

# The Functional Role of Nutrition and Anorexia Nervosa: Food is Medicine

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## *“It’s not about the food.”*

This phrase, used widely in eating disorder recovery, is misleading and potentially harmful.

Here’s the truth – anorexia nervosa (AN) is not “about” anything other than having a certain neurobiological predisposition to this particular brain disorder, which lays dormant until activated by insufficient nutrition. Food restriction has a calming and mood-elevating effect in people with this type of brain chemistry, and people prone to AN are usually quite sensitive, anxious, and emotionally reactive.

Therefore, anorexics may restrict their food intake (either consciously or unconsciously) as a way of coping with overwhelming emotions or stressful events. So it isn’t JUST about the food; it’s about feelings and circumstances as well. But it is the disturbance in eating behavior and weight, rather than feelings or events, which causes immense physical and psychological damage.

At every step of the eating disorder process, nutrition (or lack thereof) plays a functional role. An initial period of low nutrition sets the disorder in motion. Ongoing low nutrition and low body weight perpetuate the symptoms. Sustained full nutrition and weight restoration are essential for mental and physical recovery. Continued good nutrition and maintenance of a healthy body weight for life protect patients against relapse.

The relationship between food and AN is analogous to the relationship between alcohol and alcoholism. To state that AN “isn’t about the food” is like stating that alcoholism “isn’t about drinking.” A person may be born with a predisposition to developing alcoholism due to her genetic makeup and her particular brain chemistry. However, if that person never takes a sip of alcohol, the disease will never be activated in the first place. Similarly, a person predisposed to AN will not develop the disorder in the absence of a nutritional deficit.

The development of eating disorders may be conceptualized in terms of the “four P’s:”

## **Predisposing factors**

Recent research indicates that 50-80% of the risk of developing AN is genetic (Kaye, 2007). Patients with AN typically demonstrate high levels of anxiety, harm avoidance, and behavioral inhibition (Shaw et al. 1997) – all traits which are heavily influenced by genetics (Cloninger, 1986, 1987, 1988). Perfectionism, obsessionality, and cognitive rigidity, which are also highly heritable, have been identified as risk factors for AN (Kaye et al., 2009). Most patients with AN have exhibited one or more of these traits since early childhood, long before the development of an eating disorder. These traits tend to be exacerbated during bouts of malnutrition and persist long after recovery, albeit to a lesser degree (Kaye, 2007).

Avoidance of food in AN has a physiological basis as well as a psychological one. Appetite and satiety dysregulation increase a person's vulnerability to developing AN (Steiner et al., 1991; Stice et al., 1999). Individuals with AN have alterations in the brain's insular cortex (Wagner et al., 2008), which is related to disrupted bodily sensations. Thus, people with AN may literally not recognize when they are hungry, and may actually not experience much pleasure from the taste of food.

Healthy people tend to feel irritable, tired, and sluggish when they under-eat. In contrast, people with AN feel more energetic, alert, and calm when they are fasting (Gura, 2008). Recent developments in brain imaging have revealed the neurobiological mechanisms which underlie anorexics' abnormal reaction to food. Functional Magnetic Resonance Imaging (fMRI) studies have revealed that individuals with a history of AN have an imbalance between ventral brain circuits, which regulate reward and emotion, and dorsal circuits, which are associated with consequences and planning ahead (Wagner et al., 2007). Disturbances in the serotonergic system contribute to vulnerability to restricted eating, behavioral inhibition, and excessive concern about the negative consequences of eating and gaining weight (Kaplan, 2010). Given that these impairments persist long after recovery, researchers believe that they are present before the onset of AN and may predispose certain individuals to developing AN.

Taken together, a pre-existing anxious temperament, brain-based tendencies to misperceive hunger and derive little pleasure from food, and the calming and mood-elevating effects of food restriction, create a strong predisposition towards developing AN.

### **Precipitating factors**

Anorexia nervosa is always precipitated by a period of low nutrition. The precursor to the low nutrition will vary from person to person. In the USA and other western cultures, where most females experience social pressure to be thin, AN is usually triggered by a weight-loss diet. Not every episode of AN is triggered by dieting, however. A simple desire to "eat healthy," participation in sports without appropriate caloric compensation, a bout with the stomach flu, an episode of depression, or simply loss of appetite during a period of stress - any one of these unintentional, seemingly benign periods of low nutrition can trigger AN in a vulnerable child. Any weight loss in a child or adolescent should be a red flag (O'Toole, 2010).

