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Worrying Ourselves Sick: Biological, Psychological, and Social Components to Stress-Related Disease

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**WORRYING OURSELVES SICK: BIOLOGICAL, PSYCHOLOGICAL, AND
SOCIAL COMPONENTS TO STRESS-RELATED DISEASE**

**A thesis submitted to
Regis College
The Honors Program
in partial fulfillment of the requirements
for Graduation with Honors**

**by
Shannon Quirk
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I. INTRODUCTION

“We’ve entered the gilded genomics era just in time to have to admit that most of our ills have to do with extraordinary un-genomic things like...psychological makeup... social status and the society in which you [live], your lifestyle. And at the center of this nexus is stress—what stressors we are exposed to and how we cope.”

~ Robert Sapolsky (Qtd. in McEwen, 2002)

On a sunny morning in March, my mom, dad, and little sister decided to go snowshoeing with Fritz, our German Shepherd dog, just outside the mountain town of Frisco, Colorado. While alone on the trail, they suddenly spotted a full-grown female moose about fifty yards away. At that instant, the moose started to run towards my family and Fritz until it was only five feet away. Whereas my mom and little sister immediately ran and hid in a tree well, my dad’s protective instincts kicked in as he continued to hold onto Fritz, who stood up on his back two legs and barked at the moose. Just when my dad was about to let go of Fritz and run away, the moose thankfully ended up retreating back into the forest without harming my family. In this example of *acute stress*, a functioning fight-or-flight response allowed my family and dog to think clearly and react quickly in the presence of a charging moose.

Let us now compare this story to a case of *chronic stress*. During the summer of 2011, I had the amazing opportunity to spend one month living in La Vereda, a rural

farming community located within the verdant mountains of the Dominican Republic. Our group of health professionals and students set out to provide easily-accessible medical and dental clinics and live with host families. I will always remember talking with Gisela, a mother of three children who lived in one of the poorest houses in the community. Even though her family lacked basic amenities like dependable electricity, an umbrella, an indoor bathroom, and a means for transportation, Gisela was always so spirited and appreciative of her simple lifestyle. Yet, a constant stressor in her life involved her children's lack of opportunities. Despite the fact that her 11-year-old daughter, Nena, was incredibly intelligent, mature, and driven, Gisela was constantly anxious that Nena would never have the opportunity to accomplish her dream of becoming a doctor. Sadly enough, Gisela was the only patient out of over 300 to ask us for anxiety medication.

Clearly, stress can materialize in markedly different circumstances—ranging anywhere from physical threats to emotional stimuli. But what exactly is stress? In an attempt to unify the wide range of multidisciplinary definitions of the term, Walsh et al. (2011) describe stress as a constellation of events, consisting of a stimulus (or stressor), that precipitates a reaction in the brain (called stress perception), that activates physiological fight-or-flight systems in the body (or the stress response). As opposed to other vertebrates where the stressor is almost always physical, the nature of the stimulus can vary widely in humans. It can be positive, like excitement about a first date. But it can also be something negative, such as a life-threatening situation or an intense emotional burden.

The important point to note here is that Gisela experienced a similar (but not identical) stress perception and stress response as my family and dog did during their moose encounter. The same cascade of neurotransmitters and hormones raced through her body, giving rise to the same general physiological changes. Some of these alterations include elevated levels of blood glucose, an increased heart rate, faster breathing, enhanced cognitive ability, and inhibition of expensive long-term projects in the body (such as reproduction, digestion, and growth). Although these particular physiological adaptations are essential for surviving life-or-death situations, we will soon see that a protracted activation of the stress response can really wreak havoc on our bodies, both psychologically and physically.

One of the primary differences between the stress responses in the two examples above is that acute stress only lasts for a matter of minutes to hours, whereas chronic stress lasts anywhere from weeks to years. As expressed in *Why zebras don't get ulcers: The acclaimed guide to stress, stress-related diseases, and coping*, by Robert Sapolsky (2004), for both a zebra sprinting for its life and a hungry lion pursuing its prey in the savanna, the fight-or-flight response is quintessential for dealing with short-term physical challenges. Without it, the zebra and lion would not last very long in the wild and we could never react quickly enough if, for instance, a driver in front of us suddenly slammed on her breaks. Furthermore, the stress response does a fairly good job of protecting organisms during chronic physical challenges, such as drought and/or famine.

Even though the stress response clearly plays an instrumental role in life, its intensity and duration are important factors to consider. As previously mentioned, a major paradox in this field involves the fact that too much stress can ultimately render the stress response inefficient or deleterious. Take salmon, for example. In a process called smoltification, salmon are born in fresh water, swim downriver to the ocean as adults, and return to fresh water to mate and die (Björnsson, Stefansson, & McCormick, 2011). During the nine-month journey upstream, stress hormone levels skyrocket, providing crucial energy for the struggling salmon (McEwen, 2002). Interestingly enough, these very hormones ultimately kill the adult salmon (usually after they mate). This leaves enough nutrients for the newborns to thrive in the fresh water pools, and eventually start the whole cycle anew. Because persistent stress hormones make it possible for the adults to complete the difficult journey upstream, but also kill them, this is an extreme example of the stress response acting as a “double-edged sword.” Evidently, the vertebrate stress response does not function perfectly when it is overused.

But why should we care about salmon stress levels? One reason is that humans are among the most susceptible organisms to stress-related health problems. Not only do we experience physical stressors, but our species is also one of the few to experience sustained *psychological and social stress*. As stated by Sapolsky (2005), “for 99 percent of the beasts on this planet, stress is about the three minutes of screaming terror as you sprint for your life on the savanna, after which it’s either over with or you’re over with.” Whereas wildebeests do not have thirty-year sprints from loins, we can be chronically stressed with thirty-year mortgages or a plethora of other concerns that do not typically

impair our immediate survival—such as our relationships with others, our grades in school, our financial status, and our pursuit of a meaningful life. In other words, we do not always use the stress response as it was intended.

In fact, anxiety about our fast-paced, demanding world has been around for centuries. Consider the following quote: “But the present world is a different one. Grief, calamity, and evil cause bitterness...Evil influences strike from early morning until late at night...they injure the mind and reduce its intelligence and they also injure the muscles and the flesh” (Qtd. in Benson, 2000). Although this may sound like a contemporary observation, the author was actually a Chinese physician who practiced 4,600 years ago. Many of us are also familiar with the ancient Greeks’ story of Sisyphus, a man who eternally pushed a boulder up a mountain only to have gravity bring it back down every time. Perhaps humans are programmed to view the world as a place where we must push our own boulders up hills over and over again. As we will see in the following pages, this kind of a mindset can ultimately give rise to fatigue, distress, and eventually ill health.

Yet, stress-related disease is a recent occurrence, especially within societies that are rich enough to have sufficiently dealt with malnutrition, poor sanitation, and inaccessible medical care. Instead of prematurely dying from childbirth and infectious diseases (e.g., tuberculosis, diphtheria, and pneumonia), disease patterns are markedly different today, as demonstrated by [Figure 1](#). Innovations in medicine and technology over the past hundred years have generally allowed us to live longer (to about 78 years in the United States). In fact, life expectancy increased by a drastic 56% during the 20th

century (Guyer, Fredman, Strobin, & Sondik, 2000). This means that we are now prone to acquiring degenerative maladies that accumulate over a lifetime. For instance, heart disease, cancer, and stroke account for the most deaths in the United States and in much of the world.

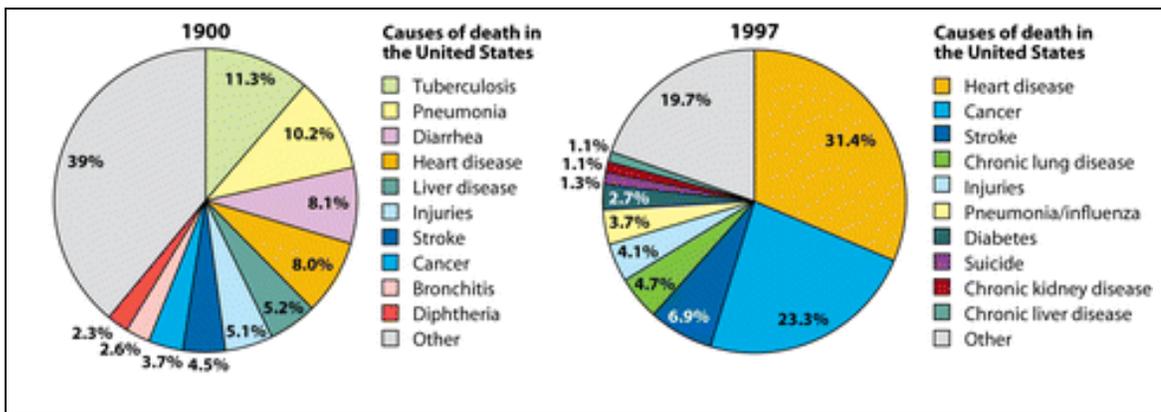


Figure 1: Causes of death in 1900 vs. 1997 (Smith & Guégan, 2010)

Stress plays a big part in worsening the degenerative diseases (especially heart disease and stroke) that fill up much of the 1997 pie chart in Figure 1. In fact, the economic toll of stress-related illness in the United States has been estimated to exceed \$200 billion annually (McEwen, 2002). In order to prevent this number from rising in the future, both clinicians and patients must focus more on preventing stress-related conditions before they ever arise. Clearly, this un-ignorable issue deserves our attention now more than ever.

This thesis has three main objectives. First, I will explain the physiological mechanisms behind our inborn stress response, setting the stage for a discussion about the diverse ways in which excessive stress physically undermines our long-term health.

While attempting to be as inclusive as possible, I will primarily focus upon its effects on the cardiovascular, endocrine, immune, nervous, and digestive systems. In the next section, we will consider why people with a lower socioeconomic status (SES) are more likely to suffer from the undesirable consequences of chronic stress. Finally, this thesis will argue that being stressed out is not an inescapable part of life. Whereas coping mechanisms such as predictability, controllability, the presence of outlets, and social support have un-ignorable caveats, our internal perception of the world around us can help us manage stress most effectively.

II. HOW DOES STRESS OPERATE IN OUR BODIES?

“The irony is this: Our bodies react to stress in exactly the same way whether or not we have a good reason for being stressed. The body doesn't care if we're right or wrong.”

~Doc Childre and Howard Martin, 1999

All of us realize the importance of balance in a world filled with conflicting tensions. We seek out this balance every day at work or school, and in our personal lives. Hence, I continue to be amazed by the body's instinctual maintenance of an internal state of harmony. Although the word “stress” may initially cause us to cringe, we must appreciate the fact that the stress response plays a quintessential role in fostering this equilibrium.

