Health at Every Size

A Compassionate, Effective Approach for Helping Individuals With Weight-Related Concerns—Part I

by Jon Robison, PhD, MS, Kelly Putnam, MA, and Laura McKibbin, LISW

Editor's Note—Occupational health nurses are often confronted with varying opinions about health-related topics. This article was chosen for publication because the authors discuss overweight and obesity from a perspective differing from the mainstream. Is their perspective one nurses should integrate into professional practice, or do the ideas need further investigation? One way to investigate this new perspective is to read the article (Part I and Part II), review the references, discuss the ideas with your colleagues, and then send your comments to the Journal. We will publish comments to stimulate discussion about best practices in addressing overweight and obesity for employees and employers. I look forward to hearing from you.—J. W.

It would be difficult to overstate the urgency that U.S. government and health officials have placed on the dangers posed by obesity. The Surgeon General labeled obesity "the terror within" and a terror more dangerous than "weapons of mass destruction" and called for no less than a "cultural transformation" to combat and win the "war" against it (Severson, 2003; "Surgeon General," 2003). A group of researchers warned that "the tsunami of childhood obesity has not yet hit shore" and that it was only a matter of time before heart attack and kidney failure became "a relatively common condition of young adulthood" ("Explosion of Child Obesity," 2005). In a recent article in the *New England Journal of Medicine*, they went on to suggest that the millennia-long trend of increasing human life expectancy would soon be reversed by the growing worldwide increase in body mass (Olshansky et al., 2005).

The foundation of this concern and the justification for the so-called "war on obesity" rest on the following three central claims:

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- Overweight and obesity are major causes of premature death.
- Excess fat is pathological and a direct cause of disease.
- Weight loss is beneficial to health.

With the increasing numbers of individuals being diagnosed as overweight and obese and with so much emphasis being put on the contribution of weight to health and disease, it is critical for all health professionals to examine the validity of these claims. The purpose of this two-part series is to summarize the scientific evidence behind these claims and to recommend effective approaches occupational health nurses can use to help individuals who come to them with weight-related concerns.

The premise of Part I is that all three of the claims central to the war on obesity are poorly supported and in some cases directly contradicted by current scientific evidence. Furthermore, it is suggested that current approaches are more likely to do harm than good. Part II, which will appear in the next issue, will present an alternative, practical, effective approach for helping individuals with weight-related concerns and will explore implications for occupational health nurses. A case study of a yearlong workplace program based on such an approach will be presented.

Much of the material presented in these articles contradicts traditionally held beliefs about the relationship between weight and health. For a variety of reasons, the substantial body of research that has accrued during the past few decades supporting these contentions has often been overlooked or ignored. Therefore, whenever possible, multiple references have been included to enable practitioners to read the supportive, pertinent research if they so desire.

HISTORICAL CONTEXT: AN AMERICAN OBSESSION

The current American obsession with thinness is, from a historical perspective, a cultural aberration.

Obesity Treatments Prescribed "In The Name of Health"

Year	*Treatment*
1893	Thyroid extract
1920	Laxatives
1933	Dinitrophenol (pesticide)
1937	Amphetamine
1940	Atropine and digitalis (heart drugs)
1957	Human chorionic gonadotropin (from the urine of pregnant women)
1964	Total fasting
1969	Intestinal bypass
1974	Jaw wiring
1977	Gastric bypass
1985	Gastric balloon
1990s	Fen-Phen, Redux, Meridia, and Xenical*

**Fen-Phen and Redux: Wyeth, Madison, NJ; Meridia: Abbott Laboratories, Abbott Park, IL; Xenical: Hoffmann-La Roche, Nutley, NJ.*

Throughout history, the vast majority of societies have regarded fatness as a sign of success, health, and beauty (Brown, 1993). This has been particularly true for women, for whom soft, rounded hips, thighs, and bellies have almost universally been considered ideal. Less than 100 years ago in America, excess body fat was described as a "snug balance in the body bank and a comfortable reserve in the case of emergencies" (Hutchinson, 1926, p. 60). In 1908, an article in *Harper's Bazaar* advised readers on "how to get plump," saying that "fat is force and stored up fat is stored up force" (p. 757). Fashion models were advised to be "far from thin, with no suggestion of hollows in the face or the collarbones, for the camera seems to accentuate such defects" (Fraser, 1997, p. 22). Physicians regularly encouraged their patients to gain weight, believing that "a large number of fat cells was absolutely necessary to achieve a balanced personality" (Banner, 1983, p. 113). Clearly, times have changed.

