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CONFLICT-RELATED N2 ERP COMPONENT IN CO-EXISTENT DEPRESSION AND BURNOUT IS UNIQUELY PREDICTED BY DEPRESSION

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Abstract

The objective of the present study was to refine the understanding of conflict processing mechanisms in overlapping depression and burnout by (1) analyzing the N2 component of event-related potentials (ERPs), and (2) applying covariate statistical models to control for the shared variance between depression and burnout in a subclinical sample. N2 ERP component was recorded in 43 participants during a Flanker task. PHQ-9 and OLBI inventories were used to assess depression and burnout, respectively. Performance and N2 were analyzed using ANCOVA with simultaneous PHQ-9, OLBI and their interaction covariates. Analysis accounting for shared variance showed that (1) performance depended on syndrome interaction, and (2) only depression affected N2 during high-conflict information processing. Together, the findings indicate that depression is the primary determinant of altered conflict-monitoring, with behaviour being modulated by syndromes interaction. New evidence is provided that, although burnout and depression overlap at the phenomenological level, they are not neurophysiologically identical when analyzed under appropriate statistical control.

Key words: depression, burnout, ERP N2 component, overlapping syndromes

Introduction. Burnout and depression are recognized as two socially and clinically significant phenomena that have profound impact on individual well-being and public health. However, it has been argued that the contextual specificity of burnout, i.e., the work-related attribution of symptoms, is not sufficient

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to justify a separate diagnostic category. Rather, burnout is suggested to be more accurately conceptualized as a form of job-related depression as manifesting moderate to strong correlations with depressive symptoms [1, 2]. Understanding the distinction between the two syndromes directly impacts on diagnostic practices, prevention strategies, intervention, and allocation of healthcare and organizational resources [3].

One advantageous strategy to disentangle the two syndromes relies on the employment of objective indicators of neurocognitive functioning in order to move beyond exclusive reliance on self-report measures, which are inherently vulnerable to shared variance and conceptual circularity. On the contrary, such measures target a level of analysis that can reveal if similar symptom profiles of the two syndromes are supported by common or dissociable neural mechanisms. Event-related potentials (ERPs), in particular, offer fine-grained temporal resolution of the neural mechanisms associated with sensory, motor and cognitive functions [4].

Using ERPs has previously demonstrated that both depression and burnout manifest deviations in one of the fundamental mechanisms of cognitive control, namely performance monitoring [5–9]. Performance monitoring is critical for adaptive behaviour as it enables the detection and resolution of conflicts, specifically the detection of competing response tendencies under conditions of interference and uncertainty [10]. However, previous ERP studies typically examine depression and burnout separately, which does not allow direct comparisons. Even when co-existing syndromes are evaluated in a single sample, the large overlapping of burnout and depression [1] prevents the reliable separation of groups with only depression (without burnout) and only burnout (without depression) [7,11]. In addition, in previous ERP research, depression is traditionally examined in patients with clinically manifested Major Depressive Disorder (MDD) [12], and rarely in subclinical individuals [7, 11] whereas burnout is typically explored at a subclinical level. Both the overlapping syndromes and the different stages of symptom expression may have affected analysis outcomes.

In view of these previous confounders, the major objective of the present study was to analyze ERPs and explore the neural correlates of conflict monitoring in co-existing subclinical depression and burnout as reflected by the fronto-central N2 ERP by controlling for syndrome overlap. The N2 component was chosen for analysis as being consistently associated with conflict detection and inhibitory engagement within medial frontal systems of the brain [4, 10, 13]. To manipulate conflict monitoring, the Eriksen Flanker task was employed as one of the most established paradigms for exploring conflict monitoring and interference control [14]. In addition and most importantly, the present study used covariate statistical models to control for the shared variance between depressive and burnout symptoms. Dissociating syndrome-specific and common effects would help to clarify whether depression and burnout exert distinct or overlapping influences on neural systems supporting conflict monitoring. Also, population with

high-risk for occupational stress was included, yet none of the individuals manifested any clinical symptoms.

The results provide evidence that, although burnout and depression overlap at the phenomenological level, they are not neurophysiologically identical when analyzed under appropriate statistical control.

Material and methods. ***Participants.*** Forty-three healthy participants from stress-related jobs were included (public healthcare and social sectors $n = 17$, business companies $n = 19$, and other $n = 7$). Participants' mean age was 42.6 ± 1.8 years. Females were 65.12%. Inclusion criteria required the absence of sensory, neurological and psychiatric disorders like clinically diagnosed major depression or burnout so that only persons with subclinical burnout and depression participated. The study was approved by the local ethics committee at the Institute of Neurobiology, Bulgarian Academy of Sciences (KBE 52/02.09.2024). All subjects gave written informed consent in accordance with the Declaration of Helsinki.

