Obesity and Life Underwriting
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Abstract: Obesity is increasing in the US population and currently affects one-third of adults. The physiology of obesity is complex and predisposition to obesity is influenced by multiple genes and environment. Obesity may be measured by body fat percentage, body mass index (BMI), or visceral adiposity. Life insurance companies generally use height and weight (build) determinations. The purpose of this paper is to review the life risks and physiology of obesity, and to suggest that the current trend to liberalize traditional build table ratings may not be prudent. A case history will be utilized to demonstrate these points.

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Introduction
Understanding the physiology behind obesity is important for life insurance underwriting for two reasons: 1) obesity is related to excess mortality and yet concomitantly, 2) life insurance tables have tended to liberalize weight tables allowing greater weights before applying an appropriate rating. The purpose of this article is to review the life risks of obesity, the metabolic abnormalities that occur with obesity, and suggest that further liberalization of build tables may not be prudent. A case history will be utilized to demonstrate these points.

Background
Obesity may be defined in many ways. Three commonly used definitions are 1) an increase in the percent of body weight that is fat (>25% for men, and >35% for women), 2) an increase in body mass index (BMI) which is calculated as weight in kilograms divided by height in meters squared (kg/m2), and 3) an increase in the waist to hip ratio or visceral adiposity. For research or clinical settings, percent body fat may be accurately measured by radioactively labeled water isotope scanning or by dual X-ray absorptiometry. In addition, visceral adiposity may be accurately measured by CT scan. However, neither of these measurements is practical for the routine insurance paramedical examination.

For insurance purposes, utilization of the BMI is most useful. According to the National Center for Health Statistics, the following table outlines the ranges for BMI:

<table>
<thead>
<tr>
<th></th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>normal</td>
<td>20.0-27.7 kg/m2</td>
<td>20.0-27.2 kg/m2</td>
</tr>
<tr>
<td>overweight</td>
<td>27.8-31.0 kg/m2</td>
<td>27.3-32.2 kg/m2</td>
</tr>
<tr>
<td>obese</td>
<td>&gt;31.0 kg/m2</td>
<td>&gt;32.2 kg/m2</td>
</tr>
</tbody>
</table>

Table 1. BMI for males and females with ranges of normal, overweight, and obese.

BMI is easy to calculate from the weight and height measurements obtained during the paramedical examination.
Visceral adiposity is an important measurement as many of the metabolic abnormalities of obesity are related to excess visceral fat. In adults, waist to hip values (by circumferential measurements or by CT scan) greater than or equal to 0.95 in men and greater than or equal to 0.85 in women are suggestive of excess visceral adiposity. The paramedical examination might also obtain indirect measurements of visceral adiposity by measuring circumferential waist and hip dimensions. However, this measurement is likely to be impractical for many reasons. Applicants are likely to object to this measurement, and there are also concerns about reliability, reproducibility, and additional examination costs.

Implications
Overall, obesity contributes to 300,000 deaths per year. Mortality ratios increase with increasing BMI. Table 2 below is adapted from data in Brackenridge on mortality ratios and BMI. The original data were derived from three different sources each with more than 750,000 persons: American Cancer Society, life insurance actuarial tables, and a Norwegian population. Thus, the data represent a wide cross-section of people from various geographical areas. Mortality ratios are higher for men at lower BMI than women. This may reflect differences in visceral adiposity and will be discussed later.

<table>
<thead>
<tr>
<th>All-cause Mortality Ratio</th>
<th>BMI kg/m2</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
</table>
| 1.0                      | 21-24     | 21-26
| 1.5                      | 32        | 34   |
| 2.0                      | 36        | 38   |
| 2.5                      | 38        | 40   |

Table 2. Mortality ratios related to approximate BMI for men and women. Data adapted from Brackenridge.

In addition to increasing mortality ratios with BMI, in adults ages 20-44, obesity is associated with increased relative risk (RR) for the development of other impairments. These impairments affect survival as well. The RR and impairment are displayed in the Table 3 below:

<table>
<thead>
<tr>
<th>Impairment</th>
<th>RR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypercholesterolemia</td>
<td>2.1</td>
</tr>
<tr>
<td>Type II diabetes mellitus</td>
<td>3.8</td>
</tr>
<tr>
<td>Hypertension</td>
<td>5.6</td>
</tr>
</tbody>
</table>

Table 3. Relative risk of developing hypercholesterolemia, type II diabetes mellitus or hypertension when obese.

Thus, obesity increases the relative risks for hypercholesterolemia, Type II diabetes mellitus, and hypertension. All are major risk factors for atherosclerosis. Not surprisingly, obesity is associated with a three-fold increase risk for myocardial infarction. It is unclear if obesity without complications is associated with excess mortality, except for visceral adiposity as discussed below.

