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SEROTONIN (5-HT) IN THE LUNG COULD LEAD TO PANIC DISORDERS IN THE BRAIN

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Introduction and aimes: We would like to consider a single model which posits that hyperfunction of suffocation receptors in the lung could be the cause of Panic Disorder (PD).
Methods: The pulmonary neuroepithelial bodies (NEBs), which are situated at the bifurcation point of small bronchi, act as O2 sensors responding to a number of airway stimuli, including hypoxia, hypercapnia, and smoking, and release 5-hydroxytriptamine (5-HT) from their secretory granules. If we suppose that PD might represent an inflammation of the NEBs, bradykinin (BK) which augments the airway hyper-response to diverse inducers might cause these cells to release 5-HT along with peptides and panneuroendcrine markers from their secretary granules.
Results: Since it was revealed that BK with 5-HT could cross the blood-brain barrier (BBB), when 5-HT released from NEBs along with BK cross the BBB, the serotonergic neurons will be inhibited by the 5-HT1A autoreceptors. It is easy to suppress the periaqueductal gray (PAG), which inhibits flight reactions to impending danger, pain or asphyxia. In short, the hyperfunction of inflammatory NEBs might bring about panic reactions.
Conclusins: PD could be a lung desease that affects the brain directly and reversibly through the effects of 5-HT with BK. Future therapies for PD might be inhalants that can stabilize inflammatory NEBs, inhibit 5-HT release and BK receptors.

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