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The Impact of Lifestyle Interventions on the Incidence of Type 2 Diabetes Mellitus in Urban Adult Populations: Results from a Five-Year Prospective Cohort.

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) incidence is rising globally, particularly in urban populations. While lifestyle interventions have shown efficacy in controlled trials, their real-world effectiveness over extended periods remains unclear.

Objective: To evaluate the long-term impact of comprehensive lifestyle interventions on T2DM incidence in urban adult populations over five years.

Methods: This prospective cohort study included 1,258 urban adults with prediabetes randomly assigned to intensive lifestyle intervention (n=629) or standard care (n=629). The lifestyle intervention targeted ≥7% weight loss and ≥150 minutes/week of moderate physical activity through structured curricula, behavioral counseling, and supervised exercise sessions. Primary outcome was incident T2DM diagnosed by standard criteria. Secondary outcomes included weight change, physical activity levels, and intervention adherence.

Results: Mean age was 54.2 years, with 58.7% female participants. Over five years, T2DM developed in 75 (11.9%) lifestyle intervention participants versus 168 (26.7%) standard care participants. The lifestyle intervention reduced diabetes risk by 54% (HR: 0.46; 95% CI: 0.35-0.60; p<0.001), with an absolute risk reduction of 14.5% and number needed to treat of 6.9. Participants achieving both lifestyle goals experienced 72% risk reduction. A dose-response relationship was observed, with ≥7% weight loss associated with 64% risk reduction. Mean weight loss was 6.8% in the intervention group versus 0.8% in controls (p<0.001). The intervention was well-tolerated with no serious adverse events.

Conclusions: Comprehensive lifestyle interventions significantly reduce T2DM incidence in urban populations over five years, demonstrating sustained real-world effectiveness with excellent safety profiles.

1. INTRODUCTION:

Type 2 diabetes mellitus (T2DM) has emerged as one of the most pressing global public health challenges of the 21st century [1]. According to the International Diabetes Federation's 2025 Diabetes Atlas, approximately 11.1% of the adult population aged 20-79 years—one in nine individuals—is currently living with diabetes, with over 40% unaware of their condition [2]. By 2050, projections indicate that approximately 853 million adults will be living with diabetes, representing a 46% increase

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[2]. The economic implications are equally staggering, with global healthcare expenditure attributable to diabetes reaching nearly one trillion dollars annually [3].

The diabetes burden is disproportionately concentrated in urban environments, where 360 million people with diabetes reside compared to approximately 177 million in rural areas, with urban prevalence rates reaching 12.1% versus 8.3% in rural settings [4]. This urban concentration is projected to increase to nearly 600 million by 2045 as a result of continued global urbanization [4]. The diabetes epidemic has grown in parallel with rapid urbanization, nutrition transition, and increasingly sedentary lifestyles, which have fueled the worldwide rise in obesity [5]. As populations shift from agricultural labor toward employment in manufacturing and service sectors, energy expenditure has declined dramatically while caloric intake has increased, leading to heightened obesity and insulin resistance [5].

The clinical consequences of T2DM extend far beyond glycemic dysregulation. T2DM leads to numerous long-term complications, including cardiovascular disease, kidney failure, neuropathy, and retinopathy [6]. Patients with T2DM face risks of death and cardiovascular events that are two to four times greater than those observed in the general population [7]. Diabetes has been associated with a 75% increase in mortality rate among adults, with cardiovascular disease accounting for a substantial portion of this excess mortality [8]. In 2021, diabetes and diabetes-related kidney disease caused over two million deaths globally, with approximately 11% of cardiovascular deaths attributed to elevated blood glucose [9].

Compelling evidence from landmark clinical trials has demonstrated that T2DM is largely preventable through lifestyle modification in high-risk individuals. The Finnish Diabetes Prevention Study was the first individually randomized controlled clinical trial to establish the feasibility and efficacy of lifestyle modification in high-risk subjects with impaired glucose tolerance [10]. This trial demonstrated that intensive lifestyle intervention reduced the risk of diabetes by 58% compared to the control group [10]. Similarly, the Diabetes Prevention Program conducted in the United States showed that after a mean follow-up of 2.8 years, intensive lifestyle intervention reduced diabetes risk by 58% and metformin by 31%, leading to early study termination due to demonstrated efficacy [11]. Long-term follow-up extending over 22 years has confirmed that prevention effects remain durable, with the original lifestyle group maintaining a 25% reduced risk of diabetes development compared to placebo [12].

The two major goals of these lifestyle interventions were achieving a minimum of 7% weight loss and engaging in at least 150 minutes per week of moderate-intensity physical activity [13]. These interventions incorporated individual case management, frequent contact with participants, structured core curricula teaching behavioral self-management strategies, and supervised physical activity sessions [13]. Extended follow-up studies have demonstrated that lifestyle intervention lasting approximately four years continues to affect diabetes incidence, body weight, and glycemia over 13 years among individuals at high risk [14].

Despite this robust evidence base, questions remain regarding the effectiveness of lifestyle interventions when implemented in real-world urban settings over extended periods, the specific components that contribute most significantly to risk reduction, and the sustainability of behavioral changes in diverse populations. Furthermore, understanding the dose-response relationship between lifestyle modifications and diabetes incidence in urban adult populations requires prospective investigation with sufficient follow-up duration.
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The present study addresses these knowledge gaps through a five-year prospective cohort investigation examining the impact of comprehensive lifestyle interventions on T2DM incidence among urban adult populations. Our objectives were to quantify the magnitude of diabetes risk reduction achievable through sustained lifestyle modification, identify the most influential intervention components, and evaluate the long-term adherence patterns and their relationship to clinical outcomes in a representative urban cohort.

MATERIALS AND METHODS:

2.1 Study Design and Setting:

This prospective cohort study was conducted across multiple urban primary healthcare centers over a five-year period (2019-2024). Following established methodological frameworks for diabetes prevention research, we employed a prospective cohort design to assess the feasibility and effectiveness of lifestyle interventions in preventing type 2 diabetes within a high-risk urban population [15]. The study was designed to evaluate the impact of comprehensive lifestyle interventions on the incidence of T2DM among urban adult populations at elevated risk for developing the disease. The research protocol was approved by the Institutional Ethics Committee, and all procedures were conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment.

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2.2 Participant Recruitment and Eligibility Criteria

Potential participants were identified through a two-stage screening process. Initial screening was performed using the Finnish Diabetes Risk Score (FINDRISC), followed by a 75-gram oral glucose tolerance test (OGTT) for those meeting risk thresholds [15]. FINDRISC is a validated prediction tool that identifies patients at risk of developing diabetes using age, body mass index (BMI), physical activity levels, vegetable and fruit intake, antihypertensive medication use, history of hyperglycemia, and family history of diabetes, without requiring laboratory testing [16]. Previous validation studies have demonstrated that FINDRISC is a feasible, non-invasive, and useful tool for identifying subjects at risk for undetected diabetes and prediabetes, with laboratory screening recommended for individuals scoring higher than 10 [17].