Stress results from a natural drive for allostasis, a term coined by Bruce McEwen (2002) in *The end of stress as we know it*. The principal goal of allostasis is to keep the body internally stable in the face of major outside changes. Before the allostatic concept was developed, the homeostatic concept was widely used. Whereas homeostasis refers to the idea that there is a single optimum for any measure in the body (such as an internal temperature of 98.6°F or an ideal pH level), allostasis suggests that different circumstances call for different homeostatic set points. For instance, blood pressure can either be very high (after encountering a hostile moose on a trail) or substantially lower (while we are asleep). The same idea applies to the concentration of stress hormones in

circulation. Because allostatic systems have much wider parameters than homeostatic systems, maintaining allostasis typically involves far-flung regulatory changes throughout the body (Sapolsky, 2004), as opposed to the local adjustments that take place in homeostatic systems.

One way to think about the role of allostasis in the stress response is McEwen's "seesaw model" (2002). Allostatic equilibrium is relatively easy to maintain when levels of stress hormones are low, as illustrated by the two children on the lower seesaw in Figure 2. Conversely, in response to chronic physical or psychological insults (or purely in expectation of them), allostatic balance is much more difficult to obtain, as depicted by the two sumo wrestlers on the upper seesaw. Not only does it require an enormous amount of energy to balance the large weights of the sumo wrestlers, but they also wear out the seesaw faster and cannot easily dismount. Thus, an over-activated stress response can eventually lead to a self-destructive state called "allostatic load." Allostatic load can also arise from obvious risk factors such as sleep deprivation, drug and substance abuse, an unhealthy diet and/or lack of regular exercise. Interestingly enough, the human mind is so powerful that it can cause allostatic load in the absence of these conventional health risks.

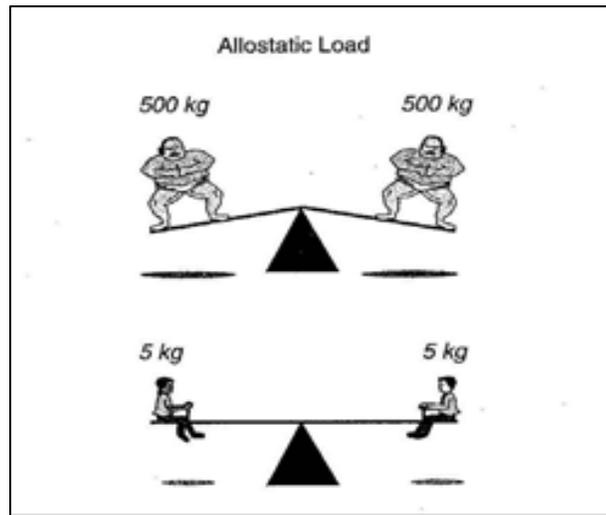


Figure 2. The seesaw model of allostasis (McEwen, 2002)

Mechanisms of Allostatic Load

With McEwen's analogy in mind, chronic stress gives rise to the pathological conditions associated with allostatic load in four different manners—or through any combination (Table 1). Allostatic load transpires when the body produces insufficient stress hormones, when the stress response is activated too frequently, when individuals cannot adjust to acute stress, and/or when the stress response is not turned off in a timely fashion.

Table 1. The four manners of acquiring stress-related allostatic load

Source of allostatic load	Example(s)
Insufficient stress hormones	When there are not enough stress hormones to properly shut off the stress response, autoimmune disease and allergies can arise.
Over-activated stress response	Elevated stress levels kill salmon as they migrate upstream. Monkeys die when forced to establish a new social hierarchy every day.
Inability to adjust to acute stress	Whereas most habituate to a novel stressor (such as a new leadership position), some cannot.
Genetic predisposition against turning off the stress response appropriately	A serotonin deficiency makes it more difficult to get over stressful situations.

First, deficient stress hormones can give rise to the adverse consequences of allostatic load. Although this may seem counterintuitive at first, the stress response is negatively inhibited by the very stress hormones it disseminates throughout the body. This means that when there are high hormone concentrations circulating throughout the blood, the stress response is normally stimulated to shut itself off. Hence, the underproduction of stress hormones prevents proper termination. For instance, a normally-functioning stress response temporarily buffers the immune system. However, if there are not enough hormones to cause negative inhibition, the immune system can become over-activated, giving rise to allergic reactions to harmless substances like dust and cat dander (McEwen, 2002). Similarly, stress can play a role in the onset of autoimmune diseases, in which cells of the immune system attack normal self-cells. As suggested by Arndt, Smith, and Tausk (2008), early stress plays a significant role in the pathogenesis and severity of Atopic dermatitis (AD), a chronic autoimmune skin disease characterized by rashes in infancy or early childhood. Partially because individuals with

AD inherit an under-responsive stress response, the immune system starts to run wild by attacking healthy skin cells. Because of this maladaptive stress response, rashes worsen with increased stress as the immune system gets weighed down with additional allostatic load.

Next, trouble occurs when the stress response reacts to a repeated or drawn-out event in a damaging manner simply due to over-activation. This role of unremitting stress in allostatic load will be the primary focus throughout this thesis. As previously discussed in the introduction, adult salmon are killed by the very stress hormones they utilize to migrate upstream. Another example of this scenario is a U.S. Department of Agriculture (USDA) policy concerning the transport of laboratory animals overseas, as discussed by Fernstro et al. (2008). In 1980, the department thought that monkeys (used for research purposes) needed one hour per day to socialize during shipment, instead of being cooped up inside individual cages the entire time. Lo and behold, this practice actually ended up killing monkeys. In essence, the constant stress of resolving a new dominance hierarchy each day raised stress hormones to life-threatening levels. Thankfully, Sapolsky and other scientists convinced the USDA to revoke this policy soon after its implementation. Fernstro et al. (2008) suggest that stress during overseas transportation is most controlled for when monkeys are housed as compatible pairs, instead of being forced to socialize with a large group.

Allostatic load also comes about from an inability to adjust to acute stress. Usually, we stop activating the flight-or-fight response once we get used to an unfamiliar

challenge that is not lengthy or severe, such as assuming a new leadership position within the workplace or community. However, a study by Wüst, Federenko, Van Rossum, Koper, and Hellhammer (2005) demonstrates that some people are not able to effectively habituate to initially stressful situations. The male participants in this study performed a speech and a mental arithmetic task in front of an audience and camera on three different occasions. Although 52% of the men became increasingly comfortable with the required tasks and showed the normal response habituation, 16% became increasingly anxious across the three test sessions. The authors conclude that these nervous men were not able to habituate due to low self-confidence.

This brings me to the last catalyst for allostatic load: some individuals are genetically ill-disposed to appropriately shut off the stress response in a timely manner. For instance, because the neurotransmitter serotonin (5-HT) acts as a calming mediator of the effects of environmental stress, the relationship between serotonin deficiency and stress-induced depression has been investigated in a longitudinal study by Caspi et al. (2003). Those who became depressed were more likely than non-depressed individuals to carry a small deletion on the promoter region of the gene responsible for generating serotonin. They found that a genetically-derived serotonin deficiency gives rise to an agitated or depressive personality style, deterring certain individuals from letting go after unfavorable life experiences. In other words, these people experience constant ruminations about daily events. If this role of genetic makeup entices you, rest assured that we will come back to it in the following chapter.

The vertebrate stress response

Now that we have a better understanding of the four scenarios in which allostatic load materializes, let us turn to the nitty-gritty physiological mechanisms through which the stress response functions. The endocrine system counters stress-induced imbalances in two steps: one network in the brain signals the activation of particular parts of the nervous system, while another signals the secretion of hormones throughout the bloodstream. The major player in the first part of this innate process is the sympathetic nervous system (SNS). The involuntary autonomic nervous system (ANS) can be broken down into two branches that work in an opposing manner. The SNS arouses, mobilizes and activates the organism by releasing stored energy. On the other hand, the parasympathetic nervous system (PNS) relaxes the organism and stores energy during calm vegetative activities (coined as “resting and digesting”) (Dallman & Hellhammer, 2011). Hence, during real emergencies, or what we *perceive* to be emergencies, the SNS takes precedence over the PNS.

The primary wave of the stress response involves the sympathetic adrenal-medullary (SAM) axis (McEwen, 2002). The response is initiated by the SNS at the base of the brain in the hypothalamus, which stimulates the release of epinephrine (commonly known as adrenaline) from the adrenal glands situated on top of the kidneys. In order to ensure an immediate response, the hypothalamus utilizes a circuit of nerves that go directly to the adrenal medulla, which is surrounded by an outer coating called the adrenal cortex. Meanwhile, norepinephrine (or noradrenaline) is secreted by all the other

sympathetic nerve endings in the body (Sapolsky, 2004). These two neurotransmitters (categorized as catecholamines) work together to quickly stimulate several major changes throughout the body. First, the pulse quickens and blood vessels constrict in order to quickly deliver blood to muscles and oxygen to the brain and lungs. The adrenaline rush also triggers the dissipation of fibrinogen to promote blood clotting. Finally, glucose is released from energy stores and delivered to muscles. During this primary stage of the stress response, the eyes become dilated so that the organism can see the surrounding environment better. Also, the hypothalamus secretes endorphins (or natural pain killers) from the neighboring pituitary gland. Long-term building projects in the body, such as tissue growth, reproduction, and digestion are halted due to the inhibition of the PNS. Therefore, one of the first signs of stress-induced inhibition of digestion is a dry mouth—clearly the production of salivary amylase is not a big concern in the presence of an attacker.

Next, the hypothalamic-pituitary-adrenal axis (HPA axis) ([Figure 3](#)) enhances the primary wave of the response. Since virtually all cells in the body have receptors for the neurotransmitters and hormones proliferated during these two waves, widespread changes ensue (Dhabhar, 2009). In the second stage, the hypothalamus secretes a hormone called corticotrophin releasing factor (CRF), which triggers the brain's anterior pituitary gland to release ACTH (the adrenocorticotrophic hormone) (Sapolsky, 2004). After ACTH travels through the bloodstream to the adrenal cortex, the release of cortisol (a type of glucocorticoid) occurs within a few minutes (Hafen, Karren, Frandsen, & Smith, 1996). Whereas epinephrine and norepinephrine kick in immediately, cortisol brings about its

effects over time. It modulates gene expression by diffusing into the cell nucleus, inhibiting or activating transcription of certain genes.

