1. General abstract

Psychotic-like experiences (PLEs) consist of both hallucination-like experiences (HLEs) and delusion-like experiences (DLEs). They are defined as subclinical experiences characterised by milder intensity and constituting a category of often transient experiences that do not fully meet the accepted criteria for psychotic symptoms. In the current literature, PLEs are considered symptoms on a continuum ranging from the absence of such experiences to full-blown psychosis. Estimates suggest that between 5 and 10% of adults in the general population report experiencing PLEs at some point in their lives (Drvaric & Bagby, 2021). The prevalence of PLEs has been observed to peak among young adults (18–35 years old), where it may reach as high as 32 to 35% (Rep et al., 2023). While the majority of individuals' PLEs (~80%) spontaneously resolve over time, a significant minority (~7%) exhibit an intensification of symptoms (Linscott & Van Os, 2013). This suggests that PLEs may be a risk factor for the development of full-blown psychosis in the future. Therefore, understanding the risk factors and mechanisms underlying their development remains an important area of research.

According to the integrated sociodevelopmental-cognitive model of psychosis (Howes & Murray, 2014), schizophrenia is conceptualised as a neurodevelopmental disorder, in the etiology of which a complex interaction of biological, cognitive, and environmental factors plays a significant role. The neurodevelopmental approach posits that schizophrenia can be regarded as a consequence of the interplay between events during early development and the co-occurrence of genetic predispositions associated with other conditions whose clinical symptoms are already present in childhood. This multifactorial process determines an individual's risk of developing the disorder, even in the absence of psychotic symptoms. Research findings consistently indicate that adverse experiences during early childhood also constitute a significant risk factor for the future development of psychosis. In the model proposed by Howes and Murray (2014), it is suggested that these experiences may affect the individual's cognitive schemas, which are used to interpret the surrounding environment. This, in turn, may result in the dysregulation of the dopaminergic system and its heightened activity, leading to the activation of a mechanism involving the attribution of excessive significance to neutral or ambiguous stimuli (Aberrant Salience). These stimuli, then, are interpreted by the individual through the distorted cognitive schemas that have been previously developed. This process can result in paranoid interpretations, hallucinations, or heightened monitoring of potential threats in a neutral environment. It creates a vicious cycle in which psychotic experiences generate further stress, intensifying dopamine dysregulation and reinforcing the resulting experiences. From a biological perspective, experienced psychosocial stress triggers a series of mechanisms, including activation of the hypothalamic-pituitary-adrenal axis (HPA). Short-term activation of the HPA axis enables the adaptive response of the body to stress; however, its chronic or excessive activation can lead to increased reactivity to stressors and dopaminergic system hypersensitisation. However, it is important to emphasise that psychosocial stress or traumatic experiences are neither necessary nor enough to cause PLEs, which highlights the multifactorial nature of this phenomenon.

Among the neurodevelopmental factors associated with the etiology of PLEs, increasing emphasis is being given to the role of Attention Deficit Hyperactivity Disorder (ADHD). In individuals exhibiting ADHD symptoms, HPA axis dysfunction is observed, which is associated with cognitive deficits (e.g., difficulty concentrating) and behavioural deficits (e.g., impulsivity). Research has demonstrated that individuals with ADHD symptoms are more likely to report elevated levels of subjectively perceived stress in daily life. A comparable relationship is observed in individuals experiencing PLEs, which may also be associated with HPA axis dysregulation in this group. Consequently, these individuals become more sensitive to stress, and the resulting cognitive distortions become entrenched. This, in turn, may lead to a further intensification of PLEs. Furthermore, the heightened stress reactivity reported in both groups – encompassing emotional processes (e.g. increased overall stress levels or experiencing a greater number of negative emotions), cognitive processes (e.g. a tendency towards rumination, impaired emotion regulation, misattribution of meaning to irrelevant stimuli), and physiological processes (e.g. heightened cortisol levels), appears to reinforce the links between ADHD symptoms, stress, and PLEs. In particular, these associations are clinically significant, as ADHD and psychotic experiences are frequently reported as comorbid conditions.

Despite the growing body of research focusing on the relationships between neurodevelopmental, psychosocial, and physiological factors, the underlying mechanisms and processes remain not fully understood. In particular, there is a lack of studies that comprehensively address affective, cognitive, and physiological processes in the relationship between the co-occurrence of ADHD symptoms and PLEs.

The present dissertation consists of three articles employing self-report measures and longitudinal data collected using the Experience Sampling Method (ESM). The main objective of this dissertation was to examine the role of ADHD symptoms in exacerbating PLEs by taking into account selected risk factors, such as adverse social experiences, as well as cognitive, emotional, and physiological factors. The studies encompass analyses of the interaction effect between PTSD (Post-Traumatic Stress Disorder) and ADHD symptoms, in the intensity of PLEs (**Study 1**); a serial mediation model that tests the mediating role of ruminations and negative emotions in the relationship between ADHD symptoms and PLEs, and a moderated serial mediation model that examines whether traumatic experiences strengthen this relationship (**Study 2**); and complex network analysis models which demonstrate the relationships between cortisol levels, PLEs, negative emotions, ruminations, threat anticipation, and aberrant salience in individuals with varying severity of ADHD symptoms (**Study 3**).

