Influencing Your Appetite

Causes Of Hunger & Satiety

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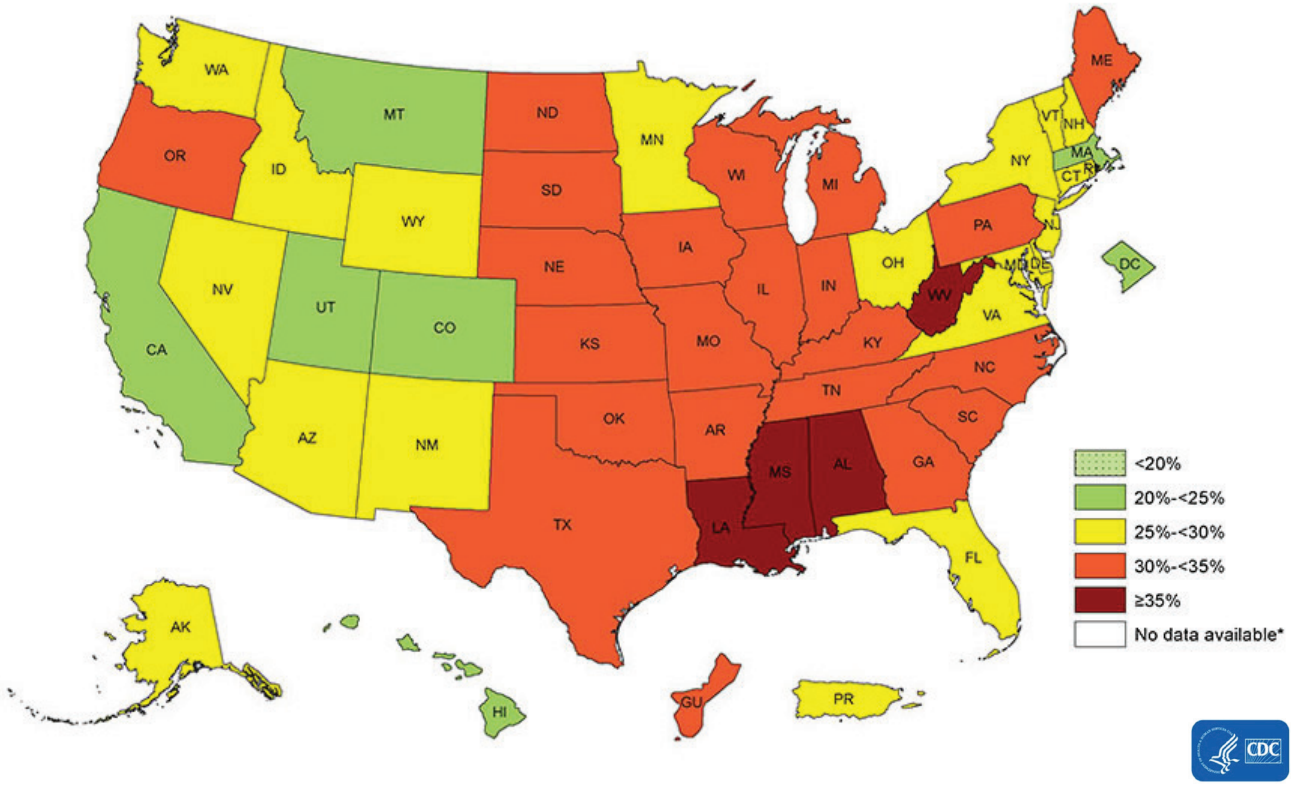
More than one-third (36.5%) of U.S. adults have obesity.



• No state had a prevalence of obesity less than 20%.

• In 6 states (California, Colorado, Hawaii, Massachusetts, Montana, and Utah) and the District of Columbia, obesity ranged from 20% to less than 25%.

• 19 states and Puerto Rico had a prevalence of obesity between 25% and less than 30%.

• Obesity prevalence in 21 states and Guam was 30% to less than 35%.

• Four states (Alabama, Louisiana, Mississippi, and West Virginia) had an obesity prevalence of 35% or greater.

• The South had the highest prevalence of obesity (31.2%), followed by the Midwest (30.7%), the Northeast (26.4%), and the West (25.2%).

What Causes Hunger?

