Development of the Health Index (HI) Statistical Equation as an Alternative Clinical Parameter to the Body Mass Index in the Prediction of Body Fat Percentage

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Citation

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Abstract

Metabolic syndrome is increasing in prevalence in the United States. Body Mass Index is a formula most commonly used to assess body fat percentage. However, the formula contains significant limitations and inefficiencies. As a result, the Health Index equation was developed as an alternative to the BMI. Data from 79 healthy individuals, as defined by the absence of metabolic syndrome risk factors, was collected and a relationship was established between their BMI and body fat percentage. The results showed the BMI produced a poor correlation with the body fat percentage while the Health Index metric produced a stronger correlation to actual body fat percentage.

INTRODUCTION

Metabolic syndrome, a combination of risk factors which increases the risk for cardiovascular disease, diabetes, which all-cause mortality, affects as much as one quarter of the United States population.¬[1-5] The prevalence of metabolic syndrome is increasing in the United States[6] and the cause may be visceral fat mediated insulin resistance, through various cytokines.[7] Central obesity is perhaps the most important risk factor.[8-13] However, other risk factors, such as stress[14-15], sedentary lifestyle[8], aging[8], lipodystrophy[8], and rheumatic diseases have also been proposed.[16]

In most cases of metabolic syndrome, the Body Mass Index (BMI), a statistical measure used to estimate healthy body weight based on a person's height, is the tool used to distinguish between underweight, overweight, and obese patients.[19-22] However, it has significant limitations. First of all, BMI is stature dependent. Therefore, individuals with shorter legs have higher BMIs.[23] Further, the BMI differs between cultures. For instance, in Japan, the BMI's upper limit of normal is 8% which is lower than standards used in the United States.[24-25]

Perhaps most importantly, the BMI cannot distinguish between mass due to muscle and mass due to fat, thus making it "as much a measure of lean body mass as it is a measure of fatness or obesity."[23] Therefore, when a patient loses muscle (and gains proportionately in fat) it actually decreases (because muscle is denser than fat) and when a patient gains muscle and loses fat, it increases (for the same reason). As a result, BMI is an inconsistent predictor of physical health and obesity-related health risk. Other measures, such as waist-circumference are much better predictors.[26-32] In fact, studies have shown that even at low BMIs, high body fat percentage is an important predictor of cardiovascular risk.[33]

Since visceral fat is an important etiologic cause of metabolic syndrome complications, the BMI must be adjusted or replaced by an equation which is based on body fat percentage, since this may lead to better prediction of clinical end points (such as diabetes, coronary heart disease, and stroke).[34-38]

METHODS

Health data was collected from public sources containing the following variables: height, weight, body fat percentage, diabetes mellitus, myocardial infarction and stroke. A total of 79 individuals (32 males and 47 females) with a "perfect" state of health in their prime were selected. A "perfectly healthy" individual, for the purposes of our analysis, was defined as a 25-35 year old male or female, with no known risk factors for metabolic syndrome, body fat percentage of around 12% for males and around 20% for females, and no history of diabetes, myocardial infarction, or stroke.

With this data, we ran multiple regressions in order to establish a standard relationship between the BMI and body fat percentage. We then modified the BMI depending on the variables described above in order to improve the predictive value of the BMI with regards to estimated body fat percentage. The equations developed are shown in Table 1.

It must be noted that we made several assumptions. Our first assumption is that patients with risk factors have 7% more body fat per risk factor than those who don't have that risk factor. This assumption is based on research showing that patients with a risk factor, such as diabetes, have a BMI predicted fat percentage which is ~5% higher than those who do not have the risk factor.[39,40] In our population, individuals with a risk factor had 12% higher BMI predicted body fat percentage. For simplicity, we used 7%, though it is a somewhat arbitrary figure.

Moreover, patients who smoke (or have smoked for a significant period of time, as defined by more than 5 pack years), get an increase of their Health Index by 2.5. This is based on data that shows that smoking increases BP by 5%[41], cholesterol by 33%[42], risk of diabetes by 29-61% (depending on amount of cigarettes consumed per day)[43], risk of PVD (peripheral vascular disease) by 184%[44], risk of stroke by 414%[45], and risk of CAD by 500%.[46] In comparison, rates for coronary disease in the non-smoking population are approximately 2%.[46] Given this data, smoking increases the absolute risk of soft end points by approximately 5 to 15 percent. Since the average Health Index is 27.76, the increase in Health Index from smoking should be 1.4 to 4 points. For simplicity, we used 2.5.

