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Prevention of Type 2 Diabetes in U.S. Hispanic Youth: A Systematic Review of Lifestyle Interventions

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Abstract

Context—Prevalence of type 2 diabetes mellitus (T2DM) in youth has increased rapidly in recent decades along with rises in childhood obesity. Disparities in risk and prevalence of T2DM are evident in Hispanic youth when compared with non-Hispanic whites. Targeted diabetes prevention programs have been recommended to reduce risk prior to adulthood in this population. This systematic review explores the effectiveness of lifestyle-based diabetes prevention interventions for Hispanic youth.

Evidence acquisition—PubMed, PsychInfo, Web of Science, and CENTRAL were searched from database inception to March 1, 2017, for studies that evaluated lifestyle-focused prevention trials targeting U.S. Hispanic youth under age 18 years. Fifteen publications met criteria for inclusion.

Evidence synthesis—Eleven of fifteen studies were RCTs; four were uncontrolled. Interventions were heterogeneous in intensity, content, and setting. Duration of most trials was 12–16 weeks. Mean age of participants ranged from 9.8 to 15.8 years, sample sizes were generally small, and the majority of participants were overweight (BMI ≥85th percentile). Three studies reported statistically significant reductions in mean BMI, four in BMI z-score, and six in fasting glucose/insulin. Study quality was moderate to high. Effect sizes were generally small to medium.

Conclusions—Evidence for the impact of lifestyle-based diabetes prevention interventions targeting U.S. Hispanic youth remains limited. Few interventions demonstrated success in reducing BMI and glucose regulation and follow-up times were brief. More studies are needed that recruit larger samples sizes, extend follow-up times, explore innovative delivery modalities, and examine effectiveness across sex and age.

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Context

The prevalence of childhood obesity in the U.S. has risen markedly in recent decades, contributing to heightened risk for type 2 diabetes mellitus (T2DM) over the lifespan and increased public health costs. Insulin resistance, a cardinal etiologic component of T2DM, is common in the context of childhood obesity and is associated with development of metabolic syndrome, T2DM, and cardiovascular disease,[1] and predicts risk of cardiometabolic morbidity and mortality during adulthood.[1] Hispanic/Latino (hereafter referred to as "Hispanic") youth are disproportionally affected by these trends, and prevalence of cardiometabolic risk factors in this population are concerning. In youth aged 8–16 years in the multi-site Study of Latino Youth, 19.9% were overweight, 26.5% were obese,[2] and 16.5% had prediabetes or diabetes.[2] Though the overall prevalence of T2DM in U.S. youth is low (e.g., 0.046% in 2009 in the SEARCH for Diabetes in Youth study[3]), prevalence has increased by 30% from 2001, and rates are significantly higher in Hispanic youth (0.079%) compared with their non-Hispanic white counterparts (0.017%).[3] In the National Health and Nutrition Examination Survey, Hispanic youth exhibited higher prevalence of obesity than non-Hispanic white youth (2011–2014, 21.9% versus 14.7%)[4] and metabolic syndrome (2001–2006, 11.2% versus 8.9%).[5] Further, Hispanic children are more insulin resistant than non-Hispanic white children independent of levels of adiposity.[6] Once diagnosed with T2DM, Hispanic individuals have poorer glucose control, more frequent organ and vascular complications, and increased depression, cardiovascular disease, and overall mortality.[7–9] Thus, early prevention is critical.

Multiple RCTs have shown that lifestyle interventions such as the Diabetes Prevention Program that emphasize changes in diet and physical activity (PA) and incorporate behavior change strategies can delay or prevent T2DM in adults[10–12] and reduce diabetes risk in youth.[13–16] Many of these programs, however, have struggled to engage and retain minority youth,[17–22] which may contribute to disparities in diabetes risk and outcomes. In attempt to more effectively reach high-risk individuals, tailored and adapted approaches have been recommended that modify content, language, mode of delivery, theoretical approach or other intervention components to improve engagement and outcomes.[23–25] Although multiple interventions targeting Hispanic Americans have been created with this goal, it is unclear to what extent programs have targeted Hispanic youth, and if they have been effective in reducing risk for T2DM. No publications have systematically aggregated data on diabetes prevention interventions for Hispanic youth, or examined the extent to which existing programs were able to modify critical risk reduction outcomes (e.g., reduction in BMI or improved insulin and glucose regulation or both, as defined by fasting insulin or glucose).

The current systematic review examines all peer-reviewed publications of lifestyle-based diabetes prevention interventions targeting Hispanic youth. The primary aim is to investigate the effectiveness of these interventions in lowering risk for T2DM via reductions in adiposity, as measured by BMI, or glucose dysregulation —variables known to be associated with development of diabetes in youth.[26,27] Secondarily, the review examines effectiveness in modifying health behaviors critical to diabetes risk in youth: PA and dietary intake.

Evidence Acquisition

Data Sources

This systematic literature review was conducted and reported in alignment with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.[28] Search protocols were designed for PubMed, PsychInfo, the Cochrane Central Register of Controlled Trials, and Web of Science to capture all studies from database inception to March 1, 2017. Search terms included medical subject (MeSH) headings and keywords in three domains: disease focus (*diabetes*), intervention type (*prevention*, *risk reduction*), and population (*Hispanic/Latino* and *children/adolescents*). For example, the PubMed search included the following terms: [*diabetes mellitus/prevention and control* (MeSH) OR *diabetes prevention* (keyword) OR *diabetes risk reduction* (keyword)] AND [*intervention* (keyword) OR *health promotion* (MeSH)] AND [*Hispanic Americans* [MeSH] OR *Hispanic* (keyword) OR *Latin**(keyword)] AND [*child* (MeSH) OR *adolescent* (MeSH) *OR youth* OR *teen**]. The MeSH term [*Hispanic Americans*] includes all Hispanic nationalities (e.g., Mexican, Cuban, Puerto Rican). Hand searching of reference lists of included publications was conducted to identify additional eligible studies. Due to absence of peer-review and risk of bias, grey literature (e.g., dissertations, conference proceedings) was not included.

