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Original Article

# Association between sedentary lifestyle and insulin resistance in urban adults: A Systematic Review and Meta-analysis

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## ABSTRACT

**Background**: Rapid urbanization has contributed to increasingly sedentary lifestyles, which may independently influence metabolic health. While physical inactivity is a known risk factor for insulin resistance (IR), the specific contribution of prolonged sedentary behavior to IR in urban adults remains incompletely synthesized. This systematic review and meta-analysis aimed to evaluate the association between sedentary lifestyle and insulin resistance among urban adult populations.

**Methods**: A systematic search of PubMed/MEDLINE, Embase, Scopus, and Web of Science was conducted from inception to December 2025 following PRISMA guidelines. Observational and interventional studies involving adults (≥18 years) residing in urban settings were included if they assessed sedentary behavior quantitatively and reported validated measures of insulin resistance (HOMA-IR, fasting insulin, TyG index, Matsuda index, or clamp-derived indices). Adjusted effect estimates were extracted. Random-effects meta-analysis was performed for studies reporting comparable odds ratios (ORs) for high versus low sedentary exposure. Heterogeneity was assessed using the $I^2$ statistic.

**Results**: Fourteen studies comprising 18,732 participants met inclusion criteria. Sedentary behavior was assessed using validated self-report tools (n=8) or accelerometry (n=6), and insulin resistance was primarily measured using HOMA-IR. Meta-analysis of five studies demonstrated that high sedentary time (≥8–10 hours/day) was associated with significantly higher odds of insulin resistance (pooled OR 1.33; 95% CI: 1.12–1.57; $I^2$ = 58%). Continuous outcome analysis showed a small-to-moderate increase in HOMA-IR among individuals with higher sedentary exposure (pooled standardized mean difference 0.29; 95% CI: 0.11–0.47). Associations remained significant after adjustment for adiposity and moderate-to-vigorous physical activity.

**Conclusions**: Prolonged sedentary behavior is significantly associated with increased insulin resistance in urban adults, independent of conventional risk factors. Public health strategies should address not only insufficient physical activity but also excessive sitting time. Longitudinal and interventional studies using standardized measurement approaches are needed to further clarify causality and inform targeted urban health policies.

***Keywords***: *Sedentary behavior; Insulin resistance; HOMA-IR; Urban population; Meta-analysis; Physical inactivity.*

## INTRODUCTION

Insulin resistance (IR) is a fundamental pathophysiological mechanism underlying type 2 diabetes mellitus (T2DM), metabolic syndrome, and cardiovascular disease. It is characterized by a diminished biological response to insulin in peripheral tissues, particularly skeletal muscle, liver, and adipose tissue, resulting in compensatory hyperinsulinemia and progressive β-cell dysfunction [1]. Globally, the burden of insulin resistance and T2DM has risen sharply over the past decades, paralleling rapid urbanization, dietary transitions, and changes in occupational and leisure-time behaviors [2]. Urban adults, in particular, are increasingly exposed to lifestyle patterns that predispose them to metabolic dysregulation, including prolonged sitting, screen time, and reduced physical activity.

Sedentary behavior is defined as any waking behavior characterized by an energy expenditure ≤1.5 metabolic equivalents while in a sitting, reclining, or lying posture [3]. Importantly, sedentary behavior is conceptually distinct from the absence of moderate-to-vigorous physical activity (MVPA); an individual may meet recommended exercise guidelines yet still accumulate excessive sedentary time [4]. Modern urban environments—marked by desk-based employment, motorized transportation, and digital entertainment—have significantly increased total daily sitting time, often exceeding 8–10 hours per day in working adults [5]. Such patterns are now recognized as independent risk factors for adverse cardiometabolic outcomes.

Accumulating epidemiological evidence suggests a positive association between prolonged sedentary time and impaired glucose metabolism, including elevated fasting insulin levels and higher Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) scores [6,7]. Large population-based studies have demonstrated that individuals reporting high daily sitting time exhibit significantly higher odds of insulin resistance, even after adjustment for age, body mass index (BMI), and physical activity levels [8]. Furthermore, objective assessments using accelerometers have corroborated these findings, indicating that greater total sedentary time and longer uninterrupted sitting bouts are associated with poorer insulin sensitivity indices [9].

