





The Leibniz Institute on Aging - Fritz Lipmann Institute (FLI) is a research institute of the Gottfried Wilhelm Leibniz Association, which is financed by the Federal State of Thuringia and the Federal Ministry of Education and Research with 50% each. The main focus of research is to delineate basic molecular mechanisms and its consequences for the development of dysfunctions and aging-related diseases. Around 350 employees work and conduct research at the FLI, with international employees from over 40 nations.

The **Research Group von Maltzahn** of the Leibniz Institute on Aging—Fritz-Lipmann-Institute e.V. in Jena invites applications for a

PhD student positions (TV-L E13/2) (Job ID 1919)

We are looking for **a highly motivated PhD students** to support our team in research focusing on Stem Cells in Regeneration of Skeletal Muscle. (for projects see next page).

The FLI is excellently equipped with state-of-the-art facilities like next-generation DNA sequencing (NGS) technology, proteomics, functional genomics, imaging and more.

If you are interested, check out the application guideline at **http://lgsa.leibniz-fli.de/application/ application-guideline/**, and send your application to **lgsa@leibniz-fli.de**.

We offer:

A position in a well-equipped research group of a high quality institute for age research, which harbors several state-of-the-art facilities. Our work is embedded in the Beutenberg Campus, an interdisciplinary base for innovative research. The PhD position will be integrated in our Leibniz Graduate School on Aging and Age Related Diseases

The contract conditions and the salary will be according to the collective labor agreement for public service employees of the Federal State of Germany (TV-L E13) with 50% in the first year and 65% from the second year .

Application:

Please acquaint yourself with the LGSA procedures and with the application procedure on our website **http://lgsa.leibniz-fli.de/**, fill in the application form and send it electronically to the LGSA.

Please note that your application will be made available to the LGSA selection committee for assessment. Please check here for FLI data protection (www.leibniz-fli.de/dataprotection) regulation and the processing of personal information according to the EU-GDPR.

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Lab Julia von Maltzahn

Skeletal muscle serves a multitude of functions in the organism including voluntary locomotion and exhibits a remarkable ability to adapt to physiological demands. Satellite cells are the stem cells of skeletal muscle and are associated with its growth, maintenance and regeneration. Aged skeletal muscle shows a significantly impaired regenerative potential. Evidence in the literature suggests that functionality of satellite cells in aged skeletal muscle is impaired due to the aged environment, but also due to intrinsic differences between adult and aged satellite cells.



My lab investigates the intrinsic differences between adult and aged satellite cells. We identified already factors which are differentially expressed in old and young satellite cells and are investigating their

function at the moment. This work will provide insights into pathways that are perturbed in aged satellite cells and allow for modification of these pathways thereby rejuvenating aged muscle.

Furthermore, we are interested in age-related diseases such as cancer cachexia (muscle wasting due to cancer) or diabetes and how satellite cells behave under those conditions.

Another research question in the lab is how rhabdomyosarcoma develop, a skeletal muscle driven cancer.

Example for a PhD project:

Intrinsic differences between satellite cells in adult and aged skeletal muscle

- FACS to isolate pure populations of satellite cells from adult and aged mice
- RNA-Seq to detect changes in gene expression between adult and aged satellite cells
- Culture of single myofibers with associated satellite cells
- siRNA mediated knockdown of pathway members detected in microarray analysis and investigation of proliferation and differentiation potential of satellite cells
- transplantation of satellite cells from adult and aged mice in young recipient mice



Regeneration potential of satellite cells in aged skeletal muscle and approaches to normalize satellite cell function in the aged

- damage of skeletal muscle in adult and aged mice and investigation of the regeneration potential (immunohistochemistry, histological stainings, etc.)
- analysis of loss of function in satellite cells during regeneration of skeletal muscle (aged and young) by injection of self-delivering siRNAs and/or normalization of tissue homeostasis in the aged (injection of chemical compounds in regenerating skeletal muscle and analysis of regeneration process)

selected recent publications:

- 1. Schmidt M*, Schüler SC*, Hüttner SS, von Eyss B and von Maltzahn J. Adult stem cells at work: regenerating skeletal muscle, 2019, Cell Mol Life Sci, epub ahead of print. *authors contribute equally.
- 2. Ahrens HE*, Huettemeister J*, Schmidt M, Kaether C and von Maltzahn J. Klotho expression is a prerequisite for proper muscle stem cell function and regeneration of skeletal muscle, 2018, Skelet Muscle, 8:20. *authors contribute equally.
- 3. Mayerl S, Schmidt M, Doycheva D, Darras VM, Hüttner SS, Boelen A, Visser TJ, Kaether C, Heuer H^{*} and von Maltzahn J^{*} **Thyroid hormone transporters MCT8 and OATP1C1 control skeletal muscle regeneration**. 2018, Stem Cell Rep accepted, *corresponding authors.
- 4. Lukjanenko L, Jung MJ, Hegde N, Perruisseau-Carrier C, Migliavacca E, Rozo M, Karaz S, Jacot G, Schmidt M, Li L, Metairon S, Raymond F, Lee U, Sizzano F, Wilson DH, Dumont NA, Palini A, Fässler R, Steiner P, Descombes P, Rudnicki MA, Fan CM, von Maltzahn J, Feige JN#, and Bentzinger CF#. Loss of Fibronectin from the Aged Stem Cell Niche Affects the Regenerative Capacity of Skeletal Muscle, 2016, Nat Med, 22: 897-905. #corresponding authors.
- 5. Price FD*, von Maltzahn J*, Bentzinger CF, Yin H, Wilson DH, Grayston A and Rudnicki MA. Inhibition of JAK/STAT Signaling Rejuvenates the Function of Aging Satellite Cells, 2014, Nat Med, 10:1174-81. *authors contribute equally