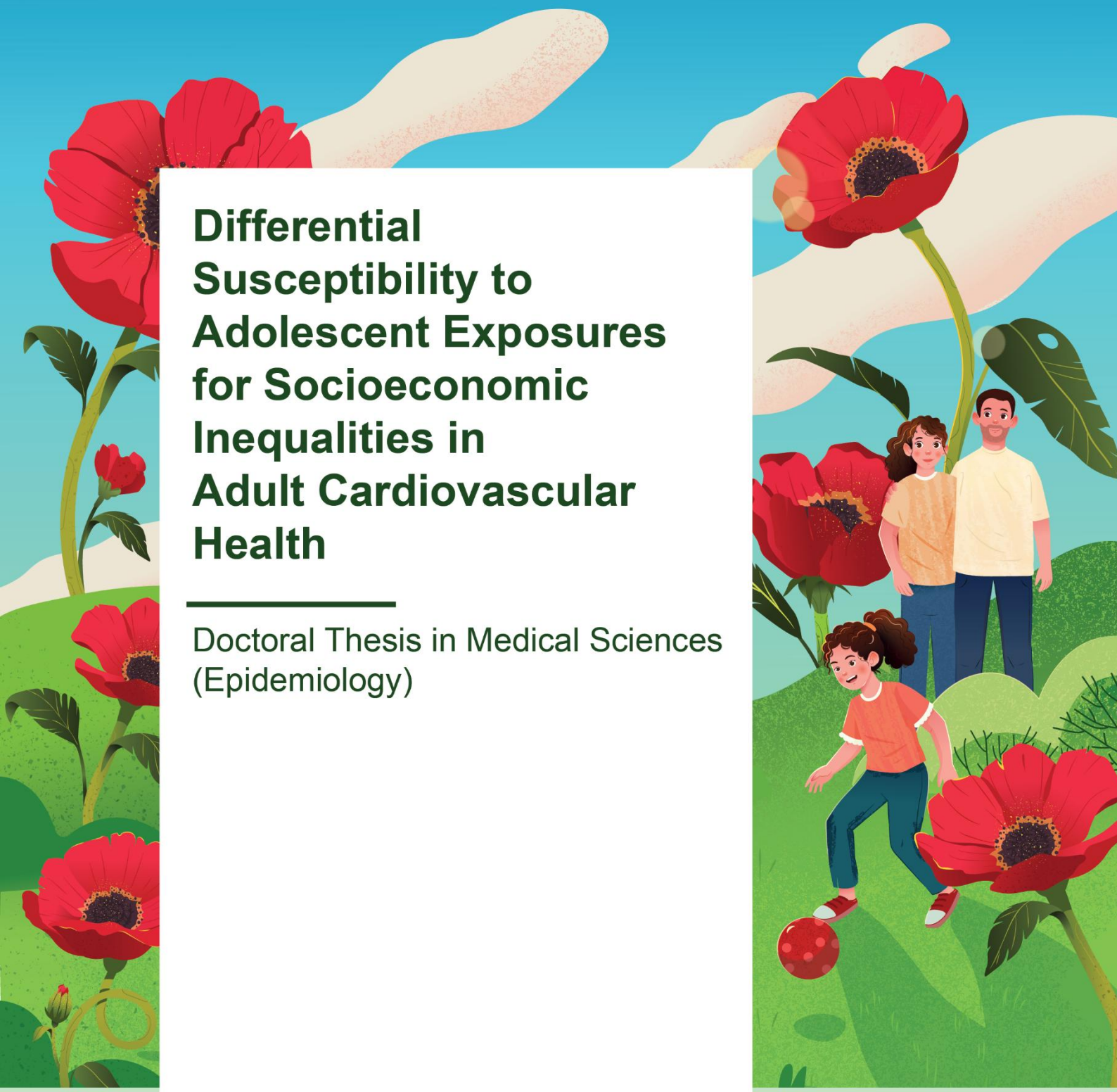


Differential Susceptibility to Adolescent Exposures for Socioeconomic Inequalities in Adult Cardiovascular Health

Doctoral Thesis in Medical Sciences (Epidemiology)

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For the children of Gaza, at least 20,000 of whom never made it to adulthood.

“The children are always ours, every single one of them, all over the globe; and I am beginning to suspect that whoever is incapable of recognizing this may be incapable of morality.” –James Baldwin

Department of Community Health

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Differential Susceptibility to Adolescent Exposures for Socioeconomic Inequalities in Adult Cardiovascular Health

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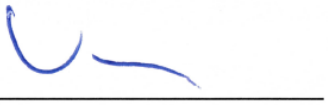
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Summaries

Abstract

Cardiovascular diseases (CVDs) and hypertension are among the leading causes of morbidity and mortality worldwide, topping the list of disease burden in 2023, despite declining cardiovascular mortality rates globally. These conditions are marked by persistent socioeconomic inequalities, with individuals from socioeconomically disadvantaged backgrounds facing higher cardiovascular risks than their more advantaged peers. Life course epidemiology posits that these inequalities develop through exposures operating across different stages of life, including social and behavioral exposures such as health-related behaviors and family environments, yet adolescence remains comparatively underexamined. Additionally, much of the literature has focused on differential exposure to harmful conditions and health-related behaviors, with less attention to differential susceptibility, a mechanism whereby the same exposures produce different magnitudes of effect across socioeconomic groups, leading to health inequalities.

In this thesis, titled “Differential Susceptibility to Adolescent Exposures for Socioeconomic Inequalities in Adult Cardiovascular Health”, I focus on adolescence as a stage in the life course during which exposures may lead to socioeconomic inequalities in cardiovascular outcomes in adulthood. Chapter 1 introduces the theoretical framework of the thesis. Drawing on social determinants of health frameworks, particularly the layered model of influence described by Dahlgren and Whitehead, we examine adolescent exposures across two domains, namely individual health behaviors and family environments, and quantify whether their effects on adult cardiovascular outcomes differ by family financial ability.

Chapter 2 sets out the objectives of this thesis. The overall objective is to assess the effect of social and behavioral exposures during adolescence on adult cardiovascular outcomes and whether the effects differ according to family financial situation. More specifically, the objective of the first study of this thesis is to quantify the differential effects of health behaviors among adolescents on adult cardiovascular conditions based on their family financial situation. The second study of this thesis aims to quantify the effect of parenting styles during adolescence on the risk of developing cardiovascular conditions in adulthood, and to assess whether family financial difficulty during adolescence modifies the effect of parenting styles on adult cardiovascular outcomes.

Chapter 3 describes the data and methods used to address these objectives. The analyses draw on the National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally representative cohort of adolescents in the United States followed into adulthood over more than two decades. Cardiovascular outcomes, specifically CVDs and hypertension, are measured at ages 33 to 43. Our analyses apply longitudinal designs and causal inference approaches to estimate the long term effects of adolescent exposures on adult cardiovascular outcomes.

In Chapter 4, we explore the first layer of influence, i.e. individual behavior and examine whether the long term cardiovascular effects of adolescent health behaviors differ according to family financial ability. Our analyses indicate that certain health behaviors do indeed have greater effects on cardiovascular outcomes among socioeconomically disadvantaged groups compared to those in more advantaged groups. After 21 years of follow up, 32.9% of participants had CVD or hypertension. Moderate to frequent alcohol use resulted in 10.8 percentage points (95% CI: 2.3 – 19.2) more cases among adolescents with family financial hardship compared to 1.1 percentage points (95% CI: -2.0 – 4.3) among those without, corresponding to a differential effect of 9.7 percentage points (95% CI: 0.8 – 18.5). Concurrent alcohol use and smoking exhibited a similar pattern, with 11.4 percentage points (95% CI: 0.9 – 22.0) more cases among disadvantaged adolescents compared to 0.4 percentage points (95% CI: -4.1 – 4.8) among advantaged adolescents, corresponding to a differential effect of 11.1 percentage points (95% CI: 0.1 – 22.1). In contrast, smoking when considered on its own showed little evidence of differential effects (2.0 percentage points, 95% CI: -7.2 – 11.2), and insufficient physical activity likewise showed no clear differential pattern (2.6 percentage points, 95% CI: -5.5 – 10.7). Breakfast skipping is associated with higher cardiovascular risk in our main analysis (5.4 percentage points, 95% CI: -3.7 – 14.6), but this effect is not consistently observed across sensitivity analyses. Thus, our results suggest that differential susceptibility is at play for some health behaviors, but not others.

Chapter 5 examines the next layer of influence i.e. family environment, here focusing on parenting styles during adolescence and their effect on adult cardiovascular outcomes. In our analysis, three parenting styles emerged: authoritative (11.1%), permissive (77.9%) and indifferent (11.0%). Overall, there were modest to negligible differences in CVD and hypertension risk for permissive or indifferent parenting compared with authoritative parenting (-1.0%, 95% CI: -5.3% – 3.3% and -1.8%, 95% CI: -7.8% – 4.2%, respectively). Permissive parenting appears protective among those from financially disadvantaged backgrounds in the main analysis, with a lower risk of -15.9% (95% CI: -28.4% – -3.3%), but this pattern is not consistent across sensitivity analyses and is likely explained by residual confounding. These

findings provide limited evidence that parenting styles during adolescence have long term causal effects or differential effects on cardiovascular outcomes at ages 33 to 43.

Chapter 6 discusses the findings from Chapters 4 and 5, situating them within a life course perspective and examining their implications for policy, practice and future research, particularly in relation to identifying which adolescent exposures contribute to cardiovascular inequalities and may serve as targets for intervention.

Based on the previous chapters, in Chapter 7, I conclude that adolescent exposures do not operate uniformly in their effects (or lack thereof) on cardiovascular risk in adulthood. Certain health behaviors may contribute to socioeconomic inequalities in cardiovascular outcomes through differential susceptibility. On the other hand, parenting styles as examined in our study appear to have limited relevance for long term cardiovascular outcomes in our study population. These results highlight the importance of identifying specific exposures that contribute to cardiovascular inequalities, and under what conditions, from a life course perspective, which may guide effective policies and interventions to improve overall population health and reduce health inequalities.

Résumé

Les maladies cardiovasculaires et l'hypertension comptent parmi les principales causes de morbidité et de mortalité dans le monde. Malgré la baisse des taux de mortalité cardiovasculaire à l'échelle mondiale, elles occupent la première place du classement du fardeau de la maladie en 2023. Ces pathologies se caractérisent par des inégalités socio-économiques persistantes, les personnes issues de milieux socio-économiquement défavorisés étant exposées à des risques cardiovasculaires plus élevés que leurs pairs plus favorisés. L'épidémiologie du parcours de vie postule que ces inégalités se développent à travers des expositions opérant à différentes étapes de la vie, y compris des expositions sociales et comportementales telles que les comportements liés à la santé et les environnements familiaux. Cependant, l'adolescence reste relativement peu étudiée. De plus, une grande partie de la littérature s'est concentrée sur l'exposition différentielle à des conditions nocives et aux comportements liés à la santé. Ceci accordant moins d'attention à la susceptibilité différentielle, un mécanisme par lequel les mêmes expositions produisent des effets d'ampleur différente selon les groupes socio-économiques, conduisant à des inégalités en matière de santé.

Dans cette thèse, intitulée « Differential Susceptibility to Adolescent Exposures for Socioeconomic Inequalities in Adult Cardiovascular Health », je m'intéresse à la phase du parcours de vie de l'adolescence pendant laquelle certaines expositions peuvent entraîner des inégalités socio-économiques dans les maladies cardiovasculaires à l'âge adulte. Le 1er chapitre présente le cadre théorique de la thèse, en nous appuyant sur les déterminants sociaux de la santé, particulièrement le modèle d'influence à plusieurs niveaux décrit par Dahlgren et Whitehead. Nous examinons les expositions pendant l'adolescence dans deux domaines, focalisant sur les comportements individuels en matière de santé et les environnements familiaux, et nous quantifions si leurs effets sur les résultats cardiovasculaires à l'âge adulte diffèrent en fonction de la situation financière de la famille.

Le 2e chapitre présente les objectifs de cette thèse. L'objectif général est d'évaluer l'effet des facteurs sociaux et comportementaux à l'adolescence sur les maladies cardiovasculaires à l'âge adulte, et de déterminer si ces effets varient en fonction de la situation financière de la famille. Plus précisément, l'objectif de la première étude de cette thèse est de quantifier les effets différentiels des comportements de santé chez les adolescents sur les maladies cardiovasculaires à l'âge adulte en fonction de la situation financière de leur famille. La deuxième étude de cette thèse vise à quantifier l'effet des styles parentaux pendant l'adolescence sur le risque de développer des maladies cardiovasculaires à l'âge adulte, et à

évaluer si les difficultés financières de la famille pendant l'adolescence modifient l'effet des styles parentaux sur les résultats cardiovasculaires à l'âge adulte.

Le 3e chapitre décrit les données et les méthodes utilisées pour ces études. Les analyses s'appuient sur l'étude « National Longitudinal Study of Adolescent to Adult Health (Add Health) », qui présente une cohorte représentative à l'échelle nationale d'adolescents aux États-Unis suivis jusqu'à l'âge adulte pendant plus de deux décennies. La présence des maladies cardiovasculaires et l'hypertension sont mesurées entre 33 et 43 ans. Nos analyses utilisent des plans d'étude longitudinaux et des approches d'inférence causale pour estimer les effets à long terme de l'exposition pendant l'adolescence sur les maladies cardiovasculaires à l'âge adulte.

Au 4e chapitre, nous explorons le premier niveau d'influence : le comportement individuel. Nous examinons si les effets cardiovasculaires à long terme selon des comportements de santé chez les adolescents diffèrent en fonction de la situation financière de la famille. Nos analyses indiquent que certains comportements de santé ont effectivement des effets plus marqués sur les résultats cardiovasculaires chez les groupes socio-économiquement défavorisés que chez les groupes plus favorisés. Après 21 ans de suivi, 32,9 % des participants présentaient une maladie cardiovasculaire ou une hypertension. Une consommation d'alcool modérée à fréquente entraînait 10,8 points de pourcentage (IC à 95 % : 2,3 – 19,2) plus élevé de maladies cardiovasculaires chez les adolescents issus de familles en difficulté financière, contre 1,1 point de pourcentage (IC à 95 % : -2,0 – 4,3) chez ceux qui n'ont pas de difficulté financière. Ceci correspond à un écart de 9,7 points de pourcentage (IC à 95 % : 0,8 – 18,5). La consommation simultanée d'alcool et de tabac a présenté une tendance similaire, avec une augmentation de 11,4 points de pourcentage (IC à 95 % : 0,9 – 22,0) chez les adolescents défavorisés, contre 0,4 point de pourcentage (IC à 95 % : -4,1 – 4,8) chez les adolescents favorisés, ce qui correspond à un effet différentiel de 11,1 points de pourcentage (IC à 95 % : 0,1 – 22,1). En revanche, le tabagisme, comme exposition isolé, ne présentait pas de signes d'effets différentiels (2,0 points de pourcentage, IC à 95 % : -7,2 – 11,2). Le manque d'activité physique ne montrait pas non plus de tendance différentielle (2,6 points de pourcentage, IC à 95 % : -5,5 – 10,7). Dans notre analyse principale, le fait de ne pas prendre de petit-déjeuner est associé à un risque cardiovasculaire plus élevé (5,4 points de pourcentage, IC à 95 % : -3,7 à 14,6), mais cet effet n'est pas systématiquement observé dans les analyses de sensibilité. Nos résultats suggèrent donc qu'une susceptibilité différentielle est présent pour certains comportements liés à la santé, mais pas pour d'autres.

Le 5e chapitre examine le niveau d'influence suivant : l'environnement familial, en se concentrant sur les styles parentaux pendant l'adolescence et leur effet sur les maladies cardiovasculaires à l'âge adulte. Notre analyse a mis en évidence trois styles parentaux : autoritaire (11,1 %), permissif (77,9 %) et indifférent (11,0 %). Dans l'ensemble, les différences en matière de risque de maladie cardiovasculaire et d'hypertension étaient modestes, voire négligeables, entre les styles parentaux permissif ou indifférent et le style autoritaire (-1,0 %, IC à 95 % : -5,3 % – 3,3 % et -1,8 %, IC à 95 % : -7,8 % – 4,2 %, respectivement). Le style parental permissif semble avoir un effet protecteur chez les personnes issues de milieux financièrement défavorisés dans l'analyse principale, avec un risque réduit de -15,9 % (IC à 95 % : -28,4 % – -3,3 %), mais cette tendance n'est pas constante dans les analyses de sensibilité et s'explique probablement par le biais de confusion résiduels. Ces résultats fournissent des preuves limitées que les styles parentaux pendant l'adolescence ont des effets causaux à long terme ou des effets différentiels sur les résultats cardiovasculaires entre 33 et 43 ans.

Le 6e chapitre examine les résultats présentés aux 4e et 5e chapitres, en les transmettant dans une perspective du parcours de vie et en analysant leurs implications pour les politiques, les pratiques et les recherches futures. On se focalise sur ce qui concerne l'identification des expositions subies à l'adolescence qui contribuent aux inégalités cardiovasculaires et pourraient servir comme des cibles d'intervention.

Sur la base des chapitres précédents, je conclus au 7e chapitre que les expositions subies à l'adolescence n'ont pas toutes les mêmes effets (ou l'absence d'effets) sur le risque cardiovasculaire à l'âge adulte. Certains comportements de santé peuvent contribuer aux inégalités socio-économiques en matière de résultats cardiovasculaires par le biais d'une susceptibilité différentielle. En revanche, les styles parentaux examinés dans notre étude semblent avoir une incidence limitée sur les maladies cardiovasculaires à long terme au sein de notre population étudiée. Ces résultats soulignent l'importance d'identifier les expositions spécifiques, autant que les conditions socio-économiques dans le contexte du parcours de vie, qui contribuent aux inégalités devant les maladies cardiovasculaires. Ceci pourrait orienter des politiques et des interventions efficaces visant à améliorer la santé globale de la population et à réduire les inégalités dans la santé.

Chapter 1: Introduction

Cardiovascular diseases (CVDs) and hypertension remain among the leading causes of morbidity and mortality worldwide. In recent decades, the absolute number of cardiovascular deaths has increased, driven partly by population growth and aging, even as age-standardized mortality rates have declined in many regions with their global burden continuing to rise in recent decades (Dattani, 2025; Hay et al., 2025). These aggregate trends mask important heterogeneity, as the decline in cardiovascular risk has not been uniform across populations, with persistent and ubiquitous socioeconomic inequalities. Across populations, individuals from socioeconomically disadvantaged backgrounds experience higher risks of cardiovascular morbidity and mortality than their more advantaged peers (Kim et al., 2008). Understanding how these inequalities arise remains a key challenge for epidemiology and public health.

This thesis takes a life course perspective to examine the effects of social and behavioral exposures during adolescence on inequalities in cardiovascular risk in adulthood. In this chapter, I introduce core concepts in health inequalities, particularly differential susceptibility as a key pathway through which cardiovascular inequalities may emerge. Further, I posit life course epidemiology as the theoretical framework for understanding how exposures in early life contribute to cardiovascular risk in later life. Finally, I situate in this chapter adolescence within this framework, describing its biological and social characteristics and its relevance for the development of cardiovascular inequalities.

Health Inequalities and Differential Susceptibilities

In virtually every society, health follows a social gradient. Individuals positioned lower in the socioeconomic hierarchy tend to live shorter lives and experience a higher burden of disease than those with greater socioeconomic advantage (WHO, 2008). This gradient has been documented across multiple indicators of socioeconomic position (SEP), whether it be education, occupation or income and across a wide range of health outcomes (Marmot et al., 1978; Braveman and Tarimo, 2002; Mackenbach, 2017; Tanaka et al., 2019). This gradient is not confined to those experiencing extreme deprivation, rather, health improves stepwise with each increase in SEP, indicating that health inequalities extend across the full social spectrum (Kawachi et al., 2002; Marmot, 2004).

The persistence and ubiquity of these patterns have shaped theoretical approaches to the social causation of disease. Link and Phelan's fundamental cause theory posits that

socioeconomic conditions influence health through access to resources such as knowledge, money, power and social connections, which can be used to avoid risks or minimize the consequences of disease (Link and Phelan, 1995). Complementary frameworks in social epidemiology have emphasized the processes through which social inequalities become biologically embodied. Krieger's eco-social theory, for example, highlights how repeated exposure to adverse material, psychosocial and environmental conditions over time shapes biological processes and contributes to the patterned distributions of disease (Krieger, 2005).

The social determinants of health framework further situates individual health outcomes within broader social, economic and political contexts. The Dahlgren and Whitehead model conceptualizes health as shaped by concentric layers of influence, ranging from individual characteristics and behaviors to social networks, living and working conditions, and wider structural determinants, as shown in Figure 1 (Dahlgren and Whitehead, 1991). Building on this model, Solar and Irwin describe how these determinants produce health inequalities, drawing attention to the role of governance, policy and power relations (and later, commercial influences) in structuring exposure to health-promoting or health-damaging conditions across populations (Solar and Irwin, 2010). According to Solar and Irwin, health inequalities are not simply the result of variations in individual characteristics but are socially reproduced through mechanisms of social stratification that operate across the life course.

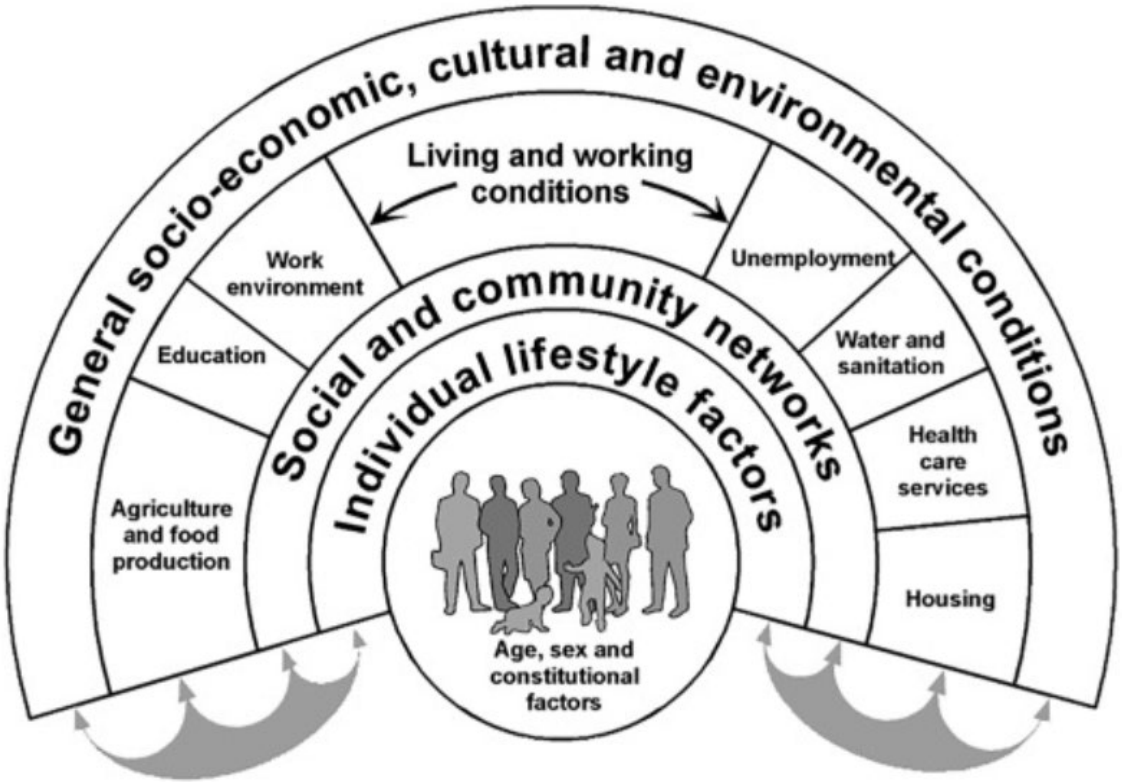


Figure 1: Dahlgren & Whitehead Model of Social Determinants of Health. Dahlgren and Whitehead (1991). Reproduced with permission.

Cardiovascular diseases exhibit a social gradient. Socioeconomic inequalities in CVDs and hypertension have been consistently documented across low-, middle- and high-income settings, with disadvantaged groups experiencing higher incidence, poorer prognosis and higher mortality (Schultz et al., 2018; Chaturvedi et al., 2024). Much of the existing studies examining the causes of these inequalities have focused on the differential exposure to health-related behaviors such as smoking, alcohol use, diet and physical activity. This mechanism, known as differential exposure, explains the emergence of health inequalities as a result of the uneven distribution of harmful exposures across socioeconomic groups, where socially disadvantaged groups are more likely to encounter adverse environments, constrained opportunities for healthy behaviors and cumulative risk profiles across the life course (Diderichsen et al., 2001). For example, recent evidence suggests that socioeconomic differences in cardiovascular risk are associated with socially-patterned clustering of behavioral and metabolic risk factors from early adulthood onwards (Ho et al., 2022).

However, differential exposure alone may not fully account for observed inequalities in cardiovascular outcomes. Another less studied mechanism is differential susceptibility, which posits that the same exposure may produce different health effects depending on individuals' social or biological circumstances (Diderichsen et al., 2019). Through differential susceptibility, socioeconomic disadvantage may amplify the harmful effects of exposures through mechanisms such as cumulative stress, reduced physiological reserve, or the accumulation of prior adversity (Diderichsen et al., 2001; Diderichsen et al., 2019).

Distinguishing between differential exposure and differential susceptibility is important because each mechanism points to different points for intervention for reducing health inequalities. Addressing differential exposure is important for targeting the unequal upstream distribution of risk factors, while addressing differential susceptibility requires attention to the social and biological processes that condition vulnerability to those risks (Diderichsen et al., 2012). In practice, these mechanisms are interrelated and often operate simultaneously across the life course, reinforcing one another over time (Diderichsen et al., 2019). Figure 2 illustrates these two mechanisms schematically.

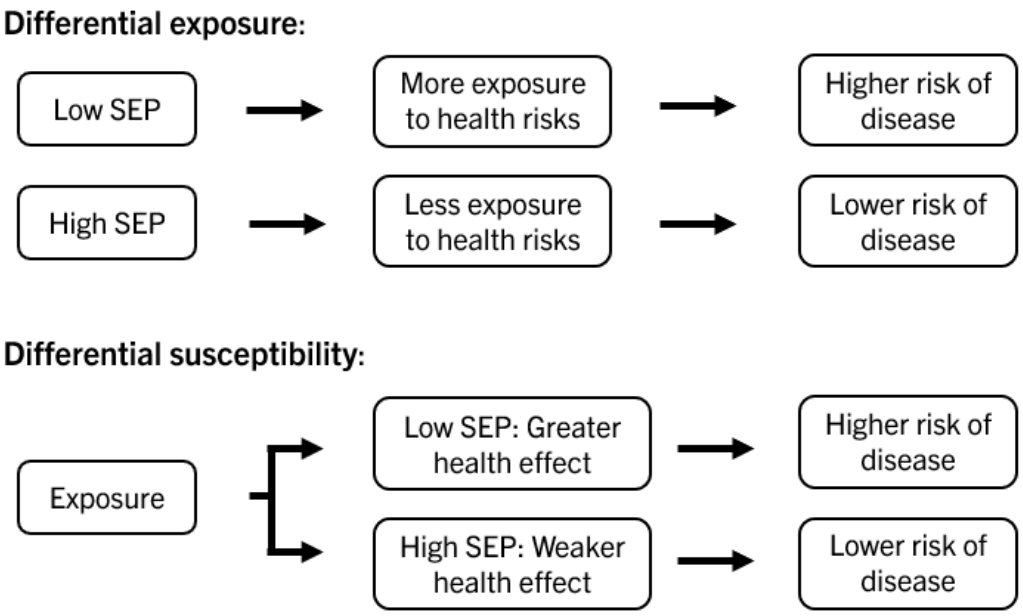


Figure 2: Conceptual illustration of differential exposure and differential susceptibility mechanisms in the production of health inequalities. SEP = socioeconomic position. Author's illustration.

Understanding why health inequalities matter extends beyond their contribution to overall disease burden. Large and persistent health inequalities signal underlying social arrangements that systematically constrain opportunities for health among substantial segments of the population. Comparative studies suggest that societies with greater socioeconomic inequality tend to exhibit worse average health outcomes, higher levels of morbidity and poorer social functioning, even among more advantaged groups (Wilkinson and Pickett, 2009). Health inequalities therefore reflect both the differences between social groups and also the extent to which overall population health is shaped by the distribution of socioeconomic resources. Mechanisms such as differential exposure to harmful conditions and differential susceptibility to their effects help explain how social advantage and disadvantage become translated into patterns of health inequality. Understanding how these mechanisms unfold over time is therefore central to explaining the emergence of health inequalities and identifying effective points for intervention.

Life Course Epidemiology

The life course perspective provides a powerful conceptual and analytical framework for examining how health inequalities develop over time. Life course epidemiology is defined as the study of long-term biological, behavioral and psychosocial processes that link adult health and disease risk to physical or social exposures acting during gestation, childhood, adolescence, earlier in adult life or across generations (Kuh et al., 2003; Wagner et al., 2024).

Rather than viewing health outcomes and exposures at single points, life course epidemiology recognizes that health is shaped by various exposures over the life span, whereby earlier exposures can influence subsequent trajectories, and advantages or disadvantages may accumulate, interact, or be modified at different stages of life (Kuh and Ben Shlomo, 2004).

Life course epidemiology is particularly relevant for research into the causes of chronic diseases where long latency periods and cumulative risk processes are involved in disease development (Ben-Shlomo and Kuh, 2002). This includes cardiovascular conditions, as highlighted in a 2002 report by the World Health Organization (WHO) which noted that cardiovascular health and diseases develop due to the combined influence of early growth patterns, childhood and adolescent socioeconomic conditions and behavioral trajectories, rather than the effects of exposures during adulthood alone (Aboderin et al., 2002). The report further noted that socioeconomic disadvantage acting across multiple stages of life contributes to the social inequalities of cardiovascular outcomes through biological and behavioral pathways.

Since then, a growing body of empirical evidence continues to suggest that socioeconomic disadvantages experienced earlier in life are the causes of increased cardiovascular risk in adulthood (Kivimaki et al., 2006; Walsemann et al., 2016; Nguyen et al., 2025). These findings have informed calls for life course approaches to cardiovascular prevention, including the Lancet Commission on hypertension, which called for a life course strategy to address the global burden of raised blood pressure (Olsen et al., 2016). Despite this, much empirical research on cardiovascular inequalities continues to focus on adult behaviors and clinical risk factors, with comparatively limited attention to how exposures earlier in the life course contribute to socioeconomic inequalities in cardiovascular outcomes.

Early contributions to life course epidemiology proposed a range of conceptual models to describe how exposures at different stages of life influence later health (Ben-Shlomo and Kuh, 2002; Kuh et al., 2003). Building on this, Lynch and Smith emphasized that life course models must account for the socially patterned accumulation of disadvantage and the structuring of pathways through education, occupation and health-related behaviors, especially for cardiovascular outcomes (Lynch and Smith, 2005). More recently, building on Kuh et al. (2003), Wagner et al. (2024) put forward four main models in which exposures across the life course affect health outcomes, shown in Figure 2 (Kuh et al., 2003; Wagner et al., 2024).

