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Adapted to Flee Famine: Adding an Evolutionary Perspective on Anorexia Nervosa

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Anorexia nervosa (AN) is commonly attributed to psychological conflicts, attempts to be fashionably slender, neuroendocrine dysfunction, or some combination of these factors. Considerable research reveals these theories to be incomplete. Psychological and societal factors account for the decision to diet but not for the phenomenology of the disorder; theories of biological defects fail to explain neuroendocrine findings that suggest coordinated physiological mechanisms. This article presents evidence that AN's distinctive symptoms of restricting food, denial of starvation, and hyperactivity are likely to be evolved adaptive mechanisms that facilitated ancestral nomadic foragers leaving depleted environments; genetically susceptible individuals who lose too much weight may trigger these archaic adaptations. This hypothesis accounts for the occurrence of AN-like syndromes in both humans and animals and is consistent with changes observed in the physiology, cognitions, and behavior of patients with AN.

This article adds an evolutionary perspective to current theories about the etiology of anorexia nervosa (AN). AN has presented a challenging problem to clinicians and researchers. A singular and distinctive syndrome, it is characterized by an odd cluster of symptoms. Despite having lost (by definition) at least 15% of their average body weight (ABW), people with AN eat little. They often feel "boundless energy" (Garfinkel & Garner, 1982) and exercise strenuously (Beumont, Arthur, Russell, & Touyz, 1994). Perhaps the most striking symptom is that they see themselves as well, even fat, when they are dangerously thin.

It is estimated that 90% of AN's victims are women and girls (*Diagnostic and Statistical Manual of Mental Disorders*, 4th ed., text rev. [*DSM–IV–TR*]; American Psychiatric Association, 2000). Incidence appears to have increased in Europe and the United States since 1960; lifetime prevalence among women is now 0.5%, and its mortality rate of 10% makes it America's most deadly psychiatric illness (American Psychiatric Association, 2000). A paradox of this disorder is that it often affects young women who are socially compliant, perfectionist high achievers (Bastiani, Rao, Weltzin, & Kaye, 1995; Bruch, 1978; Garfinkel & Garner, 1982; Srinivasagam et al., 1995).

The causes of the peculiar ideations and behaviors of AN remain a puzzle. People with AN are sometimes as mystified by their symptoms as are those around them. Why are their minds telling them that they are fat when their loved ones are frightened by their weight loss? Why do they feel driven to exercise? Why does abstaining from food feel so virtuous and so easy (Chisholm, 2002; Gottlieb, 2000; Hornbacher, 1998)?

Our current understanding of AN is derived primarily from psychosocial and biomedical theorizing and research. The seriousness of AN's life-threatening and even delusional symptoms has led clinicians to assume serious psychopathology in the patients or in their families. The consensus of the field is that AN develops when psychopathology and social pressures to be thin act in concert with biological vulnerability (Walsh & Devlin, 1998). Psychiatry's *DSM–IV–TR* assumes that people with anorexia eat little and exercise because of their "*refusal* to maintain normal body weight" (American Psychiatric Association, 2000, p. 583; italic added).

What if researchers have assumed the wrong direction of causality? Several lines of evidence, considered together, suggest that, rather than psychological or medical pathology causing the bizarre behaviors and cognitions of AN, it is weight loss that leads to the symptoms. If the interpretation of the direction of causality is reconsidered, a number of discordant observations fall into place.

Levels of Analysis

Psychological and physical disorders can be explained from several different perspectives. For example, the question "Why is this person obese?" may be answered meaningfully but differently from psychological, sociological, neuroendocrine, physiological, or evolutionary perspectives. Theoretical misunderstandings sometimes arise from semantic confusion about which level of analysis is being used (Sherman, 1988). For example, say one wants to understand why a 35-year-old male Pima Indian living on a reservation is obese and diabetic. Proximate or "how" questions investigate the individual ontogeny of the disorders, including family factors, personality variables, societal conditions, and hormonal and neurochemical mechanisms. Ultimate or "why" questions might investigate the evolutionary origins of obesity as an adaptation to an environment characterized by frequent famine

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with diabetes secondary to these adaptations in food-rich environments.

The proximate level has been the focus of most current theorizing and research on AN. Evolutionary explanations and animal models have generally been overlooked. Although considerable data, reviewed later, support the various psychological, sociological, and biomedical theories, some facts are inconsistent with each theory, and no theory accounts for all aspects of the disorder.

When humans diet severely or otherwise lose over 15% ABW, their bodies are subjected to the equivalent of famine, a situation that has exerted strong selective pressures. This article argues that some individuals have inherited a genetic ability to respond to such low body weight with specific adaptations that originally evolved to facilitate leaving food-depleted areas. The article describes what is known about AN's enigmatic psychological, epidemiological, and medical presentation and argues that, when a Darwinian perspective is added, many of these observations make sense.

Psychoanalytic Theories

The term *anorexia nervosa* literally means lack of appetite because of nerves, and researchers have searched diligently for psychological antecedents. A history of theories of AN mirrors the history of psychoanalysis and later clinical theory. Freud wrote to his colleague Wilhelm Fliess that "the well known *anorexia nervosa* of girls seems to be a melancholia occurring where sexuality is undeveloped" (Freud, 1889/1954, p. 201). Early orthodox psychoanalytic theorists believed the syndrome resulted from unconscious conflict over sexual drives or as a defense against fears of oral impregnation (Kaufman & Heiman, 1964).

The most important early psychoanalytic theorist of AN was Hilde Bruch. She proposed that food refusal represents a struggle for psychological autonomy and control (Bruch, 1973, 1978, 1988), and she chastised the doctors and psychotherapists "who approach and evaluate anorexic patients in a critical manner, with a certain guardedness and prejudice, even an open dislike . . . because of the therapist's often unexamined suspicion that the patient's denial of illness indicates dishonesty . . . as if patients could change it through an act of will" (Bruch, 1988, p. 110). Bruch brought a compassionate stance to patients she saw as "engaged in a desperate fight against feeling enslaved and exploited" (Bruch, 1978, Preface, p. x) by their mothers. Her work provided the conceptual framework for psychodynamic and family systems work that followed.

Other influential writers extended or expanded Bruch's understanding of AN within a psychodynamic model. Russell (1977) became convinced that AN was the result of a "morbid fear" of fatness, a formulation that became part of the *DSM–IV–TR* criteria. Working from a developmental model, Crisp (1980) proposed that self-starvation is an attempt to regress to a prepubertal state. Other object relations theorists proposed that anorexia is the weakened self's paranoid reaction against a powerful bad object that has become equated with the physical self (Sours, 1980). Casper (1983) noted an instability in self-concept among patients who restrict food as a defense against sexuality, as an ascetic repudiation of bodily urges, or in a drive for thinness. According to Johnson and Conners (1987), preoedipal failures of separation–individuation between 12 and 36 months play fundamental roles in causing eating disorders. Self psychologists found failures in empathic mirroring and idealization during childhood to be responsible for the symptoms of AN (Geist, 1984). Family theorists saw a family as using an anorectic child to maintain pathological homeostasis within the family system (Minuchin, Rosman, & Baker, 1978; Palazzoli, 1974). Exercise has been viewed as "a hypomanic defense against a fear of paralyzing depression, a form of masochistic self-punishment, a goal oriented pursuit of achievement serving narcissistic/exhibitionistic concerns, or as a general mechanism for regulating such tension states as anxiety, anger, and the like" (Johnson, 1984, p. 24).

Parents of anorectics have often appeared conscientious and well meaning; researchers, frustrated in attempts to find consistent overt psychopathology, hypothesized hidden dysfunction. For example, Palazzoli (1974) wrote that parents concealed disillusionment with themselves and their marriage behind a facade of respectability and good works. Bruch (1973) described the anorectic's parents as controlling, experience denying, and competitive about who was making the most sacrifices for their families. According to one reviewer, "Most recent writings have emphasized the parents' more covert and buried psychopathology despite their apparent healthy adjustment" (Hsu, 1990, p. 37).

The thoughtful clinicians laboring in this field and listening to patients' surreal thought processes were trying to understand the strange self-starvation of anorectics using the conceptual tools at their disposal. However, a number of facts are not in complete accord with these traditional psychological explanations. Although the parents of people with AN have been intensely studied with the expectation that their pathology would explain their child's terrible illness, as with research on the parents of autistic and schizophrenic youngsters, psychopathology is not consistently found. As Humphrey (1992) noted, "empirical research on family contributions to eating disorders has lagged well behind the richly articulated and elaborated theories" (p. 265). Contrary to theoretical expectations, anorectics' families do not reliably demonstrate serious psychopathology in general or the specific psychopathology hypothesized by Bruch and others (Halmi, 1992; Rutherford, McGuffin, Katz, & Murray, 1993; Strober, Lampert, Morrell, Burroughs, & Jacobs, 1990). Twin studies found that, in contrast to genetic factors, family environment had little or no effect on the transmission of AN (Rutherford et al., 1993). Studies that earlier seemed to correlate family depression and AN were later questioned because this familial loading occurs in the subgroup of anorectic probands who themselves have major depression (Strober et al., 1990).

