Do the Stress Reactions of Our Brain Control the Immune System -- Or Is It the Other Way Around?

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Prof. Hermona Soreq, The Huffington post

In the well-known 17th-century text *Le Malade Imaginaire*, Molière wrote, "the mind has great influence over the body, and maladies have their origin there." Since Molière, we now know that a diseased body and malfunctioning immune system also affect the mind. Inflammation of the nervous system has been increasingly recognized as an important factor in multiple conditions, including Alzheimer's and Parkinson's disease. New technologies in genetic engineering and drug development are rapidly progressing to implement this knowledge for early diagnosis and creative treatment strategies that take into consideration both the brain and the body.

One area that has benefited considerably from these developments is the study of anxiety. Historically, human stress reactions enabled our ancestors to survive attacks and protect the body from injury. But even though these reactions aren't necessities like they were for our ancestors, the same response patterns have continued to the present. An unpleasant discussion at work rarely leads to physical attacks, but our body nevertheless prepares for such attacks; it elevates the blood pressure to prepare for running, and produces more white blood cells as protection from anticipated injury. While such reactions are useful for immediate protection -- since alertness can assist us in reacting to stressful experiences faster and more efficiently -- the consequences of stress responses may also entail long-term damages spanning muscle and nerve cells malfunctions, neurodegenerative diseases like Alzheimer's and Parkinson's, and inflammatory diseases.

The long-term and disease-provoking consequences of stress responses may take years to develop. This was not a concern for early humans, whose life span was shorter than ours (by the time stress could take its toll, they were no longer alive). But today, humans live much longer, and as a result, stress-associated diseases -- especially in the elderly -- have become a major social and financial burden.

Over the past two decades, I, along with my colleagues at the Edmond and Lily Safra Center for Brain Sciences (ELSC) at The Hebrew University of Jerusalem, have pioneered and developed innovative strategies for investigating the consequences of traumatic experiences and designed new strategies to combat neurodegenerative diseases. Our work has proven that in both brain neurons and blood cells, the genetic information that processes events are vulnerable to changes under stress. Moreover, we’ve discovered that both inherited and acquired defects in neurons, as well as traumatic experiences or exposure to a contaminated environment, contribute to delayed susceptibilities to stress-associated
diseases. The consequences may be seriously harmful, affect seemingly unrelated functions (e.g. learning
and memory, the day-and-night cycle, muscle fatigue, inflammation), and last over the life spans of
individuals and communities.

A good portion of our research has been devoted to a small chemical called acetylcholine, the first known
neurotransmitter (a small chemical compound which is capable of activating neurons to send electrical
signals) and the primary communicator between the body and the brain. Discovered 100 years ago by the
Nobel laureate Otto Loewi, acetylcholine comes from the brain through the vagus nerve and is responsible
for muscle twitching. We've discovered that acetylcholine signals produced in the brain affect psychological
stress responses, inflammation, aging, and the recovery from acute ischemic stroke.

As part of our research, we isolated the genes controlling acetylcholine degradation in humans and
identified stress-induced changes in their expression in brain neurons and blood cells alike. To pinpoint the
physiological role of these genes, we engineered mice with excess or deficient amounts of the genes'
protein products and watched their learning, muscle functioning and behavior. We learned three basic
things:

1) Either too much or too rapid degradation of acetylcholine may cause cognitive deterioration, which
intensifies anxiety reactions and can also intensify them, creating a vicious cycle;

2) The mice with excess breakdown of acetylcholine suffer constant inflammation, demonstrating the
power of brain-to-body communication for our immune system and showing that individuals who carry
inherited small changes in these genes are at increased risks for Parkinson's and Alzheimer's disease;

3) Different products of these genes may either enhance the progression of brain pathology in Alzheimer's
and Parkinson's disease or help protect against them, and they also determine one's prospects to survive
and recover from ischemic stroke. Measuring such changes by a simple blood test can hence predict one's
prospects for recovery and the risks for post-traumatic stress disorder.

New discoveries in stress-related pathologies are occurring at a quickening pace. Just recently, we've
developed a synthetic DNA-based drug for treating patients with inflammatory bowel disease. With the help
of a $5 million grant approved by the Leona M. and Harry B. Helmsley Charitable Trust, we hope that such
developments will bring us closer to finding more permanent treatments for neurodegenerative and other
diseases that have, unfortunately, become all too common.

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Our Int'l Ph.D. program provides outstanding students with top-notch courses in computational neuroscience.

The Jerusalem Brain Sciences Building will provide a state-of-the-art research and teaching facility for the Edmond and Lily Safra Center for Brain Sciences.

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