Study Selection

The following inclusion criteria were used:

1. study evaluated a behavioral lifestyle intervention (i.e., an intervention that targeted behavior change in nutrition/dietary intake or PA or both; not a pharmaceutical intervention);
2. the intervention aimed to reduce risk for T2DM (e.g., not simply a weight loss/ obesity intervention);
3. study targeted and recruited primarily Hispanic youth participants (Hispanic youth comprised ≥50% of the sample); and
4. the publication reported an outcome measure of BMI or a glycemic regulation variable (e.g., fasting insulin, fasting glucose, oral glucose tolerance test [OGTT]) or both.

The criterion of ≥50% Hispanic youth in the sample was selected to capture studies that targeted and recruited large numbers of Hispanic youth, but allowed participants of other ethnicities as well (e.g., school-based interventions). Studies were excluded if participants had diabetes, if published in a language other than English or Spanish, or if they were secondary analyses of trials already included.

Data Extraction and Quality Assessment

Two reviewers independently conducted review of abstracts, data extraction, and quality appraisal. Screening and inclusion details are shown in the flow diagram (Figure 1). The following information was extracted from each publication: participant characteristics, study design and setting, intervention components, and select clinical and behavioral outcomes (BMI or glycemic regulation or both, PA, and dietary intake). Raw mean difference and Hedges' g effect sizes were calculated for changes in clinical outcomes (e.g., BMI, fasting glucose). Consistent with Cochrane systematic review guidelines,[29] effect sizes were not aggregated in a meta-analysis because of the heterogeneity of study design, intervention content, and intervention intensity. The Effective Public Health Practice Project Quality Assessment Tool (EPHPP; available online at www.ephpp.ca/tools.html) was utilized to rate data quality.

Evidence Synthesis

Study Characteristics

The search strategy identified 151 unique publications, and 15 studies were included in the final review (Figure 1). Characteristics of included studies are presented in Table 1 and clinical outcomes are presented in Table 2. Eleven studies were RCTs and four were uncontrolled. Samples were mostly convenience or clinic-referred. Sample sizes were generally small, varying from N=15 to N=102, with the exception of two large community-based trials that included N=1,419[30] and N=4,603[31] children across multiple schools. Mean age of participants ranged from 9.8 to 15.8 years. In addition to age, eight studies measured and reported Tanner stage, an assessment of physical maturation ranging from 1 (pre-adolescent) to 5 (post-puberty/sexual maturity). Of these, four studies contained samples of youth in stages 3–5[32–35] and four reported youth in stages 4–5 only.[36–39] Three studies recruited only females[33,36,37] and two recruited only males.[34,35] The majority of participants were overweight or obese (age- and sex-specific BMI ≥85th or 95th percentile, respectively). Twelve of 15 studies used risk-based inclusion criteria, either BMI percentile (generally, BMI ≥85th percentile) or the American Diabetes Association consensus panel risk score. One intervention included a corresponding intervention group of parents[40]; child data were extracted for this review.

Interventions

Detailed information about each intervention is presented in Appendix Table 1. Interventions took place across multiple settings: clinics,[32,36–39,41] schools,[30,31,40,42] community settings,[35,43] participant homes,[34] and online.[44] One intervention compared parallel home- and clinic-based interventions.[33] Most interventions targeted nutrition and increased PA, but four studies targeted PA only,[34,35,37,38] and one study targeted nutrition only.[33] All utilized face to face delivery of content, except for two arms of a web-based intervention.[44] Interventions generally involved weekly contact with youth and varied in duration from 10 weeks to 2.5 years, the majority lasting 12–16 weeks. Follow-up assessments occurred at the post-intervention time point in all but one study, which measured outcomes after 1 year.[41] Ten studies utilized a group modality, two provided individual sessions,[34,35] one involved both group and individual,[38] and two compared outcomes across group and individual arms.[33,44] The following behavior change strategies were common: guided PA/PA education (13 studies), nutrition education/counseling (ten studies), diabetes-specific education (five studies), motivational interviewing (five studies), goal setting (six studies), self-efficacy/self-esteem building (six studies), and parent engagement (six studies). Two studies reported no behavior change strategies aside from PA training.[35,38]

Clinical Outcomes

Reduction of risk for diabetes was measured by a variety of outcomes: changes in weight, BMI, cardiovascular fitness, total fat and lean muscle mass, and multiple outcomes of glucose regulation or insulin sensitivity (e.g., fasting glucose, fasting insulin, OGTT, homeostatic model assessment of insulin resistance [HOMA-IR], and insulin sensitivity measured by frequency-sampled intravenous glucose tolerance test [FSIVGTT]). In line with the a priori inclusion criteria of this review, data were extracted for outcomes of BMI and glucose regulation variables. Given the heterogeneity in variables measured, the four most commonly reported outcomes were selected for presentation in Table 2: BMI, BMI z-score, fasting glucose, and fasting insulin.

