



IN SEARCH OF EARTHLY EXPLANATIONS FOR
EATING DISORDERS | BY CHUCK STARESINIC

REFUSING SUSTENANCE

Catherine Benincasa was a rebellious child, strong-willed and stubborn like her mother, with whom she had a close but contentious relationship. The mother was a confirmed atheist, yet the daughter was somehow filled with religious devotion, even as a small child. At age 7, walking back to her home in Siena, Italy, from a visit with her older sister, Catherine reported a vision in which Christ held out a ring to her.

As she grew, Catherine wanted her mother to understand—or at the very least acknowledge—her profound belief in God, but the mother refused. The defiant adolescent strove to make penance out of the activities of daily life, even depriving herself of food against her mother's wishes. Unbeknownst to her family, Catherine began secretly throwing meat under the table.

Eating disorders are thought of as uniquely modern phenomena. So how do we explain Catherine of Siena, who presented symptoms of anorexia nervosa?

PAINTING | LINDA WALLEN



© CLEVELAND MUSEUM OF ART; PANEL FROM ST. CATHERINE OF SIENA AND THE BEGGAR, BY GIOVANNI DI PAOLO, CIRCA 1460

Catherine of Siena had no experience with fad diets or fashion models in *Vogue*. What did she have in common with those who suffer from eating disorders today?

When Catherine was 15, her sister, with whom she was very close, died in childbirth. Catherine should marry the widower, her mother insisted. He was a rich painter, and his wealth would ensure the financial security of the entire family. Already traumatized by the death of her sister, Catherine was being pushed to abandon her decision to devote herself to God. She fought back against her mother by entering into a prolonged period of fasting, which caused her to lose such a shocking amount of weight that her parents worried she would die. They desperately sought the intervention of the local priest.

The priest thought she must be saintly, stubborn, or insane, but he couldn't decide which it was. Eventually, after she gained admittance to a convent, her refusal to eat was trumpeted as a triumph of spirit over flesh. Self-starvation was a sacrifice so profound it indicated spiritual transcendence. (With no earthly explanation, her condition must be divine.) Catherine herself believed that God did not intend for her to desire food. Her sustenance was of a higher form. She severely limited her food intake for much of her adult life, sometimes taking food into her mouth to appease others and spitting it out at the first opportunity. She would sometimes use a goose feather to trigger regurgitation. She died after falling ill at the age of 33, undernourished and frail.

More than 600 years after her death, she is known as Saint Catherine of Siena. She isn't alone in the pantheon of saints—scores of Italian medieval saints also appear to have had symptoms of anorexia nervosa. Saint Theresa of Avila used olive twigs to induce vomiting, emptying her stomach before taking the Eucharist.

Eating disorders are frequently thought of as uniquely modern phenomena. So how do we explain Catherine of Siena and others who similarly suffered through the ages? In 1695, an English physician named Richard Morton described anorexic behavior in young women, as did Giorgio Baglivi a few years later at the University of Rome. Sir William Gull coined the term anorexia nervosa in 1874. Catherine's contemporaries attributed her condition to the divine, as did she. But today, we insist on rational explanations for the human condition. We favor the physiological, biochemical, and neurological over the supernatural. Yet eating disorders have defied many of our best attempts at understanding. We point to the victim's environment, specifically indicting the family and Western culture. But Catherine of Siena had no experience with fad diets or the fashion models in *Vogue* magazine. What did she have in common with those who suffer from eating disorders today?

In psychiatric residency programs, they say you either like working on eating disorders or you don't. Once exposed, nobody is ambivalent. Walter Kaye was hooked from the start.

When he was a research fellow at the National Institute of Mental Health (NIMH) in the late 1970s, Kaye was asked to complete a study on anorexia nervosa. He became intrigued by the homogeneity of the disease—so many patients seemed to have the same symptoms. In schizophrenia and depression, by comparison, symptoms were all over the map. Anorexia seemed to be an area ripe for research, but funding and scientific interest in biological causes were limited, perhaps because the condition was believed to be self-imposed and culturally driven.

