Rising Childhood Income Inequality and Declining Americans' Health

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Abstract

Morbidity and mortality are on the rise among Baby Boomers and younger cohorts. This study investigates whether this unfavorable health trend across birth cohorts 1925–1999 is related to rising income inequality Americans face during childhood. We use two nationally representative datasets: National Health and Nutrition Examination Surveys (NHANES) 1988–2018 and Panel Studies of Income Dynamics (PSID) 1968–2013, and two health outcomes: biomarkers of physiological dysregulation, and a chronic disease index. Childhood income inequality is measured by the average of the Gini index at the national level each birth cohort is exposed to between birth and age 18, where the Gini index from 1925 to 2016 is computed based on Internal Revenue Service income data. By merging childhood income inequality to individual level data from NHANES or PSID based on birth cohort, we find childhood income inequality is positively associated with the risk of physiological dysregulation in adulthood for all gender and racial groups in the NHANES data. It is also significantly related to the risk of chronic disease in the PSID data. This association is robust to controls for individual level childhood health and family background, adulthood socioeconomic and marital status, and contemporary macro socioeconomic factors. More importantly, childhood income inequality exposure explains a substantial amount of variation in these two health outcomes across cohorts, a pattern not observed for other early life exposures that display negative temporal trends similar to those for childhood income inequality. This study provides important evidence that income inequality experienced during childhood may have a long-lasting negative consequence for adult health, which partially explains the adverse health trends experienced by Baby Boomers and younger cohorts in the United States.

Keywords

Childhood income inequality; Early-life conditions; Adult health; Physiological dysregulation; Chronic diseases; Health trend; Health decline

Introduction

Stagnating and declining life expectancy since 2015 has become an alarming population health crisis in the United States. This declining life expectancy has been attributed to rising drug-, alcohol-, and suicide-related mortality combined with slowing progress in combating

heart disease (Barbieri, 2019; Case and Deaton, 2017) although their relative contributions may differ by demographic groups (i.e., race and gender) (National Academies of Sciences, Engineering, and Medicine (NASEM), 2021). Much attention has been focused on Baby Boomers who reached middle age and early old age in the past two decades, while recent studies report the declining health trend has extended to younger age groups or recent birth cohorts (NASEM, 2021; Zang et al., 2019). For example, physiological dysregulation (PD), the early stage of the morbidity process (Crimmins et al., 2010) and indicated by several biological risk factors, such as inflammation, metabolic syndrome, and deterioration of lung and renal function (Seeman et al. 2001), has continuously increased for Baby Boomers and members of the Generation X and Generation Y cohorts (Zheng and Echave, 2021).

At the same time, income inequality has been dramatically increasing since the mid-1940s when Baby Boomers were born (Frank, 2014). In fact, the deteriorating trend in PD across cohorts very closely mirrors the trend of income inequality (indicated by the Gini coefficient) experienced during childhood (ages 0–18) across cohorts as shown in Figure 1. Both PD and childhood income inequality declined from Early Children of the Depression (1925–1930) until War Babies (1943–1945) and increased since early Baby Boomers (1946–1955). May the declining health across cohorts partially result from rising income inequality Americans faced during their childhoods?

Exposure to income inequality in early life may impact later life health through material and psychosocial pathways. First, income inequality may lead to the reduction of socioeconomic resources people need to produce health. In societies with rising inequalities, the interests of economic elites begin to diverge from those of average citizens (Kawachi and Kennedy, 1999). The economic elites demand specialized services or policies that help maintain their wealth such as tax breaks and are less likely to support income redistribution or public expenditure policies (e.g., public education and health care) that do not directly benefit them (Bartels, 2008; Osberg et al., 2004; Ponzetto and Troiano, 2018). Studies show politicians are more likely to support policies that match the interests of the affluent because they have a vested interest in maintaining a strong economy and are reliant on the economic resources of the affluent to advance their political agendas (e.g., campaign contributions) (Gilens, 2012; Gilens and Page, 2014). That is a major reason why countries with greater income inequality tend to devote fewer resources to public expenditure policies (Osberg et al., 2004). Therefore, children growing up in a period of rising income inequality may face reduced social spending, which can lead to a reduction of life opportunities and deterioration of life circumstances throughout their life course (Bradley and Taylor, 2013; Davey, 1996; Kaplan et al., 1996).

Second, rising income inequality can also intensify relative deprivation, which emphasizes the gap between one's own income and incomes of those richer than oneself. Relative deprivation is associated with risky behaviors (e.g., smoking, obesity) and heightened mortality (Eiber et al., 2004; Eibner and Evans, 2005). That is because relative deprivation induces psychological stress (e.g., negative self-assessment, frustration, and depression), which can harm health in the long run (Kawachi and Kennedy, 1999; Kondo et al., 2008). Third, income inequality leads to loss of social cohesion and the erosion of social capital (Wilkinson, 1992, 1996), suggesting that inequality acts as a social stressor (Pickett and

Wilkinson, 2015). In more unequal societies, people show less solidarity, less concern for social harmony, and less willingness to help others (de Vries et al., 2011; Paskov and Dewilde, 2012; Putnam and Garrett, 2020). Lack of social cohesion has been found to be harmful for cognitive, emotional, and behavioral outcomes (DeWall et al., 2011) and mediate the association between income inequality and health (Delhey and Dragolov, 2014; Kawachi et al., 1997; Zimmerman and Bell, 2006). These psychosocial pathways suggest that children growing up in a period of rising income inequality are exposed to exacerbated relative deprivation and social isolation, which can have long-lasting negative consequences for their mental and physical well-being (Kawachi and Kennedy, 1999).

Based on these discussions, we hypothesize that childhood income inequality has long-lasting consequences for adult health. Exposure to rising childhood inequality contributes to declining adult health across cohorts. We use two independent datasets -- the National Health and Nutrition Examination Surveys (NHANES) and Panel Studies of Income Dynamics (PSID)) -- to test this hypothesis and account for various confounders across the life course. This study provides the first investigation on how childhood income inequality since the second half of the last century may have a lingering impact on U.S. population health.

