Is Satiety (Lack Thereof) the Culprit with Obesity?

Individuals with mental illness are more likely to be overweight or obese than the general population. When institutionalization was the norm, and individuals spent decades in psychiatric hospitals, the thinking was that high starch diets, restricted environments, and lack of exercise combined to cause the problem. Then psychoactive agents, both traditional and newer versions, were added to the blame package. Now, new research with a focus on individuals with Prader-Willi syndrome suggests another causal possibility (Tingley, 2015).

Prader-Willi syndrome has a rare chromosomal abnormality, with chief symptoms of low muscle tone and impaired signaling between the brain and stomach. “Because no sensation of satiety tells them to stop eating or alerts their body to throw up, they can accidentally consume enough in a single binge to fatally rupture their stomach” (Tingley, 2015, para. 9).

Tingley (2015) reports that researchers (Andrea Prader, Heinrich Willi, and Alexis Labhart) at a Swiss children’s hospital first identified the syndrome in 1956. For 25 years, there seemed to be no clinical interest or further research on the syndrome. Beyond the inability to experience satiety, characteristics include “short stature, disproportionately short arms and legs and small hands and feet; a tendency to pick skin until it bleeds; a high pain tolerance; infertility; excellent spatial memory; and cognitive disabilities” (Tingley, 2015, para. 15). Children affected by this illness all show an early failure to thrive, followed by obesity. The article includes several stories about these children and how their parents have tried to cope with the illness and been blamed for the problem (Tingley, 2015).

The relevance for psychiatric nurses is that an understanding of Prader-Willi syndrome and new avenues of research, including pharmaceuticals, would inform their clinical practices. Thinking about obesity in the clients whom they serve needs to shift to considering the error on the chromosome 15 segment, which occurs at conception, and is one of the few known genetic causes of obesity. No other genetic disorder produces the extreme lack of satiety or urge to obtain food. How the dozens of genes within chromosome 15 govern fullness is still unknown. What is known is that Prader-Willi syndrome disrupts the functioning of the hypothalamus, but the process is unknown. Individuals with psychiatric illness who are obese and others in the general population may have a chromosome 15 error, but not full-blown Prader-Willi syndrome.

Leptin and other hormones are known to signal the hypothalamus, affecting sensations of hunger. Leptin is a hormone produced by fat cells and interacts with genetic and environmental factors to influence whether overeating occurs. Determining this signaling error could result in a treatment for almost all cases of obesity. Jennifer Miller, an endocrinologist at the University...
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of Florida, quoted by Tingley (2015), states: “I feel that Prader-Willi holds the key…. If you could control their appetite, you could control the appetite of anyone” (para. 19).

In June 2013, the American Medical Association’s (AMA) House of Delegates voted to recognize obesity as a disease. NBC Nightly News (Snyderman, 2013) and several major newspapers covered this news. Reporters for the Los Angeles Times (Gorman & Healy, 2013) stated: “[this] vote is certain to step up pressure on health insurance companies to reimburse physicians for the time-consuming task of discussing obesity’s health risks with patients whose body mass index [BMI] exceeds 30.” The AMA’s Council on Science and Public Health did not agree with the vote. Their rationale was that BMI is simplistic and flawed.

Zafgen, a pharmaceutical company, has successfully tested beloranib to reduce weight, body fat, and insatiability in patients with Prader-Willi syndrome. Another firm, Ferring Pharmaceuticals, is clinically testing a drug to improve the signaling of fullness in the hypothalamus. The human growth hormone replaces a deficiency in the pituitary gland and has also been tried as a part of treatment (Tingley, 2015).

Individuals with psychiatric diagnoses often also have comorbid issues with street drugs of various kinds and alcohol. The hypothalamus regulates the impulse to eat and is linked to the reward centers of the brain. Brain scans can show how anticipation of drugs and alcohol, or their ingestion, are imaged. Brain scans are also affected when individuals are shown images of food, in conditions when they have or have not had prior intake of glucose. Food, drugs, and alcohol produce similar pleasure sensations.

The Center for Prader-Willi Syndrome is within the Children’s Institute of Pittsburgh, Pennsylvania. In 1982, it was the first institute to start a research and treatment center. Gregory Cherpes, MD, is the Center’s Director of Behavioral Health. Although their primary patient population is children, they also see some teens and young adults who were not diagnosed with the syndrome earlier in their lives. These patients are directed to a geneticist, who will provide testing for an abnormality on chromosome 15. Genetics testing discovers that in 70% to 75% of cases there is a deletion, with multiple genes involved. This gene area is referred to as 15-q11-q13 (G. Cherpes, personal communication, February 2015). This defect is inherited from the father, but, in another version, the inheritance is from the mother, with two chromosomal defects. “My Deadly Appetite” is a documentary developed by Discovery Life (2010), which can be seen on YouTube.

Since the early 1980s, several other centers for this syndrome have been established. Researchers hope to discover how individuals with Prader-Willi syndrome switch in early childhood from an extreme disinterest in food to being insatiable and describing, not hunger, but physical pain constantly. The nature of appetite and how it is experienced also affects the lives of individuals with other eating disorders, such as anorexia and bulimia.

A few group homes exist for adults with Prader-Willi syndrome, but demand exceeds the supply. The homes have to be able to control food supply, as well as monitor drugs being used, and assure that exercise occurs. Patients cannot manage to be in community settings without supervision, as the food temptations are too great. When individuals with Prader-Willi syndrome are in a neighborhood where there are many stores and shops selling all types of food, they can easily elude a staff member, make a purchase, and consume their choice within minutes. Maintaining a balance between allowing some measure of independent living and assuring that food cravings are controlled is not easy.

What is control, so far as ingestion of food is concerned? What is the source of feeling full or feeling hungry? These are the critical questions now being raised since the early work on Prader-Willi syndrome. Differentiating between the drive to seek food when hungry and then overeating or being unable to stop is the key to understanding obesity. What will be learned in studies about Prader-Willi syndrome will help answer these questions and benefit entire populations.

REFERENCES


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