Lipids in Health and Disease

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Decoding metabolic connections: the role of salivary amylase activity in modulating visceral fat and triglyceride glucose index

Gita Erta[1*], Gita Gersone[1], Antra Jurka[1] and Peteris Tretjakovs[1]

Abstract

Background Salivary amylase activity (SAA) is recognized as a potential biomarker for metabolic health. Previous studies suggest an association between SAA and insulin sensitivity, but the mechanisms remain unclear. This study investigates the relationship between SAA, visceral fat (VF), and the triglyceride-glucose (TyG) index to clarify the pathways linking SAA to metabolic risk factors.

Methods This cross-sectional study analysed data from women of reproductive age who were classified as overweight. Linear regression models were used to assess associations between salivary amylase activity (SAA), visceral fat (VF) and the triglyceride-glucose (TyG) index, while adjusting for confounding variables such as age, body mass index (BMI), physical activity and dietary patterns. Mediation analysis was conducted to determine whether VF mediates the relationship between SAA and the TyG index.

Results Higher SAA was inversely associated with VF (β = -0.45, 95% CI: -0.65 to -0.25, $p < 0.001$). No direct association was observed between SAA and TyG index (β = -0.10, 95% CI: -0.25 to 0.05, $p = 0.18$) after adjustment for covariates. Mediation analysis revealed that visceral fat significantly mediated the relationship between SAA and the TyG index. The indirect effect of SAA on the TyG index through VF (A × B) was statistically significant (β = -0.16, 95% CI: -0.26 to -0.08), accounting for 45% of the total effect.

Conclusions These findings suggest that higher SAA may confer metabolic benefits by reducing VF, thereby indirectly influencing the TyG index. This highlights the critical role of VF in mediating the protective effects of SAA on metabolic health and provides insights into potential pathways for intervention.

Keywords Salivary amylase activity, Visceral fat, Triglyceride-glucose index, Metabolic health, Mediation analysis, Overweight, Biomarkers

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Background

Salivary amylase activity (SAA), an enzyme that initiates starch hydrolysis in the oral cavity, has attracted interest due to its potential influence on metabolic health. Emerging evidence suggests that variations in SAA activity can modulate key metabolic parameters, including visceral fat (VF) accumulation and insulin resistance (IR), thus contributing to individual differences in metabolic risk [1, 2].

Visceral adiposity, a well-established marker of metabolic risk, is strongly associated with IR and the development of cardiovascular diseases. The TyG index has emerged as a robust surrogate for assessing IR, with recent studies highlighting its strong correlation with visceral fat in diverse populations [3].

However, the relationship between SAA activity and metabolic health remains controversial. Some studies suggest that elevated SAA is associated with enhanced glycaemic control after starch ingestion, implying a protective metabolic role [2]. For example, a study in overweight and obese non-diabetic Qatari women demonstrated that higher SAA activity correlated with lower adiposity (BMI, waist, and hip circumference), elevated High-Density Lipoprotein cholesterol, and improved markers of systemic inflammation, such as reduced levels of CRP, TNF-α, and IL-6, as well as increased adiponectin and ghrelin levels [4]. On the contrary, other research has linked higher SAA activity with increased visceral adiposity and IR, indicating a potentially adverse metabolic impact [5].

These disparate findings suggest that SAA may exert sex- and adiposity-dependent effects on metabolic regulation, highlighting the complexity of its role in metabolic health.

This study aims to elucidate the associations between SAA activity, VF, and the TyG index in women of reproductive age classified as overweight. By investigating these interrelationships, we seek to clarify the mechanistic role of SAA in the modulation of metabolic risk and contribute to the identification of novel biomarkers for metabolic health.

Methods

Study design and participants

This cross-sectional study was conducted to evaluate the relationship between SAA, VF, and the TyG index. The study was carried out in Riga, Latvia, with participants recruited from a health centre during routine health check-ups. Importantly, these individuals were not patients but volunteers who agreed to participate in the study.

