Remission is a beginning. What helps it last?



Original AI-generated conceptual art; no study participants or data depicted.

From The Arising, October 31, 2026, feature 01, pp. 7-9. Results paper online October 1; this is a new reading, not a new trial.

What the trial found

RESET randomized 96 adults aged 18-45 with early-onset type 2 diabetes and obesity, not using insulin, across three English and Canadian centres. A supervised low-energy diet and exercise program was compared with usual care. At 24 weeks, remission occurred in 27 of 50 participants (54%) versus 2 of 46 (about 4%). The reported absolute difference was about 50 percentage points (95% confidence interval 33-64). [1]

The result, the limits, the next question

Remission meant HbA1c below 6.5%, without glucose-lowering medication for at least 12 weeks. Participants knew their assignments; endpoint assessment was blinded. Missing outcomes counted as no remission. Eighty-six participants attended final assessments. At least one adverse event occurred in 43/50 versus 6/46, mainly early constipation, headache, dizziness or fatigue; no serious adverse events were reported. [1]

A program, not a slogan

The original article makes clear that this was an organized intervention, with an intensive phase followed by maintenance and clinical supervision. It should not be translated into “just eat less,” an instruction to stop medication, or a test of someone's character. The intervention included support that a headline can easily erase.

The 2022 published protocol specified adults within six years of diagnosis and a 24-week primary endpoint. It provides design context, not proof of results. [2] The trial did not include a diet-only comparison: the combined program's result cannot tell us how much benefit exercise added on its own.

Keep the horizon visible

Remission at six months is encouraging. It does not establish a permanent cure, fewer future cardiovascular events or longer life. This small, selected population also cannot stand in for everyone with diabetes. Longer follow-up and delivery outside research centres remain important questions.

The human reason to care is the possibility of changing a disease trajectory early. The practical research question is how to make a successful program sustainable, accessible and compatible with everyday life. Neither question requires pretending the uncertainty has disappeared. Intensive dietary programs and medication changes need clinical supervision.

Sources, dates and scope

[1] Dasgupta and colleagues. RESET for Remission results, The Lancet Regional Health - Americas, online October 1, 2026. This update checked the publisher's indexed abstract and funding summary; full results text, supplements and author disclosures were unavailable for this review. Funders listed: UK Medical Research Council, Canadian Institutes of Health Research and John R. McConnell Foundation. No full-text audit is claimed.

[2] Published trial protocol, September 21, 2022; ISRCTN15487120. The registry's current full record could not be retrieved in this review. The protocol is not the results paper.

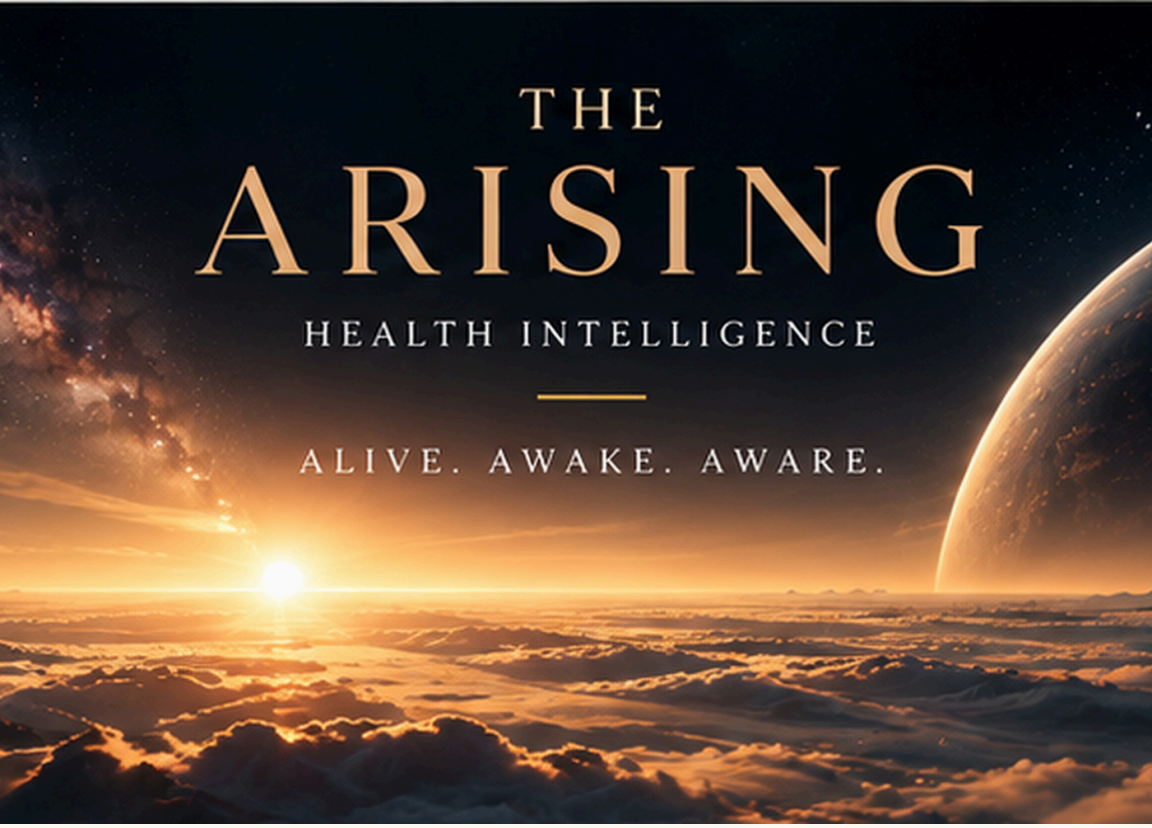
Editorial source: The Arising, October 31, 2026, full feature 01, pages 7-9, “Low-Calorie Diet Plus Exercise Put 54% of Younger Diabetics in Remission.” That article relied on secondary coverage. This adaptation corrects its observational-study badge to randomized trial, and uses serious rather than severe for the results abstract's adverse-event wording. Original source preserved. This is an update to the nutrition collection, not a newly published full newsletter.

