Enabling #EEGManyLabs: Quality in Automatically Preprocessed EEG Data and Psychopathological Associations of Frontal Alpha Asymmetry

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III List of Abbreviations

EEG	electroencephalography
QRP	questionable research practices
DF	researcher degrees of freedom
RS	resting state
MRI	magnetic resonance imaging
MDD	Major Depressive Disorder
GAD	Generalized Anxiety Disorder
COVID-19	Sars-2-COVID-19
STAI	State Trait Anxiety Inventory
PSWQ	Penn State Worry Questionnaire
EHI	Edinburgh Handedness Inventory
LQ	laterality quotient
BIS/BAS	behavioral inhibition/activation system
CESD-R	Center for Epidemiologic Studies Depression Scale-Revised
CSS	COVID Stress Scales
PA/NA	positive affect/negative affect
RBC	Ratio of Bad Channels
OHA	Ratio of data with overall high amplitude
THV	Ratio of timepoints of high variance
CHV	Ratio of channels of high variance
EC	eyes closed condition
EO	eyes open condition
AI	Asymmetry Index
ICC	intraclass correlation coefficient
CSD	

Abstract

Neuroscience is facing a replication crisis. Little effort is invested in replication projects and low power in many studies indicates a potentially poor state of research. To assess replicability of EEG research, the #EEGManyLabs project aims to reproduce the most influential original EEG studies. A spin-off to the main project shall investigate the relationship between frontal alpha asymmetries and psychopathological symptoms, the predictive qualities of which have lately been considered controversial. To ensure that preprocessing of EEG data can be conducted automatically (via Automagic), we tested 47 healthy participants in an EEG resting state paradigm and collected psychopathological measures. We analyzed reliability and quality of manual and automated preprocessing and performed multiple regressions to investigate the association of frontal alpha asymmetries and depression, worry, trait anxiety and COVID-19 related worry. We hypothesized comparably good interrater reliability of preprocessing methods and higher data quality in automatically preprocessed data. We expected associations of leftward frontal alpha asymmetries and higher depression and anxiety scores and significant associations of rightward frontal alpha asymmetries and higher worrying and COVID-19-related worrying. Interrater reliability of preprocessing methods was mostly good, automatically preprocessed data achieved higher quality scores than manually preprocessed data. We uncovered an association of relative rightward lateralization of alpha power at one electrode pair and depressive symptoms. No further associations of interest emerged. We conclude that Automagic is an appropriate tool for large-scale preprocessing. Findings regarding associations of frontal alpha asymmetries and psychopathology likely stem from sample limitations and shrinking effect sizes.

Keywords: #EEGManyLabs, EEG, preprocessing, frontal alpha asymmetry, psychopathology

1 Introduction

In recent years, the psychological community has become increasingly aware and worried about the possibility of a replication crisis of psychological research. Replication crisis refers to the phenomenon that researchers are unable to reproduce findings of original studies using similar or identical methodology (Maxwell et al., 2015). In many cases, such failures to replicate are characterized by false positive results of original studies. Replicability of original results enables conclusions about whether the finding in question presents a true effect or merely a false positive. Understanding replicability as a key concept of scientific research, poor replicability thus casts doubt over reliability of psychological findings as a whole (Simmons et al., 2011). While not immune to any form of errors, replication efforts of a study with clearly disclosed methodology should ideally produce similar results and effect sizes in a large majority of cases. Too often, however, replication efforts do not find effects that were reported in the original study. For instance, the Open Science Collaboration (2015) demonstrated how, upon thorough replication of previously significant original studies, effect sizes were halved, and significant findings were reduced to about one third of the reported effect sizes. Inclusion of freshly acquired data also led to a reduction of significant results. This apparent lack of reproducibility can be attributed to a variety of factors.

Firstly, insufficient sample sizes, and thus reduced power, hinder discovery of true effects while also favoring overestimation of effect sizes (Bishop, 2019; Button et al., 2013). Seeing statistical power as the probability to correctly reject the null hypothesis and therefore correctly detect an effect, low power consequently results in reduced detection of actual effects. This low detection rate, in turn, favors the detection of more extreme effect sizes if an effect reaches significance, thus inflating true effect sizes (Ioannidis, 2008). Secondly, overestimation of true effect sizes is further strengthened through publication bias (Dwan et al., 2008). Publication bias is the practice of selectively publishing research that provides novel findings or strong effects while disregarding statistically non-significant findings. The implications of this publication bias manifest in a culture of under-publication of non-significant results (Bishop, 2019). Therefore, more significant effects are known even though the population effect might differ. Furthermore, studies reporting low power null-effect findings are more prone to rejection during publication process, showing an interaction of power and publication bias (Button et al., 2013; Easterbrook et al., 1991). Thirdly, because of competitive publication practices, researchers might, at times unintentionally, resort to questionable (QRPs) or even fraudulent (Bhattacharjee, 2013) research practices to produce and publish their research. Among said QRPs is the capitalization on researcher degrees of freedoms (DF; Simmons et al.,

2011). DF are problematic, yet often undisclosed, flexible practices (e.g., lack of randomization or blinding of participants) that can span across the entire scientific process (Wicherts et al., 2016). These practices, in turn, might result in a body of false-positive results which replication efforts subsequently fail to replicate (Shrout & Rodgers, 2018). Despite the overall acknowledgement of the importance of replication efforts, replication studies are often considered rather unattractive compared to original research and thus only make up a small proportion of publications. This mindset is often amplified by the abovementioned culture of publication. Further, since many original studies barely reach significance, a replication sample size larger than the original sample size would be required, rendering replication efforts even more unattractive (Button et al., 2013). Lastly, Maxwell et al. (2015) pointed out, that apparent failures to replicate are not necessarily true failures to replicate. The authors argued that, in order to assert that an original effect is indeed close or equal to zero, very large samples, Bayesian analysis methods, and multiple replication studies would be required.

While attention regarding replication crisis was mostly directed at the presumably poor replicability of social psychology, other fields of psychological research, such as neuroscience, are not exempt from poor replicability and shrinking effect sizes. As already discussed, low power poses a major issue for detection of true effects. Neuroscientific studies often suffer from small sample sizes. Therefore, power is consequently reduced, and effect sizes are overestimated (Schäfer & Schwarz, 2019). It has been confirmed that statistical power in neuroscience studies remains at “an unacceptably low level” (Button et al., 2013; Szucs & Ioannidis, 2017, p. 13). This state of research is alarming and supports growing concern that many of the reported results in various scientific fields, but specifically neuroscience, might be false (Ioannidis, 2005).

A well-established measure of neuroscience and neuroimaging is the electroencephalogram (EEG). First introduced by Berger in 1924, the EEG has since become a widely used tool for both neurological diagnostics and neuroscientific research. EEG systems offer a variety of advantages, such as low acquisition and maintenance costs, non-invasive recording, accessible use, good mobility, and high temporal resolution (ms). Interest in EEG research has thus been increasing steadily, with 6,672 publications in 2020 (PubMed; “eeg[Title/Abstract]”, Filters applied: from 2020/1/1-2021/1/1). EEG replication publications for the same time frame, searched via the same search procedure, however, revealed only 38 results (PubMed, “(eeg[Title/Abstract]) AND (replication[Title/Abstract])”, Filters applied: from 2020/1/1-2021/1/1). Therefore, EEG research appears to suffer from a similar, if not more extreme, culture of under-replication. EEG research, as most scientific research, underlies a

culture of publication bias. It produces noisy data and allows for a lot DF in data handling (Kriegeskorte et al., 2009; Poldrack et al., 2017; Wicherts et al., 2016). EEG preprocessing and processing pipelines offer a multitude of different options of data handling. Artifact removal, for instance, is still largely performed via visual inspection. Further steps during preprocessing, as well as experimental conditions (Kappenman & Luck, 2010) only increase possible data and processing variability and thus hinder replicability. As pointed out by Pedroni et al. (2019), large-scale EEG datasets are therefore rare. To ensure satisfactory replicability of EEG research, however, large-scale EEG datasets of comparable quality with standardized methods and processing pipelines are needed.

1.1 #EEGManyLabs

The #EEGManyLabs project (Pavlov et al., 2021), an international effort to investigate replicability of EEG research, aims to correct these emerging shortcomings of neuroscientific research. To this end, the #EEGManyLabs team identified a body of influential EEG literature and provided a list of eligible literature for participating labs to vote on. The 32 most voted studies were subsequently checked for the required replication sample size and reduced to 27 studies, all of which would require $n \leq 200$ to replicate. Under the lead of one lab, multiple labs will perform data acquisition for their assigned original study. The Lead Replicating Laboratories will provide standardized and reviewed protocols that are faithful to the original methodology to the highest possible degree. Data analysis therefore follows the original methodology. This thorough documentation, along with a (pre-) registration of replication studies, serves as the project's main strategy to avoid the abovementioned flaws in scientific research. Scripts, protocols, materials, and analysis pipeline will be published via OSF following the FAIR principles (Wilkinson et al., 2016). Overall, #EEGManyLabs strives for as much transparence and open-science as possible. Raw EEG data will be stored according to the Brain Imaging Data Structure (BIDS; Gorgolewski et al., 2016; Pernet et al., 2019). Meta-analysis will be conducted by the respective Lead Replicating Lab and published as a replication report. A replication will be deemed successful at statistical significance ($p < .02$) of the random-effects meta-analytic estimate. Upon completion of the individual replication reports, results will be accumulated and summarized in a final report. This report shall provide a comprehensive overview of effect sizes (contrasted to original studies' effect sizes) and further statistical outcomes (e.g., Bayes factors) as well as a follow up on possible causes of variance within the findings. Through its large datasets, Bayesian approaches, and multiple separate data acquisitions, the project therefore satisfies the demands proposed by (Maxwell et al., 2015).

Apart from its main replication effort, #EEGManyLabs also features a set of spinoff projects. #EEGManyLabs Automation aims to implement an automated preprocessing and processing pipeline. As the overall replication effort proceeds to generate an enormous amount of data, manual (human-based) preprocessing alone will become increasingly resource-intensive and time-consuming. Moreover, even slight variations in preprocessing methodology might significantly alter results (Robbins et al., 2020). DF as well as QRPs could further reduce reproducibility of findings. While inter-rater reliability of manual EEG preprocessing generally appears to be satisfactory (Shirk et al., 2017), it has been demonstrated that manual and automated preprocessing produce similar results with comparable reliability (Hatz et al., 2015). The appeal of an automated approach lies in its objectivity of artifact rejection, resource efficiency as well as its replicability. Given the original parameters and data, researchers should be able to replicate the preprocessing in its entirety. Therefore, #EEGLabsAutomation will employ the Automagic preprocessing pipeline (Pedroni et al., 2019; see 2.2.2).

The spinoff project #EEGManyLabs Asymmetry will make use of EEG resting state (RS) data recorded before each replication study's sessions to enable a large-scale RS database. This database shall serve as the basis to investigate associations of EEG RS asymmetry and psychopathology, emotion, and personality (refer to 1.2) and will be made available for further research.

1.2 EEG Alpha Asymmetries

For decades, neuroimaging techniques have been established as means of diagnosis (e.g., MRI; Edelman, 2014) or prediction (e.g., EEG in epileptic seizures; Lehnertz et al., 2003; Petrosian et al., 2000) of neurological conditions. Such application of neuroimaging for diagnosis and prediction of psychiatric disorders or personality traits could drastically improve mental health services and reduce costs to the public health systems. Support for this notion stems from evidence that frontal EEG alpha power can be utilized to predict depressive episodes early (Mitchell & Pössel, 2012; Nusslock et al., 2011) and as a means of neurofeedback therapy of depression (Choi et al., 2011). Bridging the gap between neuroscientific research and (neuro-)psychiatric practice, the field of EEG asymmetry research aims to identify lateralized brain activity as biomarkers of psychopathology (Allen & Reznik, 2015). Most frequently associated with personality traits and approach and withdrawal motivation (Davidson et al., 1990; Harmon-Jones & Gable, 2018; Harmon-Jones & Sigelman, 2001), emotion processing (Coan & Allen, 2004; Davidson, 1998; Harmon-Jones, 2003; Reznik & Allen, 2018), suicidal behavior (Graae et al., 1996), and psychopathology (Allen & Cohen, 2010; Allen & Reznik, 2015; Meyer et al., 2018; Nusslock et al., 2018), lateralization of power in the frontal alpha-

EEG band (8-13 Hz) poses one of the most promising fields of EEG asymmetry research. EEG alpha asymmetry "is a relative measure of the difference in electroencephalogram (EEG) alpha power between the right and left […] regions" (Allen & Reznik, 2015, p. 93). The underlying assumption of alpha asymmetry research is that alpha activity is a marker of cortical hypoactivation, inversely correlated with activity in the respective area (Goldman et al., 2002; Oakes et al., 2004). If alpha power is relatively greater in the left hemisphere, cortical activity would therefore be assumed to be relatively stronger in the homologous area of the right hemisphere. The appeal of the marker frontal alpha asymmetry stems from its temporal stability and trait properties (Hagemann et al., 2002, 2005) as well as sensitivity towards state changes and state properties (Coan & Allen, 2004; Hagemann et al., 2005; Reznik & Allen, 2018). Alpha asymmetries arise from varying strengths of synchronized neuronal activity within the frequency of 8-13 Hz. Interestingly, such alpha oscillations do not appear to originate from a single neuronal mechanism (Cohen, 2017), but rather a multitude of generators, such as cortical pyramidal neurons (Silva et al., 1991; Steriade et al., 1990) and thalamocortical circuits (Lopes da Silva et al., 1980). Inferring intercranial source activity, Smith et al. (2018) identified lateral midfrontal regions as a generator of frontal alpha asymmetries. However, little is known about the neural basis of frontal alpha asymmetries still.

Typically, alpha asymmetries have been investigated in RS or emotionally evocative paradigms. Most research efforts particularly focus on alpha asymmetries in frontal and parietal regions of the brain. Historically, however, EEG alpha asymmetry research has suffered from inconsistent considerations and reporting of potentially confounding factors such as handedness, age, gender, sleep, medication and referencing method, thus reducing its applicability as a biomarker (Smith et al., 2017; Thibodeau et al., 2006).

