Original Research Article

Body Mass Index Versus Dual Energy X-Ray Absorptiometry-Derived Indexes: Predictors of Cardiovascular and Diabetic Disease Risk Factors

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Objectives: The body mass index (BMI), a ratio of weight/height^2, dominates estimation of adiposity in population studies. BMI, however, does not distinguish among fat, muscle, or bone mass. Accordingly, its usage to assess and manage obesity in the population is limited. This study compares the use of BMI with direct measures of fat- and lean-mass to predict established cardiovascular and diabetes risk factors: blood pressure, lipids, and glucose.

Methods: The entire Buffalo Police Department was the object of recruitment to a baseline study of physiological and psychological stress. Four hundred nine officers constitute the sample for this analysis. Regression methods focusing on explained variance in blood pressure, high density lipoprotein (HDL) cholesterol, and blood glucose compare the use of BMI to that of fat- and lean-mass indexes derived from dual energy X-ray absorptiometry (DEXA).

Results: DEXA indexes explain 1.6%–3.3% ($P < 0.05$, all risk factors) more variance than BMI. Fat mass drives the association for blood pressure, trunk lean mass for HDL cholesterol, and both for blood glucose. High degrees of multicollinearity complicate interpretation of predictive models jointly containing BMI and DEXA indexes.

Conclusions: In police officers, DEXA indexes are better predictors of cardiovascular disease and diabetes risk factors. However, populations with different distributions of fitness, diet, and health conditions may demonstrate different features. In contrast to BMI, DEXA-derived measurements suggest avenues to explore metabolic processes, which relate to an index's underlying association with risk and may suggest more effective intervention strategies. Am. J. Hum. Biol. 00:000–000, 2012. †Published 2012 Wiley Periodicals, Inc.

Defined by Quetelet in 1832 (Eknoyan, 2008), the body mass index (BMI) attempts to standardize the relation between weight and height to assess weight disproportionate to height. BMI is derived by weight/height^2. The metrics are, by convention, kilograms and meters. Other indexes have been considered; however, since the 1960s, BMI has dominated (Billewicz et al., 1962; Khosla and Lowe, 1967).

In developed nations, weight in excess of height is the disproportion of concern. Excess weight is usually attributed to obesity, although the validity of this view resides with underlying features of a target population. A younger population may have different morphological features, e.g., muscle mass, than older populations where excess weight is more likely driven by increased fat mass correlated with loss of muscle mass (Baumgartner et al., 1995; National Center for Health Statistics (U.S.) and National Health and Nutrition Examination Survey (U.S.), 2010). In the elderly, longitudinal changes—including menopausal effects—can be detected within 1 to 2 years (Visser et al., 2003). Differences can occur comparing birth cohorts even at the same age or ethnically distinct populations—Europeans, Asians, and Africans (Araujo et al., 2007; Deurenberg et al., 2002; Gallagher et al., 1996; Lu et al., 2011; Nam et al., 2010; Nelson et al., 1995; Pollitzer and Anderson, 1989; Rush et al., 2007; Sun et al., 2003; Tobias et al., 1994).

This report from the Buffalo Cardio-Metabolic Occupational Police Stress (BCOPS) cohort is limited to examining morphological constituents of body weight measured by dual energy X-ray absorptiometry (DEXA). DEXA partitions weight into fat-, lean-, and bone-mass. Statistical associations of three well-established cardiovascular and diabetes risk factors with indexes created for fat- and lean-mass partitions are compared with established associations with BMI (Lewis et al., 2009). Do these partitions perform better than BMI?

METHODS AND PROCEDURES

The cohort

BCOPS was established in 2004 as a collaboration between the National Institute for Occupational Safety and Health and the State University of New York at Buffalo (Violanti et al., 2006). The Buffalo Police Department was the target population. Now a longitudinal study, baseline recruitment commenced May 21, 2004, the first participant examined on June 4, and the last in October 2009. During recruitment, the eligible target population decreased from 710 in 2004 to 635 in 2007.

