Published in final edited form as:
J Am Geriatr Soc. 2012 April ; 60(4): 707–712. doi:10.1111/j.1532-5415.2011.03865.x.

Selective Contribution of Regional Adiposity, Skeletal Muscle and Adipokines to Glucose Disposal in Older Adults

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Abstract

Objectives—To study the relationships of muscle mass, regional adiposity, and adipokines with glucose disposal in an older population.

Design—Cross-sectional analysis.

Setting—Community-dwelling volunteers from the Baltimore Longitudinal Study of Aging.

Participants—280 men and 259 women, mean age of 71.1 ± 0.4 years (age range 55-96 years) with complete data on fasting plasma adiponectin and leptin, oral glucose tolerance test (plasma glucose available at t = 0, 20, 40, 60, 80, 100, and 120min), thigh CT, physical activity levels and anthropometric measures.

Measurements—Participants were classified into eight groups according to presence of global adiposity (BMI > 27 kg/m^2), central adiposity (waist circumference > 88 cm for women and > 102 cm for men), and low muscle mass (CT thigh, lowest sex-specific tertile [93.8 cm^2 in women and 110.7 cm^2 in men] of adjusted thigh muscle area). Linear regression models were used to estimate the contribution of these eight groups to early glucose area under the curve (AUC) (t = 0-40min), late glucose AUC (t = 60-120min), and total glucose AUC (t = 0-120min).

Results—Irrespective of muscle mass, individuals with a combination of central and global adiposity were more likely to have delayed glucose disposal rates (P <0.05). We also found a strong negative association between circulating adiponectin levels and glucose disposal rates (early AUC; β= -0.14, late AUC; β= -0.20, and total AUC; β= -0.20, P <0.05 for early, late and total glucose AUC) after adjusting for regional adiposity, muscle mass, circulating leptin levels, physical activity, age, and sex.

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Author Contributions RR: Study concept and design, analysis and interpretation of data and preparation of manuscript.
KSG: Study concept and design, analysis and interpretation of data.
EJM: Study concept and design, acquisition of subjects and/or data, analysis and interpretation of data.
JME: Acquisition of subjects and/or data and preparation of manuscript.
CWC and LF: Study concept and design, acquisition of subjects and/or data, analysis and interpretation of data and preparation of manuscript.

Conflict of Interest: The editor in chief has reviewed the conflict of interest checklist provided by the authors and has determined that the authors have no financial or any other kind of personal conflicts with this paper.

Disclosure: The authors have no relevant conflicts of interest to disclose.

Conclusion—Older individuals with global and central adiposity may be at a greater risk of glucose intolerance unrelated to low muscle mass.

Keywords

low muscle mass; regional adiposity; glucose disposal

INTRODUCTION

Aging is associated with a progressive decline of glucose tolerance.[1] It is still unclear whether this decline in glucose tolerance is directly due to aging per se or rather indirectly due to the age-related changes of muscle mass and fat regional distribution. Previous studies that addressed this question were limited by the lack of information on lean body or skeletal muscle mass, [2, 3] which may be problematic because changes in lean body mass and fat mass may not occur in parallel with aging. In some individuals, an accelerated decline in muscle mass with age accompanies a substantial increase in fat mass configuring the syndrome of "sarcopenic obesity".[4] In others, changes in fat mass are more variable; fat mass appears to increase, remain stable or even decline in different individuals and at different ages.[5] Furthermore, fat tends to deposit preferentially in the visceral compartment in some individuals, while it assumes a more global and diffuse pattern in others.[6] It is still not understood how these complex patterns of body composition affect glucose metabolism.

