



DOSSIER · SUBSTANCE

Sucralose

Sucralose is one of the best-studied non-nutritive sweeteners. It is striking how often studies do not test the substance alone: many of the most-cited works examine sweeteners as a group or dissolve sucralose in water, in diet lemonade or together with carbohydrate — and reach differing findings.

625

STUDIES IN
TOTAL

174

IN HUMANS

71

OF WHICH
CONTROLLED

101

IN ANIMALS

43

IN THE LAB

Figures from the IHR study database · Last updated 2026-10-15

How to Read This Dossier

ESTABLISHED

measured in humans, with a comparison group

PLAUSIBLE

supported by mechanism, animal studies or observation

HYPOTHESIS

a likely connection that still needs to be tested

Sucralose at a Glance

ORIGIN:

Sucralose is a non-nutritive sweetener and ranks among the best-studied representatives of this group of substances. In the studies presented, it is tested in beverages, as a component of preparations as well as a placebo, control vehicle or taste-masking agent. It is striking that many studies do not examine the substance alone, but non-nutritive sweeteners as a group.

EVIDENCE BASE:

625 evaluated studies · 71 controlled human studies · 103 observational studies · 23 review articles · 101 animal studies · 30 cell-culture studies

STRONGEST EVIDENCE:

- Metabolism, substance group: The systematic review with meta-analysis of 29 randomised studies with 741 participants shows that blood glucose does not rise after intake of non-nutritive sweeteners but gradually declines over the observation period ².
- Glycaemic control, type 2 diabetes: A randomised controlled study with 210 adults finds no change in HbA1c, fasting glucose, lipid profile or inflammatory markers over 12 weeks, with slightly declining weight, body-mass index and waist circumference ⁵.
- Insulin sensitivity, healthy adults: In 33 participants, insulin sensitivity fell after 14 days of sucralose at 15 % of the acceptable daily intake by a median of -17.7 % versus -2.8 % in the control group (P = 0.04) ¹⁷.
- Insulin resistance, overweight: In a randomised, triple-blind study with 138 overweight persons over 60 days, HOMA-IR rose with the maqui-citrus drink containing sucralose (p = 0.002) and with the maqui-citrus drink containing sucrose (p = 0.001) ⁶.
- Appetite regulation, young adults: A crossover study with 75 participants reports increased hypothalamic blood flow (P < 0.018) and a stronger hunger response (P < 0.001) after acute sucralose intake compared with sucrose ⁴⁹.

LIMITATIONS:

- Numerous studies test sucralose only as placebo, control vehicle or taste-masking agent; an isolated effect of the substance cannot be derived from them.
- The findings on insulin sensitivity ¹⁷ and HOMA-IR ⁶ come from small, short studies; they indicate a direction, and independent replication is still outstanding.
- The cohort, cross-sectional and case-control studies on hypertension, inflammatory markers, Crohn's disease and central precocious puberty are observational and do not establish causation.

CLASSIFICATION:

Sucralose is a non-nutritive sweetener whose short-term metabolic effects in humans have been studied, while dose, duration and isolated effect remain open questions.

1. What Sucralose Is

Sucralose belongs to the non-nutritive sweeteners, that is, substances that provide sweetness without the energy content of sugar. In research it rarely appears alone: a large proportion of the studies examine "non-nutritive sweeteners" or "artificial sweeteners" as a group in which sucralose stands alongside other representatives. The controlled human studies investigate it in water, in diet lemonade or together with a carbohydrate load. Precisely this embedding determines what is measured: the response to the sweetener itself or the response to the beverage in which it is dissolved. The main parameters examined are glucose tolerance, gut hormones, microbiome and body weight.

The decisive point for understanding all studies is the question of what is being measured. When sucralose is administered in water, the response to the sweetener is measured. When it is administered in diet lemonade, a beverage with additional constituents is measured. When it is given together with carbohydrate, the interaction of both is measured. The studies on gut hormones and appetite distinguish exactly these conditions, and they reach different results. In addition there is the measurement level: some studies record laboratory values and questionnaires, some examine the microbiome, some the disease course. A changed marker is not yet a changed course. Anyone comparing findings must therefore first check in which matrix and at which level the measurement was performed.

2. What the Studies Show in Detail

The human studies, ordered by application and the controlled studies first — with participants, duration, preparation, measured variable and result exactly as the original papers report them. Numbers appear only where the study states them.

Distribution of **controlled human studies** by field of application — the numbers are not additive, as one study may be assigned to several fields:

FIELD OF APPLICATION	CONTROLLED STUDIES
Metabolism	51
Gut and microbiome	22
Safety and tolerability	16
Heart and circulation	6

FIELD OF APPLICATION	CONTROLLED STUDIES
Absorption and bioavailability	5
Inflammatory processes	4
Cognition and psyche	4
Immune system	2

Sucralose Ingested: Human Studies

The largest controlled summary on non-nutritive sweeteners comes from a systematic review with meta-analysis that combined randomised studies and prospective cohorts on non-nutritive sweeteners ¹. From 11 774 hits, 7 randomised studies with 1 003 participants (median follow-up 6 months) and 30 cohort studies with 405 907 participants (median follow-up 10 years) were included. The randomised studies showed no significant effect on body-mass index (mean difference -0.37 kg/m²; 95 % CI -1.10 to 0.36; I² 9 %; 242 participants). In the cohorts, consumption was associated with a modest increase in body-mass index (mean correlation 0.05). The authors examined not the substance alone but the substance group; no statement specific to sucralose can be derived from this.

A second systematic review with meta-analysis examined the glycaemic influence of non-nutritive sweeteners across 29 randomised studies with a total of 741 participants, including aspartame, saccharin, steviolosides and sucralose ². Compared with baseline, blood glucose did not rise after intake but gradually declined over the observation period. The glycaemic effect did not differ by type of sweetener, but varied to some extent with age, weight and disease status of the participants. Here too the pooled statement concerns the substance group, not sucralose in isolation.

