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DRUG REPURPOSING FOR MULTIPLE SCLEROSIS

Technology Offer

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Branch
oral drug therapy, multiple sclerosis

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Offer
license or co-development

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Introduction

Multiple sclerosis (MS) is a serious autoimmune disease which leads to the demyelination of neurones in the central nervous system (CNS). While in earlier stages remission may partially occur, the disease ultimately becomes chronically progressive. Standard drug therapy is still reliant on parenteral treatments, though oral therapies have been introduced in recent years, but all drugs used in MS are associated with serious side-effects. Well tolerated chronic oral therapy is still being sought.

Invention

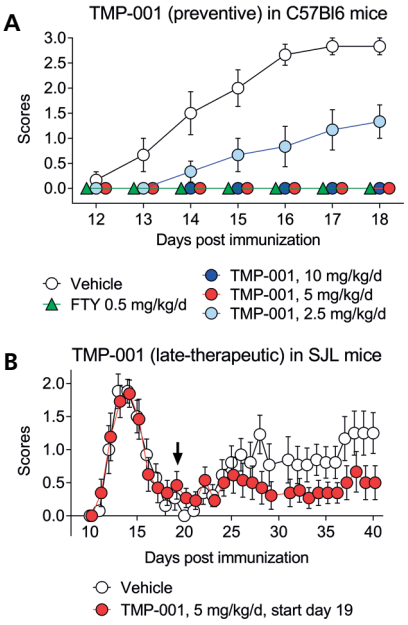
The invention relates to the use of a drug (TMP-001), marketed for many years for another indication, in the treatment of MS. It has been tested extensively, though without significant success, in Alzheimer's disease patients in which safety of high doses was comparable to placebo. Surprisingly, animal studies reveal a novel spectrum of activity in models of MS. The major advantages of this asset are the extensive clinical experience with the drug, its excellent oral safety profile and novel efficacy spectrum. TMP-001 offers a relatively straightforward development pathway to a novel oral therapy for MS.

Market Potential

Potential commercial applications include as an add-on oral drug for therapy of MS, as single oral drug therapy of MS or for pain relief in MS patients.

Development Status

In rodent models of experimental autoimmune encephalomyelitis (EAE), TMP-001 showed both prophylactic and therapeutic efficacy, being more effective in relapsere-mitting EAE than current oral drugs for MS. Its mechanism of action appears to be multifactorial, including actions on endocannabinoids, lipid metabolism and regulatory T cells. Phase I studies have confirmed the clinical safety and pharmacokinetic behaviour of TMP-001, which is currently undergoing a phase IIa clinical trial in patients with early MS.



Oral TMP-001 given preventively (A), dose dependently inhibits chronic progressive EAE in C57BL/6 mice, in comparison to marketed fingolimod (FTY), and given therapeutically (B, from arrow), inhibits relapse of EAE.