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Press Release

Proteins with different evolutionary histories now do the same job

University of Tübingen researchers discover convergent evolution in mitochondria in fungi and single-celled parasites

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Mitochondria are essential organelles of cells with a nucleus – known as eukaryotic cells. These are the cells which make up fungi, plants, and animals including humans. Mitochondria work like tiny power plants, processing the energy produced by the digestion of nutrients into a form the cell can use. Yet they have a number of other functions – which are the focus of research by the working group headed by Professor Doron Rapaport at the University of Tübingen's Interfaculty Institute of Biochemistry. Nearly all mitochondrial proteins are encoded in the nucleus and after being produced in the cytosol, must be imported into the mitochondria. Scientists know of a protein complex in the outer mitochondrial membrane of the bakers' yeast which mediates the integration of newly-made proteins into that membrane. However, until now it was unknown which proteins did that work in other eukaryotic cells. In a new study, the Tübingen researchers describe how they discovered the corresponding protein complex in a single-celled parasite – thereby revealing a case of convergent evolution. That is when characteristics, molecules or organs of different species are very similar, yet have developed independently in unrelated organisms. The study is now available in the latest edition of *eLife.*

The Tübingen biochemists compared yeast with trypanosomes – single-celled organisms which occur as parasites in vertebrates and are also known as the pathogenic organisms in sleeping sickness in humans. The researchers swapped the protein complexes Mim1/Mim2 from yeast and pATOM36 in trypanosomes, demonstrating that their functions correspond: "These protein complexes are not similar in their composition – which is of different amino acid building-blocks – nor in their structural arrangement," Rapaport says. "Yet pATOM36 can reproduce nearly all the functions which yeast cells need if you take away their Mim1/2." Working with a group headed by Professor André Schneider at the University of Bern, the scientists showed that the exchange worked the

other way as well: If trypanosomes don't have their pATOM36, the Mim1/Mim2 complex is able to replace it. The results also indicate that no other proteins are involved in this transport complex in the outer mitochondrial membrane.

"Mim and pATOM36 are the products of a convergent evolution. They arose only after the ancestors of fungus and trypanosomes had diverged into different evolutionary lines," Rapaport explains. Now the path is open for further comparative studies to uncover the common basic structures and the function of the two protein complexes, which have evolved via different routes but arrived at the same result. The next major challenge is to identify the protein complex, which fulfills this function in higher organisms – above all in humans.

Publication:

Daniela Vitali, Sandro Käser, Antonia Kolb, Kai S Dimmer, Andre Schneider, Doron Rapaport: Independent evolution of functionally exchangeable mitochondrial outer membrane import complexes. *eLife*, 2018;7:e34488. DOI: https://doi.org/10.7554/eLife.34488

Insight article about the new research results: https://elifesciences.org/articles/38209

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