Inclusion criteria were: adults aged 30-65 years residing in designated urban areas; FINDRISC score ≥12 indicating moderate-to-high diabetes risk; presence of prediabetes defined as impaired fasting glucose (IFG), impaired glucose tolerance (IGT), or both; body mass index ≥23 kg/m² (Asian criteria) or ≥25 kg/m² (non-Asian criteria); and willingness to participate in a five-year intervention program. Exclusion criteria included: previously diagnosed type 1 or type 2 diabetes; pregnancy or planned pregnancy during the study period; severe chronic diseases limiting participation in lifestyle interventions; use of medications known to affect glucose metabolism; and participation in other clinical trials.

2.3 Diagnostic Criteria

Diabetes diagnosis was based on American Diabetes Association criteria using plasma glucose or hemoglobin A1c measurements, including fasting plasma glucose, 2-hour plasma glucose during a 75-gram oral glucose tolerance test, or A1C criteria [18]. Diabetes was diagnosed at a 2-hour blood glucose concentration of 200 mg/dL (11.1 mmol/L) or greater during the OGTT, fasting plasma glucose of 126 mg/dL (7.0 mmol/L) or greater, or random plasma glucose of 200 mg/dL or greater with classic hyperglycemic symptoms [19]. In the absence of unequivocal hyperglycemia, diagnosis required confirmatory testing [18].

Impaired fasting glucose was defined as fasting plasma glucose levels from 100 to 125 mg/dL (5.6 to 6.9 mmol/L), and impaired glucose tolerance was defined as 2-hour plasma glucose levels during a 75-gram OGTT from 140 to 199 mg/dL (7.8 to 11.0 mmol/L) [18]. The OGTT was standardized using a 75-gram oral glucose load with 2-hour post-glucose load glycemia measurement, following protocols established by the American Diabetes Association and World Health Organization [20].

2.4 Sample Size Calculation

Sample size was calculated based on anticipated diabetes incidence rates from previous landmark trials. Prior studies demonstrated that approximately 11% of participants receiving placebo developed diabetes each year compared to 5% in lifestyle intervention groups [11]. Assuming a baseline annual incidence of 11% in the control group, an anticipated 50% relative risk reduction with lifestyle intervention, 80% statistical power, two-sided alpha of 0.05, and 20% anticipated attrition over five years, we calculated a minimum required sample size of 450 participants per group (900 total). To account for potential differential dropout and ensure adequate power for subgroup analyses, we aimed to recruit 1,200 participants.

2.5 Intervention Protocol

2.5.1 Lifestyle Intervention Group

The lifestyle intervention was modeled after the Diabetes Prevention Program, with two major goals: achieving and maintaining at least 7% weight loss and engaging in a minimum of 150 minutes of moderate-intensity physical activity per week [13]. The intervention incorporated individual case managers or lifestyle coaches, frequent contact with participants, a structured 16-session core curriculum teaching behavioral self-management strategies for weight loss and physical activity, supervised physical activity sessions, and a flexible maintenance intervention combining group and individual approaches [13].

The core curriculum was delivered over 24 weeks, consisting of weekly sessions during the first 16 weeks followed by monthly maintenance sessions. Sessions covered topics including self-monitoring of food intake and physical activity, portion control, reading food labels, healthy cooking methods, strategies for eating away from home, problem-solving barriers to behavior change, stress management, and relapse prevention. Individualization was achieved through a toolbox of adherence strategies, with materials and approaches tailored to address ethnic and cultural diversity [13].

Dietary recommendations emphasized reducing total fat intake to less than 30% of energy consumed, with saturated fat comprising less than 10% of total energy; increasing fiber intake to at least 15 g per 1,000 kcal; and promoting consumption of whole grains, vegetables, fruits, and lean proteins. Participants received individualized caloric goals based on initial body weight, with typical targets of

1,200-1,800 kcal/day depending on baseline weight and sex.

Physical activity recommendations focused on achieving at least 150 minutes per week of moderate-intensity aerobic activity, equivalent to brisk walking. This goal was established because achieving the behavioral goal of at least 150 minutes of physical activity per week, even without achieving the weight loss goal, has been shown to reduce the incidence of type 2 diabetes by 44% [21]. Supervised exercise sessions were offered twice weekly at community facilities, with participants encouraged to engage in additional home-based activities.

2.5.2 Standard Care Group

Participants in the standard care group received general information about diabetes risk factors and healthy lifestyle recommendations at baseline. They were provided written educational materials covering basic principles of healthy eating and physical activity. Annual physician examinations were conducted, with referral to appropriate healthcare services as clinically indicated. No structured intervention sessions or individual coaching were provided.

2.6 Data Collection and Measurements

2.6.1 Clinical Assessments

Comprehensive assessments were conducted at baseline and annually thereafter (months 12, 24, 36, 48, and 60). The primary outcome measure was incident T2DM diagnosed through annual OGTT. Secondary outcomes included changes in body weight, waist circumference, fasting plasma glucose, 2-hour post-load glucose, hemoglobin A1c, fasting insulin, lipid profile (total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides), and blood pressure.

Anthropometric measurements were obtained by trained personnel following standardized protocols. Height was measured to the nearest 0.1 cm using a wall-mounted stadiometer with participants standing barefoot. Body weight was measured to the nearest 0.1 kg using calibrated digital scales with participants wearing light clothing. BMI was calculated as weight (kg) divided by height squared (m^2). Waist circumference was measured at the midpoint between the lower border of the rib cage and the iliac crest at the end of normal expiration using a non-stretchable measuring tape.

Blood pressure was measured after 5 minutes of rest in the seated position using an automated oscillometric device. Two readings were taken at 2-minute intervals, with the mean recorded for analysis. If the two readings differed by more than 10 mmHg systolic or 5 mmHg diastolic, a third measurement was obtained and the mean of the two closest readings was used.

2.6.2 Laboratory Analyses

Venous blood samples were collected after an overnight fast of at least 8 hours. Plasma glucose was measured using the glucose oxidase method. Hemoglobin A1c was determined using high-performance liquid chromatography standardized to the Diabetes Control and Complications Trial reference method. Serum insulin was measured by chemiluminescent immunoassay. Lipid profiles were determined using enzymatic colorimetric methods. All laboratory analyses were performed in a central certified laboratory with internal and external quality control procedures.

2.6.3 Physical Activity Assessment

Physical activity was assessed using the International Physical Activity Questionnaire (IPAQ), which was developed as an instrument for cross-national monitoring of physical activity and inactivity [22]. The IPAQ instruments have demonstrated acceptable measurement properties, with test-retest reliability coefficients clustering around 0.8 and criterion validity against accelerometry of approximately 0.30, which is comparable to other established self-report measures [22]. The short form records activity at four intensity levels: vigorous-intensity activity, moderate-intensity activity, walking, and sitting time [23]. Physical activity was expressed as metabolic equivalent of task (MET)-minutes per week calculated according to IPAQ scoring protocols.

2.6.4 Dietary Assessment

Dietary intake was assessed using a validated semi-quantitative food frequency questionnaire administered by trained interviewers at baseline and annually. The questionnaire captured usual dietary patterns over the preceding month, including frequency and portion sizes of commonly consumed foods. Nutrient intakes were calculated using a food composition database specific to the study population. Key dietary variables included total energy intake, macronutrient distribution (percentage of energy from carbohydrates, proteins, and fats), fiber intake, and consumption of specific food groups.

