Genetics and Molecular Biology, 35, 3, 575-582 (2012)
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Research Article

Association of serum lipid components and obesity with genetic ancestry in an admixed population of elderly women

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Abstract

The prevalence of metabolic disorders varies among ethnic populations and these disorders represent a critical health care issue for elderly women. This study investigated the correlation between genetic ancestry and body composition, metabolic traits and clinical status in a sample of elderly women. Clinical, nutritional and anthropometric data were collected from 176 volunteers. Genetic ancestry was estimated using 23 ancestry-informative markers. Pearsons correlation test was used to examine the relationship between continuous variables and an independent samples *t*-test was used to compare the means of continuous traits within categorical variables. Overall ancestry was a combination of European (57.49%), Native American (25.78%) and African (16.73%). Significant correlations were found for European ancestry with body mass index (r = 0.165; p = 0.037) and obesity (mean difference (MD) = 5.3%; p = 0.042). African ancestry showed a significant correlation with LDL (r = 0.159, p = 0.035), VLDL (r = -0.185; p = 0.014), hypertriglyceridemia (MD = 6.4%; p = 0.003) and hyperlipidemia (MD = 4.8%; p = 0.026). Amerindian ancestry showed a significant correlation with triglyceride levels (r = 0.150; p = 0.047) and hypertriglyceridemia (MD = 4.5%; p = 0.039). These findings suggest that genetic admixture may influence the etiology of lipid metabolism-related diseases and obesity in elderly women.

Key words: dyslipidemia, genetic ancestry, lipid profile, obesity, postmenopausal women.

Received: January 26, 2012; Accepted: May 14, 2012.

Introduction

Obesity and lifestyle factors are the main contributors to an increased risk of chronic non-communicable (non-transmissible) diseases, especially type 2 diabetes (T2D), lipid disorders, hypertension and other metabolic-related comorbidities (Sullivan *et al.*, 2005). Many of these conditions may ultimately lead to cardiovascular disease. In the elderly, the risk of these disorders increases progressively with age (Wilson and Kannel, 2002; Cabrera *et al.*, 2007). As ageing progresses, overweight increases the risk for cardiovascular disease, mostly in women with high-risk LDL and HDL cholesterol profiles (Perissinotto *et al.*, 2002). As the incidence of these diseases increases worldwide there is an ever greater need to study the underlying genetic factors.

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Recent findings from genome-wide association studies have identified several loci associated with chronic metabolic and cardiovascular diseases. However, the allelic architecture that can explain the heritability accounting for these complex phenotypes is not well understood. Informative single nucleotide polymorphisms (SNPs) have been used to characterize the structure of the main continental populations, and the data indicate that ancestry estimates are the most important correction factors in genetic association studies, especially in studies using admixed populations such as those of Latin America (Bonilla *et al.*, 2004a; Tsai *et al.*, 2005; Gentil *et al.*, 2007; Moreno Lima *et al.*, 2007; Gentil *et al.*, 2009; Lai *et al.*, 2009; Ruiz-Narvaez *et al.*, 2010). Consequently, the clinical application of genetic risk markers may not be straightforward across different ethnic populations and may be vulnerable to spurious allelic association in genetic studies (Tsai *et al.*, 2005).

The risk factors for metabolic syndrome and other phenotypes related to postmenopausal biology have dis-

tinct frequencies among populations of different ethnic backgrounds (Deurenberg *et al.*, 1998; Wulan *et al.*, 2010). Genetic studies of admixed populations have substantially increased our knowledge of various genetic diseases through the detection and correction for population stratification (Bonilla *et al.*, 2004a; Lai *et al.*, 2009; Ruiz-Narvaez *et al.*, 2010).

Most genetic studies of metabolic phenotypes have investigated adult and middle-aged populations, with studies on elderly women being underrepresented. This group is more susceptible to chronic illnesses (Perissinotto *et al.*, 2002; Wilson and Kannel, 2002; Cabrera *et al.*, 2007) and is the fastest growing population group in developing countries (Nobrega *et al.*, 2009). The aim of the present study was therefore to investigate in a sample of elderly Brazilian women the contribution of admixture to several body composition measurements, metabolic traits and parameters of clinical status based on their correlation with genetic ancestry, estimated by ancestry-informative markers.

Subjects and Methods

Subjects

This cross-sectional study was done using data obtained from apparently healthy female outpatients from the low-income suburbs of Brasília, Brazil, who volunteered to undergo health screenings and medical, nutritional and/or pharmacological interventions as part of a larger research project entitled the Elderly Health Promotion Project (EHPP). Additional characterization of the subjects is presented elsewhere (Paula *et al.*, 2010; Nobrega *et al.*, 2011). Project eligibility criteria consisted of being female, aged 60 years or older, completing the clinical course of assessment for hypertension, diabetes and dyslipidemia, and recording and informing their dietary profile. Since clinical data and laboratory results were obtained more than once, only the most recent data collected prior to any health intervention (especially blood pressure therapy) were considered. All volunteers provided written informed consent and the research protocol was approved by the Catholic University of Brasília Ethics Committee.

Data collection

Venous blood samples were collected in EDTA-containing tubes after 12 h overnight fast. Laboratory tests involved routine clinical analyses with reagents from Boehringer-Mannheim (Germany). Low-density lipoprotein (LDL) was quantified by using Friedewald's formula. During consultation, blood pressure was measured after at least a 10 min rest in a sitting position. The blood pressure value of each subject was the mean of three physician-obtained measurements recorded 3 min apart. The body mass index (BMI = weight (kg)/height2 (m2)) of each patient was calculated by measuring body weight (kg) while the subject was wearing light clothing, and the subject's height (m), without shoes, was assessed to the nearest millimeter. Obesity was defined as a BMI = 30 kg/m2. Body composition (fat and fat-free soft tissue) was measured using a dual-energy X-ray absorptiometer (DXA; Lunar DPX-IQ model, software version 4.7e, Lunar Radiation Corp., Madison, WI, USA) according to standard procedures provided by the manufacturer.