Patients themselves were studied with the expectation that empirical research would confirm theoretical predictions of serious premorbid or comorbid psychopathology. Bruch (1973) taught that AN patients' faulty awareness of hunger and satiety was part of a generalized lack of interoceptive awareness, which she believed resulted from the mother's failure to accept and validate her daughter's authentic experience. Again, the empirical research did not substantiate the psychodynamic hypotheses. There is, in fact, no relationship between patients' disturbances in hunger and satiety and other areas of perceptual confusion or denial (Halmi, Sunday, Puglisi, & Marchi, 1989). Halmi (1992) concluded, "Disturbed eating in eating disordered patients cannot be dismissed as merely reflecting an underlying psychodynamic turmoil" (p. 83).

Furthermore, researchers generally agree that depression and social withdrawal, consistently noted problems in people with AN, are confounded with (and possibly secondary to) personality changes attributable to starvation (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950; Walsh & Devlin, 1998; Wonderlich, 1995).

As noted, the *DSM–IV–TR* criteria for AN include "a refusal to maintain" a normal body weight (American Psychiatric Association, 2000). However, rather than refusing to maintain normal body weight, many patients, in my clinical experience, desperately want to be well, seeking treatment freely and sometimes at considerable financial and personal sacrifice; yet, they report enormous difficulty eating and refraining from exercise, as though something other than their conscious will were in charge of their behavior, sensations, and cognitions. Neuroendocrine changes, discussed below, support this description. Halmi (1992) stated, "The disturbances in hunger and satiety perceptions in eating disorder patients seem to be *sui generis* . . . accumulating evidence suggests that aberrant eating behavior is directly influenced by disturbances of the [physiological] integrating processes of hunger and satiety" (p. 83).

Furthermore, Hsu and Lee (1993) have pointed out that one of the primary psychological motives, included as the second *DSM–IV–TR* criterion, "intense fear of gaining weight or becoming fat," is not always characteristic of nonwestern patients with AN, nor was it described in historical accounts until around 1930. Casper (1983) maintained that the drive for thinness was not commonly described as the motivating factor until the 1960s.

There are other reasons to doubt purely psychological theories of etiology. Despite most theoretical assumptions, the symptoms of AN are similar in men and women (Eliot & Baker, 2001). Not only pubescent girls struggling with fears of developing a womanly body but also the elderly, boys, and men develop AN (American Psychiatric Association, 2000). Symptoms explained using psychological concepts and theories that fit adolescent girls make less sense for boys and men and postmenopausal women. Refusing food, for example, is often understood as an attempt to gain autonomy in an overly controlling family system, but how are researchers to understand that symptom when people live alone and have no family?

Even more puzzling in the light of the serious nature of their symptoms is the recurrent observation that the patients themselves were often among "the best and the brightest" (Bordo, 1992; Bruch, 1978; Garfinkel & Garner, 1982). One study found that, unlike other adolescent psychiatric patients, patients with AN never had problems with impulse control, scholastic achievement, or breaking social rules (Pryor & Wiederman, 1998).

Ironically, the personality traits that lead to such exemplary behavior appear to be the patients' Achilles' heel; these traits represent the major psychological risk factor for AN. Although serious premorbid psychopathology is not found consistently, the personality traits of perfectionism and rigidity *are* reliably correlated with development of AN (Bastiani et al., 1995; Garfinkel & Garner, 1982; Srinivasagam et al., 1995; Wonderlich, 1995). Because these traits are quite common among high achievers, most of whom do not develop AN, their presence alone cannot explain either the seriousness or form of the disorder's symptoms; however, they may help explain a patient's initial perseverance with dieting.

In fact, AN has developed when a normal-weight person loses considerable weight for any reason, for example, when fasting for politics, religion, health or beauty, or when already lean individuals engage in strenuous activity (Bell, 1985; Vandereycken & van Deth, 1990). For example, a recent *New York Times* (Markel, 2000) story described a 15-year-old boy who began dieting after his obese grandfather died of a heart attack while they were playing checkers. The boy eliminated all fat from his diet, developed AN, and eventually died.

Social Theories

Social theorists have tried to explain AN symptoms with reference to the limited options available to women and girls in western patriarchal societies. Wooley (1995) has asserted that the cultural constructions of gender are central to understanding and treating eating disorders. Orbach (1986) wrote, "Anorexia symbolizes the restraint on women's desires . . . . In controlling her food so very stringently she caricatures the message beamed at all women" (pp. 29–30). Thus AN may be a hunger strike against an oppressive culture, an attempt to demonstrate personal power, or an avoidance of sexual exploitation (Orbach, 1986; Wooley, 1995). Bell (1985), who documented AN among Italian saints, believed that both medieval and modern women shared the same dilemma. Because western patriarchal culture offers girls few outlets for autonomy, self-starvation becomes part of their struggle for liberation.

Although these social theorists' observations help to understand what initiates some women's progression into AN, when one attempts to frame a comprehensive theory, social hypotheses suffer from some of the same limitations as psychological theories: They fail to account for the occurrence of anorexia-like syndromes in other animals, in men, and at other times. Furthermore, they offer little to explain the body image distortion and hyperactivity which also characterize AN.

However, social theories can explain why women and girls are more vulnerable to develop AN. Feminist social critics have cogently pointed out that the fashion and entertainment industries promote an ideal size that is impossible for most women to maintain. At the present time, women are rarely hired as models and actresses unless they are thinner than 95% of their age mates, whereas male models and actors are more often normal weight. Female fashion models generally weigh about 85% of ABW (Seid, 1994), thus meeting part of the *DSM–IV–TR*'s first criterion for AN. Although some women are naturally very thin, the majority of women would have to starve themselves to attain the current standard of beauty. There is a general consensus that this modern pressure on women to diet has contributed to increasing incidence of AN and other eating disorders specifically among women and girls (Garner & Garfinkel, 1997; Orbach, 1986; Wooley, 1995).

Thus, although social and psychological theories alone do not satisfactorily explain AN's distinctive symptoms, they do help to understand why most women in the developed world feel too fat and diet and why certain women and girls are more likely to persist in dieting to low weights. These social and psychological observations and insights help to explicate AN's epidemiology and etiology, if not the specific form of its symptoms.

Evolutionary Theories

As early as 1984, Nesse noted that the "remarkable consistency of this syndrome suggests the presence of a possible adaptive mechanism" (p. 578); however, earlier evolutionary theorists focused on the loss of menses in female anorectics and speculated that AN functions to delay reproduction (J. L. Anderson & Crawford, 1992; Surbey, 1987). This reproductive suppression hypothesis (RSH) posits that adolescent girls' desire to control their weight represents an evolutionary adaptation by which ancestral girls reduced the rapidity of their sexual maturation in response to cues about the probability of poor reproductive success. In support of the RSH is the observation that menarche is delayed in prepubertal girls with AN, and menses ceases in previously menstruating female anorectics. Presumably, starvation cues could indeed have been indicative of poor times for reproducing. However, the RSH does not help to understand the function of distorted body image and hyperactivity, why other less costly ways to stop menstruation were not selected for, why wealthy well-fed women and girls might expect poor reproductive success, or why AN's victims also include men and postmenopausal women. This article proposes an alternative evolutionary theory.

Biomedical Theories

Many researchers working from a biomedical perspective perceive a biological origin of the disorder, although they see the symptoms as the result of pathophysiological processes and not as adaptive mechanisms (J. L. Anderson & Kennedy, 1992; Casper, 1998; Kaye, Ebert, Raleigh, & Lake, 1984; Støving, Hangaard, & Hagen, 2001). Although acknowledging that AN's etiology is unknown, biomedical researchers have hypothesized that AN may result from inherited abnormalities in the endocrine system and the neurotransmitters involved in appetite (Holland, Sicotte, & Treasure, 1988). A number of hormones and neurotransmitters have been found in abnormal concentrations in individuals with AN (for a recent review, see Støving et al., 2001). Further, heritability estimates for AN are high. Although rare in the general population, if another family member has AN, the chance of developing it rises to 1 in 30, and the concordance rate in identical twins is 50%. A large population-based twin study estimated its heritability at 0.74 (Klump, Miller, Keel, McGue, & Iacono, 2001).

