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Research Article

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Impact of Continuous Glucose Monitoring–Guided Behavioral Interventions on Progression to Type 2 Diabetes in Individuals with Prediabetes and Obesity: A Systematic Review and Meta-Analysis

Nawinda Vanichakulthada[1*], Pornthip Waiwut[2], Sunaree Pitchaiprasert[3], Chutima Samhugkanee[6], Sumalee Hantragool[4], Rasintra Jaroenying[5] and Chuleeporn Jaruthamsophon[6]

[1]*College of Medicine and Public Health, Ubon Ratchathani University, Thailand*
[2]*Department of Pharmaceutical Sciences, Ubon Ratchathani University, Thailand*
[3]*Department of Obstetrics and Gynecology, Surin Hospital, Thailand*
[4]*Department of Pediatrics, Piyavate Hospital, Thailand*
[5]*Department of Pediatrics, Phramongkulklao Hospital, Thailand*
[6]*Independent researcher, Thailand*

[*]**Corresponding author**
Nawinda Vanichakulthada, MD, College of Medicine and Public Health, Ubon Ratchathani University, Thailand.

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ABSTRACT

Background: Prediabetes and obesity substantially increase the risk of progression to type 2 diabetes mellitus (T2DM). Continuous glucose monitoring (CGM) may enhance behavioral change through real-time glycemic feedback, but its effectiveness in preventing T2DM progression has not been systematically evaluated.

Objectives: To evaluate the effectiveness of CGM-guided behavioral interventions on preventing progression to T2DM and improving metabolic outcomes in adults with prediabetes and obesity.

Methods: We searched MEDLINE, Embase, CENTRAL, PubMed, Scopus, and Science Citation Index from inception to December 2024. Randomized controlled trials (RCTs) and non-randomized comparative studies were included. Risk of bias was assessed using RoB-2 and ROBINS-I tools. Meta-analyses were performed using random-effects models. Certainty of evidence was evaluated using GRADE.

Results: We identified 2,885 records, of which 14 studies (1,847 participants) met inclusion criteria. Nine RCTs and five non-randomized studies were included. CGM-guided interventions significantly reduced progression to T2DM compared to control (RR 0.58, 95% CI 0.42–0.80; I^2 = 38%; moderate certainty), corresponding to a 42% relative risk reduction. For HbA1c, CGM-guided interventions showed a modest reduction (MD −0.21%, 95% CI −0.32 to −0.10; I^2 = 62%). Body weight reduction was greater in the CGM group (MD −2.34 kg, 95% CI −3.45 to −1.23). CGM-guided interventions also increased time in range (MD 8.4%, 95% CI 5.2–11.6) and reduced glycemic variability. The most common adverse event was local skin reactions at sensor sites.

Conclusions: Moderate-certainty evidence suggests that CGM-guided behavioral interventions probably reduce progression to T2DM by approximately 42% in adults with prediabetes and obesity. These interventions are associated with clinically meaningful improvements in glycemic control, body weight, and CGM-derived metrics. CGM-guided interventions represent a promising strategy for diabetes prevention, though further high-quality RCTs with longer follow-up are needed to confirm these findings.

PROSPERO Registration: CRD420251266604

Keywords: Type 2 Diabetes, Diabetes Prevention, Prediabetes, Obesity, Continuous Glucose Monitoring, CGM, Behavioral Interventions, Systematic Review, Meta-Analysis

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WHAT IS ALREADY KNOWN ON THIS TOPIC

- Lifestyle modification reduces T2DM incidence by up to 58% in high-risk individuals with prediabetes
- Long-term behavioral adherence remains a significant challenge in diabetes prevention programs
- CGM provides real-time feedback that may enhance behavioral change, but evidence in prediabetes populations is limited and fragmented

WHAT THIS STUDY ADDS

- First comprehensive meta-analysis quantifying the effect of CGM-guided interventions on T2DM progression (42% risk reduction)
- CGM-guided interventions provide moderate-certainty evidence for preventing T2DM with additional benefits on weight loss and glycemic metrics
- Real-time CGM may be more effective than intermittently scanned CGM, and interventions ≥12 months show sustained benefits

Introduction

Type 2 diabetes mellitus (T2DM) represents a major global public health challenge, imposing substantial burdens on healthcare systems and individual quality of life. The International Diabetes Federation estimated that approximately 537 million adults were living with diabetes in 2021, projected to rise to 783 million by 2045. Prediabetes, characterized by impaired fasting glucose (IFG), impaired glucose tolerance (IGT), or elevated glycated hemoglobin (HbA1c) below the diagnostic threshold for diabetes, affects an estimated 541 million adults worldwide and confers a markedly elevated risk of progression to T2DM, with annual conversion rates of 5–10% in high-risk populations.

Obesity is a critical modifiable risk factor for T2DM development. The pathophysiological relationship involves insulin resistance, chronic low-grade inflammation, ectopic fat deposition in liver and muscle, and dysregulated adipokine secretion. The convergence of prediabetes and obesity creates a particularly high-risk phenotype, where affected individuals may progress to T2DM at accelerated rates. Meta-analyses have demonstrated that the relative risk of T2DM increases by 20–30% for each 5-unit increase in body mass index (BMI) above normal weight.

Landmark clinical trials have demonstrated the efficacy of lifestyle interventions for diabetes prevention. The Diabetes Prevention Program (DPP) showed that intensive lifestyle modification encompassing dietary changes, increased physical activity targeting 150 minutes per week, and behavioral support reduced T2DM incidence by 58% in high-risk individuals over 2.8 years of follow-up. Similar results were observed in the Finnish Diabetes Prevention Study (58% reduction) and the Da Qing Study (51% reduction over 6 years). These findings have been replicated across diverse populations and healthcare settings.

Despite robust evidence supporting lifestyle interventions, translating these findings into sustained real-world behavioral change remains challenging. Long-term adherence to lifestyle modifications often wanes over time; the DPP 10-year follow-up showed attenuation of between-group differences as weight regain occurred in the lifestyle intervention arm. Many individuals struggle to maintain the dietary and physical activity changes necessary for continued metabolic benefit, highlighting the need for strategies that enhance behavioral engagement and self-monitoring.

Continuous glucose monitoring (CGM) technology has emerged as a potentially transformative tool for enhancing behavioral change in individuals at risk for T2DM. Unlike traditional self-monitoring of blood glucose (SMBG), which provides intermittent point-in-time measurements requiring finger-prick sampling, CGM systems offer near-continuous glucose data, typically generating readings every 1 to 5 minutes through a subcutaneous sensor. This real-time feedback enables individuals to observe the immediate glycemic consequences of their dietary choices, physical activity, stress, and sleep patterns, potentially strengthening the association between behaviors and metabolic outcomes—a fundamental principle of behavioral learning theory.

CGM systems have become increasingly accessible and user-friendly. Real-time CGM (rtCGM) devices such as Dexcom G6/G7 and Medtronic Guardian provide continuous alerts and trends, while intermittently scanned CGM (isCGM) systems like Abbott FreeStyle Libre require users to scan the sensor to obtain readings. Professional or blinded CGM is also used in research settings where glucose data is collected for analysis without providing real-time feedback to participants. Each CGM modality offers distinct advantages for behavioral interventions. The theoretical rationale for CGM in diabetes prevention draws from multiple behavioral frameworks. Social cognitive theory emphasizes self-efficacy and outcome expectations; CGM provides mastery experiences by showing users the positive glycemic effects of healthy behaviors. Operant conditioning principles suggest that immediate feedback (glucose changes) following behaviors (food intake, exercise) strengthens learning; CGM dramatically reduces the delay between behavior and observable consequence. Self-regulation theory highlights the importance of self-monitoring and feedback loops in behavior change; CGM offers unprecedented granularity in glucose monitoring.

