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Frontal alpha asymmetry in major depression and comorbid anxiety disorder: a five-year follow-up study

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ABSTRACT

Objective: Major depression (MD) and anxiety disorders are both associated with higher left compared to right frontal alpha activity (rLFα). The aim of the study was to examine whether young adults with lifetime MD and anxiety disorder differ from healthy controls and whether this pattern remains stable over five years from adolescence into adulthood.
Methods: Resting frontal EEG asymmetry of n = 25 young adults with lifetime MD and anxiety (MDAnx) and n = 26 healthy controls (HC) was compared. Moreover, in a subsample of participants, the stability of frontal alpha asymmetry was analyzed from adolescence to young adulthood via intra-class-correlations.
Results: Participants with MDAnx displayed significantly more rLFα than HCs. Asymmetry showed fair stability over 5 years in the MDAnx group and poor stability in the HC group, the latter driven by increased relative right frontal alpha activity.
Conclusions: Increased rLFα could be a trait marker for comorbid MDAnx. Low stability in the HC group could derive from maturation of cognitive and affective processes, which might be impeded by the presence of lifetime MDAnx.
Significance: Results highlight that EEG asymmetry changes from adolescence to adulthood and could be impacted by lifetime MD and anxiety, irrespective of current symptomatology.

1. Introduction

Among psychiatric disorders in adolescence and young adulthood, major depression (MD) is one of the most commonly diagnosed, with 12-month prevalence rates of up to 17 % (Goodwin et al., 2022). MD often first manifests during adolescence and can lead to adverse outcomes in adulthood, including increased risk of suicide and social maladjustment (Petito et al., 2020). Even if remission from early-onset MD is achieved, early adult functioning still tends to be lower than in young adults with no psychiatric history (Costello & Maughan, 2015).

In addition, about 50 % of MD patients also display comorbid lifetime anxiety (Kessler et al., 2015). Studies show that depression and anxiety can be bidirectional risk factors for one another, with MD predicting later anxiety disorders and vice versa (Jacobson & Newman, 2017). Recurrence rates in both disorders are high, with about 57 % of patients having a recurrence of depressive or anxiety disorders within four years (Scholten et al., 2016). In addition, patients with comorbid depression and anxiety tend to have worse outcomes including more chronic forms of illness, increased hospitalization and increased rates of disability (Hirschfeld, 2001).

This naturally calls into focus the need for both the prevention of MD and anxiety and the identification of risk factors and markers for the course of both illnesses. Aside from sociodemographic factors and self-report assessments of depression and anxiety, neurophysiological measures as measured in the electroencephalogram (EEG) are a promising avenue to determine potential risk factors and markers of mental illnesses in youths (Toenders et al., 2019).

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1.1. Frontal alpha asymmetry as a marker for major depression

When it comes to neurophysiological markers of depression, frontal alpha asymmetry (FAA) is one of the most discussed potential markers of this disorder (Allen & Reznik, 2015; de Aguiar Neto & Rosa, 2019; Ippolito et al., 2022).

Alpha activity describes oscillations in the EEG between 8 and 12 Hz. In previous studies, it has often been interpreted as an inverse measure of cortical activity (Allen et al., 2004a; Bazanova & Vernon, 2014), i.e. larger alpha activity corresponding to a lower cortical activity. This is based on a number of early studies, such as PET studies, demonstrating that alpha frequency in certain areas had a negative relationship with $H_2^{15}O$ perfusion in the same areas (Cook et al., 1998), as well as fMRI studies demonstrating an inverse relationship between alpha and the BOLD signal (Goldman et al., 2002; Laufs et al., 2003).

When looking at hemispheric differences of alpha activity, greater relative right frontal alpha (compared to left frontal alpha; rRFα) is associated with appetitive motivation and approach related affect, while greater relative left frontal alpha activity (rLFα) is connected to behavioral inhibition and negative affective states (for a review, see Reznik & Allen, 2017).

Individuals with MD have been found to exhibit increased rLFα during rest compared to healthy controls (Koo et al., 2017; Xie et al., 2023). This has been found to be largely independent of current MD status or severity (Stewart et al., 2010). However, some recent studies have also called the connection between FAA and MD into question. A multiverse analyses by Kołodziej et al. (2021) over five studies found no evidence for a relationship between FAA and depressive disorders and another review by Kaiser et al. (2018) similarly concluded that the discriminative power of alpha asymmetry to separate MD individuals from healthy controls remains unclear. They specifically argue that ignoring factors, such as medication or comorbidity, could be a reason for the inconsistent findings (Kaiser et al., 2018).

1.2. Influence of comorbid anxiety disorders on frontal alpha asymmetry in depression

To date, few studies specifically investigated the influence of comorbid psychiatric disorders on the relationship between MD and FAA. However, many studies tend to include comorbid individuals and fail to control for comorbidity (Kaiser et al., 2018). As mentioned above, anxiety is a highly common comorbidity in MD, which was often not properly distinguished from MD in previous studies.

This is despite the fact that anxiety can have a substantial effect on FAA. For example, in an at-risk populations of healthy participants with elevated symptoms of depression and anxiety, it was found that both depression and anxiety symptoms were associated with increased rLFα, but the connection was stronger for anxiety (Adolph & Margraf, 2017). A twin-study has also found that FAA might be an endophenotype for both MD and anxiety, with shared genes influencing both the EEG patterns and the disease risk for both disorders, a connection that was most robust in young adult females (Smit et al., 2007).

In a previous study (Feldmann et al., 2018), we compared FAA between adolescents with current depression with and without comorbid anxiety. In this study, we found that only adolescents with comorbid anxiety showed significantly more rLFα than healthy adolescents, while no group differences emerged between adolescents with MD without a comorbid anxiety disorder and healthy controls. This study therefore demonstrated the importance of considering the presence of an anxiety disorder when looking at FAA patters in MD.

Aside from our previous work, there have been few other studies specifically researching FAA in comorbid anxiety and depression. Bruder et al. (1997) found that only the comorbid depression and anxiety group displayed greater rLFα, while the depression-only group displayed increased rRFα. A more recent study found no difference in FAA between a comorbid sample (history of MD with increased levels of anxiety) and healthy controls (Nusslock et al., 2018), however, this study did not determine whether anxiety disorders were present or not. With few available findings and contradictory results, more studies are needed to illuminate the relationship between FAA and comorbid MD and anxiety. Additionally, so far, there have been no studies researching the stability of FAA over the clinical course in MD and comorbid anxiety.

