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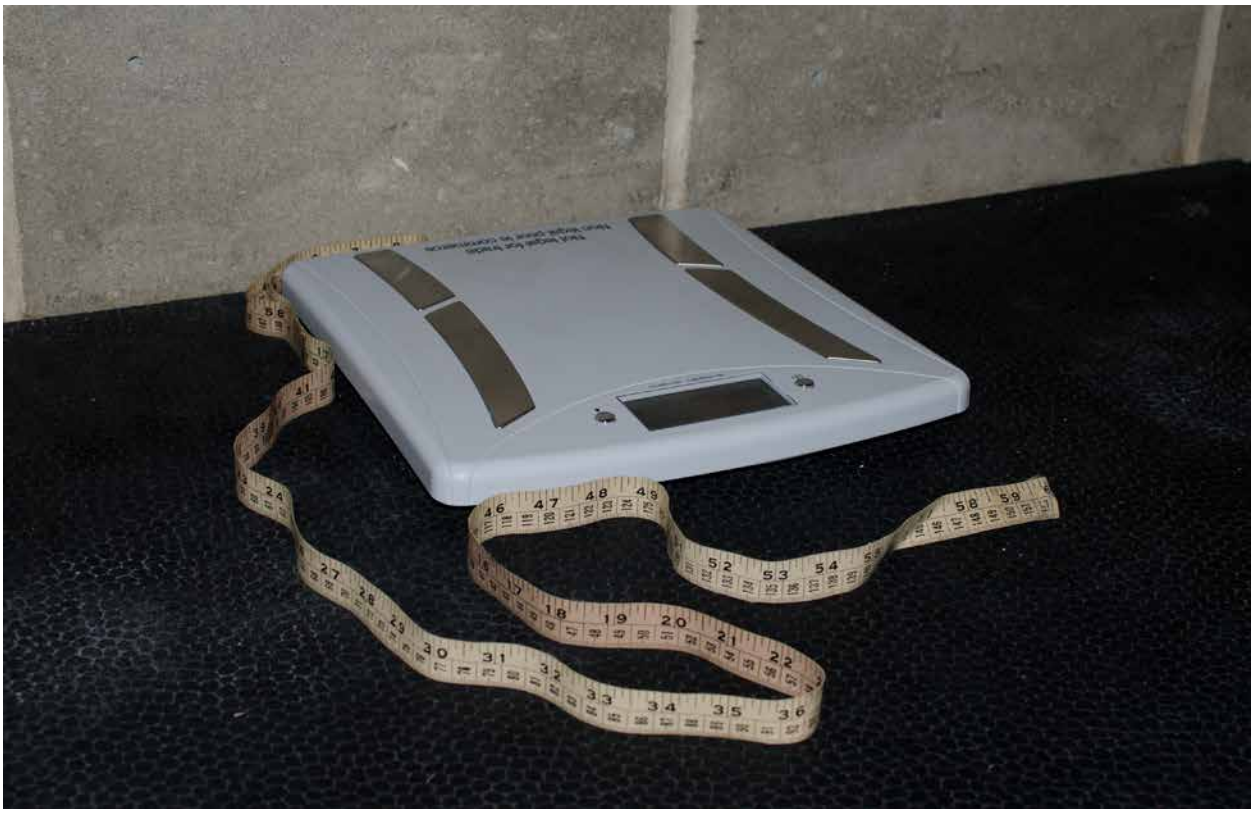
Fat of the Land:
Mismeasures and Missing the Point

Dr. Lon Kilgore finds a numbers game at the heart of the obesity epidemic.

By Dr. Lon Kilgore **October 2012**



All images: Mike Warkentin

An increased frequency of obesity is a cross-societal issue that is receiving a tremendous amount of scientific and media attention. Current estimates of obesity range from 33.8 percent in the U.S., 23.0 percent in the U.K. (with Scotland leading the way with 27.0 percent) to an international low of 3.4 percent in Japan.

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There are myriad proposed explanations for the upward trend in obesity occurring since 1960, with most explanations based on correlation and conjecture rather than causality. Despite the lack of causal data, the correlative data is used as a basis for many nations' public health policy. In the U.K., the National Health Service proposes that 55 percent of the adult population will be obese by 2050 and will represent an annual fiscal health-care burden of 4.2 billion pounds (US$6.63 billion). This is an alarming statistic and merits attention, and there is significant movement among policy makers to respond.

