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FOR STUDIES ON
HUMAN STRESS

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Genetics and Stress: Is There a Link?

By Robert-Paul Juster & Marie-France Marin

“My mother was always stressing out and so do I. There is nothing to be done, it's simply genetic!” You have no doubt heard some similar affirmation during a conversation with colleagues, your family, or your friends. Myth or reality? Several researchers are actually asking themselves the same question and attempting to answer it from many different perspectives.

Firstly, consider the fact that the human body contains an immense number of cells. Cells contain 46 **chromosomes** of which half come from your mother and the other half from your father. This means that each cell is composed of 23 pairs of chromosomes. Inside these chromosomes are **genes** which will influence numerous physical and psychological characteristics as well as countless behaviors and traits (for more details, see *Genetic Cooking Catalog*).

Where genetics are concerned, it is important to bear in mind that genes are not the sole determinants of our behaviors. It is true that for the majority of behaviors and characteristics, our genes and our environments play important roles. If you are lucky enough to grow up in a favorable environment, it is highly probable that some genetic predisposition to one disease or another might be negated. Conversely, if you are confronted by particularly stressful situations or traumas, your environment might be less favorable and your genetic vulnerabilities might become expressed.

For example, if you have experienced the death of a loved one and you have a genetic predisposition towards depression, the probability of succumbing to depression is greater in comparison to someone who has experienced the same situation, but for whom a genetic risk of depression is minimal. Furthermore, the risk of depression is also greater when compared to someone with a genetic predisposition but who has not been exposed to this situation. This ultimately means that most of the

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Genetics and Stress: Is there a Link?

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With that said, let's turn over to our favorite subject shall we? Stress! Is stress free of genetic influences? Probably not. Remember that situations that induce stress responses must be first and foremost deemed as stressful: that is, the situation must consist of at least one of four stressful ingredients that include Novelty, Unpredictability, Threat to the ego, and/or diminished Sense of control (NUTS being the acronym of this recipe). Once the brain has interpreted the situation as stressful, a stress response commences the release of stress hormones. In this manner, genetics can have an influence on normal day-to-day levels of the stress hormone cortisol, on the reactivity to stressors, and even in the way that we perceive the world.

Genetic Cooking Catalog

One way to remember genetics lingo is to imagine genes as a cellular recipe collection. Let us say that both your parents were chefs working together as were their parents before them. They pass onto you an Encyclopedia containing 23 volumes. These 23 volumes are actually the combination of their versions for the same recipes that got combined throughout generations (chromosomes). Within each volume are specific chapters of recipes (genes). The ingredients (alleles) for approximately all twenty-five thousand of these recipes (the estimated number of genes in humans) provide you with everything you need to cook. Every living cell – about 100 trillion for humans – contains this entire cooking Encyclopedia that permits the cell to flip through its inherited catalogue in order to whip up any recipe it needs to survive.

Intuitively, it makes sense to assume that stress is linked to genetics since genes influence how we perceive things and our sensitivity to stressful situations. Could it be that the reverse might also be the case? Does stress have the capacity to influence our genetic makeup, a biological constitution long considered unchangeable? Believe it or not, genes are not as untouchable as we previously thought, since stress can have an impact on them. The interaction between stress and genetics is, as you can imagine, very complex. In our 9th Mammoth Magazine issue, we aim to initiate you into this fascinating field of research.

Believe it or not, genes are not as untouchable as we previously thought, since stress can have an impact on them. The interaction between stress and genetics is, as you can imagine, very complex.

To begin, Shireen Sindi, Ph.D. student at McGill University, paints us a portrait of Dr. Michael Meaney, researcher at the Douglas Mental Health University Institute. Dr. Meaney is a world-renowned researcher known for his work on the interactions among stress and genetics. He has most notably pioneered studies using animal models to answer complex research questions. For example, how does maternal care influence the stress response of a young rat? Or maybe how might exposure to adverse environments during early life influence our reactivity to stress in adulthood? Is it possible to reverse the effects of an adverse environment on our stress response? Here are but a few research questions that Dr. Meaney and his colleagues are posing and investigating. In addition to animal research, he is also working to understand how his fascinating findings translate for humans.

