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Doświadczenia podobne do psychotycznych i mechanizmy leżące u ich podstaw – rola czynników poznawczych, emocjonalnych i fizjologicznych mierzonych za pomocą metody próbkowania doświadczeń (ESM) u osób z objawami ADHD

[Psychotic–like experiences and underlying mechanisms – the role of cognitive, emotional, and physiological factors measured via Experience Sampling Method (ESM) in individuals with ADHD symptoms]

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Spis treści

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.*

2. Streszczenie

Doświadczenia podobne do objawów psychotycznych (ang. *Psychotic-like Experiences*; PLEs) obejmują zarówno doświadczenia podobne do halucynacji (ang. *Hallucination-like Experiences*; HLEs), jak i doświadczenia podobne do urojeń (ang. *Delusion-like Experiences*; DLEs). Definiowane są one jako subkliniczne przeżycia, charakteryzujące się łagodniejszą intensywnością i stanowiące kategorię często przejściowych doświadczeń, które nie spełniają w pełni przyjętych kryteriów objawów psychotycznych. W aktualnej literaturze doświadczenia PLEs rozpatrywane są jako objawy leżące na kontinuum, obejmującego zakres od braku takich doświadczeń aż po pełnoobjawowe zaburzenia z grupy psychotycznych. Szacuje się, że od 5 do 10% osób dorosłych w populacji ogólnej relacjonuje występowanie PLEs w pewnym momencie życia (Drvaric & Bagby, 2021), a ich szczyt przypada na grupę młodych dorosłych (18 – 35 lat), w której rozpowszechnienie może sięgać nawet od 32 do 35% (Rep i in., 2023). Chociaż u większości osób doświadczenia te ustępują samoistnie wraz z upływem czasu (~80%), to u znacznej mniejszości (~7%) dochodzi jednak do ich intensyfikacji z czasem (Linscott & Van Os, 2013). Sugeruje to, że PLEs mogą stanowić czynnik ryzyka wystąpienia pełnoobjawowych zaburzeń psychotycznych w przyszłości. Dlatego poznanie czynników ryzyka i mechanizmów leżących u podstaw ich rozwoju nadal stanowi ważny przedmiot badań.

Zgodnie z socjo-poznawczo-rozwojowym modelem psychozy (Howes & Murray, 2014), schizofrenia ujmowana jest jako zaburzenie neurorozwojowe, a w jej etiologii istotną rolę odgrywa złożona interakcja czynników biologicznych, poznawczych i środowiskowych. Podejście neurorozwojowe pozwala postrzegać schizofrenię jako konsekwencję wzajemnych oddziaływań pomiędzy zdarzeniami z etapu wczesnego rozwoju a współwystępowaniem genetycznych predyspozycji związanych z innymi jednostkami chorobowymi, których objawy kliniczne występują już w okresie dzieciństwa. Ten wieloczynnikowy proces kształtuje podatność jednostki na rozwój zaburzeń jeszcze przed pojawieniem się objawów psychotycznych. Wyniki badań konsekwentnie wskazują, że niekorzystne doświadczenia z okresu wczesnego dzieciństwa również stanowią istotny czynnik ryzyka rozwoju psychozy w przyszłości. W modelu zaproponowanym przez Howesa i Murraya (2014) wskazano, że doświadczenia te mogą kształtować schematy poznawcze jednostki używane przez nią do interpretacji środowiska. To z kolei powodować może dysregulację układu dopaminergicznego i jego nadmierną aktywność, skutkując aktywacją mechanizmu polegającego na nadmiernym nadawaniu znaczeń (ang. *Aberrant Salience*). Te bodźce są następnie interpretowane przez jednostkę poprzez uprzednio już zniekształcone schematy poznawcze, prowadząc do paranoicznych interpretacji, halucynacji czy wzmożonego monitorowania potencjalnego zagrożenia w neutralnym otoczeniu. Proces ten tworzy błędne koło, w którym doświadczenia psychotyczne generują dalszy stres, nasilając dysregulację dopaminy i utrwalając powstałe doświadczenia. Z biologicznego punktu widzenia, doświadczany stres psychospołeczny uruchamia szereg mechanizmów, w tym aktywację osi podwzgórze-przysadka-nadnercza (ang. *Hypothalamic-Pituitary-Adrenal Axis;* HPA). Krótkotrwała aktywacja osi HPA umożliwia adaptacyjną odpowiedź organizmu na stres, natomiast jej

przewlekła lub nadmierna aktywacja może prowadzić do uwrażliwienia układu dopaminergicznego oraz zwiększonej reaktywności na bodźce stresowe. Należy jednak podkreślić, że stres psychospołeczny czy traumatyczne doświadczenia nie są ani konieczne ani wystarczające do wywołania PLEs, co podkreśla wieloczynnikowy charakter tego zjawiska.

Wśród czynników neurorozwojowych związanych z etiologią PLEs coraz częściej zwraca się uwagę na rolę zespołu nadpobudliwości psychoruchowej z deficytami uwagi (ang. *Attention Deficit Hyperactivity Disorder;* ADHD). U osób z objawami ADHD, obserwuje się dysfunkcję osi HPA, która związana jest m.in. z deficytami poznawczymi (np. trudność w koncentracji) i behawioralnymi (np. impulsywność). Wykazano także, że osoby z objawami ADHD częściej zgłaszają podwyższony poziom subiektywnie odczuwanego stresu w życiu codziennym. Podobną zależność obserwuje się u osób doświadczających PLEs, co również może wiązać się z dysregulacją osi HPA w tej grupie. W konsekwencji u tych osób wzrasta wrażliwość na stres, a powstałe zniekształcenia poznawcze ulegają utrwaleniu. To z kolei może prowadzić do dalszego nasilenia PLEs. Co więcej, wzmożona reaktywność na stres raportowana w obu grupach, obejmująca procesy emocjonalne (np. zwiększony ogólny poziom stresu lub doświadczanie większej liczby negatywnych emocji), poznawcze (np. skłonność do ruminacji, zaburzona regulacja emocji, błędne przypisywanie znaczeń nieistotnym bodźcom) oraz fizjologiczne (np. podwyższony poziom kortyzolu), wydaje się wzmacniać powiązania między objawami ADHD, stresem a PLEs. W szczególności, związki te są istotne w kontekście klinicznym, gdzie ADHD i doświadczenia psychotyczne często są raportowane jako zaburzenia współwystępujące.

Chociaż relacje pomiędzy czynnikami neurorozwojowymi, psychospołecznymi i fizjologicznymi stanowią coraz częstszy przedmiot badań empirycznych, to mechanizmy i procesy leżące u ich podstaw nadal nie zostały w pełni poznane. W szczególności brakuje badań kompleksowo ujmujących jednocześnie procesy afektywne, poznawcze oraz fizjologiczne w związku pomiędzy współwystępowaniem objawów ADHD i doświadczeń PLEs.

Na prezentowaną rozprawę składają się trzy artykuły wykorzystujące samoopisowe dane kwestionariuszowe oraz dane podłużne zebrane metodą intensywnego próbkowania doświadczeń (ang. *Experience Sampling Method; ESM*). Głównym celem pracy było zbadanie roli objawów ADHD w nasilaniu PLEs poprzez uwzględnienie w tej relacji wybranych czynników ryzyka, takich jak niekorzystne doświadczenia społeczne, czynniki poznawcze, emocjonalne oraz fizjologiczne. Badania obejmują analizy interakcji objawów PTSD (ang. *Post-Traumatic Stress Disorder*) i ADHD w nasileniu PLEs (**Badanie 1**); testowanie modelu szeregowej mediacji, ujmującym pośredniczącą rolę ruminacji i negatywnych emocji w związku między objawami ADHD i PLEs, oraz moderowanej mediacji szeregowej, w którym sprawdzano czy doświadczenia traumatyczne wzmacniają tę relację (**Badanie 2**); a także prezentują złożone modele analiz sieciowych testujące powiązaia pomiędzy poziomem kortyzolu, PLEs, negatywnymi emocjami, ruminacjami, antycypacją zagrożenia i nadmiernym nadawaniem znaczeń u osób z różnym nasileniem objawów ADHD (**Badanie 3**).

Pierwsze badanie w cyklu (*N = 3000*) miało na celu sprawdzenie interakcyjnego wpływu objawów PTSD i ADHD na poziom zgłaszanych doświadczeń PLEs w populacji nieklinicznej (18 – 35 lat). Dotychczas żadne z wcześniejszych badań nie badało interakcji pomiędzy objawami PTSD a ADHD w tym kontekście, a jedynie wskazywano, że mają one tendencję do współwystępowania. Wyniki naszego badania ukazały, że interakcja objawów ADHD i PTSD istotnie wpływa na poziom intensywności zgłaszanych PLEs. Najwyższy poziom doświadczeń PLEs odnotowano u osób, które uzyskały pozytywny wynik przesiewowy zarówno w kierunku ADHD, jak i PTSD. Osoby z pozytywnym wynikiem przesiewowym w kierunku tylko jednej zmiennej również zgłaszały wyższy poziom PLEs w porównaniu do osób z negatywnymi wynikami przesiewowymi dla obu zaburzeń. Wyniki te podkreślają więc znaczenie efektu interakcji pomiędzy objawami ADHD i PTSD w kontekście zgłaszanych doświadczeń PLEs.

Drugie badanie (*N = 188*) miało na celu przetestowanie modelu szeregowej mediacji, w którym ruminacje i negatywne emocje uwzględnione zostały w modelu jako mediatory w związku między objawami ADHD a PLEs. Drugim etapem tego badania było sprawdzenie, czy traumatyczne doświadczenia wzmacniają testowaną relację. Badanie drugie obejmowało uśrednione do wartości tygodniowych dane podłużne, zbierane podczas tygodniowej procedury ESM, w której monitorowano codzienne wahania emocjonalne i poznawcze osób badanych. Wyniki tego badania wskazały na obecność istotnej statystycznie mediacji szeregowej, potwierdzając tym samym postawioną przez nas hipotezę, że myśli ruminacyjne i negatywne emocje pośredniczą w relacji między objawami ADHD a PLEs. Wykazaliśmy, że objawy ADHD istotnie wiążą się ze zwiększoną ilością ruminacji, które z kolei mogą prowadzić do większej ilości doświadczanych negatywnych emocji, co w konsekwencji może prowadzić do większego nasilenia PLEs. W testowanym modelu szeregowej moderowanej mediacji nie potwierdziliśmy jednak postawionej przez nas hipotezy, że związek pomiędzy objawami ADHD a PLEs, poprzez ruminacje oraz negatywne emocje, będzie wzmocniony przez doświadczenie traumatycznych wydarzeń życiowych.

Trzecie badanie w prezentowanym cyklu (*N = 188*) bazowało na danych zebranych podczas tygodniowej procedury ESM w badaniu 2 i zaprezentowane zostało w formie dynamicznych, czasowych analiz sieciowych. W pierwszej kolejności analizy obejmowały dynamiczne powiązania pomiędzy PLEs, elementami afektywnymi (negatywne emocje), myślami ruminacyjnymi, elementami fizjologicznymi (poziom kortyzolu) oraz elementami poznawczymi (tj. przewidywanie zagrożeń i nieprawidłowe przypisywanie znaczenia neutralnym bodźcom). Modele sieci zostały oszacowane zarówno dla całej grupy badawczej, jak i osobno dla dwóch grup różniących się poziomem nasilenia objawów ADHD (wysokie ADHD, *N = 96*; niskie ADHD, *N = 92*). Drugim etapem tego badania było przeprowadzenie analiz porównawczych struktur sieciowych między grupami osób z różnym nasileniem objawów ADHD. Badanie to, łącząc ekologiczne dane i biomarker stresu z analizą sieciową, pozwoliło na uchwycenie dynamicznej natury tych złożonych związków. Wyniki pokazały, że w grupie osób z wysokim nasileniem objawów ADHD najsilniejszym predyktorem wywierającym największy wpływ na pozostałe zmienne były doświadczenia podobne do urojeń, stające się centralnym węzłem w sieci. Wynik ten sugeruje, że u osób z

nasilonymi objawami ADHD doświadczenia podobne do urojeń mogą istotnie wpływać na postrzeganie rzeczywistości, potencjalnie działając jako czynnik wyzwalający kolejne reakcje emocjonalne lub poznawcze. Ponadto w grupie z wyższym nasileniem objawów ADHD wyniki wskazały na istotne powiązanie poziomu kortyzolu z przewidywaniem zagrożenia, nieprawidłowym przypisywaniem znaczenia i spożyciem alkoholu w następnym punkcie czasowym. Natomiast w grupie osób z niższym nasileniem objawów ADHD centralnym węzłem w sieci, mającym najsilniejszy wpływ na pozostałe obszary uwzględnione w modelu, były negatywne emocje, a kortyzol wykazywał jedynie istotny związek z późniejszym spożyciem alkoholu. W końcu analizy porównań sieciowych pomiędzy grupami częściowo potwierdziły postawioną przez nas hipotezę, że poziom kortyzolu będzie silniej powiązany z PLEs w grupie z wyższym nasileniem objawów ADHD w porównaniu z grupą z niższym ich nasileniem. Wyniki pokazały, że w grupie z wyższym nasileniem objawów ADHD doświadczenia podobne do urojeń wykazały większą stabilność i trwałość w czasie. Zaobserwowaliśmy również ich silniejsze powiązanie z poszukiwaniem zagrożenia i spożyciem alkoholu, a także zidentyfikowaliśmy istotną pośrednią ścieżkę pomiędzy doświadczeniami podobnymi do urojeń a negatywnymi emocjami poprzez spożycie alkoholu. Wyniki te wskazują, że alkohol może służyć jako nieadaptacyjny styl radzenia sobie w odpowiedzi na przeżyte doświadczenia, a zaobserwowane silniejsze powiązania między poziomem kortyzolu a poszukiwaniem zagrożenia w tej grupie wskazują na związek między fizjologiczną reakcją na stres a późniejszą interpretacją sytuacji jako zagrażającej.

Podsumowując, prezentowany w rozprawie cykl trzech artykułów poprzez integrację procesów afektywnych, poznawczych i fizjologicznych przyczynia się do pogłębienia rozumienia mechanizmów leżących u podstaw PLEs w populacji nieklinicznej. Wykazaliśmy, że relacja między objawami ADHD a PLEs ma charakter złożony i wieloczynnikowy. Łącznie wyniki zaprezentowanych badań przeprowadzonych w ramach tej pracy konsekwentnie wskazały, że objawy ADHD stanowią istotny czynnik ryzyka nasilania PLEs. Ponadto wyniki mogą sugerować, że ADHD jest nie tylko współwystępującym zaburzeniem, lecz także czynnikiem powiązanym z dynamiką procesów emocjonalnych, poznawczych i fizjologicznych, który może stanowić jeden z elementów wyjaśniających indywidualne różnice w dynamice doświadczeń PLEs w populacji młodych dorosłych.

Uzyskane wyniki mają również istotne implikacje praktyczne. Przede wszystkim mogą stanowić punkt wyjścia do opracowania lepiej dopasowanych strategii wczesnych interwencji osób z objawami ADHD, które znajdują się w grupie podwyższonego ryzyka PLEs. Co więcej, zwracają uwagę na potrzebę uwzględniania w pracy z osobami z ADHD adaptacyjnych technik regulacji emocji, a w szczególności związanych z redukcją myśli ruminacyjnych czy negatywnych emocji. W konsekwencji rozprawa ta nie tylko uzupełnia lukę w dotychczasowej literaturze na temat związku pomiędzy objawami ADHD a PLEs, ale również wskazuje konkretne kierunki dalszych badań nad mechanizmami podtrzymującymi te doświadczenia oraz możliwościami konceptualizacji lepiej dopasowanych strategii interwencyjnych osób znajdujących się we wczesnych stadiach ryzyka.

Słowa kluczowe: *objawy podobne do psychotycznych; ADHD; negatywne emocje; ruminacje; kortyzol; doświadczenia traumatyczne; ESM.*

3. Wprowadzenie

Doświadczenia takie jak na przykład wrażenie słyszenia rzeczy, których nie słyszały inne osoby, chwilowe przekonanie o byciu obserwowanym/ą sprawiające poczucie niepokoju, lub przelotne uczucie bycia niepewnym/ą co do realności minionych zdarzeń nie zawsze są zarezerwowane wyłącznie dla osób z rozpoznaniem zaburzeń psychotycznych, a są nawet dość powszechnie występującymi przeżyciami w populacji ogólnej zdrowych osób dorosłych (Healy & Cannon, 2020). Określane są one jako doświadczenia podobne do objawów psychotycznych (ang. *Psychotic-like Experiences*; PLEs) i obejmują doświadczenia podobne do halucynacji i urojeń. Szacuje się, że PLEs mogą występować u 5 – 10% osób dorosłych w pewnym momencie życia (Drvaric & Bagby, 2021). Inne badania wskazują, że około ~26% osób deklaruje doświadczenie co najmniej jednego typu PLEs w ciągu życia (Bourgin i in., 2020). Ze względu na ich wysokie rozpowszechnienie w populacji ogólnej stanowią one obecnie coraz częstszy obszar badań, a ich eksploracja pozwala nie tylko na lepsze zrozumienie mechanizmów leżących u ich podstaw, ale także na identyfikację wczesnych czynników ryzyka.

3.1. Doświadczenia podobne do psychotycznych – kontinuum psychozy

W odróżnieniu od pełnoobjawowej psychozy, doświadczenia PLEs u znacznej części osób nieposzukujących pomocy mają charakter niekliniczny – są zwykle łagodne, krótkotrwałe i przejściowe (Verdoux & Van Os, 2002). Doświadczenia PLEs mogą mieć także charakter subkliniczny, w którym są bardziej intensywne i sprawiające istotne uczucie niepokoju (Van Os i in., 2009), ale nadal mogą nie spełniać w pełni przyjętych kryteriów diagnostycznych zarezerwowanych dla grupy objawów psychotycznych (Staines i in., 2022). W psychopatologii przyjmuje się, że doświadczenia te rozmieszczone są na kontinuum psychozy (DeRosse & Karlsgodt, 2015; Kaymaz i in., 2012). Na jednej ze skrajnych stron kontinuum znajduje się całkowity brak takich doświadczeń. Następnie występują łagodne i sporadyczne doświadczenia, takie jak PLEs. W niektórych przypadkach mogą one nasilać się, przechodząc w subkliniczne objawy psychotyczne, zwiększające ryzyko rozwoju stanu wysokiego ryzyka psychozy (ang. *Ultra–High–Risk*; UHR), stanowiącego kolejny etap kontinuum. Na jego przeciwległym krańcu mieszczą się utrwalone objawy psychotyczne, należące do spektrum zaburzeń psychotycznych, sprawiające istotny dyskomfort psychiczny i wyraźnie utrudniające funkcjonowanie jednostki. Doświadczenia PLEs odróżnia od klinicznych objawów psychotycznych nie tylko treść, ale także nasilenie, częstotliwość występowania, wiara w ich realność, wpływ na funkcjonowanie oraz subiektywne poczucie cierpienia wynikające z ich doświadczania (American Psychiatric Association, 2022). Analizując kontinuum psychozy i przejścia między jego kolejnymi etapami, należy uwzględniać złożoną i wielopoziomową interakcję między czynnikami indywidualnymi i środowiskowymi, obejmującą mechanizmy poznawcze i emocjonalne, ponieważ wpływają one nie tylko na ryzyko rozwoju tych doświadczeń, ale także biorą udział w ich utrwalaniu. Dlatego też postrzeganie doświadczeń PLEs jako elementu kontinuum psychozy umożliwia wielowymiarową analizę czynników leżących u ich podstaw.

3.2. Socjo–poznawczo–rozwojowy model psychozy

Zintegrowany model schizofrenii Howesa i Murraya (2014) stanowił próbę integracji powstałych we wcześniejszych latach koncepcji na temat etiologii tej choroby obejmując m.in. modele biologiczne (np. Howes & Kapur, 2009), neurorozwojowe (np. Murray & Lewis, 1987) i poznawcze (np. Garety i in., 2001), w jeden spójny schemat wyjaśniający kształtowanie się ryzyka psychozy z uwzględnieniem ich interakcji ze środowiskiem. Socjo–poznawczo–rozwojowy model psychozy (Howes & Murray, 2014) rozwija klasyczny model neurorozwojowy, zakładając, że predyspozycje neurorozwojowe wtórne do wariantów genów, wchodząc w interakcję ze wczesnymi niekorzystnymi doświadczeniami społecznymi i środowiskowymi, prowadzą do dysregulacji układu dopaminergicznego (w szczególności nadmiernej syntezy dopaminy w strukturach podkorowych). Wynikające z niekorzystnych czynników środowiskowych, zniekształcone schematy poznawcze, wykorzystywane przez jednostkę do interpretacji doświadczeń, mogą zostać wzmocnione przez nadmierną sytezę dopaminy. Skutkować może to nadmiernym nadawaniem znaczeń (ang. *Aberrant Salience*), które u części osób pośrednio prowadzić może do paranoicznych interpretacji lub/i halucynacji. Następnie wzrost poczucia stresu czy zagrożenia wynikający z doświadczania objawów psychotycznych, w połączeniu z używanymi przez jednostkę utrwalonymi zniekształconymi schematami poznawczymi, zostaje wzmocniony przez nadmierną syntezę dopaminy, tworząc tym samym błędne koło, które podtrzymuje i utrwala powstałe objawy psychotyczne. Jedną z kluczowych zalet tego modelu jest jego dynamiczny i złożony charakter, wyjaśniający nawracające epizody objawów psychotycznych, w odniesieniu do mechanizmów związanych ze wzmacniającą rolą nadmiernej syntezy dopaminy. Jednocześnie jego ograniczeniem pozostaje stosunkowo słabo rozwinięty opis mechanizmów poznawczych, odpowiedzialnych za utrwalanie błędnych przekonań na temat świata i ludzi.

Socjo–poznawczo–rozwojowy model psychozy (Howes & Murray, 2014) podkreśla istotną rolę zniekształconych schematów poznawczych jako jednego z czynników ryzyka kształtowania się objawów psychotycznych. Faktycznie, zaburzenia funkcji poznawczych występują u ponad 80% pacjentów/ek ze schizofrenią (McEvoy, 2007) i stanowią jedną z głównych przeszkód w powrocie do zdrowia (Fatouros-Bergman i in., 2014). Badania wskazują, że zaburzenia funkcji poznawczych oraz zniekształcenia poznawcze są wykrywane jeszcze przed wystąpieniem w pełni rozwinętej psychozy (Harvey i in., 2022; Howes & Murray, 2014). Zatem mogą stanowić one jedene z istotnych czynników tej choroby (Rapoport i in., 2012).

Zaburzeniem o charakterze neurorozwojowym, które również charakteryzuje się deficytami funkcji poznawczych, jest zespół nadpobudliwości psychoruchowej z deficytem uwagi (ang. Attention Deficit Hyperactivity Disorder; ADHD). Chociaż ADHD tradycyjnie kojarzone jest z problemami z koncentracją i nadpobudliwością, coraz więcej dowodów wskazuje również na jego znaczący wpływ na funkcje poznawcze osób w wieku dorosłym. Badania wskazują, że osoby z objawami ADHD wykazują deficyty funkcji wykonawczych, pamięci roboczej i elastyczności poznawczej (Eberhard i in., 2024), które znacząco utrudniają ich codzienne funkcjonowanie i obniżają ogólną jakość życia (Isaac i in., 2024). Literatura

wskazuje na powszechne współwystępowanie ADHD z innymi zaburzeniami psychicznymi, w tym również z zaburzeniami psychotycznymi (np. Syed i in., 2010; Toba-Oluboka & Dempster, 2024). Jednak jeszcze do niedawna objawy ADHD nie były bezpośrednio badane w kontekście doświadczeń psychotycznych. Dlatego też nadal brakuje empirycznych dowodów na ich związek z PLEs. Co więcej, chociaż mediacyjna rola funkcji poznawczych w relacji między różnymi czynnikami ryzyka a PLEs była już analizowana (np. Gawęda i in., 2018; Mętel i in., 2020; Pionke-Ubych i in., 2022), wciąż brakuje badań uwzględniających rolę zniekształceń poznawczych w kontekście współwystępowania objawów ADHD u osób doświadczających PLEs. Mimo że socjo–poznawczo–rozwojowy model psychozy (Howes & Murray, 2014) nie uwzględnia roli ADHD jako bezpośredniego czynnika ryzyka, zwraca jednak uwagę na istotny komponent neurorozwojowy oraz rolę zniekształceń poznawczych w kształtowaniu ryzyka psychoz. Dlatego głównym celem niniejszej pracy było zbadanie roli objawów ADHD w nasilaniu doświadczeń PLEs, ze względu na liczne wspólne czynniki ryzyka i mechanizmy leżące u ich podstaw. Uwzględniając w modelu objawy ADHD jako potencjalny czynnik ryzyka lub komponent nasilający opisane procesy, dążyliśmy do identyfikacji mechanizmów i czynników ryzyka leżących u podstaw doświadczeń PLEs w populacji nieklinicznej osób dorosłych.

3.3. Rola osi HPA – biologiczne mechanizmy stresu

Stres społeczny jest powszechnie występującym zjawiskiem, jednak fizjologiczna odpowiedź organizmu na stres i sposób radzenia sobie z nim mogą istotnie różnić się w zależności od indywidualnych predyspozycji jednostki, jej stanu psychicznego i zdolności do adaptacji. Oś podwzgórze–przysadka–nadnercza (ang. *Hypothalamic-Pituitary-Adrenal Axis*; HPA) pełni jedną z kluczowych ról w biologicznej odpowiedzi organizmu na doświadczany stres poprzez wydzielanie hormonu stresu – kortyzolu (Herman i in., 2016). Aktywacja osi HPA na bodźce stresowe ma charakter adaptacyjny. Podwyższony poziom kortyzolu w odpowiedzi na bodziec stresowy ma najczęściej charakter przejściowy. Jednak przewlekły stres może prowadzić do przedłużonej aktywacji osi HPA, skutkując długotrwałym podwyższeniem poziomu kortyzolu oraz dysregulacją osi HPA. W konsekwencji może prowadzić to do rozwoju zaburzeń somatycznych lub/i psychicznych (P & Vellapandian, 2024). Aktywacja osi HPA zależy przede wszystkim od czasu trwania i siły stresora. Prawidłowa reakcja osi HPA na ostry stres obejmuje uruchomienie jej w odpowiedzi na bodziec stresowy, a mechanizmy sprzężenia zwrotnego powinny skutecznie zakończyć jej reakcję po jego ustąpieniu (Herman i in., 2016). Jednak indywidualne predyspozycje jednostki do nieadaptacyjnych reakcji na stres zależą od wielu czynników, w tym wieku, płci, uwarunkowań genetycznych, doświadczeń wczesnodziecięcych oraz aktualnego stanu psychicznego (np. Collip i in., 2011; Gaffey i in., 2016; Goel i in., 2014; Sheerin i in., 2020).

Zaburzenia funkcjonowania osi HPA są szeroko opisywane w kontekście poważnych zaburzeń psychicznych, takich jak zaburzenia depresyjne (Keller i in., 2017), zaburzenia lękowe (Ariño-Braña i in., 2025), zaburzenia afektywne dwubiegunowe (Belvederi Murri i in., 2016) czy zaburzenia stresowe

pourazowe (Lawrence & Scofield, 2024). Badania wskazują także, że dysregulacja osi HPA uznawana jest za istotny czynnik ryzyka w rozwoju zaburzeń psychotycznych (Ciufolini i in., 2014). Dlatego też zaczęto zwracać uwagę na potencjalne znaczenie zaburzeń dysregulacji osi HPA także w odniesieniu do doświadczeń PLEs. Chociaż w literaturze nadal istnieje stosunkowo niewiele badań na temat biochemicznych aspektów tych doświadczeń, dysregulacja osi HPA stanowi jeden z ważnych mechanizmów analizowanych w tym kontekście. Badania konsekwentnie wskazują, że osoby doświadczające PLEs wykazują podwyższony poziom kortyzolu oraz większą reaktywność na stres w życiu codziennym (Collip i in., 2011; Myin-Germeys & Van Os, 2007). Wyniki sugerują nie tylko pozytywny związek pomiędzy podwyższonym poziomem kortyzolu a PLEs, ale także podkreślają, że dysregulacja osi HPA może być obecna już na subklinicznym etapie rozwoju doświadczeń psychotycznych i może stanowić jeden z biologicznych mechanizmów zwiększających ryzyko psychozy w przyszłości (Logoń i in., 2024).

W przypadku osób z objawami ADHD również obserwuje się podwyższony ogólny poziom odczuwanego stresu (Combs i in., 2015) oraz zaburzenia funkcjonowania osi HPA (Fulun i in., 2025; Randazzo i in., 2008). Dysregulacja osi HPA u osób z ADHD wiąże się m.in. z deficytami uwagi, wyższą impulsywnością oraz nadpobudliwością psychoruchową (Ma i in., 2011). Jednak fizjologiczna odpowiedź na stres różni się od tej obserwowanej u osób doświadczających PLEs. Wyniki badań dotyczących odpowiedzi kortyzolowej u osób z objawami ADHD są jednak niespójne. Niektóre wskazują na brak istotnych różnic w odpowiedzi kortyzolu na stres (np. Corominas-Roso i in., 2015), a inne pokazują opóźnioną lub zmniejszoną reakcję i spowolnione uwalnianie kortyzolu (np. Chang i in., 2021). Ponadto najnowsze badania sugerują różnice w reaktywności osi HPA i odpowiedzi kortyzolowej na stres w zależności od podtypu ADHD (np. Fulun i in., 2025). Dlatego też, chociaż raportowany podwyższony poziom subiektywnie odczuwanego stresu jest wspólny dla obu grup, fizjologiczne reakcje na bodźce stresowe oraz mechanizmy funkcjonowania osi HPA wykazują odmienne wzorce. U osób z PLEs obserwuje się zazwyczaj zwiększoną reaktywność na stres i wyższe poziomy kortyzolu (Collip i in., 2011), natomiast u osób z objawami ADHD reakcje wydzielania kortyzolu mogą być opóźnione lub spowolnione (Chang i in., 2021). Co ważne, nadal brakuje badań uwzględniających codzienne pomiary poziomu kortyzolu w warunkach ekologicznie trafnych. Utrudnia to więc jednoznaczne określenie reakcji kortyzolowej w tych grupach i tym samym wskazuje na potrzebę dalszych badań.

3.4. Rola czynników neurorozwojowych oraz stresu psychospołecznego

Zarówno schizofrenia, jak i ADHD mają charakter zaburzeń o charakterze neurorozwojowym, co może tłumaczyć częściowe nakładanie się ich czynników ryzyka oraz ich relatywnie częste współwystępowanie (Holstein i in., 2013). Faktycznie, objawy ADHD są związane z doświadczeniami PLEs (Bourgin i in., 2020), a badania wskazują nawet na pięciokrotnie wyższe ryzyko rozwoju zaburzeń psychotycznych u dzieci z diagnozą ADHD w dorosłości (Hamshere i in., 2013; Nourredine i in., 2021). Oprócz czynników neurorozwojowych istotną rolę w kształtowaniu doświadczeń PLEs odgrywają również

czynniki środowiskowe, w tym wczesne doświadczenia traumatyczne, które stanowią dobrze udokumentowane czynniki ryzyka PLEs (np. Misiak i in., 2025). Badania wskazują na dwu- a nawet trzykrotnie większe ryzyko wystąpienia objawów psychotycznych u osób, które doświadczyły co najmniej jednego traumatycznego zdarzenia w ciągu życia, a ryzyko to wzrasta wraz z liczbą traumatycznych doświadczeń, co wskazuje na addytywny charakter tego związku (np. Kilian i in., 2021; McGrath i in., 2017). Wyniki badań wskazują na dwukierunkowy związek między traumą a objawami ADHD (np. Spencer i in., 2016) oraz ich współwystępowanie (Ford & Connor, 2009). Objawy ADHD często są związane ze zwiększonym ryzykiem narażenia na traumatyczne zdarzenia, szczególnie we wczesnym okresie dorastania (Schilpzand i in., 2018), a charakterystyczne objawy na doświadczoną traumę, takie jak trudności z koncentracją czy regulacją emocji, drażliwość lub wysokie pobudzenie, mogą nakładać się na objawy ADHD lub je zaostrzać (Daud & Rydelius, 2009).

