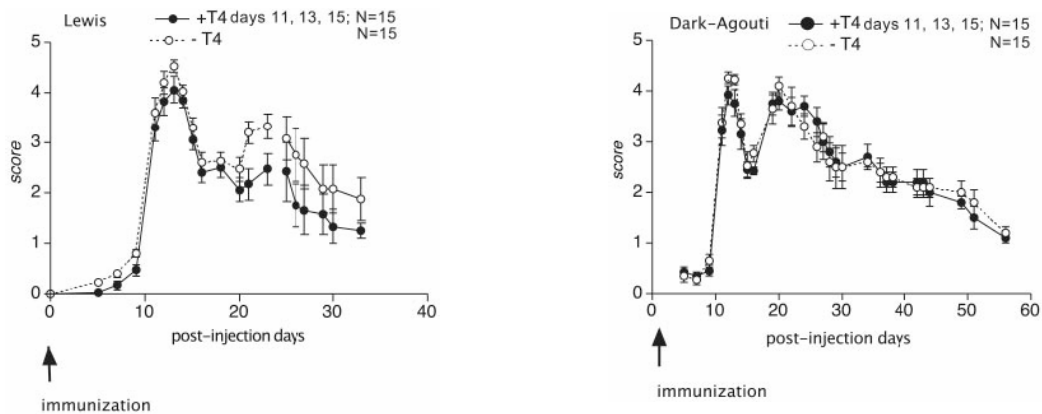


Experimental model of demyelinating diseases

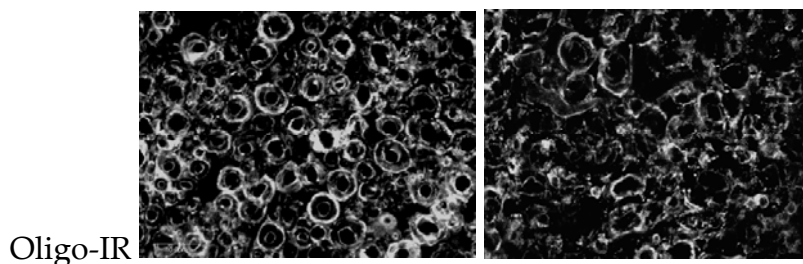
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Experimental allergic encephalomyelitis (EAE) is a widely used experimental model of multiple sclerosis. Chronic disabilities in multiple sclerosis are believed to be due to neuron damage and degeneration, which follow remyelination failure. Using this model, we have obtained positive results in promoting re-myelination and neuroprotection in EAE by recruiting progenitors and channelling them into oligodendroglial lineage through administration of thyroid hormone. This protocol is in a phase 2 clinical trial.



Control

EAE



Oligo-IR

1. Description of the model

EAE can be induced in Lewis and Dark-Agouty (DA) female rats by injecting an emulsion of guinea-pig spinal cord in complete Freund's adjuvant in both hind-paws. Eight to ten days after, animals display a severe force deficit (severe paralysis). Acute phase spontaneously recovers and subsequent course of the disease is different in Lewis and DA rats. Relapse is less severe and shorter in Lewis than in DA rats. Lewis EAE is a preferential model to study inflammation and related therapies; DA EAE is a preferential model to study demyelination and related therapies. EAE can be also induced in mouse.

We can also test effectiveness of treatments providing morphological, biochemical and molecular results (according to the established protocol).

2. Innovative aspects of the product

- Standardized parameters
- Validated model

3. Main advantages of the offer

Not available as ready to use commercial item

4. Technology keywords

Experimental allergic encephalomyelitis (EAE), thyroid hormone.

5. Current stage of development

In vivo and in vitro pilot study of active molecules have been performed.

6. Intellectual property rights

Non patentable as part of laboratory expertise

Scientific publications

Calzà L, Fernandez M, Giuliani A, D'Intino G, Pirondi S, Sivilia S, Paradisi M, Desordi N, Giardino L. Thyroid hormone and remyelination in adult central nervous system: a lesson from an inflammatory-demyelinating disease. *Brain Res Brain Res Rev* 2005, 48:339-46.

Fernandez M, Giuliani A, Pirondi S, D'Intino G, Giardino L, Aloe L, Levi-Montalcini R, Calzà L. Thyroid hormone administration enhances remyelination in chronic demyelinating inflammatory disease. *Proc Natl Acad Sci USA* 2004, 101:16363-8.

Giardino L, Giuliani A, Fernandez M, Calzà L. Spinal motoneurone distress during experimental allergic encephalomyelitis. *Neuropathol Appl Neurobiol* 2004, 30:522-31.

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