RESULTS

In healthy individuals, the BMI should be 18.5–24.9. However, in this healthy population of 79 individuals, the BMI's standard deviation was 4.9 with a mean of 21.5, tending to classify healthy females as underweight, and healthy males as overweight (see Table 2, Figure 1). When compared to the BMI, the HI produces a more consistent assessment (95% CI = 18.13-19.22, $p < 0.001$). Moreover, the HI Predicted Body Fat Percentage is better correlated to body fat percentage than the BMI (0.86 v. 0.13) in this population (see Table 3, Figure 2). As a result, the HI better approximates the true body fat percentage. In our population, the HI is, on average, within +/- 2% of actual body fat percentage, while the BMI is within +/-5%, ($p < 0.001$).

Figure 1

Table 1

HEALTH INDEX EQUATIONS

General End Point Predictor

$$HI\ (male) = BMI\ (0.95) + 0.079\ (age) - 1.29 + 2.5\ (smoking)$$

$$HI\ (male\ athlete) = BMI\ (0.78) + 0.079\ (age) - 1.29 + 2.5\ (smoking)$$

$$HI\ (female) = BMI\ (1.11) + 0.079\ (age) - 1.93 - 1 + 2.5\ (smoking)$$

$$HI\ (female - athlete, estimate) = BMI\ (1.03) + 0.079\ (age) - 1.93 - 1 + 2.5\ (smoking)$$

Body Fat % Estimation

$$HI\ Predicted\ Body\ Fat\ \%\ (male, athlete) = HI * (0.006 + \frac{6.65\ (Risk\ Factors)}{10{,}000})$$

$$HI\ Predicted\ Body\ Fat\ \%\ (male) = HI * (0.008 + \frac{6.65\ (Risk\ Factors)}{10{,}000})$$

$$HI\ Predicted\ Body\ Fat\ \%\ (female, athlete) = HI * (0.009 + \frac{6.65\ (Risk\ Factors)}{10{,}000})$$

$$HI\ Predicted\ Body\ Fat\ \%\ (female) = HI * (0.01 + \frac{6.65\ (Risk\ Factors)}{10{,}000})$$

Derivations

$$Health\ Index\ Constants = \left(\frac{\Sigma \frac{BMI_i}{20}}{n}\right) + \frac{\Sigma\left(\frac{\Sigma \frac{BMI_i}{20}}{n} - 20\right)}{n}$$

$$HI\ Predicted\ Body\ Fat\ Constant = \frac{\Sigma \frac{Actual\ Body\ Fat\ \%}{HI_i}}{n}$$

*Athlete defined as an individual performing anabolic exercise 2+ times a week or catabolic exercise 3+ times per week.
**Risk Factors include: HTN, Hyperlipidemia, DM, PVD, AAA, CAD, Age > 45 (m), and Age > 55(f). Note: HDL > 60 negates one risk factor. Tobacco use also deducts one risk factor because it decreases (rather than increases) body fat percentage.
***Risk Factor Adj = # of Risk factors. Females are deducted one risk factor because of their typically higher HDL levels.

Figure 2

Table 2

	Body Mass Index	Health Index
Sample size	79	79
Arithmetic mean	21.4994	18.6751
95% CI for the mean	20.4049 to 22.5938	18.1307 to 19.2194
Variance	23.8762	5.9062
Standard deviation	**4.8863**	**2.4303**
Standard error of the mean	0.5498	0.2734
F-test for equal variances		P < 0.001
Difference		-2.8243
Standard Error		0.6140
95% CI of difference		-4.0371 to -1.6115
Test statistic t		-4.600
Degrees of Freedom (DF)		156
Two-tailed probability		P < 0.0001

