

Cannabis could hold the key to ending multiple sclerosis misery

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Researchers investigating the role of cannabinoids - chemical substances contained within cannabis – in the treatment of multiple sclerosis (MS), have found they could significantly enhance therapy, not only by reducing nerve damage and erratic nerve impulses, but perhaps even by hindering development of the condition.

The findings, published online today (1 April, 2007) in *Nature Medicine* demonstrates for the first time how cannabis might actually slow down the progression of MS and could have major implications for the estimated 2.5 million sufferers worldwide.

Using a mouse model, a team of UK, European, Japanese and US scientists, led by David Baker, Professor of Neuroimmunology at Queen Mary, University of London, found that doses of the active component within cannabis, tetrahydrocannabinol (THC) could significantly inhibit the development and severity of MS.

Cannabis works because it stimulates molecules known as cannabinoid receptors within the body. The group had previously reported that THC could alleviate disease symptoms, and also save nerves from the damaging effects of the disease - thus potentially, via the cannabinoid receptor CB1, slowing down the development of progressive disability. They had not previously examined the influence of cannabinoids on immune aspects of the disease.

Now their most recent study has successfully separated the roles of

cannabinoid receptors CB1 and CB2 on neurons and T cells, and investigated their effect in controlling central nervous system autoimmunity. It showed that CB1 receptor expression by nerves in the brain, but not T cells, could suppress the development of an experimental MS-like disease, by stimulating the release of anti-inflammatory molecules, whilst in contrast direct stimulation of CB2 receptors by T cells was also able to control inflammation associated with the condition. This suggests that cannabis-like drugs may have the potential to block the autoimmune response which drives disease development.

Professor David Baker said: “Whilst targeting CB1 receptors for therapy runs the risk of causing the unwanted “high” to achieve these effects, we can get the same result by targeting CB2 receptors, which avoids these risks. Therefore, we can start to think about using new drugs that harness the potential medical benefits that cannabis has to offer but move away from the issues over the legality and recreational use of the plant product”.

Source: Queen Mary, University of London

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