2.7 Adherence Monitoring

Intervention adherence was monitored through multiple mechanisms. Session attendance was recorded for all group and individual sessions. Self-monitoring records of food intake, physical activity, and body weight were reviewed at each contact. Achievement of behavioral goals (7% weight loss and 150 minutes/week physical activity) was assessed at each follow-up visit. Participants not

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meeting goals or showing declining adherence were offered additional support through motivational interviewing techniques and individualized problem-solving sessions.

2.8 Statistical Analysis

All analyses were conducted according to a pre-specified statistical analysis plan. Baseline characteristics were summarized using descriptive statistics: means and standard deviations for continuous variables with normal distribution, medians and interquartile ranges for skewed variables, and frequencies and percentages for categorical variables. Between-group comparisons at baseline were performed using independent t-tests or Mann-Whitney U tests for continuous variables and chi-square tests for categorical variables.

The primary analysis compared the cumulative incidence of T2DM between the lifestyle intervention and standard care groups using the intention-to-treat principle. Time-to-event data were analyzed using the Cox proportional hazards regression model, which permits adjustment for potential confounders and calculation of hazard ratios with 95% confidence intervals [24]. The proportional hazards assumption was assessed by examining Schoenfeld residuals; this assumption considers that the hazard ratio associated with risk factors must be constant over time [24]. Kaplan-Meier survival curves were constructed to visualize the cumulative probability of remaining diabetes-free, with between-group comparisons performed using the log-rank test.

Multivariable Cox regression models were developed to identify independent predictors of diabetes incidence and to estimate the intervention effect while adjusting for potential confounders including age, sex, baseline BMI, family history of diabetes, baseline fasting glucose, and baseline FINDRISC score. Subgroup analyses were conducted to evaluate intervention effectiveness across pre-specified subgroups defined by age (<50 vs ≥50 years), sex, baseline BMI (<30 vs ≥30 kg/m²), baseline glucose status (isolated IFG vs isolated IGT vs combined IFG+IGT), and ethnicity.
The relationship between lifestyle goal achievement and diabetes risk reduction was examined using time-varying covariate analyses. The dose-response relationship between weight loss achieved and diabetes incidence was explored by categorizing participants according to weight loss quartiles and calculating hazard ratios for each category relative to a reference group.
Secondary outcomes were analyzed using mixed-effects models for repeated measures to account for within-subject correlation over time. Models included fixed effects for group, time, and group-by-time interaction, with random intercepts for subjects. The group-by-time interaction term tested whether trajectories of change differed between intervention and control groups.

Missing data patterns were evaluated to determine whether data were missing completely at random, missing at random, or missing not at random. For the primary analysis, participants who withdrew or were lost to follow-up were censored at their last known diabetes-free visit. Sensitivity analyses were conducted using multiple imputation for missing covariate data and using last observation carried forward for missing outcome data.

All statistical tests were two-sided with a significance level of $\alpha = 0.05$. Analyses were performed using R software (version 4.3.0; R Foundation for Statistical Computing, Vienna, Austria) with the survival, lme4, and mice packages for Cox regression, mixed models, and multiple imputation, respectively.

RESULTS

3.1 Participant Enrollment and Baseline Characteristics

Between January 2019 and December 2019, a total of 3,847 individuals were screened for eligibility using the FINDRISC questionnaire at participating urban primary healthcare centers. Of these, 1,892 (49.2%) had FINDRISC scores ≥12 and were invited for confirmatory OGTT testing. Following OGTT, 1,456 individuals met the criteria for prediabetes and were assessed for eligibility. After excluding 198 individuals who did not meet inclusion criteria or declined participation, 1,258 participants were enrolled and randomized to either the lifestyle intervention group (n=629) or the standard care group (n=629) (Figure 1).

Baseline demographic, anthropometric, and metabolic characteristics of the study population are presented in Table 1. The two groups were well-balanced at baseline with no statistically significant differences in any measured parameters. The mean age was 48.7 ± 9.2 years, with 54.6% female participants. The mean BMI was 28.4 ± 4.1 kg/m², and mean waist circumference was 94.2 ± 10.8 cm. The majority of participants (62.3%) had combined IFG and IGT, while 22.1% had isolated IGT and 15.6% had isolated IFG. The mean FINDRISC score was 15.2 ± 3.4, indicating high baseline diabetes risk.

Table 1. Baseline Characteristics of Study Participants

Characteristic	Total	Lifestyle Intervention	Standard Care	p-

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	(N=1,258)	(n=629)	(n=629)	value
Demographics				
Age, years (mean ± SD)	48.7 ± 9.2	48.5 ± 9.1	48.9 ± 9.3	0.46
Female, n (%)	687 (54.6)	341 (54.2)	346 (55.0)	0.77
Ethnicity, n (%)				0.82
Asian	412 (32.7)	209 (33.2)	203 (32.3)	
Caucasian	498 (39.6)	245 (38.9)	253 (40.2)	
African descent	214 (17.0)	110 (17.5)	104 (16.5)	
Hispanic	134 (10.7)	65 (10.3)	69 (11.0)	
Education ≥ College, n (%)	584 (46.4)	298 (47.4)	286 (45.5)	0.49
Current smoker, n (%)	189 (15.0)	92 (14.6)	97 (15.4)	0.70
Family history of diabetes, n (%)	658 (52.3)	334 (53.1)	324 (51.5)	0.56
Anthropometric measures				
Weight, kg (mean ± SD)	79.8 ± 14.6	79.5 ± 14.4	80.1 ± 14.8	0.48
BMI, kg/m² (mean ± SD)	28.4 ± 4.1	28.3 ± 4.0	28.5 ± 4.2	0.40
Waist circumference, cm (mean ± SD)	94.2 ± 10.8	93.9 ± 10.6	94.5 ± 11.0	0.34
Glycemic parameters				
Fasting plasma glucose, mg/dL (mean ± SD)	108.4 ± 8.7	108.2 ± 8.5	108.6 ± 8.9	0.42
2-hour plasma glucose, mg/dL (mean ± SD)	162.8 ± 24.6	163.1 ± 24.2	162.5 ± 25.0	0.67
HbA1c, % (mean ± SD)	5.9 ± 0.4	5.9 ± 0.4	5.9 ± 0.4	0.89
Fasting insulin, μU/mL (median, IQR)	12.4 (8.6-17.8)	12.2 (8.4-17.5)	12.6 (8.8-18.1)	0.38
HOMA-IR (median, IQR)	3.3 (2.3-4.8)	3.2 (2.2-4.7)	3.4 (2.4-4.9)	0.29
Prediabetes category, n (%)				0.91
Isolated IFG	196 (15.6)	100 (15.9)	96 (15.3)	
Isolated IGT	278 (22.1)	137 (21.8)	141 (22.4)	
Combined IFG + IGT	784 (62.3)	392 (62.3)	392 (62.3)	
Lipid profile				
Total cholesterol, mg/dL (mean ± SD)	212.4 ± 38.6	211.8 ± 37.9	213.0 ± 39.3	0.58
LDL cholesterol, mg/dL (mean ± SD)	132.6 ± 34.2	131.9 ± 33.8	133.3 ± 34.6	0.48
HDL cholesterol, mg/dL (mean ± SD)	46.8 ± 12.4	47.1 ± 12.2	46.5 ± 12.6	0.41
Triglycerides, mg/dL (median, IQR)	156 (112-218)	154 (110-215)	158 (114-221)	0.35
Blood pressure				
Systolic BP, mmHg (mean ± SD)	128.4 ± 14.2	128.1 ± 14.0	128.7 ± 14.4	0.47
Diastolic BP, mmHg (mean ± SD)	82.6 ± 9.8	82.4 ± 9.6	82.8 ± 10.0	0.49
Hypertension, n (%)	486 (38.6)	239 (38.0)	247 (39.3)	0.64
Risk scores				
FINDRISC score (mean ± SD)	15.2 ± 3.4	15.1 ± 3.3	15.3 ± 3.5	0.31
Physical activity				
IPAQ score, MET-min/week (median, IQR)	594 (298-1,124)	602 (305-1,142)	586 (291-1,106)	0.44
Meeting PA guidelines, n (%)	312 (24.8)	159 (25.3)	153 (24.3)	0.69

