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State-of-the-Art Review

Health disparities in cardiometabolic risk among Black and Hispanic youth in the United States

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ABSTRACT

Cardiometabolic risk factors in children and adolescents track into adulthood and are associated with increased risk of atherosclerotic cardiovascular disease. The purpose of this review is to examine the pervasive race and ethnic disparities in cardiometabolic risk factors among Black and Hispanic youth in the United States. We focus on three traditional cardiometabolic risk factors (obesity, type 2 diabetes mellitus, and dyslipidemia) as well as on the emerging cardiometabolic risk factor of non-alcoholic fatty liver disease. Additionally, we highlight interventions aimed at improving cardiometabolic health among these minority pediatric populations. Finally, we advocate for continued research on effective prevention strategies to reduce cardiometabolic risk and avert further disparities in cardiovascular morbidity and mortality.

Introduction

Atherosclerotic cardiovascular disease (CVD) is the leading cause of morbidity and mortality in the United States [1]. Cardiometabolic risk factors such as obesity, diabetes, dyslipidemia, hypertension, and non-alcoholic fatty liver disease in childhood have been associated with subclinical measures of atherosclerosis in early life [2]. Moreover, multiple studies have shown that risk factors established in childhood tend to track into adulthood [3], leading to increased risk of CVD in adulthood [4]. Thus, the increasing prevalence of cardiometabolic risk factors in children and adolescents has emerged as a public health problem in the United States.

Given the high levels of cardiovascular morbidity and mortality in adult minority populations, pediatric ethnic disparities in cardiometabolic risk factors are of critical importance for primordial prevention [5]. Hypertension is a well-known risk factor for CVD with significant disparities by race and ethnicity in youth [6–8] but space limitations prevent us from discussing this important issue. This review examines the prevalence and impact of obesity, diabetes, dyslipidemia, and non-alcoholic fatty liver disease among minority children in the US and highlights potential areas for further research and intervention. We particularly focus on non-Hispanic Black (hereafter referred to as Black) and Hispanic/Latinx (hereafter referred to as Hispanic) populations compared to non-Hispanic White (hereafter referred to as White) populations, where data is most robust. We use terminology favored by the US Census Bureau, acknowledging the limitations of this terminology to fully capture race and ethnicity information.

Obesity

The rising obesity epidemic in the United States has been documented by sequential National Center for Health Statistics surveys. The National Health Examination Survey II (NHES II), conducted from 1963 to 1965, was the first of these to include children. According to these assessments, the prevalence of overall childhood obesity was stable at approximately 5.4% until the 1980s [9], when it began to rise to epidemic proportions reaching the current 18.5% [10]. For Black and Hispanic populations, the trend is not as clear. Black and Hispanic children were first included in NHES II (1963–1965) and NHES III (1966–1970) respectively [11]. Even when the National Nutrition Surveillance System was combined with NHES to create the National Health and Nutrition Examination Survey (NHANES) in the 1970s, sufficient data was not available for many of the ethnic groups in the US [11]. The Hispanic Health and Nutrition Examination Survey was conducted from 1982 to 1984 to address this knowledge gap [11]. Starting with NHANES III (1988–1994), Black and Hispanic children were surveyed in large proportions that allow for robust comparisons [11].

