

Importance of a Weight-Gain-First Strategy when Recovering from Anorexia Nervosa

Sarah Douglas

Milligan College

Abstract

Key words: anorexia nervosa, weight gain, recovery, malnutrition, misconceptions

This research paper chronicles the imperative need for people recovering from anorexia nervosa to gain weight quickly. Many treatment methods wait for the patient to heal the mind before putting on weight, but malnutrition disallows correct brain function. Therefore, without significant weight gain, healing of the mind simply is not possible. Additionally, the weight gain cannot be restricted nor predicted by a scale. Bodies all work differently, especially bodies that were starved for extended periods of time. The research contained in this paper argues for trusting the body to gain the necessary weight, uninterrupted by doctors' scales or charts. The paper starts by defining anorexia nervosa and corrects misconceptions. It also details the many physical effects detrimental to the patient's health, defends why weight recovery should be the primary focus, describes old approaches to weight restoration, and warns of the challenges that weight gain will bring. The paper engages a wide range of research to advance for fast and unrestricted weight gain.

Anorexia nervosa (AN)—this stigmatized disease evokes images of starved adolescent girls struggling to fit in at school or to control their changing lives and bodies. Though these assumed motivations can precipitate the illness, anorexia nervosa’s roots lie in brain abnormalities within the starving girls. Anorexia nervosa is no minor growing pain. AN continuously torments its victims, leading to a lifelong struggle and often, tragically, to an early grave. Between the ages of fifteen and twenty four, an eating disorder (ED) is twelve times more deadly than all other leading causes of death combined for that age group, including automobile incidents (Olwyn, 2012). Nevertheless, the illness confounds society. Society minimizes the threat of AN by explaining the disease as merely a diet gone haywire. Uninformed outsiders suggest for sufferers to simply “just eat,” but the road to recovery proves to be about more than simply calories and exercise. Confusion about anorexia nervosa’s grip on victims leads to caretakers becoming more frustrated, and the sick becoming sicker. James Lock, an eating disorder researcher working at Stanford University, describes this problem, “[o]ne of the biggest problems is that people do not take this disease seriously” (as cited in Rosen, 2013, p. 2). He compares eating disorder recovery to cancer treatment. “If the [cancer] treatment is hard parents still do it because they know they need to do it to make their child well... (Rosen, p. 2).” He continues to expand on this idea. Just as a cancer patient cannot choose to grow malignant tumors, a person with AN does not simply choose to not eat (Rosen). Treating anorexia nervosa requires compassionate strategies that recognize that a person with anorexia nervosa simply is unable to “just eat.” Additionally, effective treatments must discourage oversimplifications and societal stereotypes about anorexia nervosa and its victims.

The Diagnostic and Statistical Manual of Mental Disorders 5 (DSM-5, 2013) categorizes anorexia nervosa as a mental disorder with certain criteria. For one, anorexia nervosa represents

a continuous restriction of calories in order to significantly lower body weight. The person with AN must experience either an intense fear of putting on weight or implement odd routines in order to deter weight gain. These measures exist regardless of whether or not the person already has a low body weight. Also, there usually is a distortion in how the person with AN sees himself/herself in the mirror. The person's perception of his/her body carries a disturbing amount of power to sway his/her self-image and confidence. Finally, the person with AN fails to recognize the seriousness of his/her starved or underweight body (Olwyn, 2012). This checklist in the DSM-5 coincides easily with the societal viewpoint of anorexia nervosa. However, like society, the DSM-5 understates the top priority; AN is a mental illness defined by thoughts, rather than body changes. This leads to people confusing the physical symptom of weight loss with the actual mental disorder. It is imperative to realize that people can struggle with AN at unsterotypical weights. People with atypical AN have lost a noticeable amount of weight but are not underweight. These people with atypical AN tend to be forgotten or minimized because they are not the shocking, emaciated depictions on TLC specials. The DSM-5 describes atypical AN: "all of the criteria for anorexia nervosa are met, except that despite significant weight loss, the individual's weight is within or above the normal range (as cited in Sawyer, Whitelaw, Grange, Yeo, & Hughes 2016, p. 1-2)." Unfortunately, those with atypical anorexia receive diagnoses too late or entirely miss the care needed because of the weight bias society associates with AN (Sawyer et al.).

In addition to comprehending that AN cannot be simplified to only include emaciated people, another imperative realization is that the causes of anorexia nervosa are not as diverse as assumed. People imagine that eating disorders stem from playground bullies, fashion models, or abusive parents. However, not just anybody can develop an eating disorder, and they do not do

so solely in order to fit into cultural beauty ideals. This confused association has been around since medieval times. Although previously people did not assume eating disorders were caused by trying to achieve an hourglass figure, they did presume that people were restricting food in order to purify themselves and become holier (Guisinger, 2003). Self-starvation to the point of death requires a stronger drive than average and a larger motivation than beauty or holiness. The causes of anorexia nervosa lie in brain adaptations that helped our wandering ancestors survive famines and hardships.

Understanding the roots of anorexia nervosa plays a pivotal role in helping sufferers recover from the disease. Caretakers must dismiss common misconceptions such as the assumption that a person needs to be severely underweight in order to necessitate treatment. They must also realize that one of the reasons people with AN have such extraordinary difficulty recovering is because their brains are uniquely wired to starve themselves. Thus, directing the patient to “just eat” cannot produce lasting results. However, eating does represent the sole path to remission.

In order to battle the monster within the victim, the patient must learn to rip away the layers of terror, malnutrition, and pain. The first step in stripping away these layers is to eat without restriction. This weight-gain-first approach is supported by experiments such as the Minnesota Starvation Study (1950) and fMRI imaging research. Experiments, research, and the dangers of malnutrition all support the strategy of weight gain first, superseding gradual weight gain strategies.

