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Neurophysiology of executive cognitive functions under depression

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Abstract

Depression is associated with executive cognitive deficits which are not well explored and treated. Such deficits have a significant impact on remission and recurrence. To understand the neurocognitive mechanisms of executive processes, a literature search was conducted using bibliographic databases in neuroscience and cognitive sciences: PubMed, ScienceDirect, EBSCOhost, and PsyArxiv, combining search terms: "depression", "executive functions", and "specific brain event-related potentials". The theoretical review focuses on experiments using electrophysiological techniques, non-invasive tools with high temporal resolution. Depression shows alterations in brain activity linked with cognition: P3 diminished amplitudes and prolonged latencies, indicating executive attentional dysfunction; similar activity characterizes mismatch negativity (MMN), reflecting difficulties for change detection, voluntary effort, and mental shifting. Besides, depression tends to increase N1 latencies related with discrimination, and amplitudes of loudness dependence of auditory evoked potentials (LDAEP), suggesting inhibitory control's deficits. Regarding feedback processing, the alterations of error-related negativity (ERN), correct response negativity (CRN), and error positivity (Pe), at anterior cingulate cortex (ACC), and frontal regions, are related with troubles for error awareness, cognitive control, and error monitoring in depression. Lastly, the ability to interpret coherently the information value of negative feedback (NF), and a propensity to commit perseverative and non-perseverative errors, need further investigation. Depressive individuals commit both errors on more occasions than controls, what seems to relate with fronto-striatal networks' alterations, producing visual attention deficits and difficulties for inhibiting incoming information. Results show a variety of brain and executive cognitive components that are impaired under depression, although further research may clarify controversies resulting from depression heterogeneity and methodology used.

Keywords

Depression, executive cognitive functions, event-related potentials (ERPs), cognition, neurophysiology, neuroscience, cognitive neurosciences, affective neurosciences

Introduction

Depression is characterized by behavioural, emotional, affective, social, and cognitive changes. Frequently, persons suffering from depression report a loss of motivation, sadness, pessimism, hopelessness, psychomotor retardation, issues with concentration, etc.

From a cognitive perspective, different mental functions seem to be compromised under depression. Among them: memory, attention, executive functions (EFs), learning, processing speed, language, and psychomotor functions [1–3]. Furthermore, structural and functional alterations in the brain could be linked with cognitive deficits under depression [4, 5].

Previous researchers have reported that cognitive deficits are not frequently explored in clinical settings, and they could continue after remission, affecting people's performance in their works, academic tasks, or daily life activities, and having a negative impact on societies' economy [6–12].

Besides, cognitive deficits could increase the likelihood of suffering another depressive episode [13–16]. Depression is a major risk factor for suicide [17]. About eight hundred thousand (800,000) people die as consequence of suicide every year in the world, and it is identified as the second cause of mortality in adults between 15 and 29 years old [18]. Furthermore, during the last years, suicide is also being accelerated [19].

In a previous work, we described a randomized experiment to explore the benefits of an exergame-based intervention on EFs under depression [20]. EFs and executive control processes, are cognitive functions that play a fundamental role in the etiology and maintenance of depression [6, 21–23].

EFs are related to the ability of organizing information, planning, reasoning, decision-making and problem-solving. These functions are essential for the daily life activities, academic, and job performance. When EFs are affected, people frequently struggle to adapt to changes, shifting mindsets, inhibiting and updating information, or searching for appropriate strategies to deal with a variety of troubles.

People suffering from depression show a wide range of impairments in EFs [24, 25]. These deficits appear to be related to other psychological alterations, and the maintenance or recurrence of depressive manifestations [26–30].

From a neuroscientific approach, evoked potential studies using electroencephalographic techniques try to explore the neural substrate of these alterations and the relationship between the cognitive deficits and the neural correlates. Evoked potential studies allow researchers to assess brain response associated to stimulus conditions in a non-invasive and cost-effective way, at a high temporal resolution (time scale of milliseconds) [31–33]. Event-related potential (ERP) technique consists in registering the brain electrical activity associated to a sensorial, perceptive, cognitive or motor task using electrodes placed on the scalp, amplifying the signal, and analysing the changes in voltage, what helps to understand individual neuro-cognitive processes by associating neuro-electrical potentials with specific events.

