Activity Anorexia: An Interplay Between Basic and Applied Behavior Analysis

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The relationship between basic research with nonhumans and applied behavior analysis is illustrated by our work on activity anorexia. When rats are fed one meal a day and allowed to run on an activity wheel, they run excessively, stop eating, and die of starvation. Convergent evidence, from several different research areas, indicates that the behavior of these animals and humans who self-starve is functionally similar. A biobehavioral theory of activity anorexia is presented that details the cultural contingencies, behavioral processes, and physiology of anorexia. Diagnostic criteria and a three-stage treatment program for activity-based anorexia are outlined. The animal model permits basic research on anorexia that for practical and ethical reasons cannot be conducted with humans. Thus, basic research can have applied importance.

Key words: activity anorexia, physical activity, animal models, behavior analysis

This paper addresses the relation between basic and applied research in behavior analysis. Unusual behavior in animals may occasionally be analogous to disordered behavior in humans. One example comes from our work on activity anorexia. Under certain environmental conditions, rats self-starve; this phenomenon is similar to seemingly willful starvation by humans. The development of an animal model and theory of activity anorexia illustrates one way that basic research has practical importance.

Anorexia nervosa is a psychiatric disorder characterized by a voluntary refusal to eat, extreme loss of weight, and in some cases, death. The syndrome poses a serious health hazard to otherwise healthy adolescents and young adults. The estimated incidence for anorexia may be as high as 3% of the population at risk, and mortality for diagnosed patients is between 5% and 21% (Yates, 1990). The *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1987) details the criteria for the diagnosis of anorexia nervosa. Several primary symptoms must be present before a person is classified as anorexic. These include behavior directed toward losing weight, weight loss, peculiar patterns of handling food, intense fear of gaining weight, disturbance of body image, and, in females, irregular or absent menstrual cycle (amenorrhea). Secondary features are not required for diagnosis but occur at some frequency. These include denial of illness and resistance to therapy, delayed psychosexual development in adolescents, and, in adults, loss of sexual interest. Several clinical researchers have presented alternative diagnostic criteria, but all have viewed anorexia nervosa as a mental or cognitive disorder that produces self-starvation (Bruch, 1965; Crisp, Hsu, Harding, & Hartshorn, 1980; Garner & Garfinkle, 1988).

Chaotic dieting, excessive activity, and physiological abnormalities are associated with human anorexia. Willful self-starvation by humans is usually diagnosed as anorexia nervosa, and patients are treated as mentally disordered. Our contention is that most cases of anorexia

Order of author is an old issue with us and (once again) was decided by a coin toss. A special thanks to Carl Cheney who inspired this line of research by encouraging the second author to pursue "starvation in rats" when he was an undergraduate student of Carl's at Eastern Washington State University. This paper was first presented as an invited address at the ABA conference in Chicago, May 1993.

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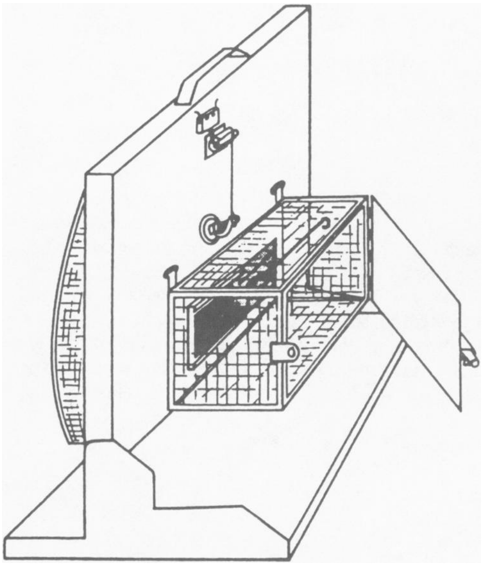

Figure 1. A standard 1.1-m Wahmann running wheel with an attached side cage. A sliding door prevents or permits access between cage and wheel. (Republished from Epling et al., 1983.)

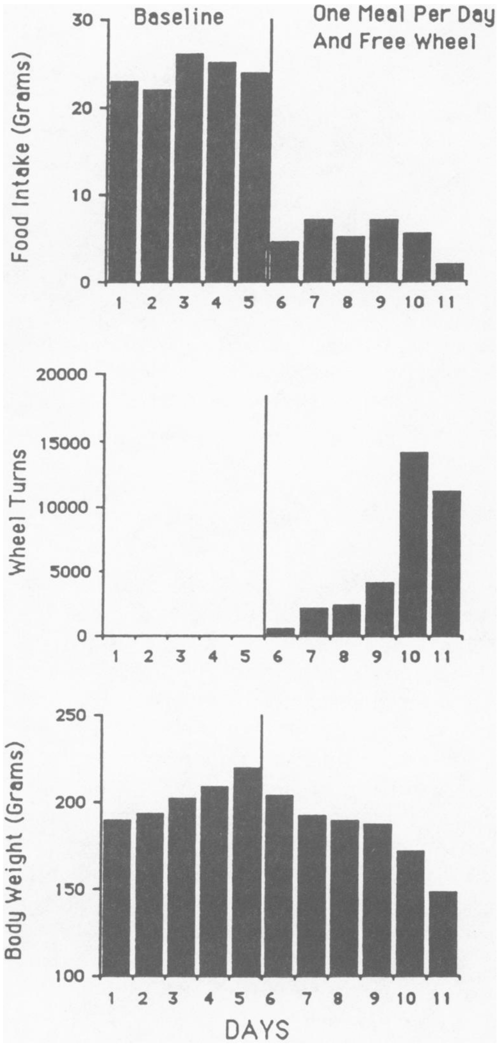

Figure 2. Effects of food restriction (one 60-min meal a day) and access to a running wheel for a typical rat. The figure shows food intake in grams (top), number of revolutions of a 1.1-m wheel (middle), and body weight in grams (bottom). The thin solid line separates baseline from experimental phases.

nervosa are in fact instances of activity anorexia (see Epling & Pierce, 1991, for detailed evidence). Activity anorexia is functionally defined and occurs when a decline in food consumption increases physical activity. As physical activity becomes excessive, food intake is further reduced; the reduction in caloric intake leads to more activity, and so on. Eventually this feedback cycle may lead to starvation and death.

