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According to the DSM-V, borderline personality disorder is “a pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity, beginning by early adulthood and present in a variety of contexts […].” It is a cluster B personality disorder, and its prevalence is somewhere between 1.6 and 5.9% of the population (DSM-V). The 9 diagnostic criteria – of which you need at least 5 to be diagnosed— are “efforts to avoid real or imagined abandonment,” unstable relationships, unstable sense of self, impulsivity (“e.g., spending, sex, substance abuse, reckless driving, binge eating”), suicidal behavior or self-harm, mood instability (“e.g., intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days”), “chronic feelings of emptiness,” dysregulated episodes of anger, and “transient, stress-related paranoid ideation or severe dissociative symptoms” (DSM-V). BPD is incredibly invasive as it affects everyday life, interpersonal relationships, possibly career success, and the like. Common treatment options include group-therapy, CBT, and more. The main goal of this paper is to explore the neurological underpinnings of the disorder as possible explanations for some of the symptomology.

Before one can delve into this task, they must first understand two major circuits in the brain: the corticolimbic circuit of recognition and reaction, and the corticostriatal circuit of motivation and action (Hariri 2015). The corticostriatal circuit’s main structures include the amygdala, the hypothalamus, the brainstem, the substantia innominata, the insula, the hippocampal formation, the prefrontal cortex, and the thalamus. Firstly, sensory input from the thalamus is relayed to the amygdala with immediate, low-resolution sensory information. The same information is filtered from the thalamus, through the sensory cortices, and to the amygdala with slower, more detailed sensory information. This information hits the basolateral complex of the amygdala (BLA) first, where it is processed and sent to either the central nucleus of the amygdala (CeA) for action or the intercalculated masses (ICMs) between the BLA and CeA for inhibition. From there, the CeA further signals the hypothalamus for hormone circulation (i.e. cortisol) and regulation of the sympathetic nervous system (“fight or flight” response). It also signals the brainstem for specific, automatic responses (such as increases in heart rate, the “freeze” response, etc.), and the BNST. These areas are responsible for interpreting and translating the information from the CeA to be more accessible for cortical areas (for higher order processing). Of these areas, the insula is responsible for conscious awareness of changes in our internal state. The insula is able to do this as it also receives input from our internal organs and somatosensory systems. The hippocampal formation, another area signaled by the BNST and NBM, is responsible for keeping track of and providing contextual information. It can also trigger the prefrontal cortex to induce an inhibitory response if this contextual information makes it clear that the situation is nonthreatening. The PFC monitors this circuit and targets the ICMs for inhibition if needed. Essentially, it either allows the amygdala to run or inhibits it from running. This circuit plays a key role in fear learning and other emotional responses.

The corticostriatal circuit consists mainly of the ventral and dorsal striatum, the ventral and dorsal palladium, the thalamus, the midbrain, the hypothalamus, motor cortices, the hippocampal formation, the BLA, and the PFC. The ventral striatum is the hub of this circuit (like the amygdala in the corticolimbic circuit). Essentially, this area is responsible for allowing intent to become action. The ventral striatum receives input from the hippocampal formation for context, the BLA for interpreted sensory cues, the PFC, and more. It has an afferent and efferent connection with the midbrain; the VS typically inhibits motivation and movement, but when signals the midbrain, the midbrain releases dopamine and blocks inhibition. Once no longer inhibited, the VS signals the ventral and dorsal palladium, the dorsal striatum, and the hypothalamus. The ventral palladium works in sync with the VS and fleshes out the reward experience or pleasure response (that serves to motivate further action). The hypothalamus serves mainly to meet your primal needs (i.e. eating, drinking, fleeing, fighting, reproduction, etc.). Different parts are activated when different goals are required. The dorsal palladium supports goal-directed behavior with action- it is responsible for the actual doing something to fulfil the desired goal. The final places that send out signals for movement (action) are the motor cortices. As per usual, the PFC is responsible for monitoring this circuit (specifically movement and goal-directed planning). In short, this circuit is responsible for motivating/ producing action and motivating the continuation of these actions.

So how does all of this relate to Borderline Personality Disorder? Structural and connectivity abnormalities that are common in this disorder, as related to these circuits, may reveal a biological basis or possible cause for some of the dysfunctional behaviors of BPD. One major finding indicates that functional connectivity between the amygdala and PFC may be lesser than that of healthy controls (New, et. al., 2007). If this is true, it would imply that functioning of the corticolimbic circuit is impaired in those with BPD. This likely takes the form of lack of regulation of the amygdala by the PFC, which would in turn impair the function of rest of the circuit. This lack of regulation may partially explain why some symptoms of BPD are intense fear of abandonment and pervasive anxiety. According to one study, “young adolescents with a genetic risk […] and exposure to physical maltreatment had a 13-fold increased risk of being in the extreme […] BPD group” (Winsper, et. al., 2016). This would imply that, due to the lack of regulation of the amygdala, those with BPD are more easily conditioned to specific fears (i.e. abandonment, maltreatment). This is further strengthened by the findings of other studies that suggest a lesser extent of functional connection between the amygdala and the hippocampal formation (Depping, et. al., 2016). In this case, fear responses may be exacerbated due to lack of or inappropriate contextual information from the HF.

Other studies have found distinct dysfunction within the corticostriatal circuit as well. Firstly, Enzi, et. al. (2013) found that, as compared to healthy controls, patients with BPD had a difficult time differentiating between rewarding and non-rewarding stimuli (specifically relating to the emotions of others). This may explain some of the relational difficulties that those with BPD have. If they cannot differentiate between positive and negative attention, and also have a fear of abandonment, they may do things that others would consider to be dysfunctional to make their partners stay with them. For example, if a man with BPD anticipated that his girlfriend was going to break up with him, he may act out in order to get any attention from her—whether positive or negative—as it is difficult for him to distinguish between the two. Another study found that dysconnectivity in the corticostriatal circuit was associated with the heightened impulsivity seen in BPD patients (Wang, et. al., 2017). It seems as if the system itself fails to inhibit action when there is a desire or motivation. In other words, where a simple thought may remain as just that in a healthy individual, a person with BPD may lack inhibitory regulation and may impulsively go through with the idea (whether or not it is a good one).

In conclusion, it appears that many of the 9 distinct symptoms of BPD may be explained by disordered function of the corticolimbic and corticostriatal circuits of the brain. Further analysis of brain structure and connectivity may reveal even more biological backing for this. Research on this topic has been done fairly recently and is somewhat limited as compared to the research of MDD and ADHD, for example. Thus, it may be beneficial to unearth and solidify these neurological abnormalities so that we can better treat and diagnose people who have BPD.

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