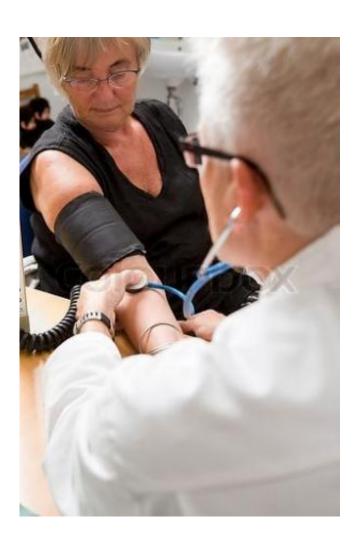


Punctured cell membranes lead to high blood pressure

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Researchers from the University of Southern Denmark have identified how a mutated protein can lead to holes in a protein sitting in a cell's



membrane. Such holes cause high blood pressure, and the discovery can now lead to new and better medication for high blood pressure.

High <u>blood pressure</u> can be caused by many things - one of them being a specific mutated protein. Now the researchers at University of Southern Denmark have found out exactly what unfortunate events in the human organism are initiated by the mutated protein.

"This knowledge can now lead to new and better medicines for <u>high</u> <u>blood pressure</u>", says the lead author of a new scientific publication, PhD student Wojciech Kopec from the Center for Biomembrane Physics (MEMPHYS) at the University of Southern Denmark.

He explains that some years ago research colleagues from University of Aarhus found out that a particular mutated protein is associated with high blood pressure. But the exact mechanism at play could not be clarified until now.

Wojciech Kopec and his colleagues, Himanshu Khandelia and Bastien Loubet from Memphys and Hanne Poulsen from University of Aarhus, have now revealed the mechanism at play: The mutated protein leads to the formation of holes in a protein sitting in a cell's membrane, and so the cell can no longer control what is allowed into and out of the cell interior. The holes are made where the cell controls its content of salts. A normal, healthy cell has full control of how much salt (sodium ions) must be removed from within the cell so that it can maintain a perfect salt balance in the organism, it is a part of.

"But when there are holes, sodium ions can penetrate into the cell, so the salt levels go up. Too high salt levels are associated with many diseases, including high blood pressure", explains Wojciech Kopec.

This specific knowledge is particularly useful for the medical industry



involved with developing new drugs.

"Medicine is molecules, and therefore it is in principle easy to develop a molecular formula that can close the holes in the membrane", says Wojciech Kopec.

The researchers found the mechanism by running a computer simulation on one of the country's most powerful computer clusters, Horseshoe 6, which is situated at University of Southern Denmark.

More information: The Molecular Mechanism of Na+, K+-ATPase Malfunction in Mutations Characteristic for Adrenal Hypertension. Wojciech Kopec, Bastien Loubet, Hanne Poulsen, and Himanshu Khandelia. *Biochemistry*. DOI: 10.1021/bi401425g . Publication Dat. (Web): 15 Jan 2014.

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