Technology Offer
F53761
May 7, 2017

Branch
oral drug therapy, multiple sclerosis

Patent Situation
EP patent filed Nov 3, 2011;
issued Nov 21, 2013
(US 2013/0309199)

Offer
license or co-development

Key Words
oral drug therapy, multiple sclerosis,
derug repositioning

Fraunhofer Institute for Molecular Biology and Applied Ecology IME
Branch for Translational Medicine and Pharmacology
Industriepark Höchst, Gebäude G879
65926 Frankfurt am Main

Contact:
Prof. Dr. Mike J. Parnham
phone: +49 69 8700-25071
michael.parnham@ime.fraunhofer.de

www.ime.fraunhofer.de

DRUG REPURPOSING FOR MULTIPLE SCLEROSIS

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 relapsing-remitting 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.