1.3. Stability of frontal alpha asymmetry

In healthy participants, FAA tends to show moderate stability, often measured with intra-class-correlations (ICCs) between two repeated measurement points. The timespans from test to retest vary, although reliabilities do not seem to differ greatly (for a review, see Lopez et al., 2023). As such, studies with shorter follow-ups from seven days to eight weeks found ICCs between 0.52 and 0.61 (Allen et al., 2004b; Koller-Schlaud et al., 2020; Metzen et al., 2022), while studies with longer follow-ups found ICCs between 0.61 and 0.65 (4 months) (Gold et al., 2013; Schneider et al., 2016). All of the cited studies have researched either adults (Gold et al., 2013; Koller-Schlaud et al., 2020; Metzen et al., 2022) or adolescents (Schneider et al., 2016). There are some studies in infancy (with ages between 5 and 12 months and follow-up timespans between 4 and 31 months) that show lower stability likely due to early processes of brain development and maturation (for a review, see Lopez et al., 2023).

One adult study employed a longer follow-up of one to three years (mean = 1.2 years), similarly finding good stability in the whole sample (ICCs between 0.54 and 0.60 for combined eyes-open and eyes-closed conditions) (Vuga et al., 2006). Aside from a healthy control group, this study also included a group of adult participants with a history of adolescent-onset MD, for which ICCs ranged between 0.47 and 0.73. Interestingly, they also found the lowest stability in male participants from the HC group, with ICCs in the 0.28 to 0.46 range. Altogether, they found that depressive symptom severity at the follow-up, as well as change in symptom severity during the follow-up period were unrelated to the stability measure. Other studies have found similar results, with antidepressant treatment, changes in clinical state and MD severity being unrelated to FAA stability (Allen et al., 2004b; van der Vinne et al., 2019).

To date, there have been no studies researching FAA stability in the transition from adolescence to adulthood. The longest follow-up during adolescence that has been researched spans four months (Schneider et al., 2016) and so cannot attest to long-term stability in this age period. This is especially important since adolescence is a time period subject to various developmental changes in social, biological and psychological aspects. Both grey and white matter develop rapidly throughout adolescence, alongside changes in, among others, social cognition, working memory, reward processing and motivation (Foulkes & Blakemore, 2018; Romine & Reynolds, 2005; Symonds et al., 2019), which are partially located in the prefrontal cortex and have been associated with hemispheric asymmetries (Basharpoor et al., 2021; Constantinidis & Luna, 2019; Papousek & Schulter, 2004; Rubia et al., 2006). As studies in early childhood suggest that FAA can show lower stability due to brain maturation processes, it would be important to research whether these developmental aspects in adolescence also have an effect on FAA stability. In addition, as FAA stability has also not been researched in adolescence in patients with MD and anxiety, it is unclear whether the presence of these psychopathologies would interact with these developmental processes.

1.4. Frontal alpha asymmetry as a predictor of clinical outcomes

While FAA does not seem to be changed by treatment of the underlying symptomatology, baseline FAA might nonetheless serve as a predictor of treatment response (Allen & Reznik, 2015). One study found that women with higher initial rRFα were more likely to show a favorable response to an SSRI, unrelated to whether comorbid anxiety was

present or not (Arns et al., 2016). In a second study, when the FAA measured eight weeks after the treatment was used to predict treatment response, it was still equally predictive as FAA measured at baseline, suggesting FAA to be a stable trait marker not influenced by changes in disease state (van der Vinne et al., 2019). It has also been found to be predictive of MD-onset; in previously healthy participants, lower rRFα at baseline was associated with higher depressive symptoms at a one-year follow-up, even when controlling for depressive symptoms at baseline, suggesting that FAA might be a risk factor for the development of MD (Stewart & Allen, 2018). However, these studies have again not taken comorbidities into account and it is unclear how their results could have been influenced by the presence of comorbid disorders, as there have been previous findings that specifically anxiety, rather than depressive symptoms, might be predicted by FAA (Blackhart et al., 2006).

As of yet, it has not been researched whether FAA could also be a predictor of the course of illness in currently or previously afflicted patients. However, other neurophysiological markers, such as a reduced P300 amplitude in the EEG (Santopetro et al., 2021) or functional connectivity measures between relevant brain networks in the fMRI have been found to be predictive of a poorer course of MD (e.g. increased depressive symptoms later on) (Pilmeyer et al., 2024), which highlights the relevance of investigating neurophysiological markers that relate to MD concerning their possible predictive power.

1.5. Aim and hypotheses

The aim of this project was threefold: First, extending our previous study (Feldmann et al., 2018) to a cross-sectional sample of young adults, we sought to research whether there would be a difference in FAA between the participants with lifetime MD and comorbid anxiety and healthy controls, irrespective of current symptomatology. Second, we aimed to research for the first time the long-term stability of FAA over five years from adolescence into young adulthood in both groups. Third, we sought to find out whether FAA can serve as a predictor for the clinical course over the time span of five years. Aims two and three were assessed in a longitudinal study design, within a subgroup of participants, who had previously taken part in our study on FAA in adolescent MD five years earlier (Feldmann et al., 2018).We posed the following hypotheses:

1) Based on previous findings in adult populations with both MD and anxiety (Koo et al., 2017; Moscovitch et al., 2011; Xie et al., 2023), we would expect that, cross-sectionally, the young adults with lifetime MD and comorbid anxiety disorder would display increased rLFα compared to the healthy control group.
2) When it comes to the stability of FAA, the longest follow-up period in adolescence was at four months (Schneider et al., 2016), in adulthood at 1–3 years (Vuga et al., 2006), with no previous studies researching a follow-up timespan as long as in this study, or in the transition from adolescence to adulthood. Based on this limited data, however, we would expect that FAA would similarly remain stable in the HC and MD with comorbid anxiety groups over the course of five years from adolescence into young adulthood.
3) As increased rLFα is a risk factor for both MD development (Stewart & Allen, 2018) and for an adverse treatment response (Allen & Reznik, 2015), we would expect that in participants with lifetime MD and comorbid anxiety, increased rLFα five years ago would be predictive of an adverse course of MD and increased anxiety at the current measurement.