Background Literature on Income Inequality and Health

The relationship between income inequality and health has been extensively studied, but current literature has provided mixed findings. Many scholars now are skeptical of the adverse impact of income inequality on health (e.g., Leigh and Jencks, 2007; Mellor and Milyo, 2003) while others believe the initial theoretical arguments and maintain that the effects may still exist in certain situations (e.g., Lillard et al., 2015; Tibber et al., 2022; Zheng, 2012). This uncertainty has high stakes as most developed and developing countries have experienced dramatic increases in income inequality in the past several decades. If income inequality adversely affects health, then even a small effect may have considerable consequences for the population as a whole (Kondo et al., 2009).

These mixed findings have been attributed to differences in countries, time periods, units of analysis, levels of income inequality, inclusion of mediating variables as controls, and timing of effects (Subramanian and Kawachi, 2004; Wilkinson and Pickett, 2006; Zheng, 2012). Timing concerns the lags between income inequality and health outcomes. Emerging studies have estimated the lagged effect of income inequality on health. Some test the aggregate association between income inequality and the population mortality rate, while others examine the relationship between income inequality and individual health or mortality risk using multilevel designs. Among the aggregate-level studies, Kim and colleagues (2008) report significant 10-year lag effects, while other studies report either non-significant or mixed results (e.g., Leigh and Jencks, 2007; Mellor and Milyo, 2003; Shi et al., 2003, 2004). Multilevel studies have reported a lag effect up to 6 years (Dahl et al., 2006), 8 years (Lochner et al., 2001), 12 years (Zheng, 2012), and 15 years (Blakely et al., 2000; Subramanian and Kawachi, 2004), while other studies do not find such long-term effects (e.g., Blakely et al., 2003; Osler et al., 2003). One study also links early life income inequality exposure to adult health (Lillard et al., 2015), which, however, has not been

corroborated in other studies. Research has yet to investigate whether income inequality experienced in childhood may contribute to health decline across cohorts in the United States.

It is reasonable to assume that early life income inequality exposure may have long-term health consequences. The fetal origins hypothesis, proposed by Barker and his colleagues (1990), holds that the risk of developing coronary heart disease in adulthood may originate from in utero biological programing associated with maternal and fetal undernutrition. Many later studies further develop the fetal origins hypothesis into a broader developmental origins of health and disease theory. This theory posits that adult health, disease, morbidity, and mortality originate in part from environmental exposures in early life (e.g., postnatal infection, early life health status, mother's education, parental income, childhood poverty) (Ben-Shlomo and Kuh, 2002; Elo and Preston, 1992; Hayward and Gorman, 2004; Lynch and Davey Smith, 2005). It usually takes years before exposures to risk factors are observed to predict chronic illness, e.g., cardiovascular diseases, cancer, stroke, diabetes (Yusuf et al., 2001). Chronic conditions have long latency periods, accounting for the lags between the accumulation of risk factors and the onset of diseases and subsequent morbidity and mortality (Lynch and Davey Smith, 2005). The conjecture of long-lasting health consequences of early life income inequality exposure is consistent with the developmental origins of health and disease theory.

Built on this developmental theory and prior studies on the lagged effect of income inequality on health, we use a cohort perspective to investigate whether income inequality experienced during childhood may have long-lasting consequences for adult health, how this may shape the cohort trend in health, and whether this consequence may vary by demographic groups (gender and race). Therefore, our study not only contributes to the evidence regarding the lagged impact of income inequality on health, but also sheds light on the underlying mechanisms shaping the health decline across U.S. cohorts.

Methods

Data

We use two large nationally representative datasets: NHANES 1988–2018 and PSID 1968–2013. NHANES is a pooled cross-sectional dataset, each wave of which collects different samples. Nonetheless, we can create synthetic cohorts and obtain cohort trends in health. NHANES data were collected from household interviews as well as physical examinations and laboratory tests performed in a mobile examination center. We combined NHANES III data, collected between 1988 and 1994, with the data collected in ten continuous waves from 1999 to 2018. Detailed descriptions of the survey designs, procedures, and methodologies are available on the NCHS website. We constrained the sample to those born in 1925–1999 to exclude very old and very young cohorts with small sample sizes. Of the 44,804 respondents born in this period and with valid PD information, we excluded respondents with missing data on education (n=535), marital status (n=1,478), poverty income ratio (n=1,656), those in "other" racial groups (n=3,248), or any without valid sampling weight (n=2,378), reaching a sample size of 35,509 individuals aged 17 years and older.

PSID is multi-cohort longitudinal dataset, which tracks individuals over time and yields a life course history of real cohorts. This survey began in 1968 with a nationally representative sample of families. The survey was administered annually until 1997 and biennially thereafter. We use information from the Family and Individual Files 1968–2013. Children from the original 1968 families were added to the Family Files after they became heads of households or the spouses of one. Our analysis period consists of the 1999 to 2013 waves when the health outcome (chronic disease index (CDI)) is measured, with information on early life conditions (early life diseases, mother's education, parental smoking) obtained from prior waves. We restricted our sample to heads of households and spouses born in 1925–1995 with valid information on CDI, which includes 17,010 individuals with 88,249 observations. After excluding respondents with missing data on age, race, and gender (n=479), early life factors (early life disease index, parental smoking before age 17, and mother's education, n=3,449), and adulthood factors (education, income, and marital status, n=158), we reached a sample size of 12,924 individuals and 68,271 observations.

Both datasets have advantages and disadvantages. NHANES data are relatively large so we can break down analysis by race and gender and examine whether childhood income inequality may explain cohort health decline for all demographic subgroups. But NHANES data do not have measures of childhood health and family background. That is why we use PSID data to test the robustness of findings produced from the NHANES data by taking into account the possible correlation between childhood income inequality and childhood health and family background. By adding these controls, however, we risk over-controlling for the full association between childhood income inequality and adult health if these controls are actually mediators. The disadvantage of the PSID data is that it has a relatively smaller sample size so we cannot disaggregate the analysis by race and gender. We provide results from both datasets and examine consistency across different measures and research designs.