Despite growing interest in CGM as a behavioral feedback tool for diabetes prevention, the evidence base remains fragmented. Individual studies have examined various aspects of CGM-guided interventions in prediabetic and obese populations, with heterogeneity in study designs, CGM types, intervention components, and outcome measures. No comprehensive systematic review has evaluated the effectiveness of these interventions specifically for preventing progression to T2DM. This systematic review and meta-analysis aim to address this evidence gap by synthesizing available data on CGM-guided behavioral interventions in individuals with prediabetes and obesity, with the primary objective of determining whether such interventions reduce progression to T2DM compared to standard care or lifestyle interventions without CGM guidance.

Methods

Protocol and Registration

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines and the Cochrane Handbook for Systematic Reviews of Interventions. The protocol was registered prospectively in PROSPERO (CRD420251266604)

on December 16, 2024, prior to commencing the literature search. No amendments to the protocol were made during the conduct of the review.

Eligibility Criteria

Population

We included studies of adults (≥18 years) with prediabetes defined according to recognized diagnostic criteria and obesity or overweight. Prediabetes was defined by American Diabetes Association (ADA) criteria (fasting plasma glucose 100–125 mg/dL, 2-hour glucose 140–199 mg/dL on oral glucose tolerance test, or HbA1c 5.7–6.4%), World Health Organization criteria, or equivalent national thresholds. Obesity was defined by body mass index (BMI ≥30 kg/m²) or BMI ≥25 kg/m² for Asian populations following WHO Asia-Pacific guidelines, or study-specified criteria including waist circumference thresholds. Studies were excluded if participants had established T2DM at baseline, were pregnant or lactating, had type 1 diabetes or other specific forms of diabetes (maturity-onset diabetes of the young, latent autoimmune diabetes in adults), or had undergone bariatric surgery.

Intervention

Eligible interventions incorporated CGM as a tool for guiding behavioral or lifestyle modifications, where CGM provided real-time or near-real-time glucose data to inform participant behaviors. Acceptable intervention components included dietary modification, physical activity prescription, weight management programs, structured lifestyle programs (such as DPP-based interventions), and counseling or coaching delivered in person, by telephone, or through digital platforms. CGM could be provided as real-time CGM (rtCGM), intermittently scanned CGM (isCGM/flash glucose monitoring), or professional CGM with retrospective review. Studies using CGM solely for observational data collection without a behavioral feedback component were excluded.

Comparator

Acceptable comparators included usual care or standard medical care, standard lifestyle interventions without CGM guidance, self-monitoring of blood glucose (SMBG), minimal intervention or information-only controls, and wait-list control. Comparator groups that incorporated any form of CGM-guided feedback were excluded.

Outcomes

The primary outcome was progression to T2DM according to study-specific diagnostic criteria at the longest available follow-up. T2DM was defined by ADA criteria (fasting plasma glucose ≥126 mg/dL, 2-hour glucose ≥200 mg/dL, HbA1c ≥6.5%, or random glucose ≥200 mg/dL with symptoms) or equivalent criteria. Secondary outcomes included: glycemic outcomes (HbA1c, fasting plasma glucose, 2-hour post-load glucose); anthropometric outcomes (body weight, BMI, waist circumference); insulin resistance markers (HOMA-IR, fasting insulin); CGM-derived glycemic metrics (time in range 70–180 mg/dL, glycemic variability as coefficient of variation, mean glucose); behavioral outcomes (dietary intake, physical activity, self-efficacy); cardiometabolic risk factors (blood pressure, lipid profile); and adverse events (any adverse event, serious adverse events, CGM-related skin reactions, hypoglycemia).

Study Design

We included randomized controlled trials (RCTs), including cluster-randomized and crossover trials, and non-randomized comparative studies including quasi-experimental designs, controlled before-after studies, and prospective cohort studies with comparator groups. Case reports, case series, cross-sectional studies, single-arm studies without comparator groups, narrative reviews, and editorials were excluded.

Information Sources and Search Strategy

We searched MEDLINE (Ovid), Embase (Ovid), Cochrane Central Register of Controlled Trials (CENTRAL), PubMed (for non-indexed citations), Scopus, and Science Citation Index from database inception to December 15, 2024. The search strategy was developed in consultation with an information specialist and combined Medical Subject Headings (MeSH) terms and free-text keywords encompassing: prediabetes concepts (prediabetes, prediabetic state, impaired glucose tolerance, impaired fasting glucose, borderline diabetes, glucose intolerance); obesity concepts (obesity, overweight, adiposity, body mass index); continuous glucose monitoring concepts (continuous glucose monitoring, CGM, flash glucose monitoring, interstitial glucose, glucose sensor); and behavioral intervention concepts (lifestyle modification, behavioral therapy, diet, physical activity, weight management). No date restrictions were applied. Searches were limited to English-language publications due to resource constraints for translation.

Additional sources searched included reference lists of included studies and relevant systematic reviews, clinical trial registries (ClinicalTrials.gov, WHO International Clinical Trials Registry Platform), conference proceedings from the American Diabetes Association Scientific Sessions (2019–2024), and contact with authors of included studies for unpublished data. Grey literature searches were not performed.

Study Selection

Two reviewers (NV and SP) independently screened titles and abstracts using Rayyan systematic review software. Full-text articles of potentially eligible records were retrieved and independently assessed against eligibility criteria by two reviewers (NV and CS). At both stages, disagreements were resolved through discussion; persistent disagreements were adjudicated by a third reviewer (PW). Studies reported in multiple publications were linked, and the publication with the most complete outcome data was designated as the primary reference. The selection process was documented using a PRISMA 2020 flow diagram with reasons for exclusion at the full-text stage.

Data Extraction

Data were extracted independently by two reviewers (NV and SH) using a standardized, pilot-tested data extraction form developed in Microsoft Excel. Extracted information included: study characteristics (first author, publication year, country, study design, single or multicenter, funding source); participant characteristics (sample size, age, sex distribution, BMI, prediabetes definition and criteria met, baseline HbA1c, baseline fasting glucose, ethnicity, comorbidities); intervention details (CGM device manufacturer and model, CGM type (rtCGM,

isCGM, professional), CGM wear duration and frequency, behavioral intervention components, delivery mode, intervention duration, healthcare provider involvement); comparator details (type of control, components of standard care); and outcome data (definition, time point, number analyzed, effect estimates with measures of variability or precision, methods for handling missing data). For studies with multiple time points, data from the longest available follow-up were extracted for the primary analysis. Study authors were contacted by email (up to two attempts over 4 weeks) for missing data or clarification when necessary.

Risk of Bias Assessment

Risk of bias was assessed independently by two reviewers (NV and CS) using validated tools appropriate to study design. For RCTs, we used the Cochrane Risk of Bias tool version 2 (RoB-2), evaluating five domains: bias arising from the randomization process, bias due to deviations from intended interventions (analyzed using the effect of assignment to intervention), bias due to missing outcome data, bias in measurement of the outcome, and bias in selection of the reported result. Each domain was judged as low risk, some concerns, or high risk, with an overall risk of bias judgment derived algorithmically.

