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Singing under the impulsiveness: impulsivity in psychiatric disorders

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ABSTRACT

Impulsivity is a key feature of numerous psychiatric disorders. This paper reviews the recent evidences on the impulsivity in elderly, relationship between impulsivity and borderline personality disorder (BPD) with attention-deficit/hyperactivity disorder (ADHD), impulsivity with anxiety and mood disorders, and the psychopharmacological approaches to impulsivity evaluating previously published studies. A literature review of the theoretical bases of the relationship between psychiatric disorders and impulsivity is presented. Measurements of impulsivity and neurobiological hypothesis are defined. Treatment approaches are discussed. Previous researches have shown significantly higher levels of impulsivity among patients with BPD, ADHD, anxiety, and mood disorders. In addition, older adults could be more impulsive than younger adults. But the nature of this relationship remains unclear. This is probably due to the fact that there is much overlap between them. Impulsivity is a risk for suicidality and influences pathogenesis, course, clinical severity of many psychiatric disorders. Pharmacological interventions for treating impulsivity should be incorporated into treatment plans for these disorders. Identifying the role of pharmacological interventions in modulating the development of trait impulsivity may prevent progression to psychiatric disorders and associated adverse consequences.

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Introduction

It has been demonstrated in several studies that impulsivity is a complex and multidimensional construct and it has been directly mentioned in the DSM-5 diagnostic criteria for several psychiatric disorders [1]. Although impulsivity is mentioned in diagnostic classifications of mental disorders, there is a lack of knowledge how to define and measure the impulsivity. In general, impulsivity has been defined as rushed responses to stimuli, swift action, and behaviour without adequate thinking often leading to undesirable outcomes [2,3]. According to Moeller “impulsivity is a predisposition to have rapid and unplanned reactions to internal and external stimuli without regard to the negative consequences of these reactions to individuals and others” [4]. Patton et al. [5] separated impulsivity into three components: (1) motor impulsivity, (2) attention impulsivity, and (3) lack of planning. Motor impulsiveness is defined as acting without inhibition and perseverance; attentional impulsiveness is the inability to focus on the ongoing task and cognitive instability; and non-planning impulsiveness is inability to plan and think carefully and orientation to the present rather than to the future [6].

Since impulsivity is a multifactorial construct, there are three main classes of instruments that measure

impulsivity; self-report measures, behavioural laboratory measures, and event-related potentials [4]. Among the psychometric instruments, the Barratt Impulsiveness Scale (BIS-11) [5] is the most common self-report scale used for the trait impulsivity assessment. It has the advantage to gather information on various acts and long-term patterns of behaviour. Behavioural measures of impulsivity includes: neuro-cognitive tasks, for example, go/no-go task, stop-signal task, commission errors, reaction time, and Iowa Gambling Task. For the event-related potential test, recording the electrical brain activity while patient performs various tasks has targeted specific waveforms as measures of predispositions to impulsiveness. Both behavioural laboratory measures and event-related potentials do not incorporate the social aspects of impulsivity but combined with other measures and they are valuable predictors [4].

Neurobiology of impulsivity and aggressive behaviour can be conceptualized as an imbalance between the top-down control, provided by the orbital frontal cortex and the anterior cingulate cortex and the bottom-up drives generated in the limbic structures such as amygdala and insula [7]. In this circuitry; serotonin regulates the prefrontal regions by acting on 5-HT₂ receptors in a different role [8]. The individuals with

induced lowering of 5-HT showed increased levels of impulsivity. 5-HT_{2A} receptors being involved in increased impulsivity and 5-HT_{2C} receptors in decreased impulsivity [7]. Like serotonin, dopamine and noradrenalin are also involved in the balance between top-down control and bottom-up drives of stimuli [7]. However, glutamate and GABA also modulate limbic regions that organize bottom-up drives [7]. A deficiency in such mechanisms may increase the response to stimuli and, therefore, the identification of the role of these neurotransmitters is critical for the treatment of impulsivity.

Since impulsivity is a common clinical problem, it may play a role in pathogenesis of neuropsychiatric disorders. It has been found as a significant contributing factor for many psychiatric disorders such as mood disorders, personality disorders, alcohol dependence, substance use disorder, attention-deficit/hyperactivity disorder (ADHD), and conduct disorder [9,10]. It is also associated with aggression, self-injury, suicide attempts, domestic violence [11], and risk-taking behaviours [12].

