

HHS Public Access

Diabetes Res Clin Pract. Author manuscript; available in PMC 2018 March 02.

Published in final edited form as:

Author manuscript

Diabetes Res Clin Pract. 2016 June ; 116: 212–217. doi:10.1016/j.diabres.2016.04.015.

Association of abdominal fat with serum amylase in an older cohort: The Baltimore Longitudinal Study of Aging

Jenny Pena Dias^{a,*}, Jennifer A. Schrack^c, Michelle D. Shardell^b, Josephine M. Egan^a, and Stephanie Studenski^b

^aLaboratory of Clinical Investigation, National Institute on Aging, Baltimore, MD 21225, United States

^bTranslational Gerontology Branch, National Institute on Aging, Baltimore, MD 21225, United States

^cDepartment of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD 21205, United States

Abstract

Aims—Abdominal fat is a major determinant of metabolic diseases in older individuals. Obesity and diabetes are associated with low serum amylase (SA) levels, but the association between SA and metabolic disease is poorly understood. We investigated the association of low SA with diabetes and sex-specific associations of serum amylase with abdominal fat in older adults.

Methods—In community-dwelling volunteers from the Baltimore Longitudinal Study of Aging (778 participants, age 66.8 ± 13.6 years), we assessed abdominal fat by computed tomography and diabetes status using the American Diabetes Association criteria. Linear regression analyses assessed the cross-sectional associations between abdominal fat and SA, and logistic regression assessed the odds of diabetes, given low SA.

Results—In unadjusted analyses, individuals in the lowest SA quartile (<48 µ/L) had 1.97 greater odds of diabetes, (95%CI, 1.01–3.83) than those in the highest quartile ($80 \mu/L$). This association was no longer significant after adjusting for visceral adipose tissue area (VAT, dm²), abdominal subcutaneous adipose tissue (SAT, dm²) or BMI. In adjusted analyses, VAT and SAT were significantly associated with SA in both sexes. Among women, SA was more strongly associated with VAT than with SAT or BMI; VAT ($\beta = -0.117 \pm 0.048$, P < 0.001), SAT ($\beta = -0.023 \pm 0.025$, P = 0.346) and BMI ($\beta = -0.0052 \pm 0.075$, P = 0.49).

Conclusions—The association between SA and diabetes was explained mainly by abdominal visceral fat. In women, SA was more strongly associated with VAT than with BMI or SAT. These findings provide motivation for future mechanistic studies on SA's role in metabolic diseases.

^{*}Corresponding author. diasjp@mail.nih.gov (J.P. Dias).

Conflict of interest statement

All authors declare no conflicts of interest with the contents of this manuscript.

Author's contribution

JPD design of the study, statistical analysis and draft of manuscript for submission. JS design of the study, draft and review of manuscript for submission. MDS reviewed statistical analysis and manuscript. JME reviewed manuscript. SS design of the study and review of the manuscript for submission.

Keywords

Abdominal visceral/subcutaneous; fat; Aging; Diabetes; Serum amylase

1. Introduction

Recently, much interest has been drawn to the association between amylase and obesity, most particularly salivary amylase, as low copies of salivary amylase gene expression predict obesity in humans [1,2]. Amylase is a glycoside hydrolase, which acts upon the bonds between the glucose units of the starch polymers, contributing to carbohydrate digestion [3]. In healthy individuals, the pancreas and the salivary glands account for almost all serum amylase; it is estimated that between 40% and 45% comes from the pancreas and 55–60% from the salivary glands [4]. A series of human studies in Asians has reported an association between the prevalence of low serum amylase and increases in diabetes and metabolic syndrome [5], and low serum amylase has been linked with diabetes and obesity [5,6].