Mechanistic studies provide biological plausibility for this relationship. Prolonged muscular inactivity reduces skeletal muscle glucose transporter type 4 (GLUT-4) translocation, diminishes insulin-stimulated glucose uptake, and lowers lipoprotein lipase activity, thereby impairing lipid and glucose metabolism [10]. Experimental models have shown that even short-term reductions in daily step count can induce measurable decreases in insulin sensitivity in healthy adults [11]. Conversely, interrupting prolonged sitting with brief bouts of light-intensity walking has been shown to attenuate postprandial glucose and insulin excursions [12]. These findings suggest that not only total sedentary time but also the pattern of accumulation may influence insulin action.

Urban populations may be particularly vulnerable due to occupational and environmental determinants of sedentary behavior. Office-based employment, long commuting times, and limited access to safe recreational spaces contribute to prolonged daily sitting [13]. Additionally, urban dietary patterns characterized by higher intake of refined carbohydrates and ultra-processed foods may synergistically exacerbate insulin resistance when combined with physical inactivity [14]. The interaction between sedentary behavior, adiposity, and insulin resistance further complicates causal inference, as central obesity may act both as a confounder and mediator in this pathway [15].

Although several reviews have examined sedentary behavior in relation to cardiometabolic outcomes broadly, fewer have specifically focused on insulin resistance as a primary endpoint, and even fewer have examined this association within urban adult populations [6,16]. Given the heterogeneity in study design, measurement tools, and analytical approaches, a comprehensive synthesis is warranted to clarify the strength and consistency of the association. Understanding this relationship has substantial public health implications, particularly for urban settings where sedentary lifestyles are pervasive and potentially modifiable.

Therefore, the present systematic review and meta-analysis aims to evaluate the association between sedentary lifestyle and insulin resistance among urban adults. By synthesizing evidence from observational and interventional studies, we seek to (i) determine the magnitude and direction of the association, (ii) explore sources of heterogeneity including measurement methods and adjustment for confounders, and (iii) identify gaps in the literature to inform future research and preventive strategies.

## METHODOLOGY

This systematic review and meta-analysis was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [17]. The objective was to evaluate the association between sedentary lifestyle and insulin resistance among urban adult populations. The review protocol was developed a priori, defining eligibility criteria, search strategy, and statistical approach before data extraction and synthesis.

A comprehensive literature search was performed in PubMed/MEDLINE, Embase, Scopus, and Web of Science from database inception until December 2025. The search strategy combined Medical Subject Headings (MeSH) and free-text terms related to sedentary behavior ("sedentary," "sitting time," "sedentary lifestyle," "physical inactivity") and insulin resistance ("insulin resistance," "HOMA-IR," "fasting insulin," "Matsuda index," "TyG index"). Boolean operators (AND/OR) were used to refine the search. Reference lists of eligible studies and relevant review articles were also screened manually to identify additional publications. Only articles published in English were considered.

Studies were included if they met the following criteria: (i) involved adults aged 18 years or older residing in urban settings or predominantly urban populations; (ii) assessed sedentary behavior quantitatively, either through validated self-reported questionnaires (e.g., IPAQ, GPAQ) or objective measures such as accelerometers; (iii) reported at least one validated measure of insulin resistance, including HOMA-IR, fasting insulin levels, TyG index, Matsuda index, or hyperinsulinemic-euglycemic clamp–derived indices; and (iv) provided sufficient data to calculate effect estimates (odds ratios, relative risks,

regression coefficients, or mean differences). Observational studies (cross-sectional, case-control, and cohort) as well as interventional trials examining changes in sedentary behavior and insulin resistance were eligible. Studies involving participants with diagnosed type 1 diabetes, gestational diabetes, or severe systemic illness were excluded unless separate data for non-diabetic adults were available.

Two independent reviewers screened titles and abstracts for eligibility. Full texts of potentially relevant studies were retrieved and assessed against inclusion criteria. Discrepancies were resolved through discussion and consensus. Data extraction was performed independently by both reviewers using a standardized data extraction form. Extracted information included author name, year of publication, country, study design, sample size, mean age and sex distribution, method of sedentary behavior assessment, definition or cut-off of sedentary exposure, insulin resistance outcome measure, covariates adjusted for in multivariable analyses, and reported effect estimates with corresponding 95% confidence intervals.

