

January 2023

Did you register for the ORS 2023 Annual Meeting yet?

Be sure to register for the Spine Symposium - you will not be able to enter the session without registration.



Register for the ORS Spine Section Scientific Symposium & Reception!

When: Friday, February 10th, 2023 3:00 PM – 9:00 PM

Join us for an expanded, half-day program with the overall theme of "Enhancing Spine Research through Diversity, Mentoring and Collaboration". At the conclusion of the Symposium, a Networking Reception will take place from 8 – 9:00 PM.

View the <u>full schedule here.</u>

Gather with your section



Congratulations 2023 Spine Section Diversity Stipend Award Winners!

This year, the Spine Section offered the 2023 Spine Section Diversity Stipend Awards. The goal of these awards is to increase diversity and equitable access to spine research.

Congratulations to the following winners:

Alex Villegas Dagoberto Pina Hosni Cherif Christian Gonzalez Kashaf Zaheer Sabrina Delva Obinna Fidelis



Pictured: Petra Cazzanelli, MSc

Research Section Member Spotlight

This issue features **Petra Cazzanelli, MSc** - Ph.D. Candidate in Biomedical and Chemical Engineering at Rochester Institute of Technology

Undergraduate Degree: BSc in Biotechnology and Food Science, University of Natural Resources and Life Sciences in Vienna (Austria)

Graduate Degree: MSc Biotechnology, University of Natural Resources and Life Sciences and Medical University of Vienna (Austria)

Who do you consider your mentors?

My supervisor and PI, Prof. Karin Wuertz-Kozak, has been an important mentor during my Ph.D. Her support and expertise have been invaluable on both a scientific and personal level. Furthermore, I am grateful for the guidance and mentoring from Prof. Lisbet Haglund (McGill University), whose extensive knowledge has been incredibly valuable.

What is your specific area of interest in research?

My research interest lies in the role of microRNAs in inflammation and mechanosensing in the context of intervertebral disc degeneration. Understanding their role in regulating and connecting these processes might provide opportunities for new therapeutic targets.

What are you currently working on?

My current work focuses on the identification of Toll-like receptor-associated microRNAs and their functional role in intervertebral disc pathophysiology, with a specific focus on inflammation, catabolism, and mechanosignaling.

What has been the biggest challenge for you lately in your research?

The intersection between bioinformatics and biological relevance. Next-generation sequencing and microRNA target prediction are wonderful tools to help us understand global changes. However, combing through the extensive amount of data and finding the biologically most relevant aspects can sometimes be challenging.

What are projects are you looking forward to?

I am looking forward to studying the role of microRNAs in mechanosensing. Changes in the reaction of IVD cells to cyclic stretching will be analyzed following the gain or loss of specific microRNAs. These studies will include transfecting human IVD cells with microRNAs and their inhibitors, followed by the cyclic loading and subsequent analysis of relevant signaling pathways and targets.

What do you like to do outside of your work?

Growing up in the Italian Alps, I like doing anything outdoors (cycling, hiking, climbing, and running) and of course anything that has to do with food.

What is the last book you read?

A Conflict of Visions: Ideological Origins of Political Struggles by Thomas Sowell

A theory on why the same people tend to end up on opposite political sides independently of the nature of the issue, even when the issues vary enormously in subject matter and sometimes hardly seem connected.

What is the most unusual/unexpected item sitting on your desk right now?

My favorite savory Indian snack Khakhara, which a friend brought me from Gujarat, India. As I am known for being a

foodie and for always snacking, friends tend to bring me food from all over the world.





Taylor & Francis

Paper Review

Petra also contributed to this research article...

The mitophagy receptor BNIP3 is critical for the regulation of metabolic homeostasis and mitochondrial function in the nucleus pulposus cells of the intervertebral disc.

Madhu V, Hernandez-Meadows M, Boneski PK, Qiu Y, Guntur AR, Kurland IJ, Barve RA, **Risbud MV**. Autophagy. 2023 Jan 10:1-23. doi: 10.1080/15548627.2022.2162245. PMID: 36628478.

Autophagy is known to be a commonly dysregulated pathway during intervertebral disc (IVD) degeneration, and recent studies have indicated a complex interplay between autophagy, apoptosis, and cell survival. Due to the avascular nature of the tissue, the metabolic adaption of nucleus pulposus (NP) cells to this hypoxic environment is essential for their survival, and a shift in their metabolic activity is a known contributor to degeneration. The role of mitochondria in their metabolism has, however not been extensively studied so far. In this study, the authors investigate the role of the outer mitochondrial membrane receptor BNIP3 in mitophagy, mitochondrial function, and the metabolism of NP cells.

The functional role of BNIP3 was studied using a loss-of-function approach. NP cells with BNIP3 knockdown were analyzed for their mitochondrial morphology, number, and mitophagy (Immunocytochemistry, Western blot, Mitochondrial morphology analysis). The effect of BNIP3 loss on the metabolic activity of NP cells was investigated by analyzing the maximum glycolytic capacity, ATP production rate, and metabolic flux (Seahorse XF analysis, 13C-Metabolic flux analysis). Furthermore, transcriptomic analysis of BNIP3 deficient cells was performed and compared to degenerative human disc transcriptomes. Lastly, the BNIP3 loss was studied in vivo in bnip3 knockout mice (µCT analysis and Immunohistochemistry).

This extensive study provides us with a deep inside into how the loss of BNIP3 in NP cells leads to mitochondrial dysfunction and causes changes in the metabolism, cellular bioenergetics, and inflammation, as well as early-stage disc degeneration in young adult mice. BNIP3 was shown to not only regulate mitophagy but also to play an important role in maintaining metabolic function and metabolism. More specifically, the loss of BNIP3 was shown to increase the mitochondrial number and the percentage of fused mitochondria, as well as overall mitophagy. Furthermore, BNIP3 deficiency reduced the glycolytic capacity of NP cells and lowered ATP production rates. Transcriptomic analysis showed the effect of BNIP3 loss on the dysregulation of membrane and cytoskeletal integrity, ECM-growth factor signaling, and the upregulation in biological processes involved in angiogenesis, inflammatory mediator, and homeostasis/metabolism. Finally, in vivo studies showed an increased autophagic flux, decreased disc height, and changes in COL10A1 expression. Overall, this study suggests that BNIP3 is critical for the regulation of metabolic homeostasis and mitochondrial function in NP cells.



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