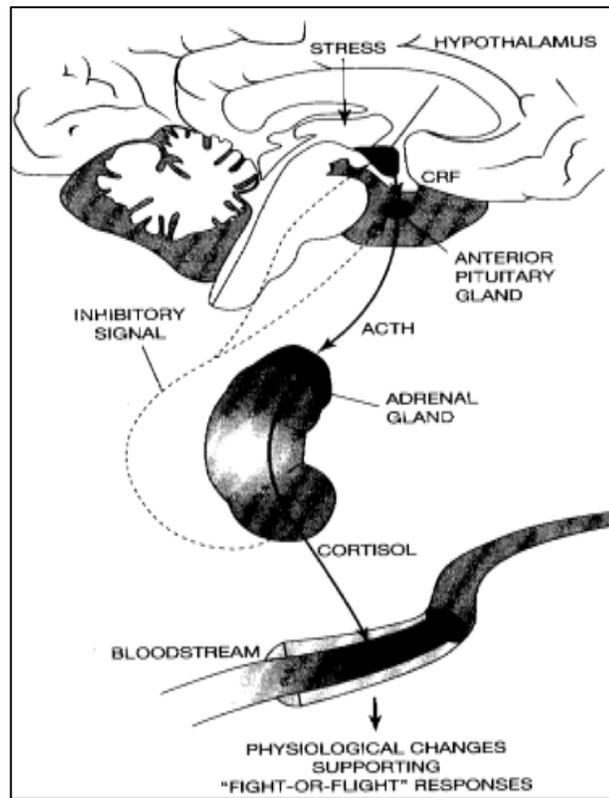


Figure 3. Overview of the HPA axis (Gutman & Nemeroff, 2011)

These two distinct phases encompass nature's fundamental survival mechanism for threatened organisms. The initial burst of cortisol augments immune function for approximately 30 minutes by mobilizing white blood cells throughout the body. This allows for rapid clearance of potential pathogens and efficient wound healing (Dhabar, 2009). Furthermore, while the SAM axis increases delivery of glucose to the brain in order to make energy more available to neurons, cortisol plays a key role in increasing signal transmission (McEwen, 2002). Together, these two factors make for better

memory formation and retrieval, allowing us to quickly decide whether something is a threat or not, and helping us remember what to do about it (Sapolsky, 2004). Moreover, the taste buds, the olfactory receptors of the nose, and the cochlear cells of the ears all require less stimulation to get excited and pass on information to the brain. This enhancement of sensory components explains why people typically remember every single detail before a traumatic event like a car accident. Lastly, when the stressor fades away, cortisol replenishes energy stores depleted by the initial adrenaline rush by stimulating the conversion of a variety of food sources into storage forms, such as fat or glycogen (McEwen, 2002). Therefore, because transient stress has a wide range of survival benefits, natural selection has favored effective fight-or-flight reactions over time. In fact, we use the stress response to our advantage every morning when we get out of bed (McEwen, 2002). Even for those who do not have troubles with waking up, stress hormones make the transition from lying down to standing up easier on our bodies.

Without a functioning stress response we would be susceptible to a variety of adverse effects. For instance, people with untreated Addison's disease are not able to secrete cortisol from the adrenal glands (usually due to adrenal tumors that block receptors) when they are faced with a major stressor. Addison's disease normally arises when the body's tissues are exposed to too much cortisol. Hence, this condition is more common in people taking glucocorticoid hormones for asthma, rheumatoid arthritis and other inflammatory diseases (Newell-Price, Bertagna, Grossman, & Nieman, 2006). Without proper medication, during stressful situations (like a car accident) they can fall into an "Addisonian" crisis—their blood pressure drops, they cannot maintain circulation,

and they potentially go into shock (Sapolsky, 2004). Thus, these life-threatening risk factors illustrate the importance of an operative HPA axis. Certain stressors can also prove fatal to those with multiple system atrophy (also known as MSA or Shy-Drager syndrome), in which impairment of the SNS prevents proper secretion of epinephrine and norepinephrine. Due to this SNS dysfunction, when people with this condition merely stand up, they can undergo a drastic drop in blood pressure, dizziness, and muscle spasms (Sapolsky, 2004).

For another example of the benefit of a functional stress response, consider a recent study by Dr. Firdaus Dhabhar (2010) of Stanford University that illustrates the role of acute stress in skin cancer prevention. In her review of Dhabhar's study, Young (2011) describes how acute stress readies the immune system to fight off disease-causing agents (like tumor cells). Dhabhar exposed hairless mice to UV-B exposure three times a week. Before exposure, one group of mice experienced short-term stress when placed in tubes that restricted their movement for a few hours. After 10 weeks, the restrained mice developed fewer tumors than the unstressed mice, indicating that the augmented immune system of the stressed group protected them. However, this protection only lasted for about 26 weeks. After that time both groups of mice developed cancerous tumors. We still need more research about the specific molecular mechanism behind this phenomenon, but perhaps someday doctors will be able to successfully exploit this stress-induced enhancement of the immune system in order to prevent cancer in humans.

Health consequences of chronic stress

To commence our discussion of the adverse stress-related conditions that have been illuminated in the last couple of decades, let us first look at a concrete example of how chronic stress impairs us on a molecular level. Typically, chronically stressed individuals have shorter telomeric DNA on the ends of chromosomes because cortisol causes oxidative damage (via the dissemination of “free radicals”) and dampens the ability of telomerase to add extra DNA segments to chromosomes with each cell division. This matters because telomeres protect the end of chromosomes from fraying. Thus, short telomeres jeopardize cell division and longevity. In order to study this, Epel et al. (2004) compared telomere length in mothers with healthy children to mothers with chronically ill children. The mother’s age was controlled for. Not surprisingly, mothers with ill children had more perceived stress. Consequently, for these anxious women, more years of caregiving corresponded to shorter telomere length, lower telomerase activity, and greater oxidative stress. On average, the telomeres of caregiving mothers were shorter by the equivalent of at least one decade of additional aging compared to the controls. Hence, this study demonstrates that the effects of stress are cumulative, real, and scientifically supported.

On top of shortened telomeres, chronic stress can make us more vulnerable to obtaining impairments in multiple bodily systems, or worsen certain preexisting diseases, as summarized by [Table 2](#). We will go into further detail about these particular health conditions in the remaining portion of this chapter.

Table 2. Adaptive and mal-adaptive aspects of stress

Body system	Acute stress	Chronic stress
Cardiovascular system	Increased heart rate. Blood vessels constrict. Oxygen and glucose is delivered to muscles, lungs and brain.	Hypertension, atherosclerosis, cardiovascular disease, heart attack, and stroke.
Endocrine system	Insulin inhibited from taking up glucose. Glucagon stimulated to release glucose.	Increased abdominal fat. For a type 1 diabetic, difficulty controlling glucose levels. Instigation of Type 2 diabetes.
Immune system	Leukocytes proliferated throughout the body to prepare for wounds. Better antibody response and recovery time.	Cortisol jeopardizes leukocyte function. Autoimmune disease and allergies can arise.
Nervous system (brain)	Alert. Glucose and oxygen travel to brain. Cortisol increases signal transmission between neurons (better memory).	Headaches. Cortisol decreases signal transmission, jeopardizes memory. Hippocampus shrinks. Nerve cells are lost.
Digestive system	Halted digestion to leave enough energy for muscles.	Irritable bowel syndrome and ulcers are more likely.

The cardiovascular system

Let us begin with the cardiovascular system. As the PNS is turned off and the SNS is activated, heart rate increases and arteries constrict, allowing blood to move faster and more forcefully. Hypertension, or chronically elevated blood pressure, usually arises first. This can then give rise to tearing in the smooth inner lining of blood vessels. Fatty acids, glucose and clotting-factors mobilized at the onset of stress can work their way into these little caverns, causing the accumulation of plaques that harden the arteries, leading to atherosclerosis (Sapolsky, 2004). Clogged blood vessels block the flow of

oxygen to the heart, which can set off a heart attack, or to the brain, which can trigger a stroke (McEwen, 2002). Cardiovascular (CV) disease is responsible for the most deaths in the United States (approximately 40%), and chronic stress only makes matters worse. In fact, ongoing stress experienced while caring for a disabled or ill person doubled a woman's chances of having an adverse CV event in a prospective study by Lee, Colditz, Berkman, and Kawachi (2003). Similarly, in a four year study, Schulz and Beach (1999) found that caregivers had a 63% higher mortality rate than non-caregivers. This increased mortality was most evident in caregivers with known CV disease, indicating that the emotional strain of care-giving enhances the severity of this condition.

The endocrine system

Next, chronic stress threatens proper functioning in the endocrine system, particularly with insulin, a hormone that lowers blood glucose levels. Increased glucagon during the stress response instigates the breakdown of triglycerides in fat cells, resulting in elevated blood glucose levels. Moreover, stress hormones act on fat cells throughout the body to make them less sensitive to insulin, thereby preventing the uptake of glucose (Sapolsky, 2004). Because this preferentially occurs in the visceral fat cells of the abdomen, chronic stress promotes an increased waist circumference (or "apple shape"), especially if an individual copes with stress by eating too much "comfort food." Abdominal fat is more of a risk-factor for metabolic disease than fat carried elsewhere on the body (such as the hips) because fat from this area more readily reaches the liver,

where it is converted to glucose (Sapolsky, 2004). This in turn potentially gives rise to elevated blood sugar and insulin resistance.

On that note, for a person with juvenile (type 1, insulin-dependent) diabetes, an autoimmune disease in which insulin cannot be secreted from the pancreas, prolonged stress can make it harder to keep the disease under control with insulin injections (Sapolsky, 2004). For instance, after surveying young adults with type 1 diabetes and measuring cortisol secretion, Hislop, Fegan, Schlaeppli, Duck, and Yeap (2007) found that over one-third of the participants experienced considerable psychological distress, including symptoms of depression. Not surprisingly, those with increased psychological distress demonstrated poorer control of glucose levels. Furthermore, insulin injections became less effective over time.

Additionally, chronic stress can trigger adult-onset (type 2, non-insulin-dependent) diabetes, in which pancreatic beta-cells fail to respond to insulin as a result of fat surplus (Sapolsky, 2004). For example, Heraclides, Chandola, Witte, and Brunner (2009) found that women who experienced a combination of high job demands, low job control (job strain), and low work social support had a twofold risk of developing type 2 diabetes. Therefore, for someone just on the verge of insulin resistance, too much stress can potentially cause that person to cross the threshold to becoming a type 2 diabetic.

The immune system

Much research has centered upon the fact that excessive stress impairs the immune system from successfully fighting off pathogenic viruses, bacteria, parasites and

fungi. As previously mentioned, the acute stress response prepares the immune system for potential wounds or infections during the first 30 minutes of a stressor: the body's "soldiers" (leukocytes, natural killer cells, B cells, and T cells) exit their "barracks" (spleen, thymus, and lungs) through the bloodstream and take their "battle stations" in the skin, lungs, gastro-intestinal and urinary-genital tracts, mucosal surfaces, and lymph nodes (Walsh et al., 2011). However, too much cortisol diminishes resting-state immune cell numbers, reduces the effectiveness of the "soldiers" in circulation, and increases immunosuppressive mechanisms (e.g. regulatory/suppressor T cells) (Dhabhar, 2009). The primary manner in which such immune suppression occurs involves the ability of glucocorticoids to shrink the spleen and thymus (Sapolsky, 2004). These two organs are involved in the production of active B cells and T cells.

