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Variations in oral performance and processing behaviours among older adults: Associations with gastric emptying, postprandial glucose and insulin responses

Dimitra Zannidi [a], Lisa Methven [a], Jayne V. Woodside [b], Gerry McKenna [b], Ciaran G. Forde [c], Kim G. Jackson [a], Amir Shafat [d], Martin Schimmel [e], Miriam Clegg [a,f,*]

[a] Department of Food and Nutritional Sciences, University of Reading, Whiteknights, Reading, RG6 6DZ, UK
[b] Centre for Public Health, School of Medicine, Dentistry and Biomedical Sciences, Queens University Belfast, UK
[c] Sensory Science and Eating Behaviour, Division of Human Nutrition and Health, Wageningen University and Research, Wageningen, the Netherlands
[d] Physiology, School of Pharmacy and Medical Sciences, University of Galway, Galway, Ireland
[e] Department of Reconstructive Dentistry and Gerodontology, School of Dental Medicine, University of Bern, Bern, Switzerland
[f] School of Food and Nutritional Sciences, University College Cork, Cork, Ireland

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ABSTRACT

Older adults often experience deterioration in oral health and oral-related abilities, with tooth loss, impaired masticatory performance, alterations to salivary flow and composition all being common. Additionally, other ageing-related physiological changes happen, including delayed gastric emptying and higher postprandial glycemic responses. The interaction between oral-related factors and metabolic responses has been researched in younger adults, but not in older age groups. This study aimed to explore oral performance measures (OPMs) and oral processing behaviours (OPBs) and their associations with gastric emptying (GE) and postprandial glucose and insulin responses in adults aged ≥65 years. Participants attended one visit after an overnight fast and were provided with a fixed-portion breakfast, which they were asked to consume in full. Eating behaviours were coded to quantify OPBs (including chewing and eating rate). OPMs (salivary flow rate, salivary alpha-amylase, bolus saliva uptake and masticatory performance) and questionnaire data were collected. Over three postprandial hours, GE was measured using labelled breath samples, and glucose and insulin responses were measured in capillary blood samples. Increased bolus salivary uptake was associated with greater masticatory performance, greater stimulated salivary flow rate and a slower eating rate. Greater masticatory performance was related to faster GE times. Faster eating rates and reduced chewing were associated with lower early postprandial glucose responses (up to peak glucose (45 min)); however, they were not associated with postprandial insulin responses or GE. This research highlights the significant, complex associations between oral performance, oral processing behaviours and postprandial metabolism in older adults.

1. Introduction

Ageing is associated with a decline in oral health, and older adults often experience high levels of tooth loss, dental caries, periodontal disease, as well as reduced saliva flow, xerostomia and other oral conditions (Petersen and Yamamoto, 2005). The ageing-related oral health deterioration can be a result of both physiological changes as well as pathological and iatrogenic effects (McKenna and Burke, 2010). For instance, ageing affects the salivary glands and has been associated with reduced production (flow rate) of saliva (Xu et al., 2019), which is linked

Abbreviations: OPB(s), oral processing behaviour(s); OPM(s), oral performance measure(s); iAUC, incremental area under the curve; SD Hue, Standard Deviation of Hue; sAA, salivary alpha amylase; sTAPM, sAA total activity per minute; GE, gastric emptying; T_{half}, half time; T_{lag}, lag phase; T_{lat}, latency time; T_{asc}, ascension time.

* Corresponding author at: School of Food and Nutritional Sciences, University College Cork, Cork, Ireland.
E-mail addresses: d.zannidi@pgr.reading.ac.uk (D. Zannidi), l.methven@reading.ac.uk (L. Methven), j.woodside@qub.ac.uk (J.V. Woodside), g.mckenna@qub.ac.uk (G. McKenna), ciaran.forde@wur.nl (C.G. Forde), k.g.jackson@reading.ac.uk (K.G. Jackson), amir.shafat@universityofgalway.ie (A. Shafat), martin.schimmel@unibe.ch (M. Schimmel), mclegg@ucc.ie (M. Clegg).

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to higher salivary alpha amylase (sAA) concentration (Nagler and Hershkovich, 2005) and impaired masticatory performance in independently living older adults aged 60 and older (Ikebe et al., 2006). Chewing ability has been related to the number and distribution of remaining teeth (Naka and Anastassiadou, 2012), with impaired masticatory performance mostly observed in older adults with tooth loss (Fontijn-Tekamp et al., 2000) or those wearing dentures (Limpuangthip et al., 2021). At the same time, changes in the food oral processing behaviours (OPBs) have been observed, with older adults consuming their meals at slower eating rates and with more chews compared to younger adults (Ketel et al., 2020). All the above changes can affect the food choices, food intake and meal-related outcomes among older adults.

Oral performance measures (OPMs), such as dentition, masticatory performance and salivary flow rate, have been linked with oral processing behaviours (OPBs), such as eating rate and number of chews during a meal. However, these associations have primarily been studied in younger adults. Dental status as well as salivary flow appear to have a strong impact on OPBs: in younger adults, an increased number of teeth, a better masticatory performance and an increased salivary flow have been previously linked to shorter consumption times and chewing cycles, faster eating rates, smaller average bite size as well as fewer perceived chewing and swallowing difficulties (Fontijn-Tekamp et al., 2004; Iwasaki et al., 2016; Ketel et al., 2020; Samnieng et al., 2012). For older adults, the literature on the above relationships is limited; self-reported data have shown that impairments in ingestion function and activities (biting and chewing) increase significantly with age and are the highest in people over 80 years (Hennequin et al., 2024). Older adults may change their OPBs as compensatory strategies to manage oral impairments while eating, such as prolonging the chewing duration, increasing the number of chewing cycles, or swallowing larger food particles (Mioche et al., 2004; Peyron et al., 2004); however, this can lead to avoidance of food textures and minimise their food choices (Yannakoulia et al., 2018). Whether caused by ageing itself or adaptation, the changes in OPBs can affect the oral stage of digestion, but also the subsequent gastrointestinal responses to food.

