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Life Expectancy and the Labor Share in the U.S.

Michael Cauvel and Miguel Alejandro Sanchez

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Abstract

We estimate the relationship between life expectancy and the labor share in the U.S., finding a positive and significant relationship. We then explore eight potential channels through which the labor share may impact life expectancy: social capital, policy, mental health, stress, crime, childhood adversity, healthcare utilization, and education. Our results suggest that healthcare utilization and education are two important channels linking life expectancy and the labor share. We expect that these relationships are driven by dynamics related to Baumol's cost disease. We also find evidence of a third channel related to policy, suggesting that rising inequality has resulted in policies that are less conducive to growth in life expectancy.

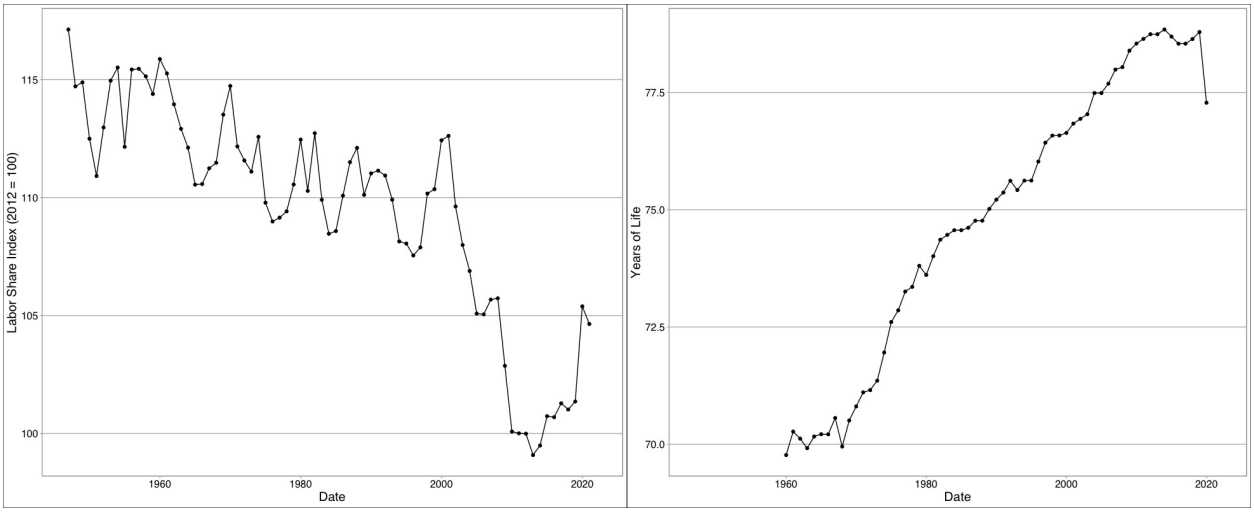
Keywords: Labor share, life expectancy, Baumol's cost disease
JEL codes: E25, I14, I24

*Assistant Professor of Economics at University of Southern Maine and Doctoral Student at Johns Hopkins University, respectively. Corresponding email: michael.cauvel@maine.edu. The authors would like to thank Vinicius Cicero and participants at the Eastern Economic Association's 49th Annual Conference for providing helpful comments.

1 Introduction

Two notable trends impacting the United States in recent decades have been rising functional inequality—i.e. a declining labor share—and slowing improvements in life expectancy. Both trends are illustrated in Figure 1. As shown in Panel (a), the labor share experienced a gradual downward trend between the 1940s and 1990s—a trend that accelerated in the early 2000s before a slight rebound occurred. Meanwhile, the growth rate of life expectancy in the U.S. has been low relative to other high-income countries (Harper et al., 2021). As Panel (b) shows, this rate has declined considerably from its high rate of 0.46% per year in the 1970s, being halved in the following three decades before falling to a paltry .05% per year in the 2010s and even turning negative in a few years in the back half of this decade. We explore whether these two trends are related and, if so, which mechanisms might connect them.

We first estimate the relationship between the labor share and life expectancy. We find a significant positive relationship, suggesting that a decline in the labor share is associated with slower growth in life expectancy (controlling for GDP growth). We then test whether several different factors, suggested by the related literature, mediate this relationship. We do not find any evidence suggesting that mental health or crime are significantly related to either life expectancy or the labor share. Three other factors—social capital, stress, and childhood adversity—are found to be significant determinants of life expectancy, but these factors are not significantly related to the labor share. We do, however, find evidence suggesting the presence of three channels linking the labor share and life expectancy—healthcare utilization, education, and policy. All three of these variables are positively related to the labor share and associated with faster growth of life expectancy. We expect that the healthcare spending and educational attainment channels reflect Baumol's cost disease, which may increase the cost of these services as productivity increases throughout the economy. Meanwhile, a declining labor share implies that productivity is growing faster than real wages, making it more



(a) Business Sector Labor Share Index **(b)** Life Expectancy at Birth

Figure 1: Trends in U.S. Labor Share and Life Expectancy
Sources: BLS and World Bank (via FRED)

difficult for workers to afford these services that positively impact health. The policy channel may be closely related to the consumption of these services as well, as the government can play a central role in improving the access to and affordability of healthcare, education, and other services that are beneficial for health.

We view our analysis as a preliminary one, noting that additional research is needed to fully understand these three channels and other complex dynamics linking the functional distribution of income to health. We should also caution that the presence of other important channels should not be ruled out based on our findings, as our results are limited to the U.S., likely to capture at most medium-run effects, and could be sensitive to measurement choices. Moreover, examining GDP growth as a potential channel linking the labor share and life expectancy is beyond the scope of this paper, but we expect that this channel may be important given various related results in the literature, as discussed below.

The remainder of this paper is organized as follows. Section 2 reviews the relevant literature, while the methodology and data are discussed in Sections 3 and 4, respectively. The results are presented in Section 5, and Section 6 concludes.

2 Literature Review

Although the decline in the labor share in the U.S. and in many other countries has recently received considerable attention from researchers, most previous work has focused on explaining the causes of this trend. Synthesizing this vast literature, Cauvel and Pacitti (2022) argue that most explanations focus on structural macroeconomic changes, including "deindustrialization, globalization, financialization, market concentration, and technological change," which operate through the same channel—labor bargaining power. In other words, the declining labor share can largely be explained as the result of an erosion in labor's ability to bargain for higher wages.

Because most researchers in this area have focused on explaining the reasons for the declining labor share, the consequences of this trend are still largely unknown. To the extent that its effects have been considered in the literature, it has been in the context of research exploring the relationship between the labor share and various measures of output. The findings generally suggest that a lower labor share is associated with slower growth in the long run (Kiefer and Rada, 2015; Blecker, 2016; Charpe et al., 2020; Barrales-Ruiz et al., 2021; David Avritzer, 2022), although there is some debate regarding the short-run effects (Stockhammer, 2017; Nogueira Rolim, 2019; Barrales-Ruiz et al., 2021; Cauvel, 2022). Beyond its connections to the business cycle and long-run growth, little is known about the ways in which a reduction in the labor share impacts society. We hope to begin filling in these gaps by exploring the relationship between the labor share and another key measure of well-being—life expectancy.