Therefore, the aim of this mini-review is to elucidate impulsivity especially in elderly, relationships between impulsivity and borderline personality disorder (BPD) with ADHD, impulsivity with anxiety, mood disorders and other psychiatric disorders, and the psychopharmacological approaches to impulsivity by evaluating previously published studies.

Impulsivity in elderly

Impulsivity is a well-established trait of several psychiatric disorders. However, it is still unknown whether impulsivity remains stable or varies in intensity over the lifespan. Despite the importance of impulsivity, few studies have been made on impulsivity in the elderly. Traditionally, old patients are regarded as a quiet, characterized by higher levels of patience and have a low level of impulsivity. However, in recent years, several studies [13] have shown some personality traits in middle and old ages opposite to traditional studies that regarded personality is stable throughout life. There are experimental studies that focused on inhibitory deficits in old people [14]. According to the inhibition deficit theory of Zacks et al. [15], impaired inhibitory processes cause many of the cognitive deficits in the elderly. According to this age-related inhibition deficit theory, older adults could be more impulsive than younger adults. The few studies performed on this issue; however, showed inconsistent and mostly contradictory results. Another large sample study ($n = 2725$) using a five well-established self-rating measure of impulsivity found that older individuals were less impulsive on self-rating behavioural inhibition and activation scales than the younger groups [16].

A relevant study on elderly sample (65 years and older) have been done to evaluate the dysfunctional and functional impulsivity [17]. A sample of 190 participants (101 females, 89 males) were evaluated. Researchers found out that the elderly sample showed higher dysfunctional impulsivity levels (e.g. tendency to make risky decisions) than the adult samples, which is consistent with the inhibition deficits. But, functional impulsivity (e.g. tendency to make quick decisions with likely personal gain) is quite stable over the lifespan. It seems that impulsivity cannot be considered to decrease with age and dysfunctional impulsivity may even increase.

Recently, a study compared impulsivity between in older 28 euthymic bipolar disorder (BD) patients and 15 older healthy controls using a range of self-rating and behavioural measures of impulsivity. The results showed that, BD patients were risk-taking, had poor decision-making, and increased delay aversion. This study suggested that, in BD, aspects of impulsivity related to reward-based decision-making persist into late adulthood [18].

In sum, since the traditional view of old people as having low impulsiveness might lead professionals to underestimate the impulsivity level of their elderly patients, clinicians should be aware of impulsivity as a risk factor that may cause many problems (e.g. suicide) in elderly patients.

Impulsivity and its relationship with between BPD and ADHD

Recent developments in personality disorder research highlighted that maladaptive personality traits might be less stable than initially believed, and their expression is likely to be worsened by psychosocial stress and psychiatric disorder. Impulsivity is one of the DSM-5 diagnostic criteria for BPD as are identity disturbance and affective instability [1]. The prevalence of ADHD comorbidity in BPD patients is quite high and impulsivity is considered as a central feature of both BPD and ADHD [19]. A number of few studies have examined the association of impulsivity as a part of the adult ADHD symptomatology in patients with BPD [20]. According to Ferrer et al. [21], BPD patients should have been separated into two subgroups according to the presence of ADHD that with impulsive comorbidity. Philipsen et al. [19] reported that ADHD should be considered as a potential aggravating factor in BPD patients with impulsive subgroup.

Recently, we have examined to determine the type of impulsivity that is observed in patients with BPD with or without ADHD symptoms. We have examined the relationship between impulsivity and attention-deficit/hyperactivity symptoms in 90 female BPD patients and 90 healthy participants. We found higher comorbidity of ADHD in the patient group and motor

impulsiveness highly predicted ADHD symptoms in BPD group [22].

We have also examined the complex relationship between impulsivity, childhood trauma, dissociation and ADHD in 165 (128 females) BPD patients and 165 healthy subjects. Our results showed that there were significant relationships between adult ADHD symptoms and impulsivity, childhood trauma, and dissociation. In addition, attentional and motor impulsiveness were found as the predictors of ADHD in BPD patients [23].

Impulsivity is one of the core factors contributing to the severity of both the BPD and ADHD, and the clinical course is strongly related to the impulsivity in the areas such as substance use disorder, impulsive gambling, physical assaults, and binge eating comorbid with these disorders [24]. In terms of the relationship between BPD, ADHD, impulsivity; BPD-ADHD has been considered as a severe, more impulsive, and homogeneous subtype of BPD and therefore, the clinical psychiatrists should carefully evaluate impulsivity among patients with BPD and ADHD to plan more appropriate treatments.

