

Consumer Continuous Glucose Monitoring in People Without Diabetes: What the Evidence Supports, and What It Does Not

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Abstract

Abstract. Continuous glucose monitors (CGMs) are sold to people without diabetes as tools for metabolic health, weight control and disease prevention. We assessed six common commercial claims against the published literature. A structured search of PubMed and Crossref returned 238 records (165 after de-duplication), of which 16 informed this synthesis, including three systematic reviews and one meta-analysis restricted to non-diabetic populations. In people with normal glucose tolerance, no appreciable glycaemic benefit of CGM use has been demonstrated. Pooled analyses show no significant effect on weight, evidence on cardiovascular risk factors is limited, and no trial reports clinical endpoints. In prediabetes, a small randomised trial reports improved glucose metrics and diet over about a month when CGM feedback is added to nutrition therapy. Measurement validity is a material risk: in one crossover trial, CGM overestimated time above 7.8 mmol/L about four-fold against capillary sampling. Claims that CGM reveals hidden dysregulation in healthy people rest on observational data and on excursion counts that sensor bias may inflate. The defensible commercial claim today is narrow: CGM as a short-term feedback tool inside a structured programme for people with prediabetes.

Keywords: continuous glucose monitoring; prediabetes; normoglycaemia; digital health; evidence assessment; due diligence

Decision summary for investment teams	
Bottom line	A CGM-for-healthy-people business cannot currently point to outcome evidence in its core market. Its evidence base sits in prediabetes, over weeks, in small samples.
Demonstrated	Short-term improvements in glucose metrics and diet in prediabetes (small RCT, 15 per arm); close agreement between two sensors of the same brand.
Not demonstrated	Benefit in normoglycaemic users; weight loss; cardiovascular or clinical outcomes; accuracy against capillary blood after meals.
Main risks	Sensor bias inflating “spikes”; industry-affiliated evidence (44% of trials in one meta-analysis); regulatory exposure of health claims.

1. Question and scope

Direct-to-consumer CGM products are marketed to people without diabetes with claims about metabolic health, weight and long-term disease risk. The question for an investor is whether those claims rest on evidence strong enough to survive scrutiny from regulators, clinicians and customers. We tested six claims that recur in the category (Table 1). The population of interest is adults with normal glucose tolerance; adults with prediabetes are reported separately because the evidence differs.

2. Methods

Search. We ran the Rapid Research Intelligence literature pipeline on 20 September 2026 with six query strings covering CGM in normoglycaemic, prediabetic and general populations, behavioural outcomes, postprandial response and cardiovascular endpoints. Sources were PubMed (E-utilities) and Crossref, up to 20 records per query and source. Semantic Scholar and OpenAlex were queried but returned rate-limit errors and contributed no records. The search yielded 238 records and 165 after de-duplication.

Selection. Titles and abstracts were screened for adults

without diabetes or with prediabetes, CGM used as an intervention or as a measurement instrument, and a reported outcome or validity estimate. Sixteen records informed the synthesis: three systematic reviews, one of them with meta-analysis restricted to non-diabetic populations (Liao et al., 2026), one meta-analysis of randomised trials across populations (Richardson et al., 2024), four trials or cohorts on intervention effects, three validity studies and five reference or observational studies.

Extraction and calibration. Effect estimates, sample sizes and conclusions were extracted from the published abstracts. Every number in this brief then passed an Evidence Calibration Review: each figure was traced to its source record, and each conclusion was checked for fidelity to the strength of the source (Section 5).

3. Findings by claim

3.1. Claim 1: CGM improves glucose control

The largest synthesis restricted to non-diabetic populations included 23 studies with 1,074 participants (Liao et al., 2026). Pooled across designs, CGM use reduced mean blood glucose compared with controls (SMD −0.54, 95%

CI -1.02 to -0.07 ; Figure 1A). The authors report that this benefit was seen in prediabetes and that “no appreciable glycemic benefit was observed in healthy normoglycemic populations”. A second meta-analysis of 25 randomised trials (2,996 participants) found HbA1c reductions of 0.28 percentage points and 7.4% more time in range with CGM feedback, but 17 of the 25 trials were in type 2 diabetes and the others in type 1 diabetes, gestational diabetes or obesity (Richardson et al., 2024).

In prediabetes, adding real-time CGM feedback to individualised nutrition therapy increased time in range and lowered mean glucose over 30 days (Basiri and Cheskin, 2024). The trial had 15 participants per arm and the abstract reports within-group significance; a between-group test is not reported. In the largest prediabetes trial ($n = 225$), both arms wore CGM, so the trial compares an algorithm-personalised diet against a Mediterranean diet rather than CGM against no CGM (Ben-Yacov et al., 2021). An uncontrolled 8-week cohort of 110 people with type 2 diabetes or prediabetes reported improved glucose control with CGM plus digital coaching; without a control group, the effect of the sensor cannot be separated from the coaching (Park et al., 2026).

