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Depression and the prefrontal-hippocampal pathway - A multimodal neuroimaging study in transgender women

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ABSTRACT

This study aims to investigate functional and neurotransmitter signaling in the prefrontal-hippocampal pathway in relation to depression in a cohort of Thai transgender women. Twenty participants completed mental health surveys and imaging between January and March 2024. Depression severity was measured by Patient Health Questionnaire-9 (PHQ-9) scores. Higher PHQ-9 scores were associated with lower GABA levels in the hippocampus, and with lower fractional amplitude of low-frequency fluctuations (fALFF) in the dorsolateral prefrontal cortex. However, removal of the hippocampal GABA outlier resulted in a non-significant relationship with PHQ-9. Therefore, future studies with larger datasets should further investigate the association between GABA and depression in a transgender cohort. These findings revealed interactions between neurotransmitter signaling and functional brain activity of the hippocampal-prefrontal circuit in depression.

1. Introduction

The transgender population experiences higher rates of depression compared to the cisgender population, with estimates in Thailand reaching around 22.7 % for the transgender community compared to 3.2 % in the overall population [1]. Depression and psychological distress in transgender individuals are influenced by complex bio-psychosocial factors, ranging from endocrinological and neuro-developmental issues, neuropsychological comorbidities, gender dysphoria (defined by distress arising from the incongruence between assigned sex at birth and gender identity) to social stigma [2–4].

Despite the high prevalence and complex nature, transgender individuals are not typically represented in neuroimaging research projects aimed at elucidating the brain basis of depression. More broadly, there is a problematic lack of diversity in neuroimaging research, which largely focuses on cisgender participants from Western, Educated, Industrialized, Rich, and Democratic (WEIRD) countries [5]. Therefore, a central goal of this study is to investigate the brain basis of depression in a cohort of Thai transgender women.

In this study, we focused on two key brain regions implicated in depression, namely the hippocampus and the dorsolateral prefrontal cortex (DLPFC). Self-referential processes such as rumination, a key symptom of depression, have been associated with hippocampal hyperactivity, and hippocampal atrophy is commonly observed in recurrent depression [6]. Furthermore, repetitive transcranial magnetic stimulation (TMS) applied to the left DLPFC has shown significant effectiveness in treating depression [7]. Indeed, the DLPFC-hippocampal pathway is central to the cognitive neurobiological model of ruminative thought in depression, reflecting both bottom-up maladaptive reactivity and decreased top-down cognitive influence [8] as confirmed in a

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cognitive reappraisal task [9].

A hierarchical model proposed by Hasler and Northoff has suggested that non-invasive measurement of neurotransmitter concentrations – specifically gamma-aminobutyric acid (GABA) - may offer endophenotypic insights into inhibitory pathways relevant to depression [10]. A recent multimodal neuroimaging study showed that hippocampal GABA was necessary to recruit DLPFC activation in a task context that required suppression of unwanted thoughts [11]. Furthermore, signal strength as measured by the fractional amplitude of low frequency fluctuations (fALFF) is consistently decreased in DLPFC and hippocampus in individuals with depression compared with healthy controls [12,13]. Despite the importance of the prefrontal-hippocampal circuit in cognitive and emotional regulation, its specific relationship with depression measurements remains poorly understood. To address this gap, we aimed to investigate functional and neurotransmitter signaling in the prefrontal-hippocampal pathway in relation to depression.

2. Material and methods

2.1. Patients and data collection

Twenty transgender women with mild-to-severe depression were recruited from the Tangerine Community Health Clinic, a specialized healthcare center for transgender people in Bangkok, Thailand [14]. Recruitment and data collection took place between January and March 2024. Mild-to-severe depression was defined based on Patient Health Questionnaire (PHQ-9) scores greater than 5 and a neuroticism score on the Five-Factor Inventory (NEO-FFI) greater than 30.1 which was previously defined as one standard deviation above the population mean. See Supplementary Table S1 for a detailed overview of inclusion and exclusion criteria. Of the twenty subjects recruited, half were currently participating in gender-affirming hormone therapy (GAHT).

This study was approved by the Institutional Review Board, Faculty of Medicine Ramathibodi Hospital, Mahidol University (COA. MURA2023/522). After obtaining informed consent, participants completed a series of mental health questionnaire assessments followed by a 1-h MRI session at the Advanced Imaging Diagnostic Center, Faculty of Medicine Ramathibodi Hospital, Bangkok, Thailand. For the mental health assessments used in this manuscript, participants performed Thai language versions of the PHQ-9 and NEO-FFI self-report questionnaires on paper prior to the MRI scanning session.