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Nutrition, movement and metabolic treatment share questions about support and sustainability. These reading links do not imply that different interventions have been compared head to head.

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13 / EVERYDAY HEALTH

Beyond the Nutrition Label

Food patterns, processing, and why a dietary name never tells the whole biological story.

JOHN GRIFFIN, MD

REVIEW EDITION · OCTOBER 2026

Begin with the food and the question

Nutrition becomes difficult to understand when the name of a diet is asked to stand in for its ingredients, energy intake, preparation, social setting, and the people eating it. A label may organize a conversation. It cannot substitute for a study description.

This booklet pairs two very different experiments. One compares acute responses to meals in people. The other examines intestinal tumor formation in genetically susceptible mice. They belong together because they show why a mechanism and a clinical recommendation require separate reasoning. [1,2]

The timescale changes the answer

A glucose response after a meal, a change in weight over months, and a disease outcome over years are different endpoints. An intervention can look favorable on one and remain uncertain on another. Matching meals on familiar nutrients can also leave differences that a study is designed to investigate.

The reader's task is to keep the comparison visible. What was actually eaten? What did the control group receive? Was the exposure measured or assigned? Were participants able to keep doing it?

A workable pattern

The practical question is broader than winning an argument about a diet. Can a person obtain, prepare, afford, and enjoy the food? Does the pattern fit their nutritional needs and medical circumstances? Is the expected benefit proportionate to its burden?

These readings encourage a more exact conversation. They do not justify translating a mouse result into a universal prohibition,

or an acute response into a promise of longer life. Food deserves the same careful distinction between interesting biology and demonstrated benefit as any other intervention.

FROM THE ARISING / 2026-10-17

Two meals can match on the label and differ in the body

A controlled experiment tests food processing beyond calories and macronutrients.

A nutrition label is useful, but it does not describe every property of a meal. A randomized crossover experiment asked whether nutritionally matched ultraprocessed and non-ultraprocessed meals produced different metabolic and brain responses.

The study involved 57 healthy-weight adults. In a crossover design, participants receive different conditions at different visits, allowing each person to contribute to both sides of the comparison. This can reduce some between-person differences, although it does not remove every source of bias or carryover.

What the experiment measured

The ultraprocessed meals produced greater insulin and energetic responses, with lower carbohydrate oxidation relative to the comparison meals. Differences in peak carbohydrate oxidation were associated with striatal responses to food cues.

Participants’ subjective food valuation did not differ between conditions. Certain brain-response relationships did. The abstract does not supply a single numerical effect that can fairly summarize all of these different physiological and imaging outcomes.

Why an acute result is not a long-term verdict

The experiment examined responses to meals, not years of eating. It did not establish future diabetes incidence, weight trajectories or lifespan. A short-term difference in insulin response is not automatically evidence of harm, and a brain-imaging result is not proof that a food is addictive.