A total of 67 overweight women of reproductive age (18–45 years) with a BMI between 25 and 29.9 kg/m² were included in the study.

Exclusion criteria

Participants were excluded if they met any of the following criteria: pregnancy or lactation, a diagnosed chronic disease (e.g., diabetes, cardiovascular disease), or the use of medications that could affect metabolism.

The study protocol was approved by the Ethics Committee of Riga Stradiņš University (Ethics Committee number: 22 – 2/479/2021), and all participants provided written informed consent in accordance with the Declaration of Helsinki.

Participant assessments

Age Verified using the participant's identification card and recorded in years.

Body Mass Index (BMI): Calculated as weight (kg) divided by height squared (m²), using measurements taken with a calibrated scale and stadiometer.

Physical activity Assessed using the International Physical Activity Questionnaire (IPAQ)] and expressed as metabolic equivalent task (MET) minutes per week.

Dietary intake: Evaluated through a 24-hour dietary recall or food frequency questionnaire (FFQ), with macronutrient composition analyzed using the Finelli database.

Salivary amylase activity measurement

Unstimulated saliva samples were collected from participants following overnight fasting. The samples were processed within one hour after collection and stored at -80 ° C until analysis. Salivary amylase activity was quantified using an enzymatic kinetic assay based on the hydrolysis of a chromogenic substrate, with results expressed in U/mL.

Classification of high vs. low salivary amylase activity

To classify participants into high and low salivary amylase activity groups, we used the median value of salivary amylase activity within the study population as a cut-off point. Participants with values above the median were classified as having high activity, while those below the median were classified as having low activity.

Visceral fat measurement

VF was estimated using a bioimpedance analysis scale (BIA) (Omron BF511, Omron Healthcare, Kyoto, Japan). Participants were instructed to stand barefoot on the device, ensuring proper electrode contact, and to hold the handgrips for accurate impedance measurement. The measurement was performed in a fasting state (e.g., after an overnight fast) and following the manufacturer's guidelines. The device generates a visceral fat score using proprietary algorithms, which serves as an indicator of visceral fat percentage.

Triglyceride-glucose index calculation

The TyG index is a widely recognized marker of insulin resistance. In this study, the TyG index was calculated using a glucose-triglyceride calculator, where fasting glucose and triglyceride concentrations were entered in mmol/L, as these were the measurement units used in our laboratory.

This formula is an adaptation of the original calculation based on mg/dl units:

TyG index = ln (fasting triglycerides (mg/dl) x fasting glucose (mg/dl)/2.

To ensure consistency with the original method, we utilized direct calculations in mmol/L, thereby avoiding unit conversion. For authoritative references on the TyG index, refer to Guerrero-Romero et al. [6].

Fasting blood samples were collected for triglyceride and glucose measurements using standardized enzymatic methods. All laboratory analyses were conducted by blinded personnel to prevent bias.

Dietary intervention and nutritional composition

Participants followed a diet intervention for a 12-week period prior to metabolic evaluations. The low-starch diet prioritized non-starchy vegetables, lean protein sources, nuts, seeds, and healthy fats while minimizing the intake of grains and starchy vegetables. The calorie-restricted diet maintained a balanced macronutrient distribution but with a controlled reduction in total energy intake. Both dietary regimens were designed to ensure adequate micronutrient intake primarily through whole foods, with supplementation provided when necessary to meet recommended daily allowances (Table 1).