Adipose tissue and skeletal muscle may affect glucose homeostasis through different mechanisms. Adipocytes secrete adiponectin and leptin that directly influence glucose homeostasis. Leptin stimulates energy expenditure and inhibits food intake thereby preventing excess adiposity.[7] Adiponectin enhances insulin sensitivity, upregulates fatty acid oxidation as well as energy expenditure and reduces hepatic gluconeogenesis.[8] High leptin[9] and low adiponectin levels[10] are associated with obesity, insulin resistance and type 2 diabetes mellitus. Skeletal muscle accounts for about 85% of post-prandial insulin mediated glucose disposal, [11] and changes in muscle mass may affect glucose disposal and insulin sensitivity. Insulin resistance has been associated with decreased muscle strength in elderly subjects even after controlling for adiposity, regardless of a diagnosis of diabetes[12, 13] suggesting that age-related sarcopenia could indeed contribute to glucose intolerance in the elderly.

In spite of the potential contributions that age related changes in body composition may have on glucose tolerance, there is no study, to our knowledge, that has considered the collective effects of muscle mass, regional adiposity, and adipokines on glucose tolerance in the elderly. We tested the hypothesis that adipokines, muscle mass and different patterns of regional adiposity independently affect glucose disposal in older persons using cross-sectional data from the Baltimore Longitudinal Study of Aging (BLSA).

MATERIALS AND METHODS

Participants

The BLSA study is an ongoing observational study of normative aging in community-dwelling volunteers conducted at and sponsored by the US National Institute on Aging (NIA) since 1958. Participants undergo medical, physiological, and psychological examinations at regular intervals. The BLSA protocol was approved by the NIA Intramural Research Program and the Institutional Review Board of the MedStar Health Research Institute, Baltimore, Maryland. All participants provided informed participation consent at each visit.

We performed a cross-sectional analysis on data from 539 BLSA participants, aged 55 and older whose latest visit fell between January 2006 to October 2009 and had the following measures: fasting plasma adiponectin and leptin, oral glucose tolerance test (OGTT; with plasma glucose values available from t = 0, 20, 40, 60, 80, 100 and 120 minutes), computer tomography (CT) scan of the thigh, physical activity levels and anthropometric measures. A 2-hr 75 gram OGTT was performed on all participants after a 10-hr overnight fast: all participants on insulin or steroid treatment within 3 months prior to study visit were excluded.

Laboratory Measurements

Plasma glucose levels were measured using a glucose analyzer (Beckman Instruments, Brea, CA). Plasma leptin was measured using ELISA with inter-assay coefficient of variation (CV) of 2.6-6.2% and intra-assay CV of 2.6-4.6% (Millipore, Billerica, MA). Plasma total adiponectin was measured using radioimmunoassay with inter-assay CV of 6.9-9.3% and intra-assay CV variation of 1.8-6.2 % (Millipore, Billerica, MA).

Assessment of Glucose Disposal—Glucose disposal was assessed using the trapezoidal rule to determine the area under the curve (AUC) of the plasma glucose values during OGTT. A high glucose AUC represents delayed glucose disposal. Conversely, a low glucose AUC signifies more rapid glucose disposal. In addition to total glucose disposal (total glucose AUC, t = 0−120 min), we also evaluated early phase glucose disposal (early glucose AUC, t = 0–40 min) and late phase glucose disposal (late glucose AUC, t = 60–120 min). The plasma glucose response during the first 30 minutes of an OGTT has been associated with hepatic glucose uptake, and the decline in plasma glucose concentration 60 minutes after glucose ingestion primarily reflects glucose uptake by skeletal muscle.[14]

Anthropometrics

Body mass index (BMI) was calculated as weight (kg)/height^2 (m^2). Waist circumference (WC) was measured just below the rib cage, at the narrowest point where the waist tapers and the average of three measurements was used in the analysis.[15]

Determination of Global and Central Adiposity—Global adiposity was defined as BMI > 27 kg/m^2 for both men and women. Central adiposity, a surrogate for visceral obesity, was defined as waist circumference > 102 cm for men and > 88 cm for women, the same thresholds used by the National Cholesterol Education Program (NCEP) Expert Panel to define metabolic syndrome.[16]