But we need to examine the historical aspects of obesity and its measurement in order to avoid alarmism and potential misdirection of national assets. To more fully understand obesity and our current straits, we need to consider whether the epidemic of obesity is authentic or an artifact of changing social values and constantly changing definitions.

The scale does not tell the whole story when it comes to assessing an individual's overall health.

Despite the flaws, height and weight tables remain in use in the assessment of obesity nearly a century after they were first developed.

Defining "Fat"

Obesity has not been considered truly unhealthy for the majority of human history. Rather, it has frequently been a valued survival trait or a symbol of social and economic status. It was not until the early 1900s that obesity began to receive significant attention via its emerging correlation to heart health and cardiac demise. At that point in history, obesity was assessed via the Metropolitan Height and Weight Charts. It must be understood that these charts were designed to be favorable to insurance-company profits. Obesity-related insurance risk, or the transition from "ideal weight" to "overweight," was set intentionally low in order to expand the population of citizens who would need to pay the higher insurance rates associated with being overweight. There are a plethora of other flaws in these tables that have been identified over the years, but despite the flaws, height and weight tables remain in use in the assessment of obesity nearly a century after they were first developed.

A common definition used for obesity is having 20 percent extra body weight. The "extra" is calculated as a body weight 20 percent greater than the ideal presented by height and weight tables. This definition fails to account for variation in lean body mass, higher amounts of which mitigate against disease. However, there are many papers in print that suggest that there is no difference in death rate from being heavy from fat mass or lean body mass. No absolute evidence can be found to state the specific composition of extra body weight associated with elevated risk.

More recently, indirect assessment of subcutaneous fat stores has been proposed as the most effective and accurate means of field measurement of obesity. Generally implemented as a set of caliper-based measures that are plugged into a predictive equation, these estimations do account for variations in lean body mass, but the resulting data can only be used to estimate an arbitrary range of "healthy" weights for an individual. A defined percent body fat above which mortality rate increases was and is not discernable from the existing data.

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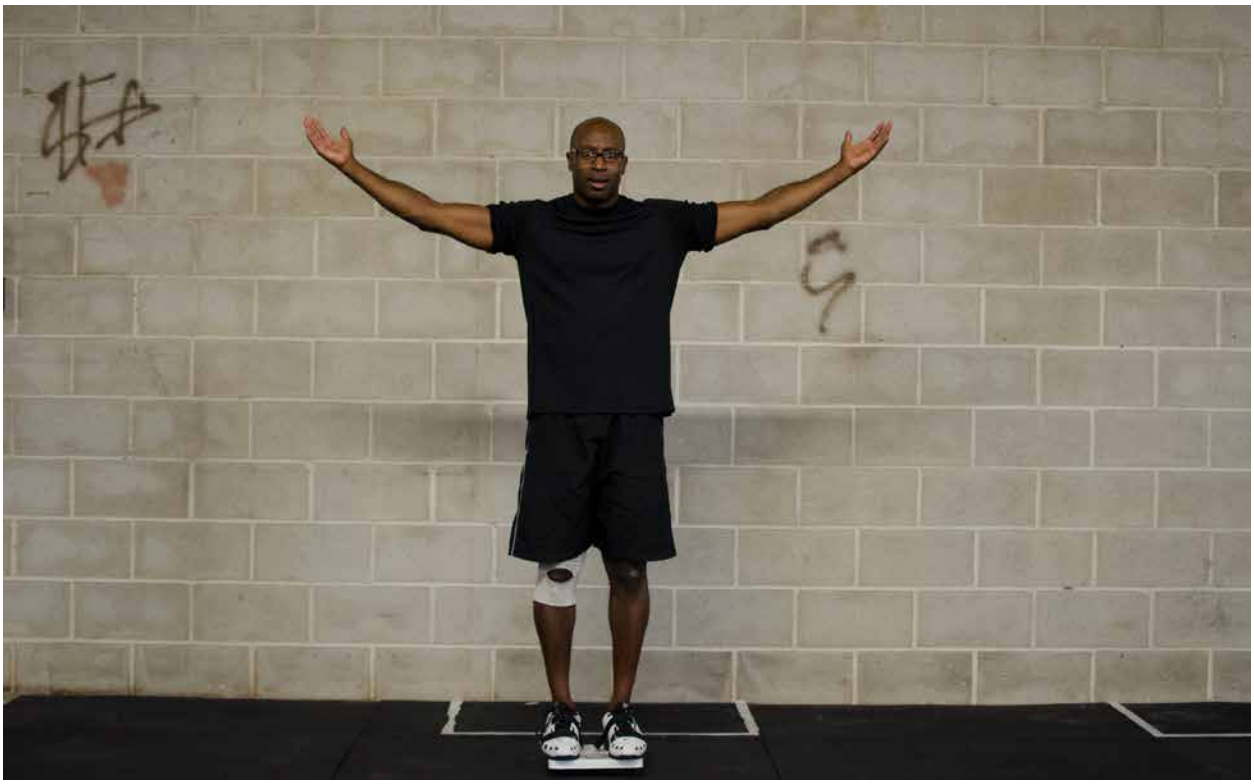
Body Mass Index has grown in popularity as an easy assessment of obesity. It was originally proposed for use as an estimation of body fat by Ancel Keyes in 1972 in large-scale population studies (the formula had originated about a century and a half earlier). By the early 1980s, the Centers for Disease Control and Prevention (CDC) and the World Health Organization (WHO) had adopted BMI as their primary means for assessing overweight status.

