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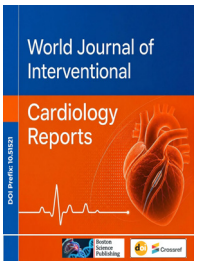

Early-Life Determinants of Cardiovascular Risk. The Impact of Nutrition, Socioeconomic Status, and Environmental Factors on Childhood Obesity and Hypertension

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A B S T R A C T

Background: Cardiovascular diseases remain the leading cause of death globally, and risk accumulation begins early in the life course. Childhood obesity is a major upstream driver of long term cardiometabolic risk, and recent surveillance confirms high and rising prevalence in both global and national contexts.

Objective: This paper examines how early life nutrition, socioeconomic status, and environmental exposures shape pathways to childhood obesity and pediatric hypertension, with a quantitative synthesis using nationally reported estimates to illustrate key gradients and links.

Methods: We developed an integrated life course conceptual framework grounded in DOHaD and social determinants theory, then conducted a secondary quantitative analysis using publicly reported NHANES based estimates from the National Center for Health Statistics and CDC reports. We extracted trend tables on obesity and severe obesity among U.S. youth ages 2 to 19, and guideline based estimates of hypertension among youth and its variation by weight status, sex, age, and race and ethnicity.

Results: U.S. obesity prevalence among ages 2 to 19 increased from 5.2% in 1971 to 1974 to 21.1% in August 2021 to August 2023, and severe obesity rose from 1.0% to 7.0% over the same horizon. In NHANES 2013 to 2016, youth hypertension prevalence under the 2017 AAP criteria was 4.11%, rising sharply with obesity, reaching 9.43% for obesity overall and 14.70% for severe obesity. Obesity prevalence also displayed a strong income gradient, ranging from 11.5% at above 350% of the federal poverty level to 25.8% at or below 130% of the federal poverty level.

Conclusion: Early life nutrition, socioeconomic disadvantage, and environmental conditions plausibly interact to increase childhood obesity and pediatric hypertension through shared behavioral, psychosocial, and biological pathways, and the obesity pathway appears central for hypertension risk concentration in youth.

Keywords: Childhood obesity. Hypertension. Early life determinants. Socioeconomic status. Nutrition. Environmental exposure. Cardiovascular risk. Life course epidemiology.

Introduction

Cardiovascular diseases account for a substantial share of global mortality, and the World Health Organization estimates 19.8 million CVD deaths in 2022, representing about 32% of all global deaths [1]. Because many cardiometabolic phenotypes track from childhood to adulthood, pediatric obesity and elevated blood pressure should be treated as early markers of cardiovascular risk accumulation rather than isolated pediatric conditions [2, 9]. In the United States, nationally representative estimates indicate that 21.1% of children and adolescents ages 2 to 19 had obesity in August 2021 to August 2023, and 7.0% met criteria for severe obesity [3]. The Centers for Disease Control and Prevention similarly summarizes that 19.7% of U.S. youth ages 2 to 19 had obesity in 2017 to March 2020, alongside clear disparities by age, race and ethnicity, and income [4].

Problem statement

Research and policy often treat nutrition, socioeconomic status, and environment as separate explanatory domains, which can obscure the reality that these exposures cluster and interact across early development

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[11]. In practice, socioeconomic disadvantage can constrain dietary options, intensify stress physiology, and elevate exposure to adverse environments, which together may amplify obesity and blood pressure risk in children [11]. At the same time, cardiovascular risk forecasts and prevention frameworks increasingly emphasize life course approaches, because upstream drivers like obesity and hypertension are likely to shape future adult burden [5].

Research aim and objectives:

The primary aim is to synthesize evidence and illustrate, with nationally reported estimates, how early life nutrition, socioeconomic context, and environmental exposures shape pathways to childhood obesity and pediatric hypertension as early determinants of cardiovascular risk [3, 4]. The specific objectives are listed below [3,4].

- To summarize life course mechanisms linking early nutrition, social position, and environments to pediatric adiposity and blood pressure phenotypes [6, 7].
- To quantify long term trends in U.S. childhood obesity and severe obesity using NHANES based NCHS Health E Stat estimates [3].
- To characterize pediatric hypertension prevalence under guideline based definitions and its gradient across weight status and demographic strata [9, 10].
- To evaluate how socioeconomic disadvantage relates to obesity prevalence and interpret implications for downstream hypertension risk concentration [4, 9].

