

# The Drug Crisis and the Living Arrangements of Children

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## Abstract

We examine the impact of the drug crisis that has unfolded over the last three decades in the United States on children's living arrangements and environments. Because the current living arrangement could be a result of events that occurred at any point in a child's life, we measure children's exposure to the crisis with the cumulative drug-related mortality of likely parents. A potential omitted variables bias complicates the analysis, as the factors that may have led parents to abuse drugs could also have altered the living arrangements of their children. Within a 2SLS framework, we instrument for the cumulative mortality of likely parents with a child's years of exposure to a non-triplicate prescription pad environment. Previous work by Alpert et al. (2019) demonstrates that pharmaceutical advertising was much more extensive in non-triplicate pad states and fostered the development of the drug crisis. Our results indicate that OLS and 2SLS estimates are nearly identical and the crisis increased both the fraction of children living away from a parent and in a household headed by a grandparent. We estimate that if drug abuse had remained at 1996 levels, 1.5 million fewer children aged 0-16 would have lived away from a parent in 2015.

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## I. Introduction

Over the last three decades, the United States has been experiencing a devastating drug abuse crisis. Drug deaths increased every year from 1987 through 2017 with more than 70,000 people dying from drug poisonings in 2017 alone. At its peak, this crisis was more severe than other recent public health crises such as the HIV epidemic (41,669 deaths in 1996) or the violence brought on by the crack epidemic (24,530 homicides in 1992).<sup>1</sup> Between 1999 and 2017, over 702,000 people died of drug poisonings—more than the number of US soldiers that died in all armed conflicts from the Spanish-American War in 1898 through the present day (about 627,000).<sup>2</sup> Fortunately, drug deaths declined in 2018, but there were still nearly 70,000 deaths in this year. As the drug crisis has developed, a growing line of work has documented the impact of the epidemic on employment (Aliprantis and Schweitzer, 2018; Harris et al., forthcoming; Currie et al., 2019), crime (Doleac and Mukherjee, 2018; Szalavitz and Rigg, 2017; Dave et al., 2020), infant health (Ziedan and Kaestner, 2020), enrollment in disability insurance programs (Cutler et al., 2017), and intimate partner violence (Stone and Rothman, 2019).<sup>3</sup>

An important feature of the crisis is that it has primarily impacted people in early adulthood through mid-life; as Figure 1 shows, the drug death rate between 1999 and 2017 was highest for those between the ages of 25 and 54. This level of death and drug use among people in this age range is likely to affect a large number of children, as many will be parents with children in the home. In this paper, we examine how this drug crisis among adults has affected the living arrangements of children. Drug use could separate a child from one or both parents through several pathways. First, a parent who is using drugs could have left the household voluntarily, or been asked to leave by a family member. The parent could be absent due to enrollment in a substance abuse treatment program, or due to incarceration because of a crime related to the abuse. If drug use results in abuse or neglect of the child, the child welfare system could become involved, increasing the chance of a court-ordered placement with another relative or a foster family. The child could also move into the home of a family friend or relative to protect him or her from the

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<sup>1</sup> Author's calculations from the Multiple Cause of Death data files.

<sup>2</sup> <https://fas.org/sgp/crs/natsec/RL32492.pdf>

<sup>3</sup> A separate literature has examined the causes of the epidemic. Several papers focus on demand-based explanations such as the decline in economic opportunities and institutions (Case and Deaton, 2015 and 2017), dislocation generated by trade (Pierce and Schott, 2020), or the expansion of health insurance (Wettstein, 2019; Averett et al., 2019). Another set of papers argues the epidemic has been generated by a series of supply shocks such as the advertising practices of pharmaceutical firms (Alpert et al., 2019), physician's prescribing behavior (Currie and Schwandt, 2020), or the reformulation of prescription opioids (Alpert et al., 2018; Evans et al., 2019).

consequences of the parent’s use. In addition, the parent of the child could have been one of the hundreds of thousands of people who have died because of the crisis.

There is abundant anecdotal evidence in the popular press that suggests that many children are indeed experiencing these kinds of events. A 2016 article in the *Wall Street Journal* reports that “Social workers say the scale of the trouble exceeds anything they saw during the crack-cocaine or methamphetamine crises of previous decades,” and quotes a child welfare worker who warns that “Honestly, if something doesn't happen with this addiction crisis, we can lose a generation of kids” (Whalen, 2016). Outlets including *Vox*, NBC, PBS, CNN, and the Associated Press have noted that grandparents are increasingly assuming parenting responsibilities because of the opioid crisis—especially in states that have been hard-hit, including Florida, Ohio, Pennsylvania, and West Virginia.<sup>4</sup> National statistics support the observations in these stories—there is a long-term, secular upward trend in children either living away from a parent or living in a household headed by a grandparent. Between 1980 and 2018, the fraction of children living without a mother or a father in the household increased from 4.9 to 8.3 percent, and 19.9 to 25.5 percent, respectively. The fraction of children living in a household headed by a grandparent more than doubled from 3.7 to 8.3 percent.<sup>5</sup>

In the last few years, researchers in the social sciences have worked to document whether the drug crisis has increased the movement of children into foster care. Meinhofer and Angler-Diaz (2019) note that between 2012 and 2017, the number of children living in and entering foster care in the U.S. increased by 12 and 8 percent, respectively. Between 2000 and 2015, the fraction of removals that were drug-related more than doubled, from 14.5 percent to 35.3 percent. Radel et al. (2018) correlates foster care entries at the county level in a cross section with opioid poisoning death rates and finds a strong correlation. Quast (2018) examines whether per capita drug prescriptions at the county level are correlated with child removals for drug use by the state, over time. He found the correlation was positive and statistically significant in 23 states, negative and statistically significant in 15 states, and statistically insignificant in 12 states. Quast et al. (2018) and Quast et al. (2019) perform a similar analysis for Florida and California, respectively.

Our paper contributes to this nascent literature in two key ways. First, as we describe in detail below, previous estimates of the relationship between the severity of the crisis and child living

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<sup>4</sup> We list some citations for the popular press articles in Appendix C.

<sup>5</sup> Data are from the 1-percent Census Public Use Micro Samples from the 1980-2000 Census and the 2010 and 2018 American Community Surveys. We use the IPUMS version of these data (Ruggles et al., 2020).

arrangements potentially suffer from both omitted variables bias and reverse causality. Changes in the economy or in the strength of institutions (for example) could affect both drug abuse rates and child living arrangements, while a separation from one's children could increase either the motivation for using drugs or the number of opportunities to do so. To overcome these challenges, we use an instrumental variables strategy that exploits variation across states and over time in children's exposure to triplicate prescription laws; Alpert et al. (2019) show that because the producers of the most frequently abused prescription opioid marketed the drug less aggressively in states with these laws, the drug crisis was less severe. Second, while the previous literature has focused on the effects of the crisis on foster care admissions, we consider its effects on parental absence and on the probability that a child lives in a household headed by a grandparent. This is an important margin, as many of the channels we describe above would not necessarily result in a foster care placement. In fact, child welfare services have increasingly sought to place at-risk children with family members, due in part to the Fostering Connections to Success and Increasing Adoptions Act of 2008, which established policies to encourage the practice (Child Welfare Information Gateway, 2019).

To conduct this analysis, we use data from the March Current Population Survey Annual Social and Economic Supplement from 1990 through 2015. We construct age-state-year cell averages of different living arrangements for children age 0-16, and pair these with estimates of children's exposure to the crisis that are constructed from Multiple Cause of Death Data from 1973 to 2017. Our measure of exposure to the crisis is the cumulative drug-related death rate for likely parents over the child's life. This measure accounts for the fact that a transition to the child's current living arrangement could have occurred at any point in the child's life, and older children have had more exposure to the crisis than younger children.

In OLS models with state, age, and year fixed-effects, we document a strong correlation between this measure of exposure to the crisis and family living arrangements. Our 2SLS estimates suggest that the relationship is causal—in all cases, OLS and 2SLS results are very similar in magnitude and we fail to reject the null hypothesis that the 2SLS estimates are equal to their OLS counterparts. We show that greater exposure to the crisis increases the chance that a child's mother or father is absent from the household and it increases the likelihood that he or she lives in a household headed by grandparent. These results hold when subjected to a wide battery of robustness tests related to our measure of exposure to the crisis, recent policies related to the opioids market, welfare reforms in the 1990s, differences in population and urban versus rural

residency of respondents, local economic conditions, trade shocks, the choice of sample, and state-specific time trends.

Given the size of the rise in the cumulative death rate of likely parents between 1996 and 2015, the implied effect on children is staggering. We estimate that by 2015, more than 1.5 million more children aged 0-16 were living away from a mother or a father and a half a million children were living in a household headed by a grandparent as a result of the drug crisis. While these estimates are very large, they are consistent with survey data. The 2018 National Survey on Drug Use and Health (NSDUH) estimates that among adults aged 18 and older, 4.2 million adults had a substance abuse disorder for illegal drugs other than marijuana, and about half that amount was for opioid abusers.<sup>6</sup> This abuse translates into millions of children living in households with a drug abuser. Using data from the 2009 NSDUH, Lipari and Van Horn (2017) estimate that 2.1 million children lived in a household with a least one parent with a past-year illegal use disorder. The number of children affected by drug-related *deaths* within their household is also large. Using data from the 1986 through 2004 mortality follow-back of the National Health Interview Survey (Blewett et al., 2019), we calculate that households that had an adult death related to drug poisoning in the four quarters after the household entered the survey included an average of 0.71 children. Assuming that this number is representative of more recent years, we estimate that the 763,000 deaths of adults aged 18 and up from 1999 through 2018 occurred in households that housed 542,000 children.<sup>7</sup> This is an underestimate of the number of children exposed to the death of a family member due to drugs, as it does not count those not living in the household (such as a father estranged from the child's mother). It is worth emphasizing that because we cannot reject the null hypothesis that OLS and 2SLS results are the same, our analysis suggests that drug deaths were affecting family structure, rather than declining family institutions leading to drug use.

Because the drug crisis is recent and currently ongoing, it is too soon to know how these changes in family structure will affect children's long-term outcome. A large body of work shows that children who have experienced trauma<sup>8</sup> have worse adult outcomes such as higher rates of

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<sup>6</sup> <https://www.samhsa.gov/data/sites/default/files/cbhsq-reports/NSDUHDetailedTabs2018R2/NSDUHDetTabsSect5pe2018.htm>

<sup>7</sup> The NHIS uses a 113-category cause of death classification system. Drug deaths would appear in three different categories: accidental poisoning, suicides not from firearms, and other unspecified accidental events, which primarily include deaths of undetermined intents. Using data from 1999 through 2018 from the National Vital Statistics System's Multiple Cause of Death data, we estimate that drug deaths represent 70 percent of deaths in these three categories.

<sup>8</sup> Trauma is frequently measured through the ACE (adverse childhood experiences) survey which asks adults 10 questions about whether as a child their parents divorced, whether someone in their household went to prison, engaged

mortality (Feletti et al., 1998), substance abuse and mental health incidence (Anda et al., 2006; Dube et al., 2003), suicide (Dube et al., 2001), involvement in criminal activity (Baglivio et al., 2014), and unemployment (Hardcastle et al., 2018; Liu et al. 2013). To make steps towards considering some of these outcomes, in Section V, we apply our same estimation strategy to investigate the impact of the crisis on the resources available in children’s households. Our results suggest that exposure to the drug crisis increases the probability that a child is living in an impoverished household or is participating in SNAP.

In the last sections of the paper, we draw on both theoretical and empirical research from across the social sciences to discuss how these changes in resources, and changes in children’s living arrangements more generally, are likely to affect outcomes. It is important to note that the variation we exploit corresponds to a particular counterfactual: a world without the drug crisis. This is particularly relevant when thinking through the likely welfare impacts of children being pushed to live in households headed by grandparents. While having a child move in with a grandparent when a parent is abusing drugs could be a constrained optimal decision conditional on being affected by the drug crisis, this does not imply that the child is better off than she would have been *in the absence of the crisis*. Overall, the evidence we discuss leads us to conclude that the changes in family structure that we document will have harmful long-term consequences for a great many of these children.

