

Journal of Psychiatry and Psychiatric Disorders

Volume 1, Issue 3

Review Article

Defining Borderline Personality Disorder Impulsivity: Review of Neuropsychological Data and Challenges that Face Researchers

Jean Gagnon^{1,2,3}

¹Department of Psychology, University of Montreal, Quebec, Canada

²Centre for Interdisciplinary Research in Rehabilitation of Greater Montreal, Canada

³Center for Research in Neuropsychology and Cognition (CERNEC), Canada

***Corresponding Author:** Jean Gagnon, Department of Psychology, University of Montreal, Centre-ville station, Montreal, Quebec, Canada, Fax: 514 343-2285; E-mail : jean.gagnon@umontreal.ca

Received: 31 May 2017; **Accepted:** 05 June 2017; **Published:** 12 June 2017

Abstract

It has been pointed out that the definition of BPD impulsivity would be improved by incorporating neurobehavioral models in order to bridge the research and the DSM behavioral criterion. Moeller et al. [1] have proposed three neuropsychological diagnostic criterions related to impulsivity in psychiatric disorders: (1) rapid, unplanned reactions to stimuli before complete processing of information; (2) lack of regard for long-term consequences and; (3) decreased sensitivity to negative consequences of behavior. The goal of this paper was to review the neuropsychological literature of BPD impulsivity in line with these neuropsychological diagnostic criterions to verify if the evidence from neuropsychological data and measurements is sufficiently strong to be integrated into the

DSM definition of BPD impulsivity. Results of the review highlight some evidence regarding neuropsychological deficits in BPD patients that may be underlying their impulsive self-damaging behaviors. However, at least five methodological challenges are pointed out and need to be addressed before these deficits can be successfully integrated into a definition of BPD impulsivity. Some solutions are proposed to face the main challenges in studying impulsivity in BPD.

Keywords: Borderline personality disorder; Impulsivity; Neuropsychology; Inhibition; Delay discounting; Decision making

1. Introduction

Borderline personality disorder (BPD) is a complex disorder characterized by several symptoms. But these patients are particularly known for their wide range of impulsive and risky behaviors which are a target priority in cognitive behavioral therapy in order to protect the patient as well as to ensure the continuation of therapy [2]. Impulsivity is one of the main factors underlying the symptomatology of the disorder and is considered as a key component of neurobehavioral models of BPD [3]. Moreover, the rate of prevalence of impulsive behaviors is very high in BPD, as can be seen in a study showing that the prevalence of various impulsive actions ranges from 43% to 99% [4]. Longitudinal studies have shown that impulsivity predicts BPD psychopathology at follow-up [5] and some impulsive patterns such as self-mutilation have a quick declining rate over time but some others such as substance abuse or excessive spending seem to be relatively stable over a period of 7 years [6].

Neuropsychology is a promising approach to develop diagnostic measures and etiological models of BPD impulsivity. In fact, it has been more than 25 years that neuropsychology has focused its attention on borderline personality disorder with the aim of describing its cognitive dysfunctions. Over the years, neuropsychological approach and methods evolve rapidly and neuropsychological deficits in several functional domains have been identified in BPD. Consequently, BPD self-damaging behaviors can be seen as the reflection of underlying cognitive impairments, notably deficits of inhibition. Moreover, several authors [1, 7, 8] have pointed out that the definition of BPD impulsivity should incorporate neurobehavioral models in order to bridge the research and the DSM. The goal of this paper is to review the neuropsychological literature of BPD impulsivity in order to verify if the evidence from neuropsychological data and measurements is sufficiently strong to be integrated into the DSM definition of BPD impulsivity and to propose solutions to the main challenges that face researchers in studying impulsivity in BPD.

2. Defining Borderline Impulsivity

The DSM-IV [9] and DSM-5 [10] define borderline impulsivity as potentially self-damaging behaviors in at least two areas such as spending, sex, substance abuse, reckless driving or binge eating. This definition is strictly based on the expression of behavior regardless of the mechanisms explaining why a person acts impulsively. According to several studies [11], one of the mechanisms that is likely to explain borderline impulsivity is a lack of inhibition. If we choose to define impulsivity as a disinhibition of mood and behavior, four other criteria could be related to impulsivity that is unstable interpersonal relationships, recurrent suicidal or self-mutilating behavior, affective instability and inappropriate anger and problems in controlling it. The opposition between the DSM behavioral criterion and a lack of inhibition to define impulsivity illustrates two very different ways of defining BPD impulsivity. The first one is based on the negative outcome of the action, whereas the second one is based on a putative underlying mechanism.

The DSM behavioral criterion to define BPD impulsivity has a number of limitations. Although behaviors such as alcohol abuse or excessive spending may occur impulsively on some occasions, individuals may also engage in these behaviors in a nonimpulsive manner on other occasions [12]. Moreover, even when the behavior is engaged in an impulsive manner, it could be the product of a predisposition or the result of an emotional or intoxication state [13]. Also, the temporal instability of impulsive-like behaviors that has been described in longitudinal studies can hinder a more temporal stability of their underlying psychological processes [14]. Indeed, symptomatic behaviors can reflect attempts at adapting to or coping with pathological impulsivity trait. Otherwise, important aspects of impulsivity such as lack of planning or perseverance are not captured in a pure behavioral approach [1]. Finally, the DSM definition of impulsivity can benefit from various findings of biological and psychological studies to determine who is impulsive, why he is impulsive and how to treat his impulsivity [15].

Moeller and collaborators [1] have proposed a definition that can be used to bridge the research and the DSM: Predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individuals or to others. The key features of the definition are predisposition which refer to a pattern of behavior rather than a single act; rapid unplanned reactions refer to actions that occur before the opportunity to consciously weigh the consequences of an act and this feature separates impulsivity from impaired judgment or compulsive behaviors in which planning occurs before behavior; and finally

without regard to the consequences of these actions refers to decision-making style of impulsive individuals and to the social aspect of impulsivity. According to the authors, a multidimensional approach combining self-report measures, behavioral laboratory measures and event-related potentials would permit to distinguish impulsive patients from non-impulsive patients, and identify psychological mechanisms underlying impulsive behaviors in order to guide treatment. In an effort to incorporate biological and behavioral models into a definition of impulsivity, these authors have proposed to include three neuropsychological diagnostic criteria: (1) rapid, unplanned reactions to stimuli before complete processing of information; (2) lack of regard for long-term consequences and; (3) decreased sensitivity to negative consequences of behavior.

