Activity-based Anorexia: A Biobehavioral Perspective

W. Frank Epling, Ph.D.
W. David Pierce, Ph.D.

A biobehavioral model of activity-based anorexia is examined in terms of recent evidence. Strenuous exercise reduces the value of food reinforcement and results in decreased food intake. Reduction of food intake increases the motivational value of physical exercise. This produces an escalation in activity that further suppresses appetite. Cultural practices of diet and exercise initiate this anorexic cycle, and once started the process is resistant to change. These anorexias may be the result of natural selection favoring those organisms that became active in times of food scarcity. Proximate physiological mechanism(s) appear to involve the endogenous opiate system that mediates the relationshp between running and eating. It is argued that classification of human self-starvation should be based on environmental and/or biological conditions that control food regulation. Activity anorexia may be one instance of such a classification that could account for many instances of "anorexia nervosa."

Our research has convinced us that a significant number of human anorexias are the result of biobehavioral processes triggered by cultural practices of diet and exercise. These processes involve the interrelation of deprivation and food schedule on physical activity and activity on food consumption (Epling, Pierce, & Stefan, 1983). From this perspective, such relationships are central to an understanding of many instances of human self-starvation.

Briefly stated, cultural requirements for health and thinness affect eating patterns and encouarge physical exercise. Strenous exercise (e.g., running) works to suppress appetite. This decrease in the value of food reinforcenment (Pierce, Epling, & Boer, 1986) results in a decline of food intake and body weight. As body weight declines, the motivational value of activity increases, and this leads to an escalation in physical exercise that further reduces the value of

---

W. Frank Epling, Ph.D. is Associate Professor of Psychology at University of Alberta, where **W. David Pierce, Ph.D.,** is Associate Professor of Sociology and Director, Center for Experimental Sociology. *Correspondence and reprint requests should be addressed to W. D. Pierce, Department of Sociology, The University of Alberta, Edmonton, Alberta, Canada, T6G 2H4.*

International Journal of Eating Disorders, Vol. 7, No. 4, 475–485 (1988)

CCC 0276-3478/88/040475-11$04.00

eating. Once initiated this cycle is "self-maintaining" and resistant to change.

Contemporary cognitive theories of anorexia nervosa have recognized that cultural practices are important to an understanding of the disorder (Garner & Garfinkel, 1978). From this perspective, an individual develops a prototypic image of the ideal person based on cultural standards. Television, magazines and other sources have frequently conveyed stereotypes of the "perfect" body. A person who has internalized these standards may become anorexic if these social cognitions are activated by situational and/or personal factors. Although the social-cognitive model offers an explanation of the tendency to diet and exercise in Western culture, it does not recognize the fundamental interrelations among food intake, physical activity, and body weight. This omission is critical because clinical reports indicate that 38–75% of anorexia nervosa patients are hyperactive (Crisp, Hsu, Harding, & Hartshorne, 1980; King, 1963; Kron, Katz, Gorzynski, & Weiner, 1978). Thus, a significant number of human anorexias may be activity induced.

EVIDENCE FOR ACTIVITY-BASED ANOREXIA

In the laboratory, the process of activity anorexia begins when rats are fed a single daily meal and given the opportunity to run on an activity wheel. These animals show increasing amounts of wheel running over days. As running increases, a suppression of eating occurs that is associated with a decline in body weight. The loss of weight further increases wheel running to excessive levels, and food intake may drop to less than 1 gram per day. If this process is allowed to continue the animals die of "self-starvation" (Routennberg, 1968; Routtenberg & Kuzenof, 1967). Importantly, control animals exposed to the same apparatus and food schedule, but *not allowed to run,* survive. This process of activity anorexia extends to other species (Epling, Pierce, & Stefan, 1981; Vincent & Pare, 1976), and Epling et al. (1983) have argued that it may account for some human self-starvation.

Activity and Anorexia

The importance of excessive activity to the onset of anorexia has been established by two facts; (1) food-deprived animals (fed a single-daily meal of 90 minutes or more) that do not run always survive, and (2) food-deprived animals that run excessively always die of starvation. A second line of evidence has demonstrated that variation in opportunity to run affects level of activity and the chances that anorexia will develop (Epling & Pierce, 1984). As the opportunity to run was varied from 2 to 22 hours the incidence of "strong" anorexias (or severe self-starvation) increased.

Excessive activity has been frequently noted by researchers of anorexia nervosa (Blitzer, Rollins, & Blackwell, 1961; Crisp, 1965; Halmi, 1974; King, 1963; Kron et al., 1978; Slade, 1973; Thoma, 1967). These investigators often view high rates of physical activity as one of several ways that the anorexic reduced calories and weight. Based on this view, excessive activity is only a "secondary symptom" of the disorder.