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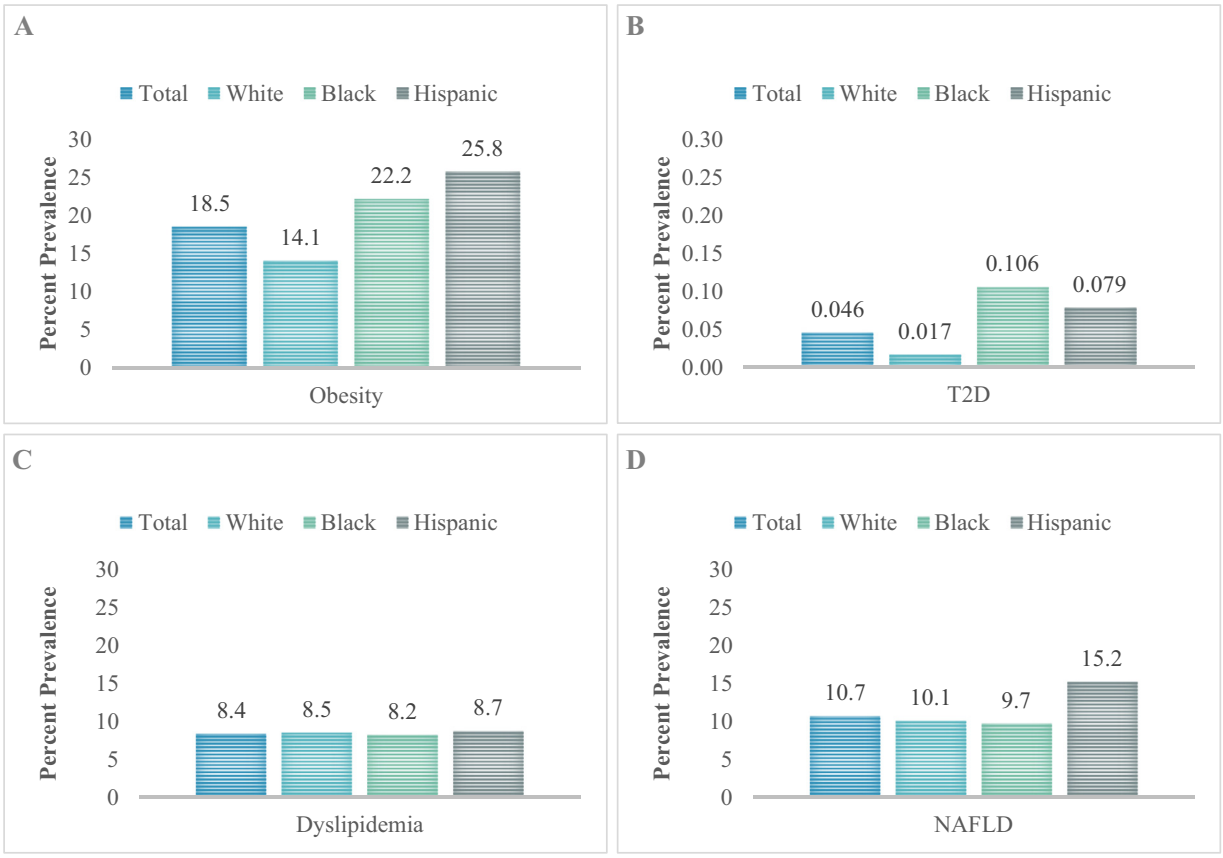

Fig. 1. Prevalence of obesity (A), type 2 diabetes (B), dyslipidemia* (C), and non-alcoholic fatty liver disease (D) among children and adolescents by race and Hispanic origin in the United States.
*Dyslipidemia is defined as high non-HDL-C.
NAFLD=non-alcoholic fatty liver disease; non-HDL-C= non high-density lipoprotein cholesterol; T2D=Type 2 diabetes mellitus.

NHANES III revealed stark ethnic disparities in body mass index (BMI) and other cardiometabolic risk factors after accounting for socioeconomic status and age. BMI levels were significantly higher among Black and Hispanic girls compared with their White counterparts [12]. These differences were evident by age 6–9 and only widened with increasing age [12]. Statistically significant differences were not observed in boys [12]. In an updated analysis conducted in 2016, Black youth (Prevalence, 19.5% [95% CI, 17.1–22.2%]; OR, 1.34 [95% CI, 1.03–1.75]) and Hispanic youth (Prevalence, 21.9% [95% CI, 20.0–23.9%]; OR, 1.48 [95% CI, 1.23–1.78]) had a higher prevalence and higher odds of obesity, compared with White children and adolescents (Prevalence, 14.7%; 95% CI, 12.3–17.3%) [13]. The latest NHANES analysis using representative data from 1999 to 2016 shows that substantial disparities by race and ethnicity remain among both girls and boys across all age groups, particularly at extreme weight categories [10]. Current estimates are that 25.8% of Hispanic and 22.2% of Black children and adolescents have obesity [10]. In contrast, White and Asian children and adolescents have much lower obesity prevalence at 14.1% and 10.7%, respectively [10] (**Fig. 1**).