Income inequality is measured by the Gini Index computed using Internal Revenue Service (IRS) data by Frank (2014). There are advantages and disadvantages to using IRS data compared to Census or Current Population Survey (CPS) data to compute inequality indexes (Atkinson et al., 2011). IRS data are better at capturing higher income brackets and income from capital gains, while CPS data are better at capturing lower income brackets (Frank 2009). The Frank (2014) data utilizes pre-tax income based on individual income tax returns and includes capital gains in addition to wages and salaries. A benefit of these data is they provide consistent measurements of inequality dating back to 1917. We use this historical data from 1925 to 2016 to construct a measure of childhood inequality. This measure is based on the average Gini Index at the national level for individuals when they were between 0 to 18 years old. For example, someone born in 1925 would have their childhood Gini Index consist of the average of the Gini Index from 1925 to 1943. This allows us to construct childhood Gini Index scores for respondents based on their birth year. Childhood Gini Index is linked to NHANES and PSID data based on birth year. We construct the childhood Gini index at the national level for both theoretical and technical reasons. Theoretically, income inequality at the national level reflects the scale of social stratification or how hierarchical a society is (Wilkinson and Pickett, 2006), and is more consequential for individual health than income inequality measured in the local context (Kondo et al., 2011; Pickett and

Wilkinson, 2015). Technically, individuals often move during childhood. It is infeasible to track local income inequality individuals may be exposed to because no such data exist.

We also collected several contemporary macro measures that were merged with NHANES (1988–2018) or PSID (1999–2013) based on survey year. Gini Index and percentage with a college degree were obtained from Frank (2014) data. GDP growth rate and unemployment rate were obtained from Federal Reserve Economic Data. Union coverage is measured as the percentage of employed workers in unions using data from Mayer (2004) and Hirsch and Macpherson (2003) that rely on the Handbook of Labor Statistics (Bureau of Labor Statistics) and Current Population Survey data.

Measures

Supplemental Information 1 and 2 display the descriptive statistics of measures from the NHANES data. Supplemental Information 3 displays the descriptive statistics of measures from PSID data. All the measures are at the individual level except childhood income inequality and contemporary period macro measures which are at the national level.

Outcome variables

Dependent variables are the biomarker measures of PD from NHANES data and CDI from PSID data. PD is operationalized as a count of high-risk physiological parameters across multiple systems, including markers of inflammation, metabolic functions, lung function, and renal function. It is based on nine markers of physiological functions listed in Supplemental Information 1, including seven markers of metabolic functions, one marker of chronic inflammation (i.e., low serum albumin), and one additional marker (i.e., urinary function-creatinine clearance). The cutoff points for high-risk levels of these markers are based on previous studies (Crimmins et al., 2003; Yang and Kozloski, 2011). PD is the summary index of positive indicators of all nine markers and indicates the number of biomarkers at high risk. It ranges from 0 to 9. We did not include additional markers of inflammation (e.g., fibrinogen, C-reactive protein) and metabolic function (e.g., fasting glucose) in this measure because these markers were not collected in more recent surveys. Specifically, fibrinogen was not collected after 2001–2002, C-reactive protein was not collected after 2009–2010, and fasting glucose was not collected for 2017–2018. We also created an alternative measure of PD based on the proportion of positive indicators among all the available markers and the overall cohort pattern was similar. The CDI consists of 10 health problems: stroke, diabetes, chronic lung disease, high blood pressure, cancer, heart attack, heart disease, emotional problems, arthritis, and asthma. A respondent's disease index score consists of the sum of all conditions he or she reported, and it ranges from 0 to 10. CDI is time varying as it comes from longitudinal PSID data.

Explanatory variables

The main explanatory variables are cohort and childhood income inequality. We categorize all single birth-year cohorts into eight historical groups: Early Children of Depression (born 1925–30), Late Children of Depression (born 1931–42), War Babies (born 1943–45), Early-Baby Boomers (born 1946–55), Late-Baby Boomers (born 1956–64), Early-Generation X (born 1965–72), Late-Generation X (born 1973–80), and Generation Y (born 1981–99).

This categorization of birth cohorts is well established in the literature (e.g., O'Rand and Hamil-Luker, 2020; Zang et al., 2019) and data collection (e.g., Health and Retirement Study). We also group them by 10-year birth cohorts (1925–1934, 1935–1944, ……, 1985–1999) and overall findings are very similar. As explained in the Data section, the childhood Gini Index was obtained by computing the average of the Gini index at the national level for respondents between the ages of 0 to 18. Gini coefficient ranges in value from 0 to 1, with higher values indicating higher levels of income inequality. Figure 1 displays the trend in childhood Gini index for cohorts born from 1925 to 1999.

Control variables

Covariates include age, age squared, gender, race, survey year, educational attainment, marital status, family poverty income ratio (NHANES) or family income-to-needs ratio (PSID), and contemporary period macro socioeconomic measures. Age and survey year are controlled to obtain the net trend of cohort. The individual level socioeconomic and demographic statuses and period macro socioeconomic characteristics are controlled to account for possible confounding factors that may contribute to the cohort trend in health. Age is grand mean centered. Race consists of three groups: non-Hispanic White (White hereafter), non-Hispanic Black (Black hereafter), and Hispanic. Educational attainment is categorized as less than high school (reference), high school diploma, and college educated or higher. Marital status is categorized as married or cohabitating (reference), divorced or separated, widowed, and never married. Poverty income ratio is the ratio of total household income to a year-specific federal poverty threshold. It is categorized into three groups: below poverty line (less than 1) as a reference, middle (1–2.99), and high (3 or more). Family income-to-needs ratio is a continuous variable and measured as a respondent's family income divided by the U.S. Census Bureau's defined needs of the family (PSID, 2013). Contemporary period macro measures include the Gini Index, GDP growth rate, unemployment rate, percentage of union workers, and percentage with a college education. The Gini Index ranges from 0 to 1 with higher values indicating greater income inequality. The unit of the other four period macro measures is percentage. GDP growth rate represents the percentage increase in real annual GDP between two adjacent years with negative percentages indicating negative growth. Unemployment rate represents the percentage of people in the labor force who are unemployed. Union coverage is measured as the percentage of employed workers in unions. Percentage of college education is the percentage of the population with a college degree.