A total of 464 officers were examined at baseline. Of these, 430 had DEXA measurements; 33 were examined after they had retired, 28 of these before the study and are included as long as complete data are available. One officer was excluded for an outlying glucose measurement. A total of 409 officers had complete data. Race strata were

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TABLE 1. Simple statistics and correlations[ab] of anthropometric, DEXA, and risk factor measurements in the BCOPS cohort (n = 409)

Variables	Mean ± SD[a]	BMI[c] (kg/m^2)	FMI[c] (kg/m$^{1.4}$)	LMI$_t$[c] (kg/m$^{1.2}$/kg$^{0.3}$)	LMI$_e$[c] (kg/m$^{1.2}$/kg$^{0.3}$)	Bone density (g/cm^2)
Age (years)	42.9 ± 8.1	0.080	0.136	0.071	−0.071	−0.031
Systolic BP (mmHg)	121.8 ± 12.2	0.229	0.276	0.168	0.096	0.018
HDL cholesterol (mg/dL)	46.6 ± 13.2	−0.225	−0.157	−0.298	−0.263	0.012
Blood glucose (mg/dL)	93.4 ± 11.6	0.232	0.241	0.247	0.144	−0.042
BMI[c] (kg/m^2)	29.1 ± 4.0	1	0.897	0.832	0.742	0.024
FMI[c] (kg/m$^{1.4}$)	10.6 ± 3.4	0.897	1	0.626	0.452	−0.093
LMI$_t$[c] (kg/m$^{1.2}$/kg$^{0.3}$)	11.7 ± 1.6	0.832	0.626	1	0.784	−0.153
LMI$_e$[c] (kg/m$^{1.2}$/kg$^{0.3}$)	11.3 ± 1.9	0.742	0.452	0.784	1	−0.026
Bone Density (g/cm^2)	1.24 ± 0.10	0.024	−0.093	−0.153	−0.026	1

[a]Means, standard deviations (SD) and correlations are adjusted for race and sex.
[b]Two-tail critical values of correlation coefficient: P = 0.05, 0.097; P = 0.01, 0.128; P = 0.001, 0.163; P = 0.0001, 0.191.
[c]BMI, body mass index; FMI, fat mass index; LMI, lean mass index; t, trunk; e, extremities. See text for definitions.

limited to Blacks and Whites, with Hispanics (n = 7) included as Whites.

Human subjects review board oversight is detailed in a previous article describing establishment of BCOPS (Violanti et al., 2006).

Study design

Analyses reported in this article reflect a cross-sectional design. The associations being assessed reflect well-established causal relationships. Similarly, population changes in obesity, muscle mass, and loss of physical activity coevolve with changes in cardiovascular and diabetes risk, thus any "snap-shot in time"—i.e., cross-section—will reflect this coevolution. This logic is sufficient to assess the fundamental question, "Do DEXA partitions perform better than BMI in explaining variation in risk factors?"

DEXA measurements

DEXA (Hologic QDR-4500A; Hologic, Waltham, MA) was used to measure whole body and segmental fat-, bone-, and lean-mass, and bone density (Haarbo et al., 1991; Tothill and Hannan, 2000). All scans were performed on the same device. Coefficients of variation (CV) were determined from duplicate measurements on 40 officers: 1.4%, 0.9%, and 0.5%, respectively, for total fat-mass, bone-mass, and bone density. A daily manufacturer quality control phantom ensured no drift. No software upgrades occurred during recruitment.

Anthropometry and cardiovascular and diabetes risk factors

Certified study staff performed anthropometric measurements, including height and weight, respectively, measured with shoes removed and recorded to the nearest ½ cm, and rounding up to the nearest ¼ lb then converted to kg. Systolic blood pressure (SBP) was measured after at least 5 min rest. Appropriately sized cuffs were matched to arm size, the inflatable inner bladder centered over the brachial artery. Three readings were recorded to the nearest even digit with rounding up; the average was used in analyses.

Analytes were assessed from blood specimens obtained after fasting at least 12 h. Serum was prepared by centrifugation. Assays were performed either on fresh or frozen sera using the Beckman Coulter LX20 clinical chemistry analyzer. An adjudication committee identified the use or nonuse of medications to treat hypertension, lipid disorders, and/or diabetes, and bivariate response variables were created for each condition—one each for hypertension and diabetes, three for lipid disorders.