Muscle Mass

A cross-sectional 10 mm CT image of the thigh was obtained from each participant at mid-femur (midpoint between the medial edge of the greater trochanter and the intercondyloid fossa in scout view image) using a Somatom Sensation 10 CT scanner (Siemens, Malvern, PA). The total mid-thigh cross-sectional area of non-adipose, non-bone tissue within the deep fascial plane was used as a proxy measure of muscle mass. The Geanie software (version 2.1, BonAlyse Oy, Jyvaskyla, Finland) was used to quantify the cross-sectional area (cm^2). This value was then divided by the subject's weight and normalized by the mean weight of the study population.

Determination of Low Muscle Mass—Low muscle mass was defined as the lowest sex-specific tertile (93.8 cm^2 in women and 110.7 cm^2 in men) of adjusted thigh muscle area. Partitioning muscle mass into tertiles allows for the exploration of muscle mass across

its entire spectrum and avoids focusing on single standard definitions of sarcopenia, an approach that has been widely criticized in the literature.[17, 18]

Definitions of body composition patterns

To study the independent effects of muscle mass and regional adiposity on glucose metabolism, participants were cross-classified according to global adiposity, central adiposity, and muscle mass into eight groups:

- Lean with normal muscle mass: BMI ≤ 27; WC ≤ 88cm for women and ≤ 102 cm for men; thigh muscle area > 93.8 cm^2 for women and > 110.7 cm^2 for men; (n = 203)
- Lean with low muscle mass: BMI ≤ 27; WC ≤ 88cm for women and ≤ 102 cm for men; thigh muscle area ≤ 93.8 cm^2 for women and ≤ 110.7 cm^2 for men; (n = 64)
- Central adiposity with normal muscle mass: BMI ≤ 27; WC > 88cm for women and > 102 cm for men; thigh muscle area > 93.8 cm^2 for women and > 110.7 cm^2 for men; (n = 21)
- Central adiposity with low muscle mass: BMI ≤ 27; WC > 88cm for women and > 102 cm for men; thigh muscle area ≤ 93.8 cm^2 for women and ≤ 110.7 cm^2 for men; (n = 23)
- Central and global adiposity with normal muscle mass: BMI > 27; WC > 88cm for women and > 102 cm for men; thigh muscle area > 93.8 cm^2 for women and > 110.7 cm^2 for men; (n = 93)
- Central and global adiposity with low muscle mass: BMI > 27; WC > 88cm for women and > 102 cm for men; thigh muscle area ≤ 93.8 cm^2 for women and ≤ 110.7 cm^2 for men; (n = 98)
- Global adiposity with normal muscle mass: BMI > 27; WC ≤ 88cm for women and ≤ 102 cm for men; thigh muscle area > 93.8 cm^2 for women and > 110.7 cm^2 for men; (n = 30)
- Global adiposity with low muscle mass: BMI > 27; WC ≤ 88cm for women and ≤ 102 cm for men; thigh muscle area ≤ 93.8 cm^2 for women and ≤ 110.7 cm^2 for men; (n = 7)

Physical Activity

Since physical activity levels may confound the association between body composition and glucose disposal, we included physical activity as a covariate in our analysis. Physical activity levels were assessed using the Leisure Time Physical Activity Questionnaire, a standardized, interviewer-administered instrument, supplemented with lower intensity activities commonly performed by older adults.[19] Total kilocalories expanded in stair climbing, walking and exercise activity per week (kcal/kg/hour) were used in this analysis.

Statistical Analysis

Distributions of plasma levels of adiponectin, leptin, and all markers of glucose disposal (early, late and total glucose AUCs) were highly skewed and were log transformed for data analysis. Linear regression models were used to estimate early glucose AUC, late glucose AUC, and total glucose AUC, across the eight groups as described above. Age, sex and physical activity levels were added into regression models as covariates. Data with normal distribution were presented as mean ± SD, and data with non-normal distribution were presented as median values (interquartile range). All analyses were performed using SPSS (version 17.0, SPSS Inc., Chicago, Illinois).