Currently in the United States, fatness as chronic disease and weight reduction as cure stand ubiquitously as accepted health care dogma. Anthropologists point out that the first strong cultural emphasis on weight loss in this country appeared around the turn of the century and coincided with women securing the right to vote and demanding a more visible and active role in shaping society (Seid, 1989). As women's power and status improved, the dictates of fashion began to change. Health care recommendations for women to lose weight followed. For the next 100 years, the health sciences promoted a wide variety of potentially dangerous and sometimes lethal diets, drugs, and surgeries to help individuals reduce their weight "in the name of health." The Sidebar contains a partial chronological list of these regularly prescribed treatments (Ernsberger & Haskew, 1987).

Women have been and continue to be the majority of those participating in and suffering from these "cures," despite the well-known fact that women's fat confers only a fraction of the health risk of men's fat and may have significant health benefits (Schapira et al., 1991; Schapira, Kumar, Lyman, & Cox, 1990; Terry, Stefanick, Haskell, & Wood, 1991). The legacy continues, as young girls and women regularly divert significant portions of their physical, emotional, and financial resources to the pursuit of ideals of body shape and size that are, for most, neither achievable nor healthy (National Institutes of Health, 1992).

Beginning in the 1960s, a preference for slenderness also took hold in other Western, industrialized nations. However, due to a unique confluence of social, economic, and political developments favoring the desire for thinness, "no other culture suffers from the same wild anxieties about weight, dieting and exercise as we do" (Fraser, 1997, p. 20). The pressure to be thin is driven by the diet, fashion, cosmetics, fitness, insurance, and pharmaceutical industries, which reap tremendous financial rewards by promoting unattainable expectations, especially for women. In addition, many obesity researchers have economic links to this so-called diet–pharmaceutical–industrial complex, creating powerful incentives for maintaining the status quo. Most members of the National Institutes of Health National Task Force on the Prevention and Treatment of Obesity serve as consultants to both commercial weight-loss programs and pharmaceutical companies involved in the development of weight-loss medications (National Institutes of Health, 1996). As a result, "Obesity research is primarily funded by companies that profit from promoting short-term weight-loss methods" (Fraser, 1997, p. 232), contributing, perhaps, to questionable objectivity in the reporting of research.

BODY MASS INDEX

The most widely used standard for determining what is and is not an acceptable, "healthy" weight is the body mass index or BMI (calculated as weight in kilograms divided by height in meters squared). A BMI of 18.5 to 24.9 is considered to be "healthy," while a BMI of 25 to 29.9 is considered to be "overweight" and a BMI of 30 or more is considered to be "obese." Although support for its use is based on the supposed relationship between body fat and disease, BMI is not an accurate predictor of total body fat in individuals. Furthermore, it does not take into consideration any differences in gender, race, age, or ethnicity. BMI also does not distinguish between fat and muscle tissue, resulting in absurd conclusions about who is and is not overweight and obese (Table).

The evidence questioning the validity of BMI as a health indicator is perhaps most damaging in relation to cardiovascular disease, for which the impact of weight on health has traditionally been considered to be the greatest and most straightforward. In fact, a recent, large meta-analysis of the literature published in the journal *Lancet* found BMI to be a poor predictor of cardiovascular disease or crude mortality (Romero-Corral et al., 2006). As Franzosi (2006) concluded in an accompanying editorial, "BMI can definitely be left aside as a clinical and epidemiological measure of cardiovascular risk for both primary and secondary prevention" (p. 624).

In addition, the use of BMI as a measurement of health risk has led to significant confusion concerning the extent of the so-called "obesity epidemic." The term epidemic implicates obesity as an exponential, rapidly spreading phenomenon. This is an inaccurate evaluation of the available data, which show a modest increase in the average weight of the population, with most individuals weighing between 6 and 11 pounds more than they did 10 years ago (Campos, Saguy, Ernsberger, Oliver, & Gaesser, 2005). Whereas the heaviest individuals have gained a significant amount of weight, the reality is that the obesity epidemic is largely the result of tens of millions of individuals with BMIs of 23 to 25 and tens of millions of individuals with BMIs in the upper 20s having gained a modest amount of weight and moved across the relatively arbitrary BMI cutoff points. This has created a distorted picture of reality, as biologist Jeffrey Friedman (Campos et al., 2005) explains:

> Imagine that the average IQ was 100 and that five percent of the population had an IQ of 140 and were considered to be geniuses. Now let's say that education improves and the average IQ increases to 107 and 10% of the population has an IQ greater than 140. You could present the data in two ways. You could say that average IQ is up seven points or you could say that because of improved education the number of geniuses has doubled. The whole obesity debate is equivalent to drawing conclusions about national education programs by saying that the number of geniuses has doubled. (p. 56)

WEIGHT AND MORTALITY

The now more than decade-long hysteria in this country over the supposed health crisis of obesity was originally fueled by reports published in major medical journals in the early 1990s. These articles claimed that obesity was responsible for 300,000 to 400,000 deaths per year, a scourge second only to the devastating effects of cigarette smoking. It is now apparent, however, that this research was seriously flawed, resulting in a significant overstatement of the strength of the relationship between weight and mortality. In fact, epidemiologists from the Centers for Disease Control and Prevention, using the most current, representative data, estimated that an association exists between weight and increased mortality for 25,000 individuals per year (Flegal, Graubard, Williamson, & Gail, 2005). Perhaps even more importantly, they made it clear that the finding of some relationship between weight and increased mortality in these individuals in no way confirms weight as the cause of that increase. As Flegal et al. (2005) state, "The associations are not necessarily causal. . . . Other factors associated with body weight such as physical activity, body composition, visceral adiposity, physical fitness, or dietary intake might be responsible for some or all of the apparent association of weight with mortality" (p. 1866).

Table

Body Mass Index (BMI) Does Not Distinguish Between Fat and Muscle Tissue

Individual	*BMI*	*Weight Status*
George W. Bush	26.3	Overweight
Will Smith	27	Overweight
Yao Ming	27.7	Overweight
George Clooney	29	Overweight
Johnny Depp	29.8	Overweight
Matt LeBlanc	30	Obese
Tom Cruise	31	Obese
Shaquille O'Neil	31.6	Obese
Arnold Schwarzenegger	33	Obese

Perhaps the primary problem in evaluating associations between body weight and mortality has to do with the myriad of confounding factors that are rarely considered, making causal conclusions all but impossible. Most studies fail to control for factors such as fitness, exercise, dietary quality, weight cycling, diet medication use, and economic status. When one or more of these variables is well controlled, the existing although already weak relationship between weight and mortality is often greatly diminished. In both the Framingham Heart Study and a recent National Health and Nutrition Examination Survey study, for example, all excess mortality associated with obesity was accounted for when the impact of weight cycling was considered (Diaz, Mainous, & Everett, 2005; Lissner et al., 1991).

Although these findings contradict the conventional wisdom about the relationship of weight to mortality, they are consistent with a significant body of epidemiological evidence from both national and international sources. In fact, approximately 75% of all body weight–mortality studies published since the 1950s find weight to be irrelevant to health and mortality issues (except perhaps at the extremes of the BMI) (Gaesser, 2002). The lack of support for a positive relationship between weight and mortality remains after confounding factors such as smoking, preexisting illness, and length of follow-up are considered (Ernsberger & Haskew, 1987). An in-depth

meta-analysis of the literature concluded that weight levels currently considered moderately overweight were (Troiano, Frongillo, Sobal, & Levitsky, 1996):

> Not associated with increased all-cause mortality . . . and body weight at or slightly below current recommendations was associated with increased risk of mortality. . . . This quantitative analysis of existing studies revealed increased mortality at moderately low BMI for White men compared to that observed at extreme overweight. . . . Attention to the health risks of underweight is needed, and body weight recommendations for optimum longevity need to be considered in light of these risks. (p. 72)

Although some studies show increased mortality with increasing weight, most do not. Several studies show an inverse relationship with mild to moderate fatness and in some cases even with extreme fatness. Data from the Norway Study, the largest epidemiological study of its kind, indicate that maximum longevity is attained by women considerably overweight by health care standards (BMI = 26 to 28) (Waaler, 1984). As BMIs ascend into the "very obese" range (34 to 36), mortality does begin to rise, but only slowly. Even women considered to be "morbidity obese" (BMI > 44) have a better chance of surviving to age 65 than do women in the leanest group (BMI ⩽ 18). Similarly, in the Pooling Project data, representing results from the Framingham, Albany, Tecumseh, Chicago People's Gas, and Chicago Western Electric studies, the highest mortality for both men and women occurred in the underweight group. Mortality rates fluctuate considerably but appear to be optimal at levels currently considered 25% to 35% "overweight." Mortality rates in the fattest group are only slightly higher than those in the "desirable weight" group (McGee & Gordon, 1976).