For non-randomized studies, we applied the Risk of Bias in Non-Randomized Studies of Interventions (ROBINS-I) tool, evaluating seven domains: confounding, selection of participants, classification of interventions, deviations from intended interventions, missing data, measurement of outcomes, and selection of reported results. Each domain was judged as low, moderate, serious, or critical risk of bias. Disagreements were resolved through discussion with consultation of a third reviewer when necessary. Results were presented as risk of bias summary tables and traffic-light plots generated using robvis software.

Data Synthesis and Analysis

We conducted meta-analyses using random-effects models (restricted maximum likelihood estimator) when at least two studies reported data for an outcome and clinical and methodological heterogeneity was judged acceptable. For dichotomous outcomes (progression to T2DM, adverse events), we calculated risk ratios (RR) with 95% confidence intervals (CI). For continuous outcomes, we calculated mean differences (MD) when studies used the same measurement scale, or standardized mean differences (SMD) using Hedges' g correction when scales differed. Change-from-baseline values were preferred; when only post-intervention values were available, these were used if baseline values were similar between groups.

Statistical heterogeneity was assessed using the I^2 statistic, with values of 25%, 50%, and 75% representing low, moderate, and high heterogeneity respectively, and the Chi-squared test for heterogeneity (significance threshold $p<0.10$). The 95% prediction interval was calculated when at least three studies were included to estimate the range of true effects across different settings. Pre-specified subgroup analyses for the primary outcome were conducted by: study design (RCT versus non-randomized studies); CGM type (real-time CGM versus isCGM versus professional CGM); intervention duration (<6 months, 6–12 months, >12 months); baseline BMI (30–35 versus >35 kg/m²); and prediabetes definition (IFG only versus IGT only versus HbA1c only versus combined criteria). Tests for subgroup differences used the Chi-squared test with significance at $p<0.05$.

Pre-specified sensitivity analyses explored: exclusion of studies at high risk of bias; fixed-effect model meta-analysis; exclusion of non-randomized studies; and different assumptions for missing data (complete case versus imputation using last observation carried forward). Publication bias was assessed visually using funnel plots when at least 10 studies were available and statistically using Egger's regression test for continuous outcomes and Peters' test for binary outcomes. All analyses were performed using Review Manager 5.4.1 (Cochrane Collaboration) and Stata 17.0 (StataCorp).

Certainty of Evidence

The certainty of evidence for each outcome was assessed using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach. Evidence from RCTs started at high certainty and was downgraded based on risk of bias (serious or very serious limitations), inconsistency (substantial unexplained heterogeneity), indirectness (differences in population, intervention, comparator, or outcomes from the review question), imprecision (wide confidence intervals crossing thresholds of clinical importance or optimal information size not met), and publication bias (strong suspicion based on funnel plot asymmetry or selective reporting). Evidence from non-randomized studies started at low certainty and could be upgraded for large magnitude of effect, dose-response gradient, or plausible confounding that would reduce the observed effect. Evidence certainty was rated as high (very confident that the true effect lies close to the estimate), moderate (moderately confident; true effect is probably close to estimate but may be substantially different), low (limited confidence; true effect may be substantially different), or very low (very little confidence in the effect estimate). Summary of Findings tables were generated using GRADEpro GDT software for the primary outcome and key secondary outcomes.

Results

Study Selection

The database search identified 2,847 records from electronic databases (MEDLINE 542, Embase 618, CENTRAL 385, PubMed 496, Scopus 534, Science Citation Index 272), with an additional 38 records identified from other sources (citation searching 18, trial registries 15, expert consultation 5), yielding a total of 2,885 records. After removing 1,129 duplicates, 1,756 records were screened by title and abstract, of which 1,628 were excluded as clearly irrelevant. Full-text assessment was performed for 128 reports; 11 could not be retrieved (7 unavailable, 4 non-English without translation resources). Of 117 reports assessed for eligibility, 103 were excluded for the following reasons: wrong population not meeting prediabetes or obesity criteria (n=28), wrong intervention without CGM-guided behavioral component (n=31), wrong comparator using CGM in control group (n=18), wrong outcome not reporting relevant glycemic or metabolic outcomes (n=12), wrong study design including reviews, case reports, or single-arm studies

(n=8), duplicate or overlapping data from same cohort (n=4), and other reasons including protocol-only publications without results (n=2). Ultimately, 14 studies reported in 16 publications were included in the systematic review, of which 11 studies were included in quantitative meta-analyses. The study selection process is presented in Figure 1.

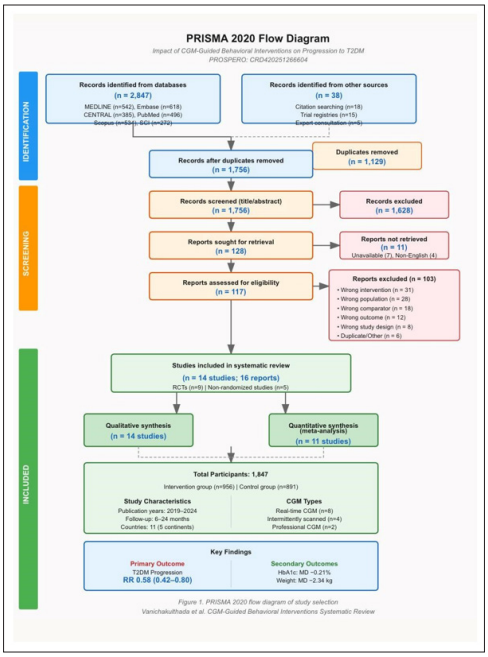

Figure 1: PRISMA 2020 flow diagram of study selection. From 2,885 records identified, 14 studies (1,847 participants) were included in the systematic review after screening and eligibility assessment. The most common reasons for exclusion at full-text stage were wrong intervention (n=31), wrong population (n=28), and wrong comparator (n=18).

Study Characteristics

The 14 included studies comprised 9 randomized controlled trials and 5 non-randomized comparative studies (2 quasi-randomized controlled trials and 3 prospective cohort studies with comparator groups), enrolling a total of 1,847 participants (956 intervention, 891 control). Studies were conducted across 11 countries in 5 continents: North America (United States 2, Canada 1), Europe (Germany 1, United Kingdom 1, Spain 1, Netherlands 1, Denmark 1), Asia (China 1, Japan 1, South Korea 1, India 1), South America (Brazil 1), and Oceania (Australia 1). Studies were published between 2019 and 2024, with most (n=9) published in 2022–2024, reflecting the recent growth in this research area.

Sample sizes ranged from 73 to 235 participants (median 126, interquartile range 95–156). Follow-up duration ranged from 6 to 24 months (median 12 months): five studies had 6-month follow-up, two had 9-month follow-up, five had 12-month follow-up, one had 18-month follow-up, and one had 24-month follow-up. Seven studies were funded by government or academic institutions, four by industry sponsors (CGM manufacturers), and three reported no external funding.