To our knowledge, no study has previously examined the relationship between health and the functional distribution of income. However, there are reasons to expect that they may be linked. Broadly speaking, there is considerable evidence that economic factors impact health (Naik et al., 2019). Recent research in this area suggests that some of the interrelated structural macroeconomic changes driving the decline in the labor share—globalization, au-

tomation, and deindustrialization—have increased mortality (Autor et al., 2019; Pierce and Schott, 2020; Adda and Fawaz, 2020; O'Brien et al., 2022). O'Brien et al. (2022) argue that there are two complementary channels through which such macroeconomic changes might impact health. The first, which they call the "material pathway," suggests that reduced wages and employment may negatively impact health because individuals reduce their utilization of healthcare (with reduced income and access to health insurance) and communities lower public spending (as tax revenue falls). The other—the "despair pathway"—suggests that a lack of economic opportunities leads to an increase in "deaths of despair" (Case and Deaton, 2017) related to substance abuse or suicide. The former is related to a well-established literature documenting the relationship between income and health, both at an individual level (Chetty et al., 2016) and at the national level, where GDP per capita is found to be a strong determinant of life expectancy (Jetter et al., 2019).

Perhaps most saliently, a considerable amount of research has examined the link between life expectancy and personal inequality. This work has spanned a number of academic disciplines, with work in economics (e.g. Monheit, 2022), demography (e.g. Zhao et al., 2021), and sizable literatures in sociology, criminology, public health, and epidemiology (Pickett and Wilkinson, 2015). Although summarizing the entirety of this expansive literature is beyond the scope of this paper, a number of literature surveys and meta analyses have explored patterns in the findings of the many existing studies and highlighted some important remaining questions (Wagstaff and van Doorslaer, 2000; Deaton, 2003; Macinko et al., 2003; Lynch et al., 2004; Subramanian and Kawachi, 2004; Wilkinson and Pickett, 2006; Kondo et al., 2009, 2012; O'Donnell et al., 2015; Pickett and Wilkinson, 2015).

There is little question that there is at least some connection between personal inequality and life expectancy, as many studies have identified a clear association between the two in the data. This association has been found using various methodologies and data from various locations at many different levels of analysis. Although the clearest evidence is found at the international level (Pickett and Wilkinson, 2015), there is a consistent correla-

tion within U.S. states and counties (Truesdale and Jencks, 2016), and recent work provides compelling evidence at the neighborhood level (Zhao et al., 2021). Similarly, although the evidence is clearest in the U.S. (Truesdale and Jencks, 2016), a similar association has also been found in studies of Europe (Hu et al., 2015) and less-developed areas, including Sub-Saharan Africa (Odusanya and Akinlo, 2021).

Although the evidence of an association between personal inequality and life expectancy is clear, the appropriate interpretation of the evidence is debated. In particular, there is an unresolved question about whether this association represents a causal relationship or a spurious one. Pickett and Wilkinson (2015) argue that the evidence is suggestive of a causal relationship, noting that "the major causal criteria of temporality, biological plausibility, consistency and lack of alternative explanations are well supported" (p. 316). Others disagree. For example, assessing many of the previous surveys of the literature—albeit curiously omitting some reviews that are more favorable towards a causal interpretation (e.g. Wilkinson and Pickett, 2006; Pickett and Wilkinson, 2015)—Monheit (2022) argues that the evidence as a whole does not support a causal interpretation.

An important component of this debate is the question of whether estimates are capturing an absolute-income effect or an independent impact of inequality itself. Gravelle (1998) argues that if there is a concave relationship between income and mortality risk, such that there are diminishing returns to income in terms of reducing mortality risk, countries with more unequal income distributions will have worse health simply because a greater share of the population has a lower absolute income. Based on this argument, evidence of poorer health in more unequal countries does not necessarily indicate that inequality in and of itself causes poorer health. This discussion has been framed as the difference between a "concavity effect" of absolute income and a "contextual effect" or "pollution effect" of inequality itself

(Subramanian and Kawachi, 2004).[1] Monheit (2022) argues that the existing evidence is suggestive of the concavity effect but not the contextual effect. On the other hand, Pickett and Wilkinson (2015) argue that multi-level studies controlling for individual income provide evidence of an additional contextual effect of inequality.

Although this is an interesting question, differentiating between these two effects is beyond the scope of this paper. As Gravelle (1998) argues, aggregate data is not well-suited to differentiating between the effects of relative and absolute income; multilevel studies are best-equipped to handle this task (Subramanian and Kawachi, 2004). Moreover, we do not view the distinction between the concavity and contextual effects to be important in the context of our study. We are interested in exploring the impact of macroeconomic changes, as captured by the decline in the labor share, on population health. For this purpose, the question of whether any potential effects are the result of slower income growth for workers or a decline in relative social standing in and of itself is largely immaterial, as the ultimate costs and policy implications are essentially the same in either case. Although studying the latter type of effect remains important for the development of a full understanding of the nature of inequality and its multifaceted impact, the effects of inequality on individuals' absolute incomes should not be downplayed on the semantic basis that they are not independent of income itself. Indeed, individual incomes and the distribution of income are inextricably linked, and slower income growth for the majority of the population is likely the most direct way in which rising inequality impacts society.

Another important issue related to the question of causality is omitted variable bias. Some studies (e.g. Hu et al., 2015) show that the relationship between inequality and life expectancy becomes insignificant when controlling for other factors, suggesting that the

[1] Truesdale and Jencks (2016) similarly note that inequality can affect individuals both directly, through their own income, and indirectly, by changing the incomes of others and by extension the social, political, and economic environment. It is also interesting to note the parallels between the framework of concavity vs. contextual effects and the material and despair pathways suggested by O'Brien et al. (2022).

relationship is spurious. However, Pickett and Wilkinson (2015) contend that many of the control variables used in the literature, such as health expenditures, alcohol or tobacco use, and education are inappropriate, as they may be channels through which inequality affects health. For example, if more education leads to better health but inequality leads to lower educational attainment, controlling for education would lead to underestimates of the true effect of inequality on health.

Although the relationship between health and income inequality has been widely studied, it is still not fully understood. Recent surveys of the literature have outlined some important issues requiring further examination. One such issue is measurement. Pickett and Wilkinson (2015) cite a need for analysis using different measures of inequality. As Monheit (2022) explains, the Gini coefficient is the measure used most commonly in the literature, although income shares at various points in the income distribution are also fairly common, and measures based on variance have occasionally been used. Notably, to our knowledge only measures of personal inequality have been tested in this context. Another important issue is treatment of lags (Pickett and Wilkinson, 2015; Truesdale and Jencks, 2016). Truesdale and Jencks (2016) argue that most studies capture only short-term effects, even though theory suggests long lags. Finally, as the discussion above illustrates, causality remains a key question. As such, further examination of specific causal mechanisms is a critical area requiring additional research (Pickett and Wilkinson, 2015; Truesdale and Jencks, 2016).