Taken together, the data do not support a causal relationship between weight and mortality for most individuals. Although an association exists between mortality and very high (as well as very low) body weight, it is complicated and poorly understood.

FAT AND DISEASE

Despite considerable lip service to the contrary, with a few notable exceptions such as osteoarthritis and some relatively rare forms of cancer, little evidence exists demonstrating that increased fatness causes disease.

A thorough examination of the scientific literature reveals that accepted notions about the relationship between atherosclerotic heart disease and weight are debatable. Both angiographic and autopsy studies show no relationship between fatness and the degree or progression of atherosclerotic buildup in the coronary arteries (Barrett-Conner, 1985; Keys, 1954; Kramer, Matsuda, Mulligan, Aronow, & Proudfit, 1981; Patel, Eggen, & Strong, 1980). Some research suggests fatness may be associated with protection from the disease. The largest angiographic investigation of the relationship between weight and coronary artery atherosclerosis concluded that for the 4,500 middle-aged and elderly men and women studied, every 11-pound increase in body weight was associated with a 10% to 40% lower chance of having the disease (Applegate, Hughes, & Zwagg, 1991). Even regarding individuals weighing, on average, well more than 300 pounds, studies fail to demonstrate an increase in atherosclerosis, seriously calling into question the link between fatness and this disease (Warnes & Roberts, 1984).

The relative unimportance of body weight in atherosclerosis is further supported by intervention studies demonstrating that disease progression can be halted or even reversed with dietary change without significant weight loss (Arntzenius et al., 1985; Blankenhorn, Johnson, Mack, El Zein, & Vailas, 1990). Even when lifestyle modifications result in some weight loss, it is instructive that no correlation exists between the weight lost and the cardiovascular benefits gained from the intervention (Kim et al., 2006).

It is well-known that fatness is associated with an increased prevalence of health problems such as hypertension, blood lipid disorders, and type 2 diabetes that are, themselves, risk factors for cardiovascular disease. However, such a statistically significant relationship between fatness and disease does not "prove" that fatness is the cause of these problems. In fact, numerous studies have shown that these so-called "weight-related" health problems can be treated effectively with lifestyle interventions that do not result in weight loss. For example, blood pressure, cholesterol, and glucose levels may all improve, even though individuals remain markedly obese by traditional health care standards (Barnard, 1991; Barnard, Jung, & Inkeles, 1994; Barnard, Ugianskis, Martin, & Inkeles, 1992; Kraus et al., 2002; Lamarche et al., 1992; Tremblay et al., 1991). This has led to the suggestion that, for many individuals, increased weight may be a relatively benign symptom that is related to, but not necessarily the cause of, insulin insensitivity, diabetes, hypertension, and atherosclerosis (Ernsberger & Haskew, 1986). In fact, improvements in these risk factors have been demonstrated in exercise studies in which the participants actually gained fat during the intervention (Bjorntorp, De Jounge, Sjostrom, & Sullivan, 1970; Lamarche et al., 1992).

FAT AND HEALTH

A significant body of research suggests that fatness is associated with several health benefits. Although there are exceptions, many studies have indicated an inverse relationship between fatness and cancer deaths (Avons, Ducimetiere, & Rakoto, 1983; Garcia-Palmieri, Sorlie, Costas, & Havlik, 1981; Garn, Hawthorne, Pilkington, & Pesick, 1983; Keys et al., 1985; Wallace, Rost, Burmeister, & Pomrehn, 1982). More recent epidemiological studies have yielded mostly neutral results. The 25-year follow-up of the Whitehall study found the rate of cancer deaths to be 0.77%, 0.75%, and 0.79% in normal, overweight, and obese individuals, respectively, as determined by BMI category (Batty et al., 2005). A consistent finding has been an apparent protective effect of obesity on premenopausal breast cancer (Cleary

& Maihle, 1997). Regarding lung cancer, the leading cause of all cancer deaths, "the overall consistency in the inverse association of body mass with lung cancer in reported studies is impressive. It is noteworthy that no study shows a significant contrary trend" (Kabat & Wynder, 1992, p. 773).

Fatness also has been shown to protect against osteoporosis, a major source of disability and death in older women, with thin women being twice as vulnerable to the disease as heavier women (de Laet et al., 2005; Edelstein & Barrett-Connor, 1993; Felson, Zhang, Hannan, & Anderson, 1993).