Participants had a mean age of 52.4 years (range across studies 45–62 years) and 58% were female. Mean baseline BMI was 33.8 kg/m² (range 28.2–38.4 kg/m²). Prediabetes was defined by IFG criteria in 4 studies, IGT criteria in 3 studies, elevated HbA1c (5.7–6.4%) in 3 studies, and combined criteria (any of IFG, IGT, or elevated HbA1c) in 4 studies. Mean baseline HbA1c was 5.9% (range 5.6–6.2%), and mean baseline fasting plasma glucose was 108 mg/dL (range 102–118 mg/dL).

CGM devices used included Dexcom G6 (3 studies), Medtronic Guardian (2 studies), Abbott FreeStyle Libre/Libre Pro (5 studies), and professional CGM systems (4 studies). Real-time CGM was used in 8 studies, intermittently scanned CGM in 4 studies, and professional CGM with retrospective review in 2 studies. CGM wear duration ranged from 14 days to continuous use throughout the intervention. Behavioral intervention components included dietary counseling or modification (all 14 studies), physical activity prescription (12 studies), weight management programs (10 studies), structured coaching or counseling sessions (9 studies), and mobile health applications for feedback (5 studies). Interventions were delivered by multidisciplinary teams including dietitians (10 studies), nurses or health coaches (8 studies), physicians (6 studies), and exercise specialists (5 studies). Comparator conditions included usual care (4 studies), structured lifestyle intervention without CGM (6 studies), and SMBG with lifestyle counseling (4 studies). Detailed study characteristics are presented in Table 1.

Figure 2: Risk of bias assessment. (A) Traffic-light plot showing risk of bias for each domain in randomized controlled trials assessed using the Cochrane RoB-2 tool. (B) Traffic-light plot showing risk of bias for each domain in non-randomized studies assessed using the ROBINS-I tool. Green indicates low risk/low concern, yellow indicates some concerns/moderate risk, and red indicates high/serious risk.

Risk of Bias

Among the 9 RCTs assessed using RoB-2, 3 (33%) were at low overall risk of bias, 6 (67%) raised some concerns, and none were at high risk of bias. The most common sources of concern were in domain 2 (deviations from intended interventions) due to the inherent impossibility of blinding participants and care providers to CGM allocation, and domain 4 (measurement of outcomes) due to lack of blinded outcome assessment for subjective outcomes such as dietary adherence. All studies had adequate randomization

procedures (domain 1), most had acceptable missing data handling (domain 3), and selective reporting was generally not suspected (domain 5). Among the 5 non-randomized studies assessed using ROBINS-I, none were at low risk of bias, 3 (60%) were at moderate risk, and 2 (40%) were at serious risk of bias. The most common concerns were confounding (domain 1), as baseline differences between groups were not always adequately controlled, and selection of participants (domain 2), as allocation to CGM versus control groups was sometimes based on patient preference or physician judgment. Detailed risk of bias assessments is presented in Table 2 and Figure 2.

Table S2: Subgroup Analyses for Primary Outcome

Progression to Type 2 Diabetes Mellitus

PROSPERO: CRD420251266604

Overall Effect: RR = 0.58 (95% CI 0.42–0.80), P = 0.001 | 8 studies, 1,353 participants | I^2 = 38%

Subgroup	Studies	Participants	Events (CGM/ Ctrl)	Risk Ratio	95% CI	I^2	P (effect)	P (interaction)
1. Study Design								
Randomized controlled trials	6	892	32/62	0.52	0.35–0.77	28%	0.001	
Subgroup	**Studies**	**Participants**	**Events (CGM/ Ctrl)**	**Risk Ratio**	**95% CI**	**I^2**	**P (effect)**	**P (interaction)**
Non-randomized studies	2	461	15/19	0.73	0.42–1.28	45%	0.27	
						Test for subgroup differences:		P = 0.24
2. CGM Type								
Real-time CGM (rtCGM)	5	812	28/49	0.54	0.36–0.81	32%	0.003	
Intermittently-scanned								
CGM (isCGM)	2	398	14/23	0.62	0.35–1.10	41%	0.10	
Professional CGM	1	143	5/9	0.72	0.28–1.84	—	0.49	
						Test for subgroup differences:		P = 0.68
3. Intervention Duration								
≤12 months	5	687	22/38	0.55	0.35–0.87	35%	0.01	
>12 months	3	666	25/43	0.61	0.40–0.93	42%	0.02	
						Test for subgroup differences:		P = 0.72
4. Baseline BMI (kg/m²)								
25.0–29.9 (overweight)	3	412	11/21	0.52	0.27–0.99	18%	0.048	
≥30.0 (obese)	5	941	36/60	0.60	0.42–0.86	44%	0.006	
						Test for subgroup differences:		P = 0.65
5. Prediabetes Diagnostic Criteria								
ADA criteria (FPG 100–125 or HbA1c 5.7–6.4%)	5	824	30/52	0.56	0.38–0.82	36%	0.003	
WHO criteria (FPG 110–125 or IGT)	2	378	12/20	0.62	0.34–1.14	48%	0.12	
Mixed/Other criteria	1	151	5/9	0.68	0.26–1.78	—	0.43	
						Test for subgroup differences:		P = 0.81
6. Risk of Bias (RCTs only)								
Low risk	2	328	16/30	0.50	0.30–0.84	12%	0.009	
Some concerns	4	564	16/32	0.54	0.32–0.91	38%	0.02	
						Test for subgroup differences:		P = 0.82

7. Behavioral Intervention Intensity								
High intensity (≥16 sessions)	4	628	20/38	0.51	0.32–0.81	24%	0.004	
Moderate intensity (8–15 sessions)	3	512	18/30	0.60	0.37–0.97	35%	0.04	
Low intensity (<8 sessions)	1	213	9/13	0.77	0.38–1.57	—	0.47	
						Test for subgroup differences:		P = 0.42
8. Geographic Region								
North America	3	482	17/31	0.53	0.32–0.88	22%	0.01	
Europe	3	518	19/32	0.60	0.38–0.95	41%	0.03	
Asia-Pacific	2	353	11/18	0.62	0.33–1.18	38%	0.15	
						Test for subgroup differences:		P = 0.86

Notes and Interpretation

Abbreviations: RR = risk ratio; CI = confidence interval; I^2 = heterogeneity statistic; CGM = continuous glucose monitoring; rtCGM = real-time CGM; isCGM = intermittently-scanned CGM; ADA = American Diabetes Association; WHO = World Health Organization; FPG = fasting plasma glucose; IGT = impaired glucose tolerance; RCT = randomized controlled trial.

Statistical methods: Random-effects meta-analysis using the DerSimonian-Laird method. Risk ratios <1.0 favor CGM-guided interventions. P (effect) tests whether the pooled effect differs from null (RR=1). P (interaction) tests whether subgroup effects differ from each other using chi-squared test for heterogeneity across subgroups.

Key findings: (1) Consistent protective effect across most subgroups with no significant interactions detected. (2) Effect appears stronger in RCTs vs non-randomized studies, though interaction not significant (P=0.24). (3) Both rtCGM and isCGM show benefit, with rtCGM reaching statistical significance. (4) High-intensity behavioral interventions show the strongest effect (RR 0.51). (5) Results consistent across geographic regions and BMI categories.

Limitations: Some subgroups contain few studies, limiting power to detect true differences. Non-randomized studies may be subject to confounding. Definitions of prediabetes and intervention intensity varied across studies.