Each subject was classified based on the consensus of Brazilian Medical Societies, particularly the 5th Brazilian Guidelines in Arterial Hypertension (Diretrizes, 2007) and the 4th Brazilian Guidelines for Dyslipidemia and Atherosclerosis Prevention (Sposito *et al.*, 2007). Briefly, hypertension was defined as a systolic blood pressure ≥ 130 mm Hg or a diastolic blood pressure ≥ 85 mm Hg, or the current use of antihypertensive medication. Type 2 diabetes mellitus (DM) was characterized by fasting blood glucose ≥ 126 mg/dL or the current use of insulin or oral anti-diabetic drugs. Glucose intolerance included DM cases and any cases with a fasting blood glucose ≥ 110 mg/dL. Hypertriglyceridemia was defined as triglyceride levels ≥ 150 mg/dL, and hypercholesterolemia was defined as total cholesterol ≥ 200 mg/dL or LDL-cholesterol ≥ 130 mg/dL. The current use of any anti-lipemic medication was considered to represent the presence of both entities. Hyperlipidemia was defined as isolated or combined hypertriglyceridemia and hypercholesterolemia, whereas dyslipidemia encompassed all cases of hyperlipidemia with or without a HDL-cholesterol < 50 mg/dL.

The study design included an estimation of fat consumption based on each subject's natural, *ad libitum* food intake. Dietary analysis was performed based on an average 3-day food intake (two weekdays and one weekend day) recorded by the patient at home. Clinical dietitians and assistants instructed each patient on how to record food intake by defining the size and number of each serving. The forms were returned at a clinical interview during which the amounts and qualities of food were checked for completion and missing data were collected. Diet composition was calculated using Diet-Pro® software, version 4.0 (A.S. Sistemas, Viçosa, MG, Brazil). Software was set for all food tables and completed with Philippi's composition table for local food (Philippi, 2002). Total lipid was expressed as the percentage of total energy. To construct categorical variables of fat consumption, intakes were classified into two groups: those with a normal lipid diet (10%-35%) and those with a hyperlipid diet (> 35%).

Marker selection and genotyping

For individual genetic ancestry estimation, we selected 23 ancestry-informative markers (AIMs) that displayed differential allele frequencies among European, African and Amerindian parental populations (Fernandez *et al.*, 2003; Bonilla e *et al.*, 2004b; Smith *et al.*, 2004; Shriver *et al.*, 2005). Some SNPs were previously used in

cross-sectional studies to correct for population structure (Gentil *et al.*, 2007, 2009; Moreno Lima *et al.*, 2007) and the informativeness of these SNPs for a three hybrid population panel has been evaluated in a Brazilian population (Lins *et al.*, 2010). The AIMs were genotyped using a modified, single-base extension protocol, as previously described (Lins *et al.*, 2007). Briefly, genomic DNA was extracted using a modified salting-out method and amplified by the polymerase chain reaction (PCR). Products were treated with Exo I/SAP enzymes and genotyping was done by single-base extension using the SNaPshot Multiplex System (Applied Biosystems, Foster City, CA), followed by capillary electrophoresis on an ABI Prism 3130XL genetic analyzer. The data were analyzed using GeneMapper software (Applied Biosystems).

Statistical analysis

Individual genetic ancestry was estimated using an algorithm based on maximum likelihood estimation (MLE) (Tsai *et al.*, 2005) that uses allele frequencies from the parental populations (European – EUR, African – AFR and Native American – AMR). Allelic frequencies were retrieved via genomic mapping in multiple panels of unrelated ethnic samples (Fernandez *et al.*, 2003; Bonilla *et al.*, 2004b; Shriver *et al.*, 2005).

The Kolmogorov-Smirnov test was used to check for normal distribution and Pearsons correlation test was applied to each continuous variable: age, weight, height, body mass index (BMI), fat mass (FM), fat-free mass (FFM), triglycerides (TG), total lipids (TL), total low-density lipoprotein (LDL), very-low density lipoprotein (VLDL), high-density lipoprotein (HDL), fasting plasma glucose (GLU), systolic blood pressure (SBP), diastolic blood pressure (DBP), dietary intake of total lipids (LIP), European ancestry proportion (EUR), African ancestry proportion (AFR) and Native American, or Amerindian, ancestry proportion (AMR). Cases were labeled as positive (affected) or negative (unaffected) with regard to metabolic and clinical features, including medication status determined from medical records, to assemble categorical variables for each metabolic disorder or trait (obesity, hypertriglyceridemia, hypercholesterolemia, hyperlipidemia, dyslipidemia, glucose intolerance, type 2 diabetes, hypertension and normolipid diet). An independent samples *t*-test was used to compare the means of continuous traits across carriers and non-carriers of the aforementioned metabolic disorders. Linear regression analysis was used to assess possible covariance for ancestry estimates and the variable rendered as interferential was later included in a covariance analysis with Bonferroni adjustments to correct for its effects. A value of $p < 0.05$ was considered statistically significant for all tests. The software SPSS, version 13 (SPSS Inc., Chicago, IL) was used for statistical calculations.

Results

The Kolmogorov-Smirnov test showed that the data for all continuous traits were normally distributed. The quantitative and categorical traits of the subjects are summarized in Table 1 and Table 2, respectively.