We have developed an animal model of the process of activity anorexia and a biobehavioral theory that incorporates the results from nonhuman studies as well as convergent evidence from humans. We show that the biobehavioral analysis of activity anorexia has assessment and treatment implications. Many people who are currently diagnosed with anorexia nervosa and treated as mentally ill may benefit from a functional analysis of activity anorexia.

A LABORATORY MODEL OF ANOREXIA

Under certain environmental conditions, rats self-starve; this phenomenon is functionally similar to so-called willful starvation by humans (anorexia nervosa). In our laboratory, adolescent male rats (approximately 60 days old) are placed in a cage that is attached to a running wheel. The wheel and side cage can be separated by closing a sliding door (see Figure 1). During the first 5 days of an experiment, the door that separates the side cage from the wheel is closed. Food is freely available in the cage, and each animal can eat as much as it wants. The amount eaten is measured daily, and the rats are also weighed each day (see Pierce & Epling, 1991, for a more complete description).

The food and weight measures provide baseline points for the experimental interventions. The experimental procedures combine food restriction and opportunity to run on a wheel. In a typical experiment, the animals are restricted to a single 60- or 90-min daily meal. Following the meal, the doors to the wheels are opened, and experimental animals are allowed to run. Control animals receive the same treatment, but the wheels will not turn.

Several procedural points are noteworthy. Experimental animals are given continuous access to the wheels, except during the feeding period. In this way, running does not compete with eating. When wheels are available, there are no programmed contingencies for running. The animals can stay in their cages, sit in the running wheels, walk rather than run on the wheels, or emit any other available response.

The initial effect of placing animals on one meal a day is a large drop in food consumption (see Figure 2). This is not surprising, because the animals have not experienced a rapid change in food supply and are not adapted to the new feeding schedule. When food restriction and the opportunity for wheel running occur together, a number of interesting effects happen. As shown in Figure 2, experimental animals begin to run on the wheels. They increase running over days, even though there is no requirement to do so. This is an unusual response, because energy expenditure increases at a time when food intake is limited. Within a week, running increases from several hundred to thousands of revolutions per day. Importantly, control animals, who cannot run, adapt to the feeding schedule within several days and remain healthy.

A more startling effect is that food intake at mealtime declines as running becomes more and more excessive. By the end of 1 week, the animal may not eat at all. The physical activity does not appear to be stressful (Spigelman, McLeod, & Rockman, 1991), and failure to eat is not due to the development of activity-stress ulcers (Doerries, Stanley, & Aravich, 1991). Recent findings by Belke and Heyman (1993) indicate that running generated by food restriction has reinforcing properties (opportunity to run will support lever pressing by rats on variable-interval schedules). The animals in the activity anorexia experiment are giving up eating based on a reinforcement process that involves increasing energy expenditure through wheel running. A typical rat may run up to 15 km per day.

If the process of activity anorexia is allowed to continue, animals become weaker and weaker, activity subsides, and they die of starvation. The seemingly willful starvation and excessive exercising of these animals appear to be similar to many cases of anorexia nervosa in humans.

ANIMAL AND HUMAN ANOREXIA

Observations like these suggest clinical applications, because the laboratory phenomena appear to be functionally similar to what has been labeled as anorexia nervosa in humans. We suggest that activity anorexia, not anorexia nervosa, is the issue. That is, many cases of activity anorexia have been incorrectly diagnosed cases of anorexia nervosa. One way to establish functional similarity between an animal model and human pathology is to gather convergent evidence. Convergent evidence involves documenting diverse findings from various sources that together support (or refute) the relationships observed in the laboratory. The strongest form of convergent evidence occurs when a researcher is unable to predict the outcome of this search. In order to extend the activity-anorexia model to the human level, we suggest five levels of functional similarity by convergent evidence (see also Pierce & Epling, 1991).

1. Excessive physical activity is associated with anorexia in humans. Based on our early research with animals (Epling, Pierce, & Stefan, 1981), we wondered if human anorectics were in fact

hyperactive. Epling, Pierce, and Stefan (1983) and Epling and Pierce (1988) reported mounting evidence for a relationship between physical activity and anorexia nervosa. We found numerous reports of hyperactivity in anorexic patients (Blitzer, Rollins, & Blackwell, 1961; Crisp, 1965; Halmi, 1974; Katz, 1986; King, 1963; Kron, Katz, Gorzynski, & Weiner, 1978; Slade, 1973; Thoma, 1967). Kron et al. (1978) conducted a retrospective study of hospitalized anorectics and concluded that hyperactivity is a central feature of anorexia nervosa. This activity is often extreme. To illustrate, Katz (1986) reported that a physician became anorexic (going from 175 to 115 lb) after starting a running program that increased to 50 miles per week. Hip pain made running difficult, and the man compensated by extensive walking and bicycling.

2. Physical activity decreases food intake. Research on nutrition and behavior confirmed our speculation that humans reduce food intake when physical activity becomes excessive (Edholm, Fletcher, Widdowson, & McCance, 1955; Epstein, Masek, & Marshall, 1978; Johnson, Mastropaolo, & Wharton, 1972; Mayer, Roy, & Mitra, 1956; Watt, Wiley, & Fletcher, 1976). For example, Endholm et al. (1955) reported that military cadets ingest less food on drilling days than they do on days of lower activity. Epstein et al. (1978) found that obese school children would voluntarily decrease food intake following a prelunch exercise period.

Generally, increasing physical activity reduces food consumption. This effect occurs when physical activity increases against an individual's base rate and subsides when activity stabilizes (Epling & Pierce, 1984, 1989). Once activity stabilizes, food intake recovers and may increase in order to compensate for the additional caloric expenditure (Tokuyama, Saito, & Okuda, 1982). Of course, when starvation becomes extreme, activity decreases. This lethargy is also observed in our laboratory rats near the end of the activity-anorexia cycle.