Using ERP techniques, a variety of brain components have been associated to depression regarding EFs. Among them, we can cite the P3 components, the N1 component, the P2 component, the mismatch negativity (MMN), the error-related negativity (ERN) or the negative feedback (NF). We will briefly describe the most relevant findings to this respect.

When a person executes a psychological experimental task or a neuropsychological test, components and subcomponents of different mental domains and subdomains activate simultaneously or sequentially, perceiving the data, organizing the information, integrating it, and providing an adequate response. Thus, components from different cognitive systems constantly interact when performing a task (perceptive systems, memory systems, attentional systems, executive systems, and so forth).

Nonetheless, experimental tasks are specially designed to assess specific functions. They do not allow to study only one mental domain, but they could be designed to increase the demand for given cognitive domains, allowing the analysis and exploration of those concrete psychological functions.

As mentioned before, EFs are related to the ability of organizing information, planning, reasoning, problem-solving, and decision-making. So, they are considered complex cognitive functions that include diverse cognitive components: cognitive control, inhibition, mental flexibility, task-switching, working memory, executive attention, or information updating.

EFs' deficits in depression have been registered in experimental and clinical settings, showing an impairment in different cognitive and brain components, that affects recovery after depression. Nonetheless, the studies about brain activity of EFs under depression show a diversity of results that needs to be clarified [34–37].

In the current work, we try to organize and review the neural information regarding the executive deficits associated to depression, increasing our understanding about these processes and helping clinical and scientific communities to develop more appropriate interventions.

Methods

A keyword search was performed using PubMed (https://pubmed.ncbi.nlm.nih.gov/), ScienceDirect (https://www.sciencedirect.com/), EBSCOhost (https://www.ebsco.com/), and PsyArxiv (https://osf.io/preprints/psyarxiv), combining search terms: "depression" AND "executive functions" AND "event-related potentials" OR specific brain components such as: "P3" OR "P2" OR "N1" OR "MMN" OR "Pe" OR "Error related negativity" OR "Error processing" OR "Feedback processing". And it was applied to article topics, titles, abstracts and keywords. Individual papers were then examined for relevance and for additional references not revealed by previous searches.

Results

Brain components associated with EFs under depression

P300/P3

Starting by the P300 brain component, a number of studies show decrease P300 amplitudes and prolonged latency under depression [38–40]. This brain component consists in a positive deflection appearing in an interval of time between 200 ms and 500 ms after the appearance of a stimulus. It has been extensively studied using experimental paradigms where unexpected stimuli are being presented among series of repetitive standard stimuli, such as the classical oddball paradigm. P300 occurs with different types of stimulus' modalities: visual, auditory, haptic, etc. And its amplitude and latency seem to be related with the analysis of a stimulus, information processing, categorization, working memory, and decision making.

The distribution of P3 is frontal, central, and parietal, and it can be divided into two subcomponents named P3a and P3b. The first one has a fronto-central scalp distribution, it happens earlier than the P3b subcomponent and is associated with a bottom-up capture of attention or orientation to an unexpected stimulus presentation. On the other hand, the P3b subcomponent has a centro-parietal scalp distribution, it is registered 300–500 ms after the target stimulus and reflects a top-down processing linked with allocation of attentional resources, working memory operations, and decision-making processes.

Most of the studies that explore P300 under depression, analyse P3b through an auditory oddball paradigm, registering attenuated auditory P3b amplitude and prolonged auditory P3b latency, what could reflect broad cognitive impairment to process target stimuli, a reduction of the ability to allocate attentional resources and updating information, and longer periods of time to allocate the sufficient amount of attentional resources to perform a task, affecting working memory and the decision making process. Besides, it can be directly associated with a tendency of persons suffering depression to be focus inward, in place of concentrate on external stimulation.

However, several studies show contradictory results in terms of amplitude and latency of P3 in depression, and the results are explained due to clinical comorbidities, severity of depression, experimental design, age, sex and so forth [31].

In relation with EFs, P300 could also be assessed using tasks with a higher executive demand than the required to perform the oddball paradigm, such as the Wisconsin Card Sorting Test (WCST), the Stroop Test, the Trail-Making Test, or the Go/No-Go Test. Behaviourally, frequent deficits linked to depression, consist in a minor number of categories completed, more errors, and slower processing speed; what could be related with the differences in amplitude and latency observed in P3 under depression, but further investigation is required [41].

