World Journal of

Psychiatry

World Journal of **Psychiatry**

Contents

Contents

ABOUT COVER

Editorial Board Member of *World Journal of Psychiatry*, Peer Reviewer, Alexander M Ponizovsky, MD, PhD, Mental Health Research Adviser, Professor, Department of Mental Health Services, Ministry of Health, Jerusalem 9446724, Israel. alexander.ponizovsky@moh.gov.il

AIMS AND SCOPE

The primary aim of *World Journal of Psychiatry* (*WJP*, *World J Psychiatr*) is to provide scholars and readers from various fields of psychiatry with a platform to publish high-quality basic and clinical research articles and communicate their research findings online.

  WJP mainly publishes articles reporting research results and findings obtained in the field of psychiatry and covering a wide range of topics including adolescent psychiatry, biological psychiatry, child psychiatry, community psychiatry, ethnopsychology, psychoanalysis, psychosomatic medicine, *etc*.

INDEXING/ABSTRACTING

The *WJP* is now abstracted and indexed in Science Citation Index Expanded (SCIE, also known as SciSearch®), Current Contents/Clinical Medicine, Journal Citation Reports/Science Edition, PubMed, and PubMed Central. The 2020 edition of Journal Citation Reports® cites the 2019 impact factor (IF) for *WJP* as 3.545; IF without journal self cites: 3.545; Ranking: 46 among 155 journals in psychiatry; and Quartile category: Q2.

RESPONSIBLE EDITORS FOR THIS ISSUE

OPINION REVIEW

Use of cognitive event-related potentials in the management of psychiatric disorders: Towards an individual follow-up and multi-component clinical approach

Salvatore Campanella

Salvatore Campanella, Laboratoire de Psychologie Médicale et d'Addictologie, ULB Neuroscience Institute (UNI), CHU Brugmann-Université Libre de Bruxelles (U.L.B.), Brussels 1020, Belgium

Corresponding author: Salvatore Campanella, PhD, Senior Research Associate FNRS, Laboratoire de Psychologie Médicale et d'Addictologie, ULB Neuroscience Institute (UNI), CHU Brugmann-Université Libre de Bruxelles (U.L.B.), 4, Place Vangehuchten, Secrétariat de Psychiatrie, Brussels 1020, Belgium. salvatore.campanella@chu-brugmann.be

Abstract

Relapse prevention remains a major challenge in psychiatry, thus indicating that the established treatment methods combining psychotherapy with neuropharmacological interventions are not entirely effective. In recent years, several intervention strategies have been devised that are aimed at improving psychiatric treatment by providing a complementary set of add-on tools that can be used by clinicians to improve current patient assessment. Among these, cognitive event-related potentials (ERPs) have been indexed as valuable biomarkers of the pathophysiological mechanisms of various mental illnesses. However, despite decades of research, their clinical utility is still controversial and a matter of debate. In this opinion review, I present the main arguments supporting the use of cognitive ERPs in the management of psychiatric disorders, stressing why it is currently still not the case despite the vast number of ERP studies to date. I also propose a clinically-oriented suitable way in which this technique could — in my opinion — be effectively incorporated into individual patient care by promotion of the use of individual ERP test-retest sessions and the use of a multi-component approach.

Key Words: Event-related potentials; Psychiatry; Cognitive disorders; Follow-up; Multi-component approach; Personalized medicine

Core Tip: Despite decades of intense research and many promising results, cognitive

event-related potentials (ERPs) have yet to be implemented in daily psychiatric care units as an add-on tool to psychotherapy and medication. I present here the main arguments supporting the notion that ERPs represent a highly suitable tool for performing individual "neuro-cognitive" assessments in psychiatric patients. Such ERP data could help clinicians to specify individual cognitive interventions that will target each patient's specific needs, thus promoting an "individualized" or "personalized" medicine.

INTRODUCTION

The 1990s have been referred to as the "Decade of the Brain", with developments such as brain imaging tools allowing patterns of distributed neural activity associated with both normal and pathological behaviors to be identified[1]. On this basis, major mental illnesses, such as schizophrenia, autism, major depression, anxiety disorders, and addictions, were redefined as brain diseases[2], with a deep impact of the environment at both the social and physical levels[1]. Nowadays, the separation of neurology and psychiatry appears arbitrary, and in the framework of modern neuroscience, psychiatrists and neurologists could be called "clinical neuroscientists" who apply neuroscientific discoveries to the care of patients with brain disorders[3]. Clearly, the management of a mentally ill patient necessarily requires consideration (mainly through psychotherapy) of a single individual embedded in a specific social-cultural context in order to encompass the social and psychogenic aspects of individual clinical symptoms[4]. However, mapping the live brain activity of a patient, by—for instance—the use of positron emission tomography (PET), magnetic resonance imaging (MRI), or electroencephalography (EEG), has demonstrated that all normal or dysfunctional mental processes are ultimately biological[2]. It is, therefore, also important to consider these cognitive, emotional, and social processes, subtended by specific choreographed patterns of brain activity that — when dysfunctional — can mediate the onset and persistence of specific clinical symptoms[5]. For instance, an alteration of mental state attribution, the ability to infer mental states of others in order to guide social interactions, is classically observed in schizophrenic patients[6]. This deficit, mainly subtended by neural alterations in the prefrontal cortex and the superior temporal sulcus[7], is associated with a poor outcome, social functioning, and social competence in schizophrenia[8]. As antipsychotic medication has been shown to have a limited impact on recovery and social cognition[9], the challenge is to develop new therapeutic strategies to specifically improve social cognition in schizophrenia. Promising results, revealing improvement of social functioning and reduction of psychotic symptoms, have been achieved through social cognition training programs and psychosocial interventions[10] as well as by brain stimulation through transcranial direct-current stimulation (tDCS)[11] or transcranial magnetic stimulation[12]. Similarly, in major depressive disorder, deficits in cognitive inhibition, subtended by a hyperactivated amygdala insufficiently controlled by a hypoactivated prefrontal region[13] appear to be a main causal factor for ruminations[14]. Sessions of training to inhibit negative thoughts[15] as well as a combined tDCS-mindfulness program resulted in a decrease in ruminations and lower depressive scores[16], while medication only has been reported to provide modest improvements[17]. In alcohol dependence, an increased salience of alcohol-related cues grasping drinkers' attention (hyperactivated mesolimbic activity[18]) combined with a lack of inhibitory resources (anterior cingulate and frontal hypoactivity[19]) defines the main neurocognitive mechanisms triggering relapse[20]. In light of the modest effect of anti-craving medications[21], cognitive bias modification training alone[22] or combined with tDCS[23] has shown promising trend on treatment outcomes by reducing craving and by improving early abstinence. Overall, convergent empirical data illustrating alterations in brain networks that underlie cognitive impairments have provided

foundational information about transdiagnostic circuits and promising targets for intervention[24]. Indeed, numerous studies have provided consistent evidence that mental illness involves significant cognitive impairments that represent valid therapeutic targets, as enhancing cognitive functioning leads to a reduction of clinical symptoms and a better quality of life. One of the main challenges at present consists of developing new ways to use neurocognitive mechanisms as an add-on tool in the clinical and conventional management of psychiatric patients.

Due to their high anatomical resolution, PET and functional MRI (fMRI) clearly constitute the most suitable tools to assess the distributed brain networks involved in diverse cognitive functions[25]. However, their coarse temporal resolution (1-2 s) does not allow definition of the temporal activation sequence, thus preventing isolation of the series of individual sensory, cognitive, affective, and motor processes that occur between a stimulus and a response[26,27]. Electrophysiological tools, by recording spontaneous electrical brain activity from multiple electrodes placed over the scalp[28], are more suitable for this purpose due to their optimal temporal resolution on the order of milliseconds[29]. EEG is an inexpensive and non-invasive tool defining a valuable clinical first-line method to exclude a diagnosis of epilepsy, drug intoxication, or sleep disorders in psychiatric patients[30]. A derivative of the EEG technique refers to event-related potentials (ERPs), *i.e.*, averaged EEG responses that are time-locked to the cognitive processing of stimuli. The past several decades have witnessed a vast number of task-dependent ERP components being described and studied among healthy people. While studies on healthy participants have helped to define the various cognitive steps needed throughout the entire information processing stream to achieve a cognitive function, such data also have great relevance in pathology. Indeed, by accessing the various cognitive steps needed to achieve a cognitive function, cognitive ERPs may then also allow definition of where a dysfunctional behavior originates at the cognitive level. This has great clinical relevance, as a similar altered behavior may be subtended by various cognitive disorders[31]. Therefore, by indexing the specific neurocognitive functions that are dysfunctional in a patient, ERPs pinpoint cognitive functions that should be rehabilitated in each patient through specific and individualized cognitive remediation procedures[26]. However, despite a solid theoretical basis[29] and decades of research showing alterations of these components in various psychiatric diseases[32], their relevance in clinical settings is still a matter of debate[33]. The scope of this paper is not to provide an exhaustive review of the literature regarding ERPs in various psychiatric diseases. Rather, my aim is to present relevant arguments supporting the notion that it is important to incorporate the use of cognitive ERPs in the management of psychiatric disorders, by also stressing why it is still not the case nowadays despite thousands of ERP studies to date. I then propose a clinically-oriented suitable way in which this technique could—in my opinion—be effectively incorporated into individual patient care.

DECADES OF ERP STUDIES IN PSYCHIATRY: WHY SO MANY HOPES AND PROMISING RESULTS FOR SUCH A MINOR CLINICAL IMPACT TO DATE?

