CELL REGULATION

AMPK TO INCREASE LIFESPAN

VALERIE KIM

A recent study led by a team at the University of California-Los Angeles has discovered a gene that could significantly influence the process of aging in the human body.

Researchers Walker et al. investigated autophagy and established that the AMPK gene is responsible for the degradation of damaged and senescent cells.[1] 5′ AMP-activated protein kinase (AMPK) plays a role in regulating cellular energy by breaking down and recycling used organelles, which allows old, deteriorating cells to make room for new, healthy cells. By manipulating the genome sequence of *Drosophila melanogaster*, the team at UCLA confirmed speculations that elevated levels of AMPK delay aging by increasing autophagic activity.[2] The researchers were amazed to find that the effects of activating the gene were more complex than simply lengthening a fruit fly's lifespan. When the AMPK gene was activated in the nervous or digestive systems of the fruit flies, autophagy increased and aging slowed down in areas beyond the brain or intestines.[3] Various organs were revitalized and this provided anti-aging protection throughout the entire body.

Thus, the findings of this study recognize the potential of decelerating the process of aging, not only on the skin to prevent wrinkles, but also in the brain and heart to prevent diseases such as Parkinson's disease. Further research into the genetics behind aging may ultimately lead to a dramatic shift in our understanding of age-related illnesses and consequently uncover potential solutions to prolong improve the quality of life.

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BIOTECHNOLOGY

NEUROPROSTHETIC EXOSKELETONS

ASHLEY LAM

The stage is being set for a world without wheelchairs. On June 12, 2014, 29-year-old paraplegic Juliano Pinto telekinetically controlled a robotic exoskeleton to kick off the 2014 World Cup opening ceremonies. Neuroscientist Dr. Miguel Nicolelis, is behind this rising technology, having led the Walk Again Project to its first live international demonstration.[1]

In the Walk Again Project, paraplegic patients are fitted with wireless caps implanted with electrodes, which pick up brain waves through electroencephalography (EEG). The process begins with simple thought commands for walking or kicking a ball. The electric signals are then transmitted to a computer found on the back of the hydraulic-powered exoskeleton. This step requires the use of Brain Machine Interfaces (BMIs), which enable the computer to read the electrical signals generated by neurons, and instruct the exoskeleton how to move.[2,3]

Temperature, pressure, and speed sensors provide feedback for the brain, allowing the patient to maintain balance and posture. Vibrations are transmitted to the arms of a paraplegic, which mimic the tactile response that would have been felt by the patient's feet. In addition, the feedback allows for restored spatial awareness.[3]

These neuroprosthetic suits are powered by hydraulics, and are composed of various circuit boards.[4] Patients require several months of training before extended use is possible.

The development of robotic exoskeletons is undoubtedly a medical breakthrough that will greatly increase the quality of life for paraplegic individuals.

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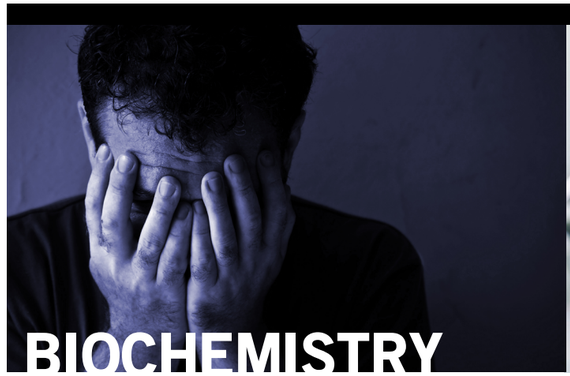

BIOCHEMISTRY

XENON TO TREAT PTSD

ANNA GOSHUA

Although xenon gas is typically utilized as an anesthetic and a tool in diagnostic imaging, a recent study published by researchers from Harvard's McLean Hospital indicates that it may possess the capacity to address post-traumatic stress disorder (PTSD), among other disorders related to emotional memory.[1] Certain events reminiscent of past trauma act as triggers for individuals afflicted with PTSD causing the brain to perceive the painful memory as if it were new. The administration of xenon may curtail these effects by inhibiting memory reconsolidation, or the remembrance and renewed impact of traumatic memories.[2]

Researchers utilized an animal model known as fear conditioning to simulate PTSD rats. Small doses of xenon gas were given to the rats upon activation of a fear response elicited by environmental stimuli. Results demonstrated a considerable decrease in these responses during tests conducted up to two weeks after treatment. However, negligible effects were observed if up to two hours elapsed between the reactivation of the fear memory and the dose of xenon. This suggests that xenon interferes in memory reconsolidation, likely due to its ability to block the brain's NDMA receptors, which are involved in memory formation.[3]

In contrast to other drugs bearing similar functions, xenon circulates through the brain rapidly, enabling an immediate response to fear memory reactivation. Another useful implication of this quality is that xenon gas only needs be administered for a brief duration. This, along with its currently established use in medicine, points toward a promising future for xenon's effectiveness in treating PTSD. For the millions suffering from this disorder, this convenient and quick "erasure" of traumatic memories would be nothing short of a wondrous relief.

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MS AND HIV

A POSSIBLE CORRELATION?

ABIRAMI KIRUBARAJAN

Multiple sclerosis (MS) is a crippling inflammatory disease that affects over 2.5 million people worldwide.[1] While it currently has no cure, a group of British, Danish and Australian researchers have discovered an unexpected connection between the HIV and MS diagnosis. Results demonstrate that HIV patients have a 40-60% less likelihood of being diagnosed with MS.

Scientists are calling this relationship "the largest protective effect of any factor yet observed in relation to the development of MS."[3] In the abundant clinical literature describing MS and HIV infection, only a single case study reported an individual with concurrent HIV and MS who was also receiving HIV antiretroviral therapies. This patient's MS symptoms (including loss of sensitivity, muscle spasms, and speech impediments) declined after HIV treatment. Researchers hypothesized that, because MS pathogenesis has previously been linked to endogenous retroviruses, there may be an interaction in the pathology and treatment of MS and HIV. In a comparative cohort study conducted by Dr. Julian Gold *et al.* the development of MS was found to be much rarer in HIV positive individuals, as compared to controls. Further scrutiny shows an 80% reduced MS diagnosis rate for HIV patients who underwent HIV treatment for over five years.[2] These results suggest a protective effect of HIV and HIV therapy against developing MS.

At the moment, the team is uncertain if the decline of MS symptoms is a result of HIV's immunodeficiency or its antiretroviral treatment; however, it is evident that the two conditions are related. Further research will focus on investigating which characteristics of HIV and HIV treatment provide protection, and can potentially lead to the approval of HIV drugs for MS use..[4]

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