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Comisión

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Abstract

Mortality rates for people in their prime (ages 25 to 54) have been increasing, a trend unseen in recent history and exclusive to the US. Most of this growth in mortality can be accounted by deaths from suicide, drug and alcohol poisonings, as well as alcohol-related liver disease, a set also known as deaths of despair. There is correlational evidence that poor labor market outcomes may be driving this increase in mortality. To move beyond correlation, we use an instrumental variable (IV)/reduced-form strategy: we estimate the impact of import penetration on US mortality rates, as an indirect approach to better understand how labor market outcomes affect mortality. Our instrument, import penetration, measures local labor market exposure to Chinese exports, and it constitutes an exogenous demand shock to local labor markets. We present the following results: growth of import exposure between 1991 and 2016 is associated with rising mortality rates in the US; the effects are strongest for white non-hispanics, and weaker for minorities. Suicide and addiction-related deaths comprise only a tiny fraction of mortality overall (less than 4%) but represent more than half of the impact that import penetration has on mortality for white non-hispanics. Whilst minorities also suffer economically from the labor market shock, their suicide and addiction-related mortality rates are unaffected. We explain these differences across race/ethnicity by exploring the cumulative disadvantage hypothesis wherein people are not driven to suicide and addiction by their struggles in the labor market *per se*, but rather by the income downward mobility resulting from labor market shocks. Import penetration also has a significant impact on health insurance coverage and opioid prescription rates, further evidence of its effect on mortality.

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Contents

1 Introduction

Mortality rates for people in their prime working years (ages 25 to 54) are increasing, a trend unseen since the second world war (Deaton, 2013), and exclusive to the United States (Fig. 1-2). While this trend is particularly pronounced for white non-hispanics, it holds true for the overall population (Fig. 3). Most of this growth in mortality is accounted for by deaths from suicide, drug and alcohol poisonings, as well as alcohol liver disease, a set also known as deaths of despair (Fig. 4-6).

This increase in mortality rates is widely recognized (Case and Deaton, 2015), but poorly understood. It coincides with a period of deteriorating labor market outcomes for people in their prime. During the "great employment sag of the 2000s" (Acemoglu et al., 2016), an estimated 4 million jobs in manufacturing were destroyed (Charles et al., 2016) and the percentage of prime age people without a job increased sharply (Fig. 7-8). In addition, a long-standing literature in health and economics has established a strong correlation between individuals' labor market outcomes and their mortality rates (Chetty et al., 2016). Could the deteriorating labor market outcomes for people in their prime be behind the growth in mortality?

This paper finds evidence that labor market outcomes are important in determining mortality rates. Using an exogenous demand shock to local labor markets, we show that when employment rates[1] and incomes go down, all-cause mortality rates go up and intermediate health outcomes deteriorate. We also show that these labor market effects on mortality are mostly accounted for by their impact on deaths of despair, the main component of the aforementioned increase in mortality. Moreover, we provide evidence that the effect on deaths of despair is limited to white non-hispanics. Whilst minorities also suffer economically from the labor market shocks, their suicide and addiction-related mortality rates are unaffected. Our results suggest that this difference may be due to varying labor market expectations across races/ethnicities.

The main challenge in establishing the impact of labor market outcomes on health and mortality is telling cause from effect: are labor market outcomes determining health outcomes or is it the other way around? An issue compounded by the fact that we cannot use an experimental approach to resolve the reverse causality problem.[2] Therefore, to study the exogenous impact of labor market outcomes on morbidity and mortality

[1]Employment to working age population ratio.

[2]Labor market effects on mortality are tiny (in the order of 1 to 100,000). Such that, to have an acceptable statistical power, an experiment would have to either create (impractical) or destroy (impractical and unethical) millions of jobs.

we use an instrumental variable (IV) approach.[3]

To isolate the impact of labor market outcomes on mortality, we take advantage of a rapid and unexpected rise in manufacturing productivity of Chinese firms. Between 1990 and 2016, the share of world manufacturing exports originating in China increased from 2% to 20% (UNCTAD, 2018). The country's share in US manufacturing imports has shown an equally sharp rise from 4% in 1991 to 26% in 2016. As the US has become increasingly reliant of goods manufactured in low income countries, some industries and jobs have all but disappeared; while others have remained unscathed by foreign competition.[4]

Using initial industry composition across US local labor markets and the level of Chinese exports across industries and time we can build an import penetration variable measuring local labor market exposure to Chinese imports over time. Autor et al. (2013) estimate that import penetration has a significant negative impact on employment rates and wages, with effects varying across industries and local labor markets. In addition, their estimates are arguably exogenous: because a growth of imports from China to the US could be related to endogenous demand factors, they use the growth of Chinese import towards other developed countries as an exogenous instrument. Following this same logic, we construct an import penetration variable using initial industry composition and exports from china to 8 developed nations other than the US.

This paper estimates the instrumental variable (IV)/reduced-form impact of import penetration on mortality rates and intermediate health variables. In doing so, we provide evidence of the effects of labor market outcomes on mortality.

Our first set of results shows that a) import penetration has a significant and positive impact on mortality of people in their prime working years, in the 1991 to 2016 period. b) suicide and addiction-related deaths represent more than half of the impact that labor market changes have on mortality. c) the effect of import penetration on deaths by other causes is more elusive. The second point is crucial, deaths of despair account for less than 4% of overall mortality in the US population from 1991 to 2016, yet they account for more than half of the effect that import penetration has on mortality. The positive effect of import penetration on all-cause mortality (point a) is consistent with two other studies: Pierce and Schott (2016) show that a liberalization of US trade policy

[3] Similar to what's called natural experiment, observational study, or cohort study in other fields.

[4] It is not only that effect sizes vary across industries, timing also varies: China's import penetration in apparel rises quickly in the 1990s, but import penetration in transistor manufacturing does not rise until the 2000s.

in 2001 had a positive impact on mortality; Donnally (2017) shows that the growth of import penetration between 2000 and 2012 also had a positive impact on mortality.[5] The specific effects of import penetration on deaths of despair and other causes are our own contribution (points b and c).

Our second set of results shows that import penetration has a significant impact on intermediate health variables. We find evidence that import penetration has a negative impact on health insurance coverage (percentage of people who have a valid health insurance plan). A result suggesting that import penetration has a positive impact on all-cause mortality, in part, because it reduces access to health care. We also find evidence that import penetration has a positive impact on legal opioid drug consumption. Which suggests that import penetration has a positive impact on deaths of despair, in part, because it leads to an increase in drug consumption.

In our third set of results, we show that import penetration has a negative impact on incomes of male children of low-income parents, for all races. But, import penetration only has a negative impact on the incomes of children of high-income parents for white non-hispanics. The incomes of minority children with high-income parents are unaffected by import penetration. This evidence is consistent with the cumulative disadvantage hypothesis (Case and Deaton, 2015, 2017); this theory claims that people are not driven to suicide and addiction by their struggles in the labor market *per se*, but by facing worse labor market opportunities than their parents. Behind this, is the idea that people's expectations from the labor market are anchored on their parent's experience, i.e. parents represent a reference group. For example, if a parent has a well-paying job in manufacturing, his children may expect to have that same kind of job. If correct, this theory could explain why mortality effects are stronger for white non-hispanics than for minorities: if labor market experiences from past generations differ across races (Fig. 9), so should vary their labor market expectations and the impact of import penetration on mortality.

Our main contribution is to elucidate how and why labor market outcomes have an impact on mortality. Whilst Donnally (2017) provided evidence on the existence of such an impact, we show that labor market outcomes influence both people's access to health care (evidence on health insurance coverage), as well as their behavior (evidence on increased opioid consumption). We find evidence that labor market outcomes mostly

[5] On a different perspective, Autor et al. (2018) look at the impact of changes in import penetration on cross-sectional gender gap in mortality. A fuller review of this work can be found on the background section.

matter because they influence people's mental health. When labor market outcomes do not meet someone's expectations they may well turn to suicide and addiction. This point underlines how economic successes or failures shape mental health. It speaks directly to previous work on the correlation between mental health and socioeconomic status (Wilkinson and Pickett, 2018), it also underlines the importance of work as more than just an income source.

The remainder of the paper is organized as follows: Section 2 sets up the background by reviewing some of the abundant correlational evidence suggesting that import penetration and mortality are related. Section 3 describes the 8 datasets we had to combine to write this paper, and how they fit together. Section 4 discusses identification: how we solve reverse causality and endogeneity problems, and estimate the effect of import penetration on mortality rates. Section 5 estimates the impact of import penetration on mortality rates. Section 6 studies how the impacts of import penetration vary according to sex, race/ethnicity, and parental income.

2 Background

Our main goal in this section is to review existing data and research on the links between mortality, labor market outcomes, and import penetration.[6]

2.1 Mortality trends in the population

In this subsection we want to make 3 points. First, the growing mortality rates for people in their prime is both exceptional and large enough that it deserves our attention. Second, a staggering growth in deaths from suicide and addiction (deaths of despair) mostly explains this increase in overall mortality. Third, we cannot tell apart the different causes of death that make up the "deaths of despair" set, in terms of intent; therefore, it makes sense to look at the set as a whole, as opposed to looking at them separately.

The broad rise in overall mortality for non-hispanic whites aged 25 to 54 is large,

[6] We find ourselves on a cross-section between several literatures. Macroeconomists would perhaps focus on how our work relates to the winners and losers of international trade literature; health economists might see this paper as a study of labor market and health outcomes relate; labor economists would perhaps see our work as a study on the consequences of the decreasing labor market participation of men in their prime. We cannot provide a literature review that satisfies everyone, but we hope we try not to miss anything egregiously central.

unprecedented, and unparalleled by other countries (Fig. 1-2). This trend was first noticed by Case and Deaton (2015), who noticed that the mortality rates of people aged 45 to 54 were increasing. With the benefit of hindsight and more recent data we can go beyond Case and Deaton's work: first, overall mortality is not only increasing for non-hispanic whites aged 45 to 54, it is increasing for the whole 25 to 54 group. Second, and contrary to what was previously believed, non-hispanic whites are not the only group suffering from an increase in all-cause mortality rates: minorities aged 25 to 54 are also dying at increasing rates. For hispanics this shift in trend started in 2012, for non-hispanic blacks it started in 2014; and just like in the case of non-hispanic whites the growth is being pushed by the increase in deaths of despair (Fig. 2); without deaths of despair, there is no increase (Fig. 4). However, the mortality growth for minorities should be taken with a pinch of salt for two reasons: first, the trends for minorities are shorter: a 2 or 4-year trend is not the same as an 18 year trend. Second, because the magnitude of the increase is smaller for minorities. But, even with those two caveats, this is still a significant finding that goes against previous reports.

The numbers involved in the growth of deaths of despair are dazing. In 2016, 146,391 people died of suicide, drug or alcohol poisoning, and alcohol liver disease in the US[7]; up from 62,591 in 1998. But this sharp rise comes after a long period of decline: between 1979 and 1998 the Mortality rate from deaths of despair went from 25.6 deaths per 100,000 living to 22.7 deaths per 100,000 living. On average that constitutes a 0.2 point drop per year. But between 1998 and 2016 the rate then shot up to 45.3 deaths per 100,000 living (Fig. 6)[8]. As we saw in the introduction this trend is even more extreme for people between the ages of 25 and 54, with the overall mortality reaching almost 80 deaths per 100,000 living (Fig. 5).

In addition, this increase in mortality in deaths of despair comes against a backdrop of declining mortality from other causes. Fig. 10 shows how deaths from cancer, cardiovascular disease, and stroke are all going down. These are the main causes of death for people in their prime working years, and the decline of death from these causes is one of the great medical victories of the 20th century (Deaton, 2013). The fact that such progress is being made on other aspects of health but in deaths of despair things seem to be getting worse adds urgency to the matter.

[7]These are conservative estimates, they only consider people whose death can be directly attributed to suicide and addiction, they exclude people who die of car accidents while on drugs or alcohol; they also exclude those for whom struggles with mental health and self-harm result in other illnesses.

[8]WONDER-CDC data, author's calculations.

This issue is of the utmost importance. To give the reader some notion of proportion, between 2000 and 2016 if the mortality rate from deaths of despair had held constant at 2000 levels, 427,127 would have been averted. If the mortality rate had continued to decrease at 0.2 points a year, 511,764 people would not have died. This is comparable to the HIV/Aids epidemic, which killed about 675,000 people between 1980 and 2016 (CDC, 2016). Furthermore, there's good reason to believe that these deaths are only the tip of the proverbial iceberg. For every person who takes their own life (by poisoning or other means) many more are struggling with mental illness and addiction. These numbers are simply dazing and as social scientists we need to do a better job at understanding exactly what is going on, and why this is happening.

But why look at deaths of despair in unison? Why not consider trends in overdose, suicide, alcohol poisoning, cirrhosis separately? We look at these causes of death together because they all stem from violent and intentional self-harm and we often cannot tell them apart, in terms of intent, by looking at death certificates[9]. For example, the line separating drug overdose form suicide is muddy at best: the victim's intent is near impossible to determine with certainty, such that misclassifications are rampant in available data (Harriss et al., 2011). Because we cannot separate these causes of death in any accurate way, we look at them together. There is also a practical reason to define deaths of despair as a set: since 2015, the definition of deaths of despair as deaths from suicide, drug or alcohol poisoning, and alcohol liver disease is well-understood; which means that tinkering with this definition would lead to confusion, without apparent upside.

2.2 Labor Market trends

In this subsection we argue that an increasing percentage of prime-age men are choosing to not partake in the labor market, one signifiant driving factor behind this trend is the growth of import penetration from low income countries.

Employment rates of men in their prime are falling: in 1970, almost 95% of men between the ages of 25 and 54 worked; in 2016, fewer than 85% of US men in this same age group have a job (Fig. 7). In West Virginia, only 73% of men in their prime go to

[9] Of course, someone could argue that smoking or eating too much also constitute self-harm; to which we would answer that smoking a cigarette is clearly not the same as injecting heroin. But this argument is the reason why we exclude all deaths where drugs or alcohol are the proximate cause; such as car accidents of people under the influence of drugs or alcohol; the line may be muddy, but we do need to draw it somewhere.

work every day in 2016 (Tab. A2). This trend of declining labor market participation started in the 1970s, but accelerated in the 2000s (Fig. 7).[10] This acceleration coincides with the increase in mortality rates.This decline in employment rates has been a matter of significant interest for labor economists for the past 10 years.