The allelic frequencies of the AIMs in the sample population differed from those reported for reference ancestral populations, thereby indicating the admixture struc-

Table 1 - Continuous variables in the studied population (n = 176).

Variable	Mean	SD	Min	Max
Age (years)	68.31	5.77	58.00	83.00
Weight (kg)	63.21	9.58	32.00	89.00
Height (m)	1.52	0.06	1.38	1.72
Body mass index (kg/m^2)	27.28	4.04	14.61	37.69
Fat mass (%)	37.28	6.84	8.05	57.74
Fat free mass (%)	58.88	6.41	39.80	87.00
Triglycerides (mg/dL)	157.24	97.11	38.00	967.00
Total lipids (mg/dL)	744.73	143.26	218.00	1117.00
Total cholesterol (mg/dL)	233.24	43.07	126.00	343.00
LDL (mg/dL)	139.93	39.58	43.00	250.00
VLDL (mg/dL)	29.84	13.12	7.60	76.00
HDL (mg/dL)	61.65	10.07	35.00	92.00
Fasting glucose (mg/dL)	104.33	29.67	70.00	324.00
SBP (mmHg)	136.42	26.54	70.00	210.00
DBP (mmHg)	81.16	15.74	40.00	120.00
LIP (%)	34.31	5.15	19.09	65.52
EUR (%)	57.49	17.21	2.00	100.00
AFR (%)	16.73	14.73	0.00	80.00
AMR (%)	25.78	14.50	0.00	65.00

AFR = African ancestry; AMR = Amerindian ancestry; DBP = diastolic blood pressure; EUR = European ancestry; HDL = high density lipoprotein; LDL = low density lipoprotein; LIP = lipid dietary intake; Max = maximum; Min = minimum; SD = standard deviation; SBP = systolic blood pressure; VLDL = very low density lipoprotein.

Table 2 - Categorical variables of the clinical and nutritional assessments in the sampled population.

Trait	Unaffected		Affected	
	n	%	n	%
Obesity	85	48.3	91	51.7
Hypertriglyceridemia	92	52.3	84	47.7
Hypercholesterolemia	41	23.3	135	76.7
Hyperlipidemia	103	58.5	73	41.5
Dyslipidemia	23	13.1	153	86.9
Normal lipid diet	59	33.5	117	66.5
Glucose intolerance	134	76.1	42	23.9
Type 2 diabetes	152	86.4	24	13.6
Hypertension	29	16.5	147	83.5

ture (Table 3). The distribution of the individual ancestry estimates (IAE) comprised a wide range of ancestry proportions in a diverse three-hybrid pattern of variation (Figure 1). The sample population displayed a genetic constitution with a marked contribution from European ancestry (57.49%) followed by Native American (25.78%) and African (16.73%) ancestries. Fewer individuals displayed a di-hybrid ancestral arrangement, *e.g.*, EUR-AFR or EUR-AMR.

When ancestry proportions were used as continuous traits, significant results were found for the European ancestry proportion, which was positively correlated with weight (r = 0.157, p = 0.037) and BMI (r = 0.165, p = 0.028), and negatively correlated with DBP (r = -0.187, p = 0.013). For the African ancestry proportion, there was a positive significant correlation with LDL (r = 0.159, p = 0.035) and a negative correlation with VLDL (r = -0.185; p = 0.014). For the Native American ancestry

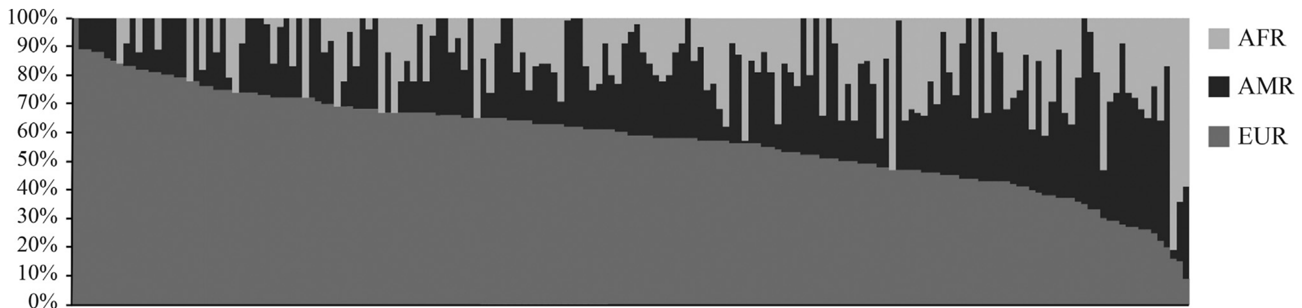

Figure 1 - Individual ancestry estimates for the sampled, admixed population. Each bar represents an individual subject and each color represents the corresponding relative ancestry proportion in relation to a parental population (light gray = African, dark gray = Amerindian and gray = European).

Table 3 - Ancestry informative markers with chromosomal positions and allele frequencies in Europeans (EUR), Africans (AFR), Amerindians (AMR) and the sampled population.

