

FIRST PERSON

First person – Marcel Nowak and Benjamin Suenkel

First Person is a series of interviews with the first authors of a selection of papers published in Journal of Cell Science, helping early-career researchers promote themselves alongside their papers. Marcel Nowak and Benjamin Suenkel are co-first authors on 'DCAF8, a novel MuRF1 interaction partner, promotes muscle atrophy', published in JCS. Marcel subsequently worked as a Product Manager for a life science company. Benjamin is a postdoc in the lab of Thomas Sommer at Max-Delbrück-Center for Molecular Medicine (MDC), Berlin-Buch, Germany, investigating protein biochemistry and quality control.

How would you explain the main findings of your paper in lay terms?

MN & BS: The strength of our muscles is tightly regulated by the amount of contractile proteins. Several diseases, but also simply immobility, can lead to loss of muscle proteins (called muscle atrophy), which eventually results in loss of muscle strength – a severe condition for elderly or bed-resting patients. One major contributor to muscle protein loss, the protein MuRF1, has been



Benjamin Suenkel

known for many years. However, its mode of action is still under discussion and it was speculated that MuRF1 may act together with further cellular factors. We have now identified such novel MuRF1 interaction partners, which will help us to fully understand the degradation of muscle proteins on the molecular level and might reveal strategies to counteract muscle atrophy.

Were there any specific challenges associated with this project? If so, how did you overcome them?

MN: Efficient transfection of cultured muscle cells is quite challenging. But for our initial high-throughput interaction screenings, high amounts of MuRF protein were a pre-requisite. The standard transfection methods didn't result in enough material so we had to switch to adenoviral transductions, which solved this problem.

BS: The low transfection efficiency of muscle cells using standard techniques remained an issue throughout the project. To validate our findings, we needed to establish knockout cell lines by CRISPR/Cas9, but our first trials were unsuccessful. By optimizing every step from transfecting myocytes, single-clone selection and propagation, we overcame this problem.

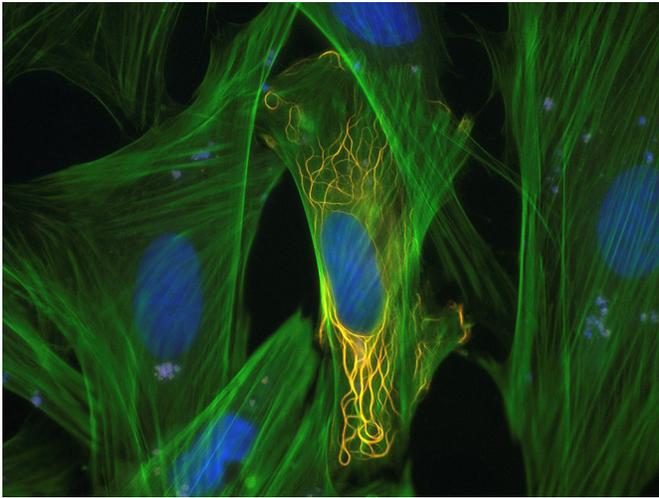
When doing the research, did you have a particular result or 'eureka' moment that has stuck with you?

MN & BS: DCAF8 was one of the most interesting interaction partners we identified for MuRF1, as it is a substrate receptor of



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A fluorescence staining of the nucleus (blue), the actin cytoskeleton (green) and MuRF proteins (orange), which are localized along fibers of rat cardiomyocytes. MN edited the picture to make it look like a piece of art for a scientific image contest.

CRL4 ubiquitin ligase complexes. After we validated the interaction between both proteins, we were interested whether DCAF8 might link MuRF1 to CRL4 complexes and started to perform co-immunoprecipitation experiments of endogenous proteins. It was really striking to us that even in the first trials we could readily detect further CRL4 components.

Why did you choose Journal of Cell Science for your paper?

MN & BS: We like that JCS publishes excellent research for a broad range of cell biologists across fields of interests. But JCS and The Company of Biologists are more than just a platform to publish research owing to their contribution to young scientists and the scientific community (e.g. travel grants, meetings and workshops). This was really appealing to us.

What motivated you to pursue a career in science, and what have been the most interesting moments on the path that led you to where you are now?

MN: Seeing living cells and their complexity under the microscope fascinated me. Of special interest to me were muscle cells, as they are constantly contracting. In order to maintain contraction, damaged muscle proteins need to be degraded and replaced rapidly. To further understand this fascinating process motivated me.

BS: I was always fascinated by proteins and how they contribute to the sophisticated machineries and pathways in our cells. Elucidating protein functions, machineries and pathways and thereby contributing to a better understanding of living cells motivates me.

Who are your role models in science? Why?

MN: I might not call him a role model, but I was really impressed by the scientific enthusiasm of Prof. Dr Friedrich Luft. Although being director of the ECRC and a well-known nephrologist, looking back on a long career, he decided to pursue another PhD in the lab of Prof. Dr Thomas Sommer. During that time, I got to know him while working together in the cell culture lab. He was so passionate about everything related to science, which really impressed me.

BS: During my biochemistry studies, we had a particular enthusiastic professor. Prof. Dr Franz X. Schmid was as passionate about his biochemical lectures and tutoring students as he was passionate about science. His analytical thinking and thoroughness on data acquisition and interpretation impressed me a lot and is the gold standard of my research.

What's next for you?

MN: After my time at the MDC, I started working as Product Manager in a life science company. Because I wanted to spend an extended time with my family, I left this job, bought a caravan and traveled with my wife and my two kids for three months around Europe. We have just returned from that trip, so I'm unpacking and returning to the routines.

BS: In December I will take a four months parental leave. Until then and afterwards, I'll be continuing the project and hope to decipher the molecular mechanism behind the MuRF1–DCAF8–CRL4 association.

Tell us something interesting about yourself that wouldn't be on your CV

MN: I am interested in all sorts of art. When I find the time, I like to paint and photograph and to edit my pictures and photos on the computer.

BS: I'm an enthusiastic hobby chef and like to cook for my friends and family. As I love Italian cuisine, I started cooking the dishes we enjoyed on a vacation and try to optimize them until they taste just like in Italy. In a way, I'm doing some research in the kitchen as well.

Reference

Nowak, M., Suenkel, B., Porras, P., Migotti, R., Schmidt, F., Kny, M., Zhu, X., Wanker, E. E., Dittmar, G., Fielitz, J. et al. (2018). DCAF8, a novel MuRF1 interaction partner, promotes muscle atrophy. *J. Cell Sci.* **132**, jcs233395. doi:10.1242/jcs.233395