Research questions and hypotheses:

RQ1. How have obesity and severe obesity prevalence changed over time among U.S. children and adolescents ages 2 to 19 [3]. RQ2. What is the prevalence of pediatric hypertension under contemporary pediatric guideline criteria, and how strongly does it vary by weight status [8,9]. RQ3 Does socioeconomic status plausibly modify nutrition related obesity pathways, and thereby contribute to differential hypertension risk through obesity mediated mechanisms [4, 11]. H1 Lower socioeconomic position is associated with higher childhood obesity prevalence, consistent with constrained resources and obesogenic environments [4, 11]. H2 Obesity substantially increases pediatric hypertension prevalence, making obesity a key mediator linking upstream determinants to blood pressure outcomes [9, 10].

Significance: Childhood obesity and pediatric hypertension affect millions of children, and their unequal distribution implies an equity sensitive prevention agenda [3, 4]. Because policy levers for nutrition, housing, school food environments, transportation, and neighborhood safety sit upstream of many individual behaviors, an integrated model can improve intervention design and targeting in a way that single factor approaches cannot [11, 17]. For Scopus indexed scholarship, this integrated framing supports stronger causal reasoning, clearer specification of mediators and moderators, and better alignment of analytic strategies with real world complexity [11].

Literature Review

Life course theory and DOHaD: The developmental origins of health and disease paradigm proposes that early exposures, including nutrition and environmental conditions, can program later cardiometabolic risk through effects on growth trajectories, endocrine systems, and epigenetic regulation [6, 7]. Barker's fetal origins hypothesis helped establish the idea that prenatal and early postnatal growth conditions can shape later cardiovascular risk, including hypertension and metabolic disease, through lifelong biological embedding [6]. Contemporary DOHaD frameworks further emphasize that these early influences operate jointly with postnatal environments and social contexts, and can act across generations through epigenetic mechanisms and intergenerational transmission of disadvantage [7, 11].

Nutrition and childhood obesity: Early feeding practices may influence obesity risk through appetite regulation, microbiome development, metabolic signaling, and learned food preferences [12]. A large review summarized on the National Academies site reports a 26% reduction in the odds of later obesity among individuals who were breastfed as infants across 105 studies, although effect sizes attenuate with stronger confounding control and in higher quality designs [12]. More recent pediatric obesity prevention literature also highlights plausible epigenetic and metabolic programming mechanisms through breastfeeding and early complementary feeding, while emphasizing that effects are modest compared with broader structural drivers [13]. In later childhood, high intake of energy dense diets and sugar sweetened beverages, along with high sodium patterns, are repeatedly implicated in obesity and elevated blood pressure risk, and guideline recommended dietary patterns such as DASH are commonly cited as beneficial for blood pressure control in youth [8, 9].

Ultra processed foods and diet quality: Ultra processed food exposure is increasingly used as a marker of dietary industrialization and low nutritional quality, with NOVA based approaches widely applied in population studies [14]. The National Cancer Institute provides guidance for applying NOVA to dietary recall systems, reflecting mainstream uptake of processing based dietary indicators in epidemiology [15]. A randomized trial focused systematic review in Frontiers reports that experimental exposure to ultra processed dietary patterns can adversely shift intermediate health outcomes, supporting biological plausibility beyond observational confounding, although trial designs vary and pediatric specific evidence remains more limited than adult evidence [16]. Despite ongoing debates about NOVA definitions, the broader evidence base supports diet quality, food environments, and processing as meaningful dimensions of cardiometabolic risk relevant to youth [14, 16].

Socioeconomic status and inequities: Social determinants research emphasizes that income, education, and social position shape health through material resources, stress exposures, neighborhood conditions, and access to health promoting opportunities, and these mechanisms unfold across lifetimes and generations [11]. Braveman and colleagues argue that upstream determinants fundamentally shape downstream behaviors, implying that individual nutrition interventions will have limited reach without structural change to resources and environments [11]. Consistent with this framework, CDC surveillance reports show obesity prevalence rises as family income decreases in U.S. children, highlighting the need to treat socioeconomic position as both a direct determinant and a modifier of nutrition and environment pathways [4].

Environmental factors: Environmental exposures relevant to childhood obesity and hypertension include ambient air pollution, neighborhood built environments, and exposure to social stressors that can influence physical activity and diet patterns [17]. Longitudinal evidence synthesized in a 39 study systematic review suggests that greenspace and recreational facilities may be protective for child weight trajectories, while greater crime and lower perceived safety can act as risk factors, though results vary by sex and context and measurement standardization remains a major challenge [17]. For cardiovascular physiology, the American Heart Association has published scientific statements describing plausible mechanistic pathways by which particulate matter contributes to cardiovascular risk through inflammation, oxidative stress, and autonomic imbalance, which can be relevant to blood pressure regulation throughout life [18]. Broader cardiovascular reviews similarly frame air pollution as a cardiovascular risk factor with population-level relevance, reinforcing the importance of environmental risk reduction within prevention strategies [19]. In addition, meta analytic evidence supports associations between air pollutants and childhood obesity and weight gain, providing a pathway by which environment can affect blood pressure partly through adiposity as well as through direct physiological mechanisms [20].