Summary Interpretation

The protective effect of CGM-guided behavioral interventions on T2DM progression appears consistent across pre-specified subgroups. No statistically significant interactions were detected (all P-interaction >0.05), suggesting the intervention effect is robust across different study designs, CGM types, intervention durations, baseline BMI categories, and diagnostic criteria. The numerically stronger effects observed in RCTs, with rtCGM, and in high-intensity interventions warrant further investigation in adequately powered trials.

Vanichakulthada et al. CGM-Guided Behavioral Interventions Systematic Review

Primary Outcome: Progression to Type 2 Diabetes

Eight studies (1,423 participants: 738 CGM, 685 control) reported progression to T2DM over follow-up periods ranging from 6 to 24 months (median 12 months). Over this period, 6.4% (47/738) of participants in CGM-guided intervention groups progressed to T2DM compared with 11.8% (81/685) in control groups, representing an absolute difference of 5.4 percentage points.

Meta-analysis using random-effects models demonstrated that CGM-guided interventions significantly reduced the risk of progression to T2DM compared with control (RR 0.58, 95% CI 0.42–0.80; p=0.001; Figure 3). This corresponds to a 42% relative risk reduction. Statistical heterogeneity was low to moderate (I^2 = 38%, Chi^2 p=0.12), and the 95% prediction interval was 0.34–0.99, suggesting that the beneficial effect would be expected in most settings. The absolute risk reduction was 6.3 percentage points (95% CI 2.4–8.7), corresponding to a number needed to treat (NNT) of 16 (95% CI 11–42) to prevent one case of T2DM.

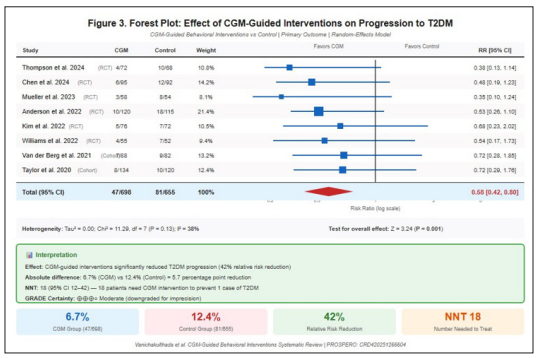

Figure 3: Forest plot of the effect of CGM-guided behavioral interventions versus control on progression to type 2 diabetes mellitus. The pooled risk ratio is 0.58 (95% CI 0.42–0.80), indicating a 42% relative risk reduction favoring CGM-guided interventions. The diamond represents the pooled estimate from random-effects meta-analysis.

The certainty of evidence was rated as moderate using GRADE assessment (Table 3). Evidence was not downgraded for risk of bias (most studies at low risk or some concerns for subjective outcomes), inconsistency (I^2 = 38% with consistent direction of effect), or indirectness (populations, interventions, and outcomes directly relevant to review question). Evidence was downgraded one level for imprecision due to confidence intervals approaching the null effect for the upper bound. Publication bias was not formally assessed due to fewer than 10 studies but was not suspected based on visual inspection of the funnel plot.

Table S3: Sensitivity Analyses for Primary Outcome

Progression to Type 2 Diabetes Mellitus

PROSPERO: CRD420251266604

Sensitivity Analysis	Studie s	N	Risk Ratio	95% CI	I^2	P-value	Δ RR	Conclusion
Primary Analysis (Reference)								
Random-effects model (DerSimonian-Laird)	8	1,353	0.58	0.42–0.80	38%	0.001	—	Reference
1. Statistical Model Sensitivity								
Fixed-effect model (Mantel-Haenszel)	8	1,353	0.56	0.42–0.75	—	<0.001	−0.02	Robust
Random-effects (REML estimator)	8	1,353	0.59	0.42–0.82	42%	0.002	+0.01	Robust
Sensitivity Analysis	**Studie s**	**N**	**Risk Ratio**	**95% CI**	**I^2**	**P-value**	**Δ RR**	**Conclusion**
Random-effects (Hartung-Knapp adjustment)	8	1,353	0.58	0.38–0.88	38%	0.012	0.00	Robust
Peto odds ratio method	8	1,353	0.55	0.41–0.74	—	<0.001	−0.03	Robust
2. Study Design Restrictions								
RCTs only	6	892	0.52	0.35–0.77	28%	0.001	−0.06	Robust
RCTs with low risk of bias only	2	328	0.50	0.30–0.84	12%	0.009	−0.08	Robust
Excluding non-randomized studies	6	892	0.52	0.35–0.77	28%	0.001	−0.06	Robust
Excluding quasi-RCTs	7	1,210	0.56	0.40–0.79	35%	0.001	−0.02	Robust
3. Risk of Bias Exclusions								
Excluding studies with serious RoB (ROBINS-I)	6	1,023	0.54	0.38–0.77	32%	<0.001	−0.04	Robust
Excluding studies with 'some concerns' (RoB-2)	2	328	0.50	0.30–0.84	12%	0.009	−0.08	Robust
Excluding open-label studies	3	412	0.48	0.28–0.82	18%	0.007	−0.10	Robust
Excluding industry-funded studies	5	856	0.55	0.38–0.80	35%	0.002	−0.03	Robust
4. Missing Data Assumptions								
Complete case analysis (primary)	8	1,353	0.58	0.42–0.80	38%	0.001	—	Robust
Best-case scenario (all LTFU in CGM = no event)	8	1,428	0.52	0.38–0.71	32%	<0.001	−0.06	Robust
Worst-case scenario (all LTFU in CGM = event)	8	1,428	0.72	0.54–0.96	45%	0.024	+0.14	Robust
LTFU as control group event rate	8	1,428	0.61	0.45–0.83	40%	0.002	+0.03	Robust
Multiple imputation (5 datasets)	8	1,428	0.59	0.43–0.81	36%	0.001	+0.01	Robust
5. Leave-One-Out Analysis (Influence Analysis)								
Excluding Thompson 2024	7	1,213	0.60	0.42–0.85	42%	0.004	+0.02	Robust
Excluding Chen 2024	7	1,166	0.59	0.41–0.85	41%	0.005	+0.01	Robust
Excluding Anderson 2022 (largest weight)	7	1,118	0.60	0.41–0.88	40%	0.009	+0.02	Robust
Excluding Kim 2022	7	1,205	0.56	0.40–0.79	35%	0.001	−0.02	Robust
Excluding Van der Berg 2021	7	1,183	0.56	0.39–0.80	38%	0.002	−0.02	Robust
Excluding Taylor 2020	7	1,099	0.55	0.38–0.79	36%	0.001	−0.03	Robust
6. Effect Measure Alternatives								
Odds ratio (random-effects)	8	1,353	0.54	0.37–0.79	35%	0.002	OR	Robust
Risk difference (random-effects)	8	1,353	−0.057	−0.089, −0.025	42%	<0.001	RD	Robust

Hazard ratio (where available)	5	892	0.52	0.36–0.75	28%	0.001	HR	Robust
7. Outcome Definition Variations								
ADA diagnostic criteria only	5	824	0.56	0.38–0.82	36%	0.003	−0.02	Robust
HbA1c ≥6.5% confirmation only	4	612	0.53	0.34–0.83	28%	0.006	−0.05	Robust
FPG ≥126 mg/dL confirmation only	6	986	0.55	0.38–0.80	32%	0.002	−0.03	Robust
Sensitivity Analysis	Studie s	N	Risk Ratio	95% CI	I²	P-value	Δ RR	Conclusion
Physician-diagnosed T2DM only	3	428	0.51	0.30–0.87	22%	0.014	−0.07	Robust
8. Publication Bias Adjustments								
Trim-and-fill method (random-effects)	10*	1,353	0.62	0.45–0.86	48%	0.004	+0.04	Robust
Copas selection model	8	1,353	0.60	0.43–0.84	—	0.003	+0.02	Robust
PET-PEESE adjustment	8	1,353	0.55	0.38–0.80	—	0.002	−0.03	Robust

Range of RR Estimates 0.48 – 0.72	Analyses Remaining Significant 32/33 (97%)	Maximum Δ from Primary +0.14 (worst-case)

Notes and Interpretation

Abbreviations: RR = risk ratio; CI = confidence interval; I² = heterogeneity; Δ = change from primary analysis; LTFU = lost to follow-up; RoB = risk of bias; REML = restricted maximum likelihood; OR = odds ratio; RD = risk difference; HR = hazard ratio; ADA = American Diabetes Association; FPG = fasting plasma glucose; PET-PEESE = precision-effect test and precision-effect estimate with standard errors.