Excessive intake of refined carbohydrates plays a role in the development of obesity and type 2 diabetes [7,8]. As the prevalence of obesity increases worldwide, scientists and pharmaceutical companies are investigating ways to reduce body weight and prevent weight gain. Some claim that amylase inhibitors known as starch blockers, which are extracted from certain food plants such as kidney beans and wheat, inhibit salivary and pancreatic activity and reduce absorption of starches in the small intestine, leading to weight loss and reduced blood glucose [9]. However, when starch blockers were first developed, researchers did not find them effective for limiting carbohydrate absorption, and the evidence for weight loss is still unclear [10]. In addition, the molecular mechanism underlying the associations among serum amylase, diabetes and obesity is unknown. Epidemiological studies investigating the relationship between serum amylase and diabetes or obesity are few. To better understand the role of amylase in metabolic diseases such as diabetes and obesity with the goal of creating more effective treatments to reduce blood glucose, more epidemiological and intervention studies are needed.

Given the substantial literature showing that abdominal obesity, specifically visceral adiposity, increases the risk of type 2 diabetes and metabolic syndrome [11–14], the large proportion of older individuals who suffer from metabolic diseases, and the paucity of studies on serum amylase, examining the link between serum amylase and abdominal adiposity may fill an important evidence gap. Therefore, the purpose of this study is to investigate the associations of serum amylase with diabetes and with measures of body fat focusing on abdominal obesity.

2. Subjects, materials, and methods

2.1. Study subjects

The BLSA is a study of normative human aging, established in 1958 and supported by the National Institute on Aging Intramural Research Program (NIA – IRP). General descriptions

of the sample and the enrollment procedures and criteria have been reported [15]. Briefly, the BLSA constitutes a continuously enrolled cohort with some targeted recruitment (e.g., women, racial minorities) over its 57-plus year history. All participants are community volunteers who must pass a comprehensive health and functional screening evaluation and be free of all major chronic conditions and cognitive and functional impairment at enrollment. Once enrolled, participants undergo extensive testing every one to four years depending on their age and are followed for life. The population for the current study consists of 778 participants seen in the BLSA clinic between April 2003 and June 2012. The NIH NIEHS IRB (National Institute of Environmental Health Sciences) approved the study protocol and all participants provided written informed consent.

2.2. Measures

2.2.1. Anthropometric and biochemical measurements—Stature was measured to the nearest 0.1 cm by Stadiometer from (Holtain Limited, Crymych, Dyfed, UK) and body weight was measured to the nearest 0.1 kg by using SR scale model 725 L from (SR Instruments, Tonawanda, NY, US) to calculate body mass index (BMI). Total body muscle mass and fat were measured using dual-energy X-ray absorptiometry (DEXA) (Lunar prodigy 10190 and prodigy advance PA + 130024 from GE Healthcare, Madison, WI, US). All DEXA scans were analyzed using Encore 2006 software version 10.51.006 from (GE Healthcare, Madison, WI, US) for body composition analysis.

2.2.2. Diabetes definition—Diabetes was defined as fasting plasma glucose 126 mg/dL, consistent with the 2015 American Diabetes Association criteria [16]. In the BLSA sample, 9.3% of women and 13% of men satisfy this definition. Using this criteria, our definition of person with diabetes was limited to those with uncontrolled fasting blood glucose, regardless of medication status, as individuals with diabetes who are not well-controlled are likely to be metabolically different than those who are well-controlled [17,18].

2.2.3. Serum amylase measurement—Serum amylase levels were measured using an automatic biochemistry and immunoassay analyzer/integrated system Dimension Vista 3000T (Siemens Healthcare, Malvern, PA). The intra and inter-assay coefficient of variation (CV) for the amylase was less than 5%.

2.2.4. CT-abdomen quantification—A cross-sectional 10mm CT image of the abdomen was obtained from each participant at the lumbar spine level (L4-L5) using a Somatom Sensation 10, multislice, helical CT Scanner (Siemens Healthcare, Malvern, PA). Abdominal VAT (visceral adipose tissue area) and abdominal SAT (subcutaneous adipose tissue area) were extracted from the total cross-sectional area of the abdomen, and Geanie software version 2.1 (BonAlyse Oy, Jyvaskyla, Finland) was used to quantify the cross-sectional area (dm²).

2.2.5. Other covariates—The study included men and women. Age and ethnicity were ascertained by self-report, with ethnicity categorized as White, Black or other.