Visceral adiposity has independent effects on mortality. In two Swedish studies of men and women separately, the highest tertile for waist to hip ratio was associated with greater probabilities of both myocardial infarction, stroke, and death from any cause. Both studies also demonstrated that high waist to hip ratio was associated with decreased survival at the highest and the lowest tertiles for BMI. In other words, high visceral adiposity correlated with myocardial infarction, stroke, and death from any cause independent of BMI. The mechanism behind greater mortality with visceral adiposity is most likely related to the activity of the visceral adipocyte. The suggestion is that visceral adipocytes contribute to excess free fatty acid release into the portal circulation. This in turn affects liver clearance of VLDL and LDL and leads to lower levels of HDL2. In addition, excess free fatty acid exposure is likely to antagonize the effect of insulin on the liver with the end result being a state of hepatic gluconeogenesis and glycogenolysis. This may lead to hyperglycemia and insulin resistance. In summary, visceral adiposity can predispose to atherosclerosis and appears to be an independent risk factor for excess mortality at all ranges of BMI. However, it is unknown what factors influence where adipose tissue accumulates. It may be related to sex steroids, gluco-
corticoids, or genetic makeup.

Obesity also contributes to an atherogenic phenotype that has been called Syndrome X or Metabolic Syndrome. Syndrome X or Metabolic Syndrome is a constellation of clinical abnormalities that includes hypertension, hyperglycemia, hypercholesterolemia, hypertriglyceridemia, and hyperuricemia. There are no mortality data on Syndrome X or Metabolic Syndrome. What is known, is that the metabolic abnormalities are most likely the result of hyperinsulinemia and insulin resistance. Hyperinsulinemia and insulin resistance occurs with greater frequency in people who are overweight and obese than in those who are of normal weight. Hyperinsulinemia has also been shown to be an independent risk factor for ischemic heart disease. Depres et al conducted a case-control study of men and noted that those who developed ischemic heart disease over a 5 year period has an 18% increase in fasting insulin levels compared to controls.

Regulation of Body Weight
Predisposition to obesity is polygenic and environmental. In any one individual it is difficult to analyze what contribution genetics, social/family structure, stress management, personal habits, etc. have on eating behavior. In terms of physiologic mechanisms, it is clear that many polypeptides influence body weight. A recently described gene product that is likely one regulator of body weight is leptin. Leptin is a hormone released by adipocytes. It acts as an afferent signal to the hypothalamus where it acts as a satiety factor. In humans, it is postulated that leptin inhibits neuropeptide Y which is an appetite stimulant. In addition, leptin likely regulates melanocortin which is an appetite suppressant. At present, it is not clear what regulates adipocyte leptin gene transcription however, glucose, insulin, glucocorticoids, and thyroid hormone have all been implicated.

Leptin levels are high in overweight and obese humans, suggesting that high levels either contribute to obesity or sustain it because of hypothalamic “leptin resistance.” Ronnemaa et al have studied identical twins discordant for body weight and discovered that high leptin levels correlated with body mass despite identical genetics. Leptin levels are also higher in women than in men for any given BMI. Many speculate that this reflects differences in body fat distribution.

Another gene product related to body fat regulation in humans is the beta 3 adrenergic receptor of brown fat. Although brown fat is found in small quantities in humans, it is an important metabolically active tissue that is partly responsible for thermogenesis. The role of beta 3 adrenergic function in obesity is undergoing extensive study. Walston et al. have noted ethnic variations in beta 3 receptor function that may be attributable to lower metabolic rates and tendency to obesity and Type II diabetes mellitus. For example, in one group of Pima Indians, a homozygous deletion for the beta 3 receptor was present, suggesting that the beta 3 receptor gene is another candidate “obesity gene.” Widen et al have made similar observations regarding beta 3 receptor gene defects in a Finnish population with a predisposition to obesity and Type II diabetes mellitus.

Treatment
Using alcoholism as a model, many physicians now approach the treatment of obesity as an illness and not a problem of willpower and self control. In addition, many view the treatment of obesity as a chronic illness, like hypertension, which requires life-long intervention and therapy.

The approach to treatment is multilevel involving diet, behavior modification, exercise, stress reduction, and pharmacological therapies. Pharmacological therapies are rapidly becoming available. Fenfluramine (Pondimin) and Dexfenfluramine (Redux) are a class of serotonin uptake inhibitors that influence weight by suppressing appetite at the level of the hypothalamus. They were FDA approved for those who are either 30% over ideal body weight or those who are 20% over
ideal body weight and also have diabetes, hypertension, or hypercholesterolemia. However, because of alarming reports about a possible causal relationship between fenfluramine (with phentermine) and dexfenfluramine and pulmonary hypertension\(^1\) or valvular heart disease,\(^2\) these medications have been pulled from the market. Furthermore, results with pharmacological agents were variable.\(^2\),\(^2\) Compared to diet and exercise, these agents had no appreciable longstanding success for achieving normal body weight.

