KEY FEATURES AND ADVANTAGES

- Whereas most current drug development initiatives focus on immune modulation OR brain repair in neuroinflammatory disorders, the current target affects both.
- The target involves a novel pathway that was not yet linked to remyelination.
- In vitro, ex vivo, and in vivo data available with (cell-specific) knockout models and compounds.
- The UHasselt Biomedical Research Institute has decades of experience in the preclinical MS field with a specific emphasis on de/remyelination and inflammatory processes.

MARKET POTENTIAL

MS affects over 2.3 million people worldwide and is the leading cause of non-traumatic neurologic disability in young adults in the developed world. Annual treatment costs using the current antibody therapies amount to over 60.000 USD.

The global MS market is growing and expected to reach 25 billion USD in the next 5 years. Current blockbuster drugs (e.g. Ocrevus) achieve annual global sales of over 5 billion USD.

OUTSTANDING OPPORTUNITY

Patent application is available for licensing.

UHasselt is searching interested parties to complete development and commercialization. More information about the target and the results is available on request and after signing an NDA.

ABOUT THE RESEARCH TEAM

Prof. dr. Jeroen Bogie studies the impact of fatty acid metabolism on the inflammatory and reparative features of immune and glial cells in neuro- and auto-inflammatory disorders.

Prof. dr. Jerome Hendriks aims at elucidating how cellular lipid metabolism impacts inflammatory and repair processes within the CNS with a focus on Multiple Sclerosis (MS).

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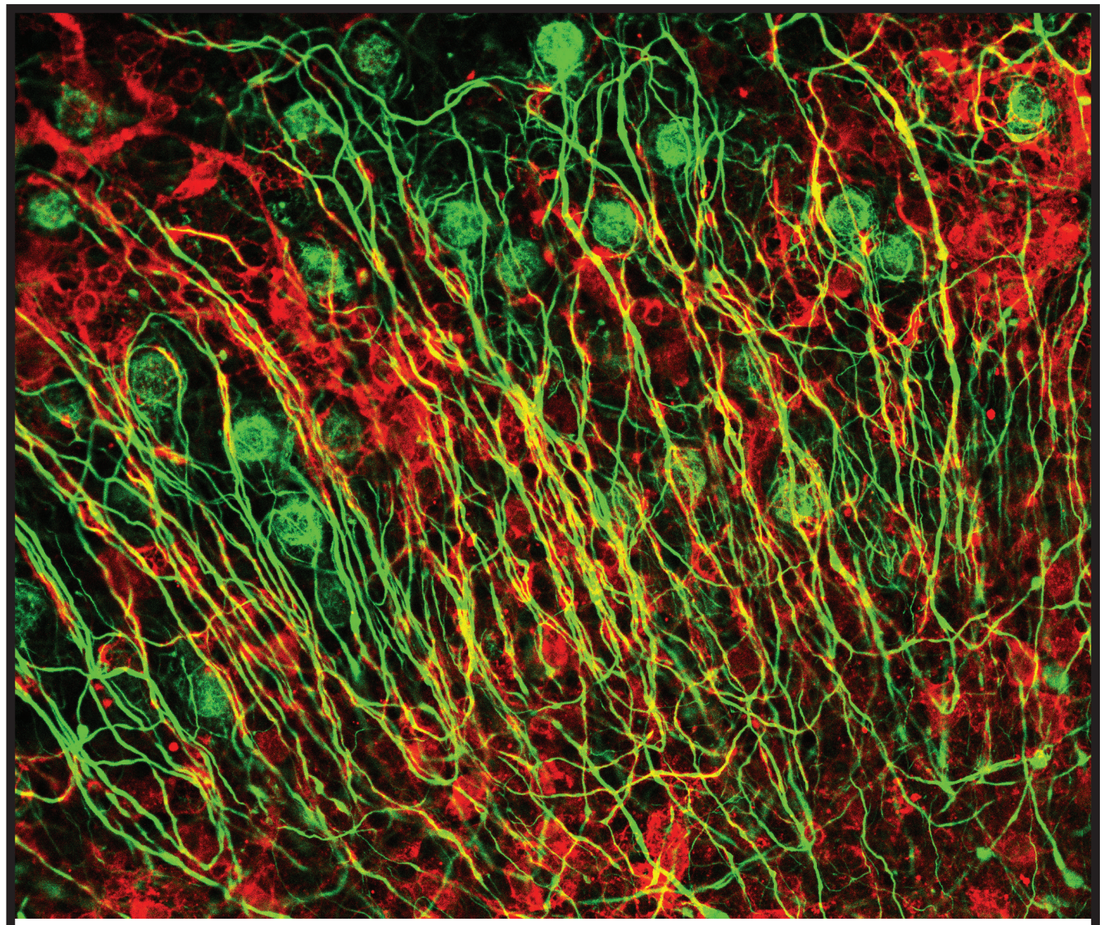

LICENSING OPPORTUNITY:

Promoting a reparative microenvironment and remyelination in multiple sclerosis

UHASSELT
KNOWLEDGE IN ACTION

BACKGROUND INFORMATION

Multiple sclerosis (MS) is a chronic inflammatory demyelinating disease of the central nervous system (CNS). Currently, there is no cure for MS and available therapies are unable to stop neurological decline and promote CNS repair. Therefore, there is an urgent need for novel therapies that are effective not only in the early disease stages, but also in the chronic progressive stage of the disease. For the induction of repair of damaged myelin sheaths, or remyelination, the chronic inflammatory response has to be resolved and oligodendrocyte precursor cells (OPCs) need to differentiate in mature, myelinating oligodendrocytes.

