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Exercise Dose, Timing and Supervision: Strategies for Gestational Diabetes Mellitus Prevention and Treatment: A Narrative Review

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Abstract

Aim. To synthesize evidence on the role of physical activity (PA) in preventing and managing gestational diabetes mellitus (GDM), covering physiology, observational data, trials, implementation barriers, and gaps.

Materials and methods. Narrative review of studies on preconception and pregnancy PA for GDM prevention and on PA interventions for treatment/control. Outcomes: incident GDM, fasting/postprandial glucose, insulin use, gestational weight gain, hypertensive disorders, delivery mode, neonatal outcomes (LGA, macrosomia, hypoglycemia), and longer-term

offspring data. Dose–response and subgroup analyses by timing, intensity, supervision, and maternal BMI.

Results. Preconception and early-pregnancy PA are linked to lower GDM risk Protective estimates span ~21–46% overall. Dose–response: ≥15–21 MET h/week preconception; in pregnancy, 10/20/50 MET h/week ≈6/11/15% lower risk. Early, supervised light–moderate programs yield the largest prevention effects; one RCT reported up to 90% risk reduction at 150–180 min/week. A single moderate bout lowers postprandial glucose for 1–2 h; repeated aerobic/resistance training with dietary counseling improves fasting/postprandial glucose and may reduce insulin needs. Intensified lifestyle care reduces gestational weight gain and LGA. Evidence for hypertensive disorders, cesarean delivery, and long-term outcomes is mixed. PA levels are low (MVPA ~11–14 min/day); barriers are clinical, symptomatic, cultural.

Conclusions. Promote PA before and early in pregnancy (≥15–21 MET h/week preconception; up to ~50 MET h/week in the first trimester when feasible). Early, supervised light–moderate programs and regular aerobic or resistance exercise plus dietary counseling are effective. More RCTs should refine optimal modality, dose, timing, supervision, and long-term outcomes.

Keywords: physical activity; gestational diabetes mellitus; pregnancy; prevention; exercise intervention; dose–response; maternal outcomes; neonatal outcomes

1. Introduction

Pregnancy is a period of profound physiological, biochemical, and psychosocial change, including hormonal shifts, increased blood volume and cardiac output, weight gain, and alterations in posture and gait; these adaptations influence maternal metabolic homeostasis and physical capacity. Because pregnancy is time-limited, the preconception and antenatal periods represent key windows of opportunity to adopt healthier behaviours—such as increased physical activity (PA), attainment of a healthy body mass index, and a plant-forward diet—that may favourably modify pregnancy outcomes and longer-term cardiometabolic risk for both mother and offspring (1).

Physical activity during pregnancy is endorsed by the World Health Organization: all pregnant and postpartum women without contraindications are recommended to undertake regular PA throughout pregnancy and the postpartum period, with a minimum target of 150 minutes per

week of moderate-intensity aerobic activity, complemented by diverse aerobic and muscle-strengthening exercises and, where appropriate, gentle stretching; women already engaging in vigorous activity may continue as tolerated (2). Importantly, the benefits of PA appear to accrue in a dose-responsive manner, yet even modest increases below guideline thresholds confer measurable health gains (3,4).

Gestational diabetes mellitus (GDM), defined as glucose intolerance first recognized in pregnancy, has risen in prevalence globally, driven by advanced maternal age, increasing preconception obesity, and changes in diagnostic criteria. GDM is a pivotal contributor to intergenerational transmission of cardiometabolic disease and identifies women at elevated lifetime risk of type 2 diabetes and cardiovascular disease, underscoring the need for preventive strategies that extend beyond pregnancy itself (5). Current prevalence estimates vary by region but are substantial—for example, ~11% across Europe with higher rates in some Eastern European populations(6), 17.5% in China, and an estimated global prevalence of 13.9%—and are associated with immediate maternal–fetal risks such as macrosomia and perinatal complications as well as long-term offspring sequelae (7,8). Demographic shifts including delayed childbearing, obesity and migration of higher-risk groups further amplify GDM incidence (9).

