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The corticolimbic circuit in the human brain serves an important role in formulating appropriate responses to our environment. The main structures of this circuit are the amygdala (the “hub”), the thalamus, sensory cortices, the hypothalamus, the brainstem, the substantia innominata, the insula, the hippocampal formation, and the prefrontal cortex. When operating properly, these structures working in conjunction with other parts of the brain and our bodies can assist in fear learning/conditioning, essential survival responses, and more.

The main structure of this circuit-- the amygdala-- is responsible for receiving sensory input and producing/ facilitating appropriate responses. The most important parts of the amygdala for this particular circuit are the lateral nucleus, the basal nucleus, and the accessory basal nucleus (commonly lumped together and referred to as the basolateral complex of the amygdala, or the BLA). The input processed by the BLA comes primarily from the thalamus and the sensory cortices. Low road (low definition) sensory information is relayed immediately to the BLA by the thalamus. Simultaneously, the thalamus sends the same information to the sensory cortices for more complex (high road) processing, which then relay the interpreted input to the BLA. Once the BLA is signaled it either signals the central nucleus of the amygdala (CeA) or the intercalculated masses (ICMs) between the two structures. This depends on the ideal course of action decided by the prefrontal cortex and other areas of the brain (discussed later).

If the CeA is signaled by the BLA, it in turn messages multiple other parts of the brain. Firstly, the CeA may signal the PVN of the hypothalamus to interact with parts of the endocrine system to get hormones circulating (i.e. cortisol). The hypothalamus also plays a role in ruling over the sympathetic portion of the autonomic nervous system to help control the “fight or flight” response. The CeA can also signal the substantia innominata (more specifically, the bed nucleus of the stria terminalis and the nucleus basalis of Meynert). These areas translate the output from the CeA so that it is suitable for cortical areas (higher order processing) like the insula, the hippocampal formation, the prefrontal cortex, and the posterior fusiform gyrus. The insula is responsible for us being aware of the changes in our internal state due to stimuli. Along with receiving input from the CeA, the insula also receives information from our internal organs (i.e. our stomach) and somatosensory systems. The hippocampal formation plays a role in recognizing and processing the context of situations. In other words, the hippocampal formation aids in making us aware of what is actually going on. Due to this, it can inhibit responses if a situation is recognized as non-threatening. The prefrontal cortex (PFC) monitors the entire process of this circuit and regulates it accordingly. It is responsible for conscious awareness of a situation. The ventral PFC receives input from the amygdala, and the dorsal PFC sends a signal back. This signal can be inhibitory (through the ICMs) or allow the response to run its course.

A practical (highly simplified) scenario in which this system would play a role would be walking up to a stranger in a dark alleyway. Firstly, sensory information about the surroundings would be processed by the thalamus and the sensory cortices and henceforth sent to the BLA. The BLA would then signal the CeA, invoking a fear response. The CeA would signal the hypothalamus, which would interact with the endocrine system to circulate norepinephrine and other hormones for sympathetic arousal. The insula would play a part in making us aware of our heartbeat increasing (and other respective stress responses). The PFC and HF would both play a role in recognizing that the situation is unfamiliar and potentially dangerous, and thus would both allow the fear response to continue. If, once approaching the “stranger,” it is determined that the person is actually an acquaintance of the subject (involving the posterior fusiform gyrus for face recognition), both the HF and the PFC would signal the ICMs of the amygdala to stop the fear response.

A particular disorder in which the effects of dysfunction within this circuit can be seen is Williams Syndrome. Williams Syndrome is a genetic disorder that involves a partial deletion of chromosome 7. The main effects of the deletion are a lack of fear of others, dysfunctional social behaviors, learning impairment, and more. fMRIs done of people with Williams Syndrome show amygdala hypoactivity in response to social threat but hyperactivity in response to general threat. For example, a child with Williams Syndrome may enthusiastically approach a dangerous looking stranger that other children would shy away from, but the same child may have debilitating arachnophobia. A theory proposed by Hariri (2015) as to why this paradox may be is that due to the increased prefrontal activity resulting from a tendency to “engage in their unique and elaborate linguistic processing of social stimuli and situations,” there may be more regulation of the normally hyperactive amygdala in social situations (p. 86). The general amygdala hyperactivity may also be attributed to abnormal regulation (or lack thereof) by the PFC. Another common characteristic of people with Williams syndrome is attention deficit issues. As this is another symptom believed to be caused by abnormal functioning of the PFC, one could be lead to believe that the problem with hyperactivity and hypoactivity resides in the PFC rather than the amygdala itself. Thus, in essence, it is possible that due to general disordered PFC functioning (likely regulation, to be specific), those who have Williams syndrome also have pervasive anxiety, are prone to phobias, and are overly-gregarious due to it not performing its role as regulator of the corticolimbic circuit. This is just one example of how a simple dysfunction in one part of the corticolimbic system can cause a multitude of problems.

References

Hariri, Ahmed R. (2015). *Looking inside the disordered brain*: *An introduction to the functional neuroanatomy of psychopathology.* Sunderland, MA: Sinauer Associates, Inc.