

## New Food-Addiction Link Found

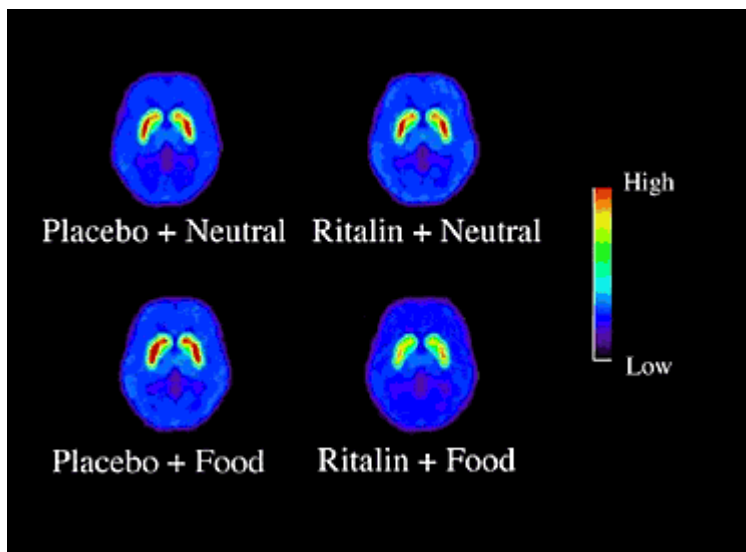
### Mere sight/smell of food spikes levels of brain “pleasure” chemical

Scientists at the U.S. Department of Energy’s Brookhaven National Laboratory have found that the mere display of food — where food-deprived subjects are allowed to smell and taste their favorite foods without actually eating them — causes a significant elevation in brain dopamine, a neurotransmitter associated with feelings of pleasure and reward. This activation of the brain’s dopamine motivation circuits is distinct from the role the brain chemical plays when people actually eat, and may be similar to what addicts experience when craving drugs.

“Eating is a highly reinforcing behavior, just like taking illicit drugs,” said psychiatrist Nora Volkow, the study’s lead investigator. “But this is the first time anyone has shown that the dopamine system can be triggered by food when there is no pleasure associated with it since the subjects don’t eat the food. This provides us with new clues about the mechanisms that lead people to eat other than just for the pleasure of eating, and in this respect may help us understand why some people overeat.” The study will appear in the June 1, 2002 issue of *Synapse* (now available [online](#) ).

Brookhaven scientists have done extensive research showing that addictive drugs increase the levels of dopamine in the brain, and that addicts have fewer dopamine receptors than non-addicts. Last year, in an effort to understand the relationship of the dopamine system to obesity, they found that obese individuals also had fewer dopamine receptors than normal control subjects.

In the new study, the scientists investigated the role of dopamine in food intake in healthy, non-obese individuals. The researchers used positron emission tomography (PET), a brain-scanning technique, to measure dopamine levels in 10 food-deprived volunteers. Each volunteer was given an injection containing a radiotracer, a radioactive chemical “tag” designed to bind to dopamine receptors in the brain. The PET camera picks up the radioactive signal to measure the level of tracer. Since the tracer competes with dopamine for binding to the receptor, the amount of bound tracer can be used to infer the concentration of dopamine (more bound tracer = less dopamine).



These brain scans can be used to infer brain dopamine levels in the four experimental conditions (with and without food stimulation, paired with and without an oral dose of Ritalin). Note that the tracer signal in

the Ritalin + food scan is significantly lower than the others. This is because the radiotracer competes with natural brain dopamine for binding to the receptor. When there is a lot of tracer bound (the first three conditions), it means there is not as much natural brain dopamine. When there is little tracer bound (as in the Ritalin + food scan), there is more natural brain dopamine occupying the receptor sites. So, it is an inverse relationship (a low tracer signal = a high dopamine level). [Hi-res image](#) (300 dpi jpeg).

Study subjects' brains were scanned four times over a two-day period, with and without food stimulation, paired with and without an oral dose of methylphenidate. Methylphenidate (Ritalin) is known to block the reabsorption of dopamine into nerve cells. The researchers wanted to see if it would amplify any subtle changes in dopamine levels.

For food stimulation, the volunteers were presented with foods they had previously reported as their favorites. The food was warmed to enhance the smell and the subjects were allowed to view and smell it, as well as taste a small portion placed on their tongues with a cotton swab. As a control, during scans when food stimulation was not used, subjects were asked to describe in as much detail as possible their family genealogy. Study participants were also instructed to describe, on a scale of 1 to 10, whether they felt hungry or desired food prior to food stimulation and then at five-minute intervals for a total of 40 minutes.

The researchers found that food stimulation in combination with oral methylphenidate produced a significant increase in extracellular dopamine in the dorsal striatum. There was also a correlation between the increase in dopamine triggered by food stimulation and methylphenidate and the changes in self-reports of 'hunger' and 'desire for food.' "This suggests the dopamine increases during the food/methylphenidate condition reflect the responses to food stimulation and not the isolated effects of methylphenidate," Volkow said.

The study demonstrates that methylphenidate, when used at low doses, amplifies weak dopamine signals. It also shows, for the first time, that the dopamine system in the dorsal striatum plays a role in food motivation in the human brain.

This relationship was not observed in the ventral striatum, which includes the nucleus accumbens, the area of the brain thought to be responsible for food reward. "We and others previously thought the nucleus accumbens was the primary brain region associated with regulating food intake by modulating reward and pleasure while eating," said study coauthor G. Wang. "These findings challenge that belief."

This study was funded by the U.S. Department of Energy, which supports basic research in a variety of scientific fields, and the National Institute on Drug Abuse.