Co istotne, zarówno trudności w regulacji emocji, większa impulsywność i deficyty uwagi charakterystyczne dla objawów ADHD (Williams i in., 2023), jak i objawy specyficzne dla doświadczonej traumy, takie jak dysregulacja emocjonalna, stałe poczucie zagrożenia, wzmożona czujność, drażliwość, wycofanie emocjonalne czy problemy z koncentracją (American Psychiatric Association, 2022), mogą przyczyniać się do zwiększonej skłonności interpretowania neutralnych sytuacji jako zagrażających oraz sprzyjać powstawaniu zniekształceń poznawczych lub/i percepcyjnych (Lyndon & Corlett, 2020; Marwaha i in., 2015). Ponadto objawy obu tych zaburzeń mogą wiązać się ze zwiększoną skłonnością do doświadczania negatywnych emocji (np. Pugach i in., 2023; Shaw i in., 2014) oraz stosowania nieadaptacyjnych strategii radzenia sobie, takich jak ruminowanie (np. Beheshti i in., 2020; Michael i in., 2007) czy używanie substancji psychoaktywnych (np. Bartoli i in., 2021; Luciano i in., 2022). Dotychczasowe badania wskazują na istotne związki między objawami ADHD czy doświadczeniami traumatycznymi, a zwiększonym ryzykiem rozwoju zaburzeń psychotycznych w przyszłości (np. Bourgin i in., 2020). Wyniki sugerują, że mechanizmy takie jak na przykład zniekształcone schematy poznawcze, nieadaptacyjne strategie regulacji emocji czy rozpamiętywanie mogą tłumaczyć część tych związków, co wskazywać może na złożone mechanizmy pośredniczące w tej relacji (np. Gibson i in., 2019; Hardy, 2017). Jednak brakuje badań uwzględniających współwystępowanie objawów ADHD i traumy w przewidywaniu doświadczeń psychotycznych. Dlatego też nadal nie jest jasne, czy ich interakcja może nasilać PLEs.

3.5. Nieadaptacyjne style radzenia sobie – ruminacje i negatywne emocje

Jednym z istotnych czynników mogących mieć znaczenie w rozwoju i podtrzymywaniu doświadczeń PLEs jest dysregulacja emocjonalna (np. Chapman i in., 2020; Leslie i in., 2025). Jest ona istotną cechą szeregu różnych zaburzeń psychicznych (Aslan i in., 2024), w tym ADHD (Shaw i in., 2014), PTSD (Conti i in., 2023) oraz schizofrenii (Henry i in., 2007). Definiowana jest ona jako niezdolność do skutecznego zarządzania i modulowania emocjami (Gross & Ford, 2024; Gross & Muñoz, 1995). Obejmuje trudności w regulacji reakcji emocjonalnych, zwiększoną reaktywność emocjonalną lub upośledzoną ekspresję

emocjonalną (Aldao i in., 2010). Badania wskazują, że nieadaptacyjne strategie regulacji emocji, takie jak ruminowanie, obwinianie się czy unikanie, są uznawane za istotne wskaźniki dysregulacji emocjonalnej i często biorą udział w rozwoju i utrzymywaniu objawów zaburzeń psychicznych (np. Aslan & Baldwin, 2021; Cisler & Olatunji, 2012). W rezultacie dysregulacja emocjonalna, w związku z podwyższonym ogólnym poziomem negatywnych emocji, (Renna, 2021) może prowadzić do niekorzystnych konsekwencji, sprzyjając tym samym zwiększeniu ryzyka doświadczeń PLEs.

Jedną z nieadaptacyjnych strategii regulowania emocji, pojawiających się w dysregulacji emocjonalnej i mogących zwiększać ryzyko nasilania PLEs, jest ruminowanie. Ruminacje definiowane są jako uporczywe nawracające myśli o sobie, swoich uczuciach, negatywnych stanach emocjonalnych czy przeszłych doświadczeniach (Treynor i in., 2003; Watkins & Roberts, 2020). Badania wskazują, że ruminacje mogą bezpośrednio kształtować indywidualne reakcje jednostki na stresujące wydarzenia poprzez przedłużanie negatywnych stanów emocjonalnych (Padilla Paredes & Calvete Zumalde, 2015; Watkins & Roberts, 2020). Jednak ekspozycja na stresujące wydarzenia również może prowadzić do nasilania ruminacji, co może wskazywać na dwukierunkowy charakter tej relacji (Nolen-Hoeksema i in., 2008). Co więcej, ruminacje powiązane zostały z wyższymi średnimi poziomami negatywnego afektu, a negatywny afekt powiązany został z ruminacyjnym skupianiem się na sobie (Kirkegaard Thomsen, 2006). Myśli ruminacyjne są powiązane z różnymi zaburzeniami psychicznymi, dlatego też rozpatruje się je jako istotny czynnik zwiększający ryzyko rozwoju innych zaburzeń psychicznych (Wong i in., 2023).

Literatura wskazuje na istotny związek między ruminacjami a psychozą (np. Hartley i in., 2014; Thomas i in., 2014) oraz na ich pozytywny związek z występowaniem urojeń u pacjentów/ek z psychozą (Freeman i in., 2015; Hartley i in., 2017). Myśli ruminacyjne są często raportowane także przez osoby z objawami ADHD (Kandeğer i in., 2024) oraz z doświadczeniem traumy (Moulds i in., 2020). Jednak pomimo tych dowodów rola myśli ruminacyjnych w kontekście doświadczeń PLEs u osób z objawami ADHD, które jednocześnie doświadczyły traumy, pozostaje niejasna. Niektóre badania sugerują, że ruminacje mogą pełnić istotną rolę w relacji pomiędzy ekspozycją na niekorzystne doświadczenia a rozwojem PLEs poprzez zwiększoną ilość myśli ruminacyjnych i ich negatywny wpływ na dobrostan psychiczny jednostek (Z. A. Shaw i in., 2024). Z drugiej strony, wyniki wskazują, że ruminacje mogą bezpośrednio wywoływać przejściowe doświadczenia psychotyczne u osób nieklinicznych (Hartley i in., 2017). Inne badania sugerują, że ruminacje mogą sprzyjać utrzymywaniu się urojeniowych przekonań poprzez pozbawienie jednostki elastyczności poznawczej oraz nadmiernego skupienia się na własnych przeżyciach (McKie i in., 2017). Wyniki te jednak pozostają niejednoznaczne, co sugerować może, że zależność między ruminacjami a PLEs może mieć charakter dwukierunkowy. Osoby z PLEs mogą mieć tendencję do częstszego ruminowania, jak również ruminacje same w sobie mogą zwiększać ryzyko doświadczania PLEs. Współzależności w kontekście objawów ADHD również pozostają niejasne. Wychodząc jednak z założenia, że u osób z objawami ADHD ruminacje mogą sprzyjać nadmiernej

reinterpretacji codziennych doświadczeń i utrwalać negatywny afekt, postawiliśmy hipotezę mówiącą, że ruminacje i negatywne emocje mogą nasilać doświadczania PLEs.

3.6. Luki w literaturze

Chociaż zarówno objawy ADHD, jak i doświadczenia traumatyczne zostały zidentyfikowane jako czynniki istotnie zwiększające ryzyko rozwoju zaburzeń psychotycznych w przyszłości (np. Kilian i in., 2021; Nourredine i in., 2021), to nadal brakuje badań testujących efekty ich interakcji w nasilaniu doświadczeń PLEs w populacji nieklinicznej. Wychodząc z założeń socjo–poznawczo–rozwojowego modelu psychozy (Howes i Murray, 2014) oraz opierając się na wynikach badań wskazujących na dwukierunkowy związek pomiędzy traumą a objawami ADHD (Spencer i in., 2016) oraz na ich pozytywny związek z objawami psychotycznymi, pierwszym celem niniejszej pracy było sprawdzenie efektu interakcji objawów ADHD i PTSD w nasilaniu doświadczeń PLEs.

Kolejnym ograniczeniem dotychczasowej literatury jest niewielka liczba badań analizujących pośredniczącą rolę wybranych czynników poznawczych i emocjonalnych w związku między objawami ADHD a doświadczeniami PLEs. Badania wskazują, że osoby z objawami ADHD wykazują zwiększone ryzyko doświadczania PLEs (Levy i in., 2015). Jednak mechanizmy leżące u podłoża tego związku nadal pozostają słabo poznane. Jednym z potencjalnych mechanizmów wyjaśniających tę relację może być dysregulacja emocjonalna, która powiązana została z nieadaptacyjnymi stylami radzenia sobie, takimi jak ruminacje, oraz podwyższonym poziomem negatywnych emocji. Ponieważ wyniki badań wskazują na istotny związek między ruminacjami a psychozą (Hartley i in., 2014) oraz istotne powiązania między negatywnym afektem a nasileniem doświadczeń PLEs (Gurnani & Georgiades, 2025) a także jego częste współwystępowanie u osób z objawami ADHD i/lub PTSD (Conti i in., 2023; Shaw i in., 2014) drugim celem niniejszego cyklu badań było przetestowanie pośredniczącej roli ruminacji i negatywnych emocji w związku między objawami ADHD a PLEs. Ponadto ze względu na istotny związek między ADHD a traumą (Spencer i in., 2016) oraz istotną rolę traumy w psychozie (Giannopoulou i in., 2023), kolejnym etapem było sprawdzenie moderującej roli traumy w badanej relacji.

Dodatkowo stosunkowo duża część dotychczasowych badań nad PLEs opiera się na retrospektywnych danych, które nie pozwalają w pełni na uchwycenie ich dynamicznego charakteru w codziennym funkcjonowaniu jednostek. W ostatnich latach coraz większą uwagę w badaniach poświęca się wykorzystywaniu metody intensywnego próbkowania doświadczeń (ang. *Experience Sampling Method*; ESM), która umożliwia śledzenie fluktuacji objawów w czasie rzeczywistym w naturalnym środowisku (Myin-Germeys i in., 2009). Pomimo rosnącego zainteresowania tą metodologią, badania wykorzystujące ESM w kontekście doświadczeń PLEs nadal pozostają stosunkowo nieliczne. Co więcej, dotychczasowe badania ESM rzadko integrowały jednocześnie procesy emocjonalne, poznawcze oraz fizjologiczne w ramach jednego modelu wyjaśniającego mechanizmy nasilania doświadczeń PLEs. W szczególności żadne z dotychczasowych badań opierających się na danych ESM nie analizowało dynamiki relacji pomiędzy

PLEs, negatywnymi emocjami, ruminacjami, procesami poznawczymi związanymi z antycypacją zagrożenia oraz nadmiernym nadawaniem znaczeń, a także fizjologicznymi wskaźnikami stresu, takimi jak poziom kortyzolu. W związku z tym, w celu lepszego zrozumienia dynamiki powiązań między tymi czynnikami w czasie rzeczywistym, trzecim celem w prezentowanym cyklu badań była analiza dynamicznych związków czasowych pomiędzy PLEs a czynnikami emocjonalnymi, poznawczymi i fizjologicznymi w codziennym funkcjonowaniu osób z różnym nasileniem objawów ADHD.

3.7. Pytania badawcze oraz hipotezy

Przedstawiona praca doktorska obejmuje trzy artykuły mające na celu systematyczne zbadanie czynników i mechanizmów leżących u podłoża doświadczeń PLEs w nieklinicznej populacji młodych osób dorosłych. Szczególną uwagę poświęcono czynnikom neurorozwojowym, takim jak objawy ADHD, oraz roli stresu psychospołecznego doświadczanego we wczesnym okresie rozwoju. W pracy analizowano czynniki takie jak myśli ruminacyjne, negatywne emocje, nadmierne poszukiwanie zagrożenia, nadmierne nadawanie znaczeń oraz poziom kortyzolu. Głównym celem projektu było zbadanie roli objawów ADHD w nasilaniu doświadczeń PLEs poprzez uwzględnienie wybranych czynników społecznych, poznawczych, emocjonalnych i fizjologicznych. Cel ten realizowano w badaniach, których główne pytania badawcze oraz hipotezy stanowiące podstawę niniejszego cyklu przedstawiono poniżej.

Badanie 1: Poprzez wykorzystanie samoopisowych kwestionariuszy sprawdziliśmy, czy efekt interakcji pomiędzy traumatycznymi doświadczeniami a objawami ADHD będzie przewidywać większe nasilenie doświadczeń PLEs. Wyniki badań konsekwentnie wskazują, że ADHD i zespół stresu pourazowego (PTSD) mają tendencję do współwystępowania i mogą być ze sobą przyczynowo powiązane (np. Spencer i in., 2016). Wykazano także, że predyspozycje genetyczne osób z objawami ADHD zwiększają wystąpienie PTSD w przyszłości (Wendt i in., 2023). Chociaż wiadomo, że każde z tych zaburzeń może być z osobna przyczynowo związane ze zwiększonym ryzykiem rozwoju zaburzeń psychotycznych w przyszłości (np. Bourgin i in., 2020; Gibson i in., 2019), to nadal nie było jasne, czy interakcja pomiędzy nimi może nasilać doświadczenia PLEs. W związku z tym postawiliśmy następujące **pytanie badawcze 1**: Czy interakcja objawów PTSD i ADHD jest związana z nasileniem doświadczania PLEs wśród młodych osób dorosłych z populacji nieklinicznej? Postawiliśmy hipotezę (**H1a**), że efekt interakcji objawów ADHD i PTSD będzie nasilać doświadczenia PLEs oraz że ich największe nasilenie będzie obserwowane w przypadku pozytywnej przesiewowej diagnozy w kierunku obu zaburzeń. Biorąc pod uwagę wyniki badań, które wskazują, że zdiagnozowane ADHD w dzieciństwie może wiązać się z podwyższonym ryzykiem wystąpienia zaburzeń psychotycznych w przyszłości (Nourredine i in., 2021) oraz badań wskazujących większą liczbę doświadczeń PLEs w grupie osób z objawami PTSD (Rodolfo Rossi i in., 2023), postawiliśmy drugą hipotezę (**H1b**), że wyższe nasilenie doświadczeń PLEs będzie obserwowane w grupie osób z pozytywnymi wynikami przesiewowymi w kierunku tylko jednego

zaburzenia, w porównaniu z osobami z bez żadnych pozytywnych wyników przesiewowych w kierunku ADHD i PTSD.

Badanie 2: Przekrojowość wyników badania 1, które ukazywało jedynie proste efekty interakcji dwóch czynników ryzyka związanych z doświadczeniami PLEs, uniemożliwiła nam wnioskowanie o związkach przyczynowo-skutkowych wpływu ADHD i PTSD na występowanie doświadczeń PLEs. Co więcej, pierwsze badanie bazowało wyłącznie na samoopisowych kwestionariuszach, które obarczone były błędem przypominania retrospektywnego. Dlatego nasze drugie badanie oparliśmy po części na danych uzyskanych z metody intensywnego próbkowania doświadczeń (ang. *Experience Sampling Method; ESM).* Badania nad ADHD i PTSD wskazują, że oba te zaburzenia charakteryzują się występowaniem dysregulacji emocjonalnej (np. Beheshti i in., 2020; Conti i in., 2023; Skirrow & Asherson, 2013) oraz podwyższonym poziomem doświadczanych negatywnych emocji (np. Pugach i in., 2023; Soler-Gutiérrez i in., 2025). Podobne zależności można zaobserwować również w badaniach prowadzonych w kontekście osób z PLEs, które wskazują, że negatywne emocje odgrywają rolę w pojawianiu się treści i objawów psychotycznych (Lam i in., 2022). Jednym z mechanizmów nieadaptacyjnego radzenia sobie z negatywnymi emocjami jest angażowanie się w ruminowanie, które uważane jest za mechanizm leżący u podstaw m.in. depresji, traumy, ADHD czy objawów ze spektrum schizofrenii (Kandeğer i in., 2024; Sellers i in., 2018). Jednak pomimo wielu dowodów na istnienie powiązań między objawami ADHD, ruminacjami, negatywnymi emocjami, traumą oraz doświadczeniami PLEs brakowało zintegrowanego modelu, który sprawdziłby konkretne zależności tej relacji. Postawiliśmy następujące pytania badawcze w celu ustalenia, czy związek pomiędzy ADHD a PLEs jest mediowany przez ruminacje i negatywne emocje oraz czy jest on wzmacniany przez traumatyczne doświadczenia. **Pytanie badawcze 2**: (**a**) Czy objawy ADHD są związane z nasileniem PLEs? (**b**) Czy związek pomiędzy ADHD a PLEs jest mediowany przez ruminacje i negatywne emocje? (**c**) Czy związek ADHD i PLEs, poprzez ruminacje i negatywne emocje, jest moderowany przez doświadczone traumatyczne wydarzenia? Postawiliśmy następujące hipotezy: (**H2a**) Objawy ADHD są związane z nasileniem doświadczeń PLEs; (**H2b**) Ruminacje i negatywne emocje pośredniczą w związku między objawami ADHD a PLEs; oraz (**H2c**) Związek między objawami ADHD a PLEs (**H2a + H2b**) jest moderowany przez traumatyczne doświadczenia życiowe, tj. pozytywny związek między objawami ADHD a PLEs pośredniczony przez ruminacje i negatywne emocje stanie się silniejszy u osób, które doświadczyły wyższego poziomu traumatycznych doświadczeń.

Badanie 3: Badanie pierwsze pozwoliło nam na zbadanie prostych efektów interakcji dwóch czynników ryzyka (tj. ADHD i PTSD) leżących u podstaw doświadczeń PLEs. Drugie badanie umożliwiło identyfikację mechanizmów pośredniczących w relacji pomiędzy objawami ADHD a PLEs, takich jak ruminacje i negatywne emocje, oraz ocenę moderującej roli traumy w tej relacji. Jednak pomimo częściowego wykorzystania danych ekologicznych w badaniu drugim zastosowane przez nas metody analiz nie pozwoliły w pełni uchwycić dynamicznych, czasowych zależności pomiędzy zmiennymi. Chociaż w badaniu 2 uwzględniliśmy procesy emocjonalne i poznawcze, to nie obejmowały one komponentu

fizjologicznego związanego ze stresem. Wydaje się on jednak być istotny w kontekście badań nad mechanizmami doświadczeń PLEs u osób z objawami ADHD, ze względu na raportowane zaburzenia regulacji osi HPA w tych grupach (np. Pauli-Pott i in., 2024; Pruessner i in., 2017). Dlatego celem trzeciego artykułu było zbadanie dynamicznej relacji pomiędzy doświadczeniami PLEs, negatywnymi emocjami, ruminacjami, antycypacją zagrożenia (ang. *Threat Anticipation*), nadmiernym nadawaniem znaczeń (ang. *Aberrant Salience*) oraz poziomem kortyzolu w warunkach codziennego funkcjonowania osób z różnym nasileniem objawów ADHD. Analiza bazowała na danych zebranych metodą ESM i obejmowała analizę sieci czasowych oraz porównanie struktur sieciowych pomiędzy grupami osób z wysokim i niskim nasileniem objawów ADHD. Postawiono następujące **Pytanie badawcze 3:** Czy dynamika czasowych powiązań pomiędzy kortyzolem, doświadczeniami PLEs, negatywnymi emocjami, ruminacjami, antycypacją zagrożenia i nadmiernym nadawaniem znaczeń różni się pomiędzy osobami z wysokim i niskim nasileniem objawów ADHD? Oraz następujące hipotezy badawcze: **(H3a)** Sieci czasowe osób z wyższym nasileniem objawów ADHD będą charakteryzowały się silniejszymi i bardziej złożonymi powiązaniami pomiędzy poziomem kortyzolu, PLEs, negatywnymi emocjami, ruminacjami, antycypacją zagrożenia oraz nadmiernym nadawaniem znaczeń w porównaniu do osób z niższym nasileniem objawów ADHD; oraz **(H3b)** Poziom kortyzolu będzie silniej powiązany z późniejszymi doświadczeniami PLEs w grupie z wysokim nasileniem objawów ADHD niż w grupie z niskim nasileniem objawów ADHD.

4. Metodologia oraz wyniki

Poniżej znajdują się syntetyczne streszczenia poszczególnych publikacji wchodzących w skład cyklu. Szczegółowe opisy metod, wyników oraz ich omówienia znajdują się w poszczególnych publikacjach składających się na prezentowaną rozprawę. Bazy danych oraz materiały uzupełniające dostępne są w repozytorium Open Science Framework (OSF) pod adresem https://osf.io/8ju6b/overview.

Publikacja 1: Gelner, H., Karska, J., Gawęda, Ł., Samochowiec, J., & Misiak, B. (2023). Effects of the interaction between PTSD and ADHD symptoms on the level of reporting psychotic-like experiences: findings from a non-clinical population. *Frontiers in psychiatry*, *14*, 1232606. https://doi.org/10.3389/fpsyt.2023.1232606

Wstęp: Czynniki neurorozwojowe i psychospołeczne leżące u podstaw doświadczeń PLEs pozostają od lat istotnym przedmiotem badań (np. Kessler i in., 2010; Kotowicz i in., 2021; Misiak i in., 2023). Brakowało jednak pracy, która jednocześnie badałaby rolę zaburzeń neurorozwojowych oraz niekorzystnych doświadczeń życiowych w tym kontekście. Dlatego celem niniejszego artykułu było uzupełnienie tej luki badawczej poprzez analizę interakcji czynników ryzyka takich jak PTSD i ADHD na poziom nasilenia doświadczeń PLEs.

Metody: Zrekrutowano łącznie 3000 osób w wieku 18–35 lat (58.3% kobiet), bez historii leczenia psychiatrycznego, które wypełniło zestaw samoopisowych kwestionariuszy online. W ankiecie pytano m.in.

o występowaniu doświadczeń PLEs ocenianych za pomocą 16–itemowej skali, obejmującej pozycje z trzech kwestionariuszy: (**1**) Zrewidowanej Skali Halucynacji (Gaweda & Kokoszka, 2011; Morrison i in., 2000, 2002); (**2**) Zrewidowanej Skali Myśli Paranoicznych Greena (Freeman i in., 2021); oraz (**3**) Kwestionariusza Prodromalnego (Ising i in., 2012). Osoby badane zostały przebadane pod kątem objawów ADHD, ocenianych za pomocą 18-itemowej przesiewowej skali do samooceny ADHD u osób dorosłych (ang. *Adult ADHD Self-Report Scale*; ASRS; Kessler i in., 2005). Wynik w kierunku wstępnej diagnozy dla ADHD określono za pomocą pierwszych sześciu pozycji kwestionariusza, które wykazują największą użyteczność w przesiewowym badaniu ADHD. Osoby, które uzyskały pozytywne wyniki w co najmniej czterech z tych pozycji z częstotliwością objawów wynoszącą co najmniej 2 – „*czasami*" (pytania 1 – 3) lub 3 – „*często*" (pytania 4 – 6), przypisano do grupy z pozytywnym wynikiem przesiewowym ADHD. Za pomocą trzech pytań z Międzynarodowego Wywiadu Neuropsychiatrycznego (ang. *Mini-International Neuropsychiatric*; M.I.N.I.; Sheehan i in., 1998), osoby poddano wstępnemu badaniu przesiewowemu w kierunku PTSD i sklasyfikowano jako pozytywne po udzieleniu twierdzących odpowiedzi na trzy pytania.

Wyniki: Wyniki potwierdziły postawioną hipotezę **H1a**, wskazując, że pozytywne wyniki przesiewowe w kierunku diagnozy PTSD i ADHD były istotnie związane z wyższym nasileniem doświadczeń PLEs w badanej populacji. Analiza ANOVA wykazała istotne efekty główne zarówno ADHD, jak i PTSD oraz interakcji ADHD x PTSD na ogólny wynik nasilenia PLEs, jak również na podskale tj. DLEs (doświadczeń podobnych do urojeń) i HLEs (doświadczeń podobnych do halucynacji), nawet po uwzględnieniu kowariantów. Wyniki wskazały, że najwyższe nasilenie PLEs obserwowane było u osób, u których potwierdzono jednocześnie wstępną diagnozę ADHD i PTSD. Potwierdziliśmy także hipotezę **H1b**, że osoby z pojedynczym pozytywnym wynikiem przesiewowym (grupa ADHD+ & PTSD– lub ADHD– & PTSD+) zgłaszały wyższe nasilenie PLEs w porównaniu z grupą bez diagnoz przesiewowych.

Dyskusja: Badanie pierwsze uzupełniło lukę w literaturze (np. Freeman & Fowler, 2009; Jannini i in., 2025; Logoń i in., 2024; Nourredine i in., 2021; Rodolfo Rossi i in., 2023), wskazując na istotny efekt interakcji objawów PTSD i ADHD na poziom nasilenia doświadczeń PLEs. Wykazaliśmy, że współwystępowanie obu zaburzeń może wiązać się z większym nasileniem PLEs, podkreślając tym samym potencjalną rolę współwystępowania tych zaburzeń w zwiększaniu ryzyka PLEs. Należy jednak podkreślić, że badanie przeprowadzone zostało na próbie nieklinicznej z wykorzystaniem samoopisowych narzędzi przesiewowych, co ogranicza możliwość wnioskowania o faktyczne rozpoznania kliniczne i pozwala jedynie na interpretację wyników w kategoriach nasilenia objawów. Co ważne, przekrojowy charakter badania nie pozwala na określenie przyczynowości ani kierunkowości analizowanych związków, dlatego nasze wyniki należy interpretować z pewną ostrożnością.

Publikacja 2: Gelner, H., Bagrowska, P., Jeronimus, B. F., Misiak, B., Samochowiec, J., & Gawęda, Ł. (2024). Psychotic-like Experiences and Underlying Mechanisms: An Integrative Model of ADHD Symptoms, Rumination, Negative Affect, and Trauma Experience. *Journal of Clinical Medicine*, *13*(22), 6727. https://doi.org/10.3390/jcm13226727

Wstęp: Dysregulacja emocjonalna jest uważana za jeden z kluczowych objawów występujących u osób z zaburzeniami neurorozwojowymi, takimi jak ADHD (American Psychiatric Association, 2013). U osób z doświadczeniami PLEs także często obserwuje się współwystępowanie zaburzeń neurorozwojowych (np. Hamshere i in., 2013; Marwaha i in., 2015), ale także objawów związanych z doświadczeniem traumy (Conti i in., 2023). Co istotne, osoby z objawami ADHD również często narażone są na zwiększoną podatność na doświadczenie traumy poprzez zwiększoną skłonność do ryzyka lub impulsywne zachowania (Magdi i in., 2025). Co ważne, objawy ADHD mogą nie tylko obniżać codzienne funkcjonowanie jednostek, ale także przyczyniać się do doświadczania większej liczby negatywnych emocji czy stosowania nieadaptacyjnych strategii regulacji emocji (Shaw i in., 2014; Yeguez i in., 2018). Podobnie jest u osób z doświadczeniami PLEs (Osborne i in., 2017). Sugeruje to więc na złożone związki między czynnikami wczesno-neurorozwojowymi, doświadczeniami traumatycznymi a mechanizmami radzenia sobie z doświadczanym stresem u osób z objawami ADHD i PLEs. Dlatego celem niniejszego badania było zbadanie pośredniczącej roli ruminacji oraz negatywnych emocji między objawami ADHD a PLEs, a także moderującej roli traumy w tej relacji.

Metody: W badaniu wzięło udział 188 osób (72.3% kobiet) w wieku 18 – 35 lat, z negatywną historią leczenia psychiatrycznego na przestrzeni życia. Badanie 1 (screeningowe) stanowiło pierwszy etap Badania 2. Główną częścią Badania 2 była siedmiodniowa procedura ESM, podczas której osoby wypełniały ankiety z pytaniami m.in. o doświadczenia PLEs, myśli ruminacyjne, negatywne emocje, oraz inne domeny nie uwzględnione w tym konkretnym badaniu. Podczas badania 1 osoby wypełniły samoopisowy kwestionariusz do pomiaru objawów ADHD (ASRS; Kessler i in., 2005) oraz potencjalnych doświadczeń traumatycznych (ang. *Traumatic Experiences Checklist;* TEC; Nijenhuis i in., 2002). Dane uzyskane poprzez procedurę ESM zostały zagregowane poprzez uśrednienie pomiarów z całego tygodnia, tak aby były porównywalne ze zmiennymi mierzonymi w jednym punkcie czasowym (dane kwestionariuszowe). Uśrednione wyniki odzwierciedlały ogólne nasilenie doświadczeń z całego tygodnia i umożliwiły wspólną analizę wszystkich zmiennych w modelu.

Wyniki: Analizy korelacji wykazały istotne związki pomiędzy wszystkimi zmiennymi uwzględnionymi w analizach ($p < 0.001$). Testowany model seryjnej mediacji potwierdził hipotezę **H2a**, zgodnie z którą objawy ADHD są związane z doświadczeniami PLEs. Wykazano istotny efekt całkowity objawów ADHD na PLEs, jednak efekt bezpośredni po uwzględnieniu mediatorów okazał się nieistotny, co wskazuje, że badany związek ma charakter wyłącznie pośredni. Potwierdza to tym samym hipotezę **H2b**, że ruminacje oraz negatywne emocje istotnie mediują związek między objawami ADHD a PLEs. Objawy ADHD wyjaśniały 18.7% wariancji PLEs, natomiast uwzględnienie w modelu mediatorów zwiększyło tę wartość do 49.5% wariancji PLEs. Sugeruje to, że związek pomiędzy objawami ADHD a PLEs realizowany jest w większości poprzez zwiększoną ilość myśli ruminacyjnych, które z kolei są związane z wyższym poziomem doświadczanych negatywnych emocji, w konsekwencji prowadząc do większego nasilenia PLEs. W końcu postawiona przez nas hipoteza **H2c**, że traumatyczne doświadczenia będą

moderować związek pomiędzy objawami ADHD a PLEs, nie została potwierdzona niezależnie od uwzględnionego w modelu rodzaju traumy (emocjonalnej, seksualnej czy cielesnej).

Dyskusja: Wyniki tego badania są zgodne z wcześniejszymi ustaleniami, że dysregulacja emocjonalna jest częstym zjawiskiem obserwowanym u osób z objawami ADHD (np. Faheem i in., 2022) i z PLEs (np. Gurnani & Georgiades, 2025; Leslie i in., 2025). Chociaż związek pomiędzy ADHD a PLEs został dobrze zgłębiony w badaniach (np. Bourgin i in., 2020; Cheng i in., 2025), to żadne z dotychczasowych badań nie uwzględniało w tej relacji pośredniczącej roli ruminacji i negatywnego afektu. Dlatego też badanie to dostarcza nowych wniosków w kontekście rozwoju PLEs u osób z objawami ADHD, wskazując konkretne czynniki pośredniczące w tej relacji, przyczyniające się do intensyfikacji tych doświadczeń. Chociaż nie potwierdziliśmy moderującej roli traumatycznych doświadczeń w naszym modelu, to pozostały one istotnym czynnikiem w badanych relacjach. Analiza korelacji wykazała istotne powiązania traumy ze wszystkimi pozostałymi zmiennymi, wskazując tym samym, że pozostaje ona istotnym czynnikiem ryzyka w rozwoju innych zaburzeń psychicznych. Nasze badanie ma jednak kilka ograniczeń. Po pierwsze, dane dotyczące objawów ADHD i traumy były oparte na kwestionariuszach samoopisowych, co ogranicza możliwość wnioskowania o konkretnych diagnozach. Po drugie, badanie ma charakter wyłącznie przekrojowy i chociaż ukazuje potencjalną ścieżkę powiązań, nie pozwala wnioskować o związki przyczynowe. Po trzecie, opiera się ono na danych uzyskanych od osób z niskim nasileniem badanych objawów i nie powinno być uogólniane na populację ogólną ze względu na znaczną przewagę kobiet w próbie. Wskazuje to więc na potrzebę dalszych badań w różnorodnych populacjach i podkreśla znaczenie interwencji ukierunkowanych na regulację emocji u osób z objawami ADHD i/lub PLEs.

Publikacja 3: Gelner, H., Bagrowska, P., Misiak, B., Samochowiec, J., & Gawęda, Ł. (2026). A comparative temporal network analysis of individuals with ADHD symptoms: an examination of the relationship between psychotic-like experiences and related factors using the Experience Sampling Method (ESM). *European archives of psychiatry and clinical neuroscience*, 10.1007/s00406-026-02203-3. Advance online publication. https://doi.org/10.1007/s00406-026-02203-3

Wstęp: Zarówno objawy ADHD, jak i PLEs wiążą się z subiektywnie podwyższonym poziomem doświadczanego stresu oraz trudnościami w regulacji emocji (Kandeğer i in., 2023; Ludwig i in., 2019). Jednocześnie badania wskazują na odmienne wzorce fizjologicznej reakcji na stres w tych grupach, określane m.in. poziomem kortyzolu, co sugeruje na różne mechanizmy funkcjonowania osi HPA w tych grupach. Osoby z PLEs wykazują zwiększoną reaktywność kortyzolu na bodźce stresowe (Collip i in., 2011), a osoby z objawami ADHD często jego opóźnioną lub obniżoną odpowiedź (Corominas-Roso i in., 2015). Ponadto osoby z objawami ADHD często wykazują deficyty w zakresie hamowania poznawczego, prowadzące do trudności w koncentracji na bodźcach istotnych, a zwiększonej reaktywności na bodźce nieistotne (Combs i in., 2015). Natomiast u osób z PLEs często obserwuje się nadmierne poszukiwanie zagrożenia (Freeman i in., 2013) oraz nadmierne nadawanie znaczeń (Kapur, 2003), które stanowią dobrze udokumentowane procesy poznawcze w tej grupie. Dotychczas brakowało badań integrujących procesy

emocjonalne, poznawcze i fizjologiczne w jednym dynamicznym modelu umożliwiającym analizę ich wzajemnych zależności czasowych. Dlatego celem niniejszego artykułu było zbadanie dynamiki relacji pomiędzy tymi zmiennymi z wykorzystaniem analiz sieciowych oraz porównanie ich struktur w grupach osób o wysokim i niskim nasileniu objawów ADHD.

