RESEARCH BRIEF · THE EVIDENCE, PLAINLY

The Dip Comes Later

ISSUE 91 · AUGUST 25, 2026 · METABOLIC NEURO

Two large continuous-glucose studies changed the shape of this topic. The first showed that people eating identical, standardized meals produce very different glucose curves, with meal composition explaining a minority of the variation. The second showed that the dip two to three hours after eating predicts hunger and later food intake better than the peak does. A third study, a 2025 randomized trial, tested the step almost everyone assumes follows from those two, that flattening the curve sharpens thinking, and did not find it.

WHAT WAS ACTUALLY STUDIED

01

Berry et al. (2020), Nature Medicine

TIER I · LARGE MULTI-COHORT OBSERVATIONAL STUDY

PREDICT 1 recruited 1,002 healthy adults in the United Kingdom, including twin pairs, and measured postprandial responses to identical standardized meals in a clinical setting and at home over roughly two weeks, with continuous glucose monitoring plus repeated blood sampling. Findings were then independently validated in a separate cohort of 100 adults in the United States. Between-person variability in response to the same meals was large: population coefficients of variation were 68 percent for glucose, 103 percent for triglyceride, and 59 percent for insulin. Variance decomposition for postprandial glycemia attributed 15.4 percent to meal macronutrient composition, 6.0 percent to person-specific factors such as gut microbiome, and 9.5 percent to genetic variants. A machine-learning model predicted glycemic responses at r = 0.77. The twin design is what makes the genetic estimate credible.

02 Wyatt et al. (2021), Nature Metabolism

TIER 1 · LARGE MULTI-COHORT OBSERVATIONAL STUDY

Drawing on the same research programme, this analysis followed 1,070 participants across a UK exploratory cohort and a US validation cohort through 8,624 standardized meals and 71,715 freely chosen meals, all under continuous glucose monitoring, with self-reported hunger and recorded energy intake. The question was which feature of the postprandial curve best tracks appetite. The answer was the dip: the average glucose level 2 to 3 hours after a meal, expressed relative to that person's own pre-meal baseline, outperformed both the 0 to 2 hour peak and the 0 to 2 hour incremental area under the curve. Larger dips predicted increased hunger at 2 to 3 hours ($r = 0.16$), a shorter interval until the next meal ($r = -0.14$), greater energy intake at 3 to 4 hours ($r = 0.19$), and greater energy intake across the following 24 hours ($r = 0.27$), all at $p < 0.001$. Directions were consistent in the US cohort. Several authors are employed by Zoe Global Ltd, a commercial personalized-nutrition company, which is disclosed in the paper and worth holding in view.

03 Mohapatra et al. (2025), European Journal of Nutrition

TIER 1 · RANDOMIZED CROSSOVER TRIAL

A decentralized randomized controlled crossover trial in 28 healthy adults aged 50 to 65, registered as NCT05801731. Each participant completed two intervention periods of three consecutive test days, consuming either a low-glycemic-index snack or a control snack twice daily. Cognitive performance, self-reported cognitive ability, mood, and appetite were sampled six times on each of the six test days through a mobile application, with continuous glucose monitoring throughout, and HbA1c plus fasting glucose taken as markers of glucoregulatory status. The low-GI snack blunted postprandial glucose responses as intended and did not affect cognitive functioning. Significant interactions between the snack effect and glucoregulatory status appeared for Spatial Memory ($p < 0.01$), Symbol Search ($p < 0.05$), and a Composite Cognition score ($p < 0.05$), with a trend for subjective cognitive ability ($p = 0.07$), all in the direction of poorer responses in participants with poorer glucose regulation. Fluctuations in blood glucose did not mediate the cognitive effects of the snacks, nor cognitive fluctuation across the test days.

WHAT THEY FOUND

The same meal does not produce the same curve in different people, and the gap is not small. Across 1,002 adults eating identical standardized meals, the population coefficient of variation for postprandial glucose was 68 percent. Put plainly: the spread of responses to one fixed meal was large enough that describing any meal as high-glycemic or low-glycemic for people in general glosses over most of what is actually happening (Berry et al., 2020).

