHR-sum-scores, while higher impairments in identity ($\beta = -0.26 \pm 0.11$, $p = .03$) were associated with lower nonper-CHR-P-sum-scores.

Discussion

In this community study, we investigated the association of personality pathology and cognitive biases with CHR-P-symptom (i.e., UHR- and basic symptom) expression. In our findings,

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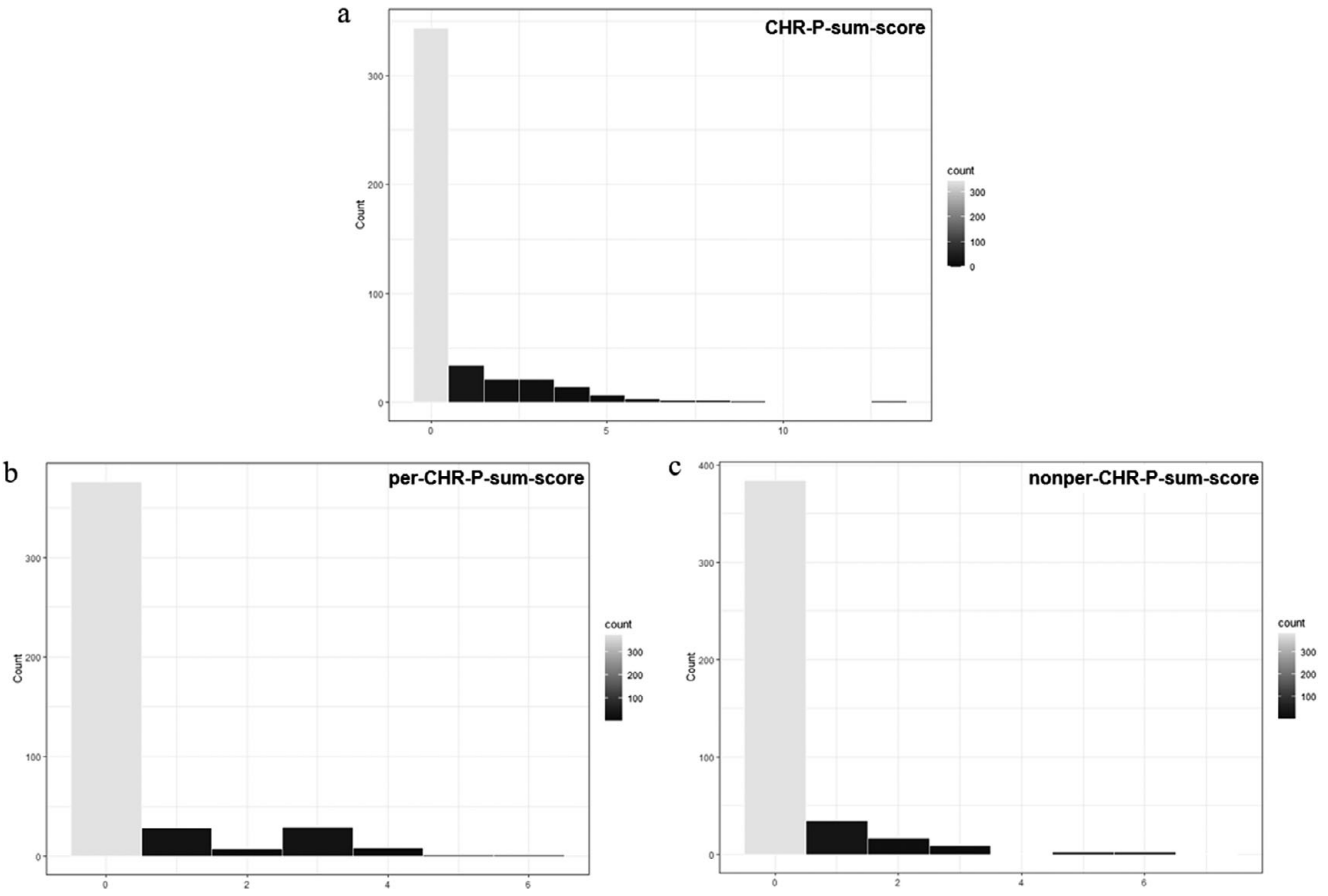