RESULTS

Baseline Characteristics

The mean age of the study population was 71.1 ± 0.4 years (age range 55-96 years). The mean and median BMI of the study population was 27.2 and 26.3 respectively; therefore, a BMI > 27 was set to define global adiposity. Of the 539 BLSA subjects, 280 (51.9 %) were men. Sixty of the 539 subjects have diabetes based on OGTT. Thirty nine of the 60 subjects are on medication for diabetes: 19 on metformin, 6 on dipeptidyl peptidase-4 (DPP 4) inhibitors, 8 on sulfonylureas, and 6 on thiazolidinediones.

The characteristics of the participants according to the eight different body composition groups are summarized in Table 1. Many participants (38 %) were considered lean with normal muscle mass, followed closely by those who were categorized as having both central and global adiposity (total 35.5 %). Interestingly, less than 10% of total subjects were considered to have either central or global adiposity alone. Participants classified in groups with low muscle mass were significantly older ($P < 0.05$ for all groups) than those with normal muscle mass. Therefore, all statistical comparisons between groups were age-adjusted. Within groups of regional adiposity, there were no differences in adiponectin associating with muscle mass. Within the groups with both central and global adiposity, and in both men and women, low muscle mass was associated with higher leptin.

Multivariable analysis

Table 2 summarized the multiple linear regression models estimating the independent contribution of the eight different body composition groups, leptin and adiponectin to different phases of glucose disposal (early, late and total glucose AUC) with the “lean with normal muscle mass” group serving as the referenced group. All analyses were adjusted for age, sex and physical activity.

The results showed no significant association between low muscle mass and glucose disposal in the lean group. Central adiposity by itself was also not associated with early, late or total glucose AUC, regardless of presence of low muscle mass. Interestingly, participants with both central and global adiposity had higher early, late and total glucose AUCs, and among them, those with low muscle mass had the highest effect on the three phases of glucose disposal (early glucose AUC: $\beta = 0.13$, $P < 0.05$; late glucose AUC: $\beta = 0.17$, $P < 0.05$: total glucose AUC: $\beta = 0.17$, $P < 0.05$). In addition, participants with global adiposity and normal muscle mass also have significantly higher early, late, and total glucose AUCs.

Within each respective model, adiponectin had a significant inverse relationship with and was one of the strongest predictors of early glucose AUC ($\beta = -0.14$, $P < 0.05$), late glucose AUC ($\beta = -0.20$, $P < 0.001$), and total glucose AUC ($\beta = -0.20$, $P < 0.001$). Age was also a strong predictor of late, and total glucose AUCs ($\beta = 0.18$, $P < 0.001$; $\beta = 0.14$, $P < 0.05$ respectively). Leptin was not a significant predictor of glucose disposal rates. To determine if this lack of association between leptin and glucose disposal was confounded by the presence of adiposity, we ran a separate backwards regression analysis (data not shown). We found a significant direct interaction between leptin and glucose disposal rates, which was attenuated when the two groups with both central and global adiposity remained in the model. Physical activity had a significant inverse relationship with early glucose AUC ($\beta = -0.10$, $P < 0.05$) but not with late or total glucose AUC.

DISCUSSION

In this study, we attempted to identify the relationship among adipokines, regional adiposity, and muscle mass with glucose disposal rates in older adults. We found evidence that

individuals with a combination of central and global adiposity were more likely to have delayed glucose disposal rates during an OGTT suggesting worsening glucose tolerance regardless of the presence of low muscle mass. We also found a strong negative association between adiponectin and glucose disposal rates after adjusting for regional adiposity, muscle mass, leptin, age, sex and physical activity levels.