Depending on the cognitive task one is confronted with and the cognitive processes one is focusing on, several ERP components have been described in recent decades in the literature. The P50, the contingent negative variation (CNV), the mismatch negativity (MMN), the P300 with its P3a and P3b subcomponents, the No-go N2 and No-go P3, the error-related negativity (ERN), and the N400 are some of the most studied ERPs. When elicited through a specific task in healthy subjects, such ERPs are the neural correlates that assess the efficiency of diverse cognitive processes, such as sensory gating[34], arousal and motor preparation[35], auditory discrimination[36], novelty processing *vs* decision making[37], cognitive and motor inhibition[38], insight[39], and semantic congruency[40]. As ERP amplitudes reflect differences in the intensity of responses whereas measurements of latency inform regarding the processing time duration[40,41], several anomalies in amplitude and/or latency of these components have been reported in various psychiatric disorders[32,42]. In such a view, ERPs could characterize biological markers of pathophysiological mechanisms[43]. Such biological markers can be state (only present during the acutely ill state but stabilized after remission) or trait (always present, during and after the disease) markers[44,45]. On the one hand, by reflecting pathophysiological processes

that are active during the disease, state markers could provide clinicians important input to assist with choosing the most appropriate treatment. For instance, a decreased amplitude of the P3b component is considered to be a state marker of depression[46]: The P3b amplitude has been shown to be increased after four weeks of antidepressant treatment[47] as well as following recovery from electroconvulsive therapy[48]. Similarly, chronic schizophrenic patients exhibit reduced MMN amplitudes compared to healthy controls[49], and antipsychotics such as aripiprazole[50] or drugs acting on the NMDA (N-Methyl-D-aspartate) receptor[51] appear to induce its recovery, while the CNV is reduced in amplitude in children[52] and adults[53] with ADHD but has been shown to exhibit an amplitude recovery with even just a single dose of stimulant medication[54]. Such instances clearly highlight how ERP state markers could be particularly useful in monitoring the efficiency of a treatment. On the other hand, trait markers can also be particularly useful, mainly as indicators of vulnerability[45]. Indeed, the amplitude reduction of the P3b as well as the absence of P50 suppression in schizophrenia[55], an enhanced ERN in child and adult anxiety disorders[56], and an altered P3 component in cocaine users[57] are examples of trait markers, indexing during and after the disease, of impaired cognitive functions that play a pivotal role in the onset and persistence of these mental diseases. But importantly, such alterations can also define a risk marker for healthy people with, for instance, a positive family history to further develop it[58-60]. Therefore, such markers, if present, can improve early detection of illness, and, as such, facilitate more effective and targeted interventions[44].

Overall, state and trait ERP markers can serve to aid diagnosis (as prognostic elements[61]), assist in choosing the most appropriate treatment for psychiatric disorders[62], and help with detecting illness at an early phase[63]. However, although such empirical data, even in meta-analyses[64-68], may appear convincing and useful, the reality is quite different as, up to now, the utility of cognitive ERPs in daily clinical care settings remains (very) modest[30]. Several explanations may account for this situation. First, at a technical level, the worldwide current ERP screening procedure favours the huge number of ERP studies, despite the heterogeneity of the data. For instance, an amplitude reduction and/or a delayed latency of the P3b and the MMN is usually considered to be a state marker of depression[69] and early psychosis[70], respectively. However, contradictory data have also been reported, suggesting no P3b and MMN differences between depressive[48] and psychotic[71] patients, respectively, with controls. Such heterogeneity has understandably led to a degree of scepticism among clinicians as it raises questions regarding reliability. The main factors accounting for these discrepancies are the clinical subtypes of patients included in these studies, with comorbidities inducing particular responses[72], higher artifact contamination in clinical patients than in typical subjects[27], a potential influence of medication[27], and differences in ERP recording protocols leading to data misinterpretations[73]. In this respect, a very interesting initiative, called "ERP CORE" (Compendium of Open Resources and Experiments)[74], has recently been launched in order to provide standardized ERP paradigms for seven widely used ERP components (N170, MMN, N2pc, N400, P3, lateralized readiness potential, and ERN). By providing researchers with a useful tool and guidelines for selected tasks to record specific ERP components, this will notably promote the possibility of comparing ERP data sets from different laboratories. At a clinical level, such guidelines already exist[75]; however, their use in studies around the world is still by no means ensured, and this could notably lead to a degree of misinterpretation of the data[73]. It is, therefore, urgent to again underscore that using such guidelines accepted by the field would clearly help with clinical implementation[76] by providing access to normative data gathered on large samples in order to follow the progression of patients as a function of the treatment, but also to control for potential confounding factors, such as gender, age, medication, and comorbid symptoms. Secondly, at a conceptual level, the potential role of ERP components in the management of mental disorders has also suffered from the predominance in psychiatry of the official nosological systems, such as the Diagnostic and Statistical Manual of Mental Disorders (DSM) and the International Classification of Diseases (ICD)[77]. In such a categorical view, patients either do or do not meet the criteria to be diagnosed with a mental illness, thus suggesting that the presence of a specific cluster of symptoms will necessarily correspond to a specific mental disease (*e.g.*, borderline personality disorder). On this basis, the psychiatrist will select what they consider the best option of treatment among the range of those appropriate for that specific diagnosis (*e.g.*, selective serotonin reuptake inhibitors; SSRIs). If this treatment proves to be ineffective, they will then have to choose another option (*e.g.*, combination of SSRIs and quetiapine[78]) or reconsider the former diagnosis[79]. A main and crucial point that makes this categorical approach still

dominant in psychiatry is that it greatly facilitates clinical communication among mental health practitioners, as all textbooks and practice guidelines have been developed based on these categories[80]. In such a categorical view, mental illnesses are discontinuous entities with distinct symptoms, etiologies, and biomarkers[81]. However, inter-individual variability in the severity of symptoms among patients receiving a similar diagnosis, common symptoms across different disorders, and an extremely usual comorbidity factor are well-documented facts in clinical experience[77]. In other words, common liabilities across these "categories"[81,82] are more suggestive of a dimensional underlying cross-cutting transdiagnostic structure for mental disorders[77]. This still ongoing debate regarding the transdiagnostic *vs* the categorical frameworks to guide psychopathology assessments[83] is not the scope of this paper. However, it clearly appears that, due to the dominance of the nosological approach in psychiatry, the imprecision of categorical psychiatric diagnoses[84] has been a limiting factor in understanding the pathophysiological mechanisms of human behavioral abnormalities. Indeed, although decades of research have provided evidence of the relevance of various ERP components as biological markers of mental illnesses[32,43], their clinical sensitivity has been hampered by the fact that their parameters (*e.g.*, amplitude and latency) are diagnostically unspecific[33]. In other words, ERP deficits are a common feature of several psychiatric afflictions, but they will not assist clinicians in deciding whether a given patient is depressed, paranoid, or an alcoholic (high sensitivity but low specificity). Moreover, cognition is not considered as a primary treatment target, being still envisaged as a particular category of symptoms (among others), and not as a core phenomenon triggering the onset and/or the persistence of the disease. Many psychiatrists still then focused on finding the best suited drugs combination in order to contain symptoms et minimize side effects. Therefore, the approach using ERPs to classify patients according to DSM categories was entirely inappropriate, and, in the next section, I will specify how I think ERPs may be genuinely useful in clinical settings, mainly as predictive biomarkers, *i.e.*, as measured indices that may be used to predict clinical responses to treatment[85]. Finally, at a clinical level, a major practical issue is that the majority of ERP-based studies compare their results with matched controls using grand-averaged data. While such "group results" have ample merit at a fundamental "research" level, there is now a need for more "individualized", "personalized" medicine[86], *i.e.*, individual data that helps with devising interventions that are specifically targeted based on each patient's needs[26]. In the next section, I will try to provide some insights on how ERPs may be used effectively in clinical settings as an individual monitoring tool to reveal (or not) expected changes in brain function in response to a treatment[87].

USING ERPS IN PSYCHIATRY: WHAT PERSPECTIVES?

In an influential paper published in 1991 arguing for the use of PET in clinical care[88], Wagner[88] stated that: "…Today's medical practice is yesterday's research. The bridge linking the two is technology assessment, which makes possible the acceptance or rejection of new technologies in the practice of medicine. Experience is the key determinant of effectiveness. If the information provided is not useful to the physician caring for the patient, the procedure will eventually fall by the wayside…". The notion that ERPs are useful for managing psychiatric diseases, such as, for instance, depression[89], alcohol disorders[90], or schizophrenia[91], is not novel at all. However, despite decades of research, ERPs have yet to be implemented in the clinical management of mental illnesses[27]. It clearly appears at present that the clinical value of ERP components as a diagnostic index is low[33], merely reflecting a common measure of brain dysfunction[92]. With this in mind, it is, therefore, now urgent to precisely define what constitutes be the best use of ERPs in the management of psychiatric disorders. In order words, there is a need to find out which properties of ERPs as a tool could be the best-suited ones to help with managing a currently still unsolved clinical question.

A prominent issue in the treatment of mental illnesses relates to the relapse rate[93], which is approximately 50% at 1 year and 70% at 5 years for manic episodes in bipolar disorders[94]; approximately 35% at 18 mo and 74% at 5 years following a first episode of schizophrenia[95]; and approximately 50% at 3 mo and 85% at 1 year for recently detoxified alcoholic patients[96]. Despite the beneficial impact of psychotherapy and neuropharmacological interventions, as well as the positive effect of more recent intervention strategies such as multisystemic[97], cognitive behavioral[98], or mindful-

ness[99] therapies, the relapse rate is still extraordinarily high. Clearly, the idea is not at all to discredit the existing treatment methods, but providing a complementary set of add-on tools to be used by clinicians to improve current patient assessments is still a major challenge. Starting with the fact that mental diseases are also brain disorders, a neurocognitive approach has emerged[100]. This can be summarized as follows: (1) Mental illness involves significant cognitive impairment[101]; (2) These cognitive alterations, subtended by perturbed brain networks, may trigger the onset and/or persistence of clinical symptoms, thereby defining valid therapeutic targets[24]; and (3) Retraining these cognitive functions (through the use of cognitive retraining programs and/or neuromodulation tools) has been reported to reduce clinical symptoms and to enhance patient quality of life[11,16,22,23]. Overall, once admitted to a psychiatric care unit, there is still a huge difficulty with assessing "partial recovery", which allows patients to leave the hospital to return home, and achievement of long-term "complete clinical recovery", defined as the reduction of psychiatric symptoms and functional disabilities[102].