In constrast to this view, Kron and his colleagues (1978) conducted a retro-

spective study of hospitalized anorexics and concluded that hyperactivity is a "central" feature of anorexia nervosa. This conclusion is further supported by recent evidence on the temporal sequence of "symptoms" of patients with anorexia nervosa. Beumont, Booth, Abraham, Griffiths, and Turner (1983) asked 25 anorexics to identify their symptoms and the order of occurrence. Of the 28 reported symptoms, only "manipulating food servings" and "increased sport activity" were present in all patients. Generally, the ordering of the symptoms indicated that behaviors involving dieting and food restriction occur early in the sequence. These changes in food allocation were followed by increased sport activity and "exercising alone." It is apparent that many patients with "anorexia nervosa" develop the disorder in a manner that is similar to the development of activity anorexia in other species.

Activity and Food Intake

There is evidence that both human and nonhuman subjects reduce food intake when activity becomes excessive. Correlational studies with humans have shown that exercise is related to decreases in calorie intake (Edholm, Fletcher, Widdowson, & McCance, 1955; Epstein, Masek, & Marshall, 1978; Johnson, Mastropaolo, & Warton, 1972; Mayer, Roy, & Mitra, 1956; Watt, Wiley, & Fletcher, 1976). Experimental research with nonhuman subjects has demonstrated a causal relationshp between activity and eating. Several investigations have shown that free-feeding sedentary rats reduced food intake as a function of spontaneous or forced exercise (Ahrens, Bishop, & Berdanier, 1972; Katch, Martin, & Martin, 1979; Levitsky, 1974).

This reduction in food consumption appears to be temporary when activity is moderate and stable. Tokuyama, Saito, and Okuda (1982) showed that food reduction by rats would recover after 5–10 days of running-wheel activity. In their study, an initial drop in food consumption was followed by an increase that exceeded baseline food intake (also see Meyer, Marshall, Vitale, Christensen, Masayeki, & Stare, 1954). Epling and Pierce (1984) suggested that *rate of change* in daily amount of activity is necessary for food reduction. Sedentary animals (e.g., confined to home cage) will reduce intake if they run at some minimal rate (i.e., revolutions/day). Food reduction will continue only if the number of revolutions per day continues to exponentially increase. If activity stabilizes or increases linearly, food intake eventually compensates for increased energy expenditure.

Research by Kanarek and Collier (1979, 1983) suggest that activity affects duration rather than initiation of eating. In one study (Kanarek & Collier, 1979) rats were required to run on a wheel in order to obtain access to a meal. As the running requirement was increased to 640 revolutions, number of meals per day decreased, but the animal "compensated" by increasing the amount eaten per meal. However, at a higher wheel-running requirement (2560 revolutions) animals ate less frequently but did not increase the amount consumed. Higher levels of activity appeared to affect the termination of eating (i.e., satiety) since animals initiated eating at each opportunity but did not eat long enough to maintain body weight.

Although satiation effects are highlighted by Kanarek and Collier (1979), they report that sometimes the animals failed to take a meal even though they

had completed the wheel-running requirement. This observation suggests that the "desire for food" may be altered by physical activity. Recent evidence by Pierce et al. (1986) adds support to this contention. In this study, rats pressed a bar to obtain food pellets. The number of bar presses per pellet was systematically increased until the animal gave up pressing. This "giving-up" point was used to index the reinforcing effectiveness of food. The reinforcing effectiveness of food was measured after wheel running or after a sedentary periood of time. Prior exercise, either voluntary or forced, reduced the reinforcing effectiveness of food.

Excessive physical activity apparently reduces food consumption in humans and other animals. This reduction of intake depends on several factors. Severe reduction of food intake occurs when activity exponentially increases over days. Importantly, suppression of eating is a temporary effect if the level of activity does not change over time (i.e., adaptation). For example, most long distance runners maintain a constant weekly milage and eat at levels that may exceed their sedentary baseline. However, when exercise is increasing against prior levels of exertion, suppression of food intake will continue. The sedentary person who begins a running program or the athlete who increases training may experience a "loss of appetite." This loss of appetite may be related to satiation effects during eating and to a decline in the reinforcing effectiveness of food.