A recent study found that sarcopenia exacerbated obesity-associated insulin resistance in the NHANES III population.[20] While we did not observe a strong effect of low muscle mass to glucose disposal rates in our study, we strengthened our analysis by further classifying adiposity into central and global adiposity to examine differences based on regional fat depots. We identified a subset of older people with both central and global adiposity who had delayed glucose disposal compared to subjects with central adiposity alone. The group with global adiposity with normal muscle mass also had reduced glucose disposal rates. The number of subjects in the group with global adiposity and low muscle mass was too small to provide any meaningful contribution to the model.

Our findings using early, late and total glucose AUCs revealed that these body composition traits adversely affect glucose disposal across the spectrum with no predilection for a specific glucose disposal phase. Age is a significant positive predictor of late and total glucose disposal rates after adjusting for body composition and adipokines. This is in agreement with the findings by Shimokata and colleagues who showed an age-dependent increase in 2-hour glucose values during OGTT.[21] Interestingly, physical activity was a significant negative predictor of early glucose AUC but not of late or total glucose AUC.

Leptin levels were found to be significantly higher in the central and global adiposity group with low muscle mass after controlling for age (Table 1). While we expect a higher leptin level in obesity, the added insult of low muscle mass appears to contribute to higher circulating leptin levels. This is in line with two recent studies that reported a correlation with raised leptin levels and low muscle mass in healthy elderly people, after adjusting for adiposity.[22, 23] A significant inverse-relationship between adiponectin and glucose disposal rates was found after controlling for age, sex, leptin, muscle mass, physical activity and adiposity. An unexpected finding was the lack of an association between leptin levels and glucose disposal rates after controlling for the same covariates. This suggests that leptin may be dependent on a combination of factors in its relationship with glucose disposal. Baratta and colleagues found that insulin sensitivity measured by euglycemic clamp studies strongly correlated with both fasting adiponectin and leptin, but only adiponectin remained significant after adjusting for age, gender, BMI and waist circumference.[24]

There are clinical implications from our findings. In our analysis, we were able to identify a sizable number of elderly subjects who not necessarily had both central and global adiposity. Forty-four (16.2%) of the 272 subjects were categorized as having central adiposity and 37 subjects (13.6%) were categorized as having global adiposity. Previous studies have shown that visceral fat is more predictive of insulin resistance, type 2 diabetes [25] and cardiovascular disease.[26] We found that subjects with a combination of both central and global adiposity had significantly delayed glucose disposal. Therefore, by using a single measure of BMI or WC, there is a possibility of ignoring risks associated with specific regional fat distributions. Thus, in the elderly, a comprehensive assessment may require the use of more than one measurement to determine regional adiposity and stratify metabolic risk.

Our study has limitations. Firstly, we might be underpowered to look at the contributions of the groups with central or global adiposity alone because of the small sample size. Secondly, a single slice of CT mid-thigh image might not be representative of the muscle mass of the

whole body. Similarly, the use of waist circumference as a measure of central adiposity can be questioned due to its inability to differentiate subcutaneous from visceral fat. In addition, oral glucose tolerance test is used as a measure of glucose disposal without taking into account insulin levels. Therefore, no inference to insulin resistance can be made. Finally, the cross-sectional nature of our study does not allow for assessment of cause-effect relationships.

In conclusion, our study results indicate that elderly subjects with a combination of central and global adiposity were more likely to have worse glucose tolerance, and the presence of low muscle mass did not profoundly magnify this relationship. With the projected increases in lifespan and incidence of obesity in older adults, it may help to identify measures aimed at quantifying specific regional adiposity patterns as potential risk factors of age related glucose impairment.

Acknowledgments

This work was supported entirely by the Intramural Research Program of the National Institutes of Health, National Institute on Aging.