Neuroendocrine abnormalities in AN are often attributed to genetic defects or the secondary effects of malnutrition (cf. Kaye, 1996); however, in "normal" starvation the pattern of neuroendocrine changes is distinctly different, leading to lethargy and hunger, not hyperactivity and food refusal. Furthermore, most of these "defects" resolve with normalization of body weight, making their roles as primary etiological factors less likely (Walsh & Devlin, 1998). Investigators seeking animal models of anorexia repeatedly comment on unanticipated, seemingly paradoxical sets of behavioral and physiological results (Hoebel, Leibowitz, & Hernandez, 1992), findings that are consistent with the existence of functionally coordinated neurophysiological mechanisms rather than simply defects. Rather than interpreting neuroendocrine abnormalities as dysfunctions or as secondary factors, it may be more appropriate to view them as evolved mechanisms to facilitate fleeing from famine.

Adapted to Flee Famine Hypothesis (AFFH)

In practice, most eating disorder specialists integrate biomedical, social, and psychological theories, assuming that most women and girls feel social pressure to diet in a thin-obsessed culture, and that, if an individual is psychologically susceptible and biologically vulnerable, she may develop AN. Such biopsychosocial models are useful as far as they go. They do not explain, however, why AN symptoms take their distinctive form or acknowledge the biological forces arrayed against recovery. I suggest that integrating conventional theories into a Darwinian framework will enable researchers to return successfully to the questions of why this disorder takes its peculiar form and suggest treatment strategies.

The AFFH proposes that AN symptoms of restricting food, hyperactivity, and denial of starvation reflect the operation of adaptive mechanisms that once facilitated migration in response to local famine. A number of medical disorders are thought to arise from defenses against problems humans faced in ancestral environments (Nesse & Williams, 1994). For example, some forms of obesity probably result from adaptations to marginal habitats by storing extra calories as fat (Neel, 1962). Although obesity is now a major health concern, in the past this "thrifty genotype" would have allowed individuals to survive famine.

Appetite and eating are components of highly regulated and tuned physiological and behavioral systems. Normally, when a person or animal begins to lose weight, behavioral and neuroendocrine mechanisms conserve energy and increase desire for food. Metabolic rate declines as much as 40% and increasing hunger prompts food searches. If starvation continues, individuals become obsessed with eating, lethargic, and depressed (G. H. Anderson & Kennedy, 1992; Keys et al., 1950; Prentice et al., 1992). Presumably, in most environments, these cognitive, affective, and physiological adaptations facilitate survival in lean times.

However, the ecological situation during most of human history was different than that of today where humans are settled in every good habitat. Many preagrarian human populations were nomadic foragers living at very low densities (Potts, 1998; Stiner, Monro, Surovell, Tchernov, & Bar-Yosef, 1999). When extreme weight loss was due to depletion of local food resources, some individuals might have done better by seeking foraging opportunities elsewhere. Then the lethargy that conserved energy and the hunger that motivated single-minded search for food could be maladaptive. To migrate efficiently, individuals' bodies would have to turn off those usual adaptations to starvation. The ability to stop foraging locally, to feel restless and energetic, and optimistically to deny that one is dangerously thin could facilitate such a last-ditch effort. Here it is proposed that core anorectic symptomology includes three distinct adaptations specifically relevant for surviving past famine conditions: ignoring food; hyperactivity; and denial of starvation, including distorted body image.

If the AFFH is correct, several predictions should be fulfilled: (a) The core symptoms should make adaptive sense for starving foragers and show evidence of evolutionary engineering; (b) AN should be relatively independent of psychopathology—that is, any practice that results in low weight should put some individuals at risk; (c) core anorexic symptoms should be described throughout recorded history; (d) food refusal ought to be present in some other species when feeding competes with activities crucial to survival

or reproduction; (e) the anorexic syndrome of ignoring food and restless energy ought to be found in some other species that inhabit similar ecological niches, when body weight drops very low; (f) AN should be more common in human lineages that most recently gave up a hunting/gathering nomadic life; (g) compulsions to move and to avoid food should be experienced as more urgent than other needs; (h) the AFFH should be consistent with the physiological, metabolic, and behavioral changes that occur during AN; and (i) the theory should account for its occurrence in both humans and animals.

The following sections review evidence for behavioral, cognitive, and biochemical alterations contributing to the food refusal, hyperactivity, and denial of starvation hypothesized to have evolved originally to facilitate leaving a food depleted area. Table 1 summarizes evidence for and against alternative hypotheses to explain AN.

Food Restriction

People with AN restrict food although they are very thin. This paradoxical behavior has also been reported in accounts of people who have lost considerable weight for any reason, including those fasting for religious and political reasons. Food refusal is found in food-restricted rats with access to a running wheel in the laboratory and in lean-bred pigs with wasting pig syndrome (Bell, 1985; Epling & Pierce, 1988; Treasure & Owen, 1997). Furthermore, a number of species stop eating and lose considerable weight during their annual migrations (Mrosovsky & Sherry, 1980).

During the Middle Ages, when many pious women fasted, some developed "holy anorexia" (Bell, 1985). The Roman Catholic Church officially canonized over 85 very thin saints, blesseds, or venerables who were recognized, in part, for their seemingly miraculous ability to live with little food. Diaries and other first-

Table 1
Summary of Evidence for and Against Alternative Hypotheses to Explain Anorexia Nervosa (AN)

Hypothesis	Supporting evidence	Contradictory evidence
Psychopathology leads to refusal to maintain body weight, intense fear of fat, and control battles with parents.	Symptoms are paradoxical and life-threatening, suggesting serious psychopathology.	There is no correlation between development of AN and family psychopathology.
	Most modern anorectics express fear of fat.	Many non-Western patients and historical anorectics, including anorectic saints, did not express fear of fat.
	Adolescents fight over control of eating and exercising.	The same symptoms are present in mature women, men, and children as in adolescent patients.
	Denial of illness and body image distortions may reach delusional proportions.	There is no correlation between serious premorbid psychopathology and AN.
	Perfectionism and rigidity are correlated with AN and contribute to dieting.	
Biomedical pathology leads to food refusal and hyperactivity.	These are consistent abnormalities in neurotransmitters and hormones that contribute to appetite and activity.	Most neurotransmitters and hormones involved in appetite and locomotion return to normal with weight restoration; abnormalities are contradictory, appear functional.
	High heritability indicates that genes make a significant contribution to AN.	Heritability can result from (polymorphic) adaptations as well as pathology.
Patriarchies' oppression of women and girls leads to symptoms.	AN represents a hunger strike against an oppressive patriarchal culture.	Symptoms are consistent across times, sexes, species, and cultures.
	Modern weight standards pressure females to diet to unnaturally low weights.	
Symptoms are archaic adaptations to flee famine.	Food refusal is found in dozens of circumstances and species when feeding competes with other activities such as migrating and breeding.	Anorectics have a great resistance to eating although food is available.
	Many animals increase activity in times of food shortage; hyperactivity can be induced in laboratory rodents by restricting feeding times.	Hyperactivity is not always present in humans throughout the illness.
	AN-like syndromes have developed in several other species at low body weight.	
	Urges to move and refuse food are experienced as desperate compulsions.	
	High heritability indicates that genes make a significant contribution to AN, suggesting that natural selection may have been involved in development of traits.	
	Most neuromodulators and hormones involved in appetite and activity function in a manner to facilitate migration by promoting activity and satiety.	Appetite-promoting neuropeptide Y and ghrelin are elevated.

hand accounts show that these women manifested typical AN symptoms of aversion to food, overactivity, and denial of starvation, although they did not express modern anorectics' fear of fat.

Some domestic animals bred for extreme leanness develop a strikingly similar constellation of symptoms. Pigs with so-called wasting pig syndrome voluntarily restrict their intake of normal food, although they may eat large amounts of straw; they are also restless, moving incessantly around their pens (Treasure & Owen, 1997). Six percent of pigs from lean breeds develop this syndrome, most frequently in adolescence. Hog farmers believe this occurs when these already thin pigs lose additional weight because of stress from maternal separation or from bullying by other pigs. The similarity in the behaviors of anorexic pigs, rats, and anorectic people from other historical epochs raises the possibility of similar biological mechanisms.

In addition, if starved in the laboratory, a number of mammal species develop AN-like symptoms in which they ignore their food and exercise excessively (Epling & Pierce, 1992). If feeding time is restricted and they have access to a running wheel, rats and eight other rodent species will self-starve in the presence of food (Epling & Pierce, 1984; Hall & Hanford, 1954; Pirke, Brooks, Wilckens, Marquard, & Schweiger, 1993; Routtenberg & Kuznesof, 1967).

Appetite also appears to be suppressed in wild animals when hunger and feeding compete with more important activities. Animals of many species stop eating, even though food is readily available, when they defend their harem or territory, incubate, molt, or migrate (Mrosovsky & Sherry, 1980). Body weight may drop considerably as these animals actively pursue other agendas (Mrosovsky & Sherry, 1980). Presumably, migrating animals do not "eat and run" because searching for food, when it is scarce, would interfere with migration.