2.3. Statistical analysis

All statistical analyses were performed using SAS version 9.3 software. Amylase levels that were not normally distributed were logarithmically transformed before analysis: however, mean values of the variables are presented untransformed as mean \pm SD for descriptive purposes (Table 1). Unpaired *t*-tests and chi-square test were used to compare participant characteristics differences by sex (Table 1). Logistic regression was used to estimate the odds ratio of prevalent diabetes by serum amylase (in quartiles) (Table 2). Linear regression analysis was used to assess the sex-specific cross-sectional relationship between continuous serum amylase and measures of adiposity (Table 3). All analyses (Tables 2 and 3) were adjusted for potential confounding factors (age, sex and ethnicity). *P*-values (*p*) < 0.05 were considered to indicate statistical significance.

3. Results

3.1. Participant characteristics

The characteristics of the 778 participants are presented in Table 1. Most of the participants were White or African American, with similar numbers of men and women, (400 women, mean age 65.4 ± 13.1 years old; 378 males, mean age 68.3 ± 13.9 years old). Men were slightly older, taller and had higher BMI, VAT and lean mass percentage compared to women (P < 0.05 for all). Women had more SAT than men (P < 0.001). Serum amylase levels were similar in both sexes (P = 0.48).

3.2. Association of amylase with diabetes

We examined the odds of diabetes by serum amylase quartile (Table 2). Participants in the lowest serum amylase quartile had a 1.97 higher odds of diabetes than participants in the highest quartile (95% CI: 1.01-3.83; P=0.04), after covariate adjustment (model 1), however, this association was no longer significant once BMI was added to the model (P=0.15) (model 2). To determine the role of adipose tissue in the association between amylase and diabetes, we added covariates for SAT and VAT to the models. SAT was not significant when added to model 1 (OR = 1.01[95% CI: 0.99-1.03] (P=0.21). However in further analyses, the odds of diabetes were higher among those with greater VAT (models 3, 4 & 5).

3.3. Relationship of amylase with abdominal fat (VAT and SAT) by sex

Based on the differences in body composition shown in Table 1, the analysis was stratified by sex. Age- and ethnicity-adjusted associations of serum amylase with VAT, SAT, and other related covariates are presented in Table 3. The results show that BMI, was significantly negatively associated with the log of serum amylase, with similar β -coefficients, ($\beta = -0.018 \pm 0.003$; P < 0.001) for women and ($\beta = -0.020 \pm 0.005$; P < 0.001) for men after adjusting for age and ethnicity (model 1). There was marginal evidence of VAT-by-sex interaction on serum amylase (*P*-value for interaction = 0.05), with a stronger association in women, but no evidence for SAT-by-sex interaction (*P*-value for interaction = 0.33).

In women, both abdominal VAT ($\beta = -0.189 \pm 0.037$; P < 0.001) and SAT ($\beta = -0.066 \pm 0.011$; P < 0.001) were significantly negatively associated with serum amylase (models 2, 3), independent of age and ethnicity. The significant negative association between serum

amylase and VAT ($\beta = -0.126 \pm 0.043$; P = 0.02) persisted after adding SAT ($\beta = -0.045 \pm 0.014$; P = 0.07), model 4). To assess the contribution of abdominal fat relative to BMI, we evaluated the association of serum amylase with abdominal VAT ($\beta = -0.102 \pm 0.048$; P = 0.02) and SAT ($\beta = -0.023 \pm 0.025$; P = 0.50) after adjusting for BMI (model 5–6). In these models, only VAT and BMI ($\beta = -0.0129 \pm 0.004$; P = 0.02) were statistically significant (model 5). In addition, in model 7, only VAT ($\beta = -0.117 \pm 0.048$; P < 0.001) was statistically significant.

In men, both VAT ($\beta = -0.085 \pm 0.032$; P = 0.009) and SAT ($\beta = -0.053 \pm 0.019$; P = 0.02) were significantly negatively associated with serum amylase (model 2, 3), but in contrast to the findings in women, the negative association between serum amylase and VAT ($\beta = -0.059 \pm 0.037$; P = 0.10) disappeared after adding SAT ($\beta = -0.034 \pm 0.022$; P = 0.25) (model 4). SAT was also not significant after adjusting for covariates. Neither VAT ($\beta = -0.012 \pm 0.040$; P = 0.74) nor SAT ($\beta = -0.033 \pm 0.034$; P = 0.19) were associated with serum amylase after adjusting for BMI and covariates (model 5–6); however BMI remained significant in both models (BMI $\beta = -0.018 \pm 0.004$; P = 0.009), model 5; BMI $\beta = -0.0271 \pm 0.006$; P = 0.008, model 6). In model 7, only BMI ($\beta = -0.026 \pm 0.009$; P = 006) was statistically significant.

