A TRP that makes our cells feel hyper

March 8 2012

A large change in the volume of a cell, from its basal level, is detrimental to its health. Therefore, our cells are equipped with mechanisms to maintain their constant volume. When a cell detects an environmental change that will alter its volume, due to changes in the osmotic pressure, it will adjust its internal water content to counter these influences. This is done through the movement of ions into, and out of, the cell through specialised ion channels. However, the identity of the molecules responsible for this phenomenon remain largely unknown. Professor Yasunobu OKADA, the Director-General of The National Institute for Physiological Sciences and the Vice-President of the National Institutes of Natural Sciences, and his research team, have identified the key molecule preventing the shrinking and eventual death (apoptosis) of cells when they are subjected to a condition of hyperosmolarity.

This is where there is an abnormal increase in the osmolarity of a solution, and can occur due to processes such as dehydration from intense exercise or superfluous salt. The molecule they describe is a variant of TRPM2, containing a truncated C terminus. TRPM2 is member of the TRP family, a group of ion channels known to be involved in altering sensations including pain, temperature, pressure, taste and vision. It was further revealed that the TRPM2ΔC molecule was activated through its binding to CD38 (cyclic ADP-ribose hydrolase), a glycoprotein associated with HIV infection, cancer, type II diabetes mellitus, and oxytocin secretion, among other things.

Professor Yasunobu OKADA expects that the newly identified molecule research should open doors into new avenues of research into these

diseases, and aid in, not only, the elucidation of their pathological characteristics, but also their potential treatments in the future.

This research result has been published in the February issue of the *Journal of Physiology* (London) and introduced in one of the Perspectives of this Journal.

This research was collaborated with Doctor Tomohiro NUMATA and Professor Yasuo MORI from Kyoto University and Professor FrankWehner from Max-Planck-Institute of Molecular Physiology and supported by a grant-in-aid for scientific research from the Japan Society for the Promotion of Science.

Provided by National Institute for Physiological Sciences