Type 2 diabetes mellitus

Previously a rare occurrence until adulthood, type 2 diabetes mellitus (T2D) has been steadily increasing among US youth over the past three decades [14], particularly among youth of racial/ethnic minority groups [15]. As in adults, the pathophysiology of youth-onset T2D involves both insulin resistance and inadequate insulin secretion [16]. However, pancreatic β-cell function decompensation appears to occur more rapidly in youth compared to adults resulting in faster loss of glycemic control [16]. Additionally, youth and adults appear to respond differently to therapy as evidenced by the Restoring Insulin Secretion (RISE) Pediatric and Adult Medication Studies. Despite utilizing the same approaches, β-cell function in youth deteriorated during treatment, whereas it improved in adults [17]. Puberty is a high-risk time for T2D development due to a physiologic reduction in insulin sensitivity, estimated as approximately 50% decrease in healthy youth with normal weight [18]. While usually transient, this reduction in insulin sensitivity may be exacerbated by overweight and obesity, which are observed in youth-onset T2D [19,20]. The compensatory increase in insulin secretion that accompanies decreased insulin sensitivity in adolescence [18] may be inadequate in youth with limited β-cell function, ultimately leading to T2D [16].

In addition to the more rapid decline in β-cell function, youth-onset T2D is particularly worrisome due to an accelerated buildup of comorbidities compared to adult-onset T2D. This is especially alarming given that the increased risk of disability and death from cardiovascular disease begins to occur 10–15 years after onset of disease [21]. High cardiovascular disease risk in T2D is related to both the effects on comorbidities such as hypertension and dyslipidemia as well as other direct macrovascular effects such as impaired endothelial function and increased low-grade inflammation. As the only randomized trial to date that has combined lifestyle and pharmacologic therapy for youth with T2D, the Treatment Options for type 2 Diabetes in Adolescents and Youth (TODAY) study provides a glimpse of the progression of disease and appearance of complications in this population. In this study, 699 youth 10–17 years of age, with mean age at entry of 14 years, participated for an average of 3.9 years [22] (**Table 1**). The primary outcome of treatment failure was defined as elevated glycated hemoglobin level

(≥8%) over a period of 6 months or persistent metabolic decompensation. With a median time of 11.5 months to treatment failure, 45.6% of participants reached the primary outcome. Hypertension prevalence increased from 11.6% at baseline to 33.8% at the end of the study. The prevalence of LDL-C ≥ 130 mg/dL or usage of lipid-lowering medication rose from 4.5% at baseline to 10.7% at the end of the study [23]. Based on the natural history of adult-onset T2D, these findings suggest that youth with diabetes may experience cardiovascular disease and other complications in their third and fourth decades of life [23]. A computer model simulation similarly predicts that youth with T2D may experience complications by their 40s and lose approximately 15 years from average remaining life expectancy even with treatment [24]. Additionally, increasing evidence suggests that the risk for CVD events increases even prior to the onset of overt clinical diabetes [25].

Adolescents from racial/ethnic minority groups have the highest prevalence of T2D. In parallel with obesity trends in the 1990s, NHANES III revealed higher levels of glycated hemoglobin in Black and Hispanic children compared to White children by ages 6–9[12]. Nationwide comparative data is limited for ethnic minorities that make up smaller segments of the population, limiting this review to Black and Hispanic youth. However, T2D is well-known as a major public health problem among American Indian youth. A study from the Indian Health Service outpatient database showed a 46% increase in T2D prevalence in the 1990s [26]. The SEARCH for Diabetes in Youth (SEARCH) study is an ongoing population-based registry of diabetes with surveillance of almost 70 million youth in five US catchment sites. The latest SEARCH prevalence estimates in 2009 demonstrate striking disparities for American Indian and other minority youth. The overall prevalence of T2D among youth aged 10–19 years old is 0.046%. American Indian youth had a prevalence of 0.12% [27]. Black and Hispanic youth followed closely with a prevalence of 0.106% and 0.079%, respectively, compared with 0.034% and 0.017% for Asian and White youth, respectively (**Fig. 1**). Incident T2D is also increasing at alarming rates in US youth with disproportionate effects in American Indian, Black, and Hispanic youth. The most recent SEARCH incidence publication in 2012 shows that minority youth aged 10–19 years old have the highest numbers of new cases with an incidence of 46.5 per 100,000 among American Indians, 32.6 among Blacks, and 18.2 among Hispanics, versus a much lower rate of 3.9 among Whites [15]. Furthermore, statistically significant incidence increases from 2002 to 2012 were observed across all race and ethnic groups except among White youth [15]. These findings portend a growing burden of T2D that will not be shared equitably and support an urgent need for aggressive prevention and treatment of T2D in vulnerable pediatric populations.