Metody: Łącznie 188 osób (72.3% kobiet) w wieku 18 – 35 lat wzięło udział w siedmiodniowej procedurze ESM, która była częścią badania 2. Rekrutacja do badania oraz procedura ESM zostały opisane w **Publikacji 2**. W badaniu trzecim grupa badawcza została podzielona na podstawie nasilenia objawów ADHD, obejmując (**1**) grupę o wysokim nasileniu objawów ADHD (*N = 96*) i (**2**) grupę o niskim nasileniu objawów ADHD (*N = 92*). Punkt odcięcia został zdefiniowany na poziomie 34 punktów względem mediany wyniku kwestionariusza ASRS dla całej próby badawczej, wynoszącej 34.5 punktów. W badaniu ESM osoby pytane były o spożycie kofeiny, tytoniu, jedzenia i napojów alkoholowych i bezalkoholowych, PLEs, obejmujące doświadczenia podobne do halucynacji i urojeń, oczekiwanie zagrożenia, nadmierne nadawanie znaczeń, myśli ruminacyjne i negatywne emocje. W celu oznaczenia poziomu kortyzolu jako wskaźnika stresu osoby pobierały próbki śliny po każdym pomiarze ESM.

Wyniki: W grupie osób z wysokim nasileniem objawów ADHD sieć czasowa wykazała znaczące efekty autoregresyjne wszystkich uwzględnionych zmiennych w czasie. Zmienną, która najsilniej przewidywała inne zmienne w modelu, były doświadczenia podobne do urojeń, a zmienną, która była najsilniej przewidywana, były ruminacje. Zaobserwowano dwukierunkowy związek między negatywnym afektem a poszukiwaniem zagrożenia. Kortyzol znacząco przewidywał poszukiwanie zagrożenia w kolejnym punkcie czasowym, ale żadna z uwzględnionych zmiennych (z wyjątkiem uwzględnionych kowariantów) nie przewidywała poziomu kortyzolu. W grupie z niskim nasileniem objawów ADHD zmienną, która miała największą siłę zewnętrzną, były negatywne emocje, a zmienną z największą siłą wewnętrzną była antycypacja zagrożenia. Wszystkie zmienne, z wyjątkiem doświadczeń podobnych do urojeń, wykazywały autoregresję w czasie. Zaobserwowano dwukierunkowe powiązanie między negatywnymi emocjami a doświadczeniami podobnymi do halucynacji. Poziom kortyzolu przewidywał jedynie późniejsze spożycie alkoholu w następnym punkcie czasowym i nie był przewidywalny przez żadną inną zmienną. Porównania sieciowe pomiędzy grupami wykazały, że różniły się one istotnie w 8 krawędziach. Efekt autoregresyjny doświadczeń podobnych do urojeń był istotnie silniejszy w grupie z wysokim nasileniem objawów ADHD. Grupa z wysokim nasileniem objawów ADHD wykazała także silniejsze efekty czasowe dla kortyzolu przewidującego poszukiwanie zagrożenia oraz doświadczeń podobnych do urojeń przewidujących negatywne emocje poprzez spożycie alkoholu w porównaniu z grupą z niskim nasileniem objawów ADHD.

Dyskusja: Wyniki tego badania są zgodne z postawioną przez nas hipotezą **H3a**, że sieci temporalne osób z wyższym nasileniem objawów ADHD będą charakteryzowały się silniejszymi i bardziej złożonymi powiązaniami. W przypadku osób z wyższym nasileniem objawów ADHD występujące objawy miały bardziej dynamiczny charakter, a poszczególne symptomy nie występowały w izolacji, lecz wzajemnie się

wzmacniały, prowadząc tym samym do samopodtrzymującej się pętli sprzężeń zwrotnych, która skutkowała utrzymywaniem się objawów (Robinaugh i in., 2020). Może to częściowo tłumaczyć większą podatność tej grupy na współwystępowanie innych zaburzeń psychicznych oraz niższy poziom funkcjonowania psychospołecznego, obserwowany również w tym badaniu. Natomiast hipoteza **H3b**, że poziom kortyzolu będzie silniej powiązany z późniejszymi doświadczeniami PLEs w grupie z wysokim nasileniem ADHD, została potwierdzona jedynie częściowo. Mianowicie, w grupie z wysokim nasileniem objawów ADHD zaobserwowano jednie bezpośrednie czasowe powiązanie pomiędzy poziomem kortyzolu a antycypacją zagrożenia, co sugerować może na silniejszy związek pomiędzy reakcją fizjologiczną na stres a późniejszą interpretacją sytuacji jako zagrażającej. Bezpośrednie powiązanie kortyzolu z PLEs nie było jednak zaobserwowane w żadnej z grup. Wskazuje to, że rola kortyzolu w nasilaniu tych doświadczeń może mieć charakter pośredni i zależny od procesów poznawczo-emocjonalnych. Wyniki tego badania należy interpretować jednak z pewną ostrożnością ze względu na jego eksploracyjny charakter, brak możliwości wnioskowania przyczynowego oraz ograniczenia próby, w tym niskiego średniego poziomu PLEs, niskiego odsetka osób spełniających kryteria kliniczne ADHD oraz przewagę kobiet w próbie.

5. Dyskusja ogólna

Głównym celem niniejszej rozprawy było systematyczne zbadanie czynników ryzyka leżących u podstaw doświadczeń PLEs w populacji nieklinicznej. Szczególną uwagę poświęcono roli objawów ADHD jako czynnika ryzyka związanego z uwarunkowaniami neurorozwojowymi, objawów PTSD jako czynnika związanego z wczesnymi niekorzystnymi doświadczeniami, myśli ruminacyjnych i negatywnego afektu związanych z rolą dysregulacji emocjonalnej oraz kortyzolu jako markera fizjologicznej reakcji na stres.

Biorąc pod uwagę ograniczone dowody na temat znaczenia interakcji objawów ADHD i PTSD w nasileniu doświadczeń PLEs, w **badaniu pierwszym** podjęliśmy próbę zgłębienia tej zależności. Wyniki badania wskazały, że jednoczesna pozytywna diagnoza przesiewowa w kierunku ADHD i PTSD wiązała się z wyższym nasileniem zgłaszanych doświadczeń PLEs. Tym samym wyniki te potwierdzają istotną interakcję między objawami ADHD i PTSD w nasilaniu PLEs, uzupełniając obecną literaturę, wskazującą, że PTSD, jak i ADHD, są związane z częstszym zgłaszaniem PLEs (Compean & Hamner, 2019; Nourredine i in., 2021). Wyniki te idą również w zgodzie z założeniami socjo-poznawczo-rozwojowego modelu psychozy (Howes & Murray, 2014), że interakcja czynników wczesno-neurorozwojowych z niekorzystnymi doświadczeniami środowiskowymi może istotnie zwiększać ryzyko doświadczeń psychotycznych, co potwierdziliśmy w naszym badaniu. Jednak badanie to bazowało wyłącznie na samoopisowych kwestionariuszach, przesiewowych diagnozach oraz opierało się na próbie nieklinicznej, więc odnosi się jedynie do pewnego wycinka kontinuum doświadczeń. Co więcej, biorąc pod uwagę fakt, że badaliśmy jedynie proste efekty interakcji dwóch potencjalnych czynników ryzyka na poziom PLEs, nie mieliśmy możliwości identyfikacji konkretnych mechanizmów wskazujących, w jaki sposób czynniki te wpływają na nasilenie PLEs.

Badanie drugie oceniało pośredniczącą rolę myśli ruminacyjnych i negatywnych emocji oraz moderującą rolę doświadczeń traumatycznych w związku między objawami ADHD a doświadczeniami PLEs. Zidentyfikowaliśmy dwa kluczowe procesy łączące objawy ADHD i PLEs – więcej ruminacji oraz bardziej nasilone negatywne emocje. Uzyskane wyniki są zgodne z wysoką częstością występowania myśli ruminacyjnych u osób z ADHD (Spinhoven i in., 2018) oraz założeniami, że ruminacje mogą bezpośrednio kształtować indywidualne reakcje stresowe jednostki poprzez przedłużanie negatywnych stanów emocjonalnych (np. Watkins & Roberts, 2020). Nadal jednak dokładna rola ruminacji w tej relacji pozostaje niejasna. Pytanie dotyczące ruminacji nie określało bezpośrednio tematyki tych myśli, a jedynie skupiało się na ich ogólnym negatywnym wydźwięku. Ogranicza to więc możliwość wnioskowania, czy myśli te związane były z PLEs (McKie i in., 2017), traumatycznymi doświadczeniami z przeszłości (Moulds i in., 2020), codziennymi stresorami związanymi z objawami ADHD (Vega i in., 2025) czy może innymi aspektami funkcjonowania jednostki. Nasze wyniki nadal nie rozstrzygają, czy osoby z PLEs mają tendencję do częstszego ruminowania, czy też może ruminacje nasilają ryzyko doświadczeń PLEs. Przyszłe badania powinny uwzględniać nie tylko częstotliwość i nasilenie ruminacji, lecz także ich treść, w celu ustalenia, czy nasilają one PLEs, są ich konsekwencją, czy współwystępującym objawem ADHD.

Kolejnym etapem badania drugiego było zbadanie moderującej roli traumy w testowanym modelu. Wyniki analiz korelacyjnych wskazały na jej istotny związek ze wszystkimi uwzględnionymi zmiennymi w badaniu. Tym samym nasze wyniki są zgodne z badaniami, które konsekwentnie identyfikują doświadczenia traumatyczne jako istotne czynniki ryzyka rozwoju przyszłych zaburzeń psychicznych (Hogg i in., 2023). Jednak wyniki analiz moderowanej szeregowej mediacji nie potwierdziły istotnego wpływu traumy na ścieżkę łączącą objawy ADHD z PLEs poprzez ruminacje i negatywne emocje. Zakładamy, że brak istotnej roli traumy w naszym modelu może wynikać z potencjalnego nakładania się objawów ADHD i traumy, takich jak trudności z koncentracją, drażliwość czy podwyższone pobudzenie (Daud & Rydelius, 2009). Mogło to więc utrudnić możliwość rozróżnienia jej dokładnego wpływu w tej relacji. W związku z tym zalecamy, aby przyszłe badania nadal uwzględniały rolę traumy w kontekście objawów ADHD i PLEs jako istotnego obszaru wartego dalszej eksploracji, mając jednak na uwadze możliwość nakładania się objawów lub nawet funkcjonowania ich jako jednego zespołu zaburzeń (Haeffel i in., 2022). Badanie drugie miało kilka ograniczeń. Poprzez agregację danych do średnich wartości z całego tygodnia nie wykorzystaliśmy w pełni możliwości, które dają dane ESM. Więc chociaż badanie skupiło się na efektach międzyosobowych, co było jego głównym celem, to jednak ograniczyło to naszą zdolność do pełnego zrozumienia dokładnej dynamiki zmian wewnątrzosobowych (Fisher i in., 2018). Co więcej, przekrojowy charakter tego badania ograniczył zdolność do ustalenia dokładnych wniosków przyczynowo-skutkowych leżących u podstaw związku pomiędzy objawami ADHD a PLEs. W kolejnym artykule przeprowadziliśmy analizy w pełni wykorzystujące potencjał danych ESM w celu lepszego zrozumienia mechanizmów leżących u podstaw badanej relacji.

Opierając się na założeniach podejścia sieciowego w psychopatologii (Bortolon & Raffard, 2019), wykorzystaliśmy temporalne analizy sieciowe w celu uchwycenia dokładniejszych interakcji między różnymi czynnikami ryzyka związanymi z doświadczeniami PLEs u osób z objawami ADHD. Analizy **badania trzeciego** rozszerzyliśmy o dwa dodatkowe dobrze udokumentowane procesy poznawcze związane z PLEs, takie jak przewidywanie zagrożeń (Freeman i in., 2013) i nadmierne nadawanie znaczeń (Kapur, 2003). Umożliwiło to bardziej precyzyjną identyfikację mechanizmów podtrzymujących lub nasilających PLEs w tej grupie. Model został uzupełniony także o komponent fizjologiczny, związany z reakcją organizmu na stres, reprezentowany w poziomach kortyzolu. Analizy sieci temporalnych wykazały, że grupa z wysokim nasileniem objawów ADHD w porównaniu z grupą z niskim ich nasileniem wykazała silniejsze i bardziej złożone powiązania między wszystkimi zmiennymi uwzględnionymi w modelu. Żaden z węzłów w sieci osób z wysokim nasileniem objawów ADHD nie był odizolowany, co podkreśla złożoność zaangażowanych procesów i ich samonapędzające się mechanizmy. Sugeruje to, że im więcej objawów, tym silniejsza staje się ich wzajemna zależność w sieci, a obecność kolejnych objawów skutkuje dodatkowym wzmocnieniem połączeń między nimi (Robinaugh i in., 2020). Analizy wykazały, że niezależnie od nasilenia objawów ADHD, negatywny afekt był najsilniejszym predyktorem myśli ruminacyjnych w kolejnym pomiarze. Wynik ten pozwala lepiej wyjaśnić rolę myśli ruminacyjnych w tej konkretnej grupie, wskazując, że są one pośrednio lub bezpośrednio przewidywane przez negatywne emocje w czasie. Jednocześnie ruminacje w każdej z grup wykazywały stabilność w czasie, przewidując same siebie w kolejnych pomiarach, co sugeruje ich samopodtrzymujący się charakter. W świetle modelu Howesa i Murraya (2014) myśli ruminacyjne, które nie były uwzględniane w modelu, mogą stanowić jego dodatkowy pośredniczący komponent poznawczo-emocjonalny między objawami ADHD a PLEs. Wyniki pomiarów centralności wskazały, że w grupie z wysokim nasileniem objawów ADHD doświadczenia podobne do urojeń stanowiły centralny węzeł w sieci, wykazując największą siłę predykcji nasilenia innych objawów w kolejnym punkcie pomiaru. Za to w grupie z niskim nasileniem objawów ADHD były to negatywne emocje. Centralność tych zmiennych uzasadnia uwzględnienie ich jako prawdopodobnie istotnych obszarów przyszłych badań nad czynnikami ryzyka PLEs w grupie osób z objawami ADHD. Analizy centralności wskazały również obszary, które były najmocniej przewidywane przez inne zmienne w kolejnym pomiarze. W grupie z wysokim nasileniem objawów ADHD były to ruminacje, a w grupie z niskim nasileniem objawów ADHD poszukiwanie zagrożenia. Wskazuje to, że w zależności od poziomu nasilenia objawów ADHD osoby przyjmują różne strategie radzenia sobie z doświadczeniami. Wyniki te mogą odzwierciedlać obserwowane u osób z ADHD różnice w deficytach funkcji wykonawczych czy ograniczonej elastyczności poznawczej, które znacząco wpływają na zdolność utrzymywania uwagi, adaptacji do zmian w środowisku czy przełączania się między zadaniami lub myślami, istotnie obniżając jakość życia (Eberhard i in., 2024; Hong i in., 2022; Luna-Rodriguez i in., 2018; Vega i in., 2025). Sugeruje to, że osoby z wyższym nasileniem objawów ADHD mogą wykazywać więcej trudności ze stosowaniem adaptacyjnych strategii regulacji emocji i przełączaniem uwagi, co może sprzyjać samonapędzaniu się

ruminacji. Natomiast osoby z niższym nasileniem objawów ADHD w reakcji na swoje emocje i doświadczenia wykazują bardziej zewnętrznie ukierunkowane strategie regulacji emocji. Zatem obszary te mogą stanowić ważny cel terapeutyczny podczas projektowania interwencji ukierunkowanych na regulację emocji osób z objawami ADHD. Wyniki te można też z pewną ostrożnością interpretować w świetle modelu Howesa i Murraya (2014), wskazując, że współwystępujące objawy ADHD mogą stanowić istotny czynnik o komponencie neurorozwojowym, nasilający doświadczenia PLEs poprzez zniekształcone schematy poznawcze oraz deficyty w adaptacyjnych strategiach regulacji emocji. Interpretacja ta wymaga jednak ostrożności ze względu na niekliniczną charakterystykę naszej grupy. Niemniej jednak wyniki sugerują, że model ten można uzupełnić o istotną rolę objawów neurorozwojowego zaburzenia takiego jak ADHD, w mechanizmach kształujących ryzyko psychoz.

W kolejnym etapie przeprowadziliśmy analizy porównawcze sieci osób z wysokim i niskim nasileniem objawów ADHD. W przeciwieństwie do analiz temporalnych, które ukazywały związki czasowe między zmiennymi w obrębie każdej z grup, analizy porównawcze pozwoliły na identyfikację silniejszych powiązań między grupami. W grupie z wysokim nasileniem objawów ADHD zaobserwowaliśmy silniejszy efekt autoregresji doświadczeń podobnych do urojeń, co może wskazywać na ich większą stabilność i trwałość w czasie niż w grupie o niskim nasileniu objawów ADHD. W grupie z wysokim nasileniem objawów ADHD w porównaniu z grupą z niskim nasileniem objawów ADHD zaobserwowaliśmy silniejsze powiązania między: (1) poziomem kortyzolu a poszukiwaniem zagrożenia, (2) doświadczeniami podobnymi do urojeń a poszukiwaniem zagrożenia oraz (3) pośrednią ścieżką między doświadczeniami podobnymi do urojeń a negatywnymi emocjami poprzez spożycie alkoholu. Wskazuje to, że osoby z wyższym nasileniem objawów ADHD używają alkoholu jako nieadaptacyjnej strategii regulacji emocji w odpowiedzi na doświadczenia podobne do urojeń, co w konsekwencji prowadzi do odczuwania negatywnych emocji. W grupie tej występował silniejszy związek między fizjologiczną reakcją na stres a późniejszą interpretacją sytuacji jako zagrażającej. Sugeruje to, że w odpowiedzi na stres osoby z wyższym nasileniem objawów ADHD częściej niż osoby z niskim nasileniem objawów ADHD interpretują środowisko jako zagrażające. Jednym z możliwych wyjaśnień tego mechanizmu mogą być deficyty w zakresie metapoznania i funkcji wykonawczych obserwowane u osób z ADHD (Butzbach i in., 2021), które mogą ograniczać ich zdolność do adekwatnej oceny własnych przeżyć i samoregulacji (Williamson i in., 2010). W konsekwencji stres, który odzwierciedlany jest w podwyższonych poziomach kortyzolu, może być błędnie przypisywany czynnikom zewnętrznym, sprzyjając tym samym nadmiernemu poszukiwaniu w nim zagrożenia. W grupie z niskim nasileniem objawów ADHD w porównaniu z grupą z wysokim ich nasileniem zaobserwowaliśmy silniejszy związek między: (1) doświadczeniami podobnymi do halucynacji a ruminacjami, (2) doświadczeniami podobnymi do halucynacji a poszukiwaniem zagrożenia oraz (3) poziomem kortyzolu a spożyciem alkoholu. Wskazuje to, że osoby z niższym nasileniem objawów ADHD, pod wpływem doświadczeń podobnych do halucynacji, więcej ruminują lub szukają wyjaśnień w środowisku zewnętrznym, a w odpowiedzi na stres stosują nieadaptacyjne strategie regulacji emocji.

Podsumowując, badanie trzecie ujawniło szereg istotnych mechanizmów leżących u podłoża PLEs u osób z różnym nasileniem objawów ADHD. Jednocześnie badanie to posiada kilka istotnych ograniczeń. Chociaż kortyzol został użyty jako wskaźnik fizjologicznej reakcji organizmu na stres, to w procedurze ESM nie uwzględniliśmy subiektywnej oceny odczuwanego stresu. Może jednak być to istotne w kontekście osób z objawami ADHD, u których obserwuje się zmniejszoną fizjologiczną reakcję na stres i spowolnione uwalnianie kortyzolu (np. Chang i in., 2021). Badania wskazują, że zarówno osoby z objawami ADHD, jak i osoby doświadczające PLEs raportują wyższy poziom stresu niż osoby z grup kontrolnych (Combs i in., 2015; DeLuca i in., 2022). Jednak wyniki naszego badania nie wykazały tego w poziomach kortyzolu. Możliwe wyjaśnienia mogą obejmować opóźnioną reakcję osi HPA u osób z ADHD, ale także ograniczenia samej procedury pobierania próbek śliny w celu określenia poziomów kortyzolu. Sześciokrotne pomiary poziomu kortyzolu mogły w pełni nie uchwycić ich dynamicznych i zależnych od stresora fluktuacji. W związku z tym zalecamy, aby przyszłe badania uwzględniały także subiektywne miary stresu, co umożliwiłoby porównanie subiektywnego poziomu stresu z fizjologiczną reakcją organizmu i lepsze zrozumienie ewentualnych dysproporcji w tym obszarze. Kolejnym ograniczeniem tego badania była przewaga kobiet w próbie. Wpłynęło to nie tylko na brak możliwości generalizacji naszych wyników na populację ogólną, ale także na ograniczoną interpretację wyników związanych z kortyzolem, ze względu na istotny dymorfizm płciowy związany z aktywnością osi HPA. Badania wskazują (np. Herman i in., 2016), że kobiety wykazują większą zmienność w aktywacji osi HPA w odpowiedzi na stres, a wahające się poziomy hormonów w cyklu menstruacyjnym mogą wpływać na fizjologiczną odpowiedź organizmu na stres. W związku z tym kolejne badania powinny uwzględniać również cykl menstruacyjny oraz oznaczać poziomy hormonów związanych z fazą cyklu. Podsumowując, uważamy, że uwzględnienie powyżej omówionych ograniczeń w przyszłych badaniach przczyniłoby się do bardziej precyzyjnego i wieloaspektowego zrozumienia roli stresu w kontekście osób z objawami ADHD doświadczających PLEs.

Przedstawiony cykl posiada także kilka istotnych ograniczeń o charakterze ogólnym. Po pierwsze, w żadnym badaniu nie byliśmy w stanie określić dokładnego kierunku zależności między badanymi zmiennymi. Chociaż w badaniu drugim zaproponowaliśmy hipotetyczny model mediacji szeregowej sugerujący związki kierunkowe, to wnioskowanie przyczynowe wymagałoby badań podłużnych lub eksperymentalnych. Po drugie, mimo że w badaniu trzecim analizowaliśmy rolę stresu reprezentowaną w fizjologicznych wartościach kortyzolu jako czynnika potencjalnie wzmacniającego doświadczenia PLEs, to warto mieć na uwadze, że same PLEs również mogą stanowić istotne źródło stresu (Logoń i in., 2024). Tym samym relacja między stresem a PLEs może mieć charakter dwukierunkowy i wymaga przyszłych badań sprawdzających tę potencjalną zależność. Kolejnym ograniczeniem naszych badań jest brak bezpośrednich pomiarów objawów ADHD w życiu codziennym. Chociaż zastosowane narzędzie do oceny objawów ADHD pozwoliło nam na ogólną ocenę ich nasilenia, to nie uwzględniliśmy ich codziennych fluktuacji w życiu codziennym, co niewątpliwie dostarczyłoby bardziej precyzyjne informacje na temat ich roli w doświadczaniu PLEs. Istotnym ograniczeniem jest także charakterystyka naszej próby. We wszystkich

trzech badaniach uczestniczyły osoby z relatywnie niskim ogólnym nasileniem objawów ADHD, oraz PLEs, co ogranicza możliwość generalizacji naszych wyników na populacje kliniczne. Co więcej, były to osoby dobrze funkcjonujące społecznie, z wykształceniem wyższym lub średnim, co ogranicza możliwość generalizacji naszych wyników na szerszą populację. Ponadto, chociaż nasze badanie miało na celu rekrutację osób z grupy niekliniczej, co potwierdziliśmy brakiem leczenia psychiatrycznego na przestrzeni całego życia i kontrolą przyjmowanych leków psychiatrycznych, to jednak osoby badane wykazywały stosunkowo wysokie rozpowszechnienie wstępnych diagnoz zaburzeń depresyjnych i lękowych na przestrzeni życia. Mimo że, podobne wskaźniki rozpowszechnienia tych zaburzeń odnotowano także w innych badaniach populacji ogólnej (np. Alqahtani i in., 2023; Bello i in., 2022), to określenie naszej grupy jako "niekliniczej" może nie być w pełni trafne. Kolejnym aspektem, który warto omówić, jest ograniczenie związane z metodologią podejścia sieciowego. Dobór zmiennych w modelu, choć oparty był na wnioskach płynących z poprzednich badań, to jednak dodanie lub ujęcie jakiejś określonej zmiennej mogłoby istotnie zmieniać strukturę sieci i dynamikę interakcji. Oznacza to więc, że uzyskane wyniki odzwierciedlają jedynie pewien wycinek bardziej złożonych procesów leżących u podłoża PLEs. Ponadto w badaniu ESM wykorzystaliśmy wyłącznie pytania zamknięte, reprezentowane w wartościach liczbowych. Ogranicza to w znacznym stopniu możliwość uchwycenia dokładniejszego kontekstu tych doświadczeń. Odpowiedzi otwarte mogłyby z pewnością dostarczyć istotnych informacji pozwalających na uchwycenie znaczenia i przyczyn objawów oraz określenie sekwencji tych zdarzeń w codziennym funkcjonowaniu. Mogłoby to ulepszyć dane ESM, osadzając je w szerszym kontekście rzeczywistych doświadczeń i zdarzeń, pogłębiając tym samym interpretację uzyskanych rezultatów (Bringmann i in., 2026).

5.1. Konkluzje

Wyniki zaprezentowanych badań w cyklu dostarczyły nowych spostrzeżeń na temat mechanizmów związanych z PLEs oraz konsekwentnie wskazały, że objawy ADHD mogą stanowić jeden z elementów wyjaśniających indywidualne różnice w dynamice ich nasilania. Wykazaliśmy, że relacja między objawami ADHD a PLEs ma charakter złożony i wieloczynnikowy. Jednocześnie pokazaliśmy, że nasilenie objawów ADHD różnicuje związki między PLEs, a czynnikami poznawczymi i mechanizmami regulacji emocji. Dlatego uważamy, że ADHD może stanowić istotny komponent o charakterystyce neurorozwojowej jako wartość dodana do modelu Howesa i Murraya (2014), na którego założeniach opieraliśmy niniejszą pracę. Uzyskane wyniki mają również istotne implikacje kliniczne. Wskazują na potrzebę identyfikacji osób z objawami ADHD jako grupy zwiększonego ryzyka wystąpienia PLEs. Podkreślają także znaczenie projektowania interwencji ukierunkowanych na poprawę regulacji emocji i stosowania adaptacyjnych strategii radzenia sobie ze stresem. Jednocześnie nasze wyniki wyznaczają potencjalne kierunki przyszłych badań. Uważamy, że wartościowe byłoby badania podłużne i eksperymentalne umożliwiające określenie kierunkowości ustalonych przez nas związków oraz ich replikacja na populacjach klinicznych w celu uzyskania pełniejszego wglądu w zaobserwowane mechanizmy.

6. Referencje

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Effects of the interaction between PTSD and ADHD symptoms on the level of reporting psychotic-like experiences: findings from a non-clinical population

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Objective: Psychotic-like experiences (PLEs) are increasingly being recognized as subclinical phenomena that might predict the development of various mental disorders that are not limited to the psychosis spectrum. Accumulating evidence suggests that attention-deficit/hyperactivity disorder (ADHD) and post-traumatic stress disorder (PTSD) are highly comorbid mental disorders. However, their interactive effect on the occurrence of PLEs has not been investigated so far. Therefore, in the present study we aimed to investigate the effect of interaction between ADHD and PTSD symptoms on the level of psychotic-like experiences (PLEs) in the non-clinical sample.

Methods: The study included 3,000 individuals aged 18–35 years with a negative history of psychiatric treatment. The symptoms of ADHD and PTSD were assessed using self-reports.

Results: There was a significant association of the interaction between ADHD and PTSD with the level of reporting PLEs. This association remained significant after adjustment for age, gender, the level of education, the current vocational situation, lifetime history of problematic substance use, and depressive symptoms. Post-hoc tests demonstrated significantly higher levels of reporting PLEs in participants with positive screening for both ADHD and PTSD compared to other subgroups of participants. Also, individuals with positive screening for one vulnerability (either ADHD or PTSD) reported significantly higher levels of reporting PLEs compared to those with a negative screening for ADHD and PTSD. In turn, no significant differences between individuals reporting one vulnerability, i.e., between those with positive screening for ADHD and those with positive screening for PTSD, were observed.

Conclusion: Findings from the present study imply that both PTSD and ADHD symptoms the interaction effect on the level of reporting PLEs that might be of importance for early intervention strategies. However, observed associations require replication in clinical samples.

KEYWORDS

ADHD, PTSD, psychosis, comorbidity, early intervention

1. Introduction

Psychotic-like experiences (PLEs) represent frequent psychopathological phenomena with the median annual prevalence estimated at 7.2% according to a systematic review and meta-analysis (1). Most commonly, PLEs are defined as subclinical phenomena that develop in the absence of underlying illness in terms of international diagnostic systems (2–4). It has been shown that the emergence of PLEs is associated with increased risk of developing mental disorders that are not limited to psychosis spectrum, suicidal behavior, multimorbidity, and poor functioning (5, 6). Although a great progress toward understanding the mechanisms contributing to the development of PLEs has been made in recent years, certain aspects remain unclear.

One of the most crucial mechanisms that might account for the appearance of psychosis spectrum symptoms are alterations in the process of neurodevelopment (7, 8). It has been reported that psychotic symptoms often occur in vulnerable populations affected by common neurodevelopmental disorders. Among them, several studies have focused on attention-deficit/hyperactivity disorder (ADHD) that might persist until adulthood, and affect even 2.5% of adults (9, 10). It has been demonstrated that ADHD diagnosed in childhood is associated with a fivefold higher risk of psychotic disorder in adulthood (11). Moreover, ADHD is highly comorbid with other mental disorders, including, i.e., autism spectrum disorder (12), mood disorders (13, 14), substance use disorders (15, 16).

Exposure to traumatic events is a well-known trigger of various psychiatric disorders, including post-traumatic stress disorder (PTSD) with prevalence rates estimated at 5.6% of trauma-exposed individuals (17). The association between PTSD and psychotic symptoms has been established by several studies and summarized in the ICD-11 for both PTSD and complex PTSD. Moreover, certain risk factors for psychotic symptoms secondary to a diagnosis of PTSD have been reported. These include trauma-related characteristics (childhood trauma and war trauma), refugee and ethnic minority status, comorbid mood disorders and substance use (18). PLEs secondary to PTSD usually include persecutory ideation and auditory or visual hallucination-like experiences (19, 20). Moreover, there is evidence that psychotherapeutic interventions based on cognitive behavioral therapy (CBT) and eye movement and desensitization and reprocessing therapy (EMDR) might be effective for people with PTSD comorbid with other mental disorders and psychotic symptoms (18).

Accumulating evidence indicates that ADHD and PTSD tend to co-occur and might be causally associated (21). Indeed, the most recent Mendelian randomization study demonstrated that ADHD genetic liability increases the risk of PTSD (22). Authors of this study found that risk-taking behaviors, household income, and educational attainment might only partially mediate this association. Specific neurobiological mechanisms underlying the association between ADHD and PTSD are yet to be determined. Nevertheless, it has been found that prenatal nicotine exposure leads to the development of ADHD-like behaviors together with impairments of fear extinction learning in mice (23, 24). The later one might also occur in people with PTSD (25). There are also studies showing beneficial effects of methylphenidate on fear extinction and PTSD symptoms (26, 27). Also, a number of risk factors for comorbid ADHD and PTSD have been reported including the effect of low intelligence quotient, a history of traumatic experiences in parents, alcohol use disorders and familial transmission of this comorbidity (28–30). It has been shown that a diagnosis of PTSD in individuals with ADHD is associated with a higher risk of psychiatric hospitalization, school impairment, worse social and cognitive performance as well as a higher likelihood of mood, conduct disorder, and anxiety disorders (29, 31).

To date, none of previous studies have investigated the interactive effects of PTSD and ADHD symptoms on the occurrence of PLEs. In the present study, we hypothesized that PTSD and ADHD symptoms exert interaction effects on the level of reporting PLEs. Our aim was to explore the interaction between PTSD and ADHD symptoms in a non-clinical population of young adults.

2. Methods

2.1. Recruitment procedures

The total sample included 3,000 individuals recruited through social media and the survey website. Recruitment was conducted via an online survey, with the advertisement targeted at individuals aged 18–35 years, without a history of psychiatric treatment. The findings reported in the present article represent a part of a bigger project investigating epigenetic mechanisms of psychosis proneness. At the screening level, the survey included only email addresses and telephone numbers for participants willing to participate in further stages of the project. Additional personal data were not collected at this stage of the project. Some findings from this study were published previously (32–34). The study was approved by the Ethics Committees at the Institute of Psychology (Polish Academy of Sciences in Warsaw, Poland, approval number: 16/VII/2022), Wroclaw Medical University (Wroclaw, Poland, approval number: 129/2022) and Pomeranian Medical University (Szczecin, Poland, approval number: KB-006/25/2022).

