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Competing Paradigms for Anorexia Nervosa

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The contributors to the April 2007 *American Psychologist* special issue on eating disorders are to be commended for acknowledging lack of progress in understanding, classifying, and treating anorexia nervosa (AN). They highlighted the acute need to refine diagnosis (Wonderlich, Joiner, Keel, Williamson, & Crosby, 2007), understand comprehensive causal mechanisms to tune treatments and transcend "hodgepodge diagnoses" (Striegel-Moore & Bulik, 2007, pp. 181–182), study functional neural circuits and link behavior with "genomic, cellular, and systems data" (Chavez & Insel, 2007, p. 164), and develop effective treatments (Wilson, Grilo, & Vitousek, 2007, p. 201). Specifically, Chavez and Insel (2007) wrote that "present-day treatments are significantly limited" and that identifying underlying pathophysiology "will be critical for developing more effective treatments and preventive strategies" (p. 160). This state of the field could suggest that a new paradigm is needed, but new paradigms are often resisted by the established scientific community (Kuhn, 1962), of which the contributors to the special issue are internationally recognized leaders.

People with AN restrict food, see fat on their emaciated bodies, and often exercise energetically although they are at least 15% below normal weight. Any dieter can attest that these are remarkable abilities. In fact, energy regulation researchers believe it is normally impossible for individuals to maintain weight below normal for long. However, the established point of view assumes that AN is caused by "successful pursuit of thinness through dietary restriction and other measures" (Wilson et al., 2007, p. 199), attributing cause to putative faulty cognitions or disturbed relationships that lead someone to have excessive weight concerns and restrict food and exercise excessively.

This view overlooks considerable evidence that the cognitive and behavioral symptoms are not strictly volitional and are themselves *caused* by weight loss in the genetically vulnerable, rather than the other way around (Guisinger, 2003; Hebebrand, Casper, Treasure, & Schweiger, 2004), as well as evidence that symptoms are biologically "engineered." In fact, Keel and Klump (2003) found that the odd beliefs and behaviors characteristic of AN are remarkably similar across age, sex, and historical era. Neuroimaging finds that body image distortion results from specific changes: When underweight anorexics look at their own bodies, the brain region responsible for body schema is activated, but only in response to the patient's own image and only when underweight (Wagner, Ruf, Braus, & Schmidt, 2003). Anorexics receive strong signals both to eat and to fast because normal starvation-induced increases in orexigenic (hunger) signals are compromised by simultaneous excesses in anorexigenic signaling (Inui, 2001). AN is heritable, with some symptoms linked to genetic polymorphisms of appetite regulators. The ability to develop AN is activated at puberty in some girls by genetic changes perhaps involving β-estrogen receptors (Klump & Gobrogge, 2005). Yet the apparent functional coherence of specific psychobiological evidence is often ignored in "hodgepodge" diagnosis and explanation.

A candidate for a new paradigm for AN and perhaps the other eating disorders sees them as evolutionary adaptations to starvation gone awry. The adapted-to-flee-famine hypothesis (AFFH) proposes that when hunter-gatherers' weight loss was due to local famine, normal responses—hunger, lethargy, and despair—would have interfered with crucial efforts to migrate. Then, individuals with abilities to ignore hunger, move energetically, and deny starvation could flee local depletion (Guisinger, 2003), resulting in selection for the abilities (or symptoms) that now comprise the AN syndrome.

Researchers may dismiss adaptationist arguments as untestable, but reasonable conclusions can be reached by weighing evidence and judging probabilities. Deciding which competing explanation provides the best explanatory fit and the most coherence is called inference to the best explanation (Lipton, 2004). If one applies this method, an evolutionary explanation for AN symptoms has more evidentiary support than other explanations (Guisinger, 2003). The AFFH accounts for the broad array of phenomena and is supported by evidence from multiple independent sources. Psychosocial explanations explain little biological data and are undermined by observations that symptoms are similar across era, sex, and culture (Keel & Klump, 2003) as well as by twin studies showing little influence of family environment (Striegel-Moore & Bulik, 2007).