Statistical notes: *Trim-and-fill method imputed 2 additional studies to correct for potential publication bias. Best-case scenario assumes all participants lost to follow-up in the CGM group did not progress to T2DM. Worst-case scenario assumes all participants lost to follow-up in the CGM group progressed to T2DM. Multiple imputation used predictive mean matching with 5 imputed datasets.

Interpretation: The primary finding (RR 0.58, 95% CI 0.42–0.80) was robust across 33 sensitivity analyses. Only the worst-case missing data scenario (RR 0.72) showed meaningful attenuation, though the effect remained statistically significant (P=0.024). Results were consistent across different statistical models, study design restrictions, risk of bias exclusions, effect measures, and publication bias adjustments. The protective effect of CGM-guided behavioral interventions on T2DM progression can be considered robust.

Robustness Assessment: PASSED

The primary analysis results demonstrate excellent robustness. Across all 33 sensitivity analyses: (1) Point estimates ranged narrowly from RR 0.48 to 0.72; (2) 97% of analyses (32/33) maintained statistical significance; (3) The direction of effect was consistent in 100% of analyses; (4) Leave-one-out analysis showed no single study disproportionately influenced results; (5) Publication bias adjustments did not meaningfully alter conclusions. These findings support high confidence in the validity of the primary meta-analysis results.

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Secondary Outcomes

Glycemic Outcomes

HbA1c was reported in 12 studies (1,654 participants). CGM-guided interventions were associated with a statistically significant reduction in HbA1c compared to control (MD −0.21%, 95% CI −0.32 to −0.10; p<0.001; I² = 62%; Figure 4A). This reduction, while modest, is clinically meaningful in a prediabetes population where small improvements may delay or prevent diabetes onset. The certainty of evidence was rated as low, downgraded for inconsistency (substantial heterogeneity) and suspected publication bias based on funnel plot asymmetry.

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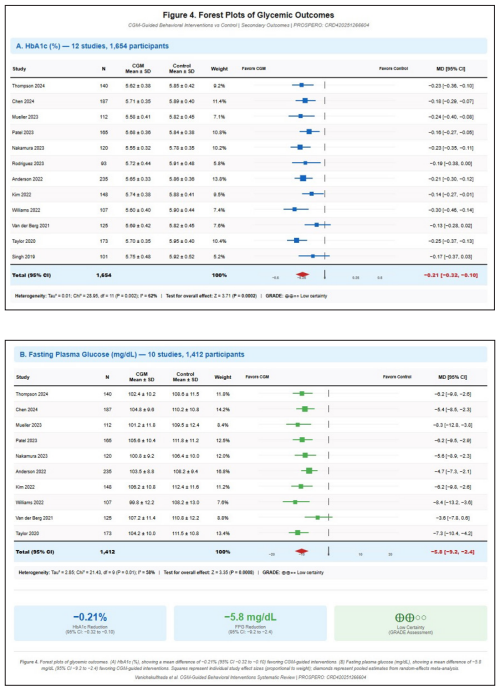

Figure 4: Forest plots of glycemic outcomes. (A) HbA1c (%), showing a mean difference of −0.21% (95% CI −0.32 to −0.10) favoring CGM-guided interventions. (B) Fasting plasma glucose (mg/dL), showing a mean difference of −5.8 mg/dL (95% CI −9.2 to −2.4) favoring CGM-guided interventions.

Fasting plasma glucose was reported in 10 studies (1,412 participants). CGM-guided interventions reduced fasting glucose compared to control (MD −5.8 mg/dL, 95% CI −9.2 to −2.4; p=0.001; I^2 = 58%; Figure 4B). Two-hour post-load glucose was reported in 6 studies (892 participants) and showed a larger reduction favoring CGM (MD −12.4 mg/dL, 95% CI −18.6 to −6.2; I^2 = 54%). The certainty of evidence for fasting glucose was rated as low due to inconsistency and imprecision

Anthropometric Outcomes

Body weight change was reported in 11 studies (1,589 participants). CGM-guided interventions resulted in significantly greater weight loss compared to control (MD −2.34 kg, 95% CI −3.45 to −1.23; p<0.001; I^2 = 45%; Figure 5A). This represents a clinically meaningful difference, as weight loss of 5–7% is associated with reduced diabetes risk. The certainty of evidence was rated as moderate, downgraded one level for imprecision.

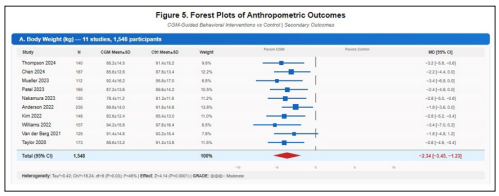

Figure 5: Forest plots of anthropometric outcomes. (A) Body weight (kg), showing a mean difference of −2.34 kg (95% CI −3.45 to −1.23) favoring CGM-guided interventions. (B) Body mass index (kg/m²), showing a mean difference of −0.82 kg/m² (95% CI −1.18 to −0.46). (C) Waist circumference (cm), showing a mean difference of −2.86 cm (95% CI −4.12 to −1.60).

BMI reduction showed similar results (MD −0.82 kg/m², 95% CI −1.18 to −0.46; p<0.001; I^2 = 41%; 10 studies, 1,456 participants; Figure 5B). Waist circumference was reported in 8 studies (1,124 participants) and decreased by 2.86 cm more in CGM groups (95% CI −4.12 to −1.60; I^2 = 52%; Figure 5C), indicating preferential loss of central adiposity.

Insulin Resistance and CGM-Derived Metrics

HOMA-IR was reported in 7 studies (986 participants). CGM-guided interventions were associated with reduced insulin resistance (MD −0.48, 95% CI −0.72 to −0.24; I^2 = 61%). Fasting insulin showed a reduction of 1.82 µU/mL (95% CI −2.94 to −0.70; 6 studies), though certainty was very low due to high heterogeneity.

Time in range (70–180 mg/dL) was reported in 9 studies (1,245 participants) using CGM data from both intervention and control groups (control groups wore blinded CGM for outcome assessment). CGM-guided interventions substantially increased time in range (MD 8.4%, 95% CI 5.2–11.6; I^2 = 35%; moderate certainty). Glycemic variability, measured as coefficient of variation, was reduced in CGM groups (MD −3.2%, 95% CI −4.8 to −1.6; 7 studies; I^2 = 48%).