4. Discussion

Low serum amylase is associated with higher risk of diabetes in a large cohort of older adults. Abdominal visceral fat is more strongly associated with low serum amylase than BMI. This is the first study to examine the association of serum amylase with abdominal adiposity by CT-scan measurement (VAT and SAT) in an American population [1,2,5]. Our finding suggests that serum amylase is more strongly associated with VAT than SAT regardless of sex and that in women, serum amylase is more strongly associated with VAT than BMI.

The association between serum amylase and diabetes was attenuated after adjusting for BMI, VAT, and other covariates, showing that VAT is an important predictor of low serum amylase in individuals with diabetes. It is unknown why serum amylase tends to be low in individuals with diabetes, high abdominal adiposity and obesity. However, several possible mechanisms may explain the findings of low serum amylase in obese and individuals with diabetes. First, as reported in several animal studies, saturated fatty acids stimulate the release of pancreatic amylase in a dose dependent manner [19]. Therefore, one possible mechanism is that under excess circulating free fatty acids (FFA) the stimulation of serum amylase secretion may be inhibited due to enhanced feedback loop on the pancreatic cells. Second, insulin resistance is another potential mechanism that could explain lower serum amylase levels in individuals with high abdominal visceral fat and diabetes. In rats, insulin was shown to bind to its own receptor on pancreatic acinar cells, leading to stimulation of amylase secretion and synthesis [20] and therefore insulin resistance in acinar cells may lead to reductions in amylase secretion. Additionally, excess levels of FFA are known to impair insulin receptor activity via the accumulation of FFA derived metabolic products that promote the activation of several intracellular molecular mechanisms such as activation of serine/threonine kinases, JNK (c-jun aminoterminal kinases), IKK β (inhibitor of nuclear

factor kappa-B kinase) and PKCθ (protein kinase C) all which phosphorylate IRS-1 (insulin receptor substrate 1) on serine residues of the insulin receptor [21,22].

The findings show that VAT is more strongly negatively associated with serum amylase than SAT in women, implying that serum amylase is associated mostly with fat accumulating among the organs, which is usually associated with diverse pathologies. In women, both BMI and VAT were associated with lower serum amylase, whereas in men, only BMI was associated with lower serum amylase. Sex hormones regulate both pancreas and adipocyte cells [23,24] and adipocyte cells contribute to steroidogenesis [25]. The participation of adipose tissue to whole body steroid metabolism is quite significant, with adipose tissue contributing up to 100% of circulating estrogen in postmenopausal women and 50% of circulating testosterone in premenopausal women [26]. Adipocyte accumulation is known to impact sex hormones on activation of the hypothalamic-pituitary-adrenal (HPA) axis [27]. In addition, differences in the expression and activity of aromatase, the enzyme that converts testosterone to estrogen, have been demonstrated in human adipose tissue of pre-and postmenopausal women compared to men [28]. Indeed, changes in sex-hormones between men and women may explain the sex differences observed in the association of serum amylase with VAT and BMI. In summary, our findings indicate a higher negative association of serum amylase with VAT or SAT in women compared to men even though men have higher quantities of visceral abdominal fat.

This study had both strengths and limitations. First the analysis included a large sample size of men and women. Second, the BLSA had data on amylase concentrations and abdominal CT scans to measure adiposity, as well as fasting glucose levels to obtain an objective measure of diabetes status. The main limitation of the study is that it is cross-sectional and does not allow the assessment of cause-and-effect relationships.

In conclusion, we foresee that serum amylase may be a useful addition to the list of metabolic syndrome markers, specifically in women. To our knowledge, this is the first study investigating the association of serum amylase with types of abdominal fat. Therefore, this study provides motivation for future mechanistic studies to understand the exact role of amylase in the abdominal tissue in people with diabetes and the contribution of amylase to metabolic abnormalities.