2.2. Measures

2.2.1. PLEs

The occurrence of PLEs was assessed using a 16-item self-report referring to the preceding month. Participants were asked to assess the frequency of PLEs on a 4-point scale (1 – "never"; 2 – "sometimes"; 3 – "often" and 4 – "almost always"), taking into consideration the experiences that had not been related to substance use. Specific items were selected from three questionnaires, including: (1) the Revised Hallucination Scale (35–37): "I hear voice speaking my thoughts aloud," „I hear people call my name and find that nobody has done so" and "I see shadows and shapes when there is nothing there", (2) the Revised Green et al., Paranoid Thoughts Scale (38): "I spent time thinking about friends gossiping about me," "People wanted me to feel threatened, so they stared at me," "I was convinced there was a conspiracy about me," "I was distressed by being persecuted" and "People have been dropping hints for me", and (3) the Prodromal Questionnaire (39): "When I look at a person, or look at myself in a mirror, I have seen the face change right before my eyes," "I have heard things other people cannot hear like voices of people whispering or talking," "I often feel that other have it in for me," "I have seen things that other people apparently cannot see," "I have had the sense that

some person or force is around me, even though I could not see anyone," "I sometimes see special meanings in advertisements, shop windows, or in the way things are arranged around me," "I sometimes smell or taste things that other people cannot smell or taste" and "I often seem to live through events exactly as they happened before." We decided to use items from various questionnaires as existing tools often record the presence of various experiences including hallucination-like experiences, delusion-like experiences, negative, and depressive symptoms (40). The total score of this questionnaire ranges between 16 and 64 points, with higher scores referring to the greater number and frequency of PLEs. The Cronbach's alpha of the questionnaire in the present study was 0.784.

2.3. The screening for ADHD

The Adult ADHD Self-Report Scale (ASRS) was administered to screen for ADHD. It includes 9 items measuring inattention and 9 items measuring impulsivity/hyperactivity over the period of the preceding 6 months (41). Each item is based on a 5-point scale (from 0 – "never" to 4 – "very often"). The total ASRS score ranges between 0 and 72. The first six items of the ASRS were found to show the highest utility in screening for ADHD. It has been found that individuals scoring positively on at least four of these items should be further assessed for a diagnosis of ADHD (41). Positively scored items include those with the symptom frequency reported to be at least 2 – "sometimes" (items 1–3) or 3 – often (items 4–6). Following this threshold, participants were divided into those with positive screening for ADHD and those with negative screening for ADHD. The Cronbach's alpha of the ASRS was 0.819 in the present study.

2.4. The screening for PTSD

Participants were screened for a diagnosis of PTSD using three questions from the Mini-International Neuropsychiatric (M.I.N.I.) (42): 1) "Have you ever experienced, witnessed or faced an extremely traumatic event, during which people have died or you and/or other persons been in danger of being killed, were severely injured or were harmed with regard to their physical integrity? Examples of traumatic events: severe accident, physical assault, rape, terrorism, hostage-taking, kidnapping, fire, discovering a dead body, sudden death of a significant other, war, natural disasters."; 2) "Do you often think of this event in a distressing way, do you dream about it or do you frequently have the impression of re-experiencing it?" 3) "Since the occurrence of this event, have you had a tendency to avoid everything that could remind you of the event?." Participants were classified as screened positive in case of positive responses to all questions.

2.5. Depressive symptoms

Depressive symptoms were measured using the Patient Health Questionnaire-9 (PHQ-9) (43). It is a 9-item questionnaire that measures the level of depressive symptoms over the preceding two weeks. Each item of the PHQ-9 is based on a 4-point scale that records the frequency of depressive symptoms (from 0 – "not at all" to 3 – "nearly every day"). The total PHQ-9 score ranges between 0 and 27 with higher scores corresponding with greater severity of depressive symptoms. The Cronbach's alpha for the PHQ-9 was 0.763 in our sample.

2.6. Other measures

Participants were also asked about age, gender, the level of education, as well a family history of schizophrenia and mood disorders. The level of education was categorized as follows: (1) primary (graduation from primary school only), (2) vocational (gives preparation for professional employment, based on education in basic vocational schools, basic schools or other equivalent schools, or study of crafts); (3) secondary (graduation from a general or vocational secondary school), (4) incomplete higher (a person studied at a university, but did not obtain a diploma), and (5) higher (obtaining a bachelor's, engineer's, master's, or equivalent degree; this category also includes the PhD degree). Current vocational situation [(unemployed, i.e., lack of employment), student (in the course of studies, i.e., first, second or third degree) without full-time job), employed (full-time job), rent (on social support/ security benefits)]. The lifetime problematic substance use was assessed using the screening question from the M.I.N.I. ("Have you ever taken any of these drugs, on one or more occasions, to get better, to get a good mood, or to change your current mood?"; the list of various substances has been provided) (42). Positive response to this question was coded as "problematic substance use."

2.7. Data analysis

The analysis of variance (ANOVA) was used to test the association of positive screening for PTSD and ADHD (the independent variables) with the level of reporting PLEs (the dependent variable). In the first ANOVA model, results of screening for PTSD and ADHD together with the interaction term (PTSD × ADHD screening results) were included as the only independent variables. The interaction term (PTSD × ADHD screening results) resulted in creating four groups: (1) individuals who screened positive for ADHD, but not for PTSD (the ADHD(+), PTSD(−) group), (2) individuals who screened positive for PTSD, but not for ADHD (the ADHD(−), PTSD(+) group), (3) individuals who screened positive for both ADHD and PTSD (the ADHD(+), PTSD(+) group), and (4) individuals who screened negative for ADHD and PTSD (the ADHD(−), PTSD(−) group). Next, sociodemographic characteristics, including age, gender, the level of education (higher or incomplete higher vs. other) and the current vocational situation (employed or student vs. other) were added as covariates. Finally, the lifetime history of problematic substance use and the PHQ-9 score were added to the ANOVA model as covariates. Post-hoc comparisons were performed using the Games-Howell test. The results of data analysis were interpreted as significant in case of $p < 0.05$. All analyses were carried out using the SPSS software (version 28).

3. Results

The majority of participants were females (58.3%), individuals with a higher level of education (40.7%), students (51.9%) and people

TABLE 1 Descriptive characteristics of the sample.

	The whole sample	ADHD(−), PTSD(−) [*n* = 2,222]	ADHD(−), PTSD(+) [*n* = 111]	ADHD(+), PTSD(−) [*n* = 553]	ADHD(+), PTSD(+) [*n* = 111]	*p*
Age	25.4 ± 4.9	25.6 ± 4.9	26.4 ± 5.1	24.8 ± 4.9	24.4 ± 4.7	< 0.001
Gender, males	1,250 (41.7)	990 (44.6)	41 (36.9)	196 (35.4)	23 (20.7)	< 0.001
Education						
Primary	101 (3.4)	67 (3.0)	3 (2.7)	22 (4.0)	9 (8.1)	
Vocational	46 (1.5)	33 (1.4)	0 (0)	9 (1.6)	4 (3.6)	0.003
Secondary	911 (30.4)	657 (29.6)	42 (37.9)	172 (31.1)	40 (36.0)	
Incomplete higher	720 (24.0)	519 (23.4)	26 (23.4)	152 (27.5)	23 (20.7)	
Higher	1,222 (40.7)	949 (42.6)	40 (36.0)	198 (35.8)	35 (31.6)	
Vocational situation						
Unemployed	113 (3.8)	89 (4.0)	5 (4.5)	15 (2.7)	5 (4.6)	
Student	1,557 (51.9)	1,133 (51.0)	54 (48.7)	315 (57.0)	55 (49.6)	0.180
Employed	1,300 (43.3)	984 (44.1)	49 (44.1)	216 (39.0)	51 (45.9)	
Pension	30 (1.0)	19 (0.9)	3 (2.7)	7 (1.3)	1 (0.9)	
Married or informal relationship	1999 (66.6)	1,490 (67.1)	80 (72.1)	358 (64.7)	71 (64.0)	0.386
Family history of schizophrenia	146 (4.9)	97 (4.4)	7 (6.3)	34 (6.1)	8 (7.2)	0.171
Family history of mood disorder	597 (19.9)	390 (17.6)	35 (31.5)	131 (23.7)	41 (36.9)	< 0.001
Lifetime problematic substance use	1,115 (37.2)	757 (34.1)	43 (38.7)	255 (46.1)	60 (54.0)	< 0.001
ASRS	30.2 ± 10.3	27.0 ± 8.5	30.0 ± 8.4	40.2 ± 8.7	44.1 ± 9.4	< 0.001
PLEs	22.5 ± 4.9	21.7 ± 4.1	24.1 ± 5.8	24.0 ± 5.2	28.6 ± 8.9	< 0.001
HLEs	8.9 ± 2.1	8.7 ± 1.8	9.5 ± 2.7	9.3 ± 2.1	10.9 ± 3.9	< 0.001
DLEs	13.6 ± 3.4	13.0 ± 2.9	14.6 ± 3.8	14.7 ± 3.7	17.7 ± 5.9	< 0.001
PHQ-9	9.4 ± 5.2	8.4 ± 4.6	11.2 ± 5.2	12.0 ± 5.2	15.5 ± 6.4	< 0.001

Data presented as mean ± SD or *n* (%).
ASRS, the Adult ADHD Self-Report Scale; DLEs, delusion-like experiences; HLEs, hallucination-like experiences; PHQ-9, the Patient Health Questionnaire-9; PLEs, psychotic-like experiences.

reporting being married or informal relationship (66.6%) (see Table 1 for sample characteristics). Family history of schizophrenia and mood disorders among first- or second-degree relatives was reported by 4.9 and 19.9% of the sample, respectively. A history of problematic substance use was declared by 37.2% of participants. The analysis of data from the questionnaires screening for ADHD and PTSD revealed the following findings: 1) 18.4% were screened positive for ADHD, but not for PTSD (the ADHD(+), PTSD(−) group); 2) 3.7% were screened positive for PTSD, but not for ADHD (the ADHD(−), PTSD(+) group) and 3) 3.7% were screened positive for both ADHD and PTSD (the ADHD(+), PTSD(+) group). Specific subgroups of participants differed significantly with respect to age, gender, education, rates of family history of mood disorders, self-reported lifetime problematic substance use, the level of reporting PLEs, and the PHQ-9 score.

The ANOVA demonstrated significant effects of screening for PTSD and ADHD as well as the ADHD × PTSD interaction on the level of reporting PLEs in general, including delusion-like experiences (DLEs) and hallucination-like experiences (HLEs), even after adding potential covariates to the model (Table 2). The models with all potential covariates explained 26.1, 27.0 and 11.9% of the variance in reporting PLEs, DLEs and HLEs, respectively (by means of the adjusted R^2). Post-hoc tests demonstrated that the ADHD(+), PTSD(+) individuals had significantly higher levels of reporting PLEs, DLEs and HLEs compared to the ADHD(+), PTSD(−) individuals as well as the ADHD(−), PTSD(+) individuals (Figure 1, Table 3). The ADHD(+), PTSD(−) individuals as well as the ADHD(−), PTSD(+) individuals did not differ significantly with respect to the level of reporting PLEs, DLEs, and HLEs. However, both groups of participants had significantly higher levels of reporting PLEs, DLEs, and HLEs compared to the ADHD(−), PTSD(−) individuals.

4. Discussion

The current study examined the effect of the interaction between PTSD and ADHD symptoms on the level of reporting PLEs in a non-clinical sample. We found that both groups of symptoms show an interaction effect on the level of reporting PLEs in this population. Hence, our results add to the literature showing that PTSD (18) as well as ADHD (11, 44) might be associated with reporting PLEs (11, 18, 29, 31, 44). It should be noted that our study was based on a non-clinical sample, and thus we refer to the level of reporting PLEs. However, some studies have also suggested that PLEs increase the risk of subsequent psychotic disorders (4). Given the fact that we investigated only a simple interaction effect of two potential risk factors on the level of reporting PLEs, we were

TABLE 2 The association of positive screening for ADHD and PTSD with PLEs after controlling for the effects of potential confounding factors.

Model	Independent variable	Dependent variable		
		PLEs	DLEs	HLEs
Model 1 (ADHD + PTSD)	ADHD	$F = 158.342$, $p < 0.001$	$F = 156.648$, $p < 0.001$	$F = 74.281$, $p < 0.001$
	PTSD	$F = 132.799$, $p < 0.001$	$F = 120.507$, $p < 0.001$	$F = 75.018$, $p < 0.001$
	ADHD × PTSD	$F = 16.329$, $p < 0.001$	$F = 12.461$, $p < 0.001$	$F = 12.558$, $p < 0.001$
Model 2 (ADHD + PTSD + sociodemographic characteristics)	ADHD	$F = 153.580$, $p < 0.001$	$F = 149.833$, $p < 0.001$	$F = 74.112$, $p < 0.001$
	PTSD	$F = 129.647$, $p < 0.001$	$F = 115.684$ $p < 0.001$	$F = 75.365$ $p < 0.001$
	ADHD × PTSD	$F = 15.672$, $p < 0.001$	$F = 11.589$, $p < 0.001$	$F = 12.570$, $p < 0.001$
	Age	$F = 8.012$, $p = 0.005$	$F = 7.084$, $p = 0.008$	$F = 4.741$, $p = 0.030$
	Gender	$F = 4.171$, $p = 0.041$	$F = 1.350$, $p = 0.245$	$F = 7.636$, $p = 0.006$
	Education	$F = 38.976$ $p < 0.001$	$F = 55.627$, $p < 0.001$	$F = 5.318$, $p = 0.021$
	Vocational status	$F = 2.151$, $p = 0.143$	$F = 1.361$, $p = 0.243$	$F = 2.138$, $p = 0.144$
Model 3 (ADHD + PTSD + sociodemographic characteristics + depression + substance use)	ADHD	$F = 52.723$, $p < 0.001$	$F = 49.430$, $p < 0.001$	$F = 25.376$, $p < 0.001$
	PTSD	$F = 65.350$, $p < 0.001$	$F = 54.156$, $p < 0.001$	$F = 39.746$, $p < 0.001$
	ADHD × PTSD	$F = 12.808$, $p < 0.001$	$F = 8.826$, $p = 0.003$	$F = 10.354$, $p = 0.001$
	Age	$F = 10.063$, $p < 0.001$	$F = 8.984$, $p = 0.003$	$F = 5.343$, $p = 0.021$
	Gender	$F = 13.752$, $p < 0.001$	$F = 7.846$, $p = 0.005$	$F = 14.036$, $p < 0.001$
	Education	$F = 32.333$, $p < 0.001$	$F = 48.866$, $p < 0.001$	$F = 3.022$, $p = 0.082$
	Vocational status	$F = 1.081$, $p = 0.299$	$F = 0.492$, $p = 0.483$	$F = 1.380$, $p = 0.240$
	Depression	$F = 540.563$, $p < 0.001$	$F = 572.34$, $p < 0.001$	$F = 196.916$, $p < 0.001$
	Substance use	$F = 8.755$, $p = 0.003$	$F = 5.811$, $p = 0.016$	$F = 7.443$ $p = 0.006$

ADHD, attention-deficit/hyperactivity disorder; DLEs, delusion-like experiences; HLEs, hallucination-like experiences; PLEs, psychotic-like experiences; PTSD, post-traumatic stress disorder.

not able to provide mechanisms of how these factors impact the occurrence of PLEs. More specifically, our results suggest that individuals who have ADHD and PTSD symptoms show significantly higher levels of reporting PLEs compared to the group of individuals without ADHD and PTSD symptoms. Importantly, our results were similar for both types of PLEs, i.e., DLEs and HLEs. In line with prior studies (11, 45), our results can be interpreted as showing an interaction between PTSD and ADHD symptoms on the occurrence of PLEs. Our study suggests a significant interaction between PTSD, ADHD and PLEs. According to our results, we recommend that this interaction should be further investigated.

When interpreting our results, it should be noted that our sample can be described as a well-functioning social group. We assessed both PTSD and ADHD symptoms as a continuum in a non-clinical sample. Hence, more severe states within narrow clinical criteria for respective disorders were not represented in the study. At the same time, several studies indicate the role of exposure to trauma in shaping the risk of psychosis, both in clinical (46) and non-clinical studies (47). In a clinical context, comorbidity of ADHD

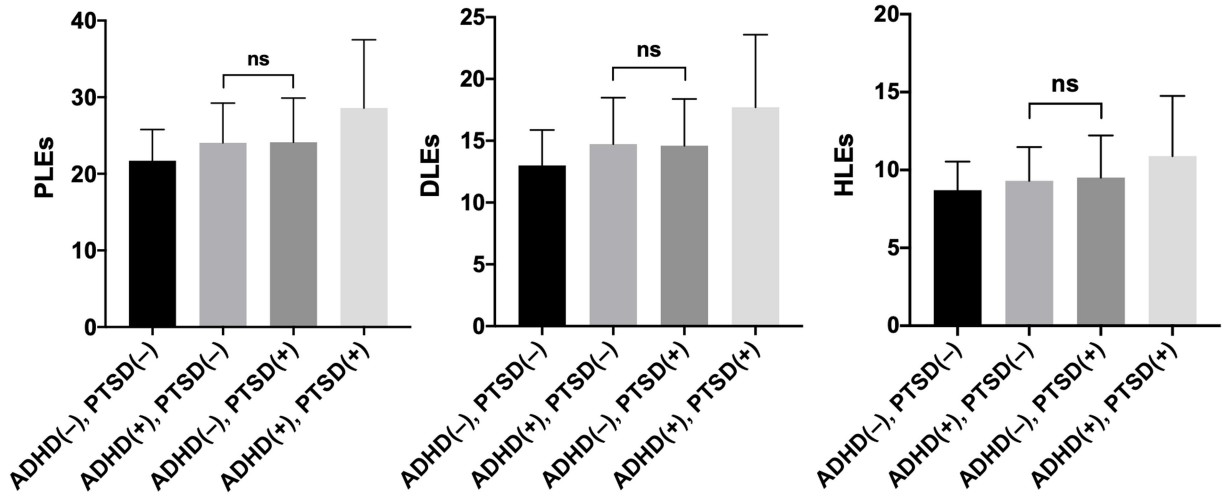

FIGURE 1
The level of psychotic-like experiences (PLEs), delusion-like experiences (DLEs) and hallucination-like experiences (HLEs) in participants with various combinations of ADHD and PTSD. Mean values and corresponding 95%CI (error bars) are shown. Post-hoc tests revealed significant differences of all bivariate comparisons, except for the comparison indicated as "ns".

TABLE 3 Results of the post-hoc comparisons.

Comparison	PLEs			DLEs			HLEs		
	Mean difference	SE	*p*	Mean difference	SE	*p*	Mean difference	SE	*p*
ADHD(−), PTSD(−) vs. ADHD(−), PTSD(+)	−2.42	0.55	< 0.001	−1.61	0.36	< 0.001	−0.81	0.26	0.012
ADHD(−), PTSD(−) vs. ADHD(+), PTSD(−)	−2.34	0.24	< 0.001	−1.74	0.17	< 0.001	−0.61	0.10	< 0.001
ADHD(−), PTSD(−) vs. ADHD(+), PTSD(+)	−6.89	0.85	< 0.001	−4.71	0.56	< 0.001	−2.19	0.37	< 0.001
ADHD(−), PTSD(+) vs. ADHD(+), PTSD(−)	0.07	0.59	0.999	−0.13	0.39	0.987	0.20	0.27	0.877
ADHD(−), PTSD(+) vs. ADHD(+), PTSD(+)	−4.48	1.01	< 0.001	−3.10	0.66	< 0.001	−1.40	0.45	0.012
ADHD(+), PTSD(−) vs. ADHD(+), PTSD(+)	−4.55	0.87	< 0.001	−2.97	0.58	< 0.001	−1.58	0.38	< 0.001

ADHD, attention-deficit/hyperactivity disorder; DLEs, delusion-like experiences; HLEs, hallucination-like experiences; PTSD, post-traumatic stress disorder.

and PTSD has already been reported in prior studies showing the impact on general functioning. For instance, results of the study by Antshel et al. (29) suggested that the co-occurrence of PTSD and ADHD in the adult group leads to a greater degree of clinical severity in terms of co-occurring psychiatric disorders and psychosocial functioning. Biederman et al. (31) demonstrated similar results showing that PTSD comorbidity in individuals with a diagnosis of ADHD leads to greater clinical severity in terms of psychiatric comorbidity and psychosocial dysfunction in adolescents. Also, it should be noted that people with ADHD are more likely to be traumatized due to their impulsivity and risky behavior (48). More specifically ADHD is associated with deficits in cognitive and emotion regulation (e.g., high impulsivity, impaired inhibitory ability) which potentially increases the risk of being exposed to the experience of behavior perceived as "socially problematic" (48). For example, during developmental periods, and later, these individuals are more likely to experience physical trauma, lower performance at work/school, more interpersonal conflict, or exposure to risky behaviors (e.g., gambling, substance abuse, or unsafe sexual behavior) that can lead to the experience of trauma or the experience of violence (48, 49).

Referring to the literature, previous studies that have examined the interaction between the occurrence of PTSD and/or ADHD and substance abuse have noted that certain risk factors for the onset of psychotic symptoms secondary to a diagnosis of PTSD and ADHD are present, including problematic substance use (28). In addition, a strong association has been shown between problematic substance use and positive screening results for PLEs (50, 51), which is also consistent with the results obtained in our study. Moreover, other studies report that in the group of individuals with ADHD, alcohol abuse can be related to a higher frequency of more severe PTSD symptoms (28). Individuals with ADHD diagnosis appear to be more likely to exposure to risky behaviors (e.g., substance abuse) which can be caused, for example, by impaired response inhibition, high impulsivity, or sensation seeking (48, 49, 52). On the other hand, problematic substance use can also be associated with prior experience of trauma (53–55). Indeed, previous studies have indicated high rates of co-occurrence of PTSD and substance abuse. Substance abuse may be, for instance, an attempt to avoid traumatic memories (including the associated distress experienced), but on the other hand, through substance abuse, individuals may expose themselves to traumatic experiences and events (55). Therefore, problematic substance use

may largely increase the chance of occurrence of PLEs in the future, especially for those diagnosed with ADHD and/or PTSD as overlapping factors. We suggest that this should be explored in more detail by future studies.

There are important limitations of the present study that need to be discussed. First, the study was based on a snowball sampling method that might be characterized by certain shortcomings related to the accuracy of assessments (56). Second, the use of a non-clinical sample with implementation of screening tools does not allow to discuss valid diagnostic constructs according to international classifications of mental disorders. This might also underlie high rates of problematic substance use found in the present sample. Third, the use of self-reports might be characterized by a recall bias. However, certain constructs assessed using self-reports in the present study might still hold some validity. For instance, there is evidence that self-reported PLEs, even if they are found to serve as false-positive observations upon clinical assessment, may still predict the development of mental disorders, including psychosis (5, 57, 58). Fourth, our sample was limited to individuals aged 18–35 years, and thus generalization of findings should be approached with caution. Finally, causal associations cannot be made as we used a cross-sectional design.

To conclude, the current study investigated the effect of the interaction between PTSD and ADHD symptoms on the risk of PLEs in a non-clinical sample. The results from our study show that both PTSD and ADHD symptoms might have an interaction effect on the level of reporting PLEs. Our results also suggest that individuals who have both PTSD and ADHD symptoms have higher levels of PLEs compared to individuals without these symptoms. This observation highlights the importance of investigating the interaction between PTSD and ADHD on the occurrence of PLEs. Future studies need to investigate our findings in longitudinal cohorts and clinical populations in order to provide specific recommendations for clinical practice. In case of replication within clinical cohorts, findings from the present study would raise awareness about the importance of PTSD and ADHD comorbidity in clinical practice. Also, the findings might provide grounds for investigating the most effective therapeutic interventions in this clinical population.

Data availability statement

The datasets presented in this study can be found in online repositories. The names of the repository/repositories and accession number(s) can be found at: https://osf.io/8ju6b.

Ethics statement

The studies involving humans were approved by the study was approved by the Ethics Committees at the Institute of Psychology (Polish Academy of Sciences in Warsaw, Poland, approval number: 16/VII/2022), Wroclaw Medical University (Wroclaw, Poland, approval number: 129/2022) and Pomeranian Medical University (Szczecin, Poland, approval number: KB-006/25/2022). The studies were conducted in accordance with the local legislation and institutional requirements. The participants provided their written informed consent to participate in this study.

Author contributions

HG and JK: writing – original draft. ŁG and JS: writing – review and editing and conceptualization. BM: conceptualization, funding acquisition, formal analysis, writing – original draft, and writing – review and editing. All authors contributed to the article and approved the submitted version.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher's note

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7.2. Publikacja 2

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Article

Psychotic-like Experiences and Underlying Mechanisms: An Integrative Model of ADHD Symptoms, Rumination, Negative Affect, and Trauma Experience

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Abstract: Background: Psychotic-like experiences (PLEs) are low-intensity subclinical phenomena, often transient in nature. The etiology of PLEs primarily involves neurodevelopmental changes, trauma exposure, and maladaptive coping styles. Attention-Deficit/Hyperactivity Disorder (ADHD) is considered to be one of the factors that increase the risk of future psychosis. Furthermore, ADHD symptoms predict a heightened incidence of traumatic experiences, ruminative thoughts, and negative affect (NA). This present study examines whether rumination and NA mediate the relationship between ADHD symptoms and PLEs and whether trauma experiences moderate these pathways. **Methods:** A total of 188 participants (72% female) aged 18–35 completed questionnaires assessing ADHD symptoms and traumatic experiences and took part in a seven-day experience sampling method (ESM) procedure, completing ratings of PLEs experiences, the intensity of ruminations, and NA. **Results:** Correlation analysis showed significant relationships between all tested variables. Serial mediation analysis revealed a significant indirect effect of rumination and NA in the link between ADHD symptoms and PLEs. There was no significant impact of trauma experience in this relationship. **Conclusions:** Our study underscores the important role of rumination and NA in the co-development of ADHD symptoms and PLEs. Future research should consider investigating the intra-individual dynamics of ADHD and trauma using ecologically valid research methods in the context of PLEs to better understand these complex relationships.

Keywords: psychosis; abuse; neurodevelopmental changes; negative repetitive thinking; negative emotions

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1. Introduction

Psychotic-like experiences (PLEs) refer to hallucinations or delusions defined as subclinical phenomena, encompassing both delusion-like experiences and perceptual abnormalities [1,2] with a limited intensity, transience, or functional impairment. They are situated at the lower end of the psychosis continuum [1], representing a category of low-grade positive psychotic symptoms that do not meet the criteria for a psychosis spectrum disorder [3,4]. Because PLEs are far more common than severe psychotic symptoms in the clinical context [5] and affect approximately 5–7% of adults in the general population [6], their impact and associated costs may be still important [7]. Individuals with PLEs report more distress [8], more depressive symptoms [9], lower general functioning [8], and increased suicide risk [10,11], among others, compared to healthy controls. While PLEs typically resolve naturally over time in most individuals (~80%), a significant minority

(∼7%) progresses into full-blown psychosis [12]. Despite a growing interest in PLEs over the past two decades, the mechanisms underlying hallucinations/delusions remain poorly understood. Research suggests that neurodevelopmental changes [13] and the experience of (early childhood) trauma are potential mechanisms of the risk of psychosis and its different stages (e.g., PLEs, full-blown psychosis; see [14–16]. Also, the use of maladaptive coping strategies, such as ruminative thought and the experience of negative affect, have been shown to increase the risk of PLEs occurrence [17,18]. An integrative theoretical model that links early developmental changes, trauma experience, and coping strategies in the context of PLEs remains necessary. Therefore, this present study focuses on the role of Attention-Deficit/Hyperactivity Disorder (ADHD) symptoms on PLE occurrences, as ADHD is a proxy for neurodevelopmental disadvantage (given the wide variety of associated neurological disorders; see [19]. The objective of this study is to test whether the association between the severity of ADHD symptoms and PLEs is mediated by the potential role of rumination and negative affect in daily life, and if the reported traumatic experiences strengthen this relationship. Hence, we examine whether (a) ADHD symptoms predict more PLEs, whether (b) rumination and negative affect mediate this association, and whether (c) the traumatic experiences over the life course moderate these processes.

1.1. The Role of ADHD and Trauma in the Context of PLEs

The sociodevelopmental–cognitive model of psychosis posits that schizophrenia is a neurodevelopmental disorder [20], which may explain the covariance with other neurodevelopmental disorders, especially Attention-Deficit/Hyperactivity Disorder (ADHD; [21,22]). ADHD symptoms are strongly associated with PLEs [23], and children diagnosed with ADHD exhibit a fivefold increased risk of developing psychotic disorders [24,25]. A systematic review of 15 studies and a meta-analysis of 12 studies conducted by Nourredine et al. (2021) [25] showed that up to 13% of patients diagnosed with schizophrenia were also diagnosed with ADHD, while adult ADHD symptom severity was associated with more paranoid thoughts and auditory hallucinations [26]. Finally, around 80% of schizophrenia patients exhibit a progressive cognitive decline from early adolescence, leading to functional disability and secondary (indirect) illness costs [27,28].

The sociodevelopmental–cognitive model of psychosis emphasizes the role of adverse childhood experiences on gene expression and dopaminergic dysregulation [13], resulting in abnormal stimulus processing, including the salience dysregulation that marks the psychosis spectrum [29,30]. Such neurodevelopmental changes arise from interactions between the environment, genes, and traumatic/adverse experiences, and dopaminergic dysregulation may result in abnormal stimulus processing, paranoid interpretations, and psychotic experiences, a process exacerbated by dysfunctional cognitive schemas [13]. Consequently, individuals with maladaptive cognitive biases are more susceptible to experiencing stress, paranoia, and/or hallucinatory experiences, potentially perpetuating or worsening psychotic beliefs.

Neurodevelopmental theories are supported by the threefold increase in the risk of developing psychotic disorders in individuals who experienced childhood trauma [31]. Childhood trauma may intensify the impact of social stress during adolescence and young adulthood when the risk of developing psychosis increases and the hypothalamic–pituitary–adrenal (HPA) axis becomes overactive or dysregulated [32]. Consequently, individuals at risk of psychosis demonstrate a heightened sensitivity to daily stressors and alterations in HPA functioning [33,34]. Furthermore, research has demonstrated that trauma is associated with an elevated risk of psychosis through the formation of cognitive biases [9,35], which in turn may render victimized people more susceptible to stress, paranoia, and/or hallucinatory experiences, and perpetuate or even exacerbate their psychotic beliefs.

1.2. The Role of Rumination Thoughts and Negative Affect in the Context of PLEs

Emotional dysregulation is a key symptom of ADHD [36] and is evidenced by deficits in self-regulation [37,38]. Self-regulation deficits are evident in low frustration tolerance and

high mood lability [39]. Indeed, individuals diagnosed with ADHD are more likely to report negative emotions, including anger, irritability, and frustration, and to ruminate more [40–42], thus causing persistent thoughts and mental images focusing on negative emotions and symptoms, pondering their causes, meanings, and consequences [43,44]. These symptoms have recently been proposed as one of the primary symptoms of ADHD [45]. Additionally, ADHD-related symptoms indirectly exacerbate depressive and anxiety symptoms through ruminative thoughts, significantly impacting an individual's functionality [43].

The experience of distress or anxiety can elicit intrusive thoughts and more negative emotions [46]. Rumination frequency predicts anxiety and depression symptom severity [44,47] and more negative emotional outcomes and distress from psychosis symptoms [48,49]. Negative emotions play a pivotal role in the emergence of psychotic content and/or symptoms and underlie all emotional and psychotic disorders [50,51]. Such negative emotions are thought to be induced by meta-cognitions ("thinking about thinking"), meta-beliefs, and poor coping that amplify psychological distress [52]. Wells and Matthew's model (1994, 1996) [53,54] on metacognitions focuses on maladaptive coping strategies and metacognitive beliefs that contribute to psychological distress by inducing negative emotions. For example, ruminative thinking, a maladaptive coping mechanism, likely plays an important role in the distressing experience of psychosis. A review reported more rumination in patients with psychosis than in healthy controls [18] and poorer self-regulatory strategies among schizophrenia patients than in the controls [55]. Furthermore, rumination or repetitive negative thinking is more common among adults who have lived through childhood adversity [56–58].

Ruminative thoughts can focus attention on threatening stimuli and create false associations between unrelated events and thoughts (salience dysregulation), which in turn perpetuates ruminative thoughts associated with delusions and more negative emotions [59]. Additionally, re-experiencing traumatic events and using maladaptive coping strategies to regulate emotions may contribute to experiencing auditory or visual hallucinations or persecutory imagery [60–62].

Despite the existence of established links between ADHD symptoms, ruminations, negative affect, trauma, and psychotic-like experiences, their interactions and codependencies remain unclear. Therefore, this present study aims to address this gap in the literature by examining whether ruminations and negative affect mediate the link between ADHD and PLEs and whether the trauma experience moderates these mediation pathways.

1.3. This Study

This present study is focused on the role of ADHD symptoms in the occurrence of PLEs. Our aim is to determine whether the relationship between ADHD and PLEs is mediated by rumination and negative affect experienced in daily life and whether this connection is exacerbated by reported traumatic events. Hence, we examine three key questions: (a) whether ADHD symptoms predict more PLEs, (b) whether the ADHD–PLE relationship is mediated by rumination and negative affect, and (c) whether these effects are moderated by differential exposure to traumatic events through life. This resulted in the following hypotheses:

H1. *ADHD symptoms predict more PLEs.*

H2. *Rumination and negative affect mediate the relationship between ADHD symptoms and PLEs (serial mediation).*

H3. *The link between ADHD symptoms and PLEs (**H1** + **H2**) is moderated by exposure to traumatic life events, such that the positive relationship between ADHD symptoms and PLEs through rumination and negative affect will become stronger in people who developed under conditions of higher levels of traumatic experience (moderated serial mediation).*

2. Methods and Materials

2.1. Participants

Data were derived from 188 participants (including participants in the control group N = 89 and participants in the experimental group N = 99) of a community sample recruited in three Polish cities (i.e., Warsaw, Wroclaw, Szczecin) for an ongoing study into epigenetic processes and associations with momentary stressors and psychotic-like experiences.