68% population coefficient of variation in postprandial glucose response across 1,002 adults eating identical, standardized meals (Berry et al., 2020, PREDICT 1).

The food explains a minority of the response. In the same study's variance decomposition for postprandial glycemia, meal macronutrient composition accounted for 15.4 percent and person-specific factors such as the gut microbiome for 6.0 percent. Genetic variants had only a modest impact on prediction, 9.5 percent for glucose, which is a separate metric from the variance split and should not be added to it. Most of the variance is not accounted for by the components this study quantified, which is itself an honest finding. What the numbers do rule out is the assumption that the plate is the dominant term (Berry et al., 2020).

When the goal is predicting how a person feels and what they do next, the descent beats the peak. Across 1,070 people and roughly 80,000 monitored meals, the glucose dip 2 to 3 hours after eating, measured against that person's own baseline, predicted hunger, time until the next meal, and energy intake at 3 to 4 hours and at 24 hours better than the peak or the total rise did. The correlations are modest, from 0.14 to 0.27, which is the expected magnitude for behavioral outcomes measured in free-living conditions, and the directions held in an independent cohort (Wyatt et al., 2021).

The step from appetite to cognition has been tested, and it did not carry. A 2025 randomized crossover trial gave 28 healthy adults a low-GI snack twice daily for three days with cognition sampled six times a day by phone. The snack blunted the glucose response as designed, and cognitive functioning did not change. Glucose fluctuation did not mediate the cognitive effects of the snacks, and did not explain cognitive fluctuation across the test days either (Mohapatra et al., 2025).

The one clear cognitive signal in that trial ran through the person, not the snack. Participants with poorer glucoregulatory status, indexed by higher HbA1c and fasting glucose while remaining non-diabetic, showed poorer cognitive responses to the low-GI intervention on Spatial Memory, Symbol Search, and the composite score. The authors note that this interaction was detectable in subjects with normal to only mildly compromised glucose regulation (Mohapatra et al., 2025).

WHERE THE EVIDENCE STANDS

Three sources, and the honest reading separates two claims that are usually sold as one.

The well-supported claim is about individuality and appetite. Glucose responses to identical meals vary enormously between people, and the food itself explains a minority of that variation (Berry et al., 2020). Across people, the average 2-to-3-hour dip measured against each person's own pre-meal baseline is the feature that best tracks hunger and subsequent eating, better than the peak that popular advice fixates on (Wyatt et al., 2021). Note the unit of analysis: these are correlations across participants' averaged responses, not a demonstration that a given person's individual dips drive their individual hunger episodes. Both come from large samples with independent validation cohorts. Both are observational, and both describe appetite and eating behavior, which is exactly what they measured.

The under-supported claim is the one about focus. The inference that a smoother glucose curve produces sharper thinking is intuitive, widely marketed, and, in the most direct test available, not observed. Blunting postprandial glucose in healthy adults produced no cognitive benefit, and glucose fluctuation did not explain the cognitive variation that did occur (Mohapatra et al., 2025). One small trial does not close a question, and a null result in 28 people is weak evidence of absence. But it is the trial that exists, and it points away from the promise rather than toward it.

What survives both readings is a smaller and more usable claim. Population-level food rules are a poor instrument for an individual whose response to the same meal may sit far from the average. Personal observation is the better instrument, the descent is the window worth observing, and the honest reason to observe it is appetite and energy, not a documented cognitive gain. Arranging demanding work away from a reliably falling window is a scheduling decision a person can make on their own data. It does not require the cognitive claim to be true, which is fortunate, because it is not yet established.

WHAT THIS DOES NOT PROVE

Berry and Wyatt are observational. They characterize variation and association, not causation, and they cannot establish that changing a glucose curve changes anything downstream. The reported correlations in the appetite analysis, 0.14 to 0.27, are modest at the individual level even though they are highly reliable across tens of thousands of meals.

Both large studies come from the same research programme, and several authors are employed by Zoe Global Ltd, a company that sells personalized-nutrition products built on exactly this science. The papers are peer-reviewed in strong journals with disclosed conflicts and independent validation cohorts, which is the right handling, and the commercial alignment still belongs in view when the findings are used to justify a purchase.

