Behavior

Bruce Bower reports from San Francisco at the annual meeting of the American Psychiatric Association

Tracing bulimia's roots . . .

Some clinicians have noted that women suffering from the eating disorder bulimia often report being sexually abused as children. That abuse, in their view, helps produce adult bulimics' urges to eat in binges and then purge their caloric excess through self-induced vomiting or other methods. However, researchers have uncovered little direct evidence for a link between childhood sexual abuse and bulimia.

A new study now finds that prolonged physical and psychological abuse, rather than sexual abuse, are more characteristic of the early family experiences of bulimic women.

"We see the pervasive effect of a type of childhood psychological water torture once practiced on bulimic women that shapes their sense of themselves and their self-esteem," asserts study director Joel Yager of the University of California, Los Angeles. "It's not just one traumatic abuse incident."

Yager's group examined 40 women currently diagnosed as bulimic, 40 formerly bulimic women who were symptom-free for at least one year, and 40 women who had never developed an eating disorder of any kind. The investigators used a series of interviews and questionnaires to probe the women's histories of childhood sexual abuse, physical abuse (such as regular spanking, punching, and beatings by parents), and psychological abuse (including persistent yelling, insults, guilt-inspiring statements, and ridicule from parents).

More frequent and extensive physical and psychological abuse occurred in the two bulimic groups, compared with the controls. But sexual abuse alone did not show up more often among bulimics, Yager says.

However, sexual abuse in combination with one or both other types of abuse appeared to boost the severity of bulimia in some women, he adds.

Long-standing psychological and physical abuse typically emerged in hostile, emotionally disturbed families, Yager says.

A few bulimic women reported no prior abuse of any kind and relatively stable early family lives. The roots of binging and purging in these cases remain unclear, he contends.

Yager's data and previous studies offer no support to those who claim that childhood sexual abuse boosts the risk of developing bulimia as an adult, argues James I. Hudson of McLean Hospital in Belmont, Mass.

. . . to early sexual abuse

Other researchers consider childhood sexual abuse to have a potentially stronger influence on bulimia than that suggested by studies such as Yager's.

Bulimic women may sometimes fail to recall early experiences of sexual abuse, maintains David B. Herzog of Massachusetts General Hospital in Boston. Painful memories of this sort may get pushed out of consciousness. And even if they emerge, some bulimics may fear others will view them as disgusting or brush them off if they reveal these incidents, Herzog points out.

Intensive two-hour interviews conducted with 20 bulimic women by Herzog and his co-workers yielded evidence of childhood sexual abuse in 13. Of those, 12 reported having experienced sexual abuse before their bulimia first appeared, Herzog says.

The interviews proved emotionally draining for both the bulimic women and the investigators, he notes. In telephone interviews conducted six weeks after the study, six of the 13 women citing sexual abuse also reported that they developed sleep problems following their face-to-face interview. But that session did not provoke a worsening of bulimic symptoms. Most women said that they had talked about their past sexual abuse only because they knew they would never see the interviewer again, Herzog says.

Biomedicine

Elizabeth Pennisi reports from Orlando, Fla., at the annual meeting of the American Association for Cancer Research

'Anti-gene' therapy shows promise . . .

Scientists are expanding the meaning of gene therapy to include treatments that thwart gene expression. Using gene-like material, these researchers target not the genes themselves, but messenger RNA, the intermediary that passes a cell's genetic information along and makes possible the production of proteins.

One approach involves small pieces of synthetic DNA, called antisense (SN: 6/10/89, p. 360). Chemical differences in the structure of antisense make this form of nucleic acid more resistant than normal DNA to being broken down by cells.

Like DNA, antisense molecules carry much more information than a typical drug molecule, says Stanley T. Crooke of Isis Pharmaceuticals in Carlsbad, Calif. This extra information enables them to home in on specific tumor targets better than traditional cancer treatments: Scientists design each antisense molecule to bind to and disarm or destroy a particular RNA.

At the University of Nebraska Medical Center in Omaha, Eliel Bayever and his colleagues have conducted a preliminary evaluation of one type of antisense in 11 people suffering from either an acute or premalignant form of myelogenous leukemia. In contrast to conventional chemotherapy for the disease, the antisense drug so far seems safe enough for patients to take in a doctor's office rather than in the hospital, says Bayever.

Another antisense molecule, this one directed against the RNA involved in the synthesis of a growth-regulating hormone, may prove useful against chronic myelogenous leukemia, says Alan M. Gewirtz of the University of Pennsylvania School of Medicine in Philadelphia. He is seeking permission from the U.S. Food and Drug Administration to test this molecule in patients.

. . . when melded with gene transfer

For a different twist on gene and antisense therapies, Jack A. Roth at the University of Texas M.D. Anderson Cancer Center in Houston packages antisense in a disabled mouse leukemia virus, much as gene therapists use viruses to transfer genes. In a planned study of 14 people with advanced lung cancer, Roth and his colleagues will administer virus containing either antisense or a functional tumor-suppressor gene, p53. The antisense will block the activity of a tumor-promoting gene in patients whose tumors contain mutations in this oncogene. The other treatment will restore p53 function to those whose tumors lack p53 or contain mutations in that gene, Roth says.

. . . and in the form of catalytic RNA

In another anti-gene approach, scientists make their own ribozymes—bits of catalytic RNA that chew up other strings of this nucleic acid (SN: 12/22&29/90, p.390). They design each ribozyme to target a specific messenger RNA, usually an RNA generated by mutated genes or oncogenes.

At Mount Sinai School of Medicine in New York City, Hiroyuki Kobayashi has made a particular "hammerhead" ribozyme, so named because of its molecular shape. It seeks out the RNA for a protein that pumps anticancer drugs out of tumor cells, making them resistant to medication. Tests in cells grown in the laboratory show that the ribozyme does chop up this RNA.

To improve on nature's ribozymes, one research team has substituted DNA for some of the RNA in these molecules. The DNA portion recognizes the base-pair sequence of the target RNA, and the RNA part breaks the sequence apart, explains John J. Rossi of the Beckman Research Institute of the City of Hope in Duarte, Calif. He and his colleagues expect these chimeric ribozymes to last longer and destroy RNA more efficiently than pure ribozymes. So far, one has proved six times more efficient than its all-RNA counterpart, says Rossi. He and his co-workers are testing the ability of these molecules to get rid of cancer-promoting gene products found in the bone marrow of people with chronic myelogenous leukemia.