The role of import penetration in the decline of manufacturing employment is among the best understood and more widely validated explanations of why employment rates have fallen.[11] To this point, Charles et al. (2016) argue that starting in the late nineties there is an accelerating decline of manufacturing employment; with the destruction of as much as 4 million jobs during the 2000s. Acemoglu et al. (2016) argue that during "the great employment sag of the 2000s", the increase of import penetration between 1991 and 2011 was responsible for the destruction of somewhere between 2 and 2.5 million jobs in manufacturing. This effect of import penetration on employment has been validated in multiple studies: Autor et al. (2013); Kollner (2016); Asquith et al. (2017); Del Angel et al. (2018), to cite only a few recent examples.[12]

The fact that the increasing mortality and the deteriorating labor market outcomes for people in their prime have evolved hand in hand suggests that, maybe, the two trends are related. As further evidence of this link, the medical and epidemiology fields have accumulated a significant amount of evidence on the correlation between labor market outcomes and health[13]; said evidence is the topic of the next section.

[10] Looking at Fig. 7 we can see that 12% of prime age males did not work both in 1975 and in 1999 (blue line). We can also see that the general trend of prime age men leaving the labor market starts early (see green line in the middle); but the cumulative figure accelerates its growth in 2000 (blue line on top).

[11] There are many alternative theories explaining why employment rates are in decline: some authors, blame the declining real wages of prime-age men without a college degree (Charles et al., 2013, 2016); there are those who point their finger to technological change and mechanization (Acemoglu and Restrepo, 2017); and others still, decry the effects of government transfers in decreasing the necessity to work (Glaeser, 2017); there are macroeconomic theories pointing to the role of hourly wage fluctuations and how they shape incentives (Elsby and Shapiro, 2012; Santos, 2014); and some more creative theories pointing to the role of video games in driving men away from paid work (Aguiar et al., 2017).

[12] As we explain in section 6, our reduced form estimates of the effect of Chinese export growth on US employment rates (results in Tab. 4) are consistent with the estimates in these papers.

[13] We have known for some time that the lives of the jobless in their prime are unhealthy. Compared to their peers who work, they smoke more, drink more, they are more likely to report chronic pain and to consume opioid drugs, their sleep schedules are more erratic, they interact less with others, they watch more television, they exercise less and are more likely to be obese, they are more likely to die before reaching retirement age, they are more likely to report poor mental health, they are more likely to commit suicide. The following papers support the previous claims: (in order) see Robroek et al. (2013) for smoking and drinking; see Krueger (2017) for chronic pain opioid use; see Gronseth et al. (2017) for sleep; see Robroek et al. (2013) for obesity lack of exercise; see Roelfs et al. (2011) for all-cause mortality before retirement age; see Milner et al. (2014) for mental health and suicide.

2.3 Mortality and the labor market

> "There is significant correlational evidence suggesting that misery haunts the lives of the long-term not working ." (Glaeser et al., 2018, p. 11)

The public health and epidemiology fields have produced abundant research analyzing the correlation between health outcomes and employment status at an individual level[14]. The main conclusions from longitudinal and cross-sectional studies are: (1) at any point in time there is significant correlation between unemployment and poor health outcomes; (2) introducing individual fixed-effects reduces the correlation but does not eliminate it, meaning that selection bias is not the only thing to the relationship between unemployment and poor health outcomes[15]; (3) longer unemployment spells have a larger negative impact on health than shorter unemployment spells (Milner et al., 2014). There is also evidence that one person's unemployment has effects on other people across the household (Moustgaard et al., 2018). These studies' main limit is that most are cross-sectional, and even the longitudinal ones usually cover a limited period of time[16]. We cannot draw causal inference from these correlational studies.

In economics, research on employment and health is more recent and more limited in breadth. Glaeser et al. (2018) suggest that the lack of employment drives people towards drug addiction and mental health problems; if they are right, then the declining employment rates of prime-age men might explain, at least in part, why deaths of despair are on the rise. Hollingsworth et al. (2018) find small but significant positive correlation between increases in unemployment and increases in deaths by opioid overdose. Charles et al. (2018) find a persistent correlation between declining manufacturing employment in the early 2000s and opioid use and mortality later on.

On the correlation between income and mortality Chetty et al. (2016) is the definitive reference. This large population study has 2 main conclusions: (1) There is a significant negative correlation between income and mortality at the individual and aggregate

[14] As for publications in economics, Krueger (2017) argues that pain and opioid addiction are pushing prime-age men out of the labor market: an estimated 44% of the prime-age males outside the labor market also reported taking opioids during the previous day. Glaeser et al. (2018, p. 12) report that almost 20% of prime-age men outside the labor force report low life satisfaction, and 30% of them report having "more than ten days of not good mental in the past month".

[15] Paul and Moser (2009) is just one example of the many meta-analyses supporting points 1 and 2. See Batic-Mujanovic et al. (2017); Zuelke et al. (2018); Brydsten et al. (2018) for recent individual studies supporting points 1 and 2.

[16] 24 months would be unusually large, see Stauder (2018) for a recent example.

levels. (2) Income matters most for mortality for lower income individuals: once above the median there seems to be little to no effect. (3) Income matters later in life, the correlation is strongest for older people, and small to none for younger people.

In sum, there is ample evidence of a strong correlation between labor market outcomes and mortality in the population. To study this relationship beyond mere correlation, we propose looking at the impact of an exogenous labor market shock (import penetration) on mortality rates.

2.4 Import penetration and the labor market

As we said earlier we are going to use import from China as an instrument to identify labor market demand shocks. In this subsection we make two arguments: first, the productivity growth of Chinese firms was large and unexpected. Second, China's increasing productivity, particularly in manufacturing, has had a significant negative effect on employment rates and earnings in US local labor markets. To conclude, we discuss our use of Chinese exports as a mere instrument helping us identify the effects of trade shocks more broadly.

The rising productivity of Chinese firms was large and unexpected. In 1991, low-income countries represented less than 10% of the US manufacturing imports; by 2007, that had risen to 28%, with China accounting for 89% of the increase (Autor et al., 2013, Table 1). Chinese productivity has increased significantly after market reforms presided by Deng Xiaoping in the 1980s; but that would not have produced the current outcome without the great rural-to-urban migration (Chen et al., 2010), among several other social forces.[17] As China became a marked-oriented economy, its comparative advantages in producing goods at a much lower cost has overcome many of the industries that used to operate in richer countries (Krugman, 2008). This process was sudden and unexpected; in the 1980s nothing of the sort had ever happened. The size of the increase in imports from China helps with identification: were it smaller we would not have enough statistical power to identify effects.[18]

The sudden nature of the effects also helps with identification. In the nineties, US firms grew and made investments unaware of the future growth of China as an exporter.

[17] See Naughton (2006) on empowerment of multinational enterprises. See Hsieh and Klenow (2009) on granting Chinese firms access to foreign technologies, capital goods, and intermediate inputs.

[18] Imagine a world where import penetration is responsible for the destruction of 2 thousand jobs and not 2 million during a 20 year period. This effect would be so small that estimates probably would not be statistically significant.

Same goes for US workers: they made human capital investments unaware of the displacement effects to come from China's growth. The transistor manufacturing sector is a good example of this: firms would not have made investments in transistor manufacturing plants in the nineties had they known that in a few years Chinese firms would be able to sell comparable products at a lower price. Workers in the transistor sector might not have made career investments in the sector. Younger people perhaps would have invested differently in their education had they known that the local transistor factory would close, victim of competition, in the 2000s. In other words, displacement effects are particularly important because economic actors cannot predict the future and make investments that, given hindsight, seem wrongheaded.

There is a large body of literature on the labor market effects of Chinese import penetration on US local labor markets. Acemoglu et al. (2016) find a negative impact on employment rates for the affected industries but also for industries supplying products and services to those affected (aka. "upstream" industries), a phenomena mostly explained by plant closures, as opposed to reductions of personnel in surviving plants (Asquith et al., 2017). Autor et al. (2013) find a negative impact of exposure to Chinese import penetration on wages; surprisingly, these wage effects are strongest for lower earners in the services sector (as opposed to manufacturing). A plausible explanation is that this is a simple price effect: as jobs disappear in manufacturing, displaced workers search for alternatives in the services sector.[19] This creates an increase in supply of labor in the services sector and precipitates a drop in the price of said labor.

The tried, tested, and reviewed research on the labor market effects of import penetration from China is central to our paper. It is because the "China shock" (Autor et al., 2013) has a negative impact on employment rates and income that it affects mortality rates. As we mentioned in the introduction, this is an instrumental variable/reduced-form approach where Chinese export growth serves as an instrument to study the effect of labor market outcomes on mortality.

At this point, it is worth mentioning that we are not interested in China per se, we merely use it as an instrument to estimate the broader consequences of import penetration and labor market shocks in general. We choose China specifically for two reasons: first, because it is the largest exporter around the world, its footprint on

[19] Del Angel et al. (2018) find that the Chinese import penetration mostly destroyed routine non-technical jobs which do not require extensive education or training. The argument being that these non-technical manufacturing workers are most likely to find work that does not require previous training in the services sector.

global markets is large and this size helps us pick up on smaller variations in US local labor markets. Second, because it was the first low-income country to increase its manufacturing capacity fast, a characteristic that helps with identification.[20]

2.5 Import Penetration and Mortality

We now review research on the impact of international trade policy and import penetration on mortality. There are two studies preceding our own, we build on their findings. We also review a third study, which relates only tangentially to ours.

Pierce and Schott (2016) are also interested in the effects of trade liberalization on mortality from a slightly different perspective. They look at the product by product change in legal framework of trade between the US and China: establishing the Permanent Normal Trade Relations (PNTR). In 2000, some products were awarded PNTR and saw their tariffs reduced, others were not.[21] The authors use this set of binary instruments (one binary variable per product) to measure the effect of PNTR on mortality within a Differences-in-differences strategy: they compare mortality outcomes before and after 2000. They find that counties exposed to the PNTR agreement[22] saw a larger growth in mortality between 1999 and 2004 than non-exposed counties. However, the labor market effects of PNTR are concentrated in the 2000 to 2004 period, and mitigated by the housing boom happening in the same period.[23] So, Pierce and Schott (2016) can achieve a good understanding of the impact of trade liberalization

[20]"While China dominates low-income-country exports to the United States, trade with middle-income nations, such as Mexico, may also matter for US labor-market outcomes. The North American Free Trade Agreement (1994) and the Central American Free Trade Agreement (2005) each lowered US barriers to imports. However, whereas China's export growth appears driven by internal conditions and global changes in trade policy toward the country, export growth in Mexico and Central America appears more related to import demand associated with US outsourcing to the region. Consequently, it is more difficult to find exogenous variation in US imports from Mexico and Central America." (Autor et al., 2013, p. 2123)

[21]It is important to note that the decision to award or withhold PNTR status to one product or another is made by legislative bodies. These bodies are acutely aware of the economic impact that granting PNTR status to one product or another would have on their constituents. As such, representatives have an incentive to lobby against granting PNTR status to products economically important in their home districts. For example, a representative from Kentucky would have a strong incentive to withhold PNTR status from Chinese-made bourbon. Which is to say that in the 2001 PNTR game, winners and losers were carefully considered.

[22]Exposed here means having plants which produce goods included in the PNTR agreement.

[23]Meaning that, the declining demand for manufacturing labor between 2001 and 2004 was, in some places, countered by a rising demand for labor in the construction sector, which was booming at the time. See Charles et al. (2016) for details on how both phenomena interact.

on the growth in mortality rates between 1999 and 2004[24], in areas where the housing boom was not too large. Which limits the scope of their findings. By looking at a broader time frame, and a more general definition of trade we hope to achieve nationally representative estimates over time that might shed light on the mechanisms linking labor market and mortality.

Donnally (2017) looks at the impact of import penetration on the increase of *all-cause* mortality in the US, a project whose underlying idea bears some resemblance to our own. Her results are in line with ours: higher levels of import penetration are associated with higher levels of mortality for the overall US population. However, this important finding raises multiple questions about the underlying mechanisms: why does import penetration matter in determining employment rates? What mechanisms link both things? Donnally (2017) cannot answer these questions because she lacks the necessary data: she only has data on *all-cause* mortality, and her analysis is limited to the 1999 to 2012 period.[25] With the benefit of fuller economic and mortality datasets we can deepen and broaden our understanding of the underlying mechanisms linking import penetration to mortality. By looking at the impact of import penetration on deaths of despair we can show that the relationship between import penetration and mortality is driven by deaths from suicide and addiction. This is a central piece of information to understand the mechanisms involved in this relationship.

At this point, we must mention another paper which is only tangentially related to ours: Autor et al. (2018) look at the impact of import penetration on a variable related to mortality rates. The dependent variable in their work is: *level* of gender gaps in accumulated deaths over 10 years. Our work, is concerned with the *growth* of mortality rates in the population in general. Since 1999, mortality *growth* for women has often been higher than mortality *growth* for men (Gelman and Auerbach, 2016), even as *levels* of mortality for men are still higher than *levels* of mortality for women. As such, the fact that places with higher import penetration growth have a higher *level* of men dying of drug overdose than women is really just a different study from ours.

2.5.1 The cumulative disadvantage hypothesis

> "If you've always been privileged, equality begins to look like oppression"[26]

[24]Theirs is a Differences-in-Differences comparing mortality outcomes in 1999 to 2004.
[25]Furthermore, the findings are hindered by a lack of data on almost 400 out of 3,115 US counties.
[26]Carol Anderson in an interview for POLITICO Magazine: (Glasser and Thrush, 2016)

As we mentioned before, previous work on the growth of mortality rates among people in their prime has shown that the trend is strongest among white non-hispanics (Case and Deaton, 2015). However, the labor market trends we described in section 2.4 are broad, and they affect all social and racial groups: men, women, whites, blacks, hispanics, etc. So, if these economic trends affect everyone shouldn't we see increases in deaths of despair in all racial and ethnic groups? How does it make sense that, under the same causes, whites would be the only ones whose mortality is increasing?

There are two answers to this question. The first answer is that, as we showed in the beginning, mortality rates of minorities are also increasing; both by deaths of despair (Fig. 5-6) and, to a lesser extent, by all-cause mortality (Fig. 3). However, this first answer does not explain why the growth in mortality for minorities is smaller and more recent than that the growth for non-hispanic whites. Any satisfactory answer would have to explain why white non-hispanics and minorities experience different trends in mortality. The second answer is Case and Deaton (2015, 2017)'s theory of cumulative disadvantage: their hypothesis is that it is not the economic strife per se that causes these deaths of despair; rather, it is doing worse that one's parents that really drives people to the brink. In other words, unemployment or low wages per se, do not have an impact on deaths of despair, but not succeeding as well as one's parents does. The underlying idea is that people's expectations are formed by looking at their parents and it is having those expectations disappointed that really takes a psychological toll. To the extent that past generations of minorities have had worse labor market experiences than whites[27], and younger generations of minorities are still able to outperform their parents in the labor market, this theory could explain the difference in growth of mortality across race/ethnicity.