Preventive and therapeutic roles for PA in GDM have been explored: while interventions of diet, PA, combined diet and PA or medication have shown limited efficacy in preventing GDM among overweight/obese women overall, PA and combined interventions consistently reduce gestational weight gain (GWG), and combined diet and PA appears most effective for GWG restriction and shows a tendency toward GDM risk reduction (10). Given the low baseline levels of PA among pregnant women and multiple barriers to activity, clarifying the effectiveness, optimal type, dose and timing of PA for GDM prevention and management remains a priority.

2. Research materials and method

A narrative literature review was conducted to synthesize evidence on the role of physical activity (PA) in the prevention and management of gestational diabetes mellitus (GDM), covering mechanistic aspects, observational data, interventional studies, implementation barriers, and research gaps. PubMed and Scopus were searched for publications available from October 2011 to October 2026, and searches were supplemented by hand-searching bibliographies of key articles.

Searches used combinations of English-language terms, including: “gestational diabetes”, “GDM”, “physical activity”, “exercise”, “aerobic exercise”, “resistance training”, “combined training”, “exercise intervention”, “preconception”, “pregnancy”, “glycaemic control”, “gestational weight gain”, “pregnancy outcomes”, “neonatal outcomes”, and “maternal metabolism”. Limits for English language and publication date were applied; additional filters (e.g., “randomized controlled trial”, “systematic review”) were used when appropriate.

Included were original experimental studies (RCTs, controlled trials), observational studies (cohort, cross-sectional), combined interventions (diet + PA), systematic and narrative reviews, and meta-analyses addressing GDM prevention (preconception/early pregnancy PA) or GDM treatment/control (PA as intervention). Outcomes of interest included incident GDM, fasting and post-load/post-exercise glucose, insulin use, gestational weight gain, hypertensive disorders, mode of delivery, neonatal outcomes (LGA, macrosomia, hypoglycaemia), and long-term offspring effects. Excluded were non-English articles, conference abstracts, commentaries/opinions without empirical data, papers without full text, and studies of manifestly poor methodological quality.

Selection was performed in two stages: initial screening of titles and abstracts, followed by full-text assessment for eligibility. Two independent reviewers conducted the process; disagreements were resolved by discussion or with a third reviewer. From included studies we extracted: population characteristics (age, BMI, risk factors), timing of intervention (preconception, first/second/third trimester), PA characteristics (modality, intensity, frequency, duration, supervision), concomitant interventions (e.g., dietary counselling), metabolic and perinatal outcomes, adherence measures, reported adverse events, and monitoring methods.

Potential publication bias, heterogeneity of interventions, limitations of self-reported PA measures, variability in GDM definitions, and short follow-up in some studies were addressed in the Discussion.

3. Research result

Physical activity as a strategy to counteract placental pathology in gestational diabetes mellitus

The placenta functions as the primary endocrine regulator during pregnancy, producing hormones such as prolactin, estrogen, and progesterone that drive maternal metabolic adaptation (11,12). These placental hormones progressively increase maternal insulin resistance throughout gestation to prioritize fetal access to glucose and other nutrients (12,13). To

compensate, maternal pancreatic β-cell insulin secretion normally increases, preserving euglycemia. In gestational diabetes mellitus (GDM), however, the balance is disturbed: exaggerated insulin resistance combined with β-cell secretory dysfunction leads to hyperglycemia (12,14). Because the placenta can sense and adapt to changes in the intrauterine environment, placental dysfunction has been implicated in the pathogenesis and complications of pregnancy, including contributions to the development and progression of GDM (12,15).