Additional control variables in PSID

Additional early-life factors from the PSID data include early life disease index, parental smoking before age 17, and mother's education. The early-life disease index consists of the number of 12 health problems a respondent reported before age 17, and scores for this index range from 0 to 12. These health problems are asthma, diabetes, respiratory disease, allergies, heart trouble, epilepsy, severe headaches/migraines, stomach issues, high blood pressure, depression, drug/alcohol abuse, and emotional/psychiatric problems. Parental smoking before age 17 is a dummy variable with 1 indicating that at least one parent/guardian smoked when the respondent was 0 to 17 years old. Mother's education consists of five categories: 1 (did not graduate from high school), 2 (high school graduate),

3 (some college), 4 (college graduate), and 5 (graduate degree). It is possible that these early life factors are mediators rather than confounders in the relationships between childhood income inequality and adult health. Thus, we may be at risk of over-controlling by including them in the model (Pickett and Wilkinson, 2015).

Statistical procedures

Because the dependent variables (i.e., PD, CDI) are count variables, we estimated Poisson mixed effects models to obtain the net effects of birth cohort after controlling for age, period, and basic sociodemographic characteristics (Yang and Land, 2006). Specifically, these models estimated the fixed effects of age and sociodemographic variables and random effects of cohort and period. Supplemental Information 4 provides a detailed explanation of model specification. Poisson regression models the log of the expected count as a function of the predictor variables, so the regression coefficient can be interpreted as follows: for a one unit change in the predictor variable, the expected count of the dependent variable will change by [exp(regression coefficient)−1]*100 percent. Model1 only includes cohort, period, and age. Model2 additionally adjusts for childhood income inequality. Sociodemographic characteristics such as educational attainment, marital status, and poverty income ratio (NHANES) or income-to-needs ratio (PSID) are added in the Model3. Period macro factors are added in Model4. Analysis based on PSID data additionally accounts for early life factors before adding adult characteristics and period factors. We accounted for both surveys' complex designs by adjusting for sampling weights. All statistical analyses were performed using SAS PROC GLIMMIX. Analyses based on NHANES data were conducted for the whole sample and separately for six gender-race-ethnicity groups: White males, White females, Black males, Black females, Hispanic males, and Hispanic females. PSID data were conducted for the whole sample due to the sample size concern.

Results

Table 1 shows the results from Poisson mixed effect models of PD based on the NHANES data. Model 1 controls for age, age squared, birth cohort and survey year. Cohort random effect coefficients indicate an increasing risk of PD from Early Baby Boomers to Generation Y. Variance components indicate a strong cohort pattern of PD. Model 2 adds the childhood Gini index, which has a significant positive association with PD. Each 0.01 unit increase in childhood Gini index is associated with a 3% (= [exp(2.989*0.01)−1]*100) increase in the level of PD. More importantly, childhood Gini index explains a substantial amount of variation in PD across cohorts. After controlling for childhood Gini index, birth cohort variance substantially reduces from 0.025 to 0.001. Additional analyses by gender and racial groups presented in Supplemental Information 5–10 show that the impact of childhood Gini index on PD is especially large among White males and Black females. Each 0.01 unit increase in childhood Gini index is associated with 3.7% and 3.2% increase in the level of PD among White males and Black females, respectively, while it is associated with 2.6% increase among White females and Hispanic females, 2.4% increase among Black males, and 2.2% increase among Hispanic males.

Figure 2 displays the cohort trend in PD for each group based on the random effect coefficients of cohort in Model 1 and Model 2 of Supplemental Information 5–10. Predicted levels of PD for each cohort were calculated and plotted for the six groups, holding age at mean value. A general pattern is that PD has substantially increased across cohorts since Baby Boomers even though the exact timing and slope of increase vary across groups. For example, PD increased since Early Baby Boomers (born 1946–1955) for White males, one historical cohort earlier than for White females. White males also experience the steepest increase in PD among males, while Black females observe a steeper increase than the other two female groups. White males and Black females happen to be most impacted by childhood income inequality exposure. After controlling for childhood Gini index, the cohort trend in PD becomes much flatter for each gender and racial group. This visually displays the substantial reduction in birth cohort variance component from Model 1 to Model 2 in these supplemental tables.

Model 3 and Model 4 of Table 1 additionally control for individual level socioeconomic statuses (educational attainment, marital status, and poverty income ratio), and five period macro socioeconomic factors (Gini index, GDP growth rate, unemployment rate, percentage of union, and percentage of college education) to ensure the patterns observed in Model 2 are not due to individual adulthood factors and contemporary period determinants. These factors, however, generally do not explain the cohort trend in PD as shown in little change in birth cohort variance components once they are controlled. More importantly, after including these variables in the models, the association between childhood Gini index and PD remains strong and significant.

Table 2 shows the results on CDI based on PSID data. Variance components suggest CDI clearly follows a cohort pattern. Model 2 controls for childhood Gini index. Each 0.01 unit increase in childhood Gini index is associated with 7% (= [exp(6.769*0.01)−1]*100) increase in the level of CDI. After controlling for childhood Gini index, birth cohort variance substantially reduces from 0.134 to 0.024. Model 3 additionally adjusts for early childhood factors, including early life disease index, parental smoking status before age 17, and mother's education, which slightly decrease the coefficient estimates of childhood Gini index and birth cohort variance. Further analysis finds that the reduction in birth cohort variance is completely due to early life disease index. In fact, it is offset by parental smoking status before age 17 and mother's education because early life disease index worsens across cohorts while the latter two improve across cohorts. Model 4 and Model 5 add individual adulthood factors and macro contemporary period determinants, which explain neither the association between childhood Gini index and CDI nor the cohort trend of CDI (cohort variance component becomes bigger). These findings corroborate those based on NHANES data.

Auxiliary analyses

Table 1 and Table 2 clearly show that childhood income inequality has long-term health consequences, and helps explain the cohort trend in deteriorating PD and CDI. The findings are also robust to control of early life disease and family socioeconomic and health behavioral factors, adulthood sociodemographic factors, and contemporary period

determinants. These findings are also not influenced by different categorizations of birth cohorts as shown in Supplemental Information 11 and 12. But are these patterns unique to income inequality people experience in childhood? In order to answer this question, we replace childhood Gini index with the other four childhood exposures (childhood percentage of union workers, childhood GDP growth rate, childhood unemployment rate, and childhood percentage with a college education). These four childhood exposures are constructed in the same way as childhood Gini index, that is as the average of these exposures between the ages of 0 and 18 for each cohort. Supplemental Information 13 displays the cohort trends in these four indicators, which show a similar negative trend except childhood percentage with a college education.