Childhood hypertension and its linkage to obesity: Pediatric hypertension definitions have evolved, and contemporary guidance emphasizes accurate measurement, age specific interpretation, and early identification given associations with later cardiovascular risk [8]. The CDC MMWR analysis of NHANES 2001 to 2016 reports that hypertension prevalence declined over time, yet the new 2017 pediatric guideline increases classification of hypertension compared with earlier thresholds, with obesity concentrated among those newly classified [9]. Multiple reviews emphasize that obesity is a dominant correlate of elevated blood pressure in children and adolescents, and that persistent high BMI trajectories in childhood increase later hypertension risk [21].

Conceptual Framework

Framework description: We propose an integrated life course framework in which early life nutrition, socioeconomic status, and environmental exposures influence childhood obesity and pediatric hypertension through shared and interacting pathways involving diet quality, physical activity, sleep, stress biology, inflammation, and metabolic regulation [7, 11]. Socioeconomic status is modeled as both a direct determinant and a moderator that shapes the strength of nutrition and environmental effects, including through resource constraints, neighborhood conditions, and exposure burden [11, 17]. Environmental

exposures are conceptualized to act through both direct physiological mechanisms relevant to vascular function and indirect pathways via adiposity promoting environments, such as limited walkability or unsafe neighborhoods that reduce activity [17, 19].

Hypothesized pathways: Nutrition influences obesity through energy balance, appetite regulation, and dietary quality, and may influence blood pressure through sodium intake patterns, vascular function, and metabolic health [8, 9]. Socioeconomic disadvantage can operate through food insecurity, constrained access to healthy foods, chronic stress, and reduced opportunities for safe physical activity, thereby strengthening the probability of sustained adiposity and elevated blood pressure trajectories [11, 17]. Environmental exposures, including air pollution and neighborhood stressors, can affect cardiometabolic outcomes directly via inflammation and autonomic pathways and indirectly via obesity promoting contextual constraints, with interaction expected because disadvantaged communities often face higher exposure burdens [11, 18].

Methodology.

Study design: This study uses a repeated cross sectional quantitative synthesis combined with an integrative narrative review, which is appropriate when nationally representative microdata are not directly analyzed but high quality national estimates and peer reviewed syntheses are available [3, 9]. The repeated cross sectional component draws on NHANES based prevalence estimates published by the National Center for Health Statistics and CDC, enabling time trend characterization and subgroup comparisons [3, 4, 9]. The integrative review component is structured around the conceptual model and focuses on early life nutrition, socioeconomic determinants, and environmental mechanisms linked to obesity and pediatric hypertension as early cardiovascular risk factors [7, 11].

Data sources: Childhood obesity trends were extracted from NCHS Health E Stat 112, which provides NHANES based estimates from 1971 to 1974 through August 2021 to August 2023, including obesity and severe obesity definitions using CDC growth chart based criteria [3]. Socioeconomic gradients in obesity were extracted from the CDC Childhood Obesity Facts summary for 2017 to March 2020, which reports obesity prevalence by federal poverty level categories [4]. Pediatric hypertension prevalence, weight status gradients, and net reclassification under new versus former pediatric guideline thresholds were extracted from the CDC MMWR report using NHANES 2001 to 2016 and detailed classification tables for 2013 to 2016 [9]. Complementary child blood pressure prevalence comparisons for 2005 to 2008 versus 2013 to 2016 were extracted from the CDC Preventing Chronic Disease analysis evaluating changes under the 2017 pediatric guideline [10].

Study population and measures: The synthesized obesity measures represent U.S. children and adolescents ages 2 to 19 in NHANES based reports, excluding pregnant females where specified [3]. Obesity is defined as BMI at or above the 95th percentile for age and sex, and severe obesity is defined as BMI at or above 120% of the 95th percentile, consistent with NCHS reporting [3]. Pediatric hypertension classification follows the American Academy of Pediatrics guideline approach used by CDC analyses, which updates percentile references and uses a 130 over 80 mm Hg threshold for adolescents 13 years or older, alongside age and percentile frameworks for younger ages [8, 9].

Analytical approach: Descriptive trend analyses were conducted by extracting prevalence estimates into structured datasets, then plotting time series and subgroup comparisons to visualize changes and disparities over time [3, 9]. Comparative analyses focus on absolute differences and relative gradients across age groups, sex, weight status strata, and socioeconomic categories, consistent with public health surveillance interpretation [4, 9]. To illustrate an integrated pathway, we computed an obesity mediated expected hypertension prevalence by applying NHANES 2013 to 2016 hypertension prevalence conditional on obesity versus healthy weight to socioeconomic obesity prevalence strata, and we label this as an illustrative scenario rather than a causal estimate [4, 9]. We also computed a simple population attributable fraction for obesity related hypertension using prevalence ratios implied by reported subgroup prevalences, and we interpret it cautiously given cross sectional measurement and potential residual confounding [9].