Most people with AN will lose weight as a consequence of low nutrition and/or excessive exercise. However, in children and adolescents, low nutrition can manifest as failure to gain weight or failure to grow. Failure to achieve expected growth and weight gain during childhood or adolescence can also be a sign of an eating disorder (O'Toole, 2010). When a child falls off her historical growth curve (for example, a child who has always tracked in the 75<sup>th</sup> percentile for height and weight falling to the 40<sup>th</sup> percentile for height and weight between her 12 year and 13 year checkup), this is a cause for alarm, even if this child is not technically in the "underweight range" on the charts.

Puberty, which involves dramatic hormonal, neurological, and physical changes coupled with new social and academic demands, is often a precipitating factor for AN. Early adolescence is a time of rapid growth and development. Between the ages of 11 and 14, a typical girl who tracks along the 50<sup>th</sup> percentile grows over 6 inches and gains over 26 pounds (CDC, 2000). A substantial increase in weight, particularly in body fat, is a necessary catalyst for puberty. Consequently, adolescents have increased nutritional requirements during early adolescence which must be met if puberty is to proceed normally. The social pressures to conform to the "thin ideal" and make oneself more attractive to the opposite sex are intensified at this age. Therefore, many pubescent girls attempt to restrict their food intake and increase their exercise in attempt to counteract their body's pubertal changes. Further, early

adolescence is the time at which many girls become involved in competitive sports or elite dance programs which increase their nutritional needs even more. The combination of very high energy requirements and dietary restriction can easily trigger an episode of AN in a vulnerable young person.

The vast majority of girls will toy around with dieting in adolescence and emerge unscathed because powerful biological forces compel them to eat plenty of food and maintain a healthy weight. However, for adolescents with a biological predisposition for AN, puberty-related hormonal changes appear to activate serotonin dysregulation (Kaye, 2007). Dieting alters serotonergic functioning in females (Goodwin, Fairburn, & Cowen, 1987), thus providing anti-anxiety and anti-depressant effects in girls who are predisposed to AN.

### **Perpetuating factors**

Continued malnutrition is largely responsible for the self-perpetuating cycle of anorexic symptoms. A starved brain is a sick brain, and people who are undernourished for any reason display many of the symptoms commonly associated with AN such as preoccupation with food, unusual food rituals, social withdrawal, irritability, and depression (Keys, 1950).

In addition, people with AN typically experience body dysmorphia, drive for thinness, and fear of weight gain. Most individuals with AN are unable to recognize how thin they are and may perceive themselves as normal or fat, despite emaciation (Mohr et al., 2010). They are terrified of eating and morbidly afraid of gaining weight. They cope with these fears by continuing to restrict their diet and remaining underweight, which of course perpetuates the symptoms of starvation. It is a vicious cycle.

Not all individuals with AN experience body dysmorphia, drive for thinness, or fear of fat. Some people with AN are well aware that they are too thin and express a desire to gain weight, but find it nearly impossible to do so. Ongoing malnourishment is a perpetuating factor for the “non fat-phobic AN” patient as well. For those who are predisposed to AN, food restriction and excessive exercise have a calming, mood-stabilizing effect (Gura, 2008). When these patients attempt to eat more or exercise less, they are flooded with extreme anxiety, mood swings, and irritability. Food restriction quickly diminishes these symptoms. And so the cycle continues.

### **Prognostic factors**

Research has indicated that full nutrition and prompt weight restoration as soon as possible after diagnosis predict better outcome in AN.

Likewise, prolonged periods of time spent at a sub-optimal weight are associated with a protracted course of illness and increased risk of irreparable damage such as infertility, osteoporosis, chronic depression, and suicide.

A recent study of inpatients with AN found that the best predictors of weight maintenance during the first year post-discharge were the level of weight restoration at the conclusion of acute treatment and the avoidance of weight loss immediately following intensive treatment (Kaplan et al., 2009). Another study found that nutrient density and variety (eating a wide range of foods, including those that are high-calorie and high-fat) were significant predictors of positive long-term outcome in weight-restored anorexics (Schebendach et al., 2008).

All of the available data suggest that eating a complete, well-balanced diet and maintaining ideal body weight are of utmost importance in recovery from AN and in preventing relapse. Full nutrition and weight restoration alone will not cure AN, but full recovery cannot occur without these essential components.

In sum, nutrition plays a functional role in all stages of AN, from the initial onset and maintenance of symptoms to physical and mental recovery to relapse prevention.

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