Table 3 shows the percentage change in birth cohort variance component across models for each early life exposure measure by race and gender from NHANES data 1988–2018. It consists of 96 models (4 models for each demographic group and early-life factor × 6 demographic groups × 4 early-life factors). The detailed results for childhood Gini index are presented in Supplemental Information 5–10, while those for childhood percentage of union workers, childhood GDP growth rate and childhood unemployment rate are available in Supplemental Information 14, 15 and 16. The results for childhood percentage of college education are not presented because either the model could not converge or it had a positive association with PD, which basically just picks up the cohort trend. The "% change" column shows the percentage change in birth cohort variance component from Model 1 to subsequent models. The setup of Model 1 to Model 4 is the same as that in Table 1. The "coef" column shows the coefficient estimate of each early life exposure factor in Model 2, Model 3, and Model 4.

For example, according to Model 2 among White females, each 0.01 unit increase in childhood Gini index is associated with 2.6% (= [exp(2.573*0.01)−1]*100) increase in the level of PD; each 1 percentage point increase in childhood union coverage is associated with 1.8% (= [exp(−0.018)−1]*100) decrease in the level of PD; each 1 percentage point increase in childhood GDP growth rate is associated with 6% (= [exp(−0.064) −1]*100) decrease in the level of PD; and each 1 percentage point increase in childhood unemployment rate is associated with 0.2% (= [exp(0.002)−1]*100) increase in the level of PD. The "% change" column from Model 1 to Model 2 shows that childhood Gini index explains over 70% of variation in PD across cohorts, while the contributions of the other three early life factors to the cohort variation in PD are substantially smaller. Supplemental Information 17–19 visually display that accounting for these three early life factors does not change the cohort trend as much as childhood Gini (Figure 2). Additional controls of individual adulthood factors (Model 3) and contemporary period macro factors (Model 4) generally do not explain the birth cohort variance component.

Discussion and Conclusion

Around the turn of the century, Americans' health began to decline. After decades of improvement in functioning and a decline in disability among the U.S. population ages 65 and older (Crimmins et al., 1997; Cutler, 2001; Freedman et al., 2004; Manton et al., 2006; Schoeni et al., 2001); starting in the late 1990s, however, newer cohorts approaching middle

age (40–59) and early old age (60–69) began to experience increasing functional limitations and disability (Freedman et al., 2013; Fuller-Thomson et al., 2009; Martin et al., 2009, 2010; Seeman et al., 2010). In the early 2000s, the worsening disability trend was accompanied by increasing mortality rates among the middle-aged (Case and Deaton, 2015; Montez and Zajacova, 2013). A more alarming picture is that this elevated morbidity and mortality is not constrained to Baby Boomers who reached middle age and early old age in the past two decades but extends to younger cohorts (Zang et al., 2019; Zheng and Echave, 2021).

Against this backdrop, this study corroborates that Americans' health has been continuously declining since the Early Baby Boomer cohort, no matter whether it is measured by physiological status based on a list of biomarkers or a disease index based on several chronic illnesses. It is consistently observed in two large nationally representative data sets. Scientific research is urgently needed to understand the cause of this health crisis. We find the cohort trend in physiological dysregulation very closely mirrors the cohort trend in early life income inequality exposure (Figure 1). By linking NHANES and PSID data to historical income inequality data, we find that early life income inequality exposure is significantly associated with poorer adulthood health, especially for White males and Black females (Table 1–2, Supplemental Information 5–10). But more importantly, we find early life income inequality exposure explains over 70% of cohort variation in PD and CDI. These findings are very robust to a wide range of controls, including early life health and family background, adulthood sociodemographic and economic factors, and contemporary macro socioeconomic determinants (e.g., contemporary income inequality). These findings are not simply due to a temporal correlation, as other similar negative temporal trends in early life exposures (i.e., childhood percentage of union membership, childhood GDP growth rate, and childhood unemployment rate) do not exhibit equivalent contributions to declining health across cohorts (Table 3, Supplemental Information 14–19).

Our study not only sheds light on the socioeconomic mechanisms that may shape the health decline in the United States, but also contributes to the substantial debate on the health consequences of income inequality. Our study suggests that income inequality experienced during childhood has a long-term impact on adult health, consistent with some prior studies (e.g., Lillard et al., 2015). Thus, childhood may be a sensitive period in which income inequality is strongly associated with poor health in adulthood. This is probably because children growing up in a period with rising income inequality may be particularly influenced by its negative socioeconomic and psychosocial consequences. Prior literature has demonstrated that income inequality can lead to decreasing public expenditures (e.g., public education and health care) (Gilens, 2012; Osberg et al., 2004), intensified relative deprivation (Kawachi and Kennedy, 1999; Kondo et al., 2008), and erosion of social capital (Kawachi et al., 1997; Wilkinson, 1992, 1996). These then reduce the socioeconomic, psychological, and healthcare resources families and children need to produce and protect health, the negative impact of which may contribute throughout the life course. The time lag between income inequality and health also may be due to both the time requirement from income inequality to the health pathways (Kondo et al., 2011) and the latency period between exposure to risk factors and diseases initiation, deterioration, and subsequent mortality (Lynch and Davey Smith, 2005).

Due to the nature of our research design, we are not able to infer causality in the link between income inequality and health from the econometrics standard. But temporality, an important criterion in the epidemiological causal framework (Pickett and Wilkinson, 2015), and robustness to control of a wide range of confounders do yield a certain degree of confidence about the validity of this link. Researchers who doubt that income inequality has an effect on health tend to argue that income inequality is a consequence of policies and social processes, therefore we should focus on the processes that generate income inequality rather than income inequality itself (Case and Deaton, 2020: 134). We, however, view income inequality as a meaningful antecedent of health in and of itself. Income inequality may be a proxy for broader institutional and systematic problems, but the health consequences of these processes and institutions work directly through inequality. Income inequality not only may have an impact on population health and mortality, but also may reinforce the processes that generate income inequality.