2.2. Neuroimaging acquisition and preprocessing

The MRI session included T1-weighted and T2-weighted structural MRI, resting-state functional MRI (rs-fMRI), and GABA magnetic resonance spectroscopy (MRS) imaging, using a Philips Ingenia Elition 3.0T scanner (software R5.7.1). We leveraged a state-of the-art imaging protocol informed by the Human Connectome Project Lifespan and Disease protocols, and the MRS utilized the MEGA-PRESS sequence with parameters based on standard guidelines [15]. The neuroimaging data were acquired as follows: For T1-weighted, 0.8 mm^3 isotropic voxel size was used with repetition time (TR) of 9 ms and echo time (TE) of 4.2 ms. For rs-fMRI, 2.5 mm^3 isotropic voxel size was acquired with TR of 0.8 s and TE of 37 ms for 20 min of both AP and PA directions. For MRS, single-voxel measurements were performed with a voxel size of $4 \times 2 \times 2$ cm^3 for the hippocampus and $4 \times 2 \times 3$ cm^3 for the dlPFC, using a TR of 1.6 s and a TE of 68 ms. Further neuroimaging acquisition parameters are listed in Supplementary Table S2.

Functional imaging data were preprocessed by FMRIB's Software Library (FSL) software. Briefly, the preprocessing steps included motion correction, distortion correction, structural co-registration, spatial normalization to Montreal Neurological Institute space using nonlinear registration, smoothing with a 5-mm kernel, and noise correction with independent component analysis. The distortion correction of echo planar imaging (EPI) of rs-fMRI was performed utilizing a technique that leverages two different phase encoding directions. Specifically, FSL's topup tool estimated distortion fields from opposite phase-encoded EPI images, and the estimated fields were applied for geometric correction (see the Supplementary Methods). Additional cleanup of rs-fMRI data was performed using independent component analysis noise removal by FSL AROMA. Regions of interest (ROIs; Fig. 1) were defined from the Harvard/Oxford Subcortical atlas [16] for hippocampus, and from a prior task-based fMRI localizer for the DLPFC [17].

For MRS images, single voxels were placed to ROIs guided by high resolution T1-weighted image. Adaptive shimming of the Philips scanner was used to optimize signals which were obtained from the left DLPFC and left hippocampus. The signals were processed with baseline initial peak subtraction with 90 % Gaussian character for fitting the power spectrum. MEGA Subtract All (ON-OFF) Spectra was performed (see the Supplementary Methods).

2.3. Data analysis

The study utilized neuroimaging parameters focused on two regions of interest: the hippocampus and the DLPFC. For the functional analyses, the bilateral hippocampus was defined based on the Harvard/Oxford Subcortical atlas and the bilateral DLPFC was defined based on prior work (see the Supplementary Methods). We extracted the mean time series of the left and right hippocampus and DLPFC following preprocessing and estimated the fractional amplitude of low-frequency fluctuation (fALFF). This measure captures the normalized strength of spontaneous signal fluctuations in each brain region by calculating the ratio of the power in the low-frequency range (0.01–0.1 Hz) to the power across the full frequency range reflecting the physiological relevance of blood oxygenation level-dependent (BOLD) signals. fALFF has also been shown to exhibit a good signal-to-noise ratio [18], and previous research has demonstrated its promising sensitivity in depression [19]. Therefore, in this study, the fALFF estimates were applied and averaged between left and right hippocampus and between left and right DLPFC before further analysis.

For the spectroscopy data, GABA concentrations were normalized to creatinine concentrations (GABA/Cr) using creatinine as a stable reference metabolite. Notably, hippocampal spectroscopy can be challenging due to the small size of the hippocampus, which can lead to low spectral resolution and a low signal-to-noise ratio [20]. To address this concern, we followed prior advice on the minimum voxel volume while also preserving spatial specificity for the regions of interest [15]. Since MRS did not provide motion estimation during scanning period, we controlled for motion-related noise by including the framewise displacement parameter from rs-fMRI as a confound in subsequent analyses which has been demonstrated to be an effective proxy for motion-related confounds in previous research [21].