3. Lower food consumption increases physical activity. There is additional evidence that lower food consumption increases physical activity in humans (Blanton, 1919; Howard, 1839; Russell-Davis, 1951) and in rats (Boer, Epling, Pierce, & Russell, 1990; Russel, Epling, Pierce, Boer, & Amy, 1987). A controlled experiment on human starvation was conducted by Keys, Brozek, Henschel, Mickelsen, and Taylor (1950). In this study, 36 conscientious objectors to World War II were required to undergo 6 months of semistarvation. Although Keys et al. emphasized the inactivity of the men, his procedure may have masked the expected increase in physical activity when food is restricted. The men were required to participate in a regular physical activity program, hike 22 miles a week, and walk 2 to 3 miles a day to the mess hall. Each man was also required to do a weekly 30-min test on a motor-driven treadmill at 3.5 miles per hour on a 10% grade. In spite of this, there is evidence that food deprivation induced excessive physical activity. The researchers stated that "some men exercised deliberately at times. Some of them attempted to lose weight by driving themselves through periods of excessive expenditure of energy" (p. 828). Overall, the study by Keys et al. and other evidence indicate that humans increase physical activity when food intake declines and then become inactive when starvation is severe (see Epling & Pierce, 1988, 1991; Epling et al., 1983, for reviews).

4. The onset of anorexia in humans and animals develops in a similar manner. Another line of convergent evidence shows that the onset of anorexia in humans is consistent with the pattern observed in nonhuman animals. In the laboratory, food restriction generates excessive physical activity that interferes with eating. A similar pattern is reported for anorexic patients (Beumont, 1991; Beumont, Booth, Abraham, Griffiths, & Turner, 1983; Katz, 1986). Beumont and associates asked 25 anorexic patients 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 occurred early in the sequence. These changes in food allocation were followed by increased sport activity and "exercising alone." Thus, the pattern of onset in humans parallels the development of activity anorexia in laboratory rats.

5. Reproductive function is disrupted for physically active rats, athletes, and anorexic patients. The estrous cycle is disrupted for female rats that are food deprived and run excessively on an activity wheel (Watanabe, Hara, & Ogawa, 1992). Anorexic humans are hyperactive and have problems with menstruation (Kaye, Picker, Naber, & Ebert, 1982). Physical activity can also produce menstrual difficulties for female athletes (Mansfield & Emans, 1989). Olympic and college track-and-field athletes often experience delayed onset of the menstrual cycle (Cumming & Rebar, 1983, 1985; Frisch et al., 1981). Ballet dancers who are very active also have a high incidence of menstrual problems and other symptoms of anorexia nervosa (Frisch, Wyshank, & Vincent, 1980; Garner & Garfinkle, 1980). Lower serum testosterone and higher serum cortisol levels have been observed for male athletes who engage in strenuous physical activity and food restriction, suggesting a disruption of reproduction similar to that seen in females (Wheeler, McFadyen, Symbaluk, Pierce, & Cumming, 1992; Wheeler, Singh, Pierce, Epling, & Cumming, 1991; Wheeler, Wall, Belcastro, & Cumming, 1984).

THE INTERPLAY OF BASIC AND APPLIED RESEARCH

Overall, the convergent evidence for activity anorexia is strong. Based on these findings, we have argued that the laboratory paradigm has functional similarity to anorexia in humans and is a useful model for exploring the determinants of anorexia under controlled conditions. When this is done, independent variables may be identified that regulate the interrelations among eating, physical activity, and starvation. Based on 12 years of research, a biobehavioral analysis of activity anorexia has been proposed (see Epling & Pierce, 1988, 1989, 1991; Epling et al., 1981, 1983). This analysis involves considerations of evolutionary biology, behavior, and physiology.

The Evolutionary Basis of Activity Anorexia

The survival value of eating is obvious; the survival value of not eating is less obvious. There are, however, anorexias that occur in many species that have resulted from natural selection (see Mrosovsky & Sherry, 1980, for a review and discussion). In these animals, anorexias often occur when the organism is engaged in other biologically relevant behavior (e.g., defending young, defending territory, molting, etc.). Organisms that are exposed to a periodic reduction in food supply (e.g., due to regular seasonal variation) may also become anorexic. For example, ground squirrels hibernate during the winter and will not eat when aroused. For these animals, anorexia during hibernation contributes to energy efficiency. During hibernation, body temperature decreases and the kidneys do not function well. The kidneys remove waste products from the bloodstream, and the animal must remain awake for efficient kidney function. Staying awake is energy expensive, because the animal must heat its body to normal temperature. Thus, refusal to eat during hibernation relates to the energy cost of waking. In fact, the more squirrels eat, the sooner they come out of hibernation (Mrosovsky & Barnes, 1974).

We contend that activity anorexia also had survival value and is an important variant of evolutionary-based anorexias. In this view, the interrelations between physical activity and food intake are based on evolved structural features of organisms (i.e., physiological characteristics). In general, natural selection favored those individuals (of some species) who became active during severe and unexpected food shortages. Animals that traveled to a new location contacted food, survived, and reproduced.

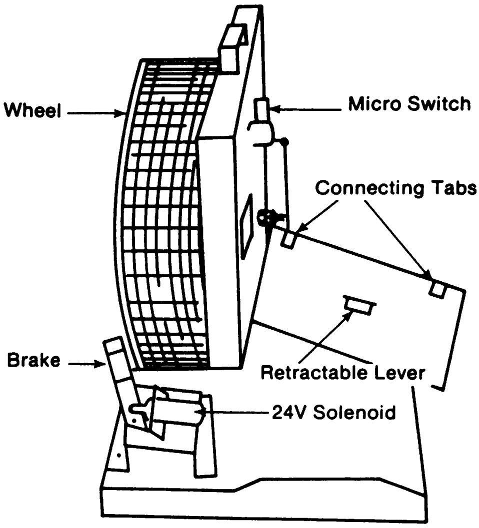

Figure 3. Modified Wahmann running wheel used to study lever pressing reinforced by 60-sec access to wheel running as a function of percentage body weight. Portrayed is the retractable lever mounted on a metal plate that fits over the entrance to the wheel. Also shown is the break that was controlled by a 24-V continuously operated solenoid. (Reprinted from Pierce et al., 1986.)