Included participants were (1) aged 18–35 years, (2) without a history of psychiatric treatment, and (3) with psychotic-like symptoms (PLEs) based on 16 items presented in the Recruitment Phase below, a global rating of 3–4 points for reference imagery and/or suspicion and persecutory imagery *(e.g., "I often have the feeling that other people are against me.", "I was worried about being stalked.", "I spend my time thinking that my friends are gossiping about me.")*, and/or a global score of 3–4 points for hallucinations *(e.g., "I hear a voice speaking my thoughts aloud.", "I can hear things that other people can't hear, such as the voices of people who are whispering or talking.", "I can see things that other people can't see.")*. Participants who scored between 48 and 64 points were assigned to the experimental group, and those with 0–32 points became the control group (a global rating of 0–2 points for reference imagery and/or suspicion and persecutory imagery and/or a global score of 0–2 points for hallucinations). Exclusion criteria included (1) a lifetime history of psychiatric treatment, (2) a current episode of major depressive disorder (MDD), and/or (3) a diagnosis of substance use disorder (other than nicotine dependence).

The study protocol was approved by the Ethics Committees of the Institute of Psychology (Polish Academy of Sciences, Warsaw, Poland, approval number: 16/VII/2022), Wroclaw Medical University (Wroclaw, Poland, approval number: 129/2022), and Pomeranian Medical University (Szczecin, Poland, approval number: KB-006/25/2022).

2.2. Materials

The participants were required to complete a seven-day experience-sampling method (ESM) procedure, a clinical interview comprising a comprehensive Mini-International Neuropsychiatric Interview (M.I.N.I.), and the Comprehensive Assessment of At-Risk Mental States (CAARMS) (see Supplementary Tables S1 and S2 for prevalence scores on M.I.N.I. and CAARMS scales in the study sample). Furthermore, the participants were required to complete a series of questionnaires, including the Traumatic Experience Checklist (TEC; [63]), all described in detail below.

Demographics. Participants were asked to self-report their age, gender, educational level, and family history of psychiatric illness (i.e., depression and schizophrenia spectrum disorders).

The Comprehensive Assessment of At-Risk Mental States (CAARMS; [64,65]) is a semi-structured interview to determine ultra-high risk (UHR) status and measure a range of subthreshold symptoms associated with the prodromal phase of psychotic disorders. The CAARMS provides an intensity and frequency score for each item. It consists of 27 items grouped into seven scales: positive symptoms, cognitive changes, emotional disturbance, negative symptoms, behavioral changes, motor/physical changes, and general psychopathology. The scores for each of the subscales range from 0 to 6. The CAARMS was selected for this study due to its demonstrated efficacy in identifying individuals at ultra-high risk (UHR) for psychosis, exhibiting superior predictive accuracy and flexibility compared to other diagnostic instruments, as evidenced by the findings of Wang et al. (2022) [66].

The Mini-International Neuropsychiatric Interview (M.I.N.I.; [67]) was developed to assess the diagnoses of psychiatric patients according to DSM-IV and ICD-10 criteria. In order to verify the diagnoses, the gold standard was applied through the use of a structured diagnostic interview M.I.N.I. All the items in the questionnaire are answered with a 'Yes' or 'No' answer, starting with the screening questions and ending with the diagnostic blocks to check whether a patient meets the diagnostic criteria. In this study, the Polish version of M.I.N.I. Plus 5.0.0 was used, including all the modules.

The Adult ADHD Self-Report Scale (ASRS; [68]) is an 18-item self-report screening scale for adult Attention-Deficit/Hyperactivity Disorder (ADHD). Responses range between 0—"never" and 4—"very often". The total score ranges between 0 and 72. The Symptom Checklist is an instrument consisting of the eighteen DSM-IV-TR criteria. Six of the eighteen questions from part A (1–6) are the most predictive of symptoms consistent with ADHD. For part A, points are summed for a range of 0–24, with a cut-point $\geq$ 14 for ADHD. The total score can be classified into four categories: 0–9 is low negative, 10–13 is high negative, 14–17 is low positive range, and 18–24 is high positive range. The remaining twelve questions (7–18) are included in part B of the Symptom Checklist. The Cronbach's alpha of the ASRS was 0.92 in our sample. The Adult ADHD Self-Report Scale (ASRS) was selected on the basis of its demonstrated reliability and validity in large-scale, cross-cultural studies, which have effectively captured ADHD symptoms in diverse populations (see [69]).

The Traumatic Experiences Checklist (TEC; [63]) is a 29-item self-report questionnaire addressing potentially traumatizing serious emotional events categorized across three types of trauma, referred to by six subscales: (1) emotional trauma captures emotional neglect and/or emotional abuse in various social settings with six items (*e.g., "Emotional neglect (e.g., being left alone, not shown enough affection) by parents or siblings. Has this happened to you?"*); (2) sexual trauma captures sexual harassment and/or sexual abuse in various social settings with six items (*"Sexual harassment (acts of a sexual nature in which there is NO physical contact) by parents or siblings. Has this happened to you?"*); and (3) bodily threat, which captures physical abuse in various social settings and intentional threat to one's life, bizarre punishment, or intense pain, with six items (*e.g., "Physical abuse (e.g., being hit, bullied or physically hurt) by parents or siblings. Has this happened to you?"*). Scale scores for emotional, sexual, and bodily trauma are calculated by summing the presence scores for the relevant items, with six items each for emotional and sexual trauma and bodily threat. The Cronbach's alpha of the TEC total was 0.74 in our sample, and the subscales were as follows: emotional trauma 0.66, sexual trauma 0.31, and bodily trauma 0.39. The Trauma Exposure Checklist (TEC) was selected on the grounds of its status as a valid and reliable tool for assessing trauma exposure and its associated risk and protective factors. This is evidenced by its robust factorial structure and psychometric properties, as demonstrated in recent studies (e.g., [70]). The experience sampling method (ESM) questionnaires covered various domains of psychotic-like experiences (PLEs). All items included in the statements are presented in Table 1. While most of the items used in the questionnaires were adopted from previous studies [71–73], we also included items adapted from the PQ-16 [74] to capture the presence of PLEs. In the ESM procedure, participants also responded to questions regarding ruminative thoughts and negative affect (NA), as well as to additional questions that were not pertinent to this present study (see Supplementary Table S3). However, only items related to PLEs, ruminations, and NA are analyzed here. The order of items was not randomized.

Table 1. Concepts used in this present study and their definitions.

Domain	#	Scale	Items	CA
PLEs	8			0.85
Hallucination-like	3	1–7	"My thoughts are so strong that I can almost hear them". "I hear things that aren't really there". "I see things that aren't really there".	0.66
Delusion-like	5	1–7	"I have the sense that some person or force is around me, although I can't see anyone". "I see special meanings in advertisements, shop windows, or in the way things are arranged around me". "I am confused whether something I experienced was real or imaginary". "My thoughts are influenced by others". "I can't get these thoughts out of my head".	0.78

Table 1. *Cont.*

Domain	#	Scale	Items	CA
Ruminative thought	1	1–7	"At the moment, I feel that I am stuck on negative thoughts and can't get away from them".	-
Negative affect	5	1–7	"I feel anxious". "I feel down". "I feel lonely". "I feel insecure". "I feel annoyed".	0.86

Note. All scales ran from "not at all" (1) to "very much" (7). # = number of items. CA = Cronbach's alpha. PLEs = Psychotic-like experiences, which comprise hallucinations and delusions experiences.

2.3. Procedure

2.3.1. Phase I

The recruitment of study participants began with an extensive screening process using snowball sampling via social media and survey websites. We recruited 4203 participants for a web-based survey, who completed a 16-item screener to identify the presence of psychotic-like experiences (PLEs) in the previous month (between April and October 2022) derived from the following questionnaires: (1) the Revised Hallucination Scale (RHS; three items, [75–77]); (2) the Revised Green Paranoid Thoughts Scale (GPTS; four items, [78]); and (3) the Prodromal Questionnaire-16 (PQ-16; nine items, [74]). Participants were also asked to complete the 14 screening questions of the Mini-International Neuropsychiatric Interview (M.I.N.I.; [67]) for depression, mania, panic attacks, anxiety, agoraphobia, social phobia, obsessive–compulsive disorder (OCD), post-traumatic stress disorder (PTSD), and alcohol/drug abuse. Participants were also asked to fulfill the Adult Attention-Deficit/Hyperactivity Disorder (ADHD) Self-Report Scale (ASRS; [68]).

2.3.2. Phase II

The second screening step included telephone interviews with participants with the highest PLEs scores, constituting the experimental group, and participants with the lowest PLEs scores, referred to as the control group. To provide clinical validation of the presence of current psychotic-like experiences, selected questions from the Comprehensive Assessment of At-Risk Mental States (CAARMS; [64,65]) were used, examining the following symptoms: (1) ideas of reference (*e.g., "Have you felt that things that were happening around you had a special meaning, or that people were trying to give you messages?"*), (2) suspiciousness and persecutory ideas (*e.g., "Do you feel like people have been talking about you, laughing at you, or watching you?"*), and (3) hallucinations (*e.g., "Do you ever hear things that other people seem not to, such as sounds, or voices?"*). Furthermore, individuals who tested positive for major depressive disorder (MDD) and substance use disorders underwent additional testing using the Mini-International Neuropsychiatric Interview (M.I.N.I.). Those who met the criteria for any of the aforementioned disorders were disqualified from participating in this study.

2.3.3. Phase III

Participants who met all inclusion criteria were invited to a face-to-face diagnostic interview, were informed about the experience sampling method (ESM) procedure, and were provided with all necessary materials. Prior to the commencement of the interview and study procedure, participants were required to complete a series of documents, including an informed consent form. This document detailed the procedures and stages involved in this study, as well as the option to withdraw from the research at any point without providing a reason. It also outlined the remuneration offered, the requirements for obtaining it, and information regarding the processing of personal data, the anonymity of the research, and collective data analysis.

2.3.4. Experience Sampling Assessment

The ESM procedure was conducted over seven days, with six assessments per day (42 assessments in total) administered between 9 a.m. and 10 p.m. with a minimum 60 min gap between prompts (a stratified randomization strategy) via the MovisensXS application on provided smartphones, Version 1.5.23 (Movisens GmbH, Karlsruhe, Germany; https://www.movisens.com, accessed on 28 April 2024). Prior to the beginning of the protocol, participants were provided with detailed instructions and a handout with all relevant information. Participants were informed that they were required to respond to each beep directly or delay it for up to 15 min. Failure to comply with these instructions resulted in the survey being considered incomplete, which accounted for a total of 3.6% of all surveys. In order to encourage participation in the study, respondents who achieved a response rate of at least 80% were awarded a prize of EUR 250. The threshold was set in accordance with recommendations identified in previous studies in the field of experiential sampling methodology [79,80]. Accordingly, the decision was taken to set the minimum response rate in the ESM to 80% in order to ensure the reliability of the collected data, while allowing for the possibility of unforeseen occurrences. Consequently, the requirements were reduced from 100% to the aforementioned 80%. Participants who encountered technical difficulties or required further clarification were encouraged to establish contact with the experimenter via email or telephone. Once the experience-sampling period was complete, participants were invited to the final face-to-face meeting with the experimenter to receive their duly compensated remuneration. Participants were asked if any unusual events had occurred during the previous week and, if necessary, provided information about the availability of psychological support.

2.4. Analysis

Statistical analyses were performed in SPSS 29. The analyses include all participants included in this study, without a distinction between the groups. It should be noted that the groups in this study were recruited for the main project. Nevertheless, the characteristics of the study groups are presented in the following section (see Table 2, Descriptives) and in the descriptive characteristics of the study subgroups (see Supplementary Tables S6 and S7).

Table 2. Descriptives.

Variable		N	%	Mean	SD	Min	Max	Range
Group	Experimental	99	52.7%					
	Control	89	47.3%					
Gender	Men	52	27.7%					
	Women	136	72.3%					
Age				25.21	5.18	18	35	18–35
Education level								
Primary		6	3.2%					
Secondary		79	42%					
Vocational		1	0.5%					
Higher		102	54.3%					
ADHD (ASRS total) [1]				34.05	14.36	0	65	0–72
Part A [2]				10.80	5.37	0	23	0–24
Part B [3]				23.24	9.88	0	46	0–48
Psychotic experiences [4,5]				11.92	5.58	6.48	36.36	
Rumination [4,6]				2.47	1.15	1	6.15	
Negative affect [4,7]				9.64	4.39	4.20	23.20	
Trauma measurement (TEC total) [8]				4.98	3.85	0	17	0–29
Emotional trauma [9]				1.85	1.61	0	6	0–6
Sexual trauma [9]				0.37	0.65	0	2	0–6
Bodily threat [9]				0.96	1.05	0	5	0–6

Note. [1] ADHD was assessed with the Adult Self-Report (ASRS). [2] Part A = Predictive list of ADHD symptoms (ASRS). [3] Part B = Control list of ADHD symptoms (ASRS). [4] As assessed with the experience sampling method (ESM), see the Methods section for details. [5] The average of Psychotic-like experiences (PLEs) over one week. [6] The average of rumination scores over one week. [7] The average of negative affect (NA) scores over one week. [8] Trauma measurement was assessed with the Traumatic Experiences Checklist (TEC). [9] Trauma measurement (TEC) subscales. N = Number of participants. SD = Standard deviation. Min = Minimum. Max = Maximum.

Pearson's correlation analyses were conducted to investigate the relationships between ADHD, PLEs, ruminations, negative affect, general trauma experience, and three subtypes of trauma (emotional, sexual, and bodily threat trauma). We use correlations (r) and betas (β) as effect size indices to express our results, which we regard to be small if they are between 0.10 and 0.19, moderate between 0.20 and 0.29, and large from 0.30 based on normative effect sizes that are commonly found [81–83]. For this typical effect size of around $r = 0.20$, one study needs at least 150 participants but, ideally, up to 250 participants to reduce estimation error in correlations [84]. The medium effect size was based on Cohen's (1988) [85] notion that it should be noticeable to the naked eye of a careful observer. We prefer practical significance (effect sizes) over statistical significance (p-values), which means we adhere to conventional p-values unadjusted for multiple testing) [85,86] and focus on effects significant at $p < 0.05$.

The strong correlations between several independent variables, including PLEs, rumination, and negative affect (ranging from 0.72 to 0.89, see Table 3), could indicate multicollinearity, which can indicate that variables are close to perfect linear combinations of one another, resulting in potentially unstable regression estimates and, thus, wide standard errors and unreliable significance tests [87]. When we examined the Variance Inflation Factors (VIFs, see Supplementary Table S4), they indicated salient but moderate inflation (all VIFs < 4.8, but close to 5, see [87]). When we examined the condition index (CI) of our correlation matrix (a function of their eigenvalue collinearity), however, there was no indication of variable collinearity problems (CI = 3.96 for the fourth and 4.81 for the fifth dimension; see Table S5 and following [87]). We, therefore, have reasonable trust in our multiple regression models estimated with robust confidence intervals, as described below.

Table 3. Correlations between study variables (N = 188).

#	Variable	Mean	SD	1.	2.	3.	4.	5.	6.	7.
1.	PLEs [1]	11.92	5.58							
2.	ADHD [2]	34.05	14.36	0.41						
3.	Ruminations [3]	2.47	1.15	0.67	0.53					
4.	NA [4]	9.64	4.39	0.69	0.56	0.89				
5.	Trauma [5]	4.98	3.85	0.37	0.31	0.33	0.30			
6.	Emotional trauma [6]	1.85	1.61	0.33	0.36	0.36	0.35	0.84		
7.	Sexual trauma [7]	0.37	0.65	0.36	0.23	0.27	0.31	0.60	0.41	
8.	Bodily threat [8]	0.96	1.05	0.26	0.24	0.26	0.19	0.75	0.51	0.34

Note. All correlations were significant at $p < 0.001$. N = Number of participants. SD = Standard deviation. [1] PLEs = Psychotic-like experiences (assessed with the 1-week aggregated ESM data). [2] ADHD = Attention-Deficit/Hyperactivity Disorder (assessed with the Adult ADHD Self-Report Scale; ASRS). [3] Ruminations (assessed with the 1-week aggregated ESM data). [4] NA = Negative affect (assessed with the 1-week aggregated ESM data). [5] Trauma experience is treated as a total score from TEC, and [6,7,8] are treated as subscales of trauma: [6] Emotional trauma captures emotional neglect and/or emotional abuse; [7] Sexual trauma captures sexual harassment and/or sexual abuse; [8] Bodily threat captures physical abuse in various social settings and intentional threats to one's life, bizarre punishment, or intense pain.

Student's t-test for independent samples was conducted to assess the group differences in outcomes between the female (N = 136) and male (N = 52) participants and for the experimental (N = 99) and control groups (N = 89) (see Supplementary Tables S6 and S7 for descriptive characteristics of the subgroups).

To test our hypotheses, serial mediation and moderated serial mediation analyses were conducted using the PROCESS macro for SPSS [88]. The dataset included variables from both ESM measures (PLEs, rumination, and negative affect) and self-report questionnaires (ADHD and trauma). The ESM data were aggregated in order to align with the other variables included in the model, which were measured at a single time point. This aggregation process involved averaging the measurements, including those obtained via ESM (negative affect, rumination, PLEs) and those collected at a single time point (ADHD symptoms, trauma) to create a single score that reflects the participants' experiences over the entire time frame. This approach enabled the analysis of all variables, including those

measured via ESM and those collected at a single time point, on an equivalent level for comparative and inferential purposes. For the ESM data, the results of those who achieved a response rate of at least 80% were included, and missing responses were not included in the calculated average of PLEs, negative affect, and rumination.

First, serial mediation analysis was used to investigate the role of ruminative thoughts (M_1) and negative affect (M_2) in the relationship before ADHD (X) and PLEs (Y), using Model 6 in the SPSS PROCESS macro. Secondly, general trauma experience scores and the three subscales of trauma separately, namely, emotional, sexual, and bodily trauma, were added as moderators of the mediation model, resulting in a moderated serial mediation analysis (using Model 85 in the PROCESS macro) to test whether trauma experience (W) would moderate the role of mediator ruminations (M_1) and negative affect (M_2) in the relationship between ADHD (X) and PLEs (Y). Third, we fit similar models separately for three subscales of trauma to explore whether any particular type of traumatic experience played a unique role in the model. We tested our hypothesis using 95% bootstrapped confidence intervals (CIs) in PROCESS, generated with 5000 bootstrapped samples.

3. Results

The study sample consisted of 72.3% female participants aged 18–35 (M = 25.21, SD = 5.18). Of these, 54.3% had completed higher education, while 42% had completed secondary education; see Table 2. The results for individual questionnaires and sample characteristics are presented in Table 2. The M.I.N.I. diagnoses are provided in Supplementary Table S1, and the CAARMS diagnoses are in Supplementary Table S2. Our sample comprised 61 individuals who met the study criteria of having an initial ADHD diagnosis according to the ASRS part A cut-off point, of which 53 individuals were in the experimental group. According to the M.I.N.I. diagnosis, 25 participants (13% total) met the criteria for ADHD (see Supplementary Table S1). The results of Student's *t*-test for independent samples (see Supplementary Table S6) indicated that women exhibited significantly higher scores in ruminative thoughts over the week ($p < 0.05$, d = 0.39), higher values in negative affect over the week ($p < 0.05$, d = 0.46), and had higher total scores on the trauma questionnaire ($p < 0.05$, d = 0.35), with a particularly notable effect observed in one of the subscales, sexual trauma ($p < 0.001$, d = 0.65). With regards to the study group comparison (High × Low) (see Supplementary Table S7), the results indicated that the participants in the experimental group were younger ($p < 0.001$, d = −0.54) and exhibited significantly higher scores on the ASRS questionnaire for ADHD symptoms ($p < 0.001$, d = 1.76), as well as in part A for the predictive list of ADHD symptoms ($p < 0.001$, d = 1.40) and in part B for the control list of ADHD symptoms ($p < 0.001$, d = 1.70). Furthermore, the results indicated significantly higher scores in the experimental group in the weekly average of PLEs experiences ($p < 0.001$, d = 1.39), higher values in ruminative thoughts over the week ($p < 0.001$, d = 1.23) and negative affect ($p < 0.001$, d = 1.51), and higher scores on the total trauma experience questionnaire ($p < 0.001$, d = 0.95), and all its subscales, including emotional trauma ($p < 0.001$, d = 0.95), sexual trauma ($p < 0.001$, d = 0.69), and bodily threat ($p < 0.001$, d = 0.5).

3.1. Power Analysis

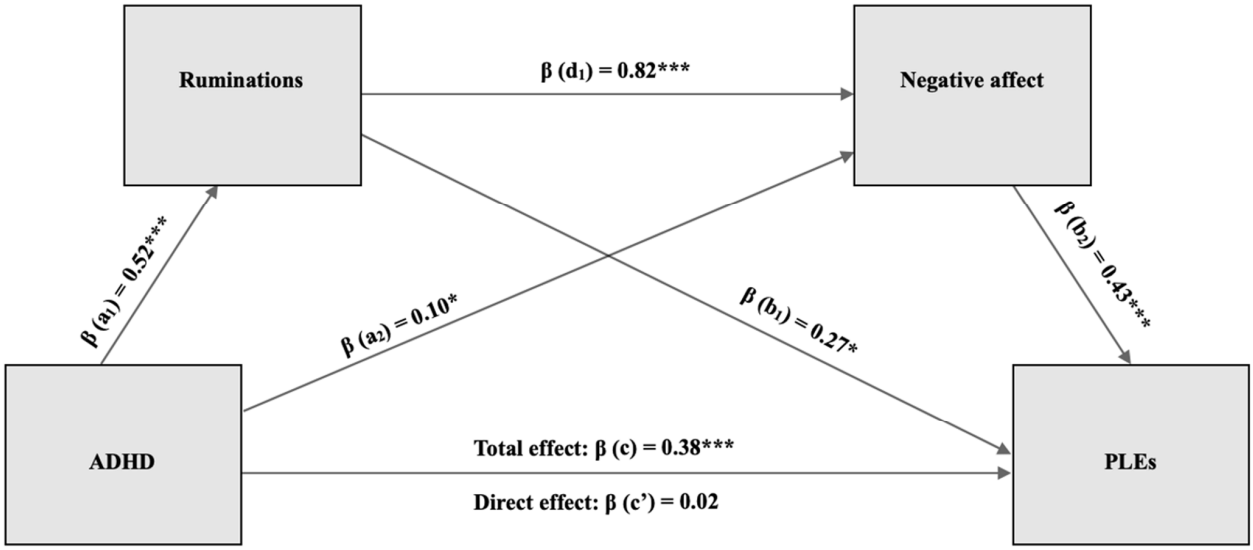
The post hoc power analysis specified a sample size of 188 and an alpha level of $p < 0.05$, considering an effect size (f^2) of 0.15 in the G*Power3 program [89], revealing a power of >0.99. The sample size was adequate for the study to detect medium-sized effects.

3.2. Correlations Analysis

We calculated correlations between the study variables, which are presented in Table 3. All variables included in the model showed a significant correlation (0.19–0.89, $p < 0.001$). Both ADHD and PLEs showed stronger covariance with rumination and negative affect (0.7) than trauma experiences (0.3).

3.3. Serial Mediation

The hypotheses that ADHD symptoms predict PLEs (**H1**) in part through the role of ruminative thoughts and negative affect (**H2**) were tested with a serial mediation analysis using Model 6 in the PROCESS macro for SPSS ([90]; see Figure 1). The results showed that the standardized total effect of ADHD on PLEs was significantly different from zero (β = 0.38, $p < 0.001$, 95% CI = 0.24 to 0.51). The direct effect of ADHD on PLEs was not significant, indicating that mediation is indirect only [91] (β = 0.02, $p = 0.81$, 95% CI = -0.11 to 0.14). The total indirect effect of ADHD on PLEs was found to be significant (β = 0.36, 95% CI = 0.25 to 0.50), as well as through NA only (β = 0.04, 95% CI = 0.003 to 0.11) and through ruminations and negative emotions (β = 0.18, 95% CI = 0.05 to 0.34). The indirect effect through ruminations only was not significant (β = 0.14, 95% CI = −0.01 to 0.31). The total effect explained 18.7% of the variance in PLEs, and the serial mediation model explained 49.5% of the variance in PLEs. Gender and age were added to the model as covariates but turned out not to be significant predictors.



Note. (*) p < 0.05 **(***)** p < 0.001

Figure 1. Serial mediation model (Model 6).

3.4. Moderated Serial Mediation

Subsequently, to test our last hypothesis (**H3**) that traumatic experiences would moderate the mediation effects, we conducted a moderated serial mediation (see Figure 1) using Model 85 in the PROCESS macro for SPSS [90], in which we added trauma experience as a moderator (W) to our model. This moderated serial mediation pathway model did not support the idea that participants with ADHD who reported more adverse (traumatic) experiences were more vulnerable to PLEs because they ruminate more and report more negative emotions. The trauma scale did not moderate any of the mediation pathways (see Table 4). Furthermore, none of the analyzed subscales of trauma (emotional, sexual, bodily) significantly moderated the serial mediation relationship between ADHD and PLEs (see Table 4). When gender and age were added to these models as covariates, they were not significant predictors in any of them.

Table 4. Moderated mediation model (Model 85) results.

Moderator (W)	Index of Moderated Mediation	S.E.	CI Lower 95%	CI Upper 95%
Trauma total	0.01	0.02	−0.03	0.05
Emotional trauma	0.01	0.02	−0.03	0.05
Sexual trauma	−0.02	0.03	−0.08	0.02
Bodily threat	−0.02	0.03	−0.08	0.03

Note. S.E. = Standard error. CI = Confidence interval.

4. Discussion

The primary objective of this study was to investigate the relationship between Attention-Deficit/Hyperactivity Disorder (ADHD) symptoms and psychotic-like experiences (PLEs) in a non-clinical sample. The results revealed a significant total effect of ADHD symptoms on PLEs, thus supporting **H1**. Moreover, the results confirm **H2**, namely that there is an indirect pathway between ADHD symptoms and PLEs. Additionally, we identified two key processes that link ADHD and PLEs: increased rumination and heightened negative affect. Collectively, these two processes explained 49.5% of the individual differences in the pathway from ADHD symptoms to PLEs. Finally, the findings of the moderated serial mediation analysis (**H3**) indicated that traumatic experiences do not exert an influence on the tested pathway from ADHD to PLEs. Therefore, **H3** was not confirmed.

The findings of this study are consistent with the high prevalence of ruminative thoughts observed in individuals with ADHD (e.g., [92]) and the detrimental impact of rumination on psychological well-being [93]. This present study demonstrates that ruminative thoughts and negative emotions serve as mediators for the majority of the relationship between ADHD symptoms and PLEs. Although mind wandering is a common phenomenon, there is evidence that prolonged rumination may be associated with the experience of negative emotions [94]. It is noteworthy that in individuals presenting with ADHD symptoms, rumination may serve as a distinctive symptom that prospectively indicates functional impairment [95]. Furthermore, given the high prevalence of ADHD and psychotic disorders [96] and the robust association between ADHD and ruminative thoughts, which predict delusional and hallucinatory experiences [97], our study suggests that ruminative thoughts and the experience of negative affect are important elements in the context of diagnosing and as a treatment focus for ADHD and PLEs symptoms. Furthermore, PLEs, as a part of the psychotic disorder continuum, are most closely related conceptually to a general factor or vulnerability for psychopathology (see [98]), and the affective dynamics are a key factor in virtually all mental health problems [99,100].

It is also noteworthy that the majority of participants in this study were young women between the ages of 18 and 35, representing 72.3% of the total sample. The results of Student's *t*-test indicated that, when compared to men, women in the study group exhibited a significantly higher frequency of ruminative thoughts and negative emotions. A substantial body of literature on this topic (e.g., [101–103]) indicates that women are significantly more likely than men to report higher levels of repetitive negative thinking, including ruminative thoughts, particularly at a young age [104]. Therefore, our results corroborate prior findings of elevated levels of ruminative thoughts and, consequently, negative emotions in a cohort of young women, underscoring the significance of these factors in the diagnosis of ADHD and PLEs. However, there is still a need for further data on the specific factors that are associated with an increased risk of mental illness. Therefore, the role of rumination and negative emotions as mediators between ADHD and PLEs may align with the perspective of transdiagnostic vulnerability and should be considered in future studies.

The objective of the second stage of analysis was to examine the potential influence of traumatic experiences on the pathway connecting ADHD symptoms to PLEs through ruminations and NA. The results of the moderated serial mediation analysis did not support the proposed hypothesis (**H3**). Moreover, none of the three subscales of trauma, including

emotional, sexual, or bodily traumatic experiences, moderated the hypothesized model. Nevertheless, the findings of this study indicated that trauma continues to exert a considerable influence on the relationship under examination. The results of the correlation analysis demonstrated a relationship between all studied variables (PLEs, ADHD symptoms, ruminations, NA, trauma, and the subscales). Furthermore, our results indicated significant gender differences in the experience of trauma. Specifically, women indicated a significantly higher total trauma score and, notably, a significantly higher score for sexual trauma. This is in line with previous research, showing that women are more likely to experience high-impact traumas with direct life-threatening exposure, such as sexual trauma [105]. It is noteworthy that, based on the M.I.N.I. interview, 27% of individuals in this current study may meet the preliminary criteria for PTSD. A review of the literature on the experience of trauma consistently identifies it as a factor that increases the risk of future PLEs [106,107]. Therefore, while traumatic experiences may not be explicitly represented in our tested models, they serve to increase the risk of developing psychiatric disorders [108,109].

Furthermore, numerous studies (e.g., [110–112]) have demonstrated a correlation between trauma and ADHD. This is primarily due to the fact that the cognitive and emotional disturbances that occur in response to experienced trauma (e.g., difficulty concentrating, irritability, high arousal) may overlap with or exacerbate ADHD symptoms (e.g., [113,114]). Furthermore, ADHD symptoms are frequently linked to an elevated probability of exposure to traumatic experiences, particularly during early childhood [115]. Consequently, although ADHD and trauma are discrete domains, their co-occurrence in clinical samples is relatively common, with prevalence rates ranging from 10% to 33% [116]. It is also noteworthy that both disorders are characterized by the experience of intrusive, ruminative thoughts (e.g., [45,117]) and the experience of negative emotions (e.g., [118,119]). It is, therefore, recommended that trauma be considered in future research as a topic for further exploration.

Research on PLEs strongly benefits from the experience sampling method (ESM) as it allows for real-time data collection, thereby capturing the full range of variability and reducing the impact of retrospective or distorted memories on the data [120]. Prior studies demonstrated the efficacy of the ESM method in accurately capturing momentary fluctuations in psychotic and/or emotional states, which is crucial for elucidating the dynamics of these variables in everyday life (e.g., [121–123]). Despite the aggregation of our ESM measures to a weekly average, the averaged results from real-time data provide a richer, more precise, and contextually valid dataset than retrospective data (e.g., retrospective questionnaires) and allow for the identification of patterns that may not be apparent with more traditional measures. Although ADHD symptoms and trauma were not assessed with the ESM, they remained significant factors in the relationships examined. Indeed, there is a clear relationship between negative affect lability and the manifestation of ADHD symptoms [124]. Moreover, some studies suggest that emotional lability may be associated with an increased risk of developing additional mental health problems, including ADHD symptoms [125,126]. Therefore, it is recommended that future research also considers ADHD symptoms in real-time to capture their individual dynamics and their potential impact on other mental health factors.

5. Limitations

Nonetheless, a number of factors should be considered in the interpretation of the results. It is possible that there is a degree of overlap between the symptoms of the two diagnostic categories, namely ADHD and PTSD. The potential for symptom overlap may complicate the ability to discern the precise influence (in this case, the moderating role) of trauma in the relationship between ADHD symptoms, trauma, and PLEs. This could result in the masking or overlapping of results. A traumatic event is one of the most common emerging risk factors for future psychosis [127], and individuals in a (pre-) psychotic state exhibit symptoms such as high impulsivity and frustration or deficits in executive functioning that overlap with clinical symptoms of ADHD [128]. Consequently, diagnoses of ADHD, trauma, and psychosis (or PLEs) should incorporate an evaluation of the other

disorders as a standard procedure, and clinicians should be mindful of the potential for these conditions to co-occur or to even function as one syndrome [129]. Such an approach can facilitate the delivery of comprehensive and accurate diagnoses and treatments.

Another consideration is that the traumatic experience scale (TEC) may not adequately capture the full range of traumatic experiences, particularly regarding their diverse forms (emotional, sexual, and bodily), and showed low reliability. Thus, our study could be replicated using a different or stronger trauma instrument. Furthermore, the TEC is a self-report scale. It is, therefore, possible that individuals reporting such experiences may have understated the significance of the traumatic events in question when completing this questionnaire. One of the coping mechanisms employed in response to traumatic events is the utilization of cognitive avoidance, which is evidenced by the suppression of thoughts and memories, rumination, and dissociation [130].