OPBs during a meal can influence the quality of the food bolus at swallow (Goh et al., 2021b) and, through this, the gastric emptying (GE) rates as well as the postprandial glucose and insulin responses (Hoebler et al., 2000). Once again, these associations have mainly been determined in younger populations and have yet to be explored among older adults. Given the links between OPMs and OPBs, these interactions may have important implications for metabolic health. GE plays a critical role in postprandial metabolism, influencing approximately 35 % of the variance in the initial postprandial glycemia (Arunachala Murthy et al., 2023; Horowitz et al., 1993). In younger adults, consuming a meal with more chews has been associated with faster GE rates (Pera et al., 2002), while a faster GE rate has been positively related to plasma glucose concentration within the initial 30 min postprandially, but inversely related to glucose concentration at 120 min (Horowitz et al., 1993). A slower GE, on the other hand, was found to delay the appearance of the peak rate of postprandial glucose and increase insulin sensitivity in healthy younger (Hinshaw et al., 2014) and middle-aged adults (Otsuka et al., 2008). Slower eating rates and longer oral exposure times were significant predictors of higher early postprandial glucose responses in younger adults for the first 30 min post-meal (Goh et al., 2021a) and of higher postprandial insulin responses for the first 60 min post-meal (Goh et al., 2021a). It is unclear whether variations in older adults' OPBs and OPMs directly affect the gastrointestinal response to a meal or indirectly, through modification of GE.

Considering the ageing-related changes that affect oral function and postprandial metabolism — including impaired glucose and insulin regulation and slower GE — it becomes essential to explore the relationships between OPMs, OPBs, and postprandial responses in older adults. Understanding how eating-related behaviours influence post-meal metabolism in this population could help inform strategies to improve glycaemic control and overall metabolic health. However, to our knowledge, these associations have yet to be investigated in older adults, highlighting a gap in the literature and the need for further research.

Following the above, the aims and hypotheses of this study were:

1. To explore associations between OPMs and OPBs in healthy older adults. Specifically, and based on the literature summarised in the introduction, we hypothesised that:
   a. OPBs such as faster eating rate, bigger average bite size, shorter oral exposure time and fewer total bites, chews and swallows per portion, would be associated with more natural teeth, greater masticatory performance, greater salivary flow rates and bolus salivary uptake, and reduced sAA.
2. To explore the associations between OPMs and OPBs with GE and postprandial metabolic responses (glucose and insulin). Specifically, we hypothesised that:
   a. Slower eating rates and more chews per portion would be associated with higher postprandial glucose and insulin responses (measured by Incremental Area Under the Curve (iAUC)).
   b. Eating rate would not influence GE in older adults. Although a slower eating rate has been previously associated with slower GE in some studies in younger adults, we predict this will not hold true in older adults, as GE rate is already slower with age (Soenen et al., 2015).

2. Methods

2.1. Participants

The main study design has been described previously (Zannidi et al., 2025). Healthy male and female participants aged 65 years or older, living in the wider Reading (Berkshire, UK) area, were recruited. The study was advertised through the University of Reading volunteer databases, online platforms, poster advertisements and word of mouth. Participants showing interest in the study were contacted for a screening call, and if the eligibility criteria were met and they were willing to consent to the study, they were booked for the study visit. The inclusion and exclusion criteria have been described previously (Zannidi et al., 2025). Briefly, participants were excluded if diagnosed with dysphagia and/or any eating or swallowing difficulties, food allergies to foods provided on the study day, existing type 1 or type 2 diabetes, loss of appetite or no remaining natural teeth.

The study was performed in accordance with the Declaration of Helsinki and was granted a favourable opinion for ethical conduct from the University of Reading Research Ethics Committee (UREC 22/29, 22/10/22). The trial has been registered at clinicaltrials.gov (NCT05671003). Written informed consent was obtained from all participants at the beginning of the visit before any data collection commenced.

2.2. Study design

The study had a cross-sectional design. Participants attended a single morning visit (5 h) after a 12-hour overnight fast. Participants were asked to avoid intake of alcohol, caffeine-containing drinks and foods high in ^{13}C (i.e. corn, sugarcane) on the day before the visit, as well as to not have any dental work performed and to restrict any participation in intense physical activity.

2.3. Data collection and analysis

Fig. 1 shows the study visit timeline and measures completed by participants.

2.3.1. Breakfast meal and OPBs analysis

On the visit day, following an overnight fast, participants were

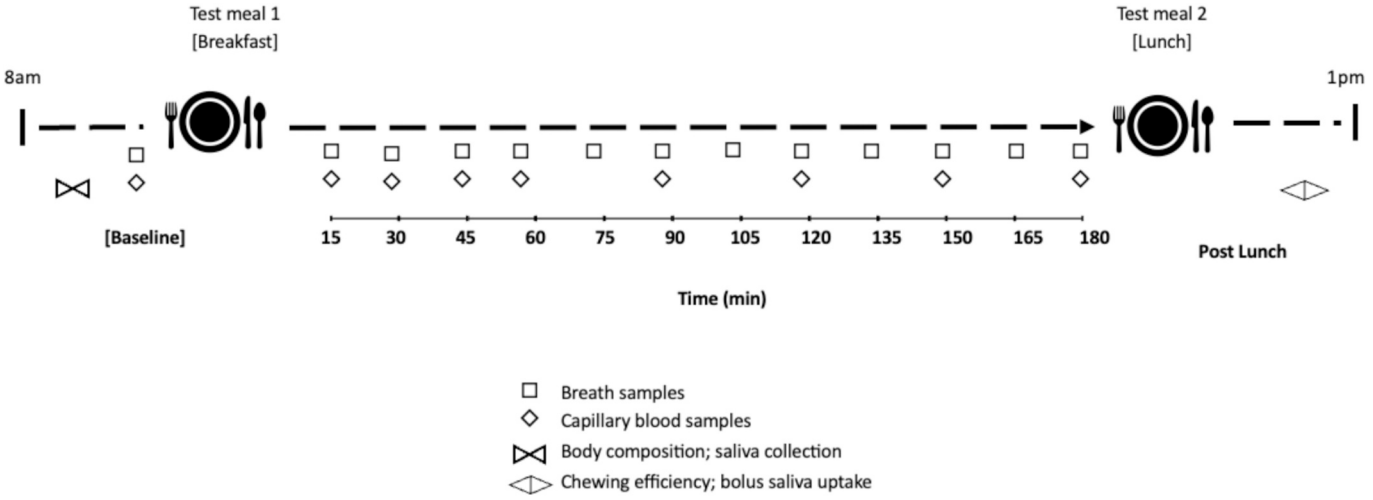

Fig. 1. Schematic representation of the visit timeline and methods.