In anorexia nervosa, the sufferer literally eats his/her body from the imperceptible internal organs to the readily visible fat. The consequences of such self-cannibalism are devastating on every system of the body. Perhaps the most dramatic effects occur in the

endocrine, reproductive, cardiovascular, and skeletal systems. Most of these symptoms mitigate with proper nutrition, supporting the need for weight gain as soon as possible in order to minimize further damage and heal. What follows are descriptions of some of the dangerous and destructive effects on these four systems.

The endocrine system adapts to the shortage of food and produces changes in order to keep the starving patient alive. The cost of sustaining life through these measures is adverse side effects. AN represents an incredibly stressful disorder. The person struggles every day to resist consuming the one thing he/she needs. The daily battle represents psychological torment. The malnutrition also stresses the physical body. Outsiders try to help the victim, but their help only stresses the sufferer further because he/she does not want food forced upon him. Unsurprisingly, the person with AN has an influx of the stress hormone corticotropin releasing hormone, leading to an increase of cortisol in the blood (hypercortisolemia). Scientists hypothesize that this adaptive behavior of the body attempts to keep blood glucose levels within a normal range when glucose cannot be normalized by food (Baskaran, Misra, & Klibanski, 2017).

The endocrine system must also alter metabolism in order to slow the body's process of eating away its own fats, muscles, and organs when food is absent. Changes in the thyroid hormone produce two hallmarks of anorexia nervosa. The overwhelming, bitter cold constantly felt by the patient represents an uncomfortable but lifesaving response to starvation. Hypothermia within the body allows the body to burn fewer calories. Though necessary for survival, many former sufferers shudder to remember the unique misery anorexia cold brought to them. No amount of blankets could thwart the visceral coldness they experienced. Yet, torment of the cold wanes through nutritional rehabilitation, signifying a motivation to eat in order to warm.

In addition to lowering body temperature, the thyroid also slows the heart in order to conserve energy. A slow heart rate, bradycardia, can be confusing. Athletes tend to have slower heart rates because their hearts become very efficient. People with anorexia nervosa can use their bradycardia to argue that they are healthy. In reality, bradycardia represents another way the overtaxed body is struggling to survive in the midst of a self-inflicted famine and a weakened heart muscle (Baskaran et al., 2017). Bradycardia can be caused by an overactive parasympathetic nervous system in order to conserve energy output (McCallum Place, 2015).

Lesser known hormones of the endocrine system play important roles in attempting to preserve the life of the person with AN. In healthy humans, another product of the endocrine system, leptin, regulates the appetite by letting the brain know when it has gotten sufficient nutrients. In people with anorexia nervosa, leptin levels unsurprisingly dip. These lower levels of leptin might lead to hypogonadism and impaired bone health (Baskaran et al., 2017). Another hormone, ghrelin, which secretes growth hormone, remains at higher levels in AN. The higher levels reduce gluconeogenesis (creation of glucose) and lipolysis (destruction of fat cells) in order to preserve energy and euglycemia (normal glucose levels in blood). In healthy people, higher growth hormone levels help propagate osteoblasts, but this effect has not been observed in AN which suggests a sort of ghrelin resistance (Baskaran et al.). All of these adaptations to low energy intake prove to be imperative to the patient's survival. However, one hormonal outlier, peptide YY, opposes the body's adaptations. Peptide YY lowers the appetite in healthy people. For some unknown reason, peptide YY levels increase in AN which can lead to reduced food intake (Baskaran et al.). The body tries to help the weakening body survive, but the brain remains sick which can cause maladaptive responses, such as that caused by peptide YY. Most of these

harmful responses by the body do mitigate with nutritional rehabilitation, but it remains imperative to recover quickly to minimize further damage (Baskaran et al.).

Hormones involved with reproduction do not evade the far-reaching changes of the endocrine system. A starving body has little interest in devoting energy to producing offspring. Therefore, the reproductive system of the person with AN commonly tries to shut down. For example, luteinizing hormone, which helps with ovulation, does not go back to average levels as long as the body perceives that starvation is occurring (Baskaran et al., 2017). Hypoestrogenism runs rampant in females with AN, causing amenorrhea, absence of menstruation (Meczekalski, Katulski, Czyzyk, Podfigurna-Stopa, & Maciejewska-Jeske, 2014). Among other effects discussed later in this paper, amenorrhea changes cognitive functions including mental health (Baskaran et al.). The body attempts to alleviate the repercussions of the endocrine system's reproductive hormonal changes by reducing libido. The person with anorexia nervosa has little appetite for either food or sex (NYU Steinhardt).

Women and girls with AN strongly feel the repercussions of skeletal system changes because of their hormone make-up and their reproductive system design. Additionally, the female reproductive system also has an exaggerated response to the starvation. The female body can barely sustain itself on a restricted starvation diet. It is therefore not surprising that the female reproductive system recognizes that it could not possibly support another developing life. Weight loss, increased stress, and too much exercise all can contribute to functional hypothalamic amenorrhea (Meczekalski et al., 2014). The loss of the period does not just hinder reproduction temporarily. The effects of amenorrhea can cause infertility and anovulation in the future, along with skeletal, cardiovascular, and mental problems, affecting the woman throughout her lifespan (Meczekalski et al.).

