

Community study traces bulimia's origins

Young women afflicted with bulimia nervosa careen between feast and famine. Frantic binges of food consumption alternate with fasting, self-induced vomiting, and other efforts to shed weight.

Many potential culprits have been proposed to explain why 1 to 3 percent of females in many Western nations develop bulimia as teenagers or young adults. But a new study, which takes a rare look at this eating disorder among women not being treated by mental health clinicians, has considerably narrowed down the factors implicated in bulimia's emergence.

Current bulimia sufferers often viewed themselves with extreme disdain during childhood, had encountered certain types of conflict with their parents, and had grappled with obesity early in life, assert psychiatrist Christopher G. Fairburn of the University of Oxford in England and his coworkers. Bouts of depression or other psychiatric conditions often preceded their bulimia, the researchers add.

"This is a terribly important study," says psychologist Kelly D. Brownell of Yale University. "It's the best analysis by far of risk factors for bulimia."

Prior research on eating disorders had mainly focused on anorexia nervosa, which is marked by self-starvation and occurs much less often than bulimia.

Fairburn's group interviewed women and girls, ages 16 to 35, selected from the patient lists of general practitioners in urban and rural parts of southern England. The investigators compared 102 participants who were diagnosed with bulimia with two other groups: 102 individuals displaying some other psychiatric disorder (mostly depression or severe anxiety) and 204 volunteers who exhibited no psychiatric problems and cited no past mental disorders.

Many traits distinguished the bulimia group from psychologically healthy controls, the scientists report in the June ARCHIVES OF GENERAL PSYCHIATRY. These included childhood physical and sexual abuse, severe physical health problems, perfectionism, and parents who had suffered from depression.

However, only a few characteristics separated bulimia from other psychiatric conditions. These consisted of several risk factors for obesity and dieting—childhood or parental obesity, current dieting by other family members, and critical comments from family members about shape, weight, or eating habits.

Bulimia also developed more frequently in women who had begun to menstruate by age 12 than in those who began later. Early changes in body shape associated with puberty may represent another spur to dieting, Fair-

burn's team suggests.

Bulimic women said that their parents maintained minimal contact with them, expressed high expectations for their daughters' achievement, and had often abused alcohol. Self-esteem reached particularly low levels in the bulimia group.

Moreover, about two-thirds of those diagnosed with bulimia had first suffered from another psychiatric disorder.

"Our findings suggest that bulimia nervosa is most likely to develop in dieters who are at risk of obesity and psychiatric disorder in general," the investigators conclude.

The new study provides a "preliminary glimpse" at the forces underlying bulim-

ia, writes Katherine A. Halmi, a psychiatrist at Cornell University Medical College in White Plains, N.Y., in an accompanying comment.

Limitations of the British project, according to Halmi, include its reliance on women's self-reports of prior behavior, rather than data gathered from childhood into young adulthood, and its failure to interview family members.

A long-term study of bulimia's onset is sorely needed but unlikely to be conducted anytime soon because of its cost, Halmi notes.

For now, school programs aimed at quelling widespread dieting among girls may need to focus on those exhibiting high risks for obesity and psychiatric disorders in general, Fairburn and his coworkers propose. —B. Bower

Ovulation cycles linked to ovarian cancer

For the female body, ovulation is hard work. An ovary secretes hormones, produces an egg, thrusts it through a wall of tissue, and afterward repairs the rupture. Four weeks later, the process repeats.

Scientists have suspected that the frequency and rigor of tissue rebuilding can lead to ovarian cancer because, after each ovulation, the manufacture of new cells requires synthesis of DNA. This cell proliferation is thought to open the door to mutations in the *p53* gene, which produces one of the body's natural cancer fighters.

Now, a new study bolsters this incessant-ovulation theory (SN:10/31/92, p. 298) and its corollary that pregnancy, breast feeding an infant, or taking oral contraceptives lessens a woman's cancer risk by giving her welcome rests from ovulation and easing wear and tear on the ovaries.

The key element in this theory is the *p53* gene, which normally blocks cell division when a cell has sustained DNA damage. If the *p53* gene itself becomes disabled, however, it allows damaged cells to proliferate, possibly leading to cancer. A sure sign of a mutated *p53* gene, researchers have discovered, is overproduction of a distorted *p53* protein.

Duke University researchers tested for overproduction of altered *p53* protein in malignant tissue from 197 women, averaging 47 years old, with ovarian cancer. They found that 105 had such an overabundance. Moreover, the researchers calculated that these 105 women each averaged 388 ovulation cycles in her life so far, nearly 30 full years' worth, while the ovarian cancer patients without the *p53* protein surplus averaged only 342 cycles.

Compared with a group of 3,363 healthy women, the women who had cancer and an overabundance of the telltale *p53* protein were nine times more likely to have had high numbers of ovulation cycles. This difference emerged after the

researchers statistically accounted for variations in age, menopausal status, and number of children, says Joellen M. Schildkraut, an epidemiologist from Duke University Medical Center in Durham, N.C., and lead author of the study, which appears in the July 2 JOURNAL OF THE NATIONAL CANCER INSTITUTE.

The researchers computed the total number of ovulations by using the women's age and their responses to survey questions. The researchers calculated menstruation to start, on average, at age 12. They then multiplied the fertile years by 13 periods per year and subtracted out the months when the women were pregnant, breast-feeding a child, or taking oral contraceptives.

The *p53* protein findings lend credence to the incessant-ovulation theory of ovarian cancer: As cells multiply each month to repair the breach in the ovarian wall, mistakes in DNA replication make some mutations more likely. "For years that's been thought to be the mechanism," says Alice S. Whittemore, a Stanford University School of Medicine epidemiologist. "This supports that theory."

Meanwhile, many of the ovarian cancer patients in the study didn't have an abundance of the abnormal *p53* protein, which suggests the cancer can use another line of attack besides taking advantage of a mutant *p53* gene, Schildkraut says. Whittemore agrees that the study raises an obvious question: "What's causing the other cancers?" So far, no one knows.

Because the average age of an ovarian cancer patient at the time of diagnosis is 59, the patients in the Duke University study, who ranged in age from 20 to 54, may not represent the majority of ovarian cancer patients, Schildkraut says. The findings leave open the possibility that later onset ovarian cancer may have a different cause. For these reasons, the study needs to be replicated on older women, she says. —N. Seppa