Dyslipidemia

Dyslipidemia is another important cardiometabolic risk factor that disproportionately affects minority children in the US. Seminal investigations such as the Bogalusa Heart Study [28] and the Pathological Determinants of Atherosclerosis in Youth (PDAY) Study [29] indicate that abnormalities in non-HDL-C levels often begin in childhood and translate into early atherosclerotic lesions. Additionally, low HDL-C is an established marker for long-term CVD risk. Thus, identifying youth with abnormal lipid profiles and treating them appropriately may reduce the risk of premature cardiovascular disease. Indeed, the Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents and the National Heart, Lung, and Blood Institute (NHLBI) released new normative values in 2011 and recommended universal screening for youth aged 9–11 and 17–21 years [30] endorsed by the American Academy of Pediatrics.

NHANES data indicate an overall favorable trend of serum lipids from 1988 to 2014 among children in the US, including reductions in LDL-C, triglycerides, non-HDL-C, and total cholesterol [8,31,32]. The latest NHANES analysis encompassing data through 2016, recapitulates the favorable trends in pediatric lipid levels in the US [33]. These trends were directionally consistent across ethnic groups but varied in magnitude and statistical significance [33]. For example, Black youth had no significant reductions in non-HDL-C, triglycerides, or LDL-C, and Hispanic youth had no significant reductions in triglycerides or LDL-C [33]. White youth also had no significant reductions in LDL-C [33]. Despite the generally favorable trends in lipid profiles among youth, the overall prevalence of dyslipidemia defined as abnormal levels of lipids or apolipoprotein B remains substantial at 19–25% [33]. The overall prevalence of high non-HDL-C in youth is 8.4%, with no statistically significant differences by race and ethnicity (**Fig. 1**).

Adherence to dyslipidemia universal screening has not yet been studied at a nationally-representative level since the 2011 guidelines were implemented. Estimates of adherence to screening guidelines vary widely. A recent study in Texas reported screening rates of 20% of pediatric patients aged 9–11 years [34]. Another study in Illinois stratified children by guideline-defined dyslipidemia risk and revealed that only 56% of high-risk patients and 6% of non-high risk patients received lipid screening by age 12 [35]. An investigation of five sites in the Cardiovascular Research Network with more than 200,000 privately-insured individuals aged 2–20 years estimated that 11% of youth were tested for lipids in 2012 [36]. An analysis conducted in Utah showed that only 3.5% of 9–11 year-olds were screened during the study period with no significant differences before and after the guidelines were implemented [37]. Factors associated with higher likelihood of lipid testing included non-White race, Hispanic ethnicity, overweight, and obesity [37]. The higher rate of screening in minority children might result from higher rates of obesity and other comorbidities known to adversely impact lipid profiles.

Non-alcoholic fatty liver disease

Increasingly considered a multisystem disease, non-alcoholic fatty liver disease (NAFLD) is an emerging cardiometabolic risk factor in pediatrics. NAFLD is characterized by the presence of macrovesicular steatosis infiltrating >5% of the liver in the absence of excessive alcohol intake, medications, infections, or autoimmune processes [38]. NAFLD ranges from simple steatosis to non-alcoholic steatohepatitis (NASH) with fibrosis and may progress to hepatocellular carcinoma [38]. While NAFLD is associated with many other cardiometabolic risk factors such as obesity, dyslipidemia, and T2D, research suggests that NAFLD also independently increases risk of cardiovascular disease. Indeed, cardiovascular disease is the primary cause of premature morbidity and mortality in patients with NAFLD [39,40]. While research on pediatric NAFLD is limited, the disease appears to be more aggressive in children compared to adults [41]. Thus, the North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition (NASPGHAN) released guidelines in 2017 endorsed by the American Academy of Pediatrics that recommend screening children 9–11 years of age who are obese or who are overweight with additional risk factors [42].