Altered placental glucose transporter expression—marked by upregulation of GLUT1 with concomitant downregulation of GLUT3 and GLUT4—reduces cellular glucose uptake and fosters an extracellular hyperglycemic milieu that suppresses NRF2 (nuclear factor erythroid 2-related factor) expression and increases reactive oxygen species (ROS) production (12). Elevated ROS disrupt calcium signaling and trigger proinflammatory cascades, notably activation of NF-κB (nuclear factor-kappa B) and the JAK/STAT3 pathway, thereby amplifying oxidative stress and inflammatory responses within the placenta (12). These interconnected pathways link metabolic dysregulation to impaired antioxidant defence and contribute to placental dysfunction in GDM (12).

In physiological pregnancy, mitochondrial dynamics (fusion and fission), biogenesis, and mitophagy cooperate to preserve mitochondrial homeostasis, enabling ATP generation via the tricarboxylic acid cycle and oxidative phosphorylation, with concomitant production of reactive oxygen species (ROS) as by-products (12). In GDM, hyperglycemia precipitates mitochondrial dysfunction, evidenced by a marked decrease in electron transport chain (ETC) complex expression, ATP depletion, excessive ROS production and increased lipid peroxidation (12). Alterations in dynamics—namely elevated OPA1 (optic atrophy 1) and reduced DRP1 (dynamin-related protein 1)—indicate a shift toward enhanced fusion, while downregulation of DsbA-L (disulfide-bond A oxidoreductase-like protein), PGC-1α (peroxisome proliferator-activated receptor-γ coactivator-1α) and TFAM (mitochondrial transcription factor A) promotes release of mitochondrial DNA (mtDNA) into the cytosol (12). Cytosolic mtDNA activates the cGAS–STING–IRF3 (cGAS: cyclic GMP-AMP synthase; STING: stimulator of interferon genes; IRF3: interferon regulatory factor 3) signaling axis, thereby amplifying placental inflammation and contributing to insulin resistance in GDM (12).

Oxidative stress and ferroptosis are mechanistically linked in the GDM placenta: oxidative stress drives lipid peroxidation, the hallmark effector of ferroptotic cell death, a process modulated by mitochondrial dysfunction and disrupted iron handling (12). Experimental in vivo models show that a high-fat, high-sugar maternal diet alters placental iron transport capacity, dysregulates ferroptosis-related gene expression, and increases

iron-dependent oxidative damage in trophoblasts, implicating ferroptosis in placental injury and dysfunction associated with GDM (12,16).

Placental inflammation is strongly associated with the development of gestational diabetes mellitus, indicating that inflammatory processes constitute a substantive component of GDM pathophysiology (17). Transcriptomic profiling of placentas from GDM patients revealed dysregulated expression of genes within the IL-1β and Toll-like receptor (TLR) pathways, including significant upregulation of IL1R, IL1RAP, TLR4, RELA and CD14; these placental changes were accompanied by increased circulating IL-1 receptor antagonist (IL-1RA) in GDM women (18).

Engagement of IL-1β with IL1R1 activates NF-κB signaling, promoting production and release of downstream proinflammatory mediators such as TNF-α, IL-6 and additional IL-1β, thereby establishing an autocrine, self-amplifying cytokine network (18) IL-1β binding induces recruitment of IL1RAP and signaling via MYD88 to IRAK-2/4 and TRAF6, which in turn mobilizes NF-κB, p38 MAPK, JAK and MAPK/ERK cascades to drive broad transcriptional inflammatory responses across multiple placental cell types (18–20).

There is substantial overlap and reciprocal activation between IL-1β and TLR pathways in the placenta; this reciprocal signaling underlies sterile inflammatory processes implicated in GDM-related placental dysfunction (18).

Exercise interventions induce concerted transcriptomic changes in the GDM placenta that are associated with reduced inflammation and oxidative stress. Structured physical activity modulates expression of key regulatory genes—CHRDL1, RORB, CCL21 and MTCO1P40—while influencing fetal growth regulators such as IGFBP1, thereby contributing to normalization of fetal birth weight trajectories (7). Exercise also upregulates antioxidant-related transcripts, notably GPX3 and MTCO1P40, which restore placental redox balance and diminish ROS-mediated damage, conferring protective effects for both mother and fetus (7).