Food Intake and Activity

The relationship between activity and food consumption appears to be reciprocal. Human studies suggest that increased body weight and food consumption are associated with inactivity (Bloom & Eidex, 1967; Mayer, 1965). Additionally, increased mobility and arousal have been described for starving populations exposed to economic hardship (Howard, 1839), malnourished German school children during Word War I (Blanton, 1919), and starving people in post-World War II Germany (Russell Davis, 1951). Of course, when starvation becomes extreme, activity decreases. The survivors of Bensen concentration camp were close to death and very inactive when liberated (Lipscomb, 1945). This same immobility was described for the slow starvation of men in prisoner-of-war camps (Leyton, 1956). Apparently, during the early stages of food restriction, physical activity increases, but as starvation continues it eventually declines. Although these human reports are interesting, the evidence is anecdotal and circumstantial. Stronger evidence for the effects of food consumption on activity can be found in the nonhuman literature on diet and activity.

There is experimental evidence that food deprivation affects level of wheel running in rats. Many studies have shown that depriving rats of food increases wheel running (Cornish & Mrosovsky, 1965; Finger, 1951; 1965; Hall & Hanford, 1954; Reid & Finger, 1955). Apparently, food restriction alone is sufficient to induce some level of activity.

Although activity increases with hours of food deprivation, there may also be an effect of meal schedule. When rats are fed once per day, activity is organized around the feeding period (Honma, Goetz, & Aschoff, 1983; Reid & Finger, 1955; Richter, 1922; Searle & Brown, 1938; Shirley, 1928; Woods, 1969). In addition researchers have noted excessive wheel running by rats when the

number of meals is changed from free-feeding (i.e., 12–16 eating bouts) to one meal per day (Routtenberg, 1968; Routtenberg & Kuznesof, 1967; Spear & Hill, 1962). Thus, the pattern and level of activity is related to food allocation.

There are, however, limitations to the generality of this statement. Although a single daily meal may increase activity, more frequent presentations of food prevent the development of excessive wheel running. In a study by Kanarek and Collier (1983), rats were fed (1) ad libitum, (2) a single 60-minute meal, (3) two 30-minute meals, or (4) four 15-minute eating opportunities. In terms of numer of wheel revolutions per day, the most running occurred for the one meal per day group. For these animals, substantial increases in running were observed over days. Two 30-minute feeding bouts also increased activity, but the rate of increase was far less than the observed for the single-meal subjects. Animals exposed to four 15-minute feeding bouts showed no increase in wheel running and were similar to the ad libitum group.

In addition to meal pattern, dietary properties of the meal may contribute to changes in physical activity. Evidence suggests that the carbohydrate-to-protein ratio of the diet is important. Experiments have shown that rats will increase wheel running as the relative amount of carbohydrate is increased (Baumeister, Harkins, & Cromwell, 1964; Chiel & Wurtman, 1981; Collier & Squibb, 1967; Reed, 1947). A report by Richter (1977) on appetite and sugar (a simple carbohydrate) suggests that high levels of carbohydrate may generate excessive wheel running (up to 40,000 revolutions per day). This effect occurred when rats were fed only dextrose and water. Thus, the absolute amount of carbohydrate in the diet may be as important as the carbohydrate-to-protein ratio.

Generally, physical activity increases or decreases with changes in body weight. For example, as body weight declines, people and other species become more active. In addition to variation in body weight, the evidence suggests that the meal schedule may have independent effects on level of activity. In rats, a single-daily meal of 1–2 hours duration generates substantial increases in wheel running. The importance of a "single daily meal" for induced activity in humans has not been demonstrated. However, animal research implies that human meal schedules play some role in generating excessive activity.

THE BIOLOGICAL BASIS OF ACTIVITY ANOREXIA

A theory of activity anorexia must explain the tendency to exercise under conditions of food restriction and the interrelation between exercise and eating. In addition, the theory must show how this kind of self-starvation generalizes to humans. Our interpretation rests on the assumption that activity anorexia depends on basic biological processes (Epling et al., 1983).

Evolutionary Basis

An evolutionary account of the interrelation between eating and running points to the survival value of such behavior. During times of food scarcity, organisms can either stay and conserve energy (e.g., hibernate) or become mo-

bile and travel to another location (e.g., migration). The particular strategy adopted by a species depends on natural selection. Thus, if travel leads to reinstatement of food supply and remaining leads to starvation, then those animals that travelled would reproduce and increase the likelihood of this behavior in the population. Many species increase locomotor activity in times of food shortage (Vincent & Pare, 1976), and this appears to extend to *Homo sapiens.*

The fact that increasing energy expenditure (e.g., by running) is accompanied by decreasing caloric intake violates common sense. From a homeostatic perspective, food intake and energy expenditure should be positively related. In fact, this is the case if an animal is given time to adjust to the new level of activity and food is *not* restricted (Tokuyama, Saito, & Okuda, 1982). However, during periods of food scarcity, travel should not stop when food is infrequently contacted, since stopping to eat may be negatively balanced against getting to a richer-food patch. Increasing contact with food would indicate a replenished supply, and travel should reduce or stop at this time. This assumption has tentative support since wheel running by anorexic animals is reduced when meal frequency is increased, but the total time of access to food is held constant (Kanarek & Collier, 1983). In humans, sociocultural facors (see below) can produce and maintain conditions of food depletion even though food is readily available (e.g., dieting to lose weight). Thus, anorexics may not experience increased contact with food, activity continues to spiral upward, and food consumption remains low or declines.