It is interesting to note that while these organizations largely abandoned height and weight tables due to their problems, BMI is based upon height and weight measures and essentially reflects the same information and same putative level of predictive accuracy as the tables replaced. Regardless, in 1985 the CDC and WHO adopted an arbitrary BMI value, 27.8, as the level above which obesity is diagnosed. At the time, this seemed appropriate and reasonable as the mean BMI among the U.S. population was 25.0 in 1960, 25.4 in 1970, 25.5 in 1980, and 26.6 in 1990. The 27.8 value represented the upper end of a two-tailed normal distribution. Thus, the rate of obesity in those decades increased mildly.

But between 1990 and 2000 there was a tremendous increase in the reported obesity rate in the U.S.: from 22.1 percent to 29.6 percent of the adult population was estimated to be overweight by BMI. This large change does not represent a commensurate increase body size in the U.S. as the average height and weights were 75.8 kilograms and 169.1 centimeters and 79.9 kilograms and 169.4 centimeters respectively.

It is a result of the CDC and WHO lowering the critical value representing a positive diagnosis of obesity from 27.8 to 25.0 in this decade that induced the change in diagnostic magnitude. Although the 1990 mean BMI was 26.6 and the 2000 mean was 28.0, the change in measurement standard downwards to 25.0 artificially elevated the rate of increased diagnosis of obesity. This alteration in standard makes epidemiologic predictions based on the new data points unreliable. The population did not magically get fat overnight; the rules changed.



What does a scale really tell you about body composition?

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It was also during this decade that the CDC and the ACSM moved obesity from being a secondary risk factor for developing heart disease (another risk-elevating factor had to be present for it to increase risk; it couldn't do it by itself) to the level of primary risk factor (independently increases risk of death). This primary-risk-factor status might have been assigned erroneously because when we simply look at the by-decade mean BMI and the absolute rate of deaths attributed to cardiovascular disease, we see that there is a very strong inverse correlation (r = -0.960) between death rate and BMI. BMI increased 3.4 units over the time period and the rate of death decreased by about 40 percent, so it seems as though obesity does not kill.

Year	Cardiovascular Deaths per Million Population	BMI
1960	55.9	25.0
1970	49.3	25.4
1980	41.2	25.5
1990	32.2	26.6
2000	25.8	28.0
2007	19.1	28.4

Although these numbers superficially seem to be a strong statement that BMI is not a valid diagnostic tool, it must be considered that advances in pharmacological and surgical therapies for cardiovascular diseases are largely responsible for the increased survival rates noted over the past 50 years. This suggests that BMI might still remain a predictor of cardiovascular mortality in some small way, although it is unlikely.

It is germane at this point to discriminate between studies that correlate BMI, body weight and body fat to mortality (actual deaths directly attributable to a specific cause) and those that correlate BMI, body weight and body fat to risk factors of disease (conditions that may be present at the same time as a disease process). The primary intent of public-health initiatives is to prevent unnecessary deaths, and its secondary intent is to minimize the occurrence of disease processes in the public. As it's apparent that BMI does not predict cardiovascular mortality, it may still possess some value in assessing risk of developing diseases that do increase mortality through contributions to cardiovascular death.

Lon Kilgore suggests BMI provides little information to scientists, nutritionists and fitness professionals.