These three criteria are based on findings from neuropsychological tasks measuring inhibition functions, delay discounting and decision making. The next section will review the performance of BPD patients on these tasks related to impulsivity.

3. Inhibition-related Functions

Even though inhibition is an old concept introduced in psychology and physiology in the beginning of the 19th century [16], the interest in the concept of inhibition in the explanation of impulsivity-related disorders is relatively recent and has increased during the past 20 years with the neurosciences [17]. At the behavioral level, disinhibition refers to an ongoing pattern of failing to inhibit, or continuing to engage in, certain appetitive behaviors that have previously led to aversive consequences [18]. For example, the individual with alcohol dependence continues to drink to excess despite repeatedly experiencing such negative outcomes as loss of friends, spouse, health, and job. At the cognitive level, it refers to any mechanism that reduces or dampens neuronal, mental, or behavioral activity [19]. A number of tasks have been developed and used to tap these processes. As it is the case with impulsivity, inhibition is conceptualized as a multidimensional construct rather than a single concept. With the use of statistical techniques, it has been possible to extract the common variance among multiple tasks and identify at least three different dimensions of inhibition [17]: (1) prepotent response inhibition; (2) resistance to distractor interference; (3) resistance to proactive interference.

3.1 Prepotent response inhibition

Prepotent response inhibition is the first and most investigated inhibition-related function in BPD. It refers to the ability to deliberately withhold or suppress dominant, automatic, or prepotent responses [20]. The two most common

paradigms to assess these processes are the Go/no-go task [21] and the Stop-signal Task [22]. Both tasks are composed of Go trials during which the participant is requested to answer. The Go/no-go task adds No-go trials during which a distinct stimulus from the Go is presented. Here, the participant is requested to not answer, to withhold his response. An inhibition failure happens when the participant answers during a No-go trial, which is called error of commission. The Stop-signal task consists of Go and No-go trials. During No-go trials, first a Go stimulus is presented, followed by a stop-signal after a certain delay. Even though a stimulus Go is presented, the Stop-signal requires a response suppression. An inhibition failure happens when the participant answers during a Stop-signal trial. Inhibition is an important executive function in our daily life when we have to withhold an inappropriate action or when there is a change in the environment and an ongoing action becomes suddenly inappropriate.

In the first study on prepotent response inhibition in BPD by Nigg and collaborators [11], the results obtained in a group composed of BPD and attention deficit and hyperactivity disorder (ADHD) patients showed that BPD symptoms were correlated with poor performance on several executive functions tasks. Also, the poor response inhibition as measured by the Stop-signal task was specific to BPD symptoms even when other personality disorder symptoms were controlled for. Moreover, poor response inhibition was a unique predictor of BPD symptoms when other executive functions problems were controlled. However, when ADHD symptoms were entered as a predictor in the statistical model, response inhibition ceased to be significantly associated with BPD symptoms, suggesting that BPD and ADHD symptoms probably overlap in the explanation of response inhibition deficit.

Studies that followed shown that BPD patients made more errors of commission, revealing problems to inhibit a prepotent response, and that poor performance was inconsistently correlated with a trait impulsivity questionnaire across studies [23, 24]. Recent studies have called upon event-related potential technique to investigate response inhibition in BPD. In an event-related potential study [25] where the participants performed a Go/no-go task, the authors focused on the Nogo-P3, which is a positive waveform with a peak latency around 300 ms that can be observed after a No-go stimulus is presented. The Nogo-P3 is related to response inhibition. BPD patients showed reduced Nogo-P3 amplitudes compared to controls, and this event-related potential component was negatively correlated with the score on a trait impulsivity questionnaire for the entire group of participants.

Three remarks can be made about response inhibition in BPD. First, BPD patients usually scored higher than controls on trait impulsivity questionnaires [26-32]. In contrast, they often show similar performances as controls on inhibition response tasks [33, 34]. Secondly, most studies did not compare BPD patients with ADHD patients and the ones that did so had obtained negative results [35, 36]. Finally, event-related potential measures proved to be useful to complement behavioral measures by showing positive results where such results were impossible to obtain with behavioral measures alone [25].

3.2 Resistance to distractor interference

The second inhibition-related function is resistance to distractor interference. This function refers to the ability to resist or resolve interference from information in the external environment that is irrelevant to the task at hand [11, 17, 37]. It has been shown that this inhibition-related function is closely related to the prepotent response inhibition [17]. Indeed, both types require focusing on the task goal in order not to get distracted by external stimuli and not to initiate an automatic response that is inappropriate in the situation. For example, while driving, one has to focus on the road and the directions and not to be distracted by the cell phone and not miss a red light. This inhibition-related function is commonly measured by the Stroop task.

In the Stroop task [38], there are at least two conditions. In the first condition, the neutral condition, examinees name the ink color of a series of rectangles arranged in rows and columns. In the second condition, the interference condition, examinees must name the ink color and ignore the content of the word, which is the name of another color. This condition is more demanding because it requires the inhibition of the strong tendency to read the word. The increased reaction time in the interference condition compared to the neutral condition is called the interference effect. Resistance to distractor interference does not seem to be problematic in BPD as three studies failed to show group differences between BPD patients and controls on the interference effect [34, 36, 39].

3.3 Resistance to proactive interference

The third inhibition-related function is resistance to proactive interference. This function refers to the ability to resist memory intrusions from information that was previously relevant to the task but has since become irrelevant [11, 17]. Friedman and Miyake [17] have shown that this inhibition-related function is not related to either prepotent response inhibition or resistance to distractor interference. Among the existing tasks to assess resistance to proactive interference, is the Directed forgetting task.

The Directed forgetting task [40] requires the participants to view words, some of which they are instructed to remember and some they are instructed to forget. They are then tested on both the to-be-remembered words, the R-cues, and the to-be-forgotten words, the F-cues. Among healthy controls, the directed forgetting effect refers to the fact that participants tend to remember words they were instructed to remember and forget words they were instructed to forget.