Locus	Position	Allele	EUR	AFR	AMR	Sample	Reference
FY-null (rs2814778)	1q23	C	0.006	0.983	0.018	0.209	Smith *et al.* (2004)
rs1129038	15q13	C	0.224	0.996	0.983	0.783	Smith *et al.* (2004)
rs3796384	3p14	C	0.154	0.783	0.875	0.626	Smith *et al.* (2004)
rs1480642	6q23	C	0.993	0.121	0.621	0.791	Smith *et al.* (2004)
rs1871534	8q24.3	C	0.981	0.071	1.000	1.000	Smith *et al.* (2004)
rs267071	5q22	C	0.654	0.088	1.000	0.421	Smith *et al.* (2004)
rs4280128	13q22	A	0.357	0.941	0.034	0.494	Smith *et al.* (2004)
rs6034866	20p12	A	0.083	0.954	0.143	0.100	Smith *et al.* (2004)
rs7349	10p11.2	A	0.067	0.969	0.000	0.221	Smith *et al.* (2004)
rs803733	9q33	C	0.880	0.013	0.410	0.636	Smith *et al.* (2004)
rs1426654	15q21	C	0.013	0.967	0.931	0.291	Smith *et al.* (2004)
TSC1102055 (rs2065160)	1q32	C	0.088	0.487	0.875	0.231	Smith *et al.* (2004)
rs730570	14q32	A	0.896	0.197	0.054	0.646	Smith *et al.* (2004)
rs1240709	1p36.3	A	0.766	0.050	0.103	0.526	Smith *et al.* (2004)
rs2278354	5p15.2	T	0.120	0.703	0.839	0.713	Smith *et al.* (2004)
rs803733	9q33	C	0.880	0.013	0.410	0.636	Smith *et al.* (2004)
rs1980888	9q22	T	0.100	0.143	0.950	0.204	Shriver *et al.* (2005)
rs1415878	Xq26	A	0.917	1.000	0.025	0.708	Shriver *et al.* (2005)
CYP3A4 (rs2740574)	7q22	G	0.040	0.800	0.040	0.211	Fernandez *et al.* (2003)
AT3 (rs3138521)	1q25	Insertion	0.282	0.858	0.061	0.429	Bonilla *et al.* (2004a)
WI-11153 (rs17203)	3p12.1	G	0.171	0.785	0.805	0.621	Bonilla *et al.* (2004a)
MID-93 (rs16383)	22q13.2	A	0.220	0.739	0.895	0.391	Bonilla *et al.* (2004a)
RB (rs2252544)	13q14	C	0.320	0.930	0.180	0.253	Bonilla *et al.* (2004a)

proportion, there was a positive correlation only with triglyceride levels (r = 0.150; p = 0.047). The correlation between variants other than ancestry also yielded significant associations and are shown in Table S1 (Supplementary Material).

Regarding the affected status, the mean difference (*MD*) of the African ancestry proportion was significantly different for hypertriglyceridemia (*MD* = 6.4%, p = 0.004) and hyperlipidemia (*MD* = 4.8% p = 0.026) (Figure 2). The contribution of European ancestry was significantly different for obesity (*MD* = 5.3%, p = 0.042) and the Native American ancestry for hypertriglyceridemia (*MD* = 4.5%, p = 0.039). The mean differences for traits other than those previously described also yielded significant associations and are shown in Table S2 (Supplementary Material). Given that lipid- and obesity-related variables yielded a significant association with ancestry, linear regression analysis used to select these phenotypes as possible covariates for African, Native American and European associations. These analyses revealed that SBP and DBP could be included as covariates for the African ancestry proportion (SBP β coefficient = -0.319, p = 0.014 and DBP β coefficient = 0.389, p = 0.003) and for the European ancestry proportion (SBP β coefficient = 0.256, p = 0.042 and DBP β coefficient = -0.404, p = 0.002). No potential predictors were identified for the Native American ancestry.

There was no divergence in the mean difference of the African ancestry proportion in the category of hypertriglyceridemia for the corrected model with SBP and DBP as covariates. For hyperlipidemia, an increase of 0.1% occurred in the corrected model for the African ancestry proportion (*MD* from 4.8% to 4.9%, p = 0.034). Using blood pressures as covariates for obesity status, the mean difference of the European ancestry levels among groups increased from 5.3% (p = 0.042) to 6.5% (p = 0.011). No other association was found for covariate-corrected models.

Discussion

The present study identified significant correlations between genetic ancestry and lipid profiles and a fluctuation in the metabolic parameters in a heterogeneous and admixed group of elderly Brazilian women. Interestingly, population ancestry estimates were relatively higher for the Native American and slightly lower for the African and European ancestry proportions than those in the general Brazilian population (Lins *et al.*, 2010) or in other elderly samples (Gentil *et al.*, 2007, 2009; Moreno Lima *et al.*,