Historic evidence of lateralization of affect dates back more than 80 years, when Goldstein (1939) reported extreme depression in patients with left frontal lesions and abnormally elevated mood in patients with right frontal lesions. These early findings are consistent with the notion that cerebral hemispheres are specialized in the processing of emotions of differing valence, also known as the valence model of emotional lateralization (Davidson, 1984). Expanding on the initial valence model, Davidson et al. (1990; Davidson, 1992) hypothesized that the anterior hemispheres are involved in the system of approach and withdrawal linked to emotional state. Left anterior regions were presumed responsible for approach motivation and positive emotions, right anterior regions specialized for withdrawal and negative emotions. Indeed, Davidson et al. (1990) reported emotion specific differences during induction of either positive (happiness) or negative withdrawal-related (disgust)

emotions. While relative left-hemispheric frontal alpha power in disgust conditions was found, no differences in frontal alpha power emerged during induction of positive emotions. Authors interpreted the results as support for the model of left specialization of approach and right specialization of withdrawal, the underlying assumption being that valence of emotions is intrinsically linked with tendencies to approach to or withdraw from a stimulus or environment.

Harmon-Jones (2003), however, criticized this lack of distinction between motivational direction and emotional valence and established the understanding that it can be difficult to disentangle motivational systems and affect. He demonstrated that anger, a negative emotion, is approach related (Carver & Harmon-Jones, 2009), giving rise to the frontal cortical asymmetry model of motivational direction. Previously, negative emotions had been understood as withdrawal motivated and positive emotions as approach related. As would be expected in a model of motivational direction, anger was shown to be associated with relative left frontal cortical activity (D'Alfonso et al., 2000; Harmon-Jones & Sigelman, 2001). These findings support the notion of lateralization of emotional valence, making a strong case for a motivation-based understanding of hemispheric lateralization.

1.2.1 Depression

Depression and depressive symptoms have long been associated with greater right than left cortical activity (Schaffer et al., 1983). Relative increased left frontal alpha power was observed in participants with acute depressive symptoms as well as lifetime depression history, but not in healthy participants (Gotlib et al., 1998). This activity pattern emerged in a multitude of studies in both depressed (Diego et al., 2001; Henriques & Davidson, 1991; Saletu et al., 2010) and formerly depressed participants (Allen et al., 1993; Henriques & Davidson, 1990). Further, frontal left-sided EEG alpha asymmetry (and cognitive vulnerability) can serve as predictors of depressive episode onset up to one year later (Nusslock et al., 2011; Mitchell & Pössel, 2012). Stability of frontal EEG RS alpha asymmetry scores was shown to be comparable to scores of healthy subjects and thus allows for reliable application of asymmetry measures in clinical practice (Allen et al., 2004). Indeed, such findings integrate well into the frontal cortical asymmetry model of motivational direction, considering that there is abundant evidence linking depression and internalizing disorders with behavioral inhibition (Heidari & Nemattavousi, 2021; Li et al., 2015; Sportel et al., 2011). While its psychological construct validity appears to be good, it is worth noting that neurophysiological construct validity of frontal alpha asymmetry, due to a lack of further neurophysiological findings, is reduced (Allen & Cohen, 2010).

Its good reliability and predictive quality make frontal EEG alpha asymmetries an attractive biomarker for depressive disorders. These findings, however, are not uncontested. While Debener et al. (2000) reported differences in frontal alpha asymmetries between patients and healthy controls, the typical left lateralization of alpha power in patients was not observed. Further, temporal stability of frontal alpha asymmetry in patients was also reported to be poor, limiting its applicability as a trait depression biomarker. Such inconsistencies were reported in major depressive disorder (MDD) samples (Segrave et al., 2011; Stewart et al., 2010), melancholic MDD (Quinn et al., 2014), depression score prediction (Blackhart et al., 2006) and reassurance-seeking depression (Minnix et al., 2004). A potential cause of such failures to replicate could stem from varying time of the year and day interfering with depressive symptomology and hormonal states (Peterson & Harmon-Jones, 2009). These factors could then result in altered hemispheric activity and emotional states.

Underlying neural mechanisms of the association between frontal alpha asymmetry and depression are insufficiently or inconclusively understood. Due to research mostly relying on relative asymmetry scores, no inferences can be made about potential generators of depression-related lateralization of alpha power. Historically, depressive symptoms in patients with left frontal damage (Goldstein, 1939; Narushima et al., 2003) and left hemispheric anesthesia (Terzian, 1964) inspired the understanding of reduced left frontal functioning in depression. Inconsistencies in research of intracranial sources of lateralization in MDD include reports of reduced left frontal activity (Lubar et al., 2003), reduced right frontal activity (Saletu et al., 2010), and no differences in lateralization (Pizzagalli et al., 2002). As pointed out by Smith et al. (2018), methodological variations as well as small sample sizes are likely causes of this inconclusiveness of research.

1.2.2 Anxiety and Worry

Like depression, anxiety disorders are one of the most prevalent mental disorders (Baxter et al., 2013). As anxiety and depression are often comorbid, Davidson (1992) not only predicted an association of left frontal hypoactivation and depression, but also of relative right frontal activity and anxiety. However, findings regarding this association have yielded mixed results. While some studies support the predicted lateralization (Papousek & Schulter, 2002; Petruzzello & Landers, 1994), others report sex-dependent differences of frontal alpha asymmetry (Baving et al., 2002), partial associations of frontal activity and anxiety dimensions (Nitschke et al., 1999), or no frontal asymmetries whatsoever (Kentgen et al., 2000).

To account for such inconsistencies, Heller (1990, 1993) and Heller et al. (1997) proposed a more nuanced perspective on anxiety and asymmetry, the valence-arousal model.

Heller expected altered lateralization patterns for anxious arousal and worry, respectively. While anxious arousal is characterized by somatic responding (e.g., sweating), anxious apprehension describes a state of worrying and rumination. Indeed, there is evidence for distinct lateralization with regards to anxiety subtype. Smith et al. (2016), following up on Heller's theory, found relative left lateralization of frontal cortical activity in participants with generalized anxiety disorder (GAD), high worrying and obsessive-compulsive symptoms. GAD patients with little worrying demonstrated more right than left frontal activity. These findings are consistent with the notion of a left-hemispheric dominance of anxious apprehension, a state associated with worry, obsessive-compulsive symptoms, and symptoms of GAD (Heller et al., 1997). It was assumed that in anxious apprehension verbal processes, most prominently verbal ruminations about the future, would result in increased left-hemispheric activation. This potential mechanism is further supported by evidence of a positive association between verbal cognitive skills and worrying scores in GAD patients (Mohlman, 2013). While there is support for this model in measures of anxious apprehension (Engels et al., 2007; Härpfer et al., 2021; Hofmann et al., 2005; Mathersul et al., 2008), other studies did not find the predicted left lateralization of worry (Nitschke et al., 1999). It appears that both approach-withdrawal model and valence-arousal model capture important features of frontal asymmetries in anxiety, yet only paint a part of the whole picture.

1.3 Sars-2-COVID-19 and Psychopathology

In December 2019 the first case of the Sars-2-COVID-19 (COVID-19) virus infection was reported in Wuhan, China. The outbreak quickly escalated into a global pandemic, causing millions of casualties and severe damage to health systems as well as international travel and trade. To contain the pandemic, many governments imposed restrictions on public and social life, at times in the form of lockdowns and curfews. Throughout the pandemic, however, public health not only suffered from COVID-19 directly, but also a worsening of mental health. MDD and GAD diagnosis increases throughout the pandemic was shown to be associated to financial hardships, COVID-19 infection, and fear of infection (Hyland et al., 2020). Overall, prevalence of depression and anxiety symptoms during the pandemic appear to increase (E. P. H. Choi et al., 2020; Sher, 2020; Varma et al., 2021), with a current meta-analysis by Bueno-Notivol et al. (2021) reporting a seven-fold increase in depression prevalence. COVID-19 Mental Disorders Collaborators (2021) estimated that the pandemic resulted in a large increase of MDD (27.6%) and anxiety disorders (25.6%) globally.

While this deterioration in mental health is very alarming, it allows for sampling of real-life induced anxiety, fear and worries. Research on the association of anxiety-related

concepts and EEG alpha asymmetries is typically conducted via unspecific measures, like the State Trait Anxiety Inventory (STAI; Spielberger et al., 1983) and the Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990). Given the increase in COVID-19-related worries, this situation could enable psychological research to investigate whether the reported lateralization of anxiety, especially within the anxious apprehension dimension, can be found in an ecologically valid anxiety-inducing situation.

1.4 Study Objectives and Hypotheses

Our objective in this study is twofold: First, we aim to establish a reliable Automagic preprocessing pipeline for application in the #EEGManyLabs Asymmetry spinoff that ensures preprocessing at a quality comparable to human raters. To this end, we designed an EEG RS paradigm capturing various measures of the final Asymmetry spinoff project (eyes-open vs. eyes-closed states, state and trait psychopathology, personality traits, etc.). Raw EEG data was preprocessed (semi-) manually by two raters and automatically via Automagic. We computed EEG alpha power at a selection of relevant electrode sites as well as measures of hemispheric alpha asymmetry. To examine the extent of reliability in manual preprocessing, inter-rater reliability was calculated between raters' preprocessed alpha asymmetry scores. Further, we also assessed inter-rater reliability between rater-preprocessed asymmetry scores and Automagic-preprocessed asymmetry scores. Finally, we compared manually and automatically preprocessed data with regards to data quality. This investigation of reliability and quality of automated preprocessing shall serve as a foundation for the #EEGManyLabs project to handle preprocessing of EEG data reliably and efficiently. We hypothesize that both inter-rater reliability within manual preprocessing as well as between manual and automated preprocessing will be good, according to Koo & Li (2016). Further, we expect automatically preprocessed data to receive better quality ratings (from the Automagic quality assessment tool) than manually preprocessed data in all four categories.

Second, we aim to investigate the association of depressive symptoms, worry, trait anxiety and overall COVID-19 worry with EEG alpha asymmetries of two frontal electrode pairs in both eyes-closed and eyes-open data preprocessed in Automagic. Previous findings on the nature of such associations have yielded inconclusive results. The cause of such inconsistencies is often attributed to flawed methodology. Here we aim to provide a study with clear and reproducible methodology, taking potential moderators of frontal alpha asymmetry and functional lateralization, such as age (Hirnstein et al., 2013; van der Vinne et al., 2017), sex (Hirnstein et al., 2019; van der Vinne et al., 2017), sleepiness (Zhang et al., 2019), and handedness (Ocklenburg et al., 2019), into consideration. It is for this reason that we expect to

replicate original findings: We predict a significant association of frontal leftward alpha asymmetry with depressive symptom and trait anxiety scores in automatically preprocessed data. We hypothesize frontal rightward alpha asymmetries to be associated with worrying behavior as well as COVID-19-related worrying.

2 Methods

2.1 Sample

We tested 48 healthy adult participants between the age of 18 and 45 (M = 23.7, SD = 5.02, Appendix 5), of which one was excluded due to errors during EEG recording (n = 47). We aimed for a sample size of n = 66 (derived from financial resources) but terminated data collection early due to time constraints. The sample was mostly recruited from students at the Ruhr-University Bochum (34 female & 13 male). The procedure comprised an online survey and a subsequent testing session (Figure 1) in the EEG laboratory of the Department of Biopsychology at the Ruhr-University Bochum. Before being invited, participants completed an online screening of pre-defined exclusion criteria via Qualtrics (Qualtrics LLC, Provo, Utah). These were based on recommendations by Babiloni et al. (2020) and consisted of diagnosed psychiatric or neurological conditions, sleep disorders, acute or chronic drug consumption, and shifted diurnal cycle (e.g., due to work or travel). Left-handed participants were not excluded for the sake of accurate representation of handedness population asymmetry within the sample. Participants with pre-existing COVID-19 risk factors were identified and excluded via online screening as a hygiene and safety measure during the pandemic.

2.2 Ethics Statement

Participants gave their informed consent and were reimbursed with 15 € or 1.5 credits (undergraduate students only). The study was conducted in accordance with the Declaration of Helsinki and approved by the local ethics commission (Ethics Statement 707) at the Faculty of Psychology of the Ruhr-University Bochum.

2.3 Materials

2.3.1 Behavioral Measures

Measures were split between the online and laboratory session of the study: In both sessions, behavioral measures were collected via Qualtrics. In the online session, participants completed temporally stable and trait measures. They commenced with the Edinburgh Handedness Inventory (EHI; Oldfield, 1971) which captures handedness via a laterality quotient LQ = ([R-L]/[R+L]) * 100. The EHI consists of 10 statements describing everyday manual activities. Participants were instructed to indicate hand preference for each activity as either strong (++

left or right), moderate (+ left or right) or equal (+ left and right). Sleepiness was assessed via Karolinska Sleepiness Schedule (KSS; Åkerstedt & Gillberg, 1990). The KSS is a self-reported measure on a one 9-point scale. Following the KSS, participants completed the BIS/BAS scales (Carver & White, 1994). The BIS/BAS scale capture the motivational systems of behavioral inhibition and behavioral activation, two core concepts of motivational direction and affect. The Center for Epidemiologic Studies Depression Scale-Revised (CESD-R; Eaton et al., 2004) served as a measure of depressive symptoms in our healthy sample. It captures symptoms and symptom severity of MDD via 20 items. Its main advantage compared to other established measures of depression (e.g., Beck's Depression Inventory; Beck et al., 1996) is that it does not assume previous MDD diagnosis and thus can be applied to non-clinical populations. Worrying behavior was assessed with the Penn State Worry Questionnaire (PSWQ; T. J. Meyer et al., 1990), a 16-item test that focuses on excessive worrying, as is often observed in patients with GAD diagnosis. A trait version of the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988) was used to compute scores of positive (PA) and negative affect (NA). The trait scale of the State-Trait Anxiety Inventory (STAI; Spielberger et al., 1983) served as a trait measure of anxiety. We used the Reinforcement Sensitivity Theory of Personality Questionnaire (Corr & Cooper, 2016) as an additional measure of approach and avoidance behavior. A short version of the Big Five Inventory (BFI-S; Gerlitz & Schupp, 2005) was employed to capture the Big Five dimensions of personality.