These factors make chronically stressed individuals more vulnerable to many different infections. For example, Pressman, Cohen, Miller, Barkin, and Rabin (2005) monitored levels of loneliness, stress, mood and health behaviors in college freshman who were first-time recipients of the influenza immunization. Entering college can be very stressful, so these researchers set out to quantify stress levels by taking salivary cortisol measures throughout the first semester. They also investigated the specific antibody response to the flu immunization via blood samples drawn on the day of vaccination, as well as one and four months post-vaccination. Those with both high levels of loneliness and a small social network had the lowest levels of antibody response. Hence, the students with perceived social support had immune systems that were significantly protected against the long-term negative effects of cortisol on antibody

function. This study helps explain why stressed-out students often get sick during final exams or throughout a difficult semester.

Whereas the link between stress and antibody responsiveness finds much support, a jeopardized immune system does not automatically imply a greater cancer risk. Several *animal* studies suggest that stress contributes to the initiation, growth, and metastasis of select tumors due to the suppression of natural killer cells that fight tumors (Sheldon, Janicki-Deverts, & Miller, 2007). The problem with cancer studies in humans is that they tend to be retrospective instead of prospective. In other words, when cancer patients look back upon their lives, they are more likely to come up with an amplified list of major stressors that would potentially explain their disease. Moreover, the evidence from prospective studies linking stress and cancer in humans is mixed at best. Part of the difficulty may lie in the fact that cancers are diagnosed only after they have been growing for many years, making an association between stress and disease onset difficult to demonstrate. It is also challenging to study the role of stress on cancer progression because of our current inability to accurately quantify the severity of cancer at a particular stage. Although research about stress and cancer has generally been inconclusive, positive thinking and avoidance of stress certainly cannot hurt when it comes to surviving a cancer diagnosis.

Because chronic stress abuses the “on-and-off switches” in the immune system, it can exacerbate allergies or asthma when uncoordinated cells of the immune system go into overdrive, attacking entities that do not pose serious threats (such as cat dander and

pollen) (McEwen, 2002). Similarly, self-cells can be targeted, resulting in autoimmune diseases like the previously discussed example of Atopic dermatitis. Moreover, stress acts as an aggravating factor in the Rheumatoid Arthritis (RA) disease course, which involves painful inflammation around joints. Research regarding the relationship between stress and RA has primarily focused on pro-inflammatory cytokines, intercellular messengers responsible for triggering tissue swelling (Younger & Zautra, 2007). In healthy individuals, the anti-inflammatory hormone cortisol acts as an immunomodulator by suppressing both the production and activity of pro-inflammatory cytokines. Despite the fact that chronic stress involves increased levels of cortisol in the bloodstream, stress increases pain in RA patients. This occurs because both pro- and anti-inflammatory agents are released during the stress response. More painful RA symptoms result from a disproportionately higher abundance of pro-inflammatory agents (such as prolactin and C-reactive protein) compared to anti-inflammatory agents.

The nervous system

Let us now shift our focus to the nervous system. Not only is the brain the central mediator of the stress response, but it also serves as a central target of stress processes. For instance, chronic stress jeopardizes proper functioning of neurons in the brain associated with retention of information. First, it is important to make a distinction between two different types of memory. Implicit memory allows us to do procedural things (e.g., riding a bike or walking down the stairs) without thinking. Stress is more detrimental to explicit memory, which involves conscious awareness of facts and events

(Sapolsky, 2004). Memories are “stored” in the cerebrum via long-term potentiation (LTP), a continuing signal enhancement in the space between two neurons called the synaptic gap. Due to repetitive stimulation, the postsynaptic neuron (the one that transmits electrical signals away from the synapse) becomes increasingly responsive to signals throughout the LTP process. The hippocampus, a component involved in placing and accessing memories, has many cortisol receptors. When cortisol is secreted during acute stress, it primarily binds to “high-affinity” receptors in the hippocampus, enhancing LTP. However, when it floods the brain during chronic stress, cortisol activates a high proportion of “low-affinity” receptors, inhibiting LTP. Thus, anxious test-takers sometimes cannot remember previously-studied facts during the heat of an exam.

In various species, chronic stress not only causes nerve cells in the hippocampus to shrink (and even die), but also suppresses the growth of new brain cells (neurogenesis). McEwen (2002) was among the first to test this phenomenon in his studies with the tree shrew, an animal that prefers solitude over interactions with its own kind. When two tree shrews are forced to coexist in the same cage for an hour, excessive cortisol levels suppress neurogenesis and give rise to shorter, less-branched neurons. Although diminished synaptic connections stifled information flow temporarily, neurons regrew when the hour-long stressor ended. High cortisol levels play the same role in the human brain, as demonstrated by a study of Cushing’s syndrome by Starkman, Giordani, Berent, Schork, and Schteingart (2001). This condition arises when tumors spontaneously secrete a deleterious amount of cortisol into the bloodstream. The more severe the hypercortisolemia, the greater the negative effects on cognitive function. Patients with

Cushingoid dementia perform significantly worse on verbal, visuospatial, learning and memory tests than controls and show a decreased hippocampus size.

Furthermore, too much stress not only precipitates individual headache episodes, but it also contributes to the onset or progression of an ongoing headache disorder in predisposed individuals (Nash & Thebarg, 2006). A vicious cycle can arise when the headache experience itself serves as a stressor, further jeopardizing an individual's well-being. Although the precise mechanisms behind this are still not perfectly clear, stress-induced headaches arise when increased blood pressure or increased tension in the muscles of the neck and scalp pinch nerves, causing pain.

The digestive system

There remains one final area of the body to consider: the digestive system. Irritable bowel syndrome (IBS) is one of the most common stress-related disorders (Sapolsky, 2004). In fact, ongoing stress both increases a person's susceptibility to IBS and worsens established cases. In essence, uncoordinated contractions in the colon jeopardize normal water absorption, resulting in either diarrhea or constipation. Moreover, stress also plays a role in the development of ulcers, or holes in the walls of organs, as explained by Sapolsky (2004). His book title makes it perfectly clear that zebras don't get digestive ulcers, right? So why is it that we do? First, we already know that stress impairs our immune system from effectively killing the causative bacterium for ulcers, *Helicobacter pylori*. Furthermore, during stressful situations, the stomach wall thins. A reduced amount of mucus is secreted, lessening the stomach's protection against

acidic digestive juices that can bring about ulcers. Lastly, the stress response diverts oxygen-rich blood flow away from the gut. Due to this diminished amount of oxygen, little cuts can appear on the stomach wall. The dead tissue in these regions serves as an ideal building block for ulcers.

Thinking about all of the health consequences attributed to excessive stress can certainly be daunting. However, I hope that this background information fully demonstrates the fact that allostatic load threatens our long-term well-being through several well-researched pathways. Although the amount of chronic stress we experience has real consequences, we will soon see that both personality and social standing are significant factors in the severity of all the conditions discussed above.

III. WHAT ROLE DO SOCIAL FACTORS PLAY?

“...if the culture in which we live shapes who we are—our thoughts, emotions, and actions—it must shape our underlying biology as well.”

~Robert Sapolsky, 2005

During every summer for the last 30 years, Robert Sapolsky (2004) has traveled to the middle of the Serengeti to research stress levels in the same troop of wild baboons (*Papio anubis*). Because these baboons only have to spend about four hours per day foraging for food, they have a lot of time left over to sort out dominance hierarchies by maliciously beating up on each other. Apparently, baboons with the lowest basal glucocorticoid levels have several unifying traits. They know how to differentiate between malicious and meaningless encounters with others, take control of threatening situations instead of acting passively, know whether they win or lose during confrontations, have outlets for frustration (e.g., other baboons to beat up on), and are social animals. Because not all baboons possess these low-glucocorticoid traits, some are more reactive to stressors than others. For instance, apprehensive, socially secluded baboons not only have an elevated risk of stress-related illness, but also have little chance of maintaining dominance over others in the troop. Sapolsky therefore suggests that baboons with more self-confidence and a higher social standing experience less stress-related illnesses than their constantly anxious counterparts.

In order to investigate this relationship between baboon social status and stress, Gesquiere et al. (2011) measured glucocorticoid levels in savannah baboons (*Papio cynocephalus*) of different ranks over a nine-year period. As depicted by Figure 4, regardless of the stability of the social hierarchy, high-ranking males had lower glucocorticoid levels than subordinate males. The major exception to this finding involved the highest-ranking alpha male. Compared to the second-ranking (beta) baboon, alpha males (rank number 1) exhibited markedly higher glucocorticoid levels. In fact, glucocorticoid levels in the alpha male were comparable to those of the lowest-ranking members. This study therefore indicates that alpha and omega baboons activate the same physiological stress response for different reasons. The baboon in charge must ceaselessly struggle to defend his authority. On the other hand, those at the bottom of the social ladder primarily experience stress when higher-ranking males use them as stress-relieving “punching bags.”

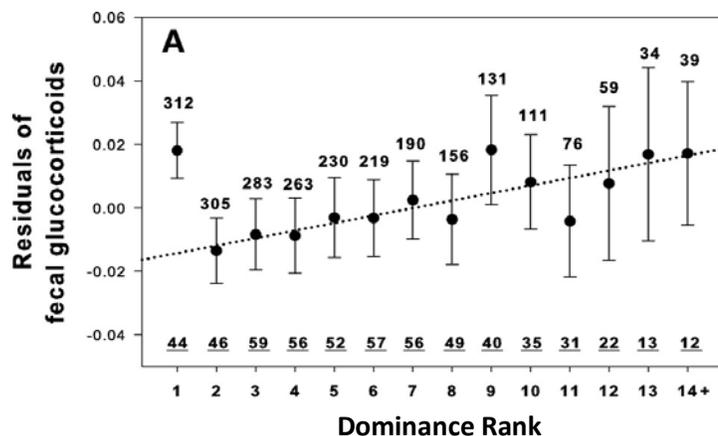


Figure 4. Glucocorticoid levels in baboons of different ranks (Gesquiere et al., 2011)

However, as we will soon see, a graph of glucocorticoid levels of humans with high and low social ranks would not necessarily show the same results as this particular baboon study. Our social ranks are not as clear-cut as baboons, and we hopefully do not go around beating each other up to sort out hierarchies. Moreover, we may be dominant in one arena (e.g., running marathons) and subordinate in another (e.g., the workforce), blurring the lines of the social ladder. In other words, if we really value our ability to outrun others, this can offset feelings of subordination at work. Hence, there is more of a cognitive influence in human hierarchies.