Additionally, the scale we used to measure ADHD symptoms is also a self-report scale based on the criteria for ADHD diagnosis in the DSM-IV [131]. While the revisions to the ADHD diagnosis in the current edition of the DSM-V [36] are subtle, they are more aligned with the current understanding of the disorder's nature. It is noteworthy that the majority of individuals in our study group exhibited relatively low levels of ADHD symptoms, particularly when assessed using part A of the ASRS questionnaire, which has been identified as the most reliable predictor of ADHD symptoms. Moreover, the majority of the sample consisted of adult women, and the diagnostic criteria for ADHD are still not particularly effective in detecting ADHD in women [132]. The presentation of ADHD symptoms differs significantly between women and men, with women exhibiting greater difficulties with inhibition and cognitive flexibility and men displaying more symptoms of hyperactivity [133]. Additionally, women face challenges in receiving an accurate diagnosis, largely due to the more subtle nature of symptoms (i.e., with less overt hyperactivity) and the potential for misdiagnosis of emotional disturbance [134]. Therefore, further research, with particular regard to the potential influence of gender on the diagnostic process, is warranted.

This present study focused on between-person effects, which was the principal objective. However, this approach also constrains our ability to fully comprehend the precise dynamics of within-person change [135]. Therefore, it would be beneficial for future studies to include real-time intrapersonal measures, particularly for ADHD symptoms and trauma, in order to more accurately capture individual variation and between-person differences in dynamics. It is also important to acknowledge the cross-sectional nature of this study, which constrains our capacity to ascertain the causal mechanisms underlying the relationship between ADHD and PLEs and to note that the findings of this study should not be generalized to the general population, as the majority of the participants were female and did not meet the criteria for a clinical diagnosis.

6. Conclusions

In conclusion, the findings of this study underscore the significant role of rumination and negative affect in the relationship between ADHD symptoms and PLEs. By identifying this association, this study provides important findings regarding the complex processes that may contribute to the development of PLEs in individuals with ADHD symptoms. This study is the first to examine this particular relationship. However, given the relative novelty of these findings, further investigation is required to gain a deeper understanding of the underlying mechanisms and potential pathways of influence. It would be beneficial for further research to aim to replicate these results in diverse populations and assess the potential clinical implications for interventions targeting rumination and affect regulation in individuals with ADHD and/or PLEs symptoms.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/jcm13226727/s1, Supplementary Table S1. M.I.N.I. diagnosis. Supplementary Table S2. CAARMS positive symptoms scores. Supplementary Table S3. ESM evaluation not included in the current study. Supplementary Table S4. Variance Inflation Factors.

Supplementary Table S5. Condition Index. Supplementary Table S6. Descriptive characteristics of the gender subgroups M (SD). Supplementary Table S7. Descriptive characteristics of the study subgroups M (SD).

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Supplementary Table S1. M.I.N.I. diagnosis

Variable	N	%
ADHD[1]	25	13.3
Depression	73	38.8
Mania	15	8
OCD[2]	29	15.4
Anxiety	67	35.6
GAD[3]	33	17.6
AUD[4]	6	3.2
SUD[5]	9	4.8
Agoraphobia	34	18.1
Social phobia	49	26.1
Specific phobia	31	16.5
PTSD[6]	52	27.7
PMDD[7]	34	18.1

Note. [1]**ADHD**=Attention-Deficit/Hyperactivity Disorder. [2]**OCD**=Obsessive-Compulsive Disorder. [3]**GAD**=Generalized Anxiety Disorder. [4]**AUD**=Lifetime Alcohol Use Disorder. [5]**SUD**=Lifetime Substance Use Disorder. [6]**PTSD**=Posttraumatic Stress Disorder. [7]**PMDD**=PreMenstrual Dysphoric Disorder. **N**=number of participants.

Supplementary Table S2. CAARMS positive symptoms scores

Domain	Max	Min	Mean	SD
Non-bizarre Ideas	6	0	1.12	1.37
Perceptual Abnormalities	10	0	1.82	2.39
Unusual Thought Content	5	0	0.62	1.10
Disorganized Speech	9	0	1.92	2.69

Note. **Max**=Maximum. **Min**=Minimum. **SD** = Standard Deviation.

Supplementary Table S3. ESM evaluation not included in the current study

Domain	#	Items	Scale
Sleep (quantity)	1	"How many hours did you sleep last night?" (Time from falling asleep to waking up - in hours)	-
Sleep (quality)	1	"How would you describe your sleep today?"	1-7

Stress event	1	"Think of the most important event that has happened to you in the last two hours. The event was for me:"	1-7
Food, drinks	3	"In the past two hours, have you consumed caffeine and/or smoked cigarettes and/or used e-cigarettes/tobacco products?" "Have you had a meal and/or non-alcoholic drink in the past two hours?" "Have you consumed any alcohol in the past two hours?"	1-2
Body image	1	"Evaluate your feelings about the appearance of your body right now:"	1-7*
Activity-related stress	3	"I would rather do something else right now." "What I am doing now is difficult for me." "I enjoy what I am doing."	1-7
Social stress	1	"Who are you currently staying with?"	1-6
Social stress	2	"Right now, I would rather be alone." "At the moment, I find it pleasant to be with these people." ***OR*** "Right now, I would prefer to be with someone." "Right now, I like being alone."	1-7
Outsider status	1	"Right now, I feel like an outsider."	1-7
Helplessness	2	"I feel upset/nervous because something unexpected has happened." "I feel that important things in my life are out of my control."	1-7
Self-efficacy	2	"I feel confident that I can handle personal problems." "I feel that things are going my way."	1-7
Area-related stress	1	"I find it unpleasant to stay in the present place."	1-7
Aberrant salience	3	"Everything attracts my attention now." "Everything seems to matter now." "I notice things now that I didn't notice before."	1-7
Threat anticipation	3	"Right now, I think something unpleasant is going to happen." "Right now, I am being cautious about protecting myself." "Right now, I am paying attention to details instead of the overall picture."	1-7

Note. Scales which ran from **1-7** "not at all" (**1**) to "very much" (**7**); "very unpleasant" (**1**)* "very pleasant" (**2**)*; Scales which ran from **1-2** "yes" (**1**) to "no" (**2**); Scales which ran from **1-6** (**1**) Alone (**2**) With family (**3**) With partner/husband/wife (**4**) With a friend(s) (**5**) With strangers (**6**) With co-workers/with classmates. #=number of items.

Supplementary Table S4. Variance Inflation Factors

Variable	**Tolerance**	**VIF**
Ruminations	.21	4.79
NA[1]	.21	4.75
Trauma[2]	.29	3.48

Emotional trauma[3]	.28	3.56

Note. VIF=Variance Inflation Factor. [1]**NA**=Negative Affect. [2]Trauma is treated as a total score from **TEC**, and [3]Emotiona trauma is treated as a subscale of trauma.

Supplementary Table S5. Condition Index

Dimension	CI
1	1.0
2	1.46
3	1.59
4	3.97
5	4.81

Note. CI=Condition Index.

Supplementary Table S6. Descriptive characteristics of the gender subgroups M (SD)

Variable	Female (N = 136)	Male (N = 52)	Group comparison
Age	25.19 (5.15)	25.25 (5.31)	n.s.
ADHD (ASRS total)[1]	35.12 (14.57)	31.23 (13.54)	n.s.
Part A[2]	11.07 (5.30)	10.12 (5.54)	n.s.
Part B[3]	24.06 (10.16)	21.12 (8.86)	n.s.
Psychotic experiences[4,5]	12.31 (5.87)	10.91 (4.66)	n.s.
Rumination[4,6]	5.60 (1.13)	2.16 (1.14)	$p < 0.05$, $d = 0.39$, **F > M**
Negative Affect[4,7]	10.18 (4.43)	8.22 (3.98)	$p < 0.05$, $d = 0.46$, **F > M**
Trauma measurement (TEC total)[8]	5.35 (4.01)	4.02 (3.22)	$p < 0.05$, $d = 0.35$, **F > M**
Emotional trauma[9]	1.99 (1.66)	1.50 (1.44)	n.s.
Sexual trauma[9]	0.49 (0.72)	0.08 (0.27)	$p < 0.001$, $d = 0.65$, **F > M**
Bodily threat[9]	0.98 (1.06)	0.92 (1.03)	n.s.

Note. [1]ADHD was assessed with the Adult Self-Report (ASRS). [2]Part A=predictive list of ADHD symptoms (ASRS). [3]Part B=control list of ADHD symptoms (ASRS). [4]As assessed with the experience sampling method (ESM), see the method section for details. [5]The average of Psychotic-like Experiences (PLEs) over one week. [6]The average of rumination scores over one week. [7]The average of negative affect (NA) scores over one week. [8]Trauma measurement was assessed with the Traumatic Experiences Checklist (TEC). [9]Trauma measurement (TEC) subscales. **N**=number of participants. **SD**=standard deviation. **F**=Female. **M**=Male.

Supplementary Table S7. Descriptive characteristics of the study subgroups M (SD)

Variable	Experimental group (N = 99)	Control group (N = 89)	Group comparison
Gender	1.77 (0.42)	1.67 (0.47)	n.s.
Age	23.92 (4.96)	26.64 (5.07)	$p < 0.001$, $d = -0.54$, **E < C**
ADHD (ASRS total)[1]	43.03 (9.51)	24.06 (12.08)	$p < 0.001$, $d = 1.76$, **E > C**
Part A[2]	13.72 (4.14)	7.56 (4.67)	$p < 0.001$, $d = 1.40$, **E > C**
Part B[3]	29.31 (6.79)	16.49 (8.29)	$p < 0.001$, $d = 1.70$, **E > C**
Psychotic experiences[4,5]	14.94 (5.79)	8.56 (2.68)	$p < 0.001$, $d = 1.39$, **E > C**
Rumination[4,6]	3.05 (1.08)	1.84 (0.86)	$p < 0.001$, $d = 1.23$, **E > C**
Negative Affect[4,7]	12.14 (4.02)	6.85 (2.83)	$p < 0.001$, $d = 1.51$, **E > C**
Trauma measurement (TEC total)[8]	6.55 (4.08)	3.24 (2.67)	$p < 0.001$, $d = 0.95$, **E > C**
Emotional trauma[9]	2.51 (1.69)	1.12 (1.14)	$p < 0.001$, $d = 0.95$, **E > C**

Sexual trauma[9]	0.58 (0.77)	0.15 (0.39)	$p < 0.001$, $d = 0.69$, **E > C**
Bodily threat[9]	1.21 (1.18)	0.69 (0.81)	$p < 0.001$, $d = 0.52$, **E > C**

Note. [1]ADHD was assessed with the Adult Self-Report (ASRS). [2]Part A=predictive list of ADHD symptoms (ASRS). [3]Part B=control list of ADHD symptoms (ASRS). [4]As assessed with the experience sampling method (ESM), see the method section for details. [5]The average of Psychotic-like Experiences (PLEs) over one week. [6]The average of rumination scores over one week. [7]The average of negative affect (NA) scores over one week. [8]Trauma measurement was assessed with the Traumatic Experiences Checklist (TEC). [9]Trauma measurement (TEC) subscales. **N**=number of participants. **SD**=standard deviation. **E**=Experimental group. **C**=Control group.

7.3. Publikacja 3

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ORIGINAL PAPER

A comparative temporal network analysis of individuals with ADHD symptoms: an examination of the relationship between psychotic-like experiences and related factors using the Experience Sampling Method (ESM)

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Abstract

Psychotic-like experiences (PLEs) refer to hallucination-like (HLEs) or delusion-like (DLEs) experiences defined as subclinical phenomena located at the lower end of the psychosis continuum. PLEs etiology includes neurodevelopmental changes, maladaptive coping styles, exposure to negative emotions, and stress. These factors frequently co-occur in individuals with Attention-Deficit/Hyperactivity Disorder (ADHD) symptoms, considered a risk factor for PLEs. A total of 188 participants from the general population completed the Adult ADHD Self-Report Scale (ASRS) questionnaire and participated in a seven-day ESM procedure. Participants were divided into High and Low ADHD groups based on self-report symptom severity scale. Saliva samples were collected to determine cortisol levels. A temporal network analysis was used to analyze targeted associations, and permutation testing to compare the differences between the groups. In the H_ADHD group, cortisol was a stronger predictor of threat anticipation (TA) than in the L_ADHD group, and DLEs were strongly associated with TA. In the L_ADHD group, HLEs had a stronger relationship with ruminations and TA than in the H_ADHD group. In both groups, negative affect (NA) was the strongest predictor of ruminations. Out-strength analysis revealed that in the H_ADHD group, DLEs exhibited the strongest predictive function on other variables, while in the L_ADHD group, it was NA. Cortisol level was associated with an increase in TA exclusively in the H_ADHD group, meanwhile in the L_ADHD with subsequent alcohol consumption. The study highlights the different temporal dynamics between factors in individuals with high/low ADHD symptomatology in a non-clinical sample. Interventions for individuals with ADHD should prioritize NA as a central element, focusing on adaptive emotion and stress regulation.

Keywords Psychotic-like experiences · Ecological momentary assessment · Neurodevelopmental changes · Cortisol levels · Negative repetitive thinking · Negative emotions

Introduction

Psychotic-like experiences (PLEs) are defined as subclinical phenomena located at the lower end of the psychosis continuum, which include hallucinations (HLEs) and delusions (DLEs) [63]. PLEs are often transient in time and characterized by a limited intensity or functional impact [28]. The occurrence of PLEs can significantly contribute to other symptoms such as anxiety or mood disorders [37], substance abuse [57], or even heightened risk of suicide [52]. As a result, the risk of developing other psychiatric disorders [46] and psychotic disorders [36] rises, affecting approximately 7% of individuals over a lifetime [42].

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PLEs have been demonstrated to be associated with several well-documented risk factors, including neurodevelopmental changes, such as Attention-Deficit/Hyperactivity Disorder (ADHD) symptoms, which have been shown to co-occur in up to 13% of patients diagnosed with schizophrenia [51]. McGrath et al. [46] indicated that approximately 5% of individuals diagnosed with ADHD experienced at least one psychotic symptom during their lifetime. Excessive or inadequate reactions to stress are frequently observed in individuals with ADHD symptoms [62] and PLEs [60]. Individuals with ADHD symptoms frequently demonstrate deficits in cognitive inhibition, which can lead to difficulties in concentrating on relevant stimuli and an increased reactivity to irrelevant stimuli [2]. Consequently, individuals with increased ADHD symptoms report experiencing a higher level of stress than neurotypical individuals [14]. This phenomenon is also observed in individuals with PLEs [53]. Emotional overreaction, attributing importance to irrelevant environmental triggers (known as aberrant salience; [35]), and expecting a situation to be more stressful than it actually may be (defined as threat anticipation) are all well-documented cognitive processes associated with PLEs [22, 35], which consequently may increase vulnerability to stress in this group.

The common denominator of PLEs and ADHD symptoms in coping with difficult/stressful situations often takes the form of maladaptive behaviors, e.g., ruminative thinking, characterized by a persistent and repetitive focus on negative emotions and experiences [66]. Both ADHD and PLEs have been found to be related to excessive rumination (e.g., Gelner et al., [23], Ludwig, [44]) and negative emotions [34], which may exacerbate emotional dysregulation and increase their vulnerability to daily stressors.

Subjectively experienced and reported stress intensity has recently been identified as an accurate predictive measure of ADHD [41] and PLEs [18]. Nevertheless, a comparison between the subjective experience of stress and its indicators, such as cortisol levels, i.e., a biological marker of stress, is warranted. The measurement of cortisol levels has gained popularity as a means of verifying hormonal response to stress (e.g., Collip et al. [13], Corominas-Roso [15], Schlotz [58]). Both individuals with ADHD symptoms and those experiencing PLEs report higher subjective stress levels in their daily functioning compared to control groups [14, 50]. However, the physiological stress response varies between these groups. For individuals with ADHD symptoms, despite higher subjective reported levels of perceived stress, studies demonstrate no significant differences in cortisol response to stress (e.g., Corominas-Roso et al. [15]). Conversely, individuals with PLEs exhibit not only higher average daily cortisol levels but also greater cortisol reactivity to stressful situations [13], which are indications of an underlying dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis. Furthermore, individuals with PLEs are more likely to perceive future situations as more stressful or threatening, leading to excessive activation of the HPA axis [16]. Conversely, studies examining the relationship between ADHD and the HPA axis have demonstrated an association between delayed or decreased stress responses and ADHD symptoms [10], suggesting a possible slowed release of cortisol in individuals with ADHD symptoms in response to stress [12]. However, the results remain inconsistent, thus emphasizing the need for further research to clarify the role of cortisol reactivity in ADHD. Finally, dysfunctional HPA axis activity in individuals with ADHD may result in reduced cortisol levels (e.g., Jue et al. [33]) and be associated with cognitive deficits (e.g., difficulties with concentration) and behavioral deficits (e.g., high impulsivity) [45]. Therefore, although a heightened subjective perception of stress is common to both groups, their physiological responses to perceived stress and HPA axis mechanisms exhibit opposite patterns.

Nevertheless, the undertaking of cortisol testing requires caution. Research has demonstrated that cortisol levels demonstrate a high sensitivity to external factors not necessarily related to stress [27]. The secretion of cortisol can be significantly affected by various factors, including the time of day, physical activity, caffeine consumption, nicotine intake, and alcohol and non-alcoholic drinks (e.g., Badrick et al. [1], Elder [17], Yang [67]). Consequently, to ensure the reliability of the results, it is necessary to take these factors into account in the analyses when testing cortisol as a stress biomarker.

The present study focuses on the actual measurement of PLEs, ruminations, negative affect, threat anticipation, aberrant salience, and cortisol levels, using the Experience Sampling Method (ESM) to assess these domains in real-time. The use of the ESM method in data collection allows for the real-time capture of participants' daily experiences and emotions, consequently minimizing the risk of recall error and providing a more reliable overview of their current emotional states [59]. Moreover, the collection of data through ESM allows for network analyses, which in turn offer a complex understanding of the dynamic relationships between symptoms and psychological factors [7]. The application of network analyses in psychological research has become popular in recent years (e.g., Beard et al. [3], Cai [11], Misiak [47]). The network approach facilitates the modeling of complex patterns of interaction between variables over time, offering insights into how one dimension (e.g., negative emotions) may influence another (e.g., psychotic experiences) at a subsequent measurement moment. Rather than hypothesizing the existence of hidden constructs, the network approach analyses symptoms as a

system of interrelated elements, thus allowing for a more detailed and process-oriented understanding of psychopathological mechanisms [4]. Therefore, the objective of the present study was to explore the dynamics of the interrelationships between variables under study by conducting a temporal network analysis and comparing network structures in groups of individuals with high and low ADHD symptom severity.

We hypothesized that: (**1**) Individuals with higher ADHD symptom severity (H_ADHD) will exhibit stronger and more complex temporal associations between cortisol levels, PLEs, ruminations, threat anticipation, aberrant salience, and NA compared to individuals with lower ADHD symptom severity (L_ADHD); (**2**) Cortisol level will be more strongly associated with subsequent psychotic-like experiences (PLEs) in the high ADHD group than in the low ADHD group.

Methodology

Participants

The present study included 188 participants aged 18–35 years (72.3% female) who participated in a seven-day Experience Sampling Method (ESM) procedure. Participants were recruited in three Polish cities: Warsaw, Wrocław, and Szczecin, as part of a larger study (grant number: 2021/41/B/HS6/02323). The study sample consisted of 201 participants, of whom 13 were excluded from the analyses due to missing data on the Adult ADHD Self-Report Scale (ASRS; [38]) questionnaire, which measures ADHD symptom severity. The selection of all study groups was made on the basis of the severity of the PLEs symptoms. Participants with the highest PLEs scores were assigned to the experimental group, while those with the lowest PLEs scores were assigned to the control group. Exclusion criteria from the study included a positive history of psychiatric treatment (pharmacological treatment) throughout lifetime, a current episode of major depressive disorder (MDD) within the past two weeks, and a current diagnosis of substance use disorders (except for nicotine dependence). Participants screened positive for major depressive disorder (MDD) and substance use disorders had an extended interview via the Mini-International Neuropsychiatric Interview (M.I.N.I.) to exclude those with a current diagnosis of depressive disorder or substance use disorder. Individuals who met the criteria for any of the above disorders were disqualified from participating in the study.

Importantly, the study sample did not consist of a clinical ADHD population. According to the results of the M.I.N.I. interview, approximately 13.3% of the participants met the diagnostic criteria for ADHD. The lifetime prevalence was as follows: 38.8% depressive disorder, 8% manic disorder, 15.4% obsessive-compulsive disorder, 17.6% generalized anxiety disorder, 15.4% anxiety disorder, 2.7% lifetime alcohol use disorder, 3.2% lifetime substance use disorder, 18.1% agoraphobia, 26.1% social phobia, 16.5% specific phobia, 27.7% posttraumatic stress disorder., and 18.1% premenstrual dysphoric disorder, among the total sample. For the specific values in the specified subgroups, see the Supplementary Table S2. For the purpose of this article, the study group was finally divided based on ADHD symptom severity determined by the ASRS questionnaire: one with high ADHD symptom severity ($N=96$) and one with low ADHD symptom severity ($N=92$). The present study's detailed characterization underscores an investigation of a community sample exhibiting varied levels of ADHD symptoms and other psychiatric conditions.

Materials

In the screening phase of the study, a set of questions was distributed to the participants, requesting demographic information regarding their age, gender, educational level, and family history of psychiatric illness (depression and schizophrenia spectrum disorders). Moreover, participants were asked to complete the Adult ADHD Self-Report Scale (ASRS; [38]), a self-report questionnaire consisting of an 18-item screening scale for adult Attention-Deficit/Hyperactivity Disorder (ADHD). Response options range from 0 to 4 (never-very often), with a maximum total score equal to 72 and a minimum equal to 0. The ASRS scale is divided into two parts: Part A (items 1–6) measures the most predictive ADHD symptoms, with scores ranging from 0 to 24 and a cut-off of ≥ 14 for screening diagnosis for ADHD. The total ASRS Part A score can be classified into four categories: 0–9 (low negative), 10–13 (high negative), 14–17 (low positive), and 18–24 (high positive). Part B consists of the remaining twelve items (7–18) and measures the symptom checklist. In the current study, the Cronbach's alpha for the ASRS was 0.92.

The baseline stage of the study consisted of two diagnostic interviews: (1) The Comprehensive Assessment of At-Risk Mental States (CAARMS; [31, 68]), which was used to measure a range of subthreshold symptoms associated with the prodromal phase of psychotic disorders, and (2) Mini-International Neuropsychiatric Interview (M.I.N.I.; Sheehan et al. [61]), which was employed to assess the psychiatric diagnoses of the participants according to the DSM-IV and ICD-10 criteria. This study used the Polish version of M.I.N.I. Plus 5.0.0, including all diagnostic modules.

Procedure

The median score for the ASRS questionnaire in the study sample was 34.5; thus, a cut-off point of 34 was used to define the high ADHD group (H_ADHD; ASRS ≥ 34) and the low ADHD group (L_ADHD; ASRS < 34). In the next stage of the study (baseline), participants were obliged to complete a clinical interview assessed by a clinical psychologist, CAARMS, and M.I.N.I. (see Supplementary Table S2). Further details of recruitment stages can be found in this reference (see *section Procedure: 2.3.1. Phase I – 2.3.3. Phase III*; [23]).

Table 1 ESM items used in the present study and their definitions

Domain	#	Scale	Item	CA
NA	5	1–7	"I feel anxious." "I feel down." "I feel lonely." "I feel insecure." "I feel annoyed."	0.81
Rumination thought	1	1–7	"At the moment, I feel that I am stuck on negative thoughts and can't get away from them."	-
TA	3	1–7	"I think that something unpleasant will happen." "At the moment, to protect myself, I am cautious." "At the moment, I pay attention to the details instead of the big picture."	0.73
AbS	3	1–7	"Everything grabs my attention right now." "Everything seems to have meaning right now." "I notice things that I haven't noticed before."	0.68
PLEs	8			0.80
HLEs	3	1–7	"My thoughts are so strong that I can almost hear them." "I hear things that aren't really there." "I see things that aren't really there."	0.57
DLEs	5	1–7	"I have the sense that some person or force is around me, although I can't see anyone." "I see special meanings in advertisements, shop windows, or in the way things are arranged around me." "I am confused whether something I experienced was real or imaginary." "My thoughts are influenced by others." "I can't get these thoughts out of my head."	0.74
CS	1	1–2	"In the past two hours, have you consumed caffeine and/or smoked cigarettes and/or used e-cigarettes/tobacco products?"	-
ED	1	1–2	"Have you had a meal and/or non-alcoholic drink in the past two hours?"	-
Alcohol	1	1–2	"Have you consumed any alcohol in the past two hours?"	-

Scales which ran from 1–7 "not at all" (1) to "very much" (7); Scales which ran from 1–2 "yes" (1) to "no" (2); #=number of items. *CA* Cronbach's alpha. *NA* Negative Affect. *TA* Threat anticipation. *AbS* Aberrant salience. *PLEs* Psychotic-like experiences, which comprise Hallucination-like experiences (HLEs) and Delusion-like experiences (DLEs). *CS* Consumption of Caffeine or Tobacco; *ED* Consumption of Food or Non-Alcoholic Drinks; *Alcohol* Consumption of Alcohol

Psychotic-like experiences (PLEs), which comprise hallucination-like experiences (HLEs) and delusion-like experiences (DLEs), threat anticipation (TA), aberrant salience (AbS), ruminations, negative affect (NA), caffeine, tobacco, food, and drink intake were assessed via the ESM method - for details see Table 1. The ESM study lasted seven days. Participants were assessed six times a day (42 measurements in total) between 9 am and 10 pm, with a minimum interval of 60 min between measurements. The assessments were carried out using the MovisensXS Version 1.5.23 application (Movisens GmbH, Karlsruhe, Germany; https://www.movisens.com) on the smartphones provided. Before the procedure, participants were given detailed instructions and information materials. Participants were informed that they had to respond immediately to the reminder or that they could delay their response for up to 15 min. Failure to respond within the specified time resulted in the survey being considered incomplete, which affected 3.6% of all measurements. For participation in the study, participants received financial incentives amounting to 250 EUR if their response rate in the ESM procedure reached or exceeded 80%. While most of the items used in the ESM were adopted from previous studies [39, 40, 55], we also included items adapted from the PQ-16 [30] to capture the presence of PLEs. The order of items was not randomized. Additional items tested as part of the ESM procedure, but not included in the current analysis, can be found in Supplementary Table S1.

Saliva samples were collected from participants once after each ESM measurement to determine cortisol levels, as an indicator of stress. Samples had to be collected within a maximum time frame set at fifteen minutes following the issuance of the ESM alert. Saliva samples were collected using cotton swabs (Salivette, Sarstedt). Participants were required to record the time and date of sampling. The determination of cortisol levels was conducted through the utilization of radioimmunoassays with fluorescence detection. The reagent used was Roche's CORTISOL II Elecsys (reference number: 06687733190). The assessment of cortisol levels was conducted by a commercial laboratory (ALAB, Warsaw, Poland).

The study protocol was approved by the Ethics Committees of the following institutions: the Institute of Psychology (Polish Academy of Sciences, Warsaw, Poland, approval number: 16/VII/2022), Wroclaw Medical University (Wroclaw, Poland, approval number: 129/2022), and Pomeranian

Medical University (Szczecin, Poland, approval number: KB-006/25/2022).

Statistical analysis

Statistical analyses were performed using SPSS 29.0.2.0 (20) and the R software (R version 4.4.2 (2024-10-31), RStudio version 2024.12.0.467) (R Core Team, [54]). All analyses were performed on the total sample and separately for the Low ADHD (L_ADHD) and High ADHD (H_ADHD) groups. Independent samples t-tests were used to assess statistical differences between groups (L_ADHD vs. H_ADHD) on demographic characteristics and all variables assessed. Effect sizes were calculated by using Cohen's d. A multilevel network approach was implemented to identify levels of association between study variables and to determine whether these associations vary between individuals with high versus low ADHD symptom severity.

Network analysis

Multivariate vector autoregression (mlVAR) analyses were conducted to examine the dynamic associations between hallucination-like experiences (HLEs), delusion-like experiences (DLEs), threat anticipation (TA), aberrant salience (AbS), negative affect (NA), ruminations, and cortisol, and their correlates (alcohol, tobacco, food, and non-alcoholic drinks consumption). The study variables were analyzed using a vector autoregression (*mlVAR*) model. According to this model, one variable (e.g., HLEs) at a given time point (t) can be predicted by other variables (e.g., NA) at a previous time point (t-1). In the present analyses, multilevel data from the Experience Sampling Method (ESM) were analyzed. The *mlVAR* model allows for the examination of temporal dynamics at both the participant and group levels, providing estimates of both mean and individual effects. For this purpose, the temporal network was implemented using a multilevel VAR model generated by the *mlVAR* package [20] . Network models were estimated for the total sample (N=188), L_ADHD (N=92), and H_ADHD groups (N=96).

Kalman filter [21] was used to impute missing data, utilizing the R package *imputeTS* [49]. Multivariate vector autoregressive (*mlVAR*) analysis relies on stationarity, i.e., the stability of statistical properties over time (see [9, 24]). Stationarity was verified using the Augmented Dickey-Fuller (ADF) test [29], using the *adf.test* function from the R *tseries* package [65], which findings demonstrated that the assumption of stationarity was fulfilled for all variables and all participants ($p < 0.01$).

Three types of networks were estimated using mlVAR on time series data and visualized using the R package *qgraph* (Epskamp et al. 2012), each of which represented a different level of connections between the variables included in the network – temporal, contemporaneous, and between-subjects networks – in order to establish a comprehensive picture of the fluctuations and connections between the variables included in the model depending on the level of ADHD symptoms. Furthermore, the in-/out-strength measures were examined to identify the most impactful variables within the network and the variables most influenced by the other variables. The networks displayed the following variables: hallucination-like experiences (HLEs), delusion-like experiences (DLEs), threat anticipation (TA), aberrant salience (AbS), negative affect (NegAffect), ruminative thoughts (Rumination), cortisol (Cortisol), alcohol (Alcohol), caffeine or tobacco (CS), and soft drink or meal (ED) consumption. The graphs illustrate the links (i.e., lines) between the variables, with the thickness of the line representing the strength of the links between the variables and the color of the line indicating the direction of the link. The absence of a link indicates no statistically significant association.

Temporal network

Temporal networks capture directional, lagged relationships between variables over time, allowing for the investigation of how changes in one variable (e.g., negative affect) predict subsequent changes in another (e.g., hallucination-like experiences). This approach is crucial for testing hypotheses concerning dynamic processes that may vary among individuals with high versus low ADHD symptoms. The temporal network model has two effects: cross-lagged and autoregressive. Cross-lagged effects represent how one variable (measured at t-1) affects another (measured at t) over time, controlling for autoregressive processes. Autoregressive effects show how one variable (t-1) predicts itself over time (t), with this measurement controlling for cross-lagged relationships [7, 8]. The relationship between the two variables is shown by a directed arrow, with the arrow pointing from one variable to the other. An autoregressive effect is characterized by an arrow from a node pointing towards itself.

Contemporaneous network

A contemporaneous network represents partial correlations between nodes that have been measured at the same time point, controlling for both time effects and all other variables within the network during the same evaluation period. A contemporaneous network model displays significant partial correlations between nodes in the form of lines devoid

of arrowheads. These networks provide a foundation for identifying synchronous interactions that may not be apparent in temporal dynamics alone, thereby highlighting co-occurring symptom patterns within individuals.

Between-subjects network

Between-subject network represents the average relationships between variables across individuals. In other words, it demonstrates how, on average, the level of one variable is related to the level of another variable across multiple participants over time. This timeframe extends across the entire ESM testing period, and it also takes into account the influence of other variables in the network. By comparing these networks across groups, we can examine whether the overall structure of the network of associations differs between individuals with high and low ADHD symptoms.

In- and out-strength

In- and out-strength are centrality measures in network analysis. Out-strength is defined as the extent to which one variable (node) predicts other variables (nodes), whereas in-strength is defined as the extent to which one variable (node) is predicted by other variables (nodes) that are directly connected to it. In- and out-strength centrality measures are used to determine the relative influence of each variable within the network. Out-strength is indicative of the extent to which a variable predicts others, while in-strength is indicative of how strongly a variable is influenced by others. These measures facilitate the identification of key variables that may exert a driving or responsive influence on network patterns characteristic of specific groups.

Group network comparison

Finally, the estimated network models were compared between groups (L_ADHD and H_ADHD). A permutation testing procedure, with 1000 permutations, was performed using the R package mnet [26] to identify significant group differences in network connections. This enables fo the the identification of significantly stronger differences between relationships between variables depending on the specific groups.