Figure 1. Sample distribution of CHR-P (Figure 1a), per-CHR-P (Figure 1b), and nonper-CHR-P (Figure 1c) sum-scores. On the *x*-axis: sum-score value; on the *y*-axis: number of participants ("count") presenting with each sum-score value.

personality functioning was specifically associated with the presence of nonper-CHR-P-symptoms, with maladaptive personality traits not substantially contributing to the respective model. In contrast, cognitive biases significantly correlated with both the presence and severity of CHR-P-symptoms, showing a differential relationship to per- and nonper-CHR-P-symptoms.

Further, exploring the association between psychopathology and socio-occupational functioning with CHR-P-symptom expression, we found a positive association across models between more axis-I diagnoses and the likelihood of CHR-P-symptoms, while socio-occupational functioning was negatively associated with nonper-CHR-P-symptom likelihood only. The implications of our findings and our exploratory analyses involving personality functioning elements and individual cognitive biases will be discussed below.

Personality functioning: Connections with nonper-CHR-P-symptoms

Overall, our results suggest a specific association between personality functioning impairment and a greater likelihood of nonper-CHR-P-symptoms, providing preliminary indications that the reported robust link between nonper-CHR-P- (especially UHR-) symptoms and impairment in psychosocial functioning [9] might extend to include personality functioning impairment. Conversely, we found no significant association between personality functioning impairment and either overall CHR-P-symptoms or per-CHR-P-symptoms. These findings highlight the need to further investigate the differential associations between personality functioning impairment and different categories of CHR-P-symptoms, for example, using data where rates of per- and nonper-CHR-P-symptoms allow for direct group comparison (see [9]). Considering (i) the established hypothesis linking nonper-CHR-P-symptoms to later-stage brain/cognitive maturation processes involving frontal regions [10] and (ii) existing evidence on frontal region activation in self- and other-referential processing relevant to personality functioning [89], future research should explore developmental and neurobiological correlates that might underlie the connection between nonper-CHR-P-symptoms and personality functioning in our study. Moreover, as negative CHR-P-symptoms were not assessed in the BEAR and BEARS-Kid studies, they were not considered in the current analysis. However, previous research has highlighted differential associations between personality pathology and positive versus negative subclinical psychotic symptoms [52], suggesting that some aspects of the relationship between personality functioning and nonper-, or even per- and overall CHR-P-symptoms, might have been masked in our analysis.

Additionally, our exploratory analysis indicated that the association between higher personality functioning impairment and greater likelihood of nonper-CHR-P-symptoms might particularly concern impairments in identity and self-direction (i.e., self-functioning). In our analysis, the nonper-CHR-P-sum-score predominantly consists of cognitive basic symptoms, which then likely weighed more on the statistical analyses than their UHR-symptom counterparts. Since basic symptoms are subjective disturbances, involving changes in mental processes that are immediately perceived to be distinct from those familiar to the self, they are by