Assessment of depression scores (PHQ-9) and burnout scores (OLBI). The questionnaire used for the assessment of burnout symptoms was the Oldenburg Burnout Inventory (OLBI) [15], including emotional exhaustion (EE) and detachment (DT) subscales. OLBI total score (OLBI-Tot) was computed as a sum of EE and DT scores. Depressive symptoms were assessed by the Patient Health Questionnaire 9 (PHQ-9) [16]. For complementary analysis participants were split according to the median of the OLBI scale into two subgroups with high burnout (BU+, OLBI-Tot ≥ 39, $n = 20$, mean 46.8, SD ± 4.9) and low burnout (BU–; OLBI-Tot < 39, $n = 23$, mean 33.83, SD ± 5.1). Depression subgroups were formed by the symptom severity categorization [16] into groups with minimal and mild depression: DE–, low to mild PHQ-9 scores 0–8, mean 4.58, SD ± 1.89, $n = 24$, and a sub-group with higher PHQ-9 scores: PHQ-9 > 8, indicating moderate to expressed depression (DE+, PHQ-9 scores 9–19, mean 12.58, SD ± 3.8, $n = 19$). The sub-groups did not differ with respect to age and sex distribution. The Pearson correlation coefficient was computed between PHQ-9 and OLBI scores and indicated a considerable overlap between subclinical burnout and depression ($r = 0.550$; $p < 0.001$).

The Flanker task. In the current study, two flankers (triangles with apexes directed to the left or right) were displayed centrally in the upper and lower visual field. After 100 ms, one of the four types of stimuli (triangle targets) appeared between the two flankers. Participants were asked to press a left or right keyboard key with the respective hand corresponding to the direction of only the target middle triangle (Go-trial). If the apex of the middle triangle was truncated, they were asked to refrain from pressing (NoGo trial). The presentation probabilities were 50% for the congruent Go (coinciding directions of flankers and targets, $n = 104$), 25% for the incongruent Go (opposite directions of flankers and targets, $n = 52$), and 25% for the NoGo stimuli ($n = 52$).

EEG and ERP methodology. EEG was recorded continuously by means of Neuvo Compumedics 128-channel system (`http://www.compumedics.com`) from 64 scalp electrodes according to the extended 10–10 system and mounted on an elastic cap (Quick-Cap HydroCell Array). For analysis, the EEG was referenced offline to linked mastoids. Vertical and horizontal electrooculogram (EOG) was recorded bipolarly. Electrode impedance was kept below 10 kΩ. The amplifier bandpass was DC-2000 Hz. EEG and EOG were recorded with a rate of 1000 Hz and resampled off-line to 250 Hz. EEG pre-processing and analysis were performed with BrainVision Analyzer (version 2.3.1, Gilching, Germany). Stimulus-related EEG epochs of 1400-ms length with a 200-ms baseline before flankers were obtained off-line. Epochs with amplitudes exceeding ±100 μV were rejected along with epochs containing technical or unwanted noise. Averaged event-related potentials (ERPs) were computed from the artefact-free epochs, separately for the congruent correct (CC, individual number of trials included in averaged ERPs between 90 and 104), incongruent correct (IC, $n = 42$–52), and NoGo correct (NoGo, $n = 42$–53) stimuli.

Amplitudes of the N2 component of the averaged ERPs were measured against the baseline as the most negative peak in the time window 350–480 ms (e.g., the 100 ms flanker duration taken into account (Fig. 1A)) at 10 relevant frontal and fronto-central electrodes.

Statistical analysis. The main analysis was a *simultaneous covariate analysis* performed separately for reaction times to CC and IC, error rates, and N2 measures. The design was a repeated-measures ANCOVAs simultaneously including as covariates PHQ-9 and OLBI scores together with their interaction, and within-subjects topographic variables (for N2) – Region (Reg) and Laterality (Lat). Covariates were mean-centred to reduce nonessential multicollinearity and improve interpretation of main and interaction effects. This approach was designed to identify effects specifically attributable to depression or burnout beyond their overlap or interaction. As a *complementary group-level analysis*, repeated-measures ANOVAs were performed separately including depression status (PHQ-9: DE+ vs. DE–) or burnout status (OLBI: BU+ vs. BU–) as a between-subjects factor, and within-subjects topographic variables.