Impulsivity and its relationship with anxiety and mood disorders

Impulsivity is a key component of manic behaviour of BD and important for the diagnosis of mania [1]. Impulsivity also contributes the complications of BD such as suicide [25] and substance abuse [26]. However, depressive episodes are also potentially associated with impulsivity, especially if suicidality is present [27]. There are few studies that have directly measured impulsivity in BD. Swann et al. [28] reported that a higher level of impulsivity, as measured by the BIS-11, in patients with bipolar disorder than in comparison subjects, even when the patients were between episodes of mania or depression. According to their results, the impulsivity among bipolar patients may be independent from state of mood. There are some views about the origins of association of impulsivity with BD such as impulsivity being a risk factor for the disorder, being a result of repeated episodes or independent factor linked with pathophysiology of BD [4].

A recent study examined impulsivity in depressed and euthymic bipolar and unipolar patients and healthy controls. Greater impulsivity was found in patients with depressed bipolar, euthymic bipolar, and depressed unipolar patients compared with the healthy controls [29]. According to the results of these studies, it is still unclear that if interepisode impulsivity is a risk factor for the disorder or a consequence of multiple episodes. Either way, impulsivity influences the clinical presentation of BD and its psychiatric management.

Anxiety disorders have some features such as harm avoidance, safety seeking, and behavioural inhibition. These features may seem opposite to the features of impulsivity such as risk-seeking, behavioural disinhibition, acting without forethought [30]. Anxiety is thought to alert the individual to potential danger and operates to inhibit behaviour under conditions of heightened threat. Traditional view suggests that anxiety might serve as a protective factor against disinhibited, potentially dangerous activities or behaviours that could lead to early mortality [31] and one might hypothesize that anxiety would be protective against impulsivity in BD. In reality, the data revealed high rates of comorbidity between anxiety disorders and impulse control disorders.

There are few studies to examine the relationship between anxiety disorders and impulsivity. Some studies found no correlations between anxiety and impulsivity [32,33] Taylor et al. examined the association between anxiety and impulsivity as measured by BIS-11 in 114 outpatients with BD. Results revealed that patients with a comorbid anxiety disorder displayed significantly higher levels of impulsivity than patients without an anxiety disorder and anxiety-related symptom domains were associated with greater impulsivity [30]. However, anxiety disorder comorbidity in BP patients was associated with negative outcomes such as increasing bipolar severity, reduced duration of euthymia periods, diminished quality of life, and functional impairments [30]. High levels of impulsivity in patients with anxiety disorders were also observed by other authors [34]. According to Del Carlo et al. [11], the patients with BD and anxiety disorder present higher levels of impulsivity than the patients without such comorbidity and anxious patients occurred more impulsive than healthy subjects in both psychometric and neurocognitive measures.

In conclusion, these results demonstrated that anxiety was positively associated with impulsivity. Since anxiety and impulsivity are also two main risk factors of suicidality [35] and have negative outcomes for BD patients, further studies are needed to elucidate the implications of and reasons for this association.

Functional neuroimaging relevant to impulsivity

The neuroimaging literature related to impulsivity has used a fairly narrow set of task paradigms, mostly contrasting the execution and inhibition of motor responses. fMRI studies in healthy adults using behavioural inhibition-related task paradigms such as the Go/NoGo and stop-signal tasks consistently implicated a right-lateralized neural circuitry [36]. Within this neural circuitry, the two critical regions were the right inferior frontal cortex (rIFC) and the subthalamic nucleus (STN). The rIFC plays a central role in

controlling behavioural inhibition opposed to motor planning/response selection. The STN, on the other hand, plays a crucial role in the stopping of a motor response, and its position within the frontostriatal circuitry is particularly well-suited for braking ongoing motor commands that are in the later stages of executions by the brain [37,38]. Functional connectivity data revealed that activation in these two structures were positively correlated [37], consistent with the view that both areas may be required for successful inhibition. Of particular interest to impulsivity researchers were studies that examined how individual differences within this frontostriatal circuitry were related to individual differences in self-reported trait impulsivity. These studies illustrated two important limitations for the study of trait impulsivity using functional neuroimaging. The first limitation was divergence across measurement instruments; i.e. different self-report instruments of impulsivity, even when administered to the same participants doing the same neurocognitive task, might implicate different brain regions. The second limitation was divergence across neurocognitive task paradigms: one and the same self-report instrument might implicate different brain regions and neural circuitries across different but conceptually similar tasks. Similarly, Brown et al. [39] correlated BIS-11 scores in the same subjects while they were scanned using two different tasks. Using an emotional face-matching paradigm, they reported positive correlations in the ventral amygdala and parahippocampal gyrus and negative correlations in the dorsal amygdala and ventral prefrontal cortex. Using a Go/NoGo paradigm, they reported a positive correlation in the caudate nucleus and anterior cingulate cortex. However, it should not come as a surprise that different task paradigms will activate different neural circuits, which may or may not be associated with self-reported trait impulsivity. However, it highlights the importance of matching impulsivity constructs with appropriate neurocognitive task paradigms when attempting to map self-reported impulsivity onto neural circuitries.