Twelve of 15 studies measured participant BMI (kg/m^2). Of these, three (two RCTs) reported a statistically significant reduction in mean BMI post intervention or compared with the control group.[36,41,42,41,42] Within-group changes in BMI were small across studies, ranging from −0.1 to −1.1 kg/m^2. Eight studies reported changes in BMI z-scores. Of these, three RCTs[31,33,36] and one uncontrolled study[42] reported significant reductions. Changes in within-group BMI z-scores were also small, ranging from −0.1 to +0.04. Six studies evaluated changes in age- and sex-adjusted BMI percentile; one reported significant intervention effects (change of −1.3%).[43]

Of ten studies that reported fasting glucose outcomes, two RCTs reported statistically significant reductions compared with controls.[30,36] Mean differences in fasting glucose ranged from −0.2 to −4.3 mg/dL. Nine studies reported fasting insulin outcomes, and four resulted in significant reduction in insulin either between[31,37] or within[38,41] groups. Mean reduction in fasting insulin across studies ranged from −0.5 to −5.9 μU/mL. Of five interventions that calculated HOMA-IR scores[32,33,37–39] two achieved significant changes compared with the control group (21% versus 4% decrease)[33] or baseline (mean change −0.8; results not shown in Table 2).[38] Seven studies measured post-load 2-hour glucose via OGTT, but only one[43] reported an improved tolerance compared with baseline (10.8% reduction). Four studies conducted FSIVGTT for direct assessment of insulin sensitivity. One of these,[35] a resistance training intervention, reported significant differences compared with baseline and controls (increase in insulin sensitivity of 0.9×10^{-4} min^{-1}* μU^{-1} mL^{-1}).

Behavioral Outcomes

As a secondary aim of this review, changes in PA and dietary intake were extracted and results are presented in Table 3. Eight studies examined self-reported changes in dietary intake[30,32,33,36,37,39,43,44] and six reported significant changes compared with controls[32,36,39,44] or baseline[33,43](Table 3). Tools utilized to assess dietary intake were 3-day dietary intake records, a self-administered food frequency questionnaire[44] and the Brief Dietary Assessment Tool for Hispanics.[43] Statistically significant changes were reported in each of the following outcomes: reduction in amount of sugar consumed,[33,36] reduction in carbohydrate intake,[32,33] reduction in dietary fat,[32,43] reduction in total energy consumed,[32,33] increase in dietary fiber,[30,33] and increase in fruit and vegetable consumption compared with controls.[44]

Five studies measured pre- and post-intervention PA (Table 3). Three assessed PA by self-report alone[39,43,44]; two utilized self-report and 7-day accelerometry measures.[36,37] Observed changes in PA ranged from no change/non-significant changes to a 29% increase in daily moderate intensity PA compared with controls, based on self-report.[39] Because of the heterogeneity in components of PA and dietary intake reported, effect sizes could not be calculated for these outcomes.

Quality of Evidence

A summary of evidence quality is presented in Appendix Table 2. Utilizing the data quality rating protocol of the EPHPP, the following characteristics were evaluated to generate a global rating for each study: selection bias, study design, confounders, blinding, assessment methods, and attrition. Four publications received a strong global rating of evidence quality[30,41,43,44]; the remaining studies received a moderate global rating. The included studies were strong on study design (11 RCTs), control of confounders, and assessment methodology. Quality ratings were often low for selection bias, attrition, and lack of blinding.

Discussion

Of the 15 interventions surveyed in this review, six resulted in reductions in either BMI or BMI z-score, and six reported changes in fasting glucose or fasting insulin. The 11 RCTs presented the strongest evidence for behavioral interventions, and reported significant changes in BMI/BMI z-score (n=3) and fasting glucose or insulin (n=4). Although evidence quality was moderate to strong, results must be interpreted cautiously as samples sizes were mostly small, and long-term effects of interventions are unknown. Considering these factors, evidence for feasibility of lifestyle-based diabetes prevention programs targeting Hispanic youth exists, but support for efficacy in reducing risk for diabetes remains limited.

It is worth noting that while effect sizes were small to medium for most interventions, incremental differences in glucose regulation can correspond to clinically meaningful changes in disease risk. Risk for incident diabetes increases markedly across the A1c range of 5.0 to 6.5%.[45] Though risk associated with small changes in fasting glucose in adolescents is less established, mean fasting glucose at baseline in many samples was 90–95 mg/dL, very close to the cut point for impaired fasting glucose/prediabetes (100–125 mg/dL). A mean increase of up to 4.4 mg/dL was observed in some groups over the course of a 16-week intervention, meaning that youth are exhibiting rapid movement towards prediabetes without intervention, or despite intervention in some cases (e.g.,[34]). In these high-risk scenarios, interventions that result in reduction of even a few mg/dL in mean fasting glucose may prevent or stall the development of prediabetes and further pathophysiological changes.

Intervention Completeness

Despite strong evidence supporting the necessity of addressing both PA and dietary intake in lifestyle interventions for effective reduction of diabetes risk,[10–12,15,16] multiple interventions addressed only one of these domains,[33–35,37,38] and some studies reported using few or no behavior change strategies. Future interventions should target both PA and nutrition using empirically supported strategies (e.g., setting achievable goals, reducing barriers, increasing self-efficacy) to provide youth an optimal chance of realizing and maintaining difficult behavior change.