Mrosovsky and Sherry (1980) speculated that lowered set point for body weight could be one physiological mechanism behind this appetite suppression. Most mammals' body weight is regulated around a genetically determined "optimal" weight range. Alterations in appetite, metabolic rate, and activity allow animals and humans to maintain stable weight despite variation in food quality, food quantity, and activity (Keesey, 1993). Signals involved in energy homeostasis control food intake, in part by modulating the response to satiety signals in the brain (Schwartz, Woods, Porte, Seeley, & Baskin, 2000; Sims, 1976). The hypothalamus plays a pivotal role in behavioral and emotional reactions and contains the major center for sensations of satiety and hunger. When satiety is reached, eating becomes aversive (Johnson et al., 2000).

Normally when individuals begin to starve, neurochemical signals of hunger are elevated and signals for satiety and activity are lowered (G. H. Anderson & Kennedy, 1992; Leibowitz, 1992; Prentice et al., 1992). In contrast, AN researchers have found that most neuromodulators and hormones influencing hunger, satiety, and activity are present in unusual concentrations that are opposite to those found in normal starvation. These findings are all consistent with adaptations to turn off eating and turn on traveling. They are also consistent with AN patients' descriptions of their subjective experience of finding it very difficult to eat and feeling restless and driven to exercise. Table 2 summarizes biochemical, behavioral, and cognitive changes found in normal starvation, underweight anorectics, and weight-recovered anorectics.

Individuals with AN often say they have difficulty eating because they feel full (satiated) after small meals. Anorexic rats are also quickly satiated (Kanarek & Collier, 1983). A number of biochemical signals could lead to this anorexic phenomenology. Leptin is a hormone manufactured in the fat cells that regulates fat stores by decreasing appetite when fat is gained. It acts on the hypothalamus by stimulating melanocortin receptors that inhibit food intake. Researchers have found that leptin levels increase in anorectic patients before substantial weight has been regained during recovery (Mantzoros, Flier, Lesem, Brewerton, & Jimerson, 1997). These findings led Hebebrand et al. (1995) to conclude that anorectics' low body weight appears to be physiologically defended as Mrosovsky and Sherry (1980) had supposed for anorexias in wild animals.

Chronic elevations in the satiety-producing biochemical signalers cholecystokinin, serotonin, and dopamine and lowered levels of appetite-promoting galanin and norepinephrine would also contribute to difficulty eating and early satiety (for reviews, see Gendall, 1999; Kaye, 1996; Pirke et al., 1993; Støving et al., 2001). Indeed, anorexic rats and people with AN are behaving just as one would expect of individuals in this fairly complex neuroendocrine state (see middle column of Table 2).

Researchers are closing in on the genetic mutations that lead to some of the biochemical alterations of appetite and satiety in AN. Leptin and melanocortin receptors and antagonists allow precise regulation of fat stores. As some obesity has been found to be caused by mutations that lead to loss of function in molecules of the leptin-melanocortin chain, it was suspected that a mutation that led to loss of function on the melanocortin receptor antagonists countering inhibition of eating might be linked to AN. Indeed, 11% of AN patients in one study carried the same genetic mutation on a melanocortin antagonist (Adan & Vink, 2001). Thus, when someone with this mutation starts to lose weight, it triggers the gene making the nonfunctional antagonist, leading to abnormally early satiety.

Researchers had also assumed that abnormal levels of appetite signalers would represent stable premorbid etiological risk factors for AN, but with two exceptions discussed later, all of these neurochemicals return to normal with weight restoration. Some workers then speculated that the abnormalities in underweight patients with AN might be a secondary result of malnutrition that perpetuates the eating disorder (Kaye, 1996; Pirke et al., 1993; Støving et al., 2001). Remaining unanswered by biomedical theorists is the question of why the malnutrition in AN results in decreased hunger and increased activity, rather than the reverse. In fact, these findings are all consistent with the adaptations to migrate when starving.

The levels of two biochemicals remain abnormal in weight-recovered anorectics. Galanin, a neuropeptide that stimulates appetite for fat, stays low in patients even after weight has been restored. Kaye (1986) suggested that this may be a "scar" secondary to years of malnutrition; or it may be a predisposing factor. It is impossible to know this without conducting prospective studies, but these are not considered feasible with such a rare disorder. Abnormally low levels of galanin could contribute to fat aversion in weight-recovered as well as in thin patients with AN.

Serotonin also remains at atypical levels in recovered anorectics. Elevated serotonin has been implicated in the perfectionism and

Table 2
Behavioral, Cognitive, Physiological, and Neuroendocrine Changes Exhibited in Normal Starvation, Underweight Anorectics, and Recovered Anorectics

Changes	Normal starvation	Underweight anorectics	Recovered anorectics
Cholecystokinin (satiety for carbohydrates)	Low	Elevated	Normal
Leptin (satiety)	Low	Elevates more rapidly than weight	Normal or elevated
Serotonin (satiety for carbohydrates)	Low	Elevated	Stays high
Dopamine (satiety for fats)	Low	Elevated	Normal or decreased
Neuropeptide Y (appetite)	Elevated	Elevated	Above normal
Ghrelin (appetite)	Elevated	Elevated	Normal
Galanin (appetite for fat)	Elevated	Low	Stays low
Norepinephrine (appetite)	—	Low	Normal
Cortisol (activity promotion)	—	Elevated	Normal
Food—seeking activity	Obsessive	Ignore/avoid	May restrict certain foods or binge eat
Locomotor activity	Decreases	Increased	More normal
Denial of starvation	Absent	Present	Normal
Basal metabolic rate	Decreases up to 40%	Decreases up to 40%	Normal

Note. Dashes indicate that the data are not available.

rigidity that characterize many people with AN (Kaye, Gendall, & Strober, 1998). As discussed earlier, these personality traits are the only consistent psychological risk factors.

More speculatively, changes in cortisol, endogenous opioids, oxytocin, and vasopressin have all been suggested as proximate mechanisms in AN. Preexisting variation in populations usually provides the raw material for natural selection. Although it is atypical, stress-seeking behavior has been described in several mammalian species, including humans (Piazza et al., 1993). Bergh and Sodersten (1996) suggested that a mechanism for the development of AN may be the reward that some individuals experience from stress. Reward is mediated by mesolimbic dopamine neurons in the brain, and adrenocortical secretions enhance the dopamine reward system. Glucocorticoids, the final product of corticotropin releasing hormone (CRH) activation, can cause euphoria in some humans, and some animals readily self-administer corticosterone (Piazza et al., 1993). In support of this stress-reward hypothesis is the finding that CRH, which can produce euphoria (Bergh & Sodersten, 1996), decreased feeding (Britton, Hoffman, Lederis, & Rivier, 1984) and hyperactivity (Sutton, Koob, Le Moal, Rivier, & Vale, 1982), increases in underweight anorectics, and returns to normal on weight restoration (Kaye et al., 1987; Støving et al., 2001). Thus, in some individuals the stress of overexercise and stringent dieting may activate reward mechanisms in the brain, so that self-starvation is initially rewarding and subsequently conditioned. This is also consistent with an adaptation to flee famine.

Other putative mechanisms for decreased appetite in AN include modifications in endogenous opioid systems. Although morphine usually stimulates increased feeding as well as sedation in mice, certain mouse strains respond to morphine with anorexia and hyperactivity (Buck & Marrazzi, 1987). These strains' atypical opiate systems may provide useful models for AN in humans. A genetic variant affecting opioid tracts may facilitate those individuals' developing AN when body weight drops below a critical level. In support of this hypothesis are observations that an opioid antagonist, naloxone, increases weight gain in AN patients (Moore, Mills, & Forster, 1981) and that AN patients show elevated endogenous opiate levels (Kaye, Pickar, Naber, & Ebert, 1982). Injections of 2-deoxy D glucose, an endogenous opioid, decreased judgment of hunger in AN subjects, although it increased hunger in normal weight subjects (Nakai, Kinoshita, Koh, Tsujii, & Tsukada, 1987).

Some female AN patients also have been found to have abnormally high levels of vasopressin and reduced levels of oxytocin that normalize with weight restoration (Kaye, 1996). These hormones have a number of effects, including effects on memory. Oxytocin is implicated in forgetting the pain of childbirth, whereas vasopressin aids for remembering aversive consequences. Although data on males is contradictory, it has been suggested that high vasopressin and low oxytocin levels in AN might enhance retention of the aversive consequences of eating (Demitrack et al., 1990).

In contrast to biochemical alterations that promote satiety, neuropeptide Y and ghrelin, potent appetite-stimulating molecules, are elevated in underweight anorectics (Cuntz et al., 2001; Kaye, 1996). This elevation may be responsible for the obsessional interest in food manifested by many people with AN, whereas the biochemical changes discussed earlier lead to food refusal. Although people with AN often feel satiated after small meals, at the same time they may experience ravenous appetite and cook elaborate dinners for others. Similar to the hungry ghosts in hell, people with AN may be dogged simultaneously by ravenous hunger and queasy satiety. Why would a powerful appetite-stimulating signaler remain high in AN if the function of the other changes was to postpone food searches until migration was completed? Presumably, if there were no hunger signaling mechanisms, prehistoric people with AN would not have begun eating when they arrived at new feeding grounds.