Figure 3

Figure 1

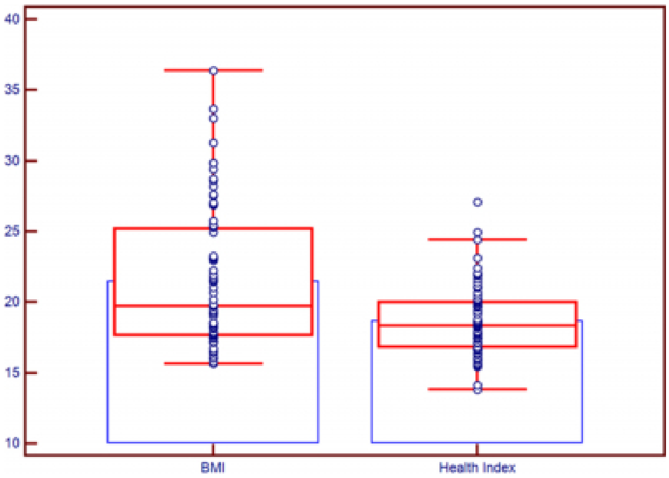


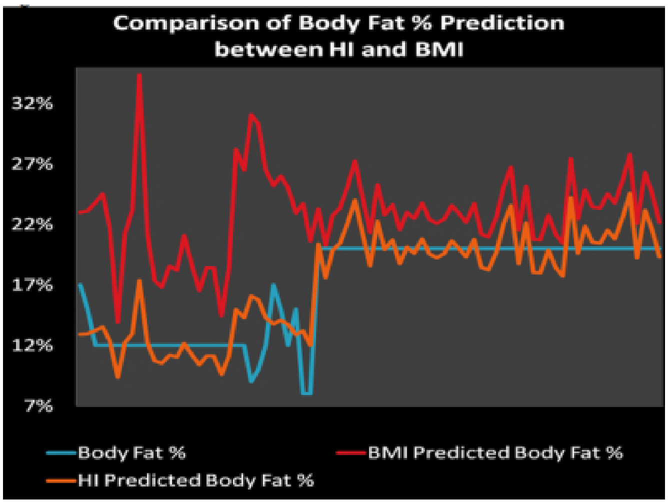
Figure 4

Table 3

Statistic	HI Predicted Body Fat %	Actual Body Fat Percentage	BMI Predicted Body Fat %
Mean	17%	17%	22%
Biased Variance	0.00183	0.00162	0.00115
Biased Standard Deviation	0.04279	0.0403	0.03392
Covariance	0.001505		0.000184
Correlation	0.861725		0.133042
Determination	0.74257		0.0177
T-Test	14.9034		1.7791
p-value (1 sided)	P < 0.001		P = 0.1212
Degrees of Freedom	77		77
Number of Observations	79		79

Figure 5

Figure 2



DISCUSSION

The results show that the HI is a more accurate predictor of body fat percentage in our study population than the BMI. In healthy individuals, the predictive model of the HI is strongly correlated to the actual body fat percentage ($R = 0.86$, $p < 0.001$), while the BMI predictive model is weakly correlated and not statistically significant ($R = 0.13$, $p = 0.12$).

The objective of this research was to create a statistical model to be tested in a prospective research project, where the HI can be used as a clinical predictor versus the BMI. Once this equation is modified based on a larger, more representative population, the accuracy and precision of the statistical model should improve. The intent of this research was to serve as a pilot study that provides the basis for a more comprehensive analysis, which is currently underway at Cleveland Clinic Florida.

It should be noted that the "Health Index" is a general equation predicting onset and complications of Metabolic Syndrome. It should not be used to predict general mortality, as it does not account for mortality associated with cachexia, cancer, and other conditions. Therefore, it should only be used to predict mortality from heart attack or stroke.

References

1. Ford ES, Giles WH, Dietz WH (2002). Prevalence of metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey. JAMA 287(3):356-359.
2. Haffner SM, Valdez RA, Hazuda HP, Mitchell BD, Morales PA, Stern MP. Prospective analysis of the insulin-resistance syndrome (syndrome X). Diabetes