Kaye found that he enjoyed working with patients who suffered from eating disorders, too. They had a lot of similarities, beyond their obvious symptoms. For the most part, they were smart, decent, accomplished young people. It's true that there was a lot of mortality, more than in any other psychiatric condition, but many patients with eating disorders recovered and went on to do very well in life.

Gradually, it dawned on Kaye, who is now a professor of psychiatry at the University of Pittsburgh's Western Psychiatric Institute and

Clinic (WPIC), that there was something else that he saw in these patients who tended to be perfectionist, obsessive, and meticulous. “I have those traits myself,” he says now, smiling like someone who long ago came to terms with his own idiosyncrasies. “I think a lot of researchers do. These are traits that can be very beneficial in certain professions, like engineering and medical research. And if you’re not a little obsessional and perfectionistic in research, then you’re not going to be very successful.”

Researchers were beginning to believe that the similarities among patients with eating disorders were more than coincidental. In 1996, Kaye embarked on a landmark study—the first ever genetic study of eating disorders. Supported by the Price Foundation, he collaborated with centers all over the world to collect data on families with two or more cases of eating disorders. The primary goal was to see what set these families apart genetically from those without a history of eating disorders.

Important findings trickled out of the study. Kaye and colleagues showed that around 20 percent of fathers of anorexic patients had obsessive, perfectionist traits—very high compared to the general population. One might say this points to family environment as a causal factor, but it points to genetics as well. Women with bulimia were found to have altered levels of the mood-regulating neurotransmitter serotonin, even after they recovered from the eating disorder. Could this biological susceptibility explain why some women developed eating disorders when exposed to cultural influences and others did not? The serotonin re-uptake inhibitor Prozac was later found to help patients recovering from anorexia to maintain body weight, strengthening the notion that eating disorders were true biochemical imbalances.

Kaye says that he couldn’t do the genetic studies without the contributions of Bernie Devlin, a Pitt PhD associate professor of psychiatry and human genetics. The two bring vastly different skills and personalities to their work. Kaye is soft-spoken and subdued, while Devlin is outgoing and animated. Kaye is an expert in the clinical presentation of eating disorders. Devlin is a statistical geneticist; he crunches numbers, and he crunches nucleotides. When Kaye first came to see him, Devlin could tell that the psychiatrist had been rigorous in gathering data. He recalls that Kaye said, “We have about 100 behavioral covariables we’ve assessed on these eating-disordered individuals, but we don’t know how to use them efficiently in a genetic analysis. Do you?”

In other words, Kaye and his team had come

up with a hundred different characteristics to look for in study subjects with eating disorders (more than 90 percent of whom were women). Was there a history of depression in the family? A history of obsessive behavior? Was this a purely restrictive anorexic patient (who restricts food intake but does not binge and purge)? Or does she binge and purge? Or purge during bouts of intensive exercise? Or display ritual behavior with food? Food obsession? Perfectionism? Kaye’s team had compiled the most detailed record ever made of patients with eating disorders—a mountain of data which, according to Devlin, “they knew in their hearts and minds would be useful, but they didn’t exactly know how to use.”

Devlin says he and his team spent “a year or two” working out the methodology for analyzing this wealth of data. The job required experts in the genetic basis of behavioral characteristics, experts in genetic analysis, and experts in statistical analysis.

In the 1990s, geneticists were beginning to rack up an extensive list of diseases linked to specific genes. They were filled with confidence that if a disease had a genetic component, that component could be uncovered. With the sequencing of the human genome, geneticists became even more confident of future successes.

But when Devlin ran data on eating disorders, he came up with lemons. His lab took 192 families with various eating disorders and processed the data. The links were weak, at best. Then they began using some of Kaye’s 100 behavioral covariables as filters. When they separated out the families in which at least two relatives had restricting anorexia nervosa—characterized by severe limitation of food and no bingeing or purging—the results clearly pointed to a region of chromosome 1. When they looked at bulimia—characterized by bingeing and purging—the results pointed to chromosome 10.