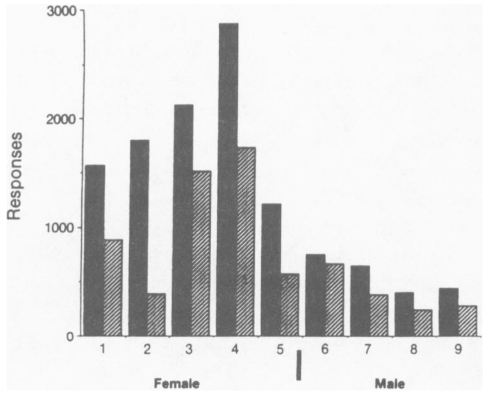

Figure 4. Total number of bar presses for wheel running on a progressive-ratio schedule at 75% (black) and 100% (striped) of free-feeding body weight for 5 female and 4 male rats. (Reprinted from Epling & Pierce, 1989.)

Anorexia induced by a migration to a new food patch is likely to have survival value. During a famine, food is difficult to obtain, and stopping to procure small amounts may be more energy costly than efficient. In other words, there may be a net negative energy balance between foraging for scarce (and difficult to obtain) prey and traveling to a more abundant food patch. Of course, these ideas are post hoc and speculative, but they may be translated into research questions.

From this perspective, activity anorexia results from the interrelations of (a) deprivation and food schedule on physical activity and (b) physical activity on food consumption. Briefly stated, strenuous activity works to suppress appetite. This decrease in the value of food reinforcement serves to affect food schedule and/or deprivation that further increases activity. We tested these ideas in the operant laboratory.

The Behavioral Basis of Activity Anorexia

Our analysis of eating and physical activity suggests that these behaviors are interrelated. Depriving an animal of food should increase the reinforcement value of exercise. Rats who press a lever in order to run on a wheel should work harder when deprived of food. In addition, engaging in exercise should reduce the reinforcement effectiveness of food. Rats who press a lever for food pellets should not work as hard following a day of exercise. We designed two experiments to test these ideas (Pierce, Epling, & Boer, 1986).

Reinforcement value of exercise. Nine adolescent rats of both sexes were used to test the reinforcement effectiveness of wheel running as food deprivation changed. The animals were trained to press a lever to obtain 60 s of wheel running. When the animal pressed the lever, a break was removed and the running wheel was free to turn. After 60 s, the break was again activated and the rat had to press the lever to obtain more time to run. Figure 3 illustrates the apparatus used in this experiment.

Once lever pressing for wheel running was consistent, each animal was tested when it was food deprived (75% of ad lib weight) and when it was at its free-feeding weight. In order to measure the reinforcement effectiveness of wheel running, the animals were required to press the lever on an increasing fixed ratio for each opportunity to run. Specifically, the rats were required to press five times to obtain 60 s of wheel running, then 10, 15, 20, 25, and so on. The point at which they gave up pressing for an opportunity to run on the wheel was used as an index of the reinforcement effectiveness of exercise. Figure 4 shows the main results of this experiment.

Note that all animals lever pressed for wheel running more when they were food deprived than when they were at normal weight. Female rats pressed more for the opportunity to run than male rats did. Although it is tempting to suggest that this is in accord with a proportionally higher number of human female anorectics, there are other explanations. Female rats are considerably lighter than males, and the deprivation operation likely had a stronger impact on them. Also, female rats become highly active during their estrus cycle, and this may have affected the reinforcement value of wheel running.

Further evidence indicated that the reinforcement effectiveness of wheel running would go up and down when an animal's weight was made to increase and decrease. For example, 1 subject pressed the bar for an opportunity to run on a wheel 1,567 times when it was food deprived, 881 times when it was at normal weight, and 1,882 times when it was again food deprived. This indicated that the effect was reversible and was tied to the level of food deprivation.

Although the reinforcement effectiveness of running increased with level of food deprivation, there was a point at which exercise began to decrease in value. When body weight was 70% or below ad lib weight, animals would not work as hard as they did with less deprivation. As previously stated, when starvation becomes extreme, an individual may be too weak to exercise. That is, such weakness may contribute to the decline in the reinforcement effectiveness of physical activity.

Reinforcement value of food. In a second experiment, we investigated the effects of exercise on the reinforcement effectiveness of food (Pierce et al., 1986, Experiment 2). Four male rats were trained to press a lever for food pellets. When lever pressing occurred reliably, we tested the effects of exercise on each animal's willingness to work for food. In this case, we expected that a day of exercise would decrease the reinforcement effectiveness of food on the next day.

Test days were arranged to measure the reinforcement effects of food. One day before each test, animals were placed in their wheels without food. On some of the days before a test, the wheel was free to turn, and on other days it was not. When the wheel was free to turn, animals could exercise or remain in an attached cage. Three of the 4 rats ran moderately (approximately 750 turns in 18 hr) in their activity wheels on exercise days. One rat did not run when given the opportunity. This animal was subsequently forced to exercise on a motor-driven wheel (see below). All animals were returned to their home cages 3 hr before each test. This insured that the effects were not due to fatigue.

Reinforcement effectiveness of food was assessed by counting the number of lever presses for food as food became more and more difficult to obtain. To illustrate, an animal had to press five times for the first food pellet, 10 for the next, then 15, 20, 25, and so on. As in the first experiment, the "giving up" point was used to measure reinforcement effectiveness. Presumably, the more effective or valuable the reinforcer (i.e., food) the harder the animal would work for it.