In my opinion, it is in this regard that ERPs may have an important clinical role in the management of psychiatric patients, as a monitoring and a predictive biomarker tool. Indeed, psychiatric evaluations are made almost entirely on the basis of clinical symptoms, and the longitudinal course is determined by a clinician speaking with the patient and informants (as well as sometimes by the use of clinical scales), but diagnostic frameworks do not usually incorporate biomarkers[103]. However, once the notion that mental illnesses are subtended by impaired neural functioning is acknowledged, a main assumption is to consider that this dysfunctional brain should undergo significant and enduring neural changes in order to be reflected in medium-to-long-term real-world behavioral modifications[104]. Indeed, test-retest ERP studies on healthy participants have, for instance, shown that subjects exhibiting improvements in inhibitory performance in a Go/No-go task had a similar residual gain in inhibition one week after post-training, albeit only when this effect was neurophysiologically indexed by faster Nogo-N2 latencies[105]. Accordingly, in neuropsychiatric conditions, several studies found that specific cognitive gains induced specific brain changes that were positively associated with decreased symptoms and better quality of life, even 6 mo later[106-108]. Taking these neural modifications into account would, therefore, be of the greatest clinical relevance, as their absence would suggest a high vulnerability to relapse. In this view, state ERP markers provide the possibility (1) to monitor the change, triggered spontaneously or by a specific treatment, in neurocognitive mechanisms that are involved in the onset and maintenance of clinical symptoms in an individual patient; and (2) to verify whether these neural changes induced by the treatment are predictive of the clinical trajectory. By fostering a longitudinal follow-up and intra-individual ERP approach, recent works have tried to verify the clinical utility of such designs, in which ERP measurements are included just as one would include measurements of symptoms. In a study by our laboratory of alcohol-dependent patients undergoing a four-week detoxification program[109], we showed that monitoring the changes in dual-processes that are well-known to trigger addictions (No-go P3 for inhibition[110] and P3 for cue reactivity[111]), at the start and at the end of the program, can provide clues about the mechanisms involved in abstinence or relapse. Indeed, specific changes in cognitive ERP markers during detoxification (a preserved oddball P3 and an enhanced No-go P3) indexed complete abstinence (over a 3-mo period) in alcoholic patients. The main clinical relevance of such test-retest ERP data is the possibility of pinpointing the change in specific neurocognitive functions (cue reactivity, inhibition) during the detoxification program that can predict further abstinence. Such a procedure necessarily implied: (1) Specification of the various cognitive mechanisms that should be considered as the primary targets subtending the clinical symptoms of interest; and (2) Selection of the appropriate cognitive tasks that will generate specific and reliable ERPs related to these specific processes at an individual level.

A crucial step, therefore, concerns the identification of the various cognitive processes of interest. As such, transdiagnostic (as opposed to disorder-specific) factors appear uniquely suited to bridge psychiatric phenomena and biological substrates of behavior[77]. Transdiagnostic impairment of cognitive control[82], self-referential processes[112], working memory[113], decision making[114], and attention[115] largely contribute to the real-world socio-occupational impairment common across disorders. Decades of research have validated reliable ERP markers for such processes (No-go P3, ERN, P300, MMN, and P50, respectively). Indeed, an altered No-go P3 response inhibition, as well as a deficit in P300 (in tasks tagging updating in working memory or decision making), are neurophysiological disorders present in psychotic[116,117], bipolar[118,119], unipolar[48,120] depressive disorders as well as

in anxiety[121,122] and substance use disorders[123,124]. Studies also commonly reported an altered ERN in anxiety disorders and in obsessive-compulsive disorders[125]; an altered MMN (indexing preattentive auditory memory processing) in major depression, schizophrenia, psychosis, and substance use disorders[36]; and a deficit in P50 sensory gating in stable schizophrenic patients and euthymic bipolar patients[58]. In such a dimensional transdiagnostic view, the lack of specificity reported for these various ERP components tagging specific cognitive functions will vanish and allow monitoring of the change in these processes during a treatment, independently of a categorical disorder. Such a proposition relies on two main "technical" recommendations for future ERP screening methods: (1) The use of individual ERP test-retest sessions; and (2) The use of a multi-component approach[126].

ERP serial recordings may be used in clinical contexts for the assessment of changes in cortical function during follow-up programs[33]. Indeed, although slight differences in the wave shape, size, and timing of ERPs between individuals are usually observed, these tend to be highly stable within an individual across recordings, as high internal consistency and high test-retest reliability have been reported[127-130]. A high degree of reliability is a key factor that makes state ERP markers highly suitable for examining changes in brain activity resulting from treatment intervention or disease progression in a single patient[12]. This could provide ERPs a novel and particularly useful role in the management of psychiatric disorders. Indeed, assessing that neural changes have been induced spontaneously and/or by a treatment is a necessary outcome to envisage medium to long-term positive behavioral changes[104]. For instance, recovering a normalized MMN amplitude or P50 suppression appears to be a good indicator of abstinence in alcohol and cocaine users, respectively[131]. In such a view, it is also particularly important to always merge ERP data obtained through "active" tasks with behavioral results. To illustrate this crucial point, we can focus on contradictory data showing that excessive alcohol users compared to controls exhibited decreased No-go P3 amplitudes[38,123], probably reflecting poor neural resources indexing a poor performance; while other studies have reported increased No-go P3 amplitudes[132], probably reflecting compensatory neuro-functional mechanisms that allow drinkers to achieve a similar performance level as controls. In other words, an increased amplitude should not necessarily be interpreted as a recovered activity, and a decreased amplitude should not necessarily be interpreted as a disrupted function. Therefore, merging test-retest ERP data with behavioral performances would allow identification of patients who need to recover more neural resources to achieve better performance (*e.g.*, an increase of inhibitory No-go P3 resources to maintain abstinence in alcoholic patients[109]), while patients exhibiting compensatory or exacerbated mechanisms may need to reduce the activity allocated to the task (*e.g.*, decreased ERN in anxiety disorders to assess better emotional regulation[56]). These types of individual assessments would help clinicians specify individual interventions that will target each patient's needs, thus providing "individualized" or "personalized" medicine[26]. For instance, a recently detoxified alcoholic patient who does not exhibit improvements on the No-go P3 component could be redirected to post-cure specific inhibitory boosting programs[109], while a patient with a severe anxiety disorder who does not exhibit a reduced ERN due to a drug treatment could be directed to therapies that address emotional regulation such as mindfulness[133]. Naturally, it is important at this point to outline that, on the basis of DSM or ICD psychiatric categories, some ERP trait markers have also been described. For instance, obsessive-compulsive disorder has been characterized by an increased ERN both before and after therapy[68] as well as for the altered P3 component in cocaine users[57]: Such trait markers, *i.e.*, neurophysiological mechanisms persisting during and after the disease, suggest that such ERP markers could be related to the risk for the disorder but not the expression of its phenotype. Even in a dimensional perspective, such trait markers, if present, can improve early detection of illness (*e.g.*, in healthy people with a positive family history), and, as such, facilitate more effective and targeted interventions[44]. Moreover, this also highlights a main challenge of future "ERP research applied to the clinic" to develop novel interventions/manipulations that could modify an ERP of interest. As such, it was found for instance that performing a secondary dual-task resulted in a reduced ERN, and this reduction was larger in patients with obsessive-compulsive disorder than in the group of healthy participants[134], as well as a single session of attention bias modification[135] or expressive writing[136], thus suggesting that increased ERNs in clinical anxiety disorders can be normalized, at least temporarily[137]. Increased sensitivity of the P3a and the P3b amplitudes to depression severity are also now observable thanks to the development of adapted new ERP protocols, such as the three-stimulus oddball design[138,139] and bimodal

oddball protocols[140,141], respectively. Research assessing the efficiency of a procedure or a treatment to impact an ERP of interest, even in healthy participants, will, therefore, remain of fundamental relevance in the ERP research area.

Many ERP studies have focused on "a single ERP component" (*i.e.*, P50, MMN, P3a, P3b, *etc.*), comparing it in a pathological population *vs* healthy matched control. However, at the clinical level, it has been suggested that although ERPs clearly exhibit high sensitivity and predictive power, they suffer from poor specificity[26,33]. The idea that a "multivariate endophenotype", based on a weighted combination of diverse electrophysiological features, may provide more information than any single endophenotype, is not novel[142]. Price and colleagues compared and contrasted four electrophysiological endophenotypes — MMN, P50, P300, and antisaccades — and showed that this combination of features decreased the impact of group heterogeneity. In the same vein, at an individual level, a prominent idea is that a psychiatric patient will exhibit various cognitive disorders, of varying severities, that will subtend their own clinical symptoms. In a dimensional view, such disturbances may evolve differentially, so that some ERP measurements that index a specific cognitive function may recover during a disorder, while others will exhibit long-lasting damage. As an example, we recently showed that a post-cure 3-mo abstinence period in alcoholic patients can be neurophysiologically indexed by an increased No-go P3 yet similar oddball P3 components between the start and the end of a detoxification program[109]. Future studies should hence adopt a multi-component approach in order to potentially increase the sensitivity of ERP recordings, as the change in the patterns of ERPs could be specific (*e.g.*, which components recover, and which remain disturbed) from one psychiatric patient to another. In my opinion, such patterns indexing the changes of various ERP components through test-retest sessions appear to be the best way for ERPs to provide clinicians with relevant information regarding change in the disease (due to a treatment) in a single patient and the residual cognitive impairments that still need to be addressed. Indeed, once cognitive disturbances have been characterized through ERP screening of individual patients, psychiatrists will be able to orient the "cognitive" treatment (individually or in groups that present homogeneous patterns of cognitive deficits). More precisely, specific cognitive retraining procedures could be used to target deficits and to increase cognitive efficiency, as both cognitive training and neuromodulation boosting methods have already been shown to reduce clinical symptoms[143,144].