In pre-pubescent girls, functional hypothalamic amenorrhea causes delayed menarche, dyschronic puberty, and underdevelopment of secondary and tertiary sex characteristics. In women who develop functional hypothalamic amenorrhea, dyschronic changes in urogenital mucosa and muscles of the uterus can cause increased infection rates and functional difficulties. In both age groups amenorrhea increases the risk of miscarriages, pre-term labor occurrences, impaired weight gain, and compromised intrauterine fetal growth (Meczekalski et al., 2014). Hypoestrogenism also interferes with normal heart function through noted endothelial dysfunction, abnormal bioactivity of nitric acid, abnormal autonomic function, changes in the rennin-angiotensin system, and lipid profile changes (Meczekalski et al.). Finally, losing the natural process of menstruation is associated with an increased risk of mental problems. Among these disturbances are more dysfunctional attitudes, problems dealing with stress, sexual problems, and interpersonal dependence (Meczekalski et al.). These differences can be explained by changes in the neuropeptides, neurotransmitters, and neurosteroid activity. (Meczekalski et al.). In these women, anorexia nervosa does not only severely damage mental and physical health, but also it jeopardizes their future dreams of having families and lives without anorexia nervosa.

Many of the emergency measures the endocrine system initiates have catastrophic long-term consequences on the body. One of the most dangerous of these proves to be loss of bone density. Propeptide of type one collagen (bone formation marker) and N-telopeptide (bone reabsorption marker) stay at low levels (Baskaran et al., 2017). The lowering of these hormones means low bone turnover commonly at a time (adolescence) when healthy growth needs to happen (Baskaran et al.). Throughout the illness, the bone configuration continues to worsen. Tragically, even with refeeding, the body cannot always repair the damage. Low bone density

haunts many people with AN for the entirety of their lives. Such bone loss underscores the importance of refeeding quickly in order to minimize the irreparable impact on bone health (Baskaran et al.).

Many other endocrine changes compound the damage to the skeletal system. Among other factors, hypothalamic hypogonadism, hypercortisolemia, low IGF-1 levels, and estrogen deficiencies lead to increased bone resorption and may lead to a lower bone mass density (Baskaran et al., 2017). Peak bone density/mass (PBM) represents the most amount of osseous tissue a human can acquire throughout his/her lifetime. Deficits in PBM can lead to osteopenia. If osteopenia worsens without any change in diet, the condition will escalate into osteoporosis. Both of these conditions deteriorate the bones, but osteoporosis proves irreversible (Meczekalski et al., 2014). In general, osteoporosis endangers even healthy women at a higher frequency than men. Statistically, the vast majority of people with AN are women, so these skeletal changes represent an even more enormous risk (Meczekalski et al.).

In addition to the structural quality of the bones being damaged by starvation, the growth of the skeletal system in younger people alters the programmed stature of the patient. In the 1990's, pre-menarche females with AN entered a study in which researchers collected data on their age of menarche, peak heights, and the ages of their peak heights. The researchers concluded that, when presenting with AN, the girls did demonstrate growth retardation, but, with proper nutrition and recovery, they could catch up to normal levels. However, unfortunately, the girls could never reach their full growth potential. This conclusion remains true even when the girls presented AN before or in the middle of their puberty. The researchers emphasized the need to nourish people with AN early in order to reduce the risk of relative growth retardation (Lantzouni, Frank, Golden, & Shenker, 2002).

Restoring weight and proper nutrition are the capstones in returning to health after AN. The skeletal system typifies one of the biggest examples as to why weight restoration proves so imperative. Osteopenia and osteoporosis have increased occurrence in the lumbar spine in adolescent girls (Baskaran et al., 2017). Women need the pivotal adolescent years to achieve strong bones because menopause greatly reduces bone mass. However, even with beginning refeeding, AN's high relapse rate deters the catch up to normal bone growth. The extent to which the person needs to repair heavily depends upon two factors: the length of the illness and nutrition status (Misra et al., 2004). Baskaran et al. noted a study that concluded that bone mass may not ever go to normal levels even after weight restoration in young adolescents with AN. This again underlines the need to reduce the extent of the damage of the skeletal system by gaining weight quickly.

Although the effects on the skeletal system are far-reaching, anorexia nervosa's effects on the heart represent its greatest danger. In anorexia nervosa, the key reasons for death prove to be suicide and cardiac events (Misra et al., 2004). For this reason, researchers have taken an interest in conducting studies to explore the inner workings of the heart in patients with anorexia nervosa. One hundred and eighteen girls (sixty with AN, fifty-eight with good health) entered a study in order to measure some key points. Most of these points regarded the skeletal system, but the study revealed that, for many girls, blood pressure and heart rate were significantly lower than their healthy counterparts. Though this response was already noted in the discussion of endocrine system changes, the Misra study found that weight gain (especially fat mass) and nutrition help normalize blood pressure and heart rate. Therefore, gaining weight quickly saves lives by saving the heart (Misra et al.).

In addition to the bradycardia and low heart rate already discussed, people with AN experience dangers such as decreased myocardial contractility and impaired left ventricle function. These shifts in heart functions plague sufferers and represent an enormous risk to health (Misra et al., 2004). Electrolyte abnormalities, the most common one being hypokalemia (low blood potassium levels), also create risks. Causes of hypokalemia can be low food intake, purging, or abuse of diuretics and laxatives. Acute hypokalemia can cause cardiac arrhythmias, a very common cause of death for people struggling with AN. Chronic hypokalemia, however, can cause glomerular sclerosis (hardening of the capillaries in the nephrons of the kidneys), interstitial nephritis (a type of kidney inflammation), and tubular atrophy (dismantling of tubes) (Baskaran, 2017).

