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Endogenous Opioid and Receptors in Panic Disorder

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Abstract. The aim of this article is to explore the role of endogenous opioid through their receptors μ, δ, κ, and σ on respiration in panic disorder. An opioidergic dysfunction as pathophysiological mechanism underlying panic disorder was proposed in some previous studies. In this mini review, we focused on (a) role of endogenous opioid as modulator for carbon dioxide sensitivity and (b) the effect of the opioid receptors on respiration in experimental studies using opioid antagonist.

Key words: endogenous opioid, CO_2 sensitivity, opioid receptor, panic disorder

Abstrak. Artikel ini bermaksud mengeksplorasi peran endogenous opioid melalui *opioid receptor* μ, δ, κ, and σ pada pernapasan pada gangguan panik. Disfungsi *opiodergic* sebagai mekanisme patofisiologis telah dibahas dalam beberapa kajian sebelumnya. Dalam *mini review* ini kami fokuskan bahasan pada kajian (a) peran endogenous opioid sebagai modulator sensitivitas terhadap karbondiosida dan (b) efek opioid receptor pada pernapasan dalam kajian eksperimental yang menggunakan administrasi antagonist opioid.

Kata kunci: opioid endogen, sensitivitas CO_2, reseptor opioid, *panic disorder*

Panic attacks come on suddenly, accompanied by shortness of breath, a pounding heart, dizziness, and chest pain, lasting less than 10 minutes. Panic disorder can cause a person to become chronically apprehensive. People may even suffer agoraphobia as a consequence. Panic attacks are almost always associated with shortness of breath or difficult breathing, responded by hyperventilating, another common symptom of panic attacks. Respiratory dysfunction has a high co-morbidity with panic disorder (PD) (Griez, 1997).

People hyperventilating exhale more carbon dioxide (CO_2) than they produce, thereby lowering CO_2 levels in the body (hypocapnia), increasing the pH, leading to alkalosis with a less solubility of Calcium. Ca in the blood and hypocalcaemia results in the light headedness and dizziness that often accompany panic attacks.

Theories of Panic Disorder

Hyperventilation Theory

This theory proposes that PD patients are chronic hyperventilators who acutely increase breathing during stress, leading to episodes of panic. However, since forced hyperventilation induces panic attacks in only a subgroup of patients and evidence for the existence of chronic hyperventilation is mixed, the primary hyperventilation hypothesis may be insufficient to account for the etiology of PD. However, this hypothesis appears to be wrong. Griez (1997) concluded that *hyperventilation* can be counted amongst panic attack symptoms and is not the reason. Klein found that air enriched with 5% CO2 frequently induced panic attacks in panic disorder patients, which is exactly the opposite of the prediction of the hyperventilation theory (Klein, 1999).

The Suffocation Alarm Theory

This theory claims that panic attacks are due to a "suffocation monitor" in the brain, erroneously signalling a lack of air components especially CO_2 level. Klein distinguishes between panic and fear, suggesting that while panic is a false suffocation alarm (FSA), fear is not. In integrating laboratory provocation studies of panic, Klein suggests that "respiratory" panicogens such as sodium lactate, CO_2, and isoproterenol elicit a false suffocation alarm (ie, a panic attack). Respiratory challenge in PD patients (hypercapnic 35% CO_2) has shown to be a reliable marker for PD (van Beek et al, 2003).

Opioid System

The mechanism of action of opium was totally unknown