CONCLUSION

The main aim of this opinion review was to present the main arguments in favour of the clinical utility of ERP components to help in the management of psychiatric disorders. ERPs may be of great value to psychiatrists for the identification and monitoring of cognitive processes that should be rehabilitated on a patient-by-patient basis[126]. Such a proposition is limited per se, as, naturally, the complexity of dealing with a mental illness encompasses a large variety of stakeholders, such as psychologists, psychiatrists, nurses, and social workers, as well as neuropsychologists and neurophysiologists. I am, therefore, fully aware that the ERP contribution would be minimal, as moreover, many other EEG tools (*e.g.*, event-related oscillations[145] or microstates[146]) could also be of interest. Also, combining ERPs data with more structural and functional information (through for instance fMRI studies[147,148]) would be of the greatest relevance in order to better capture the pathophysiological mechanisms underlying specific clinical symptoms and orient treatment. I am also fully aware that there is still a long way to go before such a proposition could be widely implemented in clinical care units: If such a procedure could be quite easy to install at a technical level for inpatients in psychiatric clinics in highly developed countries, the situation could be more problematic for lower-income countries, and even more for outpatients visiting on a punctual daily basis a psychiatric office. If such a procedure would reveal high efficiency in the future, economical discussions will have to be undertaken to furnish full access of such a material to all countries in order to (1) manage, monitor and orient treatment for inpatients; and (2) allow straight collaborations between research centers and outpatients' psychiatric office in order to deliver information to clinicians that could be of help in orienting treatment. In such a view, lack of normative data, technical artefacts linked to the recordings with patients, adoption of clear multisite guidelines, as well as a constructive dialogue between researchers and clinicians in the assessment of a suitable cognitive-ERP battery are still some of the main issues that warrant our full attention. A major issue with such a

proposal indeed relates to the fact that clinicians and researchers have to agree on a cognitive ERP-battery that could be used across centres (in terms of its content, but also, naturally, its multisite technical guidelines) and across disorders. Such a battery should be as complete as possible, but not too time-consuming in order for it to be adapted to all types of psychiatric patients (probably approximately a maximum of 45 min for a session recording?). It is nowadays well-accepted that transdiagnostic impairment of cognitive control[83], self-referential processes[112], working memory[113], decision making[114], and attention[115] largely contribute to the real-world socio-occupational impairment common across disorders. Therefore, I am inclined to suggest that such a cognitive ERP-battery should at least include two active and two passive tasks: (1) A Go/No-go task, which appears to be the best-suited task to assess cognitive control[143], and to record the No-go N2, the No-go P3, and the ERN as the main ERPs of interest; (2) A bimodal (visual-auditory) three-stimulus oddball task, in order to probe for updating memory and decision-making processes through the recording of P3a and P3b components[87,140]; (3) A passive auditory paired-stimulus paradigm, classically used to record sensory gating through the P50[149]; and (4) A passive auditory oddball design in order to access the MMN component[150]. Monitoring the changes in these components in a single patient during treatment would be of the greatest clinical interest for identifying neural changes that are positive predictors of the clinical trajectory as well as cognitive functions that still warrant being trained. Clearly, much work is still needed to achieve this aim, such as reaching an agreement regarding the battery content as well as establishing multicenter large sample recordings to obtain normative data and to test the efficiency of the procedure at a clinical level. Nevertheless, as EEG is a cheap method that can be readily implemented in any type of psychiatric care unit, and because ERPs can provide invaluable information regarding the neurocognitive status of a patient as a monitoring and a predictive biomarker tool, I very much think this method deserves attention and should be given more consideration for further development. The challenge for future studies will be to establish whether this procedure, driven by serial follow-up recordings of various ERP components in a singular patient, is efficient enough to be incorporated into novel psychiatric treatment methods.

ACKNOWLEDGEMENTS

REFERENCES

1   **Insel TR**, Quirion R. Psychiatry as a clinical neuroscience discipline. *JAMA* 2005; **294**: 2221-2224 [PMID: 16264165 DOI: 10.1001/jama.294.17.2221]

2   **Price BH**, Adams RD, Coyle JT. Neurology and psychiatry: closing the great divide. *Neurology* 2000; **54**: 8-14 [PMID: 10636118 DOI: 10.1212/wnl.54.1.8]

3   **Martin JB**. The integration of neurology, psychiatry, and neuroscience in the 21st century. *Am J Psychiatry* 2002; **159**: 695-704 [PMID: 11986119 DOI: 10.1176/appi.ajp.159.5.695]

4   **Levy N**. Addiction is Not a Brain Disease (and it Matters). *Front Psychiatry* 2013; **4**: 24 [PMID: 23596425 DOI: 10.3389/fpsyt.2013.00024]

5   **Buckholtz JW**, Meyer-Lindenberg A. Psychopathology and the human connectome: toward a transdiagnostic model of risk for mental illness. *Neuron* 2012; **74**: 990-1004 [PMID: 22726830 DOI: 10.1016/j.neuron.2012.06.002]

6   **Green MF**, Penn DL, Bentall R, Carpenter WT, Gaebel W, Gur RC, Kring AM, Park S, Silverstein SM, Heinssen R. Social cognition in schizophrenia: an NIMH workshop on definitions, assessment, and research opportunities. *Schizophr Bull* 2008; **34**: 1211-1220 [PMID: 18184635 DOI: 10.1093/schbul/sbm145]

7   **van Veluw SJ**, Chance SA. Differentiating between self and others: an ALE meta-analysis of fMRI studies of self-recognition and theory of mind. *Brain Imaging Behav* 2014; **8**: 24-38 [PMID: 24535033 DOI: 10.1007/s11682-013-9266-8]

8   **Roncone R**, Falloon IR, Mazza M, De Risio A, Pollice R, Necozione S, Morosini P, Casacchia M. Is theory of mind in schizophrenia more strongly associated with clinical and social functioning than with neurocognitive deficits? *Psychopathology* 2002; **35**: 280-288 [PMID: 12457019 DOI:

10.1159/000067062]

9　**Sergi M**, Green M, Widmark C, Reist C, Erhart S, Braff D, Kee K, Marder S, Mintz J. Social Cognition and Neurocognition: Effects of Risperidone, Olanzapine, and Haloperidol. *Am J Psychiatry* 2007; **164**: 1585-1592 [PMID: 22688151 DOI: 10.1176/appi.ajp.2007.06091515]

10　**Javed A**, Charles A. The Importance of Social Cognition in Improving Functional Outcomes in Schizophrenia. *Front Psychiatry* 2018; **9**: 157 [PMID: 29740360 DOI: 10.3389/fpsyt.2018.00157]

11　**Rassovsky Y**, Dunn W, Wynn J, Wu AD, Iacoboni M, Hellemann G, Green MF. The effect of transcranial direct current stimulation on social cognition in schizophrenia: A preliminary study. *Schizophr Res* 2015; **165**: 171-174 [PMID: 25934168 DOI: 10.1016/j.schres.2015.04.016]

12　**Mehta UM**, Thirthalli J, Basavaraju R, Gangadhar BN, Pascual-Leone A. Reduced mirror neuron activity in schizophrenia and its association with theory of mind deficits: evidence from a transcranial magnetic stimulation study. *Schizophr Bull* 2014; **40**: 1083-1094 [PMID: 24214933 DOI: 10.1093/schbul/sbt155]

13　**Mériau K**, Wartenburger I, Kazzer P, Prehn K, Lammers CH, van der Meer E, Villringer A, Heekeren HR. A neural network reflecting individual differences in cognitive processing of emotions during perceptual decision making. *Neuroimage* 2006; **33**: 1016-1027 [PMID: 16973382 DOI: 10.1016/j.neuroimage.2006.07.031]

14　**Linville P**. Attention inhibition: Does it underlie ruminative thought. *Advan Social Cogn* 1996; **9**: 121-133

15　**Daches S**, Mor N. Training ruminators to inhibit negative information: A preliminary report. *Cognit Ther Res* 2014; **38**: 160-171

16　**Monnart A**, Vanderhasselt MA, Schroder E, Campanella S, Fontaine P, Kornreich C. Treatment of Resistant Depression: A Pilot Study Assessing the Efficacy of a tDCS-Mindfulness Program Compared With a tDCS-Relaxation Program. *Front Psychiatry* 2019; **10**: 730 [PMID: 31708808 DOI: 10.3389/fpsyt.2019.00730]

17　**Dobson KS**, Hollon SD, Dimidjian S, Schmaling KB, Kohlenberg RJ, Gallop RJ, Rizvi SL, Gollan JK, Dunner DL, Jacobson NS. Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the prevention of relapse and recurrence in major depression. *J Consult Clin Psychol* 2008; **76**: 468-477 [PMID: 18540740 DOI: 10.1037/0022-006X.76.3.468]

18　**Vollstädt-Klein S**, Loeber S, Richter A, Kirsch M, Bach P, von der Goltz C, Hermann D, Mann K, Kiefer F. Validating incentive salience with functional magnetic resonance imaging: association between mesolimbic cue reactivity and attentional bias in alcohol-dependent patients. *Addict Biol* 2012; **17**: 807-816 [PMID: 21790907 DOI: 10.1111/j.1369-1600.2011.00352.x]

19　**Luijten M**, Machielsen MW, Veltman DJ, Hester R, de Haan L, Franken IH. Systematic review of ERP and fMRI studies investigating inhibitory control and error processing in people with substance dependence and behavioural addictions. *J Psychiatry Neurosci* 2014; **39**: 149-169 [PMID: 24359877 DOI: 10.1503/jpn.130052]

20　**Wiers RW**, Bartholow BD, van den Wildenberg E, Thush C, Engels RC, Sher KJ, Grenard J, Ames SL, Stacy AW. Automatic and controlled processes and the development of addictive behaviors in adolescents: a review and a model. *Pharmacol Biochem Behav* 2007; **86**: 263-283 [PMID: 17116324 DOI: 10.1016/j.pbb.2006.09.021]

21　**Palpacuer C**, Duprez R, Huneau A, Locher C, Boussageon R, Laviolle B, Naudet F. Pharmacologically controlled drinking in the treatment of alcohol dependence or alcohol use disorders: a systematic review with direct and network meta-analyses on nalmefene, naltrexone, acamprosate, baclofen and topiramate. *Addiction* 2018; **113**: 220-237 [PMID: 28940866 DOI: 10.1111/add.13974]

22　**Manning V**, Garfield JBB, Staiger PK, Lubman DI, Lum JAG, Reynolds J, Hall K, Bonomo Y, Lloyd-Jones M, Wiers RW, Piercy H, Jacka D, Verdejo-Garcia A. Effect of Cognitive Bias Modification on Early Relapse Among Adults Undergoing Inpatient Alcohol Withdrawal Treatment: A Randomized Clinical Trial. *JAMA Psychiatry* 2021; **78**: 133-140 [PMID: 33146693 DOI: 10.1001/jamapsychiatry.2020.3446]