3.2. Claim 2: CGM changes diet behaviour

Small trials report shifts in intake. In prediabetes with overweight or obesity, CGM feedback increased whole-grain and plant-protein intake (Basiri and Rajanala, 2025). This trial shares its lead author, sample size and design with Basiri and Cheskin (2024) and may report secondary outcomes from the same cohort; it should not be counted as independent replication until confirmed. Liao et al. (2026) conclude that CGM is “unlikely to achieve weight management goals independently”. Across all populations, only 4 of 25 trials measured dietary change at all (Richardson et al., 2024).

3.3. Claim 3: CGM supports weight loss

Neither meta-analysis found an effect. In non-diabetic populations the pooled BMI effect was SMD -0.25 (95% CI -0.63 to 0.12 ; $P = 0.19$) (Liao et al., 2026). Across populations, effects on BMI and weight were non-significant (Richardson et al., 2024).

3.4. Claim 4: CGM reveals hidden dysregulation

Reference studies describe what normal looks like. In 153 healthy participants wearing a Dexcom G6, median time between 70 and 140 mg/dL was 96%, with about 30 minutes a day above 140 mg/dL and 15 minutes below 70 mg/dL (Shah et al., 2019). A cohort of more than 7,000 non-diabetic adults now provides reference values (Keshet et al., 2023), and population data show glycaemic variability rising with age (Gude et al., 2017). Healthy people also register time in the hypoglycaemic range (median 1.6% below 3.9 mmol/L) (Sofizadeh et al., 2022), which a consumer app may present as a problem.

The “glucotypes” study reported that normoglycaemic individuals reached prediabetic ranges 15% of the time (Hall et al., 2018). This is an observational finding. Its clinical meaning is unknown, and it depends on the accuracy of the sensor, which is the subject of Claim 5.

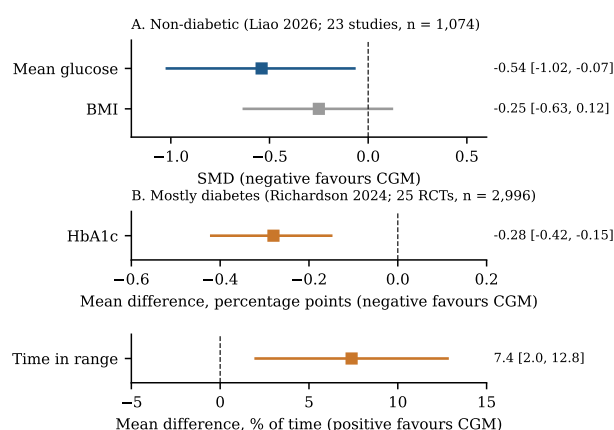


Figure 1: Pooled estimates from the two available meta-analyses. (A) Non-diabetic populations, standardised mean differences. (B, C) Randomised trials dominated by diabetes populations; shown for context, not transferable to healthy users. Values as reported in the source abstracts.

3.5. Claim 5: CGM accurately ranks foods

Two sensors of the same brand agree closely: in 394 ZOE PREDICT participants the coefficient of variation for the 2-hour glucose response was 3.7% within brand and 12.5% between brands, and meal rankings correlated at Kendall $\tau = 0.9$ and 0.7 respectively (Merino et al., 2022). Agreement between two sensors does not establish agreement with blood. In a randomised crossover trial with 15 healthy adults and seven test drinks or meals, CGM read 0.9 mmol/L higher than capillary blood both fasting and after meals, and overestimated time above 7.8 mmol/L about four-fold; baseline adjustment reduced this to about two-fold (Figure 2B) (Hutchins et al., 2025). The bias varied by food and by person. An earlier trial found a mean absolute difference of 0.66 mmol/L (10.5%) between CGM and plasma (Fechner et al., 2020).

3.6. Claim 6: CGM lowers cardiovascular risk

Two systematic reviews address this. One included seven studies and reports that “direct evidence on changes in traditional cardiovascular risk factors was limited” and that evidence on hard endpoints “remains limited” (Ahmed et al., 2025). The other states that the effect on cardiovascular outcomes in this population is unclear (Wilczek et al., 2025). We found no trial with clinical endpoints.