Pharmacological treatment of impulsivity

The psychiatric medications that have been proven effective in the treatment of impulsivity are the mood stabilizers such as lithium, carbamazepine, oxcarbazepine, valproate, and topiramate; the novel antipsychotics such as clozapine, olanzapine, and quetiapine; the beta-adrenergic blockers, the 5-HT_{1A} partial agonist buspirone, the essential fatty acid omega-3, and an alpha-2 adrenergic agonist clonidine [40].

According to the placebo control studies, most of them support the efficacy of lithium for impulsivity and aggressive behavior in children, adolescents, and adults [41,42]. Lithium is also effective in preventing

suicide in BD patients as reported by a retrospective study. But due to significant side effects, lithium has a limited usage [43]. Anticonvulsants such as phenytoin, carbamazepine, and divalproex have all been found to decrease impulsive aggression [44,45]. In a double-blind placebo-controlled study, carbamazepine leads to a reduction in impulsive behaviours in BPD [46].

Antidepressant drugs like selective serotonin reuptake inhibitors have also been used successfully to treat impulsive aggression in controlled clinical trials. In a 3-month controlled trial, Coccaro and Kavoussi [47] reported that fluoxetine significantly decreased impulsive aggressive behaviour compared to placebo in personality disorder patients. A decrease in hostility was also reported for paroxetine, in a double-blind placebo-controlled study [48] and for citalopram, compared with placebo, in patients with schizophrenia [49].

Antipsychotic medications have been used to diminish impulsivity in clinical disorders such as schizophrenia, schizoaffective disorder, and BD and clinical situations to treat impulsivity like dementia, autistic disorder, and BPD. Although these medications are appropriate only for the treatment of acute aggressive behaviour [44]. There is evidence for a more specific antiaggressive effect of atypical antipsychotic medications. Two placebo-controlled trials of risperidone in adults with dementia reported a significant decrease in aggression without significant sedation [50,51]. Similar results were reported with clozapine, olanzapine, and quetiapine [40]. Clozapine was found more effective than haloperidol, risperidone in reducing hostility [44].

Although not frequently used, beta-blockers also have been used to treat impulsive aggression. A few small placebo-controlled trials have reported efficacy of propranolol and pindolol for impulsive aggression in populations with organic brain injury [52,53]. The efficacy of psychostimulants on impulsivity is still unclear. Several controlled studies have found that psychostimulants compared with placebo reduce impulsivity in patients with ADHD [54,55]. However, other studies reported that that hyperactivity or attention was changed, but not impulsivity [56,57]. This situation can be explained as the medication is effective in controlling milder symptoms, and the lower effect was observed for severe impulsive symptoms [40].

In sum, two main treatment strategies could be aimed in the treatment of impulsivity. As impulsive behaviour could occur in a wide range of clinical situations, the treatment of impulsivity demands an alternative approach to the treatment of "primary psychiatric disorder." However, since impulsivity has some biological mechanisms that occur independently of the primary psychiatric disorder, it could be treated as a psychiatric disorder in itself.

Conclusions

Understanding the discrete mechanisms of impulsive decision-making and behaviours would offer new objectives for diagnosis and interventions. Literature indicates that impulsive symptoms comorbidity is common in patients with several psychiatric disorders. Impulsivity is a risk for suicidality and influences pathogenesis, course, clinical severity of many mental disorders. There is still a lack of information what leads to variance in this assessment. Impulsivity can be considered as a trans-diagnostic feature of many psychiatric disorders, yet our understanding of the concept and approaches to measurement have evolved significantly with advances in neuropsychiatric research. Identifying the role of pharmacological interventions in modulating the development of trait impulsivity may prevent progression to psychiatric disorders and associated adverse consequences. Further researches in this field are necessary to understand these relationships and help to guide clinicians for the best choices during the management.

Disclosure statement

No potential conflict of interest was reported by the authors.

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