Korfine and Hooley [41] called upon this paradigm to investigate memory bias in BPD patients, and used three types of words: neutrally valenced, positively valenced and borderline-related words. Borderline-related words were words presumably salient to individuals with BPD, and included themes of abandonment, rejection, anger and rage, self-harm, and others being uncaring. It was expected that BPD patients would show a memory bias for borderline words. Results indicated that BPD patients recalled significantly more of the borderline words from the forget condition than did controls, suggesting the continuation of encoding the words even after they saw the forget instruction. This continuation to encode could reflect a deficit to inhibit proactive interference in working memory. Moreover, the number of recalled words in the forget condition were correlated with BPD symptomatology. According to the authors, cognitive distortions and intrusion of affect into information processing that are observed in BPD patients might be related to a difficulty to inhibit negative thoughts about social rejection for example. This finding of an enhanced recall of negative information in BPD participants, despite the instruction to forget it, has been replicated by another study [27].

3.4 Effect of emotion on inhibition in BPD

Given that self-destructive behaviors usually happen in the context of negative affect [42] and that affective dysregulation is, with impulsivity, a central symptom in BPD [43], recent studies investigated the interaction between emotion and inhibition-related functions. More specifically, the researchers were interested to know if inhibition capacities of BPD patients were diminished in an emotional condition compared to a neutral condition. The assumption behind this question was that inhibition of emotion would be one of the main mechanisms in affective regulation, and that it enables us to regulate emotions and express them at the moment and in a manner that we want to [44]. Silbersweig and collaborators [45] called upon the fMRI technique to answer this question. The authors have investigated the functioning of prefrontal control mechanisms in the setting of negative emotional

states. They developed an emotional Go/no-go task with verbal stimuli containing themes salient for individuals with BPD and for which the affective valence could be positive, negative or neutral. Under conditions of behavioral inhibition in the context of negative emotion, BPD patients, compared with controls, made more errors of commission, suggesting a deficit in response inhibition. Also, in the same conditions, BPD patients showed decreased ventromedial prefrontal activity and increased activity of the amygdala and associated structures. Moreover, these neurodynamic changes were correlated with a questionnaire measuring negative emotional and lack of constraint temperamental traits. According to the authors, the results suggest a reciprocal functional relationship between a ventromedial prefrontal cortex playing a top-down inhibitory role, and an amygdala and associated structures playing a bottom-up emotional interfering role.

Alternatively, researchers made use of the Emotional Stroop task to investigate the effect of emotion on resistance to distractor interference [46]. In this version of the test, the participants must name the color of the stimuli as they do in the standard version, but this time the words have a positive, negative or neutral valence. Also, the words can be personally relevant or not relevant to the participants. It is assumed that the interference effect on the emotional Stroop task is related to a difficulty to inhibit emotional words, resulting in attentional bias. Studies that have manipulated the valence of stimuli only, for example comparing neutral versus negative stimuli, did not find group differences between BPD patients and controls on the interference effect [27, 47]. In contrast, studies that have manipulated not only the valence of the words but also their relevance for the participants obtained positive results more often. For example, Arntz and collaborators [48] found that BPD patients had difficulty to inhibit interference from words associated with BPD danger signals, for example rejection from others, childhood traumas and negative self-views as being bad or helpless. However, this effect was not specific to BPD patients as cluster C patients did show the same difficulty. In Sieswerda and collaborators' study [49], it was possible to obtain an emotional Stroop effect specific to BPD patients compared to cluster C patients. In this study, schema-related negative words were used. Finally, a last study [47] showed that group differences between BPD patients and controls were found but only for words related to personal stressful situations with current relevance for the participants in comparison to words with less current importance. However, in this last study, only BPD patients with post-traumatic stress disorder showed reduced inhibitory functioning compared to BPD without post-traumatic stress disorder.

Other studies investigated the effect of stress or emotion on state of impulsivity. State of impulsivity refers to the tendency to act impulsively in certain specific situations but not all the time [13]. For example, state-dependant impulsivity in individuals with BPD may be related to emotional states. State of impulsivity can be measured by questionnaire with items such as “I am in the sort of mood in which I can say whatever comes into my head”. Two studies have shown that BPD patients with or without ADHD reported that their state of impulsivity was increased after an experimental stress induction [35, 50]. In a series of two studies with non-clinical participants, Chapman and collaborators [51, 52] were interested in verifying if a negative emotional state would increase impulsive responses in a passive avoidance learning task. In a passive avoidance learning task, the participants must learn to inhibit a response that was previously punished, for example with the withdrawal of money. In the first study [51], the current emotional state was assessed by a questionnaire whereas, in the second study [52], the emotional state was manipulated by a fear induction. The fear induction consisted of watching a horror movie’s extract. Contrasting results were obtained between the two studies. In the first one, participants who were high in BPD features and were in a current negative emotional state committed fewer errors of inhibition than individuals high in BPD features but who were not in a negative emotional state. In the second study using a mood induction, participants who were high in BPD features committed a greater number of impulsive responses than low-BPD participants and these group differences were observed in the fear condition but not in the neutral condition. According to the authors, the results of those two studies suggest that incidental emotional states that accompany a situation are not strong enough to impact on impulsivity. In contrast, an acute emotional stressor may result in disruptions in behavioral control. According to the authors, impulsivity among persons with BPD features may not be a trait-like tendency but depends largely on emotional context.

The previous studies have shown that deficits of inhibition in BPD patients would be partially attributed to their negative emotional state. Some researchers interpret this as the result of a reciprocal influence between the inhibitory capacity and the intensity of emotion [45]. Other researchers question the very existence of trait impulsivity in BPD and would rather put forward the idea that impulsive behaviors of BPD patients are the result of a state of impulsivity or a by-product of their affective dysregulation [3]. Independently of the interpretation, two critiques can be said about studies that made use of emotional and personal stimuli to study the effect of emotion in inhibition. Firstly, there is a methodological bias in the selection of stimuli. Indeed, the emotionally relevant words used in some studies were based on schemas that are relevant to the BPD group only, and they were not based on

schemas that could be relevant to Cluster C patients or even control participants. Consequently, it is possible that group differences found in those studies would disappear if the words were chosen in respect with schemas of each group. This would increase the probability of getting a stronger emotional Stroop effect across all participants. The second limitation relates to the fact that most of those studies did not investigate the relationship between the interference effect and a measurement of impulsivity or BPD symptoms, and the only study that had included such a measurement did not find a significant correlation [27].