NAFLD is a rapidly emerging disease in pediatrics. A study using data from a large healthcare system revealed that incidence of NAFLD increased from 36 per 100,000 in 2009 to 58.2 per 100,000 in 2018 ($p<0.0001$) [43]. Recent estimates of the percentage of the US pediatric population affected by NAFLD range from 4.5% in an autopsy-based study of youth aged 2–19 years [44] to 10.7% in an NHANES study of adolescents aged 12–19 years [45]. Prevalence is highly [45], though not exclusively [46], associated with obesity and rises to 30–40% among youth with obesity [45,47]. US population-based studies reveal that Hispanic children have the highest prevalence of NAFLD [45,48], and also exhibit worse disease prognosis [48]. The pediatric NAFLD population is estimated as 15.2% Hispanic, 10.1% White, and 9.7% Black [45] (**Fig. 1**). In a California study of pediatric hospitalizations for liver disease, Hispanic children made up about 60% of NAFLD hospitalizations despite representing only about 50% of the state's pediatric population [49]. Notably, Black children have the lowest prevalence despite multiple risk factors and comorbidities [48].

Like with other cardiometabolic risk factors in children, the development of NAFLD is influenced by environmental and genetic factors [50]. Genetic risk for childhood NAFLD has been shown to vary by race and ethnicity, with Hispanics and Whites having higher genetic risk scores compared to other groups [51]. A *PNPLA3* variant has emerged as an important genetic risk factor for NAFLD in Hispanic children, especially those with indigenous ancestry [52]. The I148M variant is associated with increased liver fat and inflammation. Further research is needed to elucidate the genetic basis of NAFLD in general and particularly in minority ethnic groups. However, environmental and lifestyle risk factors must be emphasized in research and clinical efforts against NAFLD, given that these explain hepatic fat variation by race and ethnicity far more than genetic factors do.

The roots of racial/ethnic disparities

Racial/ethnic disparities in obesity and associated cardiometabolic risk factors are attributed to multiple factors ranging from cultural and language barriers to socioeconomic differences and unequal access to health insurance and healthcare facilities. Decreasing these barriers necessitates better characterization of the built environment [53], which encompasses neighborhood characteristics such as availability of grocery stores in comparison to fast food restaurants. In the first study of its kind, Williams et al. recently combined epidemiologic and census data to investigate the relationship between census-tract poverty level and cardiometabolic health by race and ethnicity. Their findings indicate that adolescents in the highest poverty census tracts have higher cardiometabolic dysfunction, independent of their families' socioeconomic status. This held true for White and Hispanic but not for Black youth [54]. A posthoc analysis shows that high-income Black families reside in higher poverty areas compared to White families of similar income. These data highlight key health consequences of the structural racism that fosters segregation in the US. As has been described before, the same level of socioeconomic status does not confer the same social, economic, and health benefit to ethnic minority youth as to White youth [12]. The COVID-19 pandemic will likely further exacerbate these disparities as minority families disproportionately face economic hardship and the consequent decreased access to healthy food and physical activity opportunities [55].

Multilevel, multicomponent interventions to address disparities

Decades of research on cardiovascular disease prevention have led to increasing consensus that interventions including multiple components across various settings are more effective than single-component interventions [56]. This concept takes into account the fact that the presence of cardiometabolic risk factors and disparities are both influenced by behavioral, socioeconomic, and environmental factors. Therefore, interventions addressing some or all of these aspects holistically are more likely to be successful. Additionally, these interventions can inform public health policy changes to address cardiometabolic risk disparities at the population level [57]. Some of the important interventions that have shown success in diverse pediatric populations are highlighted below and in **Table 1**.

