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Review

Auditory event-related potentials in separating patients with depressive disorders and non-depressed controls: A narrative review

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ABSTRACT

This narrative review brings together the findings regarding the differences in the auditory event-related potentials (ERPs) between patients with depressive disorder and non-depressed control subjects. These studies' results can inform us of the possible alterations in sensory-cognitive processing in depressive disorders and the potential of using these ERPs in clinical applications. Auditory P3, mismatch negativity (MMN) and loudness dependence of auditory evoked potentials (LDAEP) were the subjects of the investigation. A search in PubMed yielded 84 studies. The findings of the reviewed studies were not highly consistent, but some patterns could be identified. For auditory P3b, the common findings were attenuated amplitude and prolonged latency among depressed patients. Regarding auditory MMN, especially the amplitude of duration deviance MMN was commonly attenuated, and the amplitude of frequency deviance MMN was increased in depressed patients. In LDAEP studies, generally, no differences between depressed patients and non-depressed controls were reported, although some group differences concerning specific depression subtypes were found. This review posits that future research should investigate whether certain stimulus conditions are particularly efficient at separating depressed and non-depressed participant groups. Future studies should contrast responses in different subpopulations of depressed patients, as well as different clinical groups (e.g., depressive disorder and anxiety disorder patients), to investigate the specificity of the auditory ERP alterations for depressive disorders.

1. Introduction

Major depressive disorder (MDD) is one of the most common mental illnesses with an estimated 3.8 % of the world's population affected (Institute of Health Metrics and Evaluation, 2019). Furthermore, MDD is expected to be the leading cause of the burden of disease worldwide by 2030 (World Federation for Mental Health, 2012). In addition to the huge economic and social burden, MDD entails a lot of suffering and has a strong impact on the quality of life of depressed individuals. Therefore, efficient tools for comprehensive MDD diagnostics and treatment are needed.

MDD is a heterogeneous syndrome with a complex spectrum of possible symptoms that are often shared with other disorders (Bilello, 2016; Fried and Nesse, 2015), all of which also complicate comprehensive diagnostics. Unfortunately, there are currently no objective methods for diagnosing/characterising MDD, which may delay the start of treatment and negatively affect optimal treatment outcomes (Gaynes et al., 2009). In clinical practice, the diagnosis of MDD is currently based on structured clinical interviews to determine the presence of symptoms (typically, the Structured Clinical Interview for DSM; First et al., 2015). Hence, similar to the diagnoses of other psychiatric disorders, the diagnosis of MDD is determined by descriptive diagnostic criteria based on the symptoms reported by the patient. More objective ways to diagnose depression based on biomarkers have been the subject of research. According to Atkinson et al. (2001), a biomarker is a characteristic that can be objectively measured and evaluated as an indicator of normal or pathogenic biological processes. Biomarkers can be used, for instance, as diagnostic tools for identifying individuals with a disease (Atkinson, 2001). Important performance characteristics of biomarkers include sensitivity (the fraction of people with a disease who test positive) and specificity (the fraction of people without the disease who test negative, e.g., FDA-NIH Biomarker Working Group, 2016). It would be

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important to identify biomarkers that could provide more objective assessment techniques for MDD diagnostics. Aside from clinical interviews, biomarkers could be used as tools to differentiate between patients with MDD and non-depressed subjects, as well as to define more homogenous subtypes of MDD and to develop more personalised approaches for treatment in the future.

Previous studies indicated that markers reflecting the activity of the inflammatory, neurotransmitter, neurotrophic, neuroendocrine and metabolic systems, as well as genetic and epigenetic features might be potential candidates for MDD biomarkers, but the findings are still quite inconsistent (for reviews, see e.g., Hacimusalar and Eşel, 2018; Strawbridge et al., 2017). In biomarker research, event-related potentials (ERPs) derived from electroencephalography (EEG) in the context of MDD have also been investigated. This narrative review summarises the findings on auditory ERPs reflecting sensory-cognitive processing, which have been widely investigated in patients with MDD and which might have the potential to be developed as biomarkers for MDD. This review addresses the first phase of the research on possible future biomarkers focusing on sensitivity by investigating whether auditory ERPs could be used to differentiate between depressed and non-depressed participant groups. Furthermore, this review also discusses whether alterations in auditory ERP components are specific to some subtypes of depressive disorder, whether they are related to the severity of depression and whether there are some confounding factors (e.g., comorbid disorders and antidepressant medication) that might have an impact on the results on ERPs. This narrative review does not provide a quantitative synthesis of the findings; instead, it presents an overall picture of current knowledge comprised of four decades of research on alterations in auditory ERPs in patients with depressive disorder, providing a foundation for future research.

This review focuses on ERPs from the auditory modality. There is abundant research on auditory sensory-cognitive ERPs in the context of MDD. Nonetheless, research on these auditory ERPs for MDD has not yet been comprehensively reviewed, but these ERPs have biomarker potential for other psychiatric disorders. For instance, auditory sensory-cognitive ERPs can differentiate between schizophrenia patients and control subjects (for meta-analyses for MMN, see Erickson et al., 2016; Umbricht and Krljes, 2005; for P3, see Bramon et al., 2004; Jeon and Polich, 2003). Furthermore, pre-attentive auditory responses (e.g., N1, P2, mismatch negativity [MMN] and P3a) can be examined in the absence of participants' attention. Thus, they may be beneficial for studies investigating clinical patient groups, including MDD patients, because problems related to motivational and attentional factors in measurement can be avoided.

Visual ERPs in patients with MDD have also been investigated. These studies are beyond the scope of the present review that focuses only on auditory ERPs. MDD is characterised by deficits in emotional processing, reactivity and regulation (Bylsma, 2021). Visual ERPs elicited, for instance, by emotional pictures or in reward-related tasks can be utilised to investigate these emotion-related deficits in MDD patients. Studies on visual ERPs related to affective cognition in MDD patients have been widely reviewed, and the results indicate that ERP components reflecting emotional and motivational processing (late positive potential [LPP]) and reward processing (reward positivity [RewP]) are attenuated in MDD patients and persons who are at risk of depression (for reviews, see Hajcak and Foti, 2020; Kujawa and Burkhouse, 2017; Kujawa et al., 2020; Proudfit, 2015; Proudfit et al., 2015). Since MDD includes affective and motivational symptoms (American Psychiatric Association, 2013), these visual ERPs may also be potential future biomarkers for MDD. However, in the present narrative review, the focus on ERP research on MDD patients is different, addressing auditory ERPs reflecting basic sensory-cognitive processing. More specifically, whereas the aforementioned reviews on visual ERP components related to affective cognition provide insight into alterations in the emotional and motivational information processing in MDD patients, in the present review, the theoretical framework for the connection between auditory sensory-cognitive ERPs and MDD is mostly based on deficits in more basic cognitive functions in MDD patients (e.g., Rock et al., 2014).

ERPs are neural responses time locked to the onset of sensory stimuli or cognitive or motor functions, and they are derived from EEG measurement of neural activity by signal averaging (e.g., Luck, 2014). EEG provides a method for studying information streams in the human brain; therefore, it can also be utilised to assess pathological states, including those related to neuropsychiatric disorders (Maekawa et al., 2012). EEG may be a promising resource for the identification of MDD biomarkers because MDD is associated with changes in brain activity and neurocognitive processes (Mumtaz et al., 2015). EEG directly measures ongoing neural activity in the brain with a high temporal resolution (in a time scale of milliseconds, e.g., Sanei and Chambers, 2007). It is also a non-invasive and cost-effective resource for discovering MDD biomarkers. These advantages of the EEG method may facilitate possible future implementation in clinical practice.

There are many possible reasons for the assumption that ERPs reflecting sensory and cognitive functions can be considered potential tools for differentiating between patients with MDD and non-depressed control participants. Studies that utilised cognitive tests have shown that MDD patients exhibit deficits in attention, learning, memory, executive functions, etc. (Austin et al., 2001; Burt et al., 1995; Lee et al., 2012; Rock et al., 2014; Veiel, 1997). Information processing deficits underlying these overt cognitive dysfunctions detected with cognitive tests can be examined with a precise temporal resolution using ERPs. The high temporal resolution of ERPs allows for investigations into the serial stages of information processing at pre-attentive and attentive levels. Thus, ERP components can be used as tools to examine the differences in the specific information processing stages between MDD patients and non-depressed control participants, contributing to a better understanding of the neurophysiological underpinnings of the cognitive dysfunctions in MDD. Furthermore, ERPs are modulated by neurotransmitters, such as serotonin, dopamine, noradrenaline and glutamate (e.g., Hegerl and Juckel, 1993; Liu et al., 2009; Pogarell et al., 2011; Swick et al., 1994; Umbricht et al., 2000), that have been implicated in the pathophysiology of depression (e.g., Belujon and Grace, 2017; Malhi et al., 2005; Moret and Briley, 2011; Moriguchi et al., 2019).

In this narrative review, we bring together findings about whether there are deficits in patients with MDD in the early pre-attentive cognitive processing stage reflecting automatic change detection (MMN) and the later attentive cognitive processing stage reflecting attentional and working memory operations (P3). MMN has traditionally been studied by utilising an ignore oddball condition in which a rare deviant stimulus is interspersed with a repetitive standard stimulus (Näätänen et al., 1978; for reviews, see Näätänen et al., 2005, 2007). Several aspects suggest that MMN may be altered in MDD patients. MMN is associated with predictive coding theory, according to which the brain predicts future events based on previous sensory input (Friston, 2005). When the input does not match the prediction, a prediction error occurs, and the error signal is projected upward in the hierarchical neural network to update the predictive model (Friston, 2005). MMN has been suggested to reflect prediction errors (e.g., Chennu et al., 2013; Wacongne et al., 2011; for reviews, see Carbajal and Malmierca, 2018; Denham and Winkler, 2020). Predictive coding, as a fundamental information processing mechanism that enables adaptive behaviour, is theorised to be aberrant in psychiatric conditions, including depressive disorders (for reviews, see Kube et al., 2020; Smith et al., 2021). Moreover, at the neurochemical level, MMN is suggested to reflect the functioning of glutaminergic *N*-methyl-D-aspartate (NMDA) receptors (e.g., Javitt et al., 1996; Umbricht et al., 2000, 2002), which has been suggested to be dysfunctional in MDD patients (e.g., Adell, 2020; Inoshita et al., 2018; Sanacora et al., 2008).

P3 is a later-occurring component than MMN in the information processing chain (for a review, see Friedman et al., 2001; Kok, 2001). It is most commonly studied utilising an attend oddball task in which the

target stimuli are rarely presented among repetitive standard stimuli, and the subject is required to respond to the targets (Friedman et al., 2001; Polich and Criado, 2006; Polich, 2007). It is feasible that P3 reflecting attentional and working memory operations might differ between patients with MDD and non-depressed controls since studies investigating cognitive functions utilising cognitive tests have indicated that selective attention and working memory are especially impaired among MDD patients (e.g., Christopher and MacDonald, 2005; Landrø et al., 2001; for a meta-analysis, see Rock et al., 2014). Furthermore, dopamine and noradrenaline are neurotransmitters that contribute to the presence of depressive symptoms (e.g., Belujon and Grace, 2017; Malhi et al., 2005; Moret and Briley, 2011), and the dopaminergic and noradrenergic systems have been suggested to be associated with P3 response (for reviews, see Nieuwenhuis et al., 2005, 2011; Polich, 2007).

In this review, the findings regarding whether patients with MDD and non-depressed controls differ in terms of a sensory ERP component called the loudness dependence of auditory evoked potentials (LDAEP), also known as the intensity dependence of auditory evoked potentials, are summarised. LDAEP has primarily been defined as the change in the amplitude of the auditory N1/P2 component (a difference between N1 and P2) in response to different stimulus intensities, and it has been regarded as an indirect indicator of central serotonin function (Hegerl et al., 2001; Hegerl and Juckel, 1993). Serotonergic dysfunction in the central nervous system is suggested to be one of the major pathophysiological factors of depression (e.g., Coppen, 1967; Hasler, 2010; Kraus et al., 2017; Meltzer, 1990), which is why LDAEP in MDD patients is being investigated. However, it is obvious that in addition to serotonin, other neurotransmitters, especially other monoamines (noradrenaline and dopamine), play a role in the development of depressive disorders (for reviews, see Belujon and Grace, 2017; Malhi et al., 2005; Moret and Briley, 2011; Shao and Zhu, 2020), and various subtypes of depression may differ in terms of neurotransmitter function (Malhi et al., 2005). Thus, LDAEP might also have the potential to differentiate subgroups of depressive disorder.