Yet, this is not to say that these two studies by Sapolsky (2004) and Gesquiere et al. (2011) are irrelevant to our discussion of the social factors involved in stress-related disease. For instance, they can help explain why people with dissimilar personalities react to stressful situations in different manners. Generally speaking, some of us resemble the aloof omega baboons, some of us take after the competitive alpha males, and the rest of us fall somewhere in between. Consequently, certain people have more pronounced psychological and emotional responses to common stressors like sitting in traffic, being late, and keeping up with daily demands.

For example, in the previous chapter, we saw that those born with a deficiency in serotonin cannot recover from a stressor as easily as others, potentially giving rise to allostatic load and the accompanying health problems (Caspi et al., 2003). These individuals are more prone to having a “Type-D” personality, which involves negative emotions, referred to as “negative affect” (e.g. uneasiness, irritability, and gloom). When

Type-D people conclude that things are terrible and getting worse, it is no wonder that they tend to have higher amounts of stress hormones in circulation. However, this is not the only personality-derived predisposition to stress-related allostatic load.

Much attention has also focused upon an elevated stress response in hostilely competitive “Type-A” people, who view life as a constant struggle against others. This idea first found support in 1966 with the Western Collaborative Group Study (WCGS), by Rosenman et al. After controlling for smoking and age, this prospective study demonstrated that having a Type-A personality increased the risk of coronary heart disease (CHD) (in the 3,524 male subjects) just as much as well-known risk factors like hypertension, elevated serum lipids, and high cholesterol. Not surprisingly, health risks became most significant when a stress-related and/or conventional risk factor occurred in someone with a Type-A personality. Additionally, in a follow-up of WCGS, Hecker, Chesney, Black, and Frautschi (1988) emphasize hostility over the global Type-A characterization. It is important to note that Type-A people without hostile tendencies were not significantly susceptible to CHD. Specifically, hostility entails an inclination to express irritation, annoyance, disgust, resentment, and frustration. Because chronic hostile behavior increases stress hormones in circulation, the resulting wear on the cardiovascular system thereby considerably increased the onset and severity of CHD in these particular Type-A subjects.

Another commonly studied personality trait is neuroticism, a polygenic condition characterized by anxiousness and emotional over-reactivity to environmental demands.

Not only do neurotic people chronically feel nervous about the world around them, but they are also typically insecure and socially isolated (McAdams, 2001). Thus, neurotic individuals are more susceptible to a maladaptive stress response for several reasons. First, they tend to experience an augmented amount of stressful events on a daily basis. For instance, highly-neurotic individuals have a higher predisposition to engage in interpersonal arguments than those with low levels of the trait (McAdams, 2001). Furthermore, activation of the stress response is more pronounced due to ineffective coping strategies such as self-blame and social isolation. Lastly, even in the absence of unfavorable events, an overall negative outlook on life gives rise to plenty of distress on its own. Thus, these three different traits demonstrate the fact that we simply cannot ignore the personality component in our review of social factors.

The trend in modern medicine, though, has been to stop here. In other words, health care professionals and scientists (along with the majority of the rest of us) sometimes reduce health to tangible entities (like genetics, diet, exercise, and access to health care). “Reductionism,” in this sense, involves assuming we can fully understand a certain health condition by reducing it down to molecular components or obvious lifestyle factors. Clearly, reductionism has been of the utmost importance in achieving such medical breakthroughs as antibiotics and advanced surgical techniques. However, when we believe that the whole is smaller than the sum of the parts, we may miss the big picture. Thus, stress-related disease, or any disease for that matter, should not be considered in a social vacuum. Medicine is not about passing out painkillers whenever necessary, but rather broadening our scope of understanding in order to figure out the

source of the problem. Although the predispositions we are born with matter, the society in which we live must be considered, too.

In fact, social status and personality interact in a critical manner—a certain personality can be highly adaptive in one position in society but highly predictive of stress-related disease in another. For example, consider the case of John Henry, a 19th century folk hero who outraced a steam drill tunneling through a mountain. Using his nine pound hammer, John Henry emerged the victor, but soon dropped dead from complete physical and mental exhaustion (James, 1994). Hence, “John Henryism” involves approaching every situation with a maximal amount of personal effort and autonomy. In other words, these people always interpret themselves as being in control. Although this trait may help comfortable middle-class people succeed, it is maladaptive in poor people who face prejudices or have limited opportunities. Consequently, James (1994) found that John Henryism frequently gave rise to hypertension (and thus cardiovascular disease) among working class African Americans—but this was not the case for working-class whites or middle-class African Americans. This further emphasizes the point that there is more to stress-related disease than personality alone.

Because the social and economic conditions in which we live, work, and study vividly affect our biology and well-being, health disparities among different groups have received more consideration recently. For our purposes, “disparity” can be defined as a difference in health status between social groups (e.g., socioeconomic, racial/ethnic, gender) that is not only unnecessary and avoidable, but is also considered unfair and

unjust (Alder & Stewartz, 2010). We will primarily focus on the socioeconomic element of this definition because an unquestionable gradient in socioeconomic status (SES)—measured by education, occupation, and income—exists in world-wide health.

For instance, Marmot (2004) illustrates this gradient by describing a short ride on the subway in Washington, D.C. from the poor neighborhoods southeast of downtown to the well-off Montgomery County, Maryland. Life expectancy rises about a year and a half for each mile traveled. In fact, there is a 20-year gap between poor blacks at the start of the subway ride and rich whites at the finish. Thus, even for people living side by side in the same country, the SES gradient clearly finds expression. These findings should not lead us into thinking that the health gap is confined to bad health for certain disadvantaged individuals and good health for everybody else (Marmot, 2004). This is not a matter of “us” and “them.” Instead, the gradient affects everyone from the very bottom of the social hierarchy to the very top, giving rise to disquieting health differences all along the way.

Figure 5 provides a good representation of the mechanism behind this SES gradient. We can clearly see how the ultimate health outcome of coronary heart disease (CHD) and diabetes is influenced by the interplay of multiple factors—one’s place in society, genetic endowment, upbringing, health care access, work and home environments, health behaviors, and psychological reactions to life circumstances *all* play a vital part, here. As indicated by the “Psychosocial Work” box in the upper left hand corner of this schematic, stress cannot be ignored in all of this.

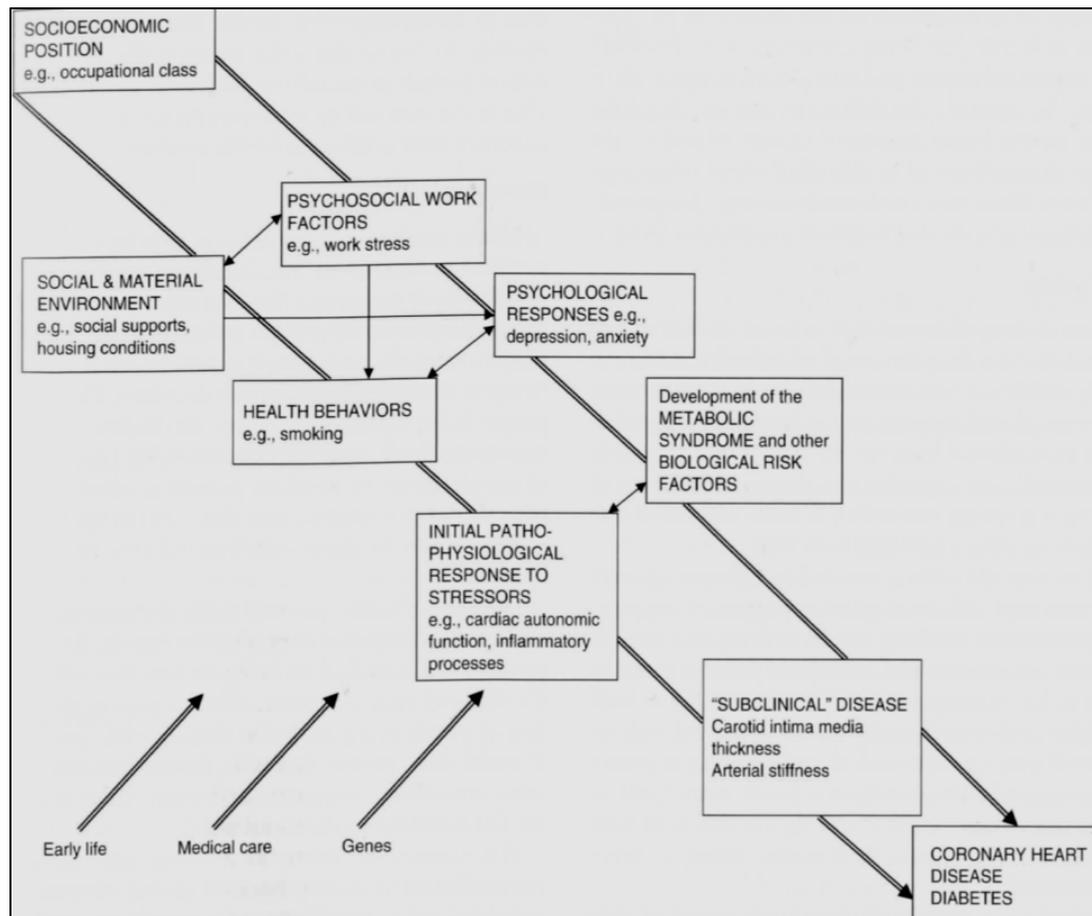


Figure 5. Interrelating factors determining disease onset (Chandola & Marmot, 2007)

Thus, much consideration has recently been devoted to the role of stress in health disparities. Many agree that those with a lower SES experience a higher degree of physical and psychological stress over a lifetime. Even though a higher SES individual (like a well-paid business executive driving around in a shiny Hummer) may *perceive* his job demands to be higher than lower SES individuals, a lower SES is associated with greater reports of prolonged stress from life events, close personal relationships, and work characteristics (Chandola & Marmot, 2007). In essence, psychological experiences of inequality can really get under our skin.

For instance, cardiovascular disease, respiratory disease, ulcers, rheumatoid disorders, psychiatric diseases, and a number of types of cancer are positively correlated with poverty (Sapolsky, 2004). There are numerous conventional explanations for this: living next to a toxic dump or in a heavily-polluted area, violence, smoking, drug abuse, limited education, restricted health care, manual labor, unemployment or insecure employment, and a general lack of control or outlets for frustration (such as a vacation or yoga class) (Sapolsky, 2004). Lifestyle factors like diet and exercise undoubtedly play a role, too. For instance, supermarkets with large selections of fresh produce and other nutritious foods are most prevalent in higher SES neighborhoods than lower ones (Morland, Wing, Diez-Roux, & Poole, 2002). Conversely, cheap and greasy fast food restaurants tend to target low SES areas. Furthermore, poorer people may be less likely to jog around the neighborhood due to safety issues. Because these conventional factors really take a toll on health, some explanations of health disparities solely focus on these things. For example, Adamson, Ebrahim, and Hunt (2006) suggest that those with lower income and less advantaged employment conditions would be at higher health risks regardless of how much stress they happen to experience.