4. Delay Discounting

The performance tasks used in neuropsychological studies to assess inhibition-related functions formed one group of behavioral paradigms of impulse control. There exists a second group of impulse control paradigms focusing on more complex behavioral patterns, such as tasks assessing delay discounting. The delay discounting principle refers to the fact that the value of a reward is discounted as a function of the delay before receiving it. According to this principle, impulsive individuals prefer smaller immediate rewards over larger delayed rewards. In return, they prefer delayed larger negative consequences over immediate smaller negative consequences. In BPD patients, this preference could explain why they choose to engage in self-harm behaviors because of the immediate benefits of temporary emotional distraction and relief from intense negative affect, despite potential long-term negative consequences. The motivational impulse control is commonly assessed with the Delay discounting task.

In the Delay Discounting task, the participant is given two choices. In the delayed choice, there is a fixed larger reward that can be received after a certain delay, for example 1000 \$ in one month. In the immediate choice, the amount of the reward is manipulated over successive trials, for example going up from 600 \$ to 900 \$. The goal of the task is to estimate the value of a delayed reward which is called the indifference point where there is a switch from a larger later reward to a smaller sooner reward. In other words, the immediate reward generates the same value as the delayed reward.

In a first study [53], the participants were given a choice between a smaller immediate real monetary reward, which was 5 cents, or a larger but progressively delayed reward, which was 15 cents. During the delayed choice, the participants must actually wait for a maximum delay of 2 minutes. Results showed that BPD patients did not select the short-delay more frequently than the controls, but their averaged longest delay before reward dropped by 50%

from the first to the second session of the task. Controls endured identical maximum delays of reinforcement across the two sessions. This pattern of results reflected a change in the BPD patients' strategy to avoid long delays in large-reward reinforcement in the second session of the task. Indeed, the BPD subjects tended to alternate between the short and long-delayed response options. However, the interpretation of the results remains ambiguous as the change in strategy may not indicate an impulsive style in choice preferences but may simply reflect the fact that BPD patients were less tolerant than controls toward the experimental condition where they had to wait for more than a minute in an isolated booth. The absence of correlation between performance on the task and a trait impulsivity questionnaire is consistent with this interpretation.

In a standard delay discounting task study [54], it was possible to confirm a preference for immediate gratification and discounting for delayed rewards. Lawrence and collaborators administered the task before and after a mood induction with the intention to verify if a change in mood would exacerbate the preference for an immediate reward. The results indicated that the BPD patients had a greater preference for immediate gratification and a higher rate of discounting the delayed reward than the controls. Also, the rate of discounting did not change for the BPD group after mood induction whereas there was a change for controls, indicating less discounting of the delayed rewards following mood induction. In other words, controls valued the delayed reward more after their mood change, suggesting a shift toward greater self-control. For BPD patients, even though they experienced a mood change as the controls did, this did not alter their reward-based decision-making, suggesting a failure to reduce impulsivity after rejection. Finally, the rate of discounting was correlated with non-planning impulsivity trait in BPD group, suggesting that the tendency to be present-oriented with limited regard for future planning leads to an immediate gratification decision-making style.

5. Decision Making

According to the somatic marker hypothesis [55], decision making is a process that depends both on conscious and unconscious processes, and it is influenced by our emotions. In fact, when facing choices or situations when the future is uncertain, our emotions through somatic signals help us to make advantageous decisions by telling us that some behaviors will be associated with positive consequences whereas other behaviors will be associated with negative consequences. Several impulsive behaviors exhibited by BPD patients such as self-destructiveness, self-harm behaviors and suicidal attempts are consistent with difficulties in emotionally guided decision-making. The most known paradigm to assess these processes is the Iowa Gambling task.

The Iowa Gambling task is designed to study the integration of emotion and cognition in decision processes [56]. It simulates real-life decision making with uncertainty concerning premises and outcomes as well as rewards and punishments. The participants are given play money and have to make a series of card selections out of four decks, with the goal of maximizing profit. Immediately after every choice, the participants receive a financial reward, although in some cases they also receive a financial punishment. Two of the decks, A and B, are considered disadvantageous and result in immediate large rewards, but also result in higher punishments at unpredictable points. The other two decks, C and D, are considered advantageous and result in immediate modest rewards, but lower punishments as well. In the long run, choosing from the advantageous decks would result in a net gain, while choosing from the disadvantageous decks would result in a net loss. The score is defined as the number of choices from the advantageous decks minus the number of choices from the disadvantageous decks over 100 trials. Through somatic markers, normal subjects are capable of anticipating the consequences associated with each deck and end up by making the right decision. In contrast, patients with a damage to ventromedial prefrontal cortex show a persistence of disadvantageous choices, a deficit interpreted as the consequence of insensitivity to future consequences, positive or negative [57].

Several studies have shown that BPD patients showed less advantageous choices on the Iowa Gambling task than did the healthy comparison subjects [39, 58]. For example, BPD patients without a substance abuse disorder made fewer advantageous choices than controls [39]. However, BPD with a substance abuse disorder made fewer advantageous choices than BPD without such a disorder, a result that is not surprising given that reduced performance has been found in patients with substance dependency [59]. However, other studies showed that BPD patients have normal performance in the Iowa Gambling task [60, 61].