In essence, the cumulative disadvantage hypothesis is telling us that labor market effects are not comparable across groups, unless we hold parental income constant. Because the distribution of parental income is not constant across groups, they are not comparable. But what if we could hold parental income constant? Case and Deaton (2017) claim that the cumulative disadvantage hypothesis cannot be tested because there was no appropriate data to do it at the time where they were writing. However, in a fortunate turn of events, Chetty et al. (2018) published (almost) exactly the data we need to test this hypothesis in March 2018: population-wide estimates of income by racial/ethnic group $\times$ sex $\times$ parental income percentile $\times$ commuting zone. In

[27] "As has often been noted, blacks are no strangers to labor market deprivations, and may be more inured to the insults of the market." (Case and Deaton, 2017, p. 432)

other words, we can test the impact of import penetration by racial/ethnic group while holding parental income constant. Therefore, we can test whether import penetration has a negative effect on intergenerational income upwards mobility by racial/ethnic group.

3 Data

We use 8 main datasets, together they provide the pieces to build variation across counties, commuting zones, and time of health and labor market outcomes as well as regional exposure to international trade. The mortality rate data by county×year×age group×racial/ethnic group×cause of death comes from one single database. Two other datasets provide county×year variation in health insurance coverage rates and opioid prescription rates outcomes. We use two separate datasets to estimate employment rates (employment to population ratios) both at the commuting zone level, and the county level for each racial/ethnic group. Trade data come from one single dataset and allows us to estimate exposure to international trade by industry. Income data come from recent research on income mobility by racial/ethnic group. Furthermore we use an alternative (eighth) dataset to study nationally representative labor market outcomes.

3.1 Mortality Data

Our first and most essential dataset consists of US mortality data by county from 1991 to 2016. We access this data through the CDC's WONDER system. This datasource gives us statistics by county×year×age group×racial/ethnic group×cause of death. To protect privacy some data by county is left censored: any county counting less than 10 deaths in one category, does not show the exact number. However, this privacy policy only precludes us from accessing data on 8 counties out of 3,115 continuous counties in continental US excluding Alaska. Specific causes of death are constructed for 1991–2016 using International Statistical Classification of Diseases and Related Health Problems 10th Revision (ICD10) codes: alcoholic liver diseases and cirrhosis (K70, K73-74), suicide (X60-84, Y87.0), and poisonings (X40-45, Y10-15, Y45, 47, 49). Poisonings are accidental and intent-undetermined deaths from alcohol poisoning and overdoses of prescription and illegal drugs. Among the deaths with drug overdose as the underlying cause, the type of opioid involved is indicated by the following ICD-10 multiple cause-of-death codes: heroin (T40.1), natural and semisynthetic opioids (T40.2), methadone

(T40.3), synthetic opioids other than methadone (T40.4), and Cocaine (T40.5).

In order to show that the increase in mortality is not some generalized phenomena, and mortality by causes other than despair is decreasing we also consider the following causes of death: all cancer (all neoplasms - C00-D48), all vascular disease (I00-99, dominated by coronary heart disease and stroke - I60-69), Central retina artery occlusion (H34.1 - stroke category), Transient cerebral ischemic attacks and related syndromes (G45 - also stroke category), and diabetes (E10-E14, Diabetes mellitus, excluding gestational). We compare the evolution of these causes of death with the evolution of deaths of despair in Fig. 10.

Mortality and morbidity data from 1979 to 1998 come in ICD-9 codes: alcoholic liver diseases and cirrhosis (571.0-571.6), Suicide (E950–E959), poisonings (E850-855, E935, E937, E939, E980.0-980.5).

Non-US Mortality Data comes from the Human Mortality Database. A project of the University of California, Berkeley, and the Max Planck Institute for Demographic Research. Available at www.mortality.org or www.humanmortality.de. We use it to generate Fig. 1 and 2.

3.2 Employment data

Our main data source for employment data is the County Business Patterns (CBP) dataset. It provides us with both an estimate of how many people are employed in a given commuting zone as a whole and how many people are employed by 4-digit industry code. The employment-by-sector data is central for estimating the size of import penetration (our instrument) across commuting zones[28]: the commuting-zones-specific industry shares allow us to weight the international trade product data and create the import penetration instrument as explained in the identification section. We use the total employment by commuting zone data to estimate the effect of import penetration on employment rates in Table 4. The CBP dataset is created from the Business Register (the Census Bureau's file of all firms), which in turn is created from multiple data sources.[29]

[28]A commuting zone is a geographic area designed to represent a local labor market, it comprises a set of counties.

[29]Census bureau has a website for the CBP dataset (https://www.census.gov/programs-surveys/cbp.html), we invite the reader to look at it as the data-generating process behind the CBP dataset is not simple, but going into the details of how the Census Bureau generates its firm data is besides this paper.

The CBP dataset does not disclose data on specific employers and county-level employment data by sector is often presented as an interval. To create industry-level estimates of employment by county we use a fixed-point algorithm proposed by Autor et al. (2013) that takes the industry employment intervals provided by the CBP data and gives us point estimates. We then aggregate these point estimates at the commuting zone level.

To produce county-level estimates of employment rates by race-ethnicity we use the 1990 and 2000 Census data: the long-form questionnaire, which is applied to 1/6th of census respondents (19 million housing units in 2000). The results of this questionnaire are summarized in the Summary File 3 (SF3) for 1980, 1990, and 2000. After 2000, the long-form census questionnaire was replaced by the American Communities Survey (which also substituted the current population survey). So, after 2000, we use ACS estimates based on 5 consecutive years of data collection to get ethnicity/race-specific employment rates. These 5-year estimates are a courtesy of the American Fact Finder project. .

To produce the national level employment statistics that feed into Fig. 7-9 we use the Public Use Microdata Series (IPUMS) based on the Current Population Survey (CPS) dataset. This dataset provides nationally representative estimates for labor market trends and descriptions. All analyses exclude people employed by the armed forces and children.

3.3 Trade data

Data on international trade for 1991 to 2016 are from the UN COMTRADE Database[30], which gives bilateral imports for six digit Harmonized Commodity Description and Coding System (HS) products. Because HS and Standard Industrial Classification (SIC) codes do not concord, we have to apply the crosswalk proposed by Pierce and Schott (2012) to obtain the required dataset. The crosswalk assigns HS codes to all but a small number of SIC industries. In order to match every industry to at least one trade code, we have to aggregate a few of the 4-digit SIC industries. This method provides us with bilateral trade data with code matching the employment data from the CBP dataset.

[30] We extracted the data from the website public API. See (https://comtrade.un.org/data/doc/api/) for instructions.

3.4 Income data

To produce Tab. 5 we use the data recently published by Chetty et al. (2018). Sample consists of children born between 1978 and 1983 and their parents. Children are assigned to commuting zones based on the first non-missing zip code of their parents, irrespective of where they live as adults. Child income is calculated as mean individual income in 2014-15, when children are in their mid-thirties. Parent income is measured as mean household income between 1994 and 2000, when their children are between the ages of 11 and 22. Parental income percentile is calculated among parents born in the same cohort. Areas with fewer than 20 children in the core sample, for which we have inadequate data to estimate mobility, or fewer than 500 residents of the children's racial group in the 2000 Census are excluded.

3.5 Health insurance data

Data on Health insurance coverage comes from the Small Area Health Insurance Estimates (SAHIE) program by the census bureau. It provides estimates of health insurance coverage for the population at the county level for the year 2000, and then 2005 to 2016. Number of people with and without health insurance is calculated using the Socioeconomic Supplement of the Current Population Survey (CPS) and the American Community Survey (ACS) as well as census and administrative data.[31]

3.6 Drug prescription data

Data on drug prescriptions comes from QuintilesIMS Transactional Data Warehouse (TDW) 2006 - 2016. QuintilesIMS TDW is based on a sample of approximately 59,000 retail (non-hospital) pharmacies, which dispense nearly 88% of all retail prescriptions in the U.S. For this database, a prescription is an initial or refill prescription dispensed at a retail pharmacy in the sample, and paid for by commercial insurance, Medicaid, Medicare, or cash or its equivalent. Does not include mail order pharmacy data. This sample includes Opioid prescriptions, including butrans (buprenorphine), codeine, fentanyl, hydrocodone, hydromorphone, methadone, morphine, oxycodone, oxymorphone, propoxyphene, tapentadol, and tramadol were identified using the National Drug Code. Cough and cold formulations containing opioids and buprenorphine, an opioid partial agonist used for treatment of opioid use disorder as well as for pain, were not included.

[31] For details see the program website: https://www.census.gov/programs-surveys/sahie.html

In addition, methadone dispensed through methadone maintenance treatment programs is not included in QuintilesIMS TDW data. The data was accessed through the CDC's website[32] (Citation as suggested by CDC).

Tab. A1 provides descriptive statistics, but regression Tables have their own descriptive statistics of the variables involved to ease interpretation.

4 Empirical strategy

In this section we discuss identification and estimation. To identify the impact of import penetration on mortality rates we use a first-difference, instrumental variable (IV)/reduced-form, approach where we regress mortality rates directly on our import penetration instrument. The first-difference estimator addresses potential endogeneity concerns; the IV approach solves the reverse causality problem.[33] We only present reduced-form estimates because import penetration has an impact on both employment rates and income, the two effects are not separable.[34]

4.1 Framework

Import Penetration The main variable of interest is import penetration, which measures the exposure of an average worker in a commuting zone to import exposure from China. We construct the import penetration instrument in a two-step process. In the first step, we estimate the national change in import exposure, ΔM_{jt}, for each industry j. We define import exposure as the total value of imports in an industry divided by a measure of initial absorption.[35] An important caveat is that, because import penetration from China to the US could be driven by changes in demand from the US and not capture a clean "supply effect", we estimate import exposure M_{jt} as imports

[32]https://www.cdc.gov/drugoverdose/maps/rxrate-maps.html

[33]Endogeneity could be a problem because some place-specific characteristic (e.g. income) could affect both labor market and health outcomes. The reverse causality is a problem can be summed up in the following question: are people out of a job because they are unhealthy, or are people unhealthy because they are out of a job?

[34]Using a two-stage-least-squares (2SLS) estimator with either employment rates or income as the instrumented variable would breach the exclusion restriction assumption because both employment rates and income are related to mortality rates. The exclusion restriction assumption is necessary for the consistency of the 2SLS estimator.

[35]$M_{jt} = Imports_{jt}/Absorption\,(j)_{1991}$. $Imports_{jt}$ is the total dollar amount of goods imported from China in industry j, year t. $Absorption\,(j)_{1991} = Y_{j,91} + M_{j,91} - V_{j,91}$ is total US manufacturing product in industry j, plus industry j imports from China, minus industry j exports to China, in 1991, when China was not a major exporter.

from China to 8 other developed nations.[36] In the second step, we calculate import penetration in each US commuting zone s by summing across industries' import exposure and weighting each element by the employment share of industry j in commuting zone s. More specifically, import penetration is defined as

$$\Delta IPO_{st} = \sum_j \frac{L_{s,j,88}}{L_{s,88}} \cdot \Delta M_{jt} \tag{1}$$

where ΔM_{jt} is the change in national industry import exposure from t-1 to t. We weight each industry by $L_{s,j,88}/L_{s,88}$ which corresponds to the industry's share of total employment in commuting zone s. To ensure that the weights are clean of any influence that Chinese imports have on industry composition, we set the weights to their 1988 value, before China became a major exporter. All in all, this import penetration variable, IPO_{st}, gives us a measure of average worker exposure to Chinese import competition, in commuting zone s, year t.[37]

The central idea behind this instrument is that the *composition* of the manufacturing workforce in 1988 locks local labor markets down a path of economic competition during the 1990s and 2000s. Having factories in sectors that shifted towards foreign production after 1991, as opposed to having factories in sectors that did not shift towards foreign production, translates into higher levels of import penetration. For example, a local labor market with many transistor factories in 1988 will have high import penetration; while a local labor market with many bedding factories in 1988 will have a low import penetration.[38] Another important source of variation is the timing of the shift: not all industries shifted production to China at the same time. Apparel shifted in the early 1990s; transistors only in the 2000s. Of course we apply this over 4-digit industries, so the resulting import penetration variable is much more composite.

Identification To identify the impact of import penetration on mortality rates we use a first differences model allowing effects to vary across racial/ethnic groups. Our

[36] Australia, Denmark, Finland, Germany, Japan, New Zealand, Spain, and Switzerland. Why these countries? Because bilateral trade with China was available, at the industry level, in 1991, the earliest of any representative set.

[37] Previous work has focused on import penetration from China to the US and instrumented this endogenous variable using import penetration from China to 8 developed nations in a two-stage-least-squares approach. In this paper we simplify things and look only at the reduced-form impact of import penetration from China to these 8 developed nations. For simplicity, we call this import penetration.

[38] Transistor production has moved to China (Shenzhen in particular), few factories factories remain in the US. Mattresses and bedding are still largely produced in the US.

model is

$$\Delta D^g_{it} = \alpha^g_t + \beta^g \Delta IPO_{st} + X^g_{it\text{-}1}\gamma^g + \varepsilon_{it} \tag{2}$$

where ΔD^g_{it} is change in the outcome variable, in county i, from t-1 to t, for group g. The central outcome variable in our paper is death rate, but we also use this identification strategy to study the effect of import penetration on other health outcomes and on employment rates.[39] In several cases we consider identification of the effects across racial/ethnic groups separately ; we consider 4 groups: white non-hispanics (WNH), black non-hispanics (BNH), hispanics, and others. α_t, λ_t and ξ_t are time-period fixed effects. $X_{it\text{-}1}$ is the share of the commuting zone population working in the manufacturing sector at t-1. Controlling for share of population working in manufacturing at baseline year is important because areas had who were heavily reliant on manufacturing before 1991 had higher growth of import penetration in later years (Autor et al., 2013).

For this model to be identified we need only for ΔIPO_{st} to be independent of ε_{it}, conditional on covariates. Something we will argue is true for the remainder of this section by going through every possible concern and checking them out one by one. To the extent that $\varepsilon_{it} \perp\!\!\!\perp \Delta IPO_{st} | X_{it\text{-}1}$ and we have a clean instrument, β^g is a consistent and unbiased estimator of the impact of import penetration on mortality rates for any group g.

Estimation In accordance with Acemoglu et al. (2016) of import penetration, we use large differences in our first-difference model. In particular, we study changes in import penetration and mortality from 1991 to 1999, 1999 to 2007, and 2007 to 2016. The reason why we look at large differences (as opposed to looking at year-to-year changes) is because we are interested in studying long-term shifts in the population. If China's total factor productivity increases from one year to anther, that might not translate into plants closures in the US immediately; but it will probably cause plants to close, eventually. As such, it makes sense to look at longer-term changes, so we can pick up on total effects and not just immediate ones.