Stability

Multilevel modeling is computationally costly, therefore, the established methods of network stability estimation, which rely on bootstrapping or Monte Carlo simulations, are not feasible for EMA data. To the best of our knowledge, a standard procedure has not been documented in the existing literature so far (Blanchard et al., 2022). In the context of cross-sectional data, Epskamp et al. [19] propose the calculation of the correlation stability coefficient (CS-coefficient) as a method to assess the stability of centrality measures. CS (cor = 0.7, sig = 0.95) is defined as the maximal proportion of cases that can be eliminated for the centrality measures to retain at least a 0.7 Spearman rank correlation with the initial centrality measures with at least 95% probability. According to the simulations conducted by Epskamp et al. 20the CS (cor = 0.7, sig = 0.95) should not fall below 0.25 to ensure the stability of the network. While it would be unfeasible to reliably calculate the value of the CS coefficient for EMA data, it is possible to estimate whether the value is smaller or larger than a predefined threshold. In their analysis, Jongeneel et al. [32] randomly selected 100 subsets, comprising 80% of the total sample, and calculated the correlations of their centrality measures with their initial values. The researchers reported that 70% of the observed correlations exceeded 0.7. This corresponds to the following inequality: CS(cor = 0.7, sig = 0.7) > 0.2. We adhere to the established protocol, but implement the values recommended by Epskamp et al., More specifically, for each network-centrality pair, we assess whether CS (cor = 0.7, sig = 0.95) > 0.25. The proportion of all correlations stronger than 0.7 can be found in the supplementary materials (see Supplementary Table S7).

Results

Data exploration

The full ESM dataset consisted of 7896 observations, nested within 188 participants, 7 days, and 6 measurements per day. The minimum required completion rate was set at 80%, which was achieved by all participants. The overall ESM completion rate for the low ADHD group was 95.98%, for the High ADHD group, 94.75%, and for the total sample, the completion rate was 95.34%.

Sample characteristics

All sample characteristics can be found in Table 2. A comparative analysis demonstrated significant differences between the L_ADHD and H_ADHD groups in ADHD symptoms severity, particularly in Part A of the questionnaire's predictive list of ADHD symptoms, indicating significantly higher scores in the H_ADHD group (M = 18.88, SD = 3.5; Cohen's d = 2.46).

Furthermore, in regard to ESM variables, participants in the H_ADHD group exhibited significantly higher scores in NA, TA, AbS, DLEs, and HLEs than L_ADHD participants,

Table 2 Descriptives; total sample $N = 188$; low ADHD = 92; high ADHD = 96

Domain		Total sample *N* (%)	Low ADHD *N* (%)	High ADHD *N* (%)	Total sample Mean (SD)	Low ADHD Mean (SD)	High ADHD Mean (SD)	Min (Total sample)	Max (Total sample)	Range	Cohen's d	*p*-value
Gender	Men	52 (27.7)	27 (29.3)	25 (26)								
	Women	136 (72.3)	65 (70.7)	71 (74)								
Education level												
Primary		6 (3.2)	1 (1.1)	5 (5.2)								
Vocational		1 (0.5)	1 (1.1)	-								
Secondary		79 (42)	32 (34.8)	47 (49)								
Higher		102 (54.3)	58 (63)	44 (45.8)								
Age					25.21 (5.18)	26.35 (5.25)	24.11 (4.9)	18	35	18–35	−0.44	<0.001
Body Mass Index (BMI)					23.18 (4.43)	23.36 (3.99)	23.01 (4.83)	10.82	40.12	-	−0.08	<0.001
ADHD (ASRS total)[1]					34.05 (14.36)	21.9 (8.2)	45.69 (7.87)	0	65	0–72	2.96	<0.001
Part A (ASRS items 1–6)[2]					10.8 (5.37)	6.55 (3.26)	14.88 (3.5)	0	23	0–24	2.46	<0.001
Part B (ASRS items 7–18)[3]					23.25 (9.88)	15.35 (6.1)	30.81 (6.18)	0	46	0–48	2.52	<0.001

ESM	Total sample Mean (SD)	Low ADHD Mean (SD)	High ADHD Mean (SD)	Min (Total sample)	Max (Total sample)	Range	Cohen's d	p-value
Negative Affect[4,5]	2.33 (1.4)	1.78 (1.1)	2.86 (1.45)	1	7	1–7	−0.84	<0.001
Rumination thought[4,5]	2.48 (1.73)	1.92 (1.45)	3.02 (1.8)	1	7	1–7	−0.67	<0.001
TA[4,5]	2.23 (1.42)	1.62 (1.1)	2.81 (1.45)	1	7	1–7	−0.93	<0.001
AbS[4,5]	2.11 (1.4)	1.57 (1.09)	2.64 (1.47)	1	7	1–7	−0.83	<0.001
PLEs[4,5]	1.56 (0.87)	1.25 (0.65)	1.87 (0.93)	1	6.88	1–7	−0.78	<0.001
HLEs[4,5]	1.54 (0.92)	1.21 (0.63)	1.86 (1.03)	1	7	1–7	−0.76	<0.001
DLEs[4,5]	1.58 (0.94)	1.27 (0.74)	1.88 (1.01)	1	7	1–7	−0.7	<0.001
Cortisol[4,5]	118.53 (211.53)	110.26 (269.23)	126.45 (134.33)	7	8904	-	−0.08	<0.001

	Total sample %*	Low ADHD %*	High ADHD %*	Min (Total sample)	Max (Total sample)	Range	Cohen's d	p-value
CS[4,5]	31.8%	26.4%	37%	1	2	1–2	0.25	<0.001
ED[4,5]	66.2%	67.4%	65.1%	1	2	1–2	−0.03	n.s.
Alcohol[4,5]	2.8%	3.1%	2.6%	1	2	1–2	−0.03	n.s.

TA Threat anticipation. *AbS* Aberrant salience. *PLEs* Psychotic-like experiences. *HLEs* Hallucinations-like experiences. *DLEs* Delusions-like experiences. *CS* Coffee/Smoke. *ED* Eat/Drink. *N* number of participants. *SD* standard deviation. *Min* minimum. *Max* maximum.*=% only for the answer "yes" (1). Scales ran from "not at all" (1) to "very much" (7). Scales ran from "yes" (1) to "no" (2). *n.s.* not significant

[1]ADHD was assessed with the Adult Self-Report (ASRS)

[2]Part A=predictive list of ADHD symptoms (ASRS)

[3]Part B=control list of ADHD symptoms (ASRS)

[4]Were assessed using the Experience Sampling Method (ESM), see the method section for details

[5]The average of the variable over one week

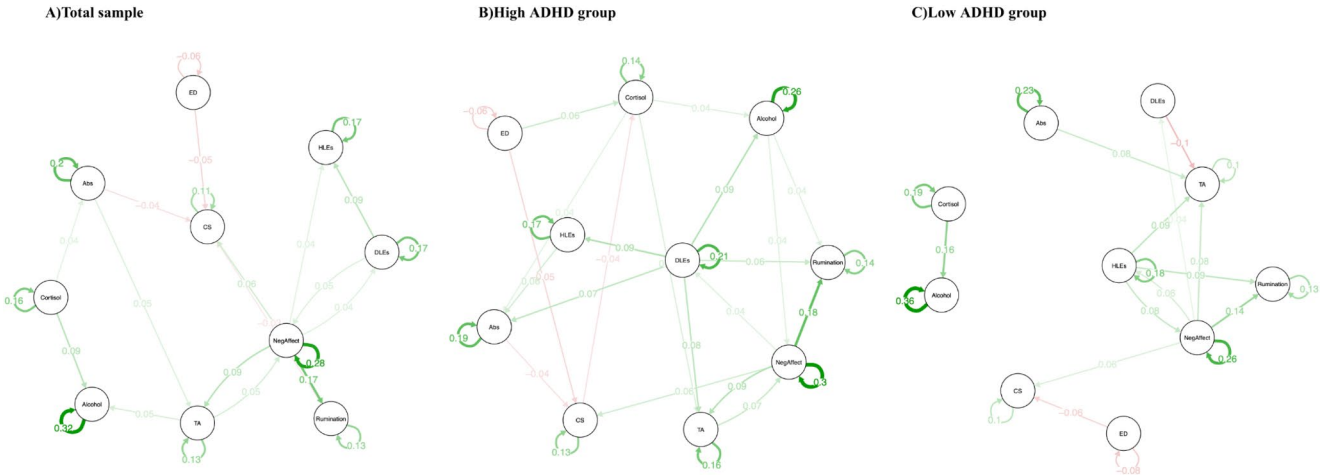

Fig. 1 Temporal network associations. *NegAffect* Negative Affect. *HLEs* Hallucinations-like experiences. *DLEs* Delusions-like experiences. *TA* Threat anticipation. *AbS* Aberrant salience. *CS* Consumption of Caffeine or Tobacco. *ED* Consumption of Food or Non-Alcoholic Drinks. *Alcohol* Consumption of Alcohol

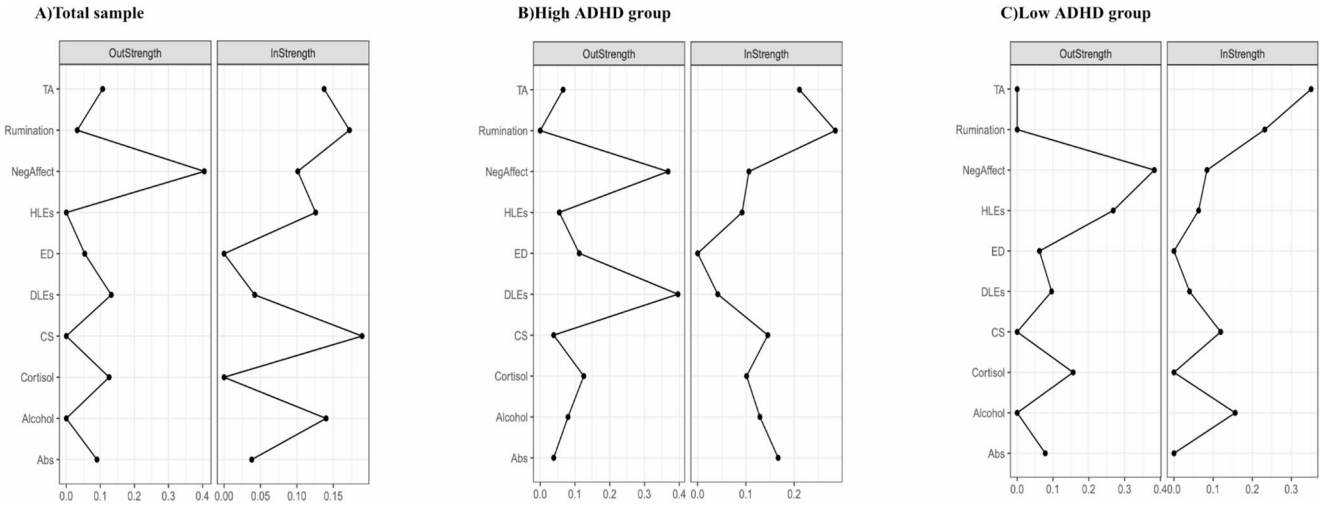

Fig. 2 In and out strength. *NegAffect* Negative Affect. *HLEs* Hallucinations-like experiences. *DLEs* Delusions-like experiences. *TA* Threat anticipation. *AbS* Aberrant salience. *CS* Consumption of Caffeine or Tobacco. *ED* Consumption of Food or Non-Alcoholic Drinks. *Alcohol* Consumption of Alcohol

with large effect sizes (Cohen's d≥−0.7). A moderate, but higher effect was observed for ruminative thoughts in the H_ADHD group (M = 3.02, SD = 1.8; Cohen's d=−0.67). However, no significant group differences were found in cortisol levels, alcohol, nicotine, and soft drink consumption (see Table 2).

Temporal network associations

Figure 1. A-C shows the temporal network models for the (A) total study sample (N = 188), (B) High ADHD N = 96), and (C) Low ADHD group (N = 92). Figure 2. A-C shows the in and out strength, respectively.

All networks included the following variables: hallucination-like experiences, delusion-like experiences, threat anticipation, aberrant salience, negative affect, ruminative thoughts, and cortisol, while taking into account the covariates: alcohol, caffeine or tobacco, and soft drink or meal consumption.

Total sample

As illustrated in Fig. 1A temporal network for the total sample indicates significant autoregressive effects of all included variables, with NA (β = 0.28, p < 0.001), aberrant salience (β = 0.2, p < 0.001), HLEs (β = 0.17, p < 0.001), and DLEs (β = 0.17, p < 0.001) showing the strongest effects over time between measurements. The variable that most strongly predicted other variables (out-strength) was NA, and the variable that was most strongly predicted by other

variables (in-strength) was ruminations (see Fig. 2A & Supplementary Table S6).

Of the 100 potential relationships between the variables included in the network analyses, 25 were found to be statistically significant. The strongest cross-lagged effect was observed in NA predicting more ruminations (β = 0.17, $p < 0.001$). Two feedback loop associations were observed, with NA significantly predicting and being predicted by DLEs (NA→DLEs, β = 0.04, $p = 0.005$; DLEs→NA, β = 0.05, $p = 0.033$), as well as with NA significantly predicting and being predicted by TA (NA→TA, β = 0.09, $p < 0.001$; TA→NA, β = 0.05, $p = 0.01$). Furthermore, NA also significantly predicted more HLEs (β = 0.04, $p = 0.018$) in the next measurement window. HLEs, in turn, were also significantly predicted by DLEs (β = 0.09, $p = 0.002$). Cortisol significantly predicted only AbS (β = 0.04, $p = 0.02$) and alcohol consumption (β = 0.09, $p < 0.001$), but the cortisol level itself was not significantly predicted by any other variable included in the network.

High ADHD

Figure 1B illustrates a temporal network for the H_ADHD group, which indicates significant autoregressive effects of all included variables over time, with NA (β = 0.3, $p < 0.001$), DLEs (β = 0.21, $p < 0.001$), AbS (β = 0.19, $p < 0.001$) and HLEs (β = 0.17, $p < 0.001$) showing the strongest effects. The variable that most strongly predicted other variables in the model (out-strength) was DLEs, and the variable that was most strongly predicted by others (in-strength) was ruminations (see Fig. 2B & Supplementary Table S6).

The analysis revealed that 21 out of 100 of the associations were statistically significant. The strongest cross-lagged effect was found in NA predicting ruminations (β = 0.18, $p < 0.001$) in the next time-point. DLEs significantly predicted increases in HLEs (β = 0.09, $p = 0.006$), TA (β = 0.08, $p = 0.009$), AbS (β = 0.07, $p = 0.02$), and rumination (β = 0.06, $p = 0.04$). Additionally, HLEs significantly predicted AbS (β = 0.06, $p = 0.03$) in the next measurement window. A bidirectional association was found between NA and TA (NA→TA, β = 0.09, $p < 0.001$; TA→NA, β = 0.07, $p = 0.01$). Cortisol was found to significantly predict TA in the next assessment (β = 0.05, $p < 0.001$), but none of the included variables (except for the included covariates) predicted the increase in cortisol levels.

Low ADHD

Figure 1C illustrates the significant associations between included variables in the temporal network model for the L_ADHD group. The variable that most strongly predicted other variables (out-strength) was NA, and the variable that was most strongly predicted by others (in-strength) was TA (see Fig. 2C & Supplementary Table S6). All variables, besides DLEs, demonstrated autoregression over time, with NA (β = 0.3, $p < 0.001$), AbS (β = 0.23, $p < 0.001$), cortisol (β = 0.19, $p = 0.01$), and HLEs (β = 0.18, $p < 0.001$) showing the strongest effects over time between measurements.

The statistical significance was established for 30 out of the 100 associations. The strongest cross-lagged effect was found in higher NA, which significantly predicted more ruminations (β = 0.14, $p < 0.001$) in the next time point. A bidirectional association was observed with NA significantly predicting and being predicted by HLEs (HLEs→NA, β = 0.08, $p = 0.001$; NA→HLEs, β = 0.06, $p = 0.01$). Moreover, more HLEs predicted more TA (β = 0.09, $p = 0.02$), higher HLEs predicted more ruminations (β = 0.09, $p = 0.030$, higher NA predicted more TA (β = 0.08, $p < 0.001$), more AbS predicted more TA (β = 0.08, $p = 0.001$), and higher NA predicted more DLEs (β = 0.04, $p = 0.04$). Other significant pathways were found to have negative associations, and besides DLEs and TA (β = −0.1, $p = 0.02$), were found between covariates ($p \leq 0.001$). Higher cortisol levels in the L_ADHD group predicted only an alcohol consumption increase in the next time point (β = 0.16, $p < 0.001$), and were not statistically predicted by any other variable in the model. Coffee/tobacco and food/non-alcoholic drinks intake were isolated variables from the rest of the network and were not connected to any of the other nodes.

Results of stability estimation

For the majority of centrality measures, a high proportion of subsamples yielded Spearman rank correlations of at least 0.7 with the original estimates, indicating adequate stability. The assessment of stability was conducted by the method described by Jongeneel et al. [32], but in accordance with the values established by Epskamp et al. [19]. Specifically, the percentage of correlations stronger than 0.7 after a random exclusion of 25% of participants was calculated for each centrality measure of each network. Comparing this percentage to the value of 0.95 corresponds to testing whether the correlation stability coefficient is higher than the recommended 0.25. However, it was observed that not all network-centrality pairs met the recommended stability criterion corresponding to the established standard. Specifically, significance ≥ 0.95 was observed in the total sample for temporal in-strength and contemporaneous network strength, it was 100% correlations, for temporal out strength-in was 97%, and in the between-subject network strength was 94%. In the H_ADHD group, only contemporaneous network strength met the established criterion in 100%. Finally, in the L_ADHD group, the temporal in-strength was 96%, and in the between-subject network

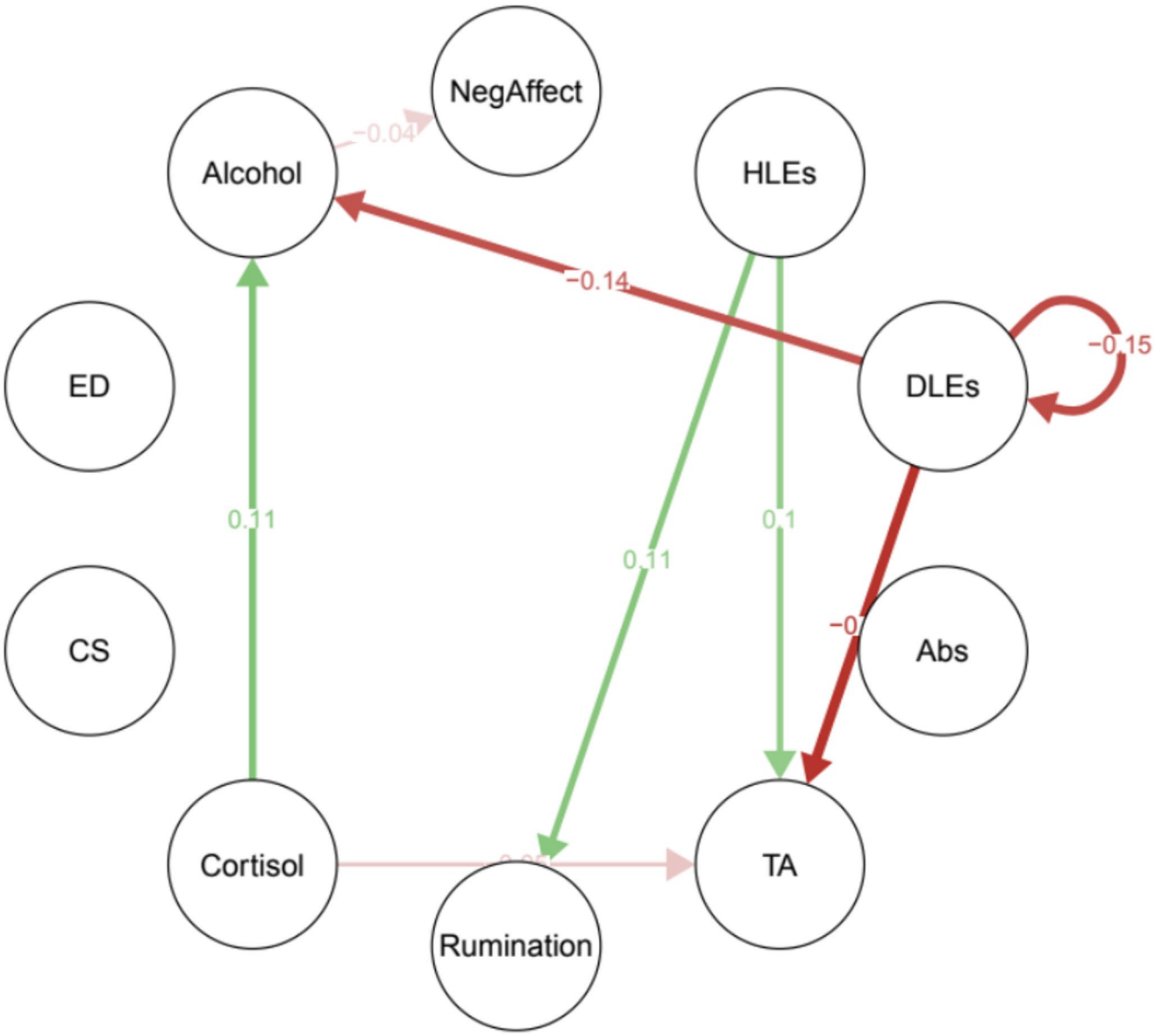

Fig. 3 Temporal network, group significant differences. Edges represent significant differences in temporal associations between groups (L_ADHD – H_ADHD). Only statistically significant differences (P<0.05). *NegAffect* Negative Affect. *HLEs* Hallucinations-like experiences. *DLEs* Delusions-like experiences. *TA* Threat anticipation. *AbS* Aberrant salience. *CS* Consumption of Caffeine or Tobacco. *ED* Consumption of Food or Non-Alcoholic Drinks. *Alcohol* Consumption of Alcohol

strength was 97%. Other values did not exceed a threshold significance ≥ 0.95 (see Supplementary Table S7).

High and low ADHD temporal network comparison

Figure 3 presents significant differences in temporal network models between the High and Low ADHD groups. The permutation tests revealed that the H_ADHD and L_ADHD groups differed significantly in 8 edges, including 1 autocorrelation and 7 cross-lagged edges. The autoregressive effect of DLEs was significantly stronger in the H_ADHD group as compared to the L_ADHD participants. Moreover, the H_ADHD group showed significantly stronger temporal effects for cortisol predicting TA (β= −0.05, p<0.05, DLEs predicting TA (β= −0.17, p<0.05), and DLEs predicting alcohol consumption (β= −0.14, p<0.05), as compared to the L_ADHD group. Meanwhile, in the L_ADHD group, significantly stronger connections were identified in HLEs predicting ruminations (β=0.11, p<0.05), HLEs predicting TA (β=0.1, p<0.05), cortisol levels predicting alcohol consumption (β=0.11, p<0.05), and alcohol consumption predicting NA (β=0.04, p<0.001) (see Supplementary Table S5).

Further analyses, with detailed descriptions, can be found in the supplement. The supplement includes contemporaneous and between-subjects network models, together with their descriptions and group assignments. It also includes a comparison model for contemporaneous and between-subjects network models (see Supplementary Results Report, and Supplementary Figs. 4. A-C & 5. A-C).

Discussion

In the present study, we employed network analyses to identify key patterns of relationships between PLEs, negative affect, aberrant salience, threat anticipation, and cortisol. We thereby demonstrated differences in their structure and centrality depending on the severity of ADHD symptoms.

Temporal network analysis revealed that, regardless of the severity of ADHD symptoms, negative affect was the strongest predictor of ruminative thoughts at the subsequent ESM measurement. While prior research has identified rumination and negative affect as characteristic of individuals with ADHD (e.g., [6]), the current findings extend this conception by indicating that the association between NA and ruminative thoughts may function apart from ADHD symptom severity, potentially reflecting a more extensive transdiagnostic mechanism. Emotional dysregulation and rumination have also been linked to PLEs [25]. More recently, rumination and negative affect have been identified as factors that increase the predisposition to subsequent hallucinatory or delusional experiences among individuals with ADHD symptoms [23]. Therefore, the results obtained in this study extend our previous findings (see [23]) regarding the mediating role of NA and rumination in the relationship between ADHD symptoms and PLEs, thereby specifying the precise direction of these associations and the variables that appear to mediate this relationship.

Analysis of the contemporaneous network model for the H_ADHD group demonstrated 47% of significant connections between variables. DLEs, AbS, HLEs, NA, TA, rumination, and cortisol exhibited a direct association, suggesting the possibility of their synchronized occurrence and reciprocal enhancement. In contrast, in the L_ADHD group, the contemporaneous network did not exhibit the same level of connectivity as observed in the H_ADHD group, with only 27% of significant connections. Our findings emphasize that the high dynamics of symptoms are specifically related to a higher level of ADHD symptoms. These results are in line with recent network approaches to psychopathology that increasingly view mental disorders as a holistic system of interconnected symptoms, where symptoms are causally linked with each other and are the result of dynamic mutual interaction [5]. The interactions between symptoms are suggested to result in a self-perpetuating feedback loop, which in turn leads to the maintenance and mutual reinforcement of symptoms, particularly in the context of co-existing disorders (e.g., co-existing ADHD and PLEs). Therefore, an increase in symptoms is indicative of a growing interconnectedness within the network, with the presence of additional symptoms resulting in the enhancement of connections between them [56].

The results of the centrality measures (out strength) of the specific variables indicated that in the H_ADHD group, DLEs appeared to play a particularly important role, i.e., being the strongest predictor of increases in other domains, emerging as a central node in the network, and in the L_ADHD group it was negative affect. Although theoretical models (e.g., [35]) suggest that attributing excessive significance to neutral stimuli (aberrant salience) may be a mechanism underlying delusional beliefs, the results of the present study suggest opposite directions for these relationships, i.e., DLEs predicted subsequent increases in AbS, as well as TA, HLEs, and rumination. In the context of this study results, it appears that in individuals with high ADHD symptom severity, it is not AbS that leads to delusion-like experiences, but that DLEs themselves can change the way reality is perceived, acting as a "trigger" for subsequent emotional or cognitive processes, which contrasts with the theoretical Kapur (2003) [35] model findings. The central role of DLEs in the H_ADHD group's temporal network may indicate not only an increased susceptibility of this group to initial delusional-like interpretations but also the activation of mechanisms for coping with these experiences.

Finally, comparisons of temporal networks for the high and low ADHD symptom severity groups revealed significant differences in eight network connections. A stronger DLEs autoregression effect was observed in the H_ADHD group, which may indicate greater stability and persistence of DLEs over time than in the L_ADHD group. In addition, stronger associations were observed between cortisol levels and TA and DLEs and TA, in comparison to the L_ADHD group. This may indicate the use of maladaptive coping styles in response to delusional-like experiences and a stronger relationship between the physiological stress response and the subsequent interpretation of a situation as threatening in the individuals within this group. Finally, an indirect pathway between DLEs and NA was observed through alcohol consumption, indicating that alcohol serves as a maladaptive coping style, leading to negative emotions. Conversely, a stronger association was observed between HLEs and ruminations, HLEs and TA, and between cortisol levels and alcohol consumption, in the L_ADHD group compared to the H_ADHD group. This may indicate that individuals with less severe ADHD symptoms, under the influence of hallucinatory-like experiences, engage in

rumination or seek explanations in the external environment, perceiving it as threatening.

Limitations

However, only a minority of participants met the diagnostic criteria for ADHD (13% of participants were diagnosed with ADHD using the M.I.N.I. interview), and overall symptom severity was relatively low (ADHD severity was self-reported, and the mean ASRS score was 34 out of 72). Consequently, the observed group differences may not fully reflect patterns that would be seen in a clinical population with more severe ADHD symptoms. Furthermore, the presence of various psychiatric disorders within the sample may have influenced the findings, underscoring the necessity for caution in interpretation. Finally, the study's low average PLEs range (1.6 on a seven-point scale) may also limit the study's generalizability of the results. Hence, it is recommended that subsequent studies examine clinical or higher-severity ADHD population groups, and (sub)clinical groups with higher PLEs symptoms to enhance the applicability of these findings in clinical contexts.

Another limitation of this study is that causal conclusions can't be drawn. The study is purely exploratory, and the subjective selection of variables may modify network dynamics, affecting results. Although variables were selected based on extant literature (e.g., [43, 48, 55]), the model could be further supplemented, e.g., with ADHD symptoms measured in real-time or isolated morning cortisol measurements. Cortisol Awakening Response (CAR) is typically considered separately due to the characteristic increase of cortisol levels after awakening [64]. However, in the present analyses, the morning cortisol measurement was included in the overall cortisol level. The ESM measurement began at 9 a.m., complicating the identification of the initial measurement's timing relative to the participant's awakening. Therefore, isolating the morning cortisol measure was difficult and limits interpretation in the context of daily cortisol dynamics.

Finally, although ESM provides longitudinal data, we cannot conclude about the experimental nature of the study. The relationships should be interpreted cautiously, considering confounding variables. The cortisol increase may not have been captured, as the actual stress response is delayed. Our network models were based on almost immediate relationships (t–1→t), which may not be optimal for physiological processes such as the HPA axis response to stress, particularly in individuals with ADHD symptoms, who tend to have a delayed release of cortisol [10, 12]. Future analyses should consider delayed relationships modeling (e.g., t–3→t), though the selection of specific delay values should be based on biological knowledge.

Clinical implications

The findings of our study indicate several practical implications for the clinical treatment of individuals with ADHD symptoms. Firstly, psychoeducation for patients may enhance their awareness of the complex dynamics of ADHD symptoms and support the conceptualization of more personalized intervention strategies. Psychological interventions for this group should prioritize stress monitoring and management, as well as the enhancement of adaptive emotion regulation techniques. Furthermore, a focus on intrusive ruminative thoughts and the use of alcohol as a stress-reducing coping mechanism is warranted. Indeed, the use of alcohol exacerbates the intensity of negative emotions and may lead to the exacerbation of preexisting psychological problems. Therefore, taking these aspects into account during the planning of therapeutic interventions can significantly improve the effectiveness of treatments and the psychosocial functioning of individuals with ADHD symptoms.

Conclusions

In summary, the results of this study indicate that different symptom dynamics occur depending on the severity of ADHD symptoms. Firstly, our findings indicated that an increased number of PLEs, more negative emotions, and more ruminative thoughts were associated with more severe ADHD symptoms. Furthermore, our results indicate that individuals with more severe ADHD symptoms exhibit a higher prevalence of psychiatric diagnoses within this group and a lower level of education. Consequently, this observation points to more complex clinical profiles among individuals with greater ADHD symptom severity, including greater psychopathological symptom severity and poorer psychosocial functioning. Future psychological interventions for individuals with ADHD symptoms should prioritize the impact of negative emotions and delusional-like interpretations, positioning them as central elements of the therapeutic approach. Given the high prevalence of maladaptive emotion regulation techniques among individuals with ADHD symptoms, it is crucial to focus on adaptive emotion regulation techniques in this specific group.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00406-026-02203-3.

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Author contributions Hanna Gelner: Writing - original draft, Conceptualization, Methodology, Investigation, Data Curation, Analysis, Resources, Visualization, Project administration.Paulina Bagrowska: Conceptualization, Methodology, Formal Analysis, Writing - Review & Editing.Błażej Misiak: Project administration, Supervision, Investigation, Funding acquisition, Writing - Review & Editing.Jerzy Samochowiec: Writing - Review & Editing.Łukasz Gawęda: Conceptualization, Methodology, Supervision, Writing - Review & Editing.

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Data availability Data supporting findings from the present study are available in the Open Science Framework (OSF) Database (file name: *network_analysis_data_20250318.sav*): [https://osf.io/8ju6b/](https://osf.io/8ju6b).

Declarations

Conflict of interest The authors declare no competing interests.

Informed consent Informed consent was obtained from all participants involved in the study.

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Supplementary Tables

Supplementary Table S1. ESM evaluation not included in the current study

Domain	#	Items	Scale
Sleep (quantity)	1	"How many hours did you sleep last night?" (Time from falling asleep to waking up - in hours)	-
Sleep (quality)	1	"How would you describe your sleep today?"	1-7
Stress event	1	"Think of the most important event that has happened to you in the last two hours. The event was for me:"	1-7
Body image	1	"Evaluate your feelings about the appearance of your body right now:"	1-7*
Activity-related stress	3	"I would rather do something else right now." "What I am doing now is difficult for me." "I enjoy what I am doing."	1-7
Social stress	1	"Who are you currently staying with?"	1-6
Social stress	2	"Right now, I would rather be alone." "At the moment, I find it pleasant to be with these people." ***OR*** "Right now, I would prefer to be with someone." "Right now, I like being alone."	1-7
Outsider status	1	"Right now, I feel like an outsider."	1-7
Helplessness	2	"I feel upset/nervous because something unexpected has happened." "I feel that important things in my life are out of my control."	1-7
Self-efficacy	2	"I feel confident that I can handle personal problems." "I feel that things are going my way."	1-7
Area-related stress	1	"I find it unpleasant to stay in the present place."	1-7

Note. *Scales which ran from* ***1-7*** *"not at all" **(1)** to "very much" (7); "very unpleasant" **(1)*** to "very pleasant" **(2)***; Scales which ran from* ***1-6*** *(**1**) Alone (**2**) With family (**3**) With partner/husband/wife (**4**) With a friend(s) (**5**) With strangers (**6**) With co-workers/ with classmates. **#**=Number of items.*

Supplementary Table S2. M.I.N.I. diagnosis; **Total Sample** N=188; **Low ADHD** N=92; **High ADHD N**=96.