This study has several limitations. First, it only examines the contextual effect of income inequality at the national level. Several scholars have demonstrated that national income inequality and social hierarchies matter more to people than local social inequality structures (e.g., Kondo et al., 2011; Wilkinson and Pickett, 2006). Further research can investigate the long-term impact of childhood income inequality at the state level on adulthood morbidity and mortality risk, which, however, requires researchers to have complete data on respondents' residential histories during their childhoods. Empirically, we think the findings will be similar as (1) we examine the association between cohort trend in childhood income inequality and cohort trend in adult health and (2) the overall temporal trend in income inequality is remarkably similar across states even though levels differ (Zheng and George, 2012). Second, even though we controlled a long list of possible confounders including childhood and adulthood socioeconomic and demographic factors and contemporary period determinants, other confounders may still influence both childhood income inequality and adult health.

There are several priority issues for future research. First, we rely on prior literature that has both conceptualized and empirically tested mechanisms linking income inequality to health (e.g., Davey, 1996; Delhey and Dragolov, 2014; Kaplan et al., 1996; Kawachi et al., 1997; Kawachi and Kennedy, 1999; Kondo et al., 2008; Pickett and Wilkinson, 2015; Zimmerman and Bell, 2006), and hypothesize that both material mechanisms (reductions in social spending) and psychosocial mechanisms (erosion of social capital and aggravation of relative deprivation) may have long-lasting consequences and contribute to the link between childhood income inequality exposure and adult health. Nonetheless, we are not able to test these and other potential mechanisms (e.g., reduced economic mobility and opportunities, increased geographic segregation, see Bor et al., 2017) due to both data limitations and the scope of this study. Future studies should directly test these and other potential mechanisms.

Second, results from the NHANES data indicate that the impact of childhood income inequality was especially strong for White males and Black females. These relationships are robust to controls for individual level childhood health and family background, adulthood socioeconomic and marital status, and contemporary macro socioeconomic factors. Given the broad array of variables controlled, it is difficult to understand why childhood income

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inequality was more important for White men and Black women than for the other four sex by race/ethnicity groups. These patterns are observed across cohorts, suggesting that the increased health risks experienced by White men and Black women reflect long-term, structural sources of vulnerability. Future research should both confirm these results in other data sets and examine possible explanations for them.

In conclusion, this study portrays a robust link between early life income inequality exposure and adult health and proposes a mechanism that may account for cohort differences in health. Although there are likely other contributing factors, our results suggest that the declining health across cohorts since the Early Baby Boomers may be partially due to increasing inequalities Americans face in early life. This may imply that reducing income inequality has a promising prospect of reversing this unfavorable trend and improving the health of current and future cohorts. Childhood income inequality exposure is linked to adult health probably through both material and psychosocial pathways. If these untested pathways are true, narrowing the income gap may require not only redistributive tax policies and a reduction in income differences before taxes from the top down (Pickett and Wilkinson, 2015) but also a strengthening of social cohesion and communities from the bottom up (Rajan, 2019).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

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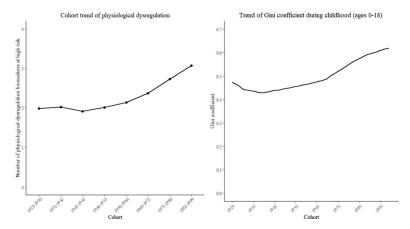
Figure 1. Cohort trend in physiological dysregulation and childhood Gini coefficient
Notes: Date source for physiological dysregulation is National Health and Nutrition
Examination Survey (NHANES) 1988–2018. Physiological dysregulation is summary index
of nine biomarkers including seven markers of metabolic functions, one marker of chronic
inflammation (i.e., low serum albumin), and one additional marker (i.e., urinary function-
creatinine clearance). The cohort trend is obtained from the random effect coefficients of
cohort based on Poisson mixed effects models with fixed effect coefficient of age and
random effect coefficients of cohort and period. Gini coefficient during childhood (ages
0–18) is computed based on Internal Revenue Service income data (Frank, 2014). The
childhood Gini coefficient is the average of Gini coefficients each cohort is exposed to
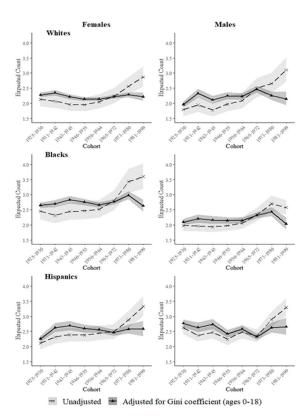between ages of 0 and 18.

Figure 2. Trend in number of physiological dysregulation biomarkers at high risk across birth cohorts with/without adjusting for childhood Gini coefficient (ages 0–18)

Table 1.

Fixed and Random Effect Estimates from Poisson Mixed Models of Physiological Dysregulation, NHANES, 1988–2018

	Model1		Model2		Model3		Model4	
	Coef.	S.E	Coef.	S.E	Coef.	S.E	Coef.	S.E
Fixed effects								
Intercept	0.765 **	0.059	0.774 **	0.018	0.998 **	0.015	0.142	0.100
Age	0.028 **	0.000	0.028 **	0.000	0.028 **	0.000	0.027 **	0.000
Age squared	0.000 **	0.000	0.000 **	0.000	0.000 **	0.000	0.000 **	0.000
Female	0.040 **	0.000	0.040 **	0.000	0.035 **	0.000	0.035 **	0.000
Race (ref: Whites)								
Blacks	0.118 **	0.000	0.118 **	0.000	0.073 **	0.000	0.073 **	0.000
Hispanics	0.149 **	0.000	0.149 **	0.000	0.067 **	0.000	0.068 **	0.000
Gini index [0–18]			2.989 **	0.219	3.028 **	0.175	2.467 **	0.167
Education (ref: Less than high school)								
High school diploma					0.003 **	0.000	0.003 **	0.000
More than high school					-0.127 **	0.000	-0.129 **	0.000
Marital status (ref: Married)								
Widowed					-0.011 **	0.000	-0.008 **	0.000
Divorced/Separated					-0.071 **	0.000	-0.070 **	0.000
Never married					-0.046 **	0.000	-0.044 **	0.000
Poverty income ratio (ref: Low (<1))								
Middle (1, <3)					-0.067 **	0.000	-0.066 **	0.000
High (3)					-0.175 **	0.000	-0.173 **	0.000
Period macro measures								
Gini index							0.721 **	0.123
Percent of college							0.012 **	0.002
Percent of union							0.040 **	0.003