Ethical considerations: This work uses publicly reported, de identified national estimates derived from NHANES based analyses and does not involve primary data collection or identifiable private information, which generally does not require additional institutional review for secondary descriptive synthesis [3, 9]. We nonetheless follow best practices for transparent documentation of definitions, units, and sources to enable reproducibility and verification [3, 9].

Results

Data and reproducibility resources: All extracted datasets, tables, and figures used in this manuscript are provided as downloadable supplements to support verification and reuse [3, 9].

Descriptive statistics and obesity trends

Nationally, obesity prevalence among U.S. youth ages 2 to 19 increased substantially across survey decades, rising from 5.2% in 1971 to 1974 to 21.1% in August 2021 to August 2023 [3]. Severe obesity rose from 1.0% to 7.0% over that same period, indicating a shifting distribution toward higher risk adiposity phenotypes rather than only modest upward drift

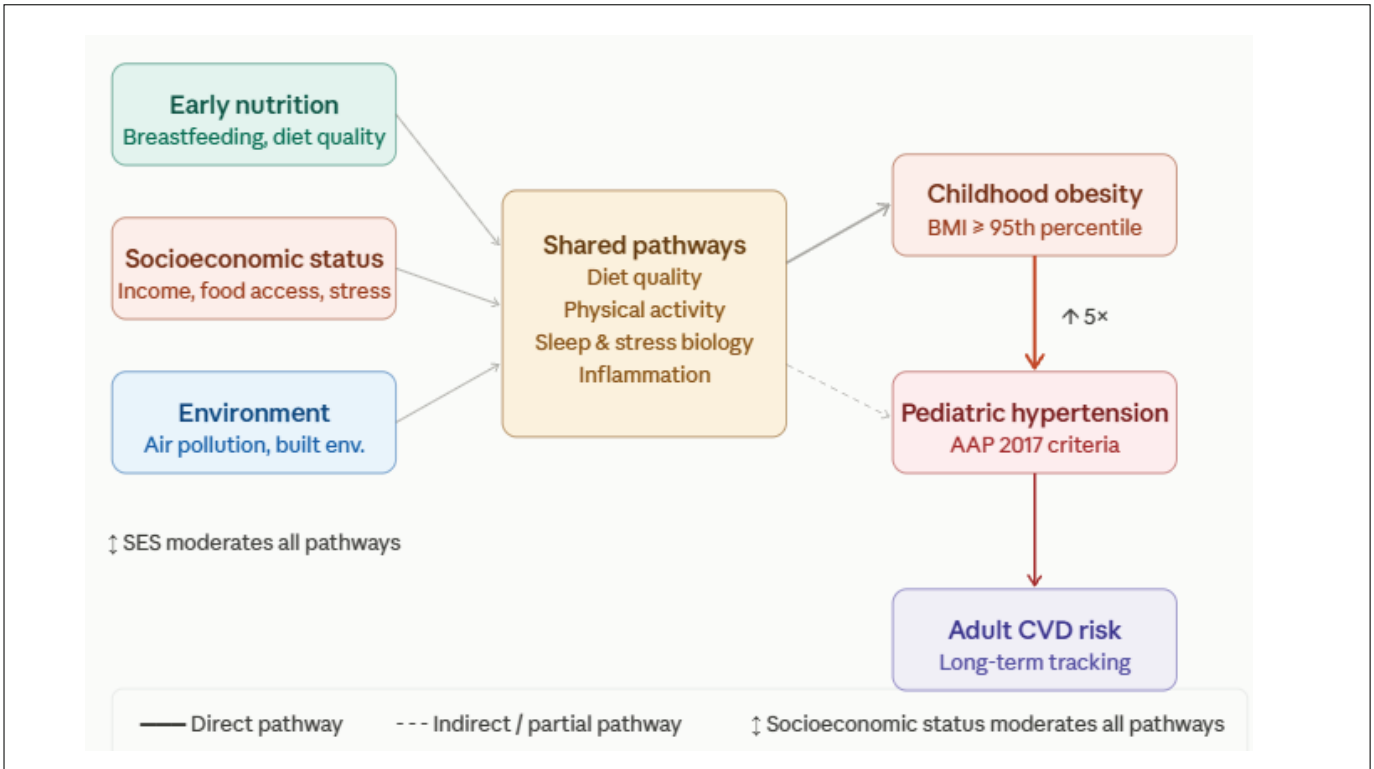

Figure 1: Conceptual framework for early-life determinants of childhood obesity and hypertension.

Table 1. Trends in overweight, obesity, and severe obesity among U.S. children and adolescents ages 2 to 19 [3].

