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Mini Review Open Acces

4QFDJGJD 2VBMJUZ PG 4FOTBUJPO %FSJWFE C

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Abstract

The most common type of cannabinoid is the tetra-hydrocannabinol, which is one of the major psychoactive components isolated from Cannabis sativa. Cannabinoids can bind to Gi-protein coupled cannabinoid type 1 receptors, which is highly expressed in the pre- and post-synaptic in brain and spinal cord, as well as the Gi-protein-coupled cannabinoid type 2 receptors that is predominantly located in the immune system. The activation of CB1 DQG &% LQKLELWV WKH IRUPDWLRQ RI LQWUDFHOOXODU F$03 KHQFH OHDGLQJ W within the neurons.

Keywords: Cannabinoid; Pain sensation; Receptors; Neurokinin; Spinal cord; Pain pathway

Introduction

In addition, the activation of CB2 can further prevent the mast cell degranulation and the release of pro-in ammatory mediators, making the reduction in pain sensation even more drastic and e ective.NE is the principal neurotransmitter of the adrenergic pathways and is synthesized from phenylalanine in the nerve terminals. Phenylalanine is converted into tyrosine and then into 3,4-dihydroxyphenylalanine by tyrosine hydroxylase. DOPA is then further converted into dopamine, which is the precursor of NE that is stored in the vesicles of the nerve terminals. e receptors of NE include 342(a)3(,in)3(,in) ,inf,hen furch1.8(ien 1)-6(ur)-9.93-Gsen fm677e redammaeureueurr lesic4(d receptors are predominantly located in postsynaptic neurons, whereas 2-Gi -protein-coupled receptors are located in presynaptic neurons [1]. us, the activation of the 2-Gi -protein-coupled receptors inhibits the Ca2+ in ux, and causes the reduction of NE release out from the synapse. On the other hand, the binding of NE with 1-Gq - and -Gs -protein-coupled receptors that are located in the postsynaptic neurons stimulates the PLC/PKC and cAMP/PKA signaling pathways, respectively, and causes excitatory e ects.

Discussion

Understanding the complex mechanisms of pain is undoubtedly essential for pain research and pain management. Hence, the present review was comprehensively discussed based on the molecular and cellular mechanisms underlying the pain pathway as a whole picture. Moreover, the major types of neurotransmitters involved in the pain transduction, transmission and modulation have been completely elaborated along with their locations and eventual pharmacological e ects. is could enlighten the understanding of the global scientists towards the pain topic and provide a useful guide for continue analgesic drug discovery in future. Tachykinins is the largest family of neuropeptides [2]. ere are three members of tachykinins family involved in the neurogenic-induced in ammation, which are SP, neurokinin A and neurokinin B. ese neuropeptides are produced from peripheral terminals of the sensory nerve bers, such as muscle and skin via proteolytic cleavage of their precursor, pre-protachykinins. e SP, NKA and NKB selectively bind to their cognate receptor according to their a nity to these receptors. For a clearer picture of the receptors that are compatible with the neuropeptides, SP binds to neurokinin type 1 receptor while NKA binds to neurokinin type 2 receptor and NKB binds to neurokinin type 3 receptor receptors, respectively. All these receptors are Gq-protein coupled receptors, which mediate through PLC/IP3 and DAG/PKC signalling pathways upon activation, hence resulting in its excitatory e ects. CGRP is widely produced in both central and peripheral nervous systems; however, it is primarily located in the primary a erent nerves. As a direct derivative of the DRG, CGRP is found in the DH of the spinal cord and associated with the conduction of noxious stimulation. CGRP is related to the excitatory e ects of SP, which results in Ca2+ release [3]. e receptors of CGRP are Gs-protein-coupled, which are known as calcitonin receptor-like receptor located in the nucleus accumbens, indicating that the CNS controls the CGRP-mediated pain transmission. BK is a well-known algogen and acts as one of the in ammatory mediators that are locally produced from the breakdown of high-molecular-weight kininogens in the site of the in amed tissue. In the nociceptive a erent nerve bers, BK binds to Gq-protein-coupled bradykinin receptor type B1 or bradykinin receptor type B2 receptors, leading to sensitization. e activation of B1 orB2 receptors causes activation of the PLC to break down the phosphatidylinositol 4,5-bisphosphate into IP3 and DAG, and subsequently, DAG activates the PKC, leading to the increase of Ca2+ conductance. Furthermore, BK can act synergistically with other algogenic substances, such as PG and NGF, to further stimulate pathways, whereas the activation of the 5-HT3 receptors induces a

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