Although it was not possible to find studies on depression, P3 and executive cognitive components, some experiments have explored the P3 brain components using the WCST, and they registered an association between P3 and error processing during decision making tasks, and also observed that the updating of attentional set, or set shifting, could be understood as a modulatory source of the P3b response [42, 43].

However, further research is required to understand better the role of the executive domains and P3 activity, and how it could be related with depression.

N100/N1, P200/P2, loudness dependence of auditory evoked potentials (LDAEP)

Different from P3, the N100 or N1 brain component, is not modality-independent. Thus, we can observe several differences in auditory N1 component versus the visual N1 component.

The visual N1 has diverse subcomponents. Earliest subcomponent peaks 100–150 ms after the appearance of stimulus and it is registered at anterior electrodes. Otherwise, the others N1 subcomponents peak later, around 150–200 ms post stimulus at posterior sites: parietal cortex and occipital cortex, and they are associated with attention and discriminative processing.

Concerning the auditory N1, it has also subcomponents. Among them, we can find a frontocentral component peaking around 75 ms, a central subcomponent peaking around 100 ms, and a lateral component peaking at 150 ms. These subcomponents seem to be related with attention too.

Regarding depression, studies frequently focus on auditory N1 component assessed through the oddball paradigm. The main reason to concentrate in auditory processing appears to be that primary auditory cortex receives many projections from neurons using a neurotransmitter closely linked to depression: serotonin [44, 45].

Neurotransmitters modulate ERPs, and ERPs' studies show higher latency and lower amplitude in N1 under depressive conditions [46]. In addition, there are studies which identify a relationship between N1 and P3 components in depression. Increase in N1 latencies is associated with a decrease in P3 amplitude, what could affect discrimination and information processing. And alteration in attention and perception is likely to affect later cognitive processes related to updating of new information in working memory [46–48].

Besides, a specific feature of auditory processing is being studied in the last years. It refers to the relationship between the N1 and P2 components. P2 component is associated with pre-attentive auditory responses. The change in amplitude of the auditory N1/P2 component (difference between N1 and P2) in response to diverse stimulus intensities, also named LDAEP (loudness dependence of auditory evoked potentials), seems to be and indirect indicator of central nervous system serotonin function [44].

Nonetheless, results were inconsistent concerning LDAEP in depression. Studies comparing LDAEP between depressed persons and non-depressed controls show an increase LDAEP in depressed in some cases, a decrease in other cases, or no differences between groups. Reasons could be related with heterogeneity of depression, experimental designs, or the complexity of the physiology of depression. Although serotoninergic dysfunction in the central nervous system is considered to be one of the major organic pathophysiological factors of depression, further neurotransmitters are implicated in depression, such as dopamine, glutamate or noradrenaline among others.

Additionally, we can find a variety of procedures for measuring LDAEP that have been used in the experiments. Often, LDAEP is defined as an amplitude change in the auditory N1/P2 component in response

to a diversity of stimulus intensities, as we have previously mentioned. But LDAEP could also be measured as amplitude change in N1, P1, P2 and P1/N1 component in response to stimulus intensities [31].

From a cognitive-behavioural perspective, N1 and P2 are not only associated with pre-attentive and discriminative functions, but also with EFs. Poor performance of No-go in Go/No-go paradigms (related to EFs, specifically with inhibitory control), could be associated with low serotonin function. As mentioned before, LDAEP could work as an indirect indicator of serotonin levels, higher LDAEP is linked to low serotonin. In fact, impulsive persons show stronger LDAEP, difficulties controlling the inhibitory response for No-go trials, and higher emotional sensitivity associated with depressive states [49, 50].

But further research is needed to clarify the variety of results in LDAEP in relation with depression, and whether inhibitory control is directly related with N1/P2 or it is the result of the effect of attentional deficits on other components.

Mismatch negativity (MMN)

Continuing with the auditory response, it is relevant to mention the MMN. This brain component consists in a negative wave that use to peak between 150–250 ms, at central and fronto-central locus, after the presence of an occasional, deviant, or mismatching stimulus, appearing in a succession of identical stimuli (e.g., a sequence of 800-Hz tones and occasionally 1,300-Hz tones).

The MMN is considered an automatic brain response elicited even when the person is not involved actively in the auditory task, but listening the sounds while performing another activity, such as reading. In addition, MMN can also be registered under other sensory modalities (i.e., somatosensory, olfactory, visual modality or even in audio-visual tasks) [51–53].