While local labor market shocks happen at the commuting zone level, we look at mortality rates at the most disaggregated level possible, counties. We do this for efficiency purposes; as recommended by Deaton (1997, pp. 67-73) for the study of ag-

[39] ΔD_{it} could refer to change in death rate, change in health insurance coverage rate, change in opioid drug prescription rate, or change in employment rate.

gregated data. The argument is that smaller aggregation is always useful to increase precision of estimates, if some variables are at a higher level of aggregation standard errors can be clustered accordingly and point estimates will still be consistent.

We estimate our model using ordinary least squares. Using weighted least squares for aggregate data, may lead to over-estimating coefficients if spillover effects are present[40]; this matter is first observed by by DuMouchel and Duncan (1983), it is discussed in detail by Dickens (1990), and Solon et al. (2015) rearrange some of the existing arguments for greater clarity. The intuition behind this result is simple: if A causes B for each person and does not affect others, then weighting can help us approach individual-level estimates; if A causes B for each person, but also for others, then aggregate effects are correlated with network size and spillover size, meaning that weights are correlated with the size of the effect, thus causing over-estimation. More to the point, Dickens (1990), proves that, in the presence of aggregated data for groups where error terms are correlated across individuals within each group, using weighted least squares can lead to type 1 error. Import penetration has been shown to cause ripple effects through local economies (for example, affecting wages in the non-manufacturing sector as we saw earlier) so spillover is most likely present; i.e. we cannot use weighted least squares.

Interpretation Interpreting the results is not simple: what does an increase in exports from China to 8 developed nations actually mean in palpable economic terms? We use the same interpretation as Acemoglu et al. (2016): the impact of a percentage point increase in import exposure since 1991. By looking at levels of exposure relative to 1991 sector size, we can give the reader a notion of how much the growth in level of exposure has affected outcomes. Consider the following example: suppose the β coefficient associated with ΔIPO_{st} in equation (2) is equal to 1 and we are calculating the effect of import penetration on the whole US population. Then, the way to interpret this coefficient is: a 1 percentage point increase in import penetration is associated with an increase in mortality rate of one yearly death per 100,000 living people. Given a US population of 325 million people, one yearly death per 100,000 living is equivalent 3,250 deaths every year. So, if $\beta = 1$ in eq. (2) and we are calculating the effect of import penetration on mortality for everyone in the US, then a 1 percentage point increase in import penetration is associated with an extra 3,250 deaths per year. Also, between 1991 and 2016, import penetration has increased by 2.51 percentage points on

[40]In our regressions weighted least squares estimated coefficients are slightly higher; we do not present these results because we suspect they may suffer from upwards bias.

average (with a standard deviation of 2.5 percentage points). Which means that the total average effect of the increase in import penetration between 1991 and 2016 is an extra 2.51 deaths per year per 100,000 living, or an extra 8,158 deaths, in the US, every year.

4.2 Exclusion restriction

Showing that import penetration from China to the US has an impact on mortality rates is, in and of itself, an important and worthwhile finding. But, the path from import penetration to mortality rates has important policy implications. If import penetration is causing an increase in mortality through labor market shocks, then policy concerns should focus on labor market interventions aimed to cushion the blow of trade shocks. If, on the contrary, hazardous products imported from China were causing the increase in mortality rates, then the policy implications would be quite different. Simply saying that β is a consistent estimator of the impact of import penetration on mortality rates is not enough, we need to discuss what that means.

Trends in health A growth in productivity of Chinese firms in one industry rather than another is itself independent of health trends in the US. For example, the fact that different firms in Shenzhen mastered the art of making cheap transistors during the 2000s should not be *directly* related to the progress of any disease in the Bay Area, California; or any other part of the US for that matter. In this sense, our instrument is valid: increase in import competition from China in one sector is, by all means, exogenous. But labor markets more heavily reliant on manufacturing in 1991, at the onset of China's productivity boom, were more likely to be disrupted by Chinese import competition during the following 25 years. At the same time, places where a large share of the population was employed in the manufacturing sector in 1991 may have distinct health trends from the rest of the country. Perhaps there are diseases or health factors directly related to working in the manufacturing sector. This would be a problem, and could be a source of bias, if the solution were't so simple: we can control for "share of the population employed in the manufacturing sector" at baseline, which we do for all our models as is standard in the literature.

Other identification concerns If the demand forces that influence import penetration in the US are somehow correlated with other developed countries imports, then the IV is contaminated, and our estimations will be biased. In other words, if demand shocks are correlated among all the countries (the US and the other 8 developed nations) then this could mean that our effect can be partially explained by US demand shocks. Acemoglu et al. (2016, S163) test this hypothesis, and squarely reject it: not only is there no evidence of the presence of correlated demand shocks, including controls for those shock does not produce significantly different estimates.[41]

Another preoccupation is that the rising Chinese imports (in all the rich countries including the US) may be associated with a productivity shock in a country other than China. Suppose that Italian clothes are very demanded by OECD countries. If Italian productivity in this sector has declined over the past decades, then the US and the others may import clothes from China rather than Italy, and we would have again correlation between our instrument and the variables of interest. But the tremendous and continuous increase in China's total factor productivity suggests the drivers of import penetration (common across the countries we study) are related to China itself.

An ultimate concern is that import penetration is mechanically related to mechanization (or any other major job-destroying trend): that could muddle estimates. If increasing usage of machines and robots is making China a better competitor, we run the risk of adjudicating to China a job destruction that is better explained by mechanization. This would make our instrument is deficient, since it would correlate with a confounding variable: mechanization. But China in 1991 has a lower robot use than the developed countries we are looking at. If anything mechanization in a given sector might make production in China less appealing, not more (Acemoglu and Restrepo, 2016). The enormous population, with a very low-wage workforce, and all the economic institution changes China has experienced, are the main drivers of the growth of Chinese import penetration in developed countries, not mechanization. This puts our mind at ease and assures us that our estimates are not related to mechanization.

5 Main Results

In this section, we estimate the reduced form impact of import penetration on mortality in general and mortality by deaths of despair in particular. We also preform two

[41] They test whether including (a) 4-digit sector dummies, (b) industry-level controls for production structure, and (c) industry-level controls for pretrends, have any influence on the magnitude or precision of the estimated impact of import penetration on employment and wages. Including these controls does not change anything meaning that if there were sector-specific shocks then they would have no bearing on the impact of import penetration on employment and wages.

falsification tests and review the impact of import penetration on intermediate variables as supporting evidence.

5.1 Import penetration and mortality across age groups

We find the impact of import penetration on mortality divides into two main components: impact on deaths of despair, and impact on death by other causes. For white non-hispanics in their prime working years, the effect of import penetration on mortality is dominated by the deaths of despair component, which accounts for over half of the effect. For older people (over 65), the effect is dominated by the other causes of deaths component, consistent with an income effect interpretation (Chetty et al., 2016). For younger people (the majority of whom have not entered the labor market) there is no effect.

Identification in this subsection is based on eq. (2) with change in death rates as de the outcome variable.

Import penetration and death of people in their prime

Our first result is that, for white non-hispanics aged 25 to 54, import penetration has a significant positive impact on all-cause mortality (Tab 1, Pan A, col 2); and more than half of that effect is accounted for by deaths of despair alone (Tab 1, Pan A, col 4). More specifically, a 1 percentage point increase in import penetration is associated with a 3.19 increase in (yearly) deaths by any cause per 100,000 living; and the same percentage point increase in import penetration is associated with a 1.65 increase in (yearly) deaths of despair per 100,000 living. Such that slightly more than half of the effect is accounted for by deaths of despair. This result is significant because between 1991 and 2016 a grand total of 63 million people died in the US; but only 2.3 million of those deaths were from suicide, overdose (poisoning), or alcoholic liver disease. This means that a set of causes of death accounting for less than 4% of deaths overall, drive more than half of the effect that import penetration has on mortality of white non-hispanics in their prime.[42]

Our second result is that, for minorities aged 25 to 54, we cannot establish a significant impact of import penetration on mortality rates, regardless of cause (see Tab 1,

[42] Let Y: deaths by all cause, Y_1: deaths of despair, Y_2: deaths by other causes. Since $Y = Y_1 + Y_2$ it is easy to see that $\beta = (X'X)^{-1} X'Y = (X'X)^{-1} X'Y_1 + (X'X)^{-1} X'Y_2 = \beta_1 + \beta_2$. It follows that the effect of import penetration on deaths by other causes is 1.53 ($= 3.19 - 1.66$).

Pan A, Cols 3 and 6). This result is consistent with previous research (Case and Deaton, 2015, 2017) pointing to a surge in mortality much stronger for white non-hispanics than for minorities (Fig. 5-6). Bellow we discuss why results could be different for white non-hispanics and minorities, and what role the cumulative disadvantage hypothesis might be playing in mediating these different effects.

Import penetration and mortality among people aged 65 and older

There is significant evidence relating household income and mortality rates of people over 65 in the US (Chetty et al., 2016), therefore, to the extent that import penetration has an impact on household income, we also expect it to have an effect on the mortality of the elderly. The results in Tab 1 Panel B seem to confirm this evidence: import penetration has a positive impact on mortality for people over 65. What is interesting is that, contrary to people in their prime, this effect is overwhelmingly concentrated on causes of death other than despair; the effect of import penetration on the mortality rates of the elderly is small, accounting for about 1 twentieth of the overall effect.

Import penetration and mortality among people aged 25 and younger (placebo test)

Contrary to those aged 65 or older there is no known evidence on the effects of income on mortality of young people, which makes sense: they either have not entered the job market yet or they are just starting. In this sense, we do not expect import penetration to significantly affect health outcomes and mortality for this group and this exercise can be seen as a placebo test. Tab. 1 Panel C presents results for the effects of import penetration on mortality of people under the age of 25; as expected, all results are close to 0 and not significant for all estimates.

A note on magnitude

Average increase in import penetration between 1991 and 2016 is 2.51[43] with a standard deviation of 2.5. This means that even considering the whole period, total effects of import penetration on all-cause mortality are relatively small: around 4% of the average mortality rate for people in their prime and 1% of the average mortality rate for the

[43]More specifically, we mean that $\frac{1}{n}\sum_s^n \Delta IPO_s = 2.51$ where $n = 3,115$, the total number of US counties in our sample, and $\Delta IPO_s = IPO_{s,2016} - IPO_{s,1991}$.

elderly.[44] This is consistent with evidence from Chetty et al. (2016) that income effects on mortality and life expectancy are modest and mainly relevant for people in the bottom half of the income distribution. But the effects of import penetration on deaths of despair is smaller in absolute terms, but much larger in relative terms.

The effect of import penetration on deaths of despair by white non-hispanics in their prime is large: the US mortality rate for this group increases 41 points between 1991 and 2016, that's 41 yearly deaths per 100,000 living. Import penetration, increases by 2.51 percentage points on average, during the same period. With $\beta = 1.66$ (Tab. 1) we have a predicted average increase in mortality rates of 4.17 points, that's an extra 4.17 deaths every year per 100,000 living. As such, import penetration predicts an increase of about 10% the size of the overall growth in mortality rates by deaths of despair, for white non-hispanics in their prime. That might seems small, but white non-hispanics in their prime are a big group, so this 4.17 point increase corresponds to 3,206 deaths in 2016 alone.[45] However, this result hides significant heterogeneity. On the one hand, in counties one standard deviation above the mean in terms of increase in import penetration, we have an accumulated increase in import penetration between 1991 and 2016 of 5 percentage points. With $\beta = 1.66$ (Tab. 1) our model predicts that a county with a 5 percentage point would see an increase in mortality rates of 8.3 points, that's an extra 8.3 deaths every year per 100,000 living. On the other hand, for 25% of the counties in our sample, import penetration increases by 1.09 percentage points or less between 1991 and 2016; which means their predicted levels of increase in mortality rates are at or bellow 1.81 points.

5.2 Falsification exercise

One simple and straightforward placebo test consists in looking at the impact of import penetration in the nineties on mortality between 1983 and 1991. Before 1991 China was not a large exporter and exports to the US were not significant. So, growth of import penetration in the nineties should not relate to mortality in the eighties; that is, unless mortality trends somehow predicted industry-specific productivity growth of Chinese firms. To preform this falsification exercise we follow equation (12), but we insert a lag in the dependent variable such that we are actually estimating $\Delta D^{g}_{i,83\text{-}91}$ as a function

[44] For ages 25 to 54 we calculate $2.51 \times 3.61/241$.

[45] $76{,}889{,}021 \times 4.17 \div 100{,}000 \approx 3{,}206$

of $\Delta IPO_{s,91\text{-}99}$.[46] We eliminate time-periods after 1999 because growth of import penetration is correlated over time. Tab. 2 presents the results of this falsification exercise. None of the results are significant, meaning that import penetration in the nineties has no bearing on mortality rates in the eighties.

5.3 Intermediate variables

Our goal in this section is to explore supportive evidence of the pathways from import penetration as a labor market shock to Health and mortality outcomes. We know from previous research (see background section) that import penetration has a significant impact on a number of economic outcomes. Our goal in this subsection is to see how import penetration could impact health through a variety of paths. We find that import penetration has a positive impact on the number of opioid prescriptions that are dispensed in a county; evidence suggesting that import penetration could be associated with increasing opioid drug consumption. An increase in consumption could explain, in part, the increase in mortality from overdose on opioid drugs. We also find that import penetration has a negative impact on health insurance coverage at the county level. Since access to health care in the US is often mediated by access to health insurance, this evidence supports the existence of an effect of import penetration on mortality.

Identification of the impact of import penetration on intermediate health variables also follows equation (2) except that the outcome variable is no longer change in mortality rates, but change in health insurance coverage rate and change in opioid drug prescriptions rate.

Health insurance coverage In the US, access to health insurance and health care have long depended on either an individual's ability to pay or their employer's ability/willingness to do so[47]; therefore, we expect any labor market shock which affects both income and employment rates to have a significant bearing on health insurance coverage rates. Results in Tab. 3, Col. 2 seem consistent with this theory: a one percentage point increase in import penetration is associated with a 0.33 percentage point decrease in health insurance coverage in the overall county population (out of all residents). While that may seem small, in a county with 100,000 residents that means 330 fewer people with health insurance. Furthermore, it is possible that this 0.33 estimate

[46] more specifically, we estimate: $\Delta D^g_{i,83\text{-}91} = \alpha^g + \beta^g \Delta IPO_{s,91\text{-}99} + X^g_i \gamma^g + \varepsilon_{it}$

[47] we only analyze data from 2000 to 2008 so more recent policy considerations such as the Affordable Care Act (ACA) should not affect our analysis.

is bellow the true effect of import penetration on health insurance coverage because we only have access to overall coverage rates.[48] The overall rate we are using can be defined as a weighted average: a county's share of people under 65 multiplied by their coverage rate and added to the share of people over 64 multiplied by their their coverage rate. This distinction is important because health insurance coverage for people over 64 is close to 100% since, in the US, everyone over 64 is covered by the Medicaid program. Because people over 64 are fully covered, the effect of import penetration on coverage for this group must be around 0.[49] Regardless of exact coefficient size, import penetration has an important and significant effect on health insurance coverage rates; this effect elucidates one possible avenue from import penetration to mortality.