Quality control included 14 blind duplicates and assessment of CVs. Established risk factors derived from blood measurements were used in data analyses—these factors already associated with BMI and risk of cardiovascular disease (CVD) and/or diabetes (Lewis et al., 2009; Wang et al., 2009). Measurements included (CV in parentheses) high density lipoprotein cholesterol (HDL cholesterol, 3.5%) and blood glucose (Gluc, 1.3%). Statistical associations involving diastolic blood pressure, total cholesterol, triglycerides, and hemoglobin A1c were examined in the course of analysis but are not reported because of the high degree of similarity with the three risk factors reported.

Statistical methods

All analyses used SAS software (Bailer et al., 2010). There were three types of exclusion: missing data (see Table 1 for variables), outlier data, and < 2 years of service. Of 464 participants, 34 had missing DEXA measurements, 16 missing clinical/laboratory data, 1 an outlying glucose, and 4 had < 2 years of service, leaving n = 409. Two factors drove the last exclusion: causal associations between DEXA measures and risk factors have latencies of years, and thus, these officers contribute no information to hypothesized associations; and the distribution of "years served as a police officer" was trimodal, the lowest mode < 2 years, thus associations risk being driven solely by modal membership—not biology.

Univariate and bivariate distributions were examined for age, weight and height, DEXA measures, and risk factors—all within joint sex and race strata. Bivariate associations between mass measurements (weight, fat-, and lean-) versus height or bone-mass revealed monotonic increasing relations within joint sex and race strata. Regression slopes were similar for White (n = 248) and Black (n = 54) males. Slopes were smaller for White (n = 75) and Black (n = 32) females, and imprecisely estimated due to small numbers.

The entire data set was used to derive DEXA indexes and to examine associations of BMI and DEXA indexes with risk factors, controlling for age, medication usage appropriate to the risk factor, and joint sex and race distributions. This strategy is equivalent to an analysis of covariance. It assumes that mass index/risk factor associations are the same across age and within joint sex and race strata and is sufficient to answer the limited question: Do DEXA partitions perform better than BMI to explain variation in risk factors? This question can be

