# e-MS Experts' Summit Season 2020

## Abstracts

### Cannabis and the endocannabinoid system

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The endocannabinoid system is widely distributed in the central and peripheral nervous system, and other peripheral tissues, where it regulates brain functions by acting on different cell types.[1] The endocannabinoid system is composed of at least two cannabinoid receptors –type 1 (CB1R) and type 2 (CB2R) – plus endogenous ligands (endocannabinoids) and their synthesizing and degrading enzymes, as well as intracellular signalling pathways and transport systems.

CB1R is the most abundant G protein-coupled receptor in the brain, with a widespread and heterogeneous distribution, and regulates multiple brain functions and pathophysiological processes. The expression of CB1R is particularly abundant in brain areas that are involved in motor control, such as the basal ganglia and the cerebellum. In contrast, CB2R was initially considered as the peripheral cannabinoid receptor due to its high expression in the peripheral immune system. However, this perception has changed dramatically in the past decade following the demonstration of CB2R expression in neurons in the central nervous system.

The endocannabinoid system is the pharmacological target of the main phytocannabinoids contained in preparations obtained from Cannabis sativa. Among the large amount of cannabinoids contained in these preparations, two of them – Delta9-tetrahydrocannabinol (THC) and cannabidiol (CBD) – are of particular interest for pharmacological purposes;[2] consequently, the mechanisms of action of THC and CBD will be covered in this session.

While the effects of THC on cannabinoid receptors have been widely reported, the mechanisms involved in the pharmacological effects of CBD are more contentious. However, THC and CBD have been reported to have a synergic effect when used together for the treatment of spasticity symptoms in patients with MS.[3] Therefore, the advantages (in terms of efficacy and safety) of combining THC and CBD in the treatment of MS will be discussed in this session.

**References**
**1.** Hu SS & Mackie K. *Handb Exp Pharmacol* 2015; 231: 59–93.
**2.** Pisanti S, Maltifano AM, Ciaglia E et al. *Pharmacol Ther* 2017; 175: 133–50.
**3.** Nielsen S, Germanos R, Weier M et al. *Curr Neurol Neurosci Rep* 2018; 18 (2): 8.