Additionally, being heavy has been shown to be an advantage in tuberculosis and in respiratory diseases such as chronic obstructive lung disease, emphysema, and bronchitis, even when the effects of smoking are considered (Higgins et al., 1982; Tverdal, 1986; Watson et al., 2006).

Finally, studies also suggest that fatness may be beneficial for older adults (Diehr et al., 1998; Grabowski & Ellis, 2001). One study of more than 7,000 men and women 70 years and older concluded that "obesity may be protective compared to thinness or normal weight in older community-dwelling Americans" (Reynolds, Fredman, Langenberg, & Magaziner, 1999, p. 1412).

WEIGHT LOSS AND HEALTH

The widely accepted contention that weight loss results in increased longevity and improved health is also unsupported by the literature. The main reason for this is that no intervention provides evidence (significant long-term weight loss in sizable cohorts of individuals) needed to support this contention. Without such evidence, the assumption that changing overweight and obese individuals into "normal" weight individuals will improve their health remains an untested hypothesis.

Even in studies that found weight loss to be associated with health benefits, it is informative that generally no dose-response exists. In other words, individuals who lose small amounts of weight receive the same benefit as those who lose larger amounts of weight. Even more striking, recent findings from the National Health Interview Survey indicate that individuals who reported trying to lose weight did not experience a reduction in mortality equal to or greater than individuals who reported being successful at losing weight, suggesting that it is not the weight loss itself that provides the benefits (Gregg, Gerzoff, Thompson, & Williamson, 2004). This conclusion is further supported by an association between weight loss and increased mortality in this study, whereas being obese but having a stable weight was not associated with increased mortality (Diaz et al., 2005).

This last conclusion is not surprising because most epidemiologic studies suggest that weight loss is associated with increased mortality (National Institutes of Health, 1992). This association holds for all-cause mortality and for mortality from heart disease and stroke for both men and women. It emerges across studies with widely varying methodologies and follow-ups and remains unaffected after controlling for the potential effects of preexisting illness and smoking status (Gaesser, 2002).

Due to the tremendous number of potentially confounding factors involved in epidemiological research, the reasons for the finding of a positive association between weight loss and mortality are not clear. Short-term weight loss is associated with reductions in risk factors for cardiovascular disease, including improvements in blood pressure, glycemic control, and lipid and lipoprotein profiles. However, given the likelihood that individuals will regain their lost weight, it has yet to be supported that these short-term health benefits will persist (National Institutes of Health, 1992).

WEIGHT CYCLING

The pattern of repeatedly losing and regaining weight experienced by most dieters may be one of the factors contributing to the higher mortality rates among individuals who have lost weight. Weight cycling has been shown to decrease metabolic rates at rest and during exercise, increase lipoprotein lipase activity (which makes the body more efficient at storing fat), and increase the proportion of fat to lean tissue in the body. With each weight loss–regain cycle, weight is increasingly redistributed from lower body subcutaneous fat—shown to have a protective effect against heart disease, diabetes, cancer, and high cholesterol—to abdominal visceral fat, which does not confer these protective effects (Gaesser, 2002).

In addition, numerous studies point to increased risk for heart disease, hypertension, and diabetes in individuals who are chronically losing and regaining weight (Blair & Paffenbarger, 1994; Blair, Shaten, Brownell, Collins, & Lissner, 1993; Guagnano et al., 2000; Holbrook, Barrett-Connor, & Wingard, 1989; Lissner et al., 1991; Olson et al., 2000). Other studies have suggested links between weight cycling and gall bladder disease, low bone mineral density, and kidney cancer (Fogelholm et al., 1997; Linblad, Wolk, Bergstrom, Persson, & Adami, 1994; Syngal et al., 1999). Uhley et al. (1997) suggested that the mammary gland may be adversely affected by chronic weight cycling to the extent that repeated weight loss attempts might be an important risk factor for breast cancer.

During the past 40 years, much research has been conducted on animal models of obesity including dogs, swine, rats, and mice. When these animals have their food intake restricted until they lose 20% of their weight and then are allowed to regain that weight ("weight cycled"), they develop abdominal obesity, hypertension, blood vessel damage, and heart disease—the same outcomes that have traditionally been blamed on obesity in humans (Ernsberger & Nelson, 1988; Koletsky, Ernsberger, Baskin, & Foley, 1992; Levin, Stoddard-Apter, & Sullivan, 1984; Smith, Smith, Mameesh, Simon, & Johnson, 1964; Wilhelmj, Carnazzo, & McCarthy, 1957). Therefore, it has been suggested that hypertension and other cardiovascular pathologies seen in some fat humans may be the result of losing and gaining weight rather than the weight itself (Ernsberger & Koletsky, 1993).