Sample Heterogeneity

A challenge in the detection of intervention effects on glucose regulation in pediatric populations is sample heterogeneity in age, sex, and level of metabolic risk, as these factors affect adiposity and glucose metabolism in complex ways. Most samples included children of both sexes who spanned multiple Tanner stages. Age and pubertal stage can affect outcomes through physiological and behavioral pathways,[1,26] including increases in growth hormone secretion and changes in adiposity,[1] and these may differ by sex. Growth hormones affect both adiposity and glucose regulation, and adiposity is associated with increased fasting insulin and insulin resistance.[1] Insulin resistance varies with pubertal level or Tanner stage, increasing roughly 25%–50% during early to mid puberty and returning to normal levels at the end of pubertal development.[26] This timeline has been shown to vary by sex,[46] with serum insulin concentrations peaking at earlier ages and remaining higher in females than males prior to age 18 years. Eight of the reviewed studies measured Tanner stage, but only three controlled for it in analyses[30,33,36] and none stratified analyses by stage. Further, few studies presented sex differences in outcomes.[30,39,44] Because of fluctuations in insulin resistance that occur during pubertal ages,[26,47] such as transient decline in insulin sensitivity,[48] sex and age/pubertal stage should be controlled for in analyses and considered in interpretation of intervention effects on glucose regulation. Including adiposity as a covariate, on the other hand, as was done by four author groups[33,35,36,39] may be over controlling, as changes in adiposity may represent an intermediate stage in the causal pathway through which lifestyle interventions lead to changes in glucose metabolism.

Regarding health behaviors, older adolescents are likely to have autonomy with PA and dietary intake, whereas younger children and adolescents rely heavily on caregivers for facilitation of activities and food.[49,50] A lifestyle intervention focused on motivation, goal setting, and decision making may therefore have more immediate success in older adolescents, whereas an intervention that prioritizes caretaker engagement may have stronger effects in younger adolescents. Studies with samples that fall across the spectrum of increasing autonomy and do not explore age as a moderator or effect modifier, as many of the studies in this review, may have difficulty detecting effects that vary by age.

Participants exhibited varying levels of metabolic risk, including normal weight, overweight with no metabolic dysregulation, and overweight/obese plus metabolic dysregulation (e.g., low high-density lipoprotein cholesterol, triglyceridemia). As glucose dysregulation progresses gradually, and is often not observed at clinical levels until adulthood, post-intervention changes may be difficult to detect in adolescents with low duration of exposure to glucose dysregulation (and little resulting β cell damage).[47,51] However, given that β cell decline is thought to progress more rapidly in youth than adults once dysregulation begins,[47,52,53] early prevention and intervention efforts are critically important.

Outcome Selection and Measurement

Although it is possible that many of the interventions reviewed were simply not effective, or not of appropriate intensity to affect BMI or insulin resistance, the choice of outcome may also have obscured true intervention effects. Fasting glucose and fasting insulin, surrogate markers of insulin resistance, were the most commonly reported outcomes in the included studies. These variables are not consistently correlated with insulin sensitivity in adolescents, however.[54,55] Fasting insulin has been shown to have low accuracy as a measure of insulin sensitivity, with correlations between fasting insulin and the gold standard euglycemic hyperinsulinemic clamp ranging from 0.42–0.91.[54] Similarly, changes in insulin sensitivity detected by the FSIVGTT or OGTT after a lifestyle intervention for overweight youth were not detected by the HOMA-IR.[55] The Insulin Resistance in Children Consensus Group[56] recommends use of the clamp technique in intervention trials, names the FSIVGTT and steady-state plasma glucose methods as reliable and valid alternatives, and recommends fasting glucose and insulin only in large epidemiological studies.[54,56] However, the clamp, FSIVGTT, and steady-state plasma glucose are time- and resource-intensive and burdensome for participants, as they require repeated intravenous blood sampling and testing done in a closely monitored clinic setting. As illustrated by the infrequent use of these measures in the trials in this review, the trade-off between validity of measure and feasibility/participant acceptability may not be worthwhile. Nonetheless, given that most youth will not develop clinical levels of diabetes pathology for many years, the lack of direct measurement of insulin sensitivity and other pathophysiological processes (e.g., insulin secretion, β cell function) may conceal or confound true intervention effects.

Despite trends of using either BMI z-score or BMI percentile for youth adiposity outcomes,[57–59] multiple studies presented simple BMI without adjustment for age and sex. BMI z-scores and percentiles are generally preferred over raw BMI due to the fluctuations in adiposity and growth that differ by sex over the course of adolescence[26,46,47,57] and because those measures are highly correlated with Dual-Energy X-ray Absorptiometry-measured adiposity[60] and fat mass z-score change,[61] though some researchers continue to report BMI without adjustment.[62,63]

Behavioral Outcomes

In the 11 studies that targeted dietary changes, outcomes were promising. Six of eight studies that measured dietary intake showed significant post-intervention increases in healthy eating. These outcomes are impressive considering the multiple domains of influence on adolescent eating behavior (e.g., school food availability, caregiver income and food provision, peer influences). Interventions appear to have been more successful in influencing renunciation of unhealthful foods (e.g., reduction in dietary sugar, sugary beverages, carbohydrates, or fat) than uptake of healthy foods (e.g., vegetables, fruit). All measurements were based on participant self-report, however, and social desirability bias may have affected responses.

Surprisingly, despite the focus on PA modification in all but one intervention,[33] fewer than half of the studies measured daily or routine PA, and only one[39] detected significant changes post-intervention. A small number of studies reported cardiovascular fitness level,[30,38] or strength[32,35] instead of PA, citing the lower reliability and validity of self-report measures of PA in children. Although objective measures of PA are preferable to self-report, omission of measurement of daily or routine PA is also problematic, as the presumption of lifestyle interventions is that dietary and PA changes will influence glucose metabolism. If PA is not measured, conclusions regarding the mechanism of action of the intervention are limited. Furthermore, sedentary behavior was not measured by any study in this review, but represents an important component of the adolescent activity spectrum. Because sedentary behavior is associated with elevated fasting insulin regardless of moderate to vigorous PA, if sedentary behavior is high, substantive changes in insulin resistance may not be observed even if PA is increasing. Investigators planning new interventions should strongly consider objective measurement of both PA and sedentary behavior via accelerometry.