	Model1		Model2		Model3		Model4	
	Coef.	S.E	Coef.	S.E	Coef.	S.E	Coef.	S.E
GDP growth rate							-0.026**	0.001
Unemployment rate							-0.040**	0.001
Random Effects								
Birth cohorts								
Early children of depression (1925–1930)	-0.107	0.056	-0.043**	0.012	-0.063**	0.009	-0.047**	0.010
Late children of depression (1931–1942)	-0.092	0.056	0.045**	0.015	0.035**	0.012	0.027*	0.012
War babies (1943–1945)	-0.149**	0.056	-0.017	0.014	-0.013	0.011	-0.029*	0.012
Early-baby Boomers (1946–1955)	-0.108	0.056	-0.011	0.013	0.006	0.010	-0.014	0.011
Late-baby Boomers (1956–1964)	-0.060	0.056	-0.015	0.011	-0.010	0.009	-0.034**	0.010
Early-gen Xers (1965–1972)	0.051	0.056	0.030	0.011	0.034**	0.009	0.010	0.009
Late-gen Xers (1973–1980)	0.173**	0.056	0.029**	0.015	0.030*	0.012	0.017	0.012
Gen Yers (1981–1999)	0.292**	0.056	-0.018	0.025	-0.019	0.020	-0.016	0.019
Survey years								
Wave3 (1988–1994)	0.081**	0.018	0.081**	0.015	0.059**	0.012	0.012**	0.003
1999–2000	0.010	0.018	0.010	0.015	0.002	0.012	-0.042**	0.002
2001–2002	0.015	0.018	0.015	0.015	0.018	0.012	-0.012**	0.002
2003–2004	0.017	0.018	0.017	0.015	0.012	0.012	0.036**	0.002
2005–2006	0.021	0.018	0.021	0.015	0.026*	0.012	0.009**	0.002
2007–2008	0.053**	0.018	0.052**	0.015	0.053**	0.012	0.022**	0.002
2009–2010	-0.070**	0.018	-0.070**	0.015	-0.067**	0.012	0.022**	0.002
2011–2012	-0.078**	0.018	-0.078**	0.015	-0.075**	0.012	0.016**	0.002
2013–2014	-0.066**	0.018	-0.066**	0.015	-0.063**	0.012	-0.045**	0.002
2015–2016	-0.063**	0.018	-0.063**	0.015	-0.055**	0.012	-0.069**	0.002
2017–2018	0.080**	0.018	0.080**	0.015	0.090**	0.012	0.050**	0.002
Variance components								
Birth cohorts	0.025*	0.013	0.001*	0.000	0.001**	0.000	0.001*	0.000

| | Model1 | | Model2 | | Model3 | | Model4 | |
	Coef.	S.E	Coef.	S.E	Coef.	S.E	Coef.	S.E
Survey years	0.004 *	0.002	0.002 **	0.001	0.002 **	0.000	0.000	0.000
Pseudo-BIC	290,040,000		290,040,000		285,620,000		285,620,000	
Sample size	35,509		35,509		35,509		35,509	

Notes:

*
<0.05

**
<0.01

Abbreviations: Coef., regression coefficients or variance estimates for random effect; S.E, standard error.

Table 2.

Fixed and Random Effect Estimates from Poisson Mixed Models of Adult Chronic Disease Index, PSID, 1999–2013

	Model1		Model2		Model3		Model4		Model5	
	Coef.	S.E	Coef.	S.E	Coef.	S.E	Coef.	S.E	Coef.	S.E
Fixed effects										
Intercept	−0.564**	0.131	−3.811**	0.566	−3.829**	0.511	−3.260**	0.554	2.406	2.687
Age	0.056**	0.000	0.056**	0.000	0.058**	0.000	0.062**	0.000	0.062**	0.000
Age squared	0.000**	0.000	0.000**	0.000	0.000**	0.000	0.000**	0.000	0.000**	0.000
Female	0.129**	0.002	0.129**	0.002	0.087**	0.002	0.037**	0.002	0.037**	0.002
Blacks	0.279**	0.002	0.279**	0.002	0.282**	0.002	0.184**	0.002	0.184**	0.002
Hispanics	−0.056**	0.004	−0.056**	0.004	−0.060**	0.004	−0.115**	0.004	−0.115**	0.004
Gini index [0–18]			6.769**	1.173	6.508**	1.059	6.712**	1.147	6.702**	1.146
Disease index before 17					0.291**	0.001	0.281**	0.001	0.281**	0.001
Parental smoking before 17					0.158**	0.002	0.135**	0.002	0.135**	0.002
Mother's education (ref: less than high school)										
High school graduate					−0.144**	0.002	−0.094**	0.002	−0.094**	0.002
Some college					−0.183**	0.003	−0.092**	0.003	−0.092**	0.003
College graduate					−0.184**	0.003	−0.068**	0.003	−0.068**	0.003
Graduate degree					−0.176**	0.005	−0.023**	0.005	−0.023**	0.005
Highest grade attained							−0.036**	0.000	−0.036**	0.000
Family income to needs ratio							−0.019**	0.000	−0.019**	0.000
Marital/cohabitating status							−0.121**	0.002	−0.121**	0.002
Period macro measures										
Gini index									0.144	1.924
Percent of college									−0.133	0.069
Percent of union									−0.268*	0.114
GDP growth rate									0.001	0.017
Unemployment rate									0.020	0.025