Although some studies have claimed that the MMN is not different from the N1 wave, and they consider the MMN response a product of a posterior subcomponent of N1 peaking around 85 ms ("N1p") and an anterior subcomponent of N1, peaking about 150 ms after stimulus ("N1a") [54], extensive research seems to demonstrate that MMN must be considered a differentiate component from N1 due to several facts.

First, the MMN latency and duration do not match the latency and duration of N1a; secondly, the MMN response could be elicited by deviants that do not produce N1 response; third, when an experiment presents a repeated sequence of tones increasing or decreasing in frequency (standards), a different MMN response could be registered, even if the settings do not allow the adaptation of N1-generating neurons that appears with deviants occurring in a sequence of similar-frequency repetitive stimuli.

Then, the MMN scalp distribution and generator locus are different from those of N1a. Auditory-cortical N1 subcomponents show larger amplitude on the hemisphere contra-lateral to the stimulated ear, while the MMN changes due to frequency, duration, and intensity of stimuli, are right-hemispheric predominant. Moreover, the MMN has a second main generator in the frontal lobes, with higher amplitudes at these sites, that cannot be observed in N1a topography.

Lately, an important difference between MMN and N1 components, concerns the effects on N1a after experimental changes, which do not mimic the effects on the MMN response. It has been explored analysing diverse effects of pharmacological interventions on N1 and MMN, or how certain lesions may eliminate the MMN but do not affect the N1 activity [55].

Regarding the MMN functions, this automatic brain response is considered a pre-attentive index of deviance detection occurring not only when the person is concentrated in the task, but also when subjects perform another activity that is not related with the auditory stimulus or another passive task [56].

It has been stated that brain may elicit MMN because it can recognize a distinct stimulation. Thus, information has been previously stored in sensory memory, and a deviant stimulus is being compared with the other sounds. Then, the brain, cognitive, and perceptive systems, can detect the discrepancy between the neural representation of the regular stimuli and the representation of the given mismatch stimulus, producing the MMN [57]. Thus, MMN could be considered an index of change detection occurring unintentionally and automatically, and also, an index associated with auditory sensory memory.

However, the auditory memory cannot be understood as a unique process, but rather as a domain including several processes. In regard to MMN, at least four steps can be identified: (1) analysis, categorization and classification of incoming input, (2) integration of information into a logical model, (3) predictions about future stimulations based on the built model, and (4) comparison of representation, between previous organized and stored information, with new information [58].

Additionally, some authors assert that the MMN could be described as a stage of the distraction effect or the involuntary capture of attention, where the sound that violates expectations trigger initially a MMN, then, in a second stage, P3 reflects the orientation of attention toward the unexpected stimulus, and finally, the reorienting negativity (RON) response appears, a negative wave occurring 500 ms after stimulus, linked to the turning attention back to the primary task [59, 60].

Nonetheless, some studies have reported contradictory results, showing that N1/MMN, P3a and RON do not form a coupled chain reflecting the three stages of auditory distraction. Indeed, P3 could appear without eliciting MMN and consequently RON response [61].

About EFs, MMN appears to be not only related with an automatic pre-attentive response or an involuntary capture of attention by deviant auditory stimuli, but also with a voluntary effort related to attention switching or mental shift.

Previous experiments have reported that the MMN originates from auditory cortex on the supratemporal region and have at least two sources. One in the right lateral temporal cortex and another in the right frontal cortex [62]. The prefrontal cortex has a relevant role in controlling the direction of attention, so it could represent the initiation of the involuntary orienting of attention to a previous detected changed by the auditory-cortex MMN. Thus, the prefrontal activation of MMN, occurring later than the temporal source of MMN, could be understood as a process of attention switching [63].

However, Toyomaki et al. [64] found a correlation between low MMN amplitude and poor EFs performance in schizophrenia patients, using non-auditory tasks, specifically using the WCST, the Trail Making Test, and the Stroop Test to assess cognitive control, cognitive witching, and response inhibition. And they concluded that the frontal component of the MMN may be associated with conscious perception of attention switch to stimulus change.

Future research needs to clarify whether the frontal source of MMN is related with a voluntary or an involuntary process, associated exclusively with attention or also with executive domains and other networks.