Survey period	Overweight % (All)	Obesity % (All)	Severe obesity % (All)
1971 to 1974	10.2	5.2	1.0
2017 to 2018	16.1	19.3	6.1
Aug 2021 to Aug 2023	15.1	21.1	7.0

Figure 2. Trends in U.S. childhood obesity and severe obesity (ages 2 to 19).

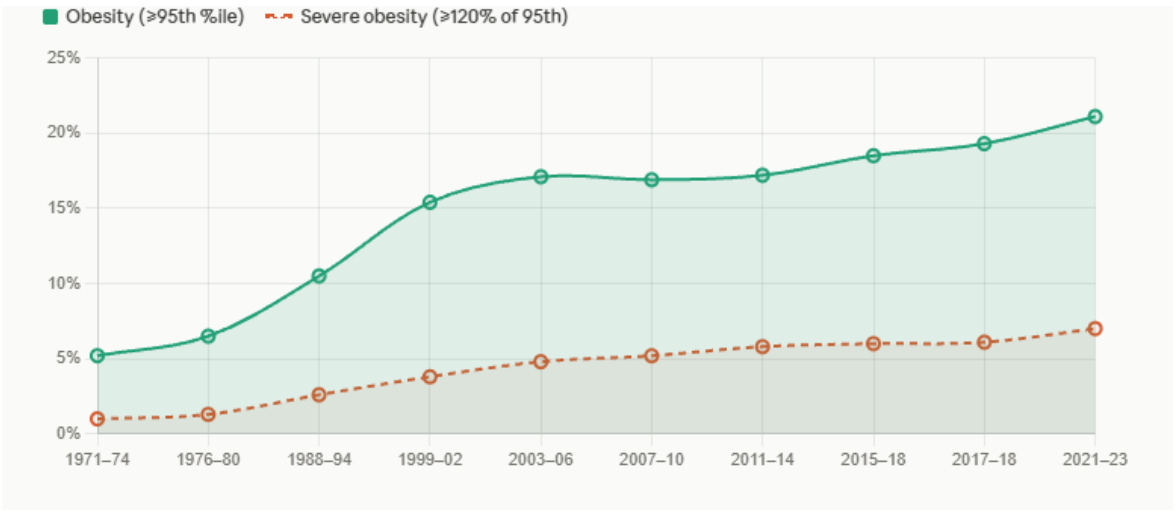

Figure 2: Trends in obesity and severe obesity among U.S. youth.

Figure 3. Obesity prevalence by age group (U.S. youth) in the 1999 to 2023 era.

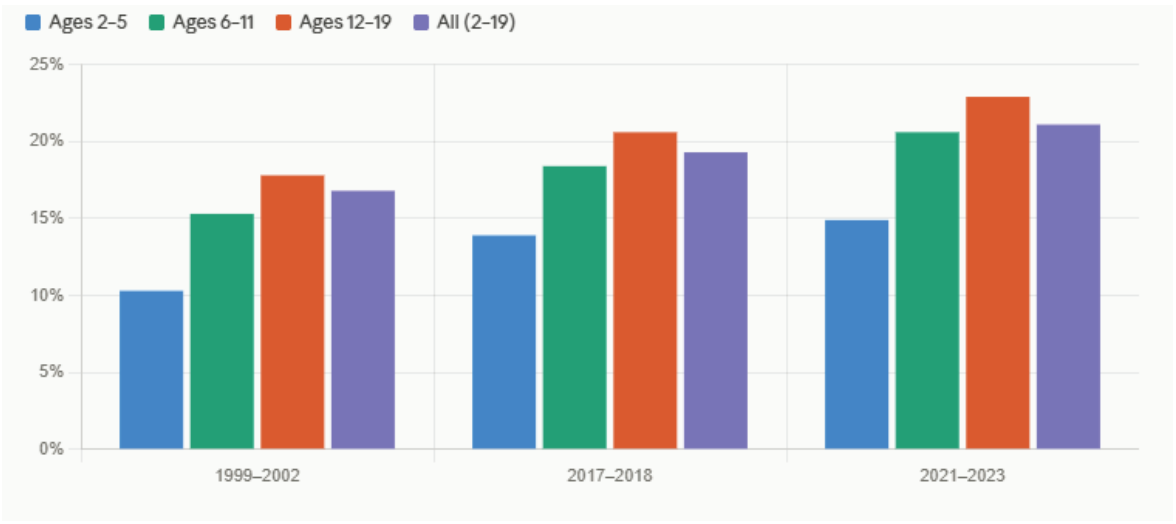

Figure 3: Obesity prevalence by age group.

[3]. Age stratified estimates show particularly high levels among older children and adolescents in recent cycles, with obesity at 22.9% among ages 12 to 19 in August 2021 to August 2023 compared with 14.9% among ages 2 to 5 [3].