## II. Measuring a Child’s Exposure to the Drug Crisis

The outcome that we are interested in is a child’s living arrangement at a given point in time, which we observe using the Annual Social and Economic Supplement (ASEC) of the March Current Population Survey (CPS). Importantly, the current arrangement is a product of all of a child’s experiences to that point in his or her life—it measures the *stock* of a child’s experience rather than the *flow*. Accordingly, a 15-year old in a given state and year has substantially more exposure to the drug crisis than a one-year old, and the 15-year old could have separated from her parent because of the parent’s drug use at any point in her life. Thus, while it is tempting to correlate a child’s current living arrangement with a contemporaneous measure of the crisis in a given state and year, we believe this would miss much of the relevant exposure to the crisis.

To that end, we construct a variable that represents a child’s cumulative exposure to the drug crisis. We call this variable  $CEXPOSURE_{ast}$ , and the subscripts indicate that the measure will vary

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in substance abuse or was mentally ill, whether they experienced neglect or felt unloved, or experienced various forms of verbal, physical, or sexual abuse.

with a child’s age ( $a$ ), state ( $s$ ), and year ( $t$ ). Our measure of the intensity of the crisis in a given state and year comes from deaths related to drug poisonings, which are available from the National Vital Statistics System’s Multiple Cause of Death (MCOB). Although drug deaths are more easily measured than drug use or abuse, they are an imperfect measure because (fortunately) not all use results in death. In 2017 there were about 72,000 drug deaths, but data from the NSDUH indicates that in that year, 4.2 million adults had a substance abuse disorder involving illegal drugs (excluding marijuana) and about half that amount included opioids. Nevertheless, because deaths are highly correlated with use, we expect that deaths will capture much of the important variation in exposure to the crisis.

To construct  $CEXPOSURE_{ast}$ , we sum the number of drug deaths of likely parents of children of age  $a$  in year  $t$  from state  $s$ , and divide by the number of likely parents at their birth.<sup>9</sup> We define likely parents as those aged 17 to 40 in the child’s year and state of birth.<sup>10</sup> A newborn’s exposure is then the drug death rate of those 17-40 years old in the child’s year of birth. As a child ages, so do her likely parents; to account for this, we age the risk set. For example, for a one year old child, we add the number of deaths among 18-41 year olds in the current year to the number of deaths among 17-40 year olds in the previous year. More precisely, we define cumulative exposure as:

$$(1) \quad CEXPOSURE_{ast} = \frac{\sum_{j=0}^a \sum_{k=17+j}^{40+j} Drug\ Deaths_{kst}}{(Population\ 17-40_{st-a} / 100,000)}.$$

Because they are rare events for people in the age range of likely parents, we express deaths as the number per 100,000 in the relevant population. Both the numerator and denominator are likely measured with error because of in- and out-migration, but for most children it should be a reasonable proxy for their cumulative exposure to the crisis. In our discussion below, we will refer to this measure as either cumulative exposure or cumulative mortality.

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<sup>9</sup> We use the child’s current state of residence from the ASEC files to assign state; unfortunately, state of birth is not contained in this dataset. Migration in response to the drug crisis would cause measurement error that could lead us to either underestimate or overestimate the effect of the crisis on living arrangements, depending on the nature of the migration response. As a check, we have used the American Community Survey (ACS) to estimate our results. These data do contain information on state of birth but the samples do not start until four years after OxyContin was introduced and the samples are much smaller in the early years of the ACS. The results in the ACS are similar to the results from the ASEC and there is little difference in estimates when we use state of birth or state of residence, leading us to conclude that selective migration is not meaningfully affecting our results.

<sup>10</sup> Data from the 1996 Natality Detail data notes that 96 percent of mothers and 93 percent of fathers are in this age range.

Given that we have ASEC data from 1990 through 2015 for children aged 0-16, we use the MCODE data from 1973 through 2015. The former year is required to calculate the cumulative exposure for children aged 16 in 1990. We obtained access to the restricted-use version of the MCODE files from 1983 to 2015, which contains state identifiers. We then supplement this with the publicly available versions of the MCODE files with state identifiers from 1973 through 1982.<sup>11</sup> In years 1973-1980, 1983-2015, the MCODE data contains a census of deaths in the US. In 1981 and 1982, there is a 50 percent sample from 19 states. Over this extended period, the MCODE data uses cause of death codes from three different versions of the International Classifications of Diseases: ICD-8 (through 1977), ICD-9 (1978-1998), and ICD-10 (1999-2015). In Appendix A we outline the codes used to identify drug and opioid deaths. As we note in the appendix, it is easier to define drug deaths across ICD systems than opioid deaths so we focus on this as the key covariate of interest.

In Figures 2 through 4, we demonstrate how children's exposure to the crisis varies with their age, state, and year. In Figure 2, we report the national time series of the cumulative mortality for likely parents of children aged 0, 4, 8, 12 and 16. To calculate these numbers, we merge the exposure measure to the ASEC samples at the age, state, and year level and weight by population across states. The rate for children aged 0 captures the contemporaneous death rate for adults aged 17-40. We note two things about this figure. First, cumulative exposure by a given age increases dramatically over time—by 290 percent for children aged 0, and by 255 percent for children aged 18. Second, the trends “fan out” over time, reflecting the fact that as the crisis has grown, the *difference* in cumulative exposure between older and younger child has grown as well.

There is also tremendous variation across birth cohorts in their exposure to the drug crisis by a given age (where we approximate year of birth as survey year minus age). In Figure 3, we report the measure for three birth cohorts (1978, 1988, 1998) as they age.<sup>12</sup> More recent cohorts have much greater exposure to the crisis at every age. By age 16, those born in 1998 have experienced about 334 deaths per 100,000 likely parents—about 3 times more than that experienced by the 1978 birth cohort.

Finally, there is variation across states in children's cumulative exposure. In Figure 4, we report the cumulative exposure over time for ten-year olds from six states: California (CA), Illinois (IL), Nebraska (NE), Ohio (OH), Virginia (VA), and West Virginia (WV). These states represent two states each with low, medium and high growth in this measure of exposure. These also include

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<sup>11</sup> These are available on the NBER website.

<sup>12</sup> The slope of a particular line represents the vertical distance between graphs at particular points in Figure 2.



two triplicate (CA and IL) and four non-triplicate states. The two largest states (California and Illinois) had substantially larger drug poisoning rates in the early part of the sample. In Illinois, a child's exposure increases by 191 percent over time, while in California it actually returns to its 1990 level by 2015. The other four states all start out with similar rates in 1990, but grow at very different rates. Nebraska and Virginia see a tripling in our measure of cumulative exposure for ten-year olds, which is typical of the nation as a whole. Meanwhile, Ohio and West Virginia are epicenters of the drug crisis, and the exposure rate for 10-year olds increases in these two states by 691 and 1,436 percent, respectively.

### **III. Data Sources and Econometric Specification**

As the data will imply the structure of the econometric model, we present information about both the data sources and the model specification in this section.

#### *A. Data on Outcomes*

Our primary data on the living arrangements of children comes from the March CPS's ASEC. The CPS is a monthly survey of the non-institutionalized population that is the primary source for monthly labor market data in the U.S. The CPS surveys about 60,000 households and 160,000 people each month, and asks a basic set of questions each month and fields special supplements most months. The ASEC is fielded in March and asks detailed questions about health insurance status, the previous year's income from a variety of sources, receipt of non-cash benefits, and a household's participation in certain federal social insurance programs. The income and family size variables are then used to construct the annual estimates of poverty.

We use the IPUMS processing of the ASEC (Flood et al., 2020) which has harmonized the data over time, allowing researchers to pool data across years. The IPUMS versions of the data also have a number of constructed variables that we use for this analysis. All respondents are a unique line number within the household and the CPS data has a detailed household roster that identifies each member's relationship to the head of the household. From these variables, IPUMS generates variables that match a child to a mother and father in the household, and the coding allows us to determine whether a mother or father is in the household. The codes do not distinguish between biological, adoptive, or step-parents. We note that there is some noise in linking children when

there are multiple subfamilies in households, and the IPUMS has explicit rules to match mothers to children in these situations.<sup>13</sup>

We use these codes to generate three key variables at the individual level that measure the living arrangements of children: 1) whether the child is living away from a mother, 2) whether the child is living away from a father, and 3) whether the child is living in a household headed by a grandparent. We can also construct other outcomes from these variables, such as indicators for whether the child is missing at least one parent, is missing *both* parents, is in a household headed by someone other than a parent, and whether the child is in foster care. On the latter, it is important to note that comparisons to administrative records from the Adoption and Foster Care Analysis and Reporting Systems (AFCARS) show that CPS estimates of the size of the foster care population are too small by about half (O’Hare, 2008). Part of the reason for this is that when indicating the child’s relationship to the household head in the CPS, relatives who are serving as foster parents are likely to indicate their familial relationship to the child rather than their foster parent status—for example, the grandmother who is also a foster parent will often indicate that the child is her grandchild (Schweizer, 2019).<sup>14</sup>

We construct these living arrangement indicator variables for all children aged 0 through 16 from 1990 through 2015. We then aggregate the data into age, state, and year cells using sample weights, and express these outcomes as the number of children in a given living arrangement per 100,000. The key outcome in our analysis is therefore a variable  $y_{ast}$  that measures the number of children per 100,000 of age  $a$  from state  $s$  in year  $t$  living in a particular household type.

## B. *The Econometric Model*

Figures 2-4 indicate there is tremendous variation in the exposure to the crisis across ages, states, and time. Our econometric model attempts to exploit this variation. Given the variables defined above, we begin with the model

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<sup>13</sup> For example, consider a mother and father that have two adult sons living with them and one of these sons has a child living in the household. This child will report their relationship to the householder (grandchild) but it is not clear which adult male (child of householder) to attach as the child’s father. In this case, IPUMS uses the oldest male as the parent, which could be an error.

<sup>14</sup> We explored using data from AFCARS; these data provide case-level information on children who enter the foster care system, where each observation is a removal event. As a result, it captures the “flow” in children’s living arrangements, rather than the “stock.” Unfortunately, this structure is not well-suited to our identification strategy, which exploits variation in children’s cumulative exposure to the crisis. Consistent with this, our 2SLS estimates of the effect of cumulative exposure on foster care admission rates are very imprecise and do not allow us to rule out large positive or negative effects of the crisis.

$$(2) \quad y_{ast} = X_{ast}\beta + CEXPOSURE_{ast}\alpha + \eta_a + \mu_s + \lambda_t + \varepsilon_{ast}$$

where  $y_{ast}$  and  $CEXPOSURE_{ast}$  are defined as above. For regressions in which the dependent variable measures the absence of a mother or father, we use a measure of  $CEXPOSURE_{ast}$  that is restricted to deaths among women and men respectively; for all other regressions, the measure includes both men and women.  $X_{ast}$  is a vector of control variables,  $\eta_a$ ,  $\mu_s$ , and  $\lambda_t$  are age, state, and time fixed effects respectively, and  $\varepsilon_{ast}$  is a random error. The data contain 17 age groups, 26 years of data, and information for 51 states so there are 22,542 observations. The control variables include the fraction of a cell that is female, Black (non-Hispanic), other race (non-Hispanic), or Hispanic (with non-Hispanic whites serving as the omitted category). Because both our outcomes and measure of exposure are expressed as the number of occurrences per 100,000 people, this allows us to interpret the coefficient on  $CEXPOSURE_{ast}$  as the number of additional children living in a particular household structure for each additional cumulative drug death. We obtain consistent estimates of the standard errors by clustering at the state level.

We are concerned about two potential sources of bias in our OLS estimates. First, there is the potential for reverse causation. There has been a general increase in the non-marital birth rate over much of this time period, and with divorce being prevalent, many fathers are living away from their children. Case and Deaton (2015 and 2017) argue that the decline in institutions such as marriage has led to a “culture of despair” where people turn to drugs as a release from their troubles. In this case, OLS estimates of  $\alpha$  will be overstated if the rise in parents living away from their children results in more drug deaths. Second, there is the potential for omitted variables bias. In the Case and Deaton framework, the decline in economic opportunities, especially for low-skilled workers has caused a shift towards drug use as a response. If the same factors encouraging drug use, such as high unemployment, low wages, or low wage gains, are also damaging families, then the OLS estimate of  $\alpha$  would also be overstated as the estimate would be capturing these same factors that are driving both high drug use and higher movements away from parents living with their children. Existing evidence suggests this could be a serious concern. For example, high state unemployment rates have been shown to lead to both higher drug deaths (Hollingsworth et al., 2017) and lower marriage rates (Schaller, 2013).<sup>15</sup> Thus, to obtain consistent estimates of  $\alpha$ , we use an instrumental variables (IV) procedure as described in the next two sections.