2. Methods

2.1. Participants

Within this study, two samples were analyzed: (1) A cross-sectional sample of young adults, who took part in the study between February 2021 and April 2022 and (2) a subsample of these participants who had additionally taken part in a study on FAA in adolescents with MD and comorbid anxiety five years earlier (Feldmann et al., 2018).

2.1.1. Whole sample

In total, $N = 51$ participants aged 18–24 were part of the whole sample for cross-sectional analysis. Of these, $n = 26$ participants had never been diagnosed with a psychiatric disorder (HC group) and $n = 25$ had a history of both MD and comorbid anxiety disorder (MDAnx group). Participants were either contacted based on their participation in the previous study (Feldmann et al., 2018) or recruited via flyers and the Department website.

2.1.2. Subsample with longitudinal data

Of the $N = 51$ participants a subsample of $n = 44$ participants had previously participated as adolescents in a study (Feldmann et al., 2018) at the Department for Child and Adolescent Psychiatry, Psychosomatics and Psychotherapy at the LMU University Hospital in Munich. Details of the procedures and materials of the previous participation can be found in the related publication (Feldmann et al., 2018). Of these $n = 44$ participants, $n = 6$ participants had to be excluded from the longitudinal analysis as they did not fulfil an MDAnx diagnosis five years earlier. As such, the final longitudinal sample includes $n = 38$ participants, $n = 19$ in the HC and $n = 19$ in the MDAnx group.

Sample characteristics for the subsample with longitudinal data can be found in Supplementary Table 1. For a comparison of the participants who participated in the current study and those who were contacted, but did not participate, please refer to Supplementary Tables 2 and 3. It should be noted that those MDAnx participants who took part in the current study and those who were invited but did not participate differed significantly in their depressive symptomatology 5 years earlier, with those who did not return displaying higher symptomatology.

2.1.3. Study procedure

Participants were invited to two study sessions: One diagnostic session, in which the diagnostic interview (for details, see Materials section) was applied (ca. 1.5–2 h) and one experimental session, in which the resting EEG was recorded. Participants received 50€ in the form of vouchers for their participation at the current measurement point which included the current resting state paradigm as well as an emotion regulation paradigm (Zsigo et al., 2024) and two short paradigms on selective attention; participants who had previously taken part five years ago were thus compensated separately for participation in the initial assessments (Feldmann et al., 2018). All study procedures were approved by the ethics committee of the Medical Faculty of the LMU Munich. Participants were informed about all procedures and aims of the study and gave written informed consent.

Inclusion criteria for the MDAnx group were the diagnosis of a lifetime disorder of major depression and a lifetime anxiety disorder

Table 1
Sample characteristics of the whole sample.

	HC ($n = 26$)	MDAnx ($n = 25$)	Test	
			t	*p*
Age in years (*M, SD*)	20.54 (1.70)	21.24 (1.62)	1.51	0.138
Age range	18–23	18–24		
Sex (% female)	76.90	80.00	0.07[a]	0.789
Handedness (% right-handed)	92.31	84.00	0.85[a]	0.367
BDI-II score (*M, SD*)	2.73 (2.31)	16.20 (12.00)	5.62	<.001
STAI State (*M, SD*)	30.42 (3.35)	39.64 (0.41)	4.70	<.001
STAI Trait (*M, SD*)	32.28 (5.26)	49.08 (11.98)	6.42	<.001

Note: HC = healthy control, MDAnx = lifetime major depression and anxiety disorder, *M* = mean, *SD* = standard deviation, BDI-II = Beck's Depression Inventory II, STAI = State Trait Anxiety Inventory.
[a] Pearson's Chi square statistic.

(specific phobia, agoraphobia, social anxiety disorder, generalized anxiety disorder, panic disorder, or separation anxiety disorder) according to the ICD-10 (World Health Organization, 1992). For details on how diagnoses were determined, see 2.2 Materials. Exclusion criteria for MDAnx group were comorbid lifetime pervasive developmental disorder, bipolar disorder or schizophrenia; other comorbidities were permitted. $N = 10$ participants showed a current comorbidity at the time of the experiment, with the most common being PTSD and OCD. At the time of the recording, $n = 9$ participants were taking continuous psychopharmacological medication (7 of those an SSRI with one using antipsychotic medication for augmentation, 1 an SNRI, 1 a MAOI). For a detailed breakdown of the participants' current and past medication, refer to Supplementary Table 6. The pattern of our results did not change upon exclusion of these participants, so the analyses detailed below include participants with psychotropic medication.

For the HC group, only participants who never met criteria for a psychiatric disorder according to the ICD-10 diagnostic criteria were included. All participants in the HC group had a BDI-II (Beck's Depression Inventory II) score < 9, which according to the BDI-II manual (Hautzinger et al., 2006) corresponds to no depression. The two groups were comparable in age and sex. Characteristics of the cross-sectional sample can be found in Table 1, for the longitudinal subsample in Supplementary Table 1.

Participants were only included in the study if they had an IQ ≥ 85, which was established via the CFT-20-R (Culture Fair Intelligence Test; Weiß, 2019) or other established IQ measures such as the WIE (Wechsler Adult Intelligence Scale, German: "Wechsler Intelligenztest für Erwachsene"; von Aster et al., 2006).

2.2. Materials

Diagnoses of psychiatric disorders were assessed according to the ICD-10 via the adult version of the Diagnostic Interview of Psychiatric Disorders (German: "Diagnostisches Interview psychischer Störungen", DIPS), which is a well-established German semi-structured clinical interview (Margraf et al., 2017; Schneider & Margraf, 2005).

Also, self-reported depressive symptomatology was measured via the German version of the Beck's Depression Inventory II (BDI-II; Hautzinger et al., 2006) and self-reported state and trait anxiety with the German version of the State-Trait-Anxiety-Inventory (STAI; Laux et al., 1981).