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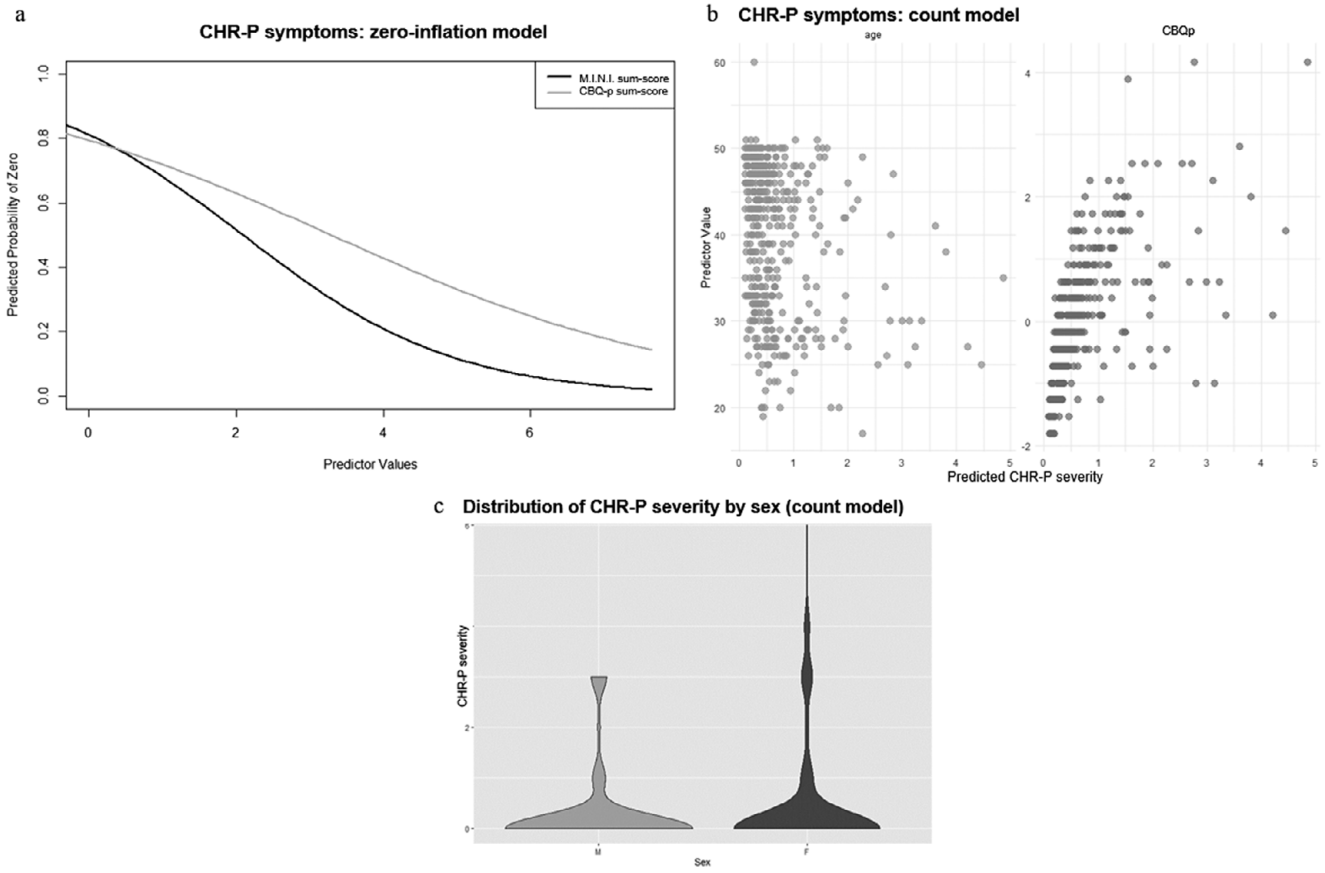

Figure 2. ZIP model results for CHR-P-symptoms. Figure 2a: Zero-inflation model. The *x*-axis shows values of the significant predictor, control variable, or covariate, while the *y*-axis shows the probability of CHR-P-symptoms being zero (e.g., the higher the CBQp-sum-score, indicating more severe cognitive biases, the lower the probability of CHR-P-symptoms being zero). Figure 2b: Count model. The *x*-axis shows predicted CHR-P-symptom severity, while the *y*-axis shows values of the significant predictor, control variable, or covariate (e.g., the younger the age, the higher the predicted CHR-P-symptom severity; the higher the CBQp-sum-score, indicating more severe cognitive biases, the higher the predicted CHR-P-symptom severity). Figure 2c: Count model. The x-axis organizes the data by the significant categorial covariate sex, while the *y*-axis shows predicted CHR-P-symptom severity. Females (F) tend to have a broader distribution of CHR-P-symptom severity, with higher participant density at both lower and higher CHR-P-symptom severity levels, compared to males (M).