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<sup>15</sup> However, both Schaller (2014) and Hellerstein and Morrill (2011) show that divorce is pro-cyclical, which would cause a negative bias in our estimates of  $\alpha$ .

### C. *Exposure to Non-Triplicate States in the OxyContin Era*

The rise in drug deaths has been driven primarily by opioids, and the drug at the center of the rise is OxyContin, a branded opioid pain killer marketed by Purdue Pharma.<sup>16</sup> Its active ingredient is oxycodone, an opioid that has been in clinical use since 1917 (Kalso, 2005). OxyContin provides oxycodone through an extended-release formulation that allows for up to 12 hours of pain relief, and as a result there is typically a high milligram (mg) content of oxycodone in the pills. Since its release in 1996, OxyContin has been one of the most successful pharmaceuticals of all time with worldwide sales totaling \$35 billion.<sup>17</sup>

OxyContin was introduced in January of 1996. Historically, opioids were reserved for those with acute pain such as post-surgical or cancer patients. Given the limited use of opioids, pain from chronic conditions often went untreated. This was viewed by many as a failure of the medical profession (e.g. Campbell, 1995; American Academy of Pain Medicine and American Pain Society, 1997). With the heightened concern about patient pain, pharmaceutical manufacturers started to market opioids directly to physicians. A key message in many presentations was that the risks of addiction were small when opioids were used appropriately. Purdue Pharma was particularly aggressive at promoting this line of argument for OxyContin (Van Zee, 2009; Quinones, 2015).

Internal documents from Purdue Pharma related to the introduction and promotion of OxyContin are now accessible as a result of various lawsuits. These documents indicate that Purdue Pharma's marketing approach was multi-faceted and included advertising in medical journals, promoting OxyContin at practitioner conventions, incentivizing pharmacies to stock the drug, advertising in drug wholesaler booklets, and much more. But Purdue Pharma believed that their most valuable tool was pharmaceutical sales representatives—employees who visit prescribers and tout the benefits of their employer's drugs.<sup>18</sup> As seen in Figure 5, Purdue Pharma's marketing expenditures increased from \$40 million in 1996 to \$260 million by 2001. The number of OxyContin prescriptions follows a similar pattern, increasing gradually from 300,000 in 1996 to 7.2 million in 2001.

Although OxyContin became available in all states in January of 1996, Purdue did not market it equally in every state. During its extensive pre-launch market research, a consistent theme

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<sup>16</sup> We refer to OxyContin ER simply as OxyContin throughout the paper.

<sup>17</sup> <http://www.latimes.com/projects/la-me-oxycotin-part3/>

<sup>18</sup> "The deployment of our most valuable resource, the sales force, will be critical to the success of OxyContin" (p. 33 in *1996 Budget Plan*).

emerged: Marketing was unlikely to be effective in “triplicate” states. Triplicate programs require prescribers to use a special, serialized pad to prescribe a Schedule II opioid such as OxyContin.<sup>19</sup> The prescriber, pharmacist, and the state each retain a copy. When Purdue Pharma launched OxyContin in 1996, California, Idaho, Illinois, New York, and Texas had active triplicate programs. These programs were an early form of prescription drug monitoring and were implemented between 1939 and 1982, well before opioid deaths began rising rapidly.

Purdue Pharma focus group research indicated that doctors found triplicate pads a hassle and did not want their prescribing practices observed by the state. In response, physicians could avoid prescribing Schedule II drugs by prescribing Schedule III opioids instead. Purdue Pharma’s research concludes: “There seems to be a definite opportunity for OxyContin as a medication for treatment of severe non-cancer pain among doctors in the non-triplicate states. More work might have to be done to determine if the product is viable in the triplicate states; however, the preliminary evidence is not encouraging.” The ability for a physician to avoid using a triplicate pad meant, “our research suggests the absolute number of prescriptions they would write each year is very small, and probably would not be sufficient to justify any separate marketing effort” (p. 8 and p. 59 in Greenbaum, 1995).

Figure 6 traces out differences in per capita opioid use across non-triplicate and triplicate states over time for specific active ingredients.<sup>20</sup> Unfortunately, the data for these graphs only extends back to 1997. To make opioids of varying strengths comparable, we convert each opioid into morphine equivalent grams and scale per 100,000 population. Triplicate and non-triplicate states had somewhat similar rates of oxycodone use (the active ingredient in OxyContin) at the start of 1997, but as Purdue’s marketing ramped up, so did the differences in oxycodone use across these groups of states. However, for most of the other opioids included in the ARCOS data, the difference between non-triplicate and triplicate states is small and remains so up through 2010. There are two important things to note about the exception, morphine: 1) because all drugs have been put into comparable units, the growth is negligible relative to the growth seen for oxycodone,

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<sup>19</sup> In 1970, the Controlled Substances Act created five “schedules” for drugs related to their potential for abuse and their medical uses. Schedule I drugs have the highest potential for abuse and no medical use (e.g. heroin). Lower scheduled drugs have either a lower potential for abuse or a larger role in medicine. Oxycodone, the main ingredient in OxyContin, is a Schedule II drug.

<sup>20</sup> This data is from the Automation of Reports and Consolidated Orders System (ARCOS), a Drug Enforcement Administration program that requires all manufacturers and distributors to report their transactions and deliveries of all Schedules I and II substances as well as a number of Schedule III-V substances. Data from 2000 forward are available on the DEA’s website. We obtained data going back to 1997 via Freedom of Information Act requests.

and 2) Purdue Pharma was also actively promoting their continuous release morphine drug, MS Contin.

There are at least two reasons to believe that Purdue’s marketing is causing these differences rather than them being directly attributable to the triplicate programs themselves. First, if the triplicate programs were the driving force, then we would have expected the seven other opioids shown in Figure 5 to display higher growth in the non-triplicate states than the triplicate states. The lack of that differential growth suggests there was something specific to oxycodone, and to a much lesser extent, morphine. Second, each of the triplicate programs was replaced with an electronic prescription drug monitoring program within a few years of OxyContin’s introduction.<sup>21</sup> Evidence reported in Alpert et al. (2019) suggests that Purdue’s marketing directly affected drug overdose death rates and was not simply picking up other factors including opioid-related policies, changes in economic conditions, or differences between urban and rural areas.

The impact of Purdue Pharma’s advertising strategy on drug deaths can be seen in Figures 7 and 8. In Figure 7, we report drug death rates for the whole population over time for triplicate and non-triplicate states. Prior to 1996, the trends for the two groups of states were similar and triplicate states actually had higher drug death rates than non-triplicate states. After 1996, drug deaths increase in both types of states, but the increase is more dramatic in non-triplicate states. Figure 8 reports the corresponding cumulative mortality risk for 10-year olds in these two state groups from 1983 through 2015.<sup>22</sup> The results mirror those in Figure 7. Because we aggregate deaths over an 11-year period to construct these numbers, the pre-treatment differences in rates across states groups are large, but the pre-treatment trends are very similar.

#### D. *The 2SLS model*

To address the potential bias in OLS estimates that we discussed in Section III.B., we implement an instrumental variables strategy that takes advantage of the fact that Purdue Pharma marketed OxyContin much less in states with triplicate programs. Specifically, we use a child’s exposure to a non-triplicate state in the post-1995 era as an instrument for our measure of his or her cumulative exposure to the crisis. We label the instrument  $YearsExpNT_{ast}$ , and it is constructed as:

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<sup>21</sup> The triplicate programs were replaced in 1997 (Idaho), 1999 (Texas), 2000 (Illinois), 2001 (New York), and 2004 (California).

<sup>22</sup> Because our MCODE files begin in 1973, 1983 is the first year for which we can construct a cumulative risk number for 10 year olds.

$$(3) \quad \text{YearsExpNT}_{ast} = \text{Min}(\text{Age}_{st}, \text{Years after 1995}) * \text{NonTriPLICATE}_{st}$$

where  $\text{NonTriPLICATE}_{st}$  is an indicator for whether the child’s state did not have a triplicate program at the time of OxyContin’s launch. For example, consider a 10-year-old in 2010 in a non-triplicate state. That child has had 10 years of exposure to living in a non-triplicate state, and consequently  $\text{YearsExpNT}_{ast} = 10$ . For a 3-year-old child in the same state and year, she has only been exposed for three years and so  $\text{YearsExpNT}_{ast} = 3$ . Children in triplicate states of all ages and in all years have  $\text{YearsExpNT}_{ast} = 0$ .

The validity of this approach rests on two assumptions. First, it must be the case that  $\text{YearsExpNT}_{ast}$  is correlated with our measure of cumulative exposure. Figure 8 suggests that this is the case, and we confirm this with regression estimates of the first-stage equation below. Second,  $\text{YearsExpNT}_{ast}$  should *not* be correlated with the error term in equation (2). While we cannot test this assumption directly, the triplicate programs were in place well before the drug crisis, and we have no reason to expect that they would have affected family structure for reasons aside from drug mortality. Moreover, the similar pre-trends for the two types of states in Figures 7 and 8 are reassuring, and we will compare pre-trends in family structure in the next section.<sup>23</sup> We cluster the standard errors by state in our main analyses, but also provide wild bootstrapped 95 percent confidence intervals in Appendix Tables B1 and B2.

## IV. Results

### A. Graphical Analysis

Before turning to our regression models, we first present a series of graphs in Figure 9 that foreshadows the results. In Figure 9A, we graph the percent of children aged 0-5 that are living away from their mothers in triplicate and non-triplicate states. In Figure 9B, we present the same graph for older children, aged 11-16. In 9C and 9D, we repeat these same graphs for the percent of children living away from their fathers, and in 9E and 9F, we show graphs for the percent of children living in a household headed by a grandparent. In each of these graphs, the high and low values on the vertical axis are the same but the range in all graphs is 10 percentage points to make comparing magnitudes more straightforward.

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<sup>23</sup> See also Alpert et al. (2019) for more event study results that demonstrate that the two types of states had similar trends in drug outcomes before the introduction of OxyContin.

We report the graphs for older and younger children because our econometric model takes advantage of variation across states, over time, and across different ages of children. In the reduced-form, for a specific age group, we are comparing outcomes over time in triplicate and non-triplicate states. However, because younger children were less exposed to the crisis in either state group in any particular year, we would expect to see larger differences across regimes in outcomes for older compared to younger children.

There is a consistent theme running through these graphs. First, for all six graphs, despite the fact that the triplicate states are a unique set of jurisdictions, their pre-1996 levels and trends in outcomes are quite similar to those of the non-triplicate states. Second, the results in these figures suggest the 2SLS models will show some effect of the crisis on these three outcomes. Comparing 9A to 9B, 9C to 9D, and 9E to 9F, there is a noticeably larger negative gap in outcomes between non-triplicate and triplicate states in the post-1996 period for the older group (B, D, and F) compared to the younger groups (A, C, and E).

#### B. *OLS and 2SLS Estimates*

In Table 1, we report the OLS and 2SLS estimates of equation (1). In the first column, we report the means of the dependent variable for the sample. Six percent of children are living away from a mother, about 24 percent are living away from a father, 3 percent are living away from both parents, and about 5 percent are living in a household headed by a grandparent.

In the next column, we report the OLS estimates of equation (1). In the first two rows, we use the cumulative death risk for likely mothers and likely fathers, respectively, as the covariate of interest. In the third row and beyond, we use the aggregate risk for adults in the appropriate age ranges. The magnitudes of the estimates in the first two rows are remarkably similar—for every likely parent that dies from a drug poisoning, about 10 children are left without a mother or are left without a father. Although this estimate might appear large, there are roughly 230 people misusing opioids or using heroin and about 43 people with a drug use disorder for this class of drugs for every person who dies from an opioid overdose.<sup>24</sup> In the next two rows, we see that for each death, approximately 15 children are missing at least one parent and about 4 children are missing *both*

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<sup>24</sup> The 2017 National Drug Use and Health Survey reports 11.8 million people that used heroin or misused pain relievers in the past year and we noted above that there are 2.1 million with a substance use disorder for this group of drugs. (<https://www.samhsa.gov/data/sites/default/files/cbhsq-reports/NSDUHDetailedTabs2017/NSDUHDetailedTabs2017.htm#tab1-1A>) We calculate about 49,000 deaths from opioids in 2017.



parents. The next two rows suggest that for each additional drug death of a likely parent, 6 children are living in a household not headed by one of their parents and 2 children are living with a grandparent as the head of household. Finally, in the last row, our OLS estimate suggests that for every drug death, 0.2 children are living in foster care. All of these results are statistically significant at conventional levels, with the exceptions of the results for grandparents and for foster care.