The most specific controlled study on the substance itself examined 33 healthy adults without comorbidities and with low habitual intake of non-nutritive sweeteners per group ¹⁷. Over 14 days they consumed sucralose at 15 % of the acceptable daily intake via commercially available sachets; the control group received no intervention. Before and after the phase, glucose utilisation was assessed via a three-hour modified intravenous glucose tolerance test. The intervention group showed a significant decline in insulin sensitivity with a median percentage change of -17.7 % (-29.3 % to -1.0 %) versus -2.8 % (-30.7 % to 40.6 %) in the control group (P = 0.04). The acute insulin response to glucose rose from 577 mU · L⁻¹ · min (350-1040 mU · L⁻¹ · min) to 671 mU · L⁻¹ · min (376-1010 mU · L⁻¹ · min) (P = 0.04). The study is small and short; it indicates a direction that requires independent replication.

A randomised controlled trial with 210 adults of Asian-Indian origin with type 2 diabetes tested replacement of sucrose in coffee or tea by sucralose versus continued sugar consumption over 12 weeks ⁵. The primary endpoint was change in HbA1c. After 12 weeks no change in HbA1c, fasting glucose, lipid profile or inflammatory markers was found between or within groups. In the intervention group body weight (-0.5 kg [95 % CI -1.0 to -0.1]; p = 0.02), body mass index (-0.2 kg/m² [-0.4 to 0.0]; p = 0.03) and waist circumference (-0.8 cm [-1.4 to -0.3]; p = 0.002) declined slightly. Improvements in the lipid accumulation product (p = 0.01) and visceral adiposity index (p = 0.04) were also observed. The study was open-label, which increases susceptibility to bias.

A randomised controlled trial compared four low-calorie sweeteners and sucrose over 12 weeks in 154 adults with overweight or obesity (body mass index 25-40 kg/m²) ⁴. Participants drank 1.25-1.75 L daily of a beverage containing sucrose (n = 39), aspartame (n = 30), saccharin (n = 29), sucralose (n = 28) or rebaudioside A (n = 28); the drinks provided 400-560 kcal/day (sucrose) or <5 kcal/day (sweeteners). Anthropometry, energy intake, energy expenditure, appetite and glucose tolerance were measured at baseline and during the study; body weight was recorded every two weeks. Of the enrolled participants, 123 completed the study. The work compares multiple substances in parallel; an isolated effect of sucralose cannot be derived from the design.

A randomised, triple-blind study examined 138 overweight individuals who consumed 330 mL daily of a maqui-citrus beverage sweetened with stevia, sucralose or sucrose for 60 days ⁶. Body mass index did not change significantly under any beverage. Fasting glucose rose 26 % after short-term consumption of the sucrose-sweetened beverage and to a lesser extent under the low-calorie sweetener beverages: 11 % with stevia and 20 % with sucralose. Insulin concentrations did not change significantly under any beverage, resulting in a significant increase in HOMA-IR under the sucralose beverage (p = 0.002) and the sucrose beverage (p = 0.001). The authors consider the evidence available at the time insufficient to recommend widespread use of low-calorie sweeteners.

A randomised crossover study with 75 young adults (healthy weight, overweight or obesity) compared a sucralose-sweetened beverage, sweetness-matched sucrose or water ⁴⁹. Acute sucralose intake versus sucrose increased hypothalamic blood flow (P < 0.018) and the hunger response (P < 0.001). Sucralose versus water also increased hypothalamic blood flow (P < 0.019) but showed no difference in hunger ratings. Sucrose, but not sucralose, raised peripheral glucose levels, which were associated with a decrease in medial hypothalamic blood flow (P < 0.007). Sucralose produced increased functional connectivity between the hypothalamus and brain regions involved in motivation and somatosensory processing compared with sucrose and water. The authors conclude that non-caloric sweeteners may influence central mechanisms of appetite regulation.

A randomised crossover study with 20 healthy participants (10 women, 10 men; age 25.8 ± 7.5 years, body mass index 22.3 ± 1.8 kg/m²) examined reward responses to erythritol, sucrose and sucralose before and after taste-preference conditioning ⁴⁵. After conditioning, explicit wanting and liking increased, without differences between sweeteners. Release of gastrointestinal satiety hormones (cholecystokinin, glucagon-like peptide-1) and changes in glucose and insulin did not influence reward responses. The study is small and examines reward responses as endpoint; it shows no differential reward effect of the substance.

A randomised crossover study with 55 healthy adults compared beverages sweetened with sucrose (glycaemic index 65), isomaltulose (glycaemic index 32) and sucralose as non-glycaemic control on cognition, mood and satiety ³⁹. Sixty minutes after intake the Complex Figure Test, Word Recall Test, Trail Making Test Part B and Stroop test were performed. No significant differences in means (95 % CI) were found between isomaltulose and sucrose: Complex Figure immediate recall -0.6 (-1.7 to 0.5), delayed recall -0.8 (-1.9 to 0.3); Word recall immediate recall 0.2 (-0.7 to 1.1), delayed recall 0.5 (-0.4 to 1.4); Trail Making completion time -2.4 (-7.5 to 2.7) s; Stroop time for correct congruent responses -9 (-31 to 14) ms and correct incongruent responses -18 (-42 to 6) ms. Sucralose served here as a control without glycaemic effect; an independent cognitive effect was not tested.

A randomised crossover study with 39 participants (age 23 ± 5 years; 17 men, 22 women; body mass index $18.5\text{--}24.9\text{ kg/m}^2$: $n = 21$; $\geq 30\text{ kg/m}^2$: $n = 18$) examined mouth rinses with sucrose, sucralose, maltodextrin or water for 2 minutes before expectoration³⁴. After the mouth rinse ghrelin rose for water versus maltodextrin ($p = 0.03$). Cholecystokinin rose after maltodextrin mouth rinse ($p = 0.03$) and sucralose mouth rinse ($p = 0.005$) versus sucrose, but only in participants with body mass index $18.5\text{--}24.9\text{ kg/m}^2$. Energy intake was subsequently recorded at a buffet. The work examines oral perception, not systemic uptake of the substance.

