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Title: Modulation of nicotinic acetylcholine receptors by cannabinoids.

With the recent legalization of medicinal and in some cases also the recreational use of marijuana by many states in the USA, there has been a significant increase in the number of marijuana users, many of which are tobacco smokers resulting in a high incidence of comorbidity of both substances. Studies have implicated the use of marijuana with the addictive potential of tobacco and viceversa suggesting that the mechanism of action of the addictive substances in both, cannabinoids and nicotine, interact with each other in the brain. However, little is known about the effects that the phytocannabinoids found on marijuana have on the function of nicotinic acetylcholine receptors (nAChRs). Interestingly, it has been shown that some endogenous cannabinoids are able to interact and alter the function of the two most abundant nAChRs found in the brain the $\alpha 7$ nAChR and the $\alpha 4\beta 2$ nAChR. The two most abundant phytocannabinoids in marijuana Δ^9-Tetrahydrocannabinol (THC) and cannabidiol (CBD) have been shown to have tremendous therapeutic potential in many neurological disorders such as convulsions and pain. However, many of their therapeutic effects are not associated with the activation of the cannabinoid system. Therefore, there is a critical need to increase our understanding of the molecular targets of phytocannabinoids in order to increase our chances of using them successfully in the clinic. For those reasons we are going to use *Xenous laevis oocytes* as a model system to study the effects of different cannabinoids both from natural (phytocannabinoids) and synthetic sources in the function of nAChRs of different subunits composition. The nAChRs are known to play major roles in the development of neurodegenerative diseases, inflammatory disorders, pain transmission and as drugs of abuse; and a lot of effort and money has been spent trying to find molecules that modulate their actions. Interestingly, some cannabinoids have been found to modulate the function of nAChRs on a subunit dependent manner, however, we lack information on the nAChR subunit selectivity of interactions between clinically relevant phytocannabinoids and nAChRs, which is needed to elucidate the mechanism of action of these cannabinoids on nAChRs.