In the third column of Table 1, we turn to our 2SLS estimates. We report the first-stage F-test of the hypothesis that the coefficient on the instrument is zero in brackets underneath the standard errors. Full results from the estimates of the first stage are reported in Table 2; given the graphs in Figures 7 and 8, it is not surprising that the years of exposure to a non-triplicate environment is strongly correlated with all death rate measures. The numbers in the first two rows of Table 2 indicate that a 16-year old with 17 years of exposure to a non-triplicate state was exposed to a cumulative death rate that was 126 times greater for likely mothers and 189 times greater for likely fathers than a 16-year old in a triplicate state. The smallest F-test in the table is 80, so there is little concern about finite sample bias in these models (Staiger and Stock, 1997).

Returning to the 2SLS estimates in the third column of Table 1, we see that all estimates are statistically significant at the 5 percent level or lower except for the those related to foster care. In the first four rows, the 2SLS estimates are close in magnitude to the OLS estimates. In the fourth column, we report the p-value for the Hausman test of the null hypothesis that the OLS and 2SLS estimates are the same. In these four cases, the p-value is very high and we cannot reject the null. In the next two rows where the outcome is the number of children per 100,000 in households headed by grandparents or headed by a non-parent, the 2SLS coefficients are somewhat larger than their OLS counterparts, but the p-values on the Hausman test are still slightly larger than 0.05. In general, the 2SLS estimates present the same story as the OLS models with similar magnitudes. Case and Deaton (2015 and 2017) suggest the drug crisis is in response to the decline in high-paying jobs for low skilled workers in sectors like manufacturing, and the resulting decline in institutions like marriage and the family. In this instance, the similarity of the OLS and 2SLS models are consistent with the interpretation that the drug crisis is leading to parents living away from their children, and *not* the other way around.

In the final two columns of Table 1, we report the 2SLS model's prediction of the number of children living in each arrangement due to the drug epidemic. In the fifth column, we give the change in the rate of likely parents' mortality risk produced by the rise in drug death rates over this period multiplied by our estimated impact of the risk on the outcome. In the final column, we

report the number of children impacted by this change.<sup>25</sup> We first note that for foster care, the point estimate is actually negative, so that our calculations suggest that 6,000 *fewer* children were living in foster care as a result of the crisis. However, the estimate is imprecise, and we also cannot rule out that many thousands of children were moved into foster care as a result of the crisis, as many popular press articles have suggested. We may also be missing children who were in formal foster care, but who were identified as a relative in the ASEC data.

What we can conclude is that any movement of children into foster care was dwarfed by the other changes in living arrangements that we consider. The estimates indicate that had the drug epidemic stayed at 1996 levels, there would be about 2.3 percentage points fewer children missing at least one parent in the household and about 0.8 percentage points fewer children living in a home headed by a grandparent. This translates into 1.5 million children living away from at least one parent and half of a million children living in a household headed by a grandparent in 2015. Perhaps the most sobering number is that 300,000 children are living away from both parents as a result of the drug crisis.

### C. *Robustness and Heterogeneity*

In this section, we summarize the findings from a battery of robustness tests; a full discussion and reporting of results can be found in Appendix B. We assess the robustness of our results to 1) alternative specification and sample choices; 2) the inclusion of additional controls; 3) omitting individual treatment states; and 4) alternative specifications to address the difference in population between triplicate and non-triplicate states.

First, recall that our exposure measure includes all drug deaths, and not only opioid deaths. While the latter are more likely to be affected by our instrument, the former is more consistently measured. When we use opioid deaths instead, the point estimates are slightly larger. The same is true when we account for the shift from prescription to illegal opioids that was induced by the reformulation of OxyContin in 2010 (documented in Alpert et al. (2018) and Evans et al. (2019)) by restricting the sample to the years before 2011. In addition, we explore whether the drug crisis affected the composition of families through changes in fertility by restricting the sample to those

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<sup>25</sup> Specifically, we calculate the change in cumulative mortality for each age 0-16 over the 20-year period 1996 to 2015 (reported in column 4), multiply this by the 2SLS coefficient on  $CEXPOSURE_{ast}$ , then multiply this by the number of children of each age in 2015 as measured by the SEER population data, and scale by 100,000 people to get levels.

born before the introduction of OxyContin in 1996; doing so yields estimates that are less precise but consistent with our main findings.<sup>26</sup> We find suggestive evidence that children born in non-triplicate states after the introduction of OxyContin were actually positively selected, in families that were less likely to have mothers missing, fathers missing, or to be headed by a grandparent. We also estimate specifications that include state-specific linear time trends to capture differential changes in population or metropolitan areas (among other things). Our results remain the same.

Second, we show that our results are not simply masking differences or changes in economic conditions across triplicate and non-triplicate states. Our results are robust to including controls for each state's per-capita real GDP or the state's unemployment rate. In addition, we show that the granting of permanent normalized trade relations to China in 2000, linked to opioid death rates by Pierce and Schott (2020), is not driving our results. We then show that our results are not merely picking up other, commonly-studied changes in the opioid market or in public policy—they are not qualitatively different if we control for subsequent opioid-related policies such as prescription drug monitoring programs, or if we control for welfare reforms which were previously shown to affect children's living arrangements (Bitler et al., 2006).

Third, we remove each triplicate state, one at a time, and re-estimate the regression. We find that no single triplicate state is driving our results.

Fourth, we address the large differences in population between triplicate and non-triplicate states and show that this is not driving our results. We show that including a fourth-order polynomial in the number of parent-aged adults does not substantively alter the results. Because the triplicate states are more urban than the non-triplicate states, we also estimate models separately for children living in metropolitan areas and for children not living in metropolitan areas. Again, our results are qualitatively similar to our main findings. In the spirit of a matching estimator, our results are also similar if we restrict our sample to include only the non-triplicate states with populations similar to triplicate states in 1990.

In our final appendix table, we explore whether the effects of the crisis vary by race. We run our analyses separately for white and Black children using race-specific measures of exposure, and

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<sup>26</sup> In addition, we explore whether the drug crisis affected the composition of families through changes in fertility using data on the population of children under age one from SEER. We find suggestive evidence that children born in non-triplicate states after the introduction of OxyContin were *less* likely to have an unmarried mother. If these children are also less likely to live in families with mothers or fathers missing or to be headed by a grandparent later in life, this selection would cause us to understate the impact of the crisis on child living arrangements.

find large impacts on white children similar to what we found for the entire sample.<sup>27</sup> Our point estimates for Black children are larger (though smaller when expressed as the percent change relative to the mean), but the estimates are imprecise and we cannot reject the null of no effect.

## **V. Changes in Family Structure and Longer-Term Effects**

The results in the previous section demonstrate that the drug crisis has had large impacts on the living arrangements of children. There is a substantial body of work in the social sciences that suggests that these changes could have important negative consequences for the affected children. One of the primary channels for such an effect is diminished household resources. Most theoretical models of child development include inputs like food, housing, and education, which are often purchased using household income.<sup>28</sup> Rigorous evidence from natural experiments and randomized trials shows that higher household income does indeed produce improvements in academic achievement, cognitive ability, and behavioral outcomes, and reductions in criminal activity (Akee et al., 2010; Duncan et al., 2011; Dahl and Lochner, 2012; Akee et al., 2018).<sup>29</sup> The absence of a parent as a result of the drug crisis will likely affect the resources available for the child (Lopoo and DeLeire, 2014; Kalil et al., 2014), though of course the magnitude of the change will depend on how much the parent would have contributed absent the crisis, and the extent to which these resources are available in the new living situation.

As a first step in directly analyzing the impacts of altered living arrangements, we estimate the same OLS and 2SLS models as before, but with various measures of a household's economic status as the dependent variable. The results are presented in Table 3 and again we find remarkably similar point estimates across the OLS and 2SLS methods for most outcomes. Broadly, the results paint a picture in which children who are more exposed to the drug crisis are more likely to live in households with fewer economic resources. If interpreted causally, the estimates suggest that because of the drug crisis, almost 700,000 more children are living in poverty and 785,000 are participating in the Supplemental Nutrition Assistance Program ("food stamps").

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<sup>27</sup> We do not distinguish based on ethnicity in these race-specific models because the ICD-8 version of the mortality data does not identify ethnicity.

<sup>28</sup> See for example Becker (1960) and Willis (1973).

<sup>29</sup> Child development models also often include time inputs or other measures of engagement from household members in the production function. Credible causal evidence on the effects of parental engagement is harder to come by, but the finding of a positive association with desired child outcomes is robust (see Sarkadi et al. (2008) and Kalil and Mayer (2016) for summaries). While these time and engagement inputs are also likely to be affected by changes in family structure, we focus on income here because we are able to measure it in our data.

While our focus in this section has been on material resources, we would be remiss if we did not acknowledge that child psychologists have long proposed that parent-child separations may have adverse effects on children’s mental and emotional health and on the development of their personalities (Rutter, 1971). It is difficult to establish causal relationships, but unstable attachment to either one’s mother *or* father throughout childhood is associated with worse biopsychosocial outcomes (Ranson and Urichuk, 2008; King and Sobolewski, 2006). The separation event itself may harm children; Fomby and Cherlin (2007) use longitudinal data on both mothers and children to show that children who experience multiple family structure transitions have worse behavioral outcomes. Separation events that result from the drug crisis may be especially traumatic; in two-thirds of foster care cases in 2017 for which drug abuse by the parent is present, the child also experienced abuse, neglect, or a parent’s death.<sup>30</sup> These types of childhood traumas are known to cause Post-Traumatic Stress Disorder, which affects “the regulation of the neurobiological stress systems, alterations in brain maturation, and neuropsychological outcomes in the developing child” (Watts-English et al., 2006). Given this line of research, it seems clear that the changes in family structure of the kind that we observe are likely to produce negative outcomes for many children.

## **VI. Discussion**

In this paper, we have examined the effect of the drug crisis of recent decades on children’s living arrangements in the United States. Using an instrumental variables strategy that exploits variation across children in exposure to the crisis as a result of pharmaceutical marketing strategies, we show that more children are living without one or both parents as a result of the crisis. Our estimates imply that the increase in the severity of the crisis between 1996 and 2015 meant that 1.5 million children were living away from at least one parent in 2015. Grandparents appear to be filling the void in part; we see that nearly half of a million children were moved into a household headed by a grandparent. Between 1996 and 2015, the fraction of children living in a household headed by a grandparent grew from 5.3 to 6.2 percent, and our results indicate that the advancing drug crisis can explain 83 percent of the increase.

The opioid crisis is one of a variety of societal changes that have indelibly altered family life for children. Our work contributes to the literature on the effects of other public health and policy crises on children’s living arrangements. Western and Wildeman (2009), Wildeman (2009),

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<sup>30</sup> Authors’ calculations, 2017 Adoption and Foster Care Analysis and Reporting Systems data.

Wakefield and Wildeman (2014), and Turney (2017) are part of a large body of work that examines the impact of mass incarceration on families, especially among African Americans. Swann and Sylvester (2006) argue that the increase in foster caseloads from 1985 through 2000 were due in part to female incarceration and the growth of crack cocaine, though Fryer et al. (2013) conclude that the crack epidemic had no impact on foster care admissions but did significantly increase unwed birth rates.

It is too soon to know how long-term outcomes for children will be affected by this change in their living situations. We show that in the intermediate stage, children are more likely to be living in impoverished households and are more likely to be using SNAP benefits. The existing literature on the likely effects of reductions in household income and of parental separations suggests that many children will be negatively impacted by these changes. We can also draw on other work in the social sciences that has documented the causal effects of changes in family structure on long-term outcomes.

While we know of no studies that have identified the causal effect of parental separation as a result of drug abuse specifically, work on the effects of separations for other reasons may be informative.<sup>31</sup> Gruber (2004) exploits variation in children's exposure to unilateral divorce laws to show that the marginal divorce leads to worse outcomes for children, including lower educational attainment and lower incomes. Using a family fixed-effects model to estimate the impact of the unexpected death of a parent in Taiwan, Chen et al. (2009) find that maternal death has large negative impacts on educational attainment. Lyle (2006) studies the impacts of parental absence due to military deployments and finds negative effects on children's test scores; see Paris et al. (2010) for a review of the literature on the effects of military deployments on children. Using variation in judges' sentencing propensities in Sweden, Dobbie et al. (2018) find that parental incarceration leads to increased risky behaviors and worse labor market outcomes for children. Finally, Doyle (2007; 2008) uses a similar strategy that exploits variation in caseworker propensities to estimate the effects of different placement outcomes for children in foster care. Doyle's findings are striking, as they show that even for children whose home environments have drawn the attention of child welfare services, remaining with the parent is better for the marginal child, as they enjoy better labor market outcomes and are less likely to enter the criminal justice system.