Next, Dr. Isabelle Ouellet-Morin, presently in post-doctoral formation at the *MRC Social Genetic Developmental Psychiatry* in London, will describe what twin studies can teach us about the genetic bases of stress reactivity. It is important to realize that twin studies are an excellent model for studying genetics. In effect, **monozygotic twins** (also called identical twins) share 100% of the same genes, while **dizygotic twins** (also called fraternal twins) share 50% of the same genes. By comparing identical twins to fraternal twins while also considering mutually shared environments and those not mutually shared, researchers are able to tease apart the estimated importance of genetics in explaining, certain behaviors or physiological responses. The stress response is a good example. Dr. Ouellet-Morin very nicely reports on



the state of knowledge in her article based on numerous findings, many of which were discovered during her doctoral research.

The third article of this Mammoth Magazine issue is written by Nadine Provençal, Doctoral student at McGill University. Nadine Provençal explains the fascinating world of **epigenetics**. As mentioned earlier and in stark contrast to previously held knowledge, it appears that genes can be modified. Epigenetics addresses the interaction between environment and genes and Nadine Provençal's article focuses on how stress can influence the expression of our genes. The good news with epigenetics is that nothing is carved in stone!

To conclude, Dr. Alexandra Fiocco, currently completing post-doctoral training at the *BayCrest Centre* in Toronto, will explore a relatively new subject in genetics called **telomeres**. These are kind of like protectors of our genetic code. The length of telomeres appears to vary from individual to individual, most notably in relation to stress, aging, and the development of certain health problems. Dr. Fiocco will highlight the fascinating nuances among these phenomena and will even provide us with advice on how to augment our chances of successful, healthy aging!

We hope that you will find reading the 9th Mammoth Magazine issue interesting and will appreciate the field of research on stress and genetics. Enjoy! 🐘

A researcher's profile:

Dr. Michael J. Meaney

Maternal care and genetics : effects on stress reactivity



By **Shireen Sindi, Ph.D.**, Candidate, Centre for Studies on Human Stress, Fernand-Seguin Research Centre, Louis-H. Lafontaine Hospital, Montreal

Dr. Michael J. Meaney is a professor in the Faculty of Medicine at McGill University and based at the Douglas Mental Health University Institute. Dr. Meaney and his research team are interested in the effects of early experiences on later development and gene expression. His work has focused on maternal care and how it alters gene expression, which then has an important influence on the capacity to respond to stress. In rats, maternal care can be determined by counting the frequency at which mothers lick and groom their offspring. Whereas some mother rats tend to lick and groom frequently, others only do so rarely.

Interestingly, when Dr. Meaney and his colleagues compared these different groups of mother rats, they found that different levels of maternal care changed the functioning of the genes involved in responding to stress in the pups. Essentially, some genes were turned off, while others were turned on. Consequently, rat pups who had received high levels of maternal care (frequent licking and grooming) responded to stress

by releasing smaller quantities of stress hormones in comparison to rat pups who had received low levels of maternal care (infrequent licking and grooming). Pups who had received higher levels of maternal care also demonstrated enhanced learning and memory on certain tests. These differences were carried through to adulthood, demonstrating the long-term effects of maternal care.

When stress hormones are produced in high quantities over extended periods of time, they have a negative impact and may be associated with a variety of physical and/or mental health outcomes. Stress hormones also have an impact on brain functioning, and especially on the brain region involved in learning and memory, known as the hippocampus. While this is true, the release of stress hormones is adaptive when responding to stress, as it allows the body to face the stressor and cope with it. Small increases in stress hormones also enhance memory functioning.


Rats born to low-licking mothers show reduced performance on certain memory tasks, as their genes related to memory do not function optimally. Yet, when these rats are raised by high-licking and grooming mothers or when placed in enriched environments until early adulthood, these same genes are turned on.

When rats are raised by low-licking mothers they respond to acute stress with larger increases in the stress hormone corticosterone, and this response is adaptive. The rat pup is thus “programmed” to interpret the environment as adverse and stressful, and therefore needing larger stress responses. On the other hand, other studies by Dr. Meaney's team have shown that rats born to low-licking mothers show reduced performance on certain memory tasks, as their genes related to memory do not function optimally. Yet, when these rats are raised by high-licking and grooming mothers or when placed in enriched environments until early adulthood, these same genes are turned on. In the same way, female rats born to low-licking mothers, who are then cared for by high-licking mothers, later become high-licking mothers themselves. These results shed light on the importance of the rearing environment and parental care in early life, which may protect development regardless of present risk factors.