In order to better understand the reason why BPD patients make disadvantageous choices, some studies used modified versions of the Gambling task to investigate the cognitive processes underlying their poor performance, for example, the feedback processing. The Iowa Gambling task simulates real-life situations in which the outcome of a decision is unpredictable. However, Svaldi and collaborators [62] reasoned that, in everyday life, individuals with BPD are most often explicitly aware of the deleterious long-term consequences of their impulsive behaviors. So they used a modified version of the Gambling task called the Game of Dice Task in which there are explicit rules for

gains and losses and where winning probabilities are obvious, stable and predictable. The results showed that BPD patients still displayed significantly more risky decision-making behaviors and these deficits in decision-making were associated with BPD symptom severity. Moreover, BPD patients continued to make risky decisions following negative feedback after a risky choice. They also continued to select advantageous choices less often after positive feedback. These results suggest a difficulty to process the feedback information. Another study [63] showed that BPD patients exhibited altered processing of information about potential losses but only when the probability of gains was simultaneously high. This result suggests a failure to properly process negative consequences in circumstances when there is a high probability of reward. For example, BPD patients would not process the probability of getting hurt after an impulsive action when they expect that this behavior will result in the reduction of intense negative affect. Finally, event-related potentials studies have identified three components in decision-making: (1) error-related negativity or ERN, a waveform that arises following the execution of erroneous responses [64]; (2) feedback-related negativity or FRN, elicited by negative performance feedback when outcomes are worse than expected [65] and (3) the feedback-related P300 associated with the significance or the value attributed to the feedback [66]. Several studies have shown that BPD patients, in contrast with controls, exhibited smaller ERN and FRN amplitudes, suggesting that BPD patients would exhibit a reduction in the monitoring of their actions and do not learn from their errors [67-70]. Also, these components were negatively correlated with a trait impulsivity questionnaire. Alternatively, the P300 amplitudes following a negative feedback were increased in BPD patients, suggesting that they would attribute more meaning to negative feedback information than positive feedback [69].

To summarize delay discounting and decision making data in BPD, there is clear evidence that BPD patients exhibit deficits in impulse control when motivational and decisional processes are involved. These deficits seem associated with their impulsivity trait and their symptoms. These deficits might be associated with a failure to properly process feedback information and to monitor action. However many questions remain open to more investigation such as the role played by a comorbidity with a substance abuse disorder in the rate of delay discounting in BPD patients, and the influence of the reinforcement valence in their poor decision-making.

6. Challenges that Need to be Addressed

The critical review of neuropsychological studies highlighted some evidence regarding neuropsychological deficits in BPD patients that may be underlying their impulsive self-damaging behaviors. These deficits relate to prepotent

response inhibition and resistance to proactive interference in the context of emotion, a preference for immediate gratification and discounting for delayed rewards, and a failure to properly process feedback information and to monitor action in decision making. However, at least five methodological challenges have to be resolved before these deficits can be integrated into a definition of BPD impulsivity.

The first challenge relates to the heterogeneous findings on neuropsychological deficits found among BPD patients. The high proportion of BPD patients with a neurological history suggested to some researchers the existence of a subgroup of BPD patients [32, 71]. Also, various degrees of severity and areas of dysfunction could simply reflect the normal variation in cognitive functioning between individuals or the co-occurrence of other mental disorders [72]. However, it is possible that one of the most problematic sources of variation could originate in the diagnostic criteria of BPD itself. Indeed, as a result of the polythetic diagnosis system, 151 possible diagnostic presentations can be found among BPD patients [73]. As recommended by Paris [74], one possible solution to the complex nature of BPD would be to diagnose it with a narrower set of criteria that cover all of its domains: affective instability, impulsivity, unstable relationships and cognitive defects. In keeping with this recommendation, future neuropsychological research should call upon diagnostic instruments in which the BPD diagnosis requires symptoms among all of these domains, such as the Revised Diagnostic Interview for Borderlines [75]. Efforts toward a more homogeneous diagnostic profile seem to be a prerequisite to all future neuropsychological studies.

A second challenge relates to the clinical relevance of neuropsychological performance found among BPD. Although studies found group differences, cognitive impairments associated with BPD are described as subtle [72]. Moreover, the individual test scores of BPD patients are generally in the average range [72]. Thus, even though deficits stand out in statistical group differences and that the performances of BPD patients are correlated with their symptoms, a question remains: Are they substantial enough to be seen as deficits that justify a neuropsychological intervention? To answer this question, single case studies describing patients with different levels of performance could be useful to draw particular profile in terms of cognitive impairment.

A third challenge relates to the causal link between neuropsychological deficits and BPD symptoms, including behavioral impulsivity. Indeed, studies that have shown relationships between cognitive deficits and BPD symptoms were correlational. Thus, longitudinal studies are needed to make progress on the issue. Furthermore, longitudinal

studies would benefit from using the event-related potential technique in order to obtain objective data on cognitive deficits among BPD patients and verify if they can predict the emergence of symptoms over time.

How to handle comorbidity with substance abuse disorder and attention deficit and hyperactivity disorder is certainly another challenge to researchers. Regarding the first comorbidity, it seems difficult to believe that the exclusion of BPD patients with a substance abuse disorder in research sampling could result in representative samples of BPD patients. This is because substance abuse may represent a maladaptive strategy to regulate intense negative affect in BPD [7]. Also, there exists an overlap between BPD and substance abuse disorder because the diagnostic criterion for impulsivity in BPD can be met with impulsive substance abuse. Moreover, levels of impulsivity and negative affect may enter into a reciprocal relationship during the development of one disorder and may contribute to the development of the second disorder. Alternatively, if BPD patients with a substance abuse disorder are included in samples, it would be difficult to disentangle the respective effect of each disorder on impulsivity. One possible solution would be to determine if the substance abuse is primary or secondary to BPD [76]. For example, in contrast with substance abuse disorder in which behaviors are caused by substance-seeking, the substance use by an individual with BPD can be a craving to soothe himself or herself in the context of dysphoric feeling states. Also, contrary to substance abuse disorder, the type of substance is not very important among BPD patients and their abuse tends to be episodic and impulsive. The use of an instrument such as the Revised Diagnostic Interview for Borderlines [75] can help to establish the differential diagnosis. Regarding the ADHD, given that almost 50% of adult BPD patients report childhood ADHD [77, 78] and that childhood ADHD symptoms predict higher rates of BPD during young adulthood [79, 80], it seems appropriate to systematically include a subgroup of BPD patients with ADHD and a subgroup of BPD patients without ADHD in future neuropsychological studies. This is consistent with the fact that some researchers argue that BPD might be subdivided in two subgroups according to the presence or absence of comorbid ADHD [81].