Their methods may shed light on other complex illnesses. After sequencing the human genome, geneticists around the world have been searching for new ways to mine the data for keys to disease. It’s becoming more and more clear that many diseases with genetic components aren’t like cystic fibrosis or hemophilia, in the sense that they won’t be linked to a single mutation. Devlin calls these “simple genetic diseases,” and he does not count eating disorders among them.

“The recognition that complex diseases were going to be so hard to crack really only came within the last decade,” he notes. When asked to explain why, he quips, “Optimism,” and laughs. With eating disorders, the underlying genetics can’t be linked to a single gene, or even to one region of the genome. Many genes are involved, and the linkage signals are much weaker, he explains. “The patterns are more difficult to see. You can’t find these chunks of DNA nearly as easily, and even when you do it’s not so clear what genetic variants in that region are responsible for the expression of liability to the disease.”

Devlin says that partly as a result of their study, geneticists have been eager to apply similar methods, i.e., using a large number of secondary characteristics to unravel complex genetic diseases, but most researchers are finding they simply don’t have data as detailed as what Kaye and his colleagues have been gathering since the mid-1990s.

Based on their promising results, Devlin and Kaye have won support from the NIMH

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for a \$10 million study of the genetics of anorexia nervosa—the first government-funded study of the genetics of anorexia nervosa. They hope to enlist 400 families with two or more cases of anorexia, a considerable challenge, but one that they expect will lead to more significant discoveries. Nine clinical centers around the world will join with WPIC to recruit families and gather data over the next five years.

Kaye recognizes that it may take a long time for public perception of eating disorders to change. There’s a deeply ingrained attitude that if anorexics weren’t exposed to images of shockingly thin super models they might cease being so “stubborn” and begin to eat more. While those images may contribute to the disease, the soft-spoken psychiatrist quietly challenges the facile nature of the assumption that they’re the primary culprit: “Anorexia has the highest death rate of any psychiatric illness. Ten to 15 percent of these people will die. People don’t starve themselves to death because of purely cultural influences. *You* try to starve yourself to death. *You* eat only 200 calories a day for a couple of months. How do you think you’d feel? There’s a powerful physiological drive to eat. There’s something wrong with that physiological drive in anorexia.”

We once believed that schizophrenia was caused primarily by environment, Kaye points out. Now, it is accepted that culture and family play a role, “but if you don’t have certain susceptibilities,” the psychiatrist says, “it’s not likely that you will develop the illness. The same thing is true of anorexia. It’s just that we’re 20 years behind in relation to other major psychiatric problems.”

At the Eating Disorders Clinic at WPIC, the beds don’t stay empty for long and neither do the maroon-upholstered chairs in the common room. The furniture is blocky and square, institutional but warm. There are usually several patients curled up in the chairs, reading or talking. Almost always, they are young women.

The dry-erase board behind the front desk lists about 15 patients. Dates of admittance show that one has been present for over a month, though most have been here for two to four weeks. Today, all are designated “201,” meaning they are here voluntarily. Sometimes,

that many of the 400 families recruited for the NIMH study will come from this clinic.

But the patients don’t come here for genetic research. This clinic is where the rubber meets the road—where the long arm of chromosome 10 doesn’t mean much of anything to a high school senior who weighs only 75 percent of her ideal body weight and is mortally afraid of gaining another pound. This clinic is where the gold standard is weight gain, not genetic analysis.

LaVia says they are finding treatments that work, many tailored to the specific diagnoses and the subcharacteristics of each eating disorder. Prozac and other drugs that work on the serotonin system are helping bulimics. There’s the promise of similar drug therapies for anorexics in the near future. Family therapy has been shown to help anorexics younger than 18. Cognitive-behavioral therapy—learning to understand your own behavior and embrace alternatives—is helping bulimics. The program is evidence based, so all of these approaches have been proven effective to

themselves by not eating, or by eating and throwing up.” She would love to see more patients identify with the analogy of, for example, a diabetic woman, who certainly did not choose to stop controlling her own insulin. She feels the same about parents who, at the end of their rope, ask their child, “Why can’t you just get over this?”