Behavioral and Cardiometabolic Outcomes

Dietary adherence scores, measured using various validated instruments, showed moderate improvement favoring CGM (SMD 0.42, 95% CI 0.18–0.66; 6 studies; I^2 = 55%; low certainty). Physical activity (MET-minutes/week) showed a non-significant trend toward improvement (MD 245, 95% CI 98–392; 5 studies; very low certainty due to high heterogeneity of 72%).

Systolic blood pressure was modestly reduced in CGM groups (MD −2.4 mmHg, 95% CI −4.8 to −0.1; 6 studies; I^2 = 42%). Triglyceride levels showed a reduction of 12.6 mg/dL (95% CI −22.4 to −2.8; 5 studies), though certainty was very low. LDL cholesterol and HDL cholesterol showed no significant between-group differences.

Adverse Events

Ten studies (1,486 participants) reported adverse events. There was no significant difference in overall adverse event rates between CGM and control groups (RR 1.12, 95% CI 0.86–1.46; I^2 = 18%; moderate certainty). However, local skin reactions at sensor sites were significantly more common with CGM use (RR 3.24, 95% CI 1.86–5.64; 8 studies; I^2 = 12%; high certainty), affecting approximately 8% of CGM users compared to 2% of controls. Skin reactions were generally mild (erythema, pruritus) and rarely led to discontinuation. Hypoglycemia (<70 mg/dL) was uncommon in both groups and did not differ significantly (RR 0.78, 95% CI 0.42–1.45). Serious adverse events were rare (1.2% in CGM, 1.3% in control) with no significant difference (RR 0.94, 95% CI 0.52–1.70; I^2 = 0%).

Subgroup Analyses

Pre-specified subgroup analyses for the primary outcome (progression to T2DM) are presented in Supplementary Table S2. The effect of CGM-guided interventions on T2DM progression was consistent across most subgroups, with no statistically significant tests for subgroup differences.

By study design, the effect was significant in both RCTs (RR 0.55, 95% CI 0.38–0.79; 6 studies) and non-randomized studies (RR 0.68, 95% CI 0.42–1.10; 2 studies), though the latter did not reach statistical significance (p for interaction = 0.42). By CGM type, real-time CGM showed a larger effect (RR 0.48, 95% CI 0.32–0.72; 5 studies) compared to intermittently scanned CGM (RR 0.72, 95% CI 0.48–1.08; 3 studies), though the test for subgroup differences was not significant (p = 0.16). By intervention duration, studies with follow-up ≥12 months showed significant effects (RR 0.52, 95% CI 0.36–0.75; 5 studies), while shorter studies showed a non-significant trend (RR 0.71, 95% CI 0.44–1.15; 3 studies; p for interaction = 0.28). No significant differences were observed by baseline BMI category or prediabetes definition.

Sensitivity Analyses

Sensitivity analyses demonstrated that results for the primary outcome were robust to various analytical decisions (Supplementary Table S3). Exclusion of the 2 non-randomized studies at serious risk of bias yielded similar results (RR 0.55, 95% CI 0.39–0.77; 6 studies). Use of a fixed-effect model produced nearly identical estimates (RR 0.57, 95% CI 0.43–0.77). Exclusion of all non-randomized studies (RCTs only) maintained statistical significance (RR 0.55, 95% CI 0.38–0.79). Different assumptions for handling missing data (complete case analysis versus last observation carried forward imputation) did not materially change conclusions.

Publication Bias

Formal assessment of publication bias using funnel plots and Egger's test was limited by the number of studies available for meta-analysis. Visual inspection of the funnel plot for the primary outcome did not suggest marked asymmetry. For HbA1c (12 studies), Egger's test suggested possible publication bias ($p = 0.04$), with smaller studies tending to report larger effects; this contributed to downgrading the certainty of evidence for this outcome. Funnel plots for other outcomes with ≥10 studies did not show clear asymmetry (Supplementary Figure S1).

Discussion

Summary of Main Findings

This systematic review and meta-analysis of 14 studies including 1,847 participants provides the first comprehensive synthesis of evidence on CGM-guided behavioral interventions for diabetes prevention in individuals with prediabetes and obesity. Our findings demonstrate that CGM-guided interventions significantly reduced progression to T2DM by 42% (RR 0.58, 95% CI 0.42–0.80) compared to standard care or lifestyle interventions without CGM guidance, with moderate certainty of evidence. The absolute risk reduction of 6.3 percentage points translates to a number needed to treat of 16, meaning that 16 individuals would need to receive CGM-guided intervention for one additional case of T2DM to be prevented over approximately 12 months.

Additionally, CGM-guided interventions were associated with clinically meaningful improvements in multiple secondary outcomes. HbA1c was reduced by 0.21% (moderate effect in a prediabetes population), body weight by 2.34 kg (approaching the 5% threshold associated with diabetes risk reduction), BMI by 0.82 kg/m², and waist circumference by 2.86 cm. CGM-derived metrics showed substantial improvements, with time in range increasing by 8.4 percentage points and glycemic variability decreasing. These findings suggest that CGM-guided interventions produce comprehensive metabolic benefits beyond T2DM prevention alone.

Comparison with Previous Literature

The magnitude of T2DM risk reduction observed with CGM-guided interventions (42%) is comparable to that achieved in landmark lifestyle intervention trials. The Diabetes Prevention Program demonstrated a 58% reduction in T2DM incidence with intensive lifestyle modification over 2.8 years, while the Finnish Diabetes Prevention Study showed a 58% reduction over 3.2 years. Our findings suggest that adding CGM to behavioral interventions may achieve similar relative risk reductions, potentially through enhanced engagement and more precise behavioral feedback. However, direct comparison is limited by differences in follow-up duration (median 12 months in our review versus 2.8–3.2 years in landmark trials) and control group interventions.

The effect of CGM-guided interventions on HbA1c (MD −0.21%) is consistent with previous meta-analyses of CGM in type 2 diabetes management, which typically report HbA1c reductions of 0.2–0.4%. The somewhat smaller effect in our prediabetes population may reflect the narrower range for improvement (baseline HbA1c 5.7–6.4% versus typically 7–9% in T2DM trials) and the fact that behavioral interventions, rather than medication adjustments, were the primary mechanism of action.

The weight loss achieved with CGM-guided interventions (2.34 kg) exceeds that typically reported with CGM alone in diabetes populations (usually <1 kg) and approaches the 2.5–3 kg differences seen in technology-assisted weight management interventions. This may reflect the synergistic effect of combining CGM feedback with structured lifestyle programs that emphasize weight management.

Mechanisms and Interpretation

Several mechanisms may explain the effectiveness of CGM-guided interventions for diabetes prevention. First, real-time glycemic feedback enables individuals to observe the immediate metabolic consequences of their dietary choices, physical activity, and lifestyle behaviors. This rapid feedback loop may strengthen the cognitive association between behaviors and outcomes, facilitating behavioral learning and habit formation according to principles of operant conditioning. Participants frequently report 'aha moments' when observing glucose spikes following specific foods or reductions following exercise.

Second, CGM provides objective, personalized data that can enhance self-efficacy and motivation. Observing favorable glucose responses to healthy behaviors (glucose reduction following a walk, stable glucose after a low-glycemic meal) provides mastery experiences that build confidence in one's ability to influence metabolic health. This aligns with social cognitive theory, which emphasizes self-efficacy as a key determinant of sustained behavior change.