One last challenge would be to better understand the role of emotional, situational and personality variables in impulse control deficits in BPD. Indeed, deficits of inhibition are modulated by emotional states or the current relevance of negative stimuli. Given that inhibition processes are composed of several functions, future studies should try to better differentiate which emotion contributes to disrupt which component of inhibition in which situation. For example, it has been shown that fear can hamper the inhibition of a previously punished response [52].

What can be said about other borderline-related feelings such as anger, abandonment, rage, sadness and anxiety? What can be said about the other components of inhibition? Otherwise, state impulsivity should be investigated in several contexts, not only in the state of negative emotions. Indeed, one could study the differences between a social versus a non-social situation. For example, what about social rejection versus a non-social frustration? Would they have the same effect on impulse control processes? Finally, personality-related variables such as personal schemas, attributional bias or defensive styles can also interact with inhibition processes in the emergence of impulsive behaviors. It would be important to include such personality variables in the future studies on inhibition, delay discounting and decision making in BPD.

7. Conclusion

The DSM behavioral criterion to define BPD impulsivity has a number of limitations. The definition of BPD impulsivity would be improved by incorporating neurobehavioral models in order to bridge the research and the DSM. Specifically, a multidimensional approach combining self-report measures, behavioral laboratory measures and event-related potentials would permit to distinguish impulsive patients from non-impulsive patients, and identify psychological mechanisms underlying impulsive behaviors in order to guide treatment. In an effort to incorporate biological and behavioral models into a definition of impulsivity, Moeller and collaborators [1] have proposed to include three neuropsychological diagnostic criteria: (1) rapid, unplanned reactions to stimuli before complete processing of information; (2) lack of regard for long-term consequences and; (3) decreased sensitivity to negative consequences of behavior. The goal of this paper was to review the neuropsychological literature of BPD impulsivity in line with these neuropsychological diagnostic criteria to verify if the evidence from neuropsychological data and measurements is sufficiently strong to be integrated into the DSM definition of BPD impulsivity.

The critical review of neuropsychological studies highlighted some evidence regarding neuropsychological deficits in BPD patients that may be underlying their impulsive self-damaging behaviors. These deficits relate to prepotent response inhibition and resistance to proactive interference in the context of emotion, a preference for immediate gratification and discounting for delayed rewards, and a failure to properly process feedback information and to monitor action in decision making. However, at least five methodological challenges have been pointed out and need to be addressed before these deficits can be successfully integrated into a definition of BPD impulsivity and

some solutions have been proposed to face the main challenges in studying impulsivity in BPD. It is wished that future studies will take into account these methodological issues considering the importance of better defining BPD impulsivity given its dramatic consequences and of finding interventions that aim the underlying cognitive and psychological mechanisms.

References

1. Moeller FG, Barratt ES, Dougherty DM, et al. Psychiatric aspects of impulsivity. *The American Journal of Psychiatry* 158 (2001): 1783-1793.
2. Linehan MM. Dialectic behavioral therapy: a cognitive behavioral approach to parasuicide. *Journal of Personality Disorders* 1 (1987): 328-333.
3. Sebastian A, Jacob G, Lieb K, et al. Impulsivity in Borderline Personality Disorder: A matter of disturbed impulse control or a facet of emotional dysregulation? *Current Psychiatry Reports* 15 (2013): 339-346.
4. Mary C. Discriminating borderline personality disorder from other axis II disorders. *The American Journal of Psychiatry* 147 (1990): 161-167.
5. Links PS, Heslegrave R, Reekum RV. Impulsivity: Core aspect of borderline personality disorder. *Journal of Personality Disorders* 13 (1999): 1-9.
6. Zanarini MC, Frankenburg FR, Hennen J, et al. The longitudinal course of borderline psychopathology: 6-year prospective follow-up of the phenomenology of borderline personality disorder. *American Journal of Psychiatry* 160 (2003): 274-283.
7. Bornoalova MA, Lejuez CW, Daughters SB, et al. Impulsivity as a common process across borderline personality and substance use disorders. *Clinical Psychology Review* 25 (2005): 790-812.
8. Lenzenweger MF, Clarkin JF, Fertuck EA, et al. Executive neurocognitive functioning and neurobehavioral systems indicators in borderline personality disorder: A preliminary study. *Journal of Personality Disorders* 18 (2004): 421-438.
9. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders—IV—Text Revision* (4th Edn.) Washington, DC (2000).
10. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders* (5th Edn.) Washington, DC (2013).

11. Nigg JT, Silk KR, Stavro G, et al. Disinhibition and borderline personality disorder. *Development and Psychopathology* 17 (2005): 1129-1149.
12. Tomko RL, Solhan MB, Carpenter RW, et al. Measuring Impulsivity in Daily Life: The Momentary Impulsivity Scale. *Psychological Assessment* 26 (2014): 339-349.
13. Wingrove J, Bond AJ. Impulsivity: a state as well as trait variable. Does mood awareness explain low correlations between trait and behavioral measures of impulsivity? *personality and Individual Differences* 22 (1997): 333-339.
14. Verheul R. Impulsivity in the diagnosis of borderline personality disorder, in Phenotype Conference of the Borderline Personality Disorder Research Foundation. New York, NY (1996).
15. Barratt ES. *Impulsivity: Integrating cognitive, behavioral, biological, and environmental data* (1993).
16. Macmillan M. The concept of inhibition in some nineteenth century theories of thinking. *Brain & Cognition* 30 (1996): 4-19.
17. Friedman NP, Miyake A. The relations among inhibition and interference control functions: a latent-variable analysis. *Journal of Experimental Psychology: General* 133 (2004): 101-135.
18. Endres MJ, Rickert ME, Bogg T, et al. Externalizing Psychopathology and Behavioral Disinhibition: Working Memory Mediates Signal Discriminability and Reinforcement Moderates Response Bias in Approach-Avoidance Learning. *Journal of Abnormal Psychology* 120 (2011): 336-351.
19. Clark JM. Contributions of inhibitory mechanisms to unified theory in neuroscience and psychology. *Brain & Cognition* 30 (1996): 127-152.
20. Cyders MA, Coskunpinar A. Measurement of constructs using self-report and behavioral lab tasks: Is there overlap in nomothetic span and construct representation for impulsivity? *Clinical Psychology Review* 31 (2011): 965-982.
21. Casey BJ, Trainor RJ, Orendi JL, et al. A Developmental Functional MRI Study of Prefrontal Activation during Performance of a Go-No-Go Task. *Journal of Cognitive Neuroscience* 9 (1997): 835-847.
22. Logan GD. *On the ability to inhibit thought and action: a users' guide to the stop signal paradigm* (1994).
23. Coffey SF, Schumacher JA, Baschnagel JS, et al. Impulsivity and risk-taking in borderline personality disorder with and without substance use disorders. *Personality Disorders: Theory, Research, and Treatment* 2 (2011): 128-141.

