CellHealth™ Institute Launches New Cell Health Formula

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CellHealth™ Institute™ launched its cell health formula, every-cell™ on May 1, 2013. Developed by a team of cell health and aging experts, and in partnership with GMP manufacturers, everycell contains more than 90 essential phytonutrients, vitamins, and minerals that the body needs to function optimally. Using natural ingredients including Fruitura™ and Aquamin™, the proprietary complexes of everycell deliver these nutrients to a person’s cells, helping to create the ideal cellular environment to support a healthy lifestyle.

The everycell formula has seven unique complexes that support: cell regulation; free radical scavenging; DNA repair; telomere maintenance; calorie restriction, digestion and absorption. These complexes were perfected over time by CellHealth Institute scientists to address the intricate infrastructure of cell health, including cell repair, aging, and replication, which directly affect the body’s ability to function on a day-to-day basis. Studies comparing the present and upcoming everycell formulations on stem cell health and function are ongoing at the University of Miami.

"It’s health," said Dr. Vincent Giampapa, CellHealth Institute’s chief medical officer. "Simply stated, when our cells are healthy, we are healthy. The everycell formula is specifically designed to be taken daily as a cell therapy to provide the natural plant-derived complexes needed to help people work, play and rest at their optimal level. It's no longer simply about the vitamins and minerals, but about creating the cellular environment for those nutrients to be utilized most effectively."

Dr. Giampapa’s previous research has shown that addition of a DNA repair-promoting ingredient to a commercially available, broad-spectrum antioxidant formulation can enhance DNA repair in human cells, while inhibiting the inflammatory cytokines, IL-1 and IL-2 (Pero, Giampapa and Vojdani, J Anti-Aging Med 2002). DNA damage and inflammation, as well as oxidative stress and epigenetic DNA and histone modification, are hallmarks of cellular senescence, a biological process that is intimately linked to aging and cancer. Improving cell health through combating the pathways linked to cellular senescence is a primary focus of ongoing research at CellHealth Institute.

Autophagy: An Emerging Anti-Aging Mechanism
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Autophagy is a cytoplasmic catabolic process that protects the cell against stressful conditions. Damaged cellular components are funneled by autophagy into the lysosomes, where they are degraded and can be re-used as alternative building blocks for protein synthesis and cellular repair. In contrast, aging is the gradual failure over time of cellular repair mechanisms that leads to the accumulation of molecular and cellular damage and loss of function. The cell’s capacity for autophagic degradation also declines with age, and this in itself may contribute to the aging process. Studies in model organisms ranging from yeast to mice have shown that single-gene mutations can extend lifespan in an evolutionarily conserved fashion, and provide evidence that the aging process can be modulated. Interestingly, autophagy is induced in a seemingly beneficial manner by many

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of the same perturbations that extend lifespan, including mutations in key signaling pathways such as the insulin/IGF-1 and TOR pathways. Here, we review recent progress, primarily derived from genetic studies with model organisms, in understanding the role of autophagy in aging and age-related diseases.

**Aging and Autophagy**

Aging is a complex process characterized by the progressive failure of maintenance and repair pathways important for cellular homeostasis, which results in a gradual accumulation of aberrant macromolecules and organelles. The accumulation of such oxidized, misfolded, cross-linked, or aggregated molecules has deleterious effects on cellular homeostasis and on tissue and organ integrity. The defective molecules can disrupt homeostasis directly or by interfering with the activity of functional molecules and organelles, which leads to further dysfunction. This progressive decline in cellular integrity leads to aging, disease, and ultimately, to death. In many model organisms, the rate of aging can be modulated by altering conserved signaling pathways and processes, suggesting that the aging process itself may ultimately be amenable to therapeutic manipulation.

Autophagy is an evolutionarily conserved intracellular recycling process that is induced in response to stresses such as nutrient deprivation, hyperthermia, and hypoxia. Autophagy is responsible for the degradation of proteins, lipids, sugars, and nucleic acids as well as larger cellular components such as organelles. During the process, damaged cellular components are sequestered into a double-membrane structure called the autophagosome, which delivers its contents to the lysosome for subsequent degradation by acidic hydrolases.

**Conclusions**

Decades of research on the subject have revealed that the aging process is influenced by genetics and that many metabolic signaling genes can affect aging by mechanisms still to be fully elucidated. Importantly, many of these genes, including the nutrient sensor TOR and AMP-dependent kinase (AMPK), are emerging as important regulators of the process of autophagy. Evidence that autophagy influences the aging process has been observed in multiple model organisms, from yeast to multicellular organisms such as worms and flies, and a more recent and exciting finding is that autophagy is implicated in neurodegenerative diseases that affect humans.

![Autophagy Diagram](image)

Conserved longevity pathways and processes, which modulate aging via autophagy. For simplicity, the interactions between the pathways are not shown, whereas the degree of conservation is indicated. While the mechanisms by which autophagy affects aging through these longevity paradigms are not yet fully elucidated, several positive regulators of autophagy have been identified, including the forkhead transcription factor FOXO, the histone deacetylase SIRT1, the energy sensor AMPK, and the forkhead transcription factor PHA-4/FoxA.

**References**


**IN THE NEWS**

**Blood Forming Stem Cells Produced in Lab**

JUNE 14, 2013 – By transferring four genes into mouse fibroblast cells, researchers at the Icahn School of Medicine at Mount Sinai have produced cells that resemble hematopoietic stem cells, which produce millions of new blood cells in the human body every day. These findings provide a platform for future development of patient-specific stem/progenitor cells, and more differentiated blood products, for cell-replacement therapy.

The study, titled, “Induction of a Hemogenic Program in Mouse Fibroblasts,” was published online in *Cell Stem Cell*. Mount Sinai researchers screened a panel of 18 genetic factors for inducing blood-forming activity and identified a combination of four transcription factors, Gata2, Gfi1b, cFos, and Etv6 as sufficient to generate blood vessel precursor cells with the subsequent appearance of hematopoietic cells.

**Source:** Icahn School of Medicine at Mount Sinai

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**NEXT MONTH’S ISSUE**

In our Spotlight section, we focus on the connections between cancer and aging, and discuss how CellHealth Institute can treat both with our new paradigm on cellular aging. Here, we highlight the similarities between cellular senescence and cancer, and show how our products and research are preventing both by keeping cells healthy. The issue will also feature the topics of senescence, cancer and aging in our Featured Publication section, which details a recent 2013 review in *Cell* by Lopez-Otin, et al., titled “The Hallmarks of Aging” 153(6):1194-217.