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REVISE THE REVISED? THE NEUROANATOMICAL HYPOTHESIS OF PANIC DISORDER

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Introduction/ objectives: In 2000 Gorman and colleagues published a widely cited revised version of their original neuroanatomical hypothesis of panic disorder from 1989. A “fear network” centered in the amygdala is supposed to be crucial in mediating fear-related responses. It is assumed that panic attacks result from a dysfunctional coordination of “upstream” (cortical) and “downstream” (brainstem) sensory information, resulting in heightened amygdalar activity with subsequent behavioral, autonomic, and neuroendocrine activation. The authors argue that many aspects of their revised neuroanatomical hypothesis are likely to prove incorrect, but hope it will stimulate research on anxiety disorders.

Methods: We reviewed neuroimaging studies on panic disorder to investigate in how far their results are in line with the neuroanatomical hypothesis and what inconsistencies emerge. Structural (CT, MRT), functional (PET, SPECT, fMRI, NIRS, EEG) and spectroscopic data (MRS) were considered.

Results: To some extent studies investigating panic disorder found structurally and functionally altered brain areas that have been implicated in fear processing as supposed by the neuroanatomical hypothesis. However, inconsistencies regarding increased vs. decreased volume and increased vs. decreased activity in specific areas indicate aspects that cannot easily be explained by the hypothesis.

Conclusions: The neuroanatomical hypothesis is partially supported by empirical data that may have therapeutic consequences. However, inconsistencies should be a starting point to expand the hypothesis and modify some aspects. This may include the consideration of genetics, task-specificity and the definition of subgroups.

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