Opioid prescription Within the Deaths of Despair set of causes of death, deaths by alcohol or drug overdose (poisoning) is one of the largest growing categories (Fig. 10). It is extremely hard to obtain accurate data on drug trafficking, there is no reliable dataset on the volume of drugs circulating every year in each county. However, past research has shown that opioid prescription practices by medical doctors have an impact on opioid deaths;[50] and there is reliable data on prescriptions. As such, we can access reliable estimates of volume of prescriptions and changes in said volume. Perhaps not all, but at least part of the increase in demand for opioids will show up on the increased prescription rates. Results in Tab. 3, Col. 1 are consistent with this theory: an increase in one percentage point in import penetration is associated with an increase of 1.97 yearly prescriptions per 100 residents. On an average county import penetration increase 0.15 percentage points, which mean a predicted prescription increase of (roughly) 0.3 prescriptions a year. That's also a small part of the average annual increase (2.07 prescriptions a year). So, the effect is significant, but only explains

[48]The reason we use overall population rates is that, for 2000, we not not have access to rates for people 18 to 65, which would have been the better estimate. Rates for people under 19 are also available but they are not comparable to rates after 2000 because of changes to the Children's Health Insurance Program (CHIP).

[49]If everyone over the age of 65 has automatic access to health insurance because they are covered by medicare, then no variable should have any impact on the health insurance coverage of people over 65, by construction. Furthermore, consider $Y = Y_1 \frac{n_1}{n} + Y_2 \frac{n_2}{n}$, where n_1 is number of people in group 1 and n_2 is number of people in group 2, and $n = n_1 + n_2$. Then $\beta = (X'X)^{-1} X'Y = (X'X)^{-1} X'Y_1 \frac{n_1}{n} + (X'X)^{-1} X'Y_2 \frac{n_2}{n} = \beta_1 \frac{n_1}{n} + \beta_2 \frac{n_1}{n}$. If the true effect of import penetration on health insurance coverage equals β_1, and the medicare policy sets $\beta_2 = 0$ artificially; it follows that the estimated β is bellow the true effect, that is, $\beta = \beta_1 \frac{n_1}{n} < \beta_1$.

[50]Schnell and Currie (2017) make a convincing argument that prescription practices have an impact on overall addiction levels. Furthermore Fig. 11 shows the growth of overdose from legal opioids .

a small part of the increase in opioid prescriptions (just as it only explains a small part of the increase in mortality, see Tab. 1).

6 The cumulative disadvantage hypothesis

The cumulative disadvantage hypothesis (Case and Deaton, 2017) posits that it is the disappointment of doing worse than one's parents in the labor market that leads people towards depression and death, as opposed to just poor labor market performance in and of itself. This hypothesis is difficult to test,[51] but if true, it could explain why the impact of import penetration differs across race/ethnicity. By looking at labor market effects of import penetration across race/ethnicity and parental income percentile, we preform an indirect test of the cumulative disadvantage hypothesis.

Employment effects

Our goal is to test whether the economic impact of import penetration varies across race/ethnicity and parental income. To do this, we start by testing the impact of import penetration on employment rates across race/ethnicity. If somehow import penetration only affected the employment rates of non-hispanic whites then varying impacts of import penetration on mortality across race/ethnicity would easily be explained away. That is not the case. We study the impact of import penetration on employment rates across race/ethnicity and present our results in Tab. 4. We see that a 1 percentage point increase in import competition (IPO) is associated with a (roughly) 1 percentage point fall in employment rates over an 8-year period.[52] This result is consistent with previous literature (Acemoglu et al., 2016, Table 7). Moreover, the effect of import penetration on employment rates is consistent across racial/ethnic groups.[53] If anything, minorities'

[51]Case and Deaton (2017, p. 430) claim it cannot be tested with data available when they were writing: "The data do not permit an analysis, but the [labor market opportunities] deterioration was likely worse for whites than blacks (...)."

[52]Estimation is based on equation (2), but ΔD_{it} is change in employment rates in this case.

[53]Yearly estimates of employment rates by commuting zone are most accurately estimated using CBP data. However, this precludes estimating employment rates by race/ethnicity since the CBP dataset does not include employees' race/ethnicity. One alternative is the current population survey, but its employment estimates by race/ethnicity are not accurate at the county level because samples are not large enough (not enough minorities interviewed in all counties). As such, we use Census long-form questionnaire (Supplementary File 3) data for in 1990 and 2000; since the long-form census questionnaire was eliminated after 2000, to obtain estimates of employment rates by race/ethnicity $\times$ county after 2000 we use the American Community Survey 5-year composite estimates for employment rates in 2016.

employment rates suffer more from import penetration than non-hispanic whites' (Tab. 4, cols 3-4). A stronger effect for minorities would be consistent with existing literature; several studies argue that in face of negative economic shocks minorities and women are usually the first to bear a larger share of the weight (Kochhar and Fry, 2014). Now that we have shown that the employment rate effects are consistent across groups we can move on to testing the cumulative disadvantage hypothesis per se.

Income effects

As we saw in the background section, at the core of the cumulative disadvantage hypothesis is the idea that, while recent labor market trends have had a negative impact on labor market outcomes for all groups, they have only led to negative intergenerational mobility for whites; this is because, for past generations, labor market outcomes for backs and hispanics are much worse than labor market outcomes for whites. In other words, it is an issue of unequal distribution of paternal income across races/ethnicities. To solve this problem we use data published by Chetty et al. (2018) on the income percentile of children while holding the income percentile of parents constant. Unfortunately, this data is only available in a cross-section, meaning that we will have to move away from estimation in a first-difference setting, as laid out in equation (2), and move into estimation in a cross-section setting, best described by the following equation

$$\begin{aligned}&\mathbb{E}\left[\text{Child income percentile rank}|\text{Parental income percentile rank}\right]_s^g\\&\qquad = \delta^g + \theta^g \Delta IPO_{s,\text{91-14}} + X_{i,91}^g \mu^g + \epsilon_{it} \quad (3)\end{aligned}$$

where the left-hand-side expected value is simply estimated as an average. While lacking panel structure for the chid income data, we still look at changes in import penetration in import penetration in the hopes that it will deliver us, partly, from the endogeneity of inherently high levels of import penetration in some commuting zones.

The results, presented in Tab. 5, can be summed up as follows: Import penetration has a negative impact on the incomes of white non-hispanic male children of high-income parents (75th income percentile), but no impact for minority children of high-income parents. For children of low-income parents (25th income percentile), import penetration has a negative impact on the incomes of all races/ethnicities. Import penetration has no impact on women's incomes, regardless of parental income percentile.

Interpretation

While they do not rule out alternative explanations completely,[54] these results are consistent with the cumulative disadvantage story: male white non-hispanic children of high-income parents who work in manufacturing expect to follow in their parent's footsteps and enjoy the same opportunities. However, import penetration has a downward mobility effect on their income by closing off some of these career avenues. It is important to mention that these are children born between 1978 and 1983; these children are between the ages of 18 and 23 in 2001, when China enters the world trade organization and the growth of import penetration accelerates in the US (Autor et al., 2013). So, for the most part, these children grow up in an environment with little to no import penetration; their labor market expectations form based on a reference group (their parents) with jobs unaffected by import penetration. However, when these children enter the labor market, import penetration is growing fast and the labor market opportunities in manufacturing are deteriorating as a consequence of import penetration. The cumulative disadvantage hypothesis (Case and Deaton, 2017) would claim that it is this disappointment of labor market expectations that explains the growth in deaths of despair.

We cannot say if the effects we find are generation-specific or they could also be present for people born before 1978 or after 1983. As we explained in the previous paragraph this is a specific cohort with very specific characteristics so we cannot really say if these are cohort-specific effects or valid for the whole population. It would be helpful to have data for other cohorts to check how this effect translates. Particularly, to check whether this effect translates into more recent generations, people who grew up aware of import penetration; hopefully, people who grew up in a context where manufacturing jobs were already in decline would make different human capital investments and not suffer from import penetration (or at least not suffer as much).

As for the male children of low-income parents, it is not surprising that they suffer from import penetration across all races/ethnicities: manufacturing work is one of

[54] The evidence we present does not confirm the cumulative disadvantage beyond reasonable doubt, other group-level characteristics could also explain the difference in effect of import penetration on mortality across races/ethnicities. Different levels of trust and support across communities could also explain why some do not fall prey to depression and self-hurt in the face of economic strife (Nunn and Wantchekon, 2011): if some communities systematically help their own when they fall onto hard times, then that would cushion the blow of an economic downturn both economically and psychologically; which could explain the difference in effects of import penetration on income and mortality across races/ethnicities.

the better-paying options for males without a college degree (along with construction, Charles et al. 2016); so, places where import penetration affects manufacturing jobs more, are also places where the incomes of male children of lower income families are lower (by the relative absence of these well-paid jobs).

Shortcomings

One issue with the above stated explanation is that it does not explain the recent increase in minorities' mortality rates. Since 2010, the mortality rates of minorities have been increasing (Fig. 3-6). As we explained in the previous section (section 5), this increase does not seem to be related to import penetration.[55] Just so, the cumulative disadvantage hypothesis does not offer any helping in understanding the recent growth in minorities' mortality rates, either. Is the growth of minorities' mortality rates of a different nature of non-hispanic whites increase? or is it of the same nature, but only delayed? Are the root causes the same as for white non-hispanics? Or are they different? We cannot answer these questions with the data we have. However, we would like to note that minorities' mortality increase does not contradict the cumulative disadvantage hypothesis because we do not know what is behind this increase; economic factors could be part of the cause, but they could also be completely irrelevant.[56]

7 Concluding remarks

In this paper, we studied the IV/reduced-form impact of import penetration on mortality as an indirect way to gain insight on the impact of labor market outcomes on mortality rates. The advantage of using import penetration as an instrument is that its impact on health is arguably exogenous, unlike labor market outcomes. Thusly, this exogenous labor demand shock provides evidence on the importance of labor market outcomes in determining mortality rates at an aggregate level.

Our findings not only confirm the existence of a significant effect of the labor market on mortality, but they also indicate that most of the effect is accounted for by deaths of despair. These suicide and addiction-related deaths comprise only a tiny fraction

[55]It is also possible we are missing the relation because the phenomena is recent, and there simply is not enough data to pick up on a significant coefficient.

[56]For example, we know that mortality rates by specific drug change in 2010, there is a rise in heroin-related deaths starting in 2010, and after 2013 there is an increase in overdose deaths related to Synthetic opioids (Fig. 11). These drug-specific trends could explain minorities' mortality increase after 2010, as opposed to economic factors.

of mortality overall (less than 4%) but represent more than half of the impact that labor market changes have on mortality. Consistent with this story, we find that labor market outcomes affect access to health insurance and drug consumption levels. We also provide evidence supporting the cumulative disadvantage hypothesis: the idea that it is doing poorly in the labor market relative to one's reference group that leads people to suicide and addiction, and not poor performance in and of itself.

To our mind, the most important implication stemming from this paper regards the social consequences of economic shocks and joblessness. A subject which has received considerable attention from both sociologists[57] and epidemiologists. As economists, we often consider work only as a source of income a means to loosen the budget restriction and increase consumption. However, the evidence in this paper, this psychological toll, shows value in working that goes beyond income: having a job provides people with structure, human interaction, daily goals, all things central to human life that, as researchers, we should not overlook.

Understanding the health consequences of poor economic outcomes is extremely important, but also extremely difficult. For ethical reasons, we cannot preform experiments where we randomly assign poor economic outcomes to people and see how their health indicators react. Without access to randomized control trials ,we simply cannot draw definite causal evidence. However imperfect, instrumental variable approaches (similar to the *cohort studies* of epidemiology) are a promising way to address this problem. Future research should look at alternative instruments (other labor market shocks, for example) and try to replicate our results. Using other instruments, future research should also explore effects on minorities specifically. Testing the cumulative disadvantage hypothesis, as well as alternative explanations of why economic outcomes have an impact on mental health could also bring promising advances in this field.

[57]Wilson (1997) is an widely known reference, but Braconnier and Dormagen (2007) and Cherlin (2014) are more recent examples. For economists the interest is much more recent, but a long time coming; if for no other reason, then at least because lives are at stake.

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Tab. 1: Import penetration and mortality, by age group.
[Reduced Form, First-Difference estimates]

Dependent Variable: ΔMortality rate									
Cause of Death:	All-cause			Death of Despair			Other causes		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Group:	All	WNH	Oth	All	WNH	Oth	All	WNH	Oth
Panel A: People in their prime working years (ages 25 to 54)									
ΔIPO_{st}	3.61**	3.19**	5.30	0.84*	1.66***	-5.30	2.77**	1.54	10.60
	(1.41)	(1.42)	(16.48)	(0.47)	(0.48)	(3.93)	(1.29)	(1.32)	(16.04)
Mortality rate (1991-2016)	241	235	256	38.7	43.5	27.3			
Panel B: People at retirement age (65) or older									
ΔIPO_{st}	18.19**	18.07**	68.72**	0.79**	0.67*	-0.82	18.99***	18.74**	69.54**
	(7.23)	(8.21)	(27.33)	(0.37)	(0.39)	(0.85)	(7.16)	(8.10)	(27.33)
Mortality rate (1991-2016)	4710	4848	4018	47	48.5	39.6			
Panel C: People aged 24 or younger									
ΔIPO_{st}	-0.57	-0.47	0.21	0.17	0.10	-0.66	-0.75	-0.56	0.87
	(0.54)	(0.58)	(0.99)	(0.14)	(0.17)	(0.69)	(0.52)	(0.56)	(0.70)
Mortality rate (1991-2016)	71.6	64.4	84.6	7.1	8.4	3.2			
sample size: 9,324 (=3×3,108)									

label: All population, All. White non-hispanics, WNH. All except white non-hispanics, Oth. $\Delta IPO_{s,t}$ is change in import penetration from China to 8 developed countries countries from 1991 to 2014. Mortality rate (1991-2016) refers to average mortality rate for the 1991 to 2016 period.

note: Identification based on equation (2). All models stack 3 consecutive differences; 1991 to 1999, 1999 to 2007, and 2007 to 2016; over 3,108 counties. All models control for share of the working age population employed in the manufacturing sector at start of the difference period. All models include time-period fixed effects. Standard errors (in parentheses) are clustered at the commuting zone level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Tab. 2: Falsification Test: Import penetration and mortality, by age group.
[Reduced Form, First-Difference estimates]