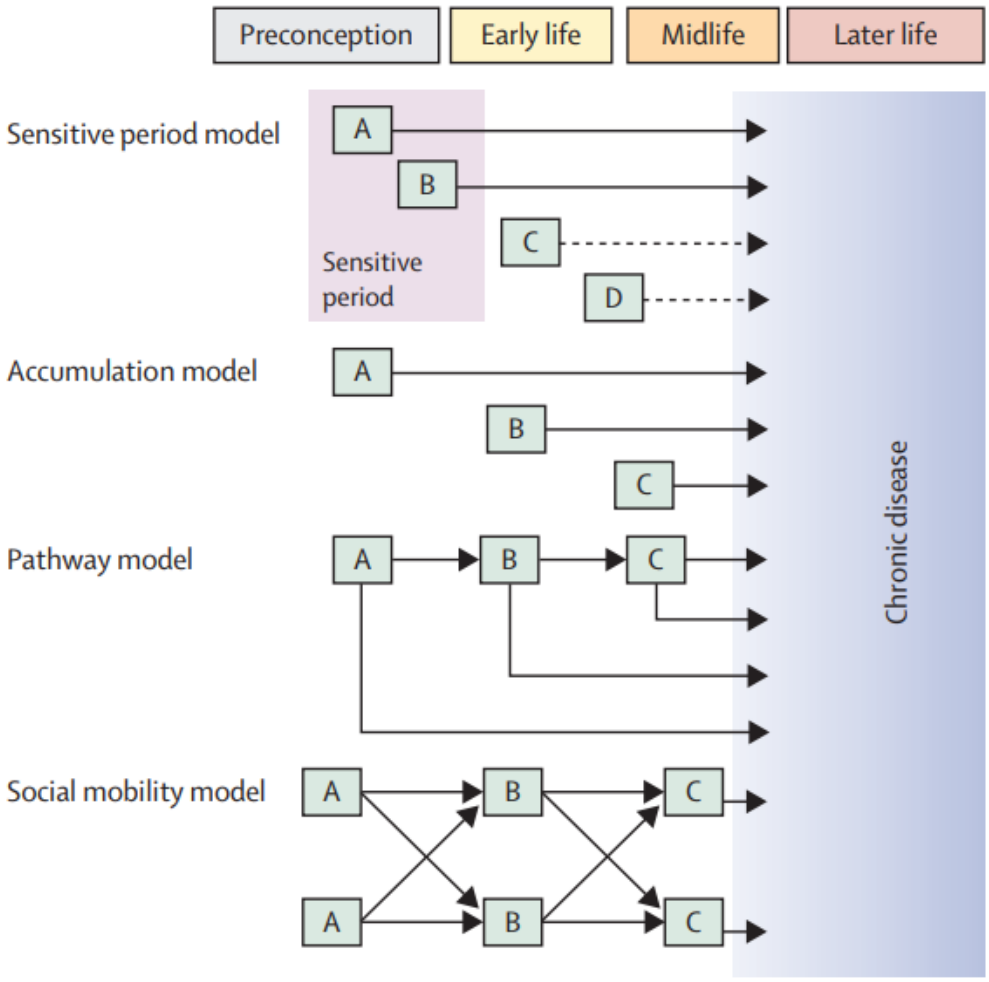


Figure 3: Life course models for the causes of chronic diseases. A, B, C and D are exposure at different times during the life course. Arrows show causal effects; dotted arrows signify weaker causal effects. Reproduced from Wagner et al., 2024.

The sensitive period model proposes that exposures have stronger associations with later health when they occur during particular developmental stages, such as early childhood or adolescence, though exposures at later stages may still matter (Ben-Shlomo and Kuh, 2002). For cardiovascular health and diseases, sensitive periods have been identified in relation to early growth, pubertal development and adolescence, where social and behavioral exposures may have lasting implications for CVDs and hypertension in later life (Aboderin et al., 2002; Viner et al., 2015; Wagner et al., 2024).

The accumulation model emphasizes the cumulative impact of disadvantage over time, whereby repeated or prolonged exposure to adverse social, behavioral or biological conditions increases cardiovascular risk in an additive or dose-response manner (Ben-Shlomo and Kuh, 2002). Evidence from cardiovascular epidemiology indicates that sustained exposure to socioeconomic disadvantage is associated with higher risks of hypertension, coronary heart

disease and stroke, independent of adult circumstances (Lynch and Smith, 2005; Kivimaki et al., 2006). The pathway model, sometimes known as the chain of risk model, refers to sequences of linked exposures, whereby early life conditions shape subsequent educational, occupational and behavioral trajectories that together influence later cardiovascular risk (Aboderin et al., 2002; Lynch and Smith, 2005).

Finally, the social mobility model considers how changes in SEP across the life course affects health outcomes. Upward or downward mobility may modify the influence of early life circumstances, either attenuating or amplifying accumulated disadvantage (Wagner et al., 2024). For CVDs, evidence suggests that mobility trajectories are associated with differential risks, although early-life conditions often retain an influence even among those who experience upward mobility (Walsemann et al., 2016).

These models are not mutually exclusive and often operate simultaneously. Indeed, different models may be more relevant at different stages of life or for different outcomes (Kuh and Ben Shlomo, 2004). Each model has distinct prevention and intervention implications, highlighting the importance of identifying when in the life course to intervene (Wagner et al., 2024). Together, these models provide a structured basis for examining how timing, duration, sequencing and change in socioeconomic circumstances contribute to inequalities in cardiovascular outcomes across the life course (Lynch and Smith, 2005; Pollitt et al., 2005).

These life course models have important implications for prevention strategies. In cardiovascular epidemiology, increasing attention has been given to primordial prevention, whereby risk factors are prevented from emerging in the first place, rather than treating established risk factors or disease (Gillman, 2015). In the context of cardiovascular prevention, primordial prevention involves intervening before the emergence of hypertension, adverse lipid profiles, obesity or cardiovascular health risk behaviors. Evidence from longitudinal and intervention studies suggests that early life interventions, including those initiated in infancy and continued through adolescence, can influence blood pressure trajectories and other cardiovascular risk markers (Gillman, 2015). Within a life course framework, primordial prevention emphasizes maintaining ideal cardiovascular health from early development onward and preventing the emergence of socially patterned risk factors across stages of life. This perspective shifts attention from treatment of established risk factors to the timing and social distribution of their origins (Epure and Chiolero, 2019).

Adolescence: Lost in Transition

Within life course research, adolescence has received comparatively less attention than other stages, particularly early childhood and later adulthood. Adolescence, typically defined as the period between ages 10 and 19 years (WHO, n.d.), has been described as a "key stage of development in the life course that has been largely neglected by the global health community" (Baird et al., 2025). Studies have predominantly concentrated on early childhood, particularly the first 1,000 days of life as emphasized by the developmental origins of health and disease framework (Barker et al., 1993; Barker, 2007; Epure et al., 2020), and on adulthood, where clinical risk factors and disease outcomes are most readily observed (Sawyer et al., 2012). Adolescence, by contrast, is often treated as a transitional phase rather than as a distinct period with long-term consequences for health. Yet, adolescence occupies a pivotal position in the life course, during which early life exposures are translated into adult trajectories, and earlier advantage or disadvantage may be reinforced or modified (Johnson et al., 2011).

Exposures during adolescence, including health-related behaviors and broader psychosocial and environmental conditions, can have long-term health effects. In a recent WHO report, "Framework to Implement a Life Course Approach in Practice", it is highlighted that early risks to health and wellbeing in adolescence are associated with up to 70% of preventable deaths from non-communicable diseases in adulthood, and explain 14% of differences in physical and mental capacities among older people (WHO, 2025). Given the importance of adolescence as a potential point of intervention, the 2025 Lancet Commission on Adolescent Health and Wellbeing notes that investing in research in adolescence health has the potential to yield a "triple dividend", leading to improvements in health in adolescence itself, as well as in later adulthood and for the next generation (Baird et al., 2025).

What makes adolescence such a pivotal moment in the life course? This period is characterized by rapid biological, psychological and social change (Sawyer et al., 2012). It is second only to infancy in the pace and breadth of development, encompassing pubertal maturation, continued brain development and processes of identity formation (Viner et al., 2015). It is in this period that adolescents experience increasing autonomy from their parents, greater influence of peer and partner relationships, and key educational and occupational transitions that shape future trajectories across various domains (Viner et al., 2015; Patton et al., 2018; Teuber et al., 2022).

These changes in adolescence often coincide with the initiation and consolidation of health-related behaviors that are relevant for cardiovascular risk. Indeed, many health-related

behaviors emerge during adolescence and show substantial tracking into adulthood. Smoking, alcohol use, dietary habits and physical activity patterns often emerge in adolescence and show substantial tracking into adulthood (Mollborn et al., 2014; Lawrence et al., 2017) These behaviors are socially patterned, with adolescents from socioeconomically disadvantaged backgrounds more likely to engage in multiple health risk behaviors and to maintain these behaviors over time (Lynch et al., 1997; Due et al., 2011). Consequently, adolescence represents an early point at which behavioral pathways contributing to adult cardiovascular inequalities begin to diverge.

In addition to behavioral pathways, biological and psychosocial changes during adolescence may heighten sensitivity to social and material conditions in ways that are relevant for later cardiovascular outcomes. Pubertal timing, stress reactivity and psychosocial adversity during adolescence have been associated with adult cardiovascular risk and outcomes (Viner et al., 2015; Doom et al., 2017). Thus, adolescence aligns with life course models that emphasize sensitive periods, as well as accumulation and pathway models, whereby risks compound and cascade across subsequent stages of life (Ben-Shlomo and Kuh, 2002; Due et al., 2011).

Additionally, family environments remain central during adolescence, even as individual agency expands. Parenting practices, household financial strain, family routines and intergenerational resources continue to shape adolescents' opportunities, stress exposures and behavioral options (Conger et al., 2010; Johnson et al., 2011).

At the same time, longitudinal evidence indicates that behavioral trajectories remain relatively malleable during adolescence and early adulthood. Adolescents from more advantaged backgrounds are more likely to discontinue risky behaviors as they transition into adulthood, whereas those from disadvantaged backgrounds are more likely to persist, contributing to the embedding of social inequalities in cardiovascular health over time (Andersson et al., 2022; Shao et al., 2023).

The relevance of adolescence for health inequalities is further underscored by its position at the intersection of individual, family and structural influences. Conceptual models such as Dahlgren and Whitehead's social determinants framework illustrate how individual behaviors are nested within social networks, living conditions and broader socioeconomic contexts (Dahlgren and Whitehead, 1991). Adolescence can be understood as a juncture at which these layers interact particularly strongly: individual agency increases, family resources and constraints remain influential and institutional contexts such as education systems and labor market pathways begin to shape future opportunities (Lynch and Smith, 2005; Johnson et al., 2011; Viner et al., 2015). Despite this, adolescence-specific applications of social

determinants frameworks remain underdeveloped, particularly with respect to their implications for long-term health inequalities.

Taken together, adolescence represents a consequential yet underexamined stage in the life course. It is a period during which early life inequalities are translated into durable behavioral and social patterns, while biological and psychosocial changes may heighten vulnerability to adverse exposures. At the same time, it offers opportunities to alter trajectories before cardiovascular risk becomes entrenched. Understanding adolescence as a distinct and consequential stage is therefore essential for explaining the emergence of socioeconomic inequalities in cardiovascular health and for identifying effective points of intervention.

Causal Approaches to Life Course Epidemiology

Investigating the long-term effects of social and behavioral exposures across the life course presents important methodological challenges. Randomized controlled trials (RCTs) are generally infeasible or unethical for many exposures of interest, particularly those related to social conditions and health-related behaviors, which often cannot be experimentally assigned (Westreich, 2020). As a result, this thesis relies on observational longitudinal data and adopts a causal inference framework to estimate the effects of adolescent exposures on adult cardiovascular outcomes. This approach involves specifying causal models based on expert knowledge, applying estimation strategies designed to approximate causal effects under clearly defined assumptions, and conducting sensitivity analyses to assess the robustness of findings. Further details on conducting causal inference using observational data are described in Chapter 3.

Summary

This chapter outlined cardiovascular health inequalities within a life course framework, emphasizing that socioeconomic differences in cardiovascular disease and hypertension reflect exposures and vulnerabilities accumulated across multiple stages of life. While much research has focused on the effects of exposures during adulthood, life course epidemiology highlights the importance of timing, duration and sequencing of social and behavioral exposures for understanding how these inequalities emerge and persist.

Despite this, important gaps remain in understanding how inequalities in cardiovascular health take shape earlier in the life course. In particular, adolescence has received comparatively limited attention, even though it represents a stage during which social, behavioral and family-related exposures are consolidated and may shape subsequent health

trajectories. What remains insufficiently understood is the extent to which adolescent exposures contribute to adult cardiovascular outcomes and whether their effects differ according to SEP. If this is the case, research cannot focus solely on differential exposure as an explanation for cardiovascular health inequalities—it is also necessary to consider the presence of differential susceptibility as a mechanism through which health inequalities are reproduced.

Against this background, this thesis focuses on adolescence as a stage in the life course through which socioeconomic inequalities in cardiovascular disease and hypertension may develop. By focusing on adolescent exposures within a life course perspective, this thesis seeks to examine how socioeconomic disadvantage becomes embedded in adult cardiovascular risk and to inform strategies for addressing cardiovascular health inequalities.

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Chapter 2: Objectives

This thesis examines how socioeconomic inequalities in CVDs and hypertension develop across the life course. The overall objective is to assess the effect of social and behavioral exposures during adolescence on adult cardiovascular outcomes and whether their effects differ according to family financial situation. Specifically, this thesis addresses two research aims:

Aim 1: Unequal Effects of Health Behaviors in Adolescence on Adult Cardiovascular Disease and Hypertension by Family Financial Situation in the US: A Cohort Study (Chapter 4)

To quantify the differential effects of health behaviors among adolescents on adult cardiovascular conditions based on their family financial situation.

Aim 2: Effects of Adolescent Parenting Styles on Adult Cardiovascular Disease and Hypertension: A Population-Based Cohort Study (Chapter 5)

- To quantify the effect of parenting styles during adolescence on the risk of developing cardiovascular disease and hypertension in adulthood.
- To assess whether family financial difficulty during adolescence modifies the effect of parenting styles on adult cardiovascular outcomes.

Chapter 3: Methodology

This chapter provides an overview of the data source and methodological approaches underlying the research presented in this thesis. Detailed descriptions of study-specific methods are provided within Chapters 4 and 5.

Add Health Cohort

To investigate the effects of exposures during adolescence adulthood on adult cardiovascular outcomes, the target population of this thesis is adolescents aged 12 and 19 living in the United States. Data were drawn from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a population-based longitudinal cohort study conducted in the United States. Add Health was initiated in the mid-1990s with a nationally representative sample of adolescents enrolled in grades 7 to 12 and has followed participants into adulthood, collecting repeated measures of social, behavioral, and health-related factors across multiple waves (Harris et al., 2019).

Initial data collection was completed through in-home interviews of both participants and their parents in 1994 and 1995 (Wave I; ages 12 – 21). Since then, five follow up periods of data collection have been completed: Wave II (1995– 1996; ages 13 – 20), Wave III (2001 – 2002; ages 18 – 26), Wave IV (2008 – 2009; ages 24 – 32), Wave V (2016 – 2018; ages 33 – 43) and Wave VI (2022 – 2025, ages 39 – 51). As the Wave VI data files were not available at the time of analysis, this thesis uses data up to Wave V. Each survey wave implemented a stratified cluster sampling design to ensure that the sample was representative of US schools with respect to region of country, urbanicity, school size, school type, and ethnicity. Participation rate at all waves is over 70% (Harris et al., 2019). Figure 4 provides general details of Waves I – V of Add Health.

The resulting dataset contains extensive information on demographic, social, familial, socioeconomic, behavioral and health characteristics across the life course, making it particularly well suited for investigating the effects of exposures during adolescence on health outcomes in adulthood.

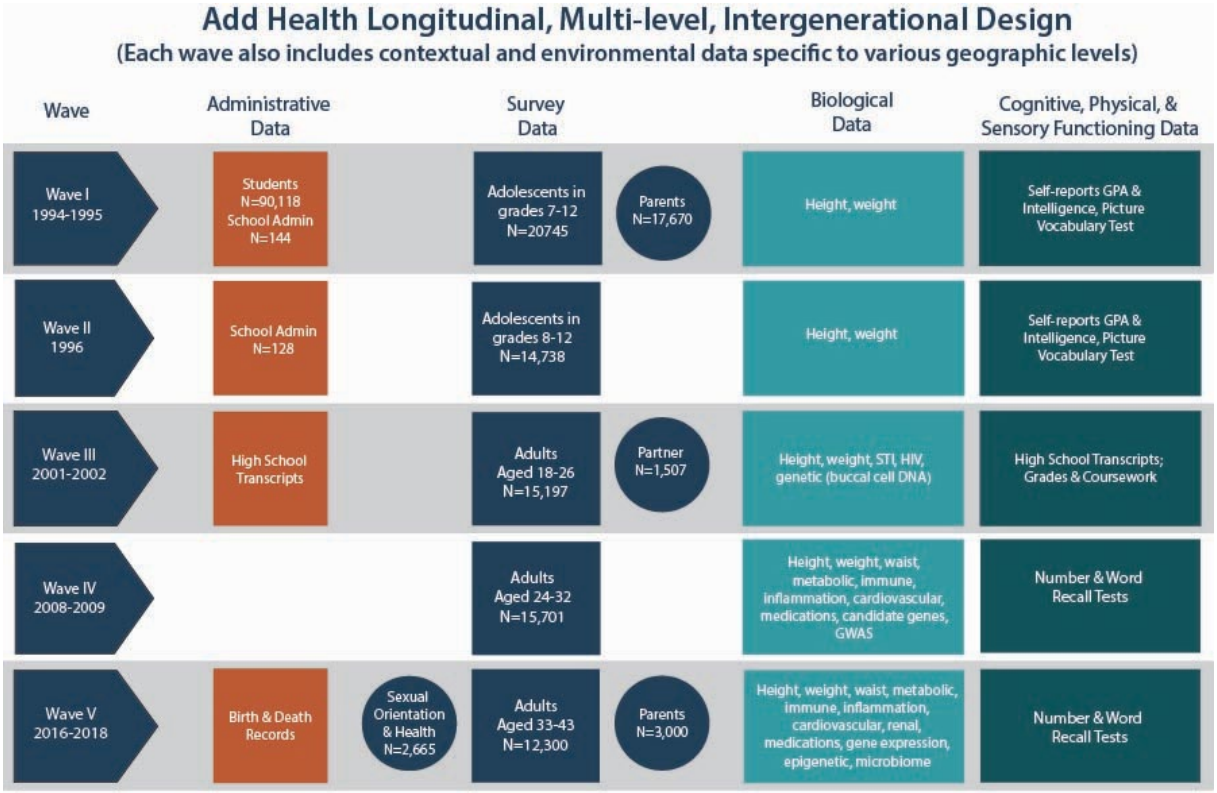


Figure 4: Add Health waves. Figure reproduced from (Add Health, n.d.).

The United States provides an important context for studying cardiovascular inequalities. Despite being a highly industrialized and high-income nation, the US also has some of the highest rates of CVDs globally, with pronounced social patterning in both life expectancy (Chetty et al., 2016) and disease outcomes (Chaturvedi et al., 2024). These health inequalities have emerged alongside widening income and wealth disparities since the 1970s, with the US ranking among industrialized countries with the highest levels of economic inequality (Chetty et al., 2016). This context provides an opportunity to examine how social and behavioral exposures during adolescence may contribute to cardiovascular outcomes within a highly stratified socioeconomic environment.

Causal Inference from Observational Data

This thesis uses causal inference methods to estimate the effects of adolescent exposures on adult cardiovascular outcomes using observational data. Identifying the causes of disease and health inequalities using observational data presents substantial methodological challenges. Randomized controlled trials are often infeasible or unethical for studying social and behavioral exposures across the life course (Westreich, 2020). As a result, epidemiological research on health inequalities relies heavily on observational data, requiring

careful attention to study design, confounding and assumptions necessary for causal interpretation. At the same time, work in social epidemiology has emphasized the importance of complementing causal methods within substantive theories of social stratification and health, rather than treating estimation strategies as purely technical exercises (Kaufman and Oakes, 2017).

This thesis adopts a causal inference framework informed by contemporary epidemiological methods. Within this framework, causal effects are conceptualized using the potential outcomes framework, in which causal effects are defined as contrasts between potential outcomes under different exposure conditions (Westreich, 2020). Because these potential outcomes cannot be simultaneously observed for the same individual, causal effects must be estimated indirectly using observed data under a set of identifying assumptions (Naimi and Whitcomb, 2023).

The assumptions required to identify causal effects from observational data include exchangeability, positivity, consistency and no interference. Exchangeability requires that, conditional on measured covariates, exposed and unexposed individuals would have had the same expected outcome in the absence of exposure (Greenland and Robins, 1986). Positivity requires that all levels of the exposure occur within levels of the covariates being conditioned on (Westreich and Cole, 2010). Consistency requires that the observed outcome corresponds to the potential outcome under the exposure actually received, while the assumption of no interference requires that the exposure status of one individual does not influence the outcome of another individual (Rehkopf et al., 2016). These assumptions form the basis for causal effect estimation using observational data.

Longitudinal data introduces additional challenges. Exposures may vary over time, cluster with other social and behavioral factors, and be influenced by prior health status. Confounding may arise from both observed and unobserved factors, and conventional regression approaches may inadequately address time-varying confounding (Westreich, 2020). Recent methodological developments, including g-methods such as inverse probability weighting (IPW), offer tools for addressing these challenges and estimating population-level effects under clearly defined assumptions (Cerdá and Keyes, 2017; Naimi et al., 2017). To assess the robustness of these estimates, sensitivity analyses can be carried out. One approach is the use of negative controls to detect unmeasured confounding or other sources of bias. Negative controls, commonly used in laboratory experiments, are variables that are not expected to have a causal effect on the outcome or the exposure of interest, but may share similar sources of bias, allowing the detection of spurious associations (Lipsitch et al., 2010).

In this thesis, these methods are applied to estimate the effects of adolescent exposures on adult cardiovascular outcomes and to quantify effect modification by family financial situation. The analytical strategy is designed to align with the life course perspective by explicitly considering the timing of exposures and their relationship to later health outcomes, while acknowledging the limitations inherent to observational data.

Exposures

The exposures examined in this thesis reflect key aspects of the adolescent social and behavioral environment, consistent with the life course frameworks and social determinants of health models described in Chapter 1, which emphasize the role of multiple layers of influence on health across the life course.

In the first study, the exposure of interest is health behaviors during adolescence, including alcohol use, cigarette smoking, breakfast eating habits and physical activity. These behaviors are often established during adolescence and are known to contribute to cardiovascular risk through both direct physiological pathways and the persistence of behavioral patterns into adulthood (Lawrence et al., 2017; Petrovic et al., 2018). In addition to examining individual behaviors, this study also tested for evidence of behavioral clustering and estimated their effects when clusters were identified, motivated by health lifestyles theory, which suggests that behaviors co-occur in structured patterns shaped by social conditions (Hussein et al., 2018; Mollborn et al., 2021).

In the second study, the exposure of interest is parenting style during adolescence. Parenting style is conceptualized along the dimensions of responsiveness and demandingness, classified using latent class analysis based on adolescent-reported indicators of parental warmth, communication and behavioral control. Parenting styles are a key feature of the family environment and have been associated with long-term health through their effects on psychosocial development, health behaviors and cardiovascular outcomes across the life course (Wolfradt et al., 2003; Vedanthan et al., 2016; Demakakos et al., 2018).

Together, these exposures capture both behavioral and family-level influences during adolescence that may contribute to cardiovascular health trajectories.

Outcomes

Across the studies included in this thesis, the primary outcome is a composite measure of CVDs and hypertension assessed during adulthood in Wave V of the Add Health study,

when respondents were aged between 33 and 43 years. CVDs and hypertension are leading contributors to morbidity and mortality worldwide (Dattani, 2025; Hay et al., 2025) and display strong socioeconomic patterning (Kim et al., 2008), making them important outcomes for studying health inequalities across the life course.

The outcome was defined as the presence of any of the following conditions: stroke, heart failure, heart disease, atrial fibrillation (collectively grouped as CVDs) or hypertension. Participants reporting any of these conditions were considered as having CVDs or hypertension.

Hypertension was identified using a combination of biomarker measurements, self-reported history of being diagnosed or current use of antihypertensive medication. Stroke, heart failure and atrial fibrillation were identified through self-reported previous diagnosis by a healthcare professional. Heart disease was identified through self-reported previous diagnosis by a healthcare professional or history of heart surgery for clogged coronary arteries.

Family Financial Situation as an Effect Modifier

A central objective of this thesis is to examine whether the effects of adolescent exposures on adult cardiovascular outcomes differ according to socioeconomic conditions during adolescence, i.e., if there is differential susceptibility. This thesis uses family financial situation as an effect modifier to examine and quantify potential differential susceptibility to adolescent exposures.

Socioeconomic position is commonly operationalized using indicators such as education, income or occupation. While these measures capture important dimensions of social stratification, they do not fully reflect material conditions or economic strain experienced by households (Oakes and Andrade, 2017). For example, education may be a less reliable proxy for material resources in the US context, where its economic returns vary substantially by race and ethnicity, and where educational expansion has reduced variability in attainment over time (Krieger et al., 1997). Income is also an imperfect indicator of SEP, as income measures are often subject to substantial non-response and reporting biases (Turrell, 2000), and may not adequately capture differences in household needs, expenditures and purchasing power (Krieger et al., 1997).

This thesis therefore focuses on family financial situation during adolescence as an indicator of material conditions. Measures of financial strain capture households' ability to meet material needs and manage economic stressors, which may be more directly relevant to adolescents' lived conditions. Financial strain is associated with adverse health outcomes

independently of conventional SEP indicators yet remains comparatively understudied in cardiovascular epidemiology (McLaughlin and Albert, 2025). A recent systematic review and meta-analysis suggests that financial strain is associated with poorer cardiovascular health and higher cardiovascular risk in adulthood (Swarup et al., 2024). In the US, over 37% of households reported difficulty paying for basic expenses in 2024 (United States Census Bureau, 2024).

Thus, family financial situation is examined as an effect modifier to assess whether adolescent exposures have differential effects on adult cardiovascular outcomes according to conditions during adolescence.

Covariates

Directed acyclic graphs (DAGs) were used to guide covariate selection and identify sufficient adjustment sets for causal estimation. DAGs provide a graphical representation of assumed causal relationships between exposures, outcomes and other variables, allowing the identification of confounding paths that must be closed to estimate causal effects (Tennant et al., 2021).

For this thesis, the covariates were selected based on DAGs constructed separately for each empirical study. These diagrams incorporated prior theoretical and empirical knowledge regarding the determinants of adolescent exposures and cardiovascular outcomes, and were used to identify appropriate adjustment sets for the causal analyses.

Statistical Analyses

In both studies included in this thesis, the analysis strategy combined latent variable modeling with causal methods to quantify the effects of adolescent exposures on adult cardiovascular outcomes.

Latent Class Analysis

Latent class analysis (LCA) is a modeling approach that identifies subgroups within a population, based on shared response patterns across multiple observed variables (Porcu and Giambona, 2017). Rather than examining individual behaviors or parenting characteristics separately, this approach allows the identification of broader patterns of exposure that may better reflect real-world behavioral and family environments. The empirical studies in this thesis

used LCAs to identify unobserved patterns in adolescent exposures, although for slightly different analytical purposes.

In the first study, LCA was used to detect the presence of adolescent health behavior clustering in our study population. The purpose of the LCA in this study was exploratory, aimed at identifying patterns of co-occurring health behaviors among adolescents in our study population, not to define exposure groups for causal analysis. Evidence of co-occurrence between certain behaviors, such as alcohol use and cigarette smoking, informed the construction of additional exposure variables used in the subsequent analyses.

In the second study, an LCA was used to classify parenting styles during adolescence. Unlike the first study, where LCA was used to detect behavioral clustering, the LCA in the second study was used to operationalize a theoretical construct. The resulting latent classes were interpreted in relation to established parenting typologies and used as the primary exposure variable in the subsequent causal analysis, by assigning individuals to their most likely latent class based on posterior class membership probabilities.

Causal Analysis

Causal effects were estimated using regression-based approaches within a counterfactual framework. Given the use of observational data where exposures were not randomly assigned, statistical adjustments were carried out to approximate exchangeability between exposure groups. Specifically, this thesis uses IPW approaches to adjust for measured confounding. Stabilized weights were estimated based on the probability of exposure conditional on the covariates identified through the DAGs. These weights create a pseudo-population in which the distribution of measured confounders is balanced across exposure groups, allowing estimation of average causal effects under the assumption of no unmeasured confounding. Weighted regression models were then used to estimate the effects of adolescent exposures on adult cardiovascular outcomes. Effect modification by family financial situation was assessed by estimating exposure effects among adolescents from a family facing and not facing difficulties paying bills.

All analyses accounted for the complex sampling design of the Add Health study through the use of survey weights to ensure population-representative estimates.

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Chapter 4: Unequal Effects of Health Behaviors in Adolescence on Adult Cardiovascular Disease and Hypertension by Family Financial Situation in the US: A Cohort Study

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Background

Cardiovascular diseases (CVDs) and hypertension are leading causes of disability and death worldwide (Hay et al., 2025). These conditions display a social gradient, with individuals from socioeconomically disadvantaged situations facing higher risks of disease and related mortality compared to those from more advantaged socioeconomic circumstances. This disparity is partly attributed to a higher prevalence of health risk behaviors including alcohol consumption, cigarette smoking, poor dietary habits, and physical inactivity among those socially disadvantaged (Petrovic et al., 2018).

While the differential exposure to health risk behaviors has been extensively documented, the complementary mechanism of differential vulnerability or susceptibility remains underexplored (Lynch et al., 1997). Differential susceptibility refers to the phenomenon whereby individuals from disadvantaged socioeconomic situations may experience stronger harmful effects of health risk behaviors, contributing to the observed socioeconomic gradient in CVDs and hypertension. Differential susceptibility can arise because of biological embedding of early life adverse experiences (Diderichsen et al., 2019; Vineis et al., 2020) or because of the clustering of health risk behaviors during adolescence which then form a lifestyle that persists into adulthood (Lawrence et al., 2017; Hussein et al., 2018). Individuals who experienced material or psychosocial adversities in early life may have a heightened stress response system (Tung et al., 2022), making them more vulnerable to the negative impacts of health risk behaviors on their health and development of chronic diseases (Solar and Irwin, 2010; Berens et al., 2017; Diderichsen et al., 2019). Parental socioeconomic position in early life is critical to determining these early life conditions and thus contributes to the development of socioeconomic health inequalities in adulthood (Houweling and Grünberger, 2024).

Despite its potential importance, most studies on differential susceptibility to health behaviors have primarily focused on adults, with limited evidence available on adolescence, a

potentially critical period when many health behaviors are established (Due et al., 2011; Sawyer et al., 2012; Nordahl et al., 2014; Probst et al., 2020). Recently, a Finnish cohort study found that socioeconomic background during adolescence can modify the long-term effects of adolescent fruit and vegetable consumption on adult blood pressure levels, although this effect modification was not observed for other health behaviors (Jackisch et al., 2025). These emerging findings suggest the need to further examine whether socioeconomic disadvantage in adolescence can modify the long-term effects of other health behaviors on cardiovascular diseases and hypertension across different settings.