The category “ultraprocessed” also contains many products. A result from matched experimental meals does not demonstrate that every food within that category has the same effect. Reproducibility across meals, populations and longer exposures matters.

What the study adds

The useful contribution is a direct experimental test of a question that observational nutrition studies cannot easily isolate: can processing-related properties matter even when calories and major nutrients are matched? The findings support further investigation rather than a universal clinical prescription.

For someone reading nutrition news, the methodological lesson travels well. Ask whether a study measured intake, weight, an acute biological response or a clinical event. Each outcome answers something different. An interesting mechanism should not silently become a claim about disease prevention.

For Arising Health, this belongs beside—not in place of—the clinical literature on dietary patterns and metabolic treatment. It is possible to take food structure seriously without suggesting that a nutrition label is useless or that one short experiment resolves all the choices at a grocery store.

Sources and evidenceMetabolic and neural responses to ultraprocessed foods: a randomized, controlled, crossover studyOctober 2026 journal issue · Acute randomized crossover experiment Open research →

FROM THE ARISING / 2026-10-13

The same diet can ask different questions of different tissues

A mouse study separates dietary fat metabolism from ketones—and should not become a human cancer verdict.

A diet’s name is not a complete description of its biological effects. Ketogenic diets change fat exposure, carbohydrate availability, insulin signaling and ketone production together. A mechanistic study asks which of those changes mattered for intestinal tumors in cancer-prone mice.

That last phrase is essential. The experiment did not test whether a ketogenic diet causes cancer in people, and it did not estimate a personal cancer risk for someone choosing a way to eat.

Following the mechanism

In mouse models of spontaneous intestinal adenoma formation, the ketogenic diet increased tumor burden and shortened survival. The investigators manipulated the production and use of ketones, but those manipulations did not explain the tumor effects.

The stronger signal involved dietary lipids and their oxidation. Removing a set of intestinal PPAR regulators weakened the diet-associated expansion and activity of intestinal stem cells. Disrupting CPT1A, a component of fatty-acid oxidation, limited adenoma formation specifically under the ketogenic diet.

This is a useful example of why a mechanism should be tested rather than inferred from a label. “Ketogenic” does not establish that ketones caused every observed outcome.

The human boundary

Genetically susceptible mouse models are designed to make particular disease processes experimentally visible. Their responses may not represent those of humans with ordinary cancer risk. The abstract does not provide a human treatment comparison, a cancer-incidence estimate or a recommended dietary prescription.

The work is relevant to questions about inherited intestinal-cancer susceptibility, but clinical advice for such a person requires evidence and context beyond this experiment. It would be equally unjustified to use the findings to promote ketone supplements: the study does not establish their safety or benefit for cancer prevention.

A more useful nutrition conversation

The editorial lesson is not that one diet has now been declared good or bad. It is that tissue, genotype, food composition and measured outcome belong in the interpretation. A diet could change weight favorably while having other effects that need separate investigation.

For future research, the question is how much of the pathway carries into human biology and under what conditions. For a reader, the immediate gain is a sharper way to evaluate a headline: who was studied, what was changed, and what actually happened?

This was an August 2026 paper revisited in the October 13 edition. Its value lies in the precision of the experiment, not in presenting older mechanistic work as today’s clinical breakthrough.

Sources and evidenceKetogenic diet mediates intestinal tumorigenesis through lipids not ketonesAugust 2026 journal issue · Mouse and mechanistic experiments
Open research →

SOURCES & READING SCOPE

Keep the claim beside its evidence

1. Zach Hutelin, Monica Ahrens, Mary Elizabeth Baugh, et al. Metabolic and neural responses to ultraprocessed foods: a randomized, controlled, crossover study. Nature metabolism. 2026 Oct;8(10):2152-2164. doi:10.1038/s42255-026-01619-4. PMID:42834140. Open source →

2. Jessica E S Shay, Fangtao Chi, Constantine N Tzouanas, et al. Ketogenic diet mediates intestinal tumorigenesis through lipids not ketones. Nature. 2026 Aug;656(8127):474-483. doi:10.1038/s41586-026-10779-y. PMID:42457955. Open source →

Selected primary publication records and abstracts checked 25–26 October 2026. This is a focused reading companion, not a systematic review, a treatment protocol, or a complete appraisal of methods, supplements and later evidence. The following readings retain the complete article prose from the October 11–25 publishing package; original study context and limitations are preserved.

KEEP FOLLOWING THE IDEA

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