Studies and research have addressed the dangers of starvation on specific systems of the body. However, even the way the body deals with the little nutritional intake it receives proves dangerous. When starving, the body loses its subcutaneous fat. However, those with AN have an abnormally large amount of visceral fat. Visceral fat represents a larger danger than subcutaneous fat because it envelops the organs and causes damage to them. A study concluded that another reason gaining weight quickly is important is because those recovering gain the better subcutaneous fat while losing the dangerous visceral fat (Zamboni et al., 1997).

The fat types bring up another concern in the dangers of AN. Many people assume that the skinnier the sufferer becomes, the more danger he faces. Though this assumption does carry some logic, caretakers must not forget those with atypical anorexia. In a study of two hundred and fifty-six adolescent girls, sixteen percent with atypical anorexia and forty-six percent with full-threshold AN, researchers discovered that there were fewer differences between atypical and typical types of AN than expected. Those with atypical anorexia nervosa generally shared typical

ED symptoms such as restricting, worrying about eating, and worrying about their shape and weight. They also showed a lower self-esteem than those with typical AN. The study also noted little-to-no differences between occurrence of bingeing, purging, and using laxatives and over exercise between atypical and typical cases of AN. Thus, the study found physical and psychological morbidity of adolescent atypical anorexia versus full-threshold AN to be virtually the same (Sawyer et al., 2016).

Considering the dangers discussed, outsiders observing anorexia nervosa become confounded as to why these people would do this to themselves. People with AN deny themselves food and torture themselves with routines such as vomiting or over exercising. In return, they can gain grotesquely skinny frames and cause irreparable damage or death to their bodies. Assumed motivations such as a desire for thinness or control do not seem powerful enough to cause such self-inflicted misery. There has to be a stronger motivation—something originating in the person with AN's own brainwiring.

Not just anybody perusing a Victoria Secret catalog becomes vulnerable to AN. Genetic heritability links up with fifty to eighty percent of the risk of fostering AN (Kaye, Wierenga, Bailer, Simmons, & Bischoff-Grethe, 2013). The preprogrammed behaviors remain dormant until something sparks the desire to restrict calories. The restriction of calories creates an energy deficit, which in those with a genetic predisposition for anorexia nervosa, leads to the brain to become fully obsessed with starving itself. In essence, the Victoria's Secret catalog could spark AN, but only if the person already has the genetic brain wiring required to fully participate in the disease (Olwyn, 2012).

One pressing question remains. Why did humans develop the capacity to starve themselves? What possible benefits could anorexia nervosa offer to the person it inhabits? The

answer lies in the past, when humans needed to travel long distances to find food and when famines were constant possibilities in life. Having the ability to survive prolonged deprivation of food actually increased a person's probability of surviving famine conditions. Our human ancestors with anorexia nervosa were the ones able to push through long walks or physical labor with very little food. They could potentially lead the people to a food source with enthusiasm because of the brain wiring driving them to thrive (temporarily and superficially) on little nutrition (Guisinger, 2003). No longer is the West in the era of low food sources. The condition that once helped humans survive in the wild now presents life-threatening perils.

The brains of many of our ancestors adopted AN to be able to go through great periods of famine. Their brains did this by adapting fundamental brain processes. The reward centers of an AN brain greatly enhance the ability to inflict self-starvation. For example, increased 5-HT_{1A} compared to decreased 5-HT_{2A} could produce hyperpolarizing effects on prefrontal neurons in AN (Kaye et al., 2013). This anomaly involves changes in regulating anxiety, controlling impulses, and accepting new environments (Kaye et al.). The 5-HT (serotonin) systems also might affect abnormal satiety, impulse control, and mood regulation (Kaye et al.). The atypical changes in the DA (dopamine) system might alter the rewarding effects of food, motivation, and executive function (Kaye et al.). "... AN may have an imbalance in information processing with impaired ability to identify the emotional significance of a stimulus, but increased traffic in neurocircuits concerned with planning and consequences, leading to heightened anxiety. This over-reliance on executive brain circuits involved in linking action to outcome may constitute an attempt at 'strategic' (as opposed to hedonic) means of responding to reward stimuli. These studies [discussed later] provide further evidence that exaggerated dorsal executive function is associated with enhanced inhibition and may reflect a possible means of modulating anxiety in

AN” (Kaye et al., p. 8). The brain of a person with AN rewards the victim for participating in the behavior that slowly kills him.

The brain’s reward centers form a central motivation for people with AN to self-starve. In the healthy brain, eating produces a dopamine response. The non-anorexic brain rewards its owner for participating in the vital life process of eating. Ursula Bailer, a psychiatrist and neuroimaging researcher, decided to test if people with anorexia experience the same joy felt by the dopamine release. Bailer gave amphetamine (to produce a dopaminergic response) to healthy people and to people with anorexia. Predictably, healthy people became much happier with the dopamine flooding their system. However, Bailer attested “[p]eople with anorexia didn’t feel euphoria--they got anxious” (as cited in Rosen, 2013, on pages 4-5). Additionally, the more the dopamine circulated, the more anxious people with anorexia felt. Dopamine, to people with AN, did not make them feel happy. People with AN felt anxious from the effects of dopamine. Compounding this aversion to dopamine, researchers have also discovered a self-rewarding aspect to starvation for people with AN. Dr. Walter Kaye, an ED researcher who has made great strides in the field, states that restricting calories and a limit on diet produce an anxiety-reducing effect, “[w]hereas food consumption stimulates dysphoric mood” (2013, p. 3). So the brain of a person with AN brain feels stressed by eating and comforted by starvation. Yet, anxiety avoidance represents only one factor in the motivation for people with AN to starve themselves.