Following the extraction of neuroimaging parameters, multiple univariate linear regressions were performed using the ordinary least squares method and standardized beta coefficient (β), p-value (p), and t-statistic (t) were reported. In these analyses, multimodal neuroimaging measures served as the independent variables, PHQ-9 scores were included as the dependent variables, and motion (framewise displacement) was included as a control variable. Mediation analyses were also conducted to investigate whether DLPFC fALFF mediated the relationship between hippocampal GABA and depression severity as measured with PHQ-9 scores. Additionally, an unpaired *t*-test was performed to investigate differences in neuroimaging measures and PHQ-9 scores between the GAHT and non-GAHT groups using permutation testing. Equations for all regression and mediation models can be found in the Supplementary Material.

3. Results

The study consisted of 20 transgender women with mean age (standard deviation) of 30.1 (±6.5) years. The mean neuroticism score

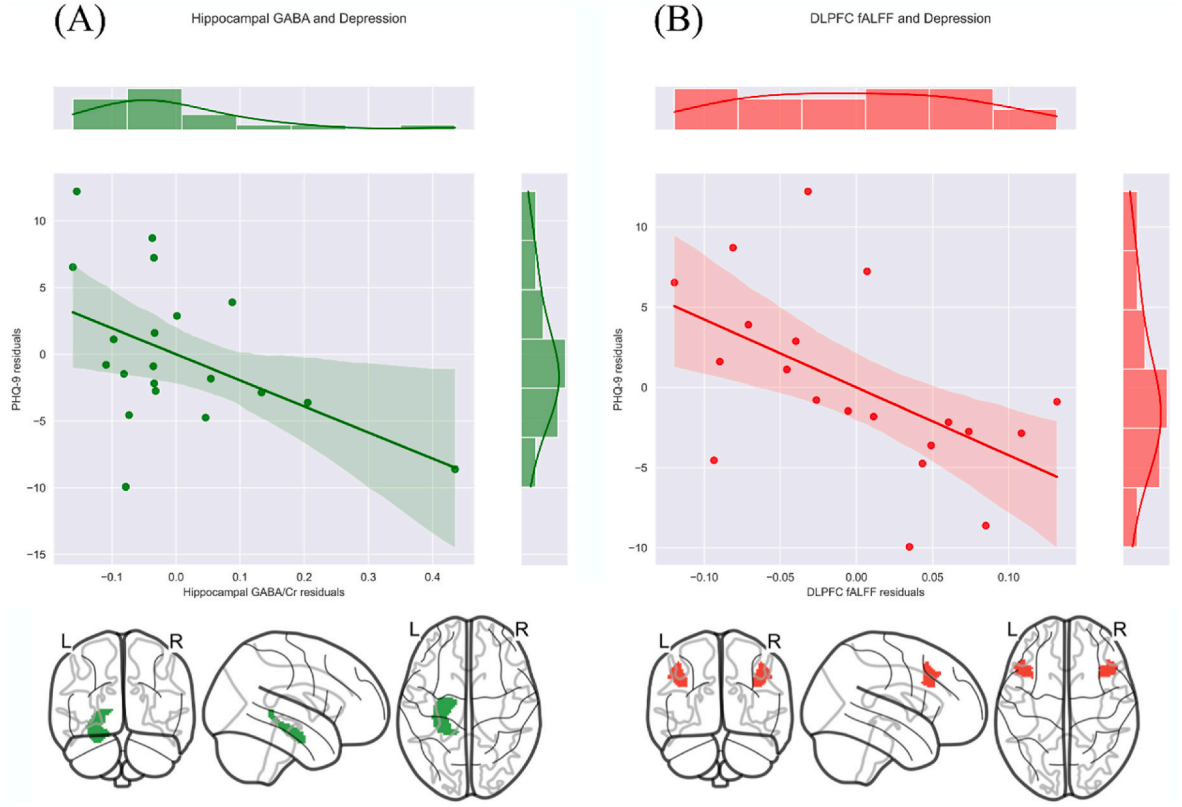

Fig. 1. Neurotransmitter and functional MRI associations with depression. Scatter plots illustrate the relationship of patient health questionnaire (PHQ-9) scores with gamma-aminobutyric acid (GABA) in the hippocampal (A) and fractional amplitude of low frequency fluctuation (fALFF) in the dorsolateral prefrontal cortex (DLPFC) (B) after controlling for the confounder. 95 % Confidence intervals and marginal histograms are also displayed. GABA/Cr indicates GABA concentrations normalized to the creatinine reference.