Table 1 Macronutrient and micronutrient composition of dietary interventions

Nutrients	Low-Starch Diet (per day)	Calorie-Restricted Diet (per day)
Energy (kcal)	~ 1800	~ 1200
Carbohydrates (%)	25–30	40–45
Carbohydrates (g)	110–135	120–150
Starch (g)	< 50	70–90
Sugars (g)	40–50	45–55
Fiber (g)	25–35	20–30
Protein (%)	25–30	20–25
Protein (g)	110–135	70–90
Fat (%)	40–45	30–35
Fat (g)	80–100	40–60
Saturated fat (g)	15–20	10–15
Monounsaturated fat (g)	30–40	15–25
Polyunsaturated fat (g)	15–20	10–15
Omega-3 (g)	1.5–2.5	1.0–2.0
Omega-6 (g)	8–12	6–10
Cholesterol (mg)	< 300	< 200
Sodium (mg)	2000–2500	1500–2000
Potassium (mg)	3500–4000	3000–3500
Calcium (mg)	1000–1200	900–1100
Magnesium (mg)	350–450	300–400
Iron (mg)	12–15	10–12
Zinc (mg)	10–12	8–10
Vitamin C (mg)	75–100	60–80
Vitamin D (IU)	800–1000	600–800
Vitamin B12 (µg)	2.4–3.0	2.0–2.5

Statistical analysis

Data were analyzed using GraphPad Prism 10 and R v4.4.2. Data normality was assessed using the Shapiro-Wilk test and visual inspection of histograms and Q-Q plots. Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range, IQR) based on data distribution. Categorical variables were presented as frequencies (percentages).

To examine the associations between salivary amylase activity (SAA), visceral fat (VF), and the triglyceride-glucose (TyG) index, we conducted multivariable linear regression models adjusted for potential confounders, including age, BMI, physical activity, and dietary intake. The regression model was specified as follows: $Y = \beta 0 + \beta 1 X + \beta 2 C + \varepsilon$.

Where *Y represents* the dependent variable (VF% or TyG index), *X* is the independent variable (SAA), *C* represents covariates, and ε is the error term. The strength of association was reported using standardized regression coefficients (β), 95% confidence intervals (CI), and p-values. Effect sizes were interpreted according to Cohen's guidelines, where $\beta \geq 0.1$ was considered a small effect, $\beta \geq 0.3$ a moderate effect, and $\beta \geq 0.5$ a large effect. We employed mediation analysis to assess whether VF% mediates the association between SAA and the TyG index, using structural equation modeling (SEM) with the lavaan package in R. The total effect (c), direct effect (c′), and indirect effect (a × b) were estimated using bootstrapping with 5,000 resamples to derive bias-corrected 95% confidence intervals (BCa 95% CI). A mediation effect was considered statistically significant if the 95% CI of the indirect effect did not include zero.

The proportion of mediation was calculated as $a \times b/c$, where a represents the effect of SAA on VF%, b represents the effet of VF% on TyG index, and c represents the total effect of SAA on TyG index.

Ethical considerations

All procedures were conducted in accordance with the Declaration of Helsinki. Data confidentiality was maintained by de-identifying participant information and securely storing it in a password-protected database. Participants were informed of their right to withdraw at any time without consequences.

Table 2 Baseline characteristics of the study population

Characteristic	Mean ± SD / Median (IQR)	Range	*n* (%)
Age (years)	29.18 ± 3.58	25–45	—
Gender -(Female)	67	—	100%
BMI (kg/m²)	27.8 ± 3.5	20.1–35.6	—
VF%	15.3 (12.1–18.5)	10.0–25.0	—
SAA (U/mL)	27.77 (10.64–56.24)	10.64–56.24	—
TyG Index	4.425	4.050–5.110	—
Physical activity	—	—	Sedentary:15 (22%) Moderate:35 (52%), Vigorous: 17 (26%)
Diet	-	-	Low starch: 30(50%) Calorie restriction:30(50%)

Table 3 Association between SAA, visceral fat, and TyG index (Multivariable linear Regression)

Predictor Variable	Outcome Variable	β Coefficient	95% CI	*p*-value
SAA	VF%	-0.45	-0.65 to -0.25	< 0.001
SAA	TyG Index	-0.10	-0.25 to 0.05	0.18
VF%	TyG Index	0.48	0.30 to 0.65	< 0.001

Results

Participant characteristics

A total of 67 participants were included in the analysis. The mean age was 29.18 ± 3.58 years, the mean BMI was 27.8 ± 3.5 kg/m².

There were no significant differences in baseline characteristics between participants with high and low salivary amylase activity (Table 2).