An evolutionary analysis provides a useful heuristic for behavioral research on activity anorexia. However, the explanation is based on "ultimate" causation, and the phylogenetic history if only indirectly inferred. Another kind of biological explanation is based on "proximate" causation involving specific physiological mechanisms.

Physiological Basis

Pierce et al. (1986) have shown that food deprivation increases the reinforcing effectiveness of running and that running decreases the reinforcing effectiveness of food. These behavioral observations suggest that some physiological mechanism(s) links the motivational value of eating and running. Recent evidence points to the role of endogenous opiates as mediators of the relationship between running and eating. One hypothesis is that excessive physical activity activates one or more endogenous opioids that reduce appetite. In support of this conjecture, Davis, Lamb, Yim, and Malven (1985) found that exercise-trained rats ate less following injection with an endogenous opioid stimulus (2-deoxy-D-glucose). Additionally, Sanger and McCarthy (1980) have shown that food-deprived animals eat less when injected with morphine. This suggests that exercise-induced opiate release would decrease eating *under conditions of food deprivation.*

Research with humans also supports an opioid hypothesis. Appenzeller, Standefer, Appenzeller, and Atkinson (1980) found elevated endorphin levels in endurance runners that lasted 2 hours following a marathon. Farrell, Gates, Maksud, Morgan, and Tseng (1981) showed that intense exercise in humans was related to increases in plasma beta-endorphin. Elevated opiate levels have

also been measured in diagnosed anorexics (Kaye, Pickar, Naber, & Ebert, 1982). Nakai, Kinoshita, Koh, Tsujii, and Tsukada (1987) reported that 2-deoxy-D-glucose increased judgements of hunger in normal-weight subjects but decreased these ratings in anorexics. This effect is in accord with animal research (see above). Finally, naloxone, an opioid antagonist, results in weight gain by anorexics (Moore, Mills, & Forster, 1981). This suggests an opiate suppression of eating induced by the intense exercise reported for these patients (Beaumont et al., 1983). Although the effects of running on eating may be mediated by endogenous opiates, no mechanism has been identified to explain the effects of food deprivation on physical activity.

There are interesting links between endogenous opiates, exercise, and menstrual problems that suggest the possibility of a human activity anorexia mediated by the opioid system. A prominent feature of anorexia nervosa in women is the cessation or delay of menstruation (Brown, 1983; Garfinkel & Garner, 1982). Similar menstrual problems have been reported for women athletes (Dale, Gerlach, & Wilhile, 1979; Feicht, Johnson, Martin, & Wagner, 1978; Frisch, Gotz-Welbergen, McArthur, Albright, Witschi, Bullen, Birnholtz, Reed, & Hermann, 1981; Speroff & Redwine, 1980). Physiological mechanisms controlling menstrual cycle are well known and involve endocrine changes arising from the "hypothalamic-pituitary-gonadal axis." Research has shown that strenuous exercise may reduce pulsatile-luteinizing hormone (LH) that contributes to the regulation of menstrual cycle and ovulation (Cumming, Vickovic, Wall, Fluker, & Belcastro, 1985; Cumming, Vickovic, Wall & Fluker, 1985). Importantly, increases in endogenous opiates appear to reduce LH levels (Robert, Quickley, & Yen, 1981).

In summary, exercise increases endorphin levels. Increased level of endorphin appears to decrease appetite when food is restricted. Anorexia-nervosa patients, and women athletes, show elevated opiate levels. Both women athletes and anorexics have a high incidence of menstrual problems. Elevated opiate levels reduce LH and this hormone is associated with the control of menstrual cycle. Thus, women who are diagnosed as having "anorexia nervosa" on the basis of reduced eating and menstrual problems may *actually* be exhibiting a form of activity-based anorexia.