Respecting depression, a recent meta-analysis including 13 studies out of 438, based on auditory paradigms, has shown that amplitude and latency of MMN regarding duration but not frequency deviants, were significantly impaired under depression in comparison with healthy controls, registering lower MMN amplitudes and prolonged MMN latencies [65].

Further projects have tried to analyse how a variety of deviant types might elicit a different MMN waves under depression, registering an increase on MMN amplitudes following tones that deviated in intensity and location [66].

Conclusively, although many studies report a decrease of MMN amplitudes and longer latencies under depression, results are inconsistent due to the variety of stimuli features, methodological issues and the severity of depression [31], and more research is required to evaluate the relationship among depression, MMN and EFs.

Feedback processing, error processing

Lately, it is important to highlight the brain activity associated with feedback processing and error processing. Once the person has provided a response, the evaluation of the feedback (correct or incorrect answer), and the ability to update the information, conflict monitoring, error processing, and the decision about continuing a response criterion or change it, are essential EFs that allow persons to adapt constantly to the environmental demands, and perform efficiently daily life activities.

ERN or error negativity (EN) consists in a negative deflection peaking around 100 ms of error commission at frontal and central electrodes, during simple decision tasks [67, 68].

Although an association between depression and ERN has been demonstrated [69, 70], it is not clear the direction of it. Some experiments have obtained enhanced ERN amplitudes in people with depression, while other show smaller ERN amplitudes under depression [71, 72].

Traditionally, depression has been characterized by excessive sensitivity to negative information. There is a tendency to amplify mistakes and consequences, a bias towards negative experiences, and a higher sensitivity to NF.

Authors who find an enhanced amplitude of the ERN component, explain it due to the hypersensitivity to errors that depressed persons show in early stages of error detection and error processing. Depression triggers an exaggerated early error response that require higher neural, cognitive and perceptive resources, generating subsequently difficulties to adapt to new situations or stimuli.

This idea is supported by neuro-structural and functional data which recognize that the anterior cingulate cortex (ACC) is likely the generator of ERN. This brain structure shows significant increase of metabolic activity under depression, coupled with a decreased activity in prefrontal regions, what could be related with the alterations observed in EFs [71, 73, 74].

However, some studies do not replicate these results. They find smaller (less negative) ERN amplitudes using different types of tasks, accounting for errors of choice (pressing an incorrect response button in an Erikson Task), and errors of commission (pressing a button when one is not supposed to, in a Go/No-go Task), that could be linked to difficulties in the evaluation of NF under depression.

In these cases, the results are in line with deficits in prefrontal functions and executive components of goal-directed behaviour, strategic reasoning, and cognitive control under depression. But functional and neuroimaging studies show decrease activation not just at prefrontal cortex but also at ACC, what contradicts previous results [75, 76].

Thus, the data regarding ERN are contradictory, and it is necessary to perform replication studies to understand the differential amplitudes of this brain component, and the neural networks implicated in error processing under depression. In addition, it is required to clarify whether depression severity and heterogeneity, together with comorbidity, could explain partly the differential results.

Moreover, wrong responses or errors are associated not only with ERN but also with a positive deflection peaking around 200 ms after the erroneous answer at posterior midline scalp distribution. This positive wave is named error positivity (Pe). Pe is independent of ERN and is linked with a later aspect of error processing. It has been related with conscious error awareness, orienting response to errors, cognitive control, and an emotional assessment process or motivational significance of errors, where mistakes are evaluated depending on the significance for the individual [67, 77, 78].

Further investigation needs to explain the relationship between Pe and depression, but several studies have found smaller Pe amplitudes under depression, what could be explained because depression could lead to a difficulty to be aware of errors and differentiate wrong and correct responses. A second explanation related to the emotional role of Pe is that a lower Pe in depressive persons is due to indifference to commit mistakes [72], what could be connected with anhedonia and apathy, indirectly associated with EFs [79, 80].

However, it is necessary to clarify why some experiments show a decrease in Pe under depression, coupled in some cases with an enhancement of ERN, and in other situations, with a reduction of ERN, and to understand its functional and clinical significance.

Pe has also been coupled with P300. Findings show reduced Pe and covariation of P3 amplitude under depression, but they seem to reflect distinct responses or manifestations of the same impairment in depression: reduced awareness and attentional allocation to stimuli [81, 82].

Similar to ERN, it is possible to identify a negative wave related to correct responses, the correct response negativity (CRN), peaking around 50 ms after the positive feedback at fronto-central regions, indicating a basic response monitoring process.