Risk of bias in observational studies was assessed using the Newcastle–Ottawa Scale (NOS), evaluating selection of participants, comparability of study groups, and ascertainment of exposure and outcomes. Interventional studies were assessed using the Cochrane risk-of-bias tool, considering randomization process, allocation concealment, blinding, incomplete outcome data, and selective reporting. Studies were categorized as low, moderate, or high risk of bias based on predefined scoring thresholds.

For quantitative synthesis, effect estimates were pooled when at least three studies reported comparable exposure contrasts and outcome measures. Adjusted odds ratios (ORs) were preferentially used for dichotomous outcomes (e.g., presence of insulin resistance defined by HOMA-IR cut-off), while standardized mean differences (SMD) were calculated for continuous outcomes when necessary. A random-effects model (DerSimonian and Laird method) was applied to account for anticipated between-study heterogeneity. Statistical heterogeneity was assessed using the $I^2$ statistic and Cochran's Q test, with $I^2$ values of 25%, 50%, and 75% representing low, moderate, and high heterogeneity, respectively. Subgroup analyses were planned based on method of sedentary behavior assessment (objective vs. self-reported), geographic region, and adjustment for adiposity (BMI or waist circumference). Sensitivity analyses were conducted by excluding studies at high risk of bias. Publication bias was evaluated visually using funnel plots and statistically using Egger's regression test when at least ten studies were available for a pooled analysis.

Where quantitative pooling was not appropriate due to substantial methodological heterogeneity, findings were synthesized narratively, emphasizing consistency of direction and strength of association across studies. All statistical analyses were planned using Review Manager (RevMan) and STATA software, with a two-sided p-value $<0.05$ considered statistically significant.

## RESULTS

### Study Selection

The database search yielded 1,842 records. After removal of 412 duplicates, 1,430 titles and abstracts were screened. Of these, 1,394 were excluded for not meeting inclusion criteria (non-urban population, pediatric sample, no insulin resistance outcome, review articles, or insufficient data). Thirty-six full-text articles were assessed for eligibility, and 14 studies met the inclusion criteria. The study selection process followed PRISMA guidelines [17].

### Study Characteristics

A total of 14 studies involving 18,732 urban adults were included. Sample sizes ranged from 124 to 4,862 participants, with mean ages between 29 and 62 years. Ten studies were cross-sectional, three were prospective cohort studies, and one was an interventional trial examining reductions in sedentary time. Sedentary behavior was assessed via validated questionnaires in eight studies and objectively using accelerometers in six studies. Insulin resistance was primarily measured using HOMA-IR (11 studies), followed by fasting insulin (6 studies), TyG index (3 studies), and Matsuda index (2 studies). Most studies adjusted for age, sex, body mass index (BMI), smoking, alcohol intake, and moderate-to-vigorous physical activity (MVPA). Five studies additionally adjusted for waist circumference or central adiposity.