The present review did not include studies in which treatment responses were predicted based on auditory ERPs, since such reviews already exist; there is growing evidence that LDAEP can act as a predictor of SSRI treatment response in MDD patients (for reviews, see Bruder et al., 2012; Leuchter et al., 2009; Mumtaz et al., 2015; Olbrich and Arns, 2013; Park, 2020; Wade and Iosifescu, 2016; for a meta-analysis, see Yoon et al., 2021). Some previous reviews have also discussed the effects of the illness stage on auditory ERPs, suggesting that, by now, neither auditory MMN nor auditory P3 has shown the potential to be a marker of a stage of a depressive disorder (Lavoie et al., 2019; McGorry et al., 2014). However, only a handful of reviews have responded to the question of whether auditory ERPs can discriminate between patients with MDD and non-depressed control subjects (e.g., Bruder et al., 2012; Mumtaz et al., 2015; Näätänen et al., 2012; Olbrich and Arns, 2013; for a meta-analysis on MMN, see Tseng et al., 2021), even though this is an essential question for the development of biomarkers for MDD diagnostics. In these previous reviews, the newest findings were naturally missing, and in some of them, the main focus was neither depressive disorders (Näätänen et al., 2012) nor auditory ERPs (Mumtaz et al., 2015; Olbrich and Arns, 2013). In the meta-analysis by Tseng et al. (2021), the main focus was on the findings of frequency deviance and duration deviance MMN responses in adults with MDD. This review extends this work by comprehensively reviewing the MMN literature, including also other deviant types. The aim of the present work is to summarise the available literature on P3, MMN and LDAEP components, focusing specifically on the comparison between MDD patients and non-depressed controls. Next, these ERP components are briefly introduced.

1.1. P3

The P3 ERP component (also known as P3b or P300) first reported by Sutton et al. (1965) is a commonly studied ERP component that is related to attention, decision-making and working memory updating during cognitive task performance (for reviews, see Kok, 2001; Polich, 2007). The latency of P3 is thought to be related to cognitive efficiency, reflecting the information processing speed (Polich, 2007). Hence, it provides a valuable tool for investigating these cognitive processes in the healthy and diseased human brain (Wronka et al., 2012). Therefore, P3 alterations in MDD patients have also been investigated.

P3 is traditionally elicited in the attend two-stimulus oddball condition, in which the target stimuli are rarely presented among repetitive standard stimuli (Friedman et al., 2001; Polich and Criado, 2006; Polich, 2007). Sometimes, a three-stimulus oddball condition is used in which there are infrequent distracter stimuli in addition to rare targets and repetitive standard stimuli (Fabiani and Friedman, 1995). In both oddball conditions, the subject is required to respond to the targets (e.g., by pressing a button).

Different experimental designs can be used to study the subcomponents of P3, including P3b, P3a and novelty P3 (Friedman et al., 2001; Polich and Criado, 2006; Polich, 2007). In an attend oddball condition, the target stimuli elicit a P3b response that peaks around 300–500 ms after the onset of the task-relevant target stimulus and that has a centro-parietal scalp distribution (Polich, 2007). The P3b elicited by the target stimuli, to which the subject is required to respond, reflects the allocation of attentional resources, stimulus classification and working memory operations, as well as decision-making (Kok, 2001). While P3b reflects top-down controlled processing (e.g., Debener et al., 2002g; Wronka et al., 2012), P3a and novelty P3 reflect the bottom-up capture of attention and orienting (e.g., Escera et al., 2000; Friedman et al., 2001; Polich, 2007). P3a is elicited by deviant stimuli in an ignore oddball condition where participants are not attending to the stimuli. A novelty P3 is elicited by novel infrequent distracter stimuli in the three-stimulus oddball condition. These stimuli include the standard stimuli, rare target stimuli and distractor stimuli. Both P3a and novelty P3 peak earlier than P3b, around 250–300 ms after the stimulus onset, and they have a fronto-central scalp distribution (Knight and Scabini, 1998). Most research on P3 activity in MDD patients has focused on the P3b subcomponent.

P3 components have been suggested to reflect the neuromodulatory effects of the noradrenergic system (Liu et al., 2009; Swick et al., 1994; for reviews, see Nieuwenhuis et al., 2005, 2011; Polich and Criado, 2006; Polich, 2007) and dopaminergic system (e.g., Liu et al., 2009; Mulert et al., 2006; Pogarell et al., 2011; for reviews, see Polich and Criado, 2006; Polich, 2007; for studies showing no such effect, see Oranje et al., 2006, 2009; Spronk et al., 2013). According to the theory connecting P3 and noradrenaline proposed by Nieuwenhuis et al. (2005), P3 components may reflect upstream activity originating from the locus coeruleus–noradrenaline system. Motivationally significant stimuli (either intrinsically motivational or task-related stimuli) increase locus coeruleus activation, leading to the release of noradrenaline in cortical projection areas. This activity is indexed by the P3 amplitude. Given the P3 response's association with noradrenergic and dopaminergic neurotransmitter systems relevant to depression (for reviews, see Belujon and Grace, 2017; Malhi et al., 2005; Moret and Briley, 2011) and motivational and attentional functions (for reviews, see Friedman et al., 2001; Kok, 2001; Polich, 2007), P3 provides a feasible target in the search for MDD biomarkers.

1.2. MMN

Pre-attentive information processing is an essential part of perception and cognition (Näätänen et al., 2010). Pre-attentive information processing can be studied utilising MMN, which reflects automatic auditory change detection (Kujala et al., 2007; Näätänen et al., 1978, 2007, 2011h). MMN can be used as an objective indicator of auditory discrimination accuracy and auditory sensory–memory duration, accuracy and capacity (Näätänen et al., 2011g, 2012). It is typically elicited

at a latency of 150–250 ms after the onset of deviance in an ignore oddball condition, in which a rare deviant stimulus is interspersed with a repetitive standard stimulus at fronto-central electrode sites in EEG recordings (Garrido et al., 2009; Näätänen et al., 1978, 2005, 2007). MMN can be produced in experimental designs that employ changes in several different stimulus properties, for example, in stimulus frequency, duration and intensity, as well as in feature combinations and abstract features (e.g., Näätänen et al., 1978, 1989; Tervaniemi et al., 1994; for reviews, see e.g., Näätänen et al., 2010, 2011h; Paavilainen, 2013; Winkler and Cowan, 2005).

MMN amplitude and latency reflect variations in cognitive abilities in healthy individuals (Bazana and Stelmack, 2002: the general factor of intelligence, *g*; Light et al., 2007: global functioning; Strömmer et al., 2017: working memory capacity and psychomotor speed), and MMN abnormality is associated with cognitive deficits (Näätänen et al., 2011g, 2012, 2014). At the neurochemical level, MMN is thought to reflect the functioning of NMDA receptors (e.g., Javitt et al., 1996; Umbricht et al., 2000; Umbricht et al., 2002), and therefore, MMN not only indexes the local neural mechanism of auditory discrimination but also cognitive performance more generally (Näätänen et al., 2012, 2014). There is an abundance of research on MMN regarding schizophrenia (for meta-analyses, see Erickson et al., 2016; Umbricht and Krljes, 2005). Impaired NMDA receptors among schizophrenia patients have been linked to reduced MMN amplitude (e.g., Lavoie et al., 2007; for reviews, see Näätänen et al., 2012, 2014). MMN alterations in MDD have been much less studied, even though the NMDA receptor system is also suggested to be dysfunctional in MDD patients (Adell, 2020; Inoshita et al., 2018; Sanacora et al., 2008).

1.3. LDAEP

Serotonergic dysfunction is postulated to be one of the main pathophysiological factors in depression (e.g., Coppen, 1967; Hasler, 2010; Kraus et al., 2017; Meltzer, 1990). High concentrations of cortical serotonin have been found in the primary auditory cortex in which serotonin behaves as a neuromodulator (Hegerl et al., 1998, 2001; Juckel et al., 1997), and auditory ERPs indirectly reflect the modulatory effects of serotonin on cortical functioning (Hegerl et al., 2001). The LDAEP is an auditory ERP component that can be used as a tool to obtain information on central serotonergic activity (Hegerl and Juckel, 1993; Hegerl et al., 2001). LDAEP is a measure that assesses the amplitude changes of the auditory N1/P2 component (a difference between N1 and P2) in response to varying loudness levels of auditory stimulation; a low LDAEP indicates high serotonergic activity, and vice versa (Hegerl and Juckel, 1993; Hegerl et al., 2001). LDAEP can be used in investigations of serotonergic dysregulation in patients with MDD (e.g., Hegerl et al., 1998). There is an abundance of research in which LDAEP has been measured to predict the treatment response of selective serotonin reuptake inhibitors (SSRIs) in MDD patients. The findings suggest that stronger baseline LDAEP values predict a favourable response to SSRIs (for reviews, see Leuchter et al., 2009; Wade and Iosifescu, 2016; for a meta-analysis, see Yoon et al., 2021). However, whether LDAEP can discriminate between MDD patients and non-depressed controls has been less studied.

2. Methods

A literature search until June 2021 was performed by entering the following keywords into the PubMed database: (‘depression’ or ‘depressive’ or ‘depressed’) and (‘P300’ or ‘P3’ or ‘P3a’ or ‘P3b’ or ‘novelty P3’ or ‘mismatch negativity’ or ‘MMN’ or ‘loudness dependence’ or ‘LDAEP’ or ‘intensity dependence’ or ‘IDAEP’). The following limits were applied: title/abstract. In addition, three studies were identified through reference lists.

Original experimental studies were included in the review if they contribute to the field of auditory ERP biomarker research concerning the discrimination between clinically diagnosed depressive disorder patients and non-depressed control participants. Studies that investigated visual and somatosensory and olfactory ERPs were excluded. The following diagnoses were included: major depressive disorder, major depression, depressive disorder, major depressive episode, depressive episode and dysthymia. Hereafter, all diagnoses are referred to as depressive disorder. Studies examining only bipolar disorder were excluded because we wanted to focus on unipolar depressive disorder, and including bipolar disorder might also cause more variability in findings. However, in five studies included, a small number of participants had been diagnosed as having a bipolar disorder (currently depressed), while a vast majority of the participants in these few studies were diagnosed as having a unipolar depressive disorder (Ancy et al., 1996; Bruder et al., 2009; Gallinat et al., 2000; Gordon et al., 1986; Tenke et al., 2010). A study that did not report the number of bipolar disorder participants was excluded (Linka et al., 2007). Only studies published in peer-reviewed journals and written in English were included. Regarding P3b, only the studies in which an oddball task had been applied were included.

3. Results

In total, 84 articles met the inclusion criteria. In the following chapters, all diagnoses are referred to as depressive disorder. The exact diagnoses are reported in Table 1, in which auditory P3, MMN and LDAEP studies that are discussed in the following sections are listed.

3.1. P3

Most ERP research on depressive disorder focused on P3b utilising a frequency deviance auditory oddball task with rare target stimuli and repetitive non-target stimuli. The participants were required to respond to the target stimuli. There are also a few studies on auditory P3a and novelty P3.

3.1.1. P3b

An attenuated auditory P3b amplitude among patients with depressive disorder was found in multiple studies (Ancy et al., 1996; Blackwood et al., 1987; Gangadhar et al., 1993; Iv et al., 2010; Jaworska et al., 2013; Karaaslan et al., 2003; Kawasaki et al., 2004; Kemp et al., 2009, 2010; Li et al., 2011; Muir et al., 1991; Murthy et al., 1997; Pfefferbaum et al., 1984; Röschke et al., 1996; Röschke and Wagner, 2003; Singh et al., 2000; Tenke et al., 2010; Urretavizcaya et al., 2003; van Dinteren et al., 2015; Wagner et al., 1997; Yanai et al., 1997; Zhou et al., 2019; difference only between suicidal depressed patients and controls: Hansenne et al., 1996). Additionally, an increased P3b amplitude in patients with depressive disorder was reported in one study (Li et al., 2014), and in one study, patients with comorbid depressive and anxiety disorders had an increased P3b amplitude compared to depressive disorder patients without anxiety disorder and control participants without depressive disorder and anxiety disorder (Bruder et al., 2002). However, several studies did not find any differences in P3b amplitude between depressed patients and controls (Barreiros et al., 2020; Bruder et al., 1998, 2002, 2009; Feldmann et al., 2018; Giedke et al., 1981; Greimel et al., 2015; Himani et al., 1999; Houston et al., 2004; Kalayam and Alexopoulos, 1999; Kaustio et al., 2002; Nan et al., 2018; Roth et al., 1981; Sara et al., 1994; Shim et al., 2019; Swanwick et al., 1996; Vandoolaeghe et al., 1998; Weir et al., 1998).

Regarding auditory P3b latency, there are many studies in which prolonged P3 latency was found in patients with depressive disorder (Himani et al., 1999; Kalayam et al., 1998; Kalayam and Alexopoulos, 1999; Karaaslan et al., 2003; Kemp et al., 2009, 2010; Kindermann et al., 2000; Nan et al., 2018; Singh et al., 2000; Tripathi et al., 2015; Urretavizcaya et al., 2003; Vandoolaeghe et al., 1998; Zhou et al., 2019). In addition, a shorter latency was reported in one study (Sumi et al., 2000). Numerous studies have found no differences in P3b latency between

Table 1
Auditory ERP studies comparing patients with depressive disorder and non-depressed control subjects in amplitude and/or latency of P3, MMN and LDAEP components. For P3 and MMN, the analysis has compared deviant and standard responses, but for LDAEP, the analyses differ between the studies. Therefore, the analysis is briefly described for LDAEP studies.