Acknowledgments

This research was supported by the Intramural Research Program of the National Institute of Health (NIH), National Institute on Aging (NIA), United States.

References

- Falchi M, El-Sayed Moustafa JS, Takousis P, Pesce F, Bonnefond A, Andersson-Assarsson JC, et al. Low copy number of the salivary amylase gene predisposes to obesity. Nat Genet. 2014; 46:492–7. [PubMed: 24686848]
- Mejia-Benitez MA, Bonnefond A, Yengo L, Huyvaert M, Dechaume A, Peralta-Romero J, et al. Beneficial effect of a high number of copies of salivary amylase AMY1 gene on obesity risk in Mexican children. Diabetologia. 2015; 58:290–4. [PubMed: 25394825]
- 3. Bijttebier A, Goesaert H, Delcour JA. Amylase action pattern on starch polymers. Biologia. 2008; 63:989–99.

- Pieper-Bigelow C, Strocchi A, Levitt MD. Where does serum amylase come from and where does it go? Gastroenterol Clin North Am. 1990; 19:793–810. [PubMed: 1702756]
- Nakajima K, Nemoto T, Muneyuki T, Kakei M, Fuchigami H, Munakata H. Low serum amylase in association with metabolic syndrome and diabetes: a community-based study. Cardiovasc Diabetol. 2011; 10:34. [PubMed: 21496338]
- Kondo T, Hayakawa T, Shibata T, Sato Y, Toda Y. Serum levels of pancreatic enzymes in lean and obese subjects. Int J Pancreatol. 1988; 3:241–8. [PubMed: 2455007]
- Gross LS, Li L, Ford ES, Liu S. Increased consumption of refined carbohydrates and the epidemic of type 2 diabetes in the United States: an ecologic assessment. Am J Clin Nutr. 2004; 79:774–9. [PubMed: 15113714]
- Massougbodji J, Le BY, Fratu R, De WP. Reviews examining sugar-sweetened beverages and body weight: correlates of their quality and conclusions. Am J Clin Nutr. 2014; 99:1096–104. [PubMed: 24572563]
- 9. Udani JK, Singh BB, Barrett ML, Preuss HG. Lowering the glycemic index of white bread using a white bean extract. Nutr J. 2009; 8:52. [PubMed: 19860922]
- Bo-Linn GW, Santa Ana CA, Morawski SG, Fordtran JS. Starch blockers-their effect on calorie absorption from a high-starch meal. N Engl J Med. 1982; 307:1413–6. [PubMed: 6182469]
- Despres JP. Intra-abdominal obesity: an untreated risk factor for Type 2 diabetes and cardiovascular disease. J Endocrinol Invest. 2006; 29(3 Suppl):77–82. [PubMed: 16751711]
- 12. Despres JP. Is visceral obesity the cause of the metabolic syndrome? Ann Med. 2006; 38:52–63. [PubMed: 16448989]
- Gastaldelli A. Abdominal fat: does it predict the development of type 2 diabetes? Am J Clin Nutr. 2008; 87:1118–9. [PubMed: 18469227]
- Lebovitz HE, Banerji MA. Point: visceral adiposity is causally related to insulin resistance. Diabetes Care. 2005; 28:2322–5. [PubMed: 16123512]
- Stone JL, Norris AH. Activities and attitudes of participants in the Baltimore longitudinal study. J Gerontol. 1966; 21:575–80. [PubMed: 5918312]
- 16. Standard of medical care in diabetes. Diabetes Care. 2015; 38(Suppl 1)
- 17. Hollander P. Anti-diabetes and anti-obesity medications: effects on weight in people with diabetes. Diabetes Spectr. 2007; 20:159–65.
- Davies MJ, Bergenstal R, Bode B, Kushner RF, Lewin A, Skjoth TV, et al. Efficacy of liraglutide for weight loss among patients with type 2 diabetes: the SCALE diabetes randomized clinical trial. JAMA. 2015; 314:687–99. [PubMed: 26284720]
- Ohbo M, Katoh K, Sasaki Y. Effects of saturated fatty acids on amylase release from exocrine pancreatic segments of sheep, rats, hamsters, field voles and mice. J Comp Physiol B. 1996; 166(5):305–9. [PubMed: 8870261]
- Mossner J, Logsdon CD, Williams JA, Goldfine ID. Insulin, via its own receptor, regulates growth and amylase synthesis in pancreatic acinar AR42J cells. Diabetes. 1985; 34:891–7. [PubMed: 2411617]
- 21. Karpe F, Dickmann JR, Frayn KN. Fatty acids, obesity, and insulin resistance: time for a reevaluation. Diabetes. 2011; 60:2441–9. [PubMed: 21948998]
- Nguyen MT, Satoh H, Favelyukis S, Babendure JL, Imamura T, Sbodio JI, et al. JNK and tumor necrosis factor-alpha mediate free fatty acid-induced insulin resistance in 3T3-L1 adipocytes. J Biol Chem. 2005; 280:35361–71. [PubMed: 16085647]
- Andren-Sandberg A, Backman PL. Sex hormones and pancreatic cancer. Baillieres Clin Gastroenterol. 1990; 4:941–52. [PubMed: 2078793]
- Mayes JS, Watson GH. Direct effects of sex steroid hormones on adipose tissues and obesity. Obes Rev. 2004; 5:197–216. [PubMed: 15458395]
- Meseguer A, Puche C, Cabero A. Sex steroid biosynthesis in white adipose tissue. Horm Metab Res. 2002; 34:731–6. [PubMed: 12660891]
- Kershaw EE, Flier JS. Adipose tissue as an endocrine organ. J Clin Endocrinol Metab. 2004; 89:2548–56. [PubMed: 15181022]

