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Stress and Aging

excerpted from *The End of Stress As We Know It*
by Bruce McEwen with Elizabeth Norton Lasley

The major systems of the body work together to provide one of the human organism's most powerful and sophisticated defenses: the stress response, also known as the fight-or-flight response. The stress response helps us to react to an emergency and cope with change. To do so it musters the brain, glands, hormones, immune system, heart, blood, and lungs. Whether

we need to fight, stand firm, bolt to safety, or concentrate on a task at hand, the stress response provides the tools–energy, oxygen, muscle power, fuel, pain resistance, mental acuity, and a temporary bulwark against infection–all at a moment's notice.

Fight or flight is *allostasis* with a sense of urgency. Allostasis comes from the Greek root, *allo*, meaning variable, and it emphasizes the point that allostatic systems help keep the body stable by being themselves able to change. Most of us notice allostasis at times that we deem to be trying, and it usually seems to kick in when we don't want it to. Increasingly, the situations that ignite the stress response are ones for which neither fight nor flight is an option–working for an overbearing boss, for example, or caring for a family member who is seriously ill. In these situations, the stress response cannot help speed us toward a resolution. And so, deprived of its natural result, the very system designed to protect us begins to cause wear and tear instead, and illness sets in. This is the type of stress, or the state of being stressed out, that I prefer to describe as *allostatic load*– the damage that the allostatic response causes when it is functioning improperly.

Allostatic load does not always denote a failure of the body's efforts to cope with change or emergency. We can create it for ourselves by living in a way that makes for internal imbalance. Sleep deprivation, for example, leads to elevations in blood glucose and cortisol; chronic elevations of this sort can lead to bone mineral loss and increase the amount of fat that accumulates in the abdominal area (considered the most unhealthy type of fat). Eating a rich diet or overeating produces a metabolic load on the body, thereby increasing fat deposition and hardening of the arteries, both risk factors for heart disease and other ailments. Lack of regular exercise is another major contributor to allostatic load; regular exercise increases energy expenditure and boosts the muscles' ability to burn glucose. Exercise works against the bad effects of a rich diet and increased body fat, enhances well-being, and improves sleep.

Anticipation or anxiety can lead to allostatic load even when it doesn't drive us to make poor lifestyle choices. The human mind is so powerful, the connections between perception and physiological response so strong, that we can set off the fight-or-flight response by just imagining ourselves in a threatening situation. This ability can be a source of power or an invitation to illness.

Stress is no respecter of age, and, inevitably, we will confront it with an aging brain. It would be nice to say that a lifetime of experiences gives us an advantage, but, alas, the brain, as it ages, becomes more vulnerable to the effects of stress. Much of the supporting research for this gloomy news comes from animal studies. In young rats, for instance, repeated stress causes down-regulation, or a decrease, in glucocorticoid receptors, meaning that fewer receptors are produced in the brain cells. This is a built-in neutralizer by which high levels of cortisol actually become less effective, because the hormones find fewer receptors to lock into. However, with increasing age, the rats tend to lose this safety catch; the receptors don't down-regulate as efficiently, leaving more of them when there should be fewer, thus giving stress-generated cortisol a broader target area. This decrease in down-regulation is particularly apparent in older rats that also happen to be cognitively impaired. When aging rats undergo restraint stress (kept for brief periods in a clear cylinder that restricts their movement), glutamate levels in the hippocampus are elevated and prolonged, increasing the potential for damage and allostatic load.

Again, not all older individuals, rats or humans, have these problems. Those that do, however, show cognitive impairments that seem to be related to high cortisol levels. Sonya Lupien, who did post-doctoral work in my laboratory and is a faculty member at McGill University, has done some studies of basically healthy people over a number of years, checking both cortisol levels and mental acuity as measured by psychological tests of explicit memory and selective attention. In the most extensive of these studies, Dr. Lupien and colleagues followed 11 healthy elderly subjects over a period of four years. In six subjects, cortisol

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levels climbed over the four years and peaked in year 4. The subjects had trouble with memory tasks such as navigating a maze designed for humans and remembering lists of words. The other five, whose cortisol levels went down or stayed the same at the end of four years, marched through the maze and rattled off the lists of words with no problem. In a follow-up, MRI revealed that the subjects with higher cortisol and poorer memory also had hippocampi that were about 14 percent smaller than their more successfully aging counterparts.

Cortisol works together with excitatory neurotransmitters, chiefly glutamate, to produce stress-related learning as well as memory damage, but there is another consequence. One result of having excess excitatory neurotransmitters like glutamate is that destructive compounds known as free radicals begin to accumulate. Free radicals are considered to be the culprits in many diseases, including cancer and neurodegenerative diseases of the brain, as well as in the normal wear and tear associated with aging. The interaction of neurons with cortisol facilitates this process, since cortisol makes neurons more sensitive to glutamate and increases the number of NMDA (n-methyl-d-aspartate) receptors. Another important consequence of elevated excitatory neurotransmitters and elevated glucocorticoids in the aging animal is suppression of the formation of new brain cells, or neurogenesis.

To keep allostasis functioning on the protective end of the spectrum, the most effective steps you can take are the simplest: exercise, a healthy diet, regular sleep, moderate-to-minimal alcohol intake, and no smoking. If this sounds suspiciously like what your grandmother always told you, all I can say is that according to the most sophisticated, up-to-the-minute, cutting-edge science available, your grandmother was right.

**Dr. Bruce McEwen will present "Stress and Aging"
on November 1st at the YRS 2003 Conference**