UHasselt researchers showed that inhibition of their target promotes CNS repair by resolving inflammation and inducing OPC differentiation.

COMPELLING RESULTS

Target deficiency promotes remyelination in the cuprizone model, an animal model for de- and remyelination. In this model, we observed less demyelination, more remyelination, and a reduced macrophage lipid load and CNS infiltration ***(Figure 1)***.

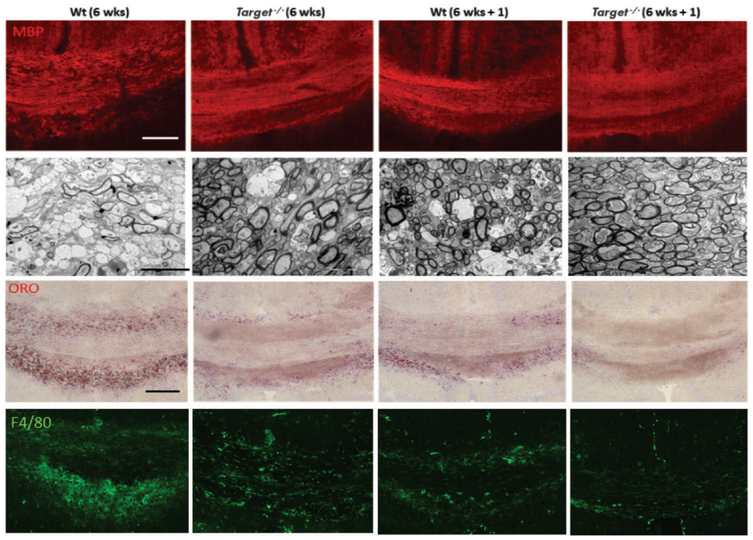

Figure 1. *Representative images of MBP (myelin)/neurofilament (neuron) stain of cerebellar brain slices (BSCs) that were demyelinated using lysolecithin. Following demyelination, BSC were treated with vehicle (DMSO) or a Target inhibitor (1μM). Fluorescence microscopy was used to assess remyelination 6d post-demyelination.*

Target deficiency leads to enhanced remyelination in ex vivo demyelinated brain slices (data not shown). These effects could be mimicked by using a Target inhibitor ***(Figure 2)***

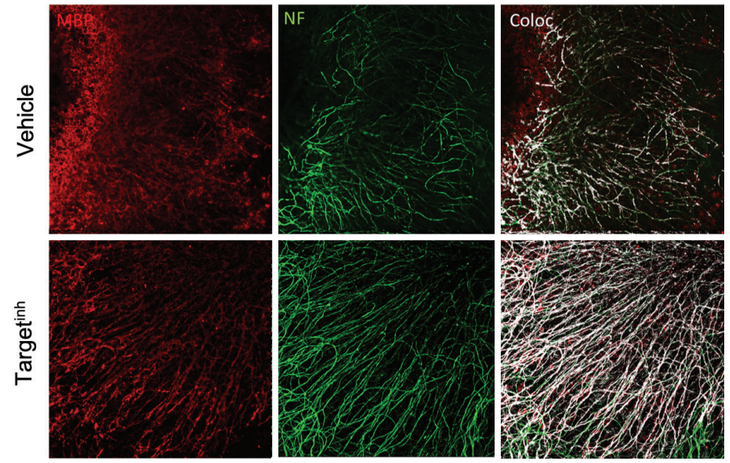

Figure 2. *Representative images of MBP (myelin)/neurofilament (neuron) stain of cerebellar brain slices (BSCs) that were demyelinated using lysolecithin. Following demyelination, BSC were treated with vehicle (DMSO) or a Target inhibitor (1μM). Fluorescence microscopy was used to assess remyelination 6d post-demyelination.*

Target deficiency promotes oligodendrocyte differentiation in vitro ***(Figure 3)***. In parallel, Target deficiency and inhibition reduces the inflammatory intracellular accumulation of lipids in macrophages in vitro, as well as increases the secretion of repair-promoting factors (data not shown).

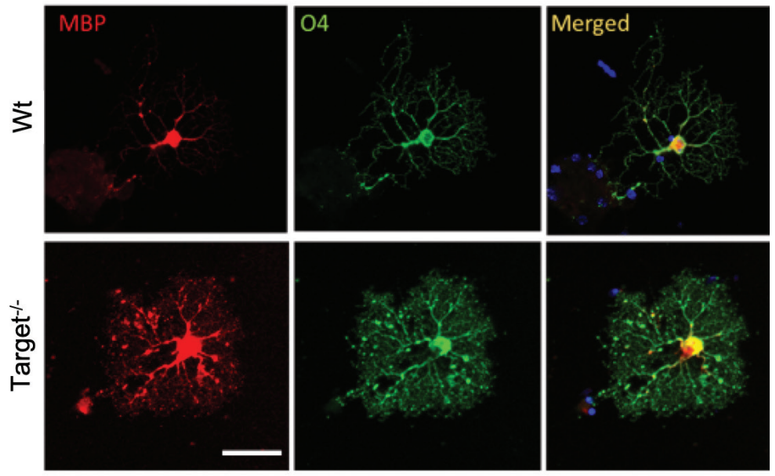

Figure 3. *Representative images of MBP (mature oligodendrocyte marker) and O4 (oligodendroglial lineage marker) stain from wt and Target-/- oligodendrocyte precursor cells (OPC). Fluorescence microscopy was used to assess OPC maturation 6 days after exposure to a differentiation cocktail.*