Bruch (1978) observed that anorectics "are often hungry, even frantically obsessed with food and eating but consider self-denial

and discipline the highest virtue and condemn satisfying their needs and desires as shameful self-indulgence" (Preface, p. x). Of course, higher brain centers can override otherwise automatic responses to hypothalamic signals and urges from the body. If feeding is inappropriate for some reason, it can be postponed. Neurons in the lateral hypothalamus receive higher cortical input from the cerebral cortex and limbic system, as well as sensory information coming up from the body. Input from limbic structures adds emotional and motivational qualities to the cortex's cognitive interpretation of sensory signals. Thus, modifications from cortical and limbic signals could theoretically have evolved to help migrating people override hunger signals.

The phenomenology and neuroendocrinology of AN suggest that lower level neuroendocrine processes signal satiety *and* hunger, whereas powerful higher level processes urge restraint. Higher cortical mechanisms are clearly involved in food refusal. Anorectics say they have difficulty eating because they feel deeply that they "should not," reporting feelings of virtue, triumph, mastery, superiority, and grandiosity about being able to abstain from food (Garner & Bemis, 1982). Although anorectics' sense of virtue in eating little has been seen in the context of a repudiation of the body's appetite or mother's control, individuals with AN of both sexes and all ages exhibit this odd symptom.

Darwin (1872) theorized that emotions evolved to motivate animals to behave adaptively. A sense of virtue in ignoring hunger pangs may constitute a component of the psychological and behavioral mechanisms for ignoring food. People often feel virtuous about withstanding painful rigors in hunting or sports, communal work, and in battle (Guisinger, 2000). The anorectic saints believed their ability to fast, even when hungry, was a sign of their piety and demonstrated that God had chosen them for a special task (Bell, 1985). Even secular anorectics describe an almost religious thrill about abstaining from food and pushing themselves to exercise harder (Hornbacher, 1998).

Hyperactivity

Restless energy has been part of the symptom picture of AN since it was first described medically. This report by William Gull in 1874 is typical: "The patient complained of no pain, but was restless and active. This was in fact a striking expression of the nervous state, for it seemed hardly possible that a body so wasted could undergo the exercise which seemed agreeable" (p. 24).

Although the *DSM–IV–TR* considers undue exercising secondary to desire for weight loss, many researchers include hyperactivity among the primary symptoms of AN (Beumont et al., 1994; Casper, 1998; Davis, Kennedy, Ravelski, & Dionne, 1994; Garfinkel & Garner, 1982; Kaye, Gwirtsman, George, & Ebert, 1986; Walsh & Devlin, 1998). Prentice et al. (1992) asserted that "the hyperactivity associated with AN stands out as the greatest difference between any of the eating disorders and famine or experimental underfeeding" (p. 259). Bergh and Sodersten (1996) called for "a return to the two symptoms included in original descriptions of the disorder that have been included in many descriptions since then: self-starvation and enhanced physical activity" (p. 21).

Hyperactivity is not invariably present throughout the illness; however, a retrospective study of anorectics found that increased activity was present in all patients at some time (Kron, Katz, Gorzynski, & Weiner, 1987). In this article hyperactivity is considered a primary symptom of AN. One anorectic describes her experience: "Pure adrenaline kicks in when you're starving—you're high as a kite, sleepless, full of a frenetic, unstable energy . . . your own drive and energy themselves are intense and focused. Your sense of power is very, very intense" (Hornbacher, 1998, p. 105).

Anorectics' hyperactivity may be a form of "migratory restlessness." Many species increase activity in times of food shortage (Vincent & Pare, 1976). *Zugunruhe*, or seasonal migratory restlessness, is common in birds and mammals (Helms, 1963), and researchers have long known that depriving laboratory rats of food increases wheel running (Hall & Hanford, 1954). Laboratory rats normally run less than 1 km/day, but if they are starved and then maintained at 70% of normal weight, they run up to 20 km per day! These emaciated rats can maintain this extraordinary activity for up to 90 days. Greater weight loss leads to greater hyperactivity. As a group, female rats become hyperactive earlier than males, a sex difference mirrored in humans. Rats also exhibit individual differences in the degree of weight loss that triggers increased activity; however, by the time rats have lost 30% of their body weight, they are all hyperactive (Pirke et al., 1993).

Even more surprising, normal weight rats develop symptoms of hyperactivity if ad lib access to food is restricted and they have an opportunity to run on a wheel (Epling & Pierce, 1984; Routtenberg & Kuznesof, 1967). Rats given constant access to food normally eat 10 to 12 small meals a day. If a rat's mealtime is restricted to 1 hr per day, it can adjust to this restricted feeding schedule and maintain its weight, but if housed with an activity wheel, it begins to run excessively. Rats maximally increase running around feeding time (Pirke et al., 1993). This starts a vicious cycle in which the rat ignores its food and runs more as weight drops (Epling & Pierce, 1984). At the end of 1 week animals may not eat at all. If the process is allowed to continue, these animals die of self-starvation.

The neuroendocrine mechanisms for hyperactivity probably include corticotrophin releasing hormone, discussed earlier, and serotonin; both of these are elevated in anorexia (Kaye et al., 1987; Støving et al., 2001). Broocks, Schweiger, and Pirke (1991) studied serotonin turnover in rats and posited that a positive feedback cycle could result in the hyperactivity of AN. They wrote that "the greatly increased serotonin turnover observed in semistarved induced hyperactivity suggests the following hypothesis: activity-induced serotonergic stimulation leads to reduced food intake and body weight, which might be a further stimulus for locomotor activity" (Broocks et al., 1991, p. 387).

Experimental conditions in which excessive running and self-starvation do *not* develop are also consistent with the AFFH. Rats do not lose weight and become anorexic if given an exercise wheel and ad lib feeding. Further, if restricted to one mealtime but given no wheel, they eat enough during the hour to maintain weight. Furthermore, if normal weight rats, housed with a wheel, are given four 15-min opportunities to feed instead of a single 1-hr bout, they also do not become hyperactive, and already anorexic rats reduce activity on this regime (Epling & Pierce, 1984; Kanarek & Collier, 1983), as though the increase in opportunities to feed is registered as an adequate foraging environment with "therapeutic" effects.

Is there evidence that ancestral humans migrated frequently? Indeed, these subtropical apes flooded over the entire globe in the blink of an eye in geologic time. Before historic times, humans were already more widely distributed than any other mammal (Tudge, 1996). A "weedy" species (Jablonski & Bottjier, 1991), humans are generalists and opportunists who disperse widely and tolerate a broad range of habitat conditions (Quammen, 1998).

Furthermore, modern humans were such effective hunters that they left a trail of decimated species behind them when they occupied Australasia and the Americas (Diamond, 2000). Thus, it can be surmised that hunters repeatedly overhunted native prey populations, leading to local famine and the need to migrate in search of new food. A smattering of individuals with the ability to become anorectic may have facilitated this dispersal.

Behavioral psychologists Epling and Pierce (1988) also proposed that rats' hyperactivity in response to underweight or feeding restriction is an evolutionary adaptation to famine. They speculated that

> during times of food scarcity, organisms can either stay and conserve energy (e.g., hibernate) or become mobile and travel to another location (e.g., migration) . . . travel should not stop when food is infrequently contacted, as stopping to eat may be negatively balanced against getting to a richer food patch. (Epling & Pierce, 1988, p. 480)

They noted parallels with human AN, but, as discussed later, their conclusions were ridiculed or ignored by AN workers.

Denial of Illness and Delusional Body Image

A startling symptom of people with AN is their sincere conviction that they are well, even fat, when perilously thin. Body image distortion and denial of illness were also characteristic of anorectic saints and other anorectics throughout history (Bell, 1985). This distorted body image often represents an extreme departure from reality and has been interpreted as an indication that patients are more disturbed than their premorbid history may have indicated.

However, such a delusion could have been adaptive in the ecological situation just described. Those starving foragers who deceived themselves about their desperate physical condition and, believing they were healthy, confidently moved on, may have survived at higher rates than those who despaired when accurately assessing their emaciated condition. If raising group morale contributed to successful migration, their optimism could also have benefited the kin groups of which they were a part.

Psychologists realized that body image is not immutable when neurologists reported extreme body image distortions in their patients, including denial of the existence of body parts and lack of awareness of the incapacitation of paralyzed body regions (Fisher, 1990). Such findings show that body image is cognitively constructed. The neuropsychological substrates that underlie cognitive constructs of body image are presumably subject to natural selection.