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<sup>31</sup> See McLanahan et al. (2013) for a review of the literature on the causal effects of father absence.

Finally, our results also show that nearly half of a million children were moved into a living situation in which the grandparent is the household head as a result of the crisis. For these children, whether and how their outcomes are affected will depend on the extent to which grandparents are able to provide resources and emotional and psychological support. Amorim (2019) finds that multigenerational households spend more on children's education than similar two-generation households. As for time inputs, Kalil, Ryan, and Chor (2014) document that children receive more caregiving time in households with a mother and grandparent than in any other household structure except for those with two biological parents present. We are not aware of any studies that estimate the causal impact of living with a grandparent on children's well-being, but among studies documenting correlations between children's living arrangements and outcomes, it is generally the case that having a grandparent as a primary caregiver is associated with worse academic outcomes and behavior problems, while results are more mixed for children who live in multi-generational households (Dunifon et al., 2018).<sup>32</sup>

This discussion highlights the fact that there will likely be heterogeneity in the effects of changes in family structure due to the drug crisis, driven by differences across children in the counterfactual living arrangement and in the resources and support available in the new home environment. Given that hundreds of thousands of children have been affected, we conclude that the existing evidence suggests that there will likely be a great number of children whose outcomes are worsened by this crisis.

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<sup>32</sup> There are also likely to be important consequences for the grandparents as well; as one example, a recent working paper found that regions where intergenerational co-residence is more common have experienced more COVID-19 deaths (Aparicio and Grossbard, 2020).

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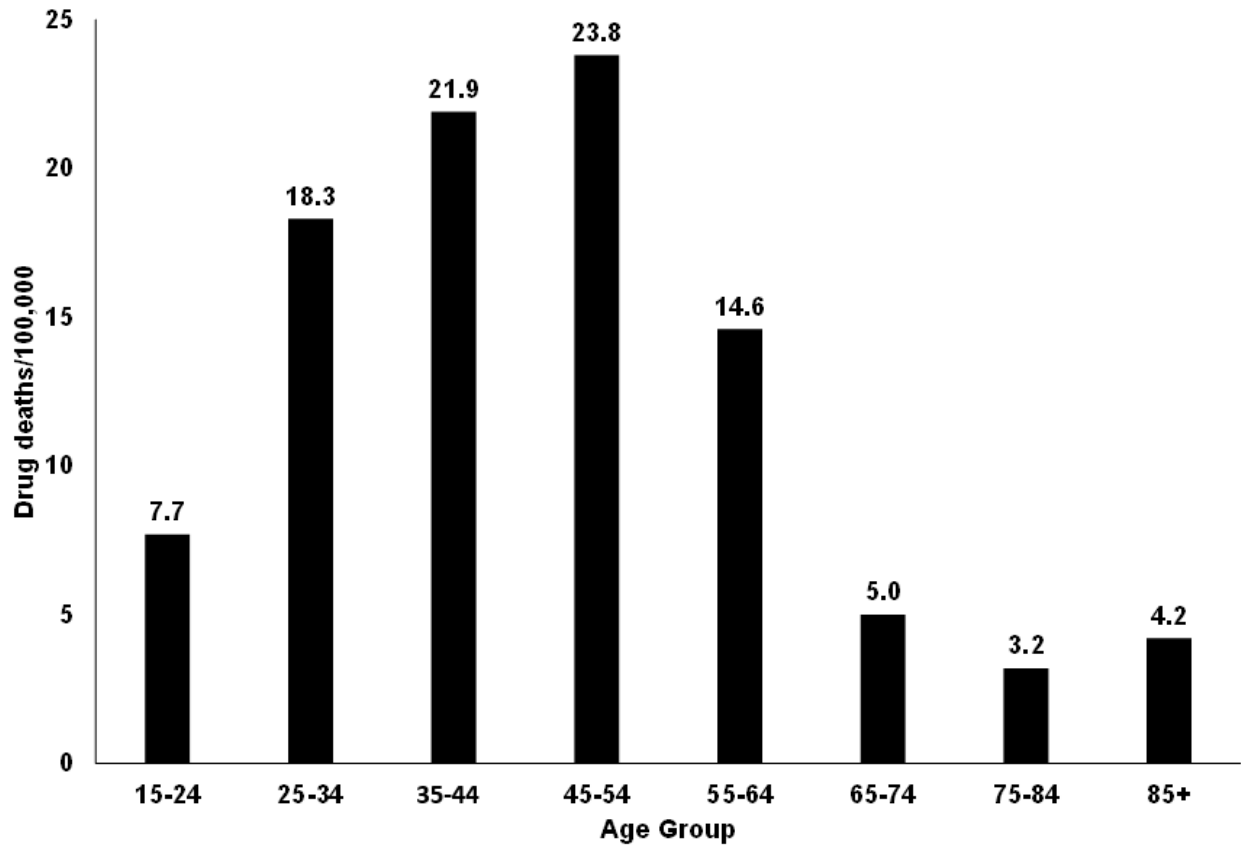
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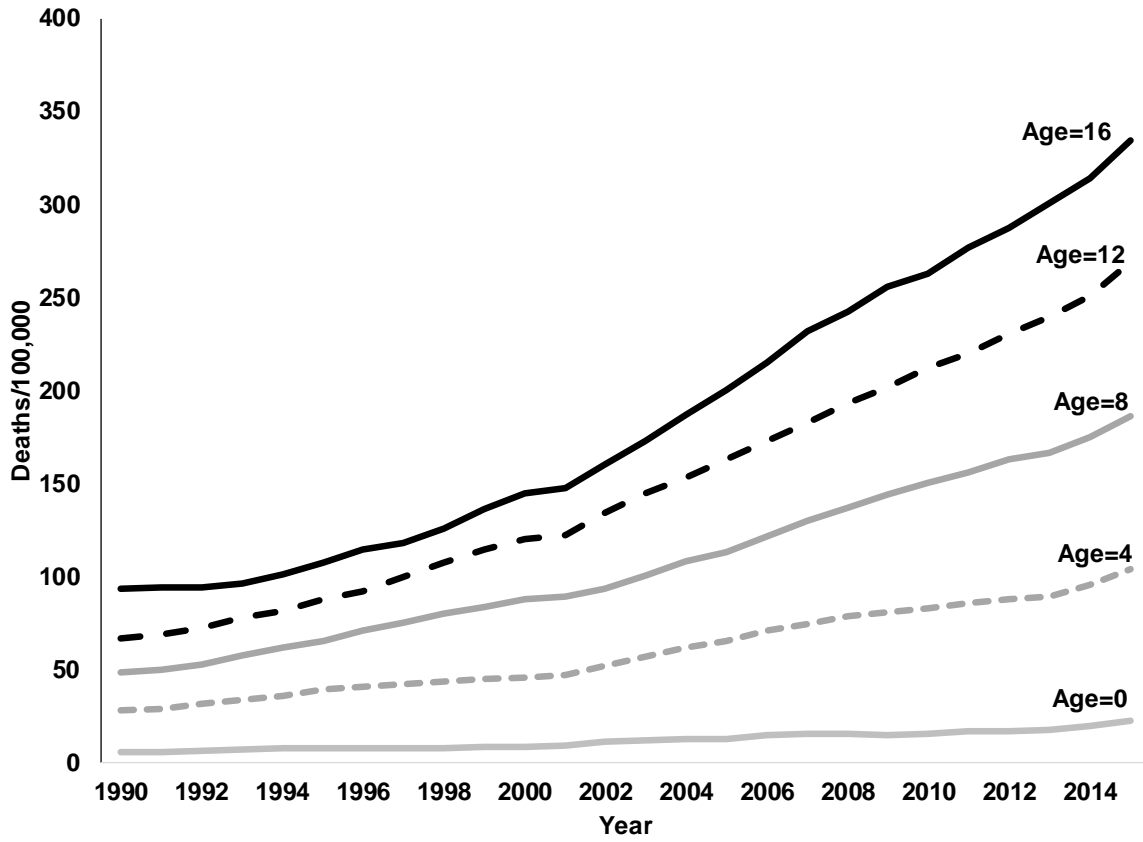
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Figure 1: Drug Death Rate by Age in the United States, 1999-2015



Data are from the 1999-2015 Multiple Cause of Death Files.

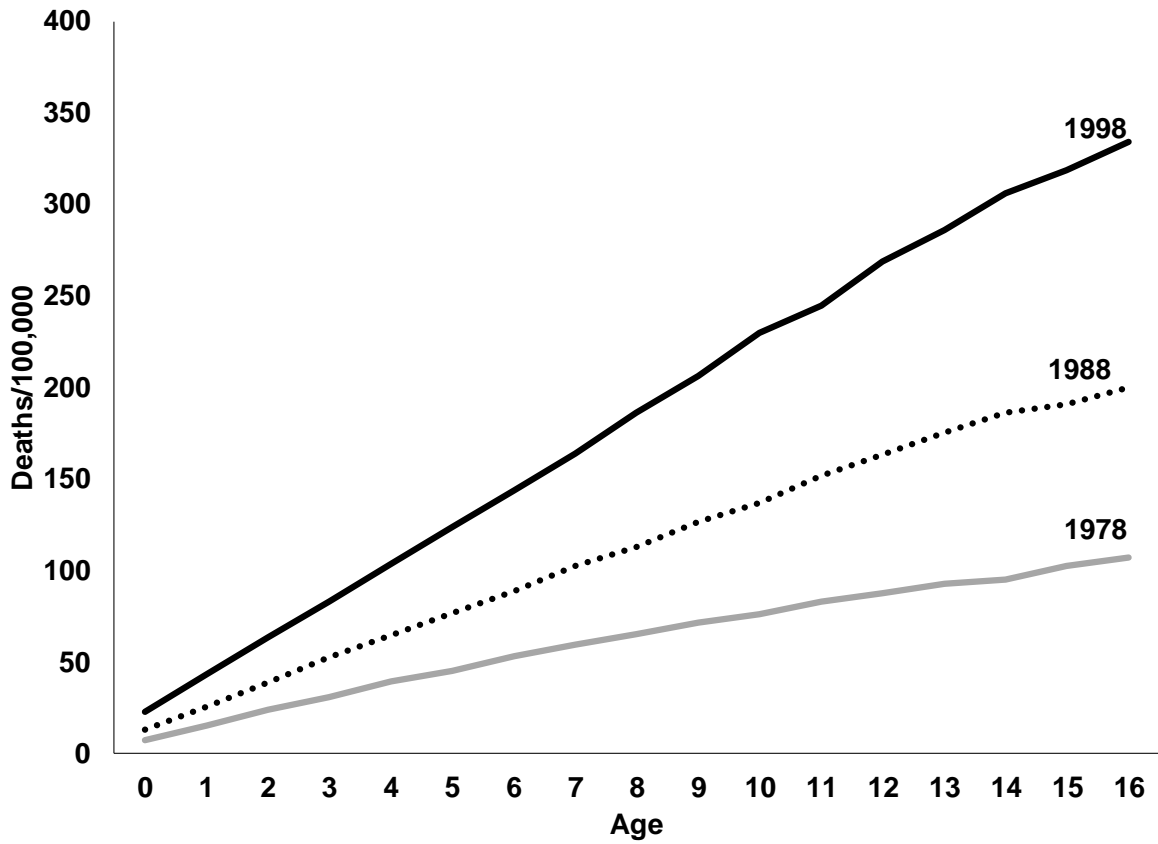
Figure 2: Cumulative Drug Death Rate of Likely Parents of Children Ages 0-16, 1990-2015, Multiple Cause of Death Data



Data are from the Multiple Cause of Death Files; see the text for details on how this measure is constructed.