Dependent Variable: ΔMortality rate 1983 to 1991									
Cause of Death:	All-cause			Death of Despair			Other causes		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Group:	All	WNH	Oth	All	WNH	Oth	All	WNH	Oth
Panel A: People in their prime working years (ages 25 to 54)									
ΔIPO_{st} 1991 to 1999	-8.77	-8.07	11.21	0.46	0.31	5.72	-9.23	-8.38	5.49
	(6.42)	(7.87)	(10.78)	(1.50)	(1.40)	(5.67)	(6.33)	(7.65)	(8.97)
Panel B: People at retirement age (65) or older									
ΔIPO_{st} 1991 to 1999	11.85	9.45	-7.30	0.56	0.63	0.27	11.29	8.81	-7.57
	(44.80)	(44.53)	(82.53)	(2.90)	(3.07)	(4.16)	(44.45)	(44.35)	(82.56)
Panel C: People aged 24 or younger									
ΔIPO_{st} 1991 to 1999	-0.45	-0.57	-1.96	0.05	0.08	0.12	-0.50	-0.65	-2.09
	(0.48)	(0.54)	(9.83)	(0.87)	(1.00)	(1.17)	(0.47)	(0.51)	(9.74)
sample size: 3,108									

label: All population, All. White non-hispanics, WNH. All except white non-hispanics, Oth. $\Delta IPO_{s,t}$ is change in import penetration from China to 8 developed countries countries from 1991 to 2014. Mortality rate (1991-2016) refers to average mortality rate for the 1991 to 2016 period.

note: Identification based on equation (2). All models stack 3 consecutive differences; 1991 to 1999, 1999 to 2007, and 2007 to 2016; over 3,108 counties. All models control for share of the working age population employed in the manufacturing sector at start of the difference period. All models include time-period fixed effects. Standard errors (in parentheses) are clustered at the commuting zone level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Tab. 3: Import penetration and intermediate Health variables
[Reduced Form, First-Difference estimates]

	(1)	(2)
Dependent Variable:	ΔOpioid prescription	ΔHealth insurance
$\Delta IPO_{s,\tau}$	1.97***	-0.33***
	(0.75)	(0.08)
Avg. ΔDependent var. (yearly)	2.07	0.09
	(4.98)	(0.29)
Avg. $\Delta IPO_{i,\tau}$ (yearly)	0.15	0.14
	(0.15)	(0.12)
Observations:	3,108	3,106
Period of analysis:	2006-2016	2000-2008

label: Opioid prescription refers to number of opioid prescriptions by medical doctors dispensed per 100 residents; varies by County. Health insurance refers to percentage of the total county population having heath insurance.

note: All models are first difference models as dectibed in equation (2). All models control for share of the employed population working in the manufacturing sector at baseline. Opioid prescription model uses one single difference: 2006 to 2016; data is only available for this period. Health insurance model uses one single difference: 2000 to 2008; data before 2000 is unavailable and data after 2008 not comparable to previous years because of the Affordable Care Act (ACA). * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Tab. 4: Import penetration and employment rates by race/ethnicity
[Reduced Form, First-Difference estimates]

Dependent Variable: Δ Employment rate				
Data source:	CBP	Census/ACS		
	(1)	(2)	(3)	(4)
Group:	All	All	WNH	Oth
ΔIPO_{st}	-0.82***	-0.91***	-0.93***	-1.08***
	(0.21)	(0.15)	(0.16)	(0.23)
Observations:	722	3,107	3,107	3,105
Period of analysis:	1999 to 2016	2000 to 2016		

label: Employment rate, employment to working age population ratio. County Business Patterns, CBP. American Community Survey, ACS. All population, All. White non-hispanics, WNH. All except white non-hispanics, Oth. ΔIPO_{st} is change in import penetration of Chinese exports into 8 developed countries, in commuting zone s, during period $t-1$ to t.

Dependent variable: Change in employment rate in commuting zone (CBP data) and county (Census/ACS data)

note: Identification based on equation (2). All models calculate impact of import penetration on share of population employed using one single difference, 1999 to 2016. All models control for share of the working age population employed in the manufacturing sector in 1999. All models include time-period fixed effects. Standard errors (in parentheses) are clustered at the commuting zone level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Tab. 5: Import penetration and income by race/ethnicity × parental income percentile
[Reduced Form, Cross-Section estimates]

Dependent Variable: Mean child income rank in 2014-2015 by commuting zone

Parent's income:		25th percentile			75th percentile		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Group:	All	BNH	WNH	His	BNH	WNH	His
Panel A: Data pooled across sex							
ΔIPO_{st}	-0.33**	-0.13	-0.39***	[NA]	[NA]	[NA]	[NA]
	(0.13)	(0.11)	(0.12)				
sample size	[722]	[610]	[722]				
Panel B: Results for men							
ΔIPO_{st}	[NA]	-0.42***	-0.57***	-0.47***	0.11	-0.47***	-0.10
		(0.15)	(0.12)	(0.16)	(0.21)	(0.09)	(0.25)
sample size		[550]	[722]	[670]	[550]	[722]	[670]
Panel C: Results for women							
ΔIPO_{st}	[NA]	0.07	-0.08	-0.03	0.31	0.07	-0.30
		(0.12)	(0.09)	(0.14)	(0.23)	(0.08)	(0.22)
sample size		[555]	[722]	[676]	[555]	[722]	[676]

label: All population, All. White non-hispanics, WNH. Black non-hispanics, BNH. ΔIPO_{st} is change in import penetration from China to 8 developed countries countries from 1991 to 2014. NA stands for Not Available; data was published in table form by Chetty et al. (2018), they did not publish everything. For example, we do not have data for Hispanics pooled across sexes, and since we do not have population data by sex we cannot fill in the blanks either.

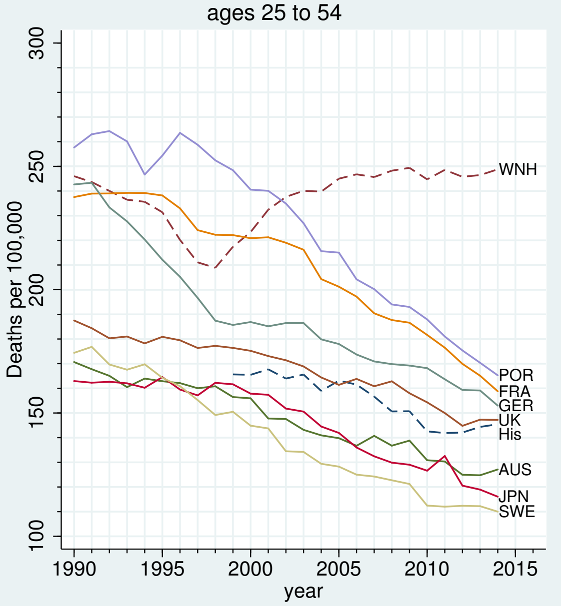
data: Sample consists of children born between 1978 and 1983 and their parents. Children are assigned to commuting zones based on the first non-missing zip code of their parents, irrespective of where they live as adults. Child income is calculated as mean individual income in 2014-15, when children are in their mid-thirties. Parent income is measured as mean household income between 1994 and 2000, when their children are between the ages of 11 and 22. Parental income percentile is calculated among parents born in the same cohort.

sample: Areas with fewer than 20 children in the core sample, for which we have inadequate data to estimate mobility, or fewer than 500 residents of the children's racial group in the 2000 Census are excluded.

note: All models regress children income percentile rank against change in import penetration according to equation (3). Heteroskedasticity-robust standard errors in parenthesis. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Fig. 1: All–cause mortality

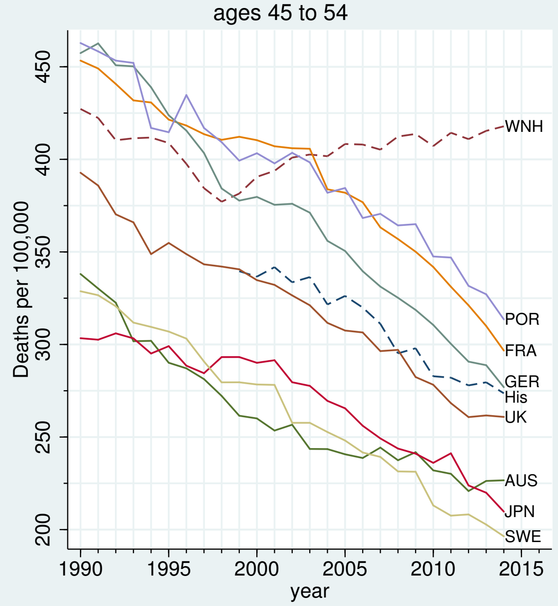
ages 25 to 54



lab: Australia, AUS. Canada, CAN. France, FRA. Germany, GER. Japan, JPN. Portugal, POR. Sweden, SWE. United Kingdon, UK. United States white non–hispanics, WNH. United States hispanics, His.
note: Mortality rates by group–country over time. This graph is a reproduction of Fig. 1 in Case and Deaton (2015), expanded to ages 25 to 54.
source: Author's calculations using Human Mortality Database.

Fig. 2: All–cause mortality

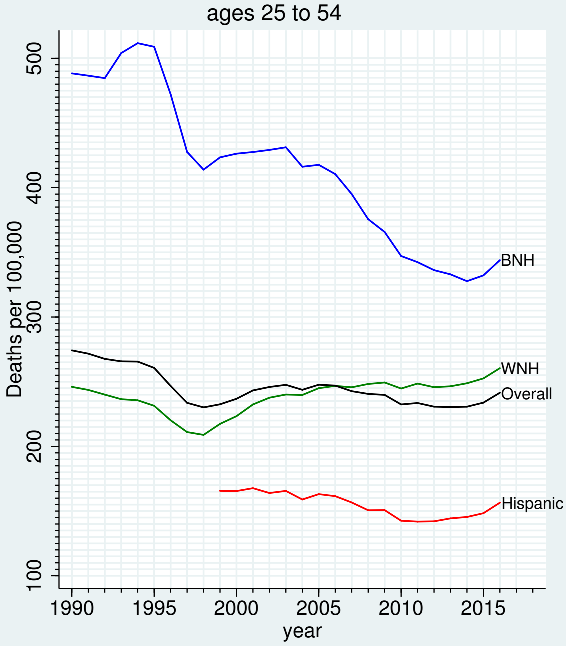
ages 45 to 54



lab: Australia, AUS. Canada, CAN. France, FRA. Germany, GER. Japan, JPN. Portugal, POR. Sweden, SWE. United Kingdon, UK. United States white non–hispanics, WNH. United States hispanics, His.
note: Mortality rates by group–country over time. This graph is a reproduction of Fig. 1 in Case and Deaton (2015).
source: Author's calculations using Human Mortality Database.

Fig. 3: All–cause Mortality by race

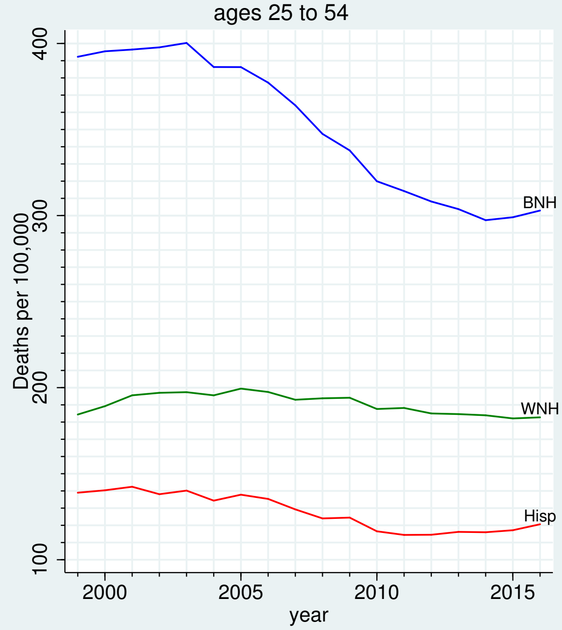
ages 25 to 54



lab: White non–hispanics, WNH. Black non–hispanics, BNH.
note: Mortality rates by race/ethnic group, all causes of death.
source: Author's calculations using the CDC WONDER mortality database.

Fig. 4: Death by any cause except despair

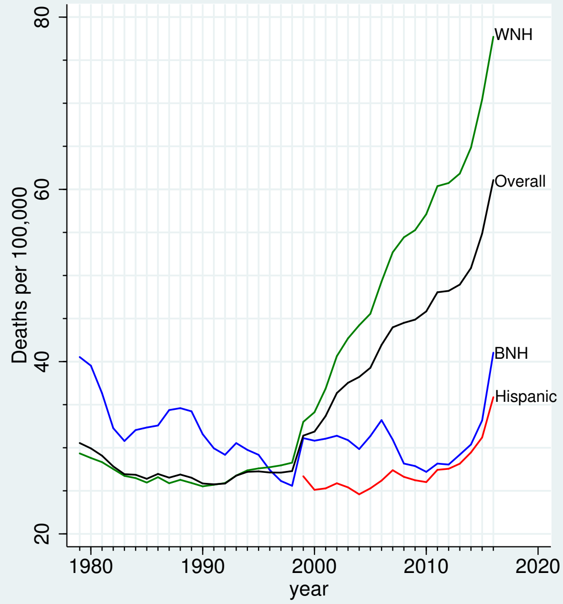
ages 25 to 54



lab: White non–hispanics, WNH. Black non–hispanics, BNH.
note: Yearly mortality rates from any cause except deaths by suicide, and/or addiction; by race/ethnic group.
source: Author's calculations using the WONDER Mortality Database.

Fig. 5: Deaths of Despair

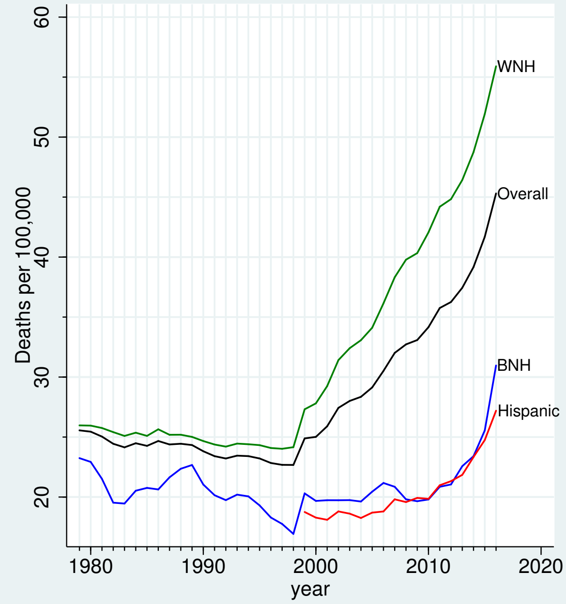
ages 25 to 54



lab: White non−hispanics, WNH. Black non−hispanics, BNH.
note: Deaths of Despair mortality rates by race/ethnic group. Deaths of Despair are deaths by suicide, and/or addiction.
source: Author's calculations using the WONDER Mortality Database.