Furthermore, evolutionary psychologists, cognitive neuroscientists, and psychoanalysts have all argued that some self-deception, that is, the active misrepresentation of reality in the conscious mind, is integral to healthy psychological functioning (Nesse & Lloyd, 1992; Trivers, 2000). According to depression researchers, healthy people consistently distort reality in ways that make it less threatening and more positive than it is (Martin, Abramson, & Alloy, 1984). People normally engage in overly positive self-evaluation, exaggerated perception of control and mastery, and unrealistic optimism (Taylor & Brown, 1988). Trivers (2000) argues that traits of positive self-deception could be selected when they serve to orient the individual favorably toward the future. If some self-deception in the service of optimism is normal and body image is constructed, it is possible to imagine selection for self-deception about one's nutritional state that is keyed to signals that fat levels have dropped below some critical value. Denial of starvation could promote optimism to travel elsewhere.

Anorectics' serene obliviousness to their emaciation has been described for centuries. Lasegue wrote in 1873 of anorectic patients' "inexhaustible optimism against which supplications and menaces are alike of no avail" (p. 626). After an initial exuberance, this optimism often alternates with depression and despair, but body image distortion generally remains firmly entrenched until normal weight is regained. The phenomenology of AN suggests that individuals with AN develop distorted perceptions about their bodies' condition and obliviousness to signs of starvation automatically when their weight drops below a critical level.

Research on the mechanisms of self-deception is in its infancy. Trivers (2000) noted that "we especially need more biological evidence on the genetics, endocrinology, physiology, and neuroanatomy of self-deception and we need to integrate very disparate findings from experimental, social and clinical psychology into the evolutionary analysis" (p. 130). However, he predicted, "Evolutionary theory promises to provide a firm foundation for a science of self-deception, which should eventually be able to predict both the circumstances expected to induce greater self-deception and the particular forms of self-deception being induced" (Trivers, 2000, p. 130).

The fact that serum levels of endogenous opioids are elevated in people with AN may contribute to feelings of euphoria and unconcern (Kaye et al., 1982). Indeed, opioids and most recreational drugs heighten self-deception. Anorectics' denial of starvation seems likely to be the result of adaptive mechanisms because it is constant across cultures and times; it is complex and apparently finely tuned; it serves a significant function; and the attitudes and behaviors it facilitates, especially the courage to travel, would be helpful to survival in times of famine.

One would also expect that if the anorectic's symptoms are highly engineered neurocognitive adaptations, they could be disrupted by neurological insult. This apparently happened when an anorectic woman developed encephalitis (Ho & Birmingham, 2000). The patient had a 12-year history of AN but lost her anorexic behaviors following an episode of encephalitis with associated hypoxic brain injury. The classic AN symptoms of body image distortion and food refusal simply disappeared. Her physicians report that the woman "began eating a full, normal diet which she had not done for many years . . . it did not seem to perturb her" (Ho & Birmingham, 2000, p. 98). During this time her weight increased from 37.8 to 51.1 kg, and her body fat content increased from 7.5% to 18.5%. Unfortunately, she was still underweight, and when the neurological sequelae resolved, the anorexic cognitions and behaviors returned. The authors concluded,

> The remission of anorexia nervosa during the episode of . . . brain injury suggests that there is key involvement of higher cortical functioning in the etiology of anorexia nervosa. This theory is supported by the return of anorexia nervosa traits, once normal neurological function resumed. (Ho & Birmingham, 2000, p. 98)

This report, suggesting that cognitive symptoms of AN probably result from coordinated mechanisms involving cortical function, along with findings that neuroendocrine changes normalize on weight restoration suggest that the symptoms associated with AN result from adaptive and finely tuned neurobehavioral mechanisms rather than from biological defects or global system breakdown secondary to advanced starvation.

Discussion

Epidemiology

There are several epidemiological puzzles that need to be considered in the context of the AFFH.

Why don't subjects in semistarvation experiments and famine victims develop AN? I believe that the primary reason AN is not identified among famine victims is that AN is polymorphic in humans; most people apparently do not have a genetic ability to develop AN (Holland et al., 1988; Strober, 1995), just as most people cannot store a great many extra calories as fat (Bouchard, 1994; Sims, 1976). Both the ability to become fat and the ability to become anorectic may have been under recent selection pressure as human populations invaded virgin or marginal habitats. In settled areas (i.e., much of Africa for the past 100,000 years) it was probably better for starving people to stay put, conserve energy, and keep searching locally for food, as traveling could result in encounters with hostile neighbors.

As predicted by the AFFH, eating disorders appear to be overrepresented in lineages that most recently gave up a nomadic life. Crago, Shisslak, and Estes (1996) reviewed reports of prevalence rates of eating disorders among major American ethnic groups. They found that eating disorders are most common among Native Americans, equally common among Hispanics and Caucasians, and least common among Asians and African Americans. Although this review lumped together AN and bulimia nervosa and reported few studies for some groups, it is suggestive that this pattern reflects the sequence of migration out of Africa.

Another reason that AN is not reported among famine victims is that observers may fail to recognize symptoms in this context. After both world wars workers in European famine relief did comment on some starving individuals' decreased appetite and increased mobility (Blanton, 1919; Keys et al., 1950; Russell Davis, 1951). In Keys et al.'s (1950) famous semistarvation experiment, the researchers were convinced that none of the 32 young men who volunteered to lose 25% ABW became anorectic. However, they noted, "Some men exercised deliberately at times. Some of them attempted to lose weight by driving themselves through periods of excessive expenditure of energy" (Keys et al., 1950, p. 828). The researchers attributed this inexplicable behavior to a futile ploy to obtain increased bread rations (Keys et al., 1950), but it may have been an AN symptom, mislabeled by the experimenters.

Furthermore, if a mother in a famine denies her own hunger and feeds her family, this behavior may be interpreted as maternal altruism, whereas it may be facilitated by anorexic symptoms. Indeed, AN may represent a hitherto unappreciated example of altruism. Although refusing food for themselves, anorectic women are often enthusiastic cooks for others, particularly their families (Garfinkel & Garner, 1982). Although this behavior is neither desirable nor truly altruistic today, people with anorexia often report that their life has a desperate, humorless quality as though they were involved in an urgent enterprise. Perhaps the highly altruistic orientation of a number of eminent women reported to have had AN, including Eleanor Roosevelt (Silverstein & Perlick, 1995), Simone Weil (Gray, 2001), and Joan of Arc (Warner, 1981), was supported partly by a sense, born of AN, of being on a grave mission.

Why are females more likely to develop AN? Accounting for AN's epidemiology requires considering social, psychological, and biological factors and their interactions.

As discussed above, modern girls and women feel social pressure to diet to very low weights. Females may try to attain these unnatural weights because, from an evolutionary perspective, a primary job of an adolescent girl is to select the best father she can obtain for her future children. In monogamous species females compete for good quality males. To this end, she tries to make herself as attractive as possible. This "vanity" is not trivial; attracting a good mate increases her chances for genetic immortality.

Unfortunately, beauty standards have sometimes involved female deformation, particularly in repressive cultures. Through the centuries girls have had their feet bound, their necks stretched, and their skulls shaped. Today's girls diet. The current epidemic of eating disorders is a result of girls and women attempting to diet below their normal body weight to look more attractive by modern standards. Currently, 2% of American girls and women have a serious eating disorder, 10% have more mild variations, and 80% of women are dissatisfied with their bodies and think they are too fat (Seid, 1994). As previously less-represented ethnic groups become more acculturated to mainstream American values, their rates of eating disorders, especially among women and girls, increase (Dolan, 1991; Lee & Lee, 1996).

The skewed sex ratio of AN is not solely a result of modern pressure on women to be thin, however. Even when thinness was not in fashion, reports of female anorectics have outnumbered those of male anorectics (Vandereycken & van Deth, 1990). Intriguingly, female rats also developed anorexia more quickly, and starving female rats were more eager to push a lever to gain access to a running wheel (Pirke et al., 1993). Perhaps female rats and humans are more likely to develop AN because traveling out of their natal territory may have been historically less risky for females. Primatologists have observed that migrating is dangerous for male chimpanzees because strange bands frequently kill males, whereas females are often integrated into the new group (Wrangham & Peterson, 1996). Thus, traveling may be differentially selected in females. Genetic evidence that women mated more exogamously than men in humans' evolutionary past supports this (Seielstad, Minch, & Cavalli-Sforza, 1998).

Why are adolescents more vulnerable? One modal period for developing AN is just after a prepubescent increase in height and before normal fat has been added to breasts and thighs to sculpt the

characteristic figure of mature women (American Psychiatric Association, 2000).

In early puberty many girls have a lower body mass index than at any other time in their lives. Hebebrand et al. (1995) have commented, "The hormonal changes associated with puberty might lead to intermittent weight loss with or without the psychopathological features of AN in genetically predisposed females during their puberty or adolescence" (p. 1624).