23　**den Uyl TE**, Gladwin TE, Rinck M, Lindenmeyer J, Wiers RW. A clinical trial with combined transcranial direct current stimulation and alcohol approach bias retraining. *Addict Biol* 2017; **22**: 1632-1640 [PMID: 27790791 DOI: 10.1111/adb.12463]

24　**Menon V**. Brain networks and cognitive impairment in psychiatric disorders. *World Psychiatry* 2020; **19**: 309-310 [PMID: 32931097 DOI: 10.1002/wps.20799]

25　**Phan KL**, Wager T, Taylor SF, Liberzon I. Functional neuroanatomy of emotion: a meta-analysis of emotion activation studies in PET and fMRI. *Neuroimage* 2002; **16**: 331-348 [PMID: 12030820 DOI: 10.1006/nimg.2002.1087]

26　**Campanella S**. Why it is time to develop the use of cognitive event-related potentials in the treatment of psychiatric diseases. *Neuropsychiatr Dis Treat* 2013; **9**: 1835-1845 [PMID: 24348040 DOI: 10.2147/NDT.S53687]

27　**Kappenman ES**, Luck SJ. Best Practices for Event-Related Potential Research in Clinical Populations. *Biol Psychiatry Cogn Neurosci Neuroimaging* 2016; **1**: 110-115 [PMID: 27004261 DOI: 10.1016/j.bpsc.2015.11.007]

28　**Biasiucci A**, Franceschiello B, Murray MM. Electroencephalography. *Curr Biol* 2019; **29**: R80-R85 [PMID: 30721678 DOI: 10.1016/j.cub.2018.11.052]

29　**Rugg MD**, Coles MG. Electrophysiology of mind: Event-related brain potentials and cognition. Oxford: Oxford University Press, 1995

30   **Campanella S**, Arikan K, Babiloni C, Balconi M, Bertollo M, Betti V, Bianchi L, Brunovsky M, Buttinelli C, Comani S, Di Lorenzo G, Dumalin D, Escera C, Fallgatter A, Fisher D, Giordano GM, Guntekin B, Imperatori C, Ishii R, Kajosch H, Kiang M, López-Caneda E, Missonier P, Mucci A, Olbrich S, Otte G, Perrottelli A, Pizzuti A, Pinal D, Salisbury D, Tang Y, Tisei P, Wang J, Winkler I, Yuan J, Pogarell O. Special Report on the Impact of the COVID-19 Pandemic on Clinical EEG and Research and Consensus Recommendations for the Safe Use of EEG. *Clin EEG Neurosci* 2021; **52**: 3-28 [PMID: 32975150 DOI: 10.1177/1550059420954054]

31   **Rossignol M**, Campanella S, Maurage P, Heeren A, Falbo L, Philippot P. Enhanced perceptual responses during visual processing of facial stimuli in young socially anxious individuals. *Neurosci Lett* 2012; **526**: 68-73 [PMID: 22884932 DOI: 10.1016/j.neulet.2012.07.045]

32   **de Tommaso M**, Betti V, Bocci T, Bolognini N, Di Russo F, Fattapposta F, Ferri R, Invitto S, Koch G, Miniussi C, Piccione F, Ragazzoni A, Sartucci F, Rossi S, Arcara G, Berchicci M, Bianco V, Delussi M, Gentile E, Giovannelli F, Mannarelli D, Marino M, Mussini E, Pauletti C, Pellicciari MC, Pisoni A, Raggi A, Valeriani M. Pearls and pitfalls in brain functional analysis by event-related potentials: a narrative review by the Italian Psychophysiology and Cognitive Neuroscience Society on methodological limits and clinical reliability-part I. *Neurol Sci* 2020; **41**: 2711-2735 [PMID: 32388645 DOI: 10.1007/s10072-020-04420-7]

33   **Pogarell O**, Mulert C, Hegerl U. Event-related potentials in psychiatry. *Clin EEG Neurosci* 2007; **38**: 25-34 [PMID: 17319589 DOI: 10.1177/155005940703800108]

34   **Nagamoto HT**, Adler LE, Waldo MC, Griffith J, Freedman R. Gating of auditory response in schizophrenics and normal controls. Effects of recording site and stimulation interval on the P50 wave. *Schizophr Res* 1991; **4**: 31-40 [PMID: 1848997 DOI: 10.1016/0920-9964(91)90007-e]

35   **Nagai Y**, Critchley HD, Featherstone E, Fenwick PB, Trimble MR, Dolan RJ. Brain activity relating to the contingent negative variation: an fMRI investigation. *Neuroimage* 2004; **21**: 1232-1241 [PMID: 15050551 DOI: 10.1016/j.neuroimage.2003.10.036]

36   **Näätänen R**, Kujala T, Escera C, Baldeweg T, Kreegipuu K, Carlson S, Ponton C. The mismatch negativity (MMN)--a unique window to disturbed central auditory processing in ageing and different clinical conditions. *Clin Neurophysiol* 2012; **123**: 424-458 [PMID: 22169062 DOI: 10.1016/j.clinph.2011.09.020]

37   **Polich J**. Updating P300: an integrative theory of P3a and P3b. *Clin Neurophysiol* 2007; **118**: 2128-2148 [PMID: 17573239 DOI: 10.1016/j.clinph.2007.04.019]

38   **Falkenstein M**, Hoormann J, Hohnsbein J. ERP components in Go/Nogo tasks and their relation to inhibition. *Acta Psychol (Amst)* 1999; **101**: 267-291 [PMID: 10344188 DOI: 10.1016/s0001-6918(99)00008-6]

39   **Hajcak G**, Moser JS, Yeung N, Simons RF. On the ERN and the significance of errors. *Psychophysiology* 2005; **42**: 151-160 [PMID: 15787852 DOI: 10.1111/j.1469-8986.2005.00270.x]

40   **Kutas M**, Federmeier KD. Thirty years and counting: finding meaning in the N400 component of the event-related brain potential (ERP). *Annu Rev Psychol* 2011; **62**: 621-647 [PMID: 20809790 DOI: 10.1146/annurev.psych.093008.131123]

41   **Warbrick T**, Mobascher A, Brinkmeyer J, Musso F, Richter N, Stoecker T, Fink GR, Shah NJ, Winterer G. Single-trial P3 amplitude and latency informed event-related fMRI models yield different BOLD response patterns to a target detection task. *Neuroimage* 2009; **47**: 1532-1544 [PMID: 19505583 DOI: 10.1016/j.neuroimage.2009.05.082]

42   **Warbrick T**, Mobascher A, Brinkmeyer J, Musso F, Stoecker T, Shah NJ, Fink GR, Winterer G. Nicotine effects on brain function during a visual oddball task: a comparison between conventional and EEG-informed fMRI analysis. *J Cogn Neurosci* 2012; **24**: 1682-1694 [PMID: 22452559 DOI: 10.1162/jocn_a_00236]

43   **Campanella S**, Petit G, Maurage P, Kornreich C, Verbanck P, Noël X. Chronic alcoholism: insights from neurophysiology. *Neurophysiol Clin* 2009; **39**: 191-207 [PMID: 19853791 DOI: 10.1016/j.neucli.2009.08.002]

44   **van der Stelt O**, Belger A. Application of electroencephalography to the study of cognitive and brain functions in schizophrenia. *Schizophr Bull* 2007; **33**: 955-970 [PMID: 17363840 DOI: 10.1093/schbul/sbm016]

45   **Bender S**, Weisbrod M, Resch F. Which perspectives can endophenotypes and biological markers offer in the early recognition of schizophrenia? *J Neural Transm (Vienna)* 2007; **114**: 1199-1215 [PMID: 17514428 DOI: 10.1007/s00702-007-0742-4]

46   **Karaaslan F**, Gonul AS, Oguz A, Erdinc E, Esel E. P300 changes in major depressive disorders with and without psychotic features. *J Affect Disord* 2003; **73**: 283-287 [PMID: 12547298 DOI: 10.1016/s0165-0327(01)00477-3]

47   **Blackwood DH**, Whalley LJ, Christie JE, Blackburn IM, St Clair DM, McInnes A. Changes in auditory P3 event-related potential in schizophrenia and depression. *Br J Psychiatry* 1987; **150**: 154-160 [PMID: 2888501 DOI: 10.1192/bjp.150.2.154]

48   **Gangadhar BN**, Ancy J, Janakiramaiah N, Umapathy C. P300 amplitude in non-bipolar, melancholic depression. *J Affect Disord* 1993; **28**: 57-60 [PMID: 8326081 DOI: 10.1016/0165-0327(93)90077-w]

49   **Umbricht D**, Krljes S. Mismatch negativity in schizophrenia: a meta-analysis. *Schizophr Res* 2005; **76**: 1-23 [PMID: 15927795 DOI: 10.1016/j.schres.2004.12.002]

50   **Zhou Z**, Zhu H, Chen L. Effect of aripiprazole on mismatch negativity (MMN) in schizophrenia. *PLoS One* 2013; **8**: e52186 [PMID: 23308105 DOI: 10.1371/journal.pone.0052186]

51 **Lavoie S**, Murray MM, Deppen P, Knyazeva MG, Berk M, Boulat O, Bovet P, Bush AI, Conus P, Copolov D, Fornari E, Meuli R, Solida A, Vianin P, Cuénod M, Buclin T, Do KQ. Glutathione precursor, N-acetyl-cysteine, improves mismatch negativity in schizophrenia patients. *Neuropsychopharmacology* 2008; **33**: 2187-2199 [PMID: 18004285 DOI: 10.1038/sj.npp.1301624]

52 **Perchet C**, Revol O, Fourneret P, Mauguière F, Garcia-Larrea L. Attention shifts and anticipatory mechanisms in hyperactive children: an ERP study using the Posner paradigm. *Biol Psychiatry* 2001; **50**: 44-57 [PMID: 11457423 DOI: 10.1016/s0006-3223(00)01119-7]

53 **Mayer K**, Wyckoff SN, Strehl U. Underarousal in Adult ADHD: How Are Peripheral and Cortical Arousal Related? *Clin EEG Neurosci* 2016; **47**: 171-179 [PMID: 25802473 DOI: 10.1177/1550059415577544]