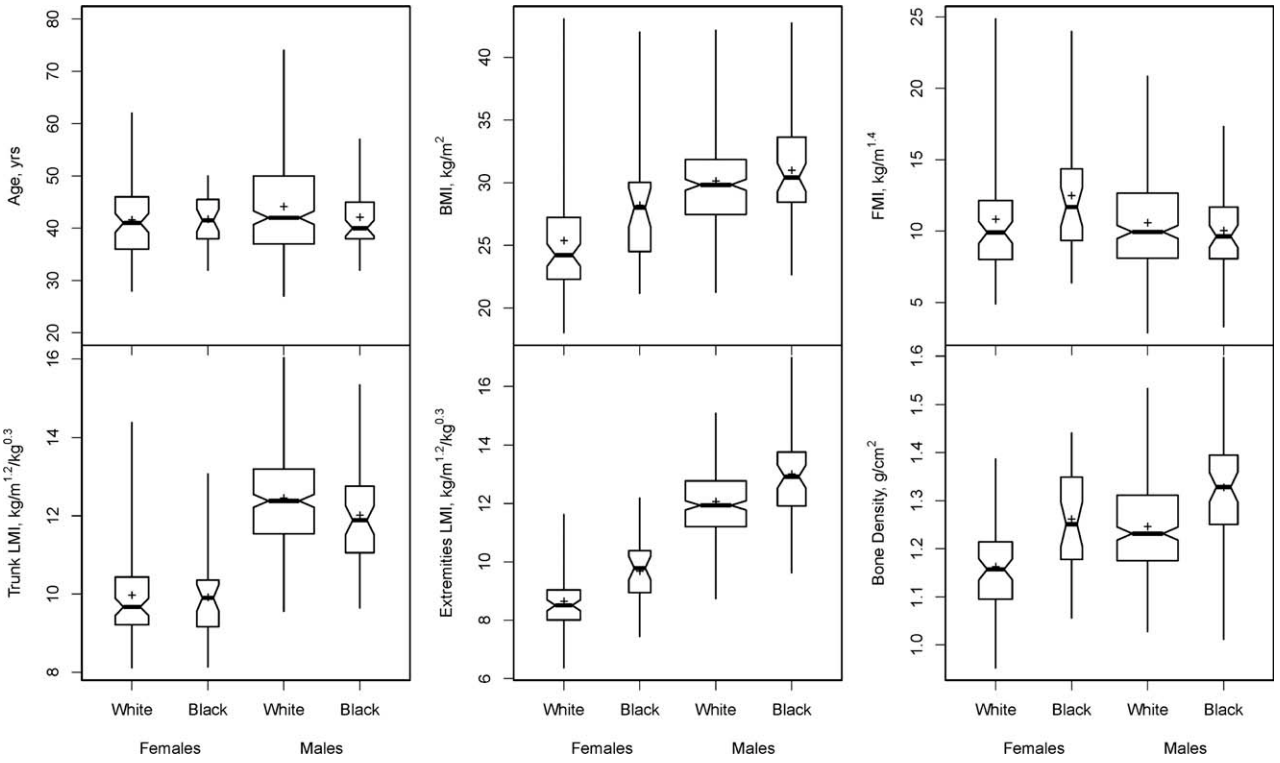

Fig. 1. Box-and-whisker plots of the distributions of age, BMI, FMI, LMI_t (trunk), LMI_e (extremities), and bone density among joint strata of sex and race (Whites and Blacks). Cross (+) denotes the mean; median is the horizontal line connecting vertices of the notch ⟩—⟨; Interquartile range (25th to 75th percentile) is denoted by lower and upper edges of box; ends of whisker lines, respectively, denote minimum and maximum values; area of box is proportional to $\sqrt{n}$. If corresponding notches do not overlap, the medians of two box plots are significantly different at approximately a 0.05 Type 1 error level.

recast as a hypothesis: DEXA partitions explain similar degrees of variance in risk factors as BMI—the null, versus DEXA partitions either explain less or more variance than BMI—the alternative.

Following suggestions that indexes better control for misclassification of obesity than BMI or percent fat-mass (Kelly et al., 2009), DEXA indexes were created for fat- and lean-mass. A fat-mass index (FMI) was derived from the regression of ln[FMI] versus ln[height]—ln refers to the natural logarithm—controlling for age, sex, and race. A coefficient of 1.4 was used to construct FMI: fat-mass (kg)/[height (m)]$^{1.4}$. A similar procedure for lean-mass revealed that ln[height] and ln[bone mass] independently correlated with ln[lean-mass]; thus, LMI = lean-mass (kg)/[height (m)]$^{1.2}$/[bone-mass (kg)]$^{0.3}$. Additional lean-mass indexes were calculated for trunk (LMI_t) and extremities (LMI_e) lean-mass, each derived from the appropriate mass divided by the aforementioned powers of height and bone-mass (justification below).

Two sets of statistical analyses were pursued. The first, intended to answer the question, "Do DEXA partitions perform better than BMI in explaining variation in risk factors," uses a partial F-test of nested multivariable regression models. The multiple R^2, a measure of explained variance, is the focus of these analyses; and four models were examined for each of the three reported risk factors as dependent variables. Model 1 includes sex, race, their interaction, and age; Model 2 adds BMI to Model 1; Model 3 adds FMI, LMI_t, and LMI_e to Model 1; and Model 4 adds BMI and the three DEXA indexes to Model 1. The partial F-test assesses the statistical significance of adding groups of variables to a more parsimonious model; the difference in magnitude of the R^2 is directly related to this test.

The second set of analyses reports magnitude of the adjusted regression coefficients, their 95% confidence limits (C.L.), and R^2s for two models of each reported risk factor as dependent variable. Model 1 contains sex, race, their interaction, age, and BMI; Model 2 contains the same demographic variables and the most parsimonious set of DEXA indexes which maximizes R^2 and produces statistically significant regression coefficients at a P-value of 0.10 or less.