Pediatric hypertension prevalence and guideline based classification

Using NHANES 2013 to 2016 and the 2017 pediatric guideline criteria, the CDC MMWR estimated hypertension prevalence among youths ages 12 to 19 at 4.11%, compared with 1.54% under the former guideline [9]. This difference translates to an estimated net increase of about 795,000 U.S. youths classified as hypertensive under the new guideline for 2013 to 2016, which CDC notes disproportionately affects older youths, males, and those with obesity [9]. CDC also reports that during 2013 to 2016, elevated blood pressure was approximately 10% and combined elevated blood pressure or hypertension was nearly 15% under the new guideline, reinforcing that a sizeable minority of adolescents exhibit above normal blood pressure phenotypes on exam day measures [9].

Table 2. Hypertension prevalence and population reclassification under new versus former pediatric guideline criteria, NHANES 2013 to 2016 [9].

Stratum	Hypertension prevalence % (new)	Hypertension prevalence % (former)	Net increase in number classified
All ages 12 to 19	4.11	1.54	795,000
Male	5.78	2.18	560,000
Female	2.42	0.88	235,000

Weight status gradient in pediatric hypertension and high blood pressure

The most consistent and policy relevant pattern is the steep weight status gradient in youth hypertension under contemporary definitions [9, 10]. In NHANES 2013 to 2016, hypertension prevalence was 1.88% among healthy weight youths but 9.43% among youths with obesity, and 14.70% among youths with severe obesity, indicating roughly a fivefold increase

Figure 4. Youth hypertension prevalence by weight status, NHANES 2013 to 2016, 2017 guideline.

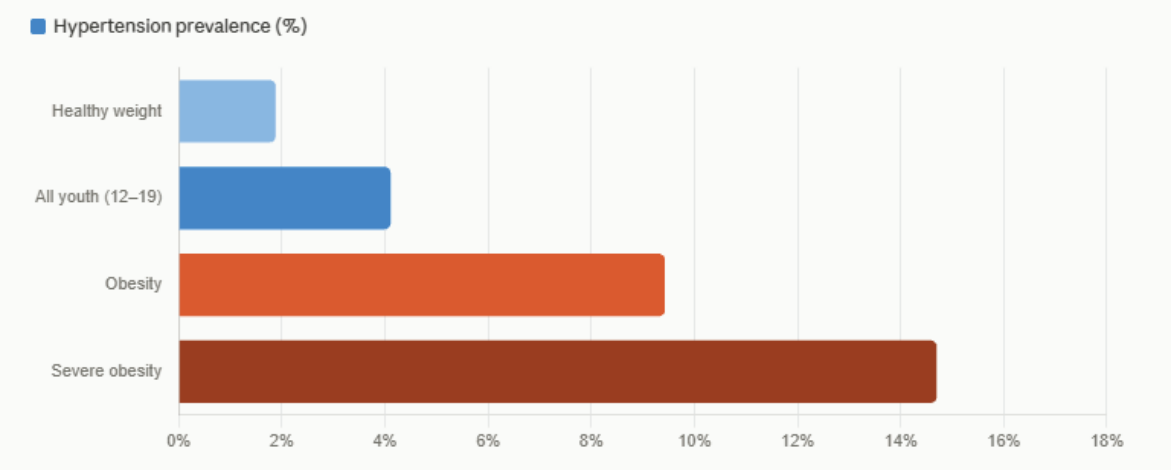

Figure 4: Hypertension prevalence by weight status.

Figure 5. High blood pressure by weight status across survey periods, AAP 2017 definition.