Mechanistically, sustained moderate-intensity exercise enhances mitochondrial efficiency and augments expression of endogenous antioxidant enzymes, thereby interrupting the positive feedback loop between inflammation and oxidative stress that characterizes GDM placental pathology (7,21). These combined anti-inflammatory and antioxidative transcriptomic adaptations support exercise as a therapeutic modality to mitigate placental dysfunction in GDM (7,21).

Routine mild-to-moderate maternal exercise during pregnancy does not increase placental oxidative or endoplasmic reticulum (ER) stress compared with inactivity, supporting its safety in healthy gestations (22). Concurrently, habitual exercise robustly upregulates

placental angiogenic signaling, exemplified by marked increases in ANG1 expression, indicating enhanced placental angiogenesis that may benefit maternal–fetal exchange and placental function (22). These findings identify ANG1 as a potential mediator of exercise-induced placental adaptation and justify larger studies to evaluate its therapeutic relevance in pathological pregnancies and across varying maternal activity levels (22).

Collectively, these findings position the placenta as a dynamic endocrine and immunometabolic hub whose dysregulation underlies the pathophysiology of gestational diabetes mellitus (GDM): hormonal drivers of maternal insulin resistance intersect with altered glucose transport (↑GLUT1; ↓GLUT3/4), NRF2 suppression, and activation of NF-κB/JAK–STAT3, while hyperglycemia provokes mitochondrial dysfunction (diminished ETC activity, ATP depletion, excess ROS), perturbs dynamics and biogenesis (↑OPA1, ↓DRP1; ↓DsbA-L, PGC-1α, TFAM), and releases mtDNA to engage cGAS–STING–IRF3, thereby amplifying inflammation; concomitantly, iron-dependent lipid peroxidation accelerates trophoblastic ferroptosis, and upregulated IL-1β/TLR signaling (e.g., IL1R, IL1RAP, TLR4, RELA, CD14) sustains a self-reinforcing cytokine milieu. This integrated mechanistic landscape not only clarifies the biological basis of placental dysfunction in GDM but also delineates modifiable targets. In particular, maternal physical activity emerges as a plausible, low-risk intervention capable of interrupting oxidative–inflammatory feedback loops, supported by exercise-induced placental transcriptomic shifts (e.g., CHRDL1, RORB, CCL21, IGFBP1), upregulation of antioxidant defenses (GPX3, MTCO1P40), improved mitochondrial efficiency, and enhanced angiogenic signaling (↑ANG1). Framed by this rationale, the subsequent section addresses evidence-based exercise prescriptions in pregnancy—type, timing, intensity, and dose—alongside safety considerations and biomarker-guided evaluation of therapeutic efficacy.

Physical activity and risk of GDM

Accumulating evidence suggests that preconception and early pregnancy physical activity (PA) are associated with substantially lower odds of developing GDM. Meta-analytic data indicate that any pre-pregnancy PA and any early-pregnancy PA are associated with approximately 30% and 21% reduced odds of GDM, respectively, while >90 minutes/week of leisure-time PA before pregnancy confers a 46% reduction in (1). Overall protective estimates for PA versus none range from 21–46% in pooled analyses, with pre-pregnancy PA reductions reported between 22% and 86% depending on PA type and duration, and early-pregnancy reductions of 11–52% (1).

Dose–response analyses reveal both linear and non-linear effects: beneficial associations in the year before pregnancy are observed at ≥15–21 MET-hours/week, and PA volumes of 10, 20 and 50 MET-hours/week during pregnancy correspond to ~6%, 11% and 15% lower GDM risk, respectively (23). First-trimester PA exhibits a non-linear (L-shaped) association with incident GDM—risk declines with increasing PA until a plateau—whereas second-trimester PA shows a linear, but attenuated, protective effect; increases in first-trimester PA appear particularly effective for prevention (23).