Models containing LMI predicted risk factors with regression coefficients counter to expectation. Conventional wisdom might suggest that lean-mass reflects muscle mass and that people with relatively high lean-mass—high values of LMI—should have risk factor values predicting protection, e.g., high LMI would predict low blood glucose (Gluc). The opposite was noted, even in models adjusting for FMI, for all risk factors—reported and unreported. These observations led to a decision to partition LMI into trunk (LMI_t) and extremities (LMI_e). The same counter intuitive associations were noted for LMI_t, adjusted for FMI; however, all associations involving LMI_e, adjusted for FMI and LMI_t, were effectively zero.

RESULTS

Displayed as box-and-whisker plots, univariate statistical assessments within joint sex and race strata demonstrate similar distributions of age (Fig. 1; see legend for

TABLE 2. Explained variance and statistical significance[a] of four regression models[b] for systolic blood pressure (SBP), high density lipoprotein (HDL) cholesterol, and blood glucose (Gluc) from BMI[c] and DEXA indexes[c] among 409 police officers

	Dependent variable		
Model	SBP (mm Hg)	HDL (mg/dL)	Gluc (mg/dL)
1	0.1149	0.2704	0.3125
2	0.1497	0.3067	0.3412
$p_{1,2}$[a]	<0.0001	<0.0001	<0.0001
3	0.1669	0.3373	0.3502
$p_{1,3}$[a]	<0.0001	<0.0001	<0.0001
4	0.1696	0.3407	0.3575
$p_{2,4}$, $p_{3,4}$[a]	0.024 , 0.26	0.0002 , 0.15	0.018 , 0.03

[a]*P*-values ($p_{j,k}$) reflect a partial *F* test statistic: whether addition of covariates in the contrast of two models—one nested in the other—accounts for additional variance. See footnote "*b*" for explanation of models.
[b]Model 1: Race, sex, their interaction, age, medications; Model 2: Adds body mass index (BMI[c]) to Model 1; Model 3: Adds three DEXA indexes[c] to Model 1; Model 4: Adds BMI and three DEXA indexes to Model 1.
[c]BMI, body mass index (kg/m^2); FMI, fat mass index (kg/m$^{1.4}$); LMI_t, trunk lean mass index (kg/m$^{1.2}$/kg$^{0.3}$); LMI_e, extremities lean mass index (kg/m$^{1.2}$/kg$^{0.3}$). See text for definitions.

TABLE 3. Regression coefficients and explained variance (R^2) comparing BMI[a] to a parsimonius model[b] of DEXA index(es)[a] as predictors of systolic blood pressure (SBP), high densiy lipoprotein (HDL) cholesterol, or blood glucose (Gluc)

		Independent variables			
		Model 1		Model 2	
Risk factor	Parameter[a]	BMI[a]	versus	FMI[a]	LMI_t[a]
SBP (mmHg)	*B*	0.59		0.84	—
	95% C.L.	0.30 , 0.88		0.51 , 1.17	—
	R^2	0.15		0.17	—
HDL (mg/dL)	*B*	−0.74		—	−3.36
	95% C.L.	−1.05 , −0.42		—	−4.42 , −2.30
	R^2	0.31		—	0.34
Gluc (mg/dL)	*B*	0.52		0.34	1.29
	95% C.L.	0.28 , 0.77		−0.03 , 0.70	0.21 , 2.36
	R^2	0.34		0.35	

[a]BMI, body mass index (kg/m^2); FMI, fat mass index (kg/m$^{1.4}$); LMI_t, trunk lean mass index (kg/m$^{1.2}$/kg$^{0.3}$); *B*, regression coefficient; C.L., confidence limit. See text for definitions of indexes.
[b]Model 1: Sex, race, their interaction, age, medications, BMI. Model 2: Sex, race, their interactionm, age, medications, identified DEXA index(es).

how to assess statistical significance). DEXA results are consistent with well-established evidence that (1) Black women tend to have higher fat mass, (2) as measured by LMI_e, men have greater lean mass than women, and Blacks have greater lean mass than Whites, and (3) Blacks have higher bone density than Whites, and White women have the lowest bone density. These observations provide external validation of the DEXA measurements in BCOPS and also justify the decision to statistically adjust for sex and race as potential confounders.