Sponsor's Role: None

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Table 1

Baseline characteristics of the study population according to the eight different body composition groups (n=539)

	LEAN		CENTRAL ADIPOSITY ONLY		CENTRAL AND GLOBAL ADIPOSITY		GLOBAL ADIPOSITY ONLY	
	Normal MM	Low MM	Normal MM	Low MM	Normal MM	Low MM	Normal MM	Low MM
Men/Women	107/96	39/25	5/16	8/15	51/42	46/52	20/10	4/3
% of total population	37.7	11.9	3.9	4.3	17.3	18.2	5.6	1.3
Age, yrs	70.1 ± 0.7	80.1 ± 1.2 †	68.9 ± 1.8	75.6 ± 1.7 *	67.1 ± 0.8	71.7 ± 0.9 †	66.1 ± 1.6	75.7 ± 2.1 ‡
BMI, kg/m^2	23.6 ± 0.2	24.1 ± 0.2	25.7 ± 0.2	25.3 ± 0.2	31.2 ± 0.3	32.9 ± 0.4 †	28.8 ± 0.3	28.0 ± 0.4
Weight, kg								
Women	59.1 ± 0.7	61.7 ± 1.1 *	70.1 ± 1.2	66.9 ± 1.3	81.8 ± 1.3	89.1 ± 1.5 †	74.8 ± 1.9	73.9 ± 2.5
Men	74.9 ± 0.7	74.6 ± 1.1 *	81.5 ± 1.2	82.3 ±1.6	96.0 ± 1.3	100.9 ± 2.0 ‡	87.0 ± 1.2	86.2 ± 3.8
Height, cm								
Women	162 ± 0.6	162 ± 1.1	165 ± 1.7	163 ± 1.8	162 ± 0.8	163 ± 0.9	161 ± 1.6	164 ± 2.4
Men	175 ± 0.7	175 ± 1.2 *	177 ± 1.0	178 ± 1.5	175 ± 0.9	177 ± 1.0 *	174 ± 1.3	174 ± 4.9
Waist Circumference, cm								
Women	77.1 ± 0.6	79.8 ± 1.2	91.6 ± 0.8	91.4 ± 0.5	96.9 ± 1.0	101.9 ± 1.1 *	84.7 ± 0.6	83.6 ± 2.3
Men	91.7 ± 0.5	93.7 ± 0.9	105.5 ± 0.9	107.1 ± 1.3	109.5 ± 0.7	113.2 ± 1.2 *	97.8 ± 0.8	98.7 ± 0.7
Muscle Mass,cm^2								
Women	115.5 ± 1.3	87.5 ± 1.6 †	107.1 ± 3.1	84.6 ± 1.4 †	109.1 ± 1.7	81.8 ± 1.2 †	105.8 ± 3.2	90.5 ± 1.6
Men	132.8 ± 1.5	99.5 ± 1.9 †	119.9 ± 2.3	94.9 ± 6.8 *	124.2 ± 1.6	96.8 ± 1.6 †	134.5 ± 4.2	103.7 ± 2.7 *
Adiponectin, µg/mL								
Women	17.4 (11.4-29.0)	16.6 (9.9-24.7)	14.1 (8.5-20.0)	15.3 (9.8-17.5)	8.7 (4.2-16.2)	10.4 (7.3-14.9)	12.5 (7.1-22.8)	11.2 (9.1-27.4)
Men	8.8 (5.1-14.8)	17.8 (7.1-27.0)	6.0 (1.8-19.9)	14.8 (5.3-26.6)	7.0 (3.6-15.0)	7.2 (5.3-12.0)	7.9 (4.4-11.2)	11.0 (7.1-25.8)
Leptin, ng/mL								
Women	12.0 (7.7-20.6)	15.2 (9.4-22.3)	25.4 (17.3-33.6)	31.9 (25.8-48.1)	29.6 (22.9-43.7)	53.8 ‡(33.9-73.2)	12.6 (8.8-42.5)	38.6 (16.6-47.8)
Men	5.4 (3.7-9.1)	7.0 (4.0-12.9)	10.7 (10.1-16.3)	7.9 (5.9-14.7)	13.2 (8.6-21.9)	19.7 * (11.1-31.1)	9.2 (6.5-15.8)	11.5 (5.6-24.4)
Fasting Glucose, mg/dl	89.8 ± 0.9	89.4 ± 1.1	89.5 ± 2.7	75.6 ± 1.7	96.5 ± 1.6	99.1 ± 2.1	101.5 ± 5.8	102.1 ± 9.0
Early AUC, mg*hr/dl	81.0 (73.3-90.2)	79.7 (73.2-87.5)	79.0 (73.8-86.6)	86.6 (77.0- 93.3)	86.6 (77.6-99.4)	85.8 (79.8- 95.6)	87.4 (77.3-97.2)	83.3 (77-91.3)
Late AUC, mg*hr/dl	125.2 (101.3-156.7)	136.8 (119.1-164.4)	125.2 (102.0-161)	131.7 (109.0- 187.2)	143.5 (116- 182)	153.3 (122-188)	151.1 (111.3-199.6)	119.5 (91.2-126.3)