Why has the evolutionary perspective been overlooked? In 2003, I wrote,

> 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 to easily associate taste with subsequent nausea because reviewers assumed the rat mind, as well as the human mind, was essentially a *tabula rasa* at birth. . . . 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, while medieval women with holy anorexia explained the same behaviors with reference to piety. Humans try to

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make sense of their behavior *post hoc*, even when it emanates from sub-cortical structures. (Guisinger, 2003, p. 757)

Kuhn (1962) wrote that normal science is "a strenuous and devoted attempt to force nature into the conceptual boxes supplied by professional education" (p. 5). In the case of anorexia in this special issue, commitment to psychosocial causation led to some odd omissions and errors. Wonderlich et al. (2007) failed to mention the many critiques of the claim made in the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2000) that hyperactivity is secondary to desire for weight loss (Hebebrand et al., 2004), which is important because it obscures the applicability of several animal models (Epling & Pierce, 1988).

Wilson et al. (2007) and Chavez and Insel (2007) downplayed or gave an odd twist to a psychotherapy outcome study that found that the control group's nutritional counseling and supportive management fared better than specialized cognitive–behavioral therapy for AN and interpersonal therapy for AN (McIntosh et al., 2005). These and other results (a) suggest that treatment succeeds (when it does) *despite* popular theories of AN, (b) emphasize the importance of communal or familial refeeding, and (c) indicate that assuming symptoms are caused by individual or interpersonal psychopathology is countertherapeutic.

AN-biopsychosocial therapy (A-BPST) combines traditional psychotherapy with a theoretically intuitive explanation for the symptoms—for example, that controlling hunger, denying starvation, and moving made sense in the context of a desperate journey. Preliminary clinical research suggests that A-BPST can improve treatment outcome (Guisinger & Schuldberg, 2007).

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Why Diets Fail—Expert Diet Advice as a Cause of Diet Failure

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In "Medicare's Search for Effective Obesity Treatments: Diets Are Not the Answer," Mann et al. (April 2007) offered the following broad, sweeping conclusion: "The benefits of dieting are simply too small and the potential harms of dieting are too large for it to be recommended as a safe and effective treatment for obesity" (p. 230). This statement is provocative and unproven in the text.

According to the laws of thermodynamics, which appear to apply everywhere in the known universe, anytime one consumes fewer Calories than one burns, there will be weight loss (Brooks, Fahey, & Baldwin, 2005, p. 22), despite the claims of some diet gurus and MDs (Cruise, 2005, p. 55; Katz, 2005). Any and every diet must work so long as a person voluntarily sustains a condition of fewer Calories in than out for a sufficient duration. Yet the article's conclusion denies this simple universal truth. This is a failing.

A major key to its failing is found in Mann et al.'s (2007) Footnote 3: "The term *dieting* has been used to refer to a wide range of behaviors, but we use it solely to refer to the specific behavior of severely restricting one's calorie intake in order to lose weight" (p. 221). Rather than explore what they term severe Calorie restriction and its ramifications, Mann et al. (2007) glossed over the matter. Perhaps an exploration of this issue should be undertaken before arriving at a media-sensational conclusion that is potentially of great harm, that is, dissuasive of Calorie limitation for weight loss.

Virtually all diets are based on the "fact" that a "safe" 1–2 pounds (0.45–0.91 kg) per week weight loss can be achieved by a daily intake reduction of 500–1,000 Calories.[1] This is false. It is impossible to lose only 1–2 pounds per week following this advice (McArdle, Katch, & Katch, 2001, pp. 840, 849).

Consequent to this approach are recommendations by acknowledged diet experts for energy intakes in a very interesting range of Calories. Table 1 shows some diet experts' schemes (in bold) and their Calorie intake recommendations compared with intakes in instances of attempted/successful murder and intentional infliction of suffering by starvation: None of the intake advice listed includes the recommended approximately 300 Calories per day to be

[1] For examples, see Berning (2005), President's Council on Physical Fitness and Sports (2004), Medical Encyclopedia: Losing Weight (n.d.), and U.S. Food and Drug Administration, Federal Trade Commission, and National Association of Attorneys General (1992).