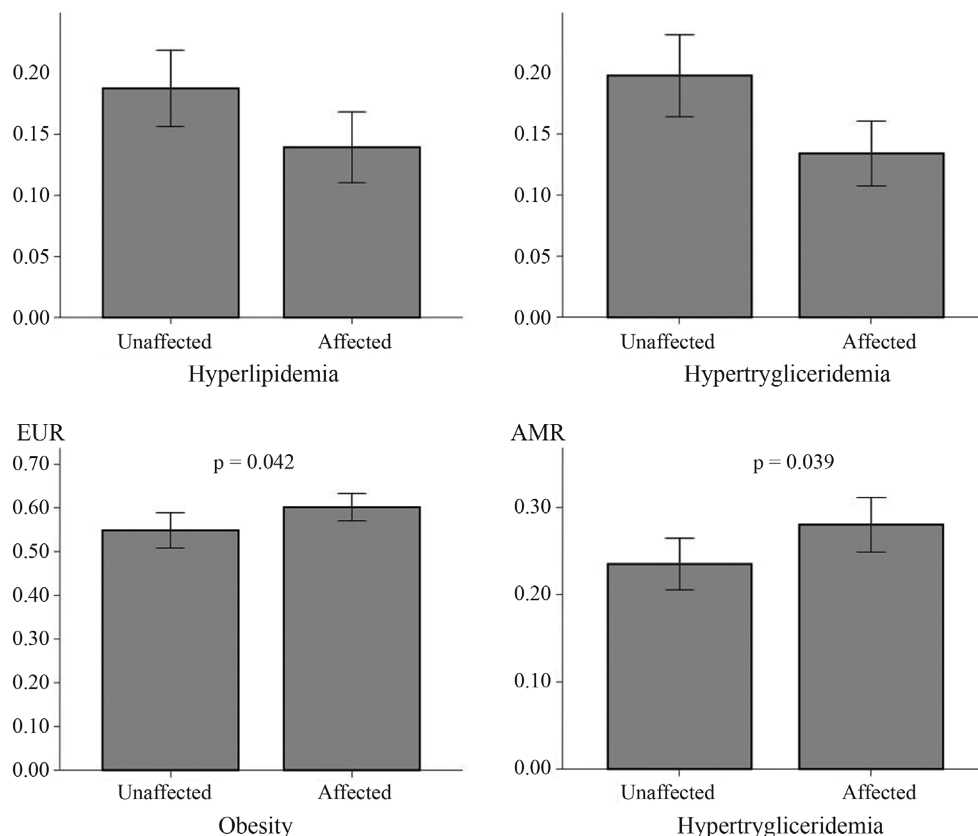

Figure 2 - Mean difference in the ancestry coefficients of significant traits. AFR = African ancestry; AMR = Amerindian ancestry; EUR = European ancestry. Error bars indicate the 95% confidence intervals.

2007), however, no significant differences in standard deviations or variance were found between the present sample population and other populations from the same region (data not shown) (Lins *et al.*, 2010, 2011). Although self-reported ancestry is socially correlated with income in most parts of Brazil, income and education status were not assessed in the present study. Notwithstanding, total lipid intake was considered as a covariate that correlates with increased income in Brazilian women (Bonomo *et al.*, 2003) and may represent an additional risk for cholesterol and lipid disorders.

Genetic and environmental factors, such as age, diet and lifestyle are known to contribute to phenotypic traits such as those evaluated in this study. Genetic ancestry has an inherent influence on the etiology of lipid and metabolism-related diseases, as demonstrated in cases such as African ancestry associations with triglyceride, LDL-cholesterol and HDL-cholesterol levels in admixed African-Americans (Deo *et al.*, 2009), or an inverse association in individuals with European ancestry (Basu *et al.*, 2009). In the present study, increased African ancestry was significantly associated with decreased levels of triglyceride-rich serum components (TG and VLDL) and augmented levels of cholesterol-rich lipoproteins (LDL), as has been reported by others (Deo *et al.*, 2009). No correlation was found for HDL levels, as previously reported (Basu *et al.*, 2009; Deo *et al.*, 2009). Although the effect of European ancestry observed here was modest, it was nevertheless significant; based on this finding, we suggest that European ancestry is an additional risk factor and that higher levels of African ancestry account for a more beneficial lipid profile in elderly women with admixed ethnicity.

The levels of LDL expression in African-American women are often described as being higher than those in European-American women. Conversely, increments of triglycerides in lean African-Americans are described as lower than in lean European-American women (Bower *et al.*, 2002). Here, African ancestry exerted a protective effect against hyperlipidemia and hypertriglyceridemia since it was correlated with higher levels of LDL and lower levels of VLDL. Therefore, in this sample population, African ancestry influenced lipid profiles, but, in contrast to previous studies (Fernandez *et al.*, 2003; Lai *et al.*, 2009), was not correlated to obesity, diabetes or hypertension. This lack of association may reflect a difference in genetic stratification (Wulan *et al.*, 2010), and the levels and range of admixture (Lins *et al.*, 2011) may confound results because of pronounced genetic effects on phenotype expression. For this reason, association studies should not be labeled as analyzing deterministic, causal relationships, but rather as an elucidative contribution to the pathophysiology of these complex phenotypes.

The prevalence of obesity varies significantly among ethnic groups, especially African- and European-Americans (Deurenberg *et al.*, 1998). In addition, some studies have demonstrated associations between European ancestry and body composition traits in admixed populations, especially bone mineral density (BMD) (Bonilla *et al.*, 2004b) and BMI (Basu *et al.*, 2009). Others have reported an association between African ancestry and BMI, FM, FFM and BMD in African-American women (Fernandez *et al.*, 2003). In the present admixed cohort, European ancestry was significantly correlated with BMI and with obesity, but in a previous study (Gentil *et al.*, 2009) this association was not detected. Fat mass and fat-free mass were not correlated with any ancestry estimate, neither in the present nor in a previous study of elderly Brazilian women (Moreno Lima *et al.*, 2007). Such associations are particularly important for understanding the role of genetic ancestry in metabolic-related traits and how interacting variables modulate these phenotypes.

Although lipid intake had no effect on overall traits, we do not advocate excluding this variable in future assessments in view of possible environmental influences on the risk of obesity and metabolic syndrome (Sichieri, 2002) or heart disease (Nobrega *et al.*, 2011). Notably, Brazilian women display significant differences in their pattern of lipid macronutrient consumption, with a trend toward increased consumption with increased income (Bonomo *et al.*, 2003) and a suggestive risk for heart disease (Nobrega *et al.*, 2011). With regard to ethnicity, in a study of the dietary pattern of a Brazilian-urban population, a correlation was identified between categories of skin color (defined by the interviewer) and dietary patterns, in which lighter skin was associated with a more varied diet (Sichieri *et al.*, 2003). However, this study was not income-adjusted, and categorical ethnicity groups might comprise a wide degree of admixture (Lins *et al.*, 2011). Consequently, other factors relating ancestry and dietary patterns to obesity and comorbidities should be investigated.