(NEO-FFI) was 45.55 (±7.5), and the mean PHQ-9 score was 14.6 (±5.73), indicating the mental health status of the sample. Nearly half of the participants (45 %) were undergoing gender-affirming hormone therapy at the time of the study. The linear regression models revealed a significant negative association between hippocampal GABA and PHQ-9 scores ($\beta = -0.48$, $p = 0.039$, $t = -2.23$; Fig. 1A), such that higher depression severity was associated with decreased hippocampal GABA. Furthermore, our findings revealed a significant negative association between DLPFC fALFF and PHQ-9 scores ($\beta = -0.54$, $p = 0.015$, $t = -2.66$; Fig. 1B), such that higher depression severity was associated with decreased fALFF in the DLPFC. However, testing the sensitivity of the hippocampal results by removing the subject with the highest GABA value revealed statistically non-significant outcome after outlier removal ($\beta = -0.34$, $p = 0.174$, $t = -1.42$). No significant associations were observed between PHQ-9 scores and either hippocampal fALFF or DLPFC GABA.

Fig. 2 illustrates the results from the mediation analysis model. We observed a trend-level mediating effect of DLPFC fALFF on the

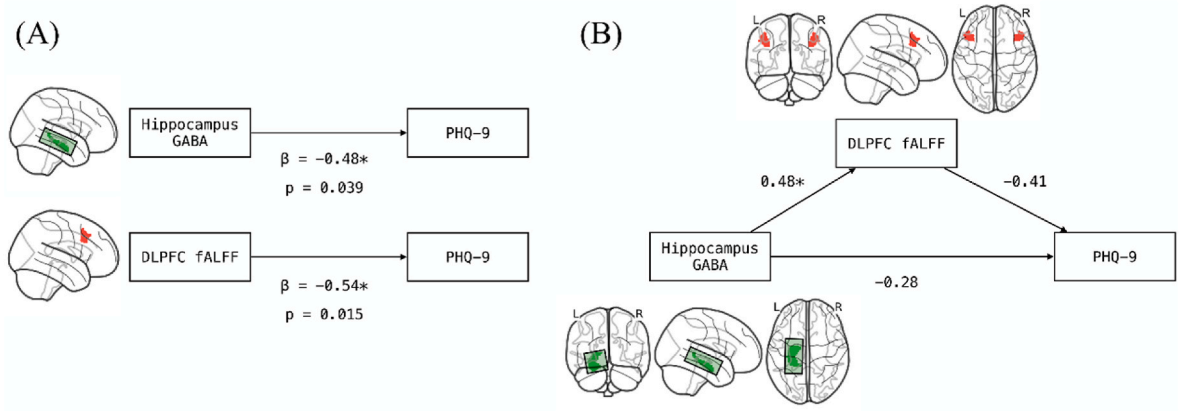

Fig. 2. DLPFC fALFF partially mediated the association between depression scores and hippocampal GABA. The right diagram displays separate linear models, while the left diagram illustrates the mediation analysis model. A) Direct associations between gamma-aminobutyric acid (GABA) in the hippocampus and patient health questionnaire (PHQ-9) scores, and between fractional amplitude of low frequency fluctuations (fALFF) in the dorsolateral prefrontal cortex (DLPFC) and PHQ-9 were estimated. B) The mediation analysis revealed a trend-level mediation effect of DLPFC fALFF on the relationship between hippocampal GABA and PHQ-9. The arrows indicate regression coefficients, and asterisks denote significance association ($p < 0.05$). L indicates the left hemisphere, and R indicates the right hemisphere.

relationship between hippocampal GABA and PHQ-9 scores. Specifically, the results showed a significant relationship between hippocampal GABA and DLPFC fALFF ($\beta = 0.48$, $p = 0.032$, $t = 2.32$) with a trend-level mediation effect between DLPFC fALFF and PHQ-9 ($\beta = -0.41$ $p = 0.084$, $t = -1.84$). In line with a mediation effect of DLPFC fALFF, the direct effect between Hippocampal GABA and PHQ-9 was no longer statistically significant ($\beta = -0.28$, $p = 0.224$, $t = -1.26$) after incorporating DLPFC fALFF into the model.

We did not observe a significant difference between GAHT groups on PHQ-9 scores or on any of the neuroimaging measurements. Although prior work has shown a decrease in mental health symptomatology following GAHT, the lack of group differences in our study is explained by our inclusion criteria which required the presence of depressive symptoms to enable the investigation of the neural correlations of depression [22].