We aim to contribute to this literature by exploring these three issues. In addition to testing the relationship between life expectancy and another measure of income inequality—the labor share—we examine issues related to lags and explore several causal channels through which inequality might affect health. Both of these issues are conducive to study using time-series analysis of aggregate data. Although aggregate analysis cannot easily differentiate between concavity and contextual effects of inequality on health, as discussed above, it has a number of advantages that make it a useful complement to individual and multilevel analysis. First, time-series econometric techniques provide the necessary tools to

appropriately consider lag specifications. We use an autoregressive distributed lag (ARDL) model in which lags can be included for all of the dependent and independent variables. The specific lag specification for each model is selected based on model fit. Moreover, analysis at the aggregate level is well-suited to the study of structural relationships that unfold over long periods of time.[2] Given the wide variety of reliable, decades-long aggregate data series that is available for a country like the U.S., it can also be used to examine several potential causal channels. Such analysis is rarely possible in individual-level analysis due to data constraints in terms of both length and breadth. This likely explains why studies do not typically empirically examine several potential causal channels at once. Despite these advantages of aggregate-level time-series analysis, relatively little research in this area has employed a longitudinal approach (Truesdale and Jencks, 2016).

We use such an approach first to examine the relationship between the labor share and life expectancy overall, and then to explore several potential causal channels relating the two in order to inform the debate regarding causality. Note that we do not examine causality in the sense of differentiating between concavity and contextual effects of inequality. Moreover, we do not argue that any observed relationship between the labor share and life expectancy is independent of the macroeconomic factors—such as automation, globalization, and deindustrialization—that influence the labor share. Rather, we view functional inequality as an important link in the causal chain that extends from structural changes in the U.S. macroeconomy to changes in life expectancy. In our view, examining the relationship between the labor share and health is useful because the labor share captures the impact of many different economic forces, and therefore allows for a comprehensive analysis.

There are a number of potential causal channels through which the labor share may affect life expectancy. We discuss nine below, eight of which we explore empirically. The

[2]Methodological choices such as the use of annual data and the differencing of some data series likely limit our analysis to at most the medium run. However, future research using different econometric techniques could capture longer-run relationships following a framework that is similar to ours.

remaining channel—economic growth—is beyond the scope of this paper, but likely of relevance to the discussion of the declining labor share. Although this is not an exhaustive list of every potential causal mechanism, these are nine of the most common theoretically plausible channels suggested by the literature.

Social Capital

One popular theory in the literature is that inequality can be "socially corrosive" (Zhao et al., 2021) and contribute to a "decay of social capital" (Truesdale and Jencks, 2016). Based on this theory, inequality may erode trust, weaken community ties, and diminish civic engagement, potentially resulting in worse health outcomes as individuals become more stressed and have fewer sources of support (Kawachi et al., 1997; Truesdale and Jencks, 2016; Zhao et al., 2021). Income inequality has been linked to lower levels of both trust (Barone and Mocetti, 2016; Kanitsar, 2022) and civic engagement (Schröder and Neumayr, 2023). Kawachi et al. (1997) find evidence suggesting that inequality reduces social capital, leading to higher mortality and increased prevalence of some other negative health outcomes. Social capital also appears to impact mental health (Silva et al., 2016), and could be related to levels of violence (Zhao et al., 2021)—both of which are additional channels discussed below.

Policy

A related channel may operate through policy. Higher inequality could lead to "political capture" wherein the wealthy gain greater ability to influence policy, resulting in cuts to social safety nets and reduced investment in public goods that are beneficial for health—e.g. parks, sanitation, and education (Truesdale and Jencks, 2016). This type of underinvestment in public goods could also be related to the decay of social capital (Truesdale and Jencks, 2016; Zhao et al., 2021). Other policy choices could impact health as well. For example, regulations can affect nutrition, substance abuse, and the quality of air and water, the government's responsiveness to the business cycle can help to determine the magnitude of health effects related to unemployment and financial insecurity, and countless policies

could potentially affect health via their effects on economic growth and the distribution of income. Research supports the view that policies are an important determinant of health (Montez et al., 2020), and spending on social safety nets in particular appears to be a strong predictor of mortality across both countries (Beckfield and Bambra, 2016) and U.S. states (Fenelon and Witko, 2021).

Regarding the other piece of the causal chain, Wisman (2013) argues that rising inequality gives wealthy and corporate interests greater control over ideology and politics, suggesting that these dynamics were ultimately responsible for policy changes including deregulation and reduced welfare spending. An empirical study by Gilens and Page (2014) supports the view that economic elites and business interests have substantial sway over policy, while average citizens have little. Montez (2020) argues that trends in U.S. life expectancy can be explained in part by state-level policy changes—especially in conservative states—driven by these interests.

Montez et al. (2021) note that corporate power is of particular importance, as corporations have a large impact on health through the products that they sell and their environmental impacts, in addition to their influence on policy. They argue that an understanding of commercial, political-economic, and legal forces is necessary to understand the determinants of health. If rising corporate power is a major driver of health via policy, this trend would likely be captured more effectively by the functional distribution of income than the personal distribution of income.

Mental Health

Another popular theory suggests that inequality leads to worse health outcomes because it fosters "status anxiety" (Layte and Whelan, 2014; Pickett and Wilkinson, 2015; Zhao et al., 2021). As Zhao et al. (2021) explain, this theory suggests that individuals making Veblenian status comparisons experience negative health effects if they are unhappy with

their relative rank. Layte and Whelan (2014) find higher levels of status anxiety across the income distribution in countries with higher levels of inequality.

However, relative status is only one avenue through which inequality might affect mental health. In addition to "relative deprivation" (Truesdale and Jencks, 2016; Zhao et al., 2021), inequality could affect mental health due to absolute deprivation. Schilbach et al. (2016) argue that poverty imposes cognitive challenges, as preoccupations with monetary concerns and other issues related to poverty can be distracting, affecting decision making and even potentially lowering the utility of consumption. Even beyond the context of poverty, lower real income can result in economic anxiety. For example, financial and employment insecurity—the latter of which is a strong determinant of the labor share (Cauvel and Pacitti, 2022) —appear to have a negative effect on mental health (Ng et al., 2013). This likely explains why recessions are associated with higher levels of depression, self-harm, and suicide (Guerra and Eboreime, 2021).

Therefore, inequality may affect mental health through either a direct concavity effect of absolute income or a contextual effect operating through relative income. Status anxiety or other forms of economic anxiety can potentially lead to worse health outcomes through a variety of channels including increased substance abuse, depression, and even suicide. For example, substance abuse appears to be related to income (Baptiste-Roberts and Hossain, 2018), and depression is associated with both higher levels of income inequality and lower absolute income (Kahn et al., 2000). Moreover, macroeconomic trends such as automation and globalization appear to be associated with increased levels of substance abuse and suicide (Adda and Fawaz, 2020; O'Brien et al., 2022), although Pickett and Wilkinson (2015) note that evidence suggests an inverse relationship between suicide and personal inequality (perhaps because individuals in more equal societies are more likely to place blame for economic outcomes on themselves).