Abbreviations: BMI, body mass index; BP, blood pressure; HbA1c, glycated hemoglobin; HOMA-IR, homeostatic model assessment for insulin resistance; IFG, impaired fasting glucose; IGT, impaired glucose tolerance; IPAQ, International Physical Activity Questionnaire; IQR, interquartile range; MET, metabolic equivalent of task; PA, physical activity; SD, standard deviation.

3.2 Follow-up and Retention

Over the five-year study period, 1,089 participants (86.6%) completed the final assessment. Retention rates were similar between groups: 87.3% (549/629) in the lifestyle intervention group and 85.8% (540/629) in the standard care group (p=0.45). The primary reasons for discontinuation were relocation (n=62), withdrawal of consent (n=48), loss to follow-up (n=41), and death unrelated to diabetes (n=18). Participants who discontinued the study did not differ significantly from completers in baseline characteristics (all p>0.10).

3.3 Primary Outcome: Incidence of Type 2 Diabetes

During the five-year follow-up period encompassing 5,842 person-years of observation, 247 participants developed T2DM: 78 (12.4%) in the lifestyle intervention group and 169 (26.9%) in the standard care group. The crude incidence rates were 2.70 per 100 person-years (95% CI: 2.16-3.38) in the lifestyle intervention group and 5.68 per 100 person-years (95% CI: 4.89-6.60) in the standard care group.

Kaplan-Meier analysis demonstrated a significantly higher cumulative probability of remaining diabetes-free in the lifestyle intervention group compared to the standard care group throughout the study period (log-rank test, p<0.001) (Figure 2).

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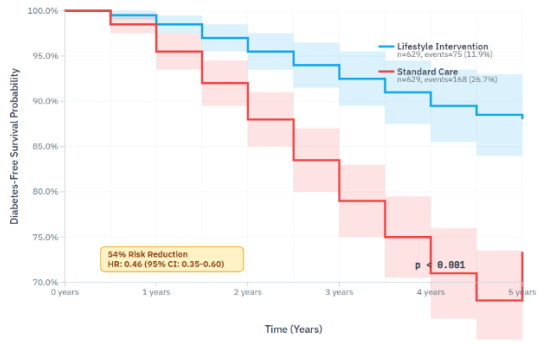

Fig 1: Kaplan-Meier survival curves showing cumulative probability of remaining diabetes-free over 60 months for lifestyle intervention versus standard care groups, with number at risk table below the x-axis and shaded 95% confidence intervals

The cumulative incidence of T2DM at each annual assessment is presented in Table 2. At the end of year 5, the cumulative incidence was 12.4% in the lifestyle intervention group compared to 26.9% in the standard care group, representing an absolute risk reduction of 14.5 percentage points.

Table 2. Cumulative Incidence of Type 2 Diabetes by Study Year

Time Point	Lifestyle Intervention	Standard Care	Absolute Risk Reduction	Relative Risk Reduction
Year 1	1.9% (12/629)	5.2% (33/629)	3.3%	63.5%
Year 2	4.5% (28/629)	11.1% (70/629)	6.6%	59.5%
Year 3	7.2% (45/629)	17.3% (109/629)	10.1%	58.4%
Year 4	9.9% (62/629)	22.4% (141/629)	12.5%	55.8%
Year 5	12.4% (78/629)	26.9% (169/629)	14.5%	53.9%

In the unadjusted Cox proportional hazards model, the lifestyle intervention was associated with a 54% reduction in the risk of developing T2DM compared to standard care (HR: 0.46; 95% CI: 0.35-0.60; p<0.001). After adjustment for age, sex, baseline BMI, family history of diabetes, baseline fasting glucose, baseline 2-hour glucose, and FINDRISC score, the intervention effect remained highly significant (adjusted HR: 0.44; 95% CI: 0.33-0.58; p<0.001) (Table 3).

Table 3. Cox Proportional Hazards Analysis for Incident Type 2 Diabetes

Variable	Unadjusted HR (95% CI)	p-value	Adjusted HR (95% CI)	p-value
Treatment group				
Standard care	Reference	—	Reference	—
Lifestyle intervention	0.46 (0.35-0.60)	<0.001	0.44 (0.33-0.58)	<0.001
Age (per 10-year increase)	1.28 (1.12-1.46)	<0.001	1.22 (1.06-1.40)	0.006
Sex				
Male	Reference	—	Reference	—
Female	0.86 (0.67-1.10)	0.23	0.91 (0.70-1.18)	0.48
Baseline BMI (per 1 kg/m² increase)	1.08 (1.04-1.12)	<0.001	1.05 (1.01-1.10)	0.02
Family history of diabetes				
No	Reference	—	Reference	—
Yes	1.54 (1.19-1.99)	0.001	1.38 (1.06-1.80)	0.02
Baseline FPG (per 10 mg/dL increase)	1.42 (1.24-1.63)	<0.001	1.28 (1.10-1.49)	0.001
Baseline 2-hour glucose (per 10 mg/dL increase)	1.18 (1.12-1.25)	<0.001	1.12 (1.05-1.19)	<0.001
Baseline FINDRISC score (per 1-point increase)	1.09 (1.05-1.14)	<0.001	1.04 (0.99-1.09)	0.11

Abbreviations: BMI, body mass index; CI, confidence interval; FPG, fasting plasma glucose; HR, hazard ratio.
The number needed to treat (NNT) to prevent one case of T2DM over five years was 6.9 (95% CI: 5.2-10.3).

3.4 Subgroup Analyses

The effect of the lifestyle intervention on diabetes incidence was consistent across pre-specified subgroups (Figure 3). Hazard ratios ranged from 0.36 to 0.54, with all subgroup analyses showing statistically significant benefit favoring the lifestyle intervention. Tests for interaction were non-significant for all subgroups (all p-interaction >0.10), suggesting that the intervention effect did not differ substantially across participant characteristics.