	LEAN		CENTRAL ADIPOSITY ONLY		CENTRAL AND GLOBAL ADIPOSITY		GLOBAL ADIPOSITY ONLY	
	Normal MM	**Low MM**	**Normal MM**	**Low MM**	**Normal MM**	**Low MM**	**Normal MM**	**Low MM**
Total AUC, mg*hr/dl	257.2 (216.2-301.2)	272.3 (237.2-301.6)	260.3 (216.2-297.8)	261.7 (235.5-344.2)	279.2 (241- 340.3)	294.4 (251.8- 341.4)	283.0 (234.0-357.3)	253 (198-301.3)
Physical Activity, kcal/week	1371 (765-2487)	1022 (378-1794)	1682 (547-2729)	730 (233-2205)	1539 (432-3112)	812 * (202-2042)	1157 (704-2624)	497 (173-1814)

Data expressed as mean ± SE or median (interquartile range); All variables have age-adjusted mean and median; MM: Muscle Mass, BMI: Body Mass Index, AUC: Area under the curve;

*$P < 0.05$,

†$P < 0.001$ and

‡$P = 0.001$ for comparisons between normal and low MM.

J Am Geriatr Soc. Author manuscript; available in PMC 2013 April 1.

Table 2

Linear Regression Models estimating Early, Late and Total glucose disposal.

	Early Glucose AUC		**Late Glucose AUC**		**Total Glucose AUC**	
	β	***P***	**β**	***P***	**β**	***P***
Lean + Low Muscle Mass (n = 64)	-0.05	0.26	0.02	0.77	-0.002	0.96
Central adiposity + Normal Muscle Mass (n = 21)	-0.01	0.70	0.02	0.79	0.01	0.83
Central adiposity + Low Muscle Mass (n = 23)	0.04	0.40	0.04	0.38	0.04	0.33
Central + Global adiposity + Normal Muscle Mass (n = 93)	**0.11**	**0.03**	**0.13**	**0.01**	**0.14**	**0.01**
Central + Global adiposity + Low Muscle Mass (n = 98)	**0.13**	**0.02**	**0.17**	**0.002**	**0.17**	**0.002**
Global adiposity + Normal Muscle Mass (n = 30)	**0.09**	**0.04**	**0.12**	**0.01**	**0.12**	**0.01**
Global adiposity + Low Muscle Mass (n = 7)	0.03	0.54	-0.05	0.27	-0.03	0.47
Adiponectin	**-0.14**	**0.003**	**-0.20**	**< 0.001**	**-0.20**	**< 0.001**
Leptin	0.07	0.23	0.04	0.52	0.04	0.46
Age	0.03	0.32	**0.18**	**< 0.001**	**0.14**	**0.003**
Sex (women = 0)	**0.16**	**0.002**	0.08	0.13	**0.11**	**0.04**
Physical Activity	**-0.10**	**0.03**	-0.07	0.09	-0.08	0.07
Adjusted R^2	**0.09**		**0.11**		**0.11**	

β represents standardized coefficients; AUC: Area Under the Curve