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Association between Δ^9-tetrahydrocannabinol (cannabis, marijuana) and the cannabinoid hyperemesis syndrome

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Communication

The use of medical (and recreational) cannabis in moderation, has been purported to ameliorate pain and discomfort from patients suffering from the effects of neoplastic diseases, administration of chemotherapy, neuropathy, inflammation, muscle spasticity and more [1,2]. The inordinate use of cannabis paradoxically, has been etiologically linked to patients presenting with intense gastrointestinal cramps, cyclical diarrhea, nausea, and vomiting - clinically referred to as cannabinoid hyperemesis syndrome or CHS [3]. Although once considered rare, CHS has markedly increased over the last number of years. Data obtained from two urban hospital Emergency Departments for example, showed > one third of cannabis users being diagnosed with CHS [4,5]. The abuse of cannabis is not considered life threatening, although two CHS-associated mortalities due to nonspecific electrolyte imbalances have been reported [6].

It wasn't until the discovery of the endocannabinoid system (ECS) in the 1990s that biomedical workers attained an insight into the mechanism of cannabis activity [7]. The ECS specifically, is a constituent of the central and peripheral nervous systems, as well as tissues and organs throughout the body, including the gastrointestinal tract [8]. Importantly, cannabinoid CB1 (CB1R) and CB2 (CB2R) and potentially other receptors of the ECS bind to both endogenous [e.g., *N*-arachidonoylethanolamine (anandamide) and 2-arachidonoylglycerol (2-AG)] as well as the exogenous cannabinoids including THC and CBD [9,10]. Interestingly, recent data obtained from clinical studies indeed appear to suggest anti-emetic activity by cannabis at lower or less frequent dosing, but a pro-emetic activity at higher or more frequent doses. Of significant note moreover, when consumed in excess, cannabis is known to effect gastric motility effecting hyperemesis [11,12].

The ECS is suggested to play an important role among chronic cannabis users through differential degrees of CBR1R downregulation [13]. Such findings however are not absolute, as genetic predisposition may also be involved. Other factors for the recognized paradoxical emetic effects on chronic cannabis use might be due to increased cannabis potency [viz. higher rations of THC to cannabidiol (CBD)] as well as duration of use.

In as much as the ECS contributes to homeostasis through a regulation of mitochondrial function, it would not be inappropriate to suggest that a cannabinoid associated down regulation of cannabinoid receptors might adversely affect mitochondrial function and in turn, prove detrimental to overall cellular function and viability [14,15]. Studies by Lipson and co-workers support the aforementioned hypothesis. Utilizing the human colon adenocarcinoma cell line as a model system, increasing concentrations of a high ratio THC to CBD formulation was found to affect an early cellular death rate and a loss of mitochondrial integrity. These findings were ascertained through cell viability testing by immunofluorescence and transmission electron microscopy, respectively [16].

The exact mechanism of THC/CBD-associated CHS still remains ill defined. Continued studies are needed to address this important issue especially on the cellular level, with special consideration to the lower gastrointestinal tract.

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