In the laboratory session, situational and state measures were completed. Once again, we assessed participant sleepiness via KSS. A state version of the PANAS was used to score state PA and NA. We captured state anxiety via the state scale of the STAI. A German translation of the COVID Stress Scales (CSS; Taylor et al., 2020) served as a measure of psychopathological symptoms and perceived strain caused by the COVID-19 pandemic. It consists of 36 items and allows for scoring on the subscales of danger and contamination, socioeconomic consequences, xenophobia, traumatic stress, compulsive checking, and a total CSS score. Apart from traumatic stress and compulsive checking, all items revolve around fear or worry regarding the pandemic, making the CSS an ideal instrument to check for associations with EEG alpha asymmetries. The translation of the CSS was not validated due to time constraints.

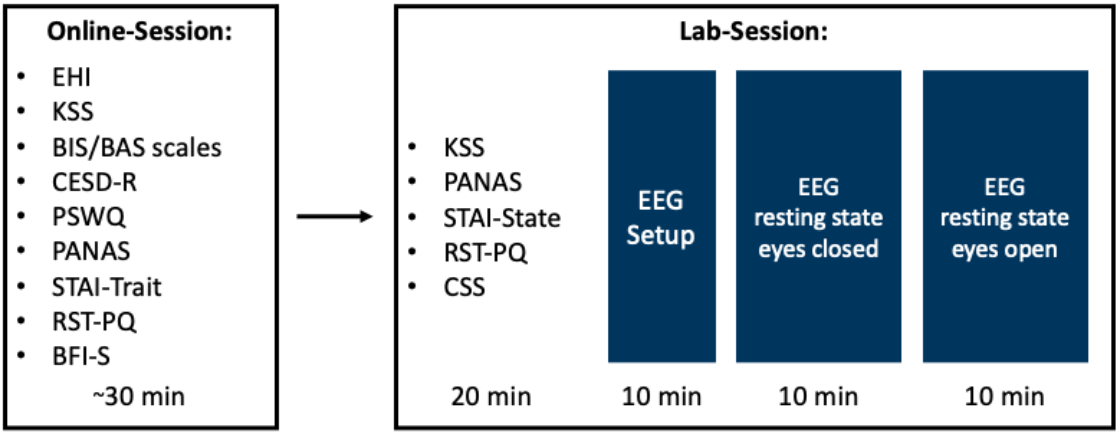

Figure 1. Testing procedure. Abbreviations as defined in Materials.

2.3.2 Automagic

Automagic (Pedroni et al., 2019) is an open-source MATLAB toolbox designed to create standardized EEG preprocessing pipelines for large EEG datasets. With its BIDS structure compatibility and configuration sharing functionality, Automagic enables accurate replicability of EEG preprocessing. Automagic features a variety of established preprocessing procedures, such as outlier trimming, bad channel detection, artefact correction, filtering, and channel interpolation as well as data quality ratings and a rating commitment system. Within Automagic, users can select a BIDS-style EEG directory for preprocessing. After configuration of the preprocessing parameters, preprocessing is automatically carried out. Upon completion users can interpolate bad channels. After interpolation, Automagic calculates four quality measures: *Ratio of Bad Channels (RBC), Ratio of data with overall high amplitude (OHA), Ratio of timepoints of high variance (THV),* and *Ratio of channels of high variance (CHV).* Higher scores indicate reduced data quality. Users are able to tune rating thresholds of these quality measures to fit their analysis. Finally, quality ratings are committed and cannot be modified any further. Automagic provides visualization of the data throughout preprocessing, as well as logfiles and the preprocessed data files. Configurations of the pipeline can be exported and replicated.

2.4 Procedure, EEG recording and Preprocessing

2.4.1 Procedure

Testing consisted of two separate sessions. For a detailed account of the procedure, refer to Figure 1. The first session was conducted online via Qualtrics survey and captured mostly trait and personality measures (see 2.2.1). The second session was conducted in the EEG laboratory of the department of Biopsychology at the Ruhr-University Bochum. To be admitted to the second session, participants had to complete the first session. In the laboratory participants

were tested for Sars-2-COVID-19 and completed a set of state measures via Qualtrics. Participants were subsequently connected to the EEG system (see 2.3.2). After successful setup, we instructed participants to close their eyes and remain in a relaxed, resting state. Participants were told to refrain from excessive movements (e.g., eye movements, chewing). Experimenters left the EEG chamber, turned off all light sources and started a 10 min recording of eyes closed RS EEG (EC). After 10 min, light was turned on to dimly illuminate the EEG chamber. Participants were instructed to open their eyes, focus a black monitor 50 cm in front of them and remain in a resting state. Once again, 10 minutes of eyes open RS EEG (EO) was recorded. Participants could then wash their hair and scalp, were reimbursed, and dismissed. Overall, testing lasted about 80 min.

2.4.2 EEG recording

EEG signal was recorded via a 64 channel actiCap system and BrainVision Recorder (Version 1.20.0802; Brain Products GmbH, Gilching, Germany). Electrode positions conformed to the international 10-20 system. The ground electrode was positioned at FPz, the reference electrode at FCz. EEG sampling rate was fixed to 1000 Hz and sampling interval was set to 1000 µS. After we connected participants to the system, we applied SuperVisc High-Viscosity Gel (EASYCAP GmbH, Herrsching, Germany) below each electrode to ensure low impedances. Impedances did not exceed 2 kΩ. Participants were instructed to keep their chin on a chinrest for the entirety of the recording. Lighting was turned on but kept dim for the EO recording. Both recordings lasted 10 min each Recording length as well as eye status practice were derived from suggestions by Hagemann (2004) and Hagemann et al. (1998). EEG data was stored in BrainVision file format (BVA).

2.4.3 Preprocessing in Automagic

To facilitate Automagic preprocessing, we converted the raw BrainVision files (.eeg, .vhdr, .vmrk) to .mat files via the MATLAB (MATLAB R2020a, Mathworks Inc., Sherborn, MA) EEGLAB toolbox (v2021.0; Delorme & Makeig, 2004). Conversion and preprocessing were performed separately for EC and EO data, configurations and quality criteria remained identical.

In Automagic, we defined the outlier trim amplitude threshold as 100 µV and range for rejection as 500 ms. Bad channels were detected via Clean_rawdata() pipeline high pass [0.25 0.75], line noise (4), and channel criterion (0.85). Residual bad channel detection was employed with High Variance Criterion (HVC) enabled at 25 SD, cutoff at 100 and a reject ratio of 0.5. Minimum Variance Criterion (MVC) was set to 1. Line power was filtered with a notch filter at 50 Hz. As high-pass filter, we employed the pop_eegfiltnew() function with a

cutoff at 0.5 Hz. We did not apply low pass filters. For artefact correction, we opted for ICLabel, saving all components extracted during the ICA. We excluded muscle, eye, heart, line noise, and channel noise components at a probability threshold of 0.8. Interpolation method was set to spherical. Quality ratings as well as additional options remained at default. Preprocessing was subsequently performed automatically. For 47 participants, preprocessing was completed after 243 min which was 4 min faster than preprocessing of the identical data in BrainVision Analyzer (BVA; Version 2.0; Brain Products GmbH, Gilching, Germany). After initial preprocessing, bad channel interpolation and quality ratings were carried out. We did not manipulate quality parameters and committed the ratings. For EC data, two datasets were rated bad, 16 datasets were rated OK, and 29 datasets were rated good. One EO dataset was rated bad, 17 EO datasets were rated OK, and 29 datasets were rated good. Preprocessed data were stored in .mat files.

2.4.4 Processing of Automagic Data

Further processing of Automagic data was conducted in MATLAB. We loaded the preprocessed Automagic data via Fieldtrip toolbox (Version 20210720; Oostenveld et al., 2011) and applied baseline correction and rereferencing to common average, as proposed in methodological literature (Davidson, 1998; Hagemann et al., 1998; van der Vinne et al., 2017). A bandpass filter with a range of 1-45 Hz was applied using the firws option. We segmented data into epochs of 1 sec. Power computation was conducted via multitaper frequency transformation and Hanning window. Power of the alpha band (8-13 Hz) was then averaged for each electrode. To enable analysis of split-half reliability, we also divided each EEG dataset into two sets of equal lengths, performed the abovementioned processing and exported average split-half alpha power scores.

2.4.5 Preprocessing and Processing in BrainVision Analyzer

Manual preprocessing of EEG data was performed in BrainVision Analyzer. To assess interrater reliability of manual preprocessing, preprocessing was carried out by two persons (Rater 1 & 2) separately, both for EC and EO data. The following procedure was applied to each dataset: We visually inspected data for bad channels (high variance or flatline) and subsequently removed them. Removed channels underwent topographic interpolation. Artifacts were removed via raw data inspection. We filtered data (zero phase shift Butterworth filter) via high pass filter at 0.53 Hz and a notch filter at 50 Hz. Finally, we performed ICA and excluded blink, eye movement and ECG components upon visual inspection of component signal and topography.

Processing was conducted after preprocessing was completed. We rereferenced data to common average reference and applied a 1-45 Hz bandpass filter. Data was segmented into 1 sec. epochs. We subsequently performed a Fast Fourier Transformation with Hanning window to compute band power. We then averaged power over the segments and exported the power values for the alpha frequency band (8-13 Hz). To analyze split-half reliability, we divided each EEG dataset into two sets of equal length, performed the abovementioned processing and exported average split-half alpha power scores.

2.4.6 Quality Measures

Via its built-in quality rating mechanisms, Automagic computed quality ratings for each dataset and stored ratings as well as quality thresholds in a corresponding logfile. We extracted OHA, THV, CHV, and RBC values separately for EC and EO data via MATLAB. Since BrainVision Analyzer does not feature such quality measures, we converted preprocessed BVA data to the EEGLAB file format and performed quality analysis on the converted data via MATLAB. Quality thresholds and parameters used in Automagic were also applied for BVA data quality ratings.

2.5 Statistical Analysis

Outlier analysis was conducted in R (Version 4.0.3) for BrainVision data and Automagic data. We identified outliers as deviations of a minimum of ±3 SD from the mean channel (F3, F4, F7, F8, P3, P4, P7, P8, O1, O2) alpha power. Outliers were subsequently excluded from analysis. Assumptions for tests were checked via Shapiro-Wilk test for normal distribution and Levene test for homogeneity of variances. In case of violation of these assumptions, we resorted to non-parametric alternative tests. Inference criterion was fixed at $\alpha = .02$.

We calculated descriptive measures of demographic and relevant values, such as age, sex, sleepiness, and handedness. The sex ratio of our sample was compared to the average German sex ratio (Destatis, 2021) via one-proportion z-test. Handedness proportion was compared to the population asymmetry (Papadatou-Pastou et al., 2020) via one-proportion z-test. We visualized the distribution of EHI and KSS scores, as well as age. To investigate if EHI scores differed significantly from zero, we performed a t-test.

As a measure of EEG alpha power asymmetry, we derived Asymmetry Indices (AI) via the formula $AI = \ln[R] - \ln[L]$ from five electrode pairs (F4-F3, F8-F7, P4-P3, P8-P7, O2-O1). Therefore, positive AIs represent relatively stronger right-sided alpha power and negative AIs represent relatively stronger left-sided alpha power. AIs were computed for BrainVision data of Rater 1 and Rater 2 (EC & EO, respectively), as well as Automagic data (EC & EO). We

plotted AIs for all electrode pairs in all conditions and performed Bonferroni-corrected t-tests ($\alpha = .0007$) of AIs against zero.

To assess split-half reliability, we calculated Spearman-Brown coefficients for the abovementioned individual channels as well as AIs. Inspired by Tavakol & Dennick (2011), we interpret Spearman-Brown coefficients between .7 and .95 as acceptable and coefficients and coefficients > .95 as excellent. We computed intraclass correlation coefficients (ICC) as a measure of interrater reliability for BrainVision data. We opted for a two-way mixed effect model ICC(3,1), as described by Shrout & Fleiss (1979). Further, ICC was also calculated to assess interrater reliability of BrainVision and Automagic preprocessing of EEG data. 95% confidence intervals were calculated for each intraclass-correlation coefficient. ICC interpretation was derived from Koo & Li (2016).

To compare the quality of preprocessed data between manual and automated preprocessing, we analyzed differences in OHA, THV, CHV, and RBC ratings for EC and EO data, respectively. Due to non-normality of quality ratings, non-parametric tests were employed. Quality differences between Rater 1, Rater 2 and Automagic were tested via Friedman test. Kendall's *w* was calculated for effect sizes. In case of significant differences, we followed up on the effect via paired Wilcoxon test. To assess whether there was an overall difference in quality of preprocessing of data, we also performed Wilcoxon tests comparing manual versus Automagic quality ratings and derived the effect size measure *r*. Quality ratings were visualized in boxplots.

We plotted the distributions of CESD-R, PSWQ, trait anxiety and CSS total scores. Spearman correlations of frontal AIs and other measures (handedness, sleepiness, CESD-R, PSWQ, trait anxiety and CSS total score) were computed and visualized via correlation heatmaps. Since correlation was high between frontal electrodes, they were entered separately into regression models. Multicollinearity of predictors within a model were assessed via variance inflation factor (VIF). As multicollinearity of some predictors was high (VIF > 10), we standardized predictors. Since our focus was the assertion of quality for Automagic preprocessing, association of psychopathological measures with frontal EEG alpha asymmetries was analyzed via Automagic data of EC and EO conditions. To this end, we performed linear multiple regressions with depression (CESD-R), worry (PSWQ), trait anxiety (STAI) and COVID-19 worry (CSS) scores as outcome. It has become increasingly clear that negligence of factors moderating hemispheric asymmetries, such as age, gender, sleepiness, and handedness, is part of the problematic methodological practices leading to inconclusive results (Hagemann, 2004; Ocklenburg et al., 2019; Reznik & Allen, 2018; Thibodeau et al.,

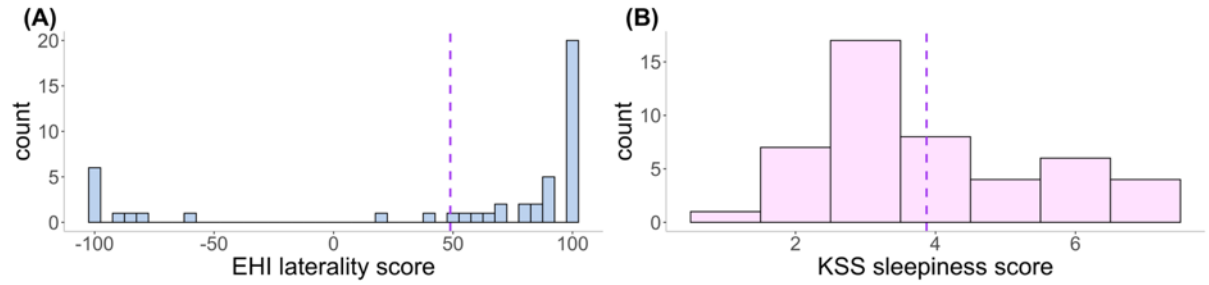

Figure 2. Distribution (*n* = 47) of **A)** EHI laterality quotient, **B)** KSS sleepiness scores. Dashed lines represent respective mean scores.