	Model1		Model2		Model3		Model4		Model5	
	Coef.	S.E	Coef.	S.E	Coef.	S.E	Coef.	S.E	Coef.	S.E
Random Effects										
Birth cohorts										
Early children of depression (1925–1930)	−0.446 **	0.130	−0.314 **	0.059	−0.284 **	0.054	−0.307 **	0.058	−0.307 **	0.058
Late children of depression (1931–1942)	−0.310 *	0.129	−0.001	0.076	−0.017	0.069	−0.021	0.074	−0.021	0.074
War babies (1943–1945)	−0.158	0.129	0.134	0.074	0.124	0.067	0.122	0.073	0.122	0.072
Early-baby Boomers (1946–1955)	−0.082	0.129	0.132 *	0.066	0.124 *	0.059	0.141 *	0.064	0.141 *	0.064
Late-baby Boomers (1956–1964)	−0.118	0.129	−0.019	0.057	−0.002	0.051	−0.001	0.056	−0.001	0.056
Early-gen Xers (1965–1972)	0.046	0.129	−0.007	0.055	−0.002	0.050	0.005	0.054	0.005	0.054
Late-gen Xers (1973–1980)	0.436 **	0.130	0.083	0.082	0.059	0.074	0.072	0.080	0.072	0.080
Gen Yers (1981–1995)	0.633 **	0.130	−0.008	0.123	−0.002	0.111	−0.011	0.121	−0.011	0.120
Survey years										
1999	−0.111 **	0.021	−0.111 **	0.021	−0.104 **	0.020	−0.103 **	0.020	0.006	0.044
2001	−0.059	0.020	−0.059 **	0.020	−0.055 **	0.020	−0.053 **	0.020	−0.040	0.033
2003	0.019	0.020	0.019	0.020	0.025	0.020	0.025	0.020	0.023	0.038
2005	0.059 **	0.020	0.059 **	0.020	0.062 **	0.020	0.063 **	0.020	0.018	0.031
2007	0.050 *	0.020	0.050 *	0.020	0.051 **	0.020	0.051 **	0.020	0.003	0.038
2009	0.023	0.020	0.023	0.020	0.020	0.020	0.022	0.020	0.026	0.040
2011	0.009	0.020	0.009	0.020	0.002	0.020	−0.001	0.020	−0.027	0.039
2013	0.010	0.020	0.010	0.020	−0.002	0.020	−0.003	0.020	−0.008	0.043
Variance components										
Birth cohorts	0.134 *	0.072	0.024 *	0.014	0.019 *	0.011	0.023 *	0.013	0.022 *	0.013
Survey years	0.003 *	0.002	0.003 *	0.002	0.003 *	0.002	0.003 *	0.002	0.002	0.002
Pseudo-BIC	2,205,878		2,205,889		1,990,867		1,930,704		1,930,770	
Sample size	12,924		12,924		12,924		12,924		12,924	
Observations	68,271		68,271		68,271		68,271		68,271	

Notes:

*
<0.05

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** <0.01

Abbreviations: Coef., regression coefficients or variance estimates for random effect; S.E, standard error.

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Table 3.

Comparison of the Percentage Change in Birth Cohort Variance Components from Model1 to Model4 by Each Early Life Exposure Measure, NHANES, 1988–2018

Models	Gini Index [0–18]		Percentage of Union [0–18]		GDP Growth Rate [0–18]		Unemployment Rate [0–18]	
	% change	coef.	% change	coef.	% change	coef.	% change	coef.
White Females								
Model 1-Model 2	94.39%	2.573**	75.10%	-0.018**	53.79%	-0.064	14.52%	0.002
Model 1-Model 3	97.36%	2.974**	42.83%	-0.018*	52.12%	-0.096*	5.50%	-0.007
Model 1-Model 4	N.C.	.	50.97%	-0.018*	34.88%	-0.096	N.C.	.
White Males								
Model 1-Model 2	87.29%	3.681**	-21.46%	-0.019	-8.63%	-0.123	-43.14%	-0.017
Model 1-Model 3	58.32%	3.814**	-7.30%	-0.019	-3.09%	-0.134	12.21%	-0.020
Model 1-Model 4	N.C.	.	-61.81%	-0.019	22.33%	-0.134*	N.C.	.
Black Females								
Model 1-Model 2	92.62%	3.128**	38.54%	-0.018*	30.02%	-0.119*	-14.83%	-0.009
Model 1-Model 3	93.26%	2.770**	50.43%	-0.016*	47.62%	-0.107*	11.07%	-0.008
Model 1-Model 4	93.26%	2.771**	50.28%	-0.016*	46.84%	-0.107*	9.62%	-0.008
Black Males								
Model 1-Model 2	78.75%	2.351**	26.88%	-0.014*	23.57%	-0.089	-19.70%	-0.006
Model 1-Model 3	79.18%	2.464**	19.25%	-0.014	19.98%	-0.094	-23.12%	-0.007
Model 1-Model 4	79.53%	2.465**	22.05%	-0.014	22.39%	-0.094	-18.35%	-0.007
Hispanic Females								
Model 1-Model 2	81.89%	2.522**	9.85%	-0.011	26.12%	-0.095	-0.87%	-0.021
Model 1-Model 3	83.43%	2.362**	19.23%	-0.010	29.74%	-0.086	15.00%	-0.020
Model 1-Model 4	83.45%	2.362**	18.67%	-0.010	29.74%	-0.086	12.09%	-0.020

	Gini Index [0–18]		Percentage of Union [0–18]		GDP Growth Rate [0–18]		Unemployment Rate [0–18]	
Models	**% change**	**coef.**	**% change**	**coef.**	**% change**	**coef.**	**% change**	**coef.**
Hispanic Males								
Model 1-Model 2	74.65%	2.161 **	60.73%	−0.017 **	−0.06%	−0.053	−12.38%	0.005
Model 1-Model 3	72.45%	1.995 **	67.47%	−0.016 **	9.07%	−0.046	1.19%	0.006
Model 1-Model 4	72.88%	1.995 **	67.35%	−0.016 **	12.26%	−0.046	−12.38%	0.006

Notes:

*
<0.05

**
<0.01

Abbreviations: Coef., regression coefficient of each early life exposure measure; % change, percentage change in birth cohort variance component from Model 1 to each model; N.C, not converged

Model1: includes age, cohort, period.

Model2: additionally adjusts for each early life exposure measure.

Model3: additionally adjusts for educational attainment, marital status, and poverty income ratio.

Model4: additionally adjusts for five period macro measures including Gini index, GDP growth rate, unemployment rate, percentage of union, and percentage of college education.