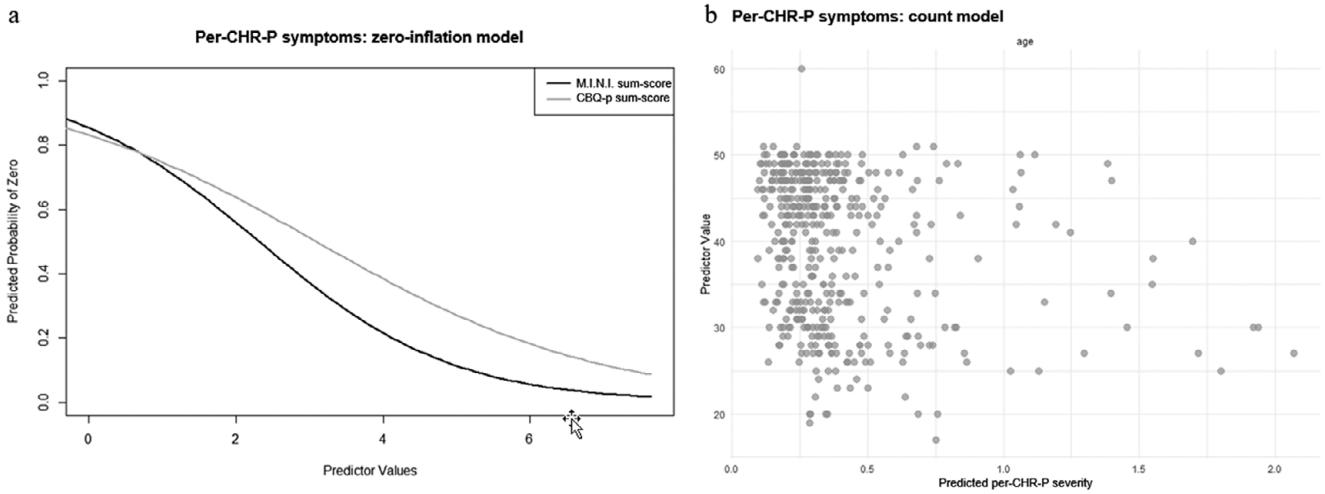

Figure 3. ZIP model results for per-CHR-P-symptoms. Figure 3a: Zero-inflation model. The *x*-axis shows values of the significant predictor, control variable, or covariate, while the *y*-axis shows the probability of per-CHR-P-symptoms being zero (e.g., the higher the CBQp-sum-score, indicating more severe cognitive biases, the lower the probability of CHR-P-symptoms being zero). Figure 3b: Count model. The *x*-axis shows predicted per-CHR-P-symptom severity, while the *y*-axis shows values of the significant predictor, control variable, or covariate (e.g., the younger the age, the higher the predicted CHR-P-symptom severity).