provided with a fixed-portion breakfast meal of a "scrambled egg sandwich" at around 9 a.m., approximately one hour after arriving at the unit. The scrambled egg sandwich consisted of 2 slices (57.7 g in total) of white bread (Warburtons, UK), 8 g of salted (1.2 %) butter (Lurpak, UK) and 50 g of liquid egg (West Horsley Dairy, UK). The meal provided 265 kcal (1109 kJ), 12.4 g of fat, 26.5 g of carbohydrates and 10.7 g of protein. Participants were asked to consume the breakfast meal in full at their natural pace, and a 15-minute timeframe was allowed for consumption. All participants complied with the instructions.

OPBs were analysed from the video recordings taken during breakfast, as previously described (Forde et al., 2013). In summary, a laptop fitted with a webcam was placed approximately 30 cm away from the participants whilst they sat in a quiet sensory booth. Participants consumed the scrambled egg sandwich using their hands and were asked to drink water (a 250 mL glass was provided) either before or after consuming the sandwich to avoid confusion in data coding (see below). During the meal, the researcher was present but not visible to participants. Participants were informed that they would be video recorded whilst eating, but they were not able to see themselves on the camera.

The video recordings were coded using a behavioural annotation software (ELAN 4.9.1, Max Plank Institute for Psycholinguistics, The Language Archive, Nijmegen, the Netherlands) (Fogel et al., 2017). Frequencies of bites, chews and swallows were measured (Forde et al., 2013). Total oral exposure time (min) was simultaneously coded (total time the food spent in the mouth). The total meal duration time (min) was the sum of total oral exposure time and the time spent with no food in the mouth (inter-bite interval in minutes). Eating rate (g/min) was calculated by dividing the food consumed (117 g) by the oral exposure time (min). Average bite size (g/bite) and number of chews taken per bite (chews/bite) were also calculated. All coding was completed by a trained researcher (DZ). Validation coding was conducted by a second, trained researcher (MC) for a minimum of 10 % of the total coded videos and had to show at least 80 % agreement between coders for the data to be accepted for analysis.

Following the completion of the breakfast meal, the timer started, and the time of all subsequent measurements is given from this point. Three hours after the breakfast meal, participants were provided with an ad libitum lunch meal to assess food intake. The ad libitum lunch meal, other appetite-related methodologies of the study, as well as the nutritional information and components of both test meals, have been described previously (Zannidi et al., 2025).

2.3.2. Body composition

Participant's weight (kg), height (m), lean body mass (kg and %) and fat mass (kg and %) were collected on arrival using bioimpedance scales (Tanita Europe BC–418 MA; Amsterdam, the Netherlands) while participants were in fasted state, and participants body mass index (BMI) was calculated.

2.3.3. Saliva collection and analysis for unstimulated and stimulated salivary flow rate and salivary alpha amylase activity

After body composition measurements and while participants were still in a fasted state, saliva samples were collected using the passive drooling method (Granger et al., 2007). More specifically, saliva samples were collected between 8.15 and 8.45 a.m. to avoid circadian variations in salivary flow rate and composition. For unstimulated saliva measurements, participants were instructed to let any saliva they had in the mouth flow naturally into a pre-weighed collection vial (5 mL, Salimetrics LLC), while gently leaning their head forward over a period of 5 min. This measurement was taken in duplicate, with a 2 min rest between collections. Then, and following another 2 min rest, stimulated saliva was collected using the above process whilst chewing on a piece of parafilm® (5 × 5 cm) for a 3 min period. The parafilm was removed from the mouth separately, and the vial containing saliva was weighed. From the weights collected, salivary flow rates were calculated as mL/min, using the assumption that 1 g of saliva equates to 1 mL. All saliva samples were stored on ice pending analysis.

The average salivary alpha amylase (sAA) activity (U/mL) for each participant was determined from the stimulated saliva, using an enzymatic kit (Salimetrics, LLC) and a Tecan SPARK plate reader (Tecan UK Limited, UK). Additionally, the sAA total activity per minute (sTAPM) was calculated as: sTAPM (U/min) = sAA activity per unit volume (U/mL) * stimulated salivary flow rate (mL/min).

2.3.4. Blood glucose and insulin analysis

Blood samples were collected using capillary blood, a consistent and validated method for measuring blood glucose and insulin responses (Brouns et al., 2005). Baseline samples were collected before the breakfast meal, while participants were in a fasted state. The next samples were collected after consumption of the breakfast meal, at the timepoints shown in Fig. 1. Blood was collected by finger prick using single-use lancets. For the total nine finger pricks, the index, middle and fourth fingers were used (with some fingers being used twice, in different parts of the fingertip). Before each finger prick, subjects were requested to wash their hands with warm water to improve blood flow. Fingertips were not squeezed to extract blood but were instead gently massaged starting from the base of the hand and moving toward the tips, to ensure minimal plasma dilution. The first two blood drops were discarded, and the third drop was used for testing blood glucose.

Blood glucose concentration (mmol/L) at each time point was

measured using one drop of capillary blood collected into a micro-cuvette (HemoCue Ltd., UK), which was then analysed with a glucose dehydrogenase assay kit (HemoCue Ltd., UK). Following that, with the same collection technique described above, a further 100 μL of blood was collected in vials treated with di-potassium EDTA and kept on ice. Immediately after the end of the study visit, the collected blood samples were centrifuged at 4200 x*g*, for 15 min, at 4 °C, and the plasma was stored at −20 °C for insulin analysis. Analysis was performed within 6 months of the blood collection date, and the plasma insulin concentration (pmol/L) was measured using a Simple Plex insulin cartridge and Ella automated ELISA platform (Bio-techne, Abingdon, UK).