EXTERNAL VALIDITY, CULTURE, AND CONTINGENCIES OF REINFORCEMENT

The most convincing evidence for activity-based anorexia comes from laboratory research with animals. In the laboratory situation, the experimenter controls food allocation and access to activity. In contrast, humans seem to "self-regulate" food intake and exercise. Given this difference, there is some reason to question the generality of the animal model. Recently, Mrosovsky (1984) commented on our animal model of self-starvation and stated that "since the experimenter, not the rat, imposes the 1-h feeding regimen, the term self-starvation is silly. To the extent that the phenomenon has a human counterpart, the patient herself adopts the role of the experimenter in setting the eating patterns. The animal analogy offers no insight into why she does this (p. 26).

The animal model describes a basic biobehavioral process that may extend to

humans. The model does not, and cannot, depict the complex sociocultural conditions that predispose some individuals to contact the basic process. There is, however, reasonable evidence that socioculture factors set the conditions that predispose some individuals to diet and exercise (Garner, Garfinkel, & Olmstead, 1983). The person who is subjected to strong "social pressure" to diet does not "willfully" restrict food intake. In both rats and humans, food restriction is imposed. Based on this observation, if "self-starvation" is an inappropriate description of the animal's behavior, it is also an inappropriate label for the human phenomenon. Also, the patient is not the "experimenter"; rather, social contingencies of reinforcement encourage and maintain dieting to lose weight (Bachrach, Erwin, & Mohr, 1965; Hallsten, 1965). In this sense, the diet and exercise culture is the "experimenter."

This viewpoint suggests that choosing to diet or exercise is determined by biological and environmental conditions. The social environment consists of the practices of other people and is a major determinant of human behavior. What a person eats or drinks, how and when the person exercises, what "inner" feelings are reported, how persons describe themselves, and what kinds of personal relationships are formed all depend in part on contingencies of reinforcement set by the group. Such behavior becomes "appropriate" to the community when specific responses are socially reinforced while others are punished or ignored (e.g., extinction).

People learn many things from the social environment both by direct experience and by observation (Bandura, 1977). Those who share common demographic and social attributes often have similar learning experiences (Ullman and Krasner, 1975). However, some experiences and social models in a person's life are unique. One person may learn to eat in moderation, another individual may learn to eat to excess. In Western culture, the mass media conveys values of thinness and physical fitness (Bruch, 1978; Kurman, 1978; Wooley & Wooley, 1980). There is, however, considerable variability in the degree of acceptance of these values. Such variation relates to differential exposure to "the message," subcultural membership, community and family characteristics, and differences in social expectations by age and sex (Schwartz, Thompson, & Johnson, 1983).

CONCLUSION

Anorexia nervosa is a diagnostic category that is based upon the medical disease model of abnormal behavior. From this perspective, the anorexic's behavior is symptomatic of an underlying personality disturbance or distorted cognitive schemata. In contrast, the biobehavioral perspective adopted here suggests that classification of anorexias should be based on the environmental or biological conditions that control food regulation. Activity anorexia may be viewed as one type of self-starvation that may occur in humans.

There are other kinds of anorexias. Mrosovsky and Sherry (1980) described "animal anorexias" in several species that occur when biologically significant activities (molting, defense of territory, incubating eggs, etc.) interfere with obtaining and eating food. Although these natural anorexias may not extend to humans, there may be other types that do. At the very least, researchers

should consider the possibility that human self-starvation may not be a unitary disorder. Severe food restriction can be the result of diverse biobehavioral processes.

Authorship of this paper is shared equally. We would like to thank Judy Cameron, M. Ed., who provided helpful suggestions and editorial assistance. Our research on activity anorexia has been supported by a grant from Alberta Mental Health Research Council, to whom we are grateful.

REFERENCES

Ahrens, R. A., Bishop, C. L., & Berdanier, C. D. (1972). Effect of age and dietary carbohydrate source on the response of rats to forced exercise. *Journal of Nutrition, 102,* 241–248.

Appenzeller, O., Standefer, J., Appenzeller, J., & Atkinson, R. (1980). Neurology of endurance training 5: Endorphins. *Neurology, 30,* 418–419.

Bachrach, A. J., Irwin, W. J., & Mohr, J. P. (1965). The control of eating behavior in an anorexic by operant conditioning techniques. In L. P. Ullman and L. Krasner (Eds.), *Case studies in behavior modification* (pp. 153–163). New York: Holt.

Bandura, A. (1977). *Social learning theory.* Englewood Cliffs, N. J.: Prentice Hall.

Baumeister, A., Harkins, J. F., & Cromwell, R. L. (1964). Need states and activity level. *Psychological Bulletin, 61,* 438–453.

Beumont, P. J. V., Booth, A. L., Abraham, S. F., Griffiths, D. A., & Turner, T. R. (1983). A temporal sequence of symptoms in patients with anorexia nervosa: A preliminary report. In P. L. Darby, P. E. Garfinkel, D. M. Garner, and D. V. Coscina (Eds.), *Anorexia nervosa: Recent developments in research* (pp. 129–136). New York: Alan R. Liss.