Quantifying differential susceptibility to health risk behaviors during adolescence is essential for developing effective public health strategies. Whole-population interventions that achieve the uniform relative reduction in health risk behaviors across socioeconomic groups could yield greater health improvement among the disadvantaged, thus narrowing inequalities. Recognizing greater susceptibility among socioeconomically disadvantaged adolescents could inform the design of targeted policies and programs that provide additional support to these groups, further mitigating health inequalities.

Given the critical role of adolescent behaviors in shaping health outcomes over the life course, this study aims to quantify the differential effect of health risk behaviors among US adolescents on adult CVDs and hypertension based on their family financial situation. We consider family financial ability, here defined as the ability to pay the bills, because it captures household economic situation related to household expenditure and wealth (Tucker-Seeley et al., 2016). Unlike traditional socioeconomic indicators such as income or education, assessing financial situation can reveal health-related inequalities across all socioeconomic levels, thereby informing public health and policy strategies that reach individuals who might otherwise be overlooked (Bialowolski and Weziak-Bialowolska, 2013; McLaughlin and Albert, 2025). Additionally, financial situation was included in the 2024 American Heart Association and American College of Cardiology report on key data elements and definitions of determinants of health in cardiology (Morris et al., 2024). Financial strain has been associated

with poorer cardiovascular health and higher cardiovascular risk in adulthood, as shown in a recent systematic review and meta-analysis (Swarup et al., 2024). Thus, it is important to examine family financial situation as an effect modifier especially in the US, where over 37% of households in 2024 reported difficulty paying for basic expenses (United States Census Bureau, 2024).

By examining the differential effects of adolescent health behaviors by family financial situation, this study seeks to expand the understanding of the mechanisms underlying social inequalities in long term health outcomes.

Methods

Study and analytic populations

Our target population is adolescents in the United States. The study population was from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a longitudinal and nationally representative sample of US adolescents (https://addhealth.cpc.unc.edu/). Initial data collection was completed through in-home interviews of both participants and their parents in 1994 and 1995 (Wave I; ages 12 – 21). Since then, four follow up periods of data collection have been completed: Wave II (1995– 1996; ages 13 – 20), Wave III (2001 – 2002; ages 18 – 26), Wave IV (2008 – 2009; ages 24 – 32), and Wave V (2016 – 2018; ages 33 – 43). Each survey wave implemented a stratified 1-level cluster sampling to ensure that the sample is representative of US schools with respect to region of country, urbanicity, school size, school type, and ethnicity (Harris et al., 2019). Participation rate at all waves is over 70%. The ensuing dataset comprises rich information on demographic, social, familial, socioeconomic, behavioral and adult health measures, making it well suited for this study (Harris, 2013; Harris et al., 2019).

The flow chart in Fig. 1 outlines the selection process of the analytic samples. After excluding Wave I participants that were older than 19 (n = 190) or with missing age (n = 17),

our study population included 20583 adolescents. The sample for the cluster analysis comprised 18400 adolescents and was derived by excluding individuals with missing health behaviors (n = 367) and with missing sampling design information at Wave I (n = 1771). The sample for the causal analysis comprised 4452 adults and was derived by excluding individuals with missing health behaviors in adolescence (n = 367), who were not sampled at Wave V for biomarker collection (n = 14869), with missing Wave V sampling design information (n = 110) and with missing parental financial situation during adolescence (n = 740). We excluded individuals with missing health behaviors and parental financial situation during adolescence because we hypothesized missingness not at random for these covariates (Hughes et al., 2019).

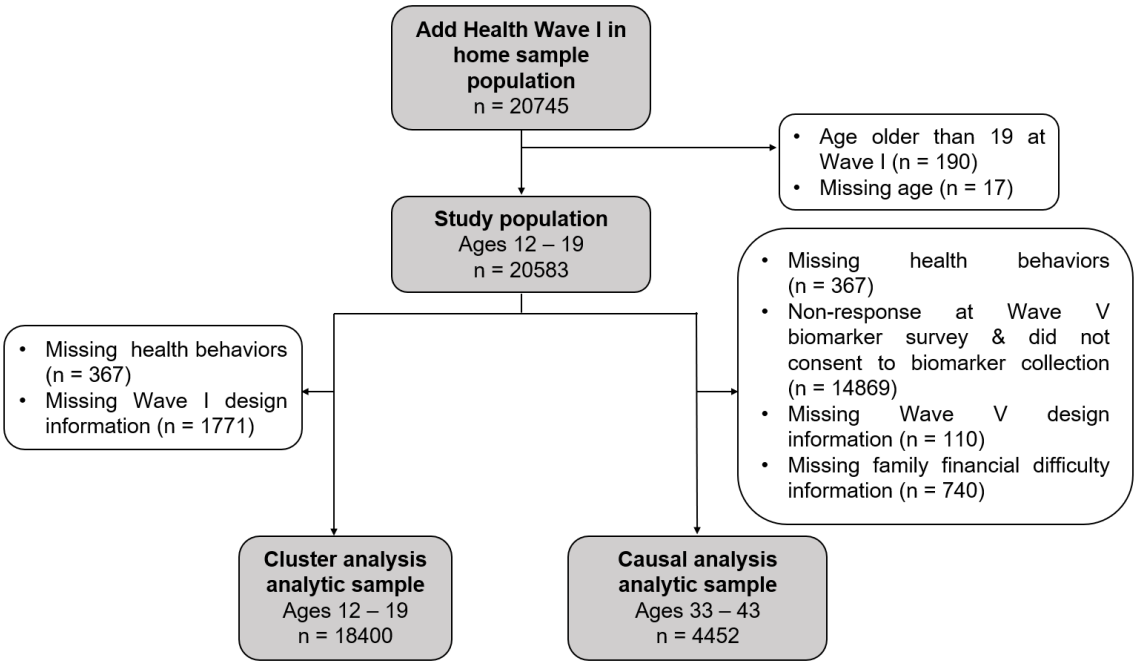


Figure 1. Flow chart of participants selection

The analytic sample is nationally representative of the US population of adolescents in grades 7 – 12 in 1994 – 1995. This is achieved using the Wave V biomarker weights, which adjusts for unequal probabilities of selection in Wave I, attrition across waves, and differential consent for biomarker collection during the Wave V in-home exam (Chen and Harris, 2020).

Causal model and measures

Based on existing literature and the knowledge of the authors (Jackisch et al., 2024), we built a conceptual causal model around the main causal path of interest, namely the effect of health behaviors in adolescence (exposure) on CVDs and hypertension in adulthood (outcome), as shown in Fig 2. Potential confounding included socioeconomic factors relative to the family and the neighborhood, parental health behaviors, history of adversities, chronic diseases, mental health disorders and excess weight.

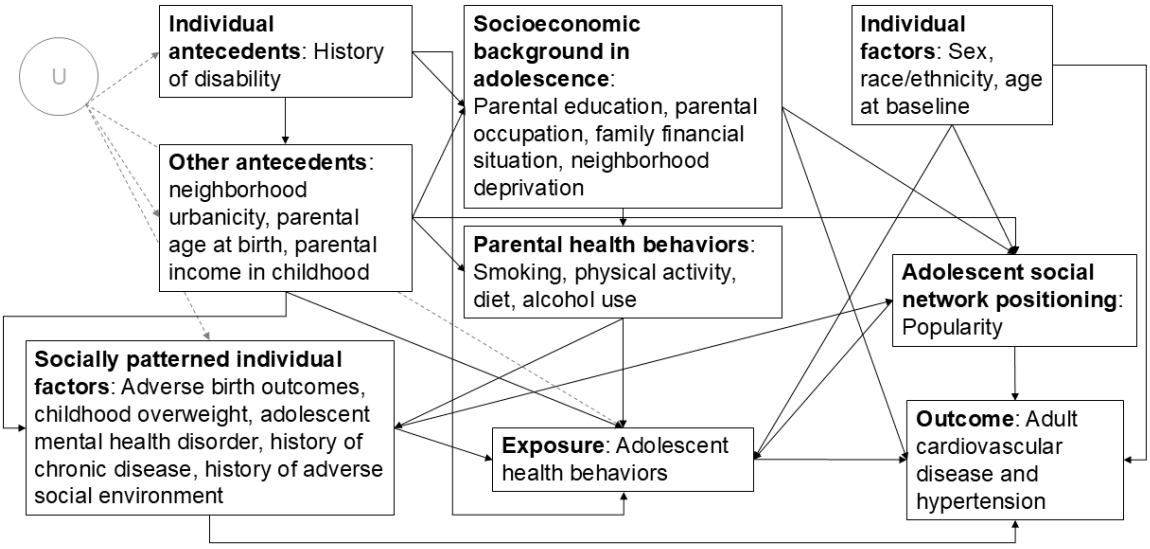


Figure 2. Conceptual causal model for adolescent health behaviors (exposure) to CVDs and hypertension in adulthood (outcome). U stands for other unidentified confounding factors that are not included in the model.

We subsequently established an analytical directed acyclic graph (DAG, see Fig. S1 in Supplementary Materials) to identify a subset of confounding factors belonging to the minimally sufficient adjustment set for the causal analysis. The effect modifier of interest, family financial situation, is illustrated within a broader super-node labelled “socioeconomic background in adolescence” in the conceptual causal model shown in Figure 2 and appears as a separate node in the full DAG in Supplementary Fig. S1.

Exposure: Health behaviors

Health behaviors during adolescence, including alcohol use, cigarette smoking, breakfast eating habits, and physical activity, were assessed using self-reports collected at Wave I (see Table S1 in Supplementary Materials for the specific questions used to measure these exposures).

Physical activity was measured using two indicators: sports frequency and exercise frequency, defined by the number of times respondents engaged in these activities per week. Sports referred to participation in active sports such as baseball, basketball or soccer, whereas exercise captured individual physical activities such as jogging, walking or dancing. Both sports and exercise frequencies were categorized into four levels: never (none), 1 – 2 times/week (low frequency), 3 – 4 times/week (moderate frequency), and 5 or more times/week (high frequency). Cigarette smoking was categorized into four levels: never smokers, past smokers, those smoking one cigarette/day, and those smoking multiple cigarettes/day. Alcohol use was categorized into five levels: never drinkers, past drinkers (those who did not drink in the past 12 months but had before), rare drinkers (those who drank alcohol 1 – 2 days in the past 12 months), moderately frequent drinkers (those who drank once a month or less or 2 – 3 days a month in the past 12 months), and frequent drinkers (those who drank more often than once a month). Breakfast eating habits were assessed based on whether respondents usually consumed breakfast, categorized into those who had nothing or only coffee/tea, those who had cereal (either alone or with other items), and those who had other types of breakfast foods. For the cluster analysis, all levels of behaviors were used.

For the causal analysis, these behaviors were recoded into risk categories in line with WHO recommendations for low risk health behaviors during adolescence (WHO, 2020) or based on existing evidence on the prevention of chronic diseases (Ofori-Asenso et al., 2019). Sports and exercise were each assigned to low risk if adolescents practiced frequent physical activity (≥5 times/week), moderate risk if they engaged 3 – 4 times/week, and insufficient/high risk if they participated 0 – 2 times/week. Smoking was designated as low risk for never

smokers, moderate risk for past smokers, and high risk for current smokers. Alcohol use was deemed low risk for never drinkers, moderate risk for past or rare drinkers, and high risk for moderate or frequent drinkers. Lastly, breakfast habit was dichotomized, where not skipping breakfast was assigned as low risk for those who usually consumed cereal or other foods, and skipping breakfast was assigned as high risk if they had nothing or only coffee or tea.

Effect modifier: Family financial situation

Family financial situation was determined using the parent survey in Wave I, where the adolescent's parent was asked if they had enough money to pay their bills (yes/no). In a sensitivity analysis, we examined equivalized parental gross income (bottom vs top income quintiles).

Outcome: CVDs and hypertension

Our outcome is defined as the presence of any of the following conditions: stroke, heart failure, heart disease, atrial fibrillation (collectively grouped as CVDs) and hypertension in adulthood as measured at Wave V, where respondents were aged between 33 and 43 years old. Participants reporting any of these conditions were considered as having CVDs or hypertension. We also conducted a separate analysis where hypertension is the sole outcome.

For hypertension, respondents were flagged as having the condition either through biomarkers, self-reported history of being diagnosed or being on medication. Stroke, heart failure and atrial fibrillation were identified through self-reported previous diagnosis by a healthcare professional. Heart disease was identified through self-reported previous diagnosis by a healthcare professional or history of heart surgery for clogged coronary arteries.

Confounding factors

For our causal analysis, the minimally sufficient adjustment set identified using the DAG included age at baseline survey, sex, race, adverse birth outcome, history of chronic disease diagnosis, childhood overweight, adolescent mental disorder, history of adverse social

environment, adolescent social network position, neighborhood deprivation, parental education, family financial situation in adolescence and parental smoking.

Age at baseline survey was measured at Wave I via self-reported date of birth and categorized into early adolescence (12 – 14 years) and mid-late adolescence (15 – 19 years) to reflect meaningful developmental differences in autonomy, exposure to health behaviors and susceptibility to social influences in different stages of adolescence (Sawyer et al., 2018).Sex at birth was self-reported at Wave I and categorized as male or female. Race/ethnicity was measured through self-report and interviewer-identified at Wave I and categorized into (non-Hispanic) White, (non-Hispanic) Black, Hispanic and (non-Hispanic) Other.

Adverse birth outcome was self-reported at Wave V, coded yes for those born preterm (before week 37 of gestational age) and no otherwise. History of chronic disease was self-reported at Wave V, defined as diagnosis of hypertension, diabetes, stroke, cardiac failure, cancer or asthma by a medical professional before the adolescent age at baseline (Wave I). Pre-adolescent overweight was measured as a diagnosis of obesity before the age of 16 by a medical professional, self-reported at Wave V.

Adolescent mental disorder was measured at Wave I as the presence of depressive symptoms or suicidal ideation. Depressive symptoms were measured using the frequency of experiencing three feelings: could not shake off the blues even with help from family and friends, felt depressed during the past 7 days, and felt sad during the past 7 days. Responses were scored as 0 (never or rarely), 1 (sometimes), 2 (a lot of the time), or 3 (most/all of the time). Participants were considered to have depressive symptoms if they scored 2 or higher on any of these three questions. Suicidal ideation was measured by whether the respondent seriously thought about committing suicide in the past 12 months. Respondents were classified as having a mental disorder if they had either depressive symptoms or suicidal ideation.

History of adverse social environment was defined as the presence of any of the following adversities: incarceration of a biological parent before age at baseline self-reported at Wave IV, school expulsion self-reported at Wave I, or physical/sexual abuse self-reported at Wave IV. Adolescent social network position is measured as popularity (Kim and Park, 2022), which is the number of friend nominations received by a respondent from other students in their school, collected at the in-school survey component of Wave I.

Neighborhood deprivation was measured at Wave I using a constructed neighborhood disadvantage score, based on Add Health participants' addresses when they were first interviewed in 1994 – 1995. The score was created based on five measures, namely the proportion of female-headed households, individuals receiving public assistance, adults with less than a high school education and adults who were unemployed. The score was Z-transformed to have zero mean and unit variance, and then categorized into terciles (20).

Parental education was dichotomized by either at least one parent who had graduated from a college or university or did not, using information from both the parent survey and the adolescent survey in Wave I. Parental smoking was determined using the parent survey in Wave I, based on whether there was a smoker in the household (yes/no).

Statistical analysis

We implemented the analysis in two parts, first a cluster analysis followed by the causal analysis. All analytical methods used Add Health's sampling design to account for unequal probability of selection and attrition.

We carried out a latent class analysis (LCA) to assess the presence of co-occurring health behaviors during adolescence. The LCA provided insights into whether health behaviors in our causal analysis should be analyzed solely as individual exposures or whether any co-occurring behavior patterns should also be considered. In other words, the aim of this analysis was to describe health behavior patterns among adolescents in our study population, not to use the latent classes as exposures in our causal analysis. Our approach is aligned with health

lifestyles theory, which conceptualizes health behaviors as co-occurring practices that emerge from the interaction of individual choices and structural conditions which often form meaningful behavioral patterns rather than independent, additive risks (Cockerham, 2005; Lawrence et al., 2017; Mollborn et al., 2021). We used the gsem package in Stata 18 to perform the LCA and determined the best number of classes by model fit statistics.

We quantified the effect modification of health behaviors on CVDs or hypertension by family financial difficulty. We estimated additive risk differences to quantify the excess burden of CVDs and hypertension across socioeconomic strata as they are commensurable to the prevalence of these diseases in the target population and are then directly interpretable for assessing the impact of potential public health interventions (Harper et al., 2010; Knol and VanderWeele, 2012). Specifically, our estimand was the difference in conditional average causal effects (cACE) between adolescents without family financial difficulty and adolescents with family financial difficulty. Each cACE represents the marginal difference in potential outcomes between counterfactual scenarios: one in which all adolescents are exposed to high risk health behavior and another in which all adolescents are exposed to low risk health behavior. As a secondary analysis, we also estimated the cACE comparing a counterfactual scenario where all adolescents were exposed to moderate risk health behavior to the counterfactual scenario where all adolescents were exposed to low risk health behavior.

To estimate the cACE, we used a doubly robust method (Robertson et al., 2021). First, we applied a multinomial logistic regression to model the polytomous exposures (sports, exercise, smoking and alcohol use) and binary logistic regression for the dichotomous breakfast skipping variable. We used these models to compute stabilized inverse probability weights, which account for measured confounding and ensure balance across exposure groups (see Fig. S2 in the Supplementary Material for assessment of weights distribution and of exposure balance before and after weighting). Second, we performed weighted logistic regression to predict the outcomes with the exposure and family financial situation (and their product term), and the measured confounding factors. Internal validity of the estimates requires

various assumptions: no residual confounding, consistency, positivity, no measurement error, and correct specification of either the exposure or the outcome regression models (Westreich, 2020). We measured effect magnitudes on the additive scale via risk differences (RD) per 100 persons or percentage for each cACE and their difference (Knol and VanderWeele, 2012).

Compatibility intervals (CI) (Cole et al., 2021) were generated by 1000 bootstrapped sampling designs with the Rao-Wu-Yue-Beaumont method (Dippo et al., 1984). Within each bootstrapped design, the effect estimates were the average of estimates from 100 imputed data sets obtained using multiple imputation by chained equations and hypothesizing missingness at random for neighborhood deprivation, adverse birth outcome, adolescent mental disorder, history of chronic diseases, history of social adversity, parental education, household smoking, childhood overweight, and adolescent social network position (see Table 1 for the numbers missing that were imputed and supplementary material for more details on the imputation model). Causal analyses were conducted in R version 4.4.0.

Sensitivity analyses

To assess sensitivity of findings when using a different operationalization of the effect modifier, we ran the analysis when financial situation was measured through levels of equivalized parental gross income (bottom income quintile vs top income quintile). Additionally, to assess sensitivity of our causal estimates to unmeasured confounding, we estimated differential effects on a negative control outcome (Lipsitch et al., 2010), that we identified in the self-reported abstention from voting in political elections.

Results

Characteristics of the analytic sample

Table 1 summarizes the characteristics of adolescents in our causal analytic sample, with weighted percentages provided in parentheses. At baseline, 72.5% of adolescents were aged 15 – 19 and 52.1% were female. The racial/ethnic composition was predominantly White

(72.4%), followed by Black (15.1%), Hispanic (8.4%) and other (4.1%). In terms of early life health conditions, 7.3% had childhood obesity, 10.5% had a history of chronic disease, and 22.5% had an adolescent mental disorder. Most adolescents were from socioeconomically advantaged backgrounds, with 66.6% of them from highly educated parents and 85.1% from families without financial difficulty. Approximately half of adolescents had high risk levels of physical activity (55.5% no sports or low frequency and 49.3% no exercise or low frequency). Almost a quarter of adolescents reported to be current smokers (24.6%). 29.2% of adolescents engaged in high risk alcohol use. Almost one in five adolescents regularly skips breakfast (19.9%). After an average follow-up of 21 years in early midlife, the prevalence of CVDs and hypertension among the adults aged 33 – 43 was 32.9%.

Infrequent sports and breakfast skipping were more common among adolescents from families with financial difficulty (66.2% and 23.3%, respectively) compared to their advantaged peers (57.1% and 18.8%, respectively), while smoking and alcohol use showed minimal differences between the groups (Supplementary Table S2). Finally, the prevalence of CVDs and hypertension was higher among adolescents from families with financial difficulty compared to their advantaged peers (36.6% and 29.7%, respectively).

Table 1. Summary of causal analytic sample characteristics

Characteristic	n	Causal analytic sample (n = 4452) (weighted %)
Covariates		
Age category		
Early adolescence (12 – 14)	1038	27.5
Mid-late adolescence (15 – 19)	3414	72.5
Sex		
Female	2669	52.1
Male	1783	47.9
Race/ethnicity		
White	2867	72.4
Black	807	15.1
Hispanic	534	8.4
Other	244	4.1
Neighborhood deprivation		
High	1165	29.8
Medium	1360	35.2
Low	1722	35.0
Missing	205	
Adverse birth outcome		
Yes	402	7.7

No	4027	92.3
Missing	23	
Adolescent mental disorder		
Yes	1062	22.5
No	3383	77.5
Missing	7	
History of chronic diseases		
Yes	503	10.5
No	3940	89.5
Missing	9	
History of social adversity		
Yes	602	14.2
No	3643	85.8
Missing	207	
Parental education		
Low	1350	33.4
High	3094	66.6
Missing	8	
Household smoking		
Yes	1849	44.5
No	2596	55.5
Missing	7	
Childhood overweight		
Yes	315	7.3
No	4097	92.7
Missing	40	
Adolescent social network position		
Not popular	1888	58.6
Popular	1357	41.4
Missing	1207	
Family financial situation		
With financial difficulty	731	14.9
Without financial difficulty	3721	85.1
Exposures		
Sports frequency		
High risk – infrequent sports		
None	1362	27.6
Low frequency	1245	27.9
Moderate risk		
Moderate frequency	812	20.7
Low risk		
High frequency	1033	23.8
Exercise frequency		
High risk – infrequent exercise		
None	676	15.9
Low frequency	1463	33.4
Moderate risk		
Moderate frequency	1057	21.5
Low risk		
High frequency	1256	29.2
Cigarette smoking		
High risk – current smokers		
Multiple cigarettes per day	764	18.9
One cigarette per day	269	5.7
Moderate risk		
Past smoker	767	18.2
Low risk		
Never smoker	2652	57.3
Alcohol use		
High risk		
Frequent drinker	343	7.2

Moderately frequent drinker	923	22.0
Moderate risk		
Rare drinker	784	18.2
Past drinker	376	8.6
Low risk		
Never drinker	2026	44.0
Breakfast habit		
High risk		
Breakfast skipping	871	19.9
Low risk – not breakfast skipping		
Has cereal at minimum	1252	25.4
Has other breakfast foods	2329	54.7
Outcomes		
Hypertension or CVDs		
Yes	1348	32.9
No	3076	67.1
Missing	28	
Hypertension		
Yes	1322	32.1
No	3129	67.9
Missing	1	
CVDs		
Yes	98	2.1
No	4297	97.9
Missing	57	

Clustering of health behaviors

The LCA identified two classes of health lifestyles among the Wave I Add Health adolescents (see Supplementary Material). Class 1 represents 60% of adolescents, characterized by high probabilities of engaging in healthier behavior, specifically never drinking alcohol (0.69), never smoking (0.82), and having breakfast usually (0.86). Class 2 represents 40% of adolescents, who exhibit low probability of engaging in healthier behaviors, particularly for never and past smoking/alcohol use (0.40 and 0.13 respectively).

Based on the LCA, alcohol use and cigarette smoking appear to co-occur, whereby adolescents who have frequent or moderately frequent alcohol use are also more likely to be current cigarette smokers and vice versa for never or past smoking/alcohol. Similar patterns of behavior clustering were observed with a three-class model and a four-class model (Supplementary Material). Guided by the results of the LCA, we analyzed the effect of concurrent smoking and alcohol in addition to the single health risk behaviors in our causal

analysis. Exposure to concurrent smoking and alcohol use was operationalized as high risk for being a current smoker and a frequent or moderately frequent alcohol user (n = 648, weighted % = 15.9%), low risk for being a never smoker and never drinker (n = 1707, weighted % = 38.2%), and moderate risk otherwise (n = 2097, weighted % = 45.9%).

Differential effects of health risk behaviors on CVDs and hypertension

Estimated average causal effects of high risk health behaviors among adolescents with and without family financial difficulty are displayed in Figure 3 and Supplementary Table S5. Among adolescents from advantaged families, the effects of infrequent sport, infrequent exercise, current smoking, moderate or frequent alcohol use, breakfast skipping, and the combination of smoking and alcohol use were small or negligible (under 5%). In contrast, adolescents from disadvantaged families experienced notably larger effects for breakfast skipping, alcohol use, and concurrent smoking and alcohol use.

Specifically, breakfast skipping resulted in an additional 10.1% (95% CI: 1.6% – 18.7%) CVD or hypertension cases for those with financial difficulty, compared to an additional 4.7% (95% CI: 0.7% – 8.7%) among those without financial difficulty. Moderate or frequent alcohol use led to 10.8% (95% CI: 2.3% – 19.2%) more CVD or hypertension cases among disadvantaged adolescents compared to 1.1% (95% CI: -2.0% – 4.3%) among advantaged adolescents. The effect of concurrent smoking and alcohol use resulted in 11.4% (95% CI: 0.9% – 22.0%) among those with financial difficulty, whereas it was only 0.4% (95% CI: -4.1% – 4.8%) among adolescents without financial difficulty.

The effect modification due to family financial difficulty is displayed in Figure 4. Breakfast skipping led to an excess of 5.4% (95% CI: -3.7% – 14.6%) CVD or hypertension cases for adolescents with financial difficulties compared to those without. Moderate or frequent alcohol use had an excess of 9.7% (95% CI: 0.8% – 18.5%), while concurrent smoking and alcohol use had an excess of 11.1% cases (95% CI: 0.1% – 22.1%). Other high risk health behaviors had negligible effect modification, indicating that their effect on CVDs or

hypertension was not substantially different between socioeconomically advantaged and disadvantaged adolescents.

The analysis comparing moderate risk to low risk adolescent health behaviors showed variable patterns across exposures (Supplementary Table S6). While moderately frequent exercise and past smoking resulted in effects with small magnitudes similarly to what observed for the corresponding high risk behaviors, moderately frequent sport among adolescents from families with financial difficulty experiencing resulted in a larger effect (11.8%, 95% CI: -0.6% – 24.3%) compared to that among their advantaged peers (2.3%, 95% CI: -2.5% – 7.1%). Rare or past alcohol use resulted in a smaller effect among disadvantaged adolescents compared to that observed for moderate or frequent alcohol use, leading to a reduced magnitude of effect modification (5.2%, 95% CI: -3.4% – 13.7%). A similar pattern was observed for the joint effect of smoking and alcohol use, with an excess risk difference of 8.5% (95% CI: 0.1% – 17.0%) for socioeconomically disadvantaged adolescents. Given the heterogeneity in smoking and alcohol use patterns within the moderate risk group, we omit this contrast.

When considering hypertension as the sole outcome, the patterns of effect modification were consistent with the main analysis (Supplementary Table S7).

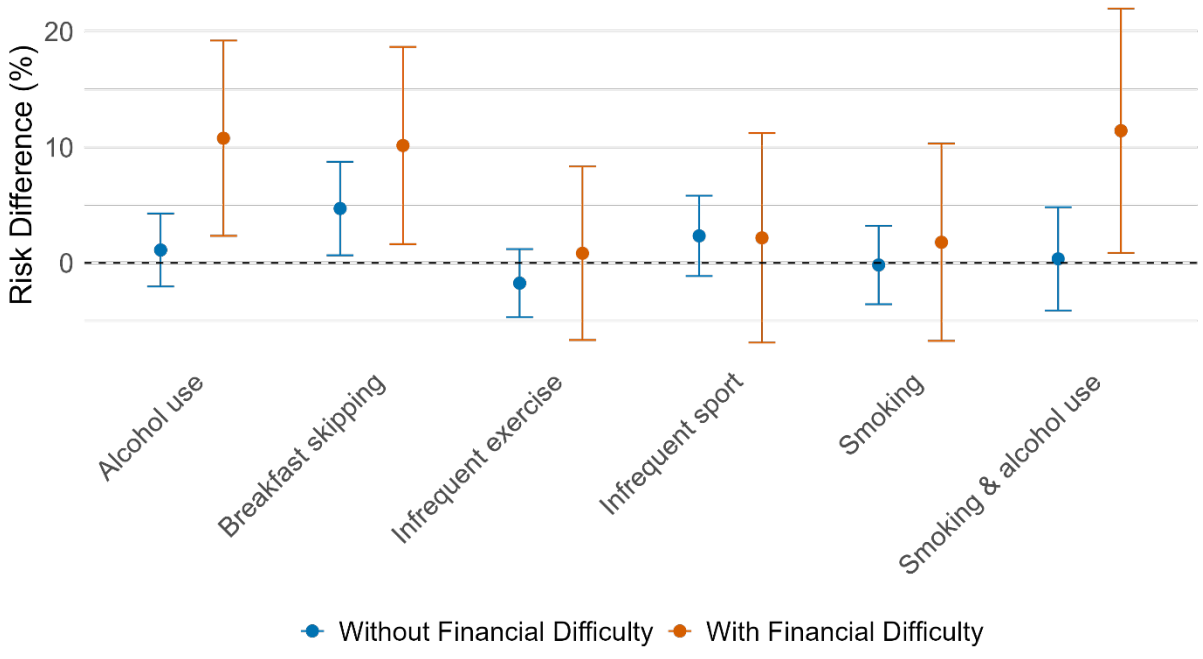


Figure 3. Average causal effect of high risk health behaviors on CVDs or hypertension among adolescents with family financial difficulty and among adolescents without family financial difficulty. Magnitude is measured via risk difference per 100 persons (% percentage). Alcohol use encompasses frequent and moderately frequent drinking. Infrequent exercise/sports encompass low frequency and no exercise/sports. Smoking encompasses current smoking (multiple cigarettes or one cigarette per day).