2.3. EEG recording, preprocessing and analysis

The EEG procedure lasted eight minutes total with one-minute blocks of an eyes-open (eO) and eyes-closed (eC) condition presented in one of two counterbalanced orders (COOCOCCO or OCCOCOOC) (see Feldmann et al., 2018; Stewart et al., 2010). Eight minutes of EEG activity has been previously reported to provide an excellent reliability in the context of asymmetry measures (for a review, see Hagemann, 2004).

Participants were seated in front of a computer screen, with light conditions kept constant across participants. The instructions "open your eyes" and "close your eyes" were both displayed on the computer screen and read aloud by the experimenter. During the eO condition, participants were instructed to look at a small fixation cross in the middle of a computer screen to avoid eye-movements. All experimental and diagnostic sessions were conducted by one experimenter with a master's degree in clinical psychology.

The EEG was recorded with a 128-channel system from Electrical Geodesics Inc. During recording, sampling rate was set to 500 Hz and Cz was used as a reference electrode. Impedances of all electrodes were kept at 50 kΩ or lower. In case of faulty electrodes, the channel was interpolated using signal from surrounding electrodes. Preprocessing and analysis of EEG data was done via BrainVision Analyser, version 2.2 from Brain Products GmbH (Gilching, Germany).

For raw data, an 8th order IIR Butterworth filter was set to a low cutoff of 0.16 Hz, a high cutoff of 40 Hz, a notch filter of 50 Hz and a 47 dB/oct roll-off. Artefacts were visually inspected and removed via a non-automatic Independent Component Analysis (ICA), which was performed by a trained person. Within the identified independent components, any major abnormality derived from electro-oculographic (EOG) artefacts, cardiac artefacts, electrodermal and other non-ocular muscular activity removed. Following ICA, remaining artefacts were removed automatically in individual channels with following settings: gradient max. 40 μV/ms, max–min 200 μV/ms for 200 ms windows, max amplitude 150 μV, min amplitude −150 μV, low activity 0.5 μV for 100 ms windows (see also Feldmann et al., 2018).

Following ICA, further analyses were performed within the channels of two regions of interest on the left (electrodes 18, 19, 20, 23, 24, 27) and right hemisphere (electrodes 3, 4, 10, 118, 123, 124), which are the same regions of interest used in previous studies of FAA on a 128-channel recording system (e.g. Feldmann et al., 2018; Gabard-Durnam et al., 2015; Zsigo et al., 2024). Sensor layout and ROI placement is also shown in Fig. 1.

Data were then re-referenced to the average of all electrodes (AVG). Previously, when investigating FAA, referencing to current source densities (CSD) has been recommended (Stewart et al., 2014), however, newer *meta*-analyses have called the utility of CSD in the context of FAA into question, stating that CSD might even results in lower probability of detecting differential effects due to the high variability of scalp topographies across individual subjects (Kołodziej et al., 2021). We therefore report the main results of the manuscript with an AVG reference. For the interested reader, results with CSD-referenced data can be found in Supplementary Table 5.

Then, data were segmented into the eyes-open (eO) and eyes-closed (eC) conditions. Only participants with at least 80 s of artifact-free data in each condition were included in the analysis, as this was shown to achieve good reliability of FAA (Towers & Allen, 2009). No participant had to be excluded due to this criterion. In the whole sample, there were no significant differences of the length of data included between the MDAnx and HC groups, in either the eO or the eC condition ($ps > 0.28$).

The signal was again segmented into 2.048 s-epochs with 50 % overlap, upon which we applied a Fast Fourier Transformation (FFT) to obtain spectral power, at a resolution of 0.5 Hz with a Hanning window (see also Smith et al., 2017). Data were then exported with alpha spectrum being defined as 8–13 Hz. Finally, data were log-transformed with the natural logarithm and values were averaged across the two regions of interest. This has been previously recommended in the literature (Allen et al., 2004a; Smith et al., 2017) in order to improve the distributional characteristics of the data, as untransformed alpha values tend to be positively skewed.

2.4. Statistical analysis

All analyses were performed with IBM SPSS Statistics version 29.0.0.0. For FAA measures, a laterality index (ln(right ROI)-ln(left ROI)) was computed for each participant (Feldmann et al., 2018; Stewart et al., 2010). As there was no significant main effect of or interaction with eye status (eyes open vs. eyes closed; details included in Supplementary Table 4) data are presented averaged over eyes open and eyes closed conditions. Details on power calculation can be found in the Supplement (Supplementary Information: Power Calculation).

2.4.1. Whole sample

First, we conducted the cross-sectional analysis, comparing the laterality index of the HC group and the MDAnx group, via a *t*-test. If groups differed significantly, we also investigated the role of the individual hemispheres in the group differences by conducting a 2 (Right Hemisphere, Left Hemisphere) x 2 (HC, MDAnx) repeated measures ANOVA. In case of a significant interaction, we conducted follow-up *t*-tests, comparing group differences within hemispheres, as well as hemisphere differences within groups. For these follow-up tests, a