Rat pups who had received high levels of maternal care (frequent licking and grooming) responded to stress by releasing smaller quantities of stress hormones in comparison to rat pups who had received low levels of maternal care (infrequent licking and grooming).

In order to assess whether studies on humans will support some of the evidence found in rats, Dr. Meaney and his collaborators are currently conducting a research project titled Maternal Adversity, Vulnerability and Neurodevelopment (MAVAN). The project aims to assess whether maternal care can have an impact on genetics and development. Researchers involved are examining how mothers' interactions with their children may influence their children's responses to stress. Cognitive and memory capacities are also measured, since stress is known to have an impact on brain

functioning and memory. The project will also look at how mothers' mental health conditions such as depression may prevent adequate bonding with their infants

Such research studies are very interesting as they demonstrate that genetics do not influence development of youths on their own, but instead in interaction with the environment. Moreover, gene expression can be modified according to the manner in which parents raise us and the quality and nurturance of our familial environment. 



Same, not the same!

How twin studies can help us better understand stress in humans

By Isabelle Ouellet-Morin, Ph.D.,
Post-doctoral trainee, *MRC Social Genetic
Developmental Psychiatry*, London
Translation: Robert-Paul Juster

It's 6:30 PM on Tuesday. Annie, age 3, and Laura, age 7, are accompanying their mother to the grocery store. Annie is crying while Laura has no idea what to do to help her younger sister. Marie, who is Annie and Laura's mother, ruminates: "Only negligent mothers arrive late to kindergarten three days in a row... and now what do I make for dinner?" Suddenly Marie reminisces about her mother when she was growing up and remembers the soupcon of anxiety felt at dinner time. Marie also recalls of her older sister who isn't doing well at the moment; "She resembles our mother more and more", she thinks. A cry interrupts Marie's thoughts. Annie is inconsolable despite Laura's attempts to calm her. Annie resembles her so much; she too had the same temperament at that age! "Apples never fall too far from the orchard", she says when reaching the cashier.

Marie's thought process resumes well the popular belief that kids replicate their parents, but why? Could it be due to the fact that they share some of the same genetic makeup or because they grow up in a similar family and are exposed to similar situations over the course of development, both beneficial (ex: love, security) and detrimental (ex: negligence, abuse)? In the same way, why do people respond differently when confronted with stressful situations? Are these differences encrypted in their genes differently or are they shaped by the environment?

Well, like so many things in life, nothing is black or white. Researchers, like the rest of the population, are challenged by this ambiguous question: what role do genetic factors and environmental factors have on stress reactivity, given the fact that they are intimately connected? The answer to this question obviously holds immense clinical implications of potential benefit to everyone. Knowing that prolonged exposure to stress hormones like cortisol is associated with

an increased risk of suffering from mental health problems (ex: depression) is crucial to better understanding the factors that render individuals more vulnerable to stress throughout the lifespan in order to prevent long-term consequences.

Twin studies: a unique tool to decipher the genetic and environmental contributions of stress reactivity

The relative contribution of genetic and environmental factors on stress reactivity can be estimated by observing identical twins. These twins are perfect human clones. They not only share the same genetic baggage, but they grow up in the same family. Observed differences between identical twins reflects exposure to unique environments (different experiences, such as belonging to different classes at school). The similarity of identical twins is then contrasted to the observed similarities among fraternal twins so that we can distinguish the contributions of genetic factors from shared environmental factors (similar experiences



such as household income). You'll recall that fraternal twins are essentially brothers and sisters (about 50% of genes are shared in common) with the exception that they are born at the same time and so are exposed to similar environments in comparison to normal brothers and sisters sometimes born several years apart.