Blanton, S. (1919). Mental and nervous changes in children of the Volkschulen of Trier, Germany, caused by malnutrition. *Mental Hygiene, 3,* 343–386.

Blitzer, J. R., Rollins, N., & Blackwell, A. (1961). Children who starve themselves. *Psychosomatic Medicine, 23,* 369–383.

Bloom, W. L., & Eidex, M. F. (1967). Inactivity as a major factor in adult obesity. *Metabolism, 16,* 679–684.

Brown, G. M. (1983) Endocrine alterations in anorexia nervosa. In P. L. Darby, P.E. Garfinkel, D. M. Garner, & D. V. Coscina (Eds.), *Anorexia nervosa: Recent developments in research* (pp. 231–247), New York: Alan R. Liss.

Bruch, H. (1978). *The golden cage.* Cambridge, Mass.: Harvard University Press.

Chiel, H. J., & Wurtman, R. J. (1981). Short-term variations in diet composition change the pattern of spontaneous motor activity in rats. *Science, 213,* 676–678.

Collier, G., & Squibb, R. L. (1967). Diet and activity. *Journal of Comparative and Physiological Psychology, 3,* 409–413.

Cornish, E. R., & Mrosovsky, N. (1965). Activity during food deprivation and satiation of six species of rodent. *Animal Behavior, 13,* 242–248.

Crisp, A. H. (1965). Clinical and therapeutic aspects of anorexia nervosa: A study of 30 cases. *Journal of Psychosomatic Research, 9,* 67–78.

Crisp, A. H., Hsu, L. K. G., Harding, B., & Hartshorne, J. (1980). Clinical features of anorexia nervosa: A study of a consecutive series of 102 female patients. *Journal of Psychosomatic Research, 24,* 179–191.

Cumming, D. C., Vickovic, M. M., Wall, S. R., & Fluker, M. R. (1985). Defects in pulsatile LH release in normally menstruating runners. *Journal of Clinical Endocrinology and Metabolism, 60,* 810–813.

Cumming, D. C., Vickovic, M. M., Wall, S. R., Fluker, M. R., & Belcastro, A. N. (1985). The effect of acute exercise on pulsatile release of luteinizing hormone in women runners. *Americal Journal of Obstetrics and Gynecology, 153,* 482–485.

Dale, E., Gerlach, D. H., & Wilhile, A. L. (1979). Menstrual dysfunction in distance runners. *Obstetrics and Gynecology, 54,* 47–53.

Davis, J. M., Lamb, D. R., Yim, G. K., & Malvern, P. V. (1985). Opioid modulation of feeding behavior following repeated exposure to forced swimming exercise in male rats. *Pharmacology and Biochemistry of Behavior, 23,* 709–714.

Edholm, O. G., Fletcher, J. G., Widdowson, E. M., & McCance, R. A. (1955). The energy expenditure and food intake of individual men. *British Journal of Nutrition, 9,* 286–300.

Epling, W. F., Pierce, W. D., & Stefan, L. (1981). Schedule-induced self-starvation. In C. M. Brad-

shaw, E. Szabadi, and C. F. Lowe (Eds.), *Quantification of steady-state operant behaviour* (pp. 393–396). Amseterdam: Elsevier/North Holland Biomedial Press.

Epling, W. F., Pierce, W. D., & Stefan, L. (1983). A theory of activity-based anorexia. *International Journal of Eating Disorders, 3,* 27–46.

Epling, W. F., & Pierce, W. D. (1984). Activity-based anorexia in rats as a function of opportunity run on an activity wheel. *Nutrition and Behavior, 2,* 37–49.

Epstein, L. H., Masek, B. J., & Marshall, W. R. (1978). A nutritionally based school program for control of eating in obese children. *Behavior therapy, 9,* 766–778.

Farrell, P. A., Cates, W. K., Maksud, M. G., Morgan, W. P., & Tseng, L. F. (1981). Plasma beta-endophine/beta-lipotropin immunoreactitity increases after treadmill exercise in man. *Medicine and Science in Sports and Exercise, 13,* 134.

Feicht, C. B., Johnson, T. S., Martin, B. J., Sparkes K. E., & Wagner, W. W. (1978). Secondary amenorrhea in athletes. *Lancet, 2,* 1145–1146.

Finger, F. W. (1951). The effect of food deprivation and subsequent satiation upon general activity in the rat. *Journal of Comparative and Physiological Psychology, 4,* 557–564.