Correlation analyses

Salivary amylase activity (SAA) exhibited a significant inverse correlation with visceral fat percentage (VF%) (r = -0.301, 95% CI: -0.481 to -0.102, p = 0.026). However, no significant correlation was observed between SAA and the triglyceride-glucose (TyG) index (r = 0.125, 95% CI: -0.250 to 0.452, p = 0.438). In contrast, VF% demonstrated a significant positive correlation with the TyG index (r = 0.479, 95% CI: 0.215 to 0.679, p < 0.001).

Table 4 Mediation analysis: indirect effects of SAA on TyG index via visceral fat

Pathway	Effect Estimate (β)	95% CI	*p*-value	Proportion of Total Effect (%)
Direct Effect (SAA → TyG)	-0.10	-0.25 to 0.05	0.18	—
Indirect Effect (SAA → VF → TyG)	-0.16	-0.26 to -0.08	< 0.001	45%
Total Effect	-0.26	-0.37 to -0.15	< 0.001	100%

Multivariable regression

After adjusting for confounding variables, SAA remained a significant predictor of VF% (β = -0.45, 95% CI: -0.65 to -0.25, p < 0.001). In contrast, SAA was not a significant predictor of the TyG index (β = -0.10, 95% CI: -0.25 to 0.05, p = 0.18). VF% was independently associated with the TyG index (β = 0.48, 95% CI: 0.30 to 0.65, p < 0.001) (Table 3).

Regression Model Specifications:

The following linear regression models were used:

1. VF% as the dependent variable:
2. TyG index as the dependent variable:

Mediation analysis

A mediation analysis was conducted to examine whether VF% mediated the relationship between SAA and the TyG index. The indirect effect of SAA on the TyG index through VF% was statistically significant (β = -0.16, 95% CI: -0.26 to -0.08) (Table 4; Fig. 1).

We employed a bootstrapping approach with 5,000 resamples to estimate confidence intervals for indirect effects. The mediation model is represented as follows:

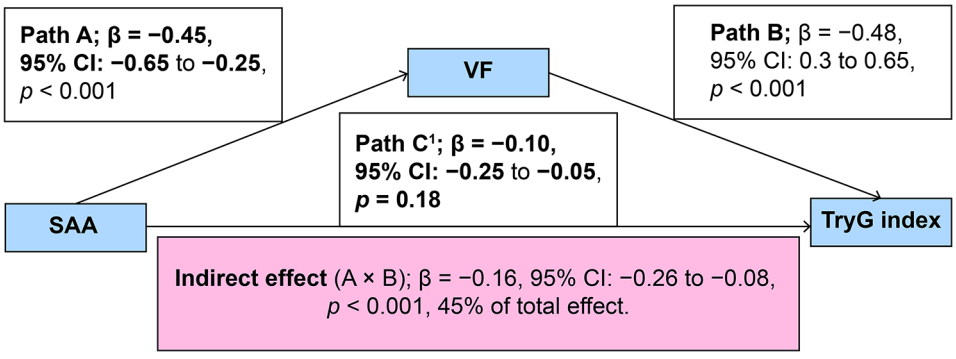

Fig. 1 Mediation analysis: indirect effects of SAA on TyG index via visceral fat

1. Effect of SAA on VF% (Path A):
2. Effect of VF% on TyG index (Path B):
3. Total effect of SAA on TyG index (Path C):
4. Direct effect of SAA on TyG index (Path C'):

Discussion

The findings of this study provide important insights into the role of SAA as a potential predictor of metabolic health. The inverse association between SAA and visceral fat, combined with the absence of a direct association between SAA and the TyG index, suggests a nuanced relationship in which VF mediates the effect of SAA on metabolic outcomes. Specifically, the mediation analysis revealed that 45% of the total effect of SAA on the TyG index could be attributed to VF, underscoring the critical role of adipose tissue distribution in linking SAA with IR.