Randomized and observational data indicate that supervised, early-initiated, and light-to-moderate intensity programmes yield the greatest reductions in GDM incidence. Exercise interventions that begin in the first trimester, are supervised, or maintain light–moderate intensity are associated with larger effects; supervision improves adherence and likely augments effectiveness (24). One RCT reported that structured PA of 150–180 min/week during pregnancy reduced GDM risk by up to 90% compared with standard care, although results vary across studies and populations (1,25).

Heterogeneity in effect estimates is influenced by maternal BMI, timing of intervention, and baseline energy expenditure: interventions are generally less effective in women with overweight/obesity or when initiated after mid-pregnancy, and lowest daily energy expenditure (≤2200 kcal) associates with highest GDM risk (1,24). Taken together, these findings support promotion of PA in the preconception period and early pregnancy—aiming for ≥15–21 MET-hours/week and, when feasible, up to ~50 MET-hours/week in the first trimester—to lower GDM risk, recognizing that individual benefits depend on intensity, duration, supervision and maternal characteristics (1,23,24)

Role of Physical Activity in the Treatment and Control of Gestational Diabetes Mellitus

Physical activity (PA) is recommended in diabetes management because it enhances peripheral insulin sensitivity, augments glucose uptake in skeletal muscle, and improves overall glycemic control (26). In the context of pregnancy, these physiological effects underpin the use of PA both prophylactically and therapeutically for gestational diabetes mellitus (GDM).

Controlled experimental studies in women at risk for GDM demonstrate that single bouts of moderate-intensity exercise (e.g., 20 min cycling) performed after a glucose load significantly reduce immediate postprandial blood glucose excursions and circulating insulin concentrations for the subsequent 1–2 hours (27,28). Comparable acute, dose-dependent reductions in postprandial glycaemia have been observed after walking and mild–moderate cycling in women with established GDM; the magnitude of glucose lowering scales with

exercise intensity and duration, but glycemic values typically return to baseline within a few hours after cessation of activity (27–29). Continuous glucose monitoring in some studies indicates that these acute improvements are transient and that a single exercise session does not produce durable glycemic changes beyond ~48 hours without repetition (27,28).

Sustained, repeated PA regimens are required to translate acute benefits into longer-term glycemic improvements. Several trials combining structured moderate-intensity aerobic or resistance exercise with dietary counselling reported greater reductions in fasting and 2-hour postprandial glucose, lower daily insulin requirements and reduced weekly mean glucose compared with dietary counselling alone; in some studies exercise plus diet achieved glycemic profiles comparable to pharmacotherapy plus diet (30). Resistance training in particular has been associated with decreased insulin utilization and improved measures of glycemic control, although results vary by protocol, adherence and baseline characteristics (27,30).

Heterogeneity in outcomes arises from differences in exercise modality (aerobic vs. resistance), intensity, frequency and supervision, timing relative to meals, baseline maternal BMI and timing of intervention initiation. Some trials report no change in daily glucose or HbA1c despite exercise interventions, which may reflect insufficient intensity/duration, late initiation (after mid-pregnancy), small sample size, or poor adherence (27,30). Systematic reviews confirm overall beneficial effects of PA on fasting, postprandial glucose and HbA1c in GDM, but they do not definitively identify a single optimal exercise prescription; only a minority of studies report insulin dose as an outcome, and findings on insulin requirement remain partially inconsistent (31,32).

Maternal outcomes

Gestational diabetes mellitus (GDM) confers a substantially increased risk of future metabolic disease, with long-term cohort data indicating up to a 70% risk of progression to type 2 diabetes mellitus (T2DM) over 28 years and estimates that in >50% of cases GDM precedes T2DM, hypertension, and cardiovascular disease within 5–15 years postpartum (1,24). GDM is also associated with adverse obstetric and perinatal events, notably hypertensive disorders of pregnancy (gestational hypertension, pre-eclampsia, eclampsia), which affect approximately 10% of pregnancies and contribute to elevated lifetime cardiovascular risk (24).