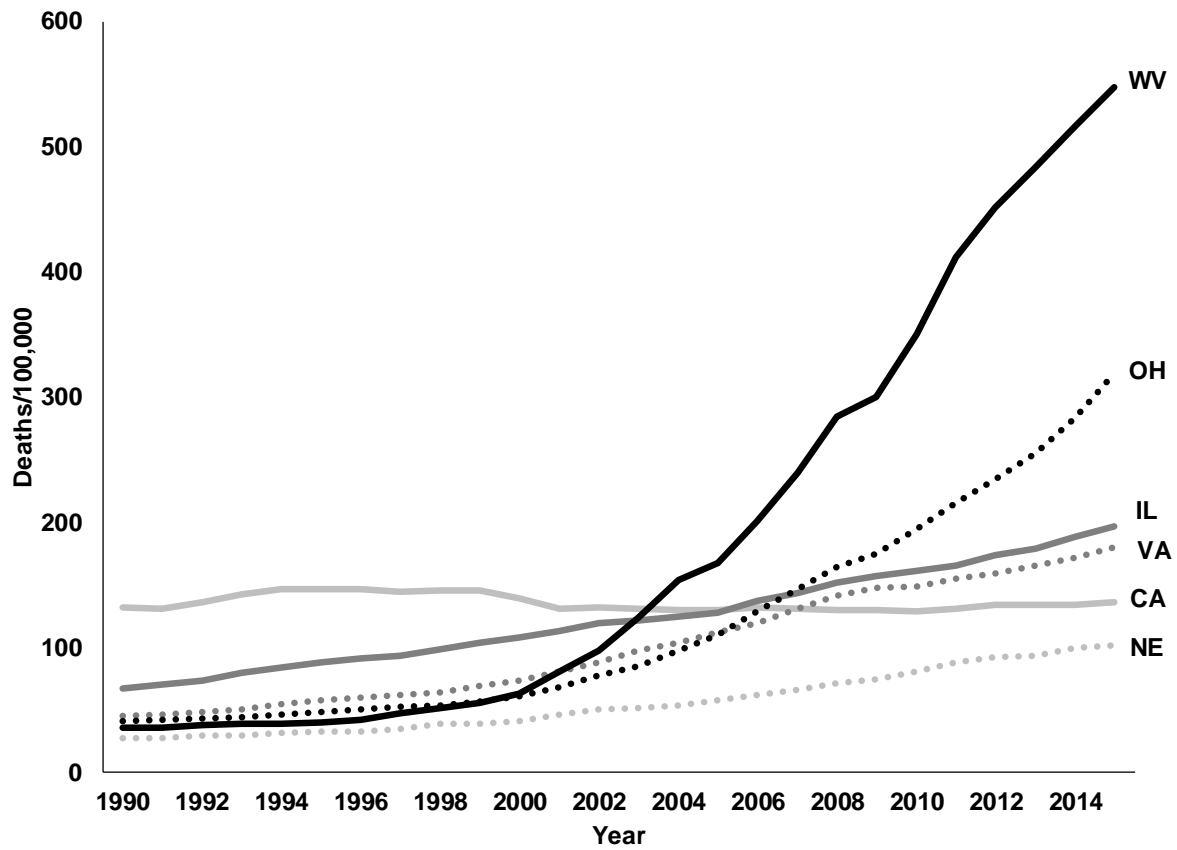


Figure 3: Cumulative Drug Poisoning Death Rate of Likely Parents by Age for Various Birth Cohorts, Multiple Cause of Death Data



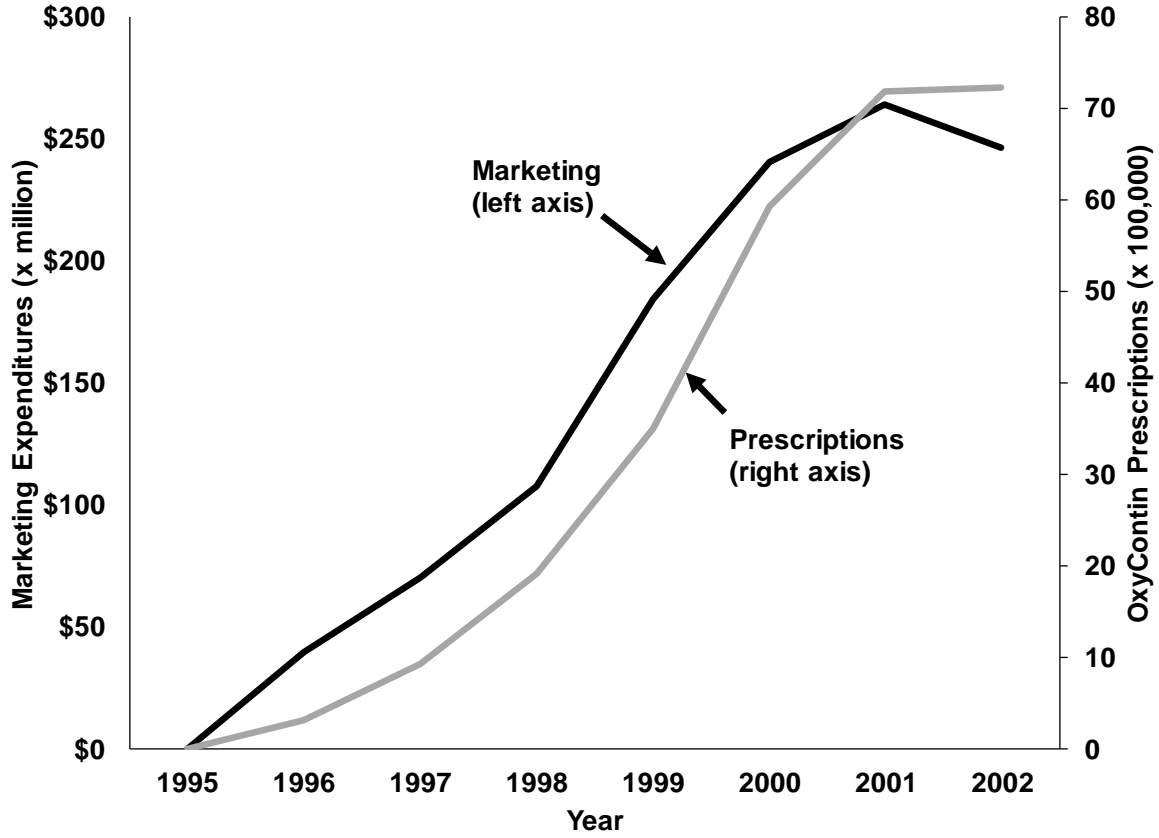
Data are from the Multiple Cause of Death Files; see the text for details on how this measure is constructed.

Figure 4: Cumulative Drug Poisoning Death Rate of Likely Parents for Children Aged 10 by State of Residence, 1990-2015, Multiple Cause of Death Data



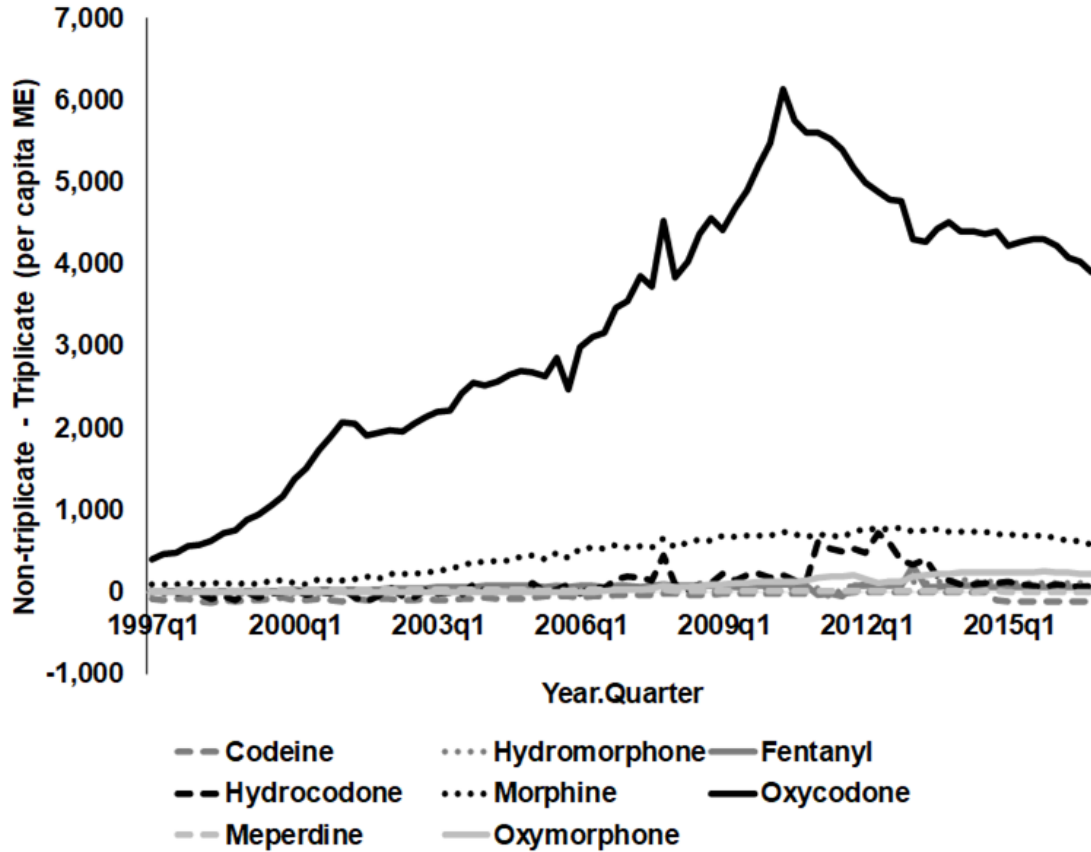
Data are from the Multiple Cause of Death Files; see the text for details on how this measure is constructed.

Figure 5: OxyContin Marketing and Prescriptions Over Time



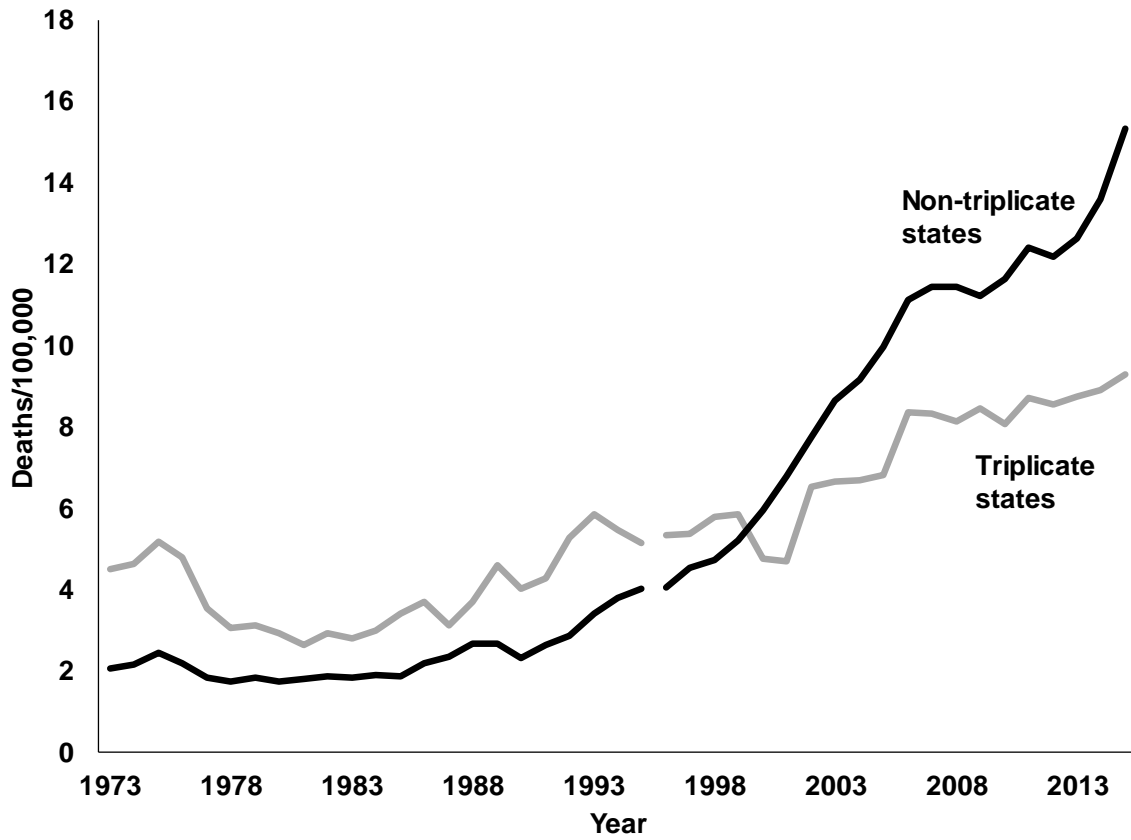
Data drawn from Purdue's *Annual Budget Plan* from 1996-2001. Figures for 2002 are Purdue's estimates made in 2001 for the following year.

