



**Press Release Cluster of Excellence, University of Cologne**

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**Researcher from the Cluster of Excellence CECAD at the University of Cologne receives Junior Prize for neuromuscular disease from the German Society for muscular disease (Deutsche Gesellschaft für Muskelkranke e.V.(DGM)).**

**Koordinator:**  
Prof. Dr. Jens Brüning

**Cologne, March 31, 2011 – Onset and progression genetically caused neuromuscular disease caused by mitochondrial dysfunction (mitochondrial myopathies) as well as age-related muscle loss (sarcopenia) can be delayed by activating mitochondrial function. A promising therapeutic approach, as determined in studies with mouse model, is administration with the already approved drug Bezafibrate, which pharmacologically activates the mitochondrial function.**

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The research interest of Dr. Tina Wenz focuses on the development of therapeutic approaches for neuromuscular diseases caused by disrupted mitochondrial function. Mitochondria are the power station of the cell and create the energy which is necessary for the cell and consequently the complete organism to live. Thereby, their function determines cell fate.

[www.exzellenzcluster.uni-koeln.de](http://www.exzellenzcluster.uni-koeln.de)

A disturbance of the functional ability of the mitochondria leads severely affects brain, heart, the liver, endocrinal system and muscle and also results in developmental disorders. While genetically contingent disturbances of the mitochondria are considered to be rare, a variety of common age-related disorders such as muscular atrophy (sarcopenia) are related to reduced mitochondrial function. In two research approaches, Dr. Tina Wenz was able to show that increased mitochondrial activity can significantly slow the process of age-related as well as genetic-related neuromuscular disorders.

A promising approach in the treatment of the genetic-related mitochondrial myopathy exists in the activation of the PPAR/PGC-1 $\alpha$  signal pathway. The metabolism of the mitochondria is improved thereby improving the energy supply of the muscle. The approved medication Bezafibrate, currently used for treatment of [hyperlipidaemia](#), pharmacologically activates the PPAR/PGC-1 $\alpha$  signal pathway, which represents a

valuable therapeutic option. Dr. Wenz's work presents the first therapeutic approach for mitochondrial myopathy, which up to date lack causal treatment.

A therapeutic effect of the activation of the PPAR/PGC-1 $\alpha$  signal pathway was also verified for age-related muscle-loss (sarcopenia). The activated mitochondrial function enhanced muscular energy supply and thereby preserved muscle mass and function. Subsequently, the retained muscle mass, as an important component of the metabolism, resulted in improved insulin and glucose During aging.

**Personal Data:**

After studying Chemistry in Kaiserslautern, Dr. Wenz graduated in 2004 from the Max-Planck-Institute for Biophysics in Frankfurt. She did her postdoctoral work at the Max-Planck-Institute in Frankfurt as well as the Department of Neurology at the University of Miami, USA. In 2010 she started her own research group at the Institute for Genetics, University of Cologne funded by the Emmy-Noether-Programm of the DFG. She is also a member of the Cluster of Excellence CECAD.

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