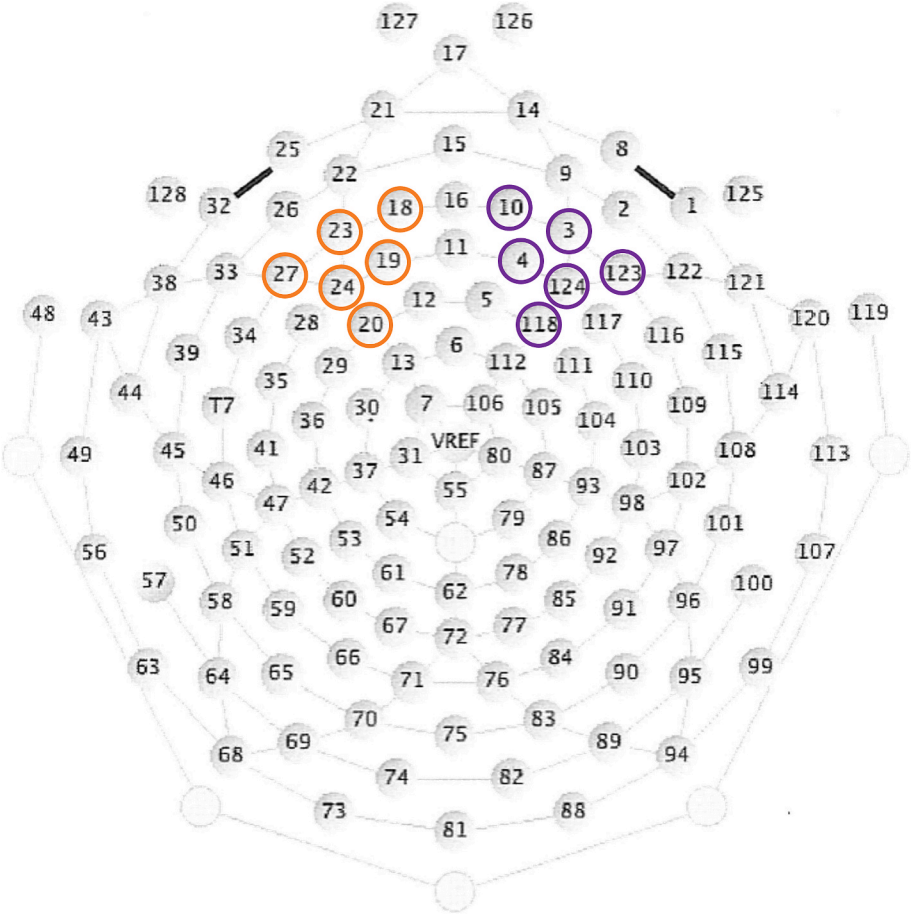

Fig. 1. Sensor layout of the Electrical Geodesics Inc. 128-channel system with the right ROI marked in purple (dark) and the left ROI marked in orange (light).

corrected alpha level of $\alpha = 0.05/4 = 0.013$ was considered. Within the MDAnx group, we also analyzed whether the laterality index correlated with self-reported depressive symptoms and state and trait anxiety, again considering a corrected alpha level of $\alpha = 0.05/3 = 0.016$.

2.4.2. Subsample with longitudinal data

Second, we determined stability of the FAA between adolescence (5 years prior, mean = 5.40 years) and young adulthood within the subsample with longitudinal data. As has been previously applied in other stability studies (Lopez et al., 2023) and recommended in current guidelines (Koo & Li, 2016), we determined stability using Intra-Class-Correlations (ICCs). As Koo et al. recommend for test–retest analyses, we applied a two-way mixed effects model with absolute agreement. We report results based on a single measurement, as has been applied in several other studies researching FAA stability (e.g. Gold et al., 2013; Koller-Schlaud et al., 2020; Metzen et al., 2022) and was recommended when EEG asymmetry is likely to change across assessments due to changes over time and in clinical status (Allen et al., 2004b). Regarding the interpretation, a review by Lopez et al. (2023) has summarized the greatest consensus as the following thresholds: <.40 = poor, 0.40-.59 = fair, 0.60-.74 = good and > .75 = excellent.

Third, in order to determine whether the laterality index can serve as a predictor for clinical variables, we conducted a prospective analysis of the MDAnx participants in the subsample with longitudinal data. As such, a linear regression was modelled to compute whether the laterality index, as measured five years earlier in the Feldmann et al. (2018) study, can be a predictor for the time participants were depressed the following five years, in months. Additionally, a second linear regression was modelled to calculate whether the laterality index five years ago is a predictor for the amount of MD episodes participants experienced in the following five years. Finally, we also calculated a linear regression determining whether the laterality index five years ago could predict an individual's current trait anxiety.

3. Results

3.1. Whole sample

The laterality index significantly differed between the two groups, with the MDAnx group displaying lower laterality scores than the HC group ($t(49) = -2.86$, $p = 0.006$, $d = 0.80$), i.e. the MDAnx group showing less rRFα than the HC group (see also Fig. 2 and Table 2).

The 2 (group) x 2 (hemisphere) repeated measures ANOVA to determine the effects of the individual hemispheres (i.e. the left and right ROIs) revealed no significant main effects of group ($F(1, 49) = 0.07$, $p = 0.797$, $\eta^2p = 0.001$) or hemisphere ($F(1, 49) = 0.91$, $p = 0.346$, $\eta^2p = 0.018$), but a significant group*hemisphere interaction ($F(1, 49) = 8.187$, $p = 0.006$, $\eta2p = 0.143$). Following up on the significant interaction, we found that the two groups did not differ in alpha activity on the left ($t(49) = 0.05$, $p = 0.96$, $d = 0.01$) or on the right hemisphere ($t(49) = -0.56$, $p = 0.57$, $d = -0.16$). In the MDAnx group, alpha activity in the two hemispheres did not differ ($t(24) = 1.19$, $p = 0.246$, $d = 0.24$), however, the HC group displayed significantly less left-frontal than right-frontal alpha; $t(25) = -3.15$, $p = 0.004$, $d = -0.62$).

Regarding the correlations in the MDAnx group, the laterality index did not correlate significantly with state anxiety ($r(25) = 0.001$, $p = 0.99$), trait anxiety ($r(25) = -0.08$, $p = 0.69$) or depressive symptom severity ($r(25) = 0.14$, $p = 0.50$).

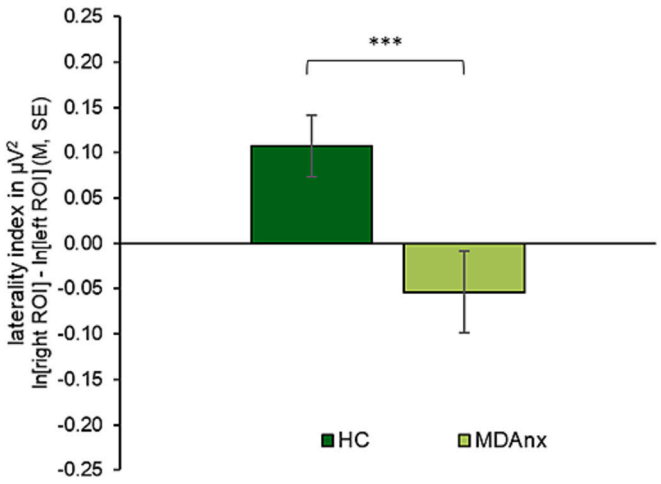

Fig. 2. Mean laterality index (ln[right ROI] − ln[left ROI]) in μV^2 in the HC and MDAnx groups. A positive up-graph represents greater right (than left) frontal alpha activity, while a negative down-graph represents greater left (than right) frontal alpha activity. Error bars indicate the standard error of the mean.