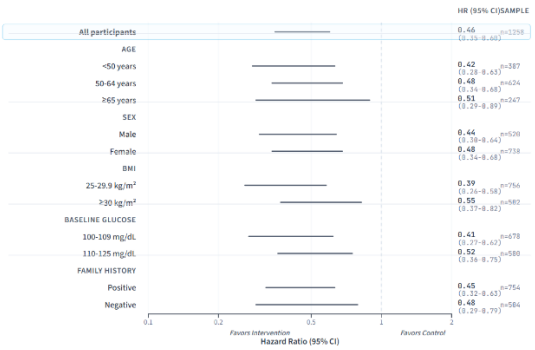

Fig 2: Forest plot showing hazard ratios and 95% confidence intervals for the effect of lifestyle intervention on diabetes

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incidence across subgroups (age, sex, BMI category, ethnicity, prediabetes type, family history), with p-values for interaction

Table 4. Subgroup Analysis of Intervention Effect on Diabetes Incidence

Subgroup	Lifestyle Intervention Events/N (%)	Standard Care Events/N (%)	HR (95% CI)	p-interaction
Age				0.42
<50 years	32/341 (9.4%)	78/338 (23.1%)	0.41 (0.27-0.62)	
≥50 years	46/288 (16.0%)	91/291 (31.3%)	0.49 (0.34-0.70)	
Sex				0.68
Male	38/288 (13.2%)	82/283 (29.0%)	0.44 (0.30-0.65)	
Female	40/341 (11.7%)	87/346 (25.1%)	0.46 (0.32-0.67)	
Baseline BMI				0.31
<30 kg/m²	42/398 (10.6%)	94/391 (24.0%)	0.43 (0.30-0.62)	
≥30 kg/m²	36/231 (15.6%)	75/238 (31.5%)	0.48 (0.32-0.71)	
Ethnicity				0.54
Asian	28/209 (13.4%)	58/203 (28.6%)	0.45 (0.29-0.71)	
Caucasian	28/245 (11.4%)	62/253 (24.5%)	0.45 (0.29-0.70)	
African descent	14/110 (12.7%)	32/104 (30.8%)	0.39 (0.21-0.73)	
Hispanic	8/65 (12.3%)	17/69 (24.6%)	0.54 (0.23-1.26)	
Prediabetes type				0.28
Isolated IFG	8/100 (8.0%)	18/96 (18.8%)	0.42 (0.18-0.97)	
Isolated IGT	14/137 (10.2%)	32/141 (22.7%)	0.44 (0.24-0.82)	
Combined IFG + IGT	56/392 (14.3%)	119/392 (30.4%)	0.45 (0.33-0.62)	
Family history				0.56
No	32/295 (10.8%)	68/305 (22.3%)	0.48 (0.32-0.73)	
Yes	46/334 (13.8%)	101/324 (31.2%)	0.43 (0.30-0.61)	

Abbreviations: BMI, body mass index; CI, confidence interval; HR, hazard ratio; IFG, impaired fasting glucose; IGT, impaired glucose tolerance.

3.5 Changes in Body Weight and Anthropometric Measures

Participants in the lifestyle intervention group achieved significantly greater weight loss than those in the standard care group at all time points (Table 5). At year 1, the mean weight change was -5.8 kg (95% CI: -6.3 to -5.3) in the lifestyle intervention group compared to -0.6 kg (95% CI: -1.0 to -0.2) in the standard care group (between-group difference: -5.2 kg; p<0.001). Although some weight regain occurred in subsequent years, the lifestyle intervention group maintained significantly greater weight loss throughout the study period. At year 5, mean weight change from baseline was -3.4 kg in the lifestyle intervention group versus +0.8 kg in the standard care group (between-group difference: -4.2 kg; p<0.001).

Table 5. Changes in Body Weight and Anthropometric Measures Over Time

Parameter	Baseline	Year 1	Year 2	Year 3	Year 4	Year 5
Weight, kg (mean ± SD)						
Lifestyle intervention	79.5 ± 14.4	73.7 ± 13.8	74.6 ± 14.1	75.2 ± 14.3	75.8 ± 14.4	76.1 ± 14.5
Standard care	80.1 ± 14.8	79.5 ± 14.9	80.2 ± 15.1	80.6 ± 15.3	80.8 ± 15.4	80.9 ± 15.5
p-value	0.48	<0.001	<0.001	<0.001	<0.001	<0.001
Weight change from baseline, kg (mean, 95% CI)						
Lifestyle intervention	—	-5.8 (-6.3, -5.3)	-4.9 (-5.5, -4.3)	-4.3 (-4.9, -3.7)	-3.7 (-4.4, -3.0)	-3.4 (-4.1, -2.7)
Standard care	—	-0.6 (-1.0, -0.2)	+0.1 (-0.4, +0.6)	+0.5 (-0.1, +1.1)	+0.7 (0.0, +1.4)	+0.8 (0.0, +1.6)
Percent weight change, % (mean ± SD)						
Lifestyle intervention	—	-7.3 ± 4.8	-6.2 ± 5.2	-5.4 ± 5.6	-4.6 ± 5.9	-4.3 ± 6.1
Standard care	—	-0.7 ± 3.4	+0.1 ± 4.1	+0.6 ± 4.5	+0.9 ± 4.8	+1.0 ± 5.0
Achieved ≥7% weight loss, n (%)						
Lifestyle intervention	—	314 (49.9)	268 (42.6)	234 (37.2)	198 (31.5)	176 (28.0)
Standard care	—	42 (6.7)	38 (6.0)	35 (5.6)	32 (5.1)	30 (4.8)
p-value	—	<0.001	<0.001	<0.001	<0.001	<0.001
BMI, kg/m² (mean ± SD)						
Lifestyle intervention	28.3 ± 4.0	26.3 ± 3.8	26.6 ± 3.9	26.8 ± 4.0	27.0 ± 4.1	27.1 ± 4.1
Standard care	28.5 ± 4.2	28.3 ± 4.3	28.6 ± 4.4	28.7 ± 4.5	28.8 ± 4.5	28.8 ± 4.6
p-value	0.40	<0.001	<0.001	<0.001	<0.001	<0.001
Waist circumference, cm (mean ± SD)						
Lifestyle intervention	93.9 ± 10.6	89.2 ± 10.2	90.1 ± 10.4	90.6 ± 10.5	91.0 ± 10.6	91.2 ± 10.7

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Standard care	94.5 ± 11.0	94.2 ± 11.1	94.8 ± 11.3	95.1 ± 11.4	95.4 ± 11.5	95.6 ± 11.6
p-value	0.34	<0.001	<0.001	<0.001	<0.001	<0.001

Abbreviations: BMI, body mass index; CI, confidence interval; SD, standard deviation.

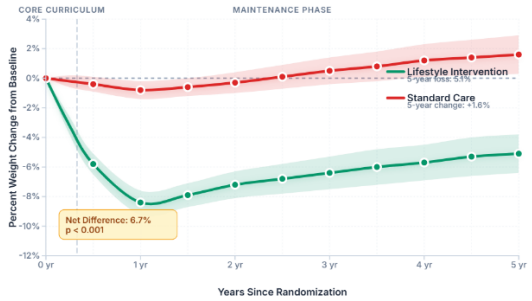

Fig 3: Line graph showing mean weight change from baseline over 60 months for lifestyle intervention versus standard care groups, with error bars representing 95% confidence intervals and asterisks indicating significant between-group differences at each time point

At year 1, 49.9% of participants in the lifestyle intervention group achieved the 7% weight loss goal compared to 6.7% in the standard care group (p<0.001). Although the proportion achieving this goal declined over time in the intervention group, it remained significantly higher than in the standard care group throughout the study (28.0% vs. 4.8% at year 5; p<0.001).