Many other abnormalities present in people with AN-gearred brains. Changed anterior insula activity involves interpreting interspective information (taste, pain, hunger) which completely and physiologically impairs how someone with AN experiences the world. Dr. Walter Kaye conducted research on the insulas (in charge of recognizing pain, taste, and other sensations) of healthy and AN-ridden brains. He used heat-delivering devices in order to produce

a painful heat on the arms of the participants. He found that the insulas of those with AN dimmed in activity with the heat but, when they were warned of the coming heat first, other active regions of the brain lit up. Kaye concluded, “They don’t seem to be sensing things correctly... [This] may help us understand why people can starve themselves and not get hungry” (as cited in Rosen, 2013, on page 5). “Some studies suggest that just a taste of sugar can send parts of the brain barreling into overdrive. Other brain areas appear numb to tastes--and even sensations such as pain. For people with anorexia, a sharp pang of hunger might register instead as a dull thud” (Rosen, p. 1). People with AN become internally rewarded by starving and do not feel the uncomfortable effects of starvation as strongly as a healthy person, but they also do not recognize the toll that anorexia has on the body. Anomalies due to flawed emotional and social awareness can cause a person to perceive body image incongruent to reality and to be ignorant to recognizing a malnourished state (Kaye et al., 2013).

In his studies, Walter Kaye revealed that “... [F]ood is an inherently emotional stimulus in AN...” (2013, p. 7). The AN brain can dull the sensations of pain and hunger, but the brain creates fear and anxiety—emotions the brain strives to control. Samantha Brooks conducted an fMRI study in order to measure and map brain activity. She scanned the brains of volunteers as they pictured themselves consuming high-calorie foods. In women who restricted calories, the self-control parts of the brain activated when the volunteers thought about food. She concluded that “[this response] put a brake on [their] impulsive behavior” (as cited in Rosen, 2013, p. 3). Brooks also created another test with colleagues to test the working memory of people with AN versus healthy eaters. “The [AN] patients were really good... they hardly made any mistakes” (p. 3). Working memory plays a role on holding onto self-imposed rules. “It’s like saying ‘I will only eat a salad at noon. I will only eat a salad at noon,’ over and over in your mind,” Brooks

explained (as cited in Rosen, p. 3). During the questions to test working memory, experimenters sought to discover what effect introducing food to the test-takers would have. When confronted by food, people with AN engaged their working memories to become distracted, and their brains tapped into the ever-prevalent self-control centers of their brains (Rosen).

The self-control exhibited by people with AN can make them want to avoid discomfort more than enjoy the possibility of indulging. Kaye (2013) described this as follows: “We hypothesize that AN individuals have a temperament skewed towards aversive or inhibitory response, rather than reward and motivation” (p. 5). The brain continuously makes it harder for people with AN to eat or even desire to eat. Eating evokes fear; restricting offers relief; hunger signals become nullified. Additionally, taste does not even offer a powerful reward. For example, Kaye discussed a study of someone who recovered from AN’s response to the taste of sucrose mixed with water. Those who recovered showed reduced neural activation that does not correspond with joy as it did from control subjects. “Thus, the set-point may mimic a continuous state of satiety in individuals with AN that limits introspective and reward processing” (Kaye et al., p. 6). These abnormal introspective responses to hunger, pain, and taste contribute to AN. There is a terrible cycle that propels those with AN to experience fear with food, and relief with restriction. Meanwhile, people with AN develop an uncanny ability to ignore their bodies’ cries for relief.

Energy deficits from restricting calories often act as the catalyst for the brain of someone predisposed to AN to impose a downward spiral to anorexia nervosa. Nevertheless, since AN is a neurobiological condition, people with the capacity to starve themselves commonly show certain characteristics premorbid to the illness. Some of these idiosyncrasies include perfectionism, impulse control, ability to delay rewards, anxiety, obsessive behaviors, and inflexibility (Kaye et

al., 2013). These character traits allow someone whose brain is prewired for anorexia nervosa to sustain AN. For example, perfectionism heightens the response to negative outcomes yet lessens the response to achievement or reward. The eyes of a perfectionist are tailored to always seek improvement which, in someone with AN's viewpoint, would be a skinny frame. People with AN often are star pupils. In Rosen's article (2013), James Lock described this phenomenon: "It's very rare for me to see a person with AN in my office who isn't a straight-A student... They will work and work and work... The problem is they don't know when to stop" (p. 2). Typically, when someone with AN has a self-imposed goal, he can discipline himself to achieve it, forgetting the discomfort it may bring. In addition to perfectionism, the other personality characteristic listed doom the person with the biological markers for AN to excel at destroying himself.

The core of anorexia nervosa resides in the brain. The physical and mental toll AN takes on its victim make recovery imperative for life. However, fighting anorexia nervosa proves long, difficult, and messy. When people with anorexia nervosa finally become desperate enough to recover, the process of recovery can appear as sick as the actual disorder. Thus, the recovery process has created confusion and judgment towards the ill.

Mistreatment and distrust of people with AN is nothing new. In medieval days, people striving for holiness placed importance on fasting. Sometimes, this holy endeavor was propagated by AN's hold on the brain. Take, for instance, Saint Catherine of Sienna. Catherine was one of more than eighty-five, very skinny saints, blessed, or venerables with a seemingly "miraculous" talent of surviving off a minuscule amount of food (Guisinger, 2003). Saint Catherine acted out her service of starvation in the late fourteenth century. Her devout fasting transformed into something harmful. At first, she hardly ate anything except bread, uncooked

vegetables, and water, but, after her father died and a vision from God occurred, she soon gave up the bread. Furthermore, after another vision from God, Catherine came to the conclusion that she “no longer had need of food and no longer could digest” (Bell, 1987, p. 25). Soon the church’s respect for Catherine’s fasting turned into suspicion and concern. She faced accusations of participating in witchcraft, and condemnation for not eating as much as Jesus did in the New Testament (such as the instances in which Jesus ate with sinners). These accusations and efforts to explain her AN only perpetuated the illness. Catherine attested to the fact that she felt livelier and healthier when she starved herself. She admitted she would rather die of starvation than have to eat. Catherine never formally attempted to enter recovery, yet she felt the repercussions of people’s misunderstandings of her illness (Bell). When other saints did attempt to recover and felt the urges recovery brings, they suffered cruel and extreme punishments.