3. Isomaa B, Almgren P, Tuomi T, et al. Cardiovascular morbidity and mortality associated with the metabolic syndrome. Diabetes Care. 2001;24:683-689
4. Trevisan M, Liu J, Bahsas FB, Menotti A. Syndrome X and mortality: a population-based study. Am J Epidemiol. 1998;148:958-966.
5. National Institutes of Health. Third Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Bethesda, Md: National Institutes of Health; 2001. NIH Publication 01-3670.
6. Ford ES, Giles WH, Mokdad AH. Increasing prevalence of the metabolic syndrome among US adults. Diab Care (2004) 27:2444–9
7. Fukuchi S, Hamaguchi K, Seike M, Himeno K, Sakata T, Yoshimatsu H. (1 June 2004). "Role of Fatty Acid Composition in the Development of Metabolic Disorders in Sucrose-Induced Obese Rats". Exp Biol Med 229 (6): 486–493.
8. Fauci, Anthony S. (2008). Harrison's principles of internal medicine. McGraw-Hill Medical.
9. Irwin ML, Ainsworth BE, Mayer-Davis EJ, Addy CL, Pate RR, Durstine JL: Physical activity and the metabolic syndrome in a tri-ethnic sample of women. Obes Res 10:1030–1037, 2002
10. Kullo IJ, Hensrud DD, Allison TG: Relation of low cardiorespiratory fitness to the metabolic syndrome in middle-aged men. Am J Cardiol 90:795–797, 2002
11. Case CC, Jones PH, Nelson K, O'Brian Smith E, Ballantyne CM: Impact of weight loss on the metabolic syndrome. Diabetes Obes Metab 4:407–414, 2002
12. Case CC, Jones PH, Nelson K, O'Brian Smith E, Ballantyne CM: Impact of weight loss on the metabolic syndrome. Diabetes Obes Metab 4:407–414, 2002
13. Gazzaruso C, Giordanetti S, La Manna A, Celsa M, De Amici E, Turpini C, Catona A, Fratino P: Weight loss after Swedish adjustable gastric banding: relationships to insulin resistance and metabolic syndrome. Obes Surg 12:841–845, 2002
14. Gohill, BC; Rosenblum, LA; Coplan, JD; Kral, JG; (July 2001). "Hypothalamic-pituitary-adrenal axis function and the metabolic syndrome X of obesity". CNS Spectr. 6 (7): 581–6, 589.
15. Tsigos, C; Chrousos, GP; (October 2002). "Hypothalamic-pituitary-adrenal axis, neuroendocrine factors and stress". J Psychosom Res. 53 (4): 865–71.
16. Quilon A III, Brent L. The primary care physician's guide to inflammatory arthritis: diagnosis. J Musculoskel Med. 2010;27:223-231
17. Katzmaryk, P.T.; Leon, Arthur S.; Wilmore, Jack H.; Skinner, James S.; Rao, D. C.; Rankinen, Tuomo; Bouchard, Claude (October 2003). "Targeting the Metabolic Syndrome with Exercise: Evidence from the HERITAGE Family Study.". Med. Sci. Sports Exerc 35 (10): 1703–1709.
18. Volek JS, Feinman RD (2005). "Carbohydrate restriction improves the features of Metabolic Syndrome. Metabolic Syndrome may be defined by the response to carbohydrate restriction". Nutr Metab (Lond) 2: 31. doi:10.1186/1743-7075-2-31
19. Keys A, Fidanza F, Karvonen MJ, Kimura N, Taylor HL. Indices of relative weight and obesity. J Chronic Dis l972, 25:329-43.
20. Landis JR, Lepkowski JM, Ekiund SA, Stehouwer SA. A statistical methodology foranalyzingdata from a complex survey: the first national health and nutrition examination survey. Hyattsville, MD: Department of Health and Human Services, 1982. [DHHS publication no (PHS) 82-2366.]
21. Eknoyan, Garabed (January 2008). "Adolphe Quetelet (1796-1874)—the average man and indices of obesity". Nephrol. Dial. Transplant. 23 (1): 47–51.
22. Cole TJ, Flegal KM, Nicholls D, Jackson AA. Body mass index cut offs to define thinness in children and adolescents: international survey. Bmj 2007;335(7612):194.
23. Garn, S.M., Leonard, W.R., & Hawthorne, V. (1986). Three limitations of the body mass index. The American Journal of Clinical Nutrition, 44, 996-997.
24. Who's fat? new definition adopted. (1998, June 17). Retrieved from http://www.cnn.com/HEALTH/9806/17/weight.guidelines/
25. Shiwaku, K., Anuurad, E., Enkhmaa, B., Nogia, A., & Kitajima, K., Yamane, Y., Oyunsuren, T. (2004). Overweight japanese with body mass indexes of 23.0-24.9 have higher risks for obesity-associated disorders: a comparison of japanese and mongolians. International Journal of Obesity, 28(1), 152-158.
26. Janssen I, Katzmarzyk PT, Ross R. Waist circumference and not body mass index explains obesity-related health risk. Am J Clin Nutr (2004) 79(3):379–384
27. Han TS, McNeill G, Seidell JC, et al. Predicting intra-abdominal fatness from anthropometric measures: the influence of stature. Int J Obes Relat Metab Disord (1997) 21(7):587–593.
28. Stewart KJ, DeRegis JR, Turner KL, et al. Usefulness of anthropometrics and dual-energy x-ray absorptiometry for estimating abdominal obesity measured by magnetic resonance imaging in older men and women. J Cardiopulm Rehabil (2003) 23(2):109–114.
29. Ho SC, Chen YM, Woo JL, et al. Association between simple anthropometric indices and cardiovascular risk factors. Int J Obes Relat Metab Disord (2001) 25(11):1689–1697.
30. Janssen I, Katzmarzyk PT, Ross R. Waist circumference and not body mass index explains obesity-related health risk. Am J Clin Nutr (2004) 79(3):379–384.
31. Wang Y, Rimm EB, Stampfer MJ, et al. Comparison of abdominal adiposity and overall obesity in predicting risk of type 2 diabetes among men. Am J Clin Nutr (2005) 81(3):555–563.
32. Wei M, Gaskill SP, Haffner SM, et al. Waist circumference as the best predictor of noninsulin dependent diabetes mellitus (NIDDM) compared to body mass index, waist/hip ratio and other anthropometric measurements in Mexican Americans—a 7-year prospective study. Obes Res. (1997) 5(1):16–23.
33. Deurenberg-Yap, M., Chew, S.K., & Deurenberg, P. (2002). Elevated body fat percentage and cardiovascular risks at low body mass index levels among singaporean chinese, malays and indians. Obesity Reviews, 3(3), 209-215.
34. Carey VJ, Walters EE, Colditz GA, et al. Body fat distribution and risk of non-insulin-dependent diabetes mellitus in women. The Nurses' Health Study. Am J Epidemiol (1997) 145(7):614–619.
35. Kaye SA, Folsom AR, Sprafka JM, et al. Increased incidence of diabetes mellitus in relation to abdominal adiposity in older women. J Clin Epidemiol (1991) 44(3):329–334.
36. Folsom AR, Prineas RJ, Kaye SA, et al. Body fat distribution and self-reported prevalence of hypertension, heart attack, and other heart disease in older women. Int J Epidemiol (1989) 18(2):361–367.
37. Rexrode KM, Carey VJ, Hennekens CH, et al. Abdominal adiposity and coronary heart disease in women. JAMA (1998) 280(21):1843–1848.