Study	Sample	Stimulus condition/ analysis	Amplitude results	Latency results
P3b				
Ancy et al. (1996)	17 melancholic depression: recurrent DD/ single episode/ bipolar disorder currently depressed, 15 CONT	An attend oddball condition with frequency deviance	DD < CONT	No difference between DD and CONT
Barreiros et al. (2020)	Cohort 1: 20 symptom-remitted MDD, 23 CONT Cohort 2: 19 symptom-remitted MDD, 19 CONT	An attend oddball condition with frequency deviance	No difference between MDD and CONT either in cohort 1 or cohort 2	No difference between MDD and CONT either in cohort 1 or cohort 2
Blackwood et al. (1987)	16 MDD (med. free), 59 CONT	An attend oddball condition with frequency deviance	MDD < CONT	No difference between MDD and CONT
Bruder et al. (1998)	40 MDD/DYS (med. free), 22 CONT	An attend oddball condition with frequency deviance	No difference between MDD/DYS and CONT	Not analysed
Bruder et al. (2002)	58 MDD/DYS (med. free), 18 MDD/DYS + anxiety disord. (med. free), 49 CONT	Attend oddball conditions with frequency deviance and syllable deviance	P3b (averaged over conditions): No difference between MDD/DYS and CONT, MDD/DYS + anxiety > MDD/ DYS, MDD/DYS + anxiety > CONT	Not analysed
Feldmann et al. (2018)	22 MD adolescents, 20 remitted MD adolescents, 32 CONT adolescents	An attend oddball condition with frequency deviance	No differences between MD, remitted MD and CONT	No differences between MD, remitted MD and CONT
Gangadhar et al. (1993)	17 MDD mel. (med. free), 22 CONT	An attend oddball condition with frequency deviance	MDD < CONT	No difference between MDD and CONT
Giedke et al. (1981)	13 primary depressive inpatients, 13 CONT	An attend oddball condition with frequency deviance	No difference between depressive patients and CONT	No difference between depressive patients and CONT
Gordon et al. (1986)	17 MDD/bipolar depressive phase, 55 CONT	An attend oddball condition with frequency deviance	Not analysed	No difference between MDD/ bipolar depressive phase and CONT
Greimel et al. (2015)	24 MD (med. free) children/ adolescents, 21 CONT children/ adolescents	An attend oddball condition with frequency deviance	No difference between MD and CONT	No difference between MD and CONT
Hansenne et al. (1996)	10 MDD with suic., 10 MD without suic., 20 CONT	An attend oddball condition with frequency deviance	MDD with suic. < CONT, No difference between MDD without suic. and CONT	No difference between MDD with suic. and CONT, No difference between MDD without suic. and CONT
Himani et al. (1999)	20 MD, 20 CONT	An attend oddball condition with frequency deviance	No difference between MD and CONT	MD > CONT
Kalayam et al. (1998)	43 MD older adults, 24 CONT older adults	An attend oddball condition with frequency deviance	Not analysed	MD > CONT
Kalayam and Alexopoulos (1999)	25 MDDpr older adults (poor response to antidepressants), 24 MDDgr older adults (good response to antidepressants), 22 CONT older adults	An attend oddball condition with frequency deviance	No differences between the groups	MDDpr > CONT, MDDpr > MDDgr, No difference between MDDgr and CONT
Karaaslan et al. (2003)	16 MDD with psyc. (med. free),	An attend oddball condition with	MDD with psych. < CONT,	MDD with psyc. > CONT, MDD without psyc. > CONT

(continued on next page)

Table 1 (*continued*)

Study	Sample	Stimulus condition/ analysis	Amplitude results	Latency results
	20 MDD without psyc. med free), 20 CONT	frequency deviance	No differences between MDD without psyc. and CONT	
Kaustio et al. (2002)	22 MD/DYS (med. free), 22 CONT	An attend oddball condition with frequency deviance	No difference between MD/DYS and CONT	No difference between MD/DYS and CONT
Kawasaki et al. (2004)	22 MDD (med. free), 22 CONT	An attend oddball condition with frequency deviance	MDD < CONT	No difference between MDD and CONT
Kemp et al. (2009)	78 MDD (med. free), 127 depressed mood (DM), 116 CONT	An attend oddball condition with frequency deviance	MDD < CONT, MDD < DM, No difference between DM and CONT	MDD > CONT, No difference between MDD and DM, No difference between DM and CONT
Kemp et al. (2010)	57 MDD mel. (med. free), 48 MDD non-mel. (med. free), 116 CONT	An attend oddball condition with frequency deviance	MDD mel. < CONT, MDD mel < MDD non-mel., No difference between MDD non-mel. and CONT	MDD mel. > CONT, No difference between MDD non-mel. and CONT, No difference between MDD mel. and MDD non-mel.
Kindermann et al. (2000)	25 MD older adults, 20 CONT older adults	An attend oddball condition with frequency deviance	Not analysed	MD > CONT
Kraiuhin et al. (1990)	11 depression older adults, 15 CONT older adults	An attend oddball condition with frequency deviance	Not analysed	No difference between depression and CONT
Li et al. (2011)	32 MDD/DYS (med. free), 30 CONT	An attend oddball condition with frequency deviance	MDD/DYS < CONT	Not analysed
Li et al. (2014)	30 MDE (med. free), 30 CONT	An attend oddball condition with frequency deviance	MDE > CONT (frontal P3b)	No difference between MDE and CONT
Muir et al. (1991)	46 MDD, 212 CONT	An attend oddball condition with frequency deviance	MDD < CONT	No difference between MDD and CONT
Murthy et al. (1997)	15 DE with somatic symptoms (med. free), 15 DYS (med. free), 15 CONT	An attend oddball condition with frequency deviance	DE < CONT, DYS < CONT, No difference between DE and DYS	No differences between the groups
Nan et al. (2018)	45 MDD, 45 CONT	An attend oddball condition with frequency and intensity deviance	No difference between MDD and CONT	MDD > CONT
Pfefferbaum et al. (1984)	17 MD (med. free), 17 MD (medicated), 115 CONT	An attend oddball condition with frequency deviance	MD (med. free) < CONT, No difference between MD (medicated) and CONT	No differences between the groups
Röschke et al. (1996)	11 MDE (med. free), 10 CONT	An attend oddball condition with frequency deviance	MDE < CONT	Not analysed
Röschke and Wagner (2003)	21 MDE (med. free), 21 CONT	An attend oddball condition with frequency deviance	MDE < CONT	Not analysed
Roth et al. (1981)	21 MDD, 28 CONT	An attend oddball condition with frequency deviance	No difference between MDD and CONT	No difference between MDD and CONT
Sara et al. (1994)	27 MD, 27 CONT	An attend oddball condition with frequency deviance	No difference between MD and CONT	No difference between MD and CONT
Shim et al. (2019)	67 MDD, 39 CONT	An attend oddball condition with frequency deviance	No difference between MDD and CONT	No difference between MDD and CONT
Singh et al. (2000)	40 MDD, 40 CONT	An attend oddball condition with	MDD < CONT	MDD > CONT

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Table 1 (*continued*)

Study	Sample	Stimulus condition/ analysis	Amplitude results	Latency results
		frequency deviance		
Sumi et al. (2000)	35 MDD/DYS older adults, 39 CONT older adults	An attend oddball condition with frequency deviance	Not analysed	MDD/DYS < CONT
Swanwick et al. (1996)	15 MDE older adults, 21 CONT older adults	An attend oddball condition with frequency deviance	No difference between MDE and CONT	No difference between MDE and CONT
Tripathi et al. (2015)	30 MDD, 30 CONT	An attend oddball condition	Not analysed	MDD > CONT
Urretavizcaya et al. (2003)	50 MD mel. (med. free), 31 CONT	An attend oddball condition with frequency deviance	MD < CONT	MD > CONT
van Dinteren et al. (2015)	1008 MDD (med. free), 336 CONT	An attend oddball condition with frequency deviance	MDD < CONT	No difference between MDD and CONT
Vandoolaeghe et al. (1998)	35 MD (med. free), 11 CONT	An attend oddball condition with frequency deviance	No difference between MD and CONT	MD > CONT
Wagner et al. (1997)	11 MDE (med. free), 10 CONT	An attend oddball condition with frequency deviance	MDE < CONT	Not analysed
Weir et al. (1998)	14 MDD, 31 CONT	An attend oddball condition with frequency deviance	No difference between MDD and CONT	No difference between MDD and CONT
Yanai et al. (1997)	16 MD older adults (med. free), 17 CONT older adults	An attend oddball condition with frequency deviance	MD < CONT	No difference between MD and CONT
Zhou et al. (2019)	30 DE, 30 CONT	An attend oddball condition with frequency and intensity deviance	DE < CONT	DE > CONT
P3a				
Chen et al. (2015)	45 MDDf (first episode) mel., 40 MDDr (recurrent) mel., 46 CONT	An ignore oddball condition with	MDDr < CONT, MDDr < MDDf,	MDDr > CONT, MDDr > MDDf,

Table 1 (*continued*)

Study	Sample	Stimulus condition/ analysis	Amplitude results	Latency results
		duration deviance	No difference between MDDf and CONT	No difference between MDDf and CONT
Kähkönen et al. (2007)	13 MDD (med. free), 12 CONT	An ignore oddball condition with frequency deviance and with novel stimuli	No difference between MDD and CONT in P3a elicited by novel stimuli	No difference between MDD and CONT in P3a elicited by novel stimuli
Lepistö et al. (2004)	10 MD children (med. free), 10 CONT children	An ignore oddball condition with /ka/ and /ta/ syllables	MD > CONT	No difference between MD and CONT
Xu et al. (2014)	32 MDD (treatment resistant) (med. free), 31 CONT	An ignore condition with 60 dB deviant stimuli and 0 dB standard stimuli (no sound)	MDD < CONT	No difference between MDD and CONT
Novelty P3				
Bruder et al. (2009)	20 MDD/DYS/ bipolar disorder (med. free), 20 CONT	An attend oddball condition with frequency deviance and novel sounds	Novelty P3: MDD < CONT, Target P3 (P3b): No difference between MDD and CONT	Not analysed
Houston et al. (2004)	28 remitted MDD female adolescents, 96 CONT female adolescents	Task 1: An attend oddball condition with frequency deviance Task 2: An attend oddball condition with frequency deviance and novel sounds	Only CONT group (not MDD group) exhibited smaller target P3 amplitudes during the task 2 as compared to task 1. Otherwise, no differences in the novelty P3 or target P3 between MDD and CONT	No difference between MDD and CONT
Iv et al. (2010)	14 MDD first episode (med. free),	An attend oddball	Novelty P3:	Not analysed

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Table 1 (*continued*)

Study	Sample	Stimulus condition/ analysis	Amplitude results	Latency results
	17 CONT	condition with frequency deviance and novel sounds	MDD < CONT, Target P3 (P3b): MDD < CONT at the right hemisphere	
Jaworska et al. (2013)	25 MDD (NDRI, SSRI non-responders), 26 MDD (NDRI, SSRI responders), 43 CONT	An attend oddball condition with frequency deviance and novel sounds	Novelty P3: MDD (non-res.) < CONT, No difference between MDD (res.) and CONT, No difference between MDD (res.) and MDD (non-res.) Target P3 (P3b): MDD (non-res.) < CONT, MDD (non-res.) < MDD (res.), No difference between MDD (res.) and CONT	No differences between the groups
Tenke et al. (2010)	49 MDD/DYS/ bipolar depressive phase (med. free), 49 CONT	An attend oddball condition with frequency deviance and novel sounds	Novelty P3: depressed patients < CONT Target P3 (P3b): depressed patients < CONT	Not analysed
MMN				
Bissonnette et al. (2020)	16 MDD, 26 CONT	An ignore multi-feature paradigm with 5 deviant tone types (frequency, duration, intensity, location, continuity)	Intensity deviance MMN: MDD > CONT, Location deviance MMN: MDD > CONT, No differences in the frequency, duration, or continuity deviance MMN between MDD and CONT	Frequency deviance MMN: MDD > CONT, No differences in the intensity, location, duration, or continuity deviance MMN between MDD and CONT
Chen et al. (2015)	45 MDDf (first episode) mel., 40 MDDr (recurrent) mel., 46 CONT	An ignore oddball condition with duration deviance	MDDf < CONT, MDDr < CONT, No differences between MDDf and MDDr	No differences between the groups
He et al. (2010)	22 treatment-resistant MDD (med. free), 22 treatment-resistant MDD + borderline personality disorder (BPD) (med. free), 32 CONT	An ignore oddball condition with frequency deviance	MDD > CONT, MDD > MDD + BPD, No difference between MDD + BPD and CONT	No differences between the groups
Hirakawa et al. (2017) (MEG study)	20 MDD, 36 CONT	An ignore oddball condition with frequency deviance	MDD < CONT in the right hemisphere, No differences between MDD and CONT in the left hemisphere	MDD < CONT
Kähkönen et al. (2007) (EEG and MEG study)	13 MDD (med. free), 12 CONT	An ignore oddball condition with 10 % and 20 % frequency deviance	EEG: 10 % frequency deviance MMN MDD > CONT, No group difference in 20 % condition MEG: MMNm No differences between MDD and CONT	No difference in MMN or MMNm latencies between MDD and CONT
Kim et al. (2020)	27 MDD, 32 CONT	An ignore oddball condition with duration deviance	No difference between MDD and CONT	Not analysed
Kuang et al. (2016)	60 MDD (med. free), 30 CONT	An ignore oddball condition with intensity and frequency deviance	Not analysed	MDD > CONT
Lepistö et al. (2004)	10 MD children (med. free), 10 CONT children	An ignore oddball condition with	No difference between MD and CONT	MD < CONT

