Leibniz Gemeinschaft

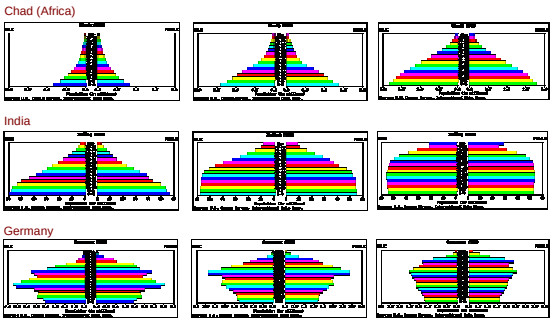
Leibniz Institute for Age Research
Fritz Lipmann Institute (FLI)
Beutenbergstrasse 11, 07745 Jena, Germany

Dr. Eberhard Fritz
Scientific Coordinator
www.fli-leibniz.de

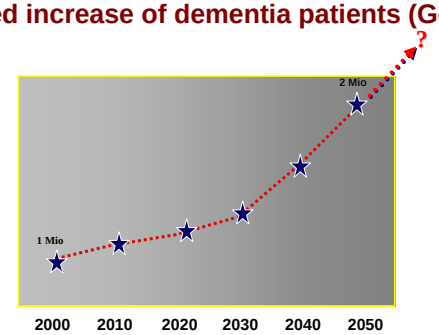
Research on age-related neurodegeneration and neurodegenerative diseases

Scientists at the Leibniz Institute for Age Research, Jena, study the molecular mechanisms of ageing and age-related diseases. Brain diseases and the decline in cognitive functions (*e.g.* Dementia), for instance in Alzheimer's disease, have especially serious effects with old age. Estimates are that at the present increase of the elderly population in Germany the number of Alzheimer's patients will have doubled to 2 million by 2050. This example strikingly illustrates the necessity to conduct research on the causes and mechanisms of neurodegenerative diseases in order to develop measures for prevention as well as new therapeutic approaches.

Demographic changes in selected countries



Predicted increase of dementia patients (Germany)



5 out of 20 research groups at FLI conduct research on neuronal diseases

Five research groups at FLI study different aspects of brain diseases and degeneration of brain functions. Research on Alzheimer's, Huntington's and other amyloid diseases includes analysis of the molecular structures of disease-causing protein deposits as well as of the mechanisms of deposit formation. We examine ways of regulating the development and function of neuronal cells and of certain brain functions by hormones and transport mechanisms in cultivated neuronal cells and suitable animal models. FLI researchers study rare diseases dependent on mutation of DNA-repair genes (such as NBS) to analyse the effects of DNA repair and genomic instability on neurodegenerative processes.

Marcus Fändrich
Alzheimer & Amyloidoses

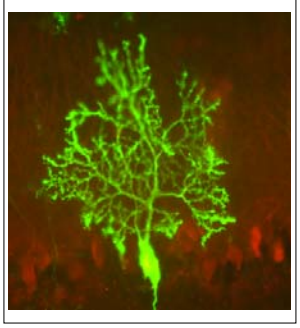
The deposition of amyloid fibrils as plaques is typically seen in degenerated brain areas of Alzheimer's patients. The pictures shows such amyloid fibrils seen by electron microscopy. The research group of Markus Fändrich works on both the molecular structure of amyloid fibrils and on the mechanism of their generation.

Picture: Marcus Fändrich

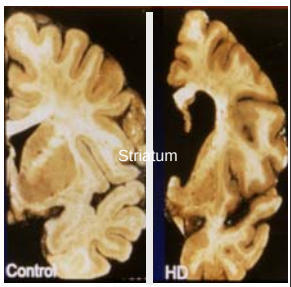
Heike Heuer
Thyroid hormones in the brain

Thyroid hormones are indispensable for normal brain functions. Their lack during development leads to severe mental and movement disorders (cretinism). The research group of Heike Heuer analyses the role of thyroid hormomes in development of neuronal cells. One particular focus relates to the question how the thyroid hormones are transported to and internalized into the neuronal cells.

Picture: Heike Heuer
GFP-stained neuronal cell of the mouse cerebellum.



Gabriele Schilling
Huntington's Disease (HD)



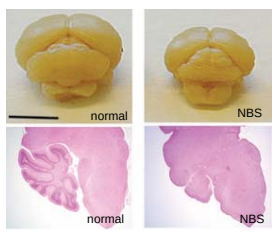
In the course of Huntington's disease, the striatum area of the brain becomes drastically degenerated. This degeneration is caused by aggregation of the large protein huntingtin. The research group of Gabriele Schilling investigates the mechanisms leading to proteolytic cleavage of huntingtin, which is thought to contribute to the aggregation. Understanding this mechanism of cleavage may allow to open new strategies for therapy.

The picture compares normal areas (control) with an area of the striatum of a Huntington patient (HD) in a clinically progressive state.

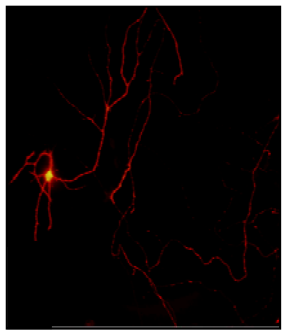
Zhao-Qi Wang
Nijmegen Breakage Syndrom (NBS)

Continuous DNA repair ensures the correct maintenance of genetic information during the lifetime of any cell, despite constant threats by endogenous and exogenously-induced DNA damage. DNA repair is severely compromised in rare human genetic diseases caused by mutations in DNA repair genes. Such diseases (*e.g.* NBS) are clinically characterized by immundeficiency, high cancer incidence, and neuronal degeneration. The latter particularly effects the Purkinje cells of the cerebellum, thus causing progressive ataxia.

Picture: Z.Q. Wang
Comparison of size, shape and histology of a normal cerebellum with a degenerated cerebellum of a NBS mouse.



Christoph Kaether
Alzheimer's Disease



The characteristic structure of neurons enables cellular transport through the axons over long distances. In neurons of Alzheimer's patients, this transport is deficient. The research group of Christoph Kaether analyses such transport mechanisms in cultivated cells. The picture shows a cultivated rat neuron with its dendrite and axonal structure. Using flourescently stained marker molecules, transport along the axons is observed by videomicroscopy.

Picture: Christoph Kaether