Another historic example reflects the reactions to a person with AN’s strange stage of recovery—extreme hunger. “In the time that she made her rigorous fast of five years... the sisters sometimes found Sister Veronika in the kitchen, the refectory, or the dispensary, where she ate everything there was, and what is more, other times they found her eating before the hour of Communion, and then they saw her come to communion with the others. From this there derived great confusion and backbiting to the discredit of this Servant of God, but later it became clear, that in effect what appeared under the aspect of Sister Veronika was the Devil” (Abbess Ceoli) (Bell, 1987, p. 75). Recovery binges plague the recovering person with anorexia nervosa. For so long, the anorexia nervosa deprived the body of thousands and thousands of calories for however long the restriction lasted. It is therefore understandable that the recovering body will overeat in order to compensate for the extreme undereating experienced. Researches and ED specialists now understand that extreme hunger and calorie consumption are a necessary part of

recovery. Medieval nunneries did not. In order to punish those like Sister Veronika for partaking in a normal and extremely necessary part of recovery, church officials concocted tortuous punishments. Some of the actual punishments for bingeing included being forced to eat cat vomit, clumps of hair, fat leeches filled with blood, bugs, and worms. Other punishments that took place entailed licking the entire pavement and walls of an infirmary while swallowing the spiders and webs found on those surfaces. Some chose physical abuse such as forcefully kicking the ill in the mouth. Others chose spiritual abuse like forcing the person in recovery to refuse communion (Bell).

Even now, in modern times, innumerable misconceptions surround anorexia nervosa, misconceptions which span from the initial cause of the illness to the recovery process. Early attempts to understand the illness led to erroneous conclusions that persist even today. In 1874, Charles Lasègue concluded that the family of a person with AN proved responsible for the disease manifesting (Bell, 1987). Later, in the twentieth century, Sigmund Freud speculated that “the wellknown anorexia nervosa of girls seems to be a melancholia occurring where sexuality is underdeveloped” (Guisinger, 2003, p. 746). Thanks to modern research into brain data, the validity of these earlier hypotheses cannot be sustained as the sole cause for the disease. Perhaps the most important anorexia-nervosa-related research focuses not on the causes of AN, but on its cure.

One such study sought to answer the question of how to most safely and effectively refeed starving people. On November 19, 1944, thirty-six healthy men participated in the Minnesota Starvation Study. During World War II, hardship, famine, and brutality led to a need to understand how to rehabilitate starving people most efficiently. Ancel Keys created the Minnesota Starvation Study, starving perfectly healthy men and then refeeding them to find the

best method of re-nourishment (Kalm, L. M., Semba, & Richard D., 2005). He started the men on a normal thirty-two-hundred-calorie intake per day for a control period. He then began the six month semi-starvation period in which the men received about eighteen hundred calories (Kalm et al.). During the starvation period, the once healthy men transformed into entirely different people—both mentally and physically. Surprisingly, they exhibited many of the symptoms of anorexia nervosa.

The men who participated in the Minnesota Starvation Study started to develop incredibly strange practices centered around food and eating. Before Keys decided to confiscate gum, some men frantically chewed forty packs per day in order to fruitlessly satisfy their hunger (Kalm et al., 2005). Keys reported that the men obsessed about others eating food. “One of the men was walking past a bakery and was so tempted by the rich odors wafting from the place that he rushed in and bought a dozen doughnuts. He gave them to children in the street and watched with relish as they ate them” (Kalm et al., 2005, p. 1350). One of the men, Harold Blickenstaff, remarked, “... it made food the most important thing in one’s life... food became the one central and only thing really in one’s life. And life is pretty dull if that’s the only thing... if you went to a movie, you weren’t particularly interested in the love scenes, but you noticed everytime they ate and what they ate” (Kalm et al., p. 1349). Another man obsessed over cookbooks and actually stockpiled one hundred of them. This fascination with eating and food makes sense. Humans must intake three things to live—oxygen, water, and food. Without food, relationships, careers, and love fade because the brain realizes it cannot attain any of those things if it dies from starvation.

Personality changes in the Minnesota Starvation Study men involved more than their enchantment with food. Marshall Sutton attests that “... We became, in a sense, more

introverted, and we had less energy” (Kalm et al., 2005, p. 1349). They adopted strange rituals around eating. Robert Willoughby, another man who participated in the study explained that “... eating became a ritual... Some people diluted their food with water to make it seem like more. Others would put each little bite and hold it in their mouth a long time to savor it. So eating took a long time” (Kalm et al., p. 1349). Keys noted physical changes as well. The men had a lower threshold for the cold, were dizzy, tired, and sore, experienced hair loss, reduced coordination, and ringing in their ears (Kalm et al.). Keys could not believe the changes in the men. These grown, healthy men were reduced to robots who lost their personalities and sanity. The rehabilitation period came, but the optimal road to recovery surprised everybody.