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Table 1 (*continued*)

Study	Sample	Stimulus condition/ analysis	Amplitude results	Latency results
		/ka/ and /ta/ syllables		
Mu et al. (2016)	20 MDD (med. free), 20 CONT	An ignore multi-feature paradigm with 6 deviant tone types (pitch, timbre, location, intensity, slide, rhythm)	Timbre deviance MMN: MDD > CONT, No differences in the pitch, location, intensity, slide, or rhythm deviance MMN between MDD and CONT	No differences between MDD and CONT
Naismith et al. (2012)	22 life-time MDD older adults, 12 CONT older adults	An ignore oddball condition with duration deviance	Temporal MMN: MDD < CONT, No difference in the frontal-central MMN between MDD and CONT	No differences between MDD and CONT
Pang et al. (2014)	18 MDD (med. free), 22 CONT	An ignore oddball condition in which meaningless syllables were spoken with either emotional (happy, angry, sad) or neutral prosodies	Sad MMN was absent among MDD patients while it was elicited in the CONT group. No differences in happy or angry MMN between MDD and CONT	No differences between MDD and CONT
Qiao et al. (2013)	24 MDD (first episode) (med. free), 24 CONT	An ignore oddball condition with duration deviance	Frontal-central MMN: MDD < CONT in the increment condition, No differences in the decrement condition, Temporal MMN: No differences between the group Females:	MDD > CONT

Table 1 (*continued*)

Study	Sample	Stimulus condition/ analysis	Amplitude results	Latency results
Qiao et al. (2015)	30 MDD (first episode) (med. free): 15 males, 15 females, 30 CONT: 15 males, 15 females	An ignore oddball condition with duration deviance	Frontal-central MMN increment condition MDD < CONT, No differences in the decrement condition, No differences between the groups in the temporal MMN. Males: No differences in the frontal-central or temporal MMN between the groups either in the increment or decrement condition	Not analysed
Restuccia et al. (2016)	16 MDD (med. free), 10 CONT	An ignore oddball condition with frequency deviance, Stimulus intensity: 70 dB 50 % of the blocks, 90 dB 50 % of the blocks	MDD > CONT in the 90 dB session, No differences between MDD and CONT in the 70 dB session	MDD < CONT in the 90 dB session, No differences between MDD and CONT in the 70 dB session
Ruohonen and Astikainen (2017)	41 DD/DYS, 21 CONT	An ignore oddball condition with intensity deviance	No differences between DD/DYS and CONT	Not analysed
Ruohonen et al. (2020)	16 MDD (young), 19 MDD (older adults), 20 CONT (young), 17 CONT (older adults) all females	An ignore oddball condition with intensity deviance	No difference between MDD and CONT	Not analysed
Takei et al. (2009) (MEG study)	14 MDD, 19 CONT	An ignore multi-feature oddball	MDD < CONT when all the deviant conditions	No differences between MDD and CONT

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Table 1 (*continued*)

Study	Sample	Stimulus condition/ analysis	Amplitude results	Latency results
		condition with frequency, duration, and vowel deviants	were analysed together, No differences between MDD and CONT in any separate condition	
Umbricht et al. (2003)	22 MD, 25 CONT	An ignore multi-feature oddball condition with frequency deviants and duration deviants	No difference between MD and CONT	No difference between MD and CONT
LDAEP				
Fitzgerald et al. (2009)	14 MDD (mel.), 13 MDD (non-mel.), 14 CONT	Tones of five intensities (60, 70, 80, 90, 100 dB) were presented / An amplitude change in N1/P2 component	MDD (mel.) < CONT, MDD (mel.) < MDD (non-mel.), No difference between MDD (non-mel.) and CONT	NA
Gallinat et al. (2000)	29 MDD/DYS/ Bipolar depression phase: 17 SSRI non-responders, 12 SSRI responders, 29 CONT	Tones of five intensities (54, 64, 74, 84, 94 dB) were presented / An amplitude change in N1/P2 component	Before SSRI treatment: SSRI non-res. < CONT, No difference between depression SSRI res. and CONT. No difference between depression SSRI non-res. and depression SSRI res.	NA
Gopal et al. (2004)	12 serotonin associated clinical depression (SSRI med.), 12 serotonin associated clinical depression (no SSRI med.), 12 CONT	Tones of five intensities (15, 25, 35, 45, 55 dB) were presented / An amplitude change in auditory brainstem response (ABR)	An amplitude change in ABR peak V and N1/P2: depression (no SSRI med.) > CONT, No difference between depression (SSRI med.) and CONT	NA

Table 1 (*continued*)

Study	Sample	Stimulus condition/ analysis	Amplitude results	Latency results
		peak V and N1/P2 component		
Graßnickel et al. (2015)	20 MDE (suic.), 20 MDE (non-suic.), 20 CONT	Tones of five intensities (79,87.5, 96, 104.5, 111 dB) were presented/ An amplitude change in N1/P2 component	No differences between the groups	NA
Hwang et al. (2021)	23 MDD (high suic.), 22 MDD (low suic.), 22 CONT	Tones of five intensities (60, 70, 80, 90, 100 dB) were presented / An amplitude change in N1/P2 component	MDD (high suic.) > MDD (low suic.), No difference between MDD (high suic.) and CONT, No difference between MDD (low suic.) and CONT	NA
Jaworska et al. (2012)	53 MDD (med. free), 43 CONT	Tones of five intensities (60, 70, 80, 90, 100 dB) were presented / An amplitude change in N1, P2 and N1/P2 components	No difference between MDD and CONT	NA
Kim et al. (2019)	20 MDD with ADHD symptoms (med. free), 20 MDD without ADHD symptoms (med. free), 20 CONT	Tones of five intensities (60, 70, 80, 90, 100 dB) were presented / An amplitude change in N1/P2 component	MDD with ADHD < CONT, MDD with ADHD < MDD without ADHD, No difference between MDD without ADHD and CONT	NA
Kim et al. (2021)	45 MDD (suicide attempts SA) (med. free), 49 MDD (suicidal ideation SI) (med. free),	Tones of five intensities (60, 70, 80, 90, 100 dB) were presented /	MDD (SA) < CONT, MDD (SI) < CONT, No difference between MDD (SA) and MDD (SI)	NA

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Table 1 (*continued*)

Study	Sample	Stimulus condition/ analysis	Amplitude results	Latency results
	36 CONT	An amplitude change in N1/P2 component		
Linka et al. (2009)	14 MDD SSRI treatment, 12 MDD SNRI treatment, 43 CONT	Tones of five intensities (60, 70, 80, 90, 100 dB) were presented / An amplitude change in P1, N1, P2, P1/N1 and N1/P2 components	No difference between MDD (SSRI) and CONT, No difference between MDD (SNRI) and CONT either before or after the treatment	NA
Manjarrez-Gutierrez et al. (2009)	9 MDD + type 1 diabetes (med. free), 8 MDD only (med. free), 11 type 1 diabetes only, 9 CONT	Tones of four intensities (40, 60, 90, 103 dB) were presented / An amplitude change in N1/P2 component	MDD + diabetes > CONT, MDD + diabetes > MDD only, MDD + diabetes > diabetes only, No differences between MDD only and CONT	NA
Ostermann et al. (2012)	86 MD, 40 CONT	Tones of five intensities (60, 70, 80, 90, 100 dB) were presented / An amplitude change in N1/P2 component	MD < CONT	NA
Park et al. (2010)	123 MDD, 55 CONT	Tones of five intensities (55, 65, 75, 85, 95 dB) were presented / An amplitude change in N1/P2 component	No difference between MDD and CONT	NA
Ruohonen et al. (2020)	16 MDD (young), 19 MDD (older adults), 20 CONT (young), 17 CONT (older adults) all females	Tones presented in oddball condition / A difference between N1 amplitude in response to low-intensity standard sounds (60 dB) and high-intensity standard sounds (80 dB)	No difference between MDD and CONT	NA
Uhl et al. (2011)	18 MDD, 18 CONT	Tones of five intensities (60, 70, 80, 90, 100 dB) were presented / An amplitude change in N1/P2 component	No difference between MDD and CONT	NA

MDD = major depressive disorder, MD = major depression, MDE = major depressive episode, DD = depressive disorder, DE = depressive episode, DYS = dysthymia, CONT = non-depressed control subjects, med. free = medication free, mel. = melancholic, non-mel. = non-melancholic, psyc. = psychotic features, suic. = suicidality, NA = not applicable.

depressed patients and controls (Ancy et al., 1996; Barreiros et al., 2020; Blackwood et al., 1987; Feldmann et al., 2018; Gangadhar et al., 1993; Giedke et al., 1981; Gordon et al., 1986; Greimel et al., 2015; Hansenne et al., 1996; Jaworska et al., 2013; Kaustio et al., 2002; Kawasaki et al., 2004; Kraiuhin et al., 1990; Li et al., 2014; Muir et al., 1991; Murthy et al., 1997; Pfefferbaum et al., 1984; Roth et al., 1981; Sara et al., 1994; Shim et al., 2019; Swanwick et al., 1996; van Dinteren et al., 2015; Weir et al., 1998; Yanai et al., 1997).

Most studies reviewed here focused on young and middle-aged adults. Regarding the research on auditory P3b alterations among children and adolescents with depressive disorder, no differences between depressed patients and non-depressed controls were found either in the P3b amplitude (Feldmann et al., 2018; Greimel et al., 2015; remitted depression: Feldmann et al., 2018; Houston et al., 2004) or latency (Feldmann et al., 2018; Greimel et al., 2015; remitted depression: Feldmann et al., 2018). As for auditory P3b alterations regarding non-demented older adults with depressive disorder, Kalayam et al. (1998) and Kindermann et al. (2000) found prolonged P3b latency among depressed patients compared to non-depressed controls. In contrast, Sumi et al. (2000) found shorter P3b latency among depressed older adults. Kraiuhin et al. (1990) and Yanai et al. (1997) did not find any differences in the latencies between non-demented older depressed patients and non-depressed non-demented controls, but Yanai et al.'s (1997) study showed that the amplitude was attenuated among depressed patients compared to controls. Swanwick et al. (1996) showed that neither P3b amplitude nor latency differed between depressed older adults and non-depressed controls.

Alterations in auditory P3b amplitude and latency have been investigated for the subgroups of depressive disorder. In the study by Murthy et al. (1997), the amplitude of P3b was attenuated among depressed patients with somatic symptoms and among dysthymia patients relative to non-depressed controls. There were no P3b latency differences

between these groups. Four studies have studied P3b alterations in melancholic depression (Ancy et al., 1996; Gangadhar et al., 1993; Kemp et al., 2010; Urretavizcaya et al., 2003). In all these studies, the P3b amplitude was attenuated in the melancholic depression group compared to the non-depressed controls. In the studies by Kemp et al. (2010) and Urretavizcaya et al. (2003), the P3 latency was also prolonged in the melancholic depression group relative to the control group.

Regarding depressive disorder with psychotic symptoms, Karaaslan et al. (2003) studied depressed patients with and without psychotic features and non-depressed control participants. The auditory P3b amplitude was attenuated in the group of patients with psychotic features compared to the controls, but there were no differences in the P3b amplitude between patients without psychotic features and controls. Both depression groups differed from the controls in respect of prolonged latency (Karaaslan et al., 2003). Kaustio et al. (2002) investigated whether P3b amplitude or latency is related to psychotic symptoms in depressed patients. Psychotic symptoms were associated with a reduction in P3b amplitude at the left temporocentral sites and with prolonged P3b latency.

In some of the reviewed studies, a relationship between auditory P3b amplitude/latency and depression severity was investigated. The most common finding was that neither the P3b amplitude (Barreiros et al., 2020; Blackwood et al., 1987; Hansenne et al., 1996; Karaaslan et al., 2003; Murthy et al., 1997; Roth et al., 1981; Sara et al., 1994; Urretavizcaya et al., 2003; Vandoolaeghe et al., 1998; Yanai et al., 1997) nor P3b latency (Blackwood et al., 1987; Karaaslan et al., 2003; Roth et al., 1981; Sara et al., 1994; Sumi et al., 2000; Urretavizcaya et al., 2003; Vandoolaeghe et al., 1998) correlated with depression severity measured by the total score of the Hamilton Depression Rating Scale (HDRS; Barreiros et al., 2020; Blackwood et al., 1987; Hansenne et al., 1996; Karaaslan et al., 2003; Murthy et al., 1997; Roth et al., 1981; Sumi et al., 2000; Urretavizcaya et al., 2003; Vandoolaeghe et al., 1998; Yanai et al., 1997) or by the Beck Depression Inventory (BDI; Sara et al., 1994). However, in a few studies, such relationships were reported. Gangadhar et al. (1993) and van Dinteren et al. (2015) found a negative correlation between P3b amplitude and depression severity as measured by HDRS. Singh et al. (2000) found a positive correlation, while Nan et al. (2018) found a negative correlation between P3b latency and depression severity (HDRS). In addition, in the study by Tripathi et al. (2015), there was a significant difference in the P3b latency between mild and severe, between mild and very severe and between moderate and severe levels of depression measured by HDRS.