The observed inverse association between SAA and VF aligns with emerging evidence suggesting that higher SAA is indicative of enhanced carbohydrate metabolism efficiency. SAA, an enzyme primarily responsible for starch digestion, has been hypothesized to contribute to glycemic regulation by modulating postprandial glucose responses. Previous studies have shown that individuals with higher SAA activity exhibit improved glucose tolerance, likely through a faster onset of carbohydrate digestion and absorption, which reduces prolonged postprandial hyperglycemia [7]. These effects may indirectly influence adiposity by reducing insulin demand and attenuating lipogenesis, mechanisms that warrant further investigation [8].

Contrary to the inverse association with VF%, no direct relationship between SAA and the TyG index was observed after adjusting for covariates. The lack of direct association in our study suggests that the influence of SAA on IR is primarily mediated by its effects on adiposity. This hypothesis is supported by the significant indirect effect of SAA on the TyG index through VF. Visceral adiposity, known for its endocrine activity and pro-inflammatory cytokine secretion, is a well-established mediator of metabolic dysfunction [9]. Our results extend this understanding by highlighting SAA as a potential upstream determinant of VF accumulation and, consequently, IR.

The mediation effect identified in this study underscores the importance of targeting visceral adiposity in metabolic health interventions. Given that SAA activity is influenced by dietary and genetic factors, it may represent a modifiable marker for predicting and managing visceral obesity. Emerging evidence suggests that dietary interventions targeting carbohydrate digestion, such as low-glycemic index diets, may modulate salivary amylase (SAA) activity and subsequently impact metabolic health outcomes [10].

From a mechanistic perspective, the link between SAA and VF may involve multiple pathways. Higher SAA activity facilitates efficient starch hydrolysis, leading to a rapid glucose supply and possibly influencing insulin dynamics [11]. Insulin, in turn, plays a critical role in regulating adipose tissue deposition. Additionally, SAA may exert systemic effects beyond digestion, as evidenced by its association with inflammatory markers and oxidative stress in studies examining stress physiology and metabolic health [12]. The interplay between these mechanisms and VF metabolism merits further exploration through experimental and longitudinal studies.

Potential mechanisms and unaccounted confounding variables

Several potential confounders could influence the observed relationships in our study. Genetic predisposition, particularly polymorphisms in the AMY1 gene, has been linked to interindividual variability in SAA activity and metabolic outcomes. Individuals with a higher AMY1 gene copy number tend to have elevated SAA activity, which may contribute to more efficient carbohydrate metabolism and lower risk of obesity and IR. Additionally, hormonal regulators such as insulin, cortisol, and catecholamines are known to modulate both SAA secretion and fat metabolism. Cortisol, for instance, is implicated in central fat accumulation, while catecholamines influence lipolysis. While our study adjusted for physical activity and dietary intake, these hormonal and genetic influences were not directly assessed, representing a limitation. Future studies incorporating genetic screening and endocrine profiling could provide further clarity on these mechanisms.

Clinical implications

Given the observed associations, SAA may serve as a novel biomarker for metabolic risk assessment. As SAA activity is influenced by dietary and genetic factors, it represents a potentially modifiable target for preventing or managing visceral obesity.

- Dietary interventions: Emerging evidence suggests that dietary modifications can influence salivary amylase (SAA) activity [10].
- Genetic screening: Assessing AMY1 gene copy number could help identify individuals with higher or lower SAA activity, enabling personalized dietary strategies.
- Metabolic risk prediction: Measuring SAA levels in clinical settings may improve early identification of individuals at risk for visceral obesity and insulin resistance.

In conclusion, this study contributes to the growing body of evidence on the metabolic relevance of SAA activity. The inverse association between SAA and VF%, along with the mediating role of visceral adiposity in the relationship between SAA and the TyG index, highlights the complex pathways linking carbohydrate digestion with systemic metabolic health. These findings support the potential utility of SAA as a biomarker for assessing metabolic risk and emphasize the need for targeted strategies to mitigate visceral adiposity in improving metabolic outcomes. Future research should aim to elucidate the molecular mechanisms underlying these associations and explore the translational potential of modulating SAA activity for metabolic health optimization.