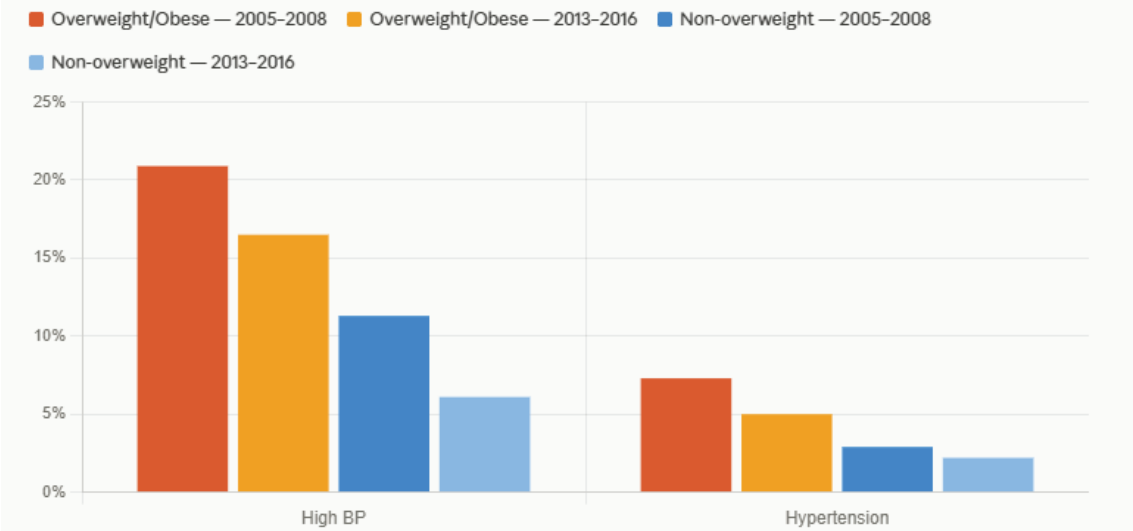

Figure 5: High blood pressure prevalence by overweight or obesity status.

Table 3. SES gradient in obesity and illustrative obesity mediated hypertension projection.

Family income category	Obesity prevalence %	Illustrative expected hypertension %
>350% FPL	11.5	2.75
130% to 350% FPL	21.2	3.48
≤130% FPL	25.8	3.83

Figure 6. Obesity prevalence by family income, U.S. youth 2017 to March 2020.

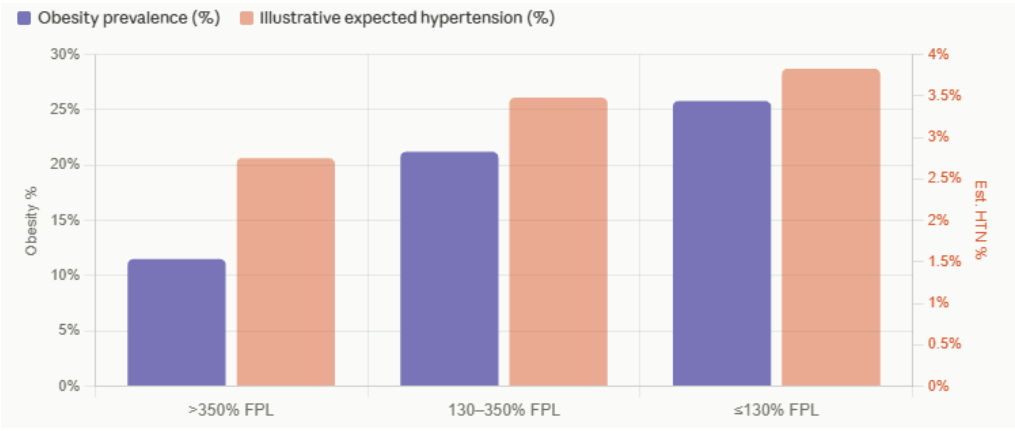

Figure 6: Obesity prevalence by family income.

for obesity versus healthy weight using reported prevalences [9]. In the CDC Preventing Chronic Disease analysis comparing 2005 to 2008 versus 2013 to 2016 under the 2017 pediatric guideline, high blood pressure prevalence among overweight or obese children decreased from 20.9% to 16.5%, while remaining much higher than the non-overweight group, which decreased from 11.3% to 6.1% [10]. Hypertension prevalence under the same analysis remained higher among overweight or obese children at 5.0% in 2013 to 2016 compared with 2.2% among those without overweight or obesity, demonstrating persistent risk concentration even as prevalence declined over time [10].

Socioeconomic gradient in obesity and illustrative downstream hypertension implications CDC reports an income gradient in obesity prevalence among U.S. youth ages 2 to 19 in 2017 to March 2020, with obesity at 11.5% for family income above 350% of the federal poverty level and 25.8% for family income at or below 130% of the federal poverty level [4]. Because obesity strongly increases hypertension prevalence in youth, this SES obesity gradient plausibly contributes to SES gradients in pediatric hypertension through obesity mediated mechanisms, even if SES also operates through direct stress, nutrition quality, and environment channels [9, 11]. Using a simplified illustrative scenario that applies NHANES 2013 to 2016 hypertension prevalence for obesity versus healthy weight to the SES specific obesity prevalence strata, expected hypertension prevalence would increase from about 2.75% in the highest income group to 3.83% in the lowest income group, purely through the obesity pathway under this crude mapping [4, 9]. This calculation is presented only to visualize how upstream SES inequalities in obesity can propagate into hypertension burden, and it should not be interpreted as a causal estimate because it mixes time periods and ignores within group heterogeneity [4, 9].