Interestingly enough, though, even when health care is provided universally to all social classes, the health gradient persists. In fact, it has actually worsened over the last century in the United Kingdom, despite the imposition of socialized health care for more than 50 years (Sapolsky, 2004). Starting in 1960, the Whitehall I study set out to determine why that was happening. Marmot, Shipley, and Rose (1984) were among the first to longitudinally investigate the role of societal factors in stress-related disease by

comparing coronary heart disease mortality rates of over ten thousand British civil servants. The British civil service was selected because of its well-defined social hierarchy. To this day, administrators at the top grade are responsible for setting policy, which is carried out by lower executives. Professionals and technical staff comprise the next group down, followed by the clerical grades and finally the office support grades. Despite the presence of these clear lines of authority, even the lowest jobs within the British civil service tend to be secure, providing adequate salaries and equal health care access to employees.

Despite these controls, those with the lowest social status had substantially higher mortality rates than the highest social class, as demonstrated by [Figure 6](#). Shockingly, for men between 40 and 64 years old, those at the bottom of the hierarchy had a four times greater mortality rate than that of the administrators at the top (Marmot, 2004). Interestingly enough, the noticeable differences between social groups did not taper off when the subjects were between 70 and 89 years old. Conventional risk factors such as smoking, hypertension and triglyceride levels in the blood only accounted for a third of the variability in the data (Sapolsky, 2004). Hence, the authors suggest that psychosocial factors from work and family life fill in the unexplained part of the social gradient. The Whitehall studies challenged the traditional idea that above a certain threshold of income, additional resources would do little to benefit health. Thus, health disparities cannot simply be erased in this manner.

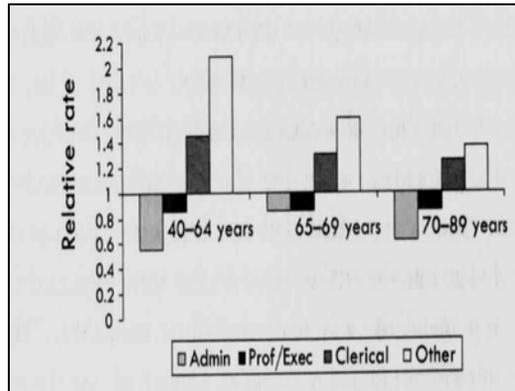


Figure 6. Relative mortality rate and occupational hierarchy level (Marmot, 2004)

Ever since the Whitehall studies, additional support for the chronic stress argument has surfaced. For example, an interesting study of mortality rates in a group of Catholic nuns was performed by Snowden, Ostwald, and Kane (1989). These nuns had the same diet, self-care activities, clothing, and health care access. Even though they thought of each other as equals, patterns of disease, dementia, and longevity were primarily determined by childhood SES. On average, sisters with bachelor's degrees lived to be 89.4 years, sisters with some high school or college education lived to be 82.2 years, and sisters with grade school education lived to be 82.0 years old. The authors suggest that nuns with lower education experienced more familial stress while growing up, generally giving rise to a shorter life.

But just because these two studies both point out earlier mortality, can we confidently conclude that there is a greater prevalence of stress-related disease among lower SES individuals? Sapolsky (2004) would answer this question by arguing that the strongest SES gradients occur for diseases with the greatest sensitivity to stress—namely,

heart disease, hypertension, psychiatric disorders—while cancer has the weakest overall gradient (because, as previously discussed, the relationship between stress and cancer is for the most part inconclusive) (Sapolsky, 2004). Therefore, a low SES truly increases a person's vulnerability to the negative health effects of chronic stress. In order to back up this claim, let us consider some more concrete stress-related explanations for the SES gradient.

First, it is important to understand that people with a lower SES have more stress hormones in circulation. As demonstrated by Cohen, Doyle, and Baum (2006), lower social status is associated with higher basal levels of cortisol, epinephrine, and norepinephrine. We already know that this can impair multiple bodily systems in the long run. For instance, Owen, Poulton, Hay, Mohamed-Ali, and Steptoe (2003) studied the effects of increased stress hormones on the immune system by looking at C-reactive protein (CRP), which is involved in inflammation during the fight-or-flight response. CRP helps explain why people with a low SES are more susceptible to coronary heart disease and immune-related disorders than higher SES individuals. It is important to note that none of the participants in this study were ill during testing periods, and even those categorized as “low SES” had health care access and incomes above the national average. Nevertheless, on average there were significantly higher concentrations of CRP in low SES participants. In essence, chronically increased inflammation induced by CRP jeopardizes blood flow through the heart, potentially increasing the risk of coronary heart disease in people with a lower social standing.

In fact, links between social standing and stress-related maladies endure throughout a lifetime. Starting with childhood, low SES families are less likely to function as a unit. Instead, elevated conflict levels, cold and negligent relationships, and harsh parenting give rise to “risky families,” a term coined by Cohen, Janicki-Deverts, Chen, and Matthews (2010). Children from risky families have increased odds of psychological and social dysfunction. Also, children in affluent schools or neighborhoods are less likely to observe or be victimized by physical violence. Moreover, higher SES schools have more consistent attendance rates, contributing order and cohesiveness to the school setting.

Thus, Cohen et al. (2010) argue that low SES children experience more psychosocial stress than their counterparts. Poor emotional regulation, difficulties with social competence, and exposure to violence or crime can all contribute to an unhealthy amount of allostatic load. For instance, in response to emotional stimuli, adults from risky families express atypical activation of the amygdala (a brain structure involved in anxiety and fear) (Cohen et al., 2010). As indicated by the results of Magnetic Resonance Imaging (MRI) tests, such kids do not appropriately detect or cope with threats, giving rise to a heightened stress response.

Chronic stress in the workforce also shapes the SES gradient in health. To name one example, Clougherty, Souza, and Cullen (2010) compared the incidence of hypertension (a stress-related condition) among full-time hourly (“blue-collar”) workers and salaried (administrative and “white-collar”) workers in a large manufacturing

company. After adjusting for income, education, and age, hourly workers had a significantly greater risk of hypertension than salaried workers. Risks among blue-collar workers were highest in those with the most years on the job, indicating that cumulative physical and chemical exposures have a serious impact on blood pressure. Increased risks of hypertension were also attributed to psychosocial factors such as chronic job strain, low job control, and stressful work conditions. Other stressors that blue-collar workers experience include decreased chances of a promotion, little participation in decision-making and unsupportive co-workers or managers (Clougherty, Souza, & Cullen, 2010). Thus, to the extent that work is perceived as stressful, resulting wear and tear increases disease risk.

Even after retirement, the SES gradient persists. We already saw this in the Whitehall studies, where mortality differences in the four occupational groups did not disappear in the 70-89 year category ([Figure 6](#)). However, the differences in mortality based on occupational status became less drastic after retirement, suggesting that work itself plays an important part in generating social inequalities in health (Marmot & Shipley, 1996). It is important to remember that social hierarchies do not simply disappear upon retirement, though, and neither does the SES gradient.

Over and over again, studies have suggested that as SES increases, stress-related disease decreases. However, it is not just rank that influences physiology, but also the sort of society in which the rank occurs, and the individual's perception of rank and society. Clearly, not all communities or individuals experience the same amount of

psychological angst about social status. Hence, SES alone is not the best predictor of stress-related disease. As discussed by Sapolsky (2004), *feeling* poor translates into bad health when it activates the stress response repeatedly. Therefore, instead of constantly comparing ourselves to others, we have the ability to choose a calmer outlook about our positions within society.

In order to demonstrate this idea, consider the first troop of baboons that Sapolsky (2005) studied. One summer, the vast majority of dominant males died after a tuberculosis outbreak that resulted from foraging for meat in a garbage dump behind a tourist lodge in Kenya. Only the “peaceful” baboons survived this outbreak because the dominant males would not let them enter the dump. Along with the females and young baboons, the subordinate males formed a society devoid of competition for social status, focusing on cooperative grooming instead of cutthroat actions. When new male baboons joined the troop, they had to assimilate to the established culture. Consequently, stress hormone levels decreased and stress-related disease no longer jeopardized these primates.

If wild baboons can undergo such a drastic behavioral change, can humans work towards a more cohesive society in which social standing does not make certain individuals more vulnerable to stress-related disease than others? Since stress-related disease is especially prevalent in underprivileged societies, this is a global issue that we should all make an effort to change—both in our personal lives and in the lives of people around us. We will see how this works in the following chapter.

IV. WHAT CAN WE DO?

“Do you suffer from...the distress that makes human wills flounder daily under the crushing number of living things and stars?”

~Annie Dillard, 1999

Annie Dillard (1999) opens *For the Time Being* “with the bad news” by describing children with bird-headed dwarfism, a genetic condition in which people grow to a maximum height of three feet, have abnormally large eyes and noses, are mentally deficient, and suffer from severely displaced hips and elbows. Dillard then goes on to say, “You cannot turn a page in *Smith’s Recognizable Patterns of Human Malformation* without your heart pounding from simple terror. Will [these] particular [dwarfs] live?” In the face of these upsetting birth defects and other forms of “the bad news” that we encounter daily, Dillard asks her readers a pertinent question: “Do you suffer [from]...the distress that makes human wills founder daily under the crushing number of living things and stars?”

The vast majority of us have indeed experienced such distress. Not only do we constantly hear about natural disasters, violence, and injustice in the world, but we also are subject to personal worries about our social status, finances, relationships, and lofty expectations at school or work. In other words, we cannot reasonably hope to live an entirely stress-free life. After all, an appropriate amount of stress adds zest to our lives

and helps us set and realize goals. So what can we do to make stress less toxic to our minds and bodies? Although difficult circumstances are inevitable at times, thankfully we can take certain steps towards moderating the negative impacts of chronic stress. For example, a large body of scientific literature underscores the efficacy of the following stress-reducers: predictability, controllability, the presence of outlets, and social support (Marmot, 2004). Let us consider a few key studies dealing with these four entities.

Predictability

First, the more predictable a negative circumstance is, the less distress it causes. When two rats were exposed to a pattern of electric shocks, for example, the rat hearing a warning bell before each shock underwent a significantly diminished stress response (Sapolsky, 2004). The importance of obtaining preparatory knowledge prior to a stressful situation also finds expression in humans, according to a study by Inzana, Driskell, Salas, and Johnston (1996). Before performing a computer-based decision-making task under high-stress conditions, Navy personnel were given either general task instructions or specific instructions. Those who received the specific training not only reported less anxiety and greater confidence while performing the task, but also made fewer errors.