Contrary to the popular idea that fatter girls are most at risk to develop this eating disorder, anorectic humans (like anorexic pigs) often start out genetically lean. Probands with AN, on average, weighed less than controls before their illness (Strober, 1995). When these already lean individuals overexercise or diet, they may trigger the symptoms of AN.

Why are perfectionist females more vulnerable? Biological and social factors account for some aspects of the epidemiology of AN, but researchers are still left to explain why, although all girls and women in western industrialized society are exposed to these very thin models, only a few attain a fashionable and dangerous 85% ABW.

Here, psychological factors enter the picture. Clinicians have long known that a risk factor for developing AN is being compliant and driven (Bruch, 1978). Perfectionism is the personality trait most consistently associated with AN (Bastiani et al., 1995; Srinivasagam et al., 1995). Hunger is generally extremely aversive; most American women say they would like to lose what would be unhealthy amounts of weight (Rodin, Silberstein, & Striegel-Moore, 1985), but thankfully, only a few actually achieve this. Presumably the same qualities that help perfectionistic individuals succeed in school and work—the ability to work hard, delay gratification, and ignore suffering in pursuit of a goal—help these dieters drive their weight dangerously low.

Evolutionary Considerations

Unpredictable environments often favor the selection of physiological and behavioral mechanisms for versatile or novel responses (Potts, 1998). In contrast to the ecological situation of relatively stable food availability in subtropical forests, the new hominid hunter-gatherers of the last 100,000 years were subject to boom and bust cycles as they pushed into temperate regions with naive prey populations. Abilities to store fat in famine-prone habitats and to migrate when resources were depleted could have been useful adaptations for surviving in diverse and unpredictable habitats. Most mammals cannot store many extra calories as fat, and no ape exhibits such variation in body weight; however, no other ape occupies so many different habitats. Humans are currently more numerous and more widely distributed than any mammal that ever lived, in part because our ancestors moved on when they had depleted the resources of an area.

How could traits be selected that, like the symptoms of AN, decrease fertility and even kill the carrier? Evolutionary biologists have repeatedly pointed out that adaptations are "makeshift" and may be dysfunctional outside the ecological context in which they evolved (Nesse & Williams, 1994). AN would have functioned more effectively in nomadic lifeways, and like obesity (which Paleolithic Venus figurines indicate was admired at one time), outside the ecological setting and social structures in which it evolved AN can be deadly. Furthermore, if the extended families of anorectics survived at higher rates, kin selection could favor these traits even if the person with AN died without reproducing (Hamilton, 1964).

In the Pleistocene era some behavioral mechanism probably helped anorectic nomadic tribal members to eat when food was once again plentiful. If the traditions of extant nomadic tribes are any indication, this mechanism involved intense social facilitation of eating. In traditional societies when more food becomes available after a shortage, it is generally offered to all in a celebratory way (Isaac, 1978). Prayers, thanksgiving, and ritual feeding of one another accompany breaking the fast. Refusing such food insults the giver. In the radically individualistic western culture, it may be hard to imagine the social pressure that is brought to bear on deviant individuals in tribal cultures. Humans are deeply social (Guisinger & Blatt, 1994), and AN probably functions best and is most easily switched off in a group context. People who have regained their weight generally attribute their recovery to social support (Gottlieb, 2000; Hornbacher, 1998). (See treatment implications below.)

The probability that anorectics were a minority in most groups (based on current prevalence) could have worked to everyone's advantage. When resources were depleted and the tribe despaired, an anorectic's energy, optimism, and grandiosity could mobilize the other members to heroic marches, much as Joan of Arc, an apparently anorectic peasant girl, inspired soldiers to march with her (Warner, 1981). When a starving tribe reached a new hunting/gathering ground, social pressure exerted by family and friends would in turn have helped the anorectic member(s) to begin eating again.

Treatment Implications

Bruch (1978) believed that patients' determined refusal to eat derived from their need to separate from intrusive and controlling parents. This conceptualization did serve to ameliorate the hostility that the anorectic's symptoms engendered in those around her, but unfortunately replaced it with parent blaming. Of course some children do begin restricting food in response to parents who are controlling, intrusive, and even incestuous, but AN also appears in the absence of parental pathology. Patients who see their families as loving and supportive are often encouraged to uncover subtle parental criticism and control in the interests of recovery.

In practice, Bruch taught that therapists are most effective when they adopt a naive stance and listen closely to the patient's story, treating the patient as a true collaborator. She emphasized that therapists should avoid theory-based interpretations of unconscious wishes (Bruch, 1973). However, many who came after her were more dogmatic in adherence to the theory.

The AFFH relieves therapists of searching for psychological or familial reasons for the bizarre symptoms, freeing them to listen without preconceptions to their patients' experiences. For example, struggles over control are a commonly reported facet of relationships between people with AN and those wanting them to get better. Starving patients' food refusal is extremely frustrating and often frightening to others. It is hard not to experience this as a power struggle, especially because traditional psychological theories attribute symbolic meaning to the symptom. What parents

and therapists believe about the meaning of food refusal can itself result in control battles over what may simply be a consequence of powerful neuroendocrine changes. The ubiquitous control struggles involving the mother, previously seen as causal, are proposed by the AFFH to arise from conflicts between the worried and uncomprehending family and the anorectic's powerful biological drives to avoid food and to exercise. Learning this, parents who were previously defensive or blaming can be more cooperative with treatment and more compassionate toward their child.

The AFFH offers a powerful cognitive intervention as a component of standard family, cognitive behavioral, and psychoeducational treatment. Of course, patients who do not believe in evolution or who believe that their AN is due to parental failings do not find the AFFH helpful. However, for many, especially the first-time AN sufferer, I have found that these biological explanations make sense and help them to resist their urges to exercise and avoid food. Patients' new belief that these powerful compulsions saved their ancestors' lives in the Pleistocene lead to awareness that they must increase weight beyond the range that triggers the symptoms. A plausible explanation for their body image distortion, fear of food, and restlessness helps them to believe their scales, doctor, therapist, and dietician instead of their internal urges. There are as yet no empirical studies on the efficacy of adding this explanation to the psychoeducational component of cognitive–behavioral therapy, but it is hoped that that this article will stimulate such research.

If the medical condition does not require immediate hospitalization, outpatient therapy, in collaboration with a nutritionist and physician, can be tried (Becker, Grinspoon, Klibanski, & Herzog, 1999). Although the symptoms may come from physiological rather than psychological causes, psychotherapy is an essential part of treatment. The nutritionist creates a refeeding plan and works closely with the therapist and physician to monitor progress. Initially, exercise is greatly restricted until a patient reaches a healthy weight that is determined in consultation with the treatment team. A timeline is set for weight restoration; if the patient is unable to regain weight on time, hospitalization is urged. When patients are very thin, frequent meetings for brief periods are more helpful than the customary 1 hr a week for psychotherapy. Deeper psychotherapeutic work is impractical when people are in the grip of AN, as Bruch (1973) noted, but patients do need considerable support and monitoring. Initially therapeutic work consists of relationship building, offering cognitive–behavioral strategies for complying with the dietician's recommendations, and coordination between the physician, parents, nutritionist, and teachers or coaches.

Although body image distortion generally decreases as weight normalizes, weight regain is threatening. I believe that one of the reasons people with AN are so frightened by the possibility of becoming obese is that, like Keys et al.'s (1950) subjects during refeeding, recovering anorectics go through periods when they are hungry all of the time, even after large meals. Patients often become terrified that their hunger has no bounds. Long-term anorectics are particularly vulnerable during this stage because they have discovered that, if they lose weight, they will eventually "regain control" of (in other words, lose) their appetite. It is important to forecast this phase for first-time recovering anorectics and to assure patients that, like Keys' volunteers, they will recover their normal satiety responses. Patients must understand that recovery of normal satiety will happen more quickly if they do not subject their bodies to famine conditions periodically. In other words, they must bring themselves to eat what the dietician has prescribed every day or, if they cannot, they must go into a hospital to get the support they need to do so. For patients who have needed the assistance of inpatient treatment for weight restoration, a discussion of the AFFH has been instrumental in convincing them to enter inpatient treatment.

For a patient in the grip of the anorectic adaptations, interpretation of his or her feelings is particularly delicate. Psychotherapeutic work often focuses on helping patients to get in touch with affect. But if anorectics listen to their feelings, they feel urged to avoid food and to exercise. One wants the patients with AN to be able to ignore feelings that come from adaptations to famine, but to attend, in the words of the poet Marge Piercy (1975), to their "own true hunger and pleasure and rage" (p. 28). Bruch finessed this problem by insisting that patients weigh at least 90 pounds before she would see them in psychodynamic therapy. Once patients' are less—biologically—in the grip of the AN adaptations, successful therapists have found that teaching patients to attend to their emotions has been healing for perfectionists who fell prey to others' standards. People with AN have typically ignored their feelings; that may be how some were initially able to lose so much weight.