**Case History**

The applicant is a 28 year old woman, who works as a retail clerk. She is applying for $50,000 of variable life insurance. The beneficiary is her mother, and the date of the application is January 1997. She reports on the application her build as 5’8” and 189 pounds (BMI=28.6). She has had weight loss in the last year due to diet. Her parents are alive and well into their 50s. Last doctor visit was on 11/96 for “diabetes and weight loss diet.” No medications.

The attending physician’s statement is as follows:

5/96: Desires weight loss. Reports history of diabetes and hypertension (unknown duration). No medicines. Build: 5’8” 286 pounds (BMI=40.7). B/P 162/104. No exam. Treatment was begun with diabetic diet and exercise program; and given phentermine (Fastin), fenfluramine (Pondimin), and furosemide (Lasix) PRN edema.

6/96: 258 pounds (BMI=39.2). B/P 126/86. FBS: 142 mg%. Triglycerides: 372 mg%.


9/96: 237 pounds (BMI=36.0). B/P 122/74. FBS: 144 mg%. Triglycerides: 324 mg%.


11/96: 227 pounds (BMI=34.5). B/P 138/98. FBS: 101 mg%. Triglycerides: 173 mg%.

12/96: Did not keep appointment.

1/97: Has been off diet and medicines and wants to restart. 241 pounds (BMI=36.6). B/P 142/101.

**Case Analysis**

This applicant illustrates many points. First, it is important to look at her BMI instead of weight alone. The excess mortality related to her weight may not be ratable, given the trend for liberalizing build tables by ten and fifteen pounds. However, calculating the BMI gives a better appreciation of the degree of obesity and the possible risk. This possible risk is inferred based on the known relative risks for the development of other impairments, because at present, the extra deaths per 1000 for each BMI are not known. The point is that if one looked at her weight alone she may not be assigned any debits.

Second, with her excess weight, she has elevated fasting blood sugars. The World Health Organization recently re-defined normal blood glucose as fasting less than or equal to 125 mg%. Fasting blood glucose greater than 125% will indicate diabetes mellitus. This applicant is diabetic according to this new standard. Most likely her obesity is contributing to a state of insulin resistance and hyperinsulinemia for maintenance of euglycemia. Insulin resistance leads to excessive hepatic glucose production in the fasted state.\(^3\) Insulin mediated inhibition of free fatty acid release is impaired when there is insulin resistance.\(^3\) She is at risk for Syndrome X or Metabolic Syndrome. In addition, she most likely has an increased mortality risk from hyperinsulinemia alone.\(^10,23\)

Third, she has fasting hypertriglyceridermia. Even though hypertriglyceridermia itself has not been shown to be attributable to excess cardiovascular disease mortality, it does contribute to changes in lipids that are detrimental. For example, hypertriglyceridermia is related to excessive VLDL and IDL production. The latter is a direct precursor to LDL. Hypertriglyceridermia may contribute to the formation of small, dense LDL which is more easily
oxidized. HDL2 levels are also decreased in the setting of hypertriglyceridemia.

Fourth, despite her young age, she is hypertensive. Again, hyperinsulinism is most likely playing a role. Like build debits, blood pressure debits are often not applied until blood pressure readings exceed 150/100. If both debits are overlooked, this would compound an inappropriate risk assessment.

Finally, pharmacological therapy was initiated with diet in her care. Her metabolic abnormalities improved when she lost weight even without attaining normal body weight. However, her weight loss was not sustained. She demonstrates what is common: initial success with weight loss, followed by weight re-gain.

Our case history demonstrates many of the risks related to obesity. She has hypertension and fasting hyperglycemia and hypertriglyceridemia. She has not sustained weight loss. If each impairment were viewed in isolation, she might not be assigned any debits. However, it is clear that she presents a risk for excess mortality when compared to the life expectancy of a select 28 year old female.

Summary
The physiology of obesity is complex. The predisposition to obesity is influenced by multiple genes and environment. The excess mortality from obesity increases with increasing BMI. In addition, visceral adiposity may pose greater risk than absolute BMI alone. The excess mortality from obesity is most likely the result of changes in the metabolic milieu, i.e. hypertriglyceridemia, hypercholesterolemia, hyperglycemia, hyperinsulinemia, possibly hyperleptinemia, and hypertension.

Obesity is increasing in the US population and currently affects one-third of the adult population. Further liberalization of build tables will likely overlook the additional risk attributable to excess weight. Measurements of BMI and visceral adiposity may better reflect the state of obesity and the degree of risk.

References