As shown in Figure 5, when test days were preceded by a day of exercise, the reinforcement effectiveness of food decreased sharply. Animals pressed the lever more than 200 times when they were not allowed to run, but pressed it no more than 38 times when running preceded test sessions. Food no longer supported lever presses following a day of moderate wheel running, even though a rest period in the home cage preceded the test. Although wheel running was moderate, it represented a large change in physical activity, because these animals had been previously sedentary. As we have noted earlier, it is the change from one level of exercise to another that reduces the value of food reinforcement (Epling & Pierce, 1984).

Prior to each test, the animals had spent an entire day without food. This depri-

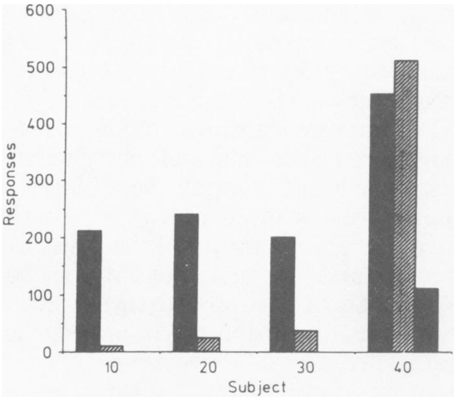

Figure 5. Total number of lever presses on a progressive-ratio schedule of food reinforcement as a function of prior exposure to a locked (black) or open (striped) wheel. Subject 40 did not run when the wheel was open and was exposed to a forced-running procedure. The effect of forced running is shown by the third bar for this subject. (Data taken from Pierce et al., 1986.)

vation should have increased the reinforcement effectiveness of food. Exercise, however, seems to override the effects of food deprivation, because responding for food went down rather than up. Other evidence from these experiments suggested that the effects of exercise were similar to feeding the animal. This finding is important for activity anorexia, because exercise appears to substitute for eating.

As stated earlier, 1 rat refused to run, but this animal sharply reduced lever pressing for food after forced exercise (Figure 5). The forced running was again moderate but substantial relative to the animal's sedentary history. Because the reinforcement effectiveness of food decreased with forced exercise, we concluded that both forced and voluntary physical activity produces a decline in the value of food reinforcement. Taken together, these findings suggest that people who increase their physical activity due to occupational requirements (e.g., ballet dancers) or for recreation may value food less and increase the chance of developing anorexia.

These relationships explain, at a behavioral level, why organisms do not eat on a famine-induced migration and why they eventually abort travel when a new food patch is contacted. During a famine, food is difficult to obtain, and caloric intake is reduced. Because food is infrequently obtained, physical activity is highly reinforcing and the organism begins to move. As the animal increases physical activity, food becomes less effective as reinforcement, and the organism is unlikely to respond for prey that requires extended effort. When food becomes abundant and more easily obtained, it is eaten. As food from easily acquired sources is taken, the reinforcement value of traveling declines and the organism stops moving. For these reasons, the animal stops migrating once a new food patch is contacted. The behavioral relations between eating and activity are a function of the organism's evolutionary history. Natural selection has resulted in physiological processes that are part of the contingencies regulating activity anorexia.

Physiology and Activity Anorexia

β-endorphin, and perhaps other endogenous opiates, appear to mediate the relation between increasing physical activity and decreasing food intake in both rats and humans (Doerries, Aravich, Metcalf, Wall, & Lauterio, 1989; see also Epling & Pierce, 1991, pp. 158–164, for a review). In general, declining food intake stimulates an increase in physical activity. Physical activity increases production of β-endorphin (Colt, Wardlaw, & Frantz, 1981), and this neural opiate functions as reinforcement for exercise (see Shainberg, 1977, for a description of "runner's high"). After reviewing the relevant evidence, Marrazzi and Luby (1986) suggested that anorexia results from addiction to opioids. In terms of activity anorexia, it is significant that anorexic patients show high plasma levels of β-endorphin (Kaye et al., 1982). Also, β-endorphin reduces the motivation to eat for anorexic patients (Kaye, 1987; Moore, Mills, & Forester, 1981; Nakai, Kinoshita, Koh, Tsujii, & Tsukada, 1987) and for rats that are forced to exercise (Davis, Lamb, Yim, & Malvern, 1985).

Endogenous opiates and fertility. Recall that disruption of the menstrual cycle is a major physical symptom of anorexia nervosa. Evidence suggests that increased levels of endorphins produced by physical activity disrupt hormones that regulate the menstrual cycle in females (Cumming & Rebar, 1983, 1985; Petraglia et al., 1986; Ropert, Quigley, & Yen, 1981; Warren, 1989) and testosterone production in males (Wheeler et al., 1991). Simply stated, it makes sense that reproduction should decrease during a time of food shortage and travel to a new food patch.

Exercise, endogenous opiates, and reinforcement. As we have noted, the release of endogenous opiates may function as reinforcement for physical activity. There is evidence of a dose–response relation between intensity of exercise and plasma β-endorphin (Radosevich et al., 1989). This implies that β-endorphin release in the central nervous system is increased. As aerobic fitness increases, more and more exercise is required to attain maximal release of endogenous opiates (McMurray, Hardy, Roberts, & Forsythe, 1989). Thus, physical activity is likely to be maintained on a schedule of reinforcement for endorphin release that requires greater and greater amounts of exercise (i.e., tolerance develops).

The reinforcement hypothesis suggests that injection of an opiate antagonist will decrease the intense wheel running of anorexic animals, because the euphoric effects of opiates are diminished by antagonizing opioid receptors. In our laboratory, research by Douglas Boer has explored the effects of the opiate antagonist naloxone on the anorexic running of male rats (Boer et al., 1990). In this study, animals were made active by feeding them a reduced amount of food and providing a running wheel. Once wheel running exceeded 5,000 revolutions per day (5 km), each animal was given naloxone (i.p., 50 mg/kg in saline) or saline (0.5 mL) on alternate days. Average daily wheel revolutions were approximately 5,800 for saline days and 4,800 on days when the animals were injected with naloxone ($p < .01$). These findings provide preliminary evidence for the hypothesis that anorexic running is strengthened and maintained by the release of endogenous opiates.