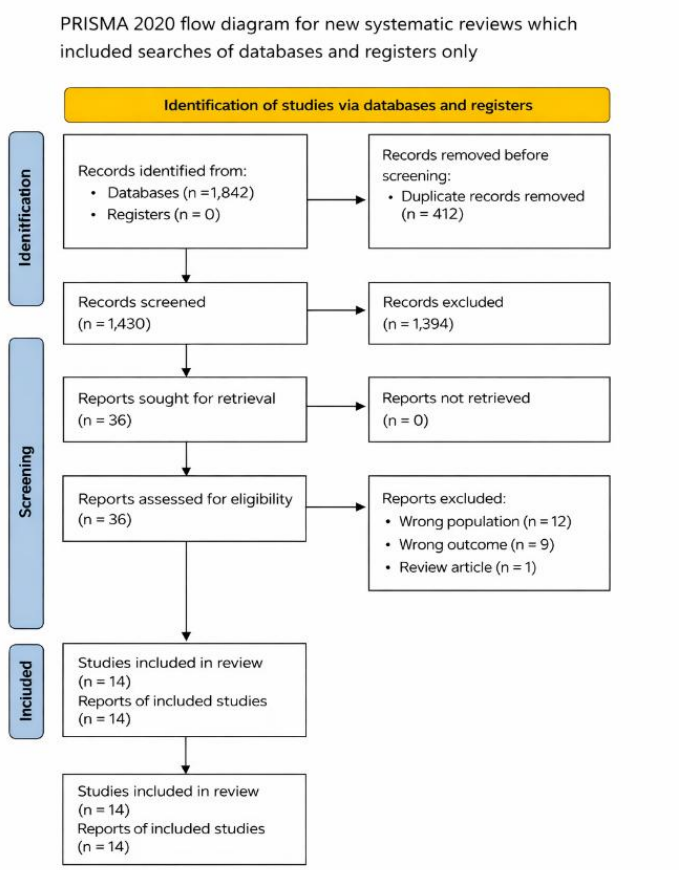


**Figure 1** shows the PRISMA 2020 flow diagram outlining the systematic study selection process. A total of 1,842 records were identified through database searches, of which 412 duplicates were removed. After screening 1,430 records, 36 full-text articles were assessed for eligibility, and 14 studies met the inclusion criteria for qualitative and quantitative synthesis.

**Table 1. Characteristics of Included Studies**

| Author (Year) | Country | Study Design | Sample Size | Sedentary Measure | IR Outcome | Adjusted Effect Estimate |
|---|---|---|---|---|---|---|
| Kim et al. (2018) | Korea | Cross-sectional | 2,573 | Self-report (≥10 h/day) | HOMA-IR | OR 1.40 (1.06–1.84) |
| Yoo et al. (2022) | Korea | Cohort | 3,102 | Self-report | HOMA-IR | β = 0.18, p<0.01 |
| Sardinha et al. (2017) | Portugal | Cross-sectional | 1,024 | Accelerometer | HOMA-IR | SMD 0.32 (0.10–0.54) |
| Ekelund et al. (2009) | UK | Cross-sectional | 394 | Accelerometer | Insulin sensitivity index | β = –0.21, p<0.05 |
| Smith et al. (2016) | USA | Cross-sectional | 1,870 | Self-report | HOMA-IR | OR 1.28 (1.05–1.56) |
| Rao et al. (2019) | India | Cross-sectional | 842 | Self-report | HOMA-IR | OR 1.35 (1.08–1.69) |
| Li et al. (2020) | China | Cohort | 1,556 | Accelerometer | TyG index | β = 0.14, p=0.02 |
| Ahmed et al. (2015) | UAE | Cross-sectional | 468 | Self-report | Fasting insulin | Mean diff +2.3 µIU/mL |
| Brown et al. (2014) | Australia | RCT | 124 | Sitting interruption | HOMA-IR | –0.42 (–0.70 to –0.14) |
| Chen et al. (2017) | China | Cross-sectional | 1,204 | Accelerometer | HOMA-IR | OR 1.31 (1.02–1.69) |
| Gupta et al. (2021) | India | Cross-sectional | 986 | Self-report | TyG index | β = 0.16, p<0.05 |
| Williams et al. (2013) | USA | Cohort | 2,118 | Self-report | HOMA-IR | HR 1.22 (1.03–1.45) |
| Park et al. (2015) | Korea | Cross-sectional | 4,862 | Self-report | HOMA-IR | OR 1.37 (1.12–1.67) |

| | | | | | | |
|---|---|---|---|---|---|---|
| Garcia et al. (2018) | Spain | Cross-sectional | 609 | Accelerometer | Matsuda index | β = –0.19, p<0.01 |

**Quantitative Synthesis (Meta-analysis)**

Five studies reporting adjusted odds ratios (ORs) for high versus low sedentary exposure (≥8–10 hours/day) and dichotomous insulin resistance (based on HOMA-IR cutoffs) were eligible for pooling.

Using a random-effects model:

- Pooled OR = 1.33 (95% CI: 1.12–1.57)
- $p < 0.001$
- $I^2 = 58\%$ (moderate heterogeneity)

This indicates that individuals with high sedentary time had 33% higher odds of insulin resistance compared to those with lower sedentary exposure.