Other associations found in this study are of interest to the clinical status of elderly women. However, they are merely well-established descriptions of metabolic health risk conditions, such as the association between lipid profile, diabetes and/or hypertension, but do not represent a novel addition to our current knowledge. The supplemental tables, available as part of the online article, contain all the association tests and their significance. Although a body of evidence advocates that the overall ancestry background influences the lipid profile of individuals, the novelty of the present paper is the approach used to address this matter. The Brazilian population displays an extensive degree of admixture that it renders suitable for estimating a variety of ancestries under standardized socio-economic conditions (Lins *et al.*, 2011).

The relatively small sample size used may be a limitation of this study. However, the sample size was sufficiently large to significantly corroborate previous reports in other populations. This lends plausibility to the present data since the replication of positive findings is an important re-

quirement for genetic studies. This scenario might help identify the relationship between ancestry backgrounds and specific phenotypes. The present study may also have been subject to other unmeasured confounders, such as the important contribution of individual interactions with environmental factors. Lifestyle behaviors in the elderly population that may be used as a covariant, such as smoking, alcohol intake, stress or depression, were not assessed here. Since this study used a cohort of elderly women, these results cannot be extended to another population strata or gender.

In conclusion, our results support the role of genetic ancestry in chronic non-communicable diseases prevalent in elderly women and suggest that caution should be taken when performing gene-based association studies in admixed populations because of the genetic heterogeneity. Our results raise the possibility of undertaking association studies based on admixture linkage disequilibrium; such studies may identify genomic loci that could explain correlations between lipid metabolic traits and ancestry.

Acknowledgments

The authors thank Cristiane Urcina Lima, Fernanda Colares-Bento and Sarah Peres da Silveira for gathering the nutritional data and for ensuring adherence to the food registration procedure. This research was supported by CNPq (grants 484318/2006-3 and 402699/2007-6) and FAPDF (grants 193.000.309/2007 and 193.000.449/2008). R.S. Paula was supported by a studentship from CAPES (PROSUP), A.S. Pires was supported by a student scholarship from CNPq (grant 550315/2008-0) and R.W. Pereira was supported by a research fellowship from CNPq.

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Supplementary Material

The following online material is available for this article:

- Table S1 - Pearson correlation for continuous traits.
- Table S2 - Independent-samples *t*-test comparing the means of continuous traits among categorical groups.

This material is available as part of the online article from http://www.scielo.br/gmb.

Associate Editor: Carlos F.M. Menck

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Table S1: Pearson Correlation of continuous traits. Correlation coefficient (r) below and p-values above the diagonal. Correlation significant at a 95% confidence level.

	Age	Weight	Height	BMI	FM	FFM	TG	TL	TC	LDL	VLDL	HDL	GLU	SBP	DBP	LIP	EUR	AMR	AFR
Age	-	<0.001	0.014	0.006	0.023	0.014	0.707	0.048	0.069	0.048	0.179	0.101	0.978	0.980	0.285	0.703	0.671	0.594	0.976
Weight	-0.290	-	<0.001	<0.001	<0.001	<0.001	0.081	0.004	0.125	0.169	0.002	0.005	0.049	0.286	0.142	0.378	0.037	0.261	0.190
Height	-0.184	0.309	-	0.018	0.981	0.909	0.580	0.856	0.764	0.634	0.482	0.770	0.713	0.618	0.742	0.007	0.849	0.356	0.494
BMI	-0.208	0.878	-0.179	-	<0.001	<0.001	0.045	0.003	0.127	0.184	<0.001	0.002	0.026	0.201	0.106	0.026	0.028	0.124	0.303
FM	-0.171	0.691	-0.002	0.717	-	<0.001	0.442	0.013	0.278	0.298	0.003	0.011	0.087	0.864	0.187	0.237	0.065	0.082	0.662
FFM	0.186	-0.704	-0.009	-0.726	-0.941	-	0.482	0.014	0.228	0.237	0.003	0.010	0.050	0.407	0.455	0.135	0.055	0.065	0.677
TG	-0.029	0.132	-0.042	0.151	0.058	-0.053	-	<0.001	<0.001	0.792	<0.001	0.282	0.001	0.012	0.006	0.485	0.941	0.047	0.063
TL	-0.149	0.218	0.014	0.223	0.188	-0.184	0.402	-	<0.001	<0.001	<0.001	0.287	0.380	0.025	0.007	0.450	0.648	0.353	0.704
TC	-0.138	0.116	0.023	0.116	0.082	-0.091	0.271	0.819	-	<0.001	0.005	<0.001	0.296	0.207	0.137	0.467	0.297	0.999	0.226
LDL	-0.150	0.104	0.036	0.101	0.079	-0.090	-0.020	0.677	0.903	-	0.761	0.219	0.044	0.709	0.606	0.343	0.230	0.475	0.035
VLDL	-0.102	0.235	-0.053	0.267	0.225	-0.227	0.643	0.611	0.209	-0.023	-	0.006	<0.001	0.003	0.004	0.904	0.433	0.118	0.014
HDL	0.124	-0.212	0.022	-0.236	-0.192	0.193	-0.082	0.081	0.276	0.093	-0.207	-	0.252	0.120	0.160	0.459	0.449	0.885	0.462
GLU	0.002	0.149	-0.028	0.167	0.129	-0.148	0.250	0.067	-0.079	-0.152	0.276	-0.087	-	0.501	0.853	0.227	0.717	0.521	0.835
SBP	-0.002	0.081	-0.038	0.097	0.013	-0.063	0.188	0.169	0.096	0.028	0.221	-0.118	0.051	-	<0.001	0.200	0.442	0.326	0.944
DBP	-0.081	0.111	-0.025	0.122	0.100	-0.057	0.207	0.201	0.112	0.039	0.214	-0.106	0.014	0.769	-	0.211	0.013	0.221	0.096
LIP	-0.029	-0.067	0.202	-0.168	-0.090	0.113	-0.053	0.057	0.055	0.072	0.009	0.056	-0.092	-0.097	-0.095	-	0.943	0.663	0.610
EUR	0.032	0.157	-0.014	0.165	0.140	-0.145	-0.006	-0.035	-0.079	-0.091	0.059	-0.057	0.028	-0.058	-0.187	0.005	-	<0.001	<0.001
AMR	-0.040	-0.085	0.070	-0.116	-0.132	0.140	0.150	0.070	0.001	-0.054	0.118	0.011	-0.049	0.074	0.093	0.033	-0.575	-	<0.001
AFR	0.002	-0.099	-0.052	-0.078	-0.033	0.032	-0.140	-0.029	0.092	0.159	-0.185	0.056	0.016	-0.005	0.126	-0.039	-0.597	-0.312	-