Stress

Stress is closely related to mental health, as it can be one physical manifestation resulting from the anxieties described above (Turner et al., 2020). As Zhao et al. (2021) note, the literature suggests an inverse relationship between status anxiety and many health outcomes. Many of these outcomes are potentially related to stress. For example, both economic positioning within countries and inequality across countries are associated with higher levels of inflammation, suggesting that inequality-related stress affects health (Layte et al., 2019). Subjective status also appears to be related to obesity (Goodman et al., 2003; Tang et al., 2016) and a number of other cardiovascular risk factors, including coronary artery disease, hypertension, and diabetes (Tang et al., 2016). Similarly, automation and globalization have been linked to higher levels of cardiovascular mortality (Adda and Fawaz, 2020; O'Brien et al., 2022). In simulations of a virtual society with differing conditions regarding income and inequality, Ryan et al. (2021) find that the distribution of income and participants' place within it can impact heart rates when making purchasing decisions (under some conditions, but not all). This physiological manifestation of stress is a strong predictor for reduced life expectancy (Jensen, 2019) and many of the diseases mentioned in the paragraph above.

Crime

Crime is one of the earliest channels linking inequality and mortality in the literature (Pickett and Wilkinson, 2015). Although inequality has been linked to rates of both violent and nonviolent crime (Nilsson, 2004), it is the former that will most directly impact health. A number of studies suggest that there is strong relationship between violent crime and inequality. For example, Rowhani-Rahbar et al. (2019) find a strong association between the Gini coefficient and firearm homicides, and Fajnzylber et al. (2002) find that a significant relationship between inequality and violent crime more generally. Similarly, O'Brien et al. (2022) find that automation—one of the factors that has been linked to the falling labor share—is associated with higher levels of homicide.

Childhood Adversity

A closely related issue is childhood adversity. Although it is not widely discussed in the life expectancy literature, other research suggests that adverse childhood experiences are related to both income inequality and mental and physical health outcomes. For example, adverse childhood experiences have been linked to cardiovascular morbidity and mortality (Cohen et al., 2010), Chronic Obstructive Pulmonary Disease (Anda et al., 2008), and physchological health issues such as major depressive disorders and substance-abuse disorders (Turner and Lloyd, 1995). Children living in poorer neighborhoods are more likely to experience some adverse outcomes. For example, Cohen et al. (2010) suggest that living in a higher-income neighborhood is incrementally and inversely related to the threats of crime, violence, and the chance of being the victim of physical violence or assault. Halfon et al. (2017) argues that reducing inequality or mitigating its effects is necessary in order to reduce the level of childhood adversity.

Healthcare Utilization

Unsurprisingly, consumption of healthcare appears to improve health. For example, healthcare expenditures are found to be a major determinant of life expectancy (Benos et al., 2019). However, as a normal good, consumption of healthcare is directly related to income (Chalise, 2020). A falling labor share implies relatively less income in the hands of the majority of the population, potentially leading to lower utilization of healthcare overall. Adda and Fawaz (2020) argue that decreased healthcare utilization is a major reason why globalization appears to contribute to worse health, as affected individuals are more likely to delay care and develop more serious conditions as a result. They also note that income is not the only factor that can contribute to lower healthcare utilization, as loss of access to employer-provided health insurance appears to be important as well. In a theoretical model, Frankovic and Kuhn (2019) show that health disparities that widen with medical advancements can result from differences in healthcare utilization for different income groups. They also show that differences in healthcare quality for different income groups can be important. It is therefore

possible that the structural macroeconomic changes driving the decline in the labor share can affect both the quantity and quality of healthcare through changes in both income and access to health insurance, although we are not able to differentiate between these effects.

Frankovic and Kuhn (2019) also highlight important dynamics related to Baumol's (1967) cost disease and the cost of healthcare, noting that productivity improvements in capital-intensive sectors are likely to both increase the costs of services like healthcare and increase inequality, potentially widening disparities in healthcare access. Although these dynamics could generate a relationship between health outcomes and any measure of inequality, they are particularly salient in the case of the labor share, which is mechanically related to labor productivity. As equation (1)—taken from Cauvel (2022)— shows, the labor share varies inversely with labor productivity, holding all else constant:

$$labor\ share = \frac{labor\ compensation}{output} = \frac{labor\ compensation/hours}{output/hours} = \frac{real\ hourly\ wage\ rate}{labor\ productivity} \tag{1}$$

A declining labor share is, therefore, indicative of wage growth that fails to keep pace with productivity growth. Given that productivity growth throughout the economy is likely to raise healthcare costs via Baumol's cost disease, workers may find it more difficult to afford healthcare when the labor share is falling.[3]

Education

Dynamics related to Baumol's cost disease could also impact another service that is important for health—education. As with healthcare, workers and their families may purchase less education as the labor share declines if the cost of education (which theory suggests will rise with productivity growth) outpaces wage growth. It is also possible that public education may decline in quality if government funding does not fully keep pace with the rising

[3]Whether medical inflation exceeds wage growth for a given change in the labor share would depend on the responsiveness of health care costs to productivity growth.

costs. Although the role of Baumol's cost disease is rarely discussed in this context, some previous studies have noted the possibility that education is a channel that links inequality and health. For example, (Pickett and Wilkinson, 2015) note this as a reason to avoid using education as a control variable.

Separate literatures have found education to be linked to both inequality and health. Income is a strong determinant of educational attainment (Plug and Vijverberg, 2005), and inequality itself has been found to negatively impact both educational attainment (Esposito and Villaseñor, 2018) and educational achievement (Workman, 2022). Moreover, education and mortality appear to be inversely related (Cutler et al., 2011; Benos et al., 2019; Leive and Ruhm, 2021). Thus, inequality could negatively affect health in the long run by hampering education.

A related argument focuses on the diffusion of health-related innovations. As Truesdale and Jencks (2016) explain, richer and more educated individuals may be quicker to take advantage of improvements in health knowledge and technology—leading to better health for these groups—and diffusion to individuals with lower income or education may be slower when inequality is high. Similarly, Frankovic and Kuhn (2019) use a theoretical model to show that medical progress can exacerbate health disparities across skill groups if there are skill-related differences in individuals' ability to effectively take advantage of state-of-the-art medical care. Although we are not able to directly test the diffusion-of-innovation channel, it provides a potential explanation for why education may impact health.