2006). Therefore, we entered the predictors age, gender, sleepiness, handedness, and frontal AI (F3F4 or F7F8) as well as interactions of each predictor with the respective AI in our regression models. While more complex moderations might explain even more variance, there is little literature justifying the inclusion of such moderations. Further, we aim to avoid overfitting of the model with overcomplex moderations.

3 Results

3.1 Descriptive Statistics and Sample Characteristics

Handedness lateralization as measured by the EHI showed a rightward shift (M = 48.85, SD = 75.95). Overall, 10 participants (21.3%) reported left handedness (Arning et al., 2015; LQ < -40) and 36 participants (76.6%) reported right handedness (LQ > 40). There was one mixed-handed participant (-40 < LQ < 40). LQs were significantly different from zero ($t(46) = 4.41$, $p < .001$). A one proportion z-test revealed a trend-significant deviation of handedness from the population average ($\chi^2(1) = 4.86$, $p = .028$) with more left-handed participants (21.3%) than in the overall population (10.4 %; Papadatou-Pastou et al., 2020). One proportion z-test revealed a deviation of sex from the German average ($\chi^2(1) = 7.96$, $p = .004$) with significantly more female participants (72.34%) than in the German population (50.7%). Sleepiness as measured by the KSS (M = 3.87, SD = 1.6) was not normally distributed ($W = 0.90$, $p < .001$). Handedness and sleepiness distributions are shown in Figure 2.

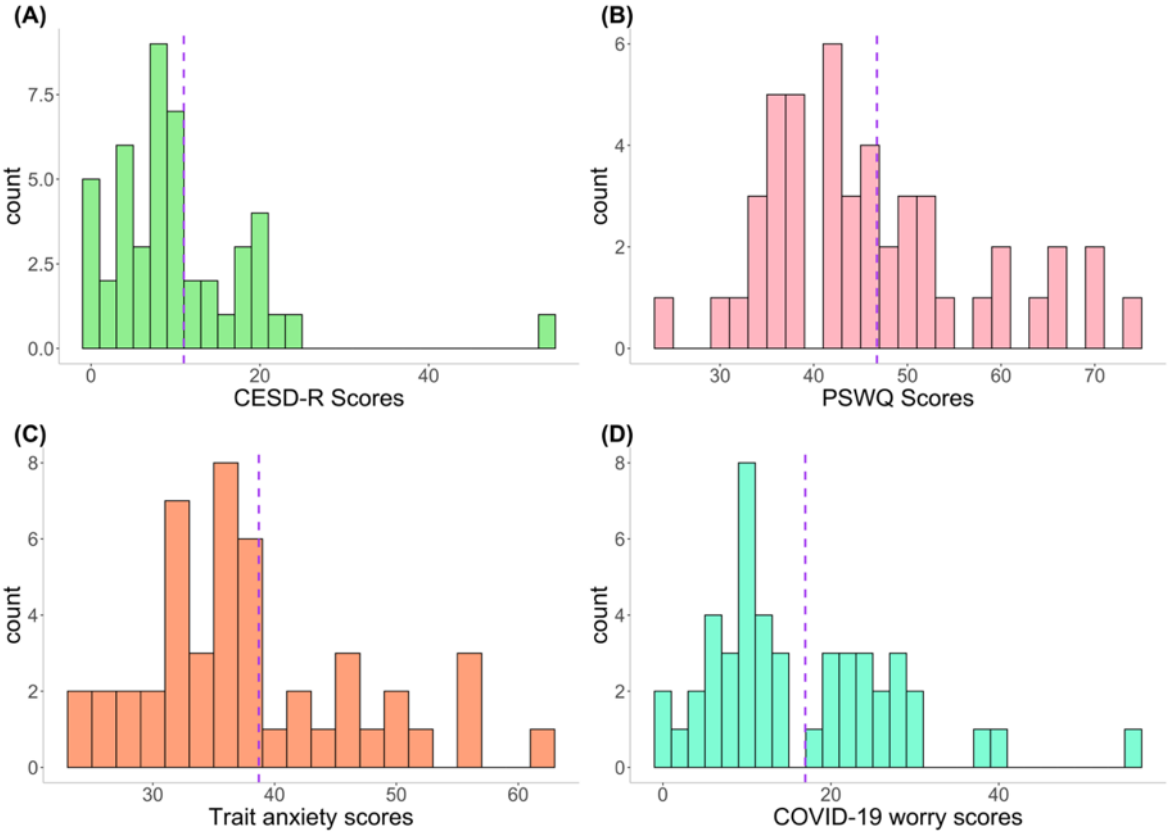

Figure 3. Distribution (n = 47) of **A)** CESD-R depression scores, **B)** PSWQ worrying scores, **C)** trait anxiety scores as measured by the STAI, **D)** CSS worrying scores. Dashed lines represent respective mean scores.

In this healthy sample, 10 participants (21%) surpassed the CESD-R cut-off score (16) for depressive symptoms. 22 participants (46%) reached a PSWQ score higher than the cut-off of 45 (Behar et al., 2003). Electrode pair AI distribution (Figure 4) was assessed for both eye conditions (EC & EO) and the three raters (Automagic, Rater 1, Rater 2). We performed Bonferroni-corrected t-tests for the 30 AIs (6 rating conditions x 5 electrode pairs) as seen in Ocklenburg et al. (2019). For Automagic EC ($t(43) = 4.95$, $p < .0007$) and Rater 1 EC ($t(43) =$

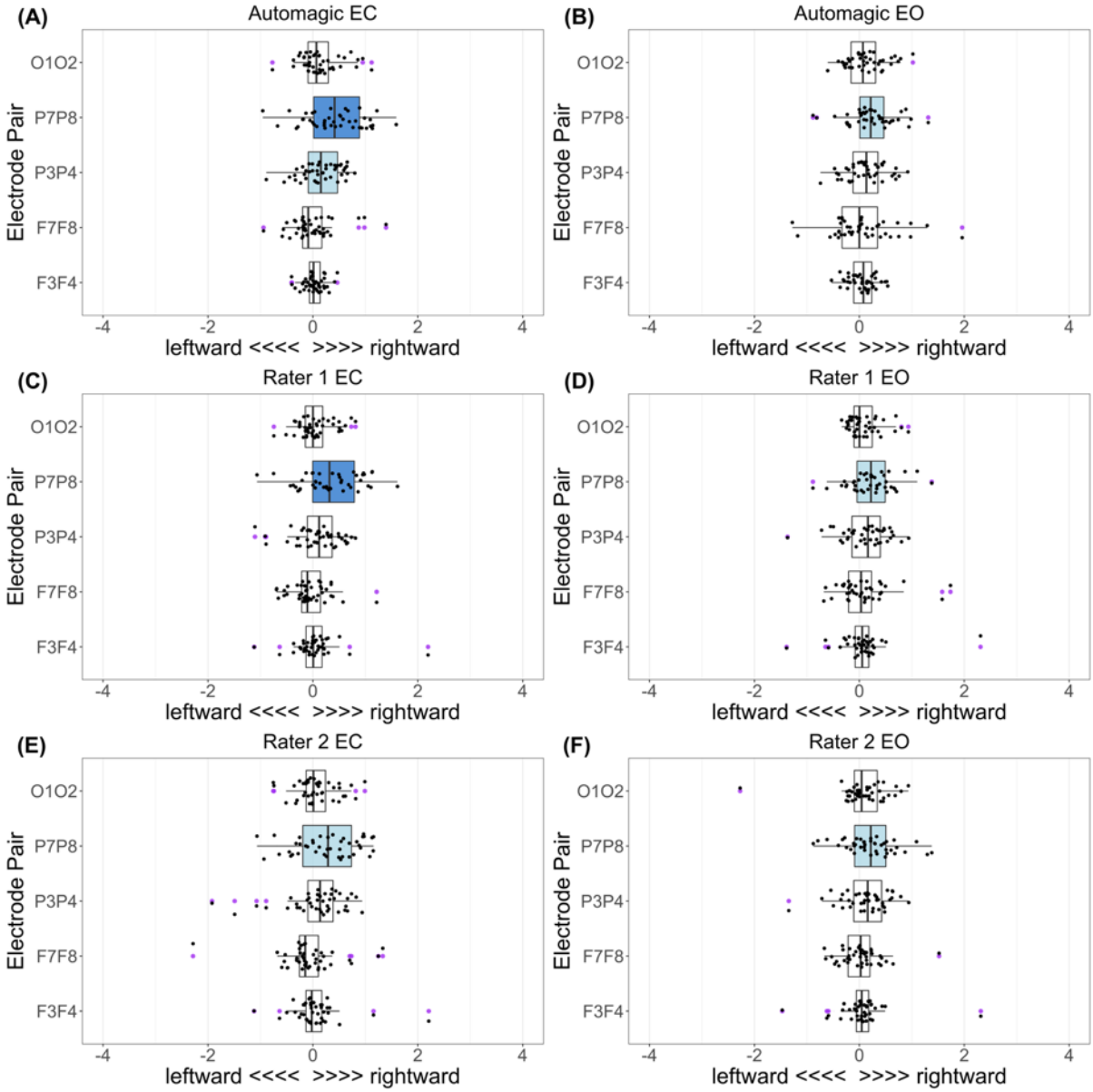

Figure 4. Distribution of alpha (8 -13 Hz) asymmetry indices (see 2.4 for formula) for the three preprocessing procedures (Automagic, Rater 1, and Rater 2) and eye status (eyes closed, eyes open). AIs are shown for 5 electrode pairs. Boxplot hinges represent the 25th and 75th percentile, whiskers extend for 1.5 * IQR. Scores exceeding the whiskers are marked purple. Significance in a t-test is indicated via colored boxplot, with red marking significant leftward and blue marking significant rightward AIs. Light colors indicate significance in uncorrected t-tests, bold colors represent significance after Bonferroni correction.

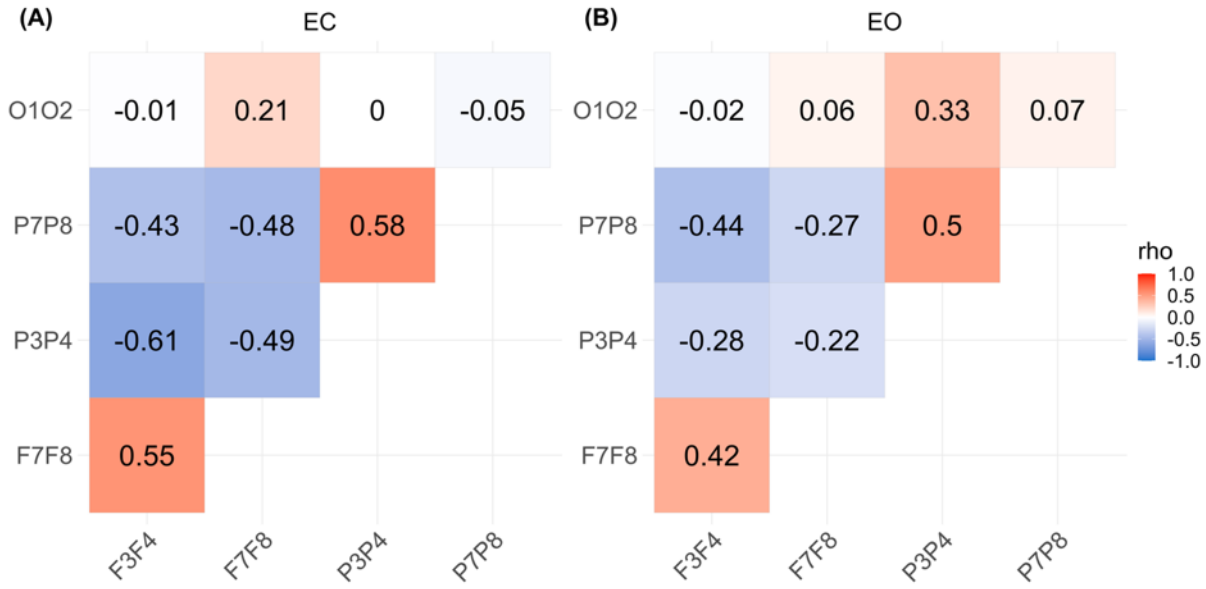

Figure 5. Correlation heatmap of alpha AIs for **A)** EC and **B)** EO Automagic data. Notes: Coefficients represent Spearman correlation coefficients. Red indicates positive correlation; blue indicates negative correlation. More intensive colors represent stronger correlations.

3.95, $p < .0007$), AIs of electrode pair P7P8 were significantly different from zero. Uncorrected significance was reached for electrode pair P7P8 in Automagic EO ($t(43) = 3.34$, $p < .01$), Rater 1 EO ($t(40) = 3.32$, $p < .01$), Rater 2 EC ($t(42) = 2.96$, $p < .01$), and Rater 2 EO ($t(41) = 2.8$, $p < .01$), and for electrode pair P3P4 in Automagic EC ($t(43) = 2.78$, $p < .01$).

We did not find handedness to be significantly correlated with AIs in either EC nor EO data (Figure 7). Correlations of F3F4 and F7F8 AIs (Figure 5) were significant and moderate (Dancey & Reidy, 2007) in both EC ($r = .55$, $p < .001$) and EO data ($r = .42$, $p = .005$).