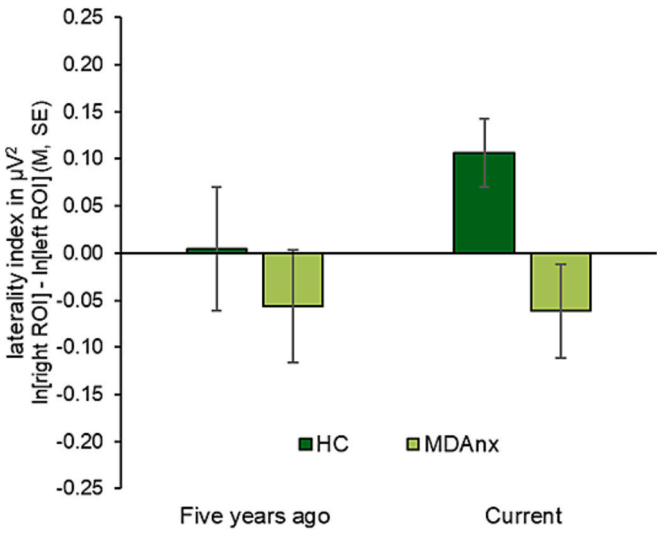

Fig. 3. Mean laterality index (ln[right ROI] − ln[left ROI]) in μV^2 in the HC and MDAnx groups, five years ago and currently. A positive up-graph represents greater right (than left) frontal alpha activity, while a negative down-graph represents greater left (than right) frontal alpha activity. Error bars indicate the standard error of the mean.

Table 2
Means and standard deviations of the laterality indices in the whole sample and the subsample with longitudinal data.

Whole Sample	HC (n = 26)	MDAnx (n = 25)
Laterality Index Current (*M, SD*)	0.108 (0.174)	−0.054 (0.226)
Longitudinal Subsample	HC (n = 19)	MDAnx (n = 19)
Laterality Index Current (*M, SD*)	0.107 (0.159)	−0.061 (0.217)
Laterality Index Five years ago (*M, SD*)	0.004 (0.287)	−0.057 (0.261)

Note: HC = healthy control, MDAnx = lifetime major depression and anxiety disorder, *M* = mean, *SD* = standard deviation. Laterality index calculated as (ln[right ROI] − ln[left ROI]), in μV^2.

3.2. Subsample with longitudinal data

3.2.1. Stability analysis

Within the MDAnx group, a fair degree of stability was found for the FAA over 5 years. The single measures ICC was 0.442 with a 95 % confidence interval from −0.020 to 0.743 ($F(18,18) = 2.50$, $p = 0.030$). Within the HC group, a poor degree of stability was found, with a single measures ICC of 0.039 with a 95 % confidence interval from −0.383 to 0.463 ($F(18,18) = 1.09$, $p = 0.432$).

For descriptive purposes, we have included the mean laterality indices of the two groups at the two measurements in Fig. 3 and Table 2. The fair stability in the MDAnx group is descriptively driven by a stable, dominant rLFα, while the poor stability of the HC group is driven by an increase in rRFα over the five-year period.

3.2.2. Prospective analysis

The regression model with the laterality index from five years earlier predicting the time participants (who had already been diagnosed with MD and anxiety five years ago) were depressed in the follow-up period (in months) was not significant ($F(1, 17) = 0.26$, $p = 0.617$). Similarly, the regression model with the laterality index from five years earlier predicting the number of MD episodes in the follow-up period was also not significant ($F(1,17) = 0.02$, $p = 0.898$). The laterality index from five years earlier was also not a predictor for current trait anxiety at ($F(1,17) = 0.60$, $p = 0.449$).

4. Discussion

This project measured for the first time whether young adults with lifetime MD and comorbid anxiety differed in FAA from healthy controls. We found that lifetime MDAnx young adults displayed increased rLFα compared to healthy controls. Within a subsample with longitudinal data, FAA of MDAnx participants showed fair stability over a period of five years from adolescence to young adulthood. In HC participants, this stability was poor, which descriptively corresponded to an increase in rRFα. Among the MDAnx participants, FAA was not predictive of the number of MD episodes and time spent depressed in the five-year follow-up period or of trait anxiety five years later.

4.1. FAA in young adulthood in MDAnx versus HC participants

First, we have confirmed our hypothesis that the MDAnx group would show more rLFα (and, conversely, less rRFα) compared to the HC group in our cross-sectional analysis. This is in line with several previous studies, which have connected rLFα to both MD and anxiety separately (Koo et al., 2017; Moscovitch et al., 2011; Xie et al., 2023), as well as with our own previous publication of the adolescent data (Feldmann et al., 2018), where we also found more rLFα in MDAnx participants compared to HCs but not in participants with MD without comorbid anxiety disorder.

When looking at these results in the context of other studies of asymmetry in the human brain, NIRS (near infrared spectrostopy) and PET studies also find a relative hypoactivity in left areas in MD patients, both during tasks and during rest (Davidson & Henriques, 2000; Nitschke et al., 2004; Ohta et al., 2008). In one study in social phobia, increased right-sided activation was also found specifically in anticipating a phobic event (Davidson et al., 2000), which corroborates this pattern of activation for anxiety as well.

When considering that alpha activity has been found to be inversely correlated with both the fMRI and PET measured brain activity level (Allen et al., 2004a; Cook et al., 1998; Goldman et al., 2002; Laufs et al., 2003), the increased leftward alpha activity in our MDAnx patients is well in line with these previous findings, as they all point toward a hypoactivation of the left hemisphere or, conversely, a hyperactive right hemisphere in MD patients. This has often been discussed to be due to the right hemisphere being generally more attuned to fear-eliciting stimuli and the experience of negative emotions, while motivation and pleasure are typically associated with the left hemisphere (for a review, see Hecht, 2010). This can also be brought in line with previous EEG

research, which has also found a connection between rLFα and avoidance, withdrawal motivation and negative affect (Reznik & Allen, 2017).