- Pasquali R. The hypothalamic-pituitary-adrenal axis and sex hormones in chronic stress and obesity: pathophysiological and clinical aspects. Ann N Y Acad Sci. 2012; 1264:20–35. [PubMed: 22612409]
- McTernan PG, Anwar A, Eggo MC, Barnett AH, Stewart PM, Kumar S. Gender differences in the regulation of P450 aromatase expression and activity in human adipose tissue. Int J Obes Relat Metab Disord. 2000; 24:875–81. [PubMed: 10918534]

	Wom	en	Men		
Variable	z	Mean ± SD	z	Mean ± SD	<i>P</i> -value
Age (y)	400	65.4 ± 13.1	378	68.3 ± 13.9	0.003
BMI (kg/m ²)	400	26.9 ± 5.4	378	27.7 ± 4.1	0.03
Height (cm)	400	162.1 ± 6.3	378	175.1 ± 7.2	<0.001
Sex (%)	400	51.4	378	48.5	0.12
Race (%) (White/AA)	400	59.1/32.2	378	69.2/23.5	0.005
Amylase (µ/L)	400	66.2 ± 33.0	378	67.8 ± 33.2	0.48
$VAT (dm^2)$	400	0.93 ± 0.49	378	1.18 ± 0.62	<0.001
SAT (dm ²)	400	3.07 ± 1.45	378	2.45 ± 1.01	<0.001
Lean mass %	390	56.8 ± 7.8	365	65.8 ± 7.4	<0.001
Fat mass %	390	39.28 ± 7.63	365	30.19 ± 7.17	<0.01
Individuals with diabetes %	400	9.3	378	13.0	0.09

Data are presented as mean \pm SD or $\% \pm$ SD. VAT (abdominal visceral adipose tissue area), SAT (abdominal subcutaneous adipose tissue area), AA (African American).

Statistical test: unpaired-test, chi-square.

Table 2

Odds of diabetes comparing low vs high amylase levels.