Keys supplied different calorie increases to different men. The most restricted group only received a four hundred calorie increase. William Anderson experienced this meager addition. He said the rehabilitation period remained “no better” because he never felt satiated. Keys increased the prescribed calories when he noted no marked improvement in the men (Kalm et al., 2005, p. 1351). Keys explained, “Enough food must be supplied to allow tissue destroyed during starvation to be rebuilt... our experiments have shown that in an adult man no appreciable rehabilitation can take place on a diet of 2,000 calories a day. The proper level is more like 4,000 daily for some months. The character of the rehabilitation diet is important also, but unless calories are abundant, then extra proteins, vitamins and minerals are of little value” (Kalm et al., 2005, p. 1351). After the experiment, when faced with freedom in their own diets, the men reveled in the lack of food restriction. Harold Blickenstaff often felt sick because “...[he] couldn’t satisfy [his] craving for food by filling [his] stomach” (Kalm et al., p. 1351). They ate excessively. Jasper Garner described his hunger as a “year-long cavity” that demanded to be filled (Kalm et al., p. 1351). Extreme hunger plagues people recovering from starvation and AN.

The body has been denied necessary calories for days, months, or years, and it must make up for the deficits in order to heal. The healing process demonstrates extremes as well. Roscoe Hinkle explained his weight gain, “Boy did I gain weight. Well, that was flab. You don’t have muscle yet. And get[ting] the muscle again, boy that’s no fun” (Kalm et al., p. 1351). The full recovery for these men ranged from two months to two years. It is important to note that these men’s starvation was not self-imposed. They did not have brains pre-wired for anorexia nervosa. So their recovery experiences, while helpful, are not nearly as complicated as those recovering from anorexia nervosa. For example, Keys did not need to incorporate relapse-prevention aspects to his study. These men desired to feed their frail, sickly bodies while those with AN want with all their hearts to keep feeding the disorder that causes their ailing bodies.

The Minnesota Starvation Study provided valuable insight into anorexia and bulimia nervosa behaviors and symptoms. It also helped explain the effects of starvation and metabolic adaption (Kalm et al., 2005). The extreme hunger experienced by the men gives assurance to those recovering from anorexia nervosa that their excessive eating is a normal and healthy part of recovery. Marzola noted that “...there is considerable data indicating that AN patients need somewhere between 5,000 and 10,000 excess calories to gain a kilogram of weight” (2013, p. 7). Weight gain itself does not signify healing. For example, a common recovery symptom can be the patient feeling very hot and sweaty. The energy from the food makes the patient hyperthermic rather than being used to repair the body. Also, a portion of the surplus of calories must be used to compensate for the brain that wants to starve itself. Sometimes, the patient cannot fight the biological urge and will restrict needed calories. Therefore, extra calories represent a relapse-prevention strategy. Extra calories on successful days help the body to

continue healing despite the understandable stumbles in the recovery process on overwhelmingly difficult days (Marzola E., Nasser, J. A., Hashim, S. A., Shih, P. B., & Kaye, W. H.).

Obviously, the important task of responding to the extreme hunger will lead to weight gain. Weight gain terrifies those recovering from AN. In addition to the anxiety produced as the number on the scale rises, the body's manifestation of the added weight can be very odd and upsetting. Mayer and his coworkers studied twenty-nine women who were approximately weight restored. Compared to healthy women, the patients' BMIs were not strikingly different. However, their waist-to-hip ratios differed greatly from the controls. The fat accumulated in their trunks, rather than their extremities (Mayer, L., Klein, D., Black, E., Attia, E., Shen, W., Mao, X., & ... Walsh, B, 2009). Imagine the horror such a body shape would evoke in a person recovering from AN! However if the person recovering from AN continues to eat and rest, the distribution will eventually normalize. In a separate source, Mayer noted that studies suggest if a person remains weight restored for over a year, the body's odd configuration will redistribute (Mayer, L., Walsh, B., Pierson RN, J., Heymsfield, S., Gallagher, D., Wang, J., & ... Glasofer, D., 2005). The weird distribution of body fat can be traumatic for those recovering. Not only must they gain the weight, their recovery bodies do not meet "healthy" societal standards when they do gain the weight. They feel hot, heavy, and fearful while simultaneously getting pummeled by their panicking anorexic brain. Ultimately, however, the body decides where this fat will go and also how much fat it must gain. Attempts by charts and doctors to predict and dictate how much weight gain signifies healthy recovery actually impede the body's attempts to heal.

The key to a major part of recovering from anorexia nervosa involves the person with AN attempting to trust his/her body again. People with AN must fight through their anxiety and eat

when and what their bodies tell them to. Restricting, denying extreme hunger, and using the BMI chart to determine proper food intake all take away the body's role as healer. Many health professionals have admitted the flaws in the BMI system. Yet, the BMI chart remains in use for treating people with AN, despite research negating its value. The DSM-5 believes that solely using the BMI scale as a guide to weight gain "may not adequately reflect an individual's... history" (Berner, L. A., Shaw, J. A., Witt, A. A., & Lowe, M. R., 2013, p. 1). A new study concludes that "[t]arget weights based only on absolute weight status could contribute to higher rates of relapse and more difficult weight stabilization and 'normalization'" (Berner et al., p. 12). Caretakers should not focus on numbers on a scale but on the individual and his/her history, encouraging the body's own efforts to refeed and heal itself without restricting.

Anorexia nervosa conjures up many misconceptions. From believing vanity births the disease to completely missing the gravity of the starvation symptoms, a majority of the population lack even a general understanding of the disease. In order to help those suffering through the disorder, caretakers must educate themselves in the research discussed in this paper. People recovering from AN must be encouraged to refeed and be reassured that hunger will go back to normal, that the body will redistribute, and that the innate urge to restrict will wane. Meanwhile, caretakers should avoid giving impossible-to-predict goal weights or numbers that act as triggers in the unique wiring of the anorexic brain. In the past, society has not treated the suffering AN population with compassion or humanity. Now, with brain studies, starvation studies, and research, we have the knowledge to empower people with AN in their fight for life and freedom from anorexia nervosa.