Results. ***Performance.*** *Simultaneous covariate analyses*: RT ANCOVA revealed a significant main effect for OLBI ($F(1/39) = 12.8$, $p < 0.001$, $\eta_\rho^2 = 0.263$) reflecting RT decrease with burnout increase, a trend effect for PHQ-9 ($F(1/39) = 3.7$, $p = 0.06$, $\eta_\rho^2 = 0.094$) reflecting RT decrease with depression increase, and a significant interaction PHQ-9 x OLBI ($F(1/39) = 4.0$, $p = 0.05$, $\eta_\rho^2 = 0.100$) reflecting response speeding only with simultaneous burnout and depression increase, and RT slowing with pure depression (without burnout) increase. Error rate did not depend on any covariate ($p > 0.2$).

Complementary group-level analyses: Error rate was significantly higher in DE+ than DE– ($F(1/41) = 4.74$, $p = 0.03$, $\eta_\rho^2 = 0.104$). In contrast, RT was faster in BU+ relative to BU– ($F(1/41) = 4.8$, $p = 0.03$, $\eta_\rho^2 = 0.113$).

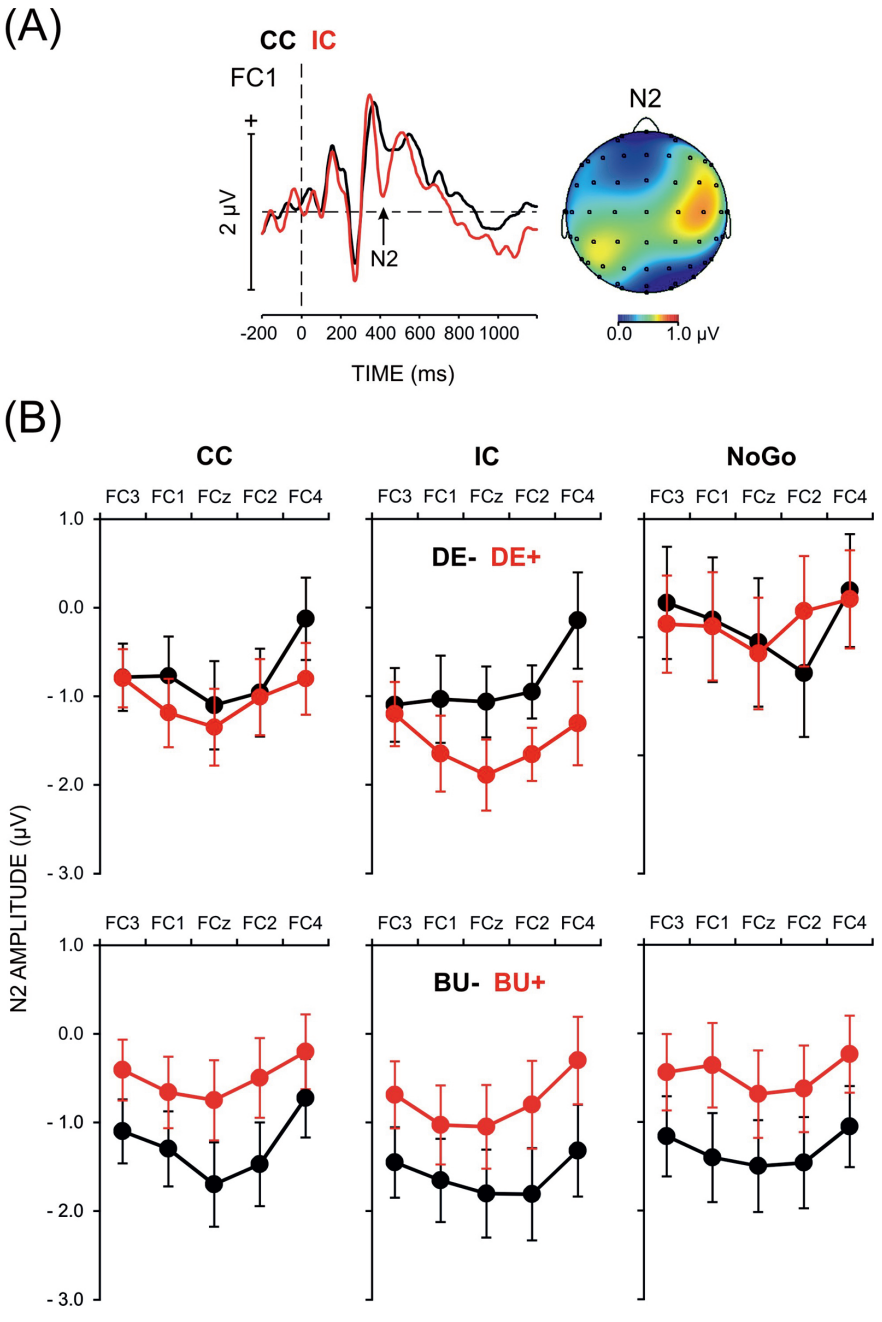

Fig. 1. (A) Event-related brain potentials (ERPs) for congruent correct (CC) and incongruent correct (IC) stimuli from FC1 electrode. Positivity upwards. The topography of N2 ERP component is shown on the right. (B) Group mean values ± SE of N2 amplitude at relevant fronto-central electrodes (FC3, FC1, FCz, FC2, FC4) for three stimulus types – CC, IC, and NoGo correct (NoGo). Upper panel demonstrates group mean N2 amplitude for depression groups DE– and DE+. Lower panel demonstrates group mean N2 amplitude for burnout groups BU– and BU+