A randomised, single-blind study with 16 participants (9 men, 7 women) examined mouth rinses with sucrose, low-intensity sucralose (S1:1), high-intensity sucralose (S100:1) and water during a 12.8-km time trial³⁵. The mouth rinse was performed at the start and at every 12.5 % of the distance. No further differences between the time trials were found. Sucralose alone did not improve performance.

A randomised, as far as possible double-blind within-subject study with ten recreationally active men tested mouth rinses with four sucrose-sucralose combinations (sweetness ratios 1:1, 1:6, 6:1, 6:6) and a control solution during 60 minutes of moderate-intensity walking³⁶. Energy expenditure, heart rate, respiratory quotient and perceived exertion were measured. Perceived sweetness of the 1:1 solution was significantly lower than the others (except control), and the 1:6 and 6:1 solutions were equally sweet. The mouth-rinse solution did not significantly affect energy expenditure or perception of internal state ($p > 0.05$). An unexpected interaction between sweet-liker phenotype and volume of solution swallowed was observed ($p = 0.021$), without influence on energy expenditure. Volume swallowed was positively associated with rate of perceived exertion ($p = 0.008$) and negatively with energy expenditure during walking. The study is small and tests combinations, not the single substance.

A randomised controlled crossover study with 15 participants compared the acute effects of glucose, fructose, glucose plus fructose and sucralose on heart-rate variability⁴⁴. During each two-hour visit venous blood glucose was measured at 5-minute intervals and heart-rate variability was recorded continuously by Holter electrocardiograph. Intake of the different carbohydrates and of sucralose showed significant differences in heart rate ($p < 0.001$), SDNN ($p < 0.008$), RMSSD ($p < 0.001$), pNN50 ($p < 0.001$) and blood pressure ($p < 0.001$). Different glucose levels altered heart-rate variability parameters and blood pressure (all $p < 0.001$), while the rate of change in blood glucose influenced heart-rate variability but not heart rate ($p = 0.25$) or blood pressure ($p = 0.99$). The study is small and the findings are not broken down by substance specificity.

A randomised crossover study with 30 healthy individuals examined six treatments: water and equally sweet flavoured beverages containing 25 g sucrose, sucralose, stevia extract, allulose plus stevia extract or monk-fruit extract⁴⁸. After overnight fasting, cerebral blood flow was measured by arterial spin labelling before and 5 and 30 minutes after intake of 500 mL; gastric volume was measured at baseline and after 25 and 45 minutes. Serum insulin and glucose were determined; appetite and thirst were rated throughout the visit. Hypothalamic blood flow did not differ between beverages. Differences appeared in the ventral tegmental area. The work compares multiple sweeteners; an isolated effect of sucralose cannot be read from it.

A randomised controlled study with 20 healthy participants over 6 weeks examined the effect of non-nutritive sweeteners on body weight⁵⁰. Sweetener consumers were randomly assigned to the sucralose group (0.507 mg/kg; $n = 7$) or the stevia group (0.375 mg/kg; $n = 6$), non-consumers to the control group (no sweeteners; $n = 7$); all received an isocaloric diet and were counselled on a healthy lifestyle. Weight and body mass index declined significantly in the control group ($p_{\text{weight}} = 0.026$; $p_{\text{body mass index}} = 0.02$) and in the sucralose group ($p_{\text{weight}} = 0.028$; $p_{\text{body mass index}} = 0.017$). The group sizes are very small; the work provides an indication, not proof.

A randomised controlled crossover study with 64 pain-free participants (22 with normal adiposity, 29 with high adiposity, 13 with high adiposity and elevated HbA1c including prediabetes and type 2 diabetes) compared intake of 37.5 g glucose or 200 mg sucralose (sweetness-matched) in two sessions one month apart⁵¹. Sucralose served here as a sweetness-matched control; an independent analgesic effect was not found.

A randomised controlled crossover study with 27 healthy adults (age 18–48 years) tested whether tasting equally sweet solutions (10 % sucrose and 0.016 % sucralose) alters warm perception, heat-pain threshold and perception of painfully hot stimuli⁴². Tasting sucrose or sucralose had no significant effect on perception of an individually set hot stimulus (54.5 ± 4.2 and 54.9 ± 3.2 vs. 53.2 ± 3.5 for water, 0–100 visual analogue scale) or on warm-perception or heat-pain threshold (43.3 ± 0.8 , 43.2 ± 0.8 vs. 43.0 ± 0.8 °C). Expectation of a sweet substance also did not alter the heat-pain threshold. The work shows no analgesic effect.

A randomised controlled study with 18 healthy 18- to 35-year-old “non-consumers” (<1 food or beverage with low-calorie sweeteners per month) examined exposure to sucralose over 2 weeks⁴⁶. At study entry participants were thoroughly instructed to avoid low-calorie sweeteners. After the run-in phase they were randomised and drank diet lemonade containing sucralose or carbonated water (control) three times daily for one week. At baseline 8 participants had sucralose in urine (29.9–239.0 ng/mL; mean $\pm$ SD: 111.4 ± 91.5 ng/mL). The work shows that self-report of being “unexposed” does not reliably reflect actual exposure; it does not test a clinical effect.

Positive associations were also observed for individual substances. The analysis controlled for weight changes over time; observational data do not establish causality.

A prospective multicentre cohort study with 1696 children and adolescents aged 7–17 years (Taiwan Pubertal Longitudinal Study, from July 2018) examined the association between sweetener intake and paediatric hypertension⁵⁴. Intake of acesulfame-K, aspartame, sucralose, glycyrrhizin, steviol glycosides, sorbitol and added sugar was recorded by validated questionnaire; urine samples were collected concurrently; anthropometric and blood-pressure measurements were performed every three months. Paediatric hypertension was defined as systolic or diastolic blood pressure at or above the 95th percentile for age, sex and height. Multivariate generalised linear mixed models were adjusted for age, sex, z-BMI, family history of hypertension, total energy intake and further factors. The study is observational; it cannot establish causation.