4. Discussion

This study aimed to investigate the multimodal prefrontal-hippocampal circuit in relation to depression in a transgender cohort. Our findings revealed negative associations between depression severity and hippocampal GABA, and negative associations between depression severity and the amplitude of functional fluctuations in the DLPFC. These findings on the prefrontal-hippocampal circuit in depression are consistent with previous research in cisgender subjects, where higher hippocampal GABA was shown to recruit the DLPFC in an unwanted thought suppression task, indicating that DLPFC top-down signaling may depend on hippocampal GABA [11]. Moreover, lower hippocampal GABA may be indicative of impaired inhibitory signaling, which could contribute to hippocampal hyperactivity [23].

Hypoactivity of the DLPFC has previously been observed in major depressive disorder patients [24]. Given the DLPFC's pivotal role in cognitive control and emotion regulation, prior work has suggested that left DLPFC fALFF could serve as a biomarker to predict treatment responses to TMS intervention [25]. Although GABA depletion is commonly observed in depression, GABA also plays a critical role in regulating neurochemical processes, including the hypothalamus-pituitary-adrenal axis and neurosteroid homeostasis [26]. Disruption of GABA signaling in premenstrual dysphoria may suggest abnormal neurochemical responses to hormonal fluctuations, particularly those driven by estradiol [27]. As such, this link between estradiol and GABA may be of interest for future research on interactions between GAHT and depression in the transgender community.

Additionally, our study supports the Hasler & Northoff depression model, which links neurotransmitter imbalances, particularly in GABAergic signaling, to functional dysconnectivity [10]. However, the potential influence of outliers driving changes in the observed relationships highlights the need for further studies with larger sample sizes to validate these findings. Further research is required to explore all potential pathways within this model, including other circuitry like limbic and salience networks and other relevant neurotransmitters such as serotonin and dopamine. The findings of this pilot study provide preliminary validation for the potential use of GABA MRS in tracking symptom severity in this underrepresented population.

Although we were underpowered to detect differences linked to GAHT, GAHT has been shown to reduce odds of depression and suicidality in transgender population [22] and has been used to treat gender dysphoria [28]. Our study did not distinguish between general depression and depression related to gender dysphoria, which could be addressed in future studies. Furthermore, future work is needed to investigate the potential mediating role of hormone therapy in mitigating depression risk in the transgender population.

Aligning with the call for inclusivity in neuroimaging research and the need to address underrepresented populations (Richard et al., 2022), transgender individuals exhibit similar neuroimaging findings but with a higher prevalence than the cisgender population. Inclusion criteria should therefore encompass these underrepresented populations to ensure broader representation in the field. Additionally, large-scale neuroimaging studies in transgender cohorts will also be crucial to further understand the relationship between sex hormones more broadly and neural circuitry dysfunction in depression.

5. Conclusion

We investigated the prefrontal-hippocampal circuit in relation to depression in a transgender cohort using multimodal neuroimaging data. Our results revealed that higher depression severity was associated with reduced hippocampal GABA and with reduced DLPFC fALFF. Larger scale neuroimaging studies are needed to further study the brain basis of depression in the transgender community and the potential impact of GAHT on depression-related neural pathways.

CRediT authorship contribution statement

Setthanan Jarukasemkit: Writing – review & editing, Writing – original draft, Visualization, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Karen M. Tam:** Investigation, Data curation. **Seksan Yoadsanit:** Investigation, Data curation. **Ty Easley:** Writing – review & editing, Conceptualization. **Hailey Modi:** Writing – review & editing. **Lyn Stahl:** Writing – review & editing. **Adun Kampaengtip:** Resources, Project administration, Methodology, Investigation, Data curation. **Thanissara Chansakul:** Supervision, Resources, Investigation, Funding acquisition, Data curation. **Rena Janamnuaysook:** Writing – review & editing, Supervision, Project administration, Conceptualization. **Akarin Hiransuthikul:** Writing – review & editing, Supervision, Conceptualization. **Chaipat Chunharas:** Writing – review & editing, Writing – original draft, Validation, Supervision, Software, Resources, Project administration, Methodology, Conceptualization. **Janine D. Bijsterbosch:** Writing – review & editing, Writing – original draft, Validation, Supervision, Software, Resources, Methodology, Formal analysis, Conceptualization.

Data sharing statement

Anonymized data will be shared on OpenNeuro upon acceptance.

Author disclosure statement

The authors declare no conflict of interest.

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Declaration of competing interest

None.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cpnec.2025.100288.

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