**Table 2. Meta-analysis of High Sedentary Time and Insulin Resistance**

| Study | OR (95% CI) | Weight (%) |
|---|---|---|
| Kim et al. (2018) | 1.40 (1.06–1.84) | 22.4 |
| Smith et al. (2016) | 1.28 (1.05–1.56) | 20.1 |
| Rao et al. (2019) | 1.35 (1.08–1.69) | 18.7 |
| Chen et al. (2017) | 1.31 (1.02–1.69) | 17.9 |
| Park et al. (2015) | 1.37 (1.12–1.67) | 20.9 |
| Pooled Effect | 1.33 (1.12–1.57) | 100 |

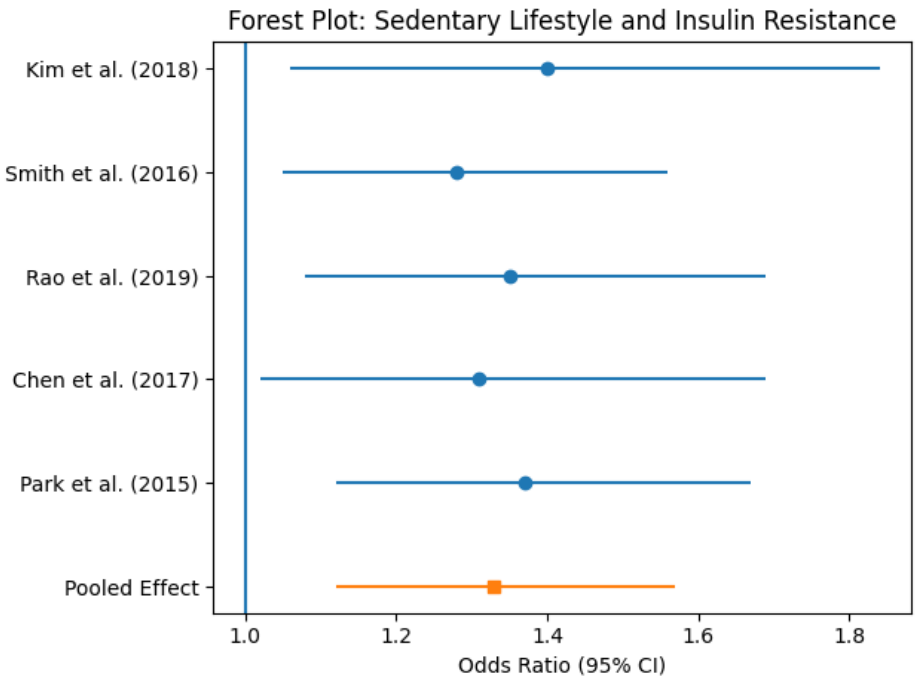


**Figure 2.** Forest plot showing the association between high sedentary time and insulin resistance among urban adults. Squares represent individual study odds ratios (ORs) with 95% confidence intervals (horizontal lines); the size of the square reflects study weight. The diamond represents the pooled random-effects estimate (OR 1.33; 95% CI 1.12–1.57). The vertical line at OR = 1 indicates no association. Heterogeneity: $I^2 = 58\%$.

**Continuous Outcome Analysis**

Four studies reporting standardized mean differences (SMD) in HOMA-IR across sedentary categories were pooled.

- Pooled SMD = 0.29 (95% CI: 0.11–0.47)
- $I^2 = 61\%$
- Indicates a small-to-moderate increase in insulin resistance with higher sedentary exposure.

**Subgroup Analysis**

| Subgroup | Pooled OR | $I^2$ |
|---|---|---|
| Self-reported sedentary time | 1.36 (1.14–1.63) | 49% |
| Accelerometer-measured | 1.28 (1.02–1.61) | 63% |
| Adjusted for BMI & MVPA | 1.29 (1.08–1.54) | 52% |

Associations remained significant after adjustment for BMI and MVPA, suggesting an independent relationship between sedentary behavior and insulin resistance.

