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The effect of acute chlorhexidine mouthwash on postprandial glucose response: a randomised cross-over trial

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Article

Keywords: chlorhexidine, carbohydrate, glucose, amylase, saliva

Posted Date: September 23rd, 2026

DOI: https://doi.org/10.21203/rs.3.rs-11115345/v1

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Additional Declarations: There is **NO** conflict of interest to disclose

Abstract

Background/Objectives: Chlorhexidine (CHX) mouthwash has strong antimicrobial effects that can influence the oral microbiome, its metabolites and interact with salivary proteins such as α-amylase that modulate glucose homeostasis. However, the acute metabolic effects of CHX in humans remain unclear. The main goal of this study was to investigate the effect of acute exposure to CHX mouthwash on the post-prandial glucose response and salivary α-amylase in healthy individuals.

Subjects/Methods: Forty-two healthy adults (29 women, 13 men) participated in a randomised, double-blind (participants, care providers and outcome assessor), placebo-controlled crossover study. Participants rinsed with either 0.2% CHX or placebo for 2 min before consuming a carbohydrate-containing breakfast. Capillary blood glucose was measured over 2 h and incremental area under the curve (iAUC) was calculated. Salivary α-amylase activity, pH, glucose, and lactate were assessed before and 2 h after the meal.

Results: A single exposure to 0.2% CHX (iAUC = 128 ± 12 mmol/L x min) mouthwash did not alter postprandial blood glucose response compared to placebo (iAUC = 137 ± 16 mmol/L x min; $P = 0.157$). Salivary α-amylase activity increased following the meal in both conditions (CHX = Δ 1.0 ± 0.5; placebo = Δ 0.8 ± 0.5; $P = 0.010$), but no differences between treatments were observed ($P = 0.938$).

Conclusions: These findings suggest that acute CHX exposure alone is insufficient to affect whole-body postprandial glucose regulation. Further studies should investigate the metabolic consequences of repeated or longer-term CHX exposure.

Introduction

Chlorhexidine (CHX) is a bisbiguanide antiseptic that has been widely used in clinical dentistry for more than four decades due to its broad-spectrum antimicrobial activity [1]. Consistent evidence has shown that CHX-containing mouthwashes reduce dental plaque accumulation and gingival inflammation and suppress the growth of oral pathogens associated with caries and periodontal disease, such as *Streptococcus mutans* and *Porphyromonas gingivalis* [2–5]. Consequently, CHX is frequently prescribed as an adjunctive therapy for the management of gingivitis and periodontitis [6]. However, in several countries, including the United Kingdom, CHX mouthwashes are readily available over the counter and are often used for prolonged periods without clinical supervision, increasing interest in their potential effects beyond plaque control.

Growing evidence indicates that CHX induces substantial alterations in the composition and metabolic activity of the oral microbiome. In particular, CHX has been shown to suppress oral nitrate-reducing bacteria, including members of the genera *Neisseria*, *Rothia*, *Actinomyces*, and *Haemophilus*, which contribute to the maintenance of a healthy oral ecosystem and the enterosalivary nitrate/nitrite/nitric oxide pathway [7–9]. Within this pathway, nitrate is reduced to nitrite by oral bacteria providing an essential substrate for the subsequent generation of nitric oxide and thereby contributing to systemic

nitric oxide bioavailability [10]. Nitric oxide is a critical signalling molecule involved in numerous physiological processes, including the regulation of glucose homeostasis through its effects on insulin signalling [11], GLUT-4 translocation [12], and skeletal muscle blood flow [13]. Consequently, disruption of the oral nitrate-reducing microbiota by CHX may impair nitric oxide production and has the potential to influence glucose metabolism and metabolic health.

In addition to its effects on microbial communities, CHX can directly bind to negatively charged proteins and glycoproteins present in saliva, potentially inducing protein aggregation and altering enzymatic activity [14]. Among these proteins, α-amylase is one of the most abundant proteins in saliva and plays a fundamental role in the initial digestion of dietary starch by hydrolysing complex carbohydrates into smaller oligosaccharides and maltose [14]. Despite the widespread use of CHX, its effects on salivary α-amylase have received considerably less attention than its effects on oral microbial communities. A plausible consequence of CHX exposure is a reduction in α-amylase activity resulting from direct protein binding and denaturation-like interactions that impair enzyme function. Furthermore, CHX-induced changes in the oral microbiome may indirectly influence α-amylase activity, as salivary proteins are continuously modified by bacterial proteases, while their structure and function are affected by changes in oral pH and microbial metabolism [15, 16]. Consequently, alterations in α-amylase activity following CHX use may reflect both direct biochemical interactions with the enzyme and indirect microbiome-mediated mechanisms.

These observations raise important questions regarding the potential metabolic consequences of CHX use. Changes in salivary α-amylase activity might influence the rate of carbohydrate breakdown and glucose availability. To date, however, no human study has directly investigated whether CHX mouthwash alters postprandial glucose responses in humans. Understanding this relationship is important because it may reveal a previously overlooked mechanism linking oral antiseptic use with nutrient metabolism and metabolic health.

Therefore, the primary aim of this study was to investigate the acute effects of CHX mouthwash on postprandial glucose responses and salivary α-amylase activity in healthy adults. We hypothesised that acute CHX exposure would reduce salivary α-amylase activity and consequently alter starch digestion, resulting in an increased postprandial glucose response.

Materials and methods

Participants

The study participants were healthy adults (> 18 y/o), including both women and men. Sample size calculation was based on preliminary data from our research group, which showed a mean glucose incremental area under the curve (iAUC) of 150 ± 48 mmol/L x min. The study was powered to detect a minimum between-group difference of 20% in glucose iAUC. Sample size estimation was performed using the ClinCalc online sample size calculator (ClinCalc.com), assuming 80% statistical power and a

two-sided significance level of 0.05 (95% confidence level). Based on these assumptions, a minimum sample size of 45 participants was required. Exclusion criteria included coeliac disease and gluten intolerance, pregnancy, smoking, the use of immunosuppressants or antibiotics and the use of antibacterial mouthwash and/or oral antibacterial ingredients within the 3 weeks preceding the study. The study was approved by the Ethics Committee of the Faculty of Health at the University of Plymouth (United Kingdom) and was conducted in accordance with the Declaration of Helsinki. All participants provided written informed consent before participating in the study.

Study design

The study (registered at www.clinicaltrials.gov, NCT07172009) followed a randomised, double-blind, crossover design. Each participant attended the Food, Exercise & Health laboratory (University of Plymouth, UK) on two occasions between 08:00 and 12:00 h under fasting conditions of at least 3 hours. Upon arrival, height and body weight were measured using a stadiometer (Seca, Birmingham, UK) and a bioimpedance analyser (Tanita, TBF-300 MA, Tokyo, Japan), respectively. An unstimulated saliva sample (2 mL) was collected into a Falcon sterile tube. Then, the saliva sample was transferred to a microtube tube and centrifuged at 13,000 rpm for 10 minutes at 4°C. Following centrifugation, the supernatant was transferred to a new tube and stored at -80°C for biochemistry analyses. At baseline, a capillary blood sample was obtained to analyse blood glucose using a glucometer (ACCU-CHEK Performa, Roche, USA). Participants were randomly allocated by an independent researcher to either the CHX or placebo group using a computer-generated block randomisation procedure (www.randomizer.org). Randomisation was conducted in blocks of 10 participants with a 1:1 allocation ratio to maintain balance between groups during enrolment. No stratification factors were used. Researchers who enrolled and those who assigned participants to the interventions had access to the random allocation sequence.

Following randomisation, participants received 10 mL of the assigned mouthwash according to the allocation schedule. The mouthwash treatments consisted of either 0.2% CHX digluconate (Corsodyl®, GlaxoSmithKline, UK) or a placebo solution (water containing yellow food colouring). Both mouthwashes were prepared and coded by an independent individual to ensure blinding of both participants and researchers. Participants rinsed their mouths with the assigned solution for 2 minutes before expectorating the mouthwash.

Following the mouth rinse, participants consume a pre-weighed breakfast meal consisting of 40 g of Hubbard's Foodstore® strawberry jam (Sainsbury's Supermarkets Ltd., London, UK) spread over 50 g of soft white bread (Sainsbury's Supermarkets Ltd., Northern Ireland, UK). Then, capillary blood glucose concentrations were measured over a 2 h period at 15, 30, 45, 60, 90 and 120 min. Changes in blood glucose concentration from baseline were calculated for each time point. Postprandial glycaemic response was assessed by calculating the incremental area under the curve (iAUC) using the trapezoidal method (Sigmaplot v16, US) with baseline glucose concentrations used as the reference value [17]. Mean iAUC values were compared between the CHX and placebo treatments. Two hours after consumption of the test meal, participants provided a second unstimulated salivary sample (2 mL). After

a washout period of at least 48 h, participants returned to the laboratory under the same conditions and repeated the protocol with the alternate mouthwash treatment.

Saliva analyses

α-amylase

Salivary α-amylase activity was measured in a subgroup of participants using a colorimetric assay kit (Novus Biologicals, Bio-Techne, USA, Cat. No. NBP3-25912). Saliva samples in duplicate were diluted 1:4 and mixed with the substrate and chromogenic agent, and incubated for 15, 30 and 5 minutes at 70, 40 and 95^{O}C, respectively. The absorbance was recorded at 540 nm using a microplate reader (Thermo Scientific™ Multiskan™ GO, Finland). α-Amylase enzymatic activity was calculated from the standard curve and expressed as units per millilitre (U/mL). The assay standard showed a linear range of 0.01–0.37 U/mL activity with a sensitivity of 0.008 U/mL.

pH

Salivary pH was measured using a single electrode digital pH meter (Lutron PH-208, Taiwan) that was calibrated following the manufacturer's instructions

Glucose and lactate

Salivary glucose and lactate concentrations were analysed using a biochemistry analyser (YSI 2500 Stat Plus, YSI Life Sciences, USA).

Statistical analysis

Data are presented as mean ± standard error of the mean (SEM) unless otherwise stated. The normality of all outcome variables was assessed using the Shapiro-Wilk test. All participants who completed both intervention visits and for whom outcome data were available were included in the analyses (per-protocol analysis). Differences in the iAUC between the CHX and placebo treatments were compared using paired-samples t-tests. Salivary variables (α-amylase activity, pH, glucose, and lactate concentrations) were analysed using a two-way repeated-measures analysis of variance (ANOVA), with treatment (placebo vs CHX) and time (pre-prandial vs post-prandial) as within-subject factors. Where a significant treatment × time interaction was observed, pairwise comparisons were performed using Bonferroni-adjusted post hoc tests. Assumptions of sphericity were assessed using Mauchly's test and, where violated, Greenhouse-Geisser corrections were applied. No subgroup or sensitivity analyses were performed. Statistical analyses were carried out using the SPSS software (SPSS Statistics, IBM® Version 24) and statistical significance was determined as $P < 0.05$.

Results

Participants

Between June 2022 and September 2025, fifty-four individuals were assessed for their participation in the study (Fig. 1). Forty-two participants completed both study visits and were included in the final analyses (Table 1). Outcome assessments were performed during each experimental visit. Standardised instructions regarding diet, physical activity, oral hygiene practices, and overnight fasting were provided before each visit and applied equally across treatment conditions. No concomitant interventions likely to influence study outcomes were permitted during the assessment periods.

Table 1
Main participant's characteristics. Data are means ± SEM.

Gender (F:M)	**29:13**
Age (years)	22.9 ± 1.0
Weight (kg)	66.8 ± 2.8
Height (cm)	170 ± 0.02
Body mass index (BMI) (kg/m^2)	23.1 ± 0.6
Blood glucose (mmol/L)	5.1 ± 0.1

Glucose

Blood glucose concentrations were similar in both treatments at baseline (placebo = 5.1 ± 0.1 mmol/L; CHX = 5.1 ± 0.1 mmol/L; *P* = 0.339). Post-prandial glucose concentrations and iAUC did not differ between treatments (Fig. 2).

Salivary α-amylase activity

Salivary α-amylase activity was measured in 28 participants (males = 10; females = 18) and it was similar in both treatments at baseline (placebo = 4.4 ± 0.3 U/mL; CHX = 3.9 ± 0.3 U/mL; *P* = 0.637) (Fig. 3). A significant increase (*P* = 0.010) was observed in both treatments 2 hours following consumption of the carbohydrate-containing breakfast (Fig. 3). No significant treatment × time interaction was observed (*P* = 0.938), indicating that the temporal change in α-amylase activity was similar in both the placebo and CHX treatments.

pH, glucose and lactate

Salivary pH, glucose and lactate levels were similar at baseline (Fig. 4). There was a significant (*P* = 0.015) decrease in salivary pH 2 hours following consumption of the carbohydrate-containing breakfast. However, no significant treatment × time interaction was observed (P = 0.620), indicating that the temporal change in salivary pH was similar following both treatments.

A significant increase was observed in glucose ($P < 0.001$) and lactate (P = 0.007) 2 hours following consumption of the carbohydrate-containing breakfast in both treatments (Fig. 4). Furthermore, a significant treatment × time interaction was detected in glucose levels (P = 0.029), indicating that CHX mouthwash significantly attenuated the increase in salivary glucose post-treatment compared to placebo. In contrast, there was no significant treatment × time interaction (P = 0.104), indicating that the change in lactate over time was similar between placebo and CHX conditions.

Discussion

To the best of our knowledge, this was the first study to investigate the acute effects of CHX mouthwash on post-prandial glucose responses in healthy individuals. Contrary to our initial hypothesis, single exposure to CHX mouthwash did not affect blood glucose concentrations, glucose iAUC, or salivary α-amylase activity following consumption of a carbohydrate-containing breakfast.

Most research on antimicrobial compounds such as CHX has traditionally focused on their efficacy in controlling dental plaque accumulation and periodontal pathogens [1, 6]. Indeed, CHX remains one of the most effective antiseptic agents used in dentistry and has well-established inhibitory effects against key periodontal pathogens, including *Porphyromonas gingivalis* and other members of the periodontal biofilm [2, 18]. However, growing evidence indicates that CHX exerts broad-spectrum antimicrobial effects that extend beyond pathogenic species and can disrupt commensal microorganisms that contribute to host physiology [8]. Current dental guidelines recommend short-term daily use of CHX mouthwash (up to three weeks) at high doses (0.2%) for the management of periodontal disease [1, 19].

In particular, CHX has been shown to reduce the abundance and activity of oral nitrate-reducing bacteria [7, 8], which contribute to maintaining nitric oxide bioavailability. Nitric oxide is an important signalling molecule involved in multiple physiological pathways relevant to glucose metabolism, including insulin signalling [11], GLUT-4 translocation [12], and skeletal muscle blood flow [13]. Consequently, interventions that suppress nitrate-reducing bacteria, such as CHX mouthwash, may theoretically influence glucose regulation by disrupting these microbial-metabolic pathways. In support of this, some observational studies have reported associations between reduced abundance and activity of nitrate-reducing bacteria and impaired glycaemic control [20, 21], while regular mouthwash use has also been linked to a greater risk of developing diabetes [22].

In contrast, prolonged use of low-concentration CHX has been associated with improvements in glycaemic control in individuals with poorly controlled type 2 diabetes [23], and experimental studies in mice have demonstrated attenuation of diet-induced metabolic dysfunction and insulin resistance following CHX treatment [24]. Similarly, no effect of CHX on glycaemic control has been reported in children with type 1 diabetes [25]. Our findings indicate that a single CHX rinse is insufficient to modify postprandial glucose regulation in healthy young adults.

To our knowledge, this was also the first study to investigate the effect of CHX mouthwash on α-amylase activity, which is one of the most abundant proteins in saliva and helps to initiate the digestion of dietary

starch [14]. The observed increase in salivary α-amylase activity following the test meal is consistent with previous studies showing a similar effect [26]. Future studies should examine the impact of prolonged CHX exposure on postprandial metabolic responses and α-amylase activity.

Secondary outcomes included salivary pH, glucose, and lactate concentrations. Consumption of the carbohydrate-containing breakfast resulted in a significant decrease in salivary pH in both treatments. This response is consistent with the well-established reduction in oral pH following carbohydrate ingestion, particularly when meals contain fermentable sugars that can be metabolised by oral bacteria [27]. The breakfast provided in the present study included jam, a source of sucrose, which may have contributed to the observed reduction in pH. Sustained or frequent exposure to fermentable carbohydrates can promote acid production within the oral cavity, leading to enamel demineralisation and increasing the risk of dental caries [27]. In previous studies, we showed that a 7-day course of CHX mouthwash caused a significant reduction in salivary pH under fasting conditions [7, 8], but this was not observed acutely [28]. We also previously observed significant increases in salivary glucose and lactate following CHX exposure [7, 8, 28]. Here, salivary lactate increased in both treatments, likely reflecting bacterial fermentation of dietary carbohydrates. Interestingly, CHX significantly attenuated the postprandial increase in salivary glucose concentrations. Because blood glucose concentrations were unaffected, these findings indicate that the observed reduction in salivary glucose was more likely to reflect a local oral effect rather than altered whole-body glucose metabolism. One possible explanation is that changes in microbial biomass or biofilm activity following CHX exposure influenced glucose utilisation or metabolism within the oral environment.

Several limitations of this study should be acknowledged. First, the intervention involved only an acute exposure to CHX mouthwash. Consequently, the present findings cannot exclude the possibility that longer-term CHX exposure may influence glucose metabolism through sustained alterations in oral microbial composition and/or salivary protein function. Second, α-amylase activity was measured in twenty-eight participants because some saliva samples were lost due to freezer malfunction. Third, other important metabolic markers, such as insulin response, were not assessed. It may be possible that subtle metabolic effects of CHX occurred without being reflected in circulating glucose concentrations. Fourth, the study was conducted in young, healthy adults with normal glucose control. The metabolic consequences of oral microbiome disruption may be more evident in populations with impaired glucose metabolism, who may also have higher prevalence of periodontal disease that can also affect glucose homeostasis. Future studies should therefore investigate the impact of repeated CHX exposure on postprandial metabolic responses, including insulin responses, in both healthy and metabolically compromised populations.

In conclusion, our findings indicate that acute CHX exposure alone does not appear to be sufficient to modify post-prandial glucose responses or the physiological increase in salivary α-amylase activity following a carbohydrate-containing breakfast in healthy individuals. These results provide an initial step towards understanding the potential relationship between oral microbial perturbation and metabolic

regulation, while highlighting the need for studies investigating the effects of repeated or longer-term oral microbiome disruption and their potential relevance to populations with impaired metabolic health.

Declarations

Authorship contribution statement

RB designed the study. RB and CJ recruited participants and were responsible for saliva and blood sample collection and processing during the experimental visits. CJ, RB and ZB conducted the pH, glucose and lactate analyses, while MM and SS performed the α-amylase analyses. CJ, RB and PCA analysed and interpreted the data and contributed to the statistical analyses. CJ, RB and PCA drafted the manuscript. MM, SS and ZB contributed to manuscript revision and interpretation of the findings. All authors reviewed and approved the final manuscript.

Competing interests

RB, CJ, MM, SS, ZB and PCA declare that there is no duality of interest associated with this manuscript. RB, CJ, MM, SS, ZB, and PCA declare that there are no conflicts of interest associated with this manuscript.

Acknowledgements

The authors would like to thank Olivia Barnes, Harry Brown, Christina Kingsman, Ngoc Truong and Zaman Zaman for their assistance with participant recruitment and data collection throughout the study. We also thank Victoria Cammack and Natalie Sweet for their assistance in the lab. This project was funded by the University of Plymouth.

Data availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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Figures

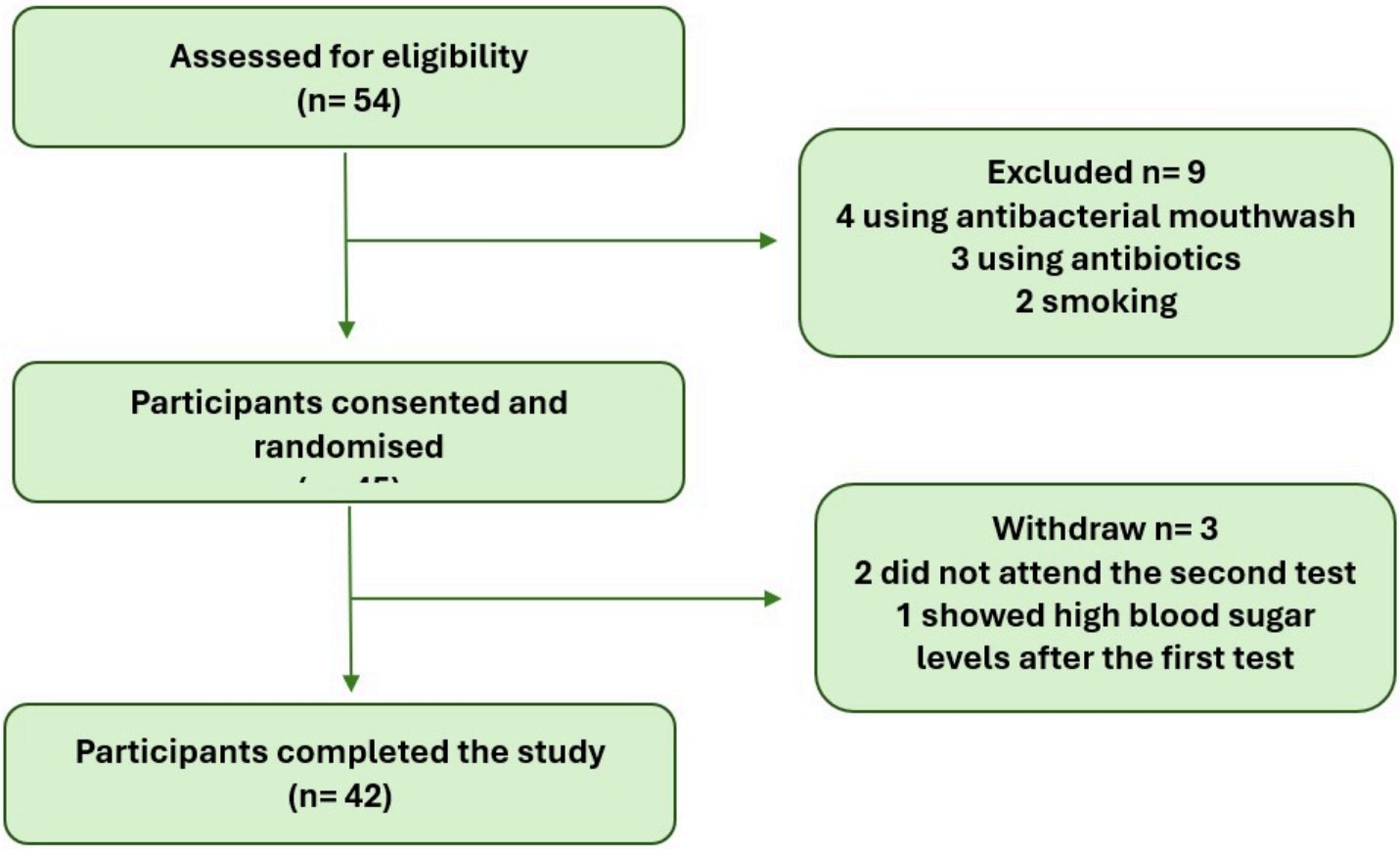


Figure 1

CONSORT flow diagram illustrating the study procedures.

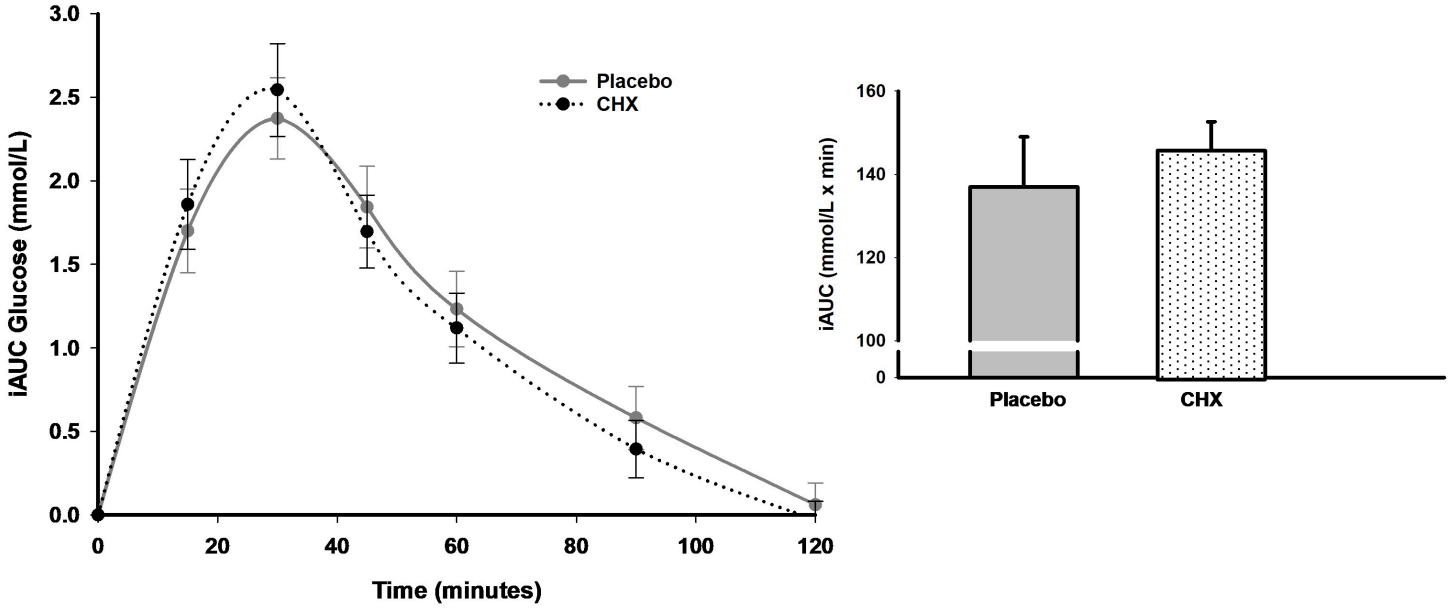


Figure 2

Changes in blood glucose concentrations and incremental area under the curve (iAUC) over 2 hours following consumption of the carbohydrate-containing breakfast and rinsing the mouth with placebo or chlorhexidine (CHX) mouthwash. Data are presented as mean ± SEM (n = 42).

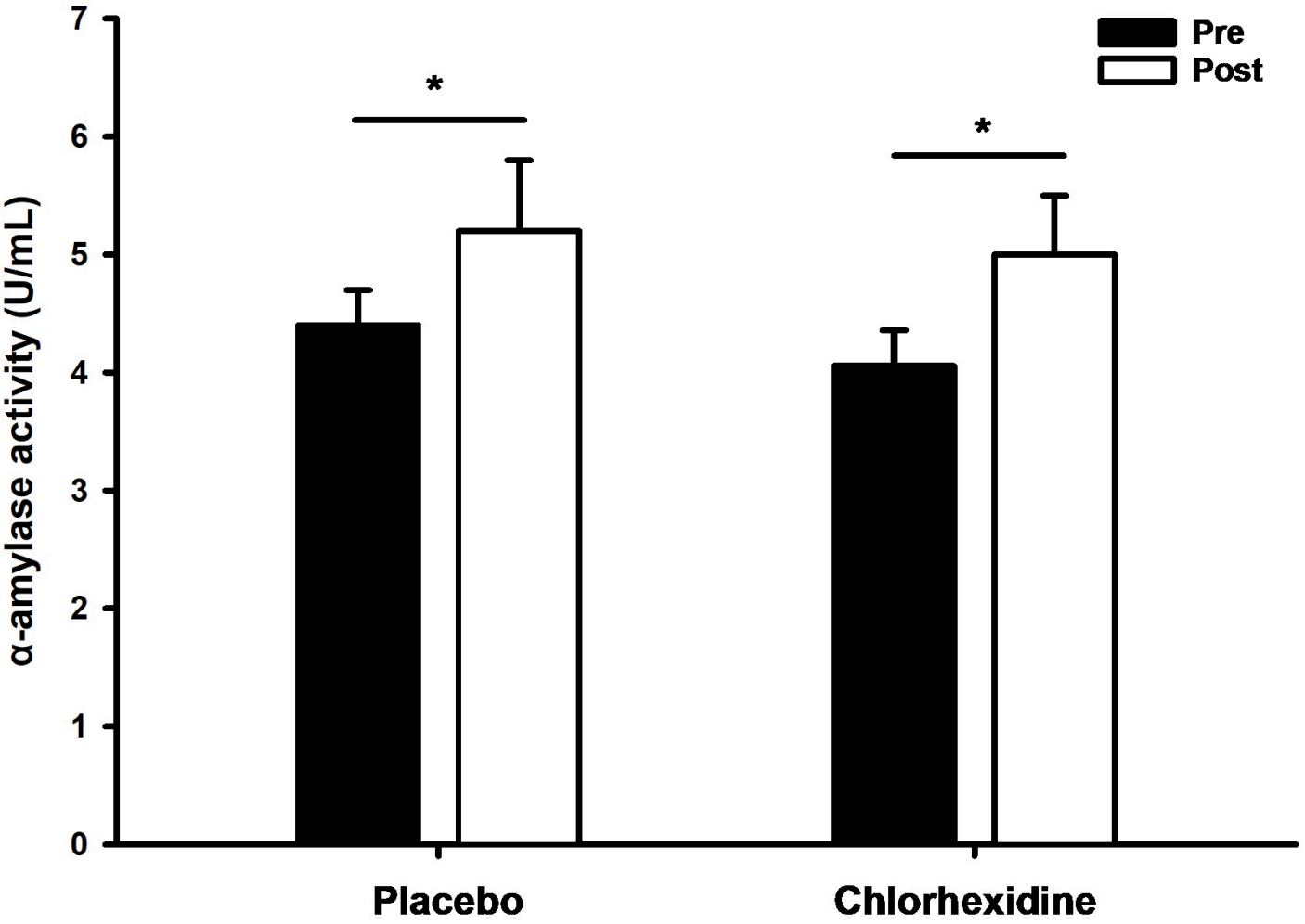


Figure 3

Salivary α-amylase activity measured before (pre) and 2 hours after (post) rinsing the mouth with placebo and chlorhexidine mouthwash and consuming a breakfast with carbohydrates. Data are presented as mean ± SEM (n = 28).

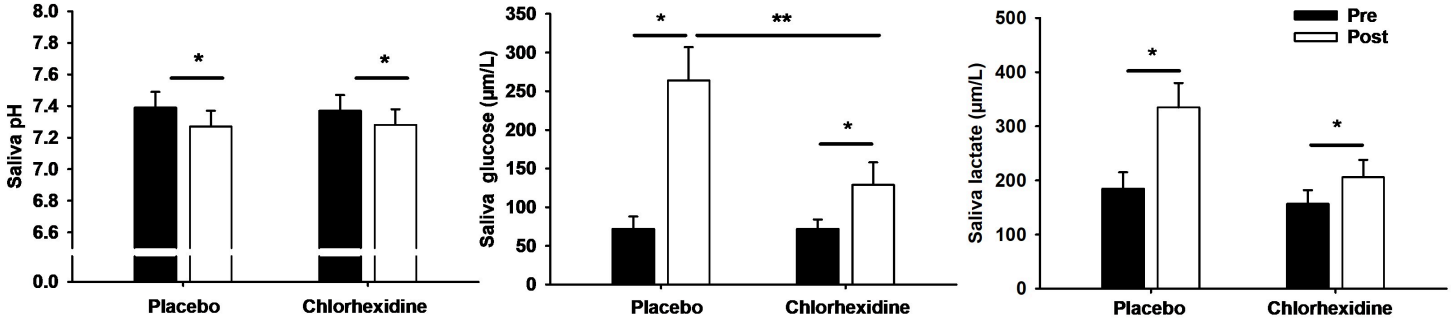


Figure 4

Salivary pH, glucose and lactate concentrations before (pre) and 2 hours after (post) rinsing the mouth with placebo and chlorhexidine mouthwash and consuming a breakfast with carbohydrates. Data are presented as mean ± SEM (n = 42).

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- CONSORTchecklist.docx
- Table1.docx
- Clinicaltrialprotocol.docx