Domain	Total sample N (%)	Low ADHD N (%)	High ADHD N (%)	p-vaule
ADHD[1]	25 (13.3)	4 (4.3)	21 (21.9)	<0.001
Depression (lifetime)	73 (38.8)	25 (27.2)	48 (50)	<0.001
Mania	15 (8)	4 (4.3)	11 (11.5)	<0.001
OCD[2]	29 (15.4)	5 (5.4)	24 (25)	<0.001
Anxiety	29 (15.4)	5 (5.4)	24 (25)	<0.001
GAD[3]	33 (17.6)	7 (7.6)	26 (27.1)	<0.001
AUD[4]	5 (2.7)	1 (1.1)	4 (4.2)	<0.001
SUD[5]	6 (3.2)	0	6 (6.3)	<0.001
Agoraphobia	34 (18.1)	10 (10.9)	24 (25)	<0.001
Social phobia	49 (26.1)	12 (13)	37 (38.5)	<0.001
Specific phobia	31 (16.5)	8 (8.7)	23 (24)	<0.001
PTSD[6]	52 (27.7)	19 (20.7)	33 (34.4)	<0.001
PMDD[7]	34 (81.1)	7 (7.6)	27 (28)	<0.001

Note. *[1]**ADHD**=Attention-Deficit/Hyperactivity Disorder. [2]**OCD**=Obsessive-Compulsive Disorder. [3]**GAD**=Generalized Anxiety Disorder. [4]**AUD**=Lifetime Alcohol Use Disorder. [5]**SUD**=Lifetime Substance Use Disorder. [6]**PTSD**=Posttraumatic Stress Disorder. [7]**PMDD**=PreMenstrual Dysphoric Disorder. **N**=Number of Participants.*

Supplementary Table S3. Network Between Subjects

Path		Total sample	L_ADHD	H_ADHD	Difference *(L_ADHD - H_ADHD)*	*p*
HLEs	NegAffect	0.077	0.219	-0.042	0.263	0.172
DLEs	NegAffect	0.053	-0.085	0.181	-0.274	0.221
DLEs	HLEs	0.589	0.581	0.617	-0.019	0.923
Abs	NegAffect	-0.288	-0.348	-0.287	-0.049	0.761
Abs	HLEs	0.214	0.13	0.148	-0.016	0.939
Abs	DLEs	0.057	0.019	0.206	-0.188	0.259
TA	NegAffect	0.433	0.464	0.341	0.119	0.452
TA	HLEs	0.075	0.185	0.09	0.095	0.62
TA	DLEs	0.107	0.128	0.008	0.137	0.472
TA	Abs	0.645	0.643	0.636	0.017	0.901
Rumination	NegAffect	0.728	0.716	0.702	0.012	0.914
Rumination	HLEs	-0.047	-0.006	-0.044	0.040	0.771
Rumination	DLEs	0.088	0.127	0.062	0.069	0.661
Rumination	Abs	0.17	0.3	0.054	0.246	0.159
Rumination	**TA**	**-0.084**	**-0.305**	**0.087**	**-0.393**	**0.016***
Cortisol	NegAffect	-0.073	-0.088	-0.097	0.014	0.945
Cortisol	HLEs	-0.043	-0.02	-0.142	0.128	0.334
Cortisol	**DLEs**	**0.101**	**-0.011**	**0.308**	**-0.327**	**0.01***
Cortisol	**Abs**	**-0.102**	**0.048**	**-0.33**	**0.388**	**0.023***
Cortisol	TA	0.095	0.02	0.244	-0.226	0.261
Cortisol	Rumination	0.073	0.097	-0.015	0.113	0.644
CS	NegAffect	0.045	0.092	0.07	0.008	0.979
CS	HLEs	-0.1	-0.057	-0.108	0.060	0.736
CS	DLEs	0.027	0.078	-0.003	0.084	0.676
CS	Abs	0.161	0.12	0.174	-0.061	0.728
CS	TA	-0.105	-0.166	-0.013	-0.163	0.406
CS	Rumination	-0.114	-0.127	-0.12	0.039	0.863
CS	Cortisol	0.023	0.142	-0.087	0.292	0.238
ED	NegAffect	-0.091	-0.071	-0.136	0.066	0.668
ED	HLEs	-0.001	-0.016	0.019	-0.035	0.763
ED	DLEs	0.137	0.112	0.15	-0.042	0.719
ED	Abs	-0.001	-0.007	0.025	-0.041	0.775
ED	TA	-0.05	-0.013	-0.131	0.118	0.353
ED	Rumination	0.127	0.015	0.261	-0.249	0.114
ED	Cortisol	-0.091	-0.136	-0.023	-0.121	0.71
ED	CS	0.189	0.27	0.141	0.092	0.617
Alcohol	NegAffect	0.01	0.012	-0.007	0.018	0.907
Alcohol	**HLEs**	**0.052**	**-0.056**	**0.158**	**-0.246**	**0.07***
Alcohol	DLEs	0.071	0.121	0.029	0.098	0.627
Alcohol	Abs	-0.024	0.09	-0.151	0.271	0.113
Alcohol	TA	-0.094	-0.095	-0.1	0.015	0.935
Alcohol	Rumination	0.064	0.083	0.027	0.052	0.695
Alcohol	Cortisol	-0.043	-0.124	0.057	-0.191	0.158
Alcohol	CS	0.211	0.237	0.243	-0.099	0.696
Alcohol	ED	-0.019	-0.006	-0.065	0.053	0.792

Note. ***L_ADHD****=Low ADHD Group;* ***H_ADHD****=High ADHD Group;* ***p****=p-value;* ***NegAffect****=Negative Affect;* ***HLEs****=Hallucinations-like Experiences;* ***DLEs=****Delusions-like Experiences;* ***TA****=Threat anticipation;* ***AbS=****Aberrant salience;* ***CS****=Consumption of Caffeine or Tobacco;* ***ED****=Consumption of Food or Non-Alcoholic Drinks;* ***Alcohol****=Consumption of Alcohol. **=p<0.05.*

Supplementary Table S4. Network Contemporaneous

Path		Total sample	L_ADHD	H_ADHD	Difference *(L_ADHD - H_ADHD)*	*p*
HLEs	NegAffect	0.067	0.088	0.054	0.038	0.296
DLEs	NegAffect	0.083	0.061	0.096	-0.02	0.444
DLEs	HLEs	0.346	0.315	0.364	-0.051	0.326
Abs	NegAffect	-0.01	-0.008	-0.01	0.003	0.945
Abs	HLEs	0.118	0.153	0.116	0.051	0.152
Abs	DLEs	0.081	0.035	0.095	-0.060	0.11
TA	**NegAffect**	**0.305**	**0.221**	**0.349**	**-0.128**	**0.001****
TA	HLEs	0.064	0.033	0.074	-0.040	0.321
TA	**DLEs**	**0.133**	**0.184**	**0.119**	**0.092**	**0.038***
TA	Abs	0.226	0.219	0.23	-0.012	0.754
Rumination	NegAffect	0.428	0.417	0.433	-0.017	0.583
Rumination	HLEs	0.019	0.043	0.003	0.047	0.179
Rumination	DLEs	0.073	0.034	0.096	-0.059	0.184
Rumination	Abs	-0.045	-0.023	-0.056	0.033	0.342
Rumination	TA	0.061	0.031	0.08	-0.048	0.18
Cortisol	NegAffect	0.003	0	0.01	-0.008	0.692
Cortisol	HLEs	0.001	-0.002	0.001	-0.003	0.88
Cortisol	DLEs	-0.033	0.005	-0.047	0.054	0.086
Cortisol	Abs	-0.016	-0.017	-0.024	0.004	0.825
Cortisol	TA	0.033	0.007	0.047	-0.040	0.2
Cortisol	Rumination	0.006	-0.003	0.031	-0.035	0.318
CS	NegAffect	-0.005	-0.015	-0.001	-0.017	0.567
CS	HLEs	-0.007	0.004	-0.012	0.020	0.431
CS	DLEs	0.007	-0.013	0.018	-0.036	0.199
CS	**Abs**	**-0.033**	**-0.002**	**-0.056**	**0.059**	**0.015***
CS	TA	-0.003	-0.015	0.007	-0.0234	0.412
CS	Rumination	0.003	0	0.002	-0.001	0.984
CS	Cortisol	-0.011	-0.003	-0.036	0.033	0.226
ED	NegAffect	-0.01	-0.013	-0.01	-0.007	0.827
ED	HLEs	0.012	0.013	0.014	-0.002	0.967
ED	DLEs	-0.005	-0.008	-0.003	-0.005	0.903
ED	Abs	-0.014	-0.021	-0.008	-0.018	0.526
ED	TA	-0.001	-0.015	0.003	-0.024	0.494
ED	Rumination	0.007	-0.007	0.018	-0.027	0.347
ED	**Cortisol**	**-0.022**	**-0.083**	**0.013**	**-0.088**	**0.023***
ED	CS	0.131	0.099	0.159	-0.063	0.067
Alcohol	NegAffect	0.029	0.043	0.017	0.026	0.358
Alcohol	HLEs	0.028	0.012	0.042	-0.040	0.209
Alcohol	DLEs	0.004	-0.022	0.017	-0.064	0.056
Alcohol	Abs	-0.027	-0.008	-0.037	0.033	0.327
Alcohol	TA	-0.005	0.003	-0.011	0.016	0.589
Alcohol	Rumination	0.016	-0.01	0.036	-0.048	0.079
Alcohol	**Cortisol**	**0.005**	**-0.058**	**0.035**	**-0.093**	**0.003***
Alcohol	CS	0.025	0.016	0.035	-0.020	0.548
Alcohol	ED	0.058	0.057	0.053	0.003	0.88

Note. ***L_ADHD****=Low ADHD Group;* ***H_ADHD****=High ADHD Group;* ***p****=p-value;* ***NegAffect****=Negative Affect;* ***HLEs****=Hallucinations-like Experiences;* ***DLEs****=Delusions-like Experiences;* ***TA****=Threat anticipation;* ***AbS****=Aberrant salience;* ***CS****=Consumption of Caffeine or Tobacco;* ***ED****=Consumption of Food or Non-Alcoholic Drinks;* ***Alcohol****=Consumption of Alcohol. *=p<0.05. **=p<0.001.*

Supplementary Table S5. Network Group Comparison

Path		Total sample	L_ADHD	H_ADHD	Difference (L_ADHD - H_ADHD)	*p*
From $_{(t-1)}$	**To** $_{(t)}$					
NegAffect	NegAffect	0.282	0.264	0.297	-0.033	0.462
NegAffect	HLEs	0.04	0.063	0.021	0.042	0.344
NegAffect	DLEs	0.042	0.04	0.042	-0.002	0.96
NegAffect	Abs	0.019	0.026	0.014	0.012	0.735
NegAffect	TA	0.09	0.082	0.087	-0.005	0.896
NegAffect	Rumination	0.172	0.141	0.182	-0.040	0.409
NegAffect	Cortisol	-0.033	-0.048	-0.013	-0.035	0.326
NegAffect	CS	0.061	0.057	0.057	0	0.997
NegAffect	ED	0.022	0.016	0.022	-0.006	0.923
NegAffect	Alcohol	0.039	0.067	0.011	0.056	0.349
HLEs	NegAffect	0.034	0.084	0.012	0.072	0.063
HLEs	HLEs	0.172	0.176	0.168	0.008	0.865
HLEs	DLEs	0.026	0.018	0.028	-0.009	0.869
HLEs	Abs	0.038	-0.022	0.055	-0.077	0.118
HLEs	**TA**	**0.027**	**0.093**	**-0.004**	**0.098**	**0.023***
HLEs	**Rumination**	**0.019**	**0.091**	**-0.017**	**0.107**	**0.028***
HLEs	Cortisol	0.007	0.002	-0.006	0.008	0.774
HLEs	CS	-0.024	-0.027	-0.021	-0.006	0.873
HLEs	ED	-0.018	-0.018	-0.017	-0.001	0.987
HLEs	Alcohol	-0.021	-0.009	-0.024	0.014	0.771
DLEs	NegAffect	0.047	0.035	0.048	-0.013	0.788
DLEs	HLEs	0.085	0.045	0.092	-0.047	0.469
DLEs	**DLEs**	**0.172**	**0.059**	**0.208**	**-0.150**	**0.004****
DLEs	Abs	0.05	-0.032	0.074	-0.107	0.086
DLEs	**TA**	**0.039**	**-0.096**	**0.078**	**-0.174**	**0****
DLEs	Rumination	0.046	-0.019	0.064	-0.084	0.148
DLEs	Cortisol	0.004	0.014	-0.004	0.019	0.574
DLEs	CS	-0.008	0.029	-0.023	0.052	0.146
DLEs	ED	-0.02	-0.033	-0.012	-0.021	0.75
DLEs	**Alcohol**	**0.041**	**-0.056**	**0.087**	**-0.143**	**0.016***
Abs	NegAffect	0.001	0.022	-0.005	0.027	0.457
Abs	HLEs	0.024	0.005	0.032	-0.027	0.438
Abs	DLEs	0.024	0.024	0.028	-0.004	0.897
Abs	Abs	0.2	0.232	0.188	0.044	0.31
Abs	TA	0.048	0.078	0.033	0.045	0.167
Abs	Rumination	-0.017	-0.017	-0.017	0	0.993
Abs	Cortisol	-0.009	-0.003	-0.014	0.010	0.692
Abs	CS	-0.042	-0.041	-0.039	-0.002	0.958
Abs	ED	0.031	0.033	0.032	0.001	0.98
Abs	Alcohol	-0.029	0.022	-0.06	0.082	0.103
TA	NegAffect	0.055	0.006	0.066	-0.060	0.202
TA	HLEs	-0.024	-0.015	-0.02	0.005	0.922
TA	DLEs	-0.012	-0.008	-0.015	0.007	0.823
TA	Abs	0.009	0.037	-0.003	0.039	0.365
TA	TA	0.132	0.099	0.161	-0.062	0.165
TA	Rumination	0.014	-0.039	0.037	-0.076	0.127
TA	Cortisol	0.008	0.011	0.015	-0.003	0.891
TA	CS	-0.012	-0.004	-0.018	0.013	0.758

TA	ED	-0.014	0.004	-0.027	0.031	0.529
TA	Alcohol	0.052	0.047	0.052	-0.004	0.92
Rumination	NegAffect	0.021	-0.017	0.042	-0.059	0.079
Rumination	HLEs	0.003	0	0.015	-0.015	0.68
Rumination	DLEs	0.017	-0.008	0.034	-0.042	0.162
Rumination	Abs	0	-0.006	0.005	-0.011	0.707
Rumination	TA	0.005	-0.023	0.023	-0.046	0.174
Rumination	Rumination	0.132	0.126	0.141	-0.014	0.733
Rumination	Cortisol	0.026	0.029	0.026	0.003	0.914
Rumination	CS	-0.032	-0.037	-0.024	-0.013	0.644
Rumination	ED	-0.002	-0.013	0.004	-0.017	0.706
Rumination	Alcohol	0.011	-0.008	0.023	-0.031	0.366
Cortisol	NegAffect	0.007	0.004	0.016	-0.011	0.638
Cortisol	HLEs	0.003	0.002	0.008	-0.006	0.638
Cortisol	DLEs	0.005	-0.002	0.017	-0.019	0.144
Cortisol	Abs	0.038	-0.01	0.037	-0.047	0.29
Cortisol	**TA**	**0.013**	**0**	**0.046**	**-0.046**	**0.039***
Cortisol	Rumination	0.003	-0.006	0.023	-0.029	0.231
Cortisol	Cortisol	0.159	0.19	0.137	0.053	0.515
Cortisol	CS	-0.007	-0.011	-0.002	-0.009	0.488
Cortisol	ED	0.003	-0.008	0.031	-0.039	0.251
Cortisol	**Alcohol**	**0.088**	**0.156**	**0.042**	**0.114**	**0.013***
CS	NegAffect	0.003	0.004	-0.002	0.006	0.638
CS	HLEs	-0.009	-0.015	-0.01	-0.005	0.771
CS	DLEs	-0.007	-0.002	-0.013	0.011	0.824
CS	Abs	-0.009	-0.016	-0.004	-0.012	0.516
CS	TA	0	0	-0.002	0.002	0.548
CS	Rumination	0.006	-0.002	0.009	-0.011	0.914
CS	Cortisol	-0.018	-0.011	-0.039	0.028	0.687
CS	CS	0.114	0.097	0.129	-0.032	0.469
CS	ED	-0.02	-0.037	-0.002	-0.035	0.378
CS	Alcohol	-0.017	-0.036	0.008	-0.044	0.252
ED	NegAffect	-0.002	0.013	-0.014	0.027	0.151
ED	HLEs	0.006	0.002	0.009	-0.007	0.664
ED	DLEs	0.002	-0.006	0.007	-0.013	0.338
ED	Abs	-0.008	-0.016	-0.005	-0.011	0.465
ED	TA	0.004	0.005	-0.001	0.007	0.708
ED	Rumination	-0.004	0.001	-0.008	0.009	0.629
ED	Cortisol	0.02	-0.001	0.063	-0.064	0.054
ED	CS	-0.054	-0.062	-0.05	-0.013	0.626
ED	ED	-0.064	-0.077	-0.058	-0.018	0.556
ED	Alcohol	-0.013	-0.007	-0.02	0.013	0.646
Alcohol	**NegAffect**	**0.019**	**0.004**	**0.041**	**-0.036**	**0.008****
Alcohol	HLEs	-0.002	-0.001	-0.003	0.002	0.975
Alcohol	DLEs	-0.004	-0.004	-0.006	0.002	0.935
Alcohol	Abs	-0.001	0.001	-0.005	0.006	0.766
Alcohol	TA	0.006	0.01	0.005	0.005	0.739
Alcohol	Rumination	0.022	0.008	0.039	-0.032	0.057
Alcohol	Cortisol	-0.059	-0.064	-0.038	-0.026	0.951
Alcohol	CS	0.01	-0.011	0.034	-0.045	0.146

Alcohol	ED	0.018	0.009	0.027	-0.017	0.6
Alcohol	Alcohol	0.315	0.361	0.262	0.098	0.375

Note. ***L_ADHD****=Low ADHD Group;* ***H_ADHD****=High ADHD Group;* ***p****=p-value;* ***NegAffect****=Negative Affect;* ***HLEs****=Hallucinations-like Experiences;* ***DLEs=****Delusions-like Experiences;* ***TA****=Threat anticipation;* ***AbS=****Aberrant salience;* ***CS****=Consumption of Caffeine or Tobacco;* ***ED****=Consumption of Food or Non-Alcoholic Drinks;* ***Alcohol****=Consumption of Alcohol. *=p<0.05. **=p<0.001.*

Supplementary Table S6. In/out-strength values

Group		TA	Rumination	NegAffect	HLEs	ED	DLEs	CS	Cortisol	Alcohol	AbS
Total sample											
	In-strength	0.14	0.17	0.1	0.13	0	0.04	0.19	0	0.14	0.04
	Out-strength	0.11	0.03	0.41	0	0.05	0.13	0	0.13	0	0.09
H_ADHD											
	In-strength	0.21	0.29	0.11	0.09	0	0.04	0.15	0.1	0.13	0.17
	Out-strength	0.07	0	0.37	0.06	0.11	0.4	0.04	0.13	0.08	0.04
L_ADHD											
	In-strength	0.35	0.23	0.08	0.06	0	0.04	0.12	0	0.16	0
	Out-strength	0	0	0.38	0.27	0.06	0.1	0	0.16	0	0.08

Note. ***L_ADHD****=Low ADHD Group;* ***H_ADHD****=High ADHD Group;* ***NegAffect****=Negative Affect;* ***HLEs****=Hallucinations-like Experiences;* ***DLEs=****Delusions-like Experiences;* ***TA****=Threat anticipation;* ***AbS=****Aberrant salience;* ***CS****=Consumption of Caffeine or Tobacco;* ***ED****=Consumption of Food or Non-Alcoholic Drinks;* ***Alcohol****=Consumption of Alcohol.*

Supplementary Table S7. Results of Stability Estimation

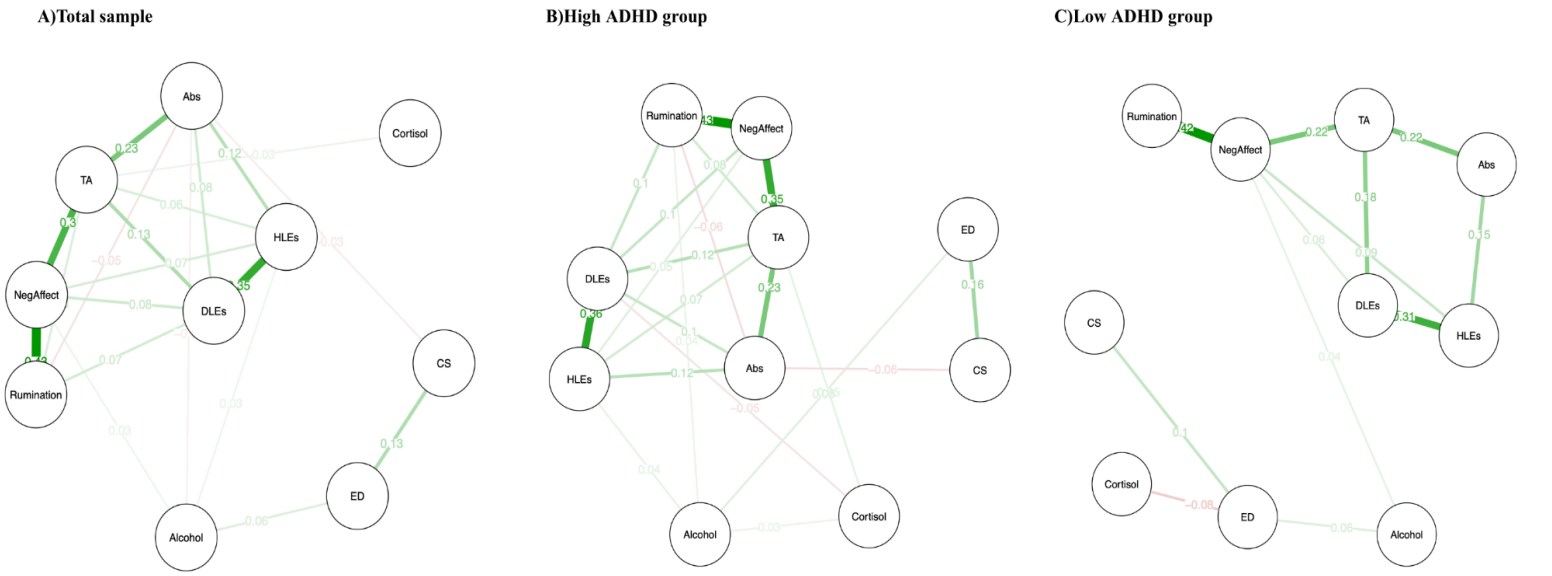
Group	Temporal in strength	Temporal out strength	Contemporaneous network strength	Between-subject network strength
Total sample	1*	0.97*	1*	0.94
H_ADHD	0.83	0.86	1*	0.93
L_ADHD	0.96*	0.94	1*	0.97*

Note. *Values represent proportions of correlations stronger than 0.7;* ***L_ADHD****=Low ADHD Group;* ***H_ADHD****=High ADHD Group; * Values larger than 0.95 indicate a CS-coefficient larger than 0.25 (Epskamp et al., 2018).*

Supplementary results report

1. Contemporaneous network models

Supplementary Figure 4. Contemporaneous network associations



Note. ***NegAffect***=Negative Affect. ***HLEs***=Hallucinations-like Experiences. ***DLEs***=Delusions-like Experiences. ***TA***=Threat anticipation. ***AbS***=Aberrant salience. ***CS***=Consumption of Caffeine or Tobacco. ***ED***=Consumption of Food or Non-Alcoholic Drinks. ***Alcohol***=Consumption of Alcohol.

Supplementary Figure 4. A-C illustrates the contemporaneous networks – i.e., the associations between the variables within the same time frame after controlling for all other temporal and contemporaneous associations. The strongest connections are described, respectively, depending on the group analyzed below. All association values can be found in **Supplementary Table 4**.

1.1. Total Sample

The statistical significance was determined for 19 out of the 45 associations (42%). The strongest association was found between ruminations and NA (β=0.43), indicating that participants who report more ruminative thoughts tend to report increased negative emotions at the same time point. Likewise, DLEs were positively associated with increased HLEs (β=0.35) at the same time frame. Furthermore, a positive association was observed between TA and both NA (β=0.31) and AbS (β=0.23), as well as DLEs (β=0.13). In other words, as the TA increases, so does the level of negative affect, and the occurrence of TA and delusion-like experiences at the same moment. Additionally, participants who reported more AbS experiences also reported more hallucination-like experiences at the same ESM assessment (β=0.12). Other positive associations between variables were β≤0.08 (see **Supplementary Figure 4A**).

On the other hand, the strongest negative association was found between ruminations and AbS (β=-0.05), indicating a slight inverse relationship, that an increase in ruminations is associated with a decrease in aberrant salience symptoms. Other negative associations were identified only between the variables under study and their covariates.

1.2. High ADHD

A total of 21 out of 45 associations were found to be statistically significant (47%). In the H_ADHD group, the strongest positive relationship at the same measurement window was found between ruminations and NA (β=0.43). Moreover, significant relationships were found between DLEs and HLEs (β=0.36), TA and NA (β=0.35), TA and AbS (β=0.23), and TA and DLEs (β=0.12). Positive associations were also found between DLEs and NA, ruminations, and DLEs, and AbS and DLEs at the level of β=0.1. Other positive associations with β≤0.08 can be found in **Supplementary Figure 4B**.

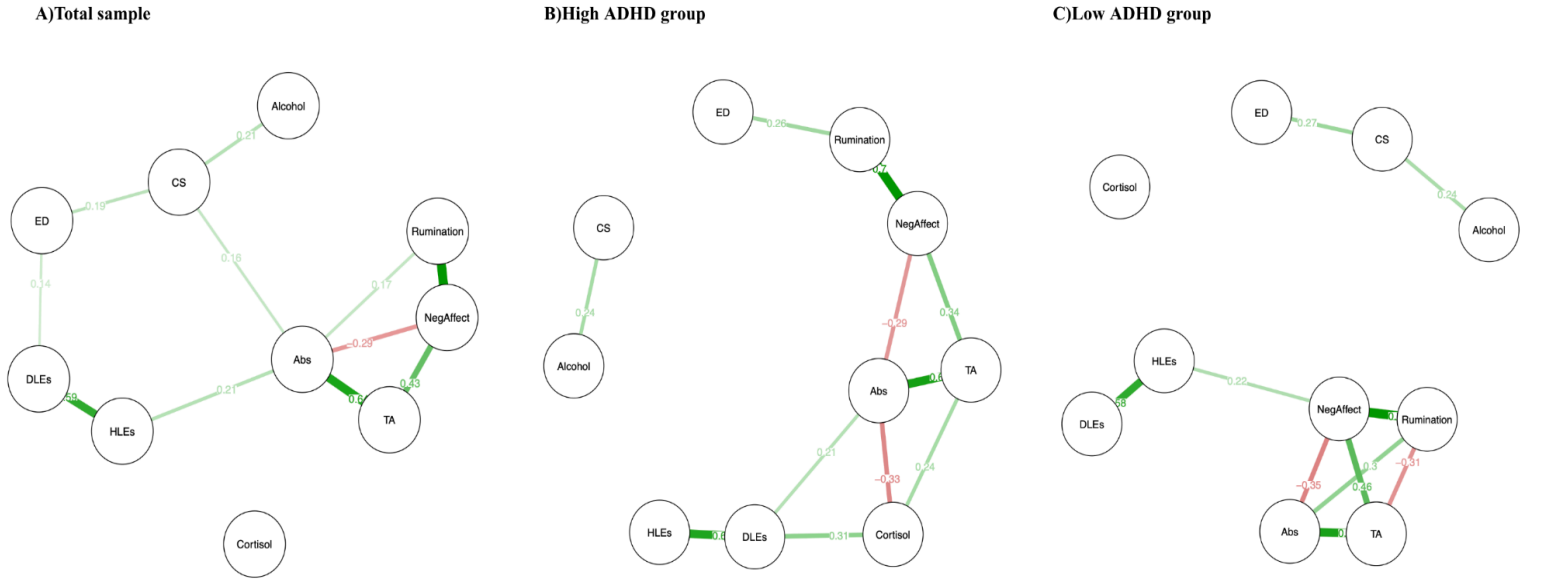
Negative associations were found between rumination and AbS (β=-0.06), and between cortisol and DLEs (β=-0.05). This indicates that, at the same time point, participants who reported more rumination or had a higher level of cortisol reported lower levels of AbS or DLEs, respectively.

1.3. Low ADHD

The analysis revealed that a total of 12/45 associations were found to be statistically significant (27%). In the L_ADHD group, only one negative association was found between the variable under study and its covariate. Other associations were positive, with ruminations and NA being the strongest (β=0.42). In this group, when reporting more DLEs experiences, participants also reported more HLEs at the same time (β=0.32). Furthermore, more TA experiences were positively associated with more NA (β=0.22), more AbS experiences (β=0.22), and more DLEs (β=0.18) at the same time. Finally, more HLEs experiences were reported when participants experienced more AbS (β=0.15). Other positive associations were found with β≤0.06 (**see Supplementary Figure 4C**) and with β≤0.1 between domains under study and their covariates.

2. Between-subjects network associations

Supplementary Figure 5. Between-subjects network associations



Note. ***NegAffect****=Negative Affect.* ***HLEs****=Hallucinations-like Experiences.* ***DLEs****=Delusions-like Experiences.* ***TA****=Threat anticipation.* ***AbS****=Aberrant salience.* ***CS****=Consumption of Caffeine or Tobacco.* ***ED****=Consumption of Food or Non-Alcoholic Drinks.* ***Alcohol****=Consumption of Alcohol.*

The between-subjects network revealed significant associations between the mean levels of the nodes in each participant, when divided by group categorization (**see Supplementary Figure 5. A-C)**, during the overall ESM assessment week.

2.1. Total Sample

The statistical significance was determined for 11 out of the 45 associations (24%). In the total sample (see **Supplementary Figure 5A)**, the strongest positive association was found between the mean levels of ruminations and the mean level of NA (β=0.73). Likewise, a positive relationship was found between the mean level of: TA and AbS (β=0.65), DLEs and HLEs (β=0.59), TA and negative affect (β=0.43), AbS and hallucination-like experiences (β=0.21), and ruminations and AbS (β=0.17). Only one negative association was observed between the mean level of AbS and NA (β= –0.29). The mean levels of cortisol did not have a significant connection to any of the nodes. Other significant associations were found only between the mean level of the variables and their covariates.

2.2. High ADHD

The analysis revealed that 11/45 of the associations were statistically significant (24%). In the H_ADHD group (see **Supplementary Figure 5B)**, the strongest positive association was found between the mean levels of ruminations and the mean level of NA (β=0.7). Likewise, a positive relationship was found between the mean level of: TA and AbS (β=0.64), DLEs and HLEs (β=0.62), TA and NA (β=0.34), and AbS and DLEs (β=0.21). Moreover, the mean level of cortisol was positively associated with delusion-like experiences (β=0.31), and TA (β=0.25), and negatively with the mean level of AbS (β=-0.33). The mean level of AbS was

also negatively correlated with the mean level of NA (β= –0.29). The other two significant positive associations were found only between the mean level of the variables and covariates.

2.3. Low ADHD

The analysis revealed that 10 out of 45 of the associations were statistically significant (22%). In the L_ADHD group (see **Supplementary Figure 5C)**, the strongest positive association was found between the mean levels of ruminations and the mean level of NA (β=0.72). Likewise, a positive relationship was found between the mean level of: TA and AbS (β=0.64), DLEs and HLEs (β=0.58), TA and NA (β=0.46), ruminations and AbS (β=0.3), and HLEs and NA (β=0.22). Only two negative significant associations were found between the mean level of AbS and NA (β=-0.35), and ruminations and TA (β=-0.31). Other significant positive associations were found only between the mean level of the variables and covariates. Finally, the mean level of cortisol did not have any significant connection with any of the nodes in the network.

3. Group differences - contemporaneous & between-subjects network models

Analysis of group differences in contemporaneous networks also revealed several significant differences between groups. In the H_ADHD, stronger associations were found between TA and NA (β=-0.13, p<0.001), alcohol consumption and cortisol levels (β=-0.09, p<0.001), and consumption of food or nonalcoholic drinks and cortisol (β=-0.09, p<0.05). Conversely, in the L_ADHD, stronger relationships were found between TA and DLEs (β=0.09, p<0.05), and coffee or tobacco consumption and AbS (β=0.06, p<0.05). For details, see **Supplementary Table 4.**

Finally, the differences in H/L_ADHD groups were also found in the between-subjects networks. Thus, in the H_ADHD group, stronger associations were identified between ruminations and TA (β=-0.39, p<0.05), cortisol levels and DLEs (β=-0.33, p<0.05), and alcohol consumption and HLEs experiences (β=-0.25, p<0.05). Meanwhile, in the L_ADHD group, only between cortisol levels and AbS (β=0.39, p<0.05). For all results, see **Supplementary Table 3.**