3.1.2. P3a and novelty P3

There are only a few studies in which auditory P3a and novelty P3 responses in patients with depressive disorder were studied in comparison to the controls.

In a study by Chen et al. (2015), P3a was obtained during an ignore oddball condition in first-episode major depression subjects, recurrent major depression subjects and non-depressed controls. The recurrent major depression group had an attenuated P3a amplitude and prolonged P3a latency compared to the controls and first-episode major depression group. No differences were found in the P3a amplitude and latency between first-episode major depression group and controls. In the study by Xu et al. (2014), P3a amplitude was attenuated in the group with treatment-resistant depression relative to the non-depressed control group. There were no differences in the P3a latency between the groups. Kähkönen et al. (2007) found no differences either in the amplitude or latency of P3a between patients with depressive disorder and non-depressed controls. P3a response in children with depression disorder was also investigated; an increased P3a amplitude was found in the depression group compared to the non-depressed control group (Lepistö et al., 2004). There were no P3a latency differences between the groups.

In studies that investigated novelty P3 utilising a novelty oddball task, the novelty P3 amplitude elicited by distractor stimuli was attenuated in patients with depressive disorder compared to the controls (Bruder et al., 2009; Iv et al., 2010; Tenke et al., 2010; difference only between depressed patients who were non-responders to antidepressants and controls: Jaworska et al., 2013). However, no differences in novelty P3 between adolescents with remitted depressive disorder and never-depressed controls were found (Houston et al., 2004).

In some of the reviewed studies that examined auditory P3a and novelty P3, the relationship between these ERP measures and depression severity was investigated. In a study by Chen et al. (2015), the P3a amplitude was negatively correlated with the severity of depression (HDRS) in first-episode depression patients and recurrent depression patients. Furthermore, the P3a amplitude deficits were positively correlated with the number of depressive episodes in recurrent depression patients. In the study by Jaworska et al. (2013), novelty P3 latency correlated positively and novelty P3 amplitude correlated negatively with depression severity measured by HDRS in male depressed patients, while no significant correlations existed in females. Bruder et al. (2009) found no significant correlation between novelty P3 amplitude and the severity of depression measured by BDI.

3.2. MMN

The differences in automatic change detection between patients with depressive disorder and non-depressed control participants were studied by investigating the amplitudes and latencies of MMN in an ignore auditory oddball condition. In contrast to earlier described P3b studies in which mostly frequency deviance oddball tasks were applied, MMN alterations in depressive disorder were investigated with several different deviating stimulus features, such as location, frequency, intensity and multi-feature and syllable deviance conditions, which were applied in the following auditory MMN studies.

Findings on the differences in auditory MMN responses between patients with depressive disorder and non-depressed controls are conflicting. An attenuated MMN amplitude among depressed patients was found in a few studies (Chen et al., 2015; Hirakawa et al., 2017; Naismith et al., 2012; Qiao et al., 2013, 2015; Takei et al., 2009). In addition, an increased MMN amplitude in patients with depressive disorder has been reported (Bissonnette et al., 2020; He et al., 2010; Kähkönen et al., 2007; Mu et al., 2016; Restuccia et al., 2016). Some studies found no differences in the MMN amplitude between depressed patients and controls (Kim et al., 2020; Lepistö et al., 2004; Ruohonen and Astikainen, 2017; Ruohonen et al., 2020; Umbricht et al., 2003).

As for auditory MMN latencies, a few studies found prolonged MMN latency among depressed patients (Bissonnette et al., 2020; Kuang et al., 2016; Qiao et al., 2013). Additionally, a shorter MMN latency has been reported in some studies (Hirakawa et al., 2017; Lepistö et al., 2004; Restuccia et al., 2016). Several studies did not find any differences in MMN latency between depressed patients and controls (Chen et al., 2015; He et al., 2010; Kähkönen et al., 2007; Mu et al., 2016; Naismith et al., 2012; Pang et al., 2014; Takei et al., 2009; Umbricht et al., 2003).

All the EEG studies that found an attenuated MMN amplitude in patients with depressive disorder applied the duration deviance oddball condition (Chen et al., 2015; Naismith et al., 2012; Qiao et al., 2013, 2015). In a study by Qiao et al. (2013), depressed patients exhibited an attenuated MMN amplitude and prolonged MMN latency compared to non-depressed controls only in the duration increment condition (deviant stimuli of longer duration than standard stimuli). Similarly, in another study by Qiao et al. (2015), an attenuated MMN amplitude in depressed patients was found only in the duration increment condition, and only in female depressed patients relative to female controls. However, among males, there were no differences in MMN amplitude between depressed patients and controls (Qiao et al., 2015). Naismith et al. (2012) found an attenuated MMN amplitude among older adult patients with lifetime depressive disorder compared to non-depressed controls. No differences in MMN latency were found between the groups (Naismith et al., 2012). In a magnetoencephalography (MEG)

study by Hirakawa et al. (2017) in which a frequency deviance paradigm was utilised, depressed patients showed attenuated magnetic global field power of MMNm (magnetoencephalographic counterpart of MMN) compared to non-depressed controls in the right hemisphere. In addition, the latency of MMNm was decreased in the group of depressed patients.

A frequency deviance oddball condition was applied in some of the studies in which increased MMN amplitude was found in patients with depressive disorder (He et al., 2010; Kähkönen et al., 2007; Restuccia et al., 2016). Restuccia et al. (2016) investigated frequency deviance MMN utilising two different stimulus intensities (70 dB and 90 dB). During the 90 dB session, MMN amplitude was increased, and latency was shorter among depressed patients, but during the 70 dB session, there were no differences in MMN amplitude or latency between depressed patients and controls. In the study by Kähkönen et al. (2007), both EEG and MEG recordings were conducted, and MMN was studied in an ignore oddball condition with deviants of 10 % and 20 % frequency change. In the EEG study, the MMN amplitude to the 10 % frequency deviance was increased in depressed patients compared to non-depressed controls, while there were no differences in the amplitude of the 20 % frequency deviance MMN between the groups. Regarding the MEG recording, no differences in MMNm amplitude between depressed patients and non-depressed controls were found in either the 10 % or 20 % frequency deviance conditions. There were no differences in the MMN or MMNm latencies between the groups.

Some studies found no MMN amplitude differences between patients with depressive disorder and controls. These studies included the duration deviance condition (Kim et al., 2020; Umbricht et al., 2003), the frequency deviance condition (Umbricht et al., 2003) and the intensity deviance condition (Ruohonen and Astikainen, 2017; Ruohonen et al., 2020). In the study by Umbricht et al. (2003), latency analyses were also conducted, and no differences in either duration deviance or frequency deviance MMN latencies were found between depressed patients and non-depressed controls.

Some studies investigated MMN in patients with depressive disorder by applying an ignore multi-feature paradigm (Bissonnette et al., 2020; Mu et al., 2016; Takei et al., 2009). In these studies, auditory MMN responses elicited by some of the stimulus features differed between depressed patients and non-depressed controls, while there were no group differences in MMN responses elicited by other stimulus features. In the study by Bissonnette et al. (2020), an ignore condition with five deviant tone types (frequency, duration, intensity, location and continuity) was utilised. Depressed patients had increased MMN amplitudes following intensity and location deviants compared to the non-depressed control group, but no differences between depressed patients and controls were found in MMN amplitudes elicited by the other deviant types. The latency of the frequency deviance MMN was prolonged among depressed patients, while there were no other MMN latency differences between depressed patients and controls (Bissonnette et al., 2020). Mu et al. (2016) measured MMN to changes in six musical sound features (pitch, timbre, location, intensity, slide and rhythm). The amplitude of the timbre deviance MMN was increased in the depressed patients compared to the non-depressed controls, but there were no group differences in the MMN amplitudes elicited by the other deviant types. No latency differences between the depression and control groups were found (Mu et al., 2016). Takei et al. (2009) studied MMNm in duration and frequency deviance as well as vowel deviance conditions utilising MEG. They found reduced magnetic global field power of the MMNm in patients with depressive disorder compared to non-depressed controls when all the deviant conditions were analysed together, but no group differences in the MMNm amplitude were found in any separate condition.

MMN has also been investigated utilising oddball conditions with vowel stimuli (Takei et al., 2009) and with syllable stimuli deviating in terms of consonant sound (Lepistö et al., 2004) or emotional prosody (Pang et al., 2014). Takei et al. (2009) used a standard stimulus of the Japanese vowel sound /a/ and the deviant stimulus of the vowel sound /o/. There were no differences in MMN amplitude or latency between depressed patients and non-depressed controls (Takei et al., 2009). Lepistö et al. (2004) investigated MMN differences among children with depressive disorder by utilising an oddball condition in which Finnish consonant-vowel syllables /ka/ and /ta/ were applied as standard and deviant stimuli. No differences in MMN amplitude between depressed and non-depressed control children were found, but a shorter MMN latency was found in children with depressive disorder (Lepistö et al., 2004). Pang et al. (2014) used an oddball paradigm in which meaningless syllables were spoken with either neutral or emotional (happy, angry and sad) prosodies. Sad MMN was absent among depressed patients, while it was elicited in the non-depressed control group. Happy, angry and neutral deviants were not processed differently between the depressed patients and controls. There were no differences in MMN latencies between the groups (Pang et al., 2014).

In most of the reviewed studies, a relationship between MMN amplitude/latency and depression severity was investigated. The results showed that neither MMN amplitude (Hirakawa et al., 2017; He et al., 2010; Kähkönen et al., 2007; Mu et al., 2016; Naismith et al., 2012; Pang et al., 2014; Qiao et al., 2013, 2015; Takei et al., 2009; Umbricht et al., 2003) nor MMN latency (He et al., 2010; Hirakawa et al., 2017; Mu et al., 2016; Pang et al., 2014; Takei et al., 2009) correlated with depression severity measured by the total score of HDRS (He et al., 2010; Hirakawa et al., 2017; Kähkönen et al., 2007; Mu et al., 2016; Naismith et al., 2012; Pang et al., 2014; Qiao et al., 2013, 2015; Takei et al., 2009; Umbricht et al., 2003) or by The Plutchik–van Praag Depression Inventory (PVP; He et al., 2010). However, in the study by Bissonnette et al. (2020) in which a multi-feature paradigm was applied, the amplitude of location deviance MMN negatively correlated with depression severity measured by a depression subscale of the Hospital Anxiety and Depression Scale (HADS), while there were no correlations between depression severity and MMN amplitudes elicited by the other deviant types.

3.3. LDAEP

In the following LDAEP studies on depressive disorder, LDAEP alterations were typically investigated for some subgroups of depressed patients; for instance, melancholic depression (Fitzgerald et al., 2009), depression with suicidality (Graßnickel et al., 2015; Hwang et al., 2021; Kim et al., 2021), depression with attention deficit hyperactivity disorder (ADHD) symptoms (Kim et al., 2019) and depression with diabetes (Manjarrez-Gutierrez et al., 2009) were studied. LDAEP has mostly been defined as the change in amplitude of the auditory N1/P2 component (a difference between N1 and P2) in response to different stimulus intensities.

LDAEP studies that investigated patients with depressive disorder and non-depressed controls have yielded quite inconsistent findings, but most commonly, no differences in LDAEP between depressed patients and controls have been found (Graßnickel et al., 2015; Hwang et al., 2021; Jaworska et al., 2012; Linka et al., 2009; Park et al., 2010; Ruohonen et al., 2020; Uhl et al., 2011). However, in a few studies, increased LDAEP (Gopal et al., 2004; Manjarrez-Gutierrez et al., 2009) and attenuated LDAEP (Fitzgerald et al., 2009; Gallinat et al., 2000; Kim et al., 2019, 2021; Ostermann et al., 2012) in depressed patients relative to non-depressed controls were reported.

In a study by Gopal et al. (2004), LDAEP was compared between SSRI medicated depressed patients, unmedicated depressed patients and non-depressed control subjects. An auditory brainstem response (ABR) peak V and late N1/P2 in response to different stimulus intensities were investigated. There was steeper amplitude growth in the ABR peak V and N1/P2 only in the unmedicated depressed group compared to the non-depressed control group (Gopal et al., 2004). Manjarrez-Gutierrez et al. (2009) compared four groups: depressed diabetics group, diabetes-only group, depression-only group and control group without depression

and diabetes. The depressed diabetic group showed significantly steeper LDAEP than the other groups. No differences between the depression-only versus control group were found.