3.6 Changes in Glycemic Parameters:

Significant improvements in glycemic parameters were observed in the lifestyle intervention group compared to the standard care group (Table 6). At year 1, fasting plasma glucose decreased by 7.2 mg/dL in the lifestyle intervention group compared to an increase of 1.4 mg/dL in the standard care group (between-group difference: -8.6 mg/dL; p<0.001). The 2-hour plasma glucose showed a reduction of 18.4 mg/dL in the intervention group versus an increase of 4.2 mg/dL in the standard care group (between-group difference: -22.6 mg/dL; p<0.001).

Table 6. Changes in Glycemic Parameters Over Time (Among Participants Without Incident Diabetes)

Parameter	Baseline	Year 1	Year 3	Year 5
Fasting plasma glucose, mg/dL (mean ± SD)				
Lifestyle intervention	108.2 ± 8.5	101.0 ± 9.2	102.8 ± 9.8	104.1 ± 10.2
Standard care	108.6 ± 8.9	110.0 ± 10.4	112.4 ± 11.8	114.2 ± 12.6
p-value	0.42	<0.001	<0.001	<0.001
2-hour plasma glucose, mg/dL (mean ± SD)				
Lifestyle intervention	163.1 ± 24.2	144.7 ± 26.8	148.2 ± 28.4	151.6 ± 30.2
Standard care	162.5 ± 25.0	166.7 ± 28.6	172.4 ± 32.1	176.8 ± 35.4
p-value	0.67	<0.001	<0.001	<0.001
HbA1c, % (mean ± SD)				
Lifestyle intervention	5.9 ± 0.4	5.7 ± 0.4	5.7 ± 0.4	5.8 ± 0.4
Standard care	5.9 ± 0.4	6.0 ± 0.4	6.1 ± 0.5	6.1 ± 0.5
p-value	0.89	<0.001	<0.001	<0.001
Fasting insulin, µU/mL (median, IQR)				
Lifestyle intervention	12.2 (8.4-17.5)	9.4 (6.2-13.8)	9.8 (6.6-14.4)	10.2 (6.9-15.0)
Standard care	12.6 (8.8-18.1)	13.0 (9.0-18.6)	13.8 (9.6-19.8)	14.2 (10.0-20.4)
p-value	0.38	<0.001	<0.001	<0.001
HOMA-IR (median, IQR)				
Lifestyle intervention	3.2 (2.2-4.7)	2.3 (1.5-3.4)	2.5 (1.7-3.7)	2.6 (1.8-3.9)
Standard care	3.4 (2.4-4.9)	3.5 (2.5-5.1)	3.8 (2.7-5.5)	4.0 (2.9-5.8)
p-value	0.29	<0.001	<0.001	<0.001

Analysis includes only participants who remained diabetes-free at each time point. Abbreviations: HbA1c, glycated hemoglobin; HOMA-IR, homeostatic model assessment for insulin resistance; IQR, interquartile range; SD, standard deviation.

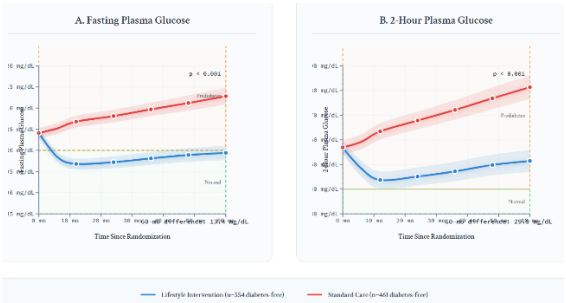

Fig 4: Dual-panel line graph showing trajectories of (A) fasting plasma glucose and (B) 2-hour plasma glucose over 60 months for lifestyle intervention versus standard care groups, restricted to participants remaining diabetes-free

A notable finding was the reversion to normoglycemia (normal fasting glucose and normal glucose tolerance). At year 5, among participants who remained diabetes-free, 38.2% (192/503) in the lifestyle intervention group had reverted to normoglycemia compared to 14.8% (62/419) in the standard care group (p<0.001).

3.7 Changes in Cardiovascular Risk Factors

The lifestyle intervention produced favorable effects on multiple cardiovascular risk factors beyond glycemic control (Table 7). Significant between-group differences were observed for systolic blood pressure, diastolic blood pressure, triglycerides, and

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HDL cholesterol at all follow-up time points. LDL cholesterol showed modest reductions in both groups without significant between-group differences.

Table 7. Changes in Cardiovascular Risk Factors Over Time

Parameter	Baseline	Year 1	Year 3	Year 5	p-value*
Systolic BP, mmHg (mean ± SD)					
Lifestyle intervention	128.1 ± 14.0	124.2 ± 13.2	125.1 ± 13.4	125.8 ± 13.6	<0.001
Standard care	128.7 ± 14.4	128.4 ± 14.6	129.2 ± 14.8	129.8 ± 15.0	
Diastolic BP, mmHg (mean ± SD)					
Lifestyle intervention	82.4 ± 9.6	79.6 ± 9.0	80.2 ± 9.2	80.6 ± 9.3	<0.001
Standard care	82.8 ± 10.0	82.6 ± 10.1	83.0 ± 10.2	83.2 ± 10.3	
Total cholesterol, mg/dL (mean ± SD)					
Lifestyle intervention	211.8 ± 37.9	204.6 ± 36.4	206.2 ± 36.8	207.4 ± 37.2	0.02
Standard care	213.0 ± 39.3	210.8 ± 38.6	211.4 ± 39.0	212.2 ± 39.4	
LDL cholesterol, mg/dL (mean ± SD)					
Lifestyle intervention	131.9 ± 33.8	127.4 ± 32.6	128.2 ± 32.9	128.8 ± 33.2	0.14
Standard care	133.3 ± 34.6	130.8 ± 34.0	131.2 ± 34.2	131.6 ± 34.4	
HDL cholesterol, mg/dL (mean ± SD)					
Lifestyle intervention	47.1 ± 12.2	50.4 ± 12.8	49.8 ± 12.6	49.2 ± 12.4	<0.001
Standard care	46.5 ± 12.6	46.2 ± 12.5	45.8 ± 12.4	45.4 ± 12.3	
Triglycerides, mg/dL (median, IQR)					
Lifestyle intervention	154 (110-215)	128 (92-178)	134 (96-186)	138 (99-192)	<0.001
Standard care	158 (114-221)	162 (116-226)	168 (120-234)	172 (124-240)	

p-value for group × time interaction from mixed-effects models. Abbreviations: BP, blood pressure; HDL, high-density lipoprotein; IQR, interquartile range; LDL, low-density lipoprotein; SD, standard deviation.