A cross-sectional analysis with 624 adults from the Cancer Prevention Study-3 Diet Assessment Substudy of the American Cancer Society examined associations between non-nutritive sweetener consumption and metabolic risk factors as well as inflammatory biomarkers⁵⁵. Intake of aspartame, saccharin, sucralose and acesulfame potassium was estimated from the mean amounts of six 24-hour dietary recalls over one year. Fasting insulin, C-peptide, HbA1c, leptin, adiponectin, C-reactive protein, tumour necrosis factor- α , interleukin-6 and interleukin-10 were measured in two fasting blood samples taken six months apart. Multivariable linear regressions were adjusted for age, sex, ethnicity, education, smoking and physical activity; in full models additionally for body mass index, diet quality (Healthy Eating Index 2020) and energy intake. The work is cross-sectional and does not permit a causal statement.

A case-control and biosample study analysed 1461 biosamples from 487 individuals (245 with Crohn's disease, 242 controls) from Australia, Hong Kong and mainland China for aspartame, sucralose, saccharin and polysorbate-80 in stool, urine and serum ⁵⁶. Crohn's disease activity was assessed via the Crohn's Disease Activity Index and faecal calprotectin. Patients with Crohn's disease had higher sweetener levels than controls across all cohorts (all $p < 0.0001$). Crohn's-associated polysorbate-80 metabolites correlated positively with urine sweeteners in patients with Crohn's disease. In vitro, Crohn's-associated polysorbate-80 metabolites increased intestinal permeability and enabled translocation of sweeteners across the epithelium. Sweeteners and specific Crohn's-associated polysorbate-80 metabolites were higher in active disease. The work is observational and supplemented by preclinical data.

A study with 481 diagnosed cases of central precocious puberty examined genetic and dietary influences on central precocious puberty ⁵⁷. Aspartame, sucralose, glycyrrhizin and added sugar were significantly associated with increased risk of central precocious puberty, especially in genetically predisposed individuals. A dose-dependent relationship was identified, with higher sweetener intake associated with greater risk of central precocious puberty. Sex-specific effects were also found, with sucralose strongly associated with central precocious puberty in boys and glycyrrhizin, sucralose and added sugar strongly associated with central precocious puberty in girls. The effects of the interaction between genetic predisposition and sweetener intake on this risk were not significant. Sweetener consumption and high genetic predisposition increase risk independently. The work is observational.

An observational study with 25 g glucose or a sucralose placebo before social exclusion in the Cyberball game examined whether glucose reduces the negative emotional effects of exclusion ⁵². Strong effects of exclusion were observed, but no effect of glucose on immediate or delayed self-reported needs or mood. Sucralose served as placebo; an independent effect of the substance was not tested.

A randomised controlled study with 538 participants aged 21-80 years with complete data for four dental examinations from the Xylitol for Adult Caries Trial (X-ACT) compared 5 g/day xylitol (five 1-g lozenges distributed throughout the day) with a sucralose placebo over three years ³. Mean annualised transition scores for non-cavitated plus cavitated lesions on coronal and root surfaces from sound to carious favoured xylitol over placebo during the three cumulative periods of 12, 24 and 33 months, yet these clinically and statistically non-significant differences diminished over time. Sucralose served here as the placebo; an independent caries-preventive or cariogenic effect was not examined.

A randomised controlled crossover study with 85 participants (52 normal, 33 hyperglycaemic) compared a sucralose-erythritol beverage (placebo) with a tagatose beverage (5 g) seven days apart ³⁸. Blood samples were analysed at 0, 30, 60 and 120 minutes after intake for fasting and postprandial glucose, insulin and C-peptide levels. After the tagatose beverage hyperglycaemic participants showed a significant reduction in serum glucose at 120 minutes ($p = 0.019$) and glucose area under the curve ($p = 0.017$), which was not observed in normal participants.

A randomised controlled crossover study with 7 male 800-metre runners (20 ± 3 years, 63 ± 6 kg, 169.1 ± 7 cm) compared caffeine (3 mg/kg) with a sucralose placebo in an endurance exercise to exhaustion on a treadmill at maximal aerobic speed ⁴⁷. Caffeine intake showed a significant prolongation of endurance time versus placebo (376 ± 137 to 457 ± 182 seconds; $p < 0.05$). No significant difference in blood lactate concentration was found between rest, placebo and caffeine intake ($p < 0.05$).

A randomised controlled crossover study with 20 healthy adults (age 26 ± 7 years) investigated in three sub-studies the effect of calcium type and simultaneous protein intake on the availability of gut hormones. In sub-study 3 ($n = 6$) milk minerals were compared with the vehicle of intake (water plus sucralose; control). Milk minerals plus protein increased the GLP-1 area under the curve approximately ninefold (43.7 ± 11.1 pmol $\cdot$ L⁻¹ $\cdot$ 120 min; $p < 0.001$) compared with calcium citrate and milk minerals, with no difference between calcium citrate (6.6 ± 3.7 pmol $\cdot$ L⁻¹ $\cdot$ 120 min) and milk minerals (5.3 ± 3.5 pmol $\cdot$ L⁻¹ $\cdot$ 120 min; $p > 0.999$). Sucralose was a component of the control vehicle; an independent effect was not examined.

An open-label randomised controlled study with 49 adults with type 2 diabetes and 48 adults with overweight or obesity without type 2 diabetes (body-mass index ≥ 23 kg/m²) investigated over 12 weeks whether replacement of sucrose in coffee and tea by sucralose alters the composition of the gut microbiome ⁴⁰. Structure and taxonomic composition were profiled by 16S rRNA sequencing in stool samples before and after the intervention; the false-discovery rate was controlled with the Benjamini-Hochberg method ($q < 0.20$). Compared with the control group, the sucralose intervention reduced α -diversity (Shannon index: $P = 0.02$; Simpson index: $P = 0.03$) and increased β -diversity ($P = 0.0...$). The study was open-label; the clinical relevance of the microbial shifts remains open.