3.2 Split-half Reliability

Split-half reliability for alpha power of single channels was overall acceptable or excellent (Appendix 1 & 2). Only in 7 of 60 investigated instances, Spearman-Brown coefficients did

Table 1
Split-half reliability of AIs in EC condition.

	Automagic		Rater 1		Rater 2	
	SB	95% CI	SB	95% CI	SB	95% CI
F3F4	0.93	CI[0.76-0.92]	0.99	CI[0.95-0.98]	0.97	CI[0.89-0.96]
F7F8	0.98	CI[0.94-0.98]	0.94	CI[0.79-0.93]	0.83	CI[0.53-0.83]
P3P4	0.94	CI[0.81-0.94]	0.69	CI[0.28-0.71]	0.63	CI[0.2-0.66]
P7P8	0.98	CI[0.94-0.98]	0.97	CI[0.89-0.97]	0.93	CI[0.78-0.93]
O1O2	0.96	CI[0.86-0.95]	0.96	CI[0.87-0.96]	0.91	CI[0.72-0.91]

Note. SB = Spearman-Brown coefficient, CI = Confidence Interval. Unacceptable < .7, acceptable ≥ .7, excellent ≥ .95.

Table 2
Split-half reliability of AIs in EO condition.

	Automagic		Rater 1		Rater 2	
	SB	95% CI	SB	95% CI	SB	95% CI
F3F4	0.98	CI[0.93-0.98]	0.99	CI[0.95-0.98]	0.98	CI[0.94-0.98]
F7F8	0.98	CI[0.94-0.98]	0.84	CI[0.54-0.84]	0.74	CI[0.35-0.75]
P3P4	0.96	CI[0.86-0.96]	0.94	CI[0.79-0.93]	0.94	CI[0.79-0.93]
P7P8	0.95	CI[0.84-0.95]	0.95	CI[0.82-0.94]	0.91	CI[0.72-0.91]
O1O2	0.95	CI[0.83-0.95]	0.99	CI[0.96-0.99]	0.94	CI[0.8-0.94]

Note. SB = Spearman-Brown coefficient, CI = Confidence Interval. Unacceptable < .7, acceptable ≥ .7, excellent ≥ .95.

not reach acceptable levels. Split-half reliability for Automagic data was excellent (≥ .94). Unacceptable reliability scores (< .70) emerged in manually preprocessed EC data only. Split-half reliability of AIs (Table 1 & 2) was mostly acceptable or excellent. Only 2 of 30 reliability scores were below the acceptability threshold, both in manually preprocessed EC data. Automagic split-half reliability once again yielded excellent results (≥ .93).

3.3 Interrater Reliability

ICC between AI values of Rater 1 and Rater 2 data (Table 3) was moderate to excellent for EC data (*M* = .80) and good to excellent for EO data (*M* = .92). ICC between Rater 1, Rater 2, and Automagic AIs (Table 1) was poor to moderate (*M* = .68) for EC data and moderate to good (*M* = .71) for EO data. Interrater reliability between Automagic AIs and single raters ranged from poor to excellent (Appendix 2).

Table 3
ICC of AIs between manual raters and between all raters.

	Manual preprocessing methods		All preprocessing methods	
	EC [95% CI]	EO [95% CI]	EC [95% CI]	EO [95% CI]
F3F4	0.94 [0.89-0.97]	1.00 [0.99-1]	0.35 [0.16-0.55]	0.57 [0.4-0.73]
F7F8	0.71 [0.52-0.83]	0.82 [0.69-0.9]	0.73 [0.6-0.84]	0.71 [0.57-0.82]
P3P4	0.82 [0.69-0.9]	1.00 [0.99-1]	0.75 [0.62-0.85]	0.81 [0.71-0.89]
P7P8	0.76 [0.59-0.86]	0.89 [0.8-0.94]	0.77 [0.64-0.86]	0.72 [0.59-0.83]
O1O2	0.79 [0.64-0.88]	0.88 [0.79-0.93]	0.79 [0.68-0.87]	0.74 [0.61-0.84]

Notes. Manual preprocessing: Rater 1 & Rater 2; All preprocessing methods: Rater 1, Rater 2 & Automagic. CI = Confidence Interval. Poor < .5, moderate < .75, good < .90, excellent ≥ .90.

3.4 Data Quality

3.4.1 OHA Scores

A Friedman test of OHA EC data quality ratings revealed significant differences between the three preprocessing procedures ($\chi^2(2) = 54.51$, $p < .001$, $w = .58$). Post-hoc pairwise Bonferroni-corrected Wilcoxon tests showed significant differences between Rater 1 and Automagic ($p < .001$) and Rater 2 and Automagic ($p < .001$) OHA scores. Automagic OHA scores were significantly lower in both cases. EO data quality also differed significantly between the three procedures ($\chi^2(2) = 36.58$, $p < .001$, $w = .406$). Once again, Automagic OHA scores were significantly lower than Rater 1 ($p < .001$) and Rater 2 ($p < .001$) scores in post-hoc analyses. Wilcoxon rank sum test revealed overall lower OHA scores in automated preprocessing of EC ($W = 3212$, $p < .001$, $r = .369$) and EO data ($W = 3131$, $p < .001$, $r = .444$).

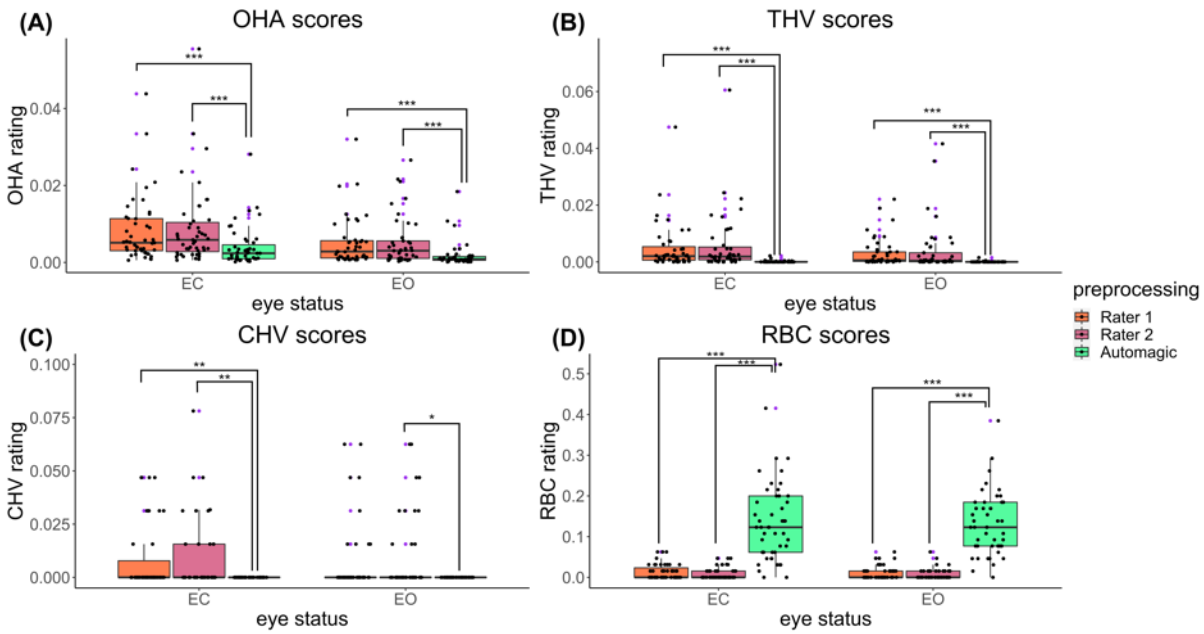

Figure 6. Quality ratings of preprocessed data. Boxplots of OHA, THV, CHV, and RBC scores. Data is presented by eye status and preprocessing method. Upper and lower hinges mark 25th and 75th percentile, respectively. Whisker length is 1.5 * IQR. Brackets and asterisks show significance of pairwise post-hoc Bonferroni-corrected Wilcoxon tests. Outliers are marked purple. * $p < .02$, ** $p < .01$, *** $p < .001$.

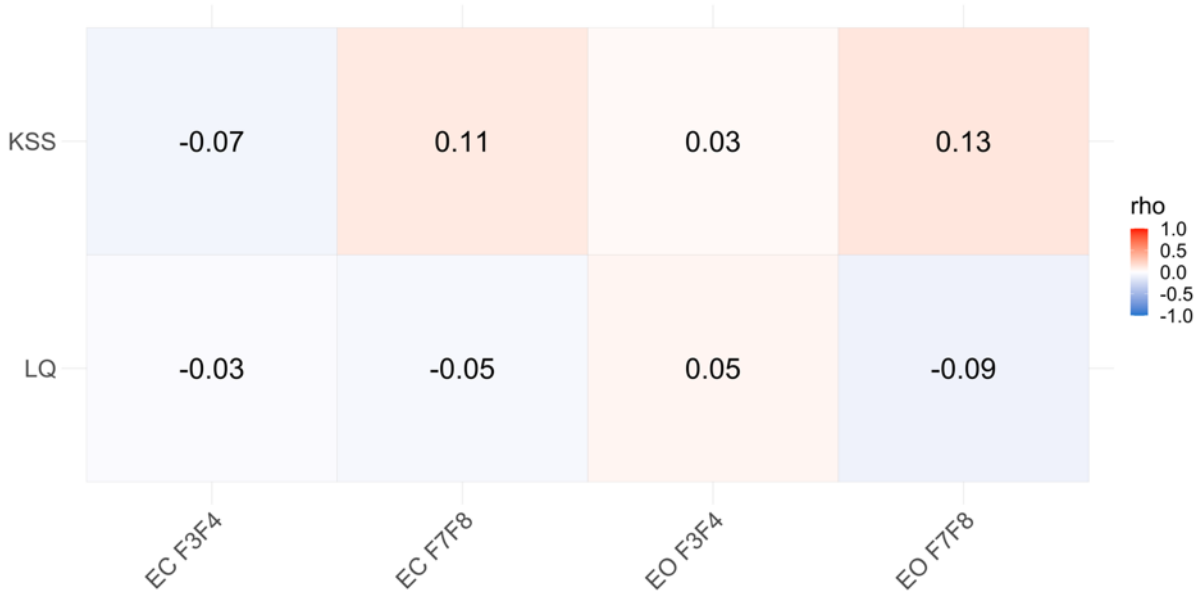

Figure 7. Correlation heatmap of alpha AIs, handedness (EHI LQ) and sleepiness (KSS) measures for EC and EO Automagic data. Coefficients represent Spearman correlation coefficients. Red indicates positive correlation, blue indicates negative correlation. More intensive colors represent stronger correlations.

3.4.2 THV Scores

The three preprocessing procedures resulted in significantly different THV scores for both EC ($\chi^2(2) = 62.52$, $p < .001$, $w = .665$) and EO data ($\chi^2(2) = 43.74$, $p < .001$, $w = .486$). Pairwise Bonferroni-corrected Wilcoxon test revealed significant THV score differences between Rater 1 and Automagic ($p < .001$) and Rater 2 and Automagic ($p < .001$) for EC and EO data. In all cases Automagic scores were lower. A Wilcoxon rank sum test showed lower THV scores in automated preprocessing of EC ($W = 4003$, $p < .001$, $r = .664$) and EO data ($W = 3557$, $p < .001$, $r = .626$).

3.4.3 CHV scores

Friedman tests uncovered significant differences in CHV scores between the three preprocessing procedures in EC ($\chi^2(2) = 21.29$, $p < .001$, $w = .226$) and EO data ($\chi^2(2) = 16.7$, $p < .001$, $w = .186$). Following up on this effect, post-hoc analysis revealed that in EC data, Automagic scores were significantly lower than Rater 1 ($p = .007$) and Rater 2 scores ($p = .003$). In EO data, only comparison of Rater 2 and Automagic scores revealed a significant difference of CHV scores ($p = .011$) with decreased scores in Automagic data. A Wilcoxon rank sum test yielded lower CHV scores in automated preprocessing of EC ($W = 2820$, $p < .001$, $r = .333$) and EO data ($W = 2475$, $p = .001$, $r = .293$).

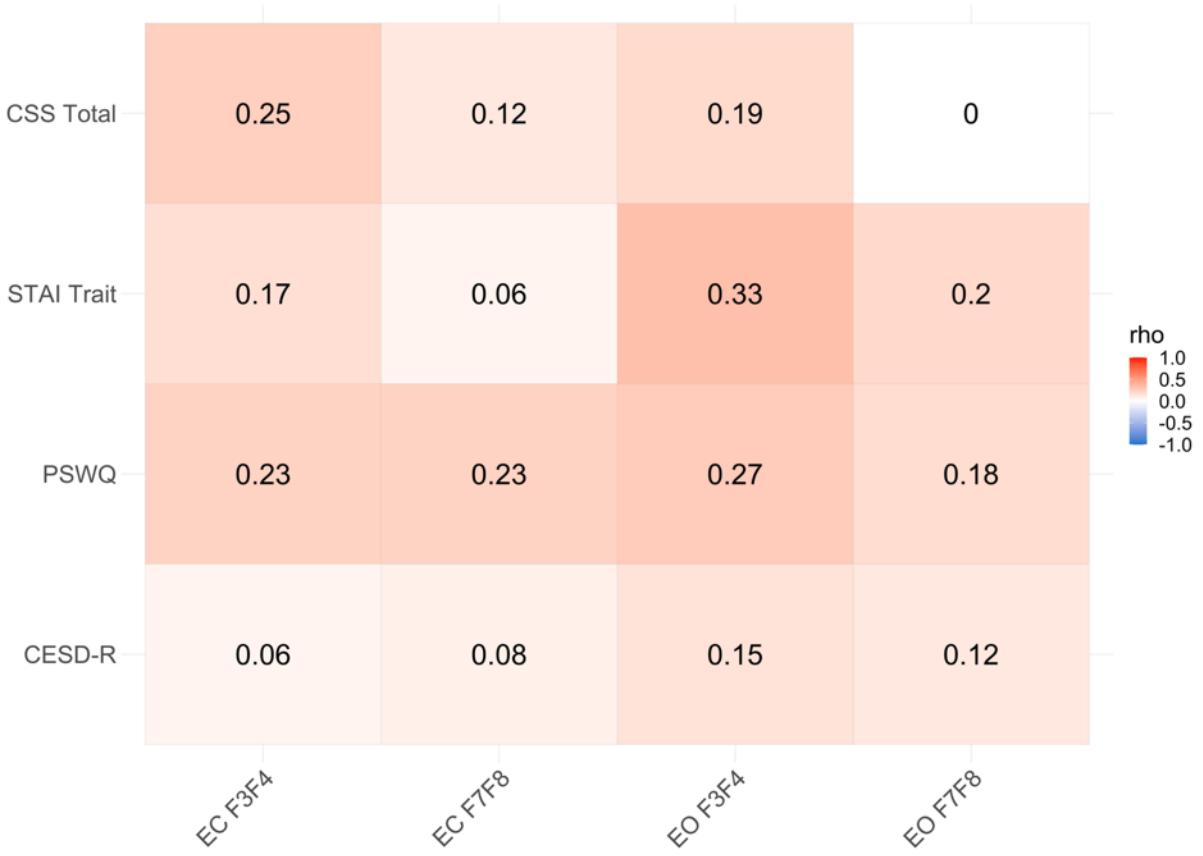

Figure 8. Correlation heatmap of electrode pair alpha AI with outcome variables for EC and EO data. Notes: Coefficients represent Spearman correlation coefficients. Red indicates positive correlation, blue indicates negative correlation. More intensive colors represent stronger correlations.