Few twin studies have estimated the contributions of genetic and environmental factors on stress reactivity, as measured using concentrations of the stress hormone cortisol, as children encounter stressful situations. One study that we conducted using 19 month-old Quebecois twins showed that identical and fraternal twins facing adverse family conditions had similar levels of stress hormones upon confronting a new situation. These results highlight how important shared environments are and also suggest that genetic factors have a negligible effect on stress reactivity for children living in adverse familial conditions. In this study, adverse familial contexts included several indices, such as maternal smoking during pregnancy, a low household income, and young maternal age at birth of children (less than 20 years old). The relative contributions of genetic and environmental factors do not, however, generalize the entire reality of all the participants. In effect, the identical twins that grew up in contexts without adversity had similar stress hormone levels to fraternal twins when exposed to a stressful situation, suggesting that genetic factors are at play here.

This study is important because it addresses the initial profile of genetic and environmental influences at an age when several brain structures implicated in stress regulation undergo development (ex: hippocampus, amygdala, and frontal cortex). Since these brain regions participate in learning, memory, and the regulation of emotions, an atypical stress hormone profile could compromise an individual's capacity to manage stress and diminish their resilience to other stressful situations (that is to say, the capacity to function adequately in spite of adversity).

The case of identical twins discordant in their exposure to adversity

Considering that it is ethically unthinkable to deliberately submit children to adverse conditions, scientists are limited when it comes to testing the possible causal relations between exposure to difficult circumstances and atypical patterns of stress reactivity. Looking at identical twins that are discordant helps clarify this question. In



Exposure to environmental adversity in early life affects stress reactivity and that it is important to intervene early in order to give kids the necessary tools to learn to effectively cope with stress.

collaboration with a British team, we observed distinct patterns of stress hormone levels in response to a psychological stressor in 12 year-old identical twins discordant with regards to the level of victimization experienced. So, one twin from the pair was victimized, while the other was not. While stress hormones rose when facing a stressful situation in the non-victimized twins (expected response), no rise was detected for victimized twins (remember that under stressful circumstances, a rise in stress hormone levels is normal and adaptive for the organism). Since the twins are genetically identical and grow up in the same family, this difference cannot be explained by genetic factors nor by shared environment effects alone. Moreover, these twins were similar in terms of their intelligence quotients and externalizing/internalizing behavioral problems, so the differences cannot be explained by these variables. The results of this study represents a big chunk of evidence that might convince the scientific community and governmental bodies that exposure to environmental adversity in early life affects stress reactivity and that it is important to intervene early in order to give kids the necessary tools to learn to effectively cope with stress. The *DeStress for Success Program* is a good example of this thinking (see Vol. 5 of *Mammoth Magazine*).

One methodological estimate among others

Twin studies allow us to piece together genetic and environmental factors and to then determine if these change as a function of the given environment (ex: adversity). Yet, many more methods of estimation exist. Some researchers study these questions using animal models to draw parallels with human functioning. Others opt for estimate associations using candidate genes (or environments) specifically related to stress reactivity. These are ideally guided by knowledge derived from twin studies. For example, the results of the first study suggest that the association between candidate genes and stress reactivity are more likely to emerge in children who were not exposed to familial adversity in the course of early life. It is by employing various methodologies that researchers will better understand the functioning of physiological systems and psychological traits implicated in stress reactivity as well as prevent more effectively the emergence of mental health problems. 🧠

Identical twins that grew up in contexts without adversity had similar stress hormone levels to fraternal twins when exposed to a stressful situation, suggesting that genetic factors are at play here.

Beyond genes: Epigenetics

Can stress modify our genes?

By Nadine Provençal, Ph.D. candidate,
McGill University, Montreal

Translation: Robert-Paul Juster

Since the time of Darwin and Lamarck, the question of what is innate or acquired is constantly being debated. Are our genes at birth the sole determinant of our development (innate) or is it experiences throughout life (acquired)? The answer is probably somewhere in the middle.