Economic Growth

Given the importance of income in determining health at both the individual and national levels, it is possible that the labor share impacts life expectancy via effects on aggregate income. Note that an effect of GDP on life expectancy differs from the concavity effect described above, wherein inequality may reduce life expectancy through individual income effects even if national income remains unchanged. As the literature has found evidence that

the labor share is positively associated with economic growth in the long run, a lower labor share could reduce life expectancy by reducing economic growth. GDP's relationships with both the labor share and life expectancy in the short to medium run are more complicated. As discussed above, there are debates in the literature about the effect of the labor share on economic activity in the short run. Similarly, there are disagreements about whether mortality varies procyclically or countercyclically over the course of the business cycle (see e.g. van den Berg et al., 2017). Because there are many complications in empirically untangling the complex interrelationships between the labor share and GDP (Stockhammer, 2017; Nogueira Rolim, 2019; Barrales-Ruiz et al., 2021; Cauvel, 2022), examining this channel directly is beyond the scope of this paper. However, further exploration of this channel remains an important area for future research.

3 Methodology

We explore the links between life expectancy and the labor share by estimating a series of ARDL models.[4] We begin by estimating the effect of the labor share on life expectancy directly. After finding evidence of a significant relationship, we then test whether this relationship is mediated by eight separate factors suggested by the literature: social capital, policy, mental health, stress, crime, childhood adversity, healthcare utilization, and education. This analysis includes two stages, as we first estimate the effects of the labor share on a proxy for each channel, and then estimate the effects of each proxy on life expectancy.

[4]Following Cauvel and Pacitti (2022), a standard representation of an ARDL model with a constant (a_0), k independent variables ($x_1, ..., x_k$) with lags $q_1, ..., q_k$, a dependent variable (y) with p lags, and error term ε is shown below, where t indexes time :

$$y_t = a_0 + \sum_{i=1}^{p} \beta_i y_{t-1} + \sum_{j=1}^{k} \sum_{l_j=0}^{q_j} \gamma_{j,l_j} x_{j,t-1} + \varepsilon_t$$

.

The ARDL model includes lags of various lengths for both the dependent and each independent variable. Using EViews, lag lengths for both the dependent and independent variables are selected using an algorithm that minimizes the Akaike Information Criterion (AIC), evaluating all possible combinations of lags up to a maximum of seven for each variable.[5] We primarily focus our analysis on long-run coefficients that summarize effects across all lags.[6] However, we note that these results are not necessarily "long-run" in an economic sense, as many capture effects across only a couple of years.

To ensure sound econometric results, we run a number of diagnostic tests for each specification including the Jarque-Bera test for normality of residuals, the Breusch-Pagan-Godfrey homoskedasticity test, the Breusch-Godfrey Lagrange multiplier to test for serial correlation up to two lags, and the Ramsey RESET general misspecification test (separately including one and two fitted terms). In specifications for which there is evidence of heteroskedasticity or serial correlation we use the Newey-West coefficient covariance matrix. The remaining diagnostic tests did not suggest any econometric issues for any of the reported specifications. However, we did include dummy variables in some models to improve diagnostic test results.[7]

[5]We experimented with higher maximum lag lengths, but these specifications were not estimable in EViews—likely due to the high number of parameters relative to the sample size. We deviated from this lag selection approach to improve our econometric specification in two cases. When regressing life expectancy on our policy variable, we reduced the maximum lag length to six because the RESET test suggested issues with the first specification. When regressing our policy variable on the labor share, we reduced the number of lags of GDP from two to one because the residuals were not normal in the initial regression. In both cases we did not consider the estimation results when making these changes, only diagnostic test results. In both cases the key results in the initial specifications are qualitatively similar to those that we present below, but we did not use them because they are not econometrically sound.

[6]A long-run coefficient is calculated as the ratio of the sum of the coefficients on all lags of a given independent variable to one minus the sum of all coefficients on lags of the dependent variable.

[7]In specifications with life expectancy as the dependent variable, we include dummy variables for the years 1968 and 2020 because pandemics in these years (the avian flu and COVID-19 respectively) lowered life expectancy for reasons unrelated to the social and economic dynamics that we are exploring, and including these dummy variables improves diagnostic test results. The 2020 dummy variable was also included in the regressions with health care spending and deaths due to cardiovascular disease, as both were impacted by the COVID-19 pandemic for non-economic reasons and accounting for a large residual in this year improves the results of the RESET test. Similarly, a dummy variable for the year 1984 is added to the specification with trust as the dependent variable to account for a large residual and address non-normal residuals. A time trend is included in this specification as well, as trust is the only dependent variable that exhibits a

Noting that many of the factors we are examining may be interrelated, we aim to avoid underestimates that could result from ruling out the effects of potential mediating variables (Pickett and Wilkinson, 2015). As such, we include very few control variables in our models. The one exception is the growth rate of GDP, which we initially include in every model to control for business cycle effects. However, in some cases we present models that exclude this variable because its inclusion does not affect the central results and we either prefer the more parsimonious model or its inclusion results in a poor econometric specification according to the diagnostic tests discussed above. In each case, the signs and significance of results relating our variables of interest in specifications excluding GDP are robust to its inclusion.

4 Data

We use annual U.S. data ranging from 1947-2021, although the sample period varies in each specification due to data limitations. Our two primary variables are the labor share, measured using the U.S. Bureau of Labor Statistics' (BLS) business sector labor share index, and life expectancy at birth, data for which comes from the World Bank. Data for real GDP, which is used as a control variable in some specifications, is compiled by the U.S. Bureau of Economic Analysis (BEA). All three series were downloaded from the St. Louis Federal Reserve Bank's FRED database.

Because many of the potential mediating factors we are interested in are difficult to observe directly, we proxy for them with measures that are closely related and have more readily available data. For several of these proxies we utilize data on cause-specific death rates from the World Health Organization (WHO). These series are age-standardized and show the number of deaths per 100,000 members of the population. We proxy for mental

trend. Finally, we include longer dummy variables in two cases to address apparent structural breaks, which we identified by visual inspection after the RESET test suggested misspecification in our initial models.

health with the suicide rate, crime with the rate of violent deaths, and stress with deaths due to cardiovascular diseases (CVD).

Our measure for childhood adversity is the U.S. Census Bureau's rate of childhood poverty. To proxy for social capital, we use a measure of general social trust based on data from the General Social Survey (GSS). The measure captures the percentage of respondents who say that "most people can be trusted" as opposed to "you can't be too careful in dealing with people" or that it depends.[8] For policy, we measure government spending on social safety nets and public goods as the amount of real non-defense government consumption expenditures as a share of GDP based on BEA data. We proxy for healthcare utilization with the amount of per capita personal consumption expenditures on healthcare, using data from the BEA obtained via FRED. This series is deflated by the BLS Consumer Price Index. Finally, to proxy for education we utilize Census Bureau data on college completion rates, measured as the percentage of people over the age of 24 who had completed four years or more of college.

Most series are found to have unit roots and are therefore transformed into growth rates by taking the log difference. The three exceptions are trust, childhood poverty, and the labor share.[9]

[8] We exclude respondents who say they do not know or give no answer and those for whom the question is not applicable. Because the survey is not administered every year, there are some years with no data. We interpolate these observations by taking the mean of the nearest data points before and after the missing year.