Regarding depression, results about CRN are also mixed, showing studies with a CRN amplitude reduction, experiments where CRN amplitudes are more negative in depression, or cases with similar CRN activity in experimental and control groups.

Besides, some studies report increased CRN under severe depression, what could be explained by an increased error monitoring on correct choices due to a tendency of depressed individuals to be uncertain about the veracity of their correct answers or the expectancy for committing more errors [83].

Nonetheless, these results need to be more investigated, because some analysis demonstrated that CRN could be considered an ERN, and the ERN may not be just understood in terms of error detection but from a broad perspective [84].

Continuing with the feedback processing, and regarding the WCST, this task requires subjects to stablish, maintain or shift sorting rules depending on the received feedback. These abilities are related with the capacity of individuals for adapting to changing environments, what is named set-shifting, mind-switching or cognitive flexibility, key components of EFs.

According to previous research, we can distinguish two moments of feedback in the WCST. The first one refers to NF coming after a sequence of positive feedback, what means that the subject must change the classification criteria. The second moment of feedback comes after the switching rule has been applied. Here, the participant could receive second-learning NF, so the new criteria is not correct, and it is required a second change; or the person can get positive feedback, indicating that the new sorting rule is right.

It is recognized that feedback has two components. The feedback could indicate whether the answer is correct or incorrect. This component is the valence of the feedback. Additionally, we can learn something else from the feedback beyond the valence, we can infer information about the rules and its implications. This component is named the informative value of feedback.

Thus, in the classic WCST we differentiate two moments: the first NF and possibly a second-learning NF; and two feedback components: the valence (correct or wrong response), and the informative value (information and implications of the rules and criteria).

Once a person gets feedback with a negative valence (incorrect answer), the person stays in the first moment or stage of NF. The person knows that it is not possible to continue with the same sorting criteria, and it should be changed according to the rules in order to get positive feedback. All these processes are related with the informative value of the NF, and this first moment could be identified as a rule-switch stage.

Then, the person tries another criterion and gets feedback (positive or negative) that is going to help to learn the new classification rule. This second moment is also called rule-learning stage. In both stages is possible to obtain NF and they are frequently denominated Switch-NF and Learn-NF.

Concerning neural activity, several studies have found diverse brain activity associated with both moments and components. Data shows that P3 is linked to the information value of feedback, and it increases in amplitude when it is necessary to extract more information from the feedback, what could be related with information updating [85, 86].

However, no information was found about the ERN and CRN for the different valence and moments of feedback in general, and specifically for the NF. And no information was found about Switch-NF and Learn-NF under depression either.

Finally, some studies using WCST, have analysed the brain components associated with the two types of errors that could be made in this task: perseverative errors and non-perseverative errors. Perseverative errors refer to the perseverance or persistence to respond according to an incorrect criterion or stimulus characteristics.

On the other hand, non-perseverative errors refer: firstly to errors that happen immediately after the sorting rule changes and indicate that it is necessary to adjust the response to a new matching criterion, and secondly, non-perseverative errors refer to failures to maintain mind-setting responding to the same sorting rule, due to the inability to maintain the focus or to inhibit the distracting interference of the other stimuli presented.

Depression characterizes by executive deficits assessed with the WCST, in terms of perseverative errors and non-perseverative errors, what could lead to poorer clinical outputs [87, 88].

If a person tends to commit perseverative errors, it likely means that experience difficulties during both moments of NF: the first NF and the second learning NF (Switch-NF and Learn-NF), and difficulties to understand the informative value of the NF.

The neural bases of this phenomenon need to be further explored, but some experiments have analysed the underlying mechanisms of perseverative and non-perseverative errors under healthy conditions, and they found distinct patterns of brain activation evoked by both types of errors.

Perseverative errors evoked larger P3b than non-perseverative errors or distractions, mainly at Pz location, what could suggest different brain processing in later stages at posterior regions as consequence of different early frontal activity. Moreover, at fronto-central regions, P2 showed higher amplitudes on distractions than on perseverative errors, and the parieto-occipital N1 was absent at perseverative errors.

It may reflect that perseverative errors are the result of alteration of fronto-extriatal network, because the absence of extrastriate N1 precedes the reduction of P2 at frontal areas, indicating troubles in visual attention to stimuli; whereas, distractions or non-perseverative errors seems to be more related with an inability to inhibit interfering information [42]. Nonetheless, further research should be conducted, specially, to understand the error and feedback processing under depression.