As such these results can be seen as neurophysiological measures mirroring the motivational and affective states of both MD and anxiety. Both disorders have been previously associated with an increase in trait avoidance and withdrawal motivation (Struijs et al., 2017), with avoidance tendencies also having been found to be a risk factor for the development of both disorders (Struijs et al., 2018).

Interestingly, this is despite the fact that our participants were not required to have current MD and anxiety diagnoses, but were also included if they had displayed MD and comorbid anxiety in the past. It has previously been found in MD that rLFα was present in adults in remission (Henriques & Davidson, 1990; Stewart et al., 2010), although the same has not been researched for anxiety. Our results provide evidence for the trait hypothesis of FAA, specifically suggesting that rLFα might be a trait marker for comorbid MDAnx independent of current symptomatology.

One question that follows is if the results are driven by the comorbidity of both disorders or simply the presence of an anxiety disorder, independent of the MD diagnosis. As summarized in the introduction, studies have found a connection between rLFα and anxiety (Adolph & Margraf, 2017; Moscovitch et al., 2011; Smit et al., 2007), but as of yet, there have been no direct comparisons between a comorbid MDAnx group and a "pure" anxiety group. However, it has been previously found that comorbid MD in anxiety and comorbid anxiety in MD are both associated with worse outcomes such as increased severity, chronicity, suicidality and worse psychosocial impairment (Pollack, 2005).

This would also explain why we have found significant differences despite studies previously calling the connection between FAA and MD into question (Kaiser et al., 2018; Kołodziej et al., 2021). As Kaiser et al. (2018) mention, one reason for the inconsistency in the association between FAA and MD might be due to many studies not discriminating between various comorbidities their participants might have had. Therefore, it is possible that the focus of this study on MD and comorbid anxiety might have identified a specific subgroup for which rLFα is a relevant marker.

Another possibility that should be considered is that differences in alpha activity could stem from neurobiological differences between participants with MDAnx and HCs. So far, research on whether there are structural differences in cortical volume in frontal areas between MD patients and HCs is inconclusive. Some studies point toward a lower volume in right hemisphere areas, such as areas involving the dorsolateral prefrontal cortex (DLPFC) (Liu et al., 2016; Zuo et al., 2019), while others, such as an analysis of the ENIGMA Consortium, found no significant altered macro-anatomical asymmetries in MD (de Kovel et al., 2019). In the future, studies should also consider structural changes possibly underlying activity asymmetries. Along the same vein, it might also be interesting to look at changes in alpha activity not only in frontal, but also for example parietal regions as previous studies have found a connection between parietal alpha asymmetry and MD (Jaworska et al., 2012; Umemoto et al., 2021).

It should also be considered that MD is associated with a variety of cognitive changes, such as impaired working memory, attention and executive function (Marazziti et al., 2010; Rose & Ebmeier, 2006), which might have in turn had an effect on neurophysiological markers such as alpha activity. Indeed, previous studies have found that FAA can be sensitive to changes in working memory, as for example in both verbal and visual working memory studies, participants displayed active rRFα during task completion (for a review, see Pavlov & Kotchoubey, 2022). In addition, the rate of event-related desynchronization between alpha and beta is directly tied to working memory demands (Erickson et al., 2019). As MD can lead to lasting cognitive deficits (Hammar et al., 2003; Semkovska et al., 2019), and, it would be interesting for future studies to consider an active cognitive or working memory paradigms including the assessment of behavioral data, in addition to the resting state to assess the influence of different active tasks on FAA.

Similarly, the effects of medication should also be investigated. While excluding medicated participants did not change the pattern of our results, past studies have found that FAA can predict responses to different SSRIs (e.g. Arns et al., 2016; van der Vinne et al., 2019) and that medication can influence frequency and localization of the alpha band (e.g. Knott et al., 2002), so the interaction between alpha and antidepressant medication presents an interesting field of study.

Finally, it should also be discussed that most of our sample consisted of female participants. There are studies that have shown that FAA can vary across the menstrual cycle (Hwang et al., 2008; Hwang et al., 2009; but see Solis-Ortiz et al., 1994) and that alpha power in general can vary with levels of estrogen / estradiol and progesterone (Becker et al., 1982; Brötzner et al., 2014). To our knowledge, there are no previous studies investigating the effect of the menstrual cycle on frontal alpha asymmetry in major depression. It would therefore be interesting to include the current phase of the menstrual cycle and / or blood hormone levels in future studies to control for this influence in a sample like ours.

4.2. Stability and predictive value of FAA from adolescence into young adulthood

This study is also the first to research the stability of FAA over five years in a sample in the transition from adolescence to adulthood from ages 18 to 24. First, in the HC group, we found poor stability. As detailed in our introduction, most of the previous studies researching FAA stability in healthy populations find higher ICCs between two sessions of repeated measurements. However, to date, there has been no investigation into FAA specifically in the transition from adolescence into young adulthood and previous follow-ups have lasted no longer than one to three years (Vuga et al., 2006).

Interestingly, in our results, the lower stability specifically in the HC group was descriptively driven by an increase of rRFα from adolescence to adulthood. Developmental studies of FAA mostly focus on early infancy (see e.g. Anaya et al., 2021; Vincent et al., 2021) and so have not investigated changes in participants coming into adulthood. There has been one study looking cross-sectionally at younger and older adults (i.e. 18–35 vs. 60 + years of age), finding that older adults generally display more rRFα than young adults (Barros et al., 2022), which, in combination with our results, could hint toward a general increase in rRFα through life.

When looking at differences between these specific age groups, previous studies have mostly found a decrease in overall alpha, without investigating laterality (Howsley & Levita, 2018). In addition, in both global and local white matter networks in a MRI study, asymmetry, specifically rightward asymmetry, has been found to be higher in adolescence than in young adults, which the authors associate with maturation of language and social cognition (Zhong et al., 2017).