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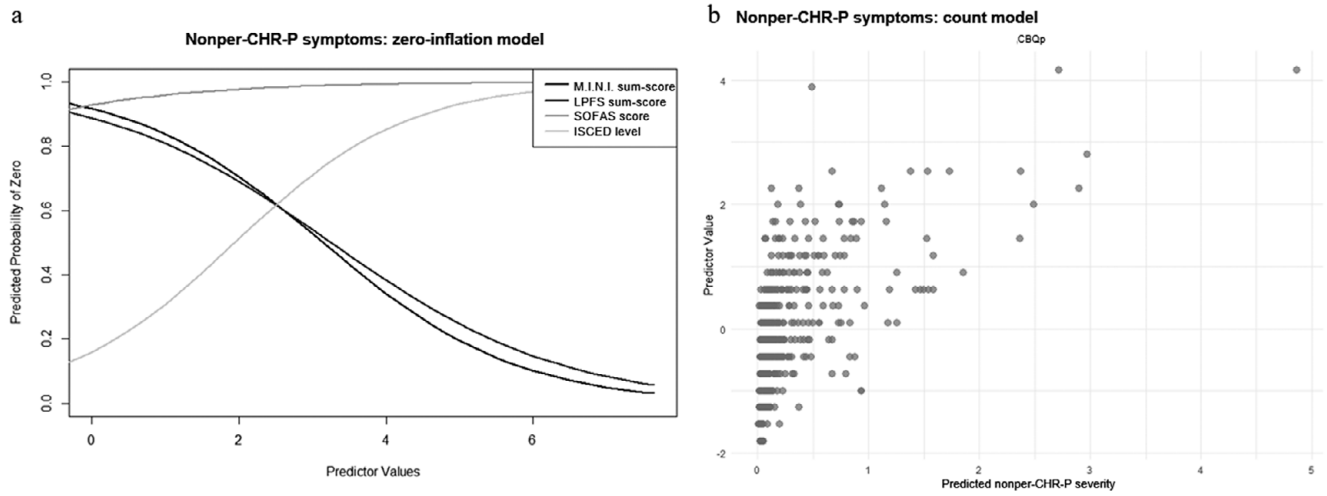

Figure 4. ZIP model results for nonper-CHR-P-symptoms. Figure 4a: Zero-inflation model. The *x*-axis shows values of the significant predictor, control variable, or covariate, while the *y*-axis shows the probability of nonper-CHR-P-symptoms being zero (e.g., the higher the SOFAS-sum-score, indicating higher socio-occupational functioning, the higher the probability of nonper-CHR-P-symptoms being zero; the higher the LPFS-sum-score, indicating higher personality functioning impairment, the lower the probability of nonper-CHR-P-symptoms being zero). Figure 4b: Count model. The *x*-axis shows predicted nonper-CHR-P-symptom severity, while the *y*-axis shows values of the significant predictor, control variable, or covariate (e.g., the higher the CBQp-sum-score, indicating more severe cognitive biases, the higher the predicted nonper-CHR-P-symptom severity).

definition related to the self [40, 90, 91]. In turn, this close association with the self might then help explain the link between nonper-CHR-P-symptoms and personality functioning in our results. Moreover, this finding aligns with research connecting deficits in the corresponding personality functioning facets (e.g., self-others boundaries, emotional regulation abilities, self-esteem, productive self-reflection) to CHR-P-symptom expression and course [27, 40, 45, 46], although specific evidence on nonper-CHR-P-symptoms is lacking. In contrast, our finding of an association between higher impairments in identity and lower severity of nonper-CHR-P-symptoms seems incoherent with this reasoning. Possibly indicating a more complex relationship between identity and nonper-CHR-P-symptom expression, this warrants investigation beyond the scope of our cross-sectional study, under consideration of potential intervening factors, such as identity formation processes or positive resources buffering against nonper-CHR-P-symptom severity [5, 92, 93]. Speculatively, identity impairment might serve as a vulnerability factor for nonper-CHR-P-symptoms in their "trait-like," "as-usual" manifestation, reflecting long-standing patterns less directly related to burden and psychosis risk [3,63]. Several other explanations for this finding, including Type I error, are also possible and should be rigorously tested in future studies. Finally, including maladaptive personality traits as a predictor of nonper-CHR-P-symptom expression did not improve this model. As our study design was guided by the AMPD, we only considered maladaptive personality traits when personality functioning showed a significant association to CHR-P-symptom expression, that is, only for nonper-CHR-P-symptoms. Therefore, while our work provides some support to the hypothesis that overarching features of personality, such as personality functioning, might be more closely associated with CHR-P expression than maladaptive traits [39], a better comprehension of their role should be pursued in future research, including all categories of CHR-P-symptoms as well as clinical samples.