At this point, it is useful to summarize the evidence for activity anorexia mediated by endogenous opiates.

1. Physical activity produces an increase in endogenous opiates.
2. Both anorexic patients and athletes show elevated levels of β-endorphin.
3. The "euphoric" effects of opiates reinforce physical activity.
4. Increasing amounts of exercise are required to produce the same level of β-endorphin.
5. When body weight is low or exercise is prominent, endogenous opiates seem to decrease appetite.
6. Anorexic patients are hyperactive and show elevated plasma levels of endogenous opiates.
7. Anorexic patients do not feel hungry when the opioid system is stimulated.
8. Opioid release decreases the level of hormones that regulate menstrual cycle in women and testosterone production in men.

Thus, people who are diagnosed as having anorexia nervosa as a result of extreme thinness, excessive exercise, and reproductive problems are probably exhibiting activity anorexia.

In general, we view activity anorexia as a normal response to an unusual set of environmental events. The process is initiated by cultural metacontingencies that lead to reinforcement for dieting and exercising. The combination of these responses produces anorexia. Once started, activity anorexia is maintained through a physiological reinforcement system, the end stage of which involves the endogenous opioids.

FROM ANALYSIS TO APPLICATION

In order to specify the functional relations that regulate activity anorexia, we

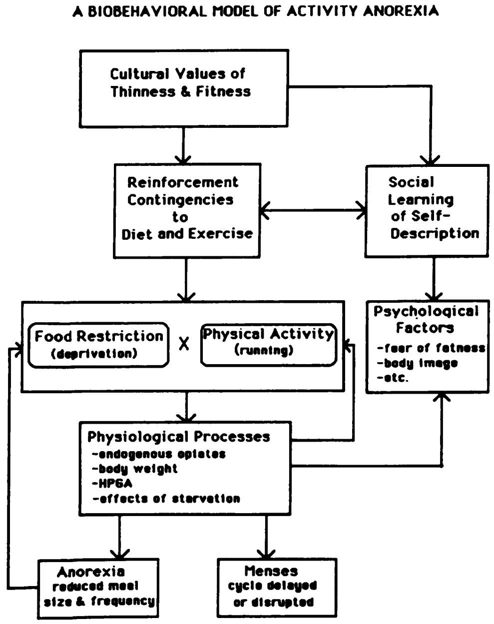

Figure 6. A biobehavioral model of activity anorexia. The model portrays the cultural impact on dieting and exercising. This behavior is supported by reinforcement contingencies set by family and friends. Under particular conditions, food restriction combines with physical activity to initiate the physiological processes of activity anorexia. (Reprinted from Epling & Pierce, 1991.)

developed a theory of the biobehavioral process. Figure 6 shows the interrelations among culture, behavior, and biology that account for activity anorexia. In addition, the theory explains how the physical and psychological symptoms attributed to anorexia nervosa are by-products of starvation and social learning. Activity anorexia is hypothesized to result from behavioral and biological processes that, in Western societies, are initiated by cultural practices based on the values of fitness and thinness.

In the laboratory, the experimenter imposes food restriction on the animals, which initiates the activity-anorexia cycle. In order to test the generality of the activity-anorexia model, it is necessary to specify the conditions that "impose" food restriction or exercise on humans. Our contention is that contingencies of reinforcement set by Western culture encourage people to diet and exercise, thereby increasing the chances that some individuals will combine food restriction and exercise in a way that initiates activity anorexia (see Epling & Pierce, 1988, 1991, pp. 169–183; Epling et al., 1983).

Western culture currently values a thin, trim appearance in women (Lakoff & Scherr, 1984; Mazur, 1986) and physical fitness in both sexes (Beck, Ward-Hull, & McLear, 1976; Garner, Rockert, Olmstead, Johnson, & Coscina, 1985). Several researchers have noted that, in this culture, the mass media convey these values (Bruch, 1978; Kurman, 1978; Wooley, Wooley, & Dyrenforth, 1979), and people learn by observation (Bandura, 1986) to uphold and promote the beauty standards. Acceptance of the thinness and fitness values means that people provide social approval, economic advantages, and privileges to individuals who attain the cultural beauty standards (Brigham, 1980; Green, Buchanan, & Heuer, 1984; Jones, Hannson, & Philips, 1978; Umberson & Hughes, 1984; Unger, Hilderbrand, & Madar, 1982). Because dieting and exercising are ways of achieving these standards, this behavior is reinforced (Garner et al., 1985). This social conditioning insures that some people will inadvertently combine dieting with physical activity in a way that produces activity anorexia (Davies & Furnham, 1986; Dwyer, Feldman, & Mayer, 1970; Jakobovits, Halstead, Kelley, Roe, & Young, 1977; Miller, Coffman, & Linke, 1980). The more severe the increase in physical activity and the larger the drop in food consumption, the greater the chances of acquiring activity anorexia (Epling & Pierce, 1984, 1991). Although the model emphasizes cultural factors as the major initiating conditions, it is important to recognize that the basic process may be triggered by other events. Famine, forced exercise, or any condition that combines food restriction and physical activity may increase the chances of activity anorexia.

If the animal model is functionally similar to human anorexia, then the psychological symptoms of anorexia nervosa are by-products of the activity-anorexia cycle. Katz (1986) reported that many of the psychological and physical symptoms of anorexia nervosa followed, rather than preceded, activity-induced starvation. Preoccupation with food, bingeing, vomiting, distortion of body image, loss of libido, and depression came after the onset of excessive exercise and food restriction (see also Beumont, 1991; Beumont et al., 1983; Keys et al., 1950).