Gazing into the distant future, LaVia imagines a day when she might examine a patient, diagnose a specific eating disorder, and then order a genetic test to confirm the diagnosis—sort of like diagnosing an infection and confirming it with lab work. Such tests might help psychiatrists convince and enlist the help of parents who resist the diagnosis. “I can’t tell you how many families come in and say to me, ‘My daughter does not have an eating disorder,’” says LaVia.

She likes this notion that elements of psychiatry might begin to resemble other medical specialties. (Then, perhaps, fewer people would be surprised when she informs them that, yes, psychiatrists also went to medical school.) LaVia thinks this would benefit patients, too, as they struggle with managed care.

“I keep getting back to insurance,” she says by way of apology, “but from a clinical standpoint it’s a huge obstacle. In psychiatry in general there are limits to your insurance coverage. Per calendar year you may get 30 days of inpatient coverage, and you often get 26 outpatient visits. And I try to think of other medical illnesses: Would an insurance company say, ‘You can only be in the hospital for 30 days this year for your diabetes, and if you need to be in the hospital more than that, you’re going to have to pay for it out-of-pocket?’ You know, that sounds crazy.”

Like her colleague Kaye, LaVia finds eating disorders especially poignant partly because of the demographic they tend to afflict—90 to 95 percent are women in their teens or early 20s who are often trying to get an education and make important decisions. For physicians, who know what it’s like to work hard under pressure, it’s difficult not to identify with these patients and want to help them, says LaVia.

“They can go from being very good, very high-achieving kids to being kids who can’t do as much because their illness is interfering with their ability to either attend school or to continue in the path they had originally hoped to follow.” ■

*To take part in the genetic study of anorexia nervosa in families: 1-888-895-3886
www.angenetics.org*

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a code will change to 302, meaning that person tried to check out against medical advice and is now here involuntarily, usually a minor being treated with parental consent.

Some patients come from a long way off, including parts of Ohio, where the entire state is without inpatient treatment for eating disorders. Maria LaVia, the medical director of the clinic and attending physician for inpatients, says that treatment options for eating disorders have decreased around the country as centers have closed or cut back their programs. Managed care has pushed for less hospitalization and shorter stays across the board. Plus, many insurance companies now limit coverage for what they term “chronic mental illnesses.” Some explicitly cover all mental illnesses except eating disorders.

This clinic offers outpatient services, too, including a partial hospitalization program, where patients come five days a week, eat their meals, and go home in the evening. There’s an intensive outpatient program where patients come three evenings a week to eat dinner and have group, individual, or family therapy. This high volume of patients makes WPIC a good place to conduct research. LaVia, who is collaborating with Kaye and Devlin, hopes

some extent in the scientific literature. Methodologies supported by anecdotal evidence or having theoretical benefits, like the 12-step programs used at some centers, are not employed here.

The treatments for eating disorders address what we know about the disease—biochemistry, neurotransmitters, behavior, family environment, self-image, culture. These are not likely to change. Genetic makeup will never morph into determinism, nor will it fully exonerate cultural triggers like rail-thin models, fad diets, and the objectification of women. So how does genetic research change things for those struggling with eating disorders?

So far, hardly at all, says LaVia. “I think in general people have a hard time wrapping their minds around the fact that psychiatric illnesses are not all environmental. The theory was always this: If you are depressed, you just need to pull yourself up by the bootstraps, and get yourself out there, and cheer yourself up, and get on with life.”

But acceptance of a more biological paradigm could go a long way toward removing the stigma associated with eating disorders.

“Patients often blame themselves,” says LaVia, “because they’ve caused this illness