Regarding studies in which decreased LDAEP among patients with depressive disorder was found, Ostermann et al. (2012) compared LDAEP between depressed inpatients and non-depressed controls, whereas the other four studies focused on different subtypes of depressive disorder (Fitzgerald et al., 2009; Gallinat et al., 2000; Kim et al., 2019, 2021). In the study by Fitzgerald et al. (2009), a melancholic depression group had a weaker LDAEP than the non-depressed control group, while there was no difference in LDAEP between the non-melancholic depression group and controls. In the study by Kim et al. (2021), LDAEP was found to be decreased in the group of depressed patients who had engaged in suicide attempts and the group of depressed patients with suicidal ideation compared to non-depressed controls. In the study by Hwang et al. (2021), LDAEP was increased in depressed patients with high suicidality compared to depressed patients with low suicidality, while there were no differences in LDAEP between depression patient groups and controls. Kim et al. (2019) found that LDAEP was decreased among depressed patients with ADHD symptoms relative to depressed patients without ADHD symptoms and controls without depression and ADHD symptoms, while there were no differences in LDAEP between depressed patients without ADHD symptoms and controls. Gallinat et al. (2000) investigated LDAEP in SSRI responder depressed patients, SSRI non-responder depressed patients and non-depressed controls. SSRI non-responders had a decreased LDAEP in comparison to non-depressed controls, but there were no differences between SSRI responders and controls.

Regarding the studies in which no differences in LDAEP were found between patients with depressive disorder and non-depressed control participants, Graßnickel et al. (2015), Hwang et al. (2021), Jaworska et al. (2012), Linka et al. (2009), Park et al. (2010) and Uhl et al. (2011) compared adults with depressive disorder and non-depressed subjects, and Ruohonen et al. (2020) compared younger and older depressed females and younger and older non-depressed control females. Graßnickel et al. (2015) did not find any differences in LDAEP between suicidal depressed patients and controls, between non-suicidal depressed patients and controls or between suicidal and non-suicidal depressed patients.

In a few of the reviewed studies, a correlation between LDAEP and depression severity measured by the HDRS, the Montgomery Åsberg Depression Rating Scale (MADRS), the Brief Symptom Inventory (BSI; a subscale of depression) and Beck Depression Inventory (BDI) were examined. The findings were inconsistent; in some studies, no correlation was found (Fitzgerald et al., 2009 [MADRS]; Gallinat et al., 2000 [HDRS]; Jaworska et al., 2012 [MADRS]) whereas in other studies, a positive correlation was reported (Kim et al., 2019 [BDI]; Ostermann et al., 2012 [BSI]).

4. Discussion

This review focused on the findings regarding the auditory ERP differences between patients with depressive disorder and non-depressed control subjects, addressing the challenge of identifying ERP-based biomarkers for depressive disorder. The findings of the reviewed studies were not highly consistent regarding auditory P3, MMN or LDAEP. However, some patterns in the findings were identified. Regarding P3b, the common findings were attenuated amplitude and prolonged latency in depressed patients compared to controls, but there were also numerous studies that showed no such differences. Regarding novelty P3, the findings were quite consistent, indicating attenuated amplitude among depressed patients, but P3a findings were more inconsistent. Concerning MMN, findings were variable. Particularly, the amplitude of duration deviance MMN seemed to be commonly attenuated, and the amplitude of frequency deviance MMN increased in depressed patients. In LDAEP studies, it was common to find no differences between depressed patients and non-depressed controls. These discrepancies in the findings, which were found for all components, may reflect the heterogeneity of depressive disorder symptoms, diagnoses and severity, as well as the differences in the neural underpinnings of different subgroups. Furthermore, diverse methodological choices related to, for instance, experimental paradigms, diagnostic criteria for depression, stage of depression (first episode vs. recurrent), severity of depression and use of antidepressant medication may explain the inconsistencies in the findings. Next, we discuss these aspects separately in the context of each component.

4.1. P3

There were only a few studies on auditory P3a and novelty P3. Regarding novelty P3, the findings were quite consistent, indicating an attenuated novelty P3 amplitude among patients with depressive disorder (Bruder et al., 2009; Iv et al., 2010; Jaworska et al., 2013; Tenke et al., 2010) while results on the P3a were more variable. By far, the largest number of ERP studies on depressive disorder, totalling 48 of the 84 studies included in this review, focused on the P3b component. An attenuated P3b amplitude and prolonged P3b latency in depressed patients were found in several studies, but there were also findings of no differences in the amplitude or latency between depressed patients and non-depressed controls.

Variable findings of auditory P3b may be related to different subtypes of depression. For instance, all studies that investigated melancholic depression found an attenuated P3b amplitude in depressed patients (Ancy et al., 1996; Gangadhar et al., 1993; Kemp et al., 2010; Urretavizcaya et al., 2003). In the studies by Kemp et al. (2010) and Urretavizcaya et al. (2003), the latency of P3b reflecting information processing speed was prolonged in patients with melancholic depression, which may be related to psychomotor retardation, which is one of the common symptoms of melancholic depression (Khan et al., 2006; Parker et al., 2013). The finding regarding prolonged P3b latency was in congruence with a finding by Schlegel et al. (1991) that demonstrated a relationship between the latency of P3b and melancholic symptoms of depression, as well as between P3b latency and motor, verbal, intellectual and emotional retardation. An attenuated P3b amplitude and prolonged latency have also been linked to psychotic features in depression (Karaaslan et al., 2003; Kaustio et al., 2002; melancholic depression: Santosh et al., 1994). These findings on P3b in melancholic and psychotic depression are in line with the studies in which cognitive functions were assessed by neurocognitive tests. The results suggest that patients with melancholic depression show more severe cognitive impairment, especially in terms of memory, executive function, attention and reaction time, than non-melancholic depressed patients (e.g., Naismith et al., 2003; Withall et al., 2010; for a review, see Bosaipo et al., 2017), and patients with psychotic depression have more severe cognitive deficits, especially in terms of memory, executive functioning and psychomotor speed, than non-psychotic depressed patients (for a review, see Fleming et al., 2004). Alterations in P3b amplitude reflecting attention, decision-making and working memory operations (e.g., Kok, 2001) and P3b latency reflecting information processing speed (e.g., Polich, 2007) may be associated with these cognitive deficits.

Regarding neurotransmitters, evidence showed that dopaminergic and noradrenergic dysfunction especially contributes to the maintenance of symptoms of melancholic and psychotic depression (for a review, see Malhi et al., 2005). Furthermore, the dopaminergic and noradrenergic systems might be associated with the P3 response (e.g., Liu et al., 2009; Mulert et al., 2006; Pogarell et al., 2011; Swick et al., 1994; for reviews, see Nieuwenhuis et al., 2005, 2011; Polich, 2007). Thus, dopaminergic and noradrenergic dysfunction might partly explain the findings regarding the P3b alterations in melancholic and psychotic depression, which were relatively consistent (Ancy et al., 1996; Gangadhar et al., 1993; Karaaslan et al., 2003; Kaustio et al., 2002; Kemp et al., 2010; Urretavizcaya et al., 2003). The consistency regarding the

findings on P3b within these studies could be at least partly explained by the diagnostic homogeneity of the samples (compared to studies in which depressive disorder as a wide diagnostic group was studied).

The inconsistency in the results regarding auditory P3b between depressed patients and non-depressed controls in general may also be related to some comorbid factors. For instance, Bruder et al. (2002) investigated the P3b amplitude in patients with depression only, anxiety only, a comorbid group (depression and anxiety) and a control group. The amplitude of P3b did not differ between the depression-only group and control group, but the comorbid group exhibited an increased P3b amplitude when compared to the other groups. Thus, the need to take comorbidities into account when studying P3b amplitude is obvious. However, in most of the reviewed P3b studies, anxiety was not considered even if anxiety symptoms occur commonly during the course of a depressive disorder (Rosellini et al., 2018), and anxiety disorders are common comorbid disorders among depressive disorder patients (Hasin et al., 2018). In the reviewed studies, anxiety symptoms were not included as a covariant in the analyses. Furthermore, 11 studies excluded anxiety disorder, and only one study excluded anxiety symptoms for depressed participants.

Regarding antidepressant medication, in 24 of the reviewed P3b studies, the participants with depressive disorder were medication-free; in 16 studies, the depressed participants were on medication; and in 8 studies, the medication condition was not reported. Importantly, in most of the studies in which an attenuated amplitude was found, the depressed patients were unmedicated. Instead, in most of the studies in which no difference in the P3b amplitude between depressed patients and non-depressed controls was found, the depressed patients were on medication, or the current medication condition was not reported. Therefore, medication might be able to recover attenuated P3b amplitude in depression. Some studies indicated that attenuated P3b amplitude indeed normalises with successful antidepressant treatments (Blackwood et al., 1987; Karaaslan et al., 2003; Yanai et al., 1997). However, there were also some contradictory findings related to P3b and antidepressants. In the study by Vandoolaeghe et al. (1998), no differences between pre-treatment and post-treatment P3b amplitudes were found, and in the study by Sara et al. (1994), there were no differences in the amplitude of P3b between unmedicated and medicated depressed patients. SSRIs are commonly used antidepressants. In healthy adults, neither the administration of SSRIs (Oranje et al., 2008; Wienberg et al., 2010) nor an acute depletion of serotonin (Ahveninen et al., 2002) had an impact on P3b amplitude. However, it is possible that antidepressants used by depressed participants had an impact on the results reviewed here, but more research is still needed to address this issue.

An important question related to the potential clinical utility of auditory P3b is whether P3b amplitude provides a trait or state marker of depressive disorder. In addition to the aforementioned studies indicating that altered P3b amplitude normalises with successful antidepressant treatment (Blackwood et al., 1987; Karaaslan et al., 2003; Yanai et al., 1997), altered P3b amplitude normalises with electroconvulsive therapy treatment (Ancy et al., 1996; Gangadhar et al., 1993; Nurminen et al., 2005). Additionally, some other results are in line with the suggestion of P3b amplitude as a state-dependent phenomenon. Barreiros et al. (2020) and Houston et al. (2004) found no differences in P3b amplitude between symptom-remitted depressive disorder patients and never-depressed controls, and Gangadhar et al. (1993) and van Dinteren et al. (2015) reported a negative correlation between P3b amplitude and depression severity. In contrast, several studies showed that P3b amplitude does not correlate with depression severity (Barreiros et al., 2020; Blackwood et al., 1987; Hansenne et al., 1996; Karaaslan et al., 2003; Murthy et al., 1997; Roth et al., 1981; Sara et al., 1994; Urretavizcaya et al., 2003; Vandoolaeghe et al., 1998; Yanai et al., 1997) suggesting that P3b might rather be a trait marker of depression. Thus, the results are conflicting, and more research is required.

In the P3b studies reviewed in this work, a frequency deviance auditory oddball task was most frequently utilised. However, these studies varied in the frequencies of the tones, interstimulus intervals, stimulus duration, number of trials and probability for target tones. There were also differences in the sample sizes between the studies. Furthermore, the inconsistencies in the P3b latency findings may partly be due to the difficulty in defining latencies accurately because P3 responses usually have a plateau shape, and *clear peaks* may have been lacking. In future studies on P3b, it would be important to manipulate different parameters (e.g., stimulus parameters) in oddball tasks to find the best options for studies investigating differences between patients with depressive disorder and controls. It is unclear whether the two-stimuli oddball task is suitable for investigations into P3b differences between depressed patients and controls. According to Bruder et al. (2012), the inconsistent findings regarding P3b among depressed patients may have been due to the use of a simple two-stimuli task that is not demanding enough to reveal a depression-related decrease in the P3b amplitude. Bruder et al. (2012) suggested measuring P3b during more challenging cognitive tasks that allow the evaluation of hypotheses concerning specific cognitive deficits in depression.

4.2. MMN

The auditory MMN was examined in the absence of participants' attention and task performance (e.g., Näätänen et al., 1978, 2012), which avoided some problems related to motivational and attentional factors in measurement (He et al., 2010; Kähkönen et al., 2007; Takei et al., 2009). However, the studies that investigated auditory MMN alterations in depressive disorder reviewed in this work showed inconsistent results regarding both amplitudes and latencies, with some reporting attenuated MMN amplitudes or shorter latencies, and others reporting increased amplitudes or prolonged latencies. In some studies, no differences between depressed patients and non-depressed controls were found. The heterogeneity of the research paradigm and stimuli, as well as the heterogeneity of depressive disorder as an illness, may partly explain this inconsistency. In contrast to studies on P3b in which an attend oddball task with sound frequency as a deviating stimulus feature has been most frequently applied, in MMN studies, an ignore oddball condition with multiple different deviating stimulus features (e.g., duration, frequency, intensity, vowel, syllable and multi-feature paradigms) has been employed.