Our biobehavioral theory proposes that these psychological factors, as well as fear of fatness, body-image distortion, and so on, arise from starvation or from contingencies of reinforcement set by others (i.e., social learning). According to this analysis, family members, friends, and health professionals teach the anorectic to describe behavior, thoughts, and feelings. Furthermore, reasonable responses to situational determinants of behavior are interpreted as personality symptoms. For example, a young woman who does not want to eat is said to resist treatment. Denial of illness, evidenced by resistance to treatment, is then taken as a symptom of anorexia nervosa (see APA, 1987). Psychological symptoms are also produced by the physiology of starvation. As noted above, the Keys et al. (1950) study found that psychologically healthy men became neurotic, preoccupied with food, and bulimic when they were forced to starve.

A multiplier effect (Figure 6) between food restriction and physical activity is a critical feature of the cycle. In animals, the effect occurs when meals are reduced to one per day (Kanarek & Collier, 1983), and many anorexic humans eat only one meal per day. The combination of food restriction and physical activity in turn generates physiological processes. There is a dose–response relation between intensity of exercise and plasma β-endorphin (Radosevich et al., 1989). In humans, more and more exercise is required to attain 80% of maximum oxygen intake and maximal release of endogenous opiates (McMurray, Forsythe, Mar, & Hardy, 1987). Endogenous opiates also contribute to the disruption of the menstrual cycle (Ropert et al., 1981; Warren, 1989) by decreasing pulsations of reproductive hormones (Mansfield & Emans, 1989); women who exercise show lowered levels of these hormones (Cumming & Rebar, 1985).

When body weight is low, β-endorphin decreases caloric intake. Food restriction further augments physical activity, and the person is locked into a cycle of declining food intake and increasing physical activity. This cycle explains why athletes, ballet dancers, gymnasts, and anorectics have eating problems (Garner & Garfinkle, 1980; Mansfield & Emans, 1989; Rosen, McKeag, Hough, & Curley, 1986).

The biobehavioral theory of activity anorexia suggests assessment criteria and treatment implications. They follow logically from the analysis and are based on research with both humans and other animals, some of which is reviewed in this article. The clinical implications of the activity-anorexia theory are beginning to be addressed by a few researchers (e.g., Beumont, 1991; Katz, 1986). The relation between the theory and its application, however, must be viewed as tentative at the present time.

Assessment Criteria for Activity Anorexia

The biobehavioral theory has been useful for developing assessment criteria for activity anorexia. We have been able to specify several primary and secondary criteria (Epling & Pierce, 1991, pp. 193–198). The criteria follow from our analysis of the biological, behavioral, and cultural determinants of the activity-anorexia cycle.

Primary criteria. The primary criteria for activity anorexia refer to the necessary conditions that must be present.

1. The person must show a history of low or declining food intake when body weight is below normative standards (based on weight adjusted for height and bone structure).

2. The person must present a history of excessive physical activity. Aerobic

exercise (e.g., long-distance running, swimming, or active sports) is particularly significant.

3. The onset of psychological symptoms often follows, rather than precedes, anorexia and excessive exercise. For people with a history of anorexia, psychological symptoms may be present during periods of low food intake and absent when eating recovers.

It is often difficult to assess personal history with regard to exercise and hyperactivity. People may not consider their activity level to be excessive, or they may not wish to report it. For this reason, it may be necessary to use a structured interview that probes for type and level of physical activity. An important point is that as starvation becomes extreme, activity may decline. Thus, the current level of physical activity may not be as important as the history of exercise.

Supplementary criteria. These criteria are often present and supplement the assessment based on primary criteria. In general, as the number of supplementary criteria increase, the greater the certainty of activity anorexia.

1. Activity is particularly significant if there is evidence of a substantial increase relative to baseline levels of exertion (e.g., sedentary to active, increased sports training, increased mileage for a long-distance runner, etc.).

2. Preoccupation of person and family with sports, fitness, dieting, and general acceptance of thinness as a beauty standard. High scores on the Eating Attitudes Test (EAT) may reflect such concerns (see Garner & Garfinkle, 1979).

3. Preoccupation with exercise, as when a person plans the day around exercise routines and/or engages in ritualistic behavior related to exercise (e.g., the sports fanatic).

4. Persons who are required by occupation to be thin (e.g., fashion models) or physically active (e.g., athletes) are at some risk. Those individuals who are required to be thin and physically active (e.g., ballet dancers, gymnasts, college wrestlers) have higher risk.

5. Persons with the time and money to belong to recreational clubs and sports facilities. High socioeconomic status is also associated with acceptance of cultural values that relate to thinness and fitness.

6. In women, menstrual problems (delayed menarche, cessation of menstruation, or irregular cycles) associated with activity and food restriction that may precede or accompany weight loss.

7. Biochemical changes, including increased endorphin levels at low weight, decreased leutinizing hormone (LH) levels, and, in men, decreased testosterone. These changes may be accompanied by decreased sexual drive in both sexes.

Treatment of Activity Anorexia

Approaches to the treatment of activity anorexia require specific techniques to modify the activity-anorexia cycle. The psychological, behavioral, and physiological symptoms of activity anorexia are expected to decrease when the food restriction and exercise spiral is interrupted. There is tentative evidence, however, that depression may occur when exercise is prevented (Angst et al., 1979). This depression appears to be associated with endogenous opiate withdrawal that occurs when excessive physical activity is reduced. Such a depressive state may be temporary and does not necessarily suggest that treatment is ineffective. A variety of techniques outlined in our treatment approach have been used to modify the behavior of some patients diagnosed with anorexia nervosa (Agras, 1987; Agras, Barlow, Chapin, Abel, & Leitenberg, 1974; Agras & Kraemer, 1983; Garner et al., 1985; Halmi, 1985).

The first phase of treatment. From a medical perspective, the first concern is the anorectic's health. If weight loss is less than 20% below normal, it may be possible to treat the person in an outpatient setting. However, when body weight is more than 25% below ideal weight, hospitalization is recommended. At this weight, serious complications such as bradycardia, low blood pressure, seizures, and imbalanced electrolytes may occur. Based on our analysis of activity anorexia, hospitalization is also recommended when weight loss is extreme, because medical staff have more control over exercise, eating, and weight gain.