[9] Augmented Dickey-Fuller (ADF) tests (with lag length selected using the Schwarz Information Criterion) failed to reject the null hypothesis of a unit root for all series except trust—wherein it was rejected at the 1% level—and childhood poverty—wherein it was rejected at the 10% level. Although this test suggests that the labor share has a unit root, a modified ADF test allowing for a structural break in 2007, as suggested by Cauvel and Pacitti (2022), rejects the null hypothesis of a unit root.

5 Results

We first estimate the relationship between the labor share and the growth rate of life expectancy. These results are presented in Table 1. We find evidence of a positive and statistically significant relationship. The estimates suggest that a one-point increase in the labor share index is associated with an increase of roughly 0.02 percentage points in the growth rate of life expectancy. Although the point estimate itself is small, we view it as economically meaningful. A one-standard deviation increase in the labor share index (an increase of roughly 4.37 units) is associated with an impact equivalent to roughly one-fifth of a standard deviation in the growth of life expectancy. To put this estimate into further context, the 13.5-point decline in the labor share index between 2001-2013 is associated with a 0.27-percentage point decline in the growth rate of life expectancy. This change is roughly 1.6 times the magnitude of the average annual growth rate of life expectancy throughout our sample period (0.17 percentage points).

We include GDP growth in these estimates to prevent capturing spurious effects that could arise if both the labor share and life expectancy are impacted by macroeconomic outcomes. The coefficient on GDP growth is negative and statistically significant, suggesting that life expectancy increases during recessions and declines during booms. This is a result that has been found elsewhere in the literature and could reflect increased stress or increased risk taking during booms (van den Berg et al., 2017). We note that our results are likely capturing at most medium-run effects due to methodological choices such as using annual data and transforming most series with log differences. As such, these estimates likely do not suggest a negative long-run effect of GDP growth on life expectancy.

Our remaining specifications separately estimate the relationship between each potential mediating variable and either life expectancy or the labor share. Results for these estimates are summarized in Tables 2 and 3, respectively. These summary tables present

Table 1: Life Expectancy–Labor Share Regression

Dependent Variable: $\Delta\ ln\ Life\ Expectancy_t$	
Constant	-0.017*
	(0.009)
$\Delta\ ln\ Life\ Expectancy_{t-1}$	-0.043
	(0.114)
$\Delta\ ln\ Life\ Expectancy_{t-2}$	0.129
	(0.114)
$Labor\ Share_t$	0.0002**
	(0.000)
$\Delta\ ln\ GDP_t$	-0.040**
	(0.018)
$Dummy\ 1968_t$	-0.010***
	(0.003)
$Dummy\ 2020_t$	-0.023***
	(0.003)
Long-run coefficients:	
$Labor\ Share$	0.0002**
	(0.000)
$\Delta\ ln\ GDP$	-0.043*
	(0.022)
$Dummy\ 1968$	-0.011***
	(0.004)
$Dummy\ 2020$	-0.026***
	(0.006)
Sample Period	1963-2020
Standard Errors	Ordinary
R^2	0.655
Adjusted R^2	0.615
AIC	-9.003
Jarque-Bera	0.318
Breush-Godfrey	0.336
Breusch-Pagan-Godfrey	0.202
RESET (1)	0.543
RESET (2)	0.786

Notes: The parenthetical numbers are standard errors. Stars represent significance levels (*** 1%, ** 5%, * 10%) based on t-statistics. The numbers presented for diagnostic tests are p-values for F-statistics. All numbers are rounded to the nearest thousandth, with the exception of coefficients less than 0.001, which are rounded to the nearest ten-thousandth.

only the long-run coefficients for each variable. Short-run coefficients for each regression are presented in Tables A1 and A2, which are located in the Appendix.

As shown in Table 2, most of the proxies that we include are found to have statistically significant associations with the growth rate of life expectancy. The only two exceptions are deaths due to suicide and violence. Although mental health and violence are certainly important aspects of public health, they do not appear to be significant determinants of life expectancy based on the measures we used. All of the other proxies are statistically significant and have the expected signs. CVD deaths and childhood poverty appear to be inversely related to life expectancy, suggesting that there are pathways through which stress and childhood adversity can reduce longevity across society.[10] Government consumption spending relative to GDP, trust, per capita healthcare spending,[11] and college completion are all positively associated with life expectancy, suggesting that policy, social capital, healthcare utilization, and education are also important factors impacting health. Health appears to be better when individuals spend more on healthcare, when the government spends more on social programs, when people are better educated, and when the social fabric is stronger.

[10]Because the null hypothesis of a unit root was only rejected at the 10% significance level, we ran a robustness check using the growth rate of childhood poverty instead of the level. In this specification, the long-run coefficient was significant at the 10% level but had a theoretically unexpected positive sign. When regressing the growth rate of childhood poverty on the labor share, we also find a theoretically implausible—but statistically significant—positive coefficient.

[11]Unreported results show that healthcare spending becomes insignificant when using a measure that is normalized by GDP rather than the size of the population. However, we believe that measuring this variable on a per capita basis is the more theoretically appropriate way of capturing the average citizen's healthcare utilization.

Table 2: Long-run Coefficients for Life Expectancy Regressions

Dependent Variable: Δ ln Life Expectancy

Key Variable	(1) Trust	(2) Policy	(3) Suicide	(4) CVD	(5) Violence	(6) Childhood	(7) Healthcare	(8) College
Key variable	0.0002*	0.078**	-0.027	-0.108***	-0.005	-0.0005***	0.053***	0.098***
	(0.000)	(0.031)	(0.027)	(0.018)	(0.004)	(0.000)	(0.016)	(0.025)
GDP	-0.026	0.014	-0.040			0.0005		
	(0.024)	(0.032)	(0.028)			(0.017)		
1968 Dummy		-0.012***	-0.012***	-0.007***	-0.010***	-0.009***	-0.012***	-0.011***
		(0.003)	(0.004)	(0.001)	(0.002)	(0.002)	(0.003)	(0.002)
2020 Dummy		-0.022***	-0.028***	-0.013***	-0.018***	-0.019***	-0.016***	-0.019***
		(0.004)	(0.009)	(0.002)	(0.003)	(0.003)	(0.003)	(0.003)
2000-2020 Dummy					-0.001			
					(0.001)			
Sample Period	1977-2018	1962-2020	1963-2020	1962-2020	1962-2020	1962-2020	1966-2020	1967-2020
Standard Errors	O	O	NW	NW	NW	O	O	O
R^2	0.277	0.738	0.653	0.827	0.608	0.728	0.751	0.717
Adjusted R^2	0.073	0.662	0.605	0.814	0.571	0.691	0.694	0.681
AIC	-9.221	-9.046	-8.962	-9.766	-8.913	-9.212	-9.142	-9.142
Jarque-Bera	0.747	0.940	0.823	0.621	0.711	0.480	0.918	0.770
Breush-Godfrey	0.463	0.595	0.022	0.005	0.038	0.555	0.386	0.457
Breusch-Pagan-Godfrey	0.684	0.682	0.515	0.009	0.337	0.104	0.827	0.668
RESET (1)	0.821	0.762	0.155	0.510	0.945	0.946	0.215	0.273
RESET (2)	0.144	0.101	0.268	0.630	0.831	0.530	0.410	0.491

Notes: The notes below Table 1 are also applicable here. Standard errors used in regression estimation are either ordinary (O) or Newey-West (NW). The key variables for each column are: (1) *Trust*, (2) Δ *ln Non − defense Government Consumption Spending / GDP*, (3) Δ *ln Suicide Rate*, (4) Δ *ln CVD Deaths*, (5) Δ *ln Rate of Violent Death*, (6) Δ *ln Childhood Poverty Rate*, (7) Δ *ln Healthcare Expenditures per Capita*, and (8) Δ *ln College Completion Rate*. Short-run coefficients for these regressions are presented in Table A1.