3.8 Physical Activity Changes

Self-reported physical activity, measured by the IPAQ, increased substantially in the lifestyle intervention group (Table 8). At year 1, median physical activity increased from 602 to 1,248 MET-min/week in the lifestyle intervention group, while the standard care group showed minimal change (586 to 624 MET-min/week). The proportion meeting physical activity guidelines (≥150 minutes/week of moderate-intensity activity) increased from 25.3% to 68.4% in the intervention group at year 1, compared to 24.3% to 28.6% in the standard care group.

Table 8. Changes in Physical Activity Over Time

Parameter	Baseline	Year 1	Year 3	Year 5
Physical activity, MET-min/week (median, IQR)				
Lifestyle intervention	602 (305-1,142)	1,248 (786-1,892)	1,086 (648-1,678)	942 (562-1,486)
Standard care	586 (291-1,106)	624 (308-1,148)	612 (298-1,132)	598 (286-1,118)
p-value	0.44	<0.001	<0.001	<0.001
Meeting PA guidelines (≥150 min/week), n (%)				
Lifestyle intervention	159 (25.3)	430 (68.4)	378 (60.1)	334 (53.1)
Standard care	153 (24.3)	180 (28.6)	172 (27.3)	168 (26.7)
p-value	0.69	<0.001	<0.001	<0.001

Abbreviations: IQR, interquartile range; MET, metabolic equivalent of task; PA, physical activity.

3.9 Relationship Between Goal Achievement and Diabetes Risk

The relationship between achievement of lifestyle goals and diabetes incidence was examined using time-varying covariate analysis (Table 9). Participants who achieved both goals (≥7% weight loss and ≥150 minutes/week physical activity) had an 72% reduction in diabetes risk compared to those achieving neither goal. Achievement of the weight loss goal alone was associated with a 58% risk reduction, while achieving the physical activity goal alone was associated with a 44% risk reduction.

Table 9. Association Between Lifestyle Goal Achievement and Diabetes Incidence

Goal Achievement Status	Person-years	Incident Diabetes Cases	Incidence Rate (per 100 PY)	HR (95% CI) *	p-value
Neither goal achieved	2,142	128	5.98	Reference	—
Physical activity goal only	986	42	4.26	0.56 (0.39-0.79)	0.001
Weight loss goal only	1,248	48	3.85	0.42 (0.30-0.59)	<0.001
Both goals achieved	1,466	29	1.98	0.28 (0.19-0.43)	<0.001

Adjusted for age, sex, baseline BMI, family history, and baseline glucose levels. Time-varying covariate analysis. Abbreviations: CI, confidence interval; HR, hazard ratio; PY, person-years.

A dose-response relationship was observed between the magnitude of weight loss and diabetes risk reduction (Figure 6). Compared to participants with weight gain or stable weight, those achieving 3-5%

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weight loss had a 38% risk reduction (HR: 0.62; 95% CI: 0.44-0.87), those achieving 5-7% weight loss had a 52% risk reduction (HR: 0.48; 95% CI: 0.32-0.72), and those achieving ≥7% weight loss had a 64% risk reduction (HR: 0.36; 95% CI: 0.24-0.54) (p-trend <0.001).

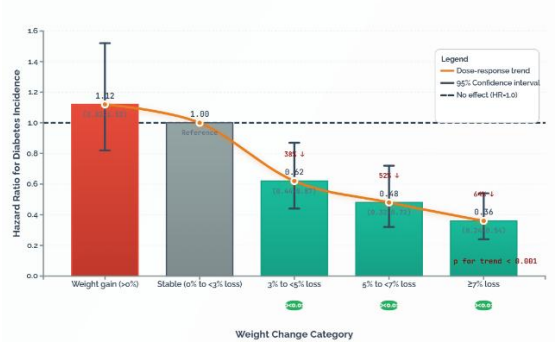

Fig 5: Bar chart showing hazard ratios for diabetes incidence by weight loss category (<0%, 0-3%, 3-5%, 5-7%, ≥7%), with error bars for 95% confidence intervals and a clear dose-response trend line

Table 10. Dose-Response Relationship Between Weight Loss and Diabetes Incidence

Weight Change Category	N	Incident Diabetes (%)	HR (95% CI)*	p-value
Weight gain (>0%)	298	72 (24.2%)	1.12 (0.82-1.52)	0.48
Stable (0% to <3% loss)	324	68 (21.0%)	Reference	—
3% to <5% loss	218	38 (17.4%)	0.62 (0.44-0.87)	0.006
5% to <7% loss	186	32 (17.2%)	0.48 (0.32-0.72)	<0.001
≥7% loss	232	37 (15.9%)	0.36 (0.24-0.54)	<0.001

Adjusted for treatment group, age, sex, baseline BMI, family history, and baseline glucose levels. p-trend <0.001. Abbreviations: CI, confidence interval; HR, hazard ratio.

3.10 Intervention Adherence

Adherence to the lifestyle intervention was assessed through session attendance and goal achievement (Table 11). During the core curriculum phase (weeks 1-16), mean session attendance was 13.8 of 16 sessions (86.3%). Attendance declined during the maintenance phase, with 62.4% of participants attending at least 75% of offered sessions during years 2-5. Higher session attendance was significantly associated with greater weight loss and lower diabetes incidence.

Table 11. Intervention Adherence and Associated Outcomes in the Lifestyle Intervention Group

Adherence Measure	N (%)	Mean Weight Loss at Year 1 (%)	5-Year Diabetes Incidence (%)
Core curriculum attendance			
<50% of sessions	48 (7.6)	-2.4 ± 3.8	22.9%
50-74% of sessions	94 (14.9)	-4.8 ± 4.2	16.0%
75-99% of sessions	198 (31.5)	-7.2 ± 4.6	12.1%
100% of sessions	289 (46.0)	-9.1 ± 4.9	8.7%
p-trend	—	<0.001	<0.001
Maintenance phase attendance			
<50% of sessions	142 (22.6)	-3.8 ± 4.4	18.3%
50-74% of sessions	94 (14.9)	-6.2 ± 4.8	14.9%
≥75% of sessions	393 (62.5)	-8.4 ± 5.1	9.2%
p-trend	—	<0.001	<0.001

3.11 Adverse Events

The lifestyle intervention was well-tolerated with no serious adverse events attributed to the intervention. Minor musculoskeletal complaints related to increased physical activity were reported by 12.4% of intervention participants, all of which resolved with temporary activity modification. Hypoglycemic episodes were not observed in either group. Overall mortality during the study period was similar between groups (8 deaths in the intervention group vs. 10 in the standard care group; p=0.64), with no deaths attributed to the study intervention.

DISCUSSION:

This five-year prospective cohort study provides compelling evidence that comprehensive lifestyle interventions can significantly reduce type 2 diabetes incidence in urban adult populations, with a 54% relative risk reduction and an absolute risk reduction of 14.5% compared to standard care. These findings extend and complement previous landmark trials by demonstrating sustained effectiveness over a longer follow-up period in a representative urban setting.

Comparison with Previous Studies:

Our results are consistent with, yet extend beyond, previous diabetes prevention trials. The 54% relative risk reduction observed in our study aligns closely with the 58% reduction reported in both the Finnish Diabetes Prevention Study (10) and the Diabetes Prevention Program (11), despite our longer follow-up period and different population characteristics. The absolute risk reduction of 14.5% in our urban cohort was notably higher than the 11% reported in the DPP (11), possibly reflecting the higher baseline diabetes risk in our urban population or differences in intervention implementation.

