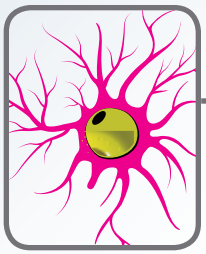

Restrictive eating disorders from the inside out: A neurobiological perspective

By Michael Gross, Farahdeba Herrawi & Thomas Field

Despite featuring the highest mortality rate of any mental illness, eating disorders are often met with misunderstanding and misattunement, primarily because of the stigma surrounding these diagnoses. All too often, these complex and multifaceted illnesses are viewed as lifestyle choices and diseases of vanity.

Long-standing misconceptions about the causes and consequences of eating disorders exist not only at the most basic level of the social microcosm but also within the medical community and among mental health clinicians. These attitudes further isolate those with restrictive eating disorders and discourage them from pursuing treatment because they fear having their experiences minimized and invalidated. This is inversely related to attitudes toward seeking counseling and can create barriers to care for clients who are struggling.

Through understanding the neuroscience of eating disorders, we have an opportunity as clinicians to gain greater insight into these complex disorders. In turn, we can use neurobiological insights to help encourage and educate our clients and their families. By incorporating neurobiology into our conversations with clients to address the nutritional, cognitive, emotional and behavioral factors that impact restrictive eating disorders, we can bring new meaning to clients' experiences and a better understanding of the illness.

Nutrition

The sequelae of medical complications caused by malnutrition have long been established. In the 1940s, when the impact of World War II led millions to die from starvation, Dr. Ancel Keys and colleagues' seminal work, *The Biology of Human Starvation*, illustrated how malnutrition causes cognitive impairment independently of the presence of an eating disorder. Malnutrition plays a significant role in cognitive functioning, the ability to regulate emotions and the ability to process information from the environment. Irrespective of body weight, lack of both adequate macro- and micronutrients can result in cardiac, renal, hepatic, immune and orthopedic complications.

Contemporarily, more credence is now given to the neurobiological changes that take place within the malnourished brain. Atrophy of the prefrontal cortex and hippocampal region of the brain can result in difficulty concentrating, impairment of memory and what some would refer to as "brain fog." Malnutrition also increases the activation of one's primal limbic system, which is responsible for humans' fight-or-flight response. Overactivation of this primary threat detector can result in a manifestation or exacerbation of both anxiety and hyperarousal symptomatology. Atrophy of the hippocampus and decreased availability of serotonin; vitamins D, B6 and B12; selenium; and magnesium can all result in a manifestation or exacerbation of depressive symptoms. Serotonergic deficits pose additional difficulties, resulting in a lack of serotonin conversion into melatonin, which consequently results in difficulty sleeping and puts the body in a state of hyperarousal.

The most urgent task of early recovery and maintenance is supporting the client around restoring adequate daily nutrition; a malnourished brain cannot recover and will not be able to process deeper emotional work. The rewiring of the brain that is associated with psychotherapy will not be robust in a brain that is malnourished. The processing of emotionally laden topics while the client is actively in a negative energy balance serves only to reinforce the use of the eating disorder as a means to cope with uncomfortable affective states.

Even after the completion of weight restoration through a recovery meal plan, the brain needs considerably more time to be refed. The brain may require more than a year of consistent and adequate nutrition for repair and optimization of functioning to take place.

Although it may be tempting to initiate the refeeding process at full throttle, precautions must be taken to monitor and treat refeeding syndrome, which can be fatal if not recognized and ameliorated. Due to increased reliance on adipose tissue and amino acids for energy (as opposed to carbohydrates) while in a state of malnourishment, insulin production is suppressed; intracellular levels of phosphate, potassium and calcium become depleted; and the synthesis of red blood cells decreases. Upon initiation of refeeding, insulin resumes normal production as synthesis of all three macronutrient groups takes place. This process requires magnesium, phosphates and potassium, which are already greatly depleted.

Replenishment of liver and muscle glycogen results in diminished intracellular levels of ATP (adenosine triphosphate) and leads to suboptimal delivery of oxygen to multiple organs in the body. As metabolism increases during the refeeding process, serum levels of electrolytes such as magnesium and phosphorus are rapidly diminished.

This can lead to cardiac arrhythmia and heart attack, which is the most common cause of death in refeeding syndrome. Supervision under a registered dietitian who specializes in eating disorders and a medical doctor is important for mitigating some of the medical risks involved in the early stages of refeeding.

Cognition

Thought process is grossly impaired by restrictive eating disorders. In both anorexia and bulimia, there is weaker structural connectivity between the insular cortex and the hypothalamus, an area that is central to feeding regulation. An increased activation of the insular cortex toward highly palatable and energy-dense foods (which can be regarded as "fear foods") results in increased inhibitory control toward food. This enables restriction of food intake for longer periods of time.

As energy deficits continue for elongated periods of time, there is typically a linear impairment in cognitive function. As restriction continues, cognitive distortions tend to increase and intensify. These can take the form of a distorted perception of one's body and unfounded beliefs surrounding food and weight gain.

The emergence of OCD-like symptomatology may also be observed in restrictive eating disorders. This can be linked to abnormal neuronal responses in the anterior cingulate cortex, resulting in an increase in maladaptive goal-directed behavior and cognitive inflexibility. Those with restrictive eating disorders have a greater tendency to delay immediate gratification for subjective larger rewards in the future, as demonstrated by enduring vigorous exercise regimens on top of heavy caloric restriction for the goal of further weight loss.