Multidimensional and primordial prevention strategies may reduce cardiometabolic risk factors in minority children. The Family-Based Approach in a Minority Community Integrating Systems-Biology for Promotion of Health (FAMILIA) trial (NCT02343341) was a preschool-based intervention to instill healthy behaviors in children belonging to a diverse and socioeconomically disadvantaged community with the goal of reducing future cardiovascular disease [58]. The children, who were 54% Hispanic and 37% Black, in 15 Head Start preschools in Harlem, New York were cluster-randomized to a 4-month educational intervention or their standard curriculum. The intervened 3–5 year olds had a statistically significant increase in their overall knowledge, attitudes, and habits score, an early impact that may cause a ripple effect of benefits in the children's future cardiovascular health.

Another strategy to reduce cardiometabolic risk factors in children involves merging high-quality clinical care and links to community resources. The Connect for Health trial (NCT02124460) randomized children with obesity to two clinical-community interventions [59]. The study population was 33.3% Black and 21.8% Hispanic. There was no statistically significant difference between the two interventions, but children in both groups had a statistically significant reduction in BMI *z*-scores at 1 year after baseline. While the magnitude of effect on BMI was modest, post-hoc analysis revealed an upward trend in *z*-scores in the year prior to enrollment. As the authors explain, this may signify an arrest of excess BMI gain but may also represent a regression to the mean. Both interventions also led to improvements in parental resource empowerment, a secondary outcome that reflects an important level of influence on pediatric patients.

Intervening at the family level in addition to clinical and community environments may also prove successful in obesity reduction among ethnically diverse children. The Bright Bodies randomized controlled trial (NCT00409422) examined a one-year intervention for inner-city impoverished children and their families [60]. The intervention, which was provided at school to address the limited transportation options of the participants, involved opportunities for exercise as well as nutrition and behavior modification education, with parents attending the nutrition component. The intervened children then returned to their usual obesity clinic appointments for the second year of the program. With one of the longest follow-up times in pediatric obesity trails, the intervention led to a statistically significant decrease in BMI *z*-scores compared to control that was sustained for 2 years [−0.16 units (95% CI: −0.23, −0.09); $p<0.0001$]. While clinical significance threshold for childhood obesity reduction has not been determined, some studies suggest that reductions of even 0.15–0.20 units are associated with healthier cardiometabolic profiles [61]. Treatment effect of the Bright Bodies intervention was also sustained at 2 years for other cardiometabolic outcomes such as total cholesterol, LDL-C, and homeostasis model assessment of insulin resistance.

Conclusion

There are pervasive and widening disparities in cardiometabolic risk factors among minority youth in the US. Further research is necessary to obtain a full epidemiologic picture of cardiometabolic risk factor disparities in youth, especially in relatively smaller minority groups such as American Indian and Asian children, who are typically underrepresented and undersampled in nationally-representative studies. Additionally, a more nuanced approach to the classification of minority populations is needed to capture their heterogeneity. For example, Hispanic is a term that characterizes people with diverse cultural traditions, geographic origin, historical background, and degrees of acculturation. Similarly, aggregating East and South East Asians as well as Pacific Islanders into a single group may lead to missing important disparities affecting some of these groups.

Most important, we need to move from simply cataloging disparities to addressing them. Because childhood cardiometabolic risk factors track into adulthood and are associated with cardiovascular events and deaths, the excessive rates of these cardiometabolic risk factors among minority youth threaten to negatively impact future morbidity and mortality rates. Acting now is imperative to prevent these dire consequences. Interventions to date have made modest improvements over short periods of time. There is an urgent need for a strong focus on the design, evaluation, and implementation of evidence-based, more aggressive, and more longitudinal cardiovascular disease prevention interventions among minority youth.

Table 1.
Interventions to reduce cardiometabolic risk factors in diverse pediatric populations.