We have long believed that our genes and genetic code were inalterable and that our environment had no effect on us. However, scientific advances have now demonstrated that the genes that we are born with are not immutable. In fact, our genes and environment are interconnected thanks to epigenetics. The term epigenetics literally means beyond genes; it essentially represents a code put on top of an existing genetic code. It is important to know that all our cells have the same genetic code, and yet, cells that make up, say, our liver, have a very different function than those that make up the neurons of our brain. How then do we explain these differences from cells possessing the same genes? It is because of epigenetic control that dictates to genes which organs to become and when to be activated. It is in a way a second genetic code. In the first place, we have DNA that determines the genetic code. In the second place, epigenetics regulates the expression of our genes. Thanks to epigenetics, it is therefore possible that our environment modifies the expression of our genes, as if genes were controlled by a series of interrupters. The food we eat, the air that we breathe, or even the cuddles and snuggles we receive can activate these interrupters of genetic expression. We all know that certain foods can render us more vulnerable to certain cancers, but today, we also know that epigenetics is the mechanism whereby our genes are activated or deactivated by different ingested foods.

Thanks to epigenetics, it is therefore possible that our environment modifies the expression of our genes, as if genes were controlled by a series of interrupters. The food we eat, the air that we breathe, or even the cuddles and snuggles we receive can activate these interrupters of genetic expression.

Can epigenetics alter our stress response?

A group of researchers from McGill University have found the answer to this question in rats.

They discovered that maternal attention has the ability to modify activity of a gene in the hippocampus (a part of the brain principally implicated in learning and memory) of newborns. Dams that licked and groomed their pups more often were in fact protecting their progeny against stress by modifying the epigenetic code of the NR3C1 gene of their little ones. This gene translates into a protein that contributes to diminishing stress hormone concentrations. A mother's attention transforms into a precise epigenetic activation of the NR3C1 genes. On the flip side, pups that have not received enough attention from their mothers live in a constant state of distress. Maternal care is therefore able to affect genes and modify the stress response in adulthood. In this way, epigenetics serves as an interface between our environment and our genes that can ultimately influence our stress responsivity.

Can stress cause epigenetic modifications?

According to an article published in *Nature Neuroscience*, stress throughout life provokes epigenetic modifications that influences future reactions

and behaviors. In effect, German researchers stressed out newborn mice by separating them from their mother for 3 hours a day over 10 days – what represents a relatively mild stressor. The mice exposed to this stressful treatment dealt with future stressful situations with greater difficulty and had poorer memory than non-treated mice. These researchers discovered that stress exerted a permanent epigenetic mark on a gene that codes a protein involved in stress; namely, vasopressin. This hormone activates the stress response and plays an important role in social behaviors. The gene in question is programmed to produce more vasopressin, rendering the mice more vulnerable to stressful environments. This means that stress, by epigenetic means, can modify behavior.

And so what about humans?

Remember that these studies were conducted in animals. Can the human genome also be modified by the environment? In humans, access to organs like the brain is not as straight-forward as with laboratory animals. It is only possible to use brain of deceased individuals that have been conserved voluntarily for research purposes. A recent study demonstrated that the same epigenetic markers are observable in humans. As was the case for rats neglected by their mothers, the NR3C1 genetic code is modified in the hippocampus of human suicide victims who were abused during childhood. These results lead us to believe that stress experienced throughout childhood has the potential to alter our epigenome and consequently modify behaviors in adulthood.

It is also possible to study in humans that are alive the effects of environments on genes by looking at blood-borne epigenetic markers. In effect, a




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recent study demonstrated that the prenatal environment can alter our epigenome. Researchers analyzed DNA in blood of adolescents who had mothers that smoked during pregnancy. Interestingly, adolescents born to mothers who smoked during pregnancy had an epigenetic mark on their blood cells at the level of the gene implicated in brain development called the BDNF gene. These results suggest that prenatal exposure to cigarettes could impact development of the brain by epigenetic means. To date, we do not understand the bottom-line impact of these modifications; however, many believe that they contribute to an increased vulnerability of developing certain mental disorders.

Once programmed, can the epigenome be reprogrammed?

In contrast to irreversible genetic mutations, epigenetic marks can be modified. It has been shown that certain medications can repair epigenetic marks left by stress. In fact, these substances have the power to alleviate epigenetic marks on DNA and in so doing reverse effects on behavior. Even though pharmacology is useful, it is important to note that a simple change in environment can have the same results. For instance, by intrusting a baby rat born from an unaffectionate mother into the hands of a caring foster mother, the pup ends up developing normally without a hypersensitivity to stress...as if destiny was not totally sealed into DNA.