Fig. 6: Deaths of Despair

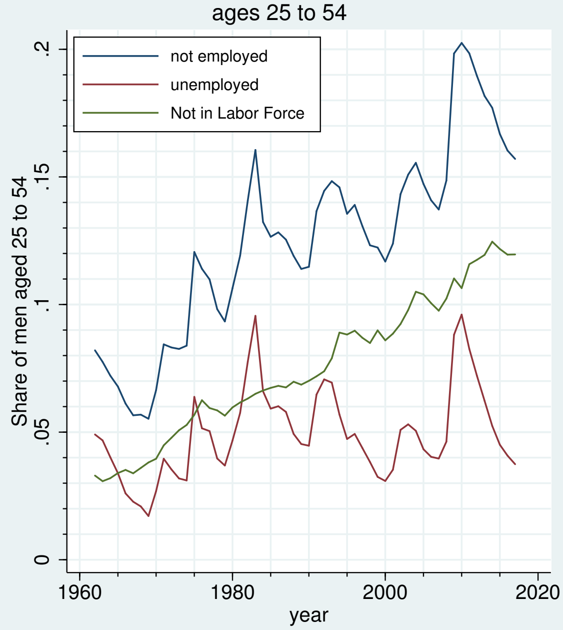
all ages



lab: White non−hispanics, WNH. Black non−hispanics, BNH.
note: Deaths of Despair mortality rates by race/ethnic group. Deaths of Despair are deaths by suicide, and/or addiction.
source: Author's calculations using the WONDER Mortality Database.

Fig. 7: non−employment of men

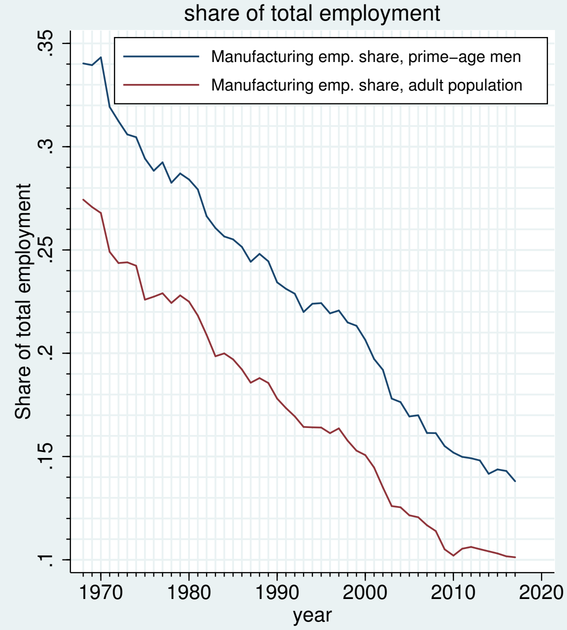
ages 25 to 54



note: Rates of prime−age male respondents from the Annual Social and Economic Supplement (ASEC) by labor market status. Calculated using the ASEC supplement survey weights. Not in labor force refers to prime age men who are neither employed nor actively looking for a job

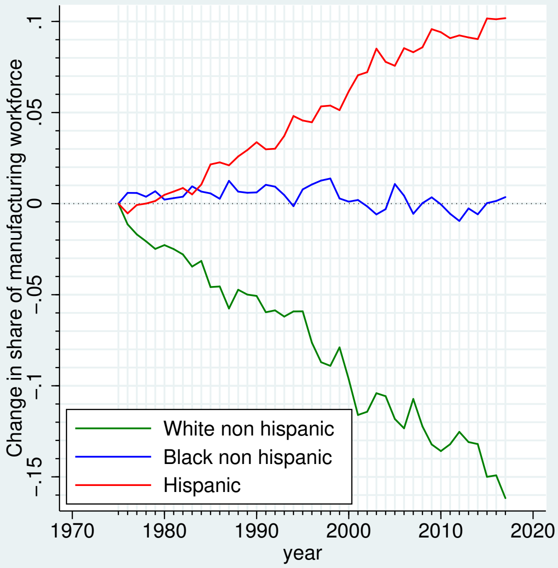
Fig. 8: Manufacturing employment

share of total employment



note: Rates of respondents to the Annual Social and Economic Supplement (ASEC) from the Current population survey/Survey of American Communities; using ASEC−specific weights.
Respondents employed in manufacturing/respondents employed, weighted.

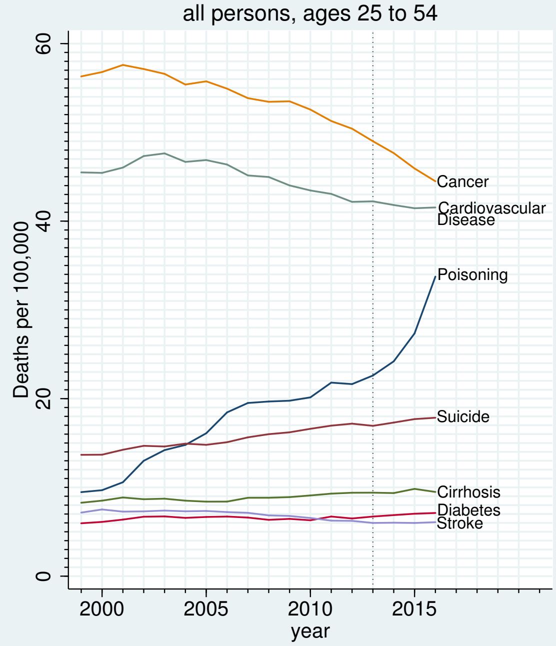
Fig 9: Change in racial/ethnic makeup of U.S. manufacturing workforce.



note: Change in share of the US manufacturing workforce makeup by racial/ethnic group. In 1975 the US manufacturing workforce was 81% white non−hispanic, 6% hispanic, and 10% black non−hispanic. In 2017 it is 65% white non−hispanics, 16% hispanic, and 10% black non−hispanics. The share held by non−hispanic whites fell by 16 percentage points, and the share held by Hispanics increased by 10 percent−age points. This change is what the graph represents.
source: author's calculations using IPMUS−CPS data

Fig. 10: Mortality by cause

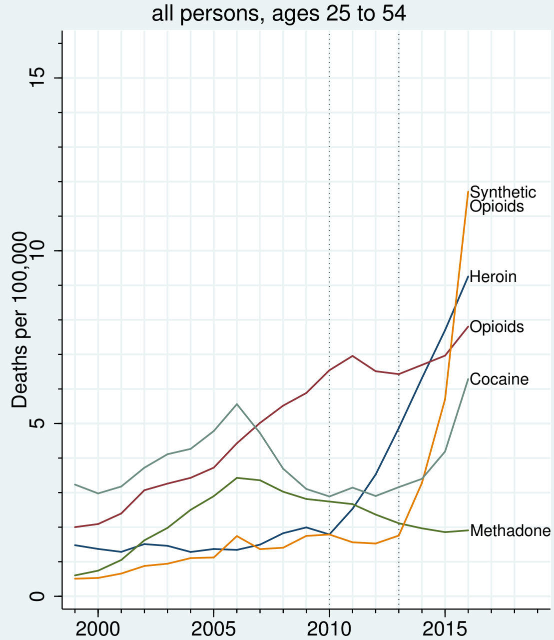
all persons, ages 25 to 54



note: ?Cardiovascular disease? references all vascular disease except stroke (ICD I00−51 and I70−99).
source: Author's calculations using the WONDER Mortality Database.

Fig. 11: poisoning mortality by drug.

all persons, ages 25 to 54



note: Opioids refers to all except Heroin, Methadone, Synthetic, and Cocaine; refers mostly to legal opioids like OxyCodone.
source: Author's calculations using the WONDER Mortality Database.

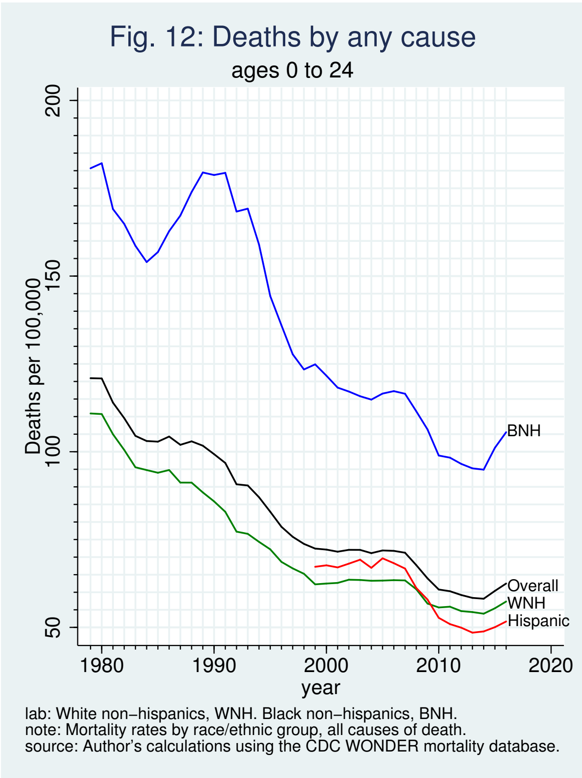


Fig. 12: Deaths by any cause
ages 0 to 24
Deaths per 100,000
200
150
100
50
1980
1990
2000
2010
2020
year
BNH
Overall
WNH
Hispanic
lab: White non−hispanics, WNH. Black non−hispanics, BNH.
note: Mortality rates by race/ethnic group, all causes of death.
source: Author's calculations using the CDC WONDER mortality database.

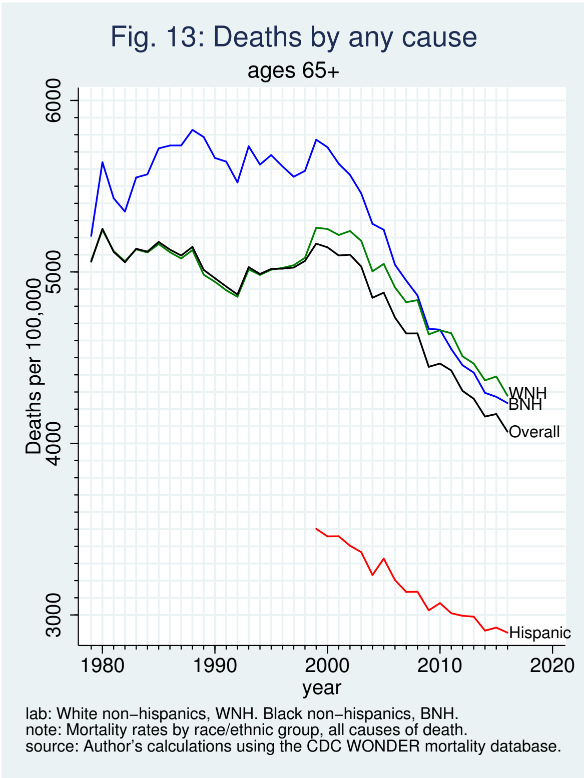


Fig. 13: Deaths by any cause
ages 65+
Deaths per 100,000
6000
5000
4000
3000
1980
1990
2000
2010
2020
year
WNH
BNH
Overall
Hispanic
lab: White non−hispanics, WNH. Black non−hispanics, BNH.
note: Mortality rates by race/ethnic group, all causes of death.
source: Author's calculations using the CDC WONDER mortality database.

Tab. A1: Descriptive statistics

	year	mean	sd	p5	p10	p25	p50	p75	p90	p95
Panel A: $\Delta Death\ rate$ from any cause for people aged 25 to 54										
All racial/ethnic groups										
D_{rate}	2016	186.35	159.68	0.00	26.32	57.11	138.30	293.23	405.91	480.44
D_{rate}	1991	135.45	137.90	0.00	0.00	19.75	73.86	233.15	319.46	378.17
Minorities (average)										
D_{rate}	2016	149.40	280.65	0.00	0.00	0.00	49.32	223.86	400.99	551.10
D_{rate}	1991	220.45	1218.50	0.00	0.00	0.00	0.00	230.68	520.16	719.42
Non-hispanic whites										
D_{rate}	2016	192.83	167.26	0.00	19.86	63.21	142.00	299.50	420.51	498.91
D_{rate}	1991	124.99	124.68	0.00	0.00	19.05	68.68	218.69	288.09	335.22
Panel B: $\Delta Death\ rate$ from deaths of despair for people aged 25 to 54										
All racial/ethnic groups										
D_{rate}	2016	63.91	49.79	0.00	0.00	35.15	58.90	85.76	117.19	147.46
D_{rate}	1991	22.56	21.65	0.00	0.00	4.96	20.42	31.79	46.34	59.21
Minorities (average)										
D_{rate}	2016	43.76	105.09	0.00	0.00	0.00	0.00	47.75	107.57	187.97
D_{rate}	1991	35.35	633.67	0.00	0.00	0.00	0.00	0.00	43.88	82.64
Non-hispanic whites										
D_{rate}	2016	68.93	50.08	0.00	0.00	36.95	65.92	96.07	130.61	155.86
D_{rate}	1991	22.22	21.95	0.00	0.00	0.00	20.13	32.61	46.86	59.41
Panel C: Economic variables										
Import penetration										
$\Delta IPO_{s,1991to2016}$		2.51	2.51	0.13	0.41	1.09	2.07	3.52	5.75	7.06
IPO_s^{CZ}	1991	0.31	0.40	0.01	0.03	0.08	0.15	0.38	0.83	1.10
IPO_s^{CZ}	1999	0.76	0.87	0.03	0.09	0.24	0.44	0.99	1.87	2.46
IPO_s^{CZ}	2007	2.00	1.84	0.07	0.31	0.81	1.46	2.70	4.40	5.80
Employment rate										
D_{rate}	1991	44.96	10.22	27.91	31.78	38.10	44.99	52.02	58.83	61.76
D_{rate}	1999	51.05	11.34	32.23	36.76	42.77	50.79	59.10	66.30	70.03
D_{rate}	2007	50.74	10.73	34.45	36.89	42.94	51.03	58.10	64.63	67.63
Share of the workforce employed in manufacturing										
Share	1991	0.23	0.12	0.04	0.08	0.14	0.22	0.30	0.39	0.44
Share	1999	0.20	0.10	0.04	0.07	0.12	0.19	0.27	0.33	0.38
Share	2007	0.16	0.09	0.04	0.06	0.09	0.14	0.22	0.27	0.31