The **first study** in this series (*N = 3000*) aimed to examine the interactive effect of PTSD and ADHD symptoms on the level of reported PLEs in a non-clinical population (aged 18–35). To date, no previous study has examined the interaction between PTSD and ADHD symptoms in this context; previous research has only indicated that they tend to co-occur. The findings of the present study demonstrated that the interaction between ADHD and PTSD symptoms exerted a significant influence on the intensity of reported PLEs. The highest level of PLEs was observed in individuals who tested positive for both ADHD and

PTSD. Individuals who exhibited a positive screening result for a single variable also demonstrated higher levels of PLEs in comparison to those who exhibited negative screening results for both conditions. These results underscore the importance of the interaction effect between ADHD and PTSD symptoms in the context of reported PLEs.

The **second study** (*N = 188*) aimed to test a serial mediation model in which rumination and negative emotions were included as mediators in the relationship between ADHD symptoms and PLEs. The second step of this study was to examine whether traumatic experiences strengthen the relationship under investigation. This study utilised weekly-averaged longitudinal data collected during a one-week ESM procedure, in which participants' daily emotional and cognitive fluctuations were monitored. The findings of this study indicated the presence of a statistically significant serial mediation, thereby confirming the hypothesis that ruminative thoughts and negative emotions mediate the relationship between ADHD symptoms and PLEs. We demonstrated that ADHD symptoms are significantly associated with increased rumination, which in turn may lead to a greater experience of negative emotions, which consequently may lead to a greater intensity of PLEs. However, in the tested model of serial moderated mediation, the relationship between ADHD symptoms and PLEs, mediated by rumination and negative emotions, was not confirmed as being strengthened by the experience of traumatic life events.

The **third study** in this series (*N = 188*) was based on data collected during the one-week ESM procedure in Study 2 and was presented in the form of dynamic, temporal network analyses. Initially, the analyses examined dynamic relationships between PLEs, affective components (negative emotions), ruminative thoughts, physiological components (cortisol levels), and cognitive components (i.e., threat anticipation and aberrant salience). Network models were estimated for the entire study group and separately for two groups differing in the severity of ADHD symptoms (high ADHD, *N = 96*; low ADHD, *N = 92*). The second stage of this study involved conducting comparative analyses of network structures between groups of individuals with varying ADHD symptom severity. By integrating ecological data and stress biomarkers with network analysis, this study revealed the dynamic nature of these complex relationships. The findings indicated that within the group exhibiting high ADHD symptom severity, delusion-like experiences emerged as the most significant predictor, exerting a substantial influence on the other variables and becoming a central node in the network. This finding suggests that in individuals with severe ADHD symptoms, delusion-like experiences may significantly influence the perception of reality, potentially acting as a trigger for subsequent emotional or cognitive reactions. Moreover, in the group with higher ADHD symptom severity, the results indicated a significant association between cortisol levels and threat anticipation, aberrant salience, and alcohol consumption at the subsequent time point. Conversely, within the group exhibiting lower ADHD symptom severity, the central node in the network, which exerted the strongest influence on the other areas included in the model, was negative emotions, while cortisol demonstrated a significant association only with subsequent alcohol consumption. Finally, network comparison analyses between the groups partially confirmed our hypothesis that cortisol levels would be

more strongly associated with PLEs in the group with higher ADHD symptom severity compared to the group with lower severity. The findings indicated that within the group with higher ADHD symptom severity, delusion-like experiences demonstrated greater stability and persistence over time. Furthermore, a stronger association was observed between these experiences and risk-seeking behaviour and alcohol consumption. In addition, a significant mediating pathway was identified between delusion-like experiences and negative emotions via alcohol consumption. The results of this study suggest that alcohol may be a maladaptive coping style in response to these experiences. The observed stronger associations between cortisol levels and risk-seeking in this group indicate a link between the physiological stress response and the subsequent interpretation of the situation as threatening.

In summary, the series of three articles presented in this dissertation contributes to a deeper understanding of the mechanisms underlying PLEs in a non-clinical population by integrating affective, cognitive, and physiological processes. We have demonstrated that the relationship between ADHD symptoms and PLEs is complex and multifactorial. Overall, the results of the studies presented in this thesis consistently indicate that ADHD symptoms constitute a significant risk factor for the intensification of PLEs. Furthermore, the results may suggest that ADHD is not only a co-occurring disorder but also a factor associated with the dynamics of emotional, cognitive, and physiological processes, which may be one of the factors explaining individual differences in the dynamics of PLEs in a population of young adults.

The findings also have significant clinical implications. First and foremost, they can serve as a point for the development of improved early intervention strategies for individuals with ADHD symptoms who are at increased risk of experiencing PLEs. Moreover, the necessity to incorporate adaptive emotion regulation techniques into work with individuals diagnosed with ADHD is emphasised, with particular focus on techniques aimed at reducing ruminative thoughts or negative emotions. Consequently, this dissertation not only fills a gap in the existing literature on the relationship between ADHD symptoms and PLEs, but also points to specific directions for further research into the mechanisms underlying these experiences and the possibilities for conceptualising better-tailored intervention strategies for individuals in the early stages of risk.

Keywords: *psychotic-like experiences; ADHD; negative emotions; ruminations; cortisol; trauma experiences; ESM.*