PSA-20
Polysialic acid for the treatment of neurodegenerative diseases

Marcus Karlstetter, Jens Kopatz, Thomas Langmann, Harald Neumann, Anahita Shahraz

Invention
There is no satisfactory therapy to prevent the loss of synapses, axons or neurons in neurodegenerative diseases, such as senile macular degeneration, multiple sclerosis and Alzheimer’s disease. Several studies demonstrate that this neuronal damage is partly mediated by proinflammatory cytokines released by tissue macrophages and by reactive oxygen species.

The present invention allows preventing the production of proinflammatory cytokines and reactive oxygen species through the application of low molecular weight polysialic acid PSA-20. In vitro studies showed that PSA-20 prevents the activation of human macrophages and human microglia thereby inhibiting the production of cytotoxic proinflammatory cytokines and reactive oxygen species. It has been shown, that this anti-inflammatory effect is mediated through the human lineage specific receptor Siglec-11.

Furthermore, in vivo studies revealed that the administration of PSA-20 prevents disease symptoms in an animal model of multiple sclerosis and suppresses the activation of retinal microglia. Thus, PSA-20 represents an ideal candidate for the treatment of neurodegenerative diseases.

Commercial Opportunities
The invention offers a novel approach for the prevention of severe neuronal damage in the course of neurodegenerative diseases.

Competitive Advantages
• PSA-20 prevents:
  - the production of reactive oxygen species and of proinflammatory cytokines
  - the activation of microglia in an animal model of macular degeneration
  - disease symptoms in an animal model of multiple sclerosis
• PSA-20 has no cytotoxic effect

Current Status
On behalf of the University Hospital Bonn and the University Cologne, PROvendis offers a patent license as well as a research collaboration with licensing option to innovative companies. In case of interest we will be pleased to inform you about the patent status.

Further Reading

An invention of the University Hospital Bonn and the University Cologne.

Contact:
Ref. No.: 3532
Dr. Juergen Walkenhorst

PROvendis GmbH
Schloßstrasse 11-15
D-45468 Mülheim an der Ruhr, Germany
Phone: +49 (0)208 94 105 25
Fax: +49 (0)208 94 105 50
Email: jw@provendis.info
Web: www.provendis.info

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