Cognitive biases: Unpacking complex associations

As a whole, more severe cognitive biases showed an association with both higher likelihood and severity of CHR-P-symptoms. Previous research has described a longitudinal link between cognitive biases and CHR-P-symptoms, proposing that cognitive biases might become a stable cognitive functioning feature, predisposing individuals to developing CHR-P-symptoms [94–96]. Furthermore, literature indicates that cognitive biases impact on multiple levels of perception, information processing, and related emotional reactions (e.g., worry), potentially interacting with stress responses that influence CHR-P-symptom severity [95, 97, 98]. While this reasoning aligns with our findings, we cannot disentangle whether (more severe) cognitive biases might be a consequence or a vulnerability/exacerbating factor of CHR-P-symptoms using our cross-sectional data [95, 99]. Addressing this question in longitudinal research might both expand our understanding of CHR-P-symptom expression and inform preventive interventions. Moreover, in our exploratory analysis, more severe dichotomous thinking, emotional reasoning, and catastrophizing were associated with higher likelihood of CHR-P-symptoms, with the first two also correlating with higher CHR-P-symptom severity. Consistent with existing data linking these cognitive biases to the presence and severity of subclinical positive symptoms in healthy individuals [100–102], these findings suggest that future research should explore their specific relevance to CHR-P-symptom expression in the community.

Furthermore, more severe cognitive biases were associated with higher likelihood of per- and severity of nonper-CHR-P-symptoms. Although our cross-sectional design precludes testing for directionality, the differential associations in our findings might reflect distinct underlying mechanisms and should be explored in future longitudinal studies. Based on our results, we might speculate that the predisposing function of cognitive biases for the development of CHR-P-symptoms is more closely related to per-CHR-P-symptoms and the connected earlier-stage maturation processes, while the impact of cognitive biases on CHR-P-symptoms rather concerns nonper-CHR-P-symptoms and the relative later-stage development processes [5, 6, 96, 102]. However, we wish to reiterate that this interpretation exceeds the scope of our study, and should only exemplify how our preliminary findings might help structuring hypotheses on the relationship between cognitive biases and per- versus nonper-CHR-P-symptoms, to then be tested elsewhere. Further, considering individual cognitive biases, the severity of dichotomous thinking and

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emotional reasoning was associated with increased likelihood of per-CHR-P-symptoms, consistent with previous findings in individuals with subclinical auditory hallucinations [100]. Similarly, we found an association of more severe dichotomous thinking and emotional reasoning with higher severity of nonper-CHR-P-symptoms, aligning with existing evidence on delusions [101]. Additionally, higher catastrophizing was associated with higher likelihood of nonper-CHR-P-symptoms, and higher intentionalizing with higher severity of nonper-CHR-P-symptoms. This reflected existing evidence on a link between catastrophizing and a higher likelihood of delusion presence and between intentionalizing and greater delusion severity [101]. Interestingly, higher intentionalizing correlated with less severe per-CHR-P-symptoms, suggesting a more complex relationship. This association might be influenced by the fact that, while evidence linked intentionalizing to perceptive symptoms via (the emotional component of) hallucinations [103], our per-CHR-P-sum-score predominantly consisted of basic symptoms. As this is true for all sum-scores, and evidence regarding the relationship between cognitive biases and basic symptoms is currently lacking, our results should overall be interpreted with caution and further investigated, especially considering our cross-sectional, explorative design. Offering an additional explanation for their correlation in our analyses, cognitive biases and (cognitive) basic symptoms both refer to aspects of cognitive functioning and thus, might have a reciprocal influence. Nonetheless, the two concepts are clearly distinct, with cognitive biases operating on the higher-level cognitive process of interpretation, which becomes systematically negatively distorted [52], whereas basic symptoms represent qualitative distortions in lower-level cognitive processes, like attention or concentration [4]. Finally, jumping to conclusions was the only cognitive bias for which severity was not associated with CHR-P-symptom expression. This aligns with indications that its influence might be specific to schizophrenia and active psychotic symptoms [56, 102, 104, 105]. Additionally, self-reporting on cognitive biases, and specifically on jumping to conclusions, might be skewed by factors like metacognitive awareness, which might lead community samples to report lower rates of jumping to conclusions (e.g., for reasons of social desirability) when compared to individuals with psychosis, whose metacognitive awareness might already be impaired. Overall, putting our results into perspective, previous research proposed that a general distorted thinking style (CBQp-sum-score) might be more clinically relevant than individual cognitive biases, for which evidence of distinct underlying distorted cognitive processes is inconsistent [56, 73, 94, 95].

