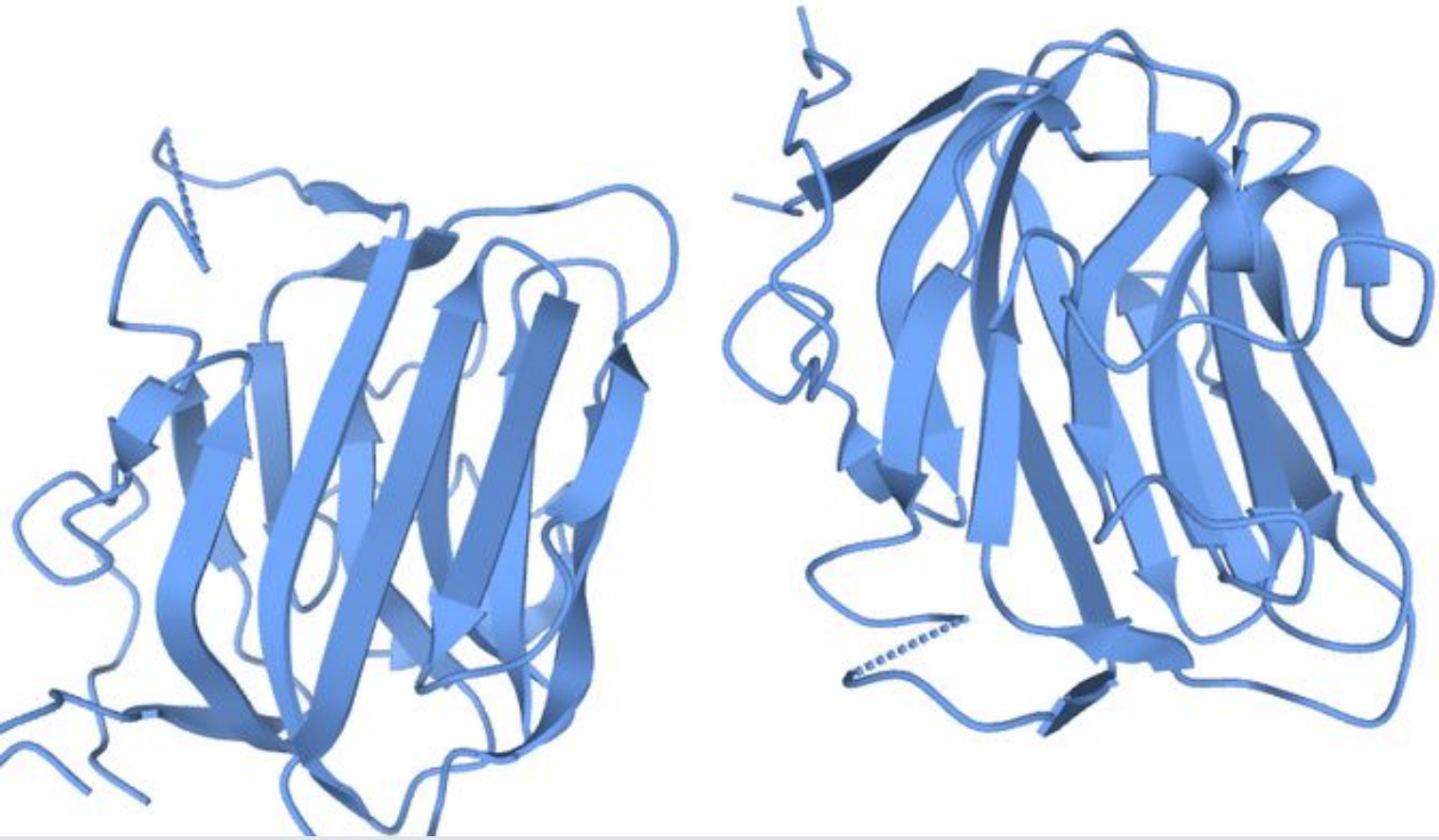
Powered by: IN-PART

A Soluble Fragment of Neural Agrin for the Treatment of Neurogenic Sarcopenia

Inactivation of agrin by neurotrypsin leads to agrin fragment which can be used as a biomarker for this subtype of neurogenic sarcopenia



Please note, header image is purely illustrative. Source: PDB ID 1PZ7, 10.2210/pdb1PZ7/pdb, CC0

Seeking

Development partner

About **LMU Munich**

Ludwig-Maximilians-Universität München is the University in the heart of Munich. LMU is recognized as one of Europe's premier academic and research institutions. The LMU Munich community is engaged in generating new knowledge for the benefit of society at large.

