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GREEK MINISTRY OF EDUCATION, RESEARCH AND RELIGIOUS AFFAIRS.
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FORTH
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Heraklion 15/3/2018

PRESS RELEASE

International scientific collaboration between IMBB-FORTH Researchers and Scientists in Paris, France uncovers a novel function of aspirin as a caloric restriction mimetic agent

The findings of the study, published in the international scientific journal *Cell Reports* reveal a novel molecular mechanism underlying the beneficial effects of aspirin on healthspan.

IMBB-FORTH Researchers, Dr. Maria Markaki and Dr. Nektarios Tavernarakis (Professor at the Medical School, University of Crete, and Chairman of the Board, FORTH) in collaboration with the team of Prof. Guido Kroemer (Université Paris Descartes/Paris V, Sorbonne), among others, demonstrate that aspirin and its active metabolite salicylate inhibit the activity of EP300 acetyltransferase and induce autophagy.

Autophagy is a conserved, tightly regulated process that degrades proteins, lipids and other macromolecules and removes unnecessary or dysfunctional organelles. It has a housekeeping role under physiological conditions, and an adaptive, homeostasis preserving role under nutrient deprivation or stress. Ageing is associated with marked decrease of autophagic activity. Deregulated autophagy often correlates with accelerated ageing and age-related pathologies such as cancer, muscular disorders and neurodegeneration. Autophagy may be considered as one of the major anti-aging mechanisms because it assures recycling and hence rejuvenation of damaged cytoplasmic components, including entire organelles such as mitochondria. Manipulations aiming at restoring or inducing autophagy can reduce the incidence of age-related disease and extend healthspan and lifespan. The nature of these interventions can be nutritional (i.e., fasting or caloric restriction), behavioural (i.e., physical activity) or pharmacological.

Using the simple nematode *Caenorhabditis elegans* and the mouse (*Mus musculus*) as model organisms, the research teams of Prof. Tavernarakis and Prof. Kroemer have now shown that aspirin and its active metabolite salicylate essentially mimic the effects of caloric restriction. This is accomplished because aspirin has the capacity to reduce the overall levels of protein acetylation and to induce autophagy. More importantly, the new data reveal that aspirin triggers cardioprotective mitophagy, a selective type of autophagy mediating the elimination of damaged mitochondria, in mice and nematodes. These findings identify aspirin as an evolutionarily conserved, caloric restriction mimetic.

The evolutionary conservation of the regulatory factors involved in this highly coordinated response to aspirin treatment suggests that similar mechanisms operate in humans. These insights could effectively contribute towards tackling numerous age-associated pathological conditions.

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Relevant links: http://www.cell.com/cell-reports/ & http://www.elegans.gr/