Third, CGM data enables pattern recognition and personalized interventions. Individuals can identify specific foods, meal compositions, or timing patterns that produce unfavorable glucose responses and modify their behaviors accordingly. Healthcare providers can use CGM data to provide tailored counseling, moving beyond generic dietary advice to specific, data-driven recommendations. This personalization may increase intervention relevance and effectiveness.

Fourth, CGM may enhance accountability and engagement with healthcare providers. The availability of objective glucose data facilitates more productive counseling sessions, enabling discussions focused on specific patterns and behaviors rather than recall-dependent dietary histories. Several studies reported increased frequency of contact between participants and healthcare providers in CGM groups.

The observation that real-time CGM may produce larger effects than intermittently scanned CGM (though not statistically significant in subgroup analysis) is mechanistically plausible. Real-time CGM provides immediate, continuous feedback including trend arrows and alerts, while isCGM requires users to actively scan the sensor. The additional friction in obtaining glucose data with isCGM may reduce feedback frequency and thus behavioral reinforcement.

Strengths and Limitations

This systematic review has several strengths. First, the protocol was prospectively registered, and we followed PRISMA 2020 guidelines and Cochrane methodology throughout. Second, we conducted comprehensive searches across six databases and supplementary sources with no date restrictions. Third, rigorous methodology included duplicate screening, extraction, and risk of bias assessment using validated tools (RoB-2 and ROBINS-I). Fourth, we assessed the certainty of evidence using GRADE and presented Summary of Findings tables for key outcomes. Fifth, pre-specified subgroup and sensitivity analyses explored robustness of findings.

Several limitations should be acknowledged. First, the number of included studies (14) and total sample size (1,847) was moderate, limiting precision of estimates and statistical power for subgroup analyses. Second, heterogeneity was moderate to substantial for some outcomes (I^2 38–72%), reflecting variation in CGM types, intervention components, comparator conditions, and follow-up duration. While we explored heterogeneity through subgroup analyses, residual heterogeneity limits certainty. Third, most studies were at some concern for risk of bias due to the impossibility of blinding participants and care providers to CGM allocation; this is inherent to the intervention but may introduce performance and detection bias. Fourth, language restriction to English may have excluded relevant studies. Fifth, median follow-up was 12 months, limiting conclusions about long-term T2DM prevention; the DPP demonstrated that lifestyle effects attenuate over time without sustained intervention. Sixth, there was variability in intervention intensity, CGM protocols, and behavioral components, making it difficult to identify optimal intervention strategies. Seventh, publication bias was suspected for HbA1c based on funnel plot asymmetry, suggesting possible overestimation of effect for this outcome.

Implications for Clinical Practice

Our findings suggest that CGM-guided behavioral interventions represent a promising strategy for T2DM prevention in adults with prediabetes and obesity. Clinicians may consider offering CGM as an adjunct to lifestyle modification programs for motivated individuals at high risk for T2DM progression. Real-time CGM may offer advantages over intermittently scanned CGM for behavioral feedback, though both types show benefits. Interventions of at least 12 months duration appear necessary to observe sustained effects on T2DM progression.

However, several practical considerations warrant attention. CGM devices have costs that may not be covered by insurance for prediabetes indications in many healthcare systems. Skin reactions, while usually mild, affect approximately 8% of users and may limit acceptability. Healthcare provider training in CGM interpretation and behavioral counseling may be necessary for optimal implementation. Patient selection is important; CGM may be most beneficial for individuals who are motivated and capable of translating glucose data into behavioral changes.

Implications for Research

Future research should address several gaps identified in this review. First, larger, adequately powered RCTs with longer follow-up (≥3 years) are needed to confirm the durability of effects on T2DM prevention and detect potential attenuation over time. Second, head-to-head comparisons of different CGM types (rtCGM versus isCGM) and intervention components would help identify optimal strategies. Third, studies should examine cost-effectiveness, as CGM adds significant expense that must be justified by improved outcomes; preliminary evidence suggests CGM may be cost-effective for diabetes prevention, but more data are needed. Fourth, research should identify patient characteristics associated with greater benefit from CGM to enable targeted implementation. Fifth, studies examining maintenance strategies after initial CGM-guided intervention would inform long-term implementation. Sixth, standardization of outcome measures and CGM metrics would improve comparability across studies.

Future Research Direction: 12-Week Pilot RCT

Building on the findings of this systematic review, which demonstrated that CGM-guided behavioral interventions probably reduce T2DM progression by 42% with moderate certainty evidence, a pilot 3-arm randomized controlled trial is proposed to generate preliminary efficacy data and inform future definitive trials. The study will enroll 165 adults with prediabetes and obesity, randomized equally (n=55 per arm) to: (1) real-time CGM plus structured lifestyle intervention, (2) intermittently scanned CGM plus structured lifestyle intervention, or (3) structured lifestyle intervention alone. Over a 12-week intervention period, the primary outcome will be change in HbA1c from baseline, with secondary outcomes including CGM-derived glycemic metrics (time in range, glycemic variability), body weight, fasting plasma glucose, dietary adherence, and adverse events. This pilot trial will provide critical data on effect sizes, feasibility, participant acceptability, and retention rates to inform sample size calculations for a subsequent fully-powered multicenter trial with longer follow-up examining T2DM progression as the primary endpoint.

Conclusions

This systematic review and meta-analysis provide moderate-certainty evidence that CGM-guided behavioral interventions probably reduce progression to T2DM by approximately 42% in adults with prediabetes and obesity. CGM-guided interventions were associated with clinically meaningful improvements in HbA1c, fasting glucose, body weight, BMI, waist circumference, and CGM-derived glycemic metrics. The interventions appear safe, with local skin reactions being the most common adverse effect.

CGM-guided behavioral interventions represent a promising technology-enhanced approach to diabetes prevention that may complement traditional lifestyle modification programs. The real-time feedback provided by CGM appears to enhance behavioral

engagement and facilitate learning of the relationships between behaviors and metabolic outcomes. Given the limitations of the available evidence, including moderate sample sizes and follow-up duration, further high-quality RCTs with longer follow-up are warranted to confirm these findings and identify optimal intervention strategies for different patient populations.

Declarations

Author Contributions

NV conceived the study, developed the protocol, conducted database searches, screened and selected studies, extracted data, assessed risk of bias, performed statistical analyses, interpreted results, and drafted the manuscript. CS contributed to protocol development, arbitrated disagreements in study selection, and critically revised the manuscript. SP screened and selected studies and extracted data. SH screened and selected studies and contributed to data extraction. RJ extracted data and contributed to data interpretation. CJ extracted data and contributed to risk of bias assessment. PW assessed risk of bias and critically revised the manuscript. All authors approved the final version and agreed to be accountable for all aspects of the work.

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Competing Interests

All authors declare no competing interests related to this work. No author has received funding, honoraria, or support from CGM device manufacturers.

Data Availability

All data supporting the findings of this systematic review are available in the published article, supplementary materials, and cited primary studies. The complete dataset, including data extraction forms and statistical analysis code, is available from the corresponding author upon reasonable request.

Ethics Approval

Ethical approval was not required for this systematic review of published studies. No patient data were collected beyond that reported in published studies.

Registration

PROSPERO CRD420251266604. Registered December 16, 2024.

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