# Behavior

*Wray Herbert reports from New York City at the meeting of the American Psychiatric Association*

## Modeling bulimia

Bulimia is an eating disorder characterized by binging and vomiting. Bulimics — mostly women — gorge themselves on as much as 20,000 calories worth of sweets and high-fat foods, then force themselves to vomit as a method of weight control. Although some bulimics are normal weight and some are even overweight, others suffer from anorexia nervosa, a disorder of self-imposed starvation. One question that has interested scientists is how bulimics can eat as much as they do: why don't they become satiated? Scientists at the New York Hospital in White Plains have now devised an animal model of bulimia, and their preliminary evidence from the model suggests that the disorder may be linked to a malfunction of certain gut-brain peptides involved in satiety. Psychiatrist James Gibbs and his co-workers have been working with rats that have been surgically altered so that food never reaches the stomach; using this "sham feeding" technique, they have been able to study the effects of taste on satiety and eating behavior.

What they have found is that, when the animals are getting no information about satiety from the stomach, they eat significantly more when the food is richer. But normally there are two peptides in the gut — called bombesin and cholecystokinin — that are released by food ingestion and circulate to the brain to induce satiety and inhibit eating. When these chemicals were given to the rats prior to feeding, they did inhibit feeding of bland food, but they were much less effective (bombesin failed completely) in controlling eating of sweet food. The potency of these natural peptides to satiate is limited by the force of sweet taste, the researchers conclude. This finding helps explain why bulimics always binge on rich foods, and it also points to the possibility of a disturbance in the central nervous system — where the competing signals of sweetness and satiety are presumably integrated.

## Anorexia and depression

Anorexia nervosa is often accompanied by classical symptoms of depression — melancholy mood, suicidal thoughts, and in some cases actual suicide — and the prevalence of depression is high among the relatives of anorectics. In order to study the possible connection between these two disorders, Jack L. Katz and his colleagues at the Montefiore Medical Center in New York did sleep polygraphs, laboratory tests and behavioral examinations on 20 young women with anorexia (including some bulimics) and ten normal controls. They found that a subgroup had a sleep abnormality typical of depression called short REM (rapid eye movement) latency: they tend, that is, to go very quickly into dream sleep. This abnormality was strikingly correlated with depressed mood. These patients also had very high levels of cortisol in their urine — a condition that suggests an overactive adrenal gland, which is quite common among depressed patients. All of the depressed anorectics were bulimic. It is unclear, the researchers say, whether anorexia develops as a defense against depression or as a result of an appetite disturbance intially associated with depression, but there appears to be a subtype of depression that is manifested primarily as an eating disorder.

## Brighter mood

The tendency of depressed people to move very quickly into REM sleep is thought by some to be just one manifestation of a generally disordered circadian clock; depressed people also tend, for example, to wake up abnormally early, and their cortisol levels seem to cycle slightly ahead of the normal pattern. Because light and dark are known to affect the secretion of a brain chemical called melatonin, it has been postulated that the use of artifical light might, by readjusting the circadian cycle, be therapeutic in treating depression; and last year scientists reported success in treating "winter depression," a seasonal disorder presumbly linked to changes in day length (SN: 3/27/82, p. 212). Now a team of California scientists has identified what they call a "critical photoperiodic interval" for treating depression with artificial light — between 5:00 and 6:00 in the morning — and they say they have successfully treated patients who remain seriously depressed year around. Psychiatrist Daniel F. Kripke and colleagues at the University of California at San Diego exposed 24 hospitalized patients to bright white light during the critical dawn hour and found that the patients' moods improved significantly by the following afternoon. Patients exposed to dim red light either at the same time or from 1:00 to 2:00 a.m. showed no improvement, indicating, according to Kripke, that sleep deprivation alone was not causing the patients' elevation in mood. Although the symptoms of depression tended to reappear when treatment was discontinued, five continuous days of exposure to bright light seemed to bring about a more lasting improvement in mood.

## A drug to remember

Amnesia resulting from heavy drinking may be a source of embarrassment for those who have had one too many, but it is of great interest to scientists studying normal memory processes. Government scientists who have been studying such amnesia report that they have found a drug that reverses the memory impairment caused by drinking — a discovery that points to one possible neurochemical pathway involved in learning and memory. According to psychiatrist Markku Linnoila and psychologist Herbert Weingartner of the National Institute of Mental Health, an experimental drug called zimelidine was given to healthy men in two experiments: in the first, it was followed three hours later by a dose of alcohol sufficient to cause memory impairment; in the second, zimelidine was given for 10 days, followed by alcohol on the last three days. In each case, the subjects were tested on various memory tasks a few hours after drinking, and it was found that the 40 percent loss of memory caused by drinking was completely reversed by the drug. Zimelidine taken by itself did not seem to improve normal memory. According to Linnoila, there was no chemical interaction between the drug and the alcohol itself, suggesting that the drug must have had its effect on the neurochemicals involved in memory. Because zimelidine, an anti-depressant drug, is known to work by increasing the supply of serotonin in the brain, Linnoila suspects that it has its effect on memory by reversing the effect of alcohol on the serotonin messenger system. The behavioral effect appears to be an elimination of one of alcohol's rewards, the ability to temporarily forget about the world; these men remember not only test items, but they also retain very clear memories of their disinhibited behavior while drinking.

## Predicting psychotic breaks

The neuroleptic drugs used to treat schizophrenia often produce undesirable side effects with long-term use, and scientists have become interested in the possibility of switching from a drug maintenance strategy to rapid drug intervention when a psychotic break is about to occur. Douglas W. Heinrichs and William T. Carpenter of the Maryland Psychiatric Research Center in Catonsville have been studying schizophrenics who experience early relapse to see if they could find reliable predictors of imminent psychosis; they report that four symptoms — hallucinations, suspiciousness, sleep disturbance and heightened anxiety — are sufficient to spot 90 percent of relapses. And for the most part, they say, a patient's behavioral signals are consistent from one relapse to another.