Frisch, R. E., Gotz-Welbergen, A. V., McArthur, J. W., Albright, T., Witschi, J., Bullen, B., Birnholtz, J., Reed, R. B., & Hermann, H. (1981). Delayed menarche and amenorrhea of college athletes in relation to age of onset of training. *Journal of the American Medical Association, 246,* 1559–1563.

Garfinkel, P. E., & Garner, D. M. (1982). *Anorexia nervosa: A multidimensional perspective.* New York: Brunner Mazel.

Garner, D. M., & Garfinkel, P. E. (1978). Socioculture factors in anorexia nervosa. *Lancet,* (Sept.), 674.

Garner, D. M., Garfinkel, P. E., & Olmsted, M. P. (1983). An overview of sociocultural factors in the development of anorexia nervosa. In P. L. Darby, P. E. Garfinkel, D. M. Garner, & D. V. Coscina (Eds.), *Anorexia nervosa: Recent developments in research* (pp. 65–82). New York: Alan R. Liss.

Hall, J. F., & Hanford, P. V. (1954). Activity as a function of a restricted feeding schedule. *Journal of Comparative and Physiological Psychology, 47,* 362–363.

Hallsten, E. A. (1965). Adolescent anorexia nervosa treated by desensitization. *Behavior Research and Therapy, 3,* 87–91.

Halmi, K. (1974). Anorexia nervosa: Demographic and clinical features. *Psychosomatic Medicine, 36* 18–26.

Honma, K., Goetz, G., & Aschoff, J. (1983). Effects of restricted daily feeding on free running circadian rhythms in rats. *Physiology and Behavior, 30,* 905–913.

Howard, R. B. (1839). *An inquiry into the morbid effect of deficiency of food chiefly with reference to their occurrence amongst the destitute poor.* London, England: Simpkin, Marshall & Co.

Johnson, R. E., Mastropalo, J. A., & Wharton, M. A. (1972). Exercise, dietary intake, and body composition. *Journal of the American Dietetic Association, 61,* 399–403.

Kanarek, R. B., & Collier, G. H. (1979). Patterns of eating as a function of the cost of the meal. *Physiology and Behavior, 23,* 141–145.

Kanarek, R., & Collier, G. H. (1983). Self-starvation: A problem of overriding the satiety signal? *Physiology and Behavior, 30,* 307–311.

Katch, F. I., Martin, R., & Martin, J. (1979). Effects of exercise intensity on food consumption in the male rat. *American Journal of Clinical Nutrition, 32,* 1401-1407.

Kaye, W. H., Picker, D. M., Naber, D., & Ebert, M. H. (1982). Cerebrospinal fluid opioid activity in anorexia nervosa. *American Journal of Psychiatry, 139,* 643–645.

King, A. (1963). Primary and secondary anorexia nervosa syndromes. *British Journal of Psychiatry, 109,* 470–479.

Kron, L. Katz, J. L., Gorzynski, G., & Weiner, H. (1978). Hyperactivity in anorexia nervosa: A fundamental clinical feature. *Comprehensive Psychiatry, 19,* 433–440.

Kurman, L. (1978). An analysis of messages concerning food, eating behaviors, and ideal body image on prime-time American network television. *Dissertation Abstracts, 39A,* 1907–1908.

Levitsky, D. (1974). Feeding conditions and intermeal relationships. *Physiology and Behavior, 12,* 779–787.

Leyton, G. B. (1946). Effect of slow starvation. *Lancet, 3,* 73–79.

Lipscomb, F. M. (1945). Medical aspects of Belsen concentration camp. *Lancet, 2,* 313–315.

Mayer, J. (1965). Inactivity as a major factor in adolescent obesity. *Annals of the New York Academry of Sciences, 131,* 502–506.

Mayer, J., Marshall, N. B., Vitale, J. J., Christensen, J. H., Masayeki, M.B., & Stare, F. J. (1954). Food intake and body weight in normal rats and genetically obese adult mice. *American Journal of Physiology, 177,* 544–548.

Mayer, J., Roy, P., & Mitra, K. P. (1956). Relation between caloric intake, body weight, and physical work: Studies in an industrial male population in West Bengal. *American Journal of Clinical Nutrition, 4,* 169–175.

Moore, R., Mills, I. H., & Forster, A. (1981). Naloxone in the treatment of anorexia nervosa: Effect on weight gain and lipolysis. *Journal of the Royal Society of Medicine, 74,* 129–131.

Mrosovsky, N. (1984). Animal models: Anorexia yes, nervosa no. In K. M. Pirke and D. Ploog (Eds.), *Psychobiology of anorexia nervosa* (pp. 22–34). New York: Springer-Verlag.