Discussion

Conclusively, executive cognitive functioning, associated with a negative impact on remission and recurrence of depression, could be analysed from a neuroscientific perspective to understand the relationship among cognitive, perceptive, affective systems and brain networks, and implement the nowadays therapeutic approaches.

The study concentrates on ERPs techniques, that are non-invasive and easy access tools that allow to get neurophysiological information at high temporal resolution.

After extensive reviewing the neuronal basis of executive cognitive functioning under depression, several conclusions are extracted from electrophysiological studies.

Most of the information concerning depression and ERPs refer to a few brain components: P3, including P3a and P3b; N1 and the relation with P2 or the LDAEP response; MMN, and the components of error and feedback processing: the ERN response, the Pe, the CRN, the Switch-NF and the Learn-NF.

Starting by P3, studies mainly focused on P3b, and they reveal that depression may cause lower amplitudes and longer latencies, what is not only associated with attentional difficulties but also with working memory and decision-making process, altering information updating and mind setting.

However, further research must differentiate between P3a and P3b when considering depression and clarify inconsistencies about its amplitude and latency and the potential effects of comorbidities, experimental design or demographic variables among others.

In second place, N1 has been studied in association with P3 and P2. Under depression, decreased P3 amplitude is linked with increased N1 latency, what could affect discrimination, information processing, and later cognitive activity.

About the relationship between N1 and P2, first it is important to mention that P2 is connected with pre-attentional processing. When considering the association N1 and P2, it refers to the difference in

amplitude between N1 and P2 in response to a variety of stimuli intensity, what is called "loudness dependence of auditory evoked potentials" or LDAEP.

People suffering from depression show lower amplitude and longer latency in N1 component, but results concerning LDAEP are contradictory. In some cases, depressed register an increase in LDAEP, in other cases, a decrease has been observed, and finally there are studies which do not show differences between depressed and non-depressed groups.

LDAEP could be considered and indirect indicator of central nervous system serotonin function, a major pathophysiological factor of depression, and it is not just associated with attentional disruption but also with EFs, specifically with inhibitory control. In cases where a high LDAEP has been registered under depression, low serotonin appears, and people show a tendency to impulsivity, difficulties to control inhibitory response, and an enhancement of sensitivity concurrent to depression states.

Third, MMN, a negative wave peaking between 150–250 ms at central and frontocentral electrodes, after the appearance of a deviant stimulus presented in a sequence of similar stimuli, is considered an index of change detection occurring unintentionally and automatically. Furthermore, it is interpreted as an index of auditory sensory memory, as well as a component of voluntary effort related to attention switching or mental shift.

Depression elicits a decrease in MMN amplitudes and prolonged latencies, what could be linked to deficits in voluntary and involuntary capture of attention and mental flexibility, but more studies should clarify discrepancy in MMN activity, accounting for depression severity and methodological issues.

Concerning the feedback processing and error processing, ERN/EN, Pe, CRN, Switch-NF and Learn-NF have been revised.

Starting with ERN, contradictory results have been found about the effect of depression. Some studies register enhanced ERN amplitudes under depression while others show smaller ERN amplitudes.

In the first case, the phenomenon observed is associated with hypersensitivity to errors in depression, what causes an exaggerated error response that requires higher neural and cognitive resources. This explanation seems to be supported by the fact that the ACC is believed to be the generator of ERN, and a significant metabolic activity is registered at ACC on depression, coupled with a decreased activity of prefrontal regions, what may explain executive deficits.

On the other hand, experiments that register lower ERN amplitudes explain that it might be the cause of difficulties for evaluating NF and adjustment response. Indeed, some studies do not show an increment of ACC, but a decrease in activation, paired with diminished prefrontal activation. Thus, those controversies must be clarified taking into account the heterogeneity of depression and comorbidities.

Together with the ERN, normally a positive wave peaks around 200 ms after the erroneous response. Pe is linked to later error processing, conscious error awareness, cognitive control and emotional significance of errors. In general, studies have found a reduction of Pe amplitudes under depression related with difficulties to be aware of errors, differentiate mistakes from correct answers, plus, indifference and apathy, indirectly related to EFs.