Table 3: Long-run Coefficients for Labor Share Regressions

Dependent Variable: Varies by Specification

Dependent Variable	(1) Trust	(2) Policy	(3) Suicide	(4) CVD	(5) Violence	(6) Childhood	(7) Healthcare	(8) College
Labor Share	-0.205	0.002***	-0.0004	-0.0004	-0.0001	0.126	0.002*	0.001**
	(0.153)	(0.001)	(0.001)	(0.001)	(0.002)	(0.142)	(0.001)	(0.001)
GDP	49.464	-0.574***	-0.242			1.392	0.695	
	(50.418)	(0.193)	(0.299)			(71.107)	(0.467)	
1984 Dummy	11.758***							
	(2.910)							
2020 Dummy				0.136***			-0.127***	
				(0.035)			(0.038)	
1980 - 2021 Dummy						2.647***		
						(0.665)		
Sample Period	1974-2018	1952-2021	1952-2020	1954-2020	1952-2020	1966-2021	1961-2021	1966-2021
Standard Errors	O	O	O	NW	O	O	O	NW
R^2	0.930	0.696	0.209	0.292	0.195	0.975	0.667	0.210
Adjusted R^2	0.907	0.662	0.118	0.234	0.158	0.964	0.615	0.180
AIC	3.573	-5.327	-4.380	-4.957	-2.650	1.355	-5.016	-5.730
Jarque-Bera	0.277	0.974	0.496	0.404	0.245	0.874	0.651	0.570
Breush-Godfrey	0.613	0.358	0.893	0.052	0.559	0.185	0.622	0.653
Breusch-Pagan-Godfrey	0.113	0.183	0.720	0.564	0.316	0.555	0.912	0.036
RESET (1)	0.158	0.441	0.650	0.746	0.459	0.869	0.143	0.500
RESET (2)	0.113	0.621	0.741	0.948	0.243	0.610	0.339	0.766

Notes: The notes below Table 2 are also applicable here. Short-run coefficients for these regressions are presented in Table A2.

Results relating these proxies to the labor share are shown in Table 3. Fewer variables are found to be significantly related to the labor share. As with the life expectancy, deaths by suicide and violence are not found to be significantly related to the labor share. The labor share coefficient is similarly insignificant in the regressions with trust, CVD deaths, and childhood poverty as the dependent variable. Our results, therefore, are not supportive of these factors as primary channels through which the labor share impacts life expectancy, even though we find that they are related to the latter. However, for a variety of reasons we caution readers against the interpretation that these results rule out these factors as potential causal channels through which inequality may impact health. First, it is possible that the labor share impacts these variables only in the long run, and our estimates likely capture at most medium-run effects. Second, given that many of these concepts are difficult to measure, results may be sensitive to the choice of proxy variable. Finally, our results focus only on one form of inequality—the functional distribution of income. It is likely that factors such as stress and social capital are influenced more heavily by the personal distribution of income than the labor share. Exploring these potential channels more fully remains an important area for future research.

We do identify three factors that are significantly related to both life expectancy and the labor share—healthcare spending, educational attainment, and government consumption spending.[12] Increases in the labor share are associated with increases in all three factors, which are in turn associated with increases in life expectancy. Our results, therefore, suggest that healthcare utilization, education, and policy are three channels though which the labor share may affect health.

One striking commonality between two of these channels is that both healthcare and education are services that have seen rapidly increasing costs in recent decades. As a result,

[12]The significance of the policy variable is sensitive to measurement. As shown here, the labor share is significantly related to government consumption spending as a share of GDP. However, when measuring this spending on a per capita basis instead, this result becomes insignificant.

college completion rates and healthcare spending have both been growing at slower rates. Although further research is needed to explore these dynamics in more depth, we expect that the strong links between the labor share and both healthcare spending and education reflect Baumol's cost disease. Services like healthcare and education typically experience slower productivity growth than goods-producing industries. As productivity rises throughout the economy, the theory behind Baumol's cost disease suggests that costs for services will rise. Meanwhile, the declining labor share implies that productivity is growing at a faster rate than real wages. Our results suggest that these two dynamics combine to have a negative impact on health. Productivity improvements make healthcare and education more expensive, while slow wage growth relative to productivity growth makes it more difficult for workers to afford these services. Lower utilization of these services, or at least slower growth in each, in turn impedes improvements in life expectancy.

Another similarity between healthcare and education is that both can be considered public goods. As such, governments often play an important role in subsidizing or directly providing these services. Therefore, to the extent that healthcare and education improve life expectancy, it is not surprising that government spending on social programs (many of which are directly related to these services) also improves health. The negative health impacts of a falling labor share could be mitigated by greater public spending on services that are important for health. However, our results suggest that the falling labor share is associated with lower public spending on social services, further contributing to declining growth in life expectancy. As we use a broad measure of government consumption spending, our results do not indicate which types of spending are impacted by the labor share and which are important for health. In addition to healthcare and education, it is possible that our results are capturing the importance of other public goods, such as parks, sanitation, or environmental protection for health. Thorough investigation of this issue requires further research.

6 Conclusion

We examine the relationship between the labor share and life expectancy in the U.S. We find a positive and significant relationship between these two variables, suggesting that the declining labor share is one factor contributing to slowing improvements, and ultimately a decline, in life expectancy. We then explore several potential causal channels through which the labor share may affect health. Our results suggest the presence of three such causal channels—healthcare utilization, education, and policy—as we find significant relationships with both the labor share and life expectancy for our proxies in each of these areas.

Regarding other potential mediating variables, we find that social capital, stress, and childhood adversity have a strong effect on life expectancy, but that these variables are not significantly impacted by the labor share. We find no evidence that mental health or crime are related to either life expectancy or the labor share. However, we caution that this preliminary analysis should not be viewed as ruling out the presence of these potential causal channels between health and inequality.

These findings have important implications for policy. They suggest that the declining labor share of income is a health issue, and that policies that increase the labor share could increase life expectancy. Alternatively, greater public spending on social programs and policies that make healthcare and education more affordable for workers could potentially limit the negative health effects of a declining labor share. More research is needed to further explore these important dynamics.