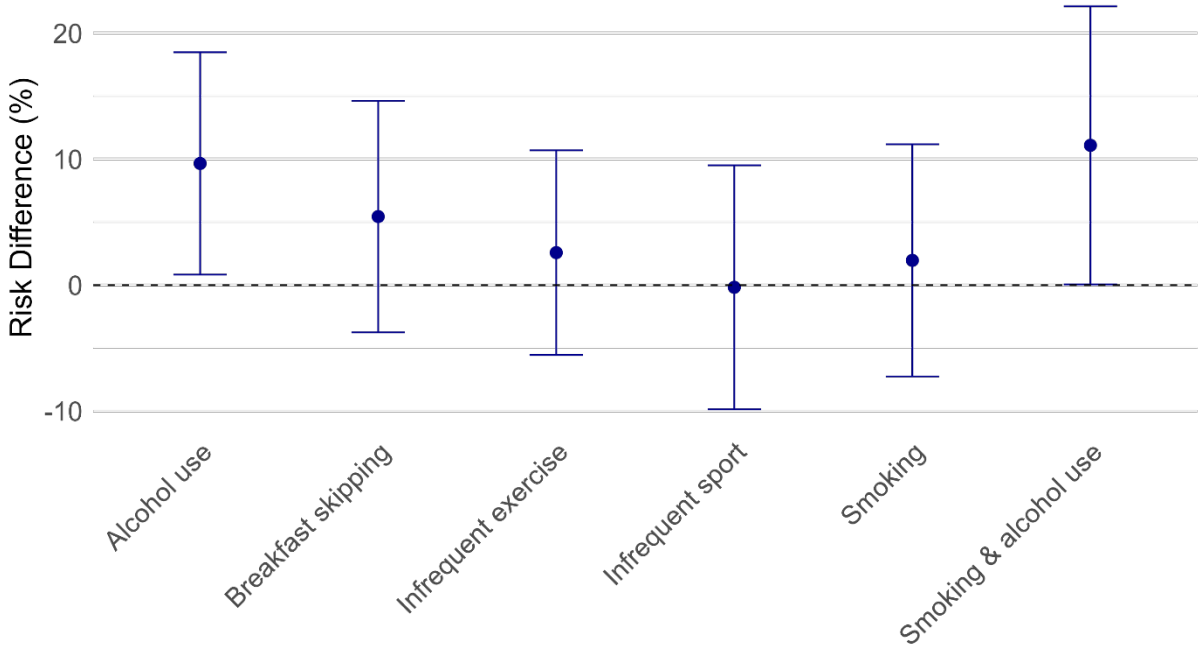


Figure 4. Effect modification associated with family financial situation. The effect modification is the difference in average causal effect between those without family financial difficulty compared to those with family financial difficulty. Alcohol use encompasses frequent and moderately frequent drinking. Infrequent exercise/sports encompass low frequency and no exercise/sports. Smoking encompasses current smoking (multiple cigarettes or one cigarette per day).

Sensitivity analyses

Substituting family financial difficulty with equivalized gross parental income as the effect modifier reaffirmed the results of our main analysis for alcohol use and concurrent smoking and alcohol use (Supplementary Figures S4 and S5). The differential effect of alcohol use was 10.8% (95% CI: 2.3% – 19.2%), which is consistent with the main analysis. Notably, there was a larger effect modification size for concurrent smoking and alcohol use compared to the main analysis, which rose to 27.3% (95% CI: 12.2% – 42.4%) when comparing the bottom income quintile group to the top income quintile group. Conversely, the effect

modification for breakfast skipping became -2.4% (95% CI: -15.4% to 10.6%), indicating variability in this estimate.

Additionally, as displayed in Supplementary Table S4, the differential effect of alcohol use during adolescence on a negative control outcome (adult abstention from voting) was -0.8% (95% CI: -8.2% – 6.7%), indicating that the main findings cannot be explained by unmeasured confounding. Similarly, the differential effect of concurrent smoking and alcohol use during adolescence on adult abstention from voting was 0.5% (95% CI: -10.3% – 11.4%), similarly indicating that the main findings are unlikely due to unmeasured confounding. In contrast, breakfast skipping showed a larger differential effect on the negative control outcome (9.1%, 95% CI: 0.6% to 17.6% among disadvantaged adolescents), which is similar to the magnitude observed in the main analysis. Thus, these results suggest that findings related to breakfast skipping could be attributable to unmeasured confounding.

Discussion

In a nationally representative sample of US adolescents, frequent alcohol consumption and concurrent cigarette smoking increased the 20-year risk of CVDs or hypertension more among adolescents from families with financial difficulty than their advantaged peers. Additionally, breakfast skipping was observed to have an unequal effect on CVDs and hypertension. However, sensitivity analysis using a negative control outcome suggested that this differential effect of breakfast skipping may be explained by unmeasured confounding factors. There was no strong evidence of differential effects of adolescent smoking and of infrequent physical activity by family financial situations.

Our main finding on the unequal effects of adolescent lifestyle pattern composed of concurrent smoking and alcohol use aligns with and expands upon existing literature. Hitherto, the evidence on differential effects of smoking and alcohol use on CVDs and hypertension is largely limited to adults. A meta-analysis of three studies reported that engaging in multiple high risk health behaviors (at least three of the following: poor diet, physical inactivity, smoking,

alcohol consumption, short sleep duration, sedentary time and television viewing time) increases the risk of cardiovascular mortality and incidence more in adults with socioeconomic disadvantage than those with socioeconomic advantage (Foster et al., 2023). Our study provides novel evidence of differential susceptibility to multiple health risk behaviors by socioeconomic situation along two dimensions: first, by identifying the specific high risk health behaviors of concurrent frequent alcohol consumption and cigarette smoking, and second, by providing evidence on adolescents. Given that concurrent smoking and alcohol use form a distinct lifestyle pattern, interventions should target both behaviors together rather than separately, particularly given the unequal effects of this pattern on cardiovascular health. Public health interventions must also consider the underlying sociocultural drivers that shape concurrent smoking and alcohol use, such as peer influences and structural factors (Bobo and Husten, 2000; Short and Mollborn, 2016).

The unequal effect of alcohol use on CVDs and hypertension for adolescents from disadvantaged backgrounds also aligns with previous discussions on the alcohol harm paradox, whereby disadvantaged groups experience disproportionate harm despite similar or lower alcohol consumption (Probst et al., 2020). Previous studies have demonstrated differential effects in alcohol-related cardiovascular mortality, with disadvantaged groups facing greater risks despite comparable consumption levels (Degerud et al., 2018; Probst et al., 2020; Zhu et al., 2024). While these studies examined alcohol use in adulthood and its impact primarily on CVD mortality, our findings contribute new evidence by showing that socioeconomic disparities in susceptibility to alcohol use emerge as early as adolescence and extend to long-term CVD and hypertension development. However, our findings differ from those of a recent Finnish cohort study which found little evidence of differential effects of adolescent alcohol use on adult cardiometabolic health (Jackisch et al., 2025). This may be partly explained by differences in the operationalization of alcohol use, where in our study, we distinguish between never drinkers and past drinkers, an important distinction given evidence that former drinkers have higher cardiovascular risk than lifetime abstainers (Fan et al., 2019).

In addition, the Finnish study examined parental education and occupation as effect modifiers, whereas the present study utilizes family financial difficulty, which may reflect more immediate material hardship. Differences in alcohol consumption patterns and health systems between Finland and the US may further contribute to the diverging observations.

When cigarette smoking was considered separately, we did not find robust evidence for differential effects. Findings on differential effects of smoking on CVDs and hypertension from previous studies are also mixed, with no clear evidence for a differential effect of smoking across socioeconomic groups (Nordahl et al., 2014; Veronesi et al., 2017; de Mestral et al., 2019).

A meta-analysis of five cohort studies reported an increased risk of CVDs associated with skipping breakfast among adults (Chen et al., 2020), supporting previous findings that (Chen et al., 2020) skipping breakfast has harmful effects for CVDs and hypertension (Ofori-Asenso et al., 2019). However, our findings were not consistent across sensitivity analyses, particularly when using a negative control outcome. One meta-analysis of two randomized controlled trials reported no effect of breakfast skipping on hypertension among adults (Bonnet et al., 2020). Additionally, a longitudinal observational study among UK adolescents aged 11 – 13 reported that after 10 years of follow-up, systolic blood pressure levels were similar between those who skipped breakfast compared to those who had breakfast regularly (Goff et al., 2019). Overall, these findings, along with our results provide limited or insufficient evidence of an effect of adolescent breakfast skipping on adult CVDs and hypertension and of its differential effect.

Similarly, infrequent physical activity, measured as either low frequency to no sport or exercise among US adolescents, did not show strong and consistent effects on adult CVDs and hypertension. This may point to either a downward bias related to social desirability-related overreporting of frequent physical activity among those who are less frequently active (Goff et al., 2019) or the crudeness of questionnaire-based assessment of physical activity (Shephard).

Future studies with more objective and thorough measurements of physical activity, for example based on accelerometer, are warranted.

Additionally, the role of differential health behavior trajectories from adolescence to adulthood is critical in understanding how cardiovascular and hypertensive disparities emerge. Adolescents from disadvantaged backgrounds are more likely to continue risky health behaviors, such as being physically inactive, smoking and drinking excessively into adulthood, while their socioeconomically advantaged peers are more likely to discontinue these behaviors as they transition into young adulthood (Andersson et al., 2022; Shao et al., 2023). These differing behavior trajectories could provide additional insights into the heightened susceptibility to joint smoking and alcohol use observed among socioeconomically disadvantaged adolescents. Future research is needed to examine this possibility.

Our study adds further understanding regarding the mechanisms underlying socioeconomic inequalities in CVDs and hypertension. Previous studies have consistently demonstrated the relevance of differential exposure to health behaviors, most notably smoking, among adults (Petrovic et al., 2018). Our findings indicate that differential susceptibility to frequent alcohol use and concurrent cigarette smoking among US adolescents, contributed to socioeconomic disparities in long-term cardiovascular diseases and hypertension. This highlights that health outcomes could be socially graded without social gradients in the prevalence of health behaviors, emphasizing the relevance of examining both mechanisms (Diderichsen et al., 2019). While these mechanisms are complementary, they offer distinct policy entry points to tackle health inequalities. In the presence of differential exposure, targeted population health promotion equalizing smoking and alcohol use would mitigate inequalities. In contrast, where differential susceptibility is present, whole-population interventions that achieve the same relative reduction of smoking and alcohol consumption across socioeconomic groups would yield greater health improvement among the disadvantaged, thus narrowing inequalities.

Our study has some limitations that should be considered. First, while we explicitly and carefully identified all potential confounding factors to the best of our knowledge to establish a causal relationship, there may still be unmeasured confounding that could result in biasing our effect estimates. Additionally, some residual confounding bias could also stem from the crude operationalization of some of the measured confounding factors. However, findings from a negative control outcome indicated that the differential effect of joint smoking and alcohol use is unlikely to be explained by residual confounding. Second, as mentioned for physical activity, reliance on self-reported data for socially sensitive behaviors as smoking and alcohol consumption may introduce reporting errors, potentially biasing upward or downward the estimated differential effect on CVDs and hypertension. Relatedly, some early life confounding factors including adolescent obesity and adverse birth outcomes were measured retrospectively in later waves, which may result in some recall bias. Third, participants without biomarker assessments at Wave V were excluded because measured blood pressure was required to define the hypertension outcome. These biomarker data were missing due to the sampling design rather than participant characteristics, so the exclusion is unlikely to bias our estimates but may reduce statistical precision. Fourth, there could be some bias from the misspecification of either the exposure or the outcome parametric models. Finally, the consistency assumption necessary for the validity of the cACE estimation might be violated as we are not measuring the effect of a specific intervention but rather a potential weighted mean effect of interventions that would change a health behavior.

Strengths of our study include the use of a nationally representative sample of US adolescents enhancing the generalizability of our findings, and the prospective design with long follow-up period of 20 years from adolescence to mid-life. Additionally, the integration of latent class analysis with contemporary methods for causal inference from observational data provides a robust approach to understanding the complex interplay between socioeconomic factors and health behaviors, offering a more comprehensive perspective on their relationship with long-term health outcomes.

Conclusions

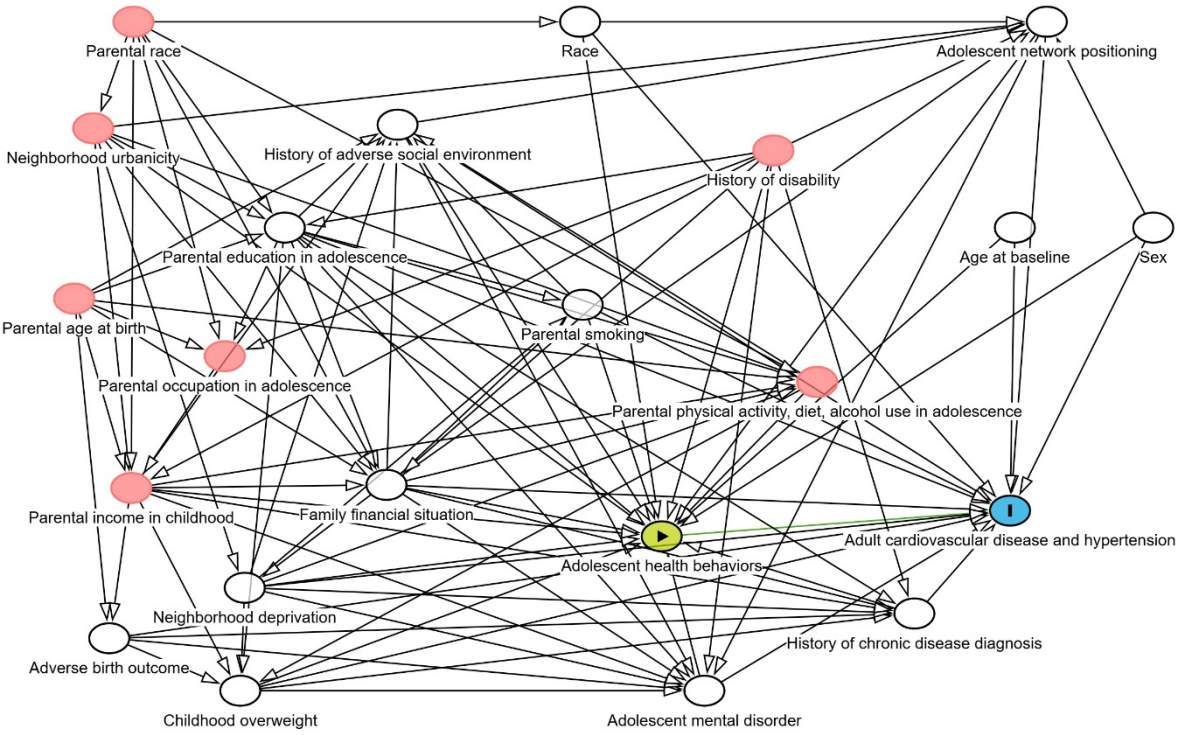
This study has two main implications for CVD and hypertension prevention. Our findings indicate that frequent alcohol use and concurrent cigarette smoking during adolescence have a more severe impact on the occurrence of cardiovascular diseases and hypertension for those from socioeconomically disadvantaged families compared to those from advantaged backgrounds. If confirmed by further research, this unequal effect of high risk behaviors points to the potential for whole-population interventions targeting alcohol use and the combination of concurrent smoking and alcohol use to mitigate inequalities in cardiovascular and hypertensive outcomes. Furthermore, acknowledging a greater susceptibility to high risk health behaviors among socioeconomically disadvantaged adolescents could guide the development of targeted policies and programs that provide additional support to these groups to address and mitigate their heightened susceptibility.

Abbreviations

cACE	Conditional average causal effect
CI	Compatibility interval
CVDs	Cardiovascular diseases
DAG	Directed acyclic graph
LCA	Latent class analysis
RD	Risk difference
UK	United Kingdom
US	United States
WHO	World Health Organization

Supplementary Materials

1. Directed Acyclic Graph (DAG)



Supplementary Fig. S1. Analytic DAG. The exposure is marked with a triangle in a green circle and the outcome with an I in a blue circle. Variables in white nodes mark the minimal sufficient adjustment set. The graph was drawn with DAGitty (1) and can be found here: https://dagitty.net/dags.html?id=fQ92WoKt

2. Add Health Questions Used for Health Behaviors

Supplementary Table S1. Add Health survey variables used to define adolescent health behavior exposures.

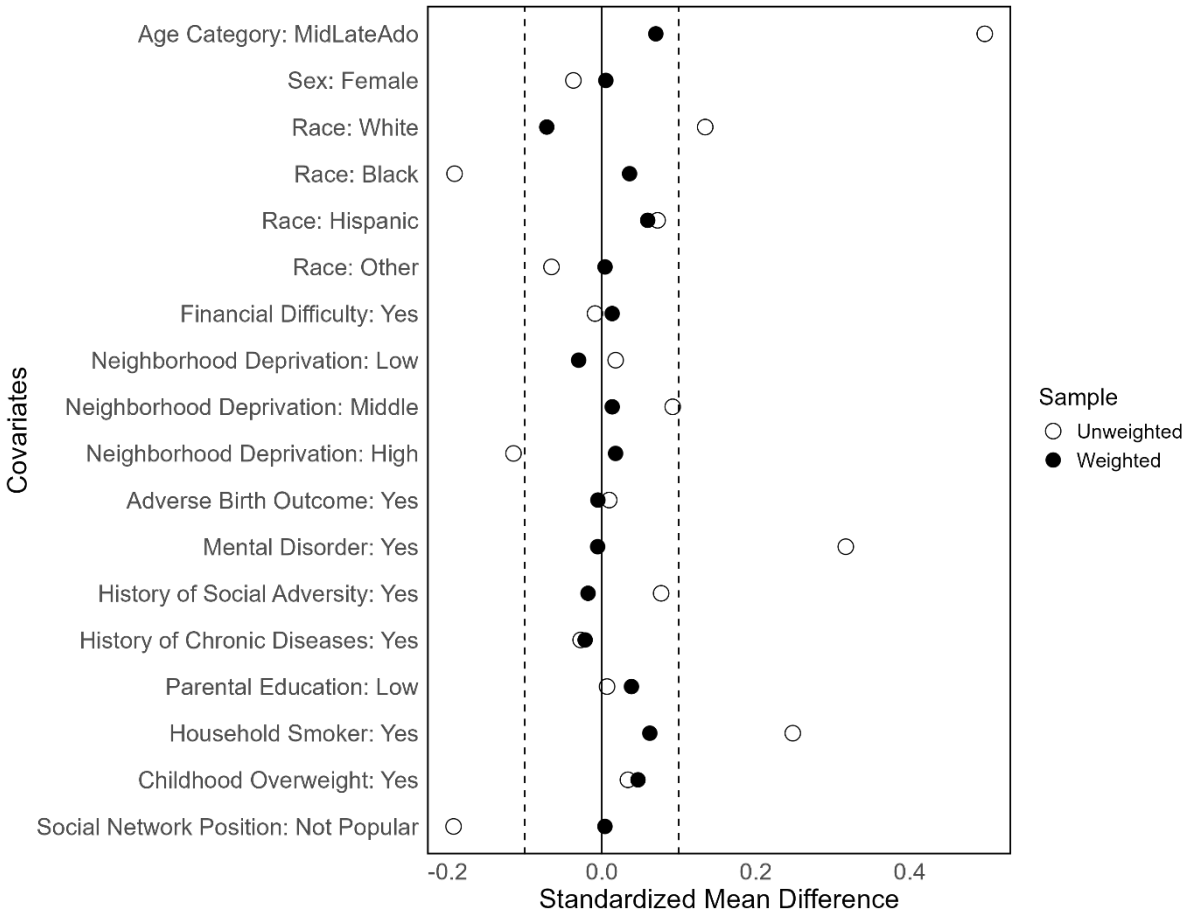
Behavior	Wave	Variable name(s)	Survey question
Sports frequency	I in-home	H1DA5	During the past week, how many times did you play an active sport, such as baseball, softball, basketball, soccer, swimming, or football?
Exercise frequency	I in-home	H1DA6	During the past week, how many times did you do exercise, such as jogging, walking, karate, jumping rope, gymnastics or dancing?
Cigarette smoking	I in-home	H1TO1	Have you ever tried cigarette smoking, even just 1 or 2 puffs?
		H1TO2	How old were you when you smoked a whole cigarette for the first time? If you have never smoked a whole cigarette, enter "0"
		H1TO5	During the past 30 days, on how many days did you smoke cigarettes?
		H1TO7	During the past 30 days, on the days you smoked, how many cigarettes did you smoke each day?
Alcohol use	I in-home	H1TO12	Have you had a drink of beer, wine, or liquor—not just a sip or a taste of someone else's drink—more than 2 or 3 times in your life?
		H1TO15	During the past 12 months, on how many days did you drink alcohol?
Breakfast habits	I in-home		What do you usually have for breakfast on a weekday morning?
		H1GH23A	Milk
		H1GH23B	Coffee or tea
		H1GH23C	Cereal

	H1GH23D	Fruit, juice
	H1GH23E	Eggs
	H1GH23F	Meat
	H1GH23G	Snack foods
	H1GH23H	Bread, toast or rolls
	H1GH23I	Other items
	H1GH23J	Nothing

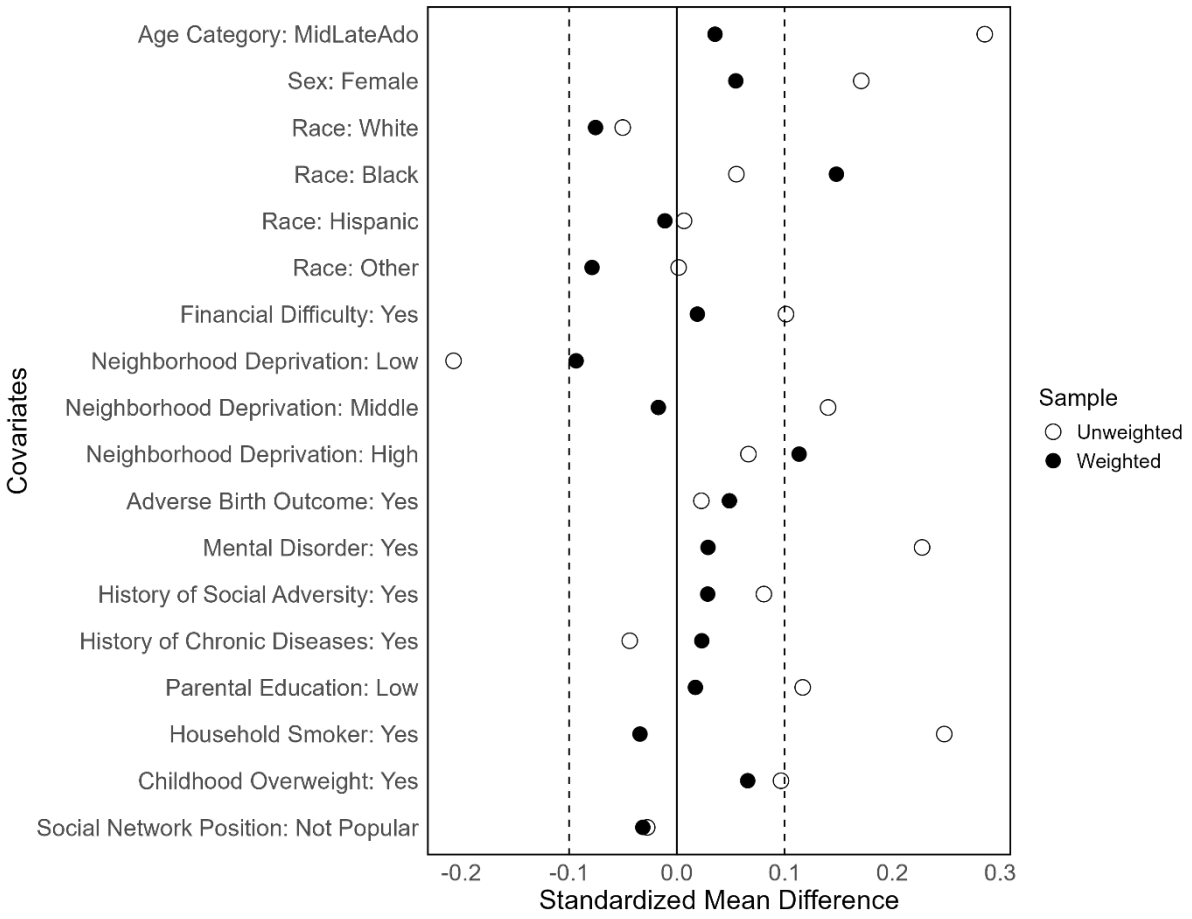
3. Standardized Mean Differences

We assessed the balance of confounders between the exposed and unexposed groups by comparing standardized mean differences (SMDs) before and after inverse probability weighting. The SMDs were calculated across 100 imputed datasets, and weights were applied to adjust for measured confounders, including age, sex, race, family financial difficulties, neighborhood deprivation, and other individual and familial factors. After applying the weights, we checked the average SMDs to ensure that balance was achieved for each behavior exposure. We conclude that a good balance was observed for all confounders (SMD <0.1).