Fronto-central N2 ERP component. *Simultaneous covariate analyses*: Including simultaneous PHQ-9 and OLBI covariates in the analysis yielded an interactive effect of only PHQ-9 on N2 (Reg × Lat × Stim × PHQ-9 ($F(8/312) = 2.3$, $p = 0.005$, $\eta_\rho^2 = 0.050$). As indexed by the interaction, the PHQ-9 covariate was significant for IC at midline and right fronto-central electrode (Reg × Lat × PHQ-9, $F(4/156) = 4.5$, $p = 0.04$, $\eta_\rho^2 = 0.080$) due to N2 increase with depression increase implying that variance in N2 amplitude associated with IC can be uniquely explained by PHQ-9, not by burnout, once their shared variance is accounted for.

Complementary group-level analyses: Figure 1 demonstrates that depression affected N2 depending on stimulus type at specific electrodes (Reg × Lat × Stim × PHQ-9: $F(8/328) = 2.5$, $p = 0.02$, $\eta_\rho^2 = 0.056$). Only for IC was N2 larger in DE+ relative to DE– (IC: Reg × Lat × PHQ-9, $F(4/164) = 4.5$, $p = 0.005$, $\eta_\rho^2 = 0.100$). In contrast to PHQ-9, N2 tended to be smaller in BU+ relative to BU– depending on stimulus type and electrode (Reg × Lat × Stim × OLBI ($F(8/328) = 2.02$, $p = 0.06$, $\eta_\rho^2 = 0.056$) (Fig. 1B), and again, this effect was most expressed for IC ($p < 0.01$).

Discussion. The present study aimed to clarify if neurophysiological processes of conflict processing differ between subclinical depression and burnout by examining behavioural performance and N2 ERP component during a Flanker task, while explicitly controlling for shared variance of the two syndromes [1]. The results provide evidence that, although burnout and depression overlap at the phenomenological level, they show dissociable effects on conflict monitoring as indexed by N2 [13].

Notably, at the group-level analysis depressive symptoms were associated with increased N2 amplitude to incongruent stimuli, whereas burnout showed an opposite tendency toward reduced N2. The divergence between increased N2 in depression and decreased N2 in burnout suggests that the two syndromes affect early cognitive control mechanisms in opposing ways: depression appears to be characterized by hyper-engagement of conflict monitoring systems, whereas burnout reflects a tendency toward hypo-engagement or functional disengagement [4, 10]. With respect to underlying mechanisms, this dissociation may reflect differential modulation of medial frontal systems [17].

Most importantly, however, the present study extends beyond prior work focused on group-level evaluation [5] by demonstrating that the apparent divergence of N2 in depression and burnout is critically modulated by shared variance between the two syndromes. When depression and burnout were modelled simultaneously as covariates, the burnout-related N2 reduction disappeared, whereas the depression-related N2 increase remained robust and specific to incongruent trials. This finding provides evidence that the variance in N2 amplitude is uniquely attributable to depressive symptoms, while burnout-related effects observed at the group level are largely driven by shared variance with depression. Hence, the N2 does not index a burnout-specific impairment in conflict monitoring per

se, but rather reflects a depression-dominant modulation that may be masked or counteracted by co-occurring burnout-related processes.

The enhanced N2 in depression found here is consistent with a body of literature linking this component to heightened conflict sensitivity and enhanced recruitment of cognitive control mechanisms, potentially driven by increased internal monitoring, error sensitivity, or negative bias [9, 18, 19] and over-activation of the anterior cingulate cortex (ACC) and medial frontal networks [17,20]. Hence, even at subclinical levels, depression is associated with a hyper-reactive conflict monitoring system, selectively engaged under conditions of response competition. Yet, participants with higher depressive symptoms committed more errors and, after controlling for burnout, showed a tendency toward slower responses. Thus, increased neural recruitment during conflict monitoring was accompanied by less efficient behavioural performance, suggesting that enhanced engagement of medial frontal control systems does not translate into improved executive functioning.

Conclusion. In summary, current findings suggest that altered conflict monitoring in subclinical populations is more strongly linked to depressive symptomatology than to burnout itself. This observation supports models proposing partially distinct neurocognitive mechanisms despite substantial phenomenological overlap between the two syndromes.

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