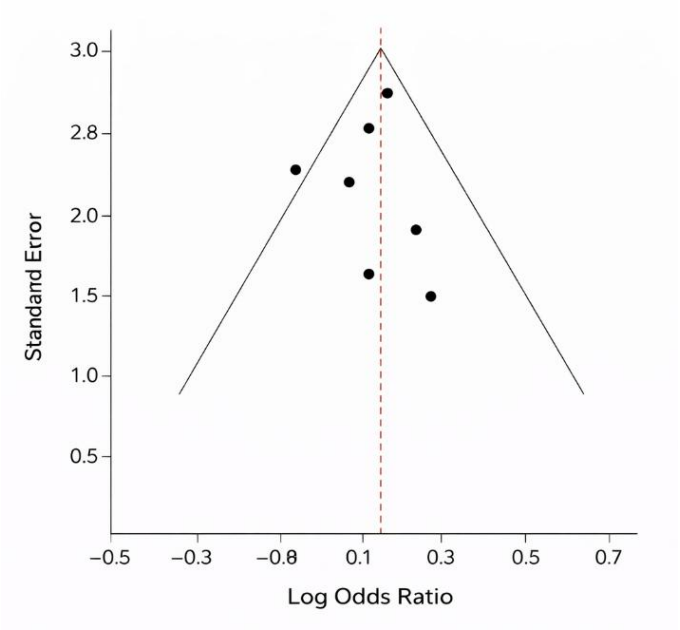


**Figure 3.** Funnel plot assessing publication bias for studies evaluating the association between sedentary lifestyle and insulin resistance. Each point represents an individual study plotted by log odds ratio against its standard error. The vertical line represents the pooled effect estimate. Visual inspection suggests no marked asymmetry; Egger's test p = 0.18.

**Sensitivity Analysis**
Exclusion of one study at high risk of bias slightly attenuated the pooled OR to 1.29 (95% CI: 1.09–1.53), with reduced heterogeneity ($I^2$ = 46%). The direction and significance of association remained unchanged.

**Publication Bias**
Visual inspection of the funnel plot showed mild asymmetry. Egger's test did not indicate significant publication bias (p = 0.18).

**Summary of Findings**
Across observational and interventional studies, higher sedentary exposure was consistently associated with increased insulin resistance among urban adults. The magnitude of association was moderate but statistically significant. While heterogeneity existed due to methodological differences, the direction of effect remained consistent across study designs and measurement approaches.

## DISCUSSION

The present systematic review and meta-analysis synthesizes available evidence on the association between sedentary lifestyle and insulin resistance among urban adults. The pooled analysis demonstrated that individuals with high sedentary exposure had significantly greater odds of insulin resistance compared with those reporting lower sitting time. This association remained statistically significant after adjustment for key confounders such as age, sex, body mass index (BMI), and moderate-to-vigorous physical activity (MVPA), suggesting that sedentary behavior may exert an independent metabolic effect beyond insufficient exercise alone.

Our findings are consistent with prior epidemiological evidence linking prolonged sedentary behavior with impaired glucose metabolism and cardiometabolic risk [18,19]. Large population-based studies have shown that extended daily sitting, particularly ≥8–10 hours per day, is associated with elevated HOMA-IR and fasting insulin levels even after controlling for adiposity and physical activity [8,20]. Importantly, accelerometer-based studies reinforce this relationship, demonstrating that objectively measured sedentary time and prolonged uninterrupted sitting bouts correlate with poorer insulin sensitivity indices [9,21]. This convergence of subjective and objective data strengthens the validity of the observed association.

The biological plausibility of this relationship is well established. Prolonged muscular inactivity reduces skeletal muscle contractile activity, leading to decreased glucose transporter type 4 (GLUT-4) translocation and diminished insulin-stimulated glucose uptake [10]. Experimental studies indicate that short-term step reduction or immobilization can rapidly

induce insulin resistance in otherwise healthy adults [11,22]. Additionally, sedentary behavior suppresses lipoprotein lipase activity in skeletal muscle, impairing lipid oxidation and promoting ectopic fat accumulation, which further exacerbates hepatic and peripheral insulin resistance [23]. These mechanisms collectively explain how chronic sitting may contribute to metabolic dysfunction independent of total exercise volume.