Figure 6: Differences in Opioid Use, Non-triplicate minus Triplicate States, ARCOS Data



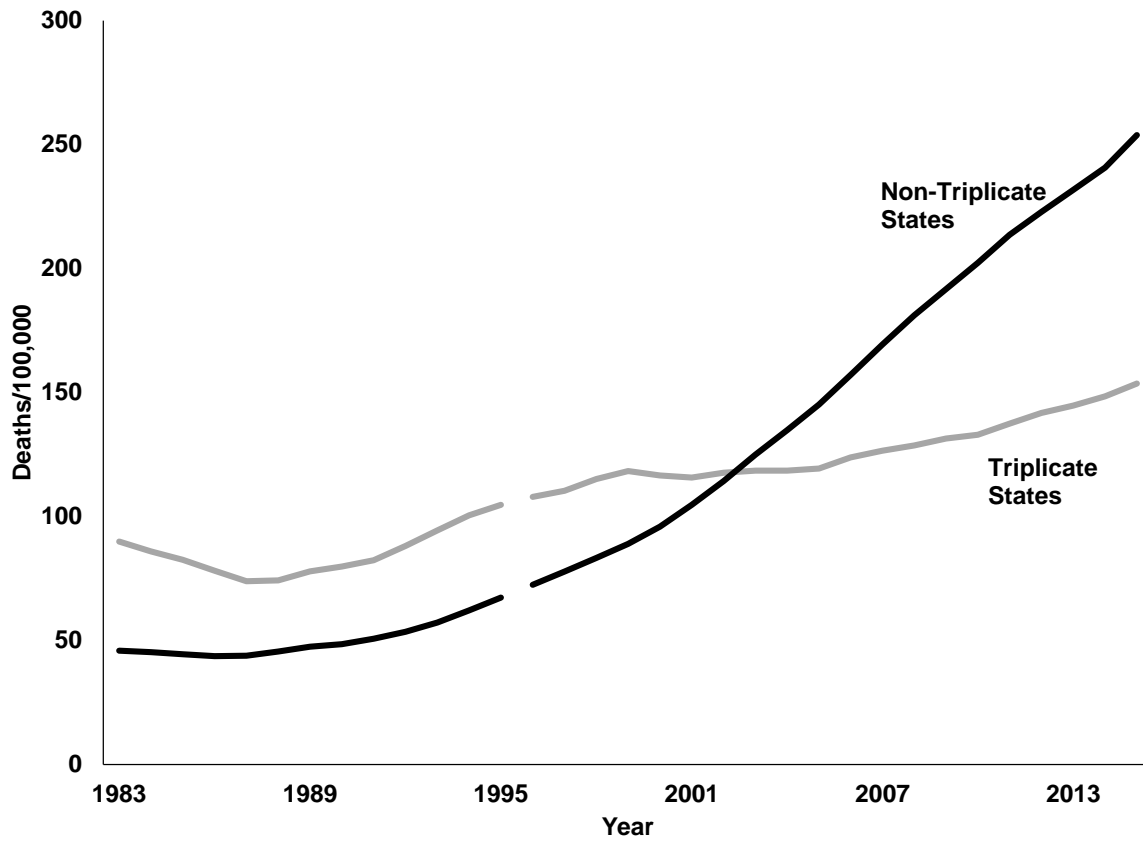
Data are from DEA’s ARCOS system. We use morphine equivalent grams per 100,000 people to put all drugs into comparable units. Codeine and hydrocodone were schedule II drugs during this time frame, but codeine combinations (e.g. Tylenol #3) and hydrocodone combinations (e.g. Vicodin) were schedule III drugs. We cannot differentiate combination from non-combination forms in the ARCOS data. All other listed opioids were schedule II drugs throughout.

Figure 7: Drug Death Rates by Triplicate State Status, 1973-2015, Multiple Cause of Death Data



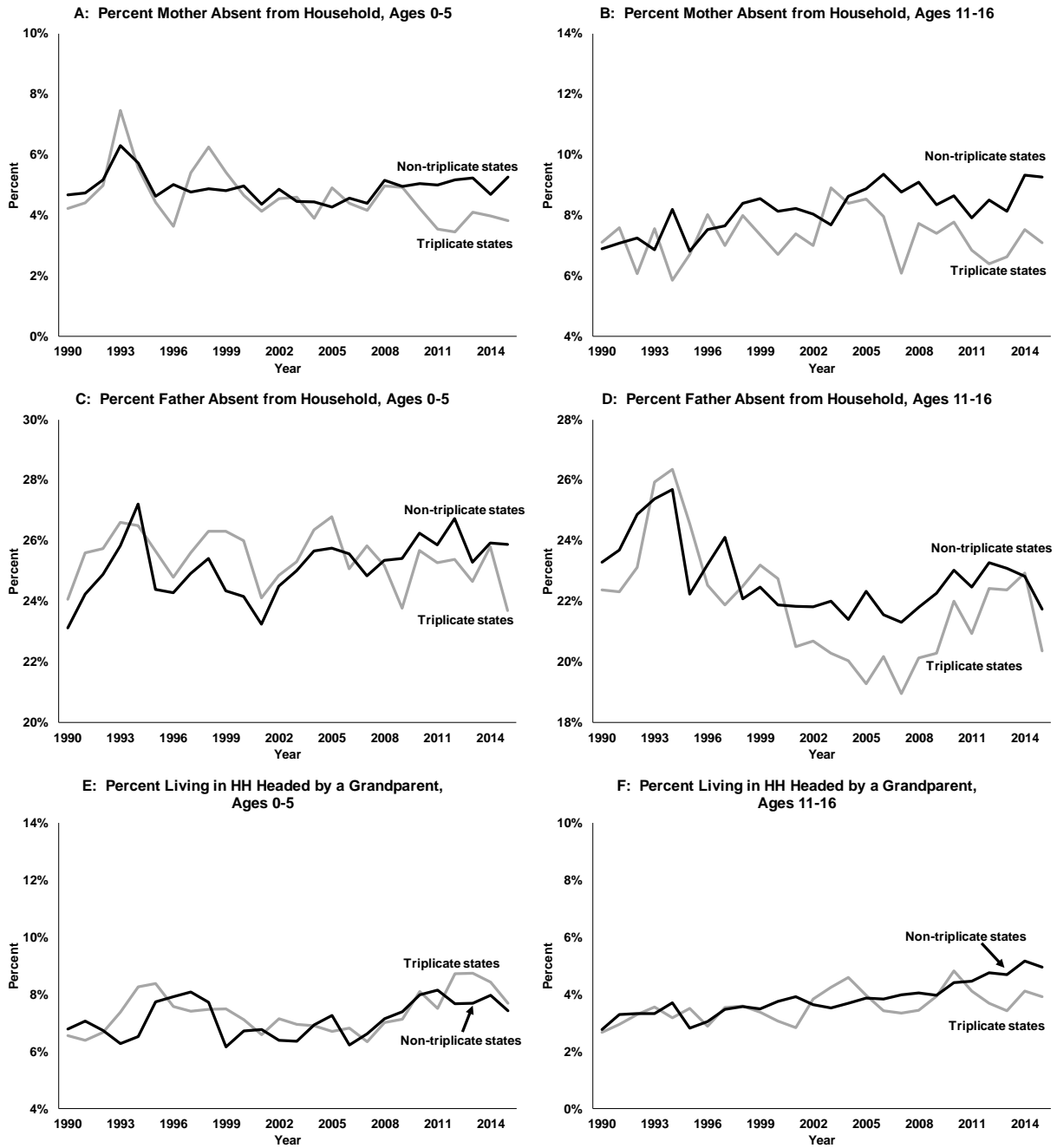
Series break point is 1996, the year Purdue Pharma released OxyContin.

Figure 8: Cumulative Drug Death Rates for Likely Parents of Children Aged 10 by Triplicate State Status, 1983-2015, Multiple Cause of Death Data



Series break point is 1996, the year Purdue Pharma released OxyContin.

**Figure 9: Living Arrangements of Children over Time and by Triplicate State Status**



Data are from the Annual Social and Economic Supplement (ASEC) of the March Current Population Survey (CPS). “HH” is an abbreviation for household.

**Table 1: OLS and 2SLS Estimates of the Impact of Cumulative Drug Deaths of Likely Parents on The Living Arrangements of Children, ASEC 1990-2015**

Parameter Estimates (Standard Errors) on  $CEXPOSURE_{ast}$

Dependent variable	Sample mean	OLS	2SLS	p-value Hausman test	Children impacted by movement of the $CEXPOSURE_{ast}$ rate from 1996 to 2015 values	
					Change in rate per 100,000	# impacted
Mom not in household/100K	6,304	10.0 (1.99)	12.88 (3.36) [84.3]	0.207	1,291	862,000
Dad not in household/100K	23,890	9.32 (1.89)	9.68 (4.34) [80.4]	0.925	1,436	954,000
Missing at least one parent/100K	27,294	15.52 (3.00)	18.29 (5.63) [92.7]	0.533	2,279	1,517,000
Missing both Mom and Dad/100K	2,900	4.38 (0.78)	3.64 (1.44) [92.7]	0.532	454	302,000
Grandparent head of HH/100K	5,282	2.09 (1.71)	6.02 (2.17) [92.7]	0.061	750	499,000
Non-parent head of HH/100k	9,533	6.41 (1.94)	11.45 (2.62) [92.7]	0.074	1,427	950,000
Foster child / 100k	309	0.20 (0.19)	-0.07 (0.48) [92.7]	0.510	-8.6	-6,000

All models include fixed effects for age, year and state, plus the fraction of observations that were female, Black (non-Hispanic), other race (non-Hispanic) and Hispanic. The model includes 17 ages x 51 states x 26 years = 22,542 observations. We calculate standard errors allowing for arbitrary correlation in errors at the state level. For the 2SLS estimates, the F-statistic for the first stage is indicated in brackets. The last column represents the number of children in the particular household in 2015 based on movement from the 1996  $CEXPOSURE_{ast}$  based on the 2SLS estimates.



**Table 2: First-Stage Estimates of Cumulative Drug Death Equations, ASEC 1990-2015**

Dependent variable	Sample mean	OLS Coefficient on $YearsExpNT_{ast}$	1 <sup>st</sup> stage F-test
Cumulative drug deaths/100K of likely mothers	75.3	7.36 (0.80)	84.2
Cumulative drug death/100K of likely fathers	146.1	11.1 (1.24)	80.3
Cumulative drug deaths/100K of likely parents	110.9	9.22 (0.96)	92.5

All models include fixed effects for age, year and state, plus the fraction of observations that were female, Black (non-Hispanic), other race (non-Hispanic) and Hispanic. The model includes 17 ages x 51 states x 26 years = 22,542 observations. We calculate standard errors allowing for arbitrary correlation in errors at the state level (in parentheses).

**Table 3: OLS and 2SLS Estimates of the Impact of Cumulative Drug Deaths of Likely Parents on Socioeconomic Outcomes of Children, ASEC 1990-2015**

Parameter Estimates (Standard Errors) on  $CEXPOSURE_{ast}$

Dependent variable	Sample mean	OLS	2SLS	p-value Hausman test	Children impacted the by movement of the $CEXPOSURE_{ast}$ rate from 1996 to 2015 values	
					Change in rate per 100,000	# impacted
# in poverty/100K	19,478	8.49 (3.55)	8.85 (4.54)	0.266	1,045	695,000
# on SNAP/100K	16,064	8.67 (4.23)	10.0 (5.07)	0.592	1,181	785,561
# without health insurance/100K	10,847	4.22 (4.66)	4.37 (5.07)	0.399	516	343,290

All models include fixed effects for age, year and state, plus the fraction of observations that were female, Black (non-Hispanic), other race (non-Hispanic) and Hispanic. The model includes 17 ages x 51 states x 26 years = 22,542 observations. We calculate standard errors allowing for arbitrary correlation in errors at the state level.

## Appendix A

### Identifying Drug and Opioid Deaths in the 1973-2015 MCODE Data

To construct cohort exposure measures for those aged 0-16 from 1990 to 2015 required data from the Multiple Cause of Death data files from 1973 through 2015. Over this period, these data sets used three different version of the International Classifications of Diseases (ICD): ICD 8 (through 1978), ICD 9 (1979 through 1998) and ICD 10 (1999 and on).