According to the findings of the auditory MMN studies, the amplitude of the duration deviance MMN might be mostly attenuated in patients with depressive disorder compared to controls (Chen et al., 2015; Naismith et al., 2012; Qiao et al., 2013, 2015), while frequency deviance MMN amplitude was found to be mostly increased in depressed patients (He et al., 2010; Kähkönen et al., 2007; Restuccia et al., 2016). However, there are also duration deviance MMN studies (Bissonnette et al., 2020; Kim et al., 2020; Takei et al., 2009; Umbricht et al., 2003) and frequency deviance MMN studies (Bissonnette et al., 2020; Mu et al., 2016; Takei et al., 2009; Umbricht et al., 2003) in which no difference between depressed patients and non-depressed controls were found, and one frequency deviance MMNm study (Hirakawa et al., 2017) discovered an attenuated MMNm amplitude among depressed patients. According to a meta-analysis by Tseng et al. (2021), only duration deviance MMN but not frequency deviance MMN amplitude was significantly altered in depressed patients compared to non-depressed controls. In the present work, findings regarding MMN responses elicited by some other stimulus types were also reviewed; for instance, Bissonnette et al. (2020) found an increased intensity deviance MMN amplitude in depressed patients, while Mu et al. (2016), Ruohonen and Astikainen (2017) and Ruohonen et al. (2020) did not find any differences in intensity deviance MMN between depressed patients and non-depressed controls. Regarding vowel and syllable deviance conditions, no differences in MMN amplitudes between depressed patients and controls were found (Lepistö et al., 2004; Takei et al., 2009).

MMN is associated with predictive coding theory, which is regarded

as a general information processing mechanism of the brain (Friston, 2005). Predictive coding mechanism has been suggested to be altered in patients with depressive disorder (for reviews, see Kube et al., 2020; Smith et al., 2021). However, the auditory MMN studies reviewed in this work showed inconsistent findings, varying in terms of deviating stimulus features. For example, duration MMN was usually attenuated, while frequency MMN was increased in amplitude in depressed patients, and the results of intensity deviance MMN were conflicting. The reason why the frequency deviance prediction error signal indexed by MMN is commonly increased while the duration deviance prediction error signal is attenuated is unknown and requires further studies in which both of these deviance types are investigated in the same subject groups.

In addition to variable stimulus features, there are also other methodological differences in auditory MMN studies investigating patients with depressive disorder. For instance, the locations of the electrodes applied in the analysis (fronto-central/temporal/midline) varied across the studies. In addition, both EEG and MEG were used in these studies. MMN research in schizophrenia patients is abundant, and the research paradigm has become standardised over the years; duration and frequency deviance oddball conditions have been used in most studies, and the findings of attenuated MMN amplitude have been consistently replicated (for meta-analyses, see Erickson et al., 2016; Umbricht and Krljes, 2005). In the future, it would be important to study MMN in depression by applying a more equal research methodology. Based on their meta-analysis, Tseng et al. (2021) suggested continuing MMN research by simultaneously applying both duration and frequency deviance conditions to investigate whether depressed patients show more impaired duration deviance MMN than frequency deviance MMN. Another interesting question related to stimulus types is whether MMN responses elicited by neutral auditory stimuli or those elicited by emotional auditory stimuli are better for differentiating between depressed patients and non-depressed control subjects. Because depression is associated with dysfunction in emotional information processing (e.g., Phillips et al., 2003), auditory MMN elicited by emotional stimuli may also be suitable to discriminate between depressed patients and controls. However, there was only one study in which auditory MMN elicited by emotional stimuli was investigated; in an MMN study by Pang et al. (2014), meaningless syllables were spoken with either neutral or emotional (happy, angry and sad) prosodies. Sad stimuli were processed differently by depressed patients and controls. Sad MMN was absent among depressed patients, while it was elicited in the non-depressed control group (Pang et al., 2014). Differences between depressed patients and non-depressed controls in the processing of emotional stimuli have also been found in early visual ERP (MMN, P1 and N170) studies, in which emotional face stimuli were utilised (Chang et al., 2010; Ruohonen et al., 2020; Wu et al., 2016; Xu et al., 2018; Zhang et al., 2016; Zhao et al., 2015) and in later visual ERPs reflecting various aspects of affective cognition (for reviews, see Hajcak and Foti, 2020; Kujawa and Burkhouse, 2017; Kujawa et al., 2020; Proudfit, 2015; Proudfit et al., 2015). More research comparing depressed and non-depressed groups in auditory MMN responses elicited by emotional stimuli is required.

In addition to different methodological issues, heterogeneity of depressive disorder diagnoses may contribute to the varying results of auditory MMN studies. In this review, for instance, first-episode depression (e.g., Chen et al., 2015; Qiao et al., 2013, 2015), lifetime depression in older adults (Naismith et al., 2012), treatment-resistant depression (TRD) (He et al., 2010) and melancholic depression (Chen et al., 2015) were investigated. In addition, comorbidities may be confounding factors in the studies that investigated MMN in depressive disorder. He et al. (2010) investigated MMN in patients with TRD, patients with borderline personality disorder (BPD), patients with TRD comorbid with BPD (TRD + BPD) and in control participants without TRD and BPD. The MMN amplitude was higher in the TRD group than in the other three groups, but there were no differences in MMN amplitude between the TRD + BPD group and controls or between the TRD + BPD group and the BPD-only group.

The use of antidepressant medication may contribute to the varying results of MMN studies. The medication condition is important to take into account in the studies that investigate MMN in patients with depressive disorder. In half of the reviewed MMN studies, depressed participants were on antidepressants. SSRIs are frequently used to treat depressive disorders. The serotonergic modulation of MMN amplitude has been investigated in depressed patients (e.g., Kuang et al., 2016) and in healthy participants (e.g., Ahveninen et al., 2002; Kähkönen et al., 2005; Leung et al., 2010; Oranje et al., 2008; Wienberg et al., 2010). Also, animal models have been utilised (e.g., Pan et al., 2020). However, these studies showed inconsistent results; in some, SSRIs increased MMN amplitude (Oranje et al., 2008; Wienberg et al., 2010), whereas in other studies, SSRIs decreased MMN amplitude (Pan et al., 2020). In the studies by Ahveninen et al. (2002) and Kähkönen et al. (2005), acute serotonin depletion led to increased MMN amplitude. It has also been suggested that the acute depletion of serotonin does not affect MMN (Leung et al., 2010). Regarding depressed participants, Kuang et al. (2016) showed that the pre-treatment latency of MMN was prolonged, but it decreased following the SSRI treatment. Antidepressants may be a confounding factor in studies investigating MMN in patients with depressive disorder. In future studies, it would be important to be able to distinguish the effects of antidepressants from the effects of depressive disorder on MMN. For instance, studies comparing unmedicated and medicated depressed patients would provide new information about this issue.

In some of the reviewed MMN studies, the relationship between MMN amplitude and the severity of depressive disorder was investigated. It seems that MMN amplitude is not related to depressive symptom severity (He et al., 2010; Hirakawa et al., 2017; Kähkönen et al., 2007; Mu et al., 2016; Naismith et al., 2012; Pang et al., 2014; Qiao et al., 2013, 2015; Takei et al., 2009; Umbricht et al., 2003). In the study by Naismith et al. (2012), patients with remitted depression demonstrated a reduced MMN amplitude, showing that the amplitude was not related to depressive symptom severity, and in a study by Bonetti et al. (2017), a relation between depression risk and MMN amplitude was found. These studies suggest that MMN may provide a trait marker of depression, but more research is still needed to confirm this, because in many studies, the conclusion was based on a null result (no correlation was found between severity and MMN amplitude). Future studies could use longitudinal designs to address the question of whether MMN is a state or trait marker of depression. This is an essential issue regarding the possible clinical use of MMN in depressive disorder diagnostics in the future.

Regarding the recurrence of depressive episodes, no correlation between MMN amplitude and the number of depressive episodes was found (Naismith et al., 2012). In addition, in the studies by Chen et al. (2015), Ruohonen and Astikainen (2017) and Umbricht et al. (2003), MMN amplitude values did not differ significantly between depressed patients with one episode of illness and those with multiple episodes. These results indicate that recurrence of depressive episodes may not cause an accumulation of impairment in the MMN amplitude.

MMN is suggested to reflect the functioning of NMDA receptors (e.g., Javitt et al., 1996; Umbricht et al., 2000, 2002), which has been suggested to be dysfunctional in depressive disorder (Adell, 2020; Inoshita et al., 2018; Sanacora et al., 2008). Therefore, in depressed patients, MMN may be an index of more general cognitive performance than auditory change detection alone (Näätänen et al., 2012, 2014). The reviewed studies showed that reduced MMN amplitude in depressed patients was associated with reduced semantic fluency and greater self-rated functional disability (Naismith et al., 2012), poor social functioning (Kim et al., 2020) and deficits in attention switching functions (Chen et al., 2015). In contrast, Ruohonen et al.'s (2020) study showed no relationship between MMN amplitude and cognitive performance, and Umbricht et al. (2003) showed MMN amplitude did not correlate with the intelligence quotient (IQ). Regarding MMN latency, Bissonnette

et al. (2020) showed that prolonged MMN latency following the duration deviant was associated with deficits in cognitive functions, such as working memory and attention.

4.3. LDAEP

LDAEP has been regarded as an indirect indicator of central serotonin function; a low LDAEP indicates high serotonergic activity and vice versa (Hegerl et al., 2001; Hegerl and Juckel, 1993). Since serotonergic dysfunction is suggested to be one of the major pathophysiological factors in depression (e.g., Coppen, 1967; Hasler, 2010; Kraus et al., 2017; Meltzer, 1990), it can be assumed that patients with depressive disorder and non-depressed control participants differ in LDAEP. However, studies comparing LDAEP between depressed patients and non-depressed controls have produced contradictory findings. Several studies have not found any differences in LDAEP between patients with depressive disorder and controls. However, an increased LDAEP in depressed patients compared to controls and attenuated LDAEP among depressed patients have been reported in a few studies. The inconsistent findings regarding LDAEP as a tool to distinguish between depressed patients and non-depressed controls may reflect the heterogeneity of depression diagnoses with multiple different symptom profiles and underlying neural mechanisms. Regarding the neurotransmitters, previous studies indicated that not only serotonin but also other neurotransmitters, especially other monoamines (noradrenaline and dopamine), contribute to the development and maintenance of depressive disorder (for reviews, see Belujon and Grace, 2017; Malhi et al., 2005; Moret and Briley, 2011; Shao and Zhu, 2020), and different subtypes of depression may differ in terms of the function of the neurotransmitters (Malhi et al., 2005). Depression is suggested to be related to functional interactions between neurotransmitter systems (Malhi et al., 2005).

Regarding subtypes of depressive disorder, melancholic depression and atypical depression have different and partly contrasting clinical symptoms. Melancholic depression is associated with a lack of reactivity of mood, psychomotor slowing, anxiety, appetite loss and insomnia, whereas atypical depression is a disorder with mood reactivity, metabolic abnormalities, increased appetite, hypersomnia, leaden paralysis and interpersonal rejection sensitivity (American Psychiatric Association, 2013). In addition to different symptom profiles, melancholic depression and atypical depression seem to have opposite characteristics in terms of LDAEP strength. Fitzgerald et al. (2009) found that the melancholic depression group had a weaker LDAEP than the non-depressed controls, whereas no difference in LDAEP existed between the non-melancholic group and control group. Lee et al. (2014) showed that patients with atypical depression had stronger LDAEP than those with non-atypical depression, which may suggest that there is deficient serotonergic activity in patients with atypical depression. This is in line with the findings that atypical depression symptoms, such as mood, sleep and appetite disturbances, are related to serotonergic dysfunction (Malhi et al., 2005). On the contrary, melancholic depression patients may have abundant serotonergic activity, as indicated by a weaker LDAEP. Several neurotransmitter systems, including dopaminergic and noradrenergic systems, are involved in melancholic depression (Malhi et al., 2005), and melancholic depression symptoms (e.g., psychomotor slowing, anhedonia and disturbances in drive, energy and volition) are attributable to dopamine and noradrenaline dysfunction (Malhi et al., 2005). According to Fitzgerald et al. (2009), noradrenergic abnormalities may explain an increase in serotonin tone in melancholic depression patients. The aforementioned studies by Fitzgerald et al. (2009) and Lee et al. (2014) suggest that melancholic and atypical depression may have different biological bases that could be separated utilising LDAEP. However, to confirm this, studies that directly compare LDAEP between melancholic depression and atypical depression are needed.

To the best of our knowledge, no studies have compared LDAEP between different depression severity groups, but the relationship between LDAEP and depression severity has been studied. The findings regarding the correlation between LDAEP and depression severity have been inconsistent (no correlation: Chen et al., 2005; Gallinat et al., 2000; Park et al., 2010; a trend toward a negative correlation: Fitzgerald et al., 2009; Jaworska et al., 2012; a negative correlation; Park and Lee, 2013; a positive correlation: Kim et al., 2019; Ostermann et al., 2012). More research on the relationship between LDAEP and depression severity is needed.

Numerous studies have investigated the relationship between LDAEP and suicidality in patients with depressive disorder (for a review, see Park, 2015) because serotonergic functions are suggested to be associated with suicidal behaviour (e.g., Miller et al., 2013; for a review, see Mann, 2013). A study by Graßnickel et al. (2015) showed no differences in LDAEP between suicidal depressed patients and non-suicidal depressed patients. In addition, in the study by Park et al. (2014), LDAEP did not differ between depressed patients with suicidal ideation and those without suicide ideation. Kim et al. (2021) demonstrated that there was no difference in LDAEP between depressed patients with suicide attempts and those with suicidal ideation. Some studies also found a sharper slope of LDAEP in depressed patients with suicidality compared to patients without suicidality (e.g., Chen et al., 2005; Hwang et al., 2021; Kim and Park, 2013; Uhl et al., 2012;). In contrast, in the study by Lee et al. (2014), a negative correlation between the LDAEP value and the suicidal ideation score was found in patients with atypical depression. Thus, the findings regarding the relationship between suicidality and LDAEP are highly inconsistent. The heterogeneity regarding the subtypes of depression and regarding how suicidality is defined (e.g., acute suicide attempts/suicide ideation) may explain the variation in the findings.