Interventional studies assessing intensified antenatal care—including dietary counselling, exercise, education, and self-monitoring—have demonstrated improvements in glycaemic control, reduced gestational weight gain, and lower rates of postpartum depression compared with standard care (33). However, pooled randomized evidence provides no clear

reduction in risk for hypertensive disorders of pregnancy or caesarean section, and shows uncertain effects on development of T2DM up to 10 years of follow-up; benefits were observed for attainment of postpartum weight goals and reduced postnatal depression (33). These findings suggest that while lifestyle interventions during pregnancy yield meaningful short-term maternal benefits, their impact on longer-term cardiometabolic outcomes remains inconclusive (33).

Neonatal outcomes

Gestational diabetes mellitus (GDM) is associated with increased neonatal morbidity, notably fetal overgrowth and macrosomia, hypoglycemia, and epigenetic alterations that may predispose offspring to obesity and type 2 diabetes later in life (1,24). Fetal overgrowth or macrosomia may affect up to 45% of pregnancies complicated by GDM, with these infants demonstrating higher long-term risks of overweight, obesity and T2DM (1,24).

Randomized trial data pooled in a Cochrane review indicate that intensified antenatal care—including additional dietary counselling, exercise, education and self-monitoring—reduces the incidence of large-for-gestational-age (LGA) births (RR 0.60, 95% CI 0.50–0.71; six trials, 2994 infants; moderate-quality evidence) and lowers birthweight and macrosomia rates (33). One trial reported a reduction in neonatal fat mass (MD −37.30 g, 95% CI −63.97 to −10.63; 958 infants; low-quality evidence) (33). There was no clear evidence of benefit for neonatal hypoglycaemia (average RR 0.99, 95% CI 0.65–1.52; six trials, ~3000 infants; moderate-quality evidence), perinatal death (RR 0.09, 95% CI 0.01–1.70; two trials, 1988 infants; low-quality evidence) or a composite of serious infant outcomes (average RR 0.57, 95% CI 0.21–1.55; two trials; very low-quality evidence) (33). In childhood, lifestyle interventions during pregnancy did not show clear effects on prevalence of BMI ≥85th percentile (RR 0.91, 95% CI 0.75–1.11; three trials, 767 children; moderate-quality evidence) (33). Long-term outcomes such as adult diabetes, adiposity and neurosensory disability were not prespecified or reported in the included trials (33).

Overall, intensified antenatal lifestyle interventions reduce fetal overgrowth and some measures of neonatal adiposity, but evidence for effects on neonatal morbidity and later childhood or adult outcomes remains limited or inconclusive (33).

Barriers to physical activity during pregnancy

Despite established benefits, only 23–29% of pregnant women meet physical activity recommendations, and incidental activity typically declines as pregnancy progresses, with

motion-sensor data from the US indicating up to 60% of daytime is spent sedentary (1). Objective assessments worldwide demonstrate generally inadequate activity levels: a Brazilian accelerometer study reported a mean of 14 minutes of moderate-to-vigorous physical activity (MVPA) per day among 2,317 pregnant women, and a US cohort recorded an average of 11.5 minutes/day during the first trimester (23). Cultural attitudes also appear influential; only 12.6% of women in a Singapore survey met the ≥150 minutes/week guideline, and multicentre data from China reported approximately 68.5% failing to achieve sufficient PA (23).

Clinical and pregnancy-related conditions further constrain activity. Pre-existing musculoskeletal or cardiac disease may limit capacity for exertion, and obstetric indications such as short cervix or threatened preterm labour frequently lead to immobilisation or prescribed bed rest despite limited supporting evidence(27). Interventions for threatened preterm delivery (e.g., antenatal corticosteroids) may transiently exacerbate insulin resistance and thereby affect activity tolerance (27). Common pregnancy complaints, including pelvic girdle pain and ligamentous laxity, and less common complications such as symptomatic varicose veins or deep venous thrombosis—particularly in the third trimester when insulin resistance peaks—often result in reduced movement and cautious behaviour (27). Together, behavioural, cultural, and clinical barriers contribute to persistently inadequate PA during pregnancy.