2.3.5. ^{13}C octanoic acid breath test (^{13}C-OABT) for GE

One hundred milligrams of 1–^{13}C octanoic acid (CK Isotopes Limited, Leicestershire, UK) were mixed with the liquid egg of the breakfast meal, as per the methodology and standard meal of Ghoos et al. (1993). ^{13}C-OABT is a safe, reliable and valid method for measuring GE (von Gerichten et al., 2022). ^{13}C labelled octanoic acid is rapidly absorbed in the duodenum and emerges in the breath as completely oxidised ^{13}C labelled carbon dioxide ($^{13}CO_2$) (von Gerichten et al., 2022). The addition of octanoic acid in the breakfast meal was not noted to affect the taste [as defined by the overall mean liking score by the participants, which was 63 (SD 20) out of 100 on VAS ratings] or the physical characteristics of the meal (as determined by the researchers).

Baseline breath samples were taken prior to breakfast meal consumption, and further samples were collected every 15 min for three hours after the breakfast meal, at the timepoints shown in Fig. 1. Breath samples were collected by blowing gently into a 12 mL Exetainer® (Labco Ltd., UK) using a straw, while wearing a nose clip, and replacing the cap just prior to the end of exhalation.

An isotope ratio mass spectrometer (ABCA, Sercon LTD, Cheshire, UK) was used to determine the ratio of $^{13}CO_2$ recovered in each breath sample, relative to a single point calibration cylinder gas [5 % CO_2 95 % N_2, −37.17 ± 0.04 Delta Vienna Pee-Dee Belemnite (δVPDB) which was measured against NBS-19; n = 15, Iso-analytical, Crewe, UK] (Werner and Brand, 2001). Abundance in δVPDB units was converted to atom fraction and used to calculate GE. Data were displayed as the percentage of $^{13}CO_2$ dose recovered per hour and the cumulative percentage $^{13}CO_2$ dose recovered over time. Carbon dioxide production was assumed to be 300 mmol/m^2 body surface area per hour (Shreeve et al., 1970). To calculate the participants' body surface area, a validated weight-height formula was used (Haycock et al., 1978), and the findings were fitted into a GE model developed by Ghoos et al. (1993). The formulae in the GE model was used to calculate the lag phase (T_{lag}), which is time taken to maximal rate of $^{13}CO_2$ excretion, the half time (T_{half}), which is the time it takes for 50 % of the ^{13}C dose to be excreted, the latency phase (T_{lat}), which is the point of intersection of the tangent at the inflection point of the $^{13}CO_2$ excretion curve representing an initial delay in the excretion curve (i.e., the time for the solid phase of the meal to reach a specific point in the digestive track after it is initially consumed), and the ascension time (T_{asc}), which is the time course between the T_{lat} and T_{half}, representing a period of high $^{13}CO_2$ excretion rates (Clegg and Shafat, 2010; Jackson et al., 2004; Schommartz et al., 1998).

2.3.6. Bolus salivary uptake

Bolus salivary uptake samples were collected following the lunch meal, after participants had rinsed the mouth with water and had taken a short rest. A 5 g piece of toasted white bread was used, and the procedure was repeated twice. Participants were instructed to chew the bread until the point of swallowing and then expectorate into a pre-weighed vial. Immediately after, they were provided with 25 g of water to rinse their mouth and then expectorate into the same vial. Expectorated bolus samples were weighed to record the weight of wet bolus (g) using the following approach: Wet bolus (g) = (weight of rinsing water (g) + wet bolus (g) + container (g)) – (weight of rinsing water (g) − weight of container (g)). Bolus salivary uptake (%) was calculated using the formula: Bolus salivary uptake (%) = ((Weight of wet bolus (g) – weight of food sample (g)) / weight of food sample (g)) x 100.

2.3.7. Masticatory performance

After the bolus salivary uptake method, the masticatory performance test was conducted, using the two-colour chewing gum test. Procedures have been described previously (Schimmel et al., 2007; Schimmel et al., 2015). In brief, one blue and one pink chewing gum (Hue-Check Gum©, University of Bern, Switzerland) were manually stuck together by slightly wetting them with water and applying moderate manual force. Participants were asked to chew the gum complex 20 times, and then the specimen was retrieved from the oral cavity by the researcher. The chewed specimen was placed in a non-reflective plastic bag, flattened to one mm thickness using a resin template, and subsequently scanned with a flatbed scanner from both sides. Images of both sides were evaluated opto-electronically using the ViewGum© software (Halazonetis et al., 2013). The software converts the images of the specimens into the HSI (Hue, Saturation, Intensity) colour characteristics and calculates the homogeneity of the colour mixture as the circular variance of Hue (SD Hue, range 0–1). Well-chewed specimens with a high degree of colour mixture present with a low SD Hue and vice versa.

2.3.8. Questionnaires

Questionnaire data were collected and managed using REDCap electronic data capture tools hosted at the University of Reading (Harris et al., 2019; Harris et al., 2009). Questionnaire data reported in this paper included data from the Xerostomia Inventory (XI) (Thomson et al., 1999) and a self-reported tooth counting questionnaire, which included information on denture wearing, number of dentures and teeth and overall oral condition (Allen et al., 2005) (Supplementary Material A).

The XI is a validated tool to assess chronic xerostomia and consists of an 11-item summated rating scale with each response to assign a score between 1 and 5, and the combined total score to be calculated into a sum ranging from 11 to 55, with higher scores indicating more severe symptoms (Thomson et al., 1999).

2.4. Statistical analysis

To define the sample size, power calculations were undertaken through an online power calculator (ClinCalc, 2024) and using a single study group design, continuous primary endpoint, 80 % power and alpha <0.05. The calculations were based on previous, published research in young adults (Li et al., 2011) and details on power calculations, which were based on the primary outcome of food and energy intake from the ad libitum meal, have been described previously (Zannidi et al., 2025).

Variables were tested for normality both visually and statistically using the quantile-quantile plot (normal Q-Q plot) and histogram prior to any statistical comparison. All variables were assessed for outliers using Tukey's method. When outliers were detected, they were excluded from the analysis and the total number of cases analysed in each test is reported in the results. Data are presented as mean and standard deviation (SD), unless otherwise stated. Independent samples *t*-tests were used to assess the significance of observed sex differences in continuous variables, and the Chi-square test of independence and Pearson's Phi coefficient to assess the significance of associations in categorical variables. Effect sizes were explored and expressed using Cohen's d for all continuous variables (with 0.2 to indicate small effect size, 0.5 medium effect size and 0.8 large effect size) and Cramer's V for all categorical variables (with a range from 0 to 1 and higher values to indicate a stronger correlation between the two variables).

The first aim was to explore associations between the OPMs (salivary flow rate, salivary amylase activity, number of teeth, masticatory performance) and OPBs (eating rate, average bite size, number of bites, number of chews, number of swallows) using a bivariate (Pearson)

correlation. Data are presented using a correlation matrix. To protect the family-wise error rate (Type 1 Error) in the total 21 pairwise correlations explored, the level of significance was adjusted using Bonferroni Correction ($p < 0.002$). Linear regression models were run between all OPMs with (a) eating rate, (b) average bite size, (c) number of bites, (d) number of chews and (e) number of swallows, while age and sex were used as covariates in all models. Results are presented as unstandardized regression coefficient (B), standard error of coefficient (SE B) and *p* values.

The second aim was to explore the associations between OPMS and OPBs with GE and postprandial metabolic responses (glucose and insulin). To explore this aim and the two associated hypotheses, linear regression models were run between (a) each OPM and OPB and postprandial glucose, expressed as incremental area under the curve (iAUC), (b) each OPM and OPB and postprandial insulin iAUC, (c) each OPM and OPB and each of GE parameters (T_{half}, Tl_{ag}, T_{lat}, T_{asc}), while age and sex were used as covariates in all models. Additionally, in order to account for the different phases of postprandial response, we calculated the incremental area under the curve (iAUC) for both glucose and insulin using the trapezoid rule and ignoring the area below the baseline, from 0 to 180 min but also for the following time segments of interest: early postprandial phase 0 to 45 min (time to average glucose peak concentration) and later postprandial phase 45 to 180 min (time after average glucose peak concentration). Results are presented as unstandardized regression coefficient (B), standard error of coefficient (SE B) and p values.

All statistical analyses were conducted in SPSS Version 27.0, IBM Corp., 2020. Armonk, NY (IBM, 2020) and a p value <0.05 was considered statistically significant.

3. Results

3.1. Descriptive characteristics of the sample population

Eighty-eight participants were included in this study, 50 % males (*n* = 44) with a mean age of 74 (SD 5) years. Participant characteristics are presented in Table 1. Variability is shown as standard deviation (SD) in all OPBs. Additionally, comparisons by sex are presented. Males (n = 44), compared to females (n = 44), had significantly greater weight and BMI, unstimulated and stimulated salivary flow rates and postprandial overall insulin responses (as expressed by iAUC). Females compared to males had slower gastric emptying postprandially (as expressed by higher GE T_{half}) and greater sAA activity; however, there were no significant differences in sTAPM (U/min) between males and females.

3.2. Correlations between OPMs (Aim 1)

Table 2 presents the Pearson's correlations observed between OPMs (Table 2). Unstimulated and stimulated salivary flow rates were positively correlated. Greater stimulated salivary flow was moderately correlated with greater bolus salivary uptake, while an inverse moderate correlation was found between the unstimulated salivary flow rate and sAA. A moderate strength correlation was found between higher values of Variance of Hue, indicating poorer masticatory performance, with a lower number of natural teeth and a weak correlation was observed between poorer masticatory performance and lower salivary uptake (Table 2).

3.2.1. Correlations of OPBs with OPMs (Hypothesis 1a)

Bolus salivary uptake was negatively associated with eating rate (B = -0.032, $p = 0.029$, $N = 76$), in the regression model that included the covariates of age and sex, indicating that greater bolus salivary uptake was associated with slower eating rate (Supplementary Table 1). Neither eating rate or number of chewing cycles were significantly linked to masticatory performance ($p > 0.05$, Supplementary Table 1).

No further significant associations were shown between OPBs (eating

Table 1
Descriptive characteristics of all sample population ($n = 88$) and comparisons between males (n = 44) and females (n = 44).

Participant characteristics	All, n = 88	Males, n = 44	Females, n = 44	Significance of sex differences (*p* value)	Effect size of sex
Age (years)	74 (5)	74 (6)	73 (4)	0.289	
Body weight (kg)	73.6 (15.1)	83.9 (12.8)	63.2 (8.9)	<0.001	1.87
BMI (kg/m^2)	25.1 (3.0), n = 86	26.2 (2.4), n = 42	24.0 (3.1), n = 44	<0.001	0.75
Salivary flow rate (mL/min)					
Unstimulated salivary flow rate	0.32 (0.19), n = 85	0.38 (0.19), n = 42	0.25 (0.16), n = 43	0.002	0.71
Stimulated salivary flow rate	1.51 (0.90)	1.72 (0.96)	1.31 (0.80)	0.033	0.46
SD Hue for masticatory performance	0.31 (0.15)	0.31 (0.16)	0.30 (0.16)	0.801	
Bolus salivary uptake[a] (%)	48.4 (40.4), n = 80	52.1 (45.0), n = 39	44.8 (35.7), n = 41	0.425	
sAA (U/mL)	91.9 (53.7), n = 85	77.7 (41.8), n = 43	106.5 (60.8), n = 42	0.012	0.53
sTAPM (U/min)	132.5 (109.8)	136.1 (119.5)	129.0 (100.5)	0.764	
Presence of partial dentures				0.401	
Yes (%, N)	17.0 (15)	20.5 (9)	13.6 (6)		
No (%, N)	83.0 (73)	79.5 (35)	86.4 (38)		
Number of natural teeth (N)	24 (4), n = 86	24 (5), n = 43	25 (4), n = 43	0.241	
XI score	22.6 (6.7)	21.7 (5.6)	23.5 (7.4)	0.193	
Gastric emptying parameters (min)					
T_{half}	70.9 (21.1), n = 85	65.3 (18.4), n = 41	76.1 (22.3), n = 44	0.018	0.22
T_{lag}	33.1 (10.4), n = 87	32.4 (12.0), n = 43	33.8 (8.7), n = 44	0.551	
T_{lat}	37.5 (7.8), n = 87	37.3 (8.8), n = 43	37.7 (6.8), n = 44	0.798	
T_{asc}	104.8 (17.0), n = 81	101.4 (14.9), n = 41	108.3 (18.4), n = 40	0.065	
Glucose iAUC (min*mmol/L)					
0–45 min	39.7 (20.2)	38.9 (18.8)	40.5 (21.7)	0.707	
45–180 min	56.4 (43.2), n = 84	60.8 (43.8), N = 42	52.0 (42.6), N = 42	0.354	
0–180 min	94.5 (52.2), n = 84	98.2 (53.1), n = 42	90.7 (51.7), n = 42	0.515	
Insulin iAUC (min*pmol/L)					

(*continued on next page*)

Table 1 (*continued*)

Participant characteristics	All, n = 88	Males, n = 44	Females, n = 44	Significance of sex differences (*p* value)	Effect size of sex
0–45 min	4110 (1593), n = 82	4075 (1582), n = 41	4146 (1622), n = 41	0.842	
45–180 min	7467 (3402), n = 83	8555 (3685), n = 42	6352 (2700), n = 41	0.003	0.68
0–180 min	11,467 (3600), n = 81	12,344 (3692), n = 40	10,610 (3333), n = 41	0.029	−0.26

Data are expressed as mean (SD); Independent samples *t*-test; p value < 0.05 is considered statistically significant. XI: Xerostomia index, iAUC: incremental area under the curve; sAA: salivary alpha-amylase; sTAPM: sAA total activity per minute; SD Hue: Standard Deviation of Hue; GE: gastric emptying; T_{half}: half time; T_{lag}: lag phase; T_{lat}: latency time; T_{asc}: ascension time; $p < 0.05$ indicates statistically significant differences between sexes; data are presented for n = 88 (all), n = 44 (males) and n = 44 (females) unless outliers are excluded, and in such cases, the actual sample size is indicated.

[a] Missing data: due to incomplete recovery of bolus samples in 8 participants.

rate, oral exposure time, number of chews and bites) and the other OPMs of number of natural teeth, presence of dentures, unstimulated salivary flow, stimulated salivary flow, sAA and bolus salivary uptake in all regression models that included age and sex ($p > 0.05$) (Supplementary Table 1).

3.3. Glucose and insulin (Aim 2)

In the whole group ($n = 88$), the mean fasting concentration of glucose was 4.7 (0.5) mmol/L and insulin was 37.2 (22.6) pmol/L. The average time to reach the postprandial peak concentration for both glucose and insulin was calculated for each participant and found to be 45 (SD 15) min postprandially. Supplementary Fig. 1(a–b) shows the incremental change in postprandial glucose and insulin responses over time (180 min) from baseline (0 min).

3.3.1. OPMs and postprandial glucose and insulin responses

Higher postprandial glucose and insulin responses between 0 and 180 min (expressed as $iAUC_{0–180\ min}$) were associated with higher sAA, when accounting for age and sex in the regression model (Table 3). No other OPMs showed significant associations with either glucose or insulin $iAUC_{0–180\ min}$ (Supplementary Table 3). When the iAUC for the early (0–45 min) and later (45–180 min) postprandial responses were explored, sAA was positively associated with glucose $iAUC_{0–45\ min}$ and insulin $iAUC_{45–180\ min}$, indicating that higher sAA was linked to higher early postprandial glucose response and higher later postprandial insulin response (Supplementary Table 3).

3.3.2. OPBs and postprandial glucose and insulin responses (Hypothesis 2a)

OPBs (eating rate, number of chews, bites and swallows) did not show associations with either glucose or insulin responses between 0 and 180 ($iAUC_{0–180\ min}$). When the early and later postprandial responses were explored (before and after the peak), a significant negative association was found between eating rate and glucose $iAUC_{0–45\ min}$ and a positive association between number of chews and glucose $iAUC_{0–45\ min}$ (Table 4). This indicates that for the timeframe before the postprandial glucose peak, a faster eating rate and a smaller number of chews were associated with lower glucose iAUC.

3.4. OPMs and OPBs associations with GE (Hypothesis 2b)

Higher *SD Hue* (indicating lower masticatory performance) was positively associated with higher GE T_{lag} ($B = 0.103$, $p = 0.014$, $n = 87$), indicating that lower masticatory performance was associated with prolonged GE T_{lag} phase. None of the other OPMs or OPBs were significantly associated with any GE parameter in the regression models, when age and sex were also included (all $p > 0.05$) (Supplementary Table 3).

It was noted that GE parameters T_{half}, T_{lag} and T_{asc} were all significantly negatively associated with postprandial glucose response ($iAUC_{0–180\ min}$) and were significantly positively associated with

Table 3
Multivariable regression of OPMs with overall postprandial glucose and insulin responses (total iAUC).

	Glucose $iAUC_{0–180\ min}$ (min*mmol/L)			Insulin $iAUC_{0–180\ min}$ (min*pmol/L)		
	B	SE B	P value	B	SE B	P value
Unstimulated salivary flow rate (mL/min)	−22.3, n = 82	32.6	0.490	730, n = 82	218.9	0.740
Stimulated salivary flow rate (mL/min)	−11.3	6.70	0.098	−169	448	0.708
SD Hue for masticatory performance	5.35, n = 84	36.5	0.884	−1691, n = 81	2383	0.480
Bolus salivary uptake (%)	−0.13, n = 77	0.15	0.398	2.74, n = 76	9.86	0.780
sAA (U/mL)	0.24, n = 82	0.11	0.038	15.5, n = 78	7.41	0.040

Age and sex are included as covariates in the model. iAUC: incremental area under the curve; sAA: salivary alpha amylase; SD Hue: Standard Deviation of Hue; B: unstandardized regression coefficient; SE B: standard error of coefficient; p value < 0.05 is considered statistically significant. Data are presented for n = 88 unless outliers are excluded and, in such cases, the actual sample size is indicated.

Table 2
Correlation matrix between OPMs.

	Unstimulated salivary flow rate (mL/min)	Stimulated salivary flow rate (mL/min)	Bolus salivary uptake (%)	sAA activity (U/mL)	SD Hue for Masticatory performance	Number of teeth (N)
Age (years)	0.020	−0.122	−0.243	0.162	0.172	−0.296
Unstimulated salivary flow rate (mL/min)		0.585*	0.222	−0.331*	−0.072	0.138
Stimulated salivary flow rate (mL/min)			0.470*	−0.261	−0.121	0.129
Bolus salivary uptake (%)				−0.178	−0.242*	0.167
sAA activity (U/mL)					0.182	−0.158
SD Hue for masticatory performance						−0.417*

Results presented when outliers are excluded. Values expressed as Pearson's r. SD Hue: Standard Deviation of Hue; sAA: salivary alpha amylase; note that higher masticatory performance is indicated by a lower SD Hue score.

* $p < 0.002$, which was considered statistically significant after adjustment for Bonferroni Correction.

Table 4
Multivariable regression between OPBs (Eating rate, Number of Chews) and incremental area under the curve for the postprandial glucose and insulin responses.

	Eating rate (g/min)			Chews (N)		
	B	SE B	P value	B	SE B	P value
Glucose $iAUC_{0–45\ min}$ (min*mmol/L)	−1.02, n = 84	0.42	0.018	0.44, n = 86	0.02	0.030
Glucose $iAUC_{45–180\ min}$ (min*mmol/L)	−1.02, n = 80	0.92	0.272	−3.90, n = 82	0.04	0.999
Glucose $iAUC_{0–180\ min}$ (min*mmol/L)	−2.01, n = 80	1.10	0.072	0.04, n = 82	0.05	0.428
Insulin $iAUC_{0–45\ min}$ (min*pmol/L)	−28.4, n = 78	36.1	0.434	2.07, n = 80	1.62	0.207
Insulin $iAUC_{45–180\ min}$ (min*pmol/L)	24.1, n = 79	71.9	0.738	−1.00, n = 81	3.24	0.758
Insulin $iAUC_{0–180\ min}$ (min*pmol/L)	−48.0, n = 77	79.0	0.546	1.85, n = 79	3.52	0.601

Age and sex are included as covariates in the model. iAUC: incremental area under the curve; B: unstandardized regression coefficient; SE B: standard error of coefficient; $p < 0.05$ is considered statistically significant. Data are presented for $n = 88$ unless outliers are excluded and, in such cases, the actual sample size is indicated.

postprandial insulin response ($iAUC_{0–180\ min}$), in the regression models when age and sex were also included (Supplementary Table 4). This indicates that slower GE (specifically, when taking longer to reach half time, lag and ascension phases) was linked to lower postprandial glucose and slower GE (as defined by T_{lat}) was associated with higher postprandial insulin.

4. Discussion

This study explored OPMs and OPBs as well as their association with GE and postprandial metabolic responses in community-living older adults (≥ 65 years). We found that a faster eating rate was associated with reduced bolus salivary uptake (Hypothesis 1). We further explored associations of OPMs and OPBs with postprandial metabolic responses and GE (Hypothesis 2), where we found significant positive associations between sAA activity and higher postprandial glucose and insulin responses (expressed as iAUC). Overall, no other OPM or OPB were related to postprandial glucose and insulin responses. However, when we explored the early and later postprandial responses separately, defined by the time before and after reaching the glucose peak concentration ($iAUC_{0–45\ min}$ and $iAUC_{45–180\ min}$), it was shown that for the time before the peak, a faster eating rate and a smaller number of chews were associated with lower early glucose response ($iAUC_{0–45\ min}$).

The study showed significant associations among OPMs. More specifically, unstimulated and stimulated salivary flow rates were positively correlated, while a higher stimulated salivary flow rate was related to an increased bolus salivary uptake and a higher unstimulated salivary flow rate to a lower sAA activity. Previous studies have reported associations between salivary flow rate, masticatory performance and age (Affoo et al., 2015; Ikebe et al., 2006). Consistent with prior research (Fontijn-Tekamp et al., 2000; Naka and Anastassiadou, 2012), we found that a greater number of natural teeth was positively correlated with better masticatory performance. The observed negative association between sAA and unstimulated salivary flow supports previous observations of increased sAA due to decreased total saliva volume in older populations (Nagler and Hershkovich, 2005). Additionally, bolus saliva uptake was positively linked to both improved masticatory performance and higher salivary flow rate, highlighting its significance and potential use as a marker of oral ability in older adults. In our study, faster eating rate was associated with reduced bolus salivary uptake, which may negatively affect the efficiency of the oral phase of ingestion; however, this could also affect the postprandial glycemia as explained below.

Previous literature has shown that in younger adults, the degree of mastication influences the amplitude of the postprandial initial glucose response and can induce a higher early insulin response (Ranawana et al., 2010); increased mastication was also shown to increase the total bolus surface and bolus salivary uptake (Goh et al., 2021b). In our study, slower eating rate and increased chewing were associated with higher glucose responses during the early postprandial phase, which could suggest that foods promoting a faster eating rate might reduce postprandial glucose responses in older people. At the same time, an opposite link was noted, as slower eating rate was related to increased bolus salivary uptake, which has been previously shown to change the kinetics of glucose release and promote insulin secretion (Hoebler et al., 2000). This suggests that a slower eating rate could assist postprandial euglycemia. However, no direct association was shown between bolus saliva uptake and postprandial glycemic responses in our study, which could highlight the direct significant association between OPBs and postprandial glycemia.

When exploring associations between OPMs and GE parameters, greater masticatory performance was linked to shorter initial delay in the stomach (shorter T_{lag}). However, in agreement with hypothesis 2b, neither eating rate nor number of chews was associated with GE. In the study of Pera et al. (2002) in younger adults, T_{lag} and T_{half} were significantly lower (i.e., GE was faster) when the test meal was chewed more. However, findings in older adults with complete dentures did not show associations between GE and mastication (Poitras et al., 1995). GE slows with age (Soenen et al., 2015), which may mask the effect of OPBs on GE rates.

In our study, faster GE was associated with higher postprandial glucose ($iAUC_{0–180\ min}$) and lower postprandial insulin responses ($iAUC_{0–180\ min}$). The review of Marathe et al. (2013) highlighted the complex relationships between gastric emptying and glycemia, showing that more rapid GE increases postprandial glycemia, possibly also through the GLP-1 and GIP secretion that promote insulin release; however, the relation between faster GE and greater insulin release is in contrast with our findings and may reflect the age-related changes in GE as well as in insulin sensitivity and glucose tolerance. As GE was not directly related to eating rate or number of chews in our study, more research is required to identify the combined associations between the above parameters (OPBs and GE) in postprandial glycemia.

Lastly, several sex related differences in OPMs were also explored in this study. The higher stimulated and unstimulated salivary flow rates found in males compared to females were consistent with previous findings and have been attributed to larger salivary gland size in males (Inoue et al., 2006). At the same time, lower sAA activity (U/mL) was found in males, which was also reported previously (Kivlighan and Granger, 2006), though conflicting results exist (Takai et al., 2007). The sex related differences in sAA were perhaps partially related to dilution, as males had a higher salivary flow rate. That was also confirmed after calculating the sTAMP (U/min), as we found no significant sex differences in sAA. SAA activity could also be influenced by the experimental conditions, ageing-related factors and other parameters (Arhakis et al., 2013). Furthermore, a faster gastric emptying rate (shorter T_{half}) was observed in males in the present study. This sex related difference was previously attributed to sex-related hormones like oestradiol and progesterone, which have a direct effect on gastrointestinal motility and can consequently affect the gastric emptying rate (Bennink et al., 1998). However, these hormones are possibly at a low level in the postmenopausal females of this study, which suggests an independent difference in GE rate between sexes. Lastly, we found higher postprandial insulin responses in males compared to females; this had been previously attributed to their increased visceral adiposity in males (Geer and

Shen, 2009). Increased visceral fat is a factor related to insulin resistance and hyperinsulinemia, whereas females typically exhibit greater peripheral fat distribution, which is linked to enhanced insulin sensitivity (Snijder et al., 2004). However, visceral or peripheral adiposity data were not determined in the present study to confirm or reject this association.

To our knowledge, this study was the first to explore oral performance and processing behaviours and their associations with GE and postprandial metabolic responses in community-living older adults (≥ 65 years). The study included a large number of participants and explored a great variety of OPMs and OPBs. However, some limitations should be considered. The study's approach utilised a cross-sectional design, which does not allow for assertions of causality. However, the study included a large group of older adults with equal number of males and females, allowing exploration of significant associations and possible sex influence on the research questions. Data from the bolus salivary uptake methodology are presented for 80 participants due to incomplete recovery of bolus samples in 8 participants.

In summary of all the above findings, this study found a number of significant associations between oral performance measures (OPMs), oral processing behaviours (OPBs) and postprandial metabolism. Both greater masticatory performance and greater stimulated salivary flow rate were associated with greater bolus saliva uptake; higher unstimulated salivary flow rate was associated with lower salivary alpha amylase (sAA). A slower eating rate was linked to increased bolus saliva uptake. Greater masticatory performance was related to shorter GE times, generally considered to be an advantage in older adults where gastric emptying can be delayed and lead to reduced appetite. The potential impact of OPMs and OPBs on postprandial glucose and insulin responses was also considered in this study, as glucose tolerance can decline with age and be accompanied by insulin resistance. Faster eating rates and reduced chewing were associated to lower initial glucose responses; perhaps suggesting that foods promoting a faster eating rate might reduce postprandial glucose responses in older adults. Additionally, lower SAA (in itself related to higher unstimulated salivary flow rates) was associated with a lower postprandial glucose response, which might indicate improved glucose tolerance. This outcome remains unclear as higher sAA was associated with higher insulin response which is considered key to improved glucose tolerance; however, when postprandial insulin responses were explored as early (before the glucose peak) and later (after the peak), higher sAA was only associated with higher later insulin responses, possibly indicating the greater insulin release following the blood glucose increase. Finally, faster GE was related to higher postprandial glucose and lower insulin responses; however, in our study, GE was not associated with eating rate or number of chews, a finding that highlights the need for future investigations on the possible combined effect of all the above parameters in the postprandial glycemia.

5. Conclusion

Physiological changes that happen with ageing can lead to alterations and great variability in food oral processing behaviours in older adults, which could have implications for their glycaemic control. Overall, this study demonstrates the complex relationships between oral processing and postprandial metabolic responses in older adults. Further research is needed into understanding the extent to which these responses can be modified, possibly through implementing approaches to influence food oral processing such as the providence of different food textures.

CRediT authorship contribution statement

Dimitra Zannidi: Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation. **Lisa Methven:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Jayne V. Woodside:** Writing – review & editing, Visualization, Software, Resources, Methodology, Funding acquisition, Data curation. **Gerry McKenna:** Writing – review & editing, Visualization, Software, Resources, Methodology, Funding acquisition, Data curation. **Ciaran G. Forde:** Writing – review & editing, Visualization, Software, Resources, Methodology, Data curation. **Kim G. Jackson:** Writing – review & editing, Software, Resources, Methodology, Data curation. **Amir Shafat:** Writing – review & editing, Resources, Methodology, Data curation. **Martin Schimmel:** Writing – review & editing, Software, Resources, Methodology, Data curation. **Miriam Clegg:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

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Declaration of competing interest

The authors declare no conflict of interest.

Data availability

Data are available on reasonable request from the corresponding author(s).

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.exger.2025.112893.

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