Similar to P3 and MMN studies, in studies that investigated LDAEP in patients with depressive disorder, the comorbid factors must be considered. For instance, Kim et al. (2019) demonstrated that LDAEP was attenuated in the depressed patients with ADHD symptoms compared to depressed patients without ADHD symptoms and controls without depression and ADHD symptoms, but there were no differences between the depressed patients without ADHD symptoms and controls. Manjarrez-Gutierrez et al. (2009) showed that LDAEP was increased in the patients with depression comorbid with diabetes compared to the control group without depression and diabetes, depression-only group and diabetes-only group, whereas there was no difference between the depression-only group and controls. This finding is in line with the suggestion that a deficit of serotonin is related to both the diabetic state (for a review, see Prabhakar et al., 2015) and depressive disorder (e.g., Coppen, 1967; Hasler, 2010; Kraus et al., 2017; Meltzer, 1990). The comorbidity of diabetes and depressive disorder may cause an accumulation of increase in the LDAEP.

Even if it seems that LDAEP cannot be used as a tool to differentiate between depressed patients and non-depressed controls, certain features in depressive disorder that are affected by a potentially higher degree of dysfunction in serotonergic functions, such as melancholy (e.g., Fitzgerald et al., 2009) and suicidality (e.g., Chen et al., 2005; Kim and Park, 2013), might be associated with LDAEP modulations. In the future, LDAEP can be investigated as a potential tool for identifying biologically different subgroups of depressive disorder. However, conclusions regarding the relationship between LDAEP and serotonin-related depressive symptoms and subtypes should be made cautiously. Most of the evidence regarding the association between LDAEP and serotonin came from animal studies (e.g., Juckel et al., 1997, 1999). Instead, studies investigating humans have provided more inconsistent findings (e.g., Debener et al., 2002h; Dierks et al., 1999; Guille et al., 2008; Kähkönen et al., 2002; Nathan et al., 2006; Segrave et al., 2006; Simmons et al., 2011; Uhl et al., 2006). Furthermore, in addition to serotonin, LDAEP is suggested to be sensitive to some other neurotransmitter systems (e.g., glutamatergic system; O'Neill et al., 2007). Thus, more research on the relationship between LDAEP and serotonin in healthy humans, as well as on LDAEP in different subgroups of depressive disorder, is needed.

In the reviewed LDAEP studies, some potential confounding factors were related to the heterogeneity of the samples. They could partly explain the inconsistent findings in the differences in LDAEP between depressed patients and non-depressed controls. An essential potential confounder is the use of antidepressants, especially SSRIs, because LDAEP is suggested to reflect central serotonergic functions (e.g., Hegerl et al., 2001). Even if there is growing evidence of LDAEP as a predictor of SSRI treatment response (e.g., Gallinat et al., 2000; Jaworska et al., 2013; Lee et al., 2005, 2015; Mulert et al., 2002; Paige et al., 1994; for a review, see e.g., Leuchter et al., 2009; for a meta-analysis, see Yoon et al., 2021), the findings of the effects of SSRIs on LDAEP are contradictory. Some studies showed no difference between pre-treatment LDAEP and post-treatment LDAEP in depressed patients (Gallinat et al., 2000; Linka et al., 2009; Paige et al., 1994). Furthermore, Gopal et al. (2004) showed that no difference in LDAEP was found between SSRI medicated and unmedicated depressed patients, and Ostermann et al. (2012) found no differences in LDAEP between depressed patients treated with SSRIs and those treated with other antidepressants. Regarding healthy adults, neither an acute administration of SSRIs (Guille et al., 2008; Uhl et al., 2006) nor an acute depletion of serotonin (Debener et al., 2002h; Dierks et al., 1999) had an impact on LDAEP. However, in a few studies investigating healthy adults, decreased LDAEP after the administration of a single dose of the SSRI (Nathan et al., 2006; Segrave et al., 2006) and chronic SSRI administration (Simmons et al., 2011) was found. Thus, more research addressing the effects of SSRIs on LDAEP is needed.

Regarding methodological issues, the procedure for measuring LDAEP varied between the studies reviewed in this work. In most studies, LDAEP was defined as an amplitude change in the auditory N1/P2 component in response to different stimulus intensities. However, there are also other ways to investigate LDAEP. For instance, in addition to N1/P2 amplitude change, amplitude change in N1, P1, P2, and P1/N1 components in response to different stimulus intensities have also been studied (Jaworska et al., 2012; Linka et al., 2009; Ruohonen et al., 2020). In the study by Gopal et al. (2004), amplitude change in response to different stimulus intensities in the brainstem ERP was also investigated. Linka et al. (2009) found associations between the LDAEP amplitude slopes and somatic symptoms of depression for N1 but not for P1/N1 nor N1/P2. Thus, the results may vary for different ERP components. Additionally, the stimulus intensities varied between the studies. However, in the reviewed studies, no systematic differences in the parameters in the study design were identified between the studies in which no between-group differences were found and the studies in which some differences between patients with depressive disorder and non-depressed controls were reported. In future research, it would be important to manipulate different parameters in LDAEP study designs to find the most effective method to separate depressed patients and controls.

5. Conclusion, limitations and future directions

This narrative review addressed the first step into researching possible future biomarkers for depressive disorder, focusing on sensitivity by investigating whether auditory P3, MMN and LDAEP components could be used to separate depressed and non-depressed participant groups. The results showed that the findings of the reviewed studies were inconsistent, but some patterns could be identified. For auditory P3b, the common finding was attenuated amplitude and prolonged latency among depressed patients. Regarding auditory MMN, especially the amplitude of duration deviance MMN was commonly attenuated, and the amplitude of frequency deviance MMN was increased in depressed patients. In LDAEP studies, commonly, no differences between depressed patients and non-depressed controls were reported, although group differences concerning specific depression subtypes were found. Still, the inconsistency in the results of the reviewed studies shows that, in general, these auditory ERPs do not have a high sensitivity in distinguishing the presence or absence of a depressive disorder.

The narrative approach enabled us to review the findings of a large range of methodologically different studies on auditory P3, MMN and LDAEP alterations in patients with depressive disorder, presenting an overall picture of the current knowledge. The broad focus of a narrative review is a strength that allows for comprehensive coverage of a topic, providing the foundation for future research. In the present review, the literature search performed using systematic selection criteria in a PubMed database yielded studies from four decades, from 1981 to 2021. However, the comprehensive approach of narrative review is also a weakness. It is obvious that compared to meta-analyses in which pooled data can be analysed statistically, narrative reviews that cannot provide a quantitative synthesis of the literature are a much less objective research design. Thus, even if differences in auditory ERPs between depressed and non-depressed groups were recognised, the magnitude of the differences is not known. Regarding research on possible future biomarkers for depressive disorder, the effect sizes are of high importance. Hence, the rigour of meta-analyses is needed to synthesise the results of studies on auditory ERP biomarkers for depression. Based on the results of the present narrative review, meta-analytic research could be continued by focusing on narrower topics.

This narrative review indicated that there are some methodological issues that should be considered in future studies on auditory P3, MMN and LDAEP in patients with depressive disorder. In many of the reviewed studies, the sample sizes were relatively small and not estimated to ensure the sufficient power of the study. There was also a lack of standardisation regarding experimental paradigms. Regarding auditory MMN studies investigating depressive disorder, duration deviance and frequency deviance oddball conditions were most often applied. Replications of these studies are still needed to draw clear conclusions regarding a suitable experimental paradigm. Additionally, in future studies on auditory MMN, as well as P3b and LDAEP, it would be important to manipulate different parameters (e.g., stimulus parameters) in experimental designs to find the best options for studies investigating differences between depressed patients and controls. Additionally, the inclusion criteria for depressed patients differed between the reviewed studies, for instance, regarding the use of antidepressant medication, the stage of illness and the subtypes of depressive disorder. These differences may partly explain the inconsistent results regarding auditory P3, MMN and LDAEP in depressive disorder in the present narrative review. Standardisation of methodological issues and inclusion criteria, as well as replications of the studies, would better enable a meta-analytic research approach in the future.

This narrative review yielded some conclusions that provide suggestions for future studies. P3b is the most frequently studied auditory ERP component in depressed patients. In the reviewed studies, attenuated auditory P3b amplitude and prolonged P3b latency among depressed patients were common findings. However, in P3b studies, and especially in LDAEP studies, the findings were more consistent when investigating some subgroups of depressive disorder instead of a heterogeneous group of depressed patients. This suggests that it would be important to study auditory ERPs systematically in clinical subgroups rather than in heterogeneous samples of depressed patients.

Furthermore, comorbid factors are important to consider. For instance, anxiety disorders and anxiety symptoms are common among depressive disorder patients. In future studies, in addition to excluding depressed patients with a comorbid anxiety disorder, it would be important to control for the effect of anxiety symptoms in the analysis.

Regarding the possible clinical utility of auditory P3, MMN and LDAEP as tools to differentiate between depressed patients and non-depressed subjects in the future, the question of whether these ERPs could be conceptualised as a trait or state marker of depressive disorder is essential. Longitudinal designs with multiple assessments could be useful to address this issue, but these kinds of studies are still very rare (for visual ERPs, see Ruohonen et al., 2020). Another essential question regarding clinical utility is related to the specificity of possible future

auditory ERP biomarkers; it would be important to investigate whether they could differentiate depressive disorder from other psychiatric disorders. For instance, while P3b is commonly found to be attenuated and prolonged in depression, these P3b findings may not be specific to depression (e.g., Blackwood et al., 1987; Roth et al., 1981; Wagner et al., 1997; Weir et al., 1998; Xu et al., 2014). Currently, research comparing auditory ERPs between depressive disorders and other psychiatric conditions is quite rare, and the findings are variable (e.g., Blackwood et al., 1987; Bruder et al., 2002; He et al., 2010; Kaur et al., 2012; Kim and Park, 2020; Kim et al., 2020; Li et al., 2011; Muir et al., 1991; Park and Lee, 2013; Röschke et al., 1996; Roth et al., 1981; Shim et al., 2019; Umbricht et al., 2003; Wagner et al., 1997; Xu et al., 2014). For this reason, more research is required.

The present review focused on auditory ERPs. In the future, it would be important to study ERP alterations in different sensory modalities in patients with depressive disorder to determine which sensory modality can best separate depressed and non-depressed groups. Regarding the visual modality, there is already growing literature indicating that visual ERPs can differentiate between depressed and non-depressed groups (for reviews, see Hajcak and Foti, 2020; Kujawa and Burkhouse, 2017; Kujawa et al., 2020; Proudfit, 2015; Proudfit et al., 2015). In addition, an essential question would be which sensory modality is the most suitable for studies comparing ERPs between groups of depressive disorder patients and groups of patients with other psychiatric disorders.

Furthermore, depressive disorder is associated with dysfunction in emotional processing (for reviews, see Bylsma, 2021; Phillips et al., 2003). However, the alterations in the information processing are usually more diverse in depressed patients (for a meta-analysis, Rock et al., 2014). The findings of the present narrative review suggest alterations in the basic sensory-cognitive information processing by showing some differences between depressed and non-depressed groups in the auditory ERPs elicited by neutral stimuli. Thus, not only emotional information processing but also the processing of basic sensory information may be altered in depressed patients. Future studies should investigate ERPs elicited by both emotional and neutral stimuli to find out whether ERP components reflecting emotional and basic sensory information processing are differently related to depressive symptoms or symptom profiles. An important question would be whether these ERP components together could have utility in separating subgroups of depressive disorder and developing more personalised approaches for treatment in the future. In the studies reviewed in the present work, ERPs reflecting sensory-cognitive processing were elicited mostly by neutral auditory stimuli. In only one ERP study (Pang et al., 2014), emotional stimuli were applied to elicit the auditory MMN component. Regarding the visual modality, ERP research investigating affective cognition in depressive disorder is already abundant; previous studies have shown that visual ERPs reflecting emotional, motivational and reward processing can differentiate between depressed and non-depressed groups (for a review, see e.g., Proudfit et al., 2015). Auditory ERPs elicited by emotional stimuli might also differ between depressed patients and non-depressed controls. Hence, research on auditory ERPs in depressed patients conducted by applying emotional stimuli is required.

In sum, the conclusions drawn from the findings of the studies reviewed in this work suggest that future research should investigate whether certain stimulus conditions are particularly efficient at separating depressed and non-depressed participant groups. Future studies should also compare responses in different subpopulations of depressed patients and different clinical groups (e.g., depressive disorder and anxiety disorder patients) to investigate the specificity of the auditory ERP alterations for depressive disorder.

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Declaration of competing interest

The authors declare no conflict of interest.

Data availability

No data was used for the research described in the article.

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