Targeting and Tailoring

Despite targeting Hispanic/minority youth, few studies reported cultural tailoring or adaptation of intervention content to improve engagement. Only four studies[30,33,40,41] described culturally-informed adaptation, and in most cases this was limited in scope, for example, incorporation of Mexican food recommendations and recipes,[40] or inclusion of “bilingual culturally and contextually relevant themes.”[30] Only one intervention was developed through a community-based participatory research approach that included Hispanic community members,[41] and only one intervention incorporated youth feedback in creation of intervention content.[44] An obvious area for improvement of intervention salience and engagement is to incorporate members of the target demographic in intervention development processes, and provide delivery modalities tailored to youth, such as mobile applications.

Limitations

This is the first systematic review to synthesize outcomes from diabetes prevention interventions targeting Hispanic youth. PRISMA guidelines were followed throughout the review process from literature search to reporting of results. A comprehensive search strategy was utilized across multiple databases, and data quality was assessed via the rigorous, structured EPHPP protocol. A notable strength of the review is the quality of data included; all evidence was moderate to strong. Limitations include the inclusion of uncontrolled trials and trials with small sample sizes. As most interventions were pilot or exploratory in nature, many were not powered to detect the clinical outcomes examined. In addition, interventions with null results may not have been included due to publication bias. Lastly, results may not be generalizable to all heterogeneous groups of Hispanic youth. Though most studies did not specify Hispanic heritage or family nationality, the majority of studies were conducted in southern California and likely involved youth of primarily Mexican background.

Conclusions

Given the heterogeneity of intervention content, lack of stratification by sex and pubertal development level, deficits in power, and small sample sizes of interventions to date, the effectiveness of lifestyle-based diabetes prevention programs for Hispanic youth has not been conclusively demonstrated. The completion of 15 lifestyle interventions in Hispanic youth lends evidence for the feasibility and relevance of targeted interventions. Nonetheless, high attrition rates indicate a need for improved tailoring and engagement strategies, recognition and reduction of barriers to completion, and novel delivery modalities. Future interventions should assess participant engagement, solicit feedback about barriers and facilitators of participation, and publish these results. New interventions would be served by employing socioecological and systems-based approaches that recognize the multiple domains of influence on adolescent behavior (e.g., family, school, peer, community). Given the prevalence of Internet and mobile device use among adolescents (92% of teens go online daily; 71% of Hispanic teens have access to a smartphone),[64] intervention designers should consider innovative mobile and online delivery formats with strategic elements that boost user engagement.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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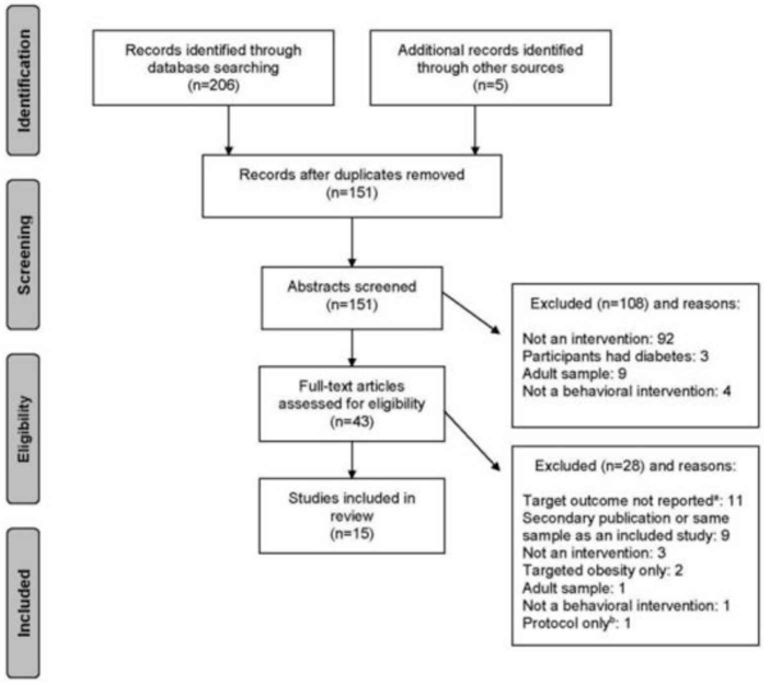

Figure 1.

Flow diagram: screening and inclusion of publications.

[a]Outcomes that were inclusion criteria for this review (BMI, glucose).

[b]Publication described protocol but not outcomes of study.