These results represent a very precious contribution towards understanding the long-term effects of stress. The recent emergence of these scientific findings indicates that adversity, like abuse and negligence during early life, can increase the risk of developing certain psychiatric problems like depression. Certain epigenetic mechanisms, such as those described in this article, can contribute to explaining this phenomenon. According to animal studies, epigenetics give us hope since it is possible to reverse the effects of stress with the help of medications or by modifying the environment... let's see where this will bring us humans! 

Does stress make our cells age faster?

Telomeres and telomerase

By **Alexandra Fiocco, Ph.D.**,
Post-doctoral trainee, Baycrest Centre, Toronto

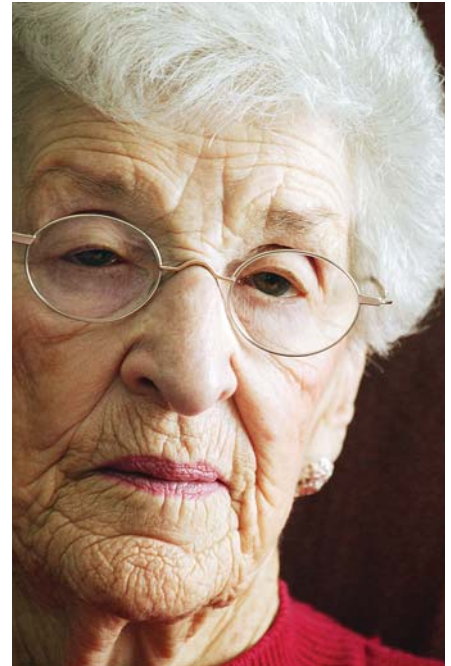
Have you ever wondered what the little plastic endings on your shoelaces are called? Anglets! Anglets are an essential backbone for our shoelace as it prevents the shoelace from unraveling and losing its functional form.

While this may seem to be a rather peculiar way to begin a piece on genetics, the anglet is the best way to describe what a telomere is. **Telomeres** are protein sequences found at the end of chromosomes that are currently very fashionable in genetics research. Although they are not genes themselves, they protect the chromosome from unraveling or binding randomly with other DNA in the cell.

In the early 1970's, scientists discovered that these chromosome "buffers" are depleted with every cycle of **mitosis**, a process where cells copy themselves. With each cell division, a piece of the original telomere sequence is lost. If not replaced, the shorter the telomere becomes, the less protection it gives to the chromosome. This ultimately leads to cell **senescence**, a fancy word for aging cells. As the cells in the body diminish in their ability to reproduce, so does the body's ability to repair against internal and external assaults that may arise.

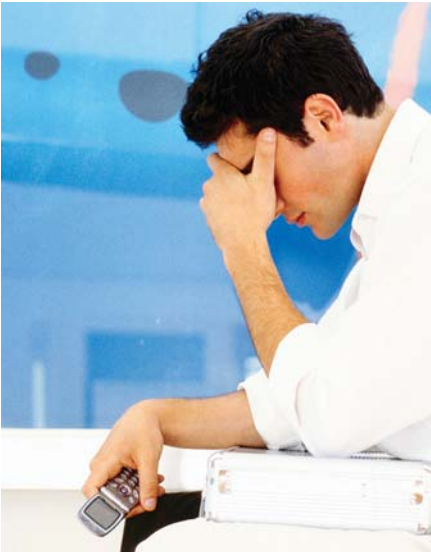
Luckily, all mammals have a built-in mechanism that regenerates telomeres and prevents the untimely death of cells over time. This protective enzyme, which is called **telomerase**, inhibits the shortening of telomeres and allows cells to divide infinitely. In fact, some might even argue that telomerase is the true fountain of youth!

Now, you may ask; why do cells die if telomerase prevents telomeres from shortening? If telomerase protects cells from aging, would it not allow for infinite cell replications and longer life spans? While the answer is not simple, evolutionary studies suggest that telomere length and telomerase maintenance are indeed associated with longevity! More striking is that there are pronounced differences in telomere length and telomerase activity from one individual to another. A big determining factor in these individual differences in telomeres and aging appears to be stress.