Table S2: Independent-samples t-test comparing means of continuous traits among categorical groups. (*) Correlation significant at 95% confidence level.

		Obesity			Hypertrygliceridemia			Hypercholesterolemia			Hyperlipidemia			Dyslipidemia		
Trait	Group	N	Mean	p	N	Mean	p	N	Mean	p	N	Mean	p	N	Mean	p
Age (years)	Aff.	91	67.59	0.091	84	67.90	0.380	135	67.99	0.164	73	67.71	0.256	153	68.05	0.122
	Unaff.	85	69.07		92	68.67		41	69.34		103	68.73		23	70.00	
Weight (kg)	Aff.	91	69.02	<0.001	84	64.93	0.022	135	64.23	0.030	73	65.23	0.016	153	64.03	0.036
	Unaff.	85	57.00	*	92	61.65	*	41	59.87	*	103	61.79	*	23	57.78	*
Height (cm)	Aff.	91	151.05	0.004	84	151.54	0.108	135	152.53	0.224	73	151.70	0.260	153	152.25	0.983
	Unaff.	85	153.52	*	92	152.89		41	151.32		103	152.63		23	152.22	
BMI (kg/m²)	Aff.	91	30.22	<0.001	84	28.28	0.001	135	27.64	0.052	73	28.37	0.002	153	27.65	0.017
	Unaff.	85	24.13	*	92	26.37	*	41	26.08		103	26.51	*	23	24.83	*
FM (%)	Aff.	91	40.94	<0.001	84	38.18	0.088	135	37.64	0.287	73	38.39	0.056	153	37.63	0.241
	Unaff.	85	33.35	*	92	36.45		41	36.07		103	36.49		23	34.91	
FFM (%)	Aff.	91	55.55	<0.001	84	57.99	0.073	135	58.55	0.297	73	57.81	0.050	153	58.58	0.285
	Unaff.	85	62.44	*	92	59.70		41	59.96		103	59.64	*	23	60.91	
Triglycerides (mg/dL)	Aff.	91	167.49	0.152	84	217.64	<0.001	135	166.82	0.002	73	219.26	<0.001	153	166.98	<0.001
	Unaff.	85	146.26		92	102.09	*	41	125.68	*	103	113.28	*	23	92.43	*
Total lipids (mg/dL)	Aff.	91	756.23	0.272	84	804.80	<0.001	135	791.69	<0.001	73	832.06	<0.001	153	769.94	<0.001
	Unaff.	85	732.43		92	689.89	*	41	590.12	*	103	682.84	*	23	577.04	*
Total cholesterol (mg/dL)	Aff.	91	233.15	0.977	84	238.01	0.160	135	248.67	<0.001	73	247.67	<0.001	153	240.24	<0.001
	Unaff.	85	233.34		92	228.89		41	182.46	*	103	223.02	*	23	186.74	*
LDL (mg/dL)	Aff.	91	140.31	0.897	84	136.57	0.280	135	152.65	<0.001	73	145.75	0.082	153	145.27	<0.001
	Unaff.	85	139.53		92	143.00		41	98.05	*	103	135.81		23	104.43	*
VLDL (mg/dL)	Aff.	91	31.92	0.029	84	39.86	<0.001	135	31.32	0.004	73	39.65	<0.001	153	31.58	<0.001
	Unaff.	85	27.61	*	92	20.69	*	41	24.95	*	103	22.89	*	23	18.30	*
HDL (mg/dL)	Aff.	91	59.52	0.003	84	60.04	0.043	135	62.29	0.099	73	60.02	0.076	153	61.50	0.498
	Unaff.	85	63.93	*	92	63.12	*	41	59.53		103	62.81		23	62.65	
Fasting glucose (mg/dL)	Aff.	91	107.99	0.088	84	112.54	0.001	135	106.48	0.008	73	114.23	0.001	153	106.22	<0.001
	Unaff.	85	100.40		92	96.83	*	41	97.20	*	103	97.30	*	23	91.75	*
SBP (mmHg)	Aff.	91	140.11	0.056	84	143.04	0.001	135	138.14	0.102	73	145.14	<0.001	153	137.29	0.240
	Unaff.	85	132.47		92	130.38	*	41	130.73		103	130.24	*	23	130.65	
DBP (mmHg)	Aff.	91	83.79	0.021	84	83.69	0.041	135	81.67	0.449	73	84.66	0.013	153	81.14	0.966
	Unaff.	85	78.35	*	92	78.86	*	41	79.52		103	78.69	*	23	81.30	
Lipid intake (%)	Aff.	91	33.66	0.083	84	34.49	0.653	135	34.54	0.244	73	34.66	0.460	153	34.41	0.512
	Unaff.	85	35.00		92	34.14		41	33.53		103	34.05		23	33.59	
EUR (%)	Aff.	91	60.15	0.042	84	58.57	0.472	135	57.69	0.896	73	59.51	0.198	153	57.50	0.808
	Unaff.	85	54.87	*	92	56.72		41	57.32		103	56.25		23	58.26	
AMR (%)	Aff.	91	24.27	0.192	84	28.01	0.039	135	25.13	0.415	73	26.56	0.476	153	26.05	0.307
	Unaff.	85	27.13		92	23.50	*	41	27.39		103	25.01		23	23.00	
AFR (%)	Aff.	91	15.57	0.281	84	13.42	0.003	135	17.19	0.434	73	13.93	0.026	153	16.44	0.447
	Unaff.	85	18.00		92	19.78	*	41	15.29		103	18.74	*	23	18.74	