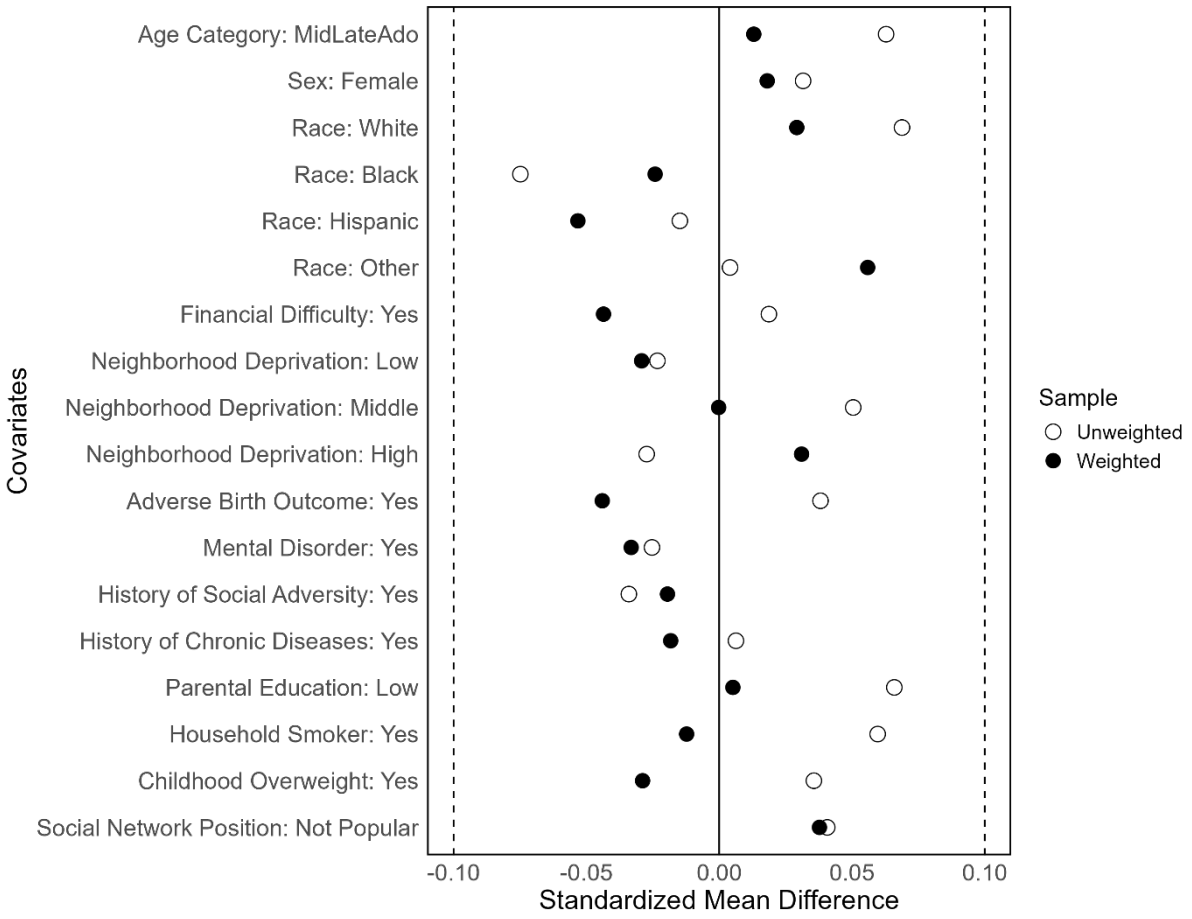
2A. Alcohol use



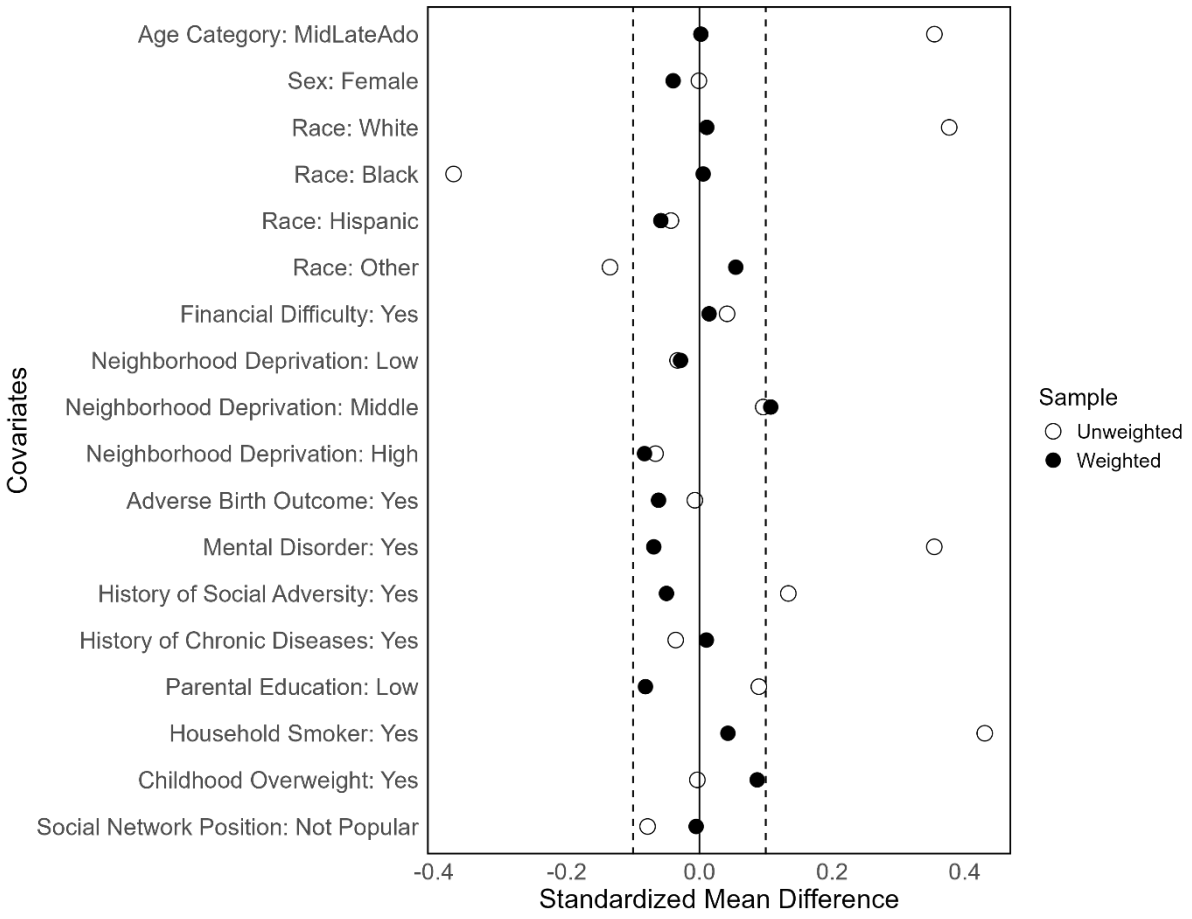
2B. Breakfast habit



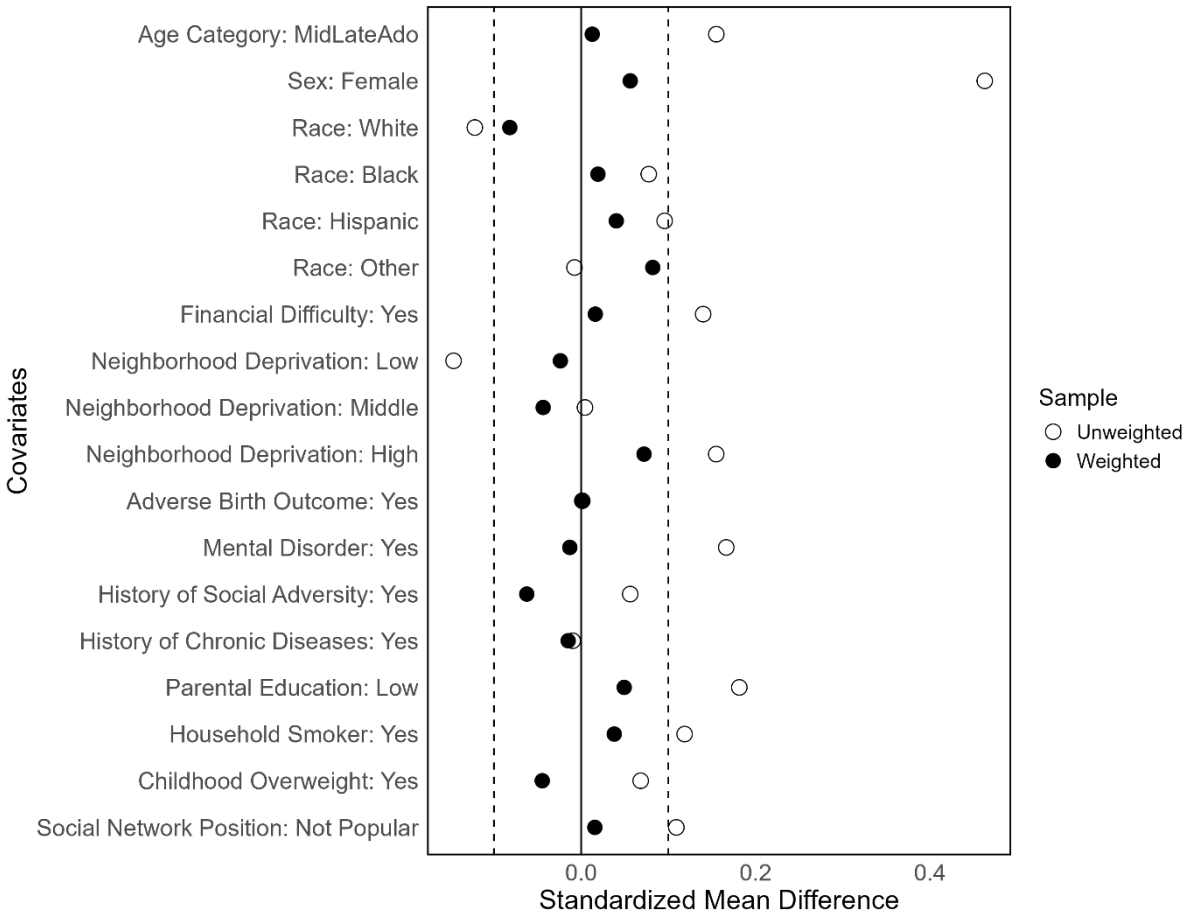
2C. Exercise frequency



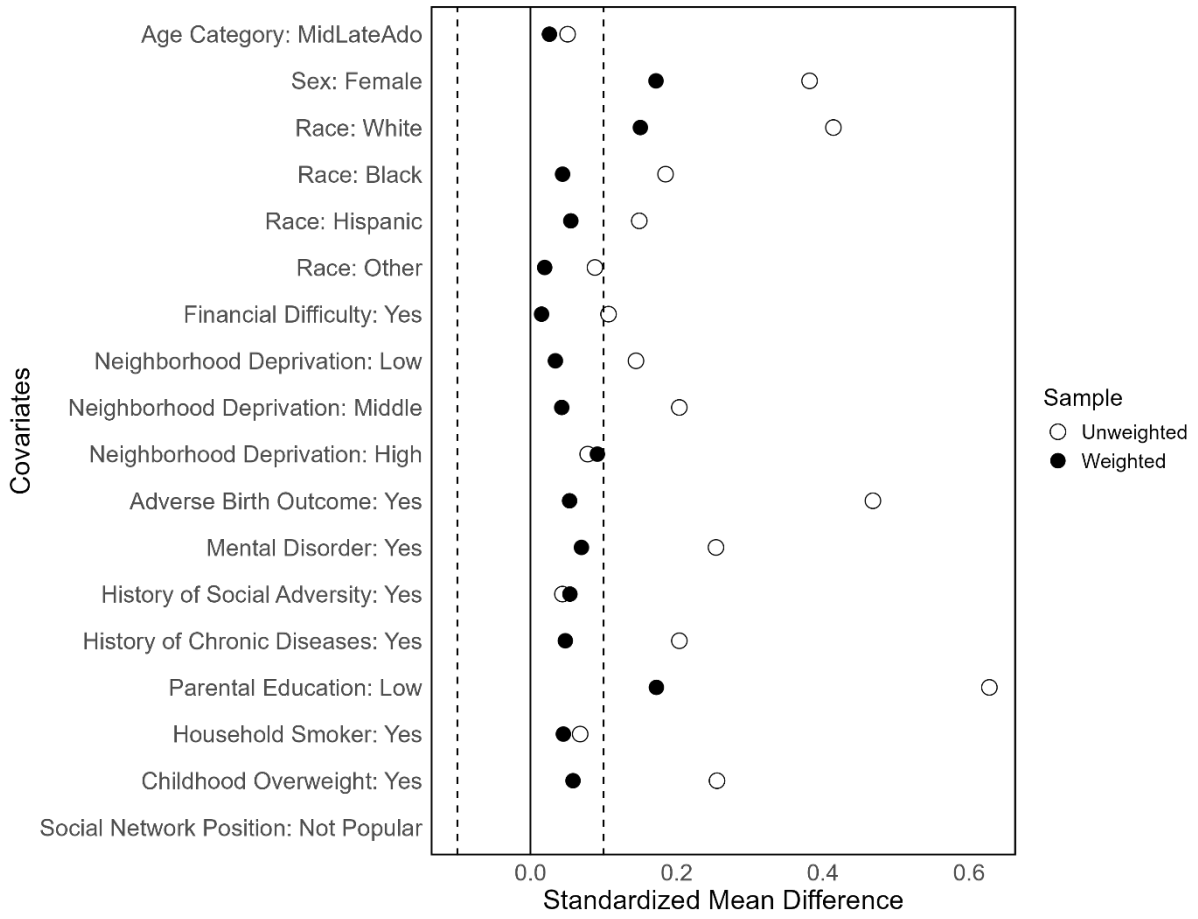
2D. Smoking status



2E. Sports frequency



2F. Alcohol use and concurrent smoking



Supplementary Fig. S2 A – F. Love plots of the covariance balance before and after inverse-probability of treatment weighting by treatment variables across the 100 imputed samples. Dashed line corresponds to abs(SMD) = 0.1, values below are usually indicative of an achieved good balance.

4. Inverse Probability Weights and Imputation Models

We used inverse probability of the exposure weights in our causal analysis. Weights were calculated from a multinomial logistic regression model relating each exposure (dependent variable) to measured confounding factors (independent variables) specified as additive and linear without product terms. Final weights were truncated at 97.5% to minimize bias arising from few participants having large weights. To assess the weights distribution, we checked whether the means were roughly 1 and that none of the weights exceeded 10. We also assessed whether the weighted population corresponded to a population in which measured confounding factors are equally distributed across exposed and unexposed groups. This was done by comparing the standardized mean differences before and after weighting (see previous section).

We specified a separate marginal structural model for each outcome variable modelled as a function of the exposure, family financial difficulties, their product term, and the measured confounding factors as specified for the exposure model. As shown by Robertson et al., this guarantees our effect or differential effect estimates are double-robust (2). We assessed the goodness of fit of these marginal structural models by estimating the Homer-Lemeshow test and the natural course outcome risk. P values of the test ranged between 0.007 and 0.02 indicating a statistically significant goodness of fit. The natural course risk of the outcome (CVDs or hypertension) was 31.6% (95% CI: 30.1% – 33.1%) for alcohol use and similarly for the other exposures. These estimates align with the observed prevalence of the outcome (see Table 1), indicating a good prediction ability of the chosen outcome model specification (3).

To deal with missingness in variables that we hypothesized were missing at random (see Table 1 and Methods), we implemented multiple imputations by chained equations using the package MICE in R (4). For each bootstrap sample, we computed 100 imputed datasets and then pooled the cACEs applying Rubin's rule (average). Predictors for the imputation were all complete covariates and three auxiliary variables that we identified in availability of cigarettes in the household reported by the adolescents or the interviewer (H1TO50 and H1IR23),

whether the adolescent participant feels safe in their neighborhood (H1NB5), and whether the adolescent participant feels socially accepted (H1PF35).

5. Inequalities in Exposures and Outcomes

Supplementary Table S2. Distribution of exposures and outcomes by effect modifier. Percentages (%) are weighted.

	Family financial difficulty			
	Yes		No	
	n	%	n	%
Sports frequency				
High risk – infrequent sports	484	66.2	2123	57.1
Moderate risk – moderate frequency	117	16.0	695	18.7
Low risk – high frequency	130	17.8	903	24.3
Exercise frequency				
High risk – infrequent exercise	350	47.9	1789	48.1
Moderate risk – moderate frequency	181	24.8	876	23.5
Low risk – high frequency	200	27.4	1056	28.4
Cigarette smoking				
High risk – current smokers	182	24.9	851	22.9
Moderate risk – past smoker	123	16.8	644	17.3
Low risk – never smoker	426	58.3	2226	59.8
Alcohol use				
High risk – moderate/frequent drinker	203	27.8	1063	28.6
Moderate risk – rare/past drinker	205	28.0	955	25.7
Low risk – never drinker	323	44.2	1703	45.8
Breakfast habit				
High risk – breakfast skipping	170	23.3	701	18.8
Low risk – not breakfast skipping	561	76.7	3020	81.2
CVDs or hypertension				
Yes	265	36.6	1097	29.7
No	460	63.4	2592	70.3
Missing	6		32	

6. Latent Class Analyses

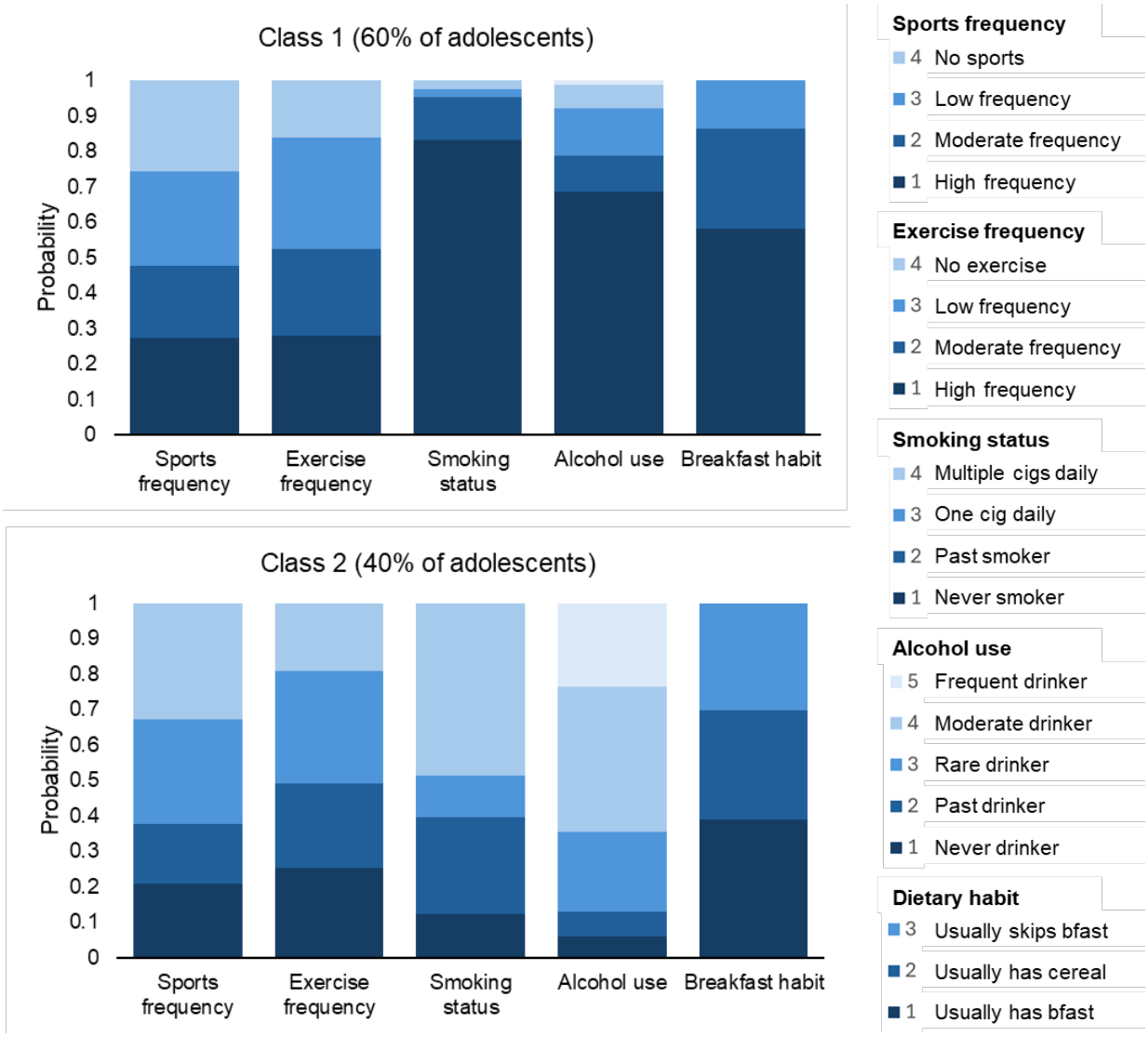
We examined the co-occurrence of behaviors within an adolescent across the participants. Sports frequency was operationalized in four levels (no sports/low/moderate/high), exercise frequency into four levels (no exercise/low/moderate/high), smoking status into four levels (multiple cigarettes daily/one cigarette daily/past smoker/never smoker), alcohol use into five levels (frequent drinker/moderately drinker/rare drinker/past drinker/never drinker), and breakfast habit into three levels (usually skips breakfast/usually has cereal at breakfast/usually has some other food for breakfast).

Latent class analysis aims to establish a categorical grouping (or latent trait) that underlies patterns of responses across the different behaviors of sports, exercise, smoking, alcohol use and diet. We fitted models from 1 – 4 classes. The latent class analysis was carried out using the gsem package in Stata 18. We compared the model fit for each of the model results (Supplementary Table S4). The conditional probabilities of health behaviors for the two-class solution, three class solution and four class solution are graphed in Supplementary Figure S3.

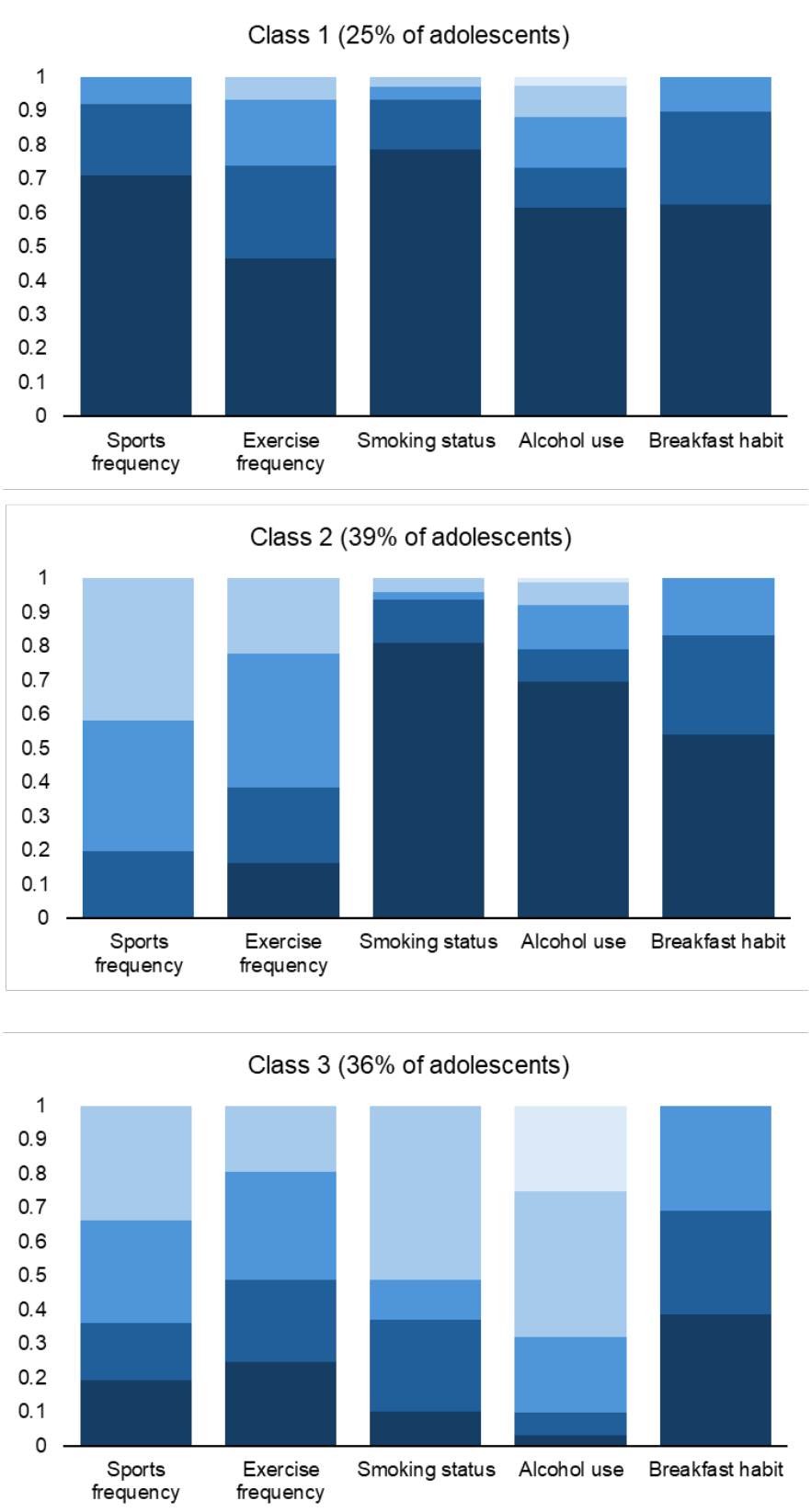
Supplementary Table S3. Model fit for 1 – 4 latent classes, n = 18,400. The best values that guided the choice of the model are in bold.

Model	**Log-likelihood**	**Resid. df**	**AIC**	**BIC**
Model 1	-138994153.2	15	277988339	277988464
Model 2	**-**135311075	31	**270622215**	**270622466**
Model 3	-135566156	47	271132409	271132786
Model 4	-136206540	63	271413208	271413710

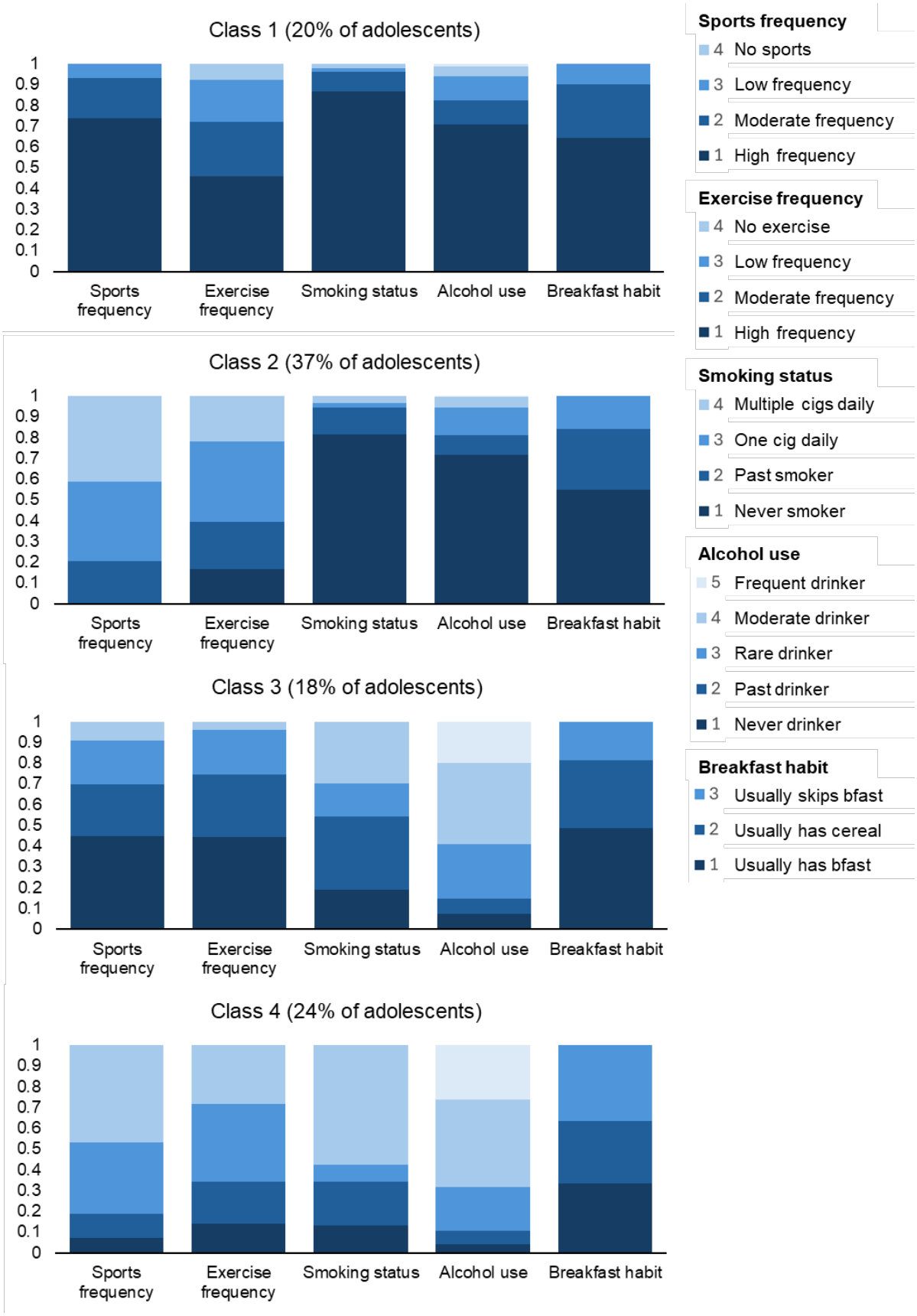
3A. Two class solution



3B. Three class solution:



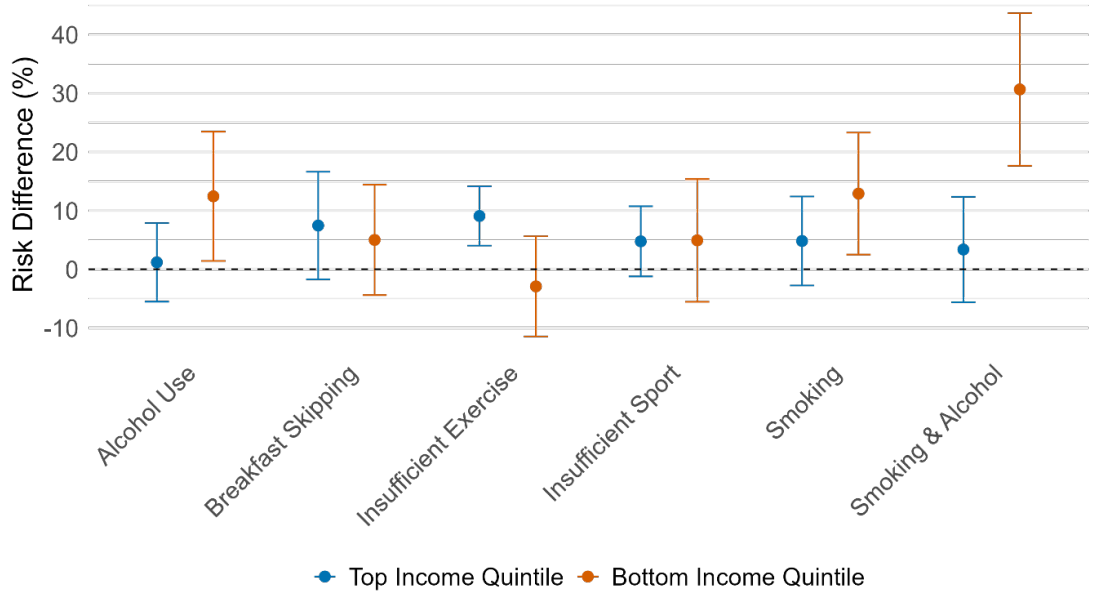
3C. Four class solution:



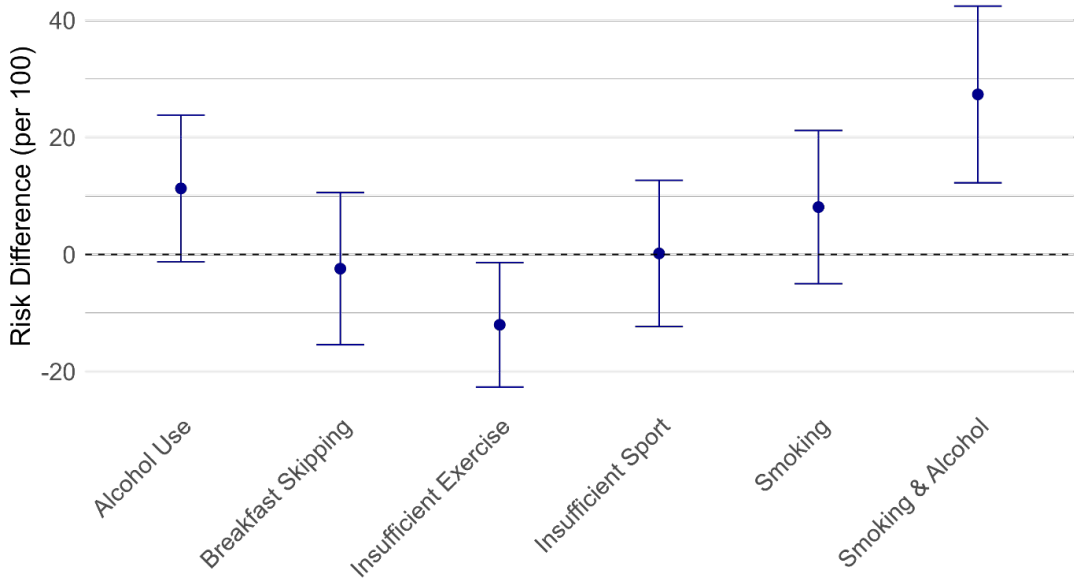
Supplementary Figure S3. Conditional probabilities of health behaviors in a two class solution, a three class solution and a four class solution.

7. Sensitivity Analyses

Using equivalized parental income (top income quintile vs bottom income quintile)
To assess the sensitivity of findings when using a different operationalization of the effect modifier, we ran the analysis when financial situation was measured through levels of equivalized parental gross income (bottom quintile vs top quintile) (n=4044).



Supplementary Fig. S4. Average causal effect of high risk health behaviors on cardiovascular conditions or hypertension among adolescents with higher parental income (top income quintile) and among adolescents with lower parental income (bottom income quintile). Magnitude is measured via risk difference per 100 persons (% percentage).



Supplementary Fig. S5. Effect modification due to parental income. The effect modification is the difference in average causal effect between those with high parental income (top income quintile) compared to those with low parental income (bottom income quintile).

Negative control outcome

To assess the sensitivity of our causal estimates to unmeasured confounding, we estimated differential effects on a negative control outcome that we identified in the self-reported voting participation in political elections. This outcome was derived from Wave V, where respondents were asked how often do they usually vote in local or statewide elections (variable H5SS8). For this analysis, we dichotomized the responses into two categories: “never” (those who responded that they never vote) and “usual” (those who reported voting sometimes, often or always).

Supplementary Table S4. Differential susceptibility to high risk health behaviors when the outcome is never/ever voting in political elections. Point estimate and 95% compatibility intervals are reported.

High risk health behaviors – Exposure	Effect among adolescents from families without financial difficulty	Effect among adolescents from families with financial difficulty	Differential effect
Frequent/moderately frequent alcohol use	3.8% (0.6% – 7.1%)	-0.8% (-8.2% – 6.7%)	-4.6% (-12.7% – 3.6%)
Breakfast skipping	-0.9% (-4.0% – 2.1%)	8.2% (0.5% – 15.8%)	9.1% (0.6% – 17.6%)
None/low frequency exercise	1.6% (-1.3% – 4.4%)	3.1% (-4.1% – 10.3%)	1.6% (-6.1% – 9.2%)
None/low frequency sport	0.8% (-2.5% – 4.1%)	-3.0% (-11.3% – 5.2%)	-3.8% (-13.1% – 5.4%)
Current smoking	4.8% (1.6% – 8.0%)	5.5% (-2.8% – 13.8%)	0.8% (-8.3% – 9.6%)
Smoking & alcohol use	3.8% (-0.6% – 8.1%)	4.3% (-5.3% – 13.9%)	0.5% (-10.3% – 11.4%)

8. Main Results Tables

Supplementary Table S5. Point estimate and 95% compatibility intervals for the cACE and the effect modification of high risk behaviors on cardiovascular diseases or hypertension, conditional on socioeconomic family financial situation as displayed in Figure 3 and 4.

Outcome	Effect among adolescents from families without financial difficulty	Effect among adolescents from families with financial difficulty	Effect modification
Frequent/Moderately frequent alcohol use	1.1 (-2.0 – 4.3)	10.8 (2.3 – 19.2)	9.7 (0.8 – 18.5)
Breakfast skipping	4.7 (0.7 – 8.7)	10.1 (1.6 – 18.7)	5.4 (-3.7 – 14.6)
None/low frequency exercise	-1.7 (-4.7 – 1.2)	0.8 (-6.7 – 8.3)	2.6 (-5.5 – 10.7)
None/low frequency sport	2.3 (-1.1 – 5.8)	2.2 (-6.9 – 11.2)	-0.2 (-9.8 – 9.5)
Current smoking	-0.2 (-3.6 – 3.2)	1.8 (-6.7 – 10.3)	2.0 (-7.2 – 11.2)
Smoking & alcohol use	0.4 (-4.1 – 4.8)	11.4 (0.9 – 22.0)	11.1 (0.1 – 22.1)

Supplementary Table S6. Point estimate and 95% compatibility intervals for the cACE and the effect modification of moderate risk behaviors on cardiovascular diseases or hypertension, conditional on socioeconomic family financial situation. Breakfast skipping is omitted as this exposure was dichotomized into high risk and low risk.