38. Kurth T, Gaziano JM, Berger K, et al. Body mass index and the risk of stroke in men. Arch Intern Med 2002; 162: 2557–62.
39. Wang, Y, Joanna, Q. The prevalence of prehypertension and hypertension among US adults according to the new national committee guidelines. Archives of Internal Medicine; 164: 2126-2134.
40. Wild, S.H., Roglic, G., Green, D., Sicree, R., King, H. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. Diabetes Care; 27: 2569.
41. Narkiewicz, K, Maraglino, G, Biasion, T, et al. Interactive effect of cigarettes and coffee on daytime systolic blood pressure in patients with mild essential hypertension. J Hypertens 1995; 13:965.
42. Akbari, M.Z.A., Sarwar, M.S., Shakoor, M. Lipid Profile in Smoking. JAMC 200; 12: 19-21.
43. Willi C, Bodenmann P, Ghali WA, Faris PD, Cornuz J. Active smoking and the risk of type 2 diabetes: a systematic review and meta-analysis. JAMA. 2007;298:2654–2664.
44. Navas-Acien A, Selvin E, Sharrett AR, Calderon-Aranda E, Silbergeld E, Guallar E. Lead, cadmium, smoking, and increased risk of peripheral arterial disease. Circulation. 2004; 109: 3196–3201.
45. Bonita R, Duncan J, Truelsen T, et al. Passive smoking as well as active smoking increases the risk of acute stroke. Tob Control 1999; 8:156–60.
46. Parish S, Collins R, Peto R, Youngman L, Barton J, Jayne K, Clarke R, Appleby P, Lyon V, Cederholm-Williams S, Marshall J, Sleight P (1995) Cigarette smoking, tar yields, and non-fatal myocardial infarction: 14,000 cases and 32,000 controls in the United Kingdom. The International Studies of Infarct Survival (ISIS) Collaborators. BMJ 311:471–477.
47. 47. Deurenberg P, Andreoli A, Borg P, et al. The validity of predicted body fat percentage from body mass index and from impedance in samples of five European populations. European Journal of Clinical Nutrition 2001; 55 (11): 973-979
48. 48. Gallagher D, Heymsfield SB, Heo M, Jebb SA, Murgatroyd PR, Sakamoto Y. Healthy percentage body fat ranges: an approach for developing guidelines based on body mass index. Am J Clin Nutr 72: 694–701, 2000.
49. 49. Deurenberg P, Weststrate, JA, Seidell J. Body mass index as a measure of body fatness: age- and sex-specific prediction formulas. British Journal of Nutrition (1991), 65:105-114.
50. 50. Zhou M, Offer A, Yang G, Smith M, Hui G, Whitlock G, Collins R, Huang Z, Peto R, Chen Z. Body mass index, blood pressure, and mortality from stroke: a nationally representative prospective study of 212 000 Chinese men. Stroke. 2008; 39: 753–759.
51. 51. Wolk R, Berger P, Lennon RJ, Brilakis ES, Somers VK. Body mass index: a risk factor for unstable angina and myocardial infarction in patients with angiographically confirmed coronary artery disease. Circulation. 2003; 108: 2206–2211.

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