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Appendix: Short-run Coefficients

Table A1: Short-run Coefficients for Life Expectancy Regressions

Dependent Variable: Δ *ln Life Expectancy*$_t$

Key Variable	(1) Trust	(2) Policy	(3) Suicide	(4) CVD	(5) Violence	(6) Childhood	(7) Healthcare	(8) College
Constant	-0.004	0.002*	0.003***	-0.0004	0.003***	0.012***	0.0002	-0.0001
	(0.003)	(0.001)	(0.001)	(0.000)	(0.001)	(0.002)	(0.001)	(0.001)
Δ *ln Life Expectancy*$_{t-1}$	-0.052	-0.012	0.016	-0.001	-0.084	-0.212*	-0.153	-0.150
	(0.159)	(0.119)	(0.164)	(0.109)	(0.146)	(0.110)	(0.114)	(0.119)
Δ *ln Life Expectancy*$_{t-2}$			0.129					
			(0.117)					
Key Variable$_t$	0.0001	0.054*	0.006	-0.108***	-0.006	0.0005	0.019	0.017
	(0.000)	(0.028)	(0.015)	(0.014)	(0.005)	(0.001)	(0.018)	(0.026)
Key Variable$_{t-1}$	-0.0002	-0.098***	-0.030**			-0.0006	-0.032	0.026
	(0.000)	(0.034)	(0.012)			(0.001)	(0.019)	(0.026)
Key Variable$_{t-2}$	-0.0000	0.100***				-0.0006	-0.001	0.069***
	(0.000)	(0.030)				(0.000)	(0.017)	(0.025)
Key Variable$_{t-3}$	0.0001	-0.031					0.009	
	(0.000)	(0.026)					(0.017)	
Key Variable$_{t-4}$	-0.0003*	0.053**					0.003	
	(0.000)	(0.021)					(0.018)	
Key Variable$_{t-5}$	0.0004**						0.008	
	(0.000)						(0.018)	
Key Variable$_{t-6}$							0.056***	
							(0.017)	
Δ *ln GDP*$_t$	-0.002	0.028	-0.034*			0.0006		
	(0.021)	(0.035)	(0.019)			(0.021)		

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Table A1 – *Continued from previous page*

Key Variable	(1) Trust	(2) Policy	(3) Suicide	(4) CVD	(5) Violence	(6) Childhood	(7) Healthcare	(8) College
$\Delta\ ln\ GDP_{t-1}$	-0.025	-0.142***						
	(0.021)	(0.043)						
$\Delta\ ln\ GDP_{t-2}$		0.109***						
		(0.036)						
$\Delta\ ln\ GDP_{t-3}$		-0.008						
		(0.031)						
$\Delta\ ln\ GDP_{t-4}$		0.026						
		(0.027)						
$Dummy\ 1968_t$		-0.012***	-0.010***	-0.007***	-0.011***	-0.011***	-0.013***	-0.012***
		(0.003)	(0.001)	(0.000)	(0.001)	(0.002)	(0.003)	(0.002)
$Dummy\ 2020_t$		-0.022***	-0.024***	-0.013***	-0.019***	-0.023***	-0.019***	-0.022***
		(0.003)	(0.002)	(0.001)	(0.001)	(0.003)	(0.003)	(0.002)
$Dummy\ 2000-2020_t$					-0.001			
					(0.001)			

Notes: For long-run coefficients, diagnostic test results, and additional information on these regressions, see Table 2.

Table A2: Short-run Coefficients for Labor Share Regressions

	Dependent Variable: Varies by Specification							
Dependent Variable	(1) Trust	(2) Policy	(3) Suicide	(4) CVD	(5) Violence	(6) Childhood	(7) Healthcare	(8) College
Constant	62.117***	-0.172***	0.053	0.011	0.012	-0.229	-0.138*	-0.060*
	(15.026)	(0.057)	(0.085)	(0.60)	(0.185)	(3.262)	(0.082)	(0.034)
Trend	-0.260***							
	(0.052)							
$Dependendent\ Variable_{t-1}$	0.422***	0.428***	-0.041	-0.002	0.285**	0.951***	0.343***	0.363**
	(0.120)	(0.115)	(0.119)	(0.127)	(0.125)	(0.147)	(0.107)	(0.147)
$Dependendent\ Variable_{t-2}$	-0.285**	-0.368***		0.274***		-0.104		
	(0.132)	(0.076)		(0.083)		(0.215)		
$Dependendent\ Variable_{t-3}$		0.082		0.262**		0.134		
		(0.070)		(0.128)		(0.210)		
$Dependendent\ Variable_{t-4}$		-0.007				-0.037		
		(0.068)				(0.203)		
$Dependendent\ Variable_{t-5}$						-0.045		
						(0.200)		
$Dependendent\ Variable_{t-6}$						0.184		
						(0.200)		
$Dependendent\ Variable_{t-7}$						-0.324**		
						(0.136)		
$\Delta\ ln\ Labor\ Share_t$	0.159	0.002***	-0.005*	-0.0002	0.016***	0.030	0.001*	0.0007**
	(0.233)	(0.001)	(0.003)	(0.001)	(0.005)	(0.031)	(0.001)	(0.000)
$\Delta\ ln\ Labor\ Share_{t-1}$	-0.335*		0.005*		-0.016***			
	(0.186)		(0.003)		(0.005)			
$\Delta\ ln\ GDP_t$	-7.147	-0.803***	-0.339**			-23.296***	0.397***	
	(11.965)	(0.092)	(0.160)			(3.342)	(0.128)	
$\Delta\ ln\ GDP_{t-1}$	9.953	0.306**	0.121			-10.326*	-0.519***	
	(17.226)	(0.134)	(0.199)			(5.228)	(0.138)	

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Table A2 – *Continued from previous page*

Dependent Variable	(1) Trust	(2) Policy	(3) Suicide	(4) CVD	(5) Violence	(6) Childhood	(7) Healthcare	(8) College
$\Delta\ ln\ GDP_{t-2}$	-15.293		-0.315**			-2.210	0.298**	
	(14.861)		(0.153)			(5.632)	(0.147)	
$\Delta\ ln\ GDP_{t-3}$	35.378**		0.281*			-0.771	0.077	
	(13.891)		(0.151)			(5.568)	(0.129)	
$\Delta\ ln\ GDP_{t-4}$	19.786					12.733**	0.204	
	(17.578)					(5.257)	(0.134)	
$\Delta\ ln\ GDP_{t-5}$						2.250		
						(5.509)		
$\Delta\ ln\ GDP_{t-6}$						13.087**		
						(5.302)		
$\Delta\ ln\ GDP_{t-7}$						8.867**		
						(4.109)		
$Dummy\ 1984_t$	10.144***							
	(1.722)							
$Dummy\ 2020_t$				0.064***			-0.084***	
				(0.005)			(0.020)	
$Dummy\ 1980-2021_t$						0.636***		
						(0.216)		

Notes: For long-run coefficients, diagnostic test results, and additional information on these regressions, see Table 3.