3.4.4 RBC scores

Preprocessing procedures achieved significantly different RBC ratings in EC ($\chi^2(2) = 75.62$, $p < .001$, $w = .804$) and EO data ($\chi^2(2) = 79.19$, $p < .001$, $w = .88$). In both EC and EO data, Automagic preprocessing resulted in significantly higher RBC scores than preprocessing by Rater 1 ($p < .001$) and Rater 2 ($p < .001$). Wilcoxon rank sum tests yielded higher RBC scores in automated preprocessing of EC ($W = 285$, $p < .001$, $r = .735$) and EO data ($W = 149$, $p < .001$, $r = .783$).

3.5 Alpha Asymmetry and Psychopathological Associations

3.5.1 Depression

Spearman correlation coefficients of CESD-R scores and AIs were calculated and visualized (Figure 8). For electrode pair F3F4, multiple regression did not reveal a significant model of EC data ($F(9,34) = 1.58$, $p = .16$), explaining 29.5% of variance (Appendix 3.1). No predictor weight reached significance ($p > .02$). The model for EO data explained 15% of variance (Appendix 3.1) and did not reach significance ($F(9,34) = 0.67$, $p = .731$). No predictor

contributed significantly to the model ($p > .02$). Multiple regression models using F7F8 AI as predictor (Appendix 3.2) did not explain a significant proportion of variance in EC ($R^2 = .12$, $F(9,34) = 0.54$, $p = .833$) and EO data ($R^2 = .24$, $F(9,34) = 1.21$, $p = .319$). For EC data, predictors did not significantly contribute to the model ($p > .02$). In EO data, significant contributions to the model were made by the AI of electrode pair F7F8 ($\beta = 4.26$, $p = .008$) and the interaction of age and F7F8 ($\beta = -4.14$, $p = .011$).

3.5.2 Worry

For heatmaps of Spearman correlations of PSWQ scores and AIs, refer to Figure 8. Multiple regression with the predictor F3F4 AI did not result in significant explanation of variance (Appendix 3.3) in EC ($R^2 = .34$, $F(9,34) = 1.92$, $p = .082$) and EO data ($R^2 = .31$, $F(9,34) = 1.72$, $p = .122$). No predictors contributed significantly to their respective models ($p > .02$). Performing multiple regression with the AI F7F8 (Appendix 3.4) did explain a significant proportion of variance in EC ($R^2 = .37$, $F(9,34) = 2.22$, $p = .045$) and EO data ($R^2 = .40$, $F(9,34) = 2.51$, $p = .025$). While in EC data no predictor significantly contributed to the model, in EO data age ($\beta = -0.50$, $p = .005$) and the interaction of sleepiness and F7F8 ($\beta = 1.17$, $p = .007$) reached significance.

3.5.3 Trait Anxiety

For heatmaps of Spearman correlations of STAI Trait Anxiety scores and AIs, refer to Figure 8. Multiple regression entering the predictor F3F4 AI (Appendix 3.5) did not yield significant explanation of variance in EC ($R^2 = .26$, $F(9,34) = 1.3$, $p = .274$) and EO data ($R^2 = .31$, $F(9,34) = 1.71$, $p = .126$). Predictors exhibited no significant contributions to the models ($p > .02$). Using AI F7F8 as predictor (Appendix 3.6) in multiple regression, we did not find models of EC ($R^2 = .28$, $F_{(9,34)} = 1.44$, $p = .211$ and EO data ($R^2 = .357$, $F(9,34) = 2.1$, $p = .058$) to explain a significant proportion of variance. In EC data, no predictor significantly contributed to the model ($p < .02$). In EO data, however, age ($\beta = -0.47$, $p = .008$), interaction of sleepiness and F7F8 ($\beta = 1.03$, $p = .02$) and interaction of age and F7F8 ($\beta = -3.82$, $p = .011$) reached significance.

3.5.4 COVID-19 Worry

For heatmaps of Spearman correlations of CSS total scores and AIs, refer to Figure 8. Multiple regression with the predictor F3F4 AI (Appendix 3.7) did not significantly explain variance in EC ($R^2 = .28$, $F(9,34) = 1.47$, $p = .20$) and EO data ($R^2 = .218$, $F(9,34) = 1.05$, $p = .42$). In the model of EC data, the interaction of age and F3F4 significantly contributed to the model ($\beta = -2.45$, $p = .009$). In the EO model, predictors made no significant contributions to the model ($p > .02$). With F7F8 as predictor (Appendix 3.8) in multiple regression, we did not uncover

significant explanation of variance for EC (R^2 = .21, F(9,34) = 0.97, p = .48 and EO data (R^2 = .13, F(9,34) = 0.56, p = .82). In EC data, the interaction of sleepiness and F7F8 significantly contributed to the model (β = -1.4, p = .017). In EO data, no predictor significantly contributed to the model (p > .02).

4 Discussion

Neuroscience is suffering from unacceptably low replicability. To combat this replication crisis, the #EEGManyLabs project will conduct replication efforts for the most influential EEG studies in neuroscience. Paving the way for the #EEGManyLabs Asymmetry and Automation spinoff projects, we aimed to assess the reliability and quality of automated preprocessing of EEG resting state data as compared to manual preprocessing. To this end, we tested 48 participants and recorded eyes closed and eyes open resting state EEG which we associated to various state and trait measures of psychopathology (depression, anxiety, worrying, COVID-19-related worrying) and personality. We found that interrater reliability of manual preprocessing is comparable to interrater reliability of manual and automated preprocessing. Automatically preprocessed data achieved better quality ratings in three out of for quality measures and worse ratings in the remaining measure. Multiple regression analysis on the association between frontal alpha asymmetries revealed a positive relationship between one AI of the electrode pair F7F8 and depression scores. No further significant associations of frontal AI and psychopathology were found. While our expectations regarding data quality were met, our findings regarding associations of psychopathology and frontal alpha asymmetry contradict out hypotheses.

4.1 Automagic Produces Higher-Quality Data

Interrater reliability between human raters ranged from moderate to excellent. This finding is in line with our hypothesis and previous research (Shirk et al., 2017). Interrater reliability between all preprocessing methods ranged from poor to moderate, indicating a less congruent preprocessing outcome. We expected interrater reliability between automatic and manual preprocessing to achieve similar reliability as manual preprocessing. It is, however, worth noting that only ICC at F3F4 was poor for all preprocessing methods. F3F4 is the only electrode pair that achieved excellent ICC when only assessing manual preprocessing. It is possible that through their knowledge of EEG alpha asymmetry research and their interest in frontal electrodes, raters were biased and thus hesitant to interpolate frontal channels or mark sections as artifacts, thereby achieving excellent interrater reliability. Automagic preprocessing is not biased and therefore could have rejected frontal channels which the raters would not reject. Other electrode pair interrater-reliability scores were of similar quality for manual-only and all-methods preprocessing. While our hypothesis was not supported in its entirety, we still conclude that automated and manual preprocessing result in comparable interrater reliability. It is worth noting, however, that ICC values should be interpreted with caution, as the mixed-

effect models ICC(3,1) mandated by our study design do not allow for generalization of interrater reliability (Koo & Li, 2016).

We showed that data preprocessed via Automagic received better quality ratings than manually preprocessed data in categories OHA, THV, and CHV. Only in RBC did manually preprocessed EEG data achieve better ratings. These findings are mostly in line with our hypothesis of favorable quality in Automagic data. We did not expect Automagic data to be rated worse in the RBC category. RBC quantifies the proportion of channels that were interpolated due to bad quality. Therefore, high RBC ratings correspond to a large ratio of channels that needed interpolation. As the underlying data for Automagic and the two raters was identical, a higher RBC score after Automagic preprocessing therefore implies that the Automagic pipeline identified significantly more channels as bad than its human counterparts. We therefore argue that Automagic simply identifies and interpolates bad channels more thoroughly than human raters. While this likely results in increased data loss, it also provides a less random and more reliable means of preprocessing for EEG data. Further, the overall extent of data loss is not only defined by the quantity of interpolated channels, but also their topographic clustering (Pedroni et al., 2019). Consequently, spatially distributed interpolations might not be as detrimental to overall data quality as the rating indicates.

4.2 Depression

Overall, we did not find frontal AIs to be associated with depression severity. This finding stands in contrast to many studies investigating the relationship between frontal EEG asymmetries and depression. However, the notion of frontal alpha asymmetry as a biomarker for depression is not unchallenged. In a recent meta-analysis, van der Vinne et al. (2017) did not find support for the hypothesis of a reliable frontal alpha asymmetry biomarker for depression, reporting effect sizes approximating to null. This result is consistent with our findings of no significant association of frontal asymmetry in three of four regression models. No model explained variance significantly, casting doubt over predictive quality of frontal alpha asymmetry as a depression marker. Further, at least 300 participants would be required to reveal stable effects of alpha asymmetry on depression (van der Vinne et al., 2017). Therefore, with 47 participants, our effort was underpowered. We only tested healthy participants that did not meet criteria for MDD diagnosis. Consequently, associations of frontal alpha asymmetry with depressive symptoms were restrained to a small range of subclinical scores within a healthy sample, whereas most original research was conducted with patients that met a clinical diagnosis (Allen et al., 2004; Allen & Reznik, 2015; Gotlib et al., 1998; Henriques & Davidson, 1991; Mitchell & Pössel, 2012; Nusslock et al., 2011; Schaffer et al.,

1983). As our participants did not fulfill diagnostic requirements for a disorder, we could therefore not categorize the continuous psychopathological predictors, as is the case in most of the original studies. By splitting data into two or more conditions and performing an ANOVA, detection of effects might have been facilitated as compared to our regression analysis. This discrepancy between original studies and our effort could in turn have been increased by the limited variability of scores within our sample.

AI of electrodes F7F8 was significantly positively associated with CESD-R scores in EO data, indicating that relative left lateralization of cortical activity is more pronounced in depressive persons. This finding of relative right lateralization of alpha power in participants with higher CESD-R scores is incompatible with our hypothesis of relative left frontal alpha asymmetry in depressed participants. However, in their meta-analysis, van der Vinne et al. (2017) revealed a similar pattern of frontal alpha asymmetry in female as compared to male participants. It is therefore conceivable that due to the overrepresentation of female participants in our sample, such inverse lateralization was found to be associated with depressive symptoms. This sex-dependent moderation of frontal alpha asymmetry in depressive symptoms would have been expected to emerge as a significant interaction of sex and AI in the regression model but did not reach significance in our data. We propose that our overall small sample size in convergence with the disproportionate sex ratio is likely the cause of this null-moderation. Even though the discriminative qualities of frontal EEG alpha asymmetries are inconclusive, with meta-analyses supporting (Thibodeau et al., 2006), disputing (van der Vinne et al., 2017) or avoiding (Kaiser et al., 2018) the understanding of frontal alpha asymmetries as predictor of depression, very few studies have reported positive associations of frontal asymmetries and depression. For instance, Jesulola et al. (2017) uncovered a pattern of greater right than left frontal alpha power in MDD participants. Consistent with the skewed sex ratio in our sample and findings by van der Vinne et al. (2017), Jesulola and colleagues discovered this inverse association in female participants only. This effect was confined to the same electrode pair (F7F8) as in our study. One further similarity of our study with the methodology of Jesulola et al. (2017) is the decision to record EEG signals in single, longer blocks of EC and EO condition to avoid disruption of the resting state through alternating instructions. As pointed out by Thibodeau et al. (2006), shorter recording episodes of RS EEG produce larger effects. This apparent reduction of effect sizes in convergence with overall low sample size could therefore lend further support to the notion that our findings do not represent a true positive association of frontal alpha asymmetry and depressive symptoms.

Our finding of a significant positive association between frontal AIs in one of four conditions does not support our hypothesis that left frontal hypoactivation is a predictor of depressive symptoms. Indeed, the only emerging significant association is in direct contradiction to our hypothesis and likely due to unrepresentative sample characteristics. As the present study mainly serves the assessment of preprocessing quality, we therefore do not recommend a generalization of our findings.

4.3 Worry, Trait Anxiety and COVID-19 Related Worry

Frontal AIs did not predict worrying symptoms in any condition. This finding contradicts our hypothesis of more left than right frontal activity in persons with pronounced worrying behavior. Previous research, though not as extensive as EEG depression research, presented conclusive evidence for the notion that worrying is predicted by relative left frontal cortical lateralization due to increased rumination via left-hemispheric verbal areas (Engels et al., 2007; Härpfer et al., 2021; Heller et al., 1997; Hofmann et al., 2005; Mathersul et al., 2008). To our knowledge, the only null-finding to date by Nitschke et al. (1999) was likely a consequence of insufficient sample sizes and potential mood induction due to the testing protocol. Notably, our effort suffered similar shortcomings regarding sample size. Moreover, none of the previous studies employed common average referencing, but either linked mastoids or CSD transformation. We, on the other hand, made use of common average referencing. While theoretically, an average reference could provide an ideal reference, topological asymmetries, imbalances, and low electrode density could result in a distortion of frontal alpha power which would be detrimental towards the objective of frontal alpha EEG research (Hagemann, 2004).