Table 1

Study Characteristics

Reference	Participants[a]	Mean age (years + SD) or range	Setting	Lifestyle component s addressed	Behavior change strategies reported	Frequency; modality; duration
RCTs						
Davis et al. (2007)	N=23 Females; BMI ≥85th percentile	14.2±1.7	*Group 1:* Home *Group 2:* Clinic	Nutrition	*All:* CD, GS, IE, NE, PI *Group 2*: +MI	Weekly 90 minute sessions; Individual (home) or group (clinic) sessions; 12 weeks
Davis et al. (2009a)	N=54 Grade 9–12; BMI ≥85th percentile	15.5±1.0	Clinic	*Group 1*: Nutrition *Group 2*: Nutrition, Physical activity	*Group 1*: NE, MI, FC *Group 2:* NE, MI, FC, ST	Weekly 90 minute sessions; Group; 16 weeks *Group 2*: +two 60 minute sessions per week
Davis et al. (2009b)	N=41 Females grade 9–12; BMI ≥85th percentile	15.2±1.1	Clinic	*Group 1*: Nutrition *Groups 2 – 3*: Nutrition, Physical activity	*Group 1:* NE+MI; *Group 2:* NE+MI+ST; *Group 3:* NE+MI+ST + aerobic PA	Weekly 90 minute sessions; Group; 16 weeks *Groups 2, 3:* +Two 60 minute PA sessions per week
Davis et al. (2011)	N=38 Females aged 14–18 years; BMI ≥85th percentile	15.8±1.1	Clinic	Physical activity	*Group 1:* ST+aerobic PA; *Group 2:* ST+aerobic PA +MI	Weekly 60–90 minute sessions; Group; 16 weeks *Group 2:* + Eight individual and eight group MI sessions
Foster et al. (2010)	N=4,603 Sixth grade at baseline; 54% Hispanic; 50% BMI ≥85th percentile	11.3±0.6	School	Nutrition, Physical activity	GS, NE, PA, PAE, SE, PI, SM	Weekly educational and PA sessions of varying length; Group; 2.5 years (sixth–eighthgrade)
Kelly et al. (2015)	N=26 Males grade 9–12; with BMI >=95th percentile	15.48±0.2	Home	Physical activity	GS, MI, PAE, ST	Two weekly 1 hour sessions + 1 monthly personal training session + weekly phone calls; Individual; 16 weeks
Patrick et al. (2013)	N=101 Age 12–16 years; with Internet access; 74.3% Hispanic; High-risk for diabetes[b]	Range: 12–16	Online/mobile	Nutrition, Physical activity	GS, NE, PAE, PI, SE	Three intervention arms: *Group 1*: website + weekly emails *Group 2*: website + monthly 90 minute groups, two monthly phone calls *Group 3*: website + weekly texts
Rosenbaum et al. (2007)	N=73 Dominican Republic background	13.6±0.2	School	Nutrition, Physical activity	DE, NE, PA, PAE	Weekly 45 minute sessions; Group; 14 weeks
Shaibi et al. (2006)	N=22 Males; BMI ≥85th percentile; Tanner stage ≥3	15.1±0.5	Community	Physical activity	ST	Two weekly 60 minute sessions; Individual;

Reference	Participants[a]	Mean age (years + SD) or range	Setting	Lifestyle component s addressed	Behavior change strategies reported	Frequency; modality; duration
						16 weeks
Treviño et al. (2004)	N=1,419 Fourth graders; 80% Mexican-American	9.7±0.5	School	Nutrition, Physical activity	CD, DE, GS, NE, PA, PI, SE	Daily (5 days/week); 50 group sessions over 7 months
Weigensberg et al. (2014)	N=35 Grades 9–12; BMI ≥95th percentile	Range: 14–17	Clinic	Nutrition, Physical activity	NE, IE, PAE, PA, SE + guided imagery	Weekly; 90 minute group sessions; 12 weeks
Uncontrolled trials						
Coleman et al. (2010)	N=62 At risk for T2DM (family history of T2DM and/or BMI ≥85th percentile)	7.5±3.1	School	Nutrition, Physical activity	CD, DE, NE, PA, PI	Weekly 90 minute sessions; Group; 10 weeks
Shaibi et al. (2010)	N=102[c] BMI ≥85th percentile	11.8±3.0	Clinic	Nutrition, Physical activity	CC, DE, GS, NE, PA, SE	NR; Group; NR
Shaibi et al. (2012)	N=15 Age 14–16 years; BMI ≥85th percentile	15.0±0.9	Community	Nutrition, Physical activity	CC, CD, DE, NE, PI, SE, PA	Weekly; Lifestyle education group class + three 60 minute PA sessions; 12 weeks
Van der Heijden et al. (2010)	N=29 Sedentary[d]; 15 obese (BMI ≥95th percentile), 14 lean (BMI <85th percentile)	15.6±0.4 (obese) 15.1±0.3 (lean)	Clinic	Physical activity	Aerobic PA	Two 30 minute group sessions + two 30 minute individual sessions per week; 12 weeks

[a]Hispanic heritage/nationality of participants (e.g., Mexican, Puerto Rican) is listed if reported in the publication.

[b]As defined by the American Diabetes Association expert consensus panel, i.e., overweight (BMI >85th percentile for age and sex, weight and height >85th percentile, or weight >120% of ideal for height) plus any two of the following risk factors: family history of T2DM in a first- or second-degree relative, race/ethnicity (American Indian, African-American, Hispanic, Asian/Pacific Islander), or signs of insulin resistance (acanthosis nigricans, hypertension, dyslipidemia, polycystic ovary syndrome).

[c]Only 50 with outcome data

[d]<45 minute light to moderate PA/week.