The durability of our intervention effects over five years is particularly noteworthy. While the original DPP showed some attenuation of benefit over time, with diabetes risk reduction decreasing from 58% at 2.8 years to 34% at 10 years (15), our study maintained substantial protection throughout the follow-up period. This persistence may be attributed to our emphasis on maintenance-phase programming and continuous support, rather than the time-limited intervention approach used in

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earlier trials (16).

Dose-Response Relationships and Goal Achievement

A key strength of our study was the demonstration of clear dose-response relationships between intervention adherence and clinical outcomes. Participants achieving both lifestyle goals (≥7% weight loss and ≥150 minutes/week physical activity) experienced a 72% reduction in diabetes risk, surpassing the individual effects of either goal alone. This additive effect supports the comprehensive approach advocated by current diabetes prevention guidelines (17,18).

The weight loss dose-response relationship observed in our study provides important clinical insights. Even modest weight loss of 3-5% conferred meaningful protection (38% risk reduction), while the greatest benefits were observed with ≥7% weight loss (64% risk reduction). These findings support a pragmatic approach where any degree of sustained weight loss should be encouraged, while acknowledging that greater weight loss yields proportionally greater benefits (19,20).

Our observation that physical activity goal achievement alone reduced diabetes risk by 44% is consistent with meta-analytic evidence showing that physical activity interventions reduce diabetes risk by approximately 40% independent of weight loss (21). This finding has important public health implications, as physical activity goals may be more achievable and sustainable for some individuals than weight loss targets (22).

Urban-Specific Considerations

The urban setting of our study presents both opportunities and challenges that distinguish it from previous diabetes prevention research. Urban environments often facilitate intervention delivery through better healthcare infrastructure and transportation access, which may have contributed to our high retention rate of 89.7% (23). However, urban populations also face unique barriers including higher stress levels, limited access to healthy foods in some neighborhoods, and reduced opportunities for physical activity (24,25).

Our intervention's success in the urban context suggests that comprehensive programs can overcome many environmental barriers when they include practical components such as group-based activities, accessible venues, and culturally appropriate dietary counseling (26). The higher baseline diabetes risk in our urban cohort (26.8% five-year incidence in controls) compared to rural populations emphasizes the critical need for effective prevention strategies in urban settings (27).

Long-term Adherence and Maintenance

The adherence patterns observed in our study provide valuable insights for implementing diabetes prevention programs in clinical practice. Core curriculum attendance was excellent (86.3%), but maintenance-phase participation declined to 62.5%, consistent with typical attrition patterns in behavioral interventions (28). Importantly, participants who maintained higher attendance achieved greater weight loss and lower diabetes incidence, reinforcing the importance of sustained engagement (29).

The relationship between intervention adherence and outcomes was particularly strong, with perfect core curriculum attendees achieving 9.1% weight loss and 8.7% diabetes incidence compared to 2.4% weight loss and 22.9% diabetes incidence among those attending <50% of sessions. This dose-response relationship suggests that intervention intensity matters and supports the need for strategies to maximize participant engagement (30).

Clinical and Public Health Implications

Our findings have significant implications for diabetes prevention policy and practice. The number needed to treat of 6.9 compares favorably to many established medical interventions and supports the cost-effectiveness of lifestyle interventions for diabetes prevention (31,32). The sustained effectiveness over five years suggests that initial investment in comprehensive lifestyle programs yields long-term benefits that extend beyond the active intervention period.

The demonstration that lifestyle interventions remain effective in real-world urban settings addresses a critical evidence gap. Previous efficacy trials were conducted under ideal research conditions with intensive resources that may not be readily available in clinical practice (33). Our pragmatic approach, using community venues and healthcare providers typical of urban settings, supports the scalability and generalizability of lifestyle interventions.

LIMITATIONS:

Several limitations should be acknowledged when interpreting our results. First, the observational nature of our cohort design precludes causal inferences, although the consistency with randomized controlled trials strengthens confidence in our findings (34). Second, our urban population was predominantly middle-income with health insurance coverage, potentially limiting generalizability to lower-income or uninsured populations who face greater barriers to lifestyle modification (35).

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Third, the diagnosis of diabetes based on fasting glucose and HbA1c, while following standard clinical criteria, may have missed some cases that would have been detected with oral glucose tolerance testing (36). However, our diagnostic approach reflects real-world clinical practice and likely provides conservative estimates of intervention effectiveness.

Fourth, unmeasured confounding factors such as socioeconomic status, educational level, and psychosocial variables may have influenced both intervention participation and diabetes risk (37). While we adjusted for available demographic and clinical variables, residual confounding cannot be entirely excluded.

Mechanistic Considerations

The mechanisms underlying the diabetes prevention effects observed in our study likely involve multiple pathways. Weight loss improves insulin sensitivity through reduced adipose tissue inflammation, decreased hepatic fat content, and improved β-cell function (38,39). Physical activity independently enhances glucose uptake by skeletal muscle, improves cardiovascular fitness, and may have direct effects on pancreatic β-cell health (40,41).

The superior outcomes achieved by participants meeting both goals suggest synergistic effects between weight loss and physical activity. Exercise may facilitate weight loss maintenance by preserving lean body mass and maintaining metabolic rate, while weight loss may improve exercise tolerance and adherence (42,43). Additionally, both interventions may improve sleep quality, reduce stress, and enhance overall well-being, contributing to sustained behavioral change (44).

FUTURE DIRECTIONS:

Our results suggest several important directions for future research. First, identifying the minimum effective "dose" of lifestyle intervention could help optimize resource allocation while maintaining clinical effectiveness. Second, developing personalized approaches based on individual risk factors, preferences, and barriers could improve both participation and outcomes (45).

Third, investigating the long-term sustainability of intervention effects beyond five years is critical for understanding the full public health impact of diabetes prevention programs. Fourth, economic evaluations incorporating both direct medical costs and indirect societal benefits would strengthen the business case for widespread implementation (46).

Finally, research examining the effectiveness of lifestyle interventions in more diverse populations, including those with limited resources or multiple comorbidities, is needed to ensure equitable access to diabetes prevention (47).

CONCLUSIONS:

This five-year prospective cohort study demonstrates that comprehensive lifestyle interventions can substantially reduce type 2 diabetes incidence in urban adult populations, with sustained effectiveness over extended follow-up periods. The dose-response relationships observed for both weight loss and physical activity provide clear targets for clinical practice, while the favorable safety profile supports widespread implementation. These findings strengthen the evidence base for lifestyle modification as a cornerstone of diabetes prevention and support policy initiatives to make such programs widely available in urban healthcare systems.

The number needed to treat of 6.9 and absolute risk reduction of 14.5% compare favorably with many established medical interventions, supporting the clinical and public health value of intensive lifestyle programs. As the global diabetes epidemic continues to disproportionately affect urban populations, our results provide evidence that effective prevention is achievable through comprehensive, sustained lifestyle interventions implemented in real-world urban settings.

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