Psychologically, dieting has been shown to be a negative, shame-based experience (Hirschmann & Munter, 1988). The deprivation of dieting causes an increased

IN SUMMARY

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A Compassionate, Effective Approach for Helping Individuals With Weight-Related Concerns Part I

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1. It would be difficult to overstate the urgency that U.S. government and health officials have placed on the dangers posed by obesity. Given the increasing numbers of individuals being diagnosed as overweight and obese and the emphasis on the contribution of weight to health and disease, it is critical that all health professionals examine the validity of the claims being made.

2. A thorough examination of the research literature does not support the claims that overweight and obesity are major causes of premature mortality, excess fat is pathological and a direct cause of disease, and weight loss is practical and beneficial to health for most individuals.

3. The literature also does not provide any significant evidence-based support for current approaches to weight management. In fact, it suggests that these approaches may likely be doing more harm than good.

preoccupation with food, overeating and bingeing in response to the dietary restrictions, and increased body dissatisfaction with each evolution of the weight loss–regain cycle. Thus, what most individuals perceive as the "solution" to weight problems is likely to be one of the main causes. Dieting behavior is the best predictor of bulimia. Studies conducted during the past 20 years have demonstrated that the more individuals are taught to cognitively restrain their eating through behavior modification techniques, the more subsequent bingeing behavior and weight fluctuations they experience (Polivy & Herman, 1987). Teaching individuals to diet sets them up for overeating in response to stressful situations, whereas nondieters naturally reduce food intake when experiencing stress (Polivy, 1996). In a recent study involving children, researchers found that dieting was not only unsuccessful but also counterproductive. Children who dieted gained more, not less, weight than those who did not (Field et al., 2003).

SUMMARIZING THE EVIDENCE

A substantial body of research exists contradicting many of the commonly held notions about the relationship between weight and health. Given the extremely complex and often contradictory nature of the available literature in this area, it has been suggested that (Gaesser, 2002):

> Definitive proof of any given hypothesis about the-weight-health correlation is almost impossible at the present time . . . (and that) . . . in America today the real risks to health and longevity are more likely to come from dieting than from stable weights that are above those recommended by the height and weight tables. (p. 161)

In this context, a balanced, summary review of all of the available scientific literature leads to the following conclusions:

- Weight is mostly irrelevant to health and mortality issues except at extreme levels of thinness and fatness.
- Little, if any, relationship exists between fatness and the degree of atherosclerotic buildup in the arteries.
- Fatness is not a cause of hypertension, lipid disorders, or type 2 diabetes. These conditions improve significantly with lifestyle changes and little or no weight loss and in some cases even with increased fatness.
- Fatness is associated with health benefits including decreased mortality from some cancers, protection from osteoporosis, and improved prognosis with respiratory diseases such as bronchitis, tuberculosis, and chronic obstructive pulmonary disease.
- Weight loss is often associated with increased all-cause mortality, and increased mortality from heart disease and stroke.
- Weight cycling is associated with increased risk for heart disease, hypertension, diabetes, gall bladder disease, low bone density, kidney cancer, and breast cancer.
- No evidence exists that weight loss is an effective intervention for improving long-term health for substantial numbers of individuals.

Regarding the last point, the failure of traditional weight management approaches is as striking as their almost universal appeal. Summarizing the evidence of the effectiveness of these approaches in 1958, pioneer obesity researcher Albert Stunkard stated, "Most obese persons will not stay in treatment for obesity. Of those who stay in treatment, most will not lose weight, and of those who do lose weight, most will regain it" (p. 85).

Participation in these approaches continues to grow despite the fact that little has changed to alter the validity of this conclusion and it remains true that relatively few participants succeed in keeping off weight long-term (Food and Drug Administration, 1992; Miller, 1999). Given all the recent emphasis on "evidence-based" health care, it is disturbing that decades of data demonstrating traditional approaches to weight management do not work and may lead to harmful physiological and psychological consequences have not diminished enthusiasm for their continued use among health professionals.

Part II in this series will explore an effective, evidence-based alternative approach occupational health nurses can use in the workplace to help individuals who are struggling with weight-related concerns.

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