This is also mirrored by other changes in cognitive function, which may develop throughout and post adolescence, such as some aspects of executive functioning. Planning and verbal fluency, for example, have been found to continue to improve in performance beyond 17 years of age and into the early adulthood period (Romine & Reynolds, 2005). These have been previously associated with hemispheric localization, such as increased coherence (i.e. the synchronicity between two electrodes) of EEG frequency bands in the left hemisphere being associated with executive function (Basharpoor et al., 2021) and increased activation in left frontal regions being predictive of better performance on verbal fluency tasks, both when measuring fMRI (Papousek & Schulter, 2004) and EEG activity (Hoptman & Davidson, 1998). Additionally, inhibitory control is often worse in adolescence than in adults, the increase in performance often being associated with increased activation in left hemispheric networks and areas (Constantinidis & Luna, 2019; Rubia et al., 2006).

In addition, other associated processes also develop throughout this time period, including those that have been previously associated with

FAA. As such, transitioning from adolescence into adulthood has been connected to an increase in the behavioral approach system, motivation and better emotion regulation (Symonds et al., 2019; Urošević et al., 2012; Zimmermann & Iwanski, 2014) – all of which have also been associated with right-frontal alpha activity (Bazanova & Vernon, 2014; Reznik & Allen, 2017). In summary, the lack of stability in FAA in the HC group could be influenced by a number of factors corresponding to important maturation processes from adolescence into adulthood.

However, we also found that stability was markedly higher in the MDAnx group, displaying fair stability compared to the poor stability of the HC group, suggesting that similar developmental processes may be impeded and / or delayed by the presence of lifetime MD and comorbid anxiety diagnoses. It is well known that MD is associated with deficits in the aforementioned areas, such as working memory and cognitive control (for a *meta*-analysis, see Rock et al., 2014). In a sample of MD patients, increased scores of anxiety have been previously associated with increased impairment in executive function performance (Liu et al., 2020) and, when comparing comorbid MDAnx patients with MD-Only patients, increased executive function and psychomotor slowing has been found (Basso et al., 2007).

While there are no studies looking at whether MDAnx specifically impedes development of the mentioned domains, it could be hypothesized that MD and anxiety might impede developmental processes, leading to deficits in areas that mature during adolescence (including the areas mentioned above), which could be an explanation of higher stability in the MDAnx group. For comparison in a structural imaging study, younger adolescents with depression, unlike healthy adolescents, did not show a decrease in grey matter volume, which is usually interpreted as a maturation process (Straub et al., 2019). However, to confirm this hypothesis, future studies would need to concurrently measure development of FAA and the factors listed above such as working memory and investigate their influences upon one another in the context of MDAnx.

The increased stability in the MDAnx group does have interesting implications. Even in young children, stable rLFα has been associated with increased avoidance and decreased autonomy in previous studies (Poole et al., 2018) and in healthy adults, increased rLFα predicts higher depressive symptomatology (Stewart & Allen, 2018) and a first depressive episode (Nusslock et al., 2011). It has also been shown to be a predictor of treatment response (Allen & Reznik, 2015; Arns et al., 2016).

In our study, we did not find such a connection. FAA during adolescence in the MDAnx group was not predictive of the included clinical markers (time depressed, number of MD episodes during the five-year follow-up and trait anxiety at follow-up). To our knowledge, this is the first study to research the predictive power of FAA on the course rather than the onset or treatment outcome of MD. As we found no effects, it is possible that FAA might not have predictive power to determine course or outcome of MD or anxiety. It should be considered however that the sample used to calculate this regression analysis was small, with only 19 participants in the MDAnx group for which data was available from five years ago, so the results should be replicated in a larger sample to draw reliable conclusions.

4.3. Strengths and limitations

An important strength of our study is the use of longitudinal data spanning over five years in a very important developmental age span from adolescence into adulthood. In addition, this was measured in two distinct groups, with one control group that was never diagnosed with any psychiatric disorder and a clinical group of participants who had at any point in their lives suffered from MD and comorbid anxiety. Diagnoses of these disorders were also determined via a semi-structured interviews conducted by a clinical psychologist, leading to a clinically well-characterized sample. The specific inclusion of only patients with lifetime MD and comorbid anxiety should also be highlighted, as comorbidities have often been insufficiently considered in the study of MD.

However, there are also limitations to consider. One is that we only contrasted a mixed MD and anxiety group with a healthy control group. We chose this design based on the results of our previous work (Feldmann et al., 2018), where we found no differences between "pure" MD and healthy control groups and therefore only focused on MDAnx in this project. However, a design with anxiety-only, MD-only, comorbid MD and anxiety and healthy control groups could further illuminate whether anxiety or comorbid MDAnx is the determining factor in our results. Additionally, the sample size in our longitudinal sample, with only 19 participants per group, is rather small, which is why a replication of our results in a larger sample would be of great importance. Finally, concerning the longitudinal sample, those who participated in the current study had displayed significantly less depressive symptomatology at the earlier assessment than those who were invited but did not participate. This is a common occurrence in studies of patients with depression and anxiety (e.g. Christensen et al., 2009; Eskildsen et al., 2010), however, this could limit the generalizability of our results onto particularly severely impacted patients.

4.4. Conclusions

The findings of this study show that young adults with lifetime MD and anxiety display more rLFα than young adults with no lifetime diagnosis of a mental disorder, which could represent increased negative affect and withdrawal motivation in the MDAnx sample. Over five years, from adolescence into adulthood, FAA does not remain stable in the control group, where rRFα increases, which could be due to processes of brain maturation in this age span. In the MDAnx group, meanwhile, fair to moderate stability was found, which could be due to the psychopathologies impeding normative development. These findings provide important insights into FAA and the fact that it is subject to developmental changes especially in the crucial period of transition from youth to adulthood. Future studies should further expand the study of FAA across the lifespan both in normal development but also in clinical groups, especially with a focus on the effects of comorbidity.

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Ethics: All included projects were approved by the ethics committee of the Medical Faculty of the LMU Munich and all procedures were in accordance with the latest version of the Declaration of Helsinki.

Consent: All participants were informed in detail about the procedures and the aims of the study and provided written informed consent.

Contributions: L.F., C.Z., G.S.K. and E.G. contributed to the study conception. C.Z. performed data collection and analyzed the data. L.F., C.Z., E.G., G.S.K., and J.B. contributed to the interpretation of the data. The manuscript was written by C.Z. under supervision of L.F. L.F., J.B., E.G. and G.S.K. commented on the manuscript draft. All authors read and approved the final manuscript.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.clinph.2025.2110794.

Data availability

Data will be made available on request.

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