In a 2018 study, the Eating Disorders Center for Treatment and Research at the University of California San Diego showed that interactions with dopaminergic reward circuits may promote eating disorder symptoms. In individuals without eating disorders, there is a dopamine spike upon consuming food. In those with anorexia, the dopamine falls flat and there is no response subsequent to food consumption. In fact, in the presence of food restriction, there is an upregulation of dopamine receptors, which accounts for the initial high experienced in response to restriction of energy intake.

Additionally, with the progression of anorexia, the overall size of the brain (both gray and white matter) may shrink. The hippocampus and prefrontal cortex will continue to atrophy as neurons die. This can lead to personality changes and to difficulties with learning, memory and concentration.

Emotion

Garnering an understanding of the atypical neurobiological changes that occur in those with restrictive eating disorders enables clinicians to better explain some of the emotional symptomatology to clients seeking counseling services. Overactivation of the amygdala causes the sufferer to develop an improper fear response to food and a prolonged sense of danger. Subsequently, dysfunction in the insular cortex causes an altered motivation to seek and consume food, which perpetuates restriction.

Another result of the serotonergic deficits mentioned earlier is a lack of disinhibition of the locus coeruleus toward activating the sympathetic

response in the amygdala. This results in behavioral urges to attempt to return to psychological and emotional homeostasis. From a biopsychosocial perspective, the experience of trauma can trigger or exacerbate difficulty in regulating affect due to an increase in neuropeptide corticotropin-releasing factor (CRF) and toxic levels of glutamate.

Those with restrictive eating disorders may present with a very narrow window of tolerance for emotions such as sadness, anxiety and anger. Often, these emotions are dulled out through restriction. Upon initiation of the refeeding process, these emotions may be experienced as intolerable by the individual, and she or he may feel a subjective sense of lacking safety due to the persistent overactivation of the amygdala.

Behavior

Behaviors can serve multiple purposes for people with restrictive eating disorders. First, there is a habitual component to these disorders. As demonstrated by the Minnesota Starvation Study (the results of which Keys and colleagues published in *The Biology of Human Starvation*), restriction of caloric intake can result in an influx of obsessive-compulsive behaviors related to food preparation and intake, food rituals, hoarding, exercise, and lower level movement compulsions.

In a 2019 study, Joanna E. Steinglass and colleagues noted that decision-making approaches indicate that the neural mechanisms of food choice among individuals with anorexia nervosa are different than those in healthy individuals. Research shows that those with restrictive eating disorders make food selections on the basis of habit through the basal ganglia, which are part of the limbic system, rather than on the basis of autonomous choice and thought through the prefrontal cortex. The basal ganglia are in part responsible for the repetitive and habitual behavior, as well as for the hyperactivity, typically associated with anorexia. Removing those with restrictive eating disorders from their usual environment, and thus threatening a change in the type of food they typically eat or preventing them from exercising, can create immense psychological and physiological arousal as well as full-blown panic.

Behaviors can also serve to distract the person with the disorder from unpleasant thoughts, feelings and memories. Although maladaptive, the repetitive nature of the behaviors and the all-consuming obsessions and compulsions that precede them prevent the person from having to confront her or his discomforting emotional experience.

Anorexia also affects the hypothalamus, leading to appetite disruption, immunosuppression, sexual dysfunction, and disruption of the circadian rhythm and sleep. A lack of adequate food means that not enough serotonin is being produced (glucose is a precursor to serotonin). This in turn can cause or exacerbate depression and negatively affect appetite and sleep.

Additional resources

- "Altered microstructure of brain white matter in females with anorexia nervosa: A diffusion tensor imaging study," by Shao-Hua Hu, Hong Feng, Ting-Ting Xu, Hao-Rong Zhang, Zhi-Yong Zhao, Jian-Bo Lai, Dong-Rong Xu & Yi Xu, *Neuropsychiatric Disease and Treatment*, November 2017 (*doi:10.2147/ndt.s144972*)

- "Impaired decision-making in symptomatic anorexia and bulimia nervosa patients: A meta-analysis," by Sebastien Guillaume, Philip Gorwood, Fabric Jollant & Frederique Van den Eynde, *Psychological Medicine*, December 2015 (*doi:10.1017/S003329171500152X*)

Implications for counseling practice

Attitudes toward eating disorders are starting to shift as the neurobiological bases for these disorders are acknowledged and integrated into treatment settings. We can see now that significant differences in brain structure and function exist between those who have eating disorders and those who do not. In understanding that eating disorders are neurobiologically influenced medical disorders requiring early intervention and specialized treatment, we can move toward developing innovative treatment modalities for our clients. In addition, exploring eating disorders through a neurobiological lens can establish a more common language for explaining these disorders to clients of all backgrounds, as well as to their families and support systems.

Restoring healthy eating and weight can help a person's eating disorder symptoms improve or go away all together, especially when practiced in conjunction with psychotherapy, skill-building and a supportive environment. Increasing our (and our clients') understanding of the mechanisms underlying eating disorders will facilitate development of more effective and personalized prevention and treatment options, eventually leading to increased recovery rates and shorter recovery times. It is our responsibility as mental health clinicians to foster greater insight into these complex conditions and to help our clients embrace the endless possibilities that their unique and individual experiences have to offer. ❖

Michael Gross and Farahdeba Herrawi are master's candidates in the Boston University School of Medicine's mental health counseling and behavioral medicine program. Contact them at *mgross6@bu.edu* and *fdeba@bu.edu*, respectively.

Thomas Field is an assistant professor of psychiatry in the Boston University School of Medicine's mental health counseling and behavioral medicine program. Contact him at *tfield@bu.edu*.

Letters to the editor: *ct@counseling.org*