NA, not assessed; NR, assessed, but not reported; CC, cultural constructs related to health; CD, cooking demonstrations/recipes/meal planning; DE, diabetes education; FC, financial compensation/gift cards; GS, goal setting; IE, intuitive eating; MI, motivational interviewing; NE, nutrition education/counseling; PA, physical activity; PAE, physical activity education; PI, parental involvement; SE, self-esteem/self-efficacy building; SM, social marketing; ST, strength training

Table 2

Raw Mean Differences and Hedges' g Effect Sizes[a] for Clinical Outcomes

Study	BMI (kg/m^2)	Effect size, *g* (95% CI)	BMI *z*-score	Effect size, *g* (95% CI)
Adiposity				
Intervention versus comparison/control[b]				
Davis et al. (2007)	+1.2	0.16 (−0.66, 0.98)	+0.1	0.21 (−0.61, 1.03)
Davis et al. (2009a)	−1.7 (N) +1.0 (N+ST)	−0.23 (−0.89, 0.42) 0.13 (−0.55, 0.12)	−0.1 (N) +0.1 (N+ST)	−0.18 (−0.83, 0.47) 0.18 (−0.51, 0.86)
Davis et al. (2009b)	**−4.6%** (N+CAST vs N+ST)[c]	–	**−7.7%** (N+CAST vs N+ST)[c]	–
Davis et al. (2011)	NS	–	NS	–
Foster et al. (2010)	NR	–	**−0.01**	-0.01 (−0.07, 0.05)
Kelly et al. (2015)	+3.8	0.74 (−0.05, 1.54)	NR	–
Patrick et al. (2013)	NR	–	−0.1 (W) −0.2 (WG) −0.1 (WSMS)	−0.22 (−0.77, 0.33) −0.44 (−0.99, 0.12) −0.22 (−0.78, 0.34)
Rosenbaum et al. (2007)	−0.8	−0.48 (−0.98, 0.01)	NR	–
Shaibi et al. (2006)	−2.2	−1.17 (−2.07, −0.26)	NR	–
Trevino et al. (2004)	NR	–	NR	–
Weigensberg et al. (2014)	NR	–	NR	–
Pre versus post intervention				
Davis et al. (2007)	−0.1 (Group-based) −0.1 (Home-based)	−0.01 (0.41, − 0.81) − 0.02 (0.43, − 0.85)	**−0.1[e] (Group-based)** **−0.1[e] (Home-based)**	−0.24 (−1.04, 0.56) −0.20 (−1.03, 0.65)
Davis et al. (2009a)	−0.1 (N) 0.0 (N+ST)	−0.02 (−0.62, 0.59) 0.00 (−0.67, 0.67)	0.0 (N) 0.0 (N+ST)	0.00 (−0.60, 0.60) 0.00 (−0.67, 0.67)
Davis et al. (2009b)	+0.3 (N) +1.1 (N+ST) −0.5 (N+CAST)	– – –	+0.02 (N) +0.08 (N+ST) −0.05 (N+CAST)	– – –
Davis et al (2011)	NR	–	NR	–
Foster et al. (2010)	NR	–	−0.05	−0.05 (−0.11, 0.01)
Kelly et al (2015)	+1.3	0.22 (−0.55, 0.99)	NR	–
Patrick et al. (2013)	NR	–	−0.1 (W) −0.2 (WG) −0.1 (WSMS)	−0.24 (−0.79, 0.30) −0.49 (−1.04, 0.06) −0.24 (−0.81, 0.32)
Rosenbaum et al. (2007)	**−0.7**	−0.48 (−0.88, −0.08)	NR	–
Shaibi et al. (2006)	+0.3	0.18 (−0.66, 1.02)	NR	–
Trevino et al. (2004)	NR	–	NR	–
Coleman et al. (2010)	−0.2	−0.04 (−0.39, 0.32)	+0.01	0.01 (−0.34, 0.36)
Shaibi et al. (2010)	**−1.1**	−2.18 (−2.68, −1.69)	NR	–
Shaibi et al. (2012)	−0.5	−0.29 (−1.01, 0.42)	**−0.1**	−0.62 (−1.35, 0.12)
Van der Heijden et al. (2010)	−0.3 (Obese youth) +0.1 (Lean youth)	−0.27 (−0.98, 0.45) 0.12 (−0.62, 0.86)	NR NR	– –
Weigensberg et al. (2014)	+0.8	0.13 (−0.59, 0.84)	+0.04	0.10 (−0.62, 0.81)
Glucose regulation	**Fasting glucose (mg/dL)**	**Effect size, *g* (95% CI)**	**Fasting insulin (μU/mL)**	**Effect size, *g* (95% CI)**