Means, standard deviations, and correlations, adjusted for joint sex and race strata, are detailed in Table 1. The BMI is highly correlated with DEXA indexes, the smallest $r = 0.742$. Correlations involving bone density are small, and with risk factors, including those not reported, no correlation was statistically significant at $P \leq 0.05$. Multivariable data analyses predicting risk factors from bone density revealed no associations, nor perturbation of magnitudes of covariate coefficients thereby suggesting no confounding (results not shown).

In Table 2, the addition of BMI (Model 2) or the three DEXA indexes as a group (Model 3) to a model containing sex, race, medication, and age variables (Model 1) significantly increases explained variance (R^2) for all three risk factor dependent variables; but notably more so for the DEXA variables. In a path in which BMI is added to a model already containing the DEXA variables (Model 3), that addition adds no statistically significant additionally explained variance for SBP and HDL (Model 4, $p_{3,4}$), and about 0.7% ($P = 0.03$) for blood glucose. However, for the contrasting path in which the DEXA variables are added to a model already containing BMI (Model 2), additional explained variance of 1.6% to 3.6% is noted and is statistically significant for all three risk factors (Model 4, $p_{2,4}$).

The DEXA variable(s) which seem to be driving the association with a risk factor depends on the risk factor (Table 3). For SBP, it is FMI; for HDL cholesterol it is LMI_t; and for blood glucose, FMI and LMI_t have independent associations (Model 2). Regression coefficients for DEXA variables not in the model did not meet 0.1 levels of statistical significance; and for all risk factors including those unreported in this article, LMI_e never demonstrated a statistically significant relationship when LMI_t was jointly included (results not shown).

DISCUSSION

Within this sampling frame of police officers and a cross-sectional design, the generalized finding is that DEXA indexes of fat- and lean-mass, compared with BMI, perform better in explaining variation in cardiovascular and diabetes risk factors. This finding contributes to points of validation raised by a recent NHANES study that DEXA-derived morphological partitions may be better predictors of risk compared with BMI (Kelly et al., 2009). These findings relate directly to the use of DEXA technology over the use of the BMI to identify associations, which underlie general concepts of causation and population distribution. Goals related to diagnosis and risk assessment for an individual represent objectives which are not addressed by the analyses and results presented in this article.

Generalized findings suggest that fat-mass may account for increased risk of hypertension, and trunk lean-mass may account for increased risks associated with levels of HDL cholesterol. Both seem to play independent roles in their association with blood glucose.

Body mass, fat-mass, and lean-mass indexes

In establishing an index of weight, or mass, to height (kg/m^2), a power coefficient of 2 for height, while a standard practice for BMI, is not the most efficient—even for BMI; indexes based on the arbitrary use of a power coefficient of 2 continue to be correlated with height in many populations, contradicting the purpose of creating an index to remove the correlation with height (Diverse Populations Collaborative Group, 2005). A decision to derive the most efficient coefficients in the analyses reported herein was predicated on the desire to maximize use of information in a relatively small population of police officers ($N = 409$), thus removing the effects of stature to derive a more pure measure of adi-

posity and lean mass. For lean mass, removing the effects of stature also required taking into account bone-mass.

However, the magnitude of these derived power coefficients reflects an averaging procedure weighted by number across joint sex and race distributions unique to BCOPS. It may not be appropriate to use these coefficients in other populations or to generalize their use to specific sex and race groups. Although an argument can be marshaled for the simplicity of using a simple power coefficient of 2 for height, this argument does not address the reality that residual correlation can remain with height, nor does it address the fact that additional standardization is possible for lean mass by taking into account bone-mass.

Risk factor associations involving LMI and LMI_t seem counter-intuitive; the expectation is that a high lean-mass index is associated with risk protection. The opposite is noted, even when controlling directly for fat-mass via inclusion of FMI in joint models. This finding is parallel to a recent observation in CARDIA (Sood et al., 2011). Women who have asthma are noted to have a higher trunk lean-mass index, even after adjustment for fat-mass, compared with women without asthma. This is explained as, "DEXA-assessed 'lean' mass is not entirely fat-free but includes the smaller and highly metabolically active ectopic fat within the skeletal muscle and viscera (Sood et al., 2011)." The larger correlation of FMI with LMI_t ($r = 0.626$, Table 1), compared with LMI_e ($r = 0.452$), may reflect this confounding effect of ectopic fat, particularly within viscera.