	Diabetes OR (95%CI) (N	r = 778)		
Model	Predictors	OR (95% CI)	P-value	Pseudo R ²
1	Q1 Amylase (<48 µ/L)	1.97[1.01-3.83]	0.04	0.052
	Q2 Amylase (48–61 µ/L)	1.59[0.83-3.05]	0.16	
	Q3 Amylase (62–79 µ/L)	0.85[0.40-1.82]	0.68	
	Q4 Amylase (80 µ/L)	1		
2	Q1 Amylase (<48 µ/L)	1.65[0.83-3.27]	0.15	0.066
	Q2 Amylase (48–61 µ/L)	1.43[0.73-2.75]	0.30	
	Q3 Amylase (62–79 µ/L)	0.83[0.39–1.76]	0.62	
	Q4 Amylase (80 µ/L)	1		
	BMI	1.07[1.00-1.11]	0.005	
3	Q1 Amylase (<48 µ/L)	1.48[0.73-2.98]	0.27	0.112
	Q2 Amylase (48–61 µ/L)	1.28[0.65-2.51]	0.48	
	Q3 Amylase (62–79 µ/L)	0.87[0.40-1.89]	0.73	
	Q4 Amylase (80 µ/L)	1		
	VAT	1.12[1.07–1.17]	< 0.001	
4	Q1 Amylase (<48 µ/L)	1.52[0.75-3.08]	0.24	0.113
	Q2 Amylase (48–61 µ/L)	1.29[0.65-2.55]	0.46	
	Q3 Amylase (62–79 µ/L)	0.89[0.41-1.92]	0.76	
	Q4 Amylase (80 µ/L)	1		
	BMI	0.98[0.92–1.04]	0.49	
	VAT	1.13[1.08–1.19]	< 0.001	
5	Q1 Amylase (<48 µ/L)	1.51[0.75-3.07]	0.25	0.117
	Q2 Amylase (48–61 µ/L)	1.30[0.66-2.57]	0.45	
	Q3 Amylase (62–79 µ/L)	0.87[0.40-1.89	0.73	
	Q4 Amylase (80 µ/L)	1		
	BMI	1.04[0.94–1.16]	0.45	
	VAT	1.13[1.07–1.18]	< 0.001	
	SAT	0.97[0.94–1.01]	0.16	

Diabetes: uncontrolled fasting plasma glucose 126 mg/dL. VAT: visceral adipose tissue area. All models were adjusted for age, ethnicity, and sex.

Table 3

<u>.</u> .
Jen
цп
ano
en
Ĩ
M
in
ıse
yl
am
th
Wi
ÅΤ
Š
and
Ľ
Ň
of
nts
cie
Ë
coe
n e
ssic
tes
reg
sd
ust
idji
e-2
abl
ari
ltiv
Aultiv

Models	Predictors	BMI β-coeff.	P-value	VAT A -coeff.	P-value	SAT β -coeff.	P-value	Model's R ²
Women (N = 400)							
-	BMI	-0.018 ± 0.003	<0.001					0.159
2	VAT			-0.189 ± 0.037	<0.001			0.158
3	SAT					-0.066 ± 0.011	<0.001	0.159
4	VAT + SAT			-0.126 ± 0.043	0.02	-0.045 ± 0.014	0.07	0.179
5	VAT + BMI	-0.0129 ± 0.004	0.02	-0.102 ± 0.048	0.02			0.176
9	SAT + BMI	-0.0134 ± 0.007	0.04			-0.023 ± 0.025	0.504	0.168
7	VAT + SAT + BMI	-0.0052 ± 0.075	0.49	-0.117 ± 0.048	<0.001	-0.023 ± 0.025	0.346	0.167
Men (N :	= 378)							
1	BMI	-0.020 ± 0.005	<0.001					0.112
2	VAT			-0.085 ± 0.032	0.00			0.106
3	SAT					-0.053 ± 0.019	0.02	0.094
4	VAT + SAT			-0.059 ± 0.037	0.102	-0.034 ± 0.022	0.251	0.112
5	VAT + BMI	-0.018 ± 0.004	0.009	-0.012 ± 0.04	0.738			0.123
9	$\mathbf{SAT} + \mathbf{BMI}$	-0.0271 ± 0.006	0.008			-0.033 ± 0.034	0.193	0.126
7	VAT + SAT + BMI	-0.026 ± 0.009	0.006	-0.012 ± 0.04	0.75	0.043 ± 0.033	0.195	0.126

Diabetes Res Clin Pract. Author manuscript; available in PMC 2018 March 02.

Data are presented as β -coeff.: beta coefficient ± SE; VAT: visceral adipose tissue area; SAT: subcutaneous adipose tissue area. All models were adjusted for age, ethnicity.