Trial, year	#	Age in years	% Black	% Hispanic	Follow-up time	Significant findings
Bright Bodies 2011 [60]	174	8–12	39%	25%	2 years	1. Design: Parallel group, RCT with a 12-month extension phase involving children with BMI≥95th percentile in a pediatric obesity clinic in CT. 2. Intervention: 1-year intervention offered at school was tailored to inner-city, ethnically diverse populations and consisted of exercise twice and nutrition/behavior modification once per week (with caregivers attending the nutrition component) vs. usual care at the obesity clinic. 3. Outcomes: Treatment effect was sustained at 2 years in the intervention group vs. control group for BMI *z*-score [−0.16 units (95% CI: −0.23, −0.09); $p<0.0001$], total cholesterol [−13.0 mg/dL (95% CI: −21.7,−4.2); $p = 0.004$], low-density lipoprotein cholesterol [−10.4 mg/dL (95% CI: −18.3, −2.4); $p = 0.01$] and homeostasis model assessment of insulin resistance [−2.05 (95% CI: −2.48, −1.75); $p = 0.0001$].
TODAY 2015 [22]	699	10–17	33%	40%	2–6.5 years	1. Design: Multicenter, RCT for youth with recent-onset T2D and BMI≥85th percentile, comparing metformin monotherapy, metformin + rosiglitazone, and metformin + intensive lifestyle intervention program. 2. Intervention: The lifestyle intervention involved a family-based, behavioral approach to weight loss. 3. Primary outcome: Rate of glycemic control loss (defined as glycated hemoglobin level of at least 8% for 6 months or sustained metabolic decompensation requiring insulin) was 51.7% for metformin alone, 38.6% for metformin + rosiglitazone, and 46.6% for metformin + lifestyle intervention. Metformin + rosiglitazone was significantly superior to metformin alone ($p = 0.006$). 4. Secondary analysis: Treatment failure rates varied by race and ethnicity: 52.8% among Blacks, 45.0% among Hispanics, and 36.6% among Whites. Metformin alone was least effective in Black participants, with 66.2% reaching glycemic control loss compared to White (44.9%, $p = 0.01$) and Hispanic (44.0%, $p<0.001$) participants.
Connect for Health 2017 [59]	721	2–12	33%	22%	1 year	1. Design: Two-arm, blinded, RCT for children with BMI≥85th percentile involving 6 pediatric primary care practices in MA to support behavior change and linkage of families to neighborhood resources. 2. Intervention: 1-year intervention of enhanced primary care (flagging and clinical decision support tools for pediatric weight management, caregiver educational materials, a Neighborhood Resource Guide, and monthly text messages) plus contextually-tailored, individual health coaching vs. enhanced primary care alone. 3. Primary outcome: BMI *z*-score change was −0.09 units (95% CI: −0.13, −0.05) in the combined intervention and −0.06 (95% CI: −0.10, −0.02) in the enhanced primary care group. There was no statistically significant difference between the intervention arms (−0.02 units; $p = 0.39$). 4. Secondary outcome: Parental resource empowerment was improved in both interventions with no statistically significant difference between them. Parent-reported health-related quality of life was improved significantly in the intervention but not the control group.
FAMILIA 2019 [58]	562	3–5	37%	54%	5 months	1. Design: Cluster-RCT involving 15 Head Start preschools in NY to teach healthy diet, physical activity, body/heart awareness, and emotion management. 2. Intervention: 4-month (50 h) educational intervention for children and caregivers. Control: Standard curriculum. 3. Primary outcome: Change from baseline to 5 months in the overall knowledge, attitudes, and habits score was ~2.2 fold higher in the intervention group [difference of 2.86 points (95% CI: 0.58–5.14 points; $p = 0.014$]. The largest changes between intervention and control groups were identified in knowledge [difference of 1.62 points (95% CI: 0.41–2.82 points; $p = 0.009$)] and attitudes [difference of 1.66 points (95% CI: 0.26–3.05 points; p 0.020)] 4. Secondary outcomes: No significant differences in BMI *z*-score or Test of Emotion Comprehension score between groups.

Authorship statement

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Resources NA.
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Supervision FR JWK.
Validation NA.
Visualization SFK JWK.
Writing - original draft SFK.
Writing - review & editing SFK FR JWK.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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