Background

Sarcopenia, the age-associated loss of muscle mass and physical performance, is a great challenge for the affected patient and society as a whole. Until today, no pharmacological therapy for sarcopenia is available. A high protein diet and physical exercise are recommended. The causes for sarcopenia are multifactorial. One reason is the loss of motoneurons and the degeneration of the neuromuscular junction (NMJ) with the consecutive atrophy of the corresponding muscle fibers. The proteoglycan agrin is responsible for the stability of the NMJ.

Tech Overview

Inactivation of agrin by neurotrypsin leads to a 22kDa large c-terminal agrin fragment (CAF) which can be used as a biomarker for this subtype of neurogenic sarcopenia. This inactivation of agrin leads to sarcopenia in a neurotrypsin overexpressing mouse model. Injection of neural agrin improves muscle pathology in the animal model. Neural agrin (NT-1654) is now also tested successfully in spinal muscular atrophy in mice.

The aim of the project is to establish CAF as a biomarker for neurogenic sarcopenia and to develop the soluble fragment of neural agrin further for the treatment of neurogenic sarcopenia.

Further Details

- Drey M, Krieger B, Sieber CC, et al (2014) Motoneuron loss is associated with sarcopenia. J Am Med Dir Assoc 15:435–439 . doi: 10.1016/j.jamda.2014.02.002
- Drey M, Sieber CC, Bauer JM, et al (2013) C-terminal Agrin Fragment as a potential marker for sarcopenia caused by degeneration of the neuromuscular junction. Exp Gerontol 48:76–80 . doi: 10.1016/j.exger.2012.05.021
- Bütikofer L, Zurlinden A, Bolliger MF, et al (2011) Destabilization of the neuromuscular junction by proteolytic cleavage of agrin results in precocious sarcopenia. FASEB J 25:4378–93 . doi: 10.1096/fj.11-191262
- Hettwer S, Lin S, Kucsera S, et al (2014) Injection of a soluble fragment of neural agrin (NT-1654) considerably improves the muscle pathology caused by the disassembly of the neuromuscular junction. PLoS One 9:e88739 . doi: 10.1371/journal.pone.0088739
- Boido M, De Amicis E, Valsecchi V, et al (2018) Increasing Agrin Function Antagonizes Muscle Atrophy and Motor Impairment in Spinal Muscular Atrophy. Front Cell Neurosci 12:17 . doi: 10.3389/fncel.2018.00017

Stage of Development

PoC: Injection of neural agrin improves muscle pathology in animal model.

The current antibody in the ELISA for the identification of neuronal CAF is not specific enough. Currently a new more specific antibody is tested.

Benefits

- Easy diagnosis of a subtype of neurogenic sarcopenia
- First therapy for neurogenic sarcopenia

Applications

- Diagnosis and treatment of a subtype of neurogenic sarcopenia

Opportunity

Searching a Partner for the establishment of CAF as a biomarker for neurogenic sarcopenia and the further development of the soluble fragment of neural agrin in the treatment of neurogenic sarcopenia.

For further information, please contact us.

Ludwig-Maximilians-Universität München (LMU Munich)

Office for Research and Technology Transfer

- Corporate Partnerships -

Dr. Barbara Blaurock

+49(0)89 2180-722 13

Dr. Laura Gerwin

+49(0)89 2180-722 12

corporatepartnerships@lmu.de

www.lmu.de/researchservices/corporatepartnerships/