Psychotherapy can also focus fruitfully on the cultural pressures, family situation, personality factors, or traumatic life events that first led the anorectic to disregard the body's hunger signals and drive weight so low. Patients need to learn to moderate their perfectionism, become more aware of affective states, and find other avenues for establishing individuality and self-esteem. Raising consciousness about oppressive sociopolitical systems, teaching resistance to pathogenic cultural norms, and fostering personal empowerment are often salutogenic. Although researchers have not yet found pharmacological interventions to counteract or normalize the various neuroendocrine changes of AN (Becker et al., 1999), it is hoped that this review may help to sharpen the focus of future research.

Although the lives of people with AN are wretched (the 10% mortality rate is due in no small part to suicide), for 50% AN becomes chronic (American Psychiatric Association, 2000). Biological, social, and psychological factors contribute to AN's high relapse rates. Neurophysiologically, short-term recovered anorectics appear ready to migrate again. The neuroendocrine changes that facilitate hyperactivity and food refusal return to normal only slowly after weight is regained (Støving et al., 2001). What this means is that the desire to exercise and restrict foods, especially fats, will generally remain high, and it is easy for the weight-restored patient to lose weight once she or he is out of an inpatient setting.

Socially, the AN symptoms have isolated sufferers from an uncomprehending world. As their interests narrow to how much to exercise and eat, friends and family become alienated, bored, and ultimately exhausted. When in recovery, people with AN need considerable social support, but they may have worn out friends and family, who naturally assume that their resistance to eating is, on some level, willful. Furthermore, the culture hasn't changed; weight standards are still unhealthy. Many patients comment ruefully on the envious praise they received from acquaintances when

they were model thin. Now, the former anorectic is again too fat for the cultural ideal, leading some patients to resume restricting food, again triggering the biological forces that tell the body it is experiencing famine.

In addition, the disorder is seductive. Some patients relapse for the same reason that people go back to smoking cigarettes or drinking alcohol—for the high. When an anorectic sometimes feels like superwoman and looks like a super model, a more cogent question may be how any are able to give up the disorder. It is also narcissistically appealing to see oneself as uniquely able to ignore appetite, hunger, and pain. Without an alternative explanation to understand this sense of virtue about abstinence, appetite is dreaded as a sign of being ordinary. An AFFH interpretation can demystify the heady experience of superhuman self-control, but the dangerous siren song of anorexia will continue to entice some.

Recognition of Evolutionary Explanations

Earlier attempts to apply evolutionary explanations of AN based on animal models have not been well received. Epling and Pierce's (1984) suggestion that starving rats' hyperactivity may be relevant for understanding AN was called silly by one researcher (Mrosovsky, 1984). Another wrote, "We cannot expect an explanation for the hyperactivity of AN patients from the study of semistarved rodents living with running wheels" (Pirke et al., 1993, p. 292). Contributors to the clinical literature on AN simply ignored their work.

Eating disorder specialists have overlooked the adaptive significance of these symptoms because current theories were developed when the pendulum in psychology and psychiatry had swung away from evolutionary explanations. For example, as late as the 1960s researchers had difficulty publishing findings showing that rats have innate abilities to learn easily to associate taste with subsequent nausea because reviewers assumed the rat mind, as well as the human mind, was essentially a tabula rasa at birth (Garcia & Koelling, 1966; Seligman & Hager, 1972). Twentieth century clinicians were not trained to look for evolutionary adaptive processes.

Furthermore, it has been difficult to see a connection between the behavior of starved animals and dieting girls because humans tend to explain behaviors and beliefs in psychological terms. Today's anorectics often attribute their self-starvation to a desire to be thin, whereas medieval women with holy anorexia explained the same behaviors with reference to piety. Humans try to make sense of their behavior post hoc, even when it emanates from subcortical structures, sometimes confabulating a plausible story to explain what they do to themselves and others (Gazzaniga, 1978).

Conclusion

Anorexia nervosa has been seen as a psychogenic illness that in the western thin-obsessed culture develops in biologically vulnerable individuals. Since Hilde Bruch's (1973, 1978, 1988) influential work, parental and patient psychopathology are assumed to cause the odd symptoms of food refusal, hyperactivity, and denial of illness. However, empirical studies have not been able to confirm the putative relationship between AN and serious psychopathology in patients or their families. Nor can traditional theories account for anorexia-like syndromes in other animals, men, and other historical times; the fact that typical neuroendocrine changes in AN support the symptoms of food refusal and hyperactivity; or that genetic studies find that AN is highly heritable.

This article proposes a new theory of AN that accounts for its unusual symptoms. In contrast to conventional psychological theories that posit symbolic or psychological meaning, here the symptoms of AN are proposed to be biological responses to low body weight. AN is likely to result from a complex of adaptations selected in the prehistoric past when migration was an effective solution to local famine conditions (i.e., when humans lived as sparsely distributed, nomadic foragers). Like obesity, AN was useful then but is deadly now. Although no longer adaptive, some individuals' bodies respond to very low body weight (caused by dieting or any other reason) as though they must migrate from famine conditions. Neuroendocrine changes turn down eating and turn on mobility, whereas cortical mechanisms appear to create body image distortion, denial of starvation, and restrained eating.

However, the answer to the question "Why do some individuals develop AN?" can, indeed must, be answered at more than one level of analysis. The various hypotheses for AN—psychological, cultural, biomedical, and evolutionary—can contribute complementary explanations and interventions; these are summarized in Table 3.

Understanding the adaptive role that AN may have played in the ancestral environment can explain the unusual form and strength of its symptoms; knowledge of its neurohormonal alterations is important in explicating the mechanisms of anorexia and suggesting pharmacological interventions; awareness of unrealistic cultural weight standards explains why most women and girls feel pressured to diet and empowers resistance, whereas knowledge of a patient's psychological issues is essential to understanding why this particular individual developed AN, helping the patient recover his or her weight, and working through individual or family issues that contributed to or arose from the illness.

AN is a devastating disorder. It typically takes over the life of a girl in adolescence and controls her thoughts, fears, and actions. So far, psychology has had too little to offer. Psychologists have blamed the victim for refusing to maintain her weight, her parents for being controlling and critical, or her defective genes. Many people do not seek treatment early because of the shame associated with having this disorder (Becker et al., 1999), and defensiveness leads some parents to ignore a child's eating disorder. Understanding the symptoms as archaic biological adaptations can free psychotherapists, physicians, and parents from indiscriminate or unjustified blaming, and it also suggests interventions that may aid AN sufferers in recovery.

The mind has been fitted through natural selection with mental structures operating below conscious awareness that process information about the body and the world and alter physiological, behavioral, and cognitive states in response to that information. Central nervous system compartmentalization leads to a lack of conscious awareness of the agendas of different mental structures (Pinker, 1997).

Psychologists are becoming more aware of the complexity, power, and diversity of these evolved adaptive mechanisms. In a recent review of unconscious mental processes in *American Psychologist* (1999), Park concluded,

Table 3
Contributions of Major Theories to Understanding Aspects of Anorexia Nervosa

Aspect	AFFH	Psychopathology	Social pathology	Biomedical pathology
Food refusal	Ignoring food facilitated migration from local famine	Perfectionistic attempts to attain model thinness; trauma or psychopathology can lead to food restriction	Weight standards for women and girls are too low, leading to extreme dieting	High levels of satiety and low levels of appetite signalers
Hyperactivity	Selected in the past because it provided energy for migration	Perfectionists drive themselves to excessive exercise	Exercise serves losing more weight	High levels of cortisol and serotonin
Denial of starvation	Selected in the past because it provided courage for migration			
Sex ratio	In monogamous species, females compete for good-quality males by enhancing attractiveness; females may have migrated more safely in evolutionary past	Ultrathin weight standards only for women and girls leads to dieting	In patriarchies women and girls are subjected to oppressive beauty standards	
Vulnerability of perfectionists		Perfectionists rigidly persist in dieting to very low weights	These individuals are more vulnerable to social norms	High levels of serotonin
Occurrence in other animals	Selected in the past to facilitate migration from local famine			Anorexic rats show same pattern of neuroendocrine changes
Usefulness for treatment	Provides way to understand symptoms and rationale to ignore bodily urges	Provides individually focused psychological interventions	Provides personal empowerment to resist social pressure to diet	Suggests pharmacological treatments

Note. AFFH = adapted to flee famine hypothesis.

> There are mental activities of which we are unaware and environmental cues to which we are not consciously attending that have a profound effect on our behavior and that help explain the complex puzzle of human motivation and actions that are seemingly inexplicable, even to the individual performing the actions. (p. 461)

The starved laboratory rat running 20 km a day while ignoring its food behaves as though neurological mechanisms have switched on forward locomotion and switched off appetite when its body weight dropped below a critical level. In the wild such responses might be adaptive, but in the context of a laboratory cage, high school, or work place these behaviors appear quite mad.

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