Psychopathology, functioning, and socio-demographics

In our analyses, current presence of more axis-I-diagnoses was associated with greater likelihood of CHR-P, per-CHR-P and nonper-CHR-P-symptoms, aligning with copious evidence of high comorbidity rates in CHR-P samples [18]. Further, lower socio-occupational functioning was associated with higher likelihood of nonper-CHR-P-symptoms, consistent with data on the close connection of especially non-perceptive UHR-symptoms with impaired functioning [9, 10, 17]. Moreover, findings of a significant link between age and overall CHR-P−/per-CHR-P-, but not nonper-CHR-P-, symptom severity are consistent with literature, but developmental implications cannot be drawn from our cross-sectional analyses [9, 10]. Finally, the link between female sex and higher CHR-P-symptom severity joins inconclusive evidence about sex effects on CHR-P expression [5]. Thus, findings involving age and sex require further investigation in future studies.

Strengths and limitations

Next to the clear strengths of our study including the innovative focus on personality functioning in relation to CHR-P-symptoms and cognitive biases, and the large community sample, some limitations should be considered. As mentioned, no directionality can be inferred from our cross-sectional data, although, given the predominantly trait-like nature of cognitive biases and personality characteristics [29, 45], it seems plausible that they precede the state-like CHR-P-symptoms [106]. Further, in our exploratory analysis, we included individual cognitive biases and personality functioning elements separately in the relevant models to avoid multicollinearity, favored by high correlations between the subscales; this, however, also prevented examination of their interplay. Moreover, while we partially corrected for this in the outcomes variables by choosing to employ ZIP models, the low levels of impairment and pathology in our sample may restrict generalizability to other populations, as statistical power to detect associations within these limited ranges may be reduced. Additionally, data on negative, general, and disorganization SIPS-symptom scales, which might add more context to our findings [52], were not available to us, as assessments in the BEAR and BEARS-Kid studies focused on criteria-relevant UHR- and basic symptoms. Finally, as our sum-scores combine both basic symptoms and UHR-symptoms, the contributions of procedural versus content-related thought disorders are not discernible in our findings.

Conclusion and future directions

The present study offers initial evidence on the intricate associations between personality functioning, cognitive biases, and CHR-P-symptomatology. First, nuanced associations of personality functioning, particularly identity and self-direction, with nonper-CHR-P manifestations emerged, alongside first indications of their relevance beyond maladaptive traits or personality disorders. Second, consistent with previous clinical studies [56], cognitive biases, and especially dichotomous thinking, emotional reasoning, and catastrophizing, arise as promising targets for future research on prevention through their association with CHR-P-symptoms likelihood and severity. Finally, our results support previous evidence on connections between nonper-CHR-P-symptoms and functioning impairment, as well as overall CHR-P expression and psychopathology [18]. Future longitudinal studies should test the associations in our findings and further investigate the complex interactions of personality pathology, psychosis risk, their related burden, and possible developmental implications, to extend our understanding of CHR-P-symptomatology.

Supplementary material. The supplementary material for this article can be found at http://doi.org/10.1192/j.eurpsy.2024.1812.

Data availability statement. Data can be made available on request via the corresponding author (C.M.).

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Competing interest. Drs Lerch, Schultze-Lutter, Schmidt, Cavelti, Kaess and Michel, and Ms Rinaldi (MSc) have declared that they have no conflicts of interest in relation to the subject of this study.

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