Outcome	Effect among adolescents from families without financial difficulty	Effect among adolescents from families with financial difficulty	Effect modification
Rare/Past alcohol use	-0.9 (-4.4 – 2.6)	4.3 (-3.6 – 12.2)	5.2 (-3.4 – 13.7)
Moderately frequent exercise	0.5 (-3.8 – 4.9)	-2.4 (-11.1 – 6.2)	-3.0 (-13.0 – 7.0)
Moderately frequent sport	2.3 (-2.5 – 7.1)	11.8 (-0.6 – 24.3)	9.5 (-2.9 – 21.9)
Past smoking	3.2 (-1.3 – 7.7)	6.8 (-3.9 – 17.4)	3.5 (-8.4 – 15.5)

Supplementary Table S7. Point estimate and 95% compatibility intervals for the cACE and the effect modification of high risk behaviors on hypertension only, conditional on socioeconomic family financial situation.

Outcome	Effect among adolescents from families without financial difficulty	Effect among adolescents from families with financial difficulty	Effect modification
Frequent/moderately frequent alcohol use	1.2 (-2.1 – 4.4)	8.5 (0.3 – 16.7)	7.4 (-1.6 – 16.3)
Breakfast skipping	5.2 (1.1 – 9.3)	10.7 (2.2 – 19.2)	5.5 (-3.7 – 14.7)
None/low frequency exercise	-0.6 (-3.6 – 2.4)	1.9 (-5.3 – 9.1)	2.5 (-5.5 – 10.5)
None/low frequency sport	2.3 (-1.4 – 6.0)	2.2 (-7.0 – 11.5)	-0.1 (-9.9 – 9.7)
Current smoking	0.1 (-3.2 – 3.3)	2.6 (-5.5 – 10.6)	2.5 (-6.3 – 11.3)
Smoking & alcohol use	1.0 (-3.5 – 5.4)	11.4 (1.0 – 21.9)	10.5 (-0.7 – 21.7)

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Chapter 5: Effects of Adolescent Parenting Styles on Adult Cardiovascular Disease and Hypertension: A Population-Based Cohort Study

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Introduction

Cardiovascular diseases (CVDs) and hypertension are leading causes of premature morbidity and mortality worldwide, with socioeconomically disadvantaged populations bearing a disproportionate burden (Ferrari et al., 2024). While research has focused on adult health behaviors as drivers of cardiovascular risk (Kaminsky et al., 2022), growing evidence suggests that family environments during early life shape health trajectories and contribute to the social gradient in cardiovascular outcomes (Luecken, 1998; Vedanthan et al., 2016; Doom et al., 2017; Hughes et al., 2017; East et al., 2020). Accordingly, family-based approaches have been proposed as part of primordial CVD prevention strategies (Vedanthan et al., 2016).

Parenting style is a key, potentially modifiable aspect of the family environment. Parenting styles can shape children's development (Bornstein and Zlotnik, 2008) and have long-term consequences for self-rated health, cognitive function, mental health and mortality in adulthood (Bornstein and Zlotnik, 2008; Demakakos et al., 2018; Ding and He, 2022). Baumrind's typology conceptualizes parenting along two dimensions: responsiveness (warmth, support) and demandingness (rules-setting, expectations, monitoring), yielding four styles: authoritative (high responsiveness and high demandingness,) authoritarian (low responsiveness and high demandingness), permissive high responsiveness and low demandingness), and indifferent (low responsiveness and low demandingness) (Maccoby et al., 1983; Baumrind, 1991). Authoritative parenting is generally considered most beneficial for child development, balancing emotional support with autonomy, and translating into positive psychosocial developmental outcomes and academic achievement (Lamborn et al., 1991; Steinberg et al., 1994; Wolfradt et al., 2003).

Despite extensive research on parenting and child development, evidence on long-term effects of parenting styles on cardiovascular outcomes remains limited. Existing research provides several reasons to consider parenting styles during adolescence as potentially relevant for adult cardiovascular risk. First, specific parenting components (warmth, support, consistent monitoring) have been associated with blood pressure and cardiovascular risk in adolescence and young adulthood (Luecken, 1998; Doom et al., 2016; Niu et al., 2018). Second, several longitudinal studies show parenting styles are associated with causal CVD risk factors including body mass index trajectories, health behaviors and psychosocial wellbeing from adolescence to adulthood (Lamborn et al., 1991; Steinberg et al., 1994; Hoeve et al., 2008; Fuemmeler et al., 2012; Chen and Harris, 2019; Ding and He, 2022). Overall, these findings suggest that parenting styles during adolescence may affect long-term cardiovascular risk and thus, underscoring the need for studies quantifying their long-term effects on CVDs and hypertension.

Additionally, financial strain may modify these effects by disrupting family functioning and amplifying negative consequences of suboptimal parenting. The Family Stress Model and the Family Investment Model both highlight distinct but interrelated mechanisms by which financial strain affects parenting and long-term health outcomes. The Family Stress Model emphasizes the psychological and relational strain induced by economic pressures, whereas the Family Investment Model highlights the material and educational investments that are enabled by financial resources (Conger and Donnellan, 2007; Conger et al., 2010). Thus, financial strain may lead to differential susceptibility, whereby social inequalities in health may arise when disadvantaged individuals experience greater harm from adverse exposures (Diderichsen et al., 2019). Understanding whether family financial ability modifies long-term effects of parenting styles on cardiovascular outcomes is essential for etiological research and public health practice. From a prevention perspective, evidence of differential susceptibility could inform whether population-wide or targeted family-based interventions are needed for reducing socioeconomic inequality in cardiovascular health.

The present study quantifies effects of parenting styles during adolescence on adult CVD and hypertension risk and differential effects by family financial ability.

Methods

This study was preregistered and the full study protocol has been published on the Open Science Framework (Noor et al., 2025).

Study and analytic populations

Our target population is adolescents living with parents in the United States. We used data from the National Longitudinal Study of Adolescents to Adult Health (Add Health), a nationally representative longitudinal study that followed adolescents from early adolescence into adulthood (Harris et al., 2019). Baseline data were collected through in-home interviews with both adolescents and their parents in 1994 and 1995 (Wave I; ages 12 – 21). Since then, five follow up periods of data collection have been completed, with Wave V (2016 – 2018; ages 33 – 43) being the latest wave available at the time of this study (Harris et al., 2019).

Figure 1 displays the participant flowchart. After excluding adolescents older than 19 years at baseline (n = 190), those with missing age (n = 17), and those not living with a parent or parental figure (n = 1349), the study population included 19189 adolescents aged 12 – 19. For the descriptive analysis, we further excluded respondents missing parenting items (n = 812) or Wave I design information (n = 1556), yielding 16821 participants. The causal analytic

sample comprised 4050 individuals who participated in the Wave V biomarker exam, had complete sampling design information, and had parent-reported family financial ability during adolescence. Survey weights accounting for unequal selection probabilities to biomarker collection at Wave V and attrition from Wave I to V were applied to obtain nationally representative estimates. Individuals with missing parenting items or family financial ability were excluded because we hypothesized missingness not at random for these variables.

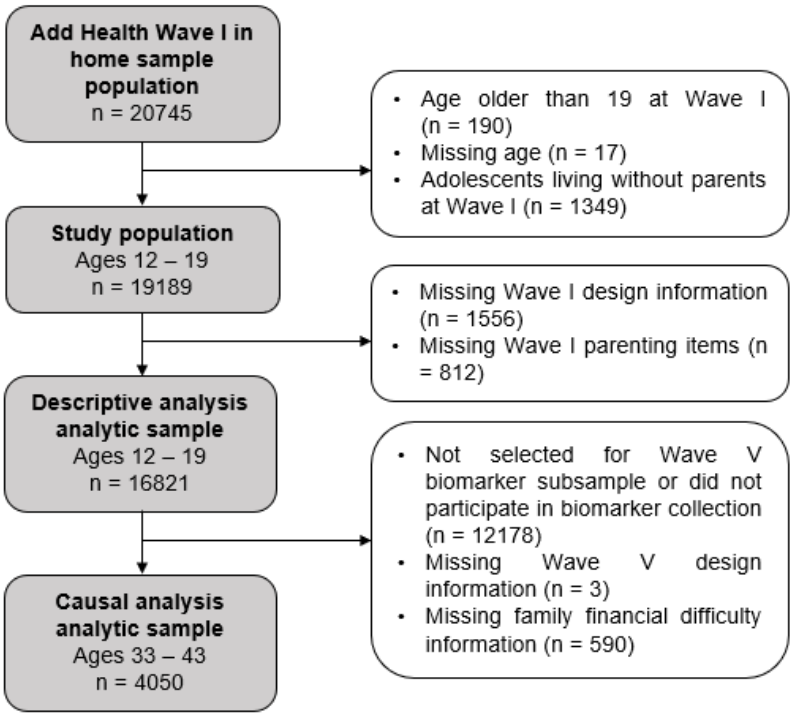


Figure 5: Flowchart of participant selection.

Measures

Exposure: Parenting style

Parenting style during adolescence was derived via latent class analysis (see Statistical analysis section 2.3.) of adolescent self-reports at Wave I of parental responsiveness and demandingness, corresponding to Baumrind's typologies. The variables used are described in Supplementary Material A).

Responsiveness was assessed through five pre-specified items capturing perceived parental warmth and involvement (Supplementary Table 1). These included how close adolescents felt to their parents, how much they believed their parents cared about them, and their perceptions of warmth and affection, communication quality, and overall relationship satisfactions. Responses were given on five-point scales ranging from 1 ("not at all") to 5 ("very

much") for closeness and caring, and from 1 ("strongly agree") to 5 ("strongly disagree") for warmth, communication, and relationship satisfaction.

Demandingness was assessed through seven pre-specified items reflecting parenting control over key aspects of the adolescent's daily lives, including curfew, friendships, clothing, hours of television watched, which television programs were watched, bedtime hours and food choices. Each item asked whether the adolescent was allowed to make their own decisions (yes/no).

Outcome: CVDs and hypertension

Our outcome is defined as the presence of stroke, heart failure, heart disease, atrial fibrillation (collectively grouped as CVDs) or hypertension in adulthood, measured at Wave V where respondents were aged between 33 and 43).

Hypertension was defined as elevated measured blood pressure, prior diagnosis, or antihypertensive medication use. Blood pressure was measured by trained field examiners using standardized protocols (see Supplementary Material B). Stroke, heart failure, and atrial fibrillation were identified through self-reported prior diagnosis by a healthcare professional. Heart disease was identified through self-reported prior diagnosis or history of heart surgery for clogged coronary arteries. We also conducted a separate analysis with hypertension as the sole outcome.

Effect modifier: Family financial ability

Family financial ability was assessed from the parent questionnaire at Wave I using the question, "Do you have enough money to pay your bills?" (yes/no). Adolescents whose parents responded "no" were classified as having family financial difficulty.

Confounding factors

Confounding factors were selected based on a causal model informed by existing literature and expert knowledge of the authors, which were established a priori and reported in the study protocol (Noor et al., 2025) (see Supplementary Material C, Fig. S1). The minimally sufficient adjustment set of confounders were age at baseline, sex, race/ethnicity, history of chronic disease, neighborhood deprivation, family structure, family-level health behaviors, parental age at birth, parental race/ethnicity, parental psychosocial stress, parental occupation, parental education, and migration background. All confounders were measured at baseline (Wave I). Detailed definitions and operationalization of all confounders are provided in the Supplementary Material D.

Statistical analysis

We conducted descriptive and causal analyses. The descriptive analysis identified parenting styles based on parental responsiveness and demandingness, which informed the causal analysis quantifying their effects on the outcome.

Descriptive analysis: We first conducted a confirmatory factor analysis to assess the dimensional structure and internal consistency of the parenting responsiveness and demandingness items. McDonald's omega was used to evaluate scale reliability, given its robustness to unequal factor loadings (Hayes and Coutts, 2020).

We then conducted an LCA to empirically identify patterns of parenting styles, with models specifying one to five latent classes were estimated in Stata 18 using weighted ordinal logistic models. The optimal number of classes was determined based on standard model fit criteria (see Supplementary Materials), while also considering class interpretability. Participants were assigned their most likely class membership based on posterior probabilities and we used these classifications as exposure in the causal analysis.

Causal analysis. We estimated the average causal effects (ACEs) of the identified parenting styles on adult CVD and hypertension, as well as the differences in these effects by family financial difficulty (conditional ACEs), using risk differences. Risk differences were chosen for their direct interpretability in relation to disease prevalence and potential intervention impacts (Harper et al., 2010). A doubly robust approach with inverse probability weighed outcome regression was implemented. Stabilized inverse probability weights were computed from an exposure regression model and truncated at the 2.5^{th} and 97.5^{th} percentiles to limit the influence of extreme values and satisfy positivity assumption (Westreich and Cole, 2010). Measured confounding balance after weighting was assessed using standardized mean differences (see Supplementary Materials E and F). Internal validity of estimates relied on assumptions of no residual confounding, consistency, no measurement error and correct model specification of either the exposure or outcome regression models.

Estimates were averaged across 100 multiple imputed datasets and compatibility intervals (CIs) were derived from 1000 bootstrapped sampling designs using the Rao-Wu-Yue-Beaumont method (see Supplementary Material G). This analysis was performed in R version 4.5.1.

Sensitivity analyses

First, to assess dependence on the data-driven latent classes, we identified parenting styles using a predefined composite approach that dichotomizes parental responsiveness and demandingness and combines them into four categories (authoritative, authoritarian, permissive, indifferent) (Driscoll et al., 2007).

Second, we used parental education as an alternative effect modifier. While parental education may be more indicative of cultural rather than purely economic capital, it is strongly correlated with parental income/financial strain in the US, and it is more stable across the life course (Oakes and Andrade, 2017).

Third, we assessed potential residual confounding using a negative control exposure, parental seatbelt use while driving. This exposure was selected as a proxy for hard-to-measure parental characteristics such as self-regulation, risk aversion and risk perception, which may influence parenting style and are also related to psychosocial stress (Sheveland et al., 2020). We assumed parental seatbelt use does not directly affect offspring CVD or hypertension risk but may share unmeasured or partially measured common causes with parenting style. Although we adjusted for parental psychosocial stress using self-reported general happiness, this measure captures only part of the construct. Therefore, an association between parental seatbelt use and offspring outcomes would suggest residual confounding by such partially measured characteristics rather than a causal effect (Lipsitch et al., 2010).

Results

Causal analysis sample characteristics

Table 1 summarizes the characteristics of adolescents in our causal analytic sample, with weighted percentages provided in parentheses. Most respondents were in mid-to-late adolescence (72.0%); 50.4% were female. The majority were White (70.0%), followed by Black (14.8%), Hispanic (11.0%) and other racial/ethnic groups (3.9%). Most (89.3%) did not have a history of chronic disease at baseline. Almost two-thirds (62.6%) of adolescents lived with both parents, 16.6% with a stepfamily and 20.8% lived with a single parent. Most parents had high education (67.0%) and manual jobs (55.2%). Most adolescents did not have a migration background (88.7% non-immigrants) and came from families without financial difficulty (83.8%). After an average follow up of 21.3 years, the prevalence of CVD or hypertension was 33.0%. Adolescents from families with financial difficulty had a higher prevalence of CVD and

hypertension compared to advantaged peers (38.8% vs. 30.9%, respectively) (see Supplementary Material H, Table S2).

Table 1. Summary of causal analytic sample characteristics

Characteristic	n	Causal analytic sample (n = 4050) (weighted %)
Covariates		
Age category		
Early adolescence (12 – 14)	962	28.0
Mid-late adolescence (15 – 19)	3088	72.0
Sex		
Female	2423	50.4
Male	1627	49.6
Race/ethnicity		
White	2636	70.0
Black	704	14.8
Hispanic	488	11.0
Other	222	3.9
Neighborhood deprivation		
High	1106	29.7
Medium	1283	32.0
Low	1476	38.2
Missing	185	
History of chronic diseases		
Yes	449	10.7
No	3594	89.3
Missing	7	
Family structure		
Living with both parents	2473	62.6
Living with stepfamily	748	16.6
Living with single parent	829	20.8
Parental alcohol consumption		
Low consumption	3108	76.5
Moderate or high consumption	934	23.5
Missing	8	
Household smoking		
Yes	1666	44.8
No	2378	55.2
Missing	6	
Parental age category		
Teen parent	475	13.2
Age 20 – 34	3300	81.7
Age 35 or older	241	5.0
Missing	34	
Parental race		
White	2697	72.4
Black	661	14.2
Hispanic	436	9.5
Other	209	3.9
Missing	47	
Parental stress		
Less stress	3897	96.7
More stress	128	3.3
Missing	25	
Parental occupation		
Non-Manual	1666	44.8
Manual	1873	55.2
Missing	511	

Parental education		
High	2841	67.0
Low	1202	33.0
Missing	7	
Migration background		
Non-immigrant	3485	88.7
Immigrant	553	11.3
Missing	12	
Family financial difficulty		
Yes	658	16.2
No	3392	83.8
Exposure		
Parenting style		
Authoritative	418	11.1
Permissive	3144	77.9
Indifferent	488	11.0
Outcomes		
CVDs or hypertension		
Yes	1229	33.0
No	2786	67.0
Missing	35	
Hypertension only		
Yes	1195	31.8
No	2854	68.2
Missing	1	

Baseline characteristics were similar between participants included in the causal analytic sample and those eligible for the Wave V biomarker assessment prior to exclusion for missing financial difficulty and parenting style items (see Supplementary Material I, Table S3).

Confirmatory factor analysis

Based on factor loadings and reliability indices (McDonald's ω = 0.89 for responsiveness; ω = 0.75 for demandingness), six of the seven pre-specified demandingness items were retained, with curfew dropped due to low factor loadings, and all five pre-specified responsiveness items were retained for the latent class analysis (see Supplementary Material J).

Latent class analysis of parenting styles

Based on model fit indices and interpretability (see Supplementary Material K), a four-class model was selected (Figure 2). One class was characterized by high responsiveness and high demandingness, which we defined as the group of adolescents who experienced authoritative parenting (11.1% of the weighted sample, Class 2 in Figure 2). Two classes (Classes 1 and 4) exhibited high responsiveness with comparatively lower demandingness, thus were grouped as permissive parenting (77.9%); while Class 4 showed high

responsiveness, both classes shared low levels of parental demandingness and did not differ meaningfully in this dimension, thus were aggregated into a single permissive category. One class showed low responsiveness and low demandingness, characterized as the indifferent parenting group (11.0%, Class 3). No class consistent with an authoritarian style was identified.

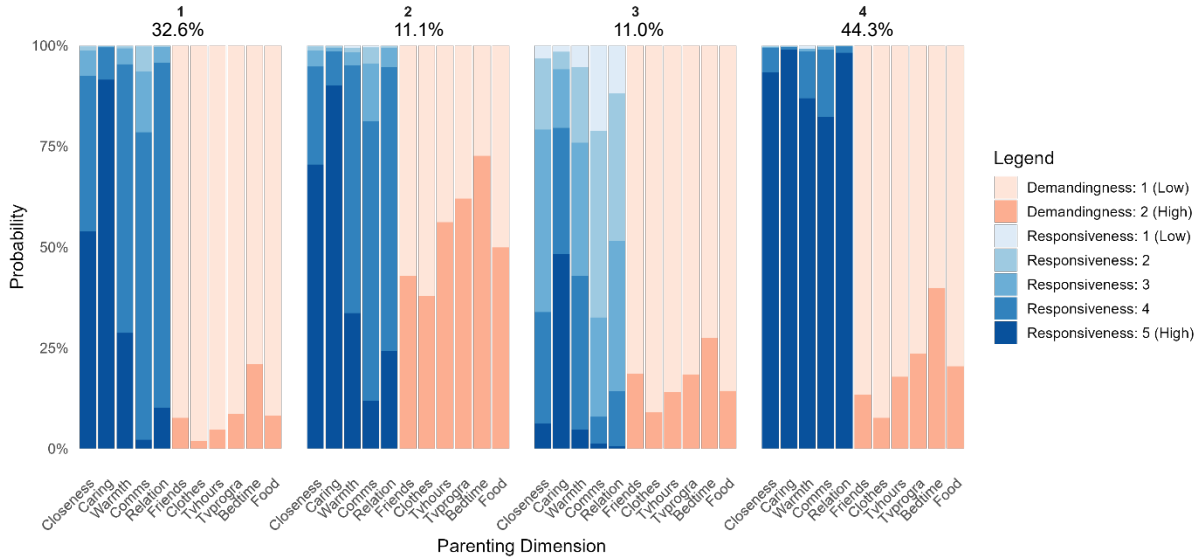


Figure 6: Item-response probabilities by latent class for five responsiveness items (perceived closeness, caring, warmth, communication quality and relationship satisfaction) and six demandingness items (control over friends, clothing, television hours, television programs, bedtime and food). Classes are labeled post-hoc as authoritative (class 2), permissive (classes 1 and 4) and indifferent (class 3).

Average and conditional causal effects of parenting styles on CVD and hypertension

Effects are shown in Figure 3. Overall, there were small risk differences for either permissive or indifferent parenting compared to authoritative parenting (-1.0%, 95% CI: -5.3% – 3.3% and -1.8%, 95% CI: -7.8% – 4.2%, respectively).

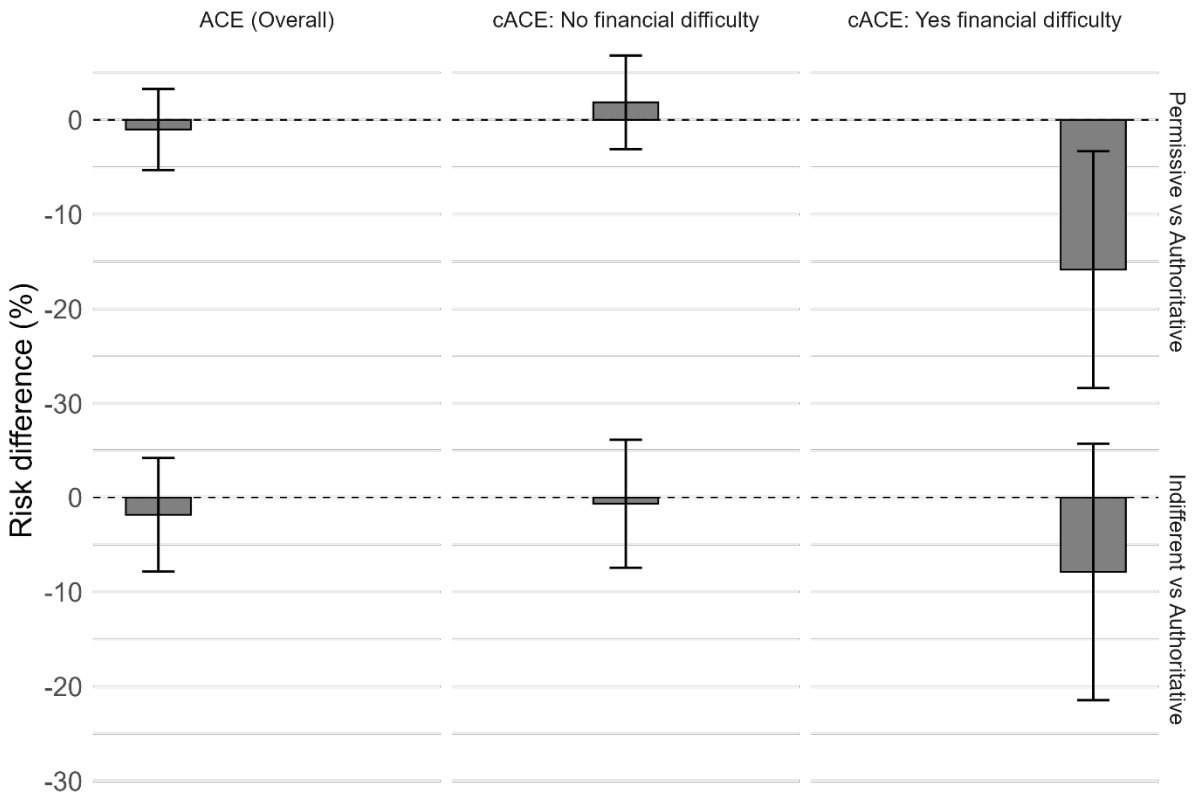


Figure 7: Average causal effect (ACE) and conditional average causal effects (cACE) of permissive vs. authoritative and indifferent vs. authoritative parenting during adolescence on CVD and hypertension in adulthood. Bars represent risk differences (%) and error bars indicate 95% compatibility intervals.

When stratified by family financial situation, a distinct pattern emerged for permissive parenting. Among families with financial difficulty, permissive parenting was associated with 15.9% fewer CVD or hypertension cases (95% CI: -28.4% – -3.3%) compared to authoritative parenting. Among families without financial difficulty, the risk difference was small (1.9% risk difference, 95% CI: -3.1% – 6.8%). The effect modification was -17.7% (95% CI: -31.2% – -4.2%), indicating that the effect of permissive parenting differed by family financial situation.

For indifferent parenting, compared to authoritative parenting, there were no clear differences by financial situation: -7.9% (95% CI: -21.5% – 5.7%) for families with difficulty and -0.7% (95% CI: -7.4% – 6.1%) for families without difficulty, with no clear effect modification (-7.2%, 95% CI: -22.4% – 7.9%).

When considering hypertension as the sole outcome, risk differences aligned with those observed for the combined CVD and hypertension outcome, although with slightly smaller magnitudes.

Sensitivity analyses

When parenting styles were defined using median splits of responsiveness and demandingness, risk differences aligned with those observed when using LCA-based parenting styles (Supplementary Material L, Figure S4).

When considering parental education as the effect modifier, effect modifications emerged in the opposite direction compared to family financial ability (Supplementary Material L, Figure S5). Lower parental education was associated with increased risk for both permissive (9.1%, 95% CI: -0.8% – 19.0%) and indifferent parenting (13.9%, 95% CI: 1.1% – 26.7%) compared to authoritative parenting.

The negative control analysis showed effect modification of similar magnitude (Supplementary Material L, Figure S6), suggesting the differential effects for permissive parenting may reflect residual confounding rather than causation (Sanderson et al., 2017).

Overall, the effect modification findings were not robust across sensitivity analyses and should be interpreted cautiously.

Discussion

In this 21-year follow-up of a nationally representative US cohort, suboptimal parenting styles during adolescence had small to negligible effects on adult CVD and hypertension risk. Permissive parenting appeared protective among families with financial difficulty, but this finding was inconsistent across sensitivity analyses and may reflect residual confounding. Indifferent parenting showed no differential effects.

Previous research has examined related constructs such as parental relationships, warmth, support and supervision during adolescence, but few studies have quantified how distinct parenting styles relate to adult CVDs and hypertension. Our small whole-population effects align with previous findings. Doom et al. (2016), using Add Health data, reported that higher maternal support in adolescence was associated with a 0.5% lower predicted 30-year CVD risk scores at ages 24 – 34 years among over 11,000 participants (Doom et al., 2016). Differences in the exposure definition (maternal warmth vs. parenting style typologies), outcome measurement (predicted risk vs. actual CVD/hypertension), and analytic approach (covariates adjustment vs causal methods) may explain variations in findings.

The absence of an authoritarian class in our LCA is also noteworthy. Two prior Add Health studies identified four distinct parenting styles aligned with Baumrind's typology (Driscoll et al., 2007; Fuemmeler et al., 2012), but used different methods: Driscoll et al. (2007)

employed median splits on warmth and control, while Fuemmeler et al. (2012) used factor mixture modeling. In our sensitivity analysis replicating Driscoll et al.'s approach, risk differences remained consistent with those obtained in our main causal analysis, indicating our effect estimates are not sensitive to the parenting style identification method.

Several factors may explain the modest effect sizes. First, parenting style typologies may obscure effects of specific traits (e.g. warmth, support or supervision individually). Second, while parenting practices have been linked to psychosocial adjustment and health behaviors in adolescence (Vedanthan et al., 2016; Chen et al., 2019) and cardiovascular outcomes in adulthood independent of adult socioeconomic conditions and health behaviors (Buchmann et al., 2010; Doom et al., 2017; Matthews et al., 2017), some longitudinal studies suggest adult socioeconomic conditions and health behaviors can mitigate these early influences (Pollitt et al., 2005; Kamphuis et al., 2013). Thus, the long-term effects of parenting styles during adolescence may be attenuated by exposures accumulated across adulthood, partly explaining our modest effect sizes.

Beyond whole-population effects, we examined whether the long-term effects differed by family financial ability. In the main analysis, permissive parenting was associated with lower CVD or hypertension risk among families experiencing financial difficulty but not among those without, suggesting differential susceptibility (Diderichsen et al., 2019). However, these differential effects had wide confidence intervals and were inconsistent across sensitivity analyses. No clear effect modification by family financial ability was observed for indifferent parenting. Overall, these findings provide limited evidence of differential susceptibility to parenting styles by family financial ability, and should be interpreted cautiously.

Our study has several limitations. Despite comprehensive confounding adjustment guided by a causal model, unmeasured or residual confounding cannot be entirely excluded, as suggested by the negative control analysis. Our measurement of demandingness relied on binary response options, which may have captured this dimension imperfectly by restricting variability. Additionally, the absence of sizeable effects could be due to insufficient measurement sensitivity. For example, the Parenting Styles and Dimensions Questionnaire provides multi-item scales validated to capture Baumrind's typologies (Robinson et al., 1995), but such instruments are often infeasible in large population-based surveys. Finally, cardiovascular outcomes were measured at ages 33 – 43 years, which may be too early to detect associations.

Our study has notable strengths. It leverages a large, nationally representative longitudinal dataset with over 20 years of follow up. The combination of LCA and doubly robust

causal inference methods provides a rigorous framework for estimating causal effects under transparent assumptions while accounting for complex survey design and missing data.

Conclusion

Our findings suggest minimal whole-population effect of parenting styles during adolescence on CVD or hypertension at ages 33 – 43, with limited and non-robust evidence of effect modification by family financial situation. Future research could assess whether more granular parenting measures, repeated assessments, or alternative pathways better capture the long-term effects of early family environments on cardiovascular outcomes. If future studies confirm the lack of differential effects by family financial situation, this would suggest that parenting style interventions during adolescence—whether population-wide or targeted to vulnerable families—may not be warranted for cardiovascular prevention.

Supplementary Materials

A. Add Health Questions Used for Parental Style

Parental responsiveness and demandingness were assessed using adolescent-reported items at Wave I. The exact questionnaire items are listed below.

Supplementary Table S1. Add Health survey variables used to identify parental style items.