Predictability protects us from negative or excessive stress responses in two different manners, as discussed by Inzana et al. (1996). First, it provides a preview of the stress environment, rendering the event less novel and fostering an expectation of self-efficacy. In other words, predictive information decreases the size of the stress response *during* the actual stressor. This means that we should strive to ask questions and gain

insight before jumping into unfamiliar and/or potentially stressful commitments. Second, the removal of a previously existing predictive signal indicates when an organism can relax. With the prior example involving rats, for instance, the rat without the warning light is constantly a half-second away from shock (Sapolsky, 2004). The warned rat, on the other hand, has a faster recovery *after* the shock is over with because he knows when to expect the stressor. Thus, predictability helps us realize what we are prone to get stressed out about, making it easier to appropriately shut off the stress response instead of letting it get the best of us.

Controllability

This leads us to the second stress-reducer on the list: controllability. The belief that one can draw upon behavioral or cognitive responses to lessen the impact of adverse events also finds expression in shocked rat experiments by Davis and Levine (1982). In this case, though, one of the rats could press a lever to decrease the likelihood of the shock. Interestingly enough, even when the lever was dysfunctional, this particular rat still had a decreased stress response. Hence, a heightened stress response is more likely to occur when the organism feels like nothing can be done to better the situation at hand.

Extrapolating these findings to the human realm, a sense of control matters to us as well. Clearly, a white-collar worker on the verge of losing her job due to company downsizing would experience more distress than someone with a more secure position. For another example of the importance of control in the workplace, consider a study by Melin, Lundberg, Söderlund, and Grandqvist (1999) involving stress levels in Volvo

factory workers. While working on the monotonous assembly line, many workers experienced higher blood pressure and epinephrine levels. However, when Volvo implemented a more flexible work day, involving interchangeable jobs and more input from the workers, health improved and decreased levels of epinephrine and norepinephrine revealed that the subjects were able to unwind more rapidly after work. Hence, a sense of personal control helps us cope with stress by encouraging us to focus on the things we *can* positively change in our lives.

The presence of outlets

Next, outlets for frustration help us manage stress appropriately. To stick with the ongoing theme, shocked rat experiments help set the stage for an explanation of this stress-reducer. Weiss (1972) demonstrated that the stress response was less severe in rats that could run over to a block of wood and gnaw on it after receiving a shock. Other examples of effective outlets noted by Sapolsky (2004) include letting the stressed rat eat or drink, or exercise on a running wheel.

Humans also experience a decreased stress response when we have the opportunity to “escape” by doing something we enjoy, such as a favorite hobby. To cite one example, Anshel (1996) compared stress responses in a group of undergraduate men enrolled in a 10-week aerobic exercise program to the responses in a control group of undergraduate men. The acute stress that participants were subjected to consisted of "losing" against a competitor of the opposite sex on a motor task while receiving negative feedback about their performance. Subjects were tested before and after the exercise

program. Aerobic exercisers responded to the stressor with less negative affect (e.g., depression and anxiety), lower heart rate, reduced systolic blood pressure, and superior motor performance. It is important to note, though, that outlets should be something positive and enjoyable for each individual. Hence, not all of us will benefit from the same outlet. As stated by Sapolsky (2004), outlets best reduce frustration by reminding us that there is more to life than whatever happens to be stressing us out at the moment.

Social support

Lastly, let us consider the positive role that genuine social relationships plays in stress management. As previously discussed at the onset of the last chapter, baboons with more playmates and grooming buddies have lower cortisol levels than secluded individuals (Sapolsky, 2004). A supportive social network is equally important when it comes to human stress levels. For instance, a study by Miller, Gaudin, Zysk, and Chen (2009) investigated the link between parental support and childhood asthma, a disease characterized by swollen and narrow lung airways. Children with asthma and healthy children filled out questionnaires about how much parental support and understanding they received. Socioeconomic conditions, cigarette exposure, disease severity, and medication use were all controlled for. Wheezing and asthma were more likely to develop (and worsen) in children whose parents had high stress, or mood problems. Miller et al. (2009) investigated the physiological mechanism behind this issue. Originally, cortisol was not the expected culprit. After all, this stress hormone serves to *suppress* inflammation. However, these authors ultimately suggest that chronic stress

fosters resistance to the anti-inflammatory properties of glucocorticoids. In fact, stressful parent-child relations worsen asthma symptoms by decreasing the molecular expression of glucocorticoid receptors, making inflammation-inducing cytokines less inhibited by cortisol. Thus, strong relationships serve as a powerful defender against the trials of life.

Yet, we must refrain from concluding that the only way to reduce stress is to maximize a sense of predictability, control, outlets, and social connections. The relationships have some subtleties. We obviously cannot foresee everything, and it is dangerous to assume that we have more control than we actually do. For instance, if we cannot seem to get over a sickness, concluding that we failed to keep our attitude positive enough to stay healthy can instigate unnecessary stress, only making matters worse. Moreover, outlets for frustration become negative when innocent bystanders take the brunt of it. For example, as previously discussed at the onset of the last chapter, a high percentage of baboon aggression occurs when stressed-out dominant males beat up on subordinates. Other unhealthy outlets for frustration include fast food binges and substance abuse, just to illustrate a few human tendencies. Finally, not all social connections are helpful in stress management. If we surround ourselves with negative or overly-competitive friends, social connections will only build up stress levels. Thus, the four coping strategies discussed have un-ignorable caveats.

In order to put these four stress-reducing factors into context, I will use my time in the Dominican Republic as a case study of sorts. As previously mentioned in the introduction to this thesis, I had the amazing opportunity to spend one month living in

solidarity with the people of La Vereda during the summer of 2011. Staying with a host family opened my eyes to the plethora of struggles that poor, rural Dominicans face on a daily basis. They lived without basic amenities that we oftentimes take for granted, such as safe drinking water, health care access, and accessible medications.

Although I obviously could not measure glucocorticoid levels in my host family like Sapolsky does with his baboon troop, I could tell that control was seriously lacking in this community. For example, many parents like Gisela were genuinely concerned that their children would not have the necessary opportunities to escape a life of poverty. Families generally could not afford books, transportation to secondary schools, or tuition for private schools. Another example of this lack of control in this poor community involves the separation of families. Even though her husband and children were all citizens of the United States, my host mother's six-year effort to gain U.S. citizenship was not making any serious headway. Her family sometimes visited her, but she was lonely for the majority of the year. In fact, even though virtually everyone in La Vereda had relatives in the United States, none of them had great prospects of leaving the Dominican Republic.

Before setting foot in La Vereda, my prior research about the relationship between SES and stress biased me into thinking that the vast majority of the rural Dominicans would be noticeably stressed out. However, these genuinely happy people not only proved me wrong, but also helped me understand what I need to emphasize more in my life. Although the people of La Vereda certainly lacked control at times, they

primarily coped with their challenging circumstances by effectively utilizing outlets for frustration, social support, and open-mindedness.

First, I will always remember the dark, rainy night, when the house was completely full of spiders and flying ants. Whereas my American roommate and I wanted to camp out by ourselves under our mosquito nets, my host mom and sister turned up the music and started dancing meringue with us. Because dancing and joking around provided us with such a great outlet, our apprehensions about all the bugs began to fade away and our friendships deepened. Other positive outlets for frustration that I observed included nightly games of dominoes and cards. As a side note, though, many men in the community turned to alcohol consumption, an unhealthy outlet for frustration.

Nevertheless, one of the most important lessons I received from the Dominicans was the importance of a tight-knit community. Instead of withdrawing into themselves, the people there cooked together, worked together and visited each other's houses constantly. For instance, when we first arrived in La Vereda, we decided that we wanted to have the medical clinic inside the church and the dental clinic outside. It was amazing to see the Dominicans clear off a concrete platform adjacent to the church by hacking away at the foliage with their machetes. Then they resourcefully used wooden sticks and plastic tarps to construct an awesome, shaded dental clinic within one hour. They really amazed me in their ability to work together as a cohesive group in order to accomplish a common goal. Whereas many of us do not know our neighbors or acquaintances very well, the Dominicans formed strong social networks and sincerely cared about each other.

Another chief Dominican value that I have come to appreciate is open-mindedness. Instead of making overwhelming to-do lists or dwelling on challenges, they approached every day with an inspiring amount of acceptance. One of their favorite phrases was “Si D os quiere,” which translates to “If God wills it.” For instance, if we were to make plans to go swimming in the river after clinic, they would say this phrase, leaving room for unexpected changes in the plan. Although predictability tends to decrease stress, the phrase “Si D os quiere” illustrates the fact that deviations away from set plans should not always be looked down upon. Because the Dominicans were not obsessed about running out of time, they really seemed relaxed and happy. This flexibility and emphasis on simplicity most likely served to combat the lack of control in this community.

Thinking back upon my experiences in La Vereda, the Dominican mindset really reminds me of Reinhold Niebuhr’s Serenity Prayer, written in 1943: “God, give us grace to accept with serenity the things that cannot be changed, courage to change the things that should be changed, and the wisdom to distinguish the one from the other” (Qtd. in Sifton, 2003). Much insight lies within this well-known mantra of Alcoholics Anonymous, regardless of a person’s religious background. Although not all losses or challenges can be easily managed, we should take charge of the parts of our lives that we can improve, while simultaneously refraining from viewing the world as an inherently hostile place.

Hence, we cannot effectively manage stress without managing our personal perceptions of the world around us, no matter the extent of turmoil that we experience. This idea clearly finds expression in Dr. Victor Frankl's *Man's Search for Meaning* (1984), an insider story about the daily struggles in Nazi concentration camps during World War II. The first portion of Frankl's novel describes the unimaginable mental stress that such prisoners were submitted to. For instance, upon arrival to Auschwitz, only about 10 percent made it through the first selection process alive. These people lost all sense of human dignity and respect, suffered from physical ailments and intense undernourishment, knew that death could happen at any instant, and had no news of family members or when the war would end. As an illustration of the severity of camp life, Frankl describes how he refrained from waking fellow prisoners up from a horrible nightmare one night because "...no dream, no matter how horrible, could be as bad as the reality of the camp which surrounded us..." Clearly, these prisoners lived in one of the most stressful environments imaginable.

Yet, a few prisoners harnessed their inner freedom to advocate for the welfare of humankind: "We who lived in concentration camps can remember the [few] men who walked through the huts comforting others, giving away their last piece of bread...they offer sufficient proof that everything can be taken from a man but one thing: the last of the human freedoms—to choose one's attitude in any given set of circumstances, to choose one's own way." Instead of letting physical and mental stress push them into an animal-like existence, these martyrs made suffering meaningful by dying for their fellow man. Frankl applauds this inner triumph by stating, "It is this spiritual freedom—which

cannot be taken away—that makes life meaningful and purposeful.” Hence, as we experience stressful situations in our own lifetimes, one of the most important lessons we can take from Frankl is that our *interior* perception of stress makes all the difference in the world.