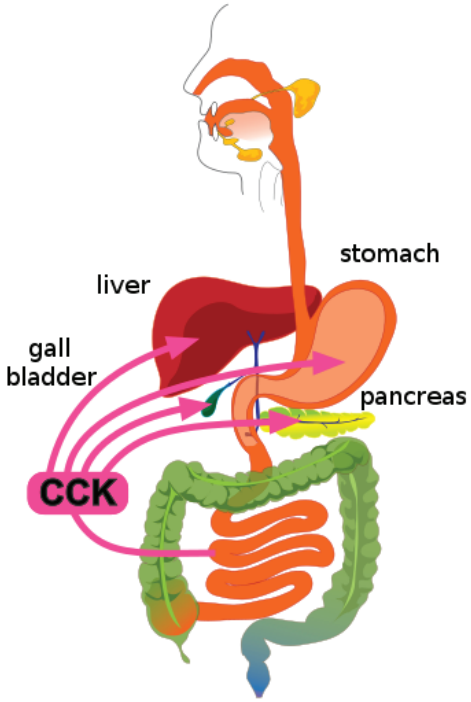
A common hurdle for weight loss patients to overcome is hunger. As one works to correct their eating habits, the body attempts to sabotage the patients' weight loss goals by telling them to eat more.

The sensations of hunger and satiety are influenced by environmental and genetic factors, as well as specific centers of the brain - especially the hypothalamus. The hypothalamus receives signals from the gastrointestinal tract which influence eating behavior. While some neurotransmitters and hormones signal lack of food and stimulate the appetite, norepinephrine, cholecystokinin, and others increase satiety and curb cravings (Guyton & Hall, 2000, p. 804). This is accomplished by inhibiting dopamine output to the brain's reward center, reducing the motivation to eat.

Decrease Hunger	Increase Hunger
• Norepinephrine • Cholecystokinin • Serotonin • Leptin • Insulin • Enterostatin	• Cortisol • Galanin • Endorphins • Orexins • Melanin-concentrating hormone • Neuropeptide Y

CHOLECYSTOKININ (CCK)

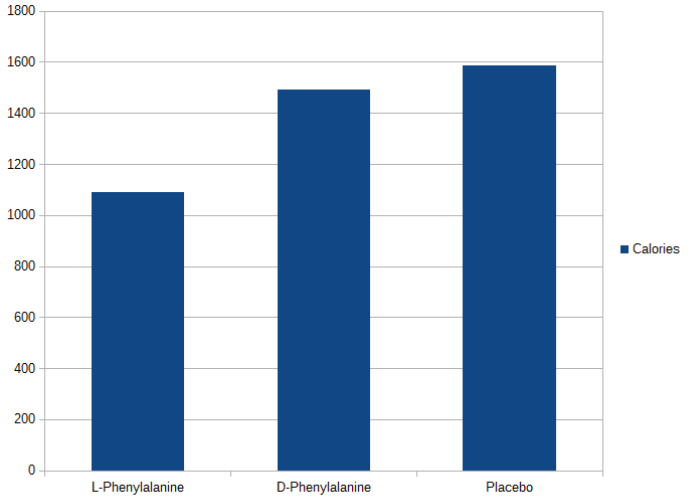
Cholecystokinin (CCK) is a peptide hormone responsible for stimulating the digestion of fat and protein. CCK is secreted by the small intestine when we eat, triggered as a response to fat entering the duodenum (Guyton & Hall, 2000, p. 806). This hormone tells the body to begin the digestive process and also acts as an appetite suppressant.

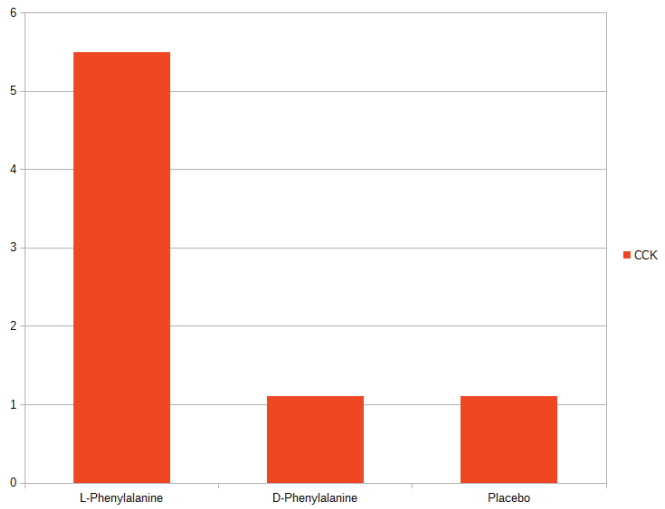


L-Phenylalanine can cause a release of CCK to influence satiety, as was demonstrated in a 1976 study:

> "Five rhesus monkeys were infused intravenously with partially purified cholecystokinin (CCK) Just prior to a test meal of solid food after overnight food deprivation; CCK produced large, rapid, dose related suppressions of feeding. The lowest dose tested produced a significant inhibition of food intake. In a second experiment, gastric preloads of a potent releaser of endogenous CCK, L-phenylalanine (L-Phe), and a weak releaser, D-phenylalanine (D-Phe) were compared for their relative abilities to suppress food intake at a test meal in nine rhesus monkeys after overnight deprivation. L-Phenylalanine produced large, rapid, dose-related suppressions of feeding, but D-Phe did not. Neither CCK nor L-Phe caused signs of illness in these experiments. The results demonstrate that intravenous exogenous CCK suppresses feeding in rhesus monkeys and suggest that endogenous CCK has the same effect; they are consistent with the hypothesis that CCK is a satiety signal." (Gibbs, 1976)

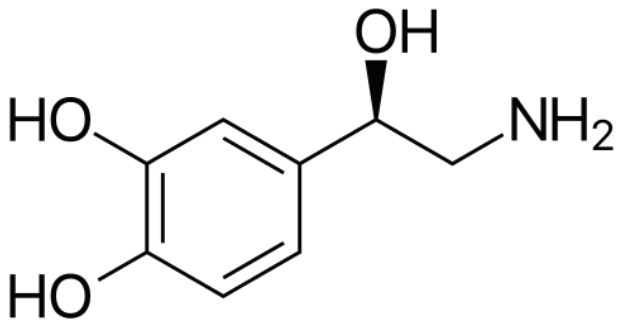
A separate study using human subjects found that CCK levels peaked 20 minutes after administration of L-Phenylalanine. Subsequently, subjects who received an administration of L-Phenylalanine showed significantly reduced calorie intake compared to D-Phenylalanine and placebo. (Ballinger, 1994)





Subjects consumed 1,089 kcal after L-Phenylalanine; 1,492 kcal after D-Phenylalanine; 1,587 kcal after placebo. Base CCK levels were 1.10 pmol/L; 20 minutes after L-Phenylalanine CCK increased to 5.49 pmol/L. There was no increase in CCK in the D-Phenylalanine and Placebo subjects.

NOREPINEPHRINE (NE)

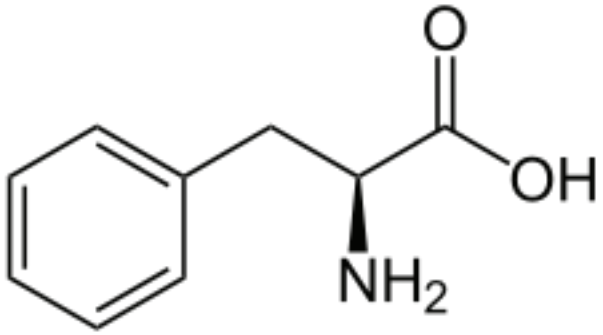


Amphetamine based appetite suppressants such as Phentermine work by causing a release of the body's NE stores. Through a mechanism similar to that of CCK, NE sends a signal to the brain that causes satiety.

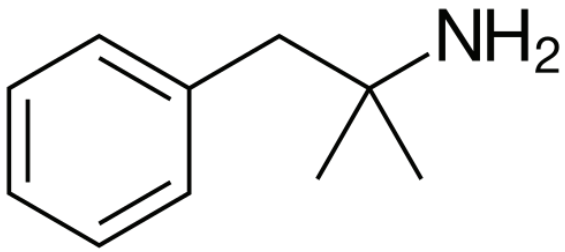
L-Phenylalanine also causes a release of NE but unlike amphetamines, L-Phenylalanine causes the brain to replenish its stores of NE. This means that there will be no depressive crash, which can potentially cause a rebound eating binge, when the patient stops using L-Phenylalanine.

This is accomplished when the body converts L-Phenylalanine to L-Tyrosine. L-Tyrosine is then converted to dopamine, norepinephrine, and epinephrine (adrenaline).

L-PHENYLALANINE



PHENTERMINE



L-Phenylalanine is an essential amino acid that is found in a variety of foods including almonds, peanuts, eggs, poultry, etc. L-Phenylalanine has been shown to influence satiety by stimulating a release of cholecystokinin and increasing the availability of norepinephrine.

While L-Phenylalanine is found in dietary sources, a larger dose is required to achieve effective results. Diucaps, from Legere Pharmaceuticals, contains L-Phenylalanine in a therapeutic dose to help reduce hunger and cravings naturally, without the side effects associated with stimulant-based appetite suppressants.

L-Phenylalanine may be a viable alternative to stimulant-based prescription appetite suppressants for patients who are not medically approved or who cannot use stimulants for job-related reasons. L-Phenylalanine may also ease the process as one transitions from stimulant-based prescription appetite suppressants by replenishing norepinephrine.

References

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