If we consider one such disease, diabetes mellitus, and its epidemiology in reference to BMI, we see another interesting relationship—rather, a lack of relationship—between diabetic death rate and BMI (r = 0.331).

Year	Diabetes Deaths per Million Population	BMI
1960	2.25	25.0
1970	2.43	25.4
1980	1.81	25.5
1990	2.07	26.6
2000	2.52	28.0
2007	2.24	28.4

So after considering two of the major causes of death globally, and with diabetes reportedly acting as a contributor to cardiovascular death, we see that BMI is not an effective tool for predicting mortality from cardiovascular causes, diabetes, or as a pair. This calls into question the practice of using BMI for any diagnostic or prognostic purpose. There is not enough direct evidence to suggest it provides any information of value to clinicians or fitness practitioners.

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According to BMI calculations, this top Regional athlete is overweight.

If we consider other measures of body size and their relationship to mortality (from cardiovascular and diabetic causes) we find nearly identical associations as with BMI:

- Cardiovascular Death to Body Weight r = -0.976
- Cardiovascular Death to Height r = -0.872
- Cardiovascular Death to Waist Circumference r = -0.975

As body size increases, the rate of cardiovascular mortality goes down. This is not intuitive, nor is this the information with which the public is presented. In fact, they are presented the exact non-factual opposite.

- Diabetic Death to Body Weight r = 0.293
- Diabetic Death to Height r = 0.067
- Diabetic Death to Waist Circumference r = 0.297

These diabetic values present an absent correlation between increased body size and death rates. Again, this data, the exact same government data available to prepare government and media reports, does not support the concepts presented to the public.

The Real Numbers

Are there any measures that are associated with decreased mortality rates? Yes. Over the past two decades, the percentage of U.S. and U.K. populations who have been physically active has risen. When treated statistically, there is an inverse association between an increasingly active population and a decreased death rate from cardiovascular causes.

Year	Cardiovascular Deaths per Million Population	Percent of Population Reporting Some Activity
1990	32.2	69.1
2000	25.8	72.2
2007	19.1	76.1

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Although the data surrounding physical activity and exercise data are tremendously soft, rife with measurement error and inconsistent definition, the correlation is quite robust (r = 0.999). Despite this, the simultaneous therapeutic and life-extending advances in medicine during the same time period make absolute association or establishment of causality impossible. However, unlike with measures of body dimension, habitual inclusion of activity and exercise into one's activity patterns presents a favorable and easily alterable association—one that is available to all populations across all social and economic divisions.

> If we fix fit first, then fat will sort itself over time.

There is emerging data suggesting that higher exercise intensities are more effective at producing longer lifespans. Epidemiologic work by Dr. Steven Blair and associates suggests that higher intensity aerobic work and resistance training increase longevity more effectively than low-intensity physical activity and exercise. This association strongly suggests that following the CrossFit model of training is much more likely to yield a longer lifespan than following the ACSM-recommended 30 minutes of accumulated low-to-moderate-intensity activity on most days of the week.

The bottom line for fitness professionals is that they should be less worried about making fat trainees skinny; rather, they should focus on making them fit. If we fix fit first, then fat will sort itself over time. By making an individual more fit, he or she should (and remember this is correlation, not causality) become less likely to die from cardiovascular causes, and that is a primary goal of our national public-health agenda: reduce premature mortality.

In this respect, every fitness trainer or coach has a more powerful influence on preventing premature deaths than any other professional population aside from physicians. For this reason, it is imperative that trainers engage actively in educational experiences and seminars that develop, expand, and refine their professional abilities in order to effectively, efficiently and safely improve fitness in the clients they serve.

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Courtesy of Dr. Lon Kilgore

About the Author

Lon Kilgore works at the University of West Scotland Institute for Clinical Exercise and Health Science. He has also held faculty appointments at Midwestern State University, Kansas State University and Warnborough University (IE). He graduated from Lincoln University with a bachelor of science in biology and earned a Ph.D. in anatomy and physiology from Kansas State University. He has competed in weightlifting to the national level since 1972 and coached his first athletes to national championship event medals in 1974. He has worked in the trenches, as a coach or scientific consultant, with athletes from rank novices to professionals and the Olympic elite, and as a collegiate strength coach. He has been a certifying instructor for U.S.A. Weightlifting for more than a decade and a frequent lecturer at events at the U.S. Olympic Training Center. His illustration and authorship efforts include books, magazine columns and research journal publications.

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