54 **Brunner JF**. Predicting clinical outcome of stimulant medication in pediatric attention, deficit/hyperactivity disorder (ADHD): Single-dose changes in event-related potentials (ERPs). *Eur Psychiatry* 2016; **33**: S144

55 **Bramon E**, Rabe-Hesketh S, Sham P, Murray RM, Frangou S. Meta-analysis of the P300 and P50 waveforms in schizophrenia. *Schizophr Res* 2004; **70**: 315-329 [PMID: 15329307 DOI: 10.1016/j.schres.2004.01.004]

56 **Meyer A**. A biomarker of anxiety in children and adolescents: A review focusing on the error-related negativity (ERN) and anxiety across development. *Dev Cogn Neurosci* 2017; **27**: 58-68 [PMID: 28818707 DOI: 10.1016/j.dcn.2017.08.001]

57 **Boutros NN**, Gooding D, Sundaresan K, Burroughs S, Johanson CE. Cocaine-dependence and cocaine-induced paranoia and mid-latency auditory evoked responses and sensory gating. *Psychiatry Res* 2006; **145**: 147-154 [PMID: 17079024 DOI: 10.1016/j.psychres.2006.02.005]

58 **Sánchez-Morla EM**, García-Jiménez MA, Barabash A, Martínez-Vizcaíno V, Mena J, Cabranes-Díaz JA, Baca-Baldomero E, Santos JL. P50 sensory gating deficit is a common marker of vulnerability to bipolar disorder and schizophrenia. *Acta Psychiatr Scand* 2008; **117**: 313-318 [PMID: 18241306 DOI: 10.1111/j.1600-0447.2007.01141.x]

59 **Meyer A**, Nelson B, Perlman G, Klein DN, Kotov R. A neural biomarker, the error-related negativity, predicts the first onset of generalized anxiety disorder in a large sample of adolescent females. *J Child Psychol Psychiatry* 2018; **59**: 1162-1170 [PMID: 29665048 DOI: 10.1111/jcpp.12922]

60 **Kamarajan C**, Porjesz B, Jones KA, Chorlian DB, Padmanabhapillai A, Rangaswamy M, Stimus AT, Begleiter H. Spatial-anatomical mapping of NoGo-P3 in the offspring of alcoholics: evidence of cognitive and neural disinhibition as a risk for alcoholism. *Clin Neurophysiol* 2005; **116**: 1049-1061 [PMID: 15826845 DOI: 10.1016/j.clinph.2004.12.015]

61 **Smith JL**, Johnstone SJ, Barry RJ. Aiding diagnosis of attention-deficit/hyperactivity disorder and its subtypes: discriminant function analysis of event-related potential data. *J Child Psychol Psychiatry* 2003; **44**: 1067-1075 [PMID: 14531589 DOI: 10.1111/1469-7610.00191]

62 **Goffin D**, Allen M, Zhang L, Amorim M, Wang IT, Reyes AR, Mercado-Berton A, Ong C, Cohen S, Hu L, Blendy JA, Carlson GC, Siegel SJ, Greenberg ME, Zhou Z. Rett syndrome mutation MeCP2 T158A disrupts DNA binding, protein stability and ERP responses. *Nat Neurosci* 2011; **15**: 274-283 [PMID: 22119903 DOI: 10.1038/nn.2997]

63 **Rudolph ED**, Ells EM, Campbell DJ, Abriel SC, Tibbo PG, Salisbury DF, Fisher DJ. Finding the missing-stimulus mismatch negativity (MMN) in early psychosis: altered MMN to violations of an auditory gestalt. *Schizophr Res* 2015; **166**: 158-163 [PMID: 26072323 DOI: 10.1016/j.schres.2015.05.028]

64 **Wang K**, Cheung EF, Gong QY, Chan RC. Semantic processing disturbance in patients with schizophrenia: a meta-analysis of the N400 component. *PLoS One* 2011; **6**: e25435 [PMID: 22022395 DOI: 10.1371/journal.pone.0025435]

65 **Euser AS**, Arends LR, Evans BE, Greaves-Lord K, Huizink AC, Franken IH. The P300 event-related brain potential as a neurobiological endophenotype for substance use disorders: a meta-analytic investigation. *Neurosci Biobehav Rev* 2012; **36**: 572-603 [PMID: 21964481 DOI: 10.1016/j.neubiorev.2011.09.002]

66 **Schwartz S**, Shinn-Cunningham B, Tager-Flusberg H. Meta-analysis and systematic review of the literature characterizing auditory mismatch negativity in individuals with autism. *Neurosci Biobehav Rev* 2018; **87**: 106-117 [PMID: 29408312 DOI: 10.1016/j.neubiorev.2018.01.008]

67 **Cheng CH**, Tsai HY, Cheng HN. The effect of age on N2 and P3 components: A meta-analysis of Go/Nogo tasks. *Brain Cogn* 2019; **135**: 103574 [PMID: 31200173 DOI: 10.1016/j.bandc.2019.05.012]

68 **Riesel A**. The erring brain: Error-related negativity as an endophenotype for OCD-A review and meta-analysis. *Psychophysiology* 2019; **56**: e13348 [PMID: 30838682 DOI: 10.1111/psyp.13348]

69 **Cavanagh J**, Geisler MW. Mood effects on the ERP processing of emotional intensity in faces: a P3 investigation with depressed students. *Int J Psychophysiol* 2006; **60**: 27-33 [PMID: 15963586 DOI: 10.1016/j.ijpsycho.2005.04.005]

70 **Pesa N**, Hermens DF, Battisti RA, Kaur M, Hickie IB, Solowij N. Delayed preattentional functioning in early psychosis patients with cannabis use. *Psychopharmacology (Berl)* 2012; **222**: 507-518 [PMID: 22402706 DOI: 10.1007/s00213-012-2676-2]

71 **Mondragón-Maya A**, Solís-Vivanco R, León-Ortiz P, Rodríguez-Agudelo Y, Yáñez-Téllez G, Bernal-Hernández J, Cadenhead KS, de la Fuente-Sandoval C. Reduced P3a amplitudes in antipsychotic naïve first-episode psychosis patients and individuals at clinical high-risk for

psychosis. *J Psychiatr Res* 2013; **47**: 755-761 [PMID: 23507048 DOI: 10.1016/j.jpsychires.2012.12.017]

72   **Rossignol M**, Philippot P, Crommelinck M, Campanella S. Visual processing of emotional expressions in mixed anxious-depressed subclinical state: an event-related potential study on a female sample. *Neurophysiol Clin* 2008; **38**: 267-275 [PMID: 18940614 DOI: 10.1016/j.neucli.2008.07.007]

73   **Campanella S**, Colin C. Event-related potentials and biomarkers of psychiatric diseases: the necessity to adopt and develop multi-site guidelines. *Front Behav Neurosci* 2014; **8**: 428 [PMID: 25540614 DOI: 10.3389/fnbeh.2014.00428]

74   **Kappenman ES**, Farrens JL, Zhang W, Stewart AX, Luck SJ. ERP CORE: An open resource for human event-related potential research. *Neuroimage* 2021; **225**: 117465 [PMID: 33099010 DOI: 10.1016/j.neuroimage.2020.117465]

75   **Duncan CC**, Barry RJ, Connolly JF, Fischer C, Michie PT, Näätänen R, Polich J, Reinvang I, Van Petten C. Event-related potentials in clinical research: guidelines for eliciting, recording, and quantifying mismatch negativity, P300, and N400. *Clin Neurophysiol* 2009; **120**: 1883-1908 [PMID: 19796989 DOI: 10.1016/j.clinph.2009.07.045]

76   **Franken IHA**, van de Wetering BJM. Bridging the gap between the neurocognitive lab and the addiction clinic. *Addict Behav* 2015; **44**: 108-114 [PMID: 25500167 DOI: 10.1016/j.addbeh.2014.11.034]

77   **Krueger RF**, Eaton NR. Transdiagnostic factors of mental disorders. *World Psychiatry* 2015; **14**: 27-29 [PMID: 25655146 DOI: 10.1002/wps.20175]

78   **McIntyre A**, Gendron A, McIntyre A. Quetiapine adjunct to selective serotonin reuptake inhibitors or venlafaxine in patients with major depression, comorbid anxiety, and residual depressive symptoms: a randomized, placebo-controlled pilot study. *Depress Anxiety* 2007; **24**: 487-494 [PMID: 17177199 DOI: 10.1002/da.20275]

79   **First MB**. Clinical utility: a prerequisite for the adoption of a dimensional approach in DSM. *J Abnorm Psychol* 2005; **114**: 560-564 [PMID: 16351379 DOI: 10.1037/0021-843X.114.4.560]

80   **First MB**.   Psychiatric classification. In: Tasman A, Kay J, Lieberman J. Psychiatry. 2nd ed. Chichester, England: Wiley, 2003: 659-676

81   **McTeague LM**, Goodkind MS, Etkin A. Transdiagnostic impairment of cognitive control in mental illness. *J Psychiatr Res* 2016; **83**: 37-46 [PMID: 27552532 DOI: 10.1016/j.jpsychires.2016.08.001]

82   **Krueger RF**. The structure of common mental disorders. *Arch Gen Psychiatry* 1999; **56**: 921-926 [PMID: 10530634 DOI: 10.1001/archpsyc.56.10.921]

83   **Stanton K**, McDonnell CG, Hayden EP, Watson D. Transdiagnostic approaches to psychopathology measurement: Recommendations for measure selection, data analysis, and participant recruitment. *J Abnorm Psychol* 2020; **129**: 21-28 [PMID: 31868384 DOI: 10.1037/abn0000464]

84   **Cosci F**, Fava GA. The clinical inadequacy of the DSM-5 classification of somatic symptom and related disorders: an alternative trans-diagnostic model. *CNS Spectr* 2016; **21**: 310-317 [PMID: 26707822 DOI: 10.1017/S1092852915000760]

85   **Baskaran A**, Milev R, McIntyre RS. The neurobiology of the EEG biomarker as a predictor of treatment response in depression. *Neuropharmacology* 2012; **63**: 507-513 [PMID: 22569197 DOI: 10.1016/j.neuropharm.2012.04.021]