Parenting dimension	**Parenting item**	**Variable name**	**Survey question**
Responsiveness	Closeness	H1WP9	How close do you feel to your mother? (1 = not at all, 5 = very much)
	Caring	H1WP10	How much do you think she cares about you? (1 = not at all, 5 = very much)
	Warmth	H1PF1	Your mother is warm and loving toward you. (1 = strongly agree, 5 = strongly disagree)
	Communication quality	H1PF4	You are satisfied with the way you and your mother communicate. (1 = strongly agree, 5 = strongly disagree)
	Relationship satisfaction	H1PF5	You are satisfied with your relationship with your mother. (1 = strongly agree, 5 = strongly disagree)
Demandingness	Curfew	H1WP1	Are you allowed to decide what time you must be home on weekend nights? (0 = no, 1 = yes)
	Friends	H1WP2	Are you allowed to decide who you hang out with? (0 = no, 1 = yes)
	Clothes	H1WP3	Are you allowed to decide what clothes you wear? (0 = no, 1 = yes)
	Television hours	H1WP4	Are you allowed to decide how many hours of television you watch? (0 = no, 1 = yes)
	Television programs	H1WP5	Are you allowed to decide which television programs you watch? (0 = no, 1 = yes)
	Bedtime	H1WP6	Are you allowed to decide what time you go to bed on weeknights? (0 = no, 1 = yes)
	Food	H1WP7	Are you allowed to decide what foods you eat? (0 = no, 1 = yes)

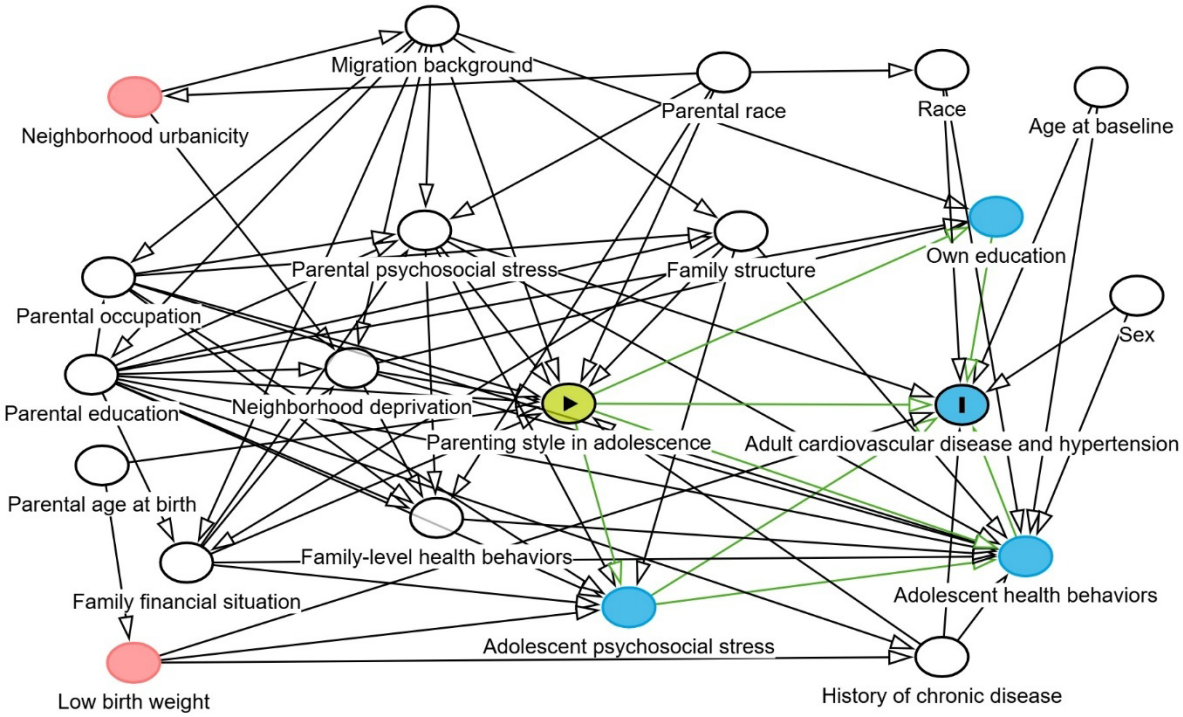
B. Hypertension classification

Hypertension status was based on an Add Health constructed variable for joint classification of hypertension, which flags respondents meeting at least one of the following criteria:

1. blood pressure classified as stage 1 or 2 hypertension using thresholds from the Seventh Report of the Joint National Committee on Prevention Detection, Evaluation, and Treatment of High Blood Pressure;
2. self-reported prior diagnosis of high blood pressure; or
3. reported use of antihypertensive medication in the past four weeks.

Blood pressure was measured three times by trained field examiners using a Microlife BP3MC1-PC-IB oscillometric monitor, and the average of the second and third readings was used to derive systolic and diastolic measures (Whitsel, Angel et al. 2020).

C. Directed Acyclic Graph (DAG)



Supplementary Fig. S1. Analytic DAG. The exposure is marked with a triangle in a green circle and the outcome with an I in a blue circle. Variables in white nodes mark the minimal sufficient adjustment set. The graph was drawn with DAGitty (Textor, van der Zander et al. 2016) and can be found here: https://dagitty.net/mDEYSZoEs

D. Details of Confounding Factors

Age at baseline survey was measured at Wave I via self-reported date of birth and categorized into early adolescence (12 – 14 years) and mid-late adolescence (15 – 19 years).

Sex at birth was self-reported at Wave I and categorized as male or female.

Race/ethnicity was measured through self-report and interviewer-identified at Wave I and categorized into (non-Hispanic) White, (non-Hispanic) Black, Hispanic and (non-Hispanic) Other.

History of chronic disease was derived from self-reported physician diagnoses of hypertension, diabetes, stroke, heart failure, cancer or asthma occurring before the baseline interview.

Neighborhood deprivation was measured using an Add Health constructed neighborhood disadvantage score linked to respondents' residential addresses at baseline, based on indicators including proportion of female-headed households, individuals living below the poverty threshold, individuals receiving public assistance, adults with less than a high school education, and adults who were unemployed. The score was standardized to have a mean of zero and standard deviation of one, and then categorized into tertiles (high, medium or low deprivation) (Belsky, Domingue et al. 2020).

Family structure during adolescence was based on adolescents' self-reported household composition at Wave I, which recorded the relationship of each household member to the respondent. Add Health constructed detailed family structure indicators categorizing whether respondents lived with both biological (or adoptive) parents, a stepparent, or a single parent (Harris 1999). For the present analysis, family structure was classified as living with both parents (biological or adoptive), living in a step-family, or living with a single parent.

Family-level health behaviors included parental alcohol consumption and household smoking. Parental alcohol consumption was reported by the responding parent and dichotomized as low (never or less than once per month) versus moderate/high (more than 2 – 3 times per month). Household smoking was coded as yes if any household member smoked.

Parental age at birth was derived from subtracting the adolescent's age at baseline from the responding parent's age, with implausible values (parents younger than 12 or mothers older than 50 years) coded as invalid. Parental age at birth was categorized into teenage parents (< 20 years), 20 – 34 years, and 35 years or older.

Parental race/ethnicity was determined from the responding parent's self-report and categorized as (non-Hispanic) White, (non-Hispanic) Black, Hispanic and (non-Hispanic) Other.

Parental psychosocial stress was proxied using the parent's self-reported general happiness ("in general, are you happy?" yes/no). Parents responding "no" were categorized as having more psychosocial stress.

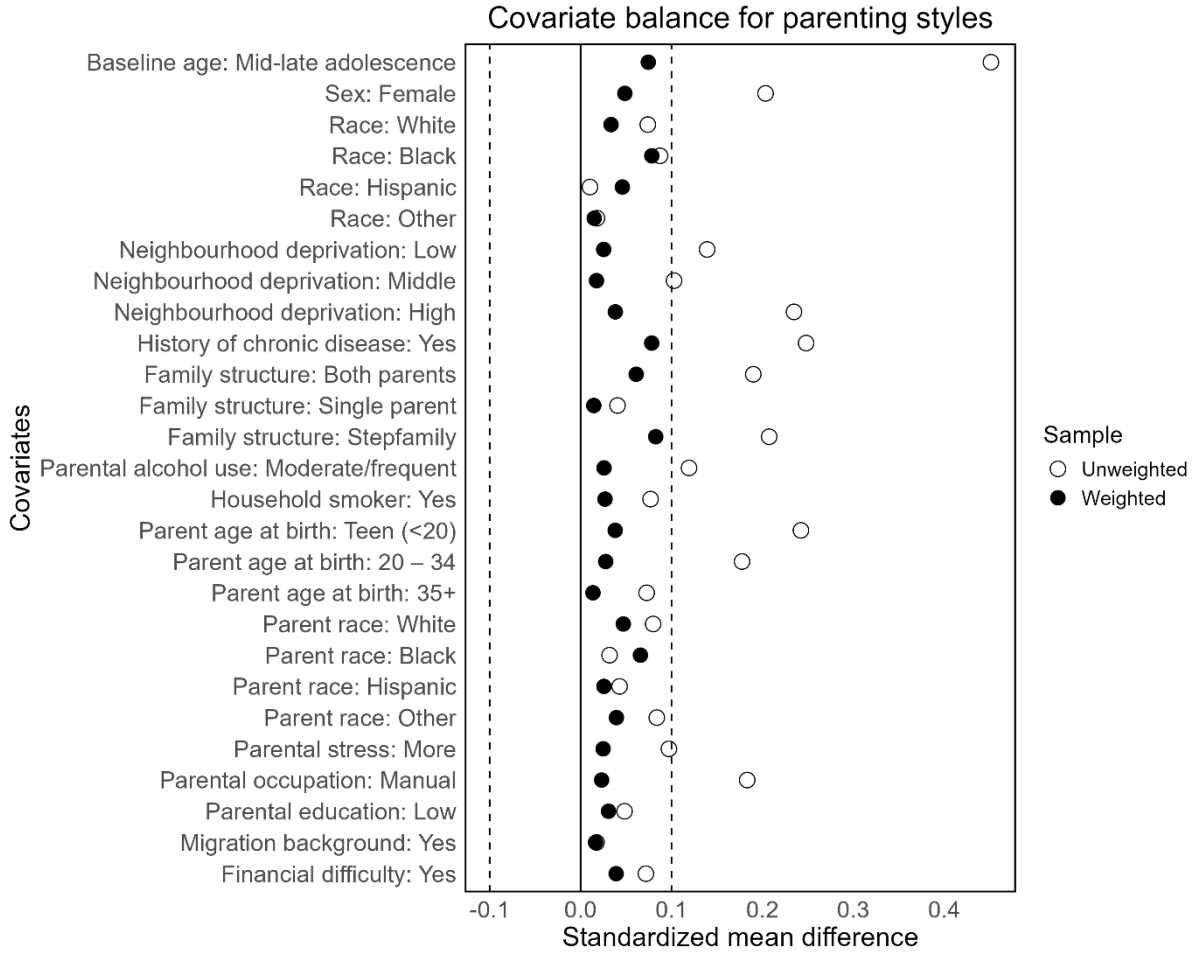
Parental occupation was based on adolescent-reported information on the current occupation of resident parents. Occupations were classified as manual (service, craft, agricultural or construction work) or non-manual (professional, managerial, technical or office work).

Parental education was coded as "high" if at least one parent had graduated from a college or university, otherwise "low", using information from both the parent survey and the adolescent survey in Wave I.

Migration background was dichotomized as immigrant (first or second generation) or non-immigrant. First-generation immigrations were respondents born outside the US, while second-generation immigrants were those born in the US with at least one foreign-born parent.

E. Standardized Mean Differences (SMDs)

We assessed covariate balance across parenting style groups by comparing standardized mean differences (SMDs) before and after applying inverse probability weighting. The SMDs were calculated for each of the 100 imputed datasets generated using multiple imputation by chained equations (mice package in R version 4.5.1) and then averaged across imputations. The SMDs were estimated using the cobalt package in R version 4.5.1, following weighting with stabilized inverse probability weights. The weighting model adjusted for measured confounders including baseline age, sex, race/ethnicity, neighborhood deprivation, family structure, parental education, parental occupation, parental alcohol use, household smoking, parental age at birth, parental race/ethnicity, parental stress, migration background and family financial difficulty. Consistent with recommendations for multi-category treatments, we considered SMD values below 0.1 to indicate adequate balance. As shown in Supplementary Figure 2 below, after weighting, SMDs were all below 0.1, which is acceptable for multinomial propensity score models (McCaffrey, Griffin et al. 2013).



Supplementary Fig. S2. Love plots of the covariance balance before and after inverse-probability of treatment weighting by treatment variables across the 100 imputed samples.

Dashed line corresponds to abs(SMD) = 0.1, values below are usually indicative of an achieved good balance.

F. Inverse Probability Weights

We used inverse probability of the exposure weights in the causal analysis. Weights were calculated from a multinomial model relating the three-category parenting style exposure (dependent variable) to measured confounding factors (independent variables), specified as additive and linear without product terms. Final weights were stabilized and truncated at the 2.5^{th} and 97.5^{th} percentiles to minimize bias arising from few participants having extreme weights. To assess the weight distribution, we checked whether the means were roughly 1 and that trimming removed excessively large values. We also assessed whether the weighted population corresponded to a population in which measured confounding factors were equally distributed across the exposure groups. This was done by comparing standardized mean differences before and after weighing (see previous section).

We specified a marginal structural outcome model for each outcome as a function of parenting style, family financial difficulty, their product term and the measured confounding factors included in the exposure model. As shown by Robertson et al., this guarantees our effect or differential effect estimates are double-robust {Robertson, 2021 #223}.

G. Imputation Models

To deal with missingness in variables that we hypothesized were missing at random, we implemented multiple imputations by chained equations using the mice package in R (van Buuren and Groothuis-Oudshoorn 2011). For each bootstrap sample, we computed 100 imputed datasets and then pooled the estimates by averaging. Predictors for the imputation included all complete covariates and several auxiliary variables that capture additional aspects of the adolescent's family and social environment: availability of cigarettes in the household reported by the adolescent or interviewer (variables H1TO50 and H1IR23 in Add Health), perceived neighborhood safety (H1NB5), perceived social acceptance (H1PF35), unmet medical care due to cost (H1GH26 and H1GH27I) and parental disability (H1RM10, H1RF10 and PA18).

H. Inequalities in Exposures and Outcomes

Supplementary Table S2. Distribution of exposures and outcomes by family financial difficulty. Percentages (%) are weighted.

	Family financial difficulty			
	Yes		No	
	n	%	n	%
Parenting style				
Authoritative	76	11.6	342	10.1
Permissive	477	72.5	2667	78.6
Indifferent	105	16.0	383	11.3
CVDs or hypertension				
Yes	238	38.8	991	30.9
No	414	61.2	2372	69.1
Missing	6		29	

J. Comparison of Analytic and Eligible Samples

Supplementary Table S3. Baseline characteristics of the eligible Wave V biomarker sample and final causal analytic sample. Percentages (%) are weighted.

Characteristic	Eligible W5 biomarker sample (n = 4,640)	Final causal analytic sample (n = 4,050)
Age category		
Early adolescence (12 – 14)	27.1%	28.0%
Mid–late adolescence (15 – 19)	72.9%	72.0%
Sex		
Female	50.8%	50.4%
Male	49.2%	49.6%
Race/ethnicity		
White	68.4%	70.0%
Black	15.7%	14.8%
Hispanic	11.6%	11.0%
Other	4.3%	3.9%
Neighborhood deprivation		
High	30.5%	29.7%
Medium	32.3%	32.0%
Low	37.1%	38.2%
History of chronic diseases		
Yes	10.6%	10.7%
No	89.4%	89.3%
Family structure		
Living with both parents	61.9%	62.6%
Living with stepfamily	16.5%	16.6%
Living with single parent	21.6%	20.8%
Parental alcohol consumption		
Low consumption	76.2%	76.5%
Moderate or high consumption	23.8%	23.5%
Household smoking		
Yes	45.1%	44.8%
No	54.9%	55.2%
Parental age category		
Teen parent	13.1%	13.2%
Age 20–34	81.8%	81.7%
Age 35 or older	5.1%	5.0%
Parental race		
White	72.0%	72.4%
Black	14.2%	14.2%
Hispanic	9.9%	9.5%
Other	3.9%	3.9%
Parental stress		
Less stress	96.8%	96.7%
More stress	3.2%	3.3%
Parental occupation		

Characteristic	Eligible W5 biomarker sample (n = 4,640)	Final causal analytic sample (n = 4,050)
Non-manual	44.3%	44.8%
Manual	55.7%	55.2%
Parental education		
High	65.6%	67.0%
Low	34.4%	33.0%
Migration background		
Non-immigrant	87.8%	88.7%
Immigrant	12.2%	11.3%

K. Confirmatory Factor Analysis (CFA) and Scale Reliability

We conducted a CFA to assess the dimensional structure of the parenting responsiveness and demandingness constructs. The CFA was performed using the lavaan package in R version 4.5.1 using the weighted least squares mean and variance (WLSMV) adjusted estimator appropriate for ordered categorical items (Xia and Yang 2019). Two latent constructs were tested: responsiveness, reflecting parental warmth and support, and demandingness, reflecting parental control and monitoring. Internal consistency was subsequently assessed using Cronbach's alpha (α) and McDonald's omega (ω) using the psych package, also in R version 4.5.1.

For responsiveness, five items capturing perceived closeness, caring, warmth, communication quality, and overall relationship satisfaction loaded onto a single latent factor (standardized factor loadings 0.75 – 0.96). Model fit indices were as follows: Comparative Fit Index (CFI) = 0.995, Tucker-Lewis Index (TLI) + 0.991, Root Mean Square Error of Approximation (RMSEA) = 0.118, and Standardized Root Mean Square Residual (SRMR) = 0.066. The internal consistency for responsiveness was high (α = 0.85, ω = 0.89).

For demandingness, an initial CFA including seven items showed that the curfew item had weak loading on the demandingness factor. Given its comparatively poor psychometric performance, this item was excluded from the final measurement model. The retained six demandingness indicators, reflecting parental control over adolescents' daily decisions (friends, clothing, television hours, television programs, bedtime and food choices) loaded onto a single latent factor (standardized loadings 0.55 – 0.78). Model fit indicators were CFI = 0.975, TLI = 0.959, RMSEA = 0.051 and SRMR = 0.059. Reliability was acceptable (α = 0.63, ω = 0.75).

Consistent with recent methodological guidance, we did not evaluate fit against fixed cutoff values (e.g. CFI > 0.95, RMSEA < 0.06), which were developed for maximum-likelihood estimation with continuous data and are not valid for ordinal data analyzed with WLSMV(Xia and Yang 2019). Under WLSMV, CFI and TLI typically appear higher and RMSEA lower than under ML, so fit indices must be interpreted in context rather than relying solely on fixed cut-offs (Xia and Yang 2019, Goretzko, Siemund et al. 2024). Given the strong and coherent loadings and good reliability, both constructs were considered adequately unidimensional and consistent with Baumrind's theoretical dimensions of responsiveness and demandingness. These constructs were therefore used as indicators to derive empirically defined parenting styles in the latent class analysis.

L. Latent Class Analysis (LCA)

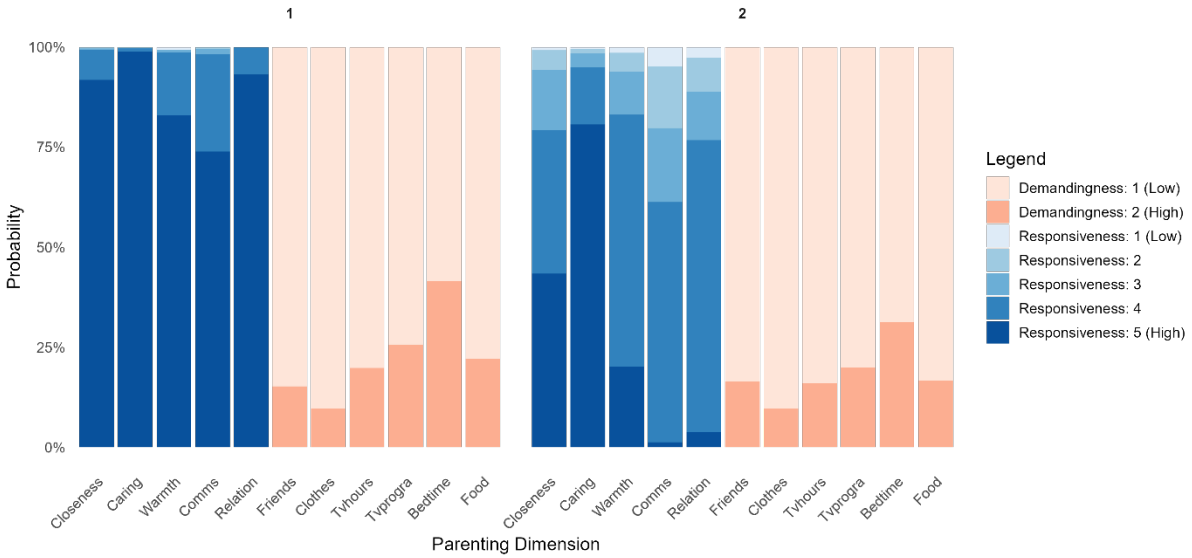
We conducted a latent class analysis (LCA) to empirically identify patterns of parenting styles, based on Baumrind's two underlying constructs of parental responsiveness and demandingness. Responsiveness was measured using five adolescent-reported items (closeness, caring, warmth, communication and satisfaction with the relationship), and demandingness was measured using six items capturing parental rules or control (friends, clothing, television hours, television programs**,** bedtime and food). All items were treated as ordinal indicators.

Models specifying one to five latent classes were estimated in Stata 18 using the gsem package. Model fit was evaluated using the Akaike Information Criterion (AIC), Bayesian Information Criterion (BIC), adjusted BIC (aBIC) and entropy, while also considering the interpretability and stability of the resulting classes. Fit statistics for all candidate models are presented in Supplementary Table S4. The conditional probabilities of parenting styles for the two-class, three-class and five-class solutions are graphed in Supplementary Figures 3A – 3C.

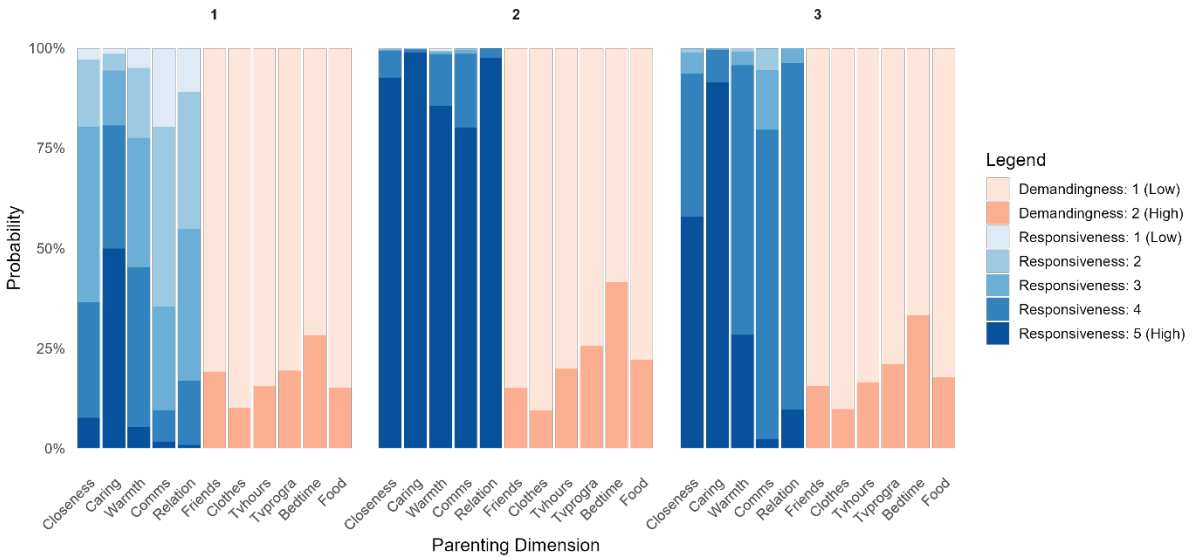
The four-class solution provided the best balance of statistical fit and substantive interpretability. After selecting the optimal number of classes, we assigned each participant to the class with the highest posterior probability. These class assignments were used as the exposure in the causal analysis.

Supplementary Table S4. Model fit for 1 – 5 latent classes, n = 16,821. The best values that guided the choice of the model are in bold.

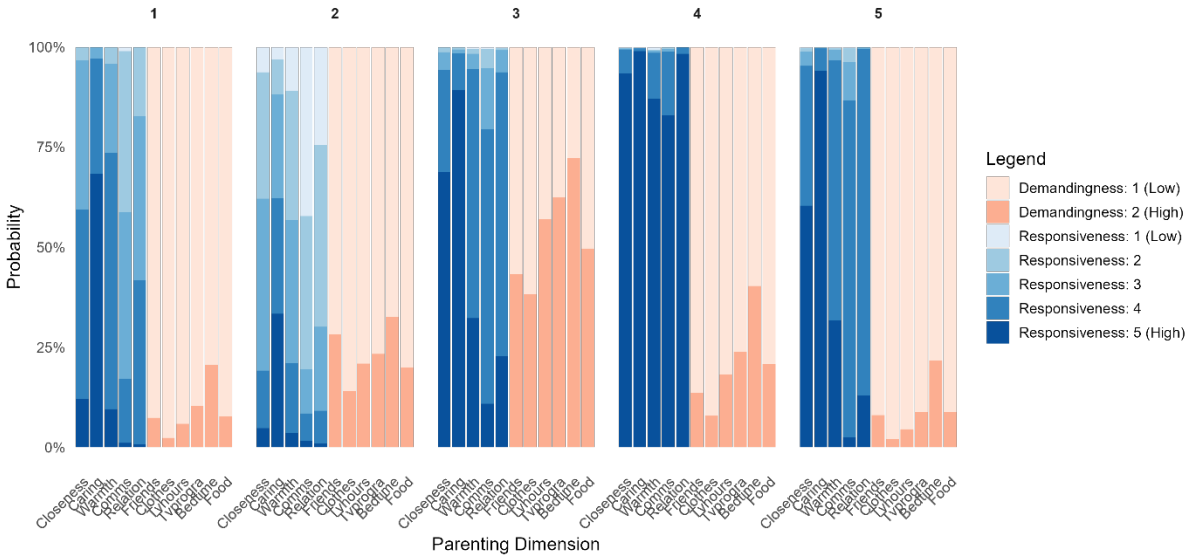
Model	**Log-likelihood**	**Resid. df**	**AIC**	**BIC**	**aBIC**	**Proportion of participants in smallest class (%)**
1	-148566024	26	297318529	297313516	297316982	N/A
2	-134637808	53	269827128	269813211	269819437	48.7
3	-129081640	80	258163510	258164784	258160873	11.8
4	**-126996488**	**107**	**254193045**	**254196351**	**254189964**	**11.0**
5	-126316338	134	252632817	252637123	252630281	5.1



Supplementary Fig. S3A. Two-class solution. Item-response probabilities by latent class for five responsiveness items (perceived closeness, caring, warmth, communication quality and relationship satisfaction) and six demandingness items (control over friends, clothing, television hours, television programs, bedtime and food).



Supplementary Fig. S3B. Three-class solution. Item-response probabilities by latent class for five responsiveness items (perceived closeness, caring, warmth, communication quality and relationship satisfaction) and six demandingness items (control over friends, clothing, television hours, television programs, bedtime and food).



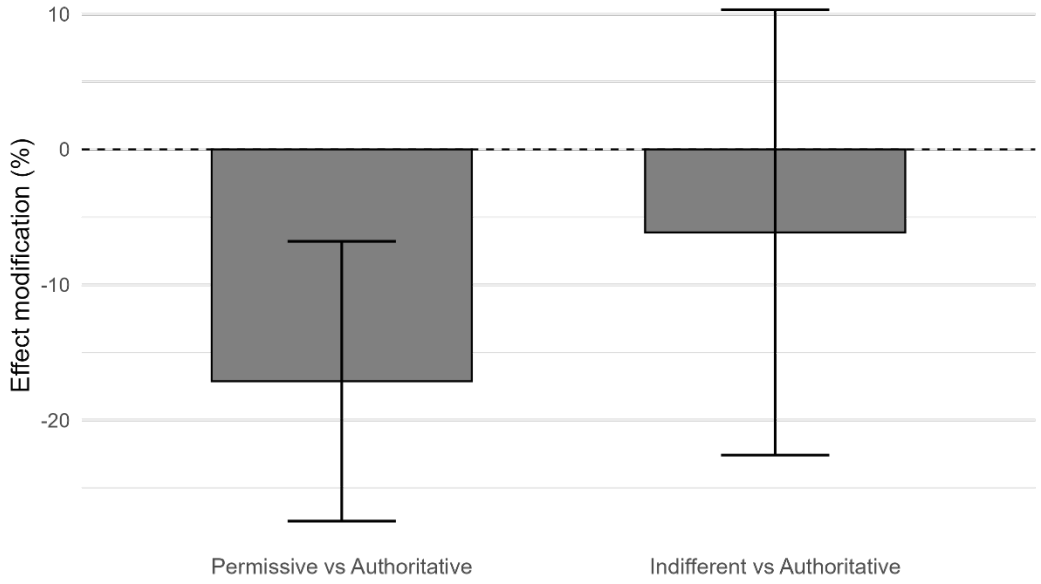
Supplementary Fig. S3C. Five-class solution. Item-response probabilities by latent class for five responsiveness items (perceived closeness, caring, warmth, communication quality and relationship satisfaction) and six demandingness items (control over friends, clothing, television hours, television programs, bedtime and food).

M. Sensitivity Analyses

We conducted three sensitivity analyses to evaluate the robustness of our causal estimates.

a. Using Predefined Composite Parenting Styles

To assess whether our findings were dependent on the data-driven latent class structure, we redefined parenting style using a predefined composite approach based on Driscoll et al. (2007). This method dichotomizes parental responsiveness and demandingness and combines them into four theoretical categories: authoritative, authoritarian, permissive and indifferent. Using this predefined typology, we repeated the causal analysis, estimating risk differences stratified by family financial difficulty and subsequently deriving effect modification estimates as the difference in conditional average causal effects (cACE) between adolescents with and without family financial difficulty.