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Recall from Volume 8 of our Mammoth Magazine that social standing influences health outcomes such as cardiovascular, respiratory, and psychiatric diseases. Extending upon this, scientists have shown that social economic status (SES) may affect telomere length. A group of researchers from the United Kingdom evaluated 1552 female twins, aged 18 to 75, and reported that women from low SES displayed shorter telomere length compared to women from high SES. This association was found independent of body size, smoking status, and physical activity, which are factors found to be associated with both SES and telomere length. The authors were further able to disentangle the effects of genetic variation by assessing twins discordant for SES (i.e. twins who lived together until the age of 16 and then separated and diverged on SES status). They found that siblings from low SES exhibited shorter telomere length compared to their discordant high SES twin.



Although there is no direct scientific evidence linking low SES to increased perceived stress, it is possible that low SES individuals are more vulnerable to the effects of an adverse environment. Indeed, when compared to high SES individuals, low SES individuals experience limited amounts of resources available to deal with the stressors in question.

Other researchers have shown how chronic stress can literally “get under the skin”. In a study that assessed healthy pre-menopausal women, perceived chronic stress was associated with shorter telomere length and lower telomerase activity. In-

in women with short telomere length compared to long telomere length. The authors also reported that decreasing telomere length was associated with decreasing hippocampal volume, a brain region important in memory and learning. Interestingly, Sonia Lupien and her colleagues showed that smaller hippocampal volume is associated with increased stress hormone production in older adults.

Telomere length is accelerated by oxidative stress and inflammation, two biological processes that are promoted by psychological stress and malfunctioning stress hormones. Given the relationship between telomere length, stress, and age-related disorders, researchers have started to examine prevention strategies that decrease stress in order to maintain telomere length. It has been suggested that techniques that decrease stress may also slow the rate of cellular aging.


Moreover, an interaction between chronic stress and physical activity on telomere length has been reported. Specifically, increased chronic stress was associated with shorter telomere length in non-physically active individuals. On the other end, this association was non-existent among the physically active group. The authors concluded that exercise may actually protect individuals from telomere shortening by serving as a buffer against the harmful effects of chronic stress. Further, shorter telomere length is related to smoking and overeating, two behaviors affected by chronic stress. Finally, another group of researchers are beginning to ask the question of whether mind-

The shortening of telomere length is not only associated with premature death, but also associated with disorders that generally occur in older age, such as cancer, osteoporosis, cardiovascular disease, diabetes, and risk of dementia. All of these age-related disorders have also been associated with chronic stress.

terestingly, women with higher levels of reported chronic stress exhibited telomere lengths that are equal to an additional decade of aging compared to women who report lower levels of chronic stress! Ultimately this means that perceived stress may accelerate the aging process, and in turn, promote disease.

The shortening of telomere length is not only associated with premature death, but also associated with disorders that generally occur in older age, such as cancer, osteoporosis, cardiovascular disease, diabetes, and risk of dementia. All of these age-related disorders have also been associated with chronic stress. A study that followed 2734 non-demented older adults between the ages of 70 and 79 years over time reported that short telomere length at baseline was associated with greater decline in global cognitive function over seven years. Similarly, it was found that the odds of developing **mild cognitive impairment** were 12-fold higher

fulness meditation may have beneficial effects on telomere length by reducing psychological stress and enhancing hormone levels that may support telomere maintenance.

Many people in today’s society are experiencing the negative impact of chronic stress. Research now suggests that stress-related outcomes may be determined by cell senescence, or rather, the shortening of telomere length over time. Understanding the link between stress, cellular aging, and health is important to help devise strategies for improving or maintaining quality of life as we age. Overall, it appears that engaging in healthy lifestyle behaviors, including not smoking, eating healthy, and exercising, may maintain the anglets at the end of our chromosomal shoelaces and thus slow cellular aging. This may in turn help prevent age-related diseases commonly associated with chronic stress and therefore keep us walking around longer. 



Next issue

Several among you are caregivers for parents with physical or mental health problems. This act of generosity and immense devotion enriches the lives of those afflicted. Unfortunately, this care can also incur a heavy cost for the caregiver. Hopelessness, stigmatization, and distress are very common and can be damaging for the caregiver’s health and well-being.

In our next issue, we shall explore this sensitive topic in the aims of informing readers of this reality and to empower them with strategies to better adapt to stress.



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