86   **Hamburg MA**, Collins FS. The path to personalized medicine. *N Engl J Med* 2010; **363**: 301-304 [PMID: 20551152 DOI: 10.1056/NEJMp1006304]

87   **Kajosch H**, Hanard F, Steegen G, Persefonis G, Cimochowska A, Michel S, Kornreich C. Monitoring the Clinical Evolution of A Psychotic Patient Presenting A First-Schizophrenic Episode Thanks to Bimodal Oddball-P300 Event-Related Potentials: First Evidence from A Single-Case Study. *Arch Clin Med Case Rep* 2020; **4**: 1194-1207

88   **Wagner HN Jr**. Clinical PET: its time has come. *J Nucl Med* 1991; **32**: 561-564 [PMID: 1849557]

89   **Brown WS**, Marsh JT, LaRue A. Event-related potentials in psychiatry: differentiating depression and dementia in the elderly. *Bull Los Angeles Neurol Soc* 1982; **47**: 91-107 [PMID: 7183369]

90   **Porjesz B**, Begleiter H.   Human brain electrophysiology and alcoholism. In: Tarter RE, van Thiel DH. Alcohol and the Brain. Boston, MA: Springer, 1985: 139-182

91   **Pritchard WS**. Cognitive event-related potential correlates of schizophrenia. *Psychol Bull* 1986; **100**: 43-66 [PMID: 2874579]

92   **Singh SM**, Basu D. The P300 event-related potential and its possible role as an endophenotype for studying substance use disorders: a review. *Addict Biol* 2009; **14**: 298-309 [PMID: 18811679 DOI: 10.1111/j.1369-1600.2008.00124.x]

93   **Kohn R**, Saxena S, Levav I, Saraceno B. The treatment gap in mental health care. *Bull World Health Organ* 2004; **82**: 858-866 [PMID: 15640922]

94   **Perry A**, Tarrier N, Morriss R, McCarthy E, Limb K. Randomised controlled trial of efficacy of teaching patients with bipolar disorder to identify early symptoms of relapse and obtain treatment. *BMJ* 1999; **318**: 149-153 [PMID: 9888904 DOI: 10.1136/bmj.318.7177.149]

95   **Robinson D**, Woerner MG, Alvir JM, Bilder R, Goldman R, Geisler S, Koreen A, Sheitman B, Chakos M, Mayerhoff D, Lieberman JA. Predictors of relapse following response from a first episode of schizophrenia or schizoaffective disorder. *Arch Gen Psychiatry* 1999; **56**: 241-247 [PMID: 10078501 DOI: 10.1001/archpsyc.56.3.241]

96   **Boothby LA**, Doering PL. Acamprosate for the treatment of alcohol dependence. *Clin Ther* 2005; **27**: 695-714 [PMID: 16117977 DOI: 10.1016/j.clinthera.2005.06.015]

97   **Henggeler SW**. Multisystemic Therapy: An overview of clinical procedures, outcomes, and policy implications. *Child Psychol Psychiatry Rev* 1999; **4**: 1-10

98   **Pike KM**, Walsh BT, Vitousek K, Wilson GT, Bauer J. Cognitive behavior therapy in the posthospitalization treatment of anorexia nervosa. *Am J Psychiatry* 2003; **160**: 2046-2049 [PMID: 14594754 DOI: 10.1176/appi.ajp.160.11.2046]

99   **Chiesa A**, Serretti A. Mindfulness based cognitive therapy for psychiatric disorders: a systematic review and meta-analysis. *Psychiatry Res* 2011; **187**: 441-453 [PMID: 20846726 DOI: 10.1016/j.psychres.2010.08.011]

100   **Andreasen NC**.   Brave new brain: Conquering mental illness in the era of the genome. Oxford: Oxford University Press, 2004

101   **Etkin A**, Gyurak A, O'Hara R. A neurobiological approach to the cognitive deficits of psychiatric disorders. *Dialogues Clin Neurosci* 2013; **15**: 419-429 [PMID: 24459409 DOI: 10.31887/DCNS.2013.15.4/aetkin]

102   **Cavelti M**, Kvrgic S, Beck EM, Kossowsky J, Vauth R. Assessing recovery from schizophrenia as an individual process. A review of self-report instruments. *Eur Psychiatry* 2012; **27**: 19-32 [PMID: 22130177 DOI: 10.1016/j.eurpsy.2011.01.007]

103   **Buchsbaum MS**. Evidence, evidence-based medicine, and evidence utility in psychiatry and electrophysiology. *Clin EEG Neurosci* 2009; **40**: 143-145 [PMID: 19534306 DOI: 10.1177/155005940904000212]

104   **Vinogradov S**, Fisher M, de Villers-Sidani E. Cognitive training for impaired neural systems in neuropsychiatric illness. *Neuropsychopharmacology* 2012; **37**: 43-76 [PMID: 22048465 DOI: 10.1038/npp.2011.251]

105   **Schroder E**, Dubuson M, Dousset C, Mortier E, Kornreich C, Campanella S. Training Inhibitory Control Induced Robust Neural Changes When Behavior Is Affected: A Follow-up Study Using Cognitive Event-Related Potentials. *Clin EEG Neurosci* 2020; **51**: 303-316 [PMID: 31858835 DOI: 10.1177/1550059419895146]

106   **Fisher M**, Holland C, Merzenich MM, Vinogradov S. Using neuroplasticity-based auditory training to improve verbal memory in schizophrenia. *Am J Psychiatry* 2009; **166**: 805-811 [PMID: 19448187 DOI: 10.1176/appi.ajp.2009.08050757]

107   **Fisher M**, Holland C, Subramaniam K, Vinogradov S. Neuroplasticity-based cognitive training in schizophrenia: an interim report on the effects 6 mo later. *Schizophr Bull* 2010; **36**: 869-879 [PMID: 19269924 DOI: 10.1093/schbul/sbn170]

108   **Wolinsky FD**, Mahncke H, Vander Weg MW, Martin R, Unverzagt FW, Ball KK, Jones RN, Tennstedt SL. Speed of processing training protects self-rated health in older adults: enduring effects observed in the multi-site ACTIVE randomized controlled trial. *Int Psychogeriatr* 2010; **22**: 470-478 [PMID: 20003628 DOI: 10.1017/S1041610209991281]

109   **Campanella S**, Schroder E, Kajosch H, Hanak C, Veeser J, Amiot M, Besse-Hammer T, Hayef N, Kornreich C. Neurophysiological markers of cue reactivity and inhibition subtend a three-month period of complete alcohol abstinence. *Clin Neurophysiol* 2020; **131**: 555-565 [PMID: 31786051 DOI: 10.1016/j.clinph.2019.10.020]

110   **Petit G**, Cimochowska A, Kornreich C, Hanak C, Verbanck P, Campanella S. Neurophysiological correlates of response inhibition predict relapse in detoxified alcoholic patients: some preliminary evidence from event-related potentials. *Neuropsychiatr Dis Treat* 2014; **10**: 1025-1037 [PMID: 24966675 DOI: 10.2147/NDT.S61475]

111   **Petit G**, Cimochowska A, Cevallos C, Cheron G, Kornreich C, Hanak C, Schroder E, Verbanck P, Campanella S. Reduced processing of alcohol cues predicts abstinence in recently detoxified alcoholic patients in a three-month follow up period: an ERP study. *Behav Brain Res* 2015; **282**: 84-94 [PMID: 25576964 DOI: 10.1016/j.bbr.2014.12.057]

112   **Elliott ML**, Romer A, Knodt AR, Hariri AR. A Connectome-wide Functional Signature of Transdiagnostic Risk for Mental Illness. *Biol Psychiatry* 2018; **84**: 452-459 [PMID: 29779670 DOI: 10.1016/j.biopsych.2018.03.012]

113   **Huang-Pollock C**, Shapiro Z, Galloway-Long H, Weigard A. Is Poor Working Memory a Transdiagnostic Risk Factor for Psychopathology? *J Abnorm Child Psychol* 2017; **45**: 1477-1490 [PMID: 27783257 DOI: 10.1007/s10802-016-0219-8]

114   **Goschke T**. Dysfunctions of decision-making and cognitive control as transdiagnostic mechanisms of mental disorders: advances, gaps, and needs in current research. *Int J Methods Psychiatr Res* 2014; **23** Suppl 1: 41-57 [PMID: 24375535 DOI: 10.1002/mpr.1410]

115   **Hsu KJ**, Beard C, Rifkin L, Dillon DG, Pizzagalli DA, Björgvinsson T. Transdiagnostic mechanisms in depression and anxiety: The role of rumination and attentional control. *J Affect Disord* 2015; **188**: 22-27 [PMID: 26340079 DOI: 10.1016/j.jad.2015.08.008]

116   **Ertekin E**, Üçok A, Keskin-Ergen Y, Devrim-Üçok M. Deficits in Go and NoGo P3 potentials in patients with schizophrenia. *Psychiatry Res* 2017; **254**: 126-132 [PMID: 28460282 DOI: 10.1016/j.psychres.2017.04.052]

117   **Salisbury DF**, Shenton ME, McCarley RW. P300 topography differs in schizophrenia and manic psychosis. *Biol Psychiatry* 1999; **45**: 98-106 [PMID: 9894581 DOI: 10.1016/s0006-3223(98)00208-x]

118   **Morsel AM**, Dhar M, Hulstijn W, Temmerman A, Morrens M, Sabbe B. Inhibitory control in euthymic bipolar disorder: Event related potentials during a Go/NoGo task. *Clin Neurophysiol* 2017; **128**: 520-528 [PMID: 28222346 DOI: 10.1016/j.clinph.2016.12.006]

119 **Barreiros AR**, Breukelaar IA, Chen W, Erlinger M, Antees C, Medway M, Boyce P, Hazell P, Williams LM, Malhi GS, Harris AWF, Korgaonkar MS. Neurophysiological markers of attention distinguish bipolar disorder and unipolar depression. *J Affect Disord* 2020; **274**: 411-419 [PMID: 32663971 DOI: 10.1016/j.jad.2020.05.048]