Strengths and limitations

Strengths

Novel Insights: This study provides novel evidence linking SAA with VF% and the TyG index, contributing to the understanding of SAA as a potential biomarker for metabolic health.

Robust Methodology: The use of a mediation analysis allowed for a detailed investigation of the indirect effect of visceral fat, providing deeper insights into the pathways connecting SAA with IR.

Comprehensive Adjustment for Covariates: By controlling for a wide range of covariates, the study ensures that the observed associations are independent of confounding variables.

Clinical Relevance: The findings highlight the significance of VF as a mediator in metabolic dysfunction, suggesting potential avenues for targeted interventions and therapeutic strategies.

Limitations

Cross-Sectional Design: The cross-sectional nature of the study limits the ability to infer causality. Longitudinal studies are needed to confirm the directionality of the observed associations.

Unaccounted Confounders: While genetic and hormonal factors may influence SAA activity and metabolic outcomes, they were not directly assessed in this study. Future research should incorporate genetic profiling (e.g., AMY1 polymorphisms) and hormonal markers (e.g., insulin, cortisol, catecholamines) to better elucidate these relationships.

Lack of Mechanistic Data: Although mediation analysis suggests a pathway involving visceral fat, direct mechanistic studies were not performed to explore how SAA influences VF metabolism or IR.

Population Specificity: The study population may not be representative of broader demographics, such as different age groups, ethnicities, or individuals with pre-existing metabolic disorders, potentially limiting the generalizability of the findings.

Potential Measurement Bias: While SAA was assessed as a proxy for enzymatic function, factors such as hydration status, circadian variation, and acute stress, which can influence SAA, were not accounted for.

Limited Exploration of Dietary Influence: Although dietary factors were adjusted as covariates, the study did not comprehensively analyze how specific dietary patterns or macronutrient compositions might modulate SAA activity and its downstream effects.

Future directions Addressing these limitations in future research is essential for validating and expanding upon our findings. Longitudinal studies and interventional trials exploring the genetic and hormonal regulation of SAA activity could provide deeper mechanistic insights. Additionally, dietary intervention studies investigating the impact of carbohydrate intake patterns on SAA activity and metabolic outcomes would help translate these findings into practical applications for metabolic health optimization.

Conclusions

This study highlights the significant role of SAA in metabolic regulation, specifically its relationship with visceral adiposity and IR. Higher SAA was inversely associated with VF%, suggesting a potential protective role of enhanced carbohydrate digestion efficiency against visceral fat accumulation. While no direct association was observed between SAA and the TyG index, VF was found to mediate the relationship, accounting for a substantial proportion of the total effect. These findings underscore the complex interplay between enzymatic activity, adipose tissue distribution, and metabolic health.

The results suggest that SAA may serve as a novel biomarker for assessing VF-related metabolic risk and highlight visceral adiposity as a critical target for intervention. Future studies should explore the mechanistic pathways linking SAA with metabolic outcomes and investigate the potential of dietary and lifestyle modifications to optimize SAA activity for improving metabolic health. Ultimately, this research contributes to the broader understanding of metabolic regulation and offers insights into strategies for mitigating metabolic disorders through personalized approaches.

Abbreviations

BMI	Body Mass Index
IR	Insulin resistance
SAA	Salivary amylase activity
TyG	Triglyceride-glucose index
VF	Visceral fat

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Author contributions
Methodology, G.G.; software, A.J.; formal analysis, G.G.; investigation, G.E.; resources, A.J.; writing—original draft, G.E.; writing—review and editing, P.T.; supervision, G.G. and P.T. All authors reviewed and approved the final manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
The study protocol was approved by the Ethics Committee of Rigas Stradiņš University (Ethics Committee number: 22 – 2/479/2021).

Consent for publication
All participants provided their written informed consent in accordance with the Declaration of Helsinki.

Competing interests
The authors declare no competing interests.

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