We suggest that treatment be initially directed at stopping excessive exercise and dieting. Direct medical intervention is necessary when starvation has progressed to a point at which it is life threatening. Confinement to bed, forced or tube feeding, and drugs may be required to save the person's life. Although we do not advocate control of behavior with drugs, medical doctors may prefer this kind of treatment. If drugs are used, specific agents that block endogenous opiates (e.g., naloxone and naltraxone) may be more effective and have fewer side effects than general tranquilizing or antidepressant drugs. Opiate blockers may be used to lower the motivation for physical activity and increase appetite.

Unfortunately, the few studies that have investigated opiate blockers for the treatment of anorexia have been uncontrolled (see Kaye, 1987; Moore et al., 1981; Marrazzi & Luby, 1986, for a review of the opiate hypothesis) and have used only a small number of patients. In addition, prolonged use of these drugs by anorexic patients has not been evaluated (Kaye, 1987), but high doses of naloxone have been associated with increased blood pressure in healthy subjects (Cohen, Cohen, Pickar, Weingartner, & Murphy, 1983). For these reasons opiate blockers should be used with caution.

The second phase of treatment. The theory of activity anorexia states that apparent self-starvation is a normal response to an unusual set of circumstances. It may be helpful for the patient and the family to recognize that the anorexic is not mentally ill or trying to manipulate significant others. Hospital personnel may describe the process of activity anorexia and its origins. That is, the staff may explain the underlying processes and how these relate to cultural practices of our society. Verbal techniques, such as articulation of beliefs, testing beliefs against evidence, and challenging "black and white" thinking about food, figure, and exercise, may be used to change self-descriptive statements (Garner & Bemis, 1985; Garner et al., 1985). Changes in self-description prompt new behavior related to diet and exercise. Similar strategies may be used to combat the patient's beliefs about combining stringent dieting with excessive exercise.

An important component of treatment is medical and nutritional advice. Nutritional counseling should address the pattern and size of meals as well as composition of the diet. Findings from animal research show that the more frequent the meals, the lower is the tendency to exercise (Kanarek & Collier, 1983). For this reason, the person should be encouraged to eat frequently throughout the day.

Unfortunately, the activity-anorexia cycle is resistant to change, and the patient may oppose medical or other advice. When opposition is intense, social psychological research has shown that a message that is backed up by several people has more impact than the same message given by a single person (Tanford & Penrod, 1984). This implies that medical staff must work together to provide consistent information about dietary practices and the activity-anorexia cycle.

Although eating is the target behavior, it is almost impossible to monitor this behavior accurately (Halmi, 1985). Often the patient will hide food or vomit after a meal. For this reason, weight gain is commonly used as an indirect measure of eating. Negative reinforcement may be used to maintain appropriate eating (Agras, 1987) and could be extended to control the level of exercise. When anorectics fail to gain weight, the contingencies should specify progressive loss of privileges and increasing medical intervention. The patient may gain privileges and remove medical treatment by eating and gaining weight. Perhaps the most important long-range consequence is getting out of the hospital (Agras, 1987).

Family members may be included in the overall treatment plan (Yager, 1982). Generalization of eating skills may be enhanced by arranging for meals that are attended by family and friends. The patient may be required to eat in a socially appropriate manner in the presence of significant others. This behavior can then be extended to cafeterias and to the home (Halmi, 1985).

It is also important to train sensible and moderate exercise habits. There is little applied research on the control of exercise for anorexic patients (but see Mavissakalian, 1982). One procedure that may be helpful involves discrimination training. Following sufficient weight gain, the person may be taught to discriminate between moderate and excessive exercising. This could be accomplished by allowing the anorectic to exercise for 20 min or less per day. If the patient exceeds 20 min, exercise is withheld on the following day. This is a "fail-safe" contingency, because the excessive exercise that generates activity anorexia is disrupted by the behavioral requirement.

As we have previously stated, the use of exercise as a reinforcer for weight gain may not be the best long-term strategy, because the reinforcement effectiveness of exercise declines as the person gains weight. Based on our experiments with rats (Pierce et al., 1986), exercise is most valued at approximately 75% of normal weight. As body weight becomes closer to normal, the patient may not eat in order to exercise. At this point, the treatment program may stall, and further weight gain will require a different source of reinforcement.

There is an additional problem with the use of exercise as reinforcement for weight gain. Evidence suggests that, at low body weight, physical activity increases endogenous opiates that automatically reinforce exercising and reduce appetite. If this occurs, a treatment program based on exercise may backfire and contribute to the eating problem. One alternative is to decrease exercise rather than to use it as a reinforcer. This procedure has been successfully used by Mavissakalian (1982) to treat 2 17-year-old female anorectics.

The third phase of treatment. Once the anorectic has reached and maintained target weight, consequences are planned to maintain weight, eating in an appropriate manner, and exercising moderately. The major consequence involves longer and longer visits home if weight remains within the negotiated range (Agras, 1987). A final target for behavior change involves family practices that may (unintentionally) encourage activity anorexia. A family that emphasizes the thin-fit standard of beauty may reinforce participation in sports, eating low-calorie foods, and a concern with body shape.

SUMMARY

The biobehavioral theory of activity anorexia began in the animal laboratory and now has applied importance. Basic research may solve practical problems when laboratory experiments are linked to the everyday world by a behavioral theory based on convergent evidence. Once an appropriate animal model and theory are developed, experiments may be carried out with animals that are unethical or impractical to conduct with human subjects. These experiments may focus on the conditions that establish, maintain, and modify the target behavior.

Behavior analysts usually emphasize the external contingencies that control behavior. It is important to note, however, that some of the relevant conditions that regulate behavior come from the internal environment. Activity anorexia results from an interplay of events that occur both outside and within the organism.

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