24. Rentrop M, Backenstrass M, Jaentsch B, et al. Response inhibition in borderline personality disorder: performance in a Go/Nogo task. *Psychopathology* 41 (2008): 50-57.
25. Ruchow M, Groen G, Kiefer M, et al. Response inhibition in borderline personality disorder: event-related potentials in a Go/Nogo task. *Journal of Neural Transmission* 115 (2008): 127-133.
26. Berlin HA, Rolls ET, Iversen SD. Borderline personality disorder, impulsivity, and the orbitofrontal cortex. *American Journal of Psychiatry* 162 (2005): 2360-2373.
27. Domes G, Winter B, Schnell K, et al. The influence of emotions on inhibitory functioning in borderline personality disorder. *Psychological Medicine* 36 (2006): 1163-1172.
28. Henry C, Mitropoulou V, New AS, et al. Affective instability and impulsivity in borderline personality and bipolar II disorders: similarities and differences. *Journal of Psychiatric Research* 35 (2001): 307-312.
29. Kunert HJ, Druecke HW, Sass H, et al. Frontal lobe dysfunctions in borderline personality disorder? Neuropsychological findings. *Journal of Personality Disorders* 17 (2003): 497-509.
30. Lemelin S, Villeneuve E. Impulsivity associated with the borderline personality disorder. *Revue Quebecoise de Psychologie* 24 (2003): 195-210.
31. van Reekum R, Links PS, Fedorov C. Impulsivity in borderline personality disorder, in *Biological and neurobehavioral studies of borderline personality disorder*, Silk KR, American Psychiatric Association: Washington, DC (1994) 1-22.
32. van Reekum R. Repeat neurobehavioral study of borderline personality disorder. *Journal of Psychiatry & Neuroscience* 21 (1996): 13-20.
33. Ferraz L, Vázquez M, Navarro JB, et al. Dimensional assessment of personality and impulsiveness in borderline personality disorder. *Personality and Individual Differences* 46 (2009): 140-146.
34. Jacob GA, Gutz L, Bader K, et al. Impulsivity in borderline personality disorder: impairment in self-report measures, but not behavioral inhibition. *Psychopathology* 43 (2010): 180-188.
35. Krause-Utz A, Sobanski E, Alm B, et al. Impulsivity in relation to stress in patients with borderline personality disorder with and without co-occurring attention-deficit/hyperactivity disorder: an exploratory study. *Journal of Nervous & Mental Disease* 201 (2013): 116-123.
36. Lampe K, Konrad K, Kroener S, et al. Neuropsychological and behavioural disinhibition in adult ADHD compared to borderline personality disorder. *Psychological Medicine* 37 (2007): 1717-1729.

37. Stahl C, Voss A, Schmitz F, et al. Behavioral components of impulsivity. *Journal of Experimental Psychology: General* 143 (2014): 850-886.
38. Golden CJ, Freshwater SM. Stroop color and word test. *Age* 15 (1978): 90.
39. LeGris J, Links PS, van Reekum R, et al. Executive function and suicidal risk in women with Borderline Personality Disorder. *Psychiatry Research* 196 (2012): 101-108.
40. Johnson HM. Processes of successful intentional forgetting. *Psychological Bulletin* 116 (1994): 274-292.
41. Korfine L, Hooley JM. Directed forgetting of emotional stimuli in borderline personality disorder. *Journal of Abnormal Psychology* 109 (2000): 214-221.
42. Linehan MM. *Cognitive-behavioral treatment of borderline personality disorder*. New York: Guilford Press (1993).
43. Lieb K. Borderline personality disorder. *Lancet* 364 (2004): 453-461.
44. Gross JJ. Emotion regulation: affective, cognitive, and social consequences. *Psychophysiology* 39 (2002): 281-291.
45. Silbersweig D, Clarkin JF, Goldstein M, et al. Failure of frontolimbic inhibitory function in the context of negative emotion in borderline personality disorder. *American Journal of Psychiatry* 164 (2007): 1832-1841.
46. Williams JM, Mathews A, MacLeod C. The emotional Stroop task and psychopathology. *Psychological Bulletin* 120 (1996): 3-24.
47. Wingefeld K, Mensebach C, Rullkoetter N, et al. Attentional bias to personally relevant words in borderline personality disorder is strongly related to comorbid posttraumatic stress disorder. *Journal of Personality Disorders* 23 (2009): 141-155.
48. Arntz A, Appels C, Sieswerda S. Hypervigilance in borderline personality disorder: A test with the emotional Stroop paradigm. *Journal of Personality Disorders* 14 (2000): 366-373.
49. Sieswerda S, Arntz A, Mertens I, et al. Hypervigilance in patients with borderline personality disorder: specificity, automaticity, and predictors. *Behaviour Research & Therapy* 45 (2006): 1011-1024.
50. Cackowski S, Reitz AC, Ende G, et al. Impact of stress on different components of impulsivity in borderline personality disorder. *Psychological Medicine* 44 (2014): 3329-3340.
51. Chapman AL, Leung DW, Lynch TR. Impulsivity and emotion dysregulation in borderline personality disorder. *Journal of Personality Disorders* 22 (2008): 148-164.