Like CARDIA investigators who offer speculations related to metabolically active visceral fat and the role of cytokines in asthma (Sood et al., 2011), perturbed metabolism may be the underlying cause of observed findings in BCOPS. For example, HDL cholesterol plays a major role in reverse cholesterol transport to the liver—a trunk viscus (Ragbir and Farmer, 2010). However, it remains to be determined if variation in HDL explained by LMI_t would be related to such a process.

Multicollinearity: BMI and DEXA indexes

Inclusion of BMI and DEXA indexes in a model predicting a risk factor produces unstable and imprecise regression coefficients (results not shown). Inspection of bivariate correlations between BMI and FMI, LMIt, or LMIe (Table 1) indicates remarkably high values. This explains the marked instability of regression coefficients and inflation of standard errors when models contain combinations of these variables (results not shown). It is likely that in a different population, the correlations and statistics would be very different. It is conceivable that issues of measurement error—both construct validity, discussed later, and laboratory error—would create different multicollinear structures for another population and study design. For these reasons, it would seem inadvisable to construct prediction models which include BMI and DEXA indexes.

Construct validity

Preceding discussions about the meaning of associations between LMI_t and risk factors raises questions about the nature of the underlying association between DEXA indexes and risk factors.

If a mass index is being examined in its association with a disease, or an antecedent marker of disease—a risk factor, then the nature of such an association is better conceptualized as a metabolic feature of a population—not a morphological feature. For example, it is possible to be fat and fit, and variation in fitness within a constant level of "fat" can account for decreased cardiovascular risk (Li et al., 2006). Thus, at the same level of adiposity measured morphologically, variation in metabolic processes related to muscle metabolism and attendant fitness can exist. Thereby, protective effects related to risk is best conceptualized as metabolic.

By-and-large, however, fit people in a population tend not to be fat. Thus, variation in morphological measures does not fully capture the underlying metabolic variation driving risk relations; but, it does capture detectable degrees of metabolic variation and thereby function as a marker. Because joint metabolic features related to fitness level, diet, and hormonal effects are the underlying causes of disease, similar or conflicting relations between a mass index and disease among populations may be confounded by differences in the distribution of these underlying causes.

DEXA indexes are closer markers to underlying metabolic processes than BMI. DEXA-derived measures have identified changes in fat-mass associated with moving from an active to a sedentary life style in early adulthood, whereas BMI suggests little or no change (Hull et al., 2007). However, in cross-sectional observational studies of general populations, it may be that DEXA-derived partitions will not reveal marked quantitative improvements, compared with BMI, in relationships with risk factors—particularly for clinical purposes of diagnostic use (Sun et al., 2010). The BCOPS study of police officers, however, would suggest that quantitative improvements do exist.

There is a suggestion by The Emerging Risk Factors Collaboration that when established risk factors are available as predictors of incident CVD, then anthropometric measures may have little if any use in the prediction of incident CVD (Wormser et al., 2011). This inference extends from a similar nested-model data analysis strategy as used herein (Table 3), i.e., determine if addition of a class of variables—anthropometrics—offers additional "explanatory" value above-and-beyond a simpler model containing only risk factors in the prediction of incident CVD.

The purpose of the analysis reported in this study of police officers is predicated on a different logic than the Collaboration efforts: DEXA partitions are morphological constituents of total body mass. The Collaboration paper views risk factors as a mediating causal variable linking variation in anthropometrics to incident CVD. This logic precludes a suggestion that anthropometrics offer little as a measure of a public health or clinical state about which effective intervention strategies should be developed to prevent disease. In part, this is because the logic is different; but it also may be that, compared with risk factors, the anthropometrics of the Collaboration analysis are more limited by issues of measurement and construct validity.

CONCLUSIONS

From an epidemiological viewpoint, DEXA-based partitions suggest avenues to explore metabolic-based processes which are believed to relate to a partition's associations with disease risk. The value of this viewpoint lies with indirect assessment of biological mechanisms manifest at a population level and how to develop effective and measureable interventions to prevent disease—lose fat mass, gain muscle mass, and improve physical fitness.

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