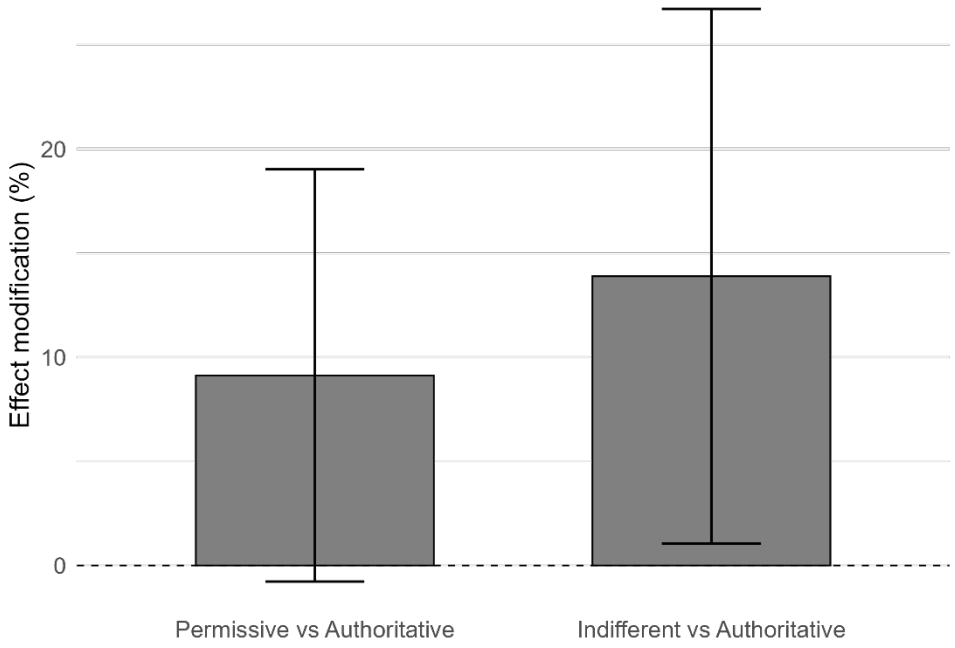


Supplementary Fig. S4. Effect modification by family financial difficulty of permissive vs. authoritative and indifferent vs. authoritative parenting during adolescence on adult CVD or hypertension, using predefined composite parenting styles based on median splits of responsiveness and demandingness. Bars represent effect modification estimates (%) comparing cACEs of adolescents with financial difficulty to those without financial difficulty; error bars indicate 95% compatibility intervals.

b. Using Parental Education as an Alternative Effect Modifier

To examine whether results differ when using an alternative effect modifier, we repeated the analysis using parental education instead of family financial ability. Parental education was defined as the highest educational attainment of either parent, derived from the Wave I parent interview and adolescent survey, and categorized as high (at least one parent graduated from a college or university) versus low education. We repeated the causal analysis estimating risk

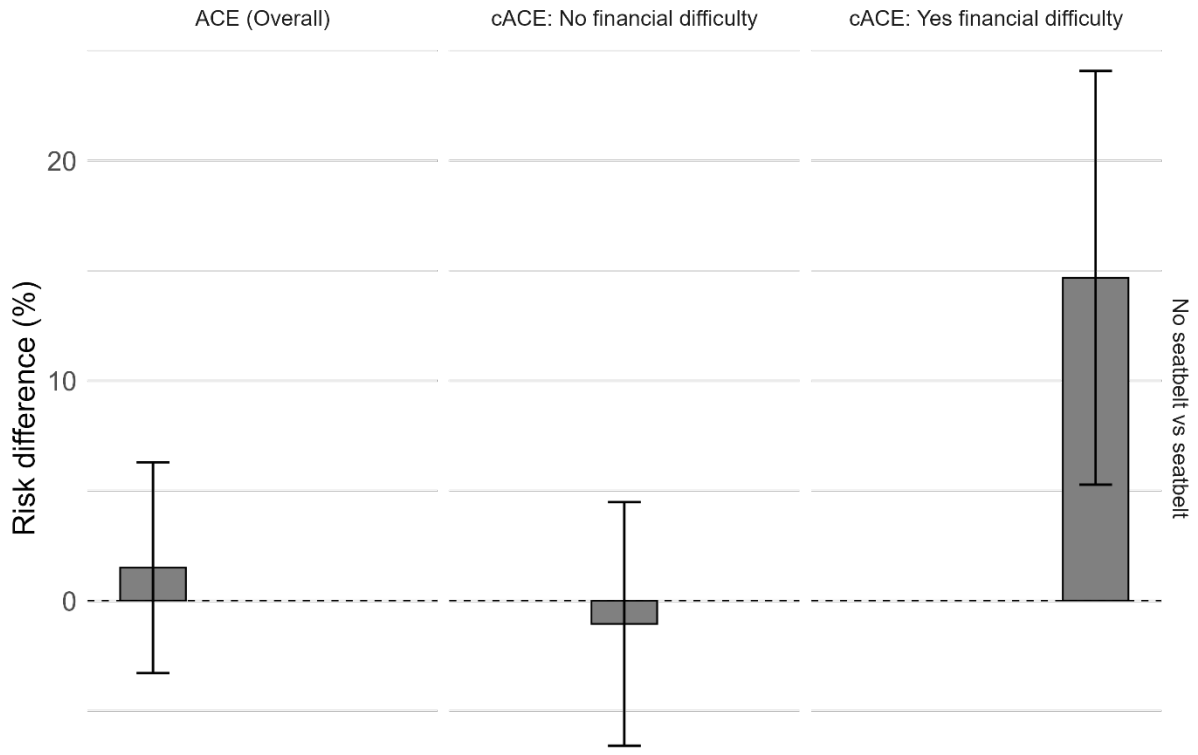
differences stratified by parental education and subsequently derived effect modification estimates as the difference in cACEs between adolescents with low versus high parental education.



Supplementary Fig. S5. Effect modification by parental education of permissive vs. authoritative and indifferent vs. authoritative parenting during adolescence on adult CVD or hypertension. Bars represent effect modification estimates (%) comparing cACEs of adolescents with low versus high parental education; error bars indicate 95% compatibility intervals.

c. Negative Control Exposure: Parental Seatbelt Use

To assess the potential impact of partially measured confounding, we conducted a negative control exposure analysis using parental seatbelt use while driving, reported in the Wave I parent survey. We selected seatbelt use because it is not expected to causally influence offspring cardiovascular or hypertension risk, but may capture underlying psychological or behavioural traits such as self-regulation, risk aversion or risk perception that could also relate to parenting style. These traits may not be fully measured by available covariates. Although our main analysis adjusted for parental psychosocial stress using self-reported general happiness as a proxy, this variable reflects only one aspect of such characteristics. Using parental seatbelt use in place of parenting style, we estimated the same risk difference contrasts to assess whether associations emerged that would be consistent with residual confounding.



Supplementary Fig. S6. Average causal effect (ACE) and conditional average causal effects (cACE) of parental seatbelt use during adolescence on CVD and hypertension in adulthood by family financial situation (yes difficulty/no difficulty). Bars represent risk differences (%) and error bars indicate 95% compatibility intervals.

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Chapter 6: Discussion

Summary of Main Findings

This thesis examined how socioeconomic inequalities in cardiovascular health may emerge due to exposures early in the life course, by investigating the effects of behavioral and parental exposures during adolescence on adult cardiovascular outcomes and whether these effects differed by family financial situation. Using data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), the empirical studies of this thesis explored two domains of adolescent exposures, namely individual health behaviors and family environments.

The first empirical study, presented in Chapter 4, investigated whether the cardiovascular effects of adolescent health behaviors differed according to family financial situation. The findings suggest that certain health behaviors have a greater effect on cardiovascular outcomes among socioeconomically disadvantaged groups. In particular, exposure to high risk alcohol use and alcohol use combined with smoking during adolescence had a greater effect on cardiovascular diseases and hypertension in adulthood among individuals who grew up in financially disadvantaged households. These results provide evidence of differential susceptibility, where the same behavioral exposure may produce larger health effects in socioeconomically disadvantaged groups, thereby amplifying existing inequalities over the life course.

The second empirical study, presented in Chapter 5, examined whether parenting styles experienced during adolescence have effects on adult cardiovascular and hypertension risk, and whether these effects differ by family financial ability. In contrast to the health behavioral exposures examined in the first study, parenting styles during adolescence showed minimal whole population effects on cardiovascular outcomes for our study population. Permissive parenting appeared protective among individuals who grew up in financially disadvantaged families in the main analysis, but this pattern was not consistent across sensitivity analyses and may reflect residual confounding. Overall, the findings provide limited evidence that parenting styles during adolescence have meaningful long term causal effects on cardiovascular outcomes at ages 33 to 43.

Considering the broader research question of the thesis, these findings suggest that not all adolescent exposures operate through the same mechanisms in their effects on long term cardiovascular outcomes, if any. Certain health behaviors during adolescence may

reinforce socioeconomic inequalities in health outcomes through differential susceptibility, whereas parenting styles during this developmental period appear to have limited long term cardiovascular importance. The results thus highlight the importance of distinguishing between exposures that affect cardiovascular risk and those that may have less of an effect when examined over the life course.

More broadly, the findings illustrate the importance of examining both differential exposure and differential susceptibility when studying the mechanisms underlying health inequalities. By examining both mechanisms, this thesis contributes to a more comprehensive understanding of how cardiovascular health inequalities may develop across the life course.

Limitations and Strengths

Several limitations should be considered when interpreting the findings of this thesis. First, exposures and covariates were primarily measured using self-reported data. Adolescent health behaviors and perceptions of parenting style were assessed through participant reports, which may be subject to reporting biases and misclassification. Adolescents may underreport behaviors such as smoking or alcohol consumption, and perceptions of parenting practices may differ from parents' own reports. Measurement error in these exposures could attenuate estimated associations and reduce the ability to detect true causal effects.

Second, some exposures may have been measured imperfectly due to limitations in the available survey instruments. For example, in Chapter 4, questionnaire-based assessments of physical activity may not capture intensity or duration of activity accurately, and objective measures such as accelerometer-based data were not available. Similarly, in Chapter 5, parenting styles were derived from a set of items measuring parental responsiveness and demandingness, which capture key aspects of Baumrind's typology but may not fully represent the complexity of family relationships.

Finally, in both studies, cardiovascular outcomes were measured when participants were aged between 33 and 43 years. Although hypertension and early manifestations of cardiovascular disease are detectable at these ages, many cardiovascular conditions develop later in adulthood. Consequently, the follow up period may not capture the full long term impact of adolescent exposures on adult cardiovascular outcomes.

Despite these limitations, this thesis has several important strengths. A major strength is the use of a large, nationally representative longitudinal cohort followed from adolescence into adulthood. The Add Health study provides rich information on social, behavioral and familial environments during adolescence and allows these early life exposures to be linked to

cardiovascular outcomes more than two decades later. Such long follow up periods are essential for examining life course processes underlying chronic disease development. Moreover, the analyses accounted for missing data and complex survey design. Multiple imputation was used to address missing covariate information, and bootstrap procedures combined with survey weights ensured that estimates reflected the complex sampling design and attrition patterns of the Add Health cohort.

Additionally, this thesis applies a rigorous causal inference framework to examine the long term effects of adolescent exposures. The use of doubly robust estimation with inverse probability weighting allowed adjustment for measured confounding while estimating causal contrasts that are interpretable for public health interventions. Effect modification was examined on the additive scale using risk differences, which directly reflect the potential impact of population-level interventions on disease burden.

Finally, the analyses integrated complementary methodological approaches to capture different dimensions of adolescent environments. Latent class analysis was used to identify behavioral patterns and parenting style typologies, while causal models were used to estimate their long term effects on cardiovascular outcomes. This combination of descriptive and causal approaches provides a more comprehensive understanding of adolescent exposures than analyses focusing solely on individual behaviors.

Implications for Policy, Practice and Future Research

The implications of this thesis' findings can be considered within an evidence-based public health (EBPH) framework, which emphasizes the integration of the best available scientific evidence with practitioner expertise, population needs and contextual factors to inform decision-making (Brownson et al., 2009). From this perspective, understanding not only whether specific exposures cause specific health outcomes, but also whether their effects differ across socioeconomic groups, is essential for designing interventions that are both effective and equitable. In particular, an EBPH approach highlights the importance of considering the reach and distribution of intervention effects across populations, rather than focusing only on average population-level effectiveness.

In line with the EBPH framework, the findings of Chapter 4 suggest that interventions targeting adolescent health behaviors, specifically alcohol use and concurrent alcohol use and cigarette smoking, may reduce socioeconomic inequalities in adult cardiovascular health. When harmful behaviors produce larger health effects in disadvantaged populations, reducing these exposures may generate greater health gains for these populations. Thus, policies that

target adolescent health behaviors have the potential not only to improve overall population health but also to mitigate socioeconomic inequalities in cardiovascular outcomes.

A range of evidence-based policy tools are relevant in this regard. Population-level measures such as increasing excise taxes on tobacco and alcohol, mass media campaigns, comprehensive restrictions on advertising of these products and limiting their physical availability have been identified as “best buy” interventions by the WHO, defined as those with demonstrated effectiveness, feasibility and cost-effectiveness in reducing non-communicable disease risk (WHO, 2024). These measures are supported by a substantial body of evidence showing reductions in initiation and consumption, including among adolescents (Ding, 2005; Farrelly et al., 2013; Stockings et al., 2016). School-based prevention programs and community interventions addressing adolescent health behaviors may also play an important role, particularly when implemented in socioeconomically disadvantaged communities (Busch et al., 2013).

At the same time, the effects of these interventions on health inequalities are not uniform. Evidence from systematic reviews suggests that public health interventions can have differential effects across socioeconomic groups, depending on how they are implemented and who they reach (Lorenc et al., 2013; Thomson et al., 2018). Interventions that rely on individual behavior change such as mass media campaigns may disproportionately benefit more advantaged groups and widen inequalities, whereas more structural approaches including fiscal policies may reduce inequalities (Lorenc et al., 2013). An umbrella review of public health policies in high-income countries on health inequalities reported similarly mixed effects, with more structural interventions such as fiscal measures and regulatory policies tending to reduce inequalities, while information-based or education-focused interventions were more likely to have no effect or to widen inequalities (Thomson et al., 2018). These reviews suggest that the equity impact of prevention strategies cannot be assumed from their overall effectiveness. Complementing population-wide approaches with strategies targeted toward socioeconomically disadvantaged adolescents may help ensure that prevention efforts effectively reduce rather than inadvertently widen inequalities in cardiovascular health. This aligns with an EBPH approach that prioritizes not only overall effectiveness but also the reach and equity of intervention impacts.

Chapter 4’s findings also provide evidence of adolescence as an important period for primordial prevention, during which health behaviors that contribute to cardiovascular outcomes may be established. In the US, national initiatives such as Healthy People 2030 include targets to reduce youth tobacco and alcohol use (National Academies of Sciences, 2019; Office of Disease Prevention and Health Promotion, n.d.), illustrating how behavioral

prevention strategies can be embedded within broader efforts to improve population health and reduce health inequalities.

Meanwhile, the findings of Chapter 5 raise questions about the role of parenting styles during adolescence as a target for cardiovascular disease prevention. Although family environments including parenting styles are widely considered important determinants of long term health (Vedanthan et al., 2016; Ortiz et al., 2024), the results of this study suggest that parenting style typologies during adolescence have limited causal effects on cardiovascular disease and hypertension in early/mid adulthood. This does not mean that parenting is wholly unimportant for shaping offspring health. Prior research has indicated that parenting styles and practices affect a range of developmental outcomes, including psychosocial wellbeing, educational attainment and the adoption of health behaviors (Matthews et al., 2017; Ding and He, 2022; Teuber et al., 2022; Stager et al., 2023), which may still be important on long term health outcomes even if direct effects on cardiovascular disease are modest when examined over long follow up periods. The present findings instead highlight the importance of identifying which aspects of early life family environments are most relevant for the development of cardiovascular inequalities. From an EBPH perspective, these findings suggest that prioritizing parenting interventions for CVD prevention should be guided by their demonstrated long-term health impact, rather than their broader importance for child development alone.

Several directions for future research can be considered from this thesis. Additional studies are needed to clarify which adolescent exposures have long term consequences for cardiovascular health and how these exposures interact with socioeconomic conditions across the life course. For example, the effects of behaviors that have increasing relevance for today's adolescence warrant greater attention. Screen time and sleep patterns are increasingly recognized as important health behaviors that may interact with socioeconomic conditions and influence long term health trajectories (Jackson et al., 2015; Horner et al., 2025). Furthermore, research using more detailed measures of family environments may provide further insight into the role of family contexts in shaping adult cardiovascular outcomes. Repeated assessments of parenting practices across adolescence or alternative conceptualizations of family dynamics may help capture changes in family relationships that are not reflected in single time point measures. Longer follow up periods can also provide useful insights, as many cardiovascular diseases develop later in adulthood.

Further research could also examine additional dimensions of family life that are not captured by parenting style typologies. For example, parental presence in daily routines, such as time spent together during meals, before and after school, or participation in shared physical and social activities such as sports may represent important aspects of family environments.

Previous research suggests that parental presence and involvement in children's daily lives, such as shared meals and time spent together, are associated with healthier dietary behaviors and improved psychosocial wellbeing among adolescents (Nolte et al., 1983; Wenk et al., 1994; Videon and Manning, 2003). However, evidence for long term cardiovascular effects remains limited and mixed, with little research examining whether these aspects of family life influence cardiovascular health in adulthood (McIntosh, 2008; Chen et al., 2019). Investigating whether patterns of parental presence and involvement have long term effects for offspring cardiovascular health, and whether the effects differ according to parental gender, may further improve understanding of how family contexts affect cardiovascular trajectories across the life course.

While the studies in this thesis examined the effects of individual- and family-level exposures, further research could also investigate the effects of the broader sociopolitical and institutional contexts on health inequalities. Reviews by Lorenc et al. (2013) and Thomson et al. (2018) indicate that population-level interventions and regulations can have differential effects across socioeconomic groups, depending on their design, implementation and reach. As a result, policies that reduce harmful exposures at the population level may not reduce inequalities, and may in some cases increase them (Lorenc et al., 2013; Thomson et al., 2018). Thus, estimating the distributional effects of population-level public health interventions may provide important insights into how policies can either mitigate or unintentionally reinforce health inequalities. While such analyses were not conducted as part of this thesis, this line of research is explored further in the proposed study "Future Work: Same Laws, Unequal Effects? Tobacco Control and Use Among Adolescents in Australia", detailed in the Appendix to this thesis.

Overall, by examining how socioeconomic disadvantage may modify susceptibility to health risk behaviors and parenting styles during adolescence, this thesis highlights the importance of empirically evaluating upstream determinants of cardiovascular health inequalities. Identifying which early life exposures have meaningful long-term effects, and how these effects are distributed across populations, is essential for designing prevention strategies that are both effective and equitable.

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Chapter 7: Conclusion

Life course epidemiology emphasizes that health outcomes in adulthood are determined not only by exposures in adulthood but also by behavioral, social and environmental exposures earlier in life. Adolescence represents a particularly important developmental period during which health behaviors are established and family environments continue to affect developmental trajectories. Exposures during this stage may contribute to the development of health inequalities observed decades later in life. Using a life course perspective, this thesis examined whether exposures during adolescence contribute to socioeconomic inequalities in cardiovascular outcomes in adulthood, with a particular focus on differential susceptibility as a potential mechanism through which health inequalities are produced.

Indeed, the evidence presented in this thesis suggests that adolescent exposures can have unequal effects on cardiovascular outcomes in later life. Certain health behaviors, specifically high risk alcohol use and concurrent alcohol use and cigarette smoking, produce long term cardiovascular effects that differ according to the socioeconomic conditions in which individuals grow up. This pattern is consistent with the hypothesis that differential susceptibility can modify the health effects of behavioral exposures, translating into health inequalities in later life. As such, socioeconomic disadvantage can affect not just the distribution of exposures but also the magnitude of their health effects. This understanding complements the emphasis on differential exposure that is predominant in studies on the etiology of cardiovascular inequalities. While much of the literature has focused on the greater prevalence of harmful exposures among socioeconomically disadvantaged populations, the findings of this thesis provide evidence that inequalities may also emerge when similar exposures produce different health effects across social contexts. Thus, considering both differential exposure and differential susceptibility can provide a more comprehensive approach for understanding the development of socioeconomic inequalities in cardiovascular health over the life course.

At the same time, given the limited whole-population and differential effects observed for parenting styles during adolescence, further nuance is necessary to understand the effects of different types of adolescent exposures. Although previous research has documented associations between early life family environments and adult health, these results suggest that parenting styles may not have causal effects on adult cardiovascular disease within the age range examined in this thesis. Their effects may instead be more relevant to indirect pathways to health, including the development of behavioral patterns or psychosocial

processes earlier in the life course. These contrasting findings illustrate the importance of distinguishing between different types of exposures when studying life course processes underlying health inequalities.

Thus, understanding which adolescent exposures have lasting cardiovascular effects remains an important task for future research. In particular, additional studies are needed to clarify which adolescent exposures have long term effects on cardiovascular health and whether these effects vary by socioeconomic position. Longitudinal cohort studies with follow up extending throughout the life course are particularly valuable for addressing these questions, especially given that many cardiovascular conditions emerge in later adulthood. Beyond identifying these effects, future research could also examine the potential impact of intervening on adolescent exposures. Simulation and modelling approaches can be used to estimate the effects of population level and targeted interventions on the burden of CVDs, providing insights into which strategies are most likely to reduce both overall risk and their unequal distribution across socioeconomic groups. Future work may also benefit from more detailed and repeated measurement of different aspects of the family environment across adolescence, to capture the effects of family dynamics throughout the life course.

A key contribution of this thesis is its empirical examination of differential susceptibility as a mechanism underlying socioeconomic inequalities in cardiovascular health. By assessing whether the effects of adolescent exposures differ according to socioeconomic contexts, this thesis extends existing research that has largely focused on the unequal distribution of exposures. Based on the findings of this thesis, the development of cardiovascular inequalities can also emerge from the unequal effects of those exposures across socioeconomic groups.

Overall, this thesis reinforces the need to examine cardiovascular inequalities through a life course lens. Health inequalities observed in adulthood are not just the product of a single exposure but instead develop through processes that accumulate across time. Early life exposures, including health behaviors and socioeconomic conditions, can interact across the life course to produce unequal cardiovascular outcomes. Thus, identifying the exposures that causally contribute to these disparities remains essential for developing effective strategies to reduce socioeconomic inequalities in cardiovascular health.

Appendix on Future Work: Same Laws, Unequal Effects? Tobacco Control and Use Among Adolescents in Australia

Background

On 1 December 2012, Australia became the first country in the world to implement plain packaging laws for tobacco products. These laws required all tobacco products to have uniform, unattractive packaging, removing branding and restricting the use of colors, logos, and promotional text. As outlined in the Tobacco Plain Packaging Act, the law aims to improve public health by discouraging tobacco use initiation, motivating cessation, preventing relapse, and reducing non-smokers' exposure to tobacco smoke (Department of Health, n.d.).

Although this policy was designed as a population-wide level of intervention, its impact on adolescents warrants particular attention. Adolescence is a critical period for the formation of health behaviors such as smoking, where people who start smoking in their adolescence are more likely to be daily smokers as adults (Australian Institute of Health and Welfare, 2017). As such, it is essential to understand how tobacco control policies shape the behaviors of adolescents specifically (Due et al., 2011). Evaluating the effects on adolescents can also provide valuable insights for other nations, especially as more countries are introducing policies and legislation specifically designed to curb tobacco use among youths (Ahluwalia et al., 2020).

As the first country to implement plain packaging, Australia offers a unique and extended timeline of evidence for assessing the policy's impact. This long-term perspective provides critical insights into both immediate and sustained effects, making it a valuable case study for global tobacco control efforts. Additionally, Australia has implemented a comprehensive range of tobacco control measures, including excise tax increases, public education campaigns, advertising bans, sponsorship restrictions, and smoke-free policies (Department of Health, 2018). Together, these measures have led to a decline in tobacco use across all age groups. Among secondary school students, for example, smoking prevalence fell from 9% in 2002 to 2% in 2017 (Guerin and White, 2020).

Despite the positive effects, these policies may have unintended consequences that need to be mitigated. For example, these policies may inadvertently worsen health inequalities

if their effects vary by socioeconomic conditions. As it is, the Australian Institute of Health and Welfare has reported that people with the most disadvantaged socioeconomic conditions, whether measured by income or by completed level of education, were almost three times more likely to smoke daily compared with people of the most advantageous backgrounds (19.9% versus 6.7%, respectively) (Guerin and White, 2020). While studies have provided evidence of the effectiveness of Australia's tobacco control measures (Diethelm and Farley, 2017), limited research has examined whether the benefits of plain packaging and other laws are equitably distributed across the adolescent population. Addressing these gaps is crucial for designing interventions that reduce disparities rather than exacerbate them.

Research Aim

This study aims to assess whether Australia's tobacco plain packaging policy had unequal effects on tobacco use among adolescents, varying by their family financial situation.

Methodology

Dataset

The Growing Up in Australia: Longitudinal Study of Australian Children (LSAC) provides a comprehensive dataset to evaluate the impact of plain packaging on inequalities in adolescent tobacco use. Variables of interest include tobacco use initiation and prevalence, patterns of alternative tobacco use (e.g. e-cigarettes), socioeconomic conditions (family financial difficulty, parental education, area deprivation indices), and covariates such as sex, parental smoking status, and peer smoking behaviors (Department of Social Services, 2024).

Statistical analysis

We plan to use a difference-in-differences approach to evaluate the impact of plain packaging laws. This method will compare changes in tobacco use behaviors in Australia (treatment group) with changes in a suitable control group, such as adolescents in New Zealand which did not have plain packaging laws during the study period (Underwood et al., 2020).

Potential challenges and considerations

Since the plain packaging laws came into effect in Australia, other tobacco control measures such as enlarged graphic warnings have also been introduced concurrently across the nation. As such, this may make it challenging to disentangle the effects of one policy from another. This has been noted in a 2016 study commissioned by the Australian Department of Health on the impact of Australia's tobacco plain packaging on smoking prevalence (Chipty, 2016). A common approach to isolate the effect of a single policy when there are co-occurring policies is to include them as covariates, which is an approach that we may consider (Matthay et al., 2021).

While traditional cigarette consumption may have declined in the last few decades, there have been increased in use of alternative tobacco products, especially e-cigarettes, which may be used to substitute traditional cigarettes (Underwood et al., 2020). These products need to be explicitly taken into account, so they do not confound our results.

Expected Outcomes

This study will provide evidence on whether plain packaging laws have a differential effect in tobacco use among adolescence. Based on the findings, we aim to provide evidence that may guide the design of tobacco control measures to address health inequities.

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List of Publications, Presentations and Courses

Publications in Peer-Reviewed Scientific Journals

1. **Noor, N.**, Jackisch, J., Baggio, S., Cullati, S., Carmeli, C. “Effects of Adolescent Parenting Styles on Adult Cardiovascular Disease and Hypertension: A Population-Based Cohort Study”. [Under Review].

2. **Noor, N.**, Jackisch, J., Chiolero, A., Mullan Harris, K., Carmeli, C. “Unequal effects of health behaviors in adolescence on adult cardiovascular disease and hypertension by family financial hardship in the US: A cohort study”. [Under Review].

3. van der Linden, BWA., Viehl, CA., **Noor, N.**, Adair, T., Vaccarella, S., Carmeli, C. (2026). “Trends in obesity-related cardiovascular and cancer mortality in Switzerland 1995 – 2019: an analysis of multiple causes of death”. American Journal of Epidemiology.

4. Jackisch, J., **Noor, N**., Raitakari, OT., Lehtimäki, T., Kähonen, M., Cullati, S., Delpierre, C., Kivimäki, M., Carmeli, C. (2025). “Differential effects of adolescent health behaviours on adult cardiometabolic health by parental and neighbourhood socioeconomic background“. European Journal of Public Health.

5. Jackisch, J., **Noor, N**., Raitakari, OT., Lehtimäki, T., Kähonen, M., Cullati, S., Delpierre, C., Kivimäki, M., Carmeli, C. (2024). *“*Does the effect of adolescent health behaviours on adult cardiometabolic health differ by socioeconomic background? Protocol for a population-based cohort study”. BMJ Open.

6. Probst-Hensch, N., Imboden, M., Jeong, A., Keidel, D., Vermes, T., Witzig, M., Cullati, S., Tancredi, S., **Noor, N**., … Corona Immunitas Research Group. (2024). “Long-term trajectories of densely reported depressive symptoms during an extended period of the COVID-19 pandemic in Switzerland: Social worries matter”. Comprehensive Psychiatry.

7. Harju, E., Speierer, A., Jungo, KT., Levati, S., Baggio, S., Tancredi, S., **Noor, N**., … Chocano-Bedoya, P. (2023). “Changes in Healthcare Utilization During the COVID-19 Pandemic and Potential Causes—A Cohort Study From Switzerland”. International Journal of Public Health.

8. Sabatini, S., Kaufmann, M., Fadda, M., Tancredi, S., **Noor, N.,** van der Linden, BW. A., Cullati, S., … von Wyl, V. (2023). “Factors Associated With COVID-19 Non-Vaccination in Switzerland: A Nationwide Study”. International Journal of Public Health.

Published Pre-Prints and Protocols

1. **Noor, N.,** Jackisch, J., Baggio, S., Cullati, S., Carmeli, C., 2026. “Effects of Adolescent Parenting Styles on Adult Cardiovascular Conditions: A Population-Based Cohort Study”. medRxiv. doi: https://doi.org/10.64898/2026.08.22.26361103

2. **Noor, N.**, Jackisch, J., Chiolero, A., Mullan Harris, K., Carmeli, C. 2026. “Unequal effects of health behaviors in adolescence on adult cardiovascular disease and hypertension by family financial situation in the US: A cohort study”. medRxiv. doi: https://doi.org/10.64898/2026.03.11.26348128

3. **Noor, N.**, Jackisch, J., Chiolero, A., Baggio, S., Mullan Harris, K., Cullati, S., Carmeli, C., 2025. “Differential Effect of Parenting Style During Adolescence on Adult Cardiovascular Disease and Hypertension by Family Financial Situation: Protocol for a Population-Based Study”. Open Science Framework. doi: 10.17605/OSF.IO/QBXCJ.

Presentations at Scientific Conferences

Date	Event and Location	Format
March 2023	Causality to Get Population Health Science Right Symposium, Lausanne, Switzerland.	Oral, Poster
October 2023	SLLS Annual International Conference, Munich, Germany.	Oral, Poster
September 2024	European Public Health Conference 2024, Lisbon, Portugal.	Poster
January 2025	Zurich Population Research Conference, Zurich, Switzerland.	Oral
June 2025	SSPH+ Annual Meeting, Bern, Switzerland	Workshop
September 2025	SLLS Annual International Conference, Fribourg, Switzerland. *Received Runner-Up Best Poster Award	Oral, Poster
November 2025	Institute for Social Science Research Conference, Brisbane, Australia	Oral
September 2026	European Congress of Epidemiology, London, UK.	Poster

Courses

Date	Course Title	Location	ECTS
October 2022	Introduction to Health Economics	Lausanne, Switzerland	2
December 2022	Public Health Surveillance and Population Health Monitoring (Advanced Module)	Lausanne, Switzerland	2
February 2023	Basics of Scientific Writing in English: A Structured Method for Organizing and Writing Better, Faster Papers	Online	2
April 2023	Understanding Social Inequalities and Social Problems in Health!	Basel, Switzerland	1
June 2023	Life Course Epidemiology and Public Health	Fribourg, Switzerland	1
September 2023	Causal Inference for Population Health Sciences (Advanced Module)	Basel, Switzerland	2
October 2023	Writing Better, Faster Scientific Papers in English: A Structured Method for Non-Native English Speakers (Advanced Module)	Online	2
January 2024	Introduction to Causal Inference for Health and Social Scientists	Leeds, UK	N/A
March 2024	Behavioral Health Economics	Online	N/A
March 2024	Cancer Epidemiology: From Description to Prevention	Online	1
June 2024	Mining Public Opinion Using Language Models	Online	N/A
September 2024	Introduction to Health Policy	Luzern, Switzerland	1
October 2024	Introduction to the Global Burden of Disease Project Data and Tools	Online	N/A
November 2024	Advancing Health Equity in Learning Health Systems	Luzern, Switzerland	2
November 2024	Foundations of Public Health	Zurich, Switzerland	2
February 2025	Designing and Using Questionnaires for Research: Practical Insights and Challenges	Lausanne, Switzerland	1

September 2025	Social Policies in a Life Course Perspective	Lausanne, Switzerland	N/A
September 2025	Cumulative Advantage/Disadvantage Workshop	Mannheim, Germany	N/A
April 2026	Analyzing the Household, Income and Labour Dynamics in Australia (HILDA) Survey	Melbourne, Australia	N/A
	Total ECTS		**19**

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