The cohorts skew toward healthy, predominantly white UK and US adults, with substantial twin representation in PREDICT 1. Generalization to other populations, ages, and metabolic states is not established by these data.

The 2025 cognition trial is small, with 28 participants aged 50 to 65 over three test days per arm. It is not powered to detect small cognitive effects, so it cannot rule out a real but modest glucose-to-cognition link. The interaction findings with glucoregulatory status are secondary analyses across multiple cognitive outcomes and should be treated as a hypothesis for larger trials rather than a settled result.

The trial tested a low-GI snack in a real-world setting, not a controlled comparison of large versus small dips, and cognition was measured by brief mobile tasks rather than a full neuropsychological battery. Different instruments and a different manipulation could produce a different answer.

None of this is medical advice, and no individual should read a group-level finding as a description of their own physiology. Persistent afternoon fatigue has many possible causes, and fasting glucose or HbA1c that is trending upward is a clinical matter that belongs with a doctor rather than with a consumer glucose monitor.

WHAT IT MEANS FOR YOU

Watch hour two, not minute thirty, because the 2-to-3-hour window is where the appetite association was actually found and the immediate post-meal peak is not the feature that predicted anything about how people felt or ate. Change one meal and hold everything else still, because between-person variation is large enough that population advice is a poor guide to your own response, and a single-variable observation over a week is the only way one person generates usable data about themselves. Put demanding work somewhere your curve is not reliably falling, which is a scheduling decision you can defend on your own observation without needing the cognitive claim to be true. Hold the flat-line

promise loosely, because the most direct available test of it blunted the glucose curve on purpose and produced no cognitive benefit, and honesty about that gap is worth more than a protocol built on an inference. And treat drifting glucose numbers as a clinician question rather than a gadget question, since the one clear cognitive signal in the 2025 trial ran through glucoregulatory status in people who were not diabetic. The newsletter walks through those five moves. The research above is why they are the five, and where each one runs out.

GO TO THE SOURCE

TIER 1 · MULTI-COHORT OBSERVATIONAL Berry, S. E., Valdes, A. M., Drew, D. A., Asnicar, F., Mazidi, M., Wolf, J., Capdevila, J., Hadjigeorgiou, G., Davies, R., Al Khatib, H., Bonnett, C., Ganesh, S., Bakker, E., Hart, D., Mangino, M., Merino, J., Linenberg, I., Wyatt, P., Ordovás, J. M., Gardner, C. D., Delahanty, L. M., Chan, A. T., Segata, N., Franks, P. W., & Spector, T. D. (2020). Human postprandial responses to food and potential for precision nutrition. Nature Medicine, 26(6), 964-973. doi.org/10.1038/s41591-020-0934-0

TIER 1 · MULTI-COHORT OBSERVATIONAL Wyatt, P., Berry, S. E., Finlayson, G., O'Driscoll, R., Hadjigeorgiou, G., Drew, D. A., Al Khatib, H., Nguyen, L. H., Linenberg, I., Chan, A. T., Spector, T. D., Franks, P. W., Wolf, J., Blundell, J., & Valdes, A. M. (2021). Postprandial glycaemic dips predict appetite and energy intake in healthy individuals. Nature Metabolism, 3(4), 523-529. doi.org/10.1038/s42255-021-00383-x

TIER 1 · RANDOMIZED CROSSOVER TRIAL Mohapatra, L., Cabral, R., Bhatnagar, M., Chan, P. W., Ng, M., Chua, X. Y., Soon, C. S., Massar, S., de Iorio, M., & Schmitt, J. A. J. (2025). Glucoregulatory status modulates acute cognitive effects of repeated low-glycaemic snack consumption in older adults: a decentralized randomized controlled trial. European Journal of Nutrition, 64(5), 189. doi.org/10.1007/s00394-025-03712-y

Tier 1 means peer-reviewed primary research or meta-analysis, the strongest evidence. Tier 2 means an expert framework or smaller study that traces to peer-reviewed work. We grade every source so you can see the weight behind each claim.

Pegasus Realm publishes Wellness Team Culture and the practice resources behind it.

Education, not medical advice.