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Corrigendum: Psychosocial stress moderates the relationship between cerebrospinal fluid lactate dehydrogenase and the duration of untreated psychosis in first-episode psychosis

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A Corrigendum on:

Psychosocial stress moderates the relationship between cerebrospinal fluid lactate dehydrogenase and the duration of untreated psychosis in first-episode psychosis

by Giné-Servén E, Boix-Quintana E, Daví-Loscos E, Cepedello S, Moreno-Sancho L, Niubó M, Hernández-Antón R, Cuesta MJ and Labad J (2024). *Front. Psychiatry* 15:1327928. doi: 10.3389/fpsyt.2024.1327928

In the published article there was an error in the **Introduction**. The authors sincerely apologize to Dr. Jai Shah and his group (Shah JL, Paquin V, McIlwaine SV, Malla AK, Joober R, Pruessner M. Examining the psychobiological response to acute social stress across clinical stages and symptom trajectories in the early psychosis continuum. *Dev Psychopathol* (2023) 28:1-13. doi: 10.1017/S0954579423000056) for the overlap, with missing attribution, between our article and theirs. This was an oversight which the authors deeply regret. The text previously read:

"Evidence for this diathesis has been gathered from various approaches (18), encompassing general epidemiology and prospective cohort studies (19–21), adverse childhood experiences (22, 23), clinical assessments documenting sensitivity to stress (24, 25), analyses of perceived (subjective) stress in patients (26, 27), and research on dopamine or hypothalamic–pituitary–adrenal (HPA) axis dysregulation (28–30).

In this manner, it is thought that early-life adversity and stress can interact with innate and acquired neurobiological factors to play a role in distress and, ultimately, the development of psychotic syndromes (28, 31)."

The corrected text appears below:

"Previous studies suggest that early life stress, particularly childhood trauma (18, 19), is a risk factor for psychotic disorders. Several studies have also reported increased perceived stress in individuals at clinical high risk of psychosis (CHR) or with psychotic disorders, measured using self-report questionnaires (20-22) or electronic sampling methods (23). Regarding hypothalamic-pituitary-adrenal (HPA) axis measures, previous longitudinal studies suggest that diurnal cortisol (24, 25), an increased cortisol awakening response (CAR) (26), and a higher stressor-cortisol concordance (27) are found in CHR individuals who will later develop psychotic symptoms. However, in first episode psychosis (FEP) patients, a blunted CAR has been described (28-30), suggesting that biological markers of stress response might be influenced by the stage of psychotic illness. Consistent with this, previous studies exploring stress responsiveness with the Trier Social Stress Test in CHR and FEP patients (31) found differences in autonomic measures between groups (elevated heart rate and blood pressure were observed in FEP patients), suggesting that the stage of illness contributes to variations in the psychobiological stress response."

In the published article there was an error in the **References**. Following the correction to the **Introduction**, the below references have been added and the reference list renumbered:

The authors apologize for this error. This update does not change the scientific conclusions of the research in any way.

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