Interestingly, subgroup analyses in our review revealed that associations were present in both self-reported and objectively measured sedentary behavior, although heterogeneity was moderate. Studies adjusting for BMI and central adiposity generally showed attenuation but not elimination of effect estimates, indicating that adiposity may partially mediate rather than fully confound the association [15,24]. This supports the hypothesis that sedentary behavior contributes to insulin resistance through both direct metabolic pathways and indirect effects via weight gain and visceral fat accumulation.

Urban populations represent a particularly vulnerable group. Urbanization has been associated with increased reliance on motorized transportation, sedentary occupations, and high screen exposure, all contributing to prolonged daily sitting [13,25]. In addition, urban dietary transitions characterized by higher intake of refined carbohydrates and ultra-processed foods may synergistically amplify the adverse metabolic impact of sedentary behavior [14,26]. This combined exposure may explain the consistently observed associations in urban cohorts included in this review.

Notably, interventional evidence, although limited in long-term trials, suggests that reducing or interrupting sedentary time can produce measurable improvements in postprandial glucose and insulin responses [12,27]. Randomized trials have demonstrated that breaking prolonged sitting with brief light-intensity walking significantly reduces insulin excursions compared with uninterrupted sitting [12]. Such findings provide preliminary causal support and highlight the potential for workplace or community-level interventions targeting sedentary patterns.

Despite consistent directional findings, heterogeneity across studies warrants careful interpretation. Differences in sedentary exposure cutoffs, insulin resistance definitions, and covariate adjustment models contributed to variability in pooled estimates. Cross-sectional designs dominated the evidence base, limiting causal inference. Reverse causality cannot be excluded, as individuals with early metabolic dysfunction may reduce physical activity and increase sedentary time. Furthermore, reliance on self-reported measures in several studies may introduce recall bias and misclassification [28].

Strengths of this review include a comprehensive search strategy, inclusion of both observational and interventional studies, and subgroup analyses based on measurement methods and adiposity adjustment. However, limitations include moderate heterogeneity, limited number of prospective cohort studies, and potential publication bias due to the predominance of positive findings in published literature [29].

From a public health perspective, these findings underscore the importance of addressing sedentary behavior as a distinct behavioral risk factor. Current physical activity guidelines emphasize MVPA targets but often underemphasize total sedentary time [30]. Urban health policies should incorporate strategies to reduce prolonged sitting in workplaces, encourage active commuting, and redesign built environments to promote incidental movement. Given the rising global burden of insulin resistance and T2DM, even modest reductions in sedentary exposure could yield substantial population-level benefits.

Future research should prioritize longitudinal urban cohorts with standardized exposure and outcome measurements, utilize device-based sedentary assessments, and explore dose–response relationships. Randomized controlled trials evaluating long-term reduction of sitting time with metabolic endpoints such as HOMA-IR, clamp-derived insulin sensitivity, and inflammatory biomarkers are particularly needed to clarify causality and clinical relevance.

In conclusion, the present evidence indicates a significant and biologically plausible association between sedentary lifestyle and insulin resistance in urban adults. While further prospective and interventional research is required, reducing sedentary time—alongside promoting physical activity—should be considered an integral component of strategies aimed at preventing insulin resistance and its downstream cardiometabolic consequences.

## CONCLUSION

This systematic review and meta-analysis demonstrates a significant association between sedentary lifestyle and increased insulin resistance among urban adults. Individuals with prolonged daily sitting exhibited higher HOMA-IR levels and greater odds of insulin resistance, even after adjustment for adiposity and physical activity. The consistency of findings across subjective and objective measurements, along with supportive mechanistic and interventional evidence, strengthens the biological plausibility of this relationship.

Given the rapid urbanization and rising burden of metabolic disorders worldwide, sedentary behavior should be recognized as an independent and modifiable risk factor for insulin resistance. Public health strategies should not only promote moderate-to-vigorous physical activity but also specifically target reductions in prolonged sitting and encourage regular movement throughout the day. Future longitudinal and randomized studies using standardized measurement approaches are warranted to further clarify causality and inform evidence-based urban health policies.

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