120 **Kaiser S**, Unger J, Kiefer M, Markela J, Mundt C, Weisbrod M. Executive control deficit in depression: event-related potentials in a Go/Nogo task. *Psychiatry Res* 2003; **122**: 169-184 [PMID: 12694891 DOI: 10.1016/s0925-4927(03)00004-0]

121 **Sehlmeyer C**, Konrad C, Zwitserlood P, Arolt V, Falkenstein M, Beste C. ERP indices for response inhibition are related to anxiety-related personality traits. *Neuropsychologia* 2010; **48**: 2488-2495 [PMID: 20434466 DOI: 10.1016/j.neuropsychologia.2010.04.022]

122 **Enoch MA**, White KV, Waheed J, Goldman D. Neurophysiological and genetic distinctions between pure and comorbid anxiety disorders. *Depress Anxiety* 2008; **25**: 383-392 [PMID: 17941097 DOI: 10.1002/da.20378]

123 **Kamarajan C**, Porjesz B, Jones KA, Choi K, Chorlian DB, Padmanabhapillai A, Rangaswamy M, Stimus AT, Begleiter H. Alcoholism is a disinhibitory disorder: neurophysiological evidence from a Go/No-Go task. *Biol Psychol* 2005; **69**: 353-373 [PMID: 15925035 DOI: 10.1016/j.biopsycho.2004.08.004]

124 **Maurage P**, Philippot P, Verbanck P, Noel X, Kornreich C, Hanak C, Campanella S. Is the P300 deficit in alcoholism associated with early visual impairments (P100, N170)? *Clin Neurophysiol* 2007; **118**: 633-644 [PMID: 17208045 DOI: 10.1016/j.clinph.2006.11.007]

125 **Weinberg A**, Dieterich R, Riesel A. Error-related brain activity in the age of RDoC: A review of the literature. *Int J Psychophysiol* 2015; **98**: 276-299 [PMID: 25746725 DOI: 10.1016/j.ijpsycho.2015.02.029]

126 **Campanella S**, Schroder E, Kajosch H, Noel X, Kornreich C. Why cognitive event-related potentials (ERPs) should have a role in the management of alcohol disorders. *Neurosci Biobehav Rev* 2019; **106**: 234-244 [PMID: 29936112 DOI: 10.1016/j.neubiorev.2018.06.016]

127 **Walhovd KB**, Fjell AM. One-year test-retest reliability of auditory ERPs in young and old adults. *Int J Psychophysiol* 2002; **46**: 29-40 [PMID: 12374644 DOI: 10.1016/s0167-8760(02)00039-9]

128 **Williams LM**, Simms E, Clark CR, Paul RH, Rowe D, Gordon E. The test-retest reliability of a standardized neurocognitive and neurophysiological test battery: "neuromarker". *Int J Neurosci* 2005; **115**: 1605-1630 [PMID: 16287629 DOI: 10.1080/00207450590958475]

129 **Kappenman ES**, Farrens JL, Luck SJ, Proudfit GH. Behavioral and ERP measures of attentional bias to threat in the dot-probe task: poor reliability and lack of correlation with anxiety. *Front Psychol* 2014; **5**: 1368 [PMID: 25538644 DOI: 10.3389/fpsyg.2014.01368]

130 **Kappenman ES**, MacNamara A, Proudfit GH. Electrocortical evidence for rapid allocation of attention to threat in the dot-probe task. *Soc Cogn Affect Neurosci* 2015; **10**: 577-583 [PMID: 25062842 DOI: 10.1093/scan/nsu098]

131 **Campanella S**, Pogarell O, Boutros N. Event-related potentials in substance use disorders: a narrative review based on articles from 1984 to 2012. *Clin EEG Neurosci* 2014; **45**: 67-76 [PMID: 24104954 DOI: 10.1177/1550059413495533]

132 **López-Caneda E**, Cadaveira F, Crego A, Gómez-Suárez A, Corral M, Parada M, Caamaño-Isorna F, Rodríguez Holguín S. Hyperactivation of right inferior frontal cortex in young binge drinkers during response inhibition: a follow-up study. *Addiction* 2012; **107**: 1796-1808 [PMID: 22487028 DOI: 10.1111/j.1360-0443.2012.03908.x]

133 **Fissler M**, Winnebeck E, Schroeter TA, Gummbersbach M, Huntenburg JM, Gärtner M, Barnhofer T. Brief training in mindfulness may normalize a blunted error-related negativity in chronically depressed patients. *Cogn Affect Behav Neurosci* 2017; **17**: 1164-1175 [PMID: 28975567 DOI: 10.3758/s13415-017-0540-x]

134 **Klawohn J**, Endrass T, Preuss J, Riesel A, Kathmann N. Modulation of hyperactive error signals in obsessive-compulsive disorder by dual-task demands. *J Abnorm Psychol* 2016; **125**: 292-298 [PMID: 26692121 DOI: 10.1037/abn0000134]

135 **Nelson BD**, Jackson F, Amir N, Hajcak G. Single-session attention bias modification and error-related brain activity. *Cogn Affect Behav Neurosci* 2015; **15**: 776-786 [PMID: 26063611 DOI: 10.3758/s13415-015-0365-4]

136 **Schroder HS**, Moran TP, Moser JS. The effect of expressive writing on the error-related negativity among individuals with chronic worry. *Psychophysiology* 2018; **55** [PMID: 28884815 DOI: 10.1111/psyp.12990]

137 **Hajcak G**, Klawohn J, Meyer A. The Utility of Event-Related Potentials in Clinical Psychology. *Annu Rev Clin Psychol* 2019; **15**: 71-95 [PMID: 31067414 DOI: 10.1146/annurev-clinpsy-050718-095457]

138 **Bruder GE**, Kroppmann CJ, Kayser J, Stewart JW, McGrath PJ, Tenke CE. Reduced brain responses to novel sounds in depression: P3 findings in a novelty oddball task. *Psychiatry Res* 2009; **170**: 218-223 [PMID: 19900720 DOI: 10.1016/j.psychres.2008.10.023]

139 **Tenke CE**, Kayser J, Stewart JW, Bruder GE. Novelty P3 reductions in depression: characterization using principal components analysis (PCA) of current source density (CSD) waveforms. *Psychophysiology* 2010; **47**: 133-146 [PMID: 19761526 DOI: 10.1111/j.1469-8986.2009.00880.x]

140 **Kajosch H**, Gallhofer B, Corten P, From L, Verbanck P, Campanella S. The bimodal P300 oddball component is decreased in patients with an adjustment disorder: An event-related potentials study. *Clin Neurophysiol* 2016; **127**: 3209-3216 [PMID: 27521621 DOI: 10.1016/j.clinph.2016.07.009]

141   **Nan C**, Wang G, Wang H, Wang X, Liu Z, Xiao L, Bai H, Wu S. The P300 component decreases in a bimodal oddball task in individuals with depression: An event-related potentials study. *Clin Neurophysiol* 2018; **129**: 2525-2533 [PMID: 30366168 DOI: 10.1016/j.clinph.2018.09.012]

142   **Price GW**, Michie PT, Johnston J, Innes-Brown H, Kent A, Clissa P, Jablensky AV. A multivariate electrophysiological endophenotype, from a unitary cohort, shows greater research utility than any single feature in the Western Australian family study of schizophrenia. *Biol Psychiatry* 2006; **60**: 1-10 [PMID: 16368076 DOI: 10.1016/j.biopsych.2005.09.010]

143   **Allom V**, Mullan B, Hagger M. Does inhibitory control training improve health behaviour? *Health Psychol Rev* 2016; **10**: 168-186 [PMID: 26058688 DOI: 10.1080/17437199.2015.1051078]

144   **Spagnolo PA**, Goldman D. Neuromodulation interventions for addictive disorders: challenges, promise, and roadmap for future research. *Brain* 2017; **140**: 1183-1203 [PMID: 28082299 DOI: 10.1093/brain/aww284]

145   **Phalen H**, Coffman BA, Ghuman A, Sejdić E, Salisbury DF. Non-negative Matrix Factorization Reveals Resting-State Cortical Alpha Network Abnormalities in the First-Episode Schizophrenia Spectrum. *Biol Psychiatry Cogn Neurosci Neuroimaging* 2020; **5**: 961-970 [PMID: 31451387 DOI: 10.1016/j.bpsc.2019.06.010]

146   **Murphy M**, Whitton AE, Deccy S, Ironside ML, Rutherford A, Beltzer M, Sacchet M, Pizzagalli DA. Abnormalities in electroencephalographic microstates are state and trait markers of major depressive disorder. *Neuropsychopharmacology* 2020; **45**: 2030-2037 [PMID: 32590838 DOI: 10.1038/s41386-020-0749-1]

147   **Stoyanov D**, Kandilarova S, Paunova R, Barranco Garcia J, Latypova A, Kherif F. Cross-Validation of Functional MRI and Paranoid-Depressive Scale: Results From Multivariate Analysis. *Front Psychiatry* 2019; **10**: 869 [PMID: 31824359 DOI: 10.3389/fpsyt.2019.00869]

148   **Beresniewicz J**, Craven AR, Hugdahl K, Løberg EM, Kroken RA, Johnsen E, Grüner R. White Matter Microstructural Differences between Hallucinating and Non-Hallucinating Schizophrenia Spectrum Patients. *Diagnostics (Basel)* 2021; **11** [PMID: 33477803 DOI: 10.3390/diagnostics11010139]

149   **Oranje B**, van Berckel BN, Kemner C, van Ree JM, Kahn RS, Verbaten MN. P50 suppression and prepulse inhibition of the startle reflex in humans: a correlational study. *Biol Psychiatry* 1999; **45**: 883-890 [PMID: 10202576 DOI: 10.1016/s0006-3223(98)00128-0]

150   **Näätänen R**, Jacobsen T, Winkler I. Memory-based or afferent processes in mismatch negativity (MMN): a review of the evidence. *Psychophysiology* 2005; **42**: 25-32 [PMID: 15720578 DOI: 10.1111/j.1469-8986.2005.00256.x]

Published by **Baishideng Publishing Group Inc**
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA
Telephone: +1-925-3991568
E-mail: bpgoffice@wjgnet.com
Help Desk: https://www.f6publishing.com/helpdesk
https://www.wjgnet.com