Identifying drug overdoses in all three versions of the ICD system is relatively straightforward. In each year, there are three sets of codes that identify unintentional poisoning deaths, intentional poisonings (e.g., suicides), and drug poisoning of unknown intent. These codes vary by the class of drug. ICD 8 has an additional code 304 that measure death due to drug dependence, which is a code under the mental health classifications. This code was dropped in subsequent versions. In the ICD 9 system, code E962 measures death from homicide due to drug poisonings. That code under the ICD 10 classification is X85. We list these codes in Table A1 below.

**Table A1**  
**Codes to Identify Drug Poisonings, ICD 8 through ICD 10**

ICD Era	Unintentional Poisonings	Intentional Poisonings	Poisonings of Unknown Intent	Other codes
ICD-8	E850.0 – E858.9	E950.0 – E950.5	E980.0 – E980.3	304
ICD-9	E850.0 – E858.9	E950.0 – E950.5	E980.0 – E980.3	E962
ICD-10	X40 – X44	X60 – X64	Y10 – Y14	X85

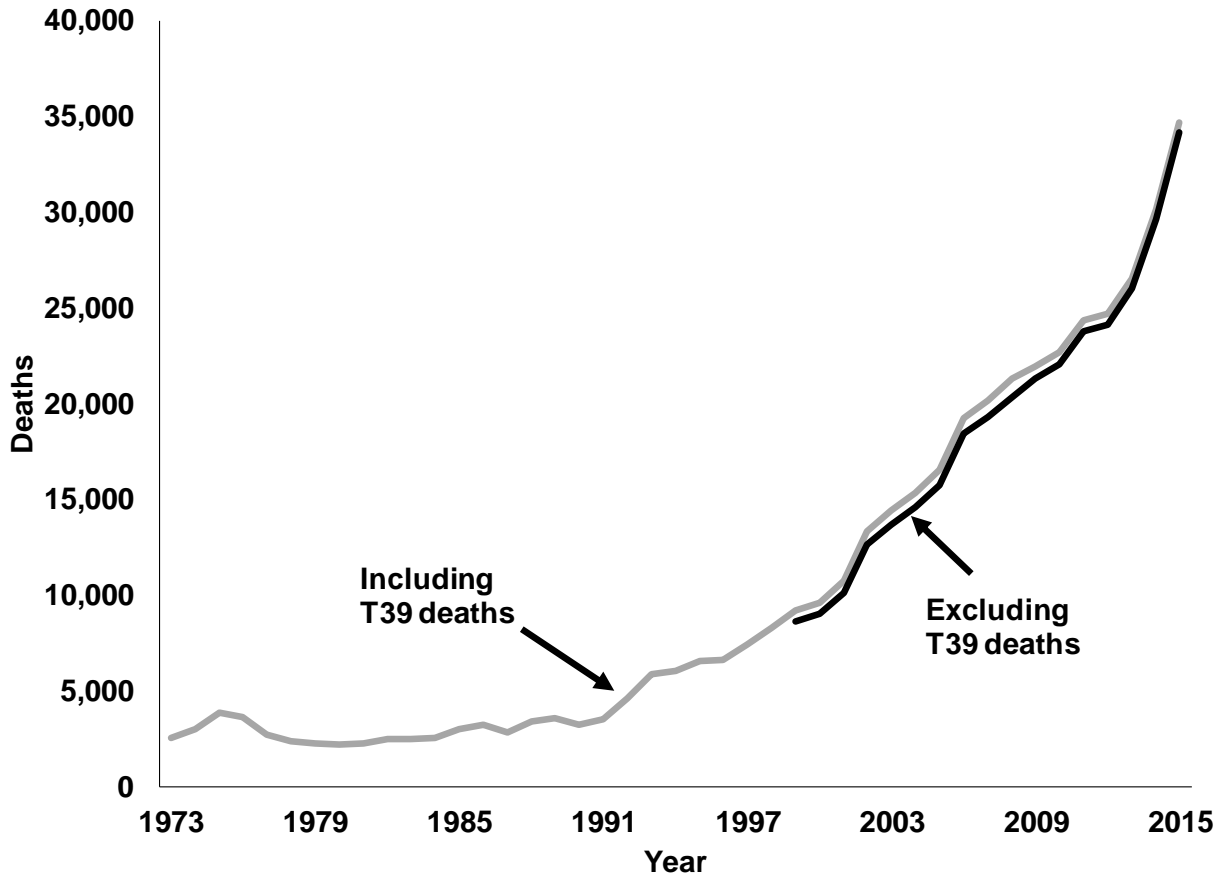
Identifying opioid deaths is relatively easy in ICD 10 as there are codes that identify conditions present at death to indicate specific drugs. These include T40.1 (heroin), T40.2 (other opioids) T40.3 (methadone), and T40.4 (synthetic opioids). Like Alpert et al. (2019), we also include T40.6 (other and unspecified narcotics) as well. There are similar codes in the ICD 9 classifications: 965.0 (opiates and related narcotics), 965.1 (heroin), 965.2 (methadone), 965.9 (other opiates and related narcotics). There is only one condition code that uniquely identifies opioids in the ICD-8 coding: 965.1 (opiates and synthetic analogues).

The problem we found is that in many cases during the ICD 8 and 9 era, the “965” condition codes are frequently not used when there was a drug death. In the ICD 9 era, we can identify opioids in some of the “E” codes – E850.0 (heroin), E850.1 (methadone), and E850.2 (opiates and related narcotics). Unfortunately, categories E950.0 and E980.0 (poisonings by analgesics, antipyretics, and antirheumatics for intentional and unknown intent, respectively) lump opiates in with other drugs (mostly non-opioid pain relievers).

In the ICD 10 era, the T39 condition code identifies non-opioid analgesics, antipyretics and antirheumatics and in 1999, there were only 759 deaths from these drugs, but 8,645 of the T40.x opioid/heroin deaths. As a result, to make a more consistent series without a noticeable jump in opioid deaths as we move from the ICD 10 back to the ICD 9 era, we use a broader opioid death rate category that includes the T39 cases. In the ICD 9 era, we consider the “965” conditions listed above, those that include non-opioid analgesics, and any E850.x code which contains opiates and the non-opioid analgesics, plus deaths with E950.0 and E980.0 codes. For ICD 8 years, we include in the broader opioid death category E5853.x codes which are opiates and other analgesics, E950.1 (suicides by salicylates and congeners), E980.1 (poisoning by salicylates and congeners of undetermined intent), and all 965.x condition codes.

In Figure A1 below, in the gray line, we report trends in the opioid death counts when we include this slightly broader set of drugs. The black line is the trend in opioid deaths that only uses the T40.x codes outlined above. The lines track each other well and the broader definition we use is greater by 501 to 952 deaths/year in the 1999-2015 period.

Figure A1  
Opioid-Related Deaths, 1973 to 2015, Including and Excluding  
Non-opioid Analgesics, Antipyretics and Antirheumatics (T39) Causes



Data are from the Multiple Cause of Death Files, 1973-2015.

## Appendix B

### Sensitivity and Robustness

In this appendix, we probe the sensitivity of our primary estimates to a multitude of sample and specification choices. In addition to these robustness results, we also report clustered wild bootstrap confidence intervals for the first stage of our 2SLS procedure (Appendix Table B1) as well as all subsequent robustness analyses (Appendix Tables B2-B6). For ease of comparison, the first column in most tables in this section contains the basic 2SLS results from Table 1.

#### *Appendix Table B2: Robustness to General Sample and Specification Choices*

In our main analyses, our cumulative risk measure is based on all drug deaths. However, our instrument generates variation most directly in opioid deaths. The drug death measure is more general and more consistently coded across ICD classification systems, but an exposure measure based on opioid deaths is more closely tied to the variation generated by the instrument. In the second column of Appendix Table B2, we show results based on measuring a child's exposure with opioid deaths rather than drug deaths. The ratio of estimates based on opioids to the estimates based on all drug deaths ranges from 1.40 to 1.46; our exposure measure based on drug deaths increased by 124.6 while the measure based on opioids increased by 89.8—a ratio of 1.38. This suggests that approximately all of the changes in family structure we estimate are generated by changes in opioid death rates.

Alpert et al. (2018) and Evans et al. (2019) demonstrate that the reformulation of OxyContin in August of 2010, which made OxyContin more difficult to abuse, encouraged the shift in drug abuse away from prescription opioids towards heroin. Although the reformulation reduced mortality associated with prescription opioids, it increased heroin mortality to the point that the reformulation had no impact on drug mortality in the short run. The market for drugs was systematically changed in 2013 by another supply shock when fentanyl appeared in large scale in illegal drug markets. The rapid increase in mortality experienced in the US from 2013 to 2018 is primarily driven by increasing use of fentanyl and other synthetic opioids. One can argue that OxyContin abuse lead to its reformulation, which then expanded use of heroin, and the heroin market begat the fentanyl market. That said, one could also argue that our instrument can best explain the movement of drug use across triplicate and non-triplicate states prior to the end of 2010. In the third column of Appendix Table B2, we estimate our basic specifications with data only

through 2010. The results are actually slightly larger in magnitude than our baseline estimates and do not suggest that changes in drug deaths in recent years are driving our results.

Exposure to the drug crisis might not only affect the living arrangements of children, but also the probability that a child is born at all. This in turn suggests that the composition of families in which children are living could be affected by the drug crisis. We test this hypothesis by limiting our sample to children born prior to the introduction of OxyContin, 1996. The benefit of this approach is that it precludes the possibility that OxyContin affected the birth of anyone in the sample; the drawback is that it cuts out approximately 50 percent of our sample and our standard errors increase considerably. The results from this exercise are shown in the fourth column of Appendix Table B2. In all but one case, missing at least one parent, the point estimates increase in size. This suggests that children born in non-triplicate states after the introduction of OxyContin tended to be positively selected, born to families less likely to have mothers or fathers absent. This is suggestive evidence that the primary mechanism through which the drug crisis affects children's living arrangements is through impacts on the family after the child is born.

As an additional check, we have included state-specific linear trends in the regressions. These trends will pick up any population, demographic, or other factors which are increasing or decreasing differentially across triplicate and non-triplicate states. Our estimated impacts of drug deaths are presented in the fifth column and again, we do not find results qualitatively different from our main estimates.

#### *Appendix Table B3: Robustness to Additional Controls*

It seems likely that economic wellbeing is an important determinant of both family structure as well as drug death rates. As such, we explore the degree to which various measures of economic conditions could be affecting our results. In the first column of results in Appendix Table B3, we report regressions in which each state's per-capita real GDP has been included as a control variable. The results are extremely similar to our baseline results.

In the second column, we include the state's unemployment rate. Hollingsworth et al. (2017) found a correlation between unemployment rates and drug overdose death rates. Again, our results are largely unaffected by this variable's inclusion. While unemployment rates might capture some of the variation in drug over dose death rates, it does not appear to be driving the portion which is related to family structure.

Using the granting of permanent normalized trade relations with China in 2000, Pierce and Schott (2020) showed that geographic regions most exposed to trade with China saw increases in opioid overdose death rates. To ensure that our results are not being driven by this same trade shock, we interact their measure of exposure with year dummies and include those variables in our regression. The results are very similar to our baseline results, suggesting that our instrument is not providing spurious results in which it happens to capture differences in exposure to trade shocks.

Triplicate programs were early versions of prescription drug monitoring programs (PDMP) systems designed to oversee and discipline the prescribing of controlled substances like opioids. In subsequent years nearly all states adopted some form of PDMP. Although evidence on the effectiveness of these subsequent PDMPs is mixed (e.g. Buchmueller and Carey, 2018), we create three different measures of PDMPs (based on Horwitz et al. (2019)) for each state and include them in the regression. Our measures are indicators for years including and after the state's PDMP 1) was legislated to be active, 2) actually became active (funding, and other, issues delayed many PDMPs), and 3) whether it was a modern, electronic system. As seen in the fourth column of Appendix Table B3, these variables have little impact on our point estimates and suggest that triplicate and non-triplicate states were not differentially enacting opioid-related legislation that was correlated with both cumulative drug mortality and family structure.

Welfare reforms took place at roughly the same time as the introduction of OxyContin, they varied across states, and they have been shown to have affected children's living arrangements (Bitler et al., 2006). Consequently, there is a possibility that our estimation strategy is partially capturing the effects of these policy reforms. Following Bitler et al. (2006), we create two variables which indicate whether the state had obtained a waiver for its Aid to Families with Dependent Children (AFDC) program and the first year in which Temporary Assistance to Needy Families (TANF) was implemented. As seen in the final column of