In contradiction to our hypothesis, we did not find trait anxiety to be associated with increased leftward alpha asymmetry. The most prominent theories of frontal asymmetry in anxiety predict altered lateralization in anxious participants. Davidson (1992) and Davidson et al. (1990) expected anxiety to be inherently avoidant, therefore resulting in greater right-sided activity. Heller (1993) and Heller et al. (1997) proposed a more nuanced model, suggesting that anxious apprehension would result in more left than right frontal cortical activity. Our results do not support these assumptions. While we did not anticipate this null finding, it is not the first instance where research did not reveal a relationship between trait anxiety and frontal asymmetries. Kentgen et al. (2000) reported no differences in alpha asymmetry at frontal sites between depressed, anxious, comorbid and control participants. Nitschke et al. (1999) found a right lateralization of frontal activity in anxious arousal participants. However, like Kentgen et al. (2000), they could not find effects for comorbid depression groups. A speculative reason for this phenomenon could lie in an equalization of alpha lateralization due to simultaneous

high rumination and depression. Considering that our sample was characterized by a high number of above-cut-off participants for both depressive and worrying symptoms, it is conceivable that co-occurrence of symptoms resulted in levelling out of frontal asymmetries. This notion is also supported by Mathersul et al. (2008), who reported more symmetrical frontal activation in nonclinical comorbid participants.

Frontal alpha asymmetries did not predict COVID-19 related worry. This finding stands in contrast to our hypothesis of stronger leftward lateralization of cortical activity in those with strong COVID-19 related worrying. We assumed that the ruminating characteristics of anxious apprehension would result in stronger left than right frontal activity (Heller, 1993; Heller et al., 1997). There are, however, methodological considerations that could account for this discrepancy. First, the measure of COVID-19 worry (CSS) was translated to German (see Appendix 4), but not validated before use. Therefore, we cannot rule out that the features of anxious apprehension of the original version are not accurately preserved in the German version. Moreover, while most items of the CSS revolve around worries about COVID-19 and the consequences of the pandemic, it also incorporates the factors *COVID compulsive checking and reassurance seeking*, and *COVID traumatic stress symptoms* (Taylor et al., 2020). Especially the compulsive component of the CSS might have contributed to this null finding. OCD is characterized by more right than left frontal cortical activity (Ischebeck et al., 2014; Kuskowski et al., 1993; Smith et al., 2016). Smith et al. (2016) also pointed out the frequent comorbidity of obsessive-compulsive symptomatology and depression as a potential cause of right-sided frontal asymmetry in OCD patients. This lateralization could potentially conflict with the expected left-sided asymmetry in worrying participants and eventually cancel out frontal asymmetries. Similar rightward lateralization, stemming from ventromedial PFC, dorsolateral PFC and amygdala, has also been reported in patients with PTSD (Kemp et al., 2010; see T. Meyer et al., 2015 for a review). As the traumatic stress factor of the CSS captures posttraumatic symptoms, such as hypervigilance and intrusions, it is possible that high scores in these items are associated with right-sided frontal activity, and thus drive the cancellation of lateralization even further.

4.4 Strengths and Limitations

Our research plays a crucial role in enabling the #EEGManyLabs projects to conduct the most extensive replication effort in EEG research to date. By employing and testing the automated EEG preprocessing pipeline Automagic on our alpha asymmetry data, we were able to assert that this pipeline is not just efficient and transparent, but also produces similarly reliable and objectively cleaner data than manual preprocessing. These findings allow for the use of

standardized preprocessing to handle the large datasets of the #EEGManyLabs effort. To our knowledge, this study was the first to compare manual and automated preprocessing via objective, standardized and transparent quality measures directly derived from the preprocessed data. Further, we sampled a large variety of measures in personality and psychopathology domains. These measures go beyond the ones analyzed for the present study, therefore allowing for future research to make use of our data. Our dataset, in the spirit of open science and transparent research, will be available to both #EEGManyLabs as well as the public via OSF. With our detailed accounts on the testing and preprocessing procedures, as well as the available data, replication and further analysis are encouraged. Finally, a strength of our study lies in the incorporation of the ongoing COVID-19 pandemic. We made use of this very rare, global, and psychologically relevant event to test ecologically valid, widespread worries for associations with frontal EEG alpha asymmetries.

However, there are also several shortcomings to our study. Firstly, distribution of sex and handedness did not match the overall population asymmetries reported (Destatis, 2021; Papadatou-Pastou et al., 2020). In this sample, female participants and left-handed participants are grossly overrepresented. Considering that both factors are associated with alterations of alpha asymmetry (Hirnstein et al., 2019; Ocklenburg et al., 2019), we cannot rule out that these sample characteristics affected the outcomes. Secondly, time of day of recordings varied within a range of eight hours between participants, with some recordings taking place shortly after awakening. As the cortisol awakening response elevates cortisol levels after awakening (Späth-Schwalbe et al., 1992), some EEG might have been recorded under high cortisol conditions. Increased cortisol levels are, in turn, associated with rightward frontal cortical lateralization of activity (Tops et al., 2005). Third, we only tested nonclinical participants for psychopathology and alpha asymmetries, thereby focusing on a different group than most original studies. As discussed by Mathersul et al. (2008), the sample discrepancy to previous studies as well as the reduced range of test scores likely results in small effect sizes. Therefore, a larger sample size might have been required to uncover said effects. This also implies that a priori power analysis of future research in nonclinical samples cannot rely on effect size measures of original studies, as they are likely not reflective of the reality in nonclinical participants.

Due to the amount of asymmetry research, the choice of one psychopathology questionnaire subsequently meant the exclusion of other measures and, consequently, methodological discrepancies with previous studies. Not only the choice of measures, but also of recording and preprocessing procedures might have ultimately reduced the conclusiveness of replication. As pointed out in many previous articles, there is considerable methodological

variation and degrees of freedom in EEG alpha asymmetry research (Kriegeskorte et al., 2009; Poldrack et al., 2017; Wicherts et al., 2016). In a way, our research, as many efforts before, fell victim to the sheer abundance of options with little consensus on how to apply them. While the tests employed in the present study are established questionnaires, various dimensions and neuropsychological subtypes are known to exist in both depression (Barlow, 1991; Drysdale et al., 2017) as well as anxious psychopathology (Barlow, 1991; Heller, 1993; Heller et al., 1997). Questionnaires in our study do capture specific subtypes (e.g., anxious arousal in STAI, anxious apprehension in PSWQ), but were not designed based on these theoretical constructs. It is therefore possible that our measures did not fully meet the complexity of the underlying psychopathological dimensions of depression and anxiety.

We did not split EC and EO phases into alternating, shorter blocks as often seen in RS EEG recordings. While in theory over the course of recording fatigue may have resulted in different alpha power, split-half reliability analysis showed high similarity of alpha power values and AIs between test halves. We interpret this as evidence that alpha EEG signals as well as AIs derived from alpha signal are a temporally (~10 min) stable and reliable measures that allow for associations with behavioral measures. The high split-half reliability indicates that EEG alpha power did not hugely vary over the course of the recording. While this does not allow for inferences about alpha strength, it implies that factors such as attention or fatigue likely do not influence alpha signal over a single block recording.

Since testing was conducted with German-speaking participants, the CSS was translated, but not validated before use. Therefore, we cannot claim that original test quality criteria were met or that linguistic details of the original scales were preserved. However, the benefits of being able to test worry in an ecologically valid situation outclassed the concerns over test quality. Our choice of an average reference may have resulted in contamination of frontal alpha power by occipital sites via mirroring. An alternative to this reference could lie within a Laplacian (CSD) transformation. An advantage of the CSD method is its potential to reduce the effect of distal electrode alpha on power of the frontal electrodes, thereby enabling a more accurate recording of ‘true’ frontal alpha power (Hagemann, 2004).

4.5 Conclusion and Future Directions

Despite these limitations to our study, it provides evidence for the applicability and suitability of Automagic-powered standardized preprocessing. Our results indicate similar reliability as and better data quality than semi-automated EEG preprocessing. These findings enable the #EEGManyLabs to preprocess their enormous database with minimal effort and cost while ensuring transparent quality assessment and reproducibility. While we could not conclusively

assert whether automatically preprocessed RS EEG alpha asymmetries could serve as a biomarker for depression, various forms of anxiety, and COVID-19 related worry we demonstrated the need for reliable and empirically supported methodological guidelines as well as large sample sizes in EEG alpha asymmetry research. Going forward, the #EEGManyLabs Asymmetry spin-off project shall investigate the association of frontal alpha asymmetry and psychopathology in a large, demographically representative sample. Additionally, a systematic evaluation of the influence of reference electrode, eye status and statistical analysis on the association of alpha asymmetry and psychopathology should be conducted. In the future, machine learning approaches and clustering approaches as discussed in Ocklenburg et al. (2021) could prove to be a more a reliable and less controversial means to identify EEG patterns as biomarkers of mental illness. In the field of EEG-based classification, there have already been several successful demonstrations regarding classification of depression (Acharya et al., 2018; Hosseinifard et al., 2013; Wan et al., 2019), diagnosis of depression subtypes (Zelenina & Prata, 2019), depression severity (Mohammadi et al., 2019), and treatment response (Hasanzadeh et al., 2019; Jaworska et al., 2019; Khodayari-Rostamabad et al., 2013).

We conclude that automated preprocessing via Automagic is a suitable means of EEG data preprocessing for the #EEGManyLabs project. Our findings highlight the need for transparent reporting and systematic evaluation of the most influential factors and DFs in EEG asymmetry research.

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Conflict of Interest

The authors declare that research was not influenced by conflicts of interest or any personal, financial, or other relationship.

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Appendix

Appendix 1: Split-half Reliability

Appendix 1.1
Split-half reliability scores of EC condition

	Automagic		Rater 1		Rater 2	
	EC	95% CI	EC	95% CI	EC	95% CI
F3	0.97	CI[0.88-0.96]	0.97	CI[0.9-0.97]	0.13	CI[-0.22-0.35]
F7	0.96	CI[0.87-0.96]	0.96	CI[0.87-0.96]	0.07	CI[-0.26-0.32]
P3	0.95	CI[0.84-0.95]	0.37	CI[-0.06-0.48]	0.09	CI[-0.24-0.33]
P7	0.97	CI[0.91-0.97]	0.97	CI[0.88-0.96]	0.96	CI[0.88-0.96]
O1	0.94	CI[0.81-0.94]	0.95	CI[0.84-0.95]	0.76	CI[0.4-0.77]
O2	0.95	CI[0.83-0.95]	0.96	CI[0.88-0.96]	0.66	CI[0.24-0.69]
P4	0.96	CI[0.88-0.96]	0.97	CI[0.9-0.97]	0.78	CI[0.43-0.78]
P8	0.98	CI[0.91-0.97]	0.98	CI[0.93-0.98]	0.96	CI[0.85-0.95]
F4	0.96	CI[0.86-0.96]	0.96	CI[0.88-0.96]	0.24	CI[-0.16-0.41]
F8	0.95	CI[0.82-0.94]	0.95	CI[0.85-0.95]	0.17	CI[-0.2-0.37]

Note: SB = Spearman-Brown coefficient, CI = Confidence Interval. Unacceptable < .7, acceptable ≥ .7, excellent ≥ .95.

Appendix 1.1
Split-half reliability scores of EO condition

	Automagic		Rater 1		Rater 2	
	EO	95% CI	EO	95% CI	EO	95% CI
F3	0.99	CI[0.98-0.99]	0.98	CI[0.93-0.98]	0.98	CI[0.92-0.98]
F7	0.99	CI[0.96-0.99]	0.97	CI[0.89-0.96]	0.98	CI[0.92-0.98]
P3	0.99	CI[0.95-0.99]	0.97	CI[0.9-0.97]	0.97	CI[0.91-0.97]
P7	0.99	CI[0.96-0.99]	0.98	CI[0.92-0.97]	0.98	CI[0.93-0.98]
O1	0.99	CI[0.95-0.99]	0.97	CI[0.91-0.97]	0.96	CI[0.86-0.96]
O2	0.99	CI[0.96-0.99]	0.98	CI[0.93-0.98]	0.98	CI[0.92-0.98]
P4	0.98	CI[0.94-0.98]	0.96	CI[0.87-0.96]	0.96	CI[0.87-0.96]
P8	1	CI[0.99-1]	0.99	CI[0.97-0.99]	0.99	CI[0.97-0.99]
F4	0.99	CI[0.95-0.98]	0.96	CI[0.88-0.96]	0.97	CI[0.89-0.97]
F8	0.98	CI[0.93-0.98]	0.78	CI[0.42-0.78]	0.79	CI[0.45-0.8]

Note: SB = Spearman-Brown coefficient, CI = Confidence Interval. Unacceptable < .7, acceptable ≥ .7, excellent ≥ .95.

Appendix 2: Interrater Reliability

Appendix 2
ICC of AIs between Automagic and single manual raters.

	Automagic & Rater 1		Automagic & Rater 2	
	EC [95% CI]	EO [95% CI]	EC [95% CI]	EO [95% CI]
F3F4	-0.12 [-0.41-0.2]	0.24 [-0.07-0.51]	-0.16 [-0.45-0.15]	0.24 [-0.07-0.5]
F7F8	0.82 [0.68-0.9]	0.76 [0.6-0.87]	0.71 [0.51-0.83]	0.56 [0.31-0.73]
P3P4	0.88 [0.79-0.94]	0.71 [0.52-0.84]	0.61 [0.37-0.77]	0.7 [0.51-0.83]
P7P8	0.91 [0.84-0.95]	0.78 [0.62-0.87]	0.69 [0.48-0.82]	0.51 [0.25-0.71]
O1O2	0.92 [0.86-0.96]	0.74 [0.56-0.85]	0.7 [0.5-0.83]	0.32 [0.02-0.56]

Note: Manual preprocessing: Rater 1 & Rater 2, All preprocessing methods: Rater 1, Rater 2 & Automagic. CI = Confidence Interval. Poor < .5, moderate < .75, good < .90, excellent ≥ .90.