Table S2: Independent-samples t-test comparing means of continuous traits among categorical groups. (*) Correlation significant at 95% confidence level.

		Glucose intolerance			Type 2 Diabetes			Hypertension			Normolipidic diet		
Trait	Group	N	Mean	p	N	Mean	p	N	Mean	p	N	Mean	p
Age (years)	Aff.	42	68.93	0.432	24	69.46	0.329	147	68.30	0.970	117	68.26	0.902
	Unaff.	134	68.11		152	68.13		29	68.34		59	68.39	
Weight (kg)	Aff.	42	64.33	0.369	24	65.33	0.238	147	63.86	0.047	117	63.97	0.132
	Unaff.	134	62.86		152	62.88		29	59.97	*	59	61.71	
Height (m)	Aff.	42	151.81	0.553	24	152.25	0.996	147	152.33	0.647	117	151.94	0.318
	Unaff.	134	152.38		152	152.24		29	151.83		59	152.85	
BMI (kg/m²)	Aff.	42	27.97	0.221	24	28.22	0.229	147	27.54	0.055	117	27.73	0.028
	Unaff.	134	27.07		152	27.13		29	26.00		59	26.40	*
FM (%)	Aff.	42	37.60	0.732	24	37.32	0.976	147	37.30	0.922	117	38.11	0.025
	Unaff.	134	37.17		152	37.27		29	37.17		59	35.62	*
FFM (%)	Aff.	42	58.62	0.774	24	58.79	0.949	147	58.84	0.845	117	58.19	0.048
	Unaff.	134	58.96		152	58.89		29	59.07		59	60.24	
Triglycerides (mg/dL)	Aff.	42	205.95	0.010	24	203.29	0.023	147	163.39	0.015	117	160.32	0.473
	Unaff.	134	141.97	*	152	149.97	*	29	126.03	*	59	151.12	
Total lipids (mg/dL)	Aff.	42	767.52	0.309	24	767.06	0.544	147	750.59	0.164	117	738.11	0.385
	Unaff.	134	737.59		152	741.21		29	715.03		59	757.86	
Total cholesterol (mg/dL)	Aff.	42	235.02	0.777	24	231.58	0.844	147	234.27	0.435	117	232.07	0.603
	Unaff.	134	232.69		152	233.51		29	228.03		59	235.58	
LDL (mg/dL)	Aff.	42	134.31	0.295	24	130.87	0.225	147	139.82	0.927	117	138.42	0.465
	Unaff.	134	141.69		152	141.36		29	140.52		59	142.93	
VLDL (mg/dL)	Aff.	42	35.82	0.002	24	38.30	0.006	147	30.80	0.036	117	29.58	0.693
	Unaff.	134	27.97	*	152	28.51	*	29	24.97	*	59	30.37	
HDL (mg/dL)	Aff.	42	62.10	0.767	24	62.08	0.852	147	61.54	0.725	117	61.43	0.673
	Unaff.	134	61.51		152	61.58		29	62.24		59	62.10	
Fasting glucose (mg/dL)	Aff.	42	135.45	<0.001	24	149.04	<0.001	147	106.23	0.001	117	105.00	0.625
	Unaff.	134	94.57	*	152	97.26	*	29	94.67	*	59	102.98	
SBP (mmHg)	Aff.	42	138.69	0.459	24	140.63	0.310	147	141.63	<0.001	117	134.62	0.232
	Unaff.	134	135.71		152	135.76		29	110.00	*	59	140.00	
DBP (mmHg)	Aff.	42	80.24	0.612	24	82.29	0.641	147	83.78	<0.001	117	80.81	0.680
	Unaff.	134	81.46		152	80.99		29	67.93	*	59	81.86	
Lipid intake (%)	Aff.	42	33.99	0.592	24	33.40	0.269	147	34.13	0.313	117	31.74	<0.001
	Unaff.	134	34.41		152	34.45		29	35.22		59	39.39	*
EUR (%)	Aff.	42	60.48	0.195	24	62.29	0.100	147	57.03	0.293	117	57.72	0.902
	Unaff.	134	56.70		152	56.86		29	60.48		59	57.37	
AMR (%)	Aff.	42	23.05	0.124	24	21.83	0.071	147	26.52	0.087	117	26.24	0.470
	Unaff.	134	26.47		152	26.26		29	21.24		59	24.49	
AFR (%)	Aff.	42	16.48	0.900	24	15.88	0.800	147	16.44	0.522	117	16.04	0.406
	Unaff.	134	16.83		152	16.88		29	18.28		59	18.14	