As mentioned in the last chapter, though, our current society has the tendency to rely too exclusively on *external* fixes for distress—on medications with unfavorable side effects and expensive appointments with psychiatrists—and not on our own natural potential for self-healing. Frankl reminds us that because man is self-determining, the choice is ours to make. In the words of Annie Dillard, we can either helplessly let our “wills flounder...under the crushing number of living things and stars,” or we can actively conquer the harmful health repercussions of chronic stress by always striving to react in a calm and collected manner. Although we obviously cannot always avoid adversity, we all are blessed with an ability to maintain composure in the midst of demanding circumstances.

As expressed in *Man’s Search for Meaning*, one manner in which to prevent stressful situations from dominating our lives involves a form of social support that we call love. Frankl’s love for his wife helps him overcome the awful mental and physical hardships he experienced as a prisoner. For instance, one morning during a cold march to the worksite, Frankl recalls his ability to escape from the prisoner’s existence by thinking about his wife. For the first time, he understood “...that love is the ultimate and highest goal to which man can aspire...The salvation of man is through love and in love.” He

goes on to say that "...a man who has nothing left in this world still may know bliss, be it only for a brief moment, in the contemplation of his beloved." It did not matter that Frankl was not sure if his wife was alive: "Love goes very far beyond the physical person of the beloved. It finds its deepest meaning in his spiritual being, his inner self." In these lines, we once again are reminded that the positive emotions associated with love can help mitigate chronic stress.

Another way to rise above the harmful effects of stress entails keeping future objectives in mind, as proposed in Frankl's novel. Without this hope for a meaningful life outside of the concentration camp, the plethora of oppressive physical and emotional stressors would stifle any sense of hope in the future. For instance, one night, when Frankl found himself worrying about "trivial things" like finding a piece of wire to replace his broken shoelace, he started to envision what he would do after his release: "Suddenly I saw myself standing on the platform of a well-lit, warm and pleasant lecture room. In front of me sat an attentive audience... [and] I was giving a lecture on the psychology of the concentration camp!" With this constructive visualization, Frankl knew that he could use his suffering to teach and encourage others.

Thus, not only does Frankl come to understand his future role as an innovative psychotherapist, but he also encourages his fellow prisoners to envision their own future purposes. For one man, "...it was his child whom he adored and who was waiting for him in a foreign country." Another man "...was a scientist and had written a series of books which still needed to be finished. His work could not be done by anyone else, any more

than another person could ever take the place of the father...” Because Frankl effectually inspires his fellow prisoners to find a personal “will to meaning,” he prevents all the stressors of the concentration camp from having the upper hand.

Frankl’s internally-derived coping strategies therefore make him an excellent role model for our purposes. According to psychologists Ryff and Singer (1996), we can learn a lot by studying the shared characteristics of effective stress-managers (like Frankl). Such individuals are great at developing a sense of self-directed purpose by setting and accomplishing goals, fostering deep connections with others, managing outside challenges and opportunities with composure, and possessing positive self-regard. Thus, what started out as a science-based thesis about the long-term effects of stress has ultimately turned into an inquiry into what is best and worst about the way in which we perceive the world around us.

In order to demonstrate the importance of perception, let us consider a recent National Public Radio (NPR) article about coping with stress, hardships, and aging. Dr. Mark Lachs (2011) writes about Helen Reichert. She unfortunately died soon after the debut of this article, just short of her 110th birthday. Not only did Reichert love chocolate truffles, Budweiser beer, and cigarettes, but she also experienced major stressors in her life, such as bereavement, gender discrimination, and medical issues. So how could this centenarian still be independent, healthy, and fully engaged in the world around her at 109 years old? Although her longevity can partially be attributed to genetics, Reichert’s ability to cope with stress stemmed from a trait called adaptive competence. Dr. Lachs

loosely defines adaptive competence as having a bright outlook despite the presence of psychological and physical stress.

Levy, Slade, and Kunkel (2002) suggest that a key factor in adaptive competence involves believing that the perceived benefits of one's life outweigh the perceived adversity. In this study, participants in their 50s were asked whether they concurred with statements like, "Things keep getting worse as I get older," and "As you get older you are less useful" (Lachs, 2011). Health and mortality were determined 23 years later. After controlling for age, functional health, gender, and socioeconomic status in this prospective study, seniors with positive self-perceptions of aging lived on average 7.5 years longer than those with less positive perceptions. Moreover, a strong will to live outweighed the influence of other variables that have been previously linked to survival (like gender, socioeconomic status, functional health, and loneliness). Thus, this study suggests that internally overcoming negative (and stress-inducing) stereotypes about the aging process can considerably lengthen a lifespan. Similar to Frankl, therefore, these authors underscore how imperative our internal perceptions are in lessening the negative health effects of an overused fight-or-flight response.

In fact, if we simply put aside 12 to 15 minutes per day to elicit the "relaxation response," a term coined by Dr. Herbert Benson of Harvard Medical School in the 1970s, we can use our mind to noticeably diminish stress-related health consequences, supplement the use of established drugs or surgical procedures, and prevent symptoms from arising in the first place (Benson, 2000). Physiologically speaking, the relaxation

response is the polar opposite of the fight-or-flight response. Activity of the sympathetic nervous system (SNS), oxygen consumption, carbon dioxide emission, blood pressure, heart rate, and respiratory rate are all reduced during this form of peaceful meditation, helping our bodies to effectively reestablish a healthy allostatic balance (Benson, 2000). This technique also fosters calmer brain activity and a sense of emotional well-being.

The relaxation response is not just another fleeting self-help fad, though. Instead, many cultures and religions throughout history have encouraged this internal focus devoid of external thoughts, ranging anywhere from Christian mystics to Buddhist monks (Benson, 2000). To name one literary example involving the relaxation response, consider Lines 42-50 of William Wordsworth's *Tintern Abbey* (1798): "...that serene and blessed mood,/ In which...the breath of this corporeal frame,/ And even the motion of our human blood,/ Almost suspended, we are laid asleep/ In body, and become a living soul:/ While with an eye made quiet by the power/ Of harmony, and the deep power of joy,/ We see into the life of things" (Qtd. in Benson, 2000). While tranquilly taking in the natural beauty of his surroundings, Wordsworth poetically describes key changes associated with the relaxation response (such as a calm mind and a decreased respiration rate and blood pressure).

So how can we reap the age-old benefits of the relaxation response? Dr. Benson starts off *Relaxation Revolution* (2010) with an overview of this process. The first step involves deciding upon a personally-meaningful focus word, phrase, image, or short prayer. In a quiet, comfortable place (usually with the eyes closed), this focus word or

phrase should be envisioned or said with each slow exhale. Lastly, Dr. Benson underscores the importance of passively dismissing any outside thoughts that may distract us. This response can be elicited while walking, jogging, practicing yoga, or sitting on an airplane. As long as the relaxation response is consistently exercised for just 12 to 15 minutes per day, the scientific data speaks well for Benson's technique. For instance, when subjects with high blood pressure practiced the relaxation response for six weeks, the average systolic blood pressure in the participants decreased from 146 millimeters of mercury to 137 (Benson, 2000). This matters because hypertension often leads to strokes and heart attacks.

Interestingly enough, the relaxation response even plays a significant role in the expression or activation of certain health-related genes. A groundbreaking study by Dusek et al. (2008) compared genetic expression in a group of 19 expert mind body practitioners (with an average of 9.4 years of practice) to a group of 19 closely matched controls with no prior experience in the relaxation response. The matching characteristics included similarities in age, race, gender, height, weight, and marital status. The latest "microarray analysis" technology was employed to check the activity of all 54,000 genes in the participants. 2,209 genes in the experienced practitioners were expressed differently than the same genes in the inexperienced group. The chances of this happening due to chance alone were less than five in 100. It was determined that for the inexperienced group, the dissimilar genetic activation was associated with stress-related medical problems like detrimental regulation of immune responses and premature aging.

Next, these researchers spent 8 weeks teaching the inexperienced group how to enter the relaxation response state. Upon completion of training, genetic analysis was performed for a second time. 1,561 genes changed in expression from the first test to the second. Once again, the chances of this being a random event were less than five in 100. Furthermore, after the relaxation training, 433 new similarities in genetic expression became evident between the experienced meditators (9.4 years of practice) and the group with short-term practice. These gene signatures were associated with important health benefits for both groups. Because the probability that such genetic changes happened by chance in both parts of the experiment was less than one in 10 billion (Benson, 2010), we can safely say that the relaxation response alters the manner in which genes interact or express themselves.

Hence, now more than ever, scientists are starting to understand the previously dismissed links between mind and body. With a little bit of self-awareness, we all have the ability to promote health when life puts us to the test. Not only should we eliminate optional stressors in our lives, but we should also save the stress response for what it was evolved for—namely, surviving life-threatening situations. After considering the cumulative damage that excessive stress can incite on our telomeres and blood pressure, stress reduction is not something to be put off for another day.

Meanwhile, the medical community should continue to pay attention to symptoms of stress-related allostatic load by measuring blood pressure, waist-to-hip ratio, blood cholesterol, glucose metabolism over a period of days, and levels of stress hormones like

cortisol and norepinephrine in overnight urine (McEwen, 2002). This could prevent heart disease and diabetes in people that show no symptoms or family history. Although hospital stress reduction and wellness programs are a good first start, health care providers need to fully explain the importance of allostatic load reducers like restful sleep, a balanced diet, and regular exercise (McEwen, 2002). Moreover, scientists should continue to investigate how the mind and body function as a unit, and the role stress plays in that relationship. Because complexity and additional stress are added to our lives with each generation, such research will be imperative.

To end on a positive note, maybe we need to try and *redeem* stressful situations for what they are, as opposed to letting them get the best of us. This idea finds expression when Annie Dillard quotes Martin Buber: “God entrusts and allots to everyone an area to redeem: this creased and feeble life, ‘the [stressful] world in which you live, just as it is and not otherwise.’ ...Here and now...an ordinary person would approach with a holy and compassionate intention ...the car pool...the retirement account. ‘Insofar as he cultivates and enjoys them in holiness...and in holiness reflects upon his business, through him...the worlds which have fallen will be delivered and renewed.” Here, Dillard offers a few important pieces of advice. First, we should accept unfortunate events for what they are, instead of ignoring or dismissing them. Moreover, instead of chronically stressing out about car pools and retirement funds, we ought to approach them with a positive outlook. Thus, as we strive to deal with everything in our busy and sometimes hectic lives, the way in which we react to experiences—both stressful and joyful—really matters for our long term health and happiness.

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