Study	BMI (kg/m^2)	Effect size, *g* (95% CI)	BMI *z*-score	Effect size, *g* (95% CI)
Intervention versus control group				
Davis et al. (2007)	−0.9	−0.14 (−0.96, 0.68)	+2.6	0.33 (−0.49, 1.15)
Davis et al. (2009a)	+2.7 (N) −0.2 (N+ST)	0.37 (−0.29, 1.02) −0.02 (−0.71, 0.66)	−1.8 (N) +1.7 (N+ST)	−0.10 (−0.75, 0.55) 0.10 (−0.58, 0.78)
Davis et al. (2009b)	**−10.3%** (N+CAST vs N)[c]	–	NS	
Davis et al. (2011)	NS	–	**24% decrease vs 6% increase** (CT vs control)[c]	–
Foster et al. (2010)	−0.08	−0.10 (−0.15, −0.04)	**−0.5**	−0.03 (−0.09, 0.02)
Kelly et al. (2015)	**+2.4**	0.44 (−0.34, 1.22)	NR	–
Patrick et al. (2013)	NR	–	NR	–
Rosenbaum et al. (2007)	0	0.00 (−0.49, 0.49)	+1.0	0.33 (−0.75, 0.55)
Shaibi et al. (2006)	+2.1	1.03 (0.12, 1.95)	−7.6	−2.81 (−4.02, −1.60)
Trevino et al. (2004)	**−2.5**	−0.25 (−0.36, −0.15)	NR	–
Weigensberg et al. (2014)	NR	–	NR	–
Pre versus post intervention				
Davis et al. (2007)	+0.9 (Home-based) −3.6 (Group-based)	0.10 (−0.73, 0.94) −0.65 (−1.47, 0.17)	−3.3 (Home-based) 0.0 (Group-based)	−0.40 (−1.25, 0.44) 0.00 (−0.80, 0.80)
Davis et al (2009a)	−0.8 (N) −2.4 (N+ST)	−0.12 (−0.73, 0.48) −0.29 (−0.97, 0.38)	−1.8 (N) −3.9 (N+ST)	−0.11 (−0.72, 0.49) −0.30 (−0.98, 0.37)
Davis et al. (2009b)	+2.5 (N) −3.6 (N+ST) − 4.3 (N+CAST)	– – –	+4.5 (N) −0.8 (N+ST) −0.5 (N+CAST)	– – –
Davis et al. (2011)	NR	–	NR	–
Foster et al. (2010)	0	0.00 (−0.06, 0.06)	**+3.80**	0.29 (0.23, 0.35)
Kelly et al. (2015)	+4.4	0.67 (−0.12, 1.46)	NR	–
Patrick et al. (2013)	NR	–	NR	–
Coleman et al. (2010)	NR	–	NR	–
Rosenbaum et al. (2007)	−1.0	−0.99 (−1.41, −0.57)	−1.0	−0.33 (−0.73, 0.07)
Shaibi et al. (2006)	+2.5	1.30 (0.38, 2.22)	−1.3	−0.54 (−1.39, 0.31)
Trevino et al. (2004)	−0.2	−0.02 (−0.13, 0.08)	NR	–
Shaibi et al. (2010)	−0.6	−0.50 (−0.89, −0.10)	**−5.9**	−2.53 (−3.05, −2.00)
Shaibi et al. (2012)	NR	–	NR	–
Van der Heijden et al. (2010)	0.0 (Obese youth) −1.0 (Lean youth)	0.00 (−0.72, 0.72) −0.97 (−1.75, −0.19)	**−3.6** (Obese youth) −0.6 (Lean youth)	−1.37 (−2.17, −0.58) −0.65 (−1.41, 0.11)
Weigensberg et al. (2014)	NR	–	NR	–

Notes: Boldface indicates statistical significance (p<0.05) as reported by study authors.

[a]Effect sizes were calculated if publication provided sufficient data (e.g., group means, SDs.

[b]All comparisons are between specified group and control group unless otherwise noted.

[c]Percentage reported because publication did not provide raw data.

CAST, combined aerobic and strength training; N, nutrition education only; NR, not reported; NS, not significant and data not reported; N+ST, nutrition education plus strength training; W, website-only intervention group; WG, website + group meetings; WSMS, website + text content

Table 3

Physical Activity and Dietary Behavior Outcomes of Diabetes Prevention Interventions for U.S. Hispanic Youth

Reference	PA outcome; measure	PA findings	Dietary outcome; measure	Dietary findings
RCTs				
Davis et al. (2007)	None	N/A	Dietary intake; 3-day diet records[a]	**Reduced intake of added sugar (33%), sugary beverages (66%), refined carbohydrates (35%), and total energy (22%); increased dietary fiber (46%) in both group and individual format**
Davis et al. (2009a)	None	N/A	Dietary intake; 3-day dietary records[a]	**N+ST group: reduced total energy intake (20%), carbohydrate intake (18%), and dietary fat intake (24%) compared to controls**
Davis et al. (2009b)	Light and MVPA; 7-day ActiGraph accelerometer wear, 3-DPAR	No significant changes	Dietary intake; 3-day diet records[a]	**Group (1): significant reduction in added sugar (38.7%); Group (3): significant reductions in total sugar consumed (45.7%)**
Davis et al. (2011)	Light and MVPA; 7-day ActiGraph accelerometer wear	No significant changes	Dietary intake; 3-day diet records[a]	No significant changes
Foster et al. (2010)	None	N/A	None	N/A
Kelly et al. (2015)	None	N/A	None	N/A
Patrick et al. (2013)	PA; 7-day PA recall interview	No significant changes	Dietary intake; Self-administered food frequency questionnaire	**Group (2): increased fruit and vegetable intake compared to controls**
Rosenbaum et al. (2007)	None	N/A	None	N/A
Shaibi et al. (2006)	None	N/A	None	N/A
Trevino et al. (2004)	None	N/A	Dietary fiber % kcal from saturated fat; three 24-hr dietary recalls administered by staff	**Increase in dietary fiber consumed compared to controls**
Weigensberg et al. (2014)	MVPA; 3-DPAR	**Moderate PA increased by 29% compared to controls**	Dietary intake; 3-day diet records[a]	Decrease in total energy intake compared to controls
Uncontrolled trials				
Coleman et al. (2010)	None	N/A	None	N/A
Shaibi et al. (2010)	None	N/A	None	N/A
Shaibi et al. (2012)	MVPA; 3-DPAR	Daily MVPA increase of 26.1%; 67% of sample (post) versus 47% (pre) met CDC recommendations of 60 minutes MVPA per day	Dietary intake; Brief Dietary Assessment Tool for Hispanics	**Reduction in servings of fat consumed per day from 3.3 (±0.3) to 2.0 (±0.2)**

Reference	PA outcome; measure	PA findings	Dietary outcome; measure	Dietary findings
Van der Heijden et al. (2010)	None	N/A	None	N/A

Notes: Boldface indicates statistical significance (p<0.05).

[a] 3-day diet recalls were self-administered and then clarified by study staff.

3DPAR, 3-Day Physical Activity Recall questionnaire; Kcal, kilocalories; MVPA, moderate to vigorous physical activity; PA, physical activity, N/A, not applicable