Similar to ERN, a negative wave appears after right choices at fronto-central regions, but results are also mixed concerning CRN and depression. Studies report increasing CRN amplitudes in some situations, decreasing amplitudes in other cases or no differences between depressive subjects and controls.

However, it seems that severe depression is associated with increased CRN amplitudes, what could be explained by increased error monitoring on correct choices due to a tendency of depressive individuals to believe that they are going to commit more errors.

Nonetheless, some authors emphasize the importance of foster research to understand whether CRN could be considered an extension of ERN, understanding EN not only in terms of error detection but from a broad perspective.

Continuing with error and feedback processing, some approaches distinguish two different moments of error processing related to tasks with a high executive demand, such as the WCST. In those cases, there is a first stage of NF where the person performing the task gets an incorrect answer and it is necessary a different response (Switch-NF), and later, a second moment of NF could appear implying that the person needs to accurately assess the information value of the feedback, adapting the choice according to a new sorting criteria (Learn-NF).

These two moments: Switch-NF, when obtaining the first feedback and Learn-NF, when getting the second NF, need to be more studied under depression, due to the connection of Learn-NF, information value, and enhanced P3 amplitudes, relevant for extracting and updating previous information.

Similarly, executive deficits measured with the WCST are linked with a growing number of perseverative and non-perseverative errors in depression.

Perseverative errors or the tendency to continue responding to a criterion that is no longer valid, may be connected to both moments of NF, Switch-NF and Learn-NF, and difficulties understanding the information value of the NF.

Although more investigation is needed, perseverative errors evoke larger P3b primarily at central posterior areas, lower P2 amplitudes at fronto-central locations, and there is an absence of parieto-occipital N1 on perseverative errors but not on non-perseverative errors or distractions. This fact indicates distinctive brain patterns connected with diverse types of errors.

Specifically, perseverative errors, augmented during depression, are associated with alterations of frontal-striatal networks, indicating troubles in visual attention to stimuli, whereas, non-perseverative errors, also increased in depression, are linked to lower P3b amplitudes at posterior areas, higher P2 amplitudes at frontal sites, and the presence of parieto-occipital N1, what seems to be related with an inability to inhibit interfering information, an important component of EFs.

Conclusion

In conclusion, this review provides important information to understand the connection between depression and neuro-executive functioning.

Summarizing (Table S1), P3b activity in depression shows lower amplitude and longer latency, implying attentional and executive deficits. Besides, P3b decreased amplitude is linked to increased N1 latency affecting discrimination and information processing.

In addition, longer N1 latency and lower amplitude in relation with P2 activity, is associated with attentional disruption and inhibitory control deficits in depression.

Moreover, depression produces decreased MMN amplitude and prolonged latency, compromising mental flexibility and the voluntary and involuntary capture of attention.

Regarding feedback processing, hypersensitivity to errors in depression is linked to an exaggerated ERN and a reduction of Pe amplitude, what is linked to difficulties for errors' awareness and executive dysfunction. Furthermore, the processing of right results in depression is associated with increased CRN amplitudes indicating an enhanced error monitoring.

Conclusively, although these findings bring to light neuro-cognitive mechanisms of depression, further research might clarify previous findings and inconsistencies resulting from heterogeneity of depression, demographic variables, and distinct methodological approaches, to implement the quality of investigations and the potential therapeutic benefits.

Abbreviations

ACC: anterior cingulate cortex

CRN: correct response negativity

EFs: executive functions

EN: error negativity

ERN: error-related negativity

ERP: event-related potential

LDAEP: loudness dependence of auditory evoked potentials

MMN: mismatch negativity

NF: negative feedback

Pe: error positivity

RON: reorienting negativity

WCST: Wisconsin Card Sorting Test

Supplementary materials

The supplementary material for this article is available at: https://www.explorationpub.com/uploads/Article/file/100696_sup_1.pdf.

Declarations

Author contributions

ERM: Conceptualization, Investigation, Methodology, Writing—original draft, Project administration, Visualization, Funding acquisition, Writing—review & editing.

Conflicts of interest

The author declares no conflicts of interest.

Ethical approval

Not applicable.

Consent to participate

Not applicable.

Consent to publication

Not applicable.

Availability of data and materials

The data that support the findings of this study are openly available in the [OPEN SCIENCE FRAMEWORK] [https://osf.io/RQ357/]. This work is available as Preprint at Open Science Framework [https://doi.org/10.31219/osf.io/f5dhq].

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