52. Chapman AL, Dixon-Gordon KL, Layden BK, et al. Borderline Personality Features Moderate the Effect of a Fear Induction on Impulsivity. *Personality Disorders-Theory Research and Treatment* 1 (2010): 139-152.
53. Dougherty DM, Bjork JM, Huckabee HC, et al. Laboratory measures of aggression and impulsivity in women with borderline personality disorder. *Psychiatry Research* 85 (1999): 315-326.
54. Lawrence KA, Allen JS, Chanen AM. Impulsivity in borderline personality disorder: reward-based decision-making and its relationship to emotional distress. *Journal of Personality Disorders* 24 (2010): 786-799.
55. Damasio AR, Everitt BJ, Bishop D. The somatic marker hypothesis and the possible functions of the prefrontal cortex. *Philosophical Transactions of the Royal Society of London - Series B: Biological Sciences* 351 (1996): 1413-1420.
56. Bechara A, Damasio H, Tranel D, et al. The Iowa Gambling Task and the somatic marker hypothesis: some questions and answers. *Trends in Cognitive Sciences* 9 (2005): 159-162.
57. Bechara A, Damasio AR, Damasio H, et al. Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition* 50 (1994): 7-15.
58. Maurex L, Zaboli G, Wiens S, et al. Emotionally controlled decision-making and a gene variant related to serotonin synthesis in women with borderline personality disorder. *Scandinavian Journal of Psychology* 50 (2009): 5-10.
59. Haaland VO, Landrø NI. Decision making as measured with the Iowa Gambling Task in patients with borderline personality disorder. *Journal of the International Neuropsychological Society* 13 (2007): 699-703.
60. Dowson J, Bazanis E, Rogers R, et al. Impulsivity in patients with borderline personality disorder. *Comprehensive Psychiatry* 45 (2004): 29-36.
61. McCloskey MS, New AS, Siever LJ, et al. Evaluation of behavioral impulsivity and aggression tasks as endophenotypes for borderline personality disorder. *Journal of Psychiatric Research* 43 (2009): 1036-1048.
62. Svaldi J, Philipson A, Matthies S. Risky decision-making in borderline personality disorder. *Psychiatry Research* 197 (2012): 112-118.
63. Kirkpatrick T, Joyce E, Milton J, et al. Altered emotional decision-making in prisoners with borderline personality disorder. *Journal of Personality Disorders* 21 (2007): 243-261.

64. Gehring WJ. The error-related negativity: an event related brain potential accompanying errors. *Psychophysiology* 27 (1990): S34.
65. Miltner WH, Braun CH, Coles MG. Event-related brain potentials following incorrect feedback in a time-estimation task: evidence for a 'generic' neural system for error detection. *Journal of Cognitive Neuroscience* 9 (1997): 788-798.
66. de Bruijn ER, Mars RB, Hulstijn W. It wasn't me... or was it? How false feedback affects performance, in Errors, conflicts, and the brain. *Current opinions on performance monitoring*, M. Ullsperger and M. Falkenstein, MPI of Cognitive Neuroscience: Leipzig (2004): 118-124.
67. de Bruijn ER, Grootens KP, Verkes RJ, et al. Neural correlates of impulsive responding in borderline personality disorder: ERP evidence for reduced action monitoring. *Journal of Psychiatric Research* 40 (2006): 428-437.
68. Ruchow M, Walter H, Buchheim A, et al. Electrophysiological correlates of error processing in borderline personality disorder. *Biological Psychology* 72 (2006): 133-140.
69. Schuermann B, Kathmann N, Stiglmayr C, et al. Impaired decision making and feedback evaluation in borderline personality disorder. *Psychological Medicine* 41 (2011): 1917-1927.
70. Vega D, Soto À, Amengual JL, et al., Negative reward expectations in Borderline Personality Disorder patients: neurophysiological evidence. *Biological Psychology* 94 (2013): 388-396.
71. Andrulonis PA, Glueck BC, Stroebel CF, et al. Organic brain dysfunction and the borderline syndrome. *Psychiatric Clinics of North America* 4 (1981): 47-66.
72. Judd PH. Neurocognitive impairment as a moderator in the development of borderline personality disorder. *Development & Psychopathology* 17 (2005): 1173-1196.
73. Skodol AE, Gunderson JG, Pfohl B, et al. The borderline diagnosis I: psychopathology, comorbidity, and personality structure. *Biological Psychiatry* 51 (2002): 936-950.
74. Paris J. The nature of borderline personality disorder: multiple dimensions, multiple symptoms, but one category. *Journal of Personality Disorders* 21 (2007): 457-473.
75. Zanarini MC, Gunderson JG, Frankenburg FR, et al. The revised diagnostic interview for borderlines: discriminating BPD from other Axis II disorders. *Journal of Personality Disorders* 3 (1989): 10-18.
76. Gunderson JG. *Borderline personality disorder: A clinical guide*. American Psychiatric Pub (2009).

77. Fossati A. History of childhood attention deficit/hyperactivity disorder symptoms and borderline personality disorder: a controlled study. *Comprehensive Psychiatry* 43 (2002): 369-377.
78. Philipsen A, Limberger MF, Lieb K, et al. Attention-deficit hyperactivity disorder as a potentially aggravating factor in borderline personality disorder. *British Journal of Psychiatry* 192 (2008): 118-123.
79. Fischer M, Barkley RA, Smallish L, et al. Young adult follow-up of hyperactive children: self-reported psychiatric disorders, comorbidity, and the role of childhood conduct problems and teen CD. *Journal of Abnormal Child Psychology* 30 (2002): 463-475.
80. Miller CJ, Flory JD, Miller SR, et al. Childhood attention-deficit/hyperactivity disorder and the emergence of personality disorders in adolescence: a prospective follow-up study. *Journal of Clinical Psychiatry* 69 (2008): 1477-1484.
81. Ferrer M, Andi3n 3, Matal3 J, et al. Comorbid attention-deficit/hyperactivity disorder in borderline patients defines an impulsive subtype of borderline personality disorder. *Journal of Personality Disorders* 24 (2010): 812-822.



This article is an open access article distributed under the terms and conditions of the [Creative Commons Attribution \(CC-BY\) license 4.0](https://creativecommons.org/licenses/by/4.0/)