Implementing and monitoring lifestyle changes during pregnancy: a practical approach.

Pregnancy represents an optimal period to initiate and consolidate lifestyle modifications owing to heightened maternal motivation driven by concern for the fetus and the increased frequency of antenatal care contacts. Pregnant women are more likely to adopt changes such as weight management, increased physical activity and improved dietary habits, particularly when these recommendations are made by their primary clinician(34). Regular monitoring of progress and ongoing motivational support are essential to sustain health-promoting behaviours. The use of standardised tools—such as the Five A's model (Ask, Advise, Assess, Assist, Arrange), originally developed for smoking-cessation interventions but also effective for dietary and physical activity counselling—may facilitate this process(35–37).

Before prescribing an exercise programme, a clinical assessment should be performed to exclude medical contraindications to physical activity. Exercise intensity should then be individualised, especially when the goal is moderate-intensity activity. It should be noted that heart rate (HR) responses in pregnant women may be blunted or remain within ranges conventionally considered normal; therefore, HR alone may not reliably indicate exercise

intensity in this population(37,38). A practical and simple method to assess intensity is the “talk test”: if the woman can maintain a conversation during exercise, the intensity is likely to be appropriate(39).

Women should maintain adequate hydration, avoid prolonged supine positioning and discontinue exercise if any of the following symptoms occur: vaginal bleeding, abdominal pain, regular painful contractions, leakage of amniotic fluid, dyspnoea at rest or before exertion, dizziness, headache, chest pain, muscle weakness affecting balance, or calf pain or swelling(37). High-intensity or prolonged exercise may predispose to hypoglycaemia; accordingly, appropriate caloric intake before activity and limits on exercise intensity and duration should be observed to minimize this risk(40).

Recommended and contraindicated activities. Forms of exercise that have been well studied and are considered safe and beneficial in pregnancy include walking, stationary cycling, aerobic exercise, dancing, resistance training (e.g., using weights or resistance bands), flexibility exercises and hydrotherapy/water-based aerobics(37). Although activities associated with a high risk of abdominal trauma or loss of balance, as well as scuba diving, are contraindicated in pregnancy(37).

4. Conclusions

The evidence supports active promotion of physical activity (PA) in the preconception period and during early pregnancy as an effective strategy to reduce the risk of gestational diabetes mellitus (GDM). Public health and clinical efforts should encourage women planning pregnancy to achieve at least 15–21 MET-hours/week of PA, and when clinically appropriate and acceptable to the woman, facilitate escalation toward ~50 MET-hours/week in the first trimester to maximize preventive benefit. Implementation should prioritise early initiation, supervision, and light–to-moderate intensity regimens to improve adherence and effectiveness; structured programmes combining regular aerobic and/or resistance exercise with dietary counselling are particularly promising for both prevention and management of established GDM. Interventions must be individualised according to maternal BMI, comorbidities, pregnancy complications, and cultural/contextual factors, with clear guidance on safe activity types and progression. Clinicians should proactively address common barriers (symptoms, clinical restrictions, socioeconomic and cultural constraints) and use supervision, remote support or group formats to sustain engagement. Finally, well-powered RCTs with diverse populations and long-term follow-up are required to establish the optimal modality, dose,

timing and supervision models, and to determine durable maternal and offspring cardiometabolic benefits and potential harms.

Disclosure

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The authors deny any conflict of interest.

Declaration of the use of generative AI and AI-assisted technologies in the writing process

In preparing this work, the authors used ChatGPT for the purpose of grammar checking and improving the readability of the text. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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