Methods: At baseline, psychotic and affective symptomatology were assessed. The same participants were contacted again 6-years later. The initial analysis aimed to assess the link between affective and negative symptoms, and the progression to PD. The independent variable, baseline symptomatology, was categorized into five groups: no Psychotic Experiences (PE)(reference), subclinical PE, subclinical PE accompanied by affective/negative symptoms, clinical PE, and clinical PE with affective/negative symptoms. In the subsequent analysis, the association between affective and negative symptoms at baseline and the onset of PE and PD at follow-up was evaluated. For this analysis, the baseline symptomatology was restructured into two categories: neither PE nor affective/negative symptoms (reference), and the presence of affective/negative symptoms without PE.
Results: The findings from the initial analysis indicated that being part of the 'subclinical PE only' group at baseline was not associated with an increased risk of developing PD at follow-up. Being part of the 'subclinical PE+affective/negative symptoms' group was not significantly associated with PD at follow-up, although a trend was observed (OR: 3.22; z=1.90; p=0.057). Moreover, being classified as having 'clinical PE only' (OR: 6.23; z=2.57; p=0.010) and 'clinical PE+affective/negative symptoms' (OR: 8.48; z=4.17; p=0.001) at baseline was associated with an increased risk of developing PD at follow-up. Results from the subsequent analysis showed that being in the 'affective/negative symptoms' group at baseline was associated with an increased risk of new subclinical PE (RR: 1.98; z=3.20; p=0.001), new clinical PE (RR: 3.14; z=4.84; p=0.001), and new PD (RR: 4.21; z=2.17; p=0.030) at follow-up, compared to the 'neither PE nor affective/negative symptoms' group.
Conclusions: The results confirm that baseline severity of positive symptoms is significant in predicting transition to PD. In addition, the findings imply that not only positive symptoms but also affective and negative symptoms might contribute to the risk of transition to PD as well as incident psychotic symptoms. Defining CHR groups based on a combination of positive, affective and negative symptoms instead of focusing only on positive symptoms likely will help more accurately predict the transition to psychosis.

Disclosure of Interest: None Declared

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Neurophysiological evidence of motor preparation dysfunction to inner speech in schizophrenia

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Introduction: Auditory verbal hallucinations (AVHs) in schizophrenia have been suggested to arise from failure of corollary discharge mechanisms to correctly predict and suppress self-initiated inner speech. However, it is unclear whether such dysfunction is related to motor preparation of inner speech during which sensorimotor predictions are formed. The contingent negative variation (CNV) is a slow-going negative event-related potential that occurs prior to executing an action. A recent meta-analysis has revealed a large effect for CNV blunting in schizophrenia. Given that inner speech, similar to overt speech, has been shown to be preceded by a CNV, the present study tested the notion that AVHs are associated with inner speech-specific motor preparation deficits.
Objectives: The present study aimed to provide a useful framework for directly testing the long-held idea that AVHs may be related to inner speech-specific CNV blunting in patients with schizophrenia. This may hold promise for a reliable biomarker of AVHs.
Methods: Hallucinating (n=52) and non-hallucinating (n=45) patients with schizophrenia, along with matched healthy controls (n=42), participated in a novel electroencephalographic (EEG) paradigm. In the Active condition, they were asked to imagine a single phoneme at a cue moment while, precisely at the same time, being presented with an auditory probe. In the Passive condition, they were asked to passively listen to the auditory probes. The amplitude of the CNV preceding the production of inner speech was examined.
Results: Healthy controls showed a larger CNV amplitude (p = .002, d = .50) in the Active compared to the Passive condition, replicating previous results of a CNV preceding inner speech. However, both patient groups did not show a difference between the two conditions (p > .05). Importantly, a repeated measure ANOVA revealed a significant interaction effect (p = .007, η_p^2 = .05). Follow-up contrasts showed that healthy controls exhibited a larger CNV amplitude in the Active condition than both the hallucinating (p = .013, d = .52) and non-hallucinating patients (p < .001, d = .88). No difference was found between the two patient groups (p = .320, d = .20).
Conclusions: The results indicated that motor preparation of inner speech in schizophrenia was disrupted. While the production of inner speech resulted in a larger CNV than passive listening in healthy controls, which was indicative of the involvement of motor planning, patients exhibited markedly blunted motor preparatory activity to inner speech. This may reflect dysfunction in the formation of corollary discharges. Interestingly, the deficits did not differ between hallucinating and non-hallucinating patients. Future work is needed to elucidate the specificity of inner speech-specific motor preparation deficits with AVHs. Overall, this study provides evidence in support of atypical inner speech monitoring in schizophrenia.

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