Appendix 3: Regression Tables

Appendix 3.1
Regression results using CESD-R Scores as the criterion

	EC		EO	
Predictor	*β*	Fit	*β*	Fit
Handedness	-0.16	R^2 = .295	-0.08	R^2 = .150
Sleepiness	0	95% CI[.00,.36]	-0.02	95% CI[.00,.17]
Sex-male	0.1		0.1	
Age	-0.23		-0.06	
F3F4	1.08		0.64	
Handedness:F3F4	0.08		0.11	
Sex-male:F3F4	0.4		0.08	
Sleepiness:F3F4	0.33		0.19	
Age:F3F4	-1.42		-0.71	

Note. β represents standardized regression weights. Bracketed values indicate lower and upper CI limits.
* indicates p < .02. ** indicates p < .01.

Appendix 3.2

Regression results using CESD-R Scores as the criterion

	EC		EO	
Predictor	β	Fit	β	Fit
Handedness	0.02	R^2 = .125	-0.02	R^2 = .243
Sleepiness	-0.01	95% CI[.00,.13]	0.16	95% CI[.00,.30]
Sex-male	0.15		0.08	
Age	-0.36		-0.44	
F7F8	1.48		4.26**	
Handedness:F7F8	-0.07		0.02	
Sex-male:F7F8	0.07		0.09	
Sleepiness:F7F8	0.19		-0.1	
Age:F7F8	-1.6		-4.14*	

Note. β represents standardized regression weights. Bracketed values indicate lower and upper CI limits.

* indicates $p < .02$. ** indicates $p < .01$.

Appendix 3.3
Regression results using *PSWQ* Scores as the criterion

	EC		EO	
Predictor	β	Fit	β	Fit
Handedness	-0.07	R^2 = .337	0.01	R^2 = .313
Sleepiness	0.01	95% CI[.00,.41]	0.14	95% CI[.00,.38]
Sex-male	-0.19		-0.26	
Age	-0.3		-0.18	
F3F4	1.37		0.83	
Handedness:F3F4	-0.25		-0.11	
Sex-male:F3F4	0.02		-0.37	
Sleepiness:F3F4	0.56		0.46	
Age:F3F4	-1.73		-0.83	

Note. β represents standardized regression weights. Bracketed values indicate lower and upper CI limits.
* indicates $p < .02$. ** indicates $p < .01$.

Appendix 3.4
Regression results using *PSWQ* Scores as the criterion

	EC		EO	
Predictor	*β*	Fit	*β*	Fit
Handedness	0.09	R^2 = .370	0.12	R^2 = .399
Sleepiness	-0.05	95% CI[.00,.44]	0.11	95% CI[.00,.47]
Sex-male	-0.2		-0.25	
Age	-0.42		-0.5**	
F7F8	1.03		2.41	
Handedness:F7F8	-0.09		-0.04	
Sex-male:F7F8	-0.08		-0.25	
Sleepiness:F7F8	0.96		1.17**	
Age:F7F8	-1.56		-3.14	

Note. β represents standardized regression weights. Bracketed values indicate lower and upper CI limits.
* indicates p < .02. ** indicates p < .01.

Appendix 3.5
Regression results using *STAI Trait Anxiety* Scores as the criterion

	EC		EO	
Predictor	β	Fit	β	Fit
Handedness	-0.08	R^2 = .256	0	R^2 = .311
Sleepiness	0.19	95% CI[.00,.32]	0.24	95% CI[.00,.38]
Sex-male	-0.02		-0.05	
Age	-0.22		-0.12	
F3F4	1.59		0.7	
Handedness:F3F4	-0.2		-0.07	
Sex-male:F3F4	0.05		-0.3	
Sleepiness:F3F4	0.33		0.59	
Age:F3F4	-1.75		-0.79	

Note. β represents standardized regression weights. Bracketed values indicate lower and upper CI limits.
* indicates p < .02. ** indicates p < .01.

Appendix 3.6
Regression results using *STAI Trait Anxiety* Scores as the criterion

	EC		EO	
Predictor	β	Fit	β	Fit
Handedness	0.08	R^2 = .276	0.12	R^2 = .357
Sleepiness	0.14	95% CI[.00,.34]	0.29	95% CI[.00,.43]
Sex-male	-0.02		-0.04	
Age	-0.37		-0.47**	
F7F8	1.61		3.18	
Handedness:F7F8	-0.15		-0.02	
Sex-male:F7F8	-0.14		-0.38	
Sleepiness:F7F8	0.53		1.03*	
Age:F7F8	-1.83		-3.82*	

Note. β represents standardized regression weights. Bracketed values indicate lower and upper CI limits.
* indicates $p < .02$. ** indicates $p < .01$.

Appendix 3.7
Regression results using *CSS Total* Scores as the criterion

	EC		EO	
Predictor	β	Fit	β	Fit
Handedness	-0.16	R^2 = .280	-0.17	R^2 = .218
Sleepiness	-0.06	95% CI[.00,.35]	0.13	95% CI[.00,.27]
Sex-male	0.12		0.12	
Age	0.02		-0.08	
F3F4	2.2		1.3	
Handedness:F3F4	-0.07		0.13	
Sex-male:F3F4	-0.37		-0.4	
Sleepiness:F3F4	0.46		-0.31	
Age:F3F4	-2.45**		-0.65	

Note. β represents standardized regression weights. Bracketed values indicate lower and upper CI limits.
* indicates p < .02. ** indicates p < .01.

Appendix 3.8
Regression results using *CSS Total* Scores as the criterion

	EC		EO	
Predictor	*β*	Fit	*β*	Fit
Handedness	-0.21	R^2 = .205	-0.14	R^2 = .129
Sleepiness	-0.01	95% CI[.00,.25]	-0.01	95% CI[.00,.14]
Sex-male	0.07		0.06	
Age	-0.26		-0.3	
F7F8	0.4		1.15	
Handedness:F7F8	0.1		-0.04	
Sex-male:F7F8	0.1		-0.14	
Sleepiness:F7F8	1.4*		0.72	
Age:F7F8	-1.86		-1.78	

Note. β represents standardized regression weights. Bracketed values indicate lower and upper CI limits.
* indicates p < .02. ** indicates p < .01.

Appendix 4: German Version of the COVID Stress Scales

Der nachfolgende Fragebogen bezieht sich auf verschiedene Arten von Sorgen, die Sie möglicherweise in den vergangenen sieben Tagen erlebt haben. In den nachfolgenden Statements bezeichnen wir COVID-19 als "das Virus".

	Gar nicht (1)	Ein bisschen (2)	Moderat (3)	Sehr (4)	Extrem (5)
1. Ich mache mir Sorgen darüber das Virus zu kriegen. (1)	○	○	○	○	○
2. Ich mache mir Sorgen, dass ich meine Familie nicht vor dem Virus schützen kann. (2)	○	○	○	○	○
3. Ich mache mir Sorgen, dass unser Gesundheitssystem meine Lieben nicht schützen können wird. (3)	○	○	○	○	○
4. Ich mache mir Sorgen, dass unser Gesundheitssystem mich nicht vor dem Virus schützen können wird. (4)	○	○	○	○	○
5. Ich mache mir sorgen, dass grundlegende Hygiene (z.B. Händewaschen) nicht ausreicht um mich vor dem Virus zu schützen. (5)	○	○	○	○	○
6. Ich mache mir Sorgen, dass Social Distancing nicht ausreicht um mich vor dem Virus zu schützen. (6)	○	○	○	○	○
7. Ich mache mir Sorgen darüber, dass den Lebensmittelgeschäften die Lebensmittel ausgehen. (7)	○	○	○	○	○
8. Ich mache mir Sorgen, dass Lebensmittelgeschäfte schließen werden. (8)	○	○	○	○	○

9. Ich mache mir Sorgen darüber, dass Geschäften die Reinigungs- und Desinfektionsmittel ausgehen. (9)	○	○	○	○	○
10. Ich mache mir Sorgen darüber, dass Geschäften die Erkältungs- und Grippemittel ausgehen. (10)	○	○	○	○	○
11. Ich mache mir Sorgen darüber, dass Lebensmittelgeschäften das Wasser ausgeht. (11)	○	○	○	○	○
12. Ich mache mir Sorgen, dass Apotheken verschreibungspflichtige Medikamente ausgehen. (12)	○	○	○	○	○
13. Ich mache mir Sorgen, dass Ausländer das Virus in meinem Land verbreiten. (13)	○	○	○	○	○
14. Würde ich in ein Restaurant für ausländisches Essen gehen, wäre ich besorgt, mich mit dem Virus anzustecken. (14)	○	○	○	○	○
15. Ich mache mir Sorgen darüber, dass ich mit Ausländern in Kontakt gerate, weil sie das Virus haben könnten. (15)	○	○	○	○	○
16. Würde ich eine Person aus dem Ausland treffen, wäre ich besorgt, dass sie das Virus hat. (16)	○	○	○	○	○
17. Wäre ich im Aufzug mit einer Gruppe Ausländer, so wäre ich besorgt, dass sie mit dem Virus infiziert sind. (17)	○	○	○	○	○

18. Ich mache mir Sorgen, dass Ausländer das Virus verbreiten, weil sie nicht so sauber sind wie wir. (18)	○	○	○	○	○
19. Ich mache mir Sorgen, dass ich mich, wenn ich etwas in der Öffentlichkeit berühren würde, mit dem Virus anstecken würde. (19)	○	○	○	○	○
20. Ich mache mir Sorgen, dass ich mich, wenn jemand in meiner Nähe husten oder niesen würde, mit dem Virus anstecken würde. (20)	○	○	○	○	○
21. Ich mache mir Sorgen, dass Leute um mich herum mich mit dem Virus infizieren werden. (21)	○	○	○	○	○
22. Ich mache mir Sorgen darüber, Wechselgeld bei Bargeldgeschäften anzunehmen. (22)	○	○	○	○	○
23. Ich mache mir Sorgen, dass ich mich beim Umgang mit Geld oder Verwenden eines EC-Kartenterminals mit dem Virus anstecken könnte. (23)	○	○	○	○	○
24. Ich mache mir Sorgen, dass meine Post vom Postboten kontaminiert worden ist. (24)	○	○	○	○	○

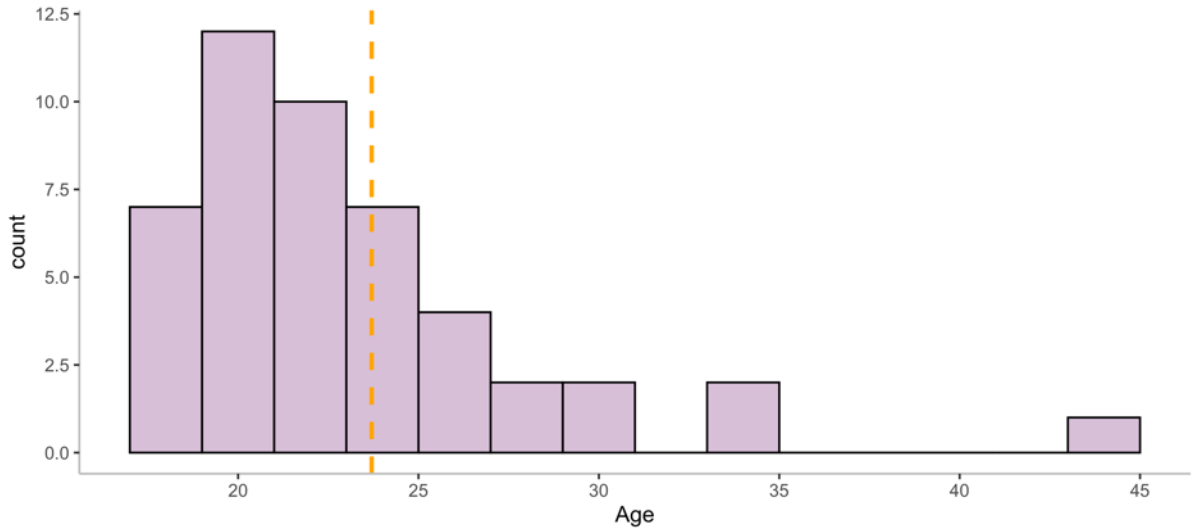
Bitte lesen Sie jede Aussage und geben Sie an, wie oft Sie jedes Problem in den letzen 7 Tagen erfahren haben.

	Nie (1)	Selten (2)	Manchmal (3)	Oft (4)	Nahezu immer (5)
25. Ich hatte Schwierigkeiten mich zu konzentrieren, weil ich an das Virus dachte. (1)	○	○	○	○	○
26. Beunruhigende geistige Bilder tauchten gegen meinen Willen auf. (2)	○	○	○	○	○
27. Ich hatte Schlafschwierigkeiten, weil ich an das Virus dachte. (3)	○	○	○	○	○
28. Ich dachte an das Virus wenn ich es nicht wollte. (4)	○	○	○	○	○
29. Erinnerungen an das Virus lösten bei mir körperliche Reaktionen, wie Schwitzen oder Herzklopfen, aus. (5)	○	○	○	○	○
30. Ich hatte schlechte Träume über das Virus. (6)	○	○	○	○	○

Die nachfolgenden Items erfragen Absicherungsverhalten. Wie oft haben sie folgende Dinge aus Sorge über COVID-19 während der letzten sieben Tage getan?

	Nie (1)	Selten (2)	Manchmal (3)	Oft (4)	Nahezu immer (5)
31. Im Internet nach Behandlungen für COVID-19 gesucht. (1)	○	○	○	○	○
32. Gesundheitsexperten (z.B. Ärzte oder Apotheker) nach Rat über COVID-19 gefragt. (2)	○	○	○	○	○
33. YouTube Videos zu COVID-19 angesehen. (3)	○	○	○	○	○
34. Den eigenen Körper auf Anzeichen einer Infektion überprüft (z.B. Temperatur messen). (4)	○	○	○	○	○
35. Bei Freunden oder Familie Rückversicherung über COVID-19 gesucht. (5)	○	○	○	○	○
36. Social Media-Posts zu COVID-19 angesehen. (6)	○	○	○	○	○

Appendix 5: Age Distribution



Appendix 5 Distribution (*n* = 47) of participant age. Dashed line represents mean age.