Mrosovsky, N., & Sherry, D. F. (1980). Animal anorexias. *Science, 207,* 837–842.

Nakai, Y., Kinoshita, F., Koh, T., Tsujii, S., & Tsukada, T. (1987). Perception of hunger and satiety induced by 2-deoxy-D-glucose in anorexia nervosa and bulimia nervosa. *International Journal of Eating Disorders, 6,* 49–57.

Pierce, W. D., Epling, W. F., & Boer, D. P. (1986). Deprivation and satiation: The interrelations between food and wheel running. *Journal of the Experimental Analysis of Behavior, 46,* 199–210.

Reed, J. D. (1947). Spontaneous activity of animals: A review of the literature since 1929. *Psychological Bulletin, 44,* 393–412.

Reid, L. S., & Finger, F. W. (1955). The rats adjustment to 23-hour food-deprivation cycles. *Journal of Comparative and Physiological Psychology, 48,* 110–113.

Richter, C. P. (1922). A behavioristic study of the acvitivy of the rat. *Comparative Psychological Monographs, 1,* N. 2.

Richter, C. P. (1977). Six common sugars as tools for the study of appetite. In J. M. Weiffenback (Ed.), *Taste and development: The genesis of sweet preference* (NIH # 77–1068), Bethesda, Maryland: U. S. Department of Health, Education, and Welfare.

Ropert, J. F., Quigley, M. E., & Yen, S. S. C. (1981). Endogenous opiates modulate pulsatile luteinizing hormone release in humans. *Journal of Clinical Endocrinology and Metabolism, 52,* 583–585.

Routtenberg, A. (1968). Self-starvation of rats living in activity wheels: Adaptation effects. *Journal of Comparative and Physioloigical Psychology, 66,* 234–238.

Routtenberg, A., & Kuznesof, A. W. (1967). "Self-starvation" of rats living in activity wheels on a restricted feeding schedule. *Journal of Comparative and Physiological Psychology, 64,* 414–421.

Russell Davis, D. (1951). *Studies in malnutrition.* M. R. C. special report series no. 275. London: H. M. Stationary Office.

Sanger, D. J., & McCarthy, P.S. (1980). Differential effects of morphine on food and water intake in food deprived and freely-feeding rats. *Psychopharmacology, 72,* 103–106.

Schwartz, D. M., Thompson, M. G., & Johnson, G. L. (1983). Eating disorders and the culture. In P. L. Darby, P. E. Garfinkel, D. M. Garner, and D. V. Coscina (Eds.), *Anorexia nervosa; Recent developments in research* (pp. 83–94). New York: Alan R. Liss.

Searle, L. V., & Brown, C. (1938). The effect of subcutaneous injections of benzedrine sulfate on the activity of white rats. *Journal of Experimental Psychology, 22,* 480–490.

Shirley, M. (1928). Studies in activity. 11. Activity rhythms: Age and activity; Activity after rest. *Journal of Comparative Psychology, 8,* 159–186.

Slade, P. D. (1973). A short anorectic behavior scale. *British Journal of Psychiatry, 122,* 83–85.

Spear, N. E., & Hill, W. F. (1962). Methodological note: Excessive weight loss in rats living in activity wheels. *Psycological Reports, 11,* 437–438.

Speroff, L., & Redwine, E. B. (1980). Exercise and menstrual function. *Physician and Sports Medicine, 8,* 42–52.

Thoma, H. (1967). *Anorexia Nervosa.* New York: International Universities Press.

Tokuyama, K., Saito, M., & Okuda, H. (1982) Effects of wheel running on food intake and weight gain of male and female rats. *Physiology and Behavior, 23,* 899–903.

Ullmann, L. P., & Krasner, L. (1975). *A psychological approach to abnormal behavior.* Englewood Cliffs, N. J.: Prentice Hall.

Vincent, G. P., & Pare, W. P. (1976). Activity-stress ulcer in the rat, hamster, gerbil and guinea pig. *Physiology and Behavior, 16,* 557-560.

Watt, E. W., Wiley, J., & Fletcher, G. F. (1976). Effect of dietary control and exercise training on daily food intake and serum lipids in post-myocardial infarction patients. *American Journal of Clinical Nutrition, 29,* 900–904.

Woods, D. J. (1969). *The effects of age and chlorpromazine on self-starvation in activity wheels.* M. A. Thesis, Department of Psychology, Northwestern University.

Wooley, S. C., & Wooley, O. W. (1980). Eating disorders: Obesity and anorexia. In A. Brodsky and R. Hare-Mustin (Eds.), *Women and psychotherapy: An assessment of research and practice.* New York: Guilford Press.