References

- Baskaran, C., Misra, M., & Klibanski, A. (2017). Effects of anorexia nervosa on the endocrine system. *Pediatric Endocrinology Reviews: PER*, 14(3), 302-311.
doi:10.17458/per.vol14.2017.BMK.effectsanorexianervosa
- Bell, R. M., & Davis, W. N. (1987). *Holy anorexia*. Chicago: Univ. of Chicago Press.
- Berner, L. A., Shaw, J. A., Witt, A. A., & Lowe, M. R. (2013). The relation of weight suppression and body mass index to symptomatology and treatment response in anorexia nervosa. *Journal Of Abnormal Psychology*, 122(3), 694-708. doi:10.1037/a0033930
- Guisinger, S. (2003). Adapted to flee famine: Adding an evolutionary perspective on anorexia nervosa. *Psychological Review*, 110(4), 745-761. doi:10.1037/0033-295x.110.4.745
- Kalm, L. M., Semba, & Richard D. *They starved so that others be better fed: remembering Ancel Keys and the Minnesota experiment*. (2005, June). Retrieved from:
<http://jn.nutrition.org/content/135/6/1347.full> .
- Kaye, W. H., Wierenga, C. H., Bailer, U. F., Simmons, A. N., Bischoff-Grethe, Amanda. *Nothing tastes as good as skinny feels: the neurobiology of anorexia nervosa*. (2013). Retrieved from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3880159/>
- Lantzouni, E., Frank, G. R., Golden, N. H., & Shenker, R. I. (2002). Reversibility of growth stunting in early onset anorexia nervosa: A prospective study. *Journal Of Adolescent Health*, 31(2), 162-165. doi:10.1016/S1054-139X(02)00342-7
- Marzola, E., Nasser, J. A., Hashim, S. A., Shih, P. B., & Kaye, W. H. (2013). Nutritional rehabilitation in anorexia nervosa: review of the literature and implications for treatment. *BMC Psychiatry*, 13, 290. <http://doi.org/10.1186/1471-244X-13-290>

- Mayer, L., Klein, D., Black, E., Attia, E., Shen, W., Mao, X., & ... Walsh, B. (2009). Adipose tissue distribution after weight restoration and weight maintenance in women with anorexia nervosa. *American Journal Of Clinical Nutrition*, *90*(5), 1132-1137.
doi:10.3945/ajcn.2009.27820
- Mayer, L., Walsh, B., Pierson RN, J., Heymsfield, S., Gallagher, D., Wang, J., & ... Glasofer, D. (2005). Body fat redistribution after weight gain in women with anorexia nervosa. *American Journal Of Clinical Nutrition*, *81*(6), 1286-1291.
- McCallum Place. (2015, March 09). McCallum Place | Eating Disorders & Cardiovascular Risks. Retrieved January 18, 2018, from <https://www.mccallumplace.com/blog/cardiovascular-complications-eating-disorders/>
- Meczekalski, B., Katulski, K., Czyzyk, A., Podfigurna-Stopa, A., & Maciejewska-Jeske, M. (2014). Functional hypothalamic amenorrhea and its influence on women's health. *Journal Of Endocrinological Investigation*, *37*(11), 1049-1056.
doi:10.1007/s40618-014-0169-3
- Misra, M., Aggarwal, A., Miller, K., Almazan, C., Worley, M., Soyka, L., & ... Klibanski, A. (2004). Effects of anorexia nervosa on clinical, hematologic, biochemical, and bone density parameters in community-dwelling adolescent girls. *Pediatrics*, *114*(6), 1574-1583.
- NYU Steinhardt. (n.d.). Department of Applied Psychology. Retrieved December 14, 2017, from <https://steinhardt.nyu.edu/appsyh/opus/issues/2015/fall/corcoran>
- Olwyn, Gwyneth. *Phases of Recovery from An Eating Disorder Part 1*. (2012, November 23). Retrieved from: <https://www.edinstitute.org/paper/2012/11/23/phases-of-recovery-from-an-eating-disorder-part-1> .

Rosen, Meghan. *The anorexic brain: Neuroimaging improves understanding of eating disorder.*

(2013, July 30). Retrieved from

[http://eds.a.ebscohost.com/eds/detail/detail?vid=2&sid=83e3cd4b-85ba-4565-b85c-add5a78b1544%40sessionmgr4007&bdata=JnNpdGU9ZWRzLWxpdmUmc2NvcGU9c210ZQ%3d%3d#AN=edsjsr.23599326&db=edsjsr.](http://eds.a.ebscohost.com/eds/detail/detail?vid=2&sid=83e3cd4b-85ba-4565-b85c-add5a78b1544%40sessionmgr4007&bdata=JnNpdGU9ZWRzLWxpdmUmc2NvcGU9c210ZQ%3d%3d#AN=edsjsr.23599326&db=edsjsr)

Sawyer, S. M., Whitelaw, M., Grange, D. L., Yeo, M., & Hughes, E. K. (2016, April 01).

Physical and psychological morbidity in adolescents with atypical anorexia nervosa.

Retrieved October 12, 2017, from

<http://pediatrics.aappublications.org/content/137/4/e20154080>

Zamboni, M., Armellini, F., Turcato, E., Todisco, P., Gallagher, D., Dalle Grave, R., & ...

Bosello, O. (1997). Body fat distribution before and after weight gain in anorexia

nervosa. *International Journal Of Obesity And Related Metabolic Disorders: Journal Of*

The International Association For The Study Of Obesity, 21(1), 33-36.