Discussion

Interpretation of findings in relation to prior studies

The quantitative synthesis confirms three consistent empirical patterns from U.S. surveillance, namely a long run rise in obesity and severe obesity, a substantial prevalence of pediatric hypertension under current guideline criteria, and a steep obesity gradient in youth hypertension [3, 9]. These findings align with the CDC conclusion that application of the 2017 pediatric guideline increases hypertension classification compared with prior definitions, and that obesity accounts for a disproportionate share of newly classified hypertension cases in youth [9]. The PCD analysis additionally indicates that high blood pressure and hypertension prevalence declined between 2005 to 2008 and 2013 to 2016, yet overweight and obesity remained a strong differentiator of risk, suggesting that upstream drivers continue to matter even when overall prevalence trends shift [10]. In life course terms, these patterns support the framing of childhood obesity as an early determinant of vascular risk and as a key pathway through which multiple upstream exposures may translate into later cardiovascular burden [5, 21].

Mechanisms linking nutrition, SES, environment to obesity and hypertension

Nutrition can influence obesity through cumulative energy balance and through early programming mechanisms involving appetite regulation and metabolic signaling, and breastfeeding related evidence suggests modest protective effects that may be stronger among children with higher genetic susceptibility to obesity [12]. Nutrition may also influence pediatric blood pressure through sodium and overall dietary patterns, and CDC explicitly references DASH style eating and physical activity promotion as strategies for youth hypertension prevention and management [9]. Socioeconomic status can intensify obesity and hypertension risk through constrained access to healthy foods, exposure to chronic psychosocial stress, time scarcity, and reduced opportunities for safe physical activity, and these mechanisms are emphasized as upstream drivers in social determinants theory [11]. Environmental context contributes through neighborhood safety, greenspace availability, and access to recreational facilities, which longitudinal evidence suggests can shape weight trajectories, and by air pollution exposures that may directly influence cardiovascular physiology while also contributing to obesity risk [17, 18, 20].

Policy implications for prevention and equity

Because obesity and hypertension cluster by socioeconomic position and environment, purely clinic centered approaches are unlikely to reduce population burden without parallel policy action on food environments, school nutrition, and neighborhood conditions [4, 11]. The CDC documented gradient by federal poverty level indicates that income related prevention must address affordability and access, including the structural drivers of diet quality rather than only education based messaging [4]. Built environment evidence supports investments in safe recreational spaces and neighborhood safety as feasible levers for modifying physical activity opportunities and stress exposures relevant to weight gain [17]. Environmental health frameworks and cardiovascular scientific statements support air pollution reduction as cardiovascular prevention, and the obesity evidence linking pollutants to childhood weight gain provides an additional rationale for environmental action framed around child health and equity [18, 20].

Strengths and limitations

A strength of this paper is its use of nationally reported, NHANES based estimates across long time horizons, enabling robust description of population trends in obesity and severe obesity and guideline based blood pressure outcomes [3, 9]. Another strength is explicit integration of a conceptual model with quantification of key gradients, which clarifies mediators and moderators for future microdata based causal analyses and SEM approaches [11]. Limitations include reliance on published estimates rather than person level microdata, which prevents full multivariable adjustment, causal mediation estimation, and direct modeling of nutrition and environmental exposures in a single unified dataset [3, 9]. Single visit blood pressure measurement in NHANES based analyses can overestimate true clinical hypertension prevalence compared with repeated visit diagnosis, and CDC notes this limitation explicitly, which should temper interpretation of prevalence magnitude [9].

Conclusion and Recommendations

Conclusion: Early life determinants of cardiovascular risk operate through interlocking pathways in which nutrition, socioeconomic disadvantage, and environmental exposures shape childhood obesity and pediatric hypertension as early phenotypes of later cardiometabolic burden. U.S. surveillance demonstrates a sustained rise in obesity and severe obesity among youth, and guideline based analyses show that hypertension prevalence, while modest in absolute terms, increases sharply with obesity and is therefore likely sensitive to upstream obesity prevention. Socioeconomic gradients in obesity imply that equity oriented interventions must address structural constraints and environmental context, not only individual behaviors, because upstream determinants shape downstream diet and activity opportunities across childhood.

Recommendations:

Clinical practice should prioritize routine blood pressure screening per pediatric guidance, with targeted follow up for children with obesity and those in high risk social contexts, because these groups carry higher prevalence and may benefit from earlier lifestyle intervention. Public health programs should combine school and community nutrition interventions with policies that improve affordability and availability of healthy foods, and that reduce exposure to ultra processed dietary patterns, while recognizing that breastfeeding support and early nutrition programs provide additional but modest upstream benefits. Community interventions should emphasize safe physical activity environments through parks, recreation, and neighborhood safety improvements, consistent with longitudinal built environment evidence and the conceptual model's mediator pathways. Environmental health policy should integrate child cardiovascular prevention with air pollution reduction and exposure mitigation strategies, reflecting cardiovascular scientific statements and evidence of pollutant associations with childhood obesity trajectories.

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