4. Implications for investors

The evidence supports a narrow product: CGM feedback for a few weeks inside a structured nutrition programme for people with prediabetes. It does not yet support the broader wellness positioning used in much of the category. Three consequences follow. First, revenue built on normoglycaemic users rests on engagement, not demonstrated health benefit. Second, marketing claims about weight, longevity or heart health carry regulatory risk in jurisdictions that require substantiation. Third, a company’s own data on “spikes” may partly reflect sensor bias; any internal outcome analysis

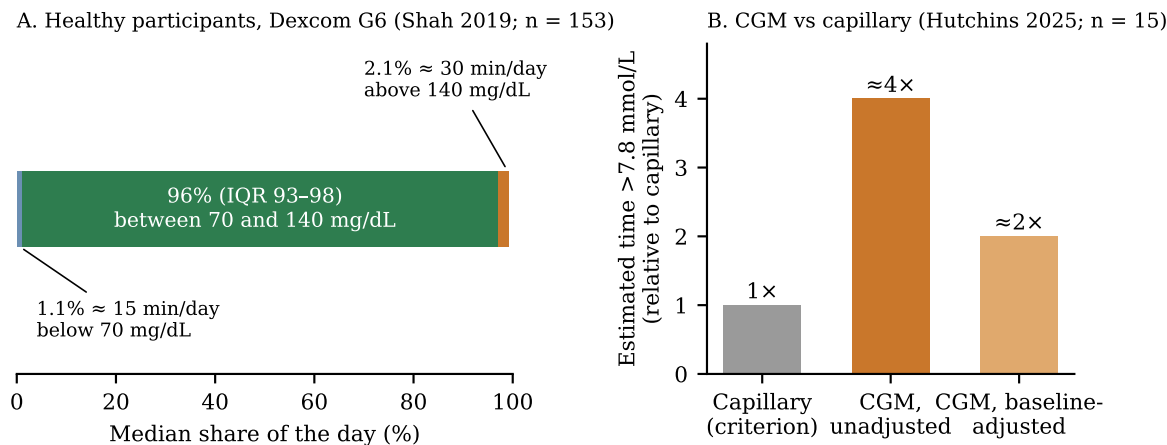


Figure 2: (A) Median distribution of sensor glucose across the day in healthy, non-obese participants (Shah et al., 2019). (B) Time above 7.8 mmol/L estimated by CGM relative to capillary sampling after standardised carbohydrate loads; approximate ratios as reported by Hutchins et al. (2025).

Table 1: Claim-by-claim evidence matrix.

Claim	Best available evidence	What it shows	Status
1. Improves glucose control	Meta-analysis, 23 studies, $n = 1,074$ (Liao et al., 2026); RCTs in prediabetes (Basiri and Cheskin, 2024; Ben-Yacov et al., 2021)	Benefit in prediabetes; none appreciable in normoglycaemia	Prediabetes only
2. Changes diet	RCT, 15 per arm (Basiri and Rajanala, 2025)	Short-term intake shifts; possible overlap with Basiri and Cheskin (2024)	Weak
3. Weight loss	Two meta-analyses (Liao et al., 2026; Richardson et al., 2024)	No significant effect on BMI or weight	Not supported
4. Hidden dysregulation	Observational (Hall et al., 2018; Shah et al., 2019; Keshet et al., 2023)	Excursions occur in healthy people; clinical meaning unknown	Unproven
5. Accurate food ranking	Validity studies (Merino et al., 2022; Hutchins et al., 2025; Fechner et al., 2020)	Sensors agree with each other; overestimate versus blood	Contested
6. Cardiovascular benefit	Systematic reviews (Ahmed et al., 2025; Wilczek et al., 2025)	No direct evidence on risk factors or endpoints	Not supported

should state how excursions were validated.

Questions to put to management.

1. Which population generated your outcome data: normoglycaemic users or prediabetes?
2. Is there a control group that wore a blinded sensor, and was the between-group difference tested?
3. Which outcomes beyond 12 weeks have you measured, and what is retention at 6 months?
4. How are glucose excursions validated against capillary or venous samples?
5. Which published studies were funded by the company or its sensor supplier?
6. Which health claims appear in your marketing, and what substantiation file supports each?

5. Limitations

This is a rapid evidence assessment, not a systematic review. Extraction was based on abstracts, by a single reviewer, from two databases; two further databases were unavailable at search time. Effect sizes are reported as published and were not re-pooled. The grading in Figure 3 reflects this assessment’s judgement and should be read with Table 1. A full-text review would be the next step before an investment decision that depends on Claims 1 or 5.

Conflict of interest

None. This brief was produced independently and was not commissioned by any company in the sector.

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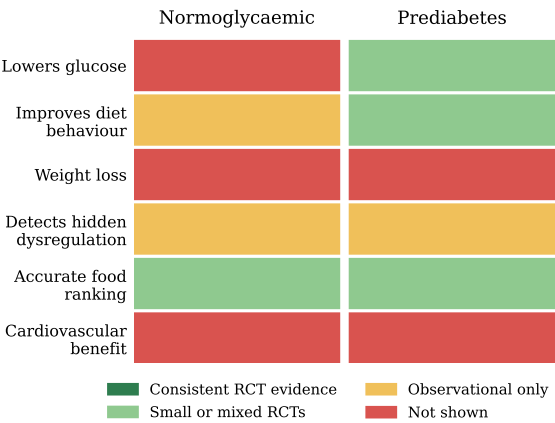


Figure 3: Evidence map: strength of support for each claim by population, as graded in this assessment from the records in Table 1. No claim reached consistent RCT support.

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