Tab. A2: Share of men aged 25 to 54 employed by state

	AL	AK	AZ	AR	CA	CO	CT	DE	DC	FL	GA	HI	ID	IL	IN	IA	KS	KY	LA	ME	MD	MA	MI	MN	MS	MO
1962	92		89	87	93	96	95	91	89	91	91			93	96	93	97	89	90		89	94	90	94	89	94
1963	86	64	91	89	93	93	94	89	81	89	95	95	91	94	96	96	98	86	86	95	91	94	94	94	92	93
1964	91	67	93	84	91	95	93	91	98	92	90	94	97	94	96	94	98	90	91	95	93	93	95	92	91	93
1965	93	93	89	86	91	96	94	98	92	94	93	100	95	96	94	93	97	84	89	91	96	92	96	94	91	92
1966	92	89	90	91	94	93	97	98	94	93	92	95	96	97	96	95	95	88	85	92	95	95	95	95	92	95
1967	89	94	93	97	93	97	97	99	92	92	93	98	97	97	97	96	95	92	93	92	95	95	94	92	92	93
1968					94		94		84	94	94			95	96			92	90		96					94
1969					93		96		89	95	95			95	97			89	91		96					96
1970					91		97		92	94	94			97	95			88	88		94					94
1971					89		87		81	90	94			93	93			88	91		93					93
1972					89		95		86	93	95			94	93			85	89		92					93
1973					89		92		89	95				94	92							91				
1974					89		92		86	89				93	94							91				
1975					87		88		75	86				90	88							88				
1976					87		87		84	88				90	91							87				
1977	87	86	90	91	88	89	90	92	81	86	90	91	88	91	91	93	92	89	86	85	92	89	89	92	90	90
1978	89	82	85	91	90	91	92	93	81	89	92	91	91	91	90	94	94	89	90	88	93	90	90	92	89	91
1979	90	79	92	91	91	93	93	93	89	91	91	88	90	90	94	94	95	88	87	87	92	88	90	93	89	91
1980	87	81	92	87	90	94	93	90	84	89	92	92	87	88	88	94	94	89	86	86	91	90	85	91	89	91
1981	86	80	88	83	87	95	94	90	80	90	91	92	88	87	88	91	93	86	87	84	90	89	83	92	88	86
1982	84	77	86	83	84	91	93	89	79	86	90	85	84	87	82	83	90	84	85	86	87	89	81	89	86	87
1983	80	78	84	83	84	86	92	88	81	84	89	85	83	85	82	85	86	81	79	83	85	87	78	85	81	83
1984	84	80	91	86	87	92	94	89	79	90	88	89	82	86	86	85	89	81	83	87	90	90	82	90	82	87
1985	86	82	92	86	87	91	93	89	82	89	91	85	86	85	86	85	92	84	82	89	92	91	82	91	84	87
1986	86	81	88	87	87	91	91	90	83	88	91	89	86	86	90	87	90	81	87	86	93	90	82	90	82	89
1987	87	76	86	84	88	86	94	89	82	91	89	86	81	86	87	91	92	82	82	90	93	91	85	89	85	88
1988	86	81	84	85	88	87	95	92	87	89	90	91	88	86	85	93	90	79	80	91	94	91	82	89	89	89
1989	87	80	87	84	89	87	94	92	85	89	88	89	89	89	91	96	93	84	84	89	91	90	86	89	87	86
1990	88	83	92	85	88	87	89	90	83	90	89	88	90	89	90	93	90	86	83	87	91	88	85	87	85	88
1991	80	81	90	84	85	90	92	87	75	87	86	93	89	87	86	91	93	82	82	82	90	82	81	90	83	86
1992	82	82	88	85	84	90	88	89	79	85	84	91	84	85	87	90	94	84	85	84	87	80	82	88	84	86
1993	84	84	87	87	82	85	89	87	77	87	86	89	86	83	89	91	90	82	85	81	88	86	85	91	89	84
1994	83	80	86	84	83	89	88	89	74	87	87	84	91	87	87	90	87	82	80	82	87	84	83	90	86	84
1995	84	82	91	88	83	89	88	90	78	87	87	85	83	89	89	89	87	82	82	83	89	85	84	91	87	84
1996	80	82	90	84	83	91	86	88	75	87	85	84	85	86	89	93	91	81	81	87	87	82	89	91	81	89
1997	85	81	88	83	85	92	85	87	78	86	87	80	87	87	89	95	95	86	84	85	89	86	88	92	86	87
1998	86	84	88	82	85	92	87	91	76	86	89	84	90	89	90	94	92	85	84	85	90	85	88	94	84	87
1999	91	79	90	87	86	92	83	89	79	87	90	85	89	90	92	91	93	83	82	85	87	87	87	91	81	89
2000	89	82	88	84	87	89	90	88	85	89	89	87	90	89	90	94	89	86	82	87	94	90	90	91	84	88
2001	87	82	89	85	87	90	88	90	79	88	87	87	89	86	89	92	89	85	83	88	93	87	86	92	86	89
2002	81	83	89	86	85	90	88	87	82	86	83	84	88	84	87	89	88	82	84	85	91	89	84	89	85	87
2003	83	83	86	82	86	89	84	85	80	87	86	86	87	83	86	89	87	82	82	83	88	84	80	89	77	88
2004	83	81	86	82	84	88	83	85	81	87	84	86	88	83	85	91	89	83	82	81	88	84	79	86	82	85
2005	83	84	89	84	85	91	85	85	81	87	84	85	89	85	85	89	88	78	82	82	86	82	84	89	80	83
2006	80	80	89	84	85	92	86	88	77	88	87	86	88	86	87	91	88	79	81	84	89	83	81	89	76	85
2007	83	82	87	84	86	91	88	84	84	89	88	88	91	87	85	89	89	80	84	83	88	83	81	88	81	86
2008	83	84	84	84	84	90	85	88	83	85	87	90	90	85	83	88	88	74	80	84	87	83	81	87	82	85
2009	75	79	77	82	79	85	83	82	81	77	81	81	86	79	79	86	87	77	77	80	84	79	74	83	79	78
2010	72	77	77	79	80	84	84	81	77	78	79	83	84	79	77	86	83	73	81	78	84	80	73	84	70	77
2011	77	81	77	78	79	83	84	84	84	79	77	80	81	80	79	87	85	77	73	78	84	81	75	85	73	79
2012	76	81	81	80	79	85	84	82	77	79	80	81	84	79	81	86	83	80	78	81	85	80	77	85	78	78
2013	76	81	80	79	82	87	81	83	84	81	80	81	85	80	78	88	88	78	72	81	84	86	80	86	78	81
2014	81	82	80	80	81	88	80	83	82	82	85	81	85	81	85	86	86	75	80	78	84	85	79	86	72	81
2015	82	79	78	78	83	89	84	85	81	81	84	83	88	83	84	89	88	78	80	77	86	85	80	89	77	87
2016	74	81	85	85	83	87	87	87	84	82	83	85	86	87	87	88	88	78	78	82	86	84	82	87	73	88
2017	80	81	87	77	83	93	87	82	85	86	82	87	85	85	87	88	85	80	76	85	87	85	83	87	78	85

label: Alabama, AL. Alaska, AK. Arizona, AZ. Arkansas, AR. California, CA. Colorado, CO. Connecticut, CT. Delaware, DE. Florida, FL. Georgia, GA. Hawaii, HI. Idaho, ID. Illinois, IL. Indiana, IN. Iowa, IA. Kansas, KS. Kentucky, KY. Louisiana, LA. Maine, ME. Maryland, MD. Massachusetts, MA. Michigan, MI. Washington, WA. Minnesota, MN West Virginia, WV. Mississippi, MS. Wisconsin, WI. Wyoming, WY.
note: Estimated employment rate for each of the 50 states (plus the District of Columbia) by year.
source: Author's calculations using IPMUS-CEP survey data.

Tab. A3: Share of men aged 25 to 54 employed by state

	MT	NE	NV	NH	NJ	NM	NY	NC	ND	OH	OK	OR	PA	RI	SC	SD	TN	TX	UT	VT	VA	WA	WV	WI	WY
1962			88		94	96	92	89		90	91	86	90	92	92		90	92	96		93	92	79	94	
1963	98	94	89	85	94	90	93	90	96	92	91	87	90	93	94	89	91	93	93	100	95	94	83	93	90
1964	97	98	93	92	96	91	92	92	96	93	95	86	91	97	94	98	93	93	97	92	94	93	86	97	71
1965	91	95	94	89	94	97	92	95	92	94	95	93	94	94	97	95	91	94	93	100	94	91	83	95	90
1966	88	98	88	97	96	94	94	94	94	96	92	94	93	96	93	96	92	94	95	97	95	92	79	97	98
1967	92	96	86	97	94	89	95	93	93	96	94	96	95	100	97	94	92	95	94	90	92	94	86	97	100
1968					95		94			96		92	94				91	97					90		
1969					95		94			95		94	95				91	96					80		
1970					96		94			93		91	94				90	95					79		
1971					93		90			93		87	93				90	93					84		
1972					92		90			93		90	91				91	94					85		
1973					93		90	93		94			91					94							
1974					94		89	93		94			92					93							
1975					88		87	86		89			87					92							
1976					88		87	89		89			86					92							
1977	93	94	90	90	89	85	86	90	89	90	93	83	88	86	88	89	87	91	91	85	92	85	82	92	97
1978	90	96	96	95	90	87	87	93	91	90	92	91	89	87	93	90	88	92	93	89	91	87	86	94	95
1979	91	93	89	93	89	91	89	91	93	92	93	89	89	92	91	91	89	93	94	93	91	92	82	94	97
1980	89	95	93	95	91	86	88	90	93	88	90	86	88	89	88	93	85	92	91	90	92	91	83	93	95
1981	89	93	92	91	90	83	87	88	87	86	93	84	87	88	85	90	84	93	92	87	91	84	77	89	95
1982	87	91	87	89	89	82	87	87	88	82	89	81	84	83	84	88	81	92	90	89	89	82	82	84	91
1983	85	91	87	90	88	81	85	84	88	81	86	81	79	84	82	88	82	89	87	88	90	84	69	80	85
1984	83	93	88	93	90	83	86	89	92	84	90	83	82	89	88	89	81	90	90	86	91	87	72	89	86
1985	89	92	86	92	91	84	85	89	88	86	90	84	86	88	89	88	85	89	90	87	91	84	77	89	86
1986	85	93	92	92	92	84	86	89	86	86	83	82	84	91	90	89	84	86	91	90	90	86	75	86	87
1987	81	91	90	94	92	83	89	89	89	86	86	84	86	90	88	92	86	84	91	92	91	86	79	86	88
1988	83	91	90	94	93	80	89	90	89	86	87	87	90	90	90	93	87	88	91	90	92	88	80	89	86
1989	85	92	87	96	92	80	86	91	88	86	85	86	90	90	89	92	86	89	91	88	92	89	82	92	89
1990	88	95	90	91	91	84	88	91	89	89	89	90	88	87	90	91	86	89	91	91	92	89	81	91	92
1991	84	94	90	89	88	83	85	87	88	87	86	91	86	85	86	88	85	89	90	87	90	85	77	87	90
1992	86	94	88	86	88	83	83	89	88	86	85	86	85	84	85	91	84	88	90	86	89	86	76	89	89
1993	87	92	85	84	84	82	83	88	89	86	85	84	84	82	83	89	81	86	92	85	91	86	73	92	87
1994	87	95	88	88	87	81	81	88	91	85	85	87	85	84	85	91	85	88	91	85	89	85	74	90	91
1995	87	94	88	89	87	82	83	89	90	87	84	89	86	81	86	91	84	88	95	89	92	88	78	89	91
1996	84	92	90	89	88	78	82	89	94	87	88	89	85	89	88	92	87	89	92	88	88	83	77	92	92
1997	82	92	92	91	88	81	83	91	92	88	89	88	85	81	92	89	82	88	94	87	89	88	76	91	88
1998	85	93	92	90	88	81	85	89	90	87	87	91	87	88	91	87	87	90	91	91	92	88	77	90	89
1999	85	92	89	92	88	85	84	90	88	88	88	90	87	86	89	91	86	89	96	90	92	87	81	87	89
2000	87	92	92	92	90	85	86	90	90	87	87	90	88	86	90	92	88	88	92	89	89	87	78	90	89
2001	85	93	87	92	89	84	84	88	90	89	89	88	87	85	86	90	85	88	94	90	91	88	80	90	91
2002	83	90	89	90	86	84	82	84	90	86	89	83	85	87	84	93	84	88	90	89	89	84	75	86	90
2003	83	90	87	91	86	83	82	85	86	85	85	82	85	85	83	91	83	85	86	86	88	83	76	86	90
2004	82	90	88	89	88	83	81	85	89	86	86	83	84	82	83	88	83	85	88	88	85	83	74	87	90
2005	86	91	92	93	91	84	82	87	89	85	87	83	85	85	83	87	82	86	91	88	88	84	73	87	90
2006	85	88	88	90	88	81	83	87	92	84	83	90	86	83	84	92	84	87	93	86	91	89	79	87	90
2007	89	89	88	89	88	81	84	87	90	84	81	85	86	88	86	91	86	88	94	86	89	90	78	86	94
2008	85	90	88	89	86	82	84	85	91	86	85	82	86	85	85	92	81	87	91	84	88	87	79	88	91
2009	82	86	84	85	82	73	81	80	89	78	85	74	81	81	75	86	77	84	86	86	84	81	71	81	87
2010	80	88	75	85	83	73	79	81	89	77	83	80	81	78	78	84	79	83	82	82	82	81	73	78	87
2011	80	88	79	88	81	75	79	82	89	79	83	79	80	77	79	88	81	84	85	83	83	81	70	85	89
2012	79	91	78	88	81	74	80	81	90	81	85	81	84	81	77	87	79	85	88	84	83	82	74	83	85
2013	82	89	80	88	85	78	80	82	89	76	83	81	81	80	77	89	81	85	90	85	86	83	74	84	87
2014	86	92	83	87	86	79	79	82	94	78	80	80	81	79	78	90	84	87	87	84	86	81	68	88	88
2015	80	90	85	88	82	79	82	85	89	81	84	86	82	80	81	87	80	86	89	83	87	84	71	87	88
2016	82	88	83	90	87	79	82	85	86	83	81	87	84	81	81	88	83	86	89	85	83	86	72	88	86
2017	84	91	84	89	89	77	82	82	89	82	83	86	84	84	80	88	82	86	89	89	85	85	73	89	86

label: Alabama, AL. Alaska, AK. Arizona, AZ. Arkansas, AR. California, CA. Colorado, CO. Connecticut, CT. Delaware, DE. Florida, FL. Georgia, GA. Hawaii, HI. Idaho, ID. Illinois, IL. Indiana, IN. Iowa, IA. Kansas, KS. Kentucky, KY. Louisiana, LA. Maine, ME. Maryland, MD. Massachusetts, MA. Michigan, MI. Washington, WA. Minnesota, MN West Virginia, WV. Mississippi, MS. Wisconsin, WI. Wyoming, WY.
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source: Author's calculations using IPMUS-CEP survey data.