EATING DISORDERS
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## *Eating Disorders:*

# The Biochemical Connection

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Anorexia nervosa and bulimia are disorders with well-recognized psychological components. However, many researchers now believe that these conditions have a physiological origin, which may ultimately improve the treatment options for those who are so afflicted.

It was in the early 1970's that researchers began to study seriously the links between food intake and the brain. More recently, these researchers have sought to find applications in psychiatry. A steady increase in scientific knowledge holds the promise that new medications might be found which can create alterations in brain chemistry.

It is argued that people suffering from eating disorders are actually attempting to alter brain chemistry imbalances by manipulating their intake of foods. Eating patterns and macronutrient (protein, carbohydrate, fat) selection affect the production of neurotransmitters, i.e. epinephrine or serotonin. This theory results from reasearch which shows in great detail how food choices or fasting affect the brain and how a given food increases or decreases the production of specific neurotransmitters, the chemicals which transmit signals between brain cells.

The nutrients which affect these brain chemicals are chiefly amino acids, components of protein. A small number of these building blocks of protein have the unique property, once they are digested and enter the blood, of readily crossing the barrier between the brain and most compounds in the blood. Much of the research has focused on two of these animo acids, tryptophan and tyrosine. These substances are known as precursors, signaling the brain to produce certain brain chemicals. Tryptophan affects the neurotransmitter serotonin, while tyrosine affects a number of neurotransmitters including epinephrine.

Experiments conducted on animals indicate that injection of epinephrine directly into the hypothalamus causes normally satiated animals to consume large quantities of food—particularly carbohydrates (starches or sweets). The injection of serotonin into the hypothalamus produces the opposite effect, a reduction carbohydrate ingestion. Clinical studies indicate that these neurotransmitters may cause the same reactions in humans. This is evidenced by the fact that antidepressant drugs, which influence the brain's norepinephrine system, produce in some people a "craving" for carbohydrates. The anorexic drug fenfluramine, which activates serotonin reduces the appetite for carbohydrates.

After consumption of carbohydrates it is known that levels of the amino acid tryptophan increase, providing the essential precursor for serotonin synthesis. With increased serotonin production in the hypothalamus, norepinephrine activity is then inhibited and a normal satiety for carbohydrates develops. This leads to the cessation of the ingestion of carbohydrates, and either the ingestion of protein or fat, or a period of abstinence from food.

A number of studies have indicated that anorexia and bulimia are associated with particular aberrations in the regulation of carbohydrate ingestion. Anorexics consciously avoid carbohydrate-rich foods, whereas bulimics usually binge on carbohydrate-rich foods.

Although the evidence of the connection between brain chemistry anomalies and eating disorders is convincing, the etiology of anorexia nervosa and bulimia remains unknown. These conditions are complex are associated with multiple determinants including familial and cultural influences, alterations in brain chemistry and genetic predisposition. Drugs are clearly useful in the amelioration of the symptoms of eating disorders. However, the life-threatening nature and multifaceted etiology of eating disorders suggest an interdisciplinary approach to treatment including: medical/biochemical, psychological and nutritional components. ●

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