A study protocol describes a double-blind, randomised, placebo-controlled crossover study with 33 adults with type 2 diabetes in Malaysia ⁴¹. Participants are to take sucralose (5 mg/kg body weight), saccharin (2 mg/kg body weight) or a placebo (calcium carbonate) daily in capsule form for 7 days per intervention arm, with a four-week wash-out period. Anthropometric measurements, biochemical determinations of glycaemic control, dietary records, physical activity and stool samples are to be recorded. The homeostasis model assessment of insulin resistance is a planned endpoint. This is a protocol; results are not yet available.

A further study protocol describes a randomised, controlled crossover study with 23 participants with a body-mass index between 25 and 39.9 kg/m² ⁴³. Two intervention phases with sucralose (2 mg/kg/day, corresponding to 40 % of the acceptable daily intake) or glucose (control) are planned for 4 weeks each, separated by a four-week wash-out period. The glycaemic response is to be determined in a meal-tolerance test, subjective appetite by visual analogue scale, and changes in the gut microbiome by next-generation sequencing of the bacterial 16S rRNA gene from stool samples. All measurements are planned before and after the intervention phases. The protocol was approved by the ethics committee of the UJAT (No. 0721) and registered in the Australian New Zealand Clinical Trials Registry. Results are still awaited.

Sucralose on Skin and Mucous Membranes: Studies in Humans

A randomised, double-blind crossover study with 24 healthy adults compared the oesophageal clearance of three oral viscous budesonide formulations ²⁶. Honey- and xanthan-gum formulations were compared with the conventional sucralose formulation; each participant received the sucralose formulation and either the honey or the xanthan-gum formulation. Clearance was determined as area under the curve by 1 millicurie technetium-99m sulphur colloid and nuclear-medicine scintigraphy. Xanthan gum had a longer mucosal contact time than sucralose, shown by a higher area under the curve after 3 minutes ($P = 0.002$), whereas honey showed no significant difference in oesophageal clearance compared with sucralose. Taste ratings were significantly higher in the honey group, while ratings for xanthan gum did not differ. Sucralose served here as a reference formulation; an independent therapeutic effect was not examined.

The primary efficacy variable was the change in total nasal symptom score (rhinorrhoea, sneezing, itching, nasal congestion) from baseline to day 14. The original and the reformulated spray achieved comparable improvements in total nasal symptom score at both doses. There was a dose-related difference between the 1- and 2-spray doses. Sucralose was contained in the reformulated preparation as a taste-masking agent; an independent effect was not investigated.

A pharmacokinetic study with 18 healthy volunteers and a one-year safety study with patients with chronic rhinitis (new formulation $n = 430$, original $n = 432$) examined the safety, tolerability and pharmacokinetics of a reformulated azelastine hydrochloride nasal spray with sorbitol vehicle and sucralose as taste-masking agent³¹. In the pharmacokinetic study, 18 healthy volunteers received either a single dose of the new or the original formulation, and pharmacokinetic parameters were determined. In the one-year safety study, tolerability and possible adverse effects on the nasal mucosa were recorded. The new formulation was safe and well tolerated on long-term use and had a comparable pharmacokinetic profile to the original formulation. Sucralose was a component of the new formulation; an independent effect on the nasal mucosa was not examined.

Reviews and Mechanisms of Action

A 2024 review traces the effects of sucralose on metabolism and its possible role in the development of obesity⁵⁸. It covers topics from discovery to analytical detection methods in food samples and examines metabolic effects. The work investigates the association between sucralose intake and obesity and questions the conventional assumption of a weight-regulating role. It reviews in-vivo studies and describes implications for insulin resistance, the gut microbiome and metabolic syndrome. In addition, effects on glucose and lipid metabolism, blood pressure and cardiovascular health as well as a possible involvement in the development of malignancies are discussed. The work concludes with a call for greater public attention, education and updated dietary guidelines. The future perspective emphasises the need for longitudinal studies, examination of alternative sweeteners and refinement of acceptable daily intakes.

A 2026 review addresses the ecotoxicity of sucralose, acesulfame, saccharin and cyclamate⁵⁹. Because of high stability and water solubility, some artificial sweeteners enter the aquatic environment and remain there. Although sucralose, acesulfame, saccharin and cyclamate have been identified in the literature for more than a decade as emerging contaminants, no environmental risk assessment of these substances has yet been carried out. The European Food Safety Authority (EFSA) is currently conducting a re-evaluation of the authorisation of artificial sweeteners, which is still ongoing for sucralose and cyclamate. The review shows that all four sweeteners have been identified as persistent and highly mobile in the aquatic environment. Acute toxicity studies in aquatic model organisms found no general acute hazard. However, there is a pronounced lack of chronic toxicity studies, and data at environmentally relevant concentrations remain still emerging. The review points out that numerous studies with alternative endpoints have found effects within environmentally relevant concentrations.

A 2026 review examines mechanistic aspects of the metabolic effects of sucralose on satiety, obesity, glycaemic control and adipogenesis as well as gut dysbiosis, inflammation and disruption of intestinal permeability⁶⁰. Sucralose (also known as Splenda in combination with dextrose and maltodextrin) is a common non-nutritive sweetener in beverages marketed with health and fitness benefits. Some evidence suggests that sucralose may alter appetite regulation, taste perception and energy intake. In addition, safety concerns exist regarding carcinogenic potential and epigenetic effects on the foetus with consistent maternal consumption. On the basis of current findings on non-nutritive sweeteners, it is concluded that sucralose may be useful short-term for weight reduction as a non-nutritive sweetener, but this must be weighed against possible long-term metabolic side effects and safety precautions.

A 2026 review considers the gut-immune axis and sucralose⁶¹. Sucralose is regarded as one of the most widely used non-nutritive sweeteners and has long been considered metabolically inert and safe within established acceptable daily intakes. Recent evidence, however, suggests that chronic sucralose exposure can alter the microbial composition of the gut, epithelial-barrier function, mucosal inflammation and immune responses. The review examines experimental and clinical evidence on the effects of sucralose on the gut-immune axis with particular attention to possible implications for colitis and colorectal carcinoma. Preclinical studies suggest that sucralose can reduce beneficial short-chain fatty-acid-producing taxa, alter microbial metabolic pathways, disrupt epithelial-barrier-associated molecules and promote inflammatory and immunological changes associated with colitis severity and inflammation-driven tumourigenesis. Preclinical findings also suggest that sucralose can impair the fitness of CD8⁺ T cells and, in experimental models, reduce the response to immune-checkpoint inhibitors via microbiome-dependent mechanisms with altered arginine and citrulline metabolism; whether this occurs in humans has not been investigated. Human studies show that sucralose can alter the composition of the gut and oral microbiome and influence metabolic responses, although these effects appear heterogeneous and context-dependent.

A 2023 review addresses non-sugar sweeteners and cancer⁶². It provides a comprehensive quantitative overview of toxicological and epidemiological evidence on the possible association between non-sugar sweeteners and cancer. The toxicological part includes assessment of genotoxicity and carcinogenicity data for acesulfame K, advantame, aspartame, cyclamates, saccharin, steviol glycosides and sucralose. The epidemiological part contains the results of a systematic search for cohort and case-control studies. The majority of the 22 cohort studies and 46 case-control studies showed no associations. Some risks for bladder, pancreatic and haematopoietic cancers found in a few studies were not confirmed in other studies. On the basis of the review of both the experimental data on genotoxicity or carcinogenicity of the individual non-sugar sweeteners evaluated and the epidemiological studies, it is concluded that there is no evidence for a cancer risk associated with the consumption of non-sugar sweeteners.

An animal study from 2024 examined the long-term metabolic effects of non-nutritive sweeteners under normal and obesogenic conditions⁶³. Eight-week-old male C57BL/6 mice received acesulfame K (535.25 mg/L), aspartame (411.75 mg/L), sucralose (179.5 mg/L), saccharin (80 mg/L) or steviol glycoside (Reb M, 536.25 mg/L) in drinking water, against the background of a regular or high-fat diet (60 % of calories from fat in the high-fat diet). Water or fructose-sweetened water (82.3 g/L) served as controls. Anthropometric and metabolic parameters as well as microbiome composition were analysed after 20 weeks of exposure. Under a regular diet, chronic intake of non-nutritive sweeteners did not significantly alter body weight, fat mass or glucose metabolism compared with water, although aspartame showed impaired glucose tolerance. Under diet-induced obesity the exposure differed. The work is an animal study; the findings are preclinical.

Overall Picture

Across applications a divided picture emerges. On one side stand a randomised controlled study that found no change in HbA1c, fasting glucose or lipid profile under sucralose⁵, and a meta-analysis that demonstrates no rise in blood glucose after non-nutritive sweeteners². On the other side stand indications of reduced insulin sensitivity after 14 days in healthy adults¹⁷, a rise in HOMA-IR with a sucralose-sweetened maqui-citrus beverage in a 60-day comparison⁶, and altered hypothalamic blood-flow and hunger responses⁴⁹. The cohort and cross-sectional studies show associations with type 2

diabetes⁵³, paediatric hypertension⁵⁴, inflammatory markers⁵⁵, Crohn's disease⁵⁶ and central precocious puberty⁵⁷; these are observational and do not establish causation. The reviews collate mechanistic and preclinical findings on insulin resistance, the gut microbiome, inflammation and barrier disruption^{58, 60, 61}; a further review of various non-sugar sweeteners finds, in the synthesis of toxicological and epidemiological data, no indication of a cancer risk⁶².

It remains open which dose and exposure duration trigger the observed metabolic and microbial changes and whether they translate into clinically relevant endpoints. The two available study protocols on sucralose in type 2 diabetes⁴¹ and in overweight or obesity⁴³ are precisely directed at these questions; their results are still awaited. Likewise, an independent replication of the findings on insulin sensitivity¹⁷ and on HOMA-IR after a sucralose-sweetened maqui-citrus beverage⁶ is still required before they can be regarded as established. In studies on nasal sprays and the oesophagus, sucralose was examined as a taste-masking agent or reference formulation^{26, 31, 37}; an independent effect on the skin was not investigated. What is lacking are long-term, blinded studies with clinical endpoints that test the substance in isolation and at relevant doses.

Methodology of the evidence base

The material comprises randomised controlled studies with parallel and crossover designs, systematic reviews with meta-analysis, prospective cohort and cross-sectional studies, case-control and biosample analyses, animal studies and two study protocols. Intervention duration ranges from a single dose through 7 days to 12 weeks, in cohorts up to a median follow-up of 10 years. Participant numbers range from 6 to 405 907. Endpoints are body-mass index, body weight, waist circumference, HbA1c, fasting glucose, insulin sensitivity, HOMA-IR, lipid profiles, inflammatory markers, gut microbiome, hypothalamic blood flow, heart-rate variability, pain thresholds, cognition, appetite, satiety and performance. Several works examine sucralose only as placebo, control vehicle or taste-masking agent; an isolated effect of the substance cannot be inferred from these.

3. How Robust the Research Is

The research is clearly distributed. The largest block consists of basic-science work, followed by observations in humans and animal studies; controlled studies in humans are present but in the minority. Within the controlled human studies, metabolism predominates by a wide margin, followed by work on the gut and on safety. Cardiovascular, bioavailability, inflammation and cognition are represented by single studies, immune system and oncology by very few. Reviews summarise mainly metabolism and the safety question. The field is therefore broadly conceived but unevenly deep: for metabolism and gut there are several controlled examinations; for other application areas there are individual ones.

Methodologically the field has two strengths and two weaknesses. A strength is the number of randomised, double-blind crossover studies that test the substance under controlled conditions; two systematic reviews with meta-analysis pool the evidence on cardiometabolic health and on glycaemic effect. A further strength is that several works explicitly vary the accompanying matrix. A weakness is grouping: a considerable proportion of the cited works examine sweeteners collectively, so that no statement about sucralose alone can be derived from them. A second weakness is the short observation period of many studies; long-term metabolic consequences are the subject of reviews, not of completed long-term trials.

Group findings are not sucralose findings

The most frequently cited works in the field do not speak of sucralose but of non-nutritive sweeteners. The 2017 meta-analysis pools randomised and prospective cohort studies on cardiometabolic health; the 2022 work examines microbiome-mediated effects of non-nutritive sweeteners on glucose tolerance; the 2019 randomised study compares four calorie-free sweeteners with sucrose. Such designs are useful for the authorisation question but limited for the classification of the individual substance: from a group examination no effect of sucralose alone can be derived. Readers of the citations must therefore distinguish whether sucralose was the test substance or only part of the collective.

The second fault line runs through the results themselves. A randomised controlled trial from 2018 reports that sucralose reduces insulin sensitivity in healthy individuals. A 2015 study describes that sucralose enhances GLP-1 release and lowers blood glucose when carbohydrate is ingested at the same time — but not in people with type 2 diabetes. A 2011 study finds no corresponding appetite effect in normal-weight individuals. These findings do not necessarily contradict each other; they measure different things: different accompanying matrices, different population groups, different markers. That is precisely why the matrix question is not a side issue, but the core.

4. What Is Frequently Claimed — and What the Data Support

CLAIM	STUDY SITUATION
Sucralose supports weight loss	The randomised 2019 study compares four low-calorie sweeteners with sucrose in overweight adults. It examines the group, not sucralose alone; it does not
Sucralose is metabolically neutral	A randomised controlled study from 2018 reports reduced insulin sensitivity in healthy individuals, a 2015 study reports increased GLP-1 release with carb
Sucralose damages the gut microbiome	The 2020 study examines aspartame and sucralose together and reports microbiome parameters as secondary endpoints; the 2019 study examines short-term

CLAIM	STUDY SITUATION
Sucralose is carcinogenic	The 2023 review surveys toxicological and epidemiological evidence on non-sugar sweeteners; the 2022 cohort study investigates artificial sweeteners as a g

5. Profile: Molecule and Charge

Sucralose is not a single molecule, but a **mixture** — there is no molecular formula and no fixed molecular mass. What can be described are functional groups, charge behaviour and redox properties; that is exactly what the next section is about.

6. Charge, Electrons, Radicals

PROVEN Charge, cation exchange and electron transfer of sucralose have not yet been measured. Only the formation of reactive oxygen species (ROS) has been recorded. In human HepG2 cells, sucralose increased ROS formation and triggered endoplasmic reticulum stress ²⁰. In mesenchymal stromal cells, sucralose promoted the accumulation of ROS and adipogenesis ²⁵; ROS were detected via Mitosox-Red staining ²⁵. In cell culture, four artificial sweeteners, including sucralose, increased ROS formation and favoured the plasmid-mediated conjugative transfer to the gut microbiome ²⁴.

PROVEN In human keratinocytes, sucralose increased cell proliferation via PKA-, AMPK-, SIRT-1- and ERK-dependent mechanisms, promoted wound healing and reduced cell death after UVB irradiation ²². A PKA inhibitor completely abolished the sucralose-induced AMPK phosphorylation ²³; PKA and AMPK inhibitors attenuated the sucralose-induced SIRT-1 activity ²¹. **Hypothesis:** The findings come from cell culture of human keratinocytes; they touch on redox processes but do not prove electron transfer by sucralose itself. Whether charge, cation exchange or electron transfer are measurable in humans remains to be determined.

7. Interactions with Other Substances

With which other substances of the platform this substance is **jointly** investigated or discussed in human studies and review articles — combination, comparison or common mechanism of action. The number indicates studies in which both occur.

SUBSTANCE	JOINT STUDIES	OF WHICH CONTROLLED IN HUMANS
Kunsttherapie	58	18
Acceptance and Commitment Therapy (ACT)	55	18
Darmmikrobiom	45	10
Hochintensives Intervalltraining (HIIT)	27	7
Transkranielle Gleichstromstimulation (tDCS)	24	4
Kalium	19	5
Lugol's Solution (Iodine and Potassium Iodide)	18	4
Myofasziale Release	18	7
Chronic Inflammation	13	4
Therapeutic Fasting and Intermittent Fasting	13	8

For sucralose, an interaction with a medicinal substance in the formulation is described: In extruded filaments, the bitter active ingredient diphenhydramine hydrochloride was combined with hydrophilic carriers such as hydroxypropylcellulose and a non-ionic surfactant as well as sucralose and strawberry flavour. The evaluation showed complete taste masking of the bitter diphenhydramine and a synergistic effect of sweetener and strawberry flavour on sweetness, fruitiness and aftertaste ²⁹. In L-cells in vitro, sucralose stimulates the secretion of GLP-1; this effect is inhibited by the simultaneous administration of a T1r2+T1r3 inhibitor ¹⁰.

Observations in humans are available for the uptake of accompanying substances: Sucrose did not offer significantly higher bioavailability of compounds compared to sucralose or stevia ²⁸. In urine samples, it was examined whether gender and the added sweetener (sucrose, sucralose or stevia) influence the bioavailability of (poly)phenol metabolites ³⁰; biostatistical and machine-learning methods were applied to the quantified metabolites ³⁰. Another study found greater somatosensory activity for sucrose compared to sucralose ²⁷. Whether CYP enzymes are involved is an open question.

8. Overdosage, Tolerability, Limits

Sucralose belongs to the group of non-nutritive sweeteners. Figures on overdosage or an upper limit cannot be derived from this.

PLAUSIBLE Non-nutritive sweeteners are considered safe and tolerable, yet their effect on glucose intolerance, sweet taste receptors and the gut microbiota is controversial ³². Artificial sweeteners are found in thousands of food and beverage brands and are currently being re-evaluated by the European Food Safety Authority, the World Health Organization and other health authorities ³³. Dose-finding studies on intake amounts and adverse effects would need to be clarified.

9. The Most Robust Studies

Ordered by strength of evidence, not by familiarity: Reviews and studies with many participants first. The complete evaluation includes significantly more studies; the most robust ones are listed here.

Controlled Studies in Humans

- ¹ Nonnutritive sweeteners and cardiometabolic health: a systematic review and meta-analysis of randomized controlled trials and prospective cohort ... (2017) — Übersichtsarbeit, 405.907 Teilnehmer
- ² Glycemic impact of non-nutritive sweeteners: a systematic review and meta-analysis of randomized controlled trials (2018) — Übersichtsarbeit, 741 Teilnehmer
- ³ Visual scoring of non cavitated caries lesions and clinical trial efficiency, testing xylitol in caries-active adults (2014) — 538 Teilnehmer
- ⁴ A randomized controlled trial contrasting the effects of 4 low-calorie sweeteners and sucrose on body weight in adults with overweight or obesity (2019) — 154 Teilnehmer
- ⁵ Effect of Replacing Sucrose in Beverages with Nonnutritive Sweetener Sucralose on Cardiometabolic Risk Factors Among Asian Indian Adults with Type 2 ... (2024) — 210 Teilnehmer
- Pharmacokinetic characteristics and safety and tolerability of a reformulated azelastine hydrochloride nasal spray in patients with chronic rhinitis (2009) — 432 Teilnehmer
- ⁶ Stevia, sucralose and sucrose added to a maqui-Citrus beverage and their effects on glycemic response in overweight subjects: A randomized clinical ... (2021) — 138 Teilnehmer
- Effects of desensitizing products on the reduction of pain sensitivity caused by in-office tooth bleaching: a 24-week follow-up (2020) — 108 Teilnehmer
- ⁷ Non-Nutritive Sweeteners Intake and Its Association With Sugars Intake Among Chilean Toddlers (2025) — 450 Teilnehmer
- ⁸ Personalized microbiome-driven effects of non-nutritive sweeteners on human glucose tolerance (2022)
- ⁹ Effects of carbohydrate sugars and artificial sweeteners on appetite and the secretion of gastrointestinal satiety peptides (2011)
- ¹⁰ Effects of oral ingestion of sucralose on gut hormone response and appetite in healthy normal-weight subjects (2011)
- Clinical trial: effect of active lactic acid bacteria on mucosal barrier function in patients with diarrhoea-predominant irritable bowel syndrome (2008)
- ¹¹ Novel multi-sugar assay for site-specific gastrointestinal permeability analysis: a randomized controlled crossover trial (2013)
- ¹² The Effects of Non-Nutritive Artificial Sweeteners, Aspartame and Sucralose, on the Gut Microbiome in Healthy Adults: Secondary Outcomes of a ... (2020)
- ¹³ Short-term impact of sucralose consumption on the metabolic response and gut microbiome of healthy adults (2019)
- ¹⁴ Sucralose enhances GLP-1 release and lowers blood glucose in the presence of carbohydrate in healthy subjects but not in patients with type 2 diabetes (2015)
- ¹⁵ Effects of diet soda on gut hormones in youths with diabetes (2012) — 25 participants
- ¹⁶ Hormonal responses to non-nutritive sweeteners in water and diet soda (2016) — 31 participants
- ¹⁷ Sucralose decreases insulin sensitivity in healthy subjects: a randomized controlled trial (2018) — 33 participants
- ¹⁸ Effects of sucralose on insulin and glucagon-like peptide-1 secretion in healthy subjects: a randomized, double-blind, placebo-controlled trial (2018)
- Diet and Headache: Part 1 (2016)
- Development and Validation of Test for "Leaky Gut" Small Intestinal and Colonic Permeability Using Sugars in Healthy Adults (2021)
- ¹⁹ A 12-week randomized clinical trial investigating the potential for sucralose to affect glucose homeostasis (2017)

10. Summary

In humans, the systematic review with meta-analysis of 29 randomised studies with 741 participants shows that blood glucose levels do not rise after intake of non-nutritive sweeteners, but gradually fall over the observation period ². A randomised controlled trial with 210 adults with type 2 diabetes finds no change in HbA1c, fasting glucose, lipid profile and inflammatory markers over 12 weeks, with slightly decreasing weight, body mass index and waist circumference ⁵. Both statements concern the substance group; they carry no statement for sucralose in isolation.

It is plausible that the findings depend on the preparation and the accompanying food: the studies dissolve sucralose in water, in diet lemonade or together with carbohydrate and arrive at different results. **Hypothesis:** The decline in insulin sensitivity after 14 days ¹⁷ and the increase in HOMA-IR after 60 days with a maqui-citrus drink containing sucralose ⁶ could indicate a common early response of insulin regulation that no longer appears with longer observation of clinical endpoints. It could follow from this that, in addition to sucralose, the accompanying food and the duration of exposure determine the direction of the findings.

11. What Remains Open

- **Accompanying matrix:** Does a study clarify whether the observed hormone and glucose effects depend on the sweetener, the drink or the simultaneously ingested carbohydrate?
- **Single substance:** Which randomised trial investigates sucralose alone, instead of sweeteners as a group, and thereby separates the individual finding from the group finding?
- **Long term:** Which controlled study observes metabolic markers over months instead of over hours or days?

12. Further Paths

Topics, mechanisms of action and chains in which this substance occurs — for all who wish to look beyond the substance.

- Interaction network

Frequently Asked Questions

Should I treat sucralose differently from other sweeteners in counselling?

The study situation rarely separates the substances so far. Many of the most-cited works test non-nutritive sweeteners together, and the controlled human studies on sucralose focus on metabolism and the gut. No separate recommendation can be derived from this; for counselling it is important that findings on the group cannot be transferred directly to the individual substance.

What does it mean that sucralose is tested in water, in diet lemonade and with carbohydrate?

It means that the studies measure different things. In water the focus is on the reaction to the sweetener, in diet lemonade the reaction to a drink with further constituents, with carbohydrate the interplay of both. The works on gut hormones and appetite explicitly distinguish these conditions. Comparisons between studies are therefore only permissible when the accompanying matrix matches.

How robust are the findings on insulin sensitivity?

Controlled studies with differing directions stand opposite each other here. A randomised work from 2018 reports reduced insulin sensitivity in healthy persons, a work from 2015 reports increased GLP-1 release and lower blood glucose with simultaneous carbohydrate intake. Both are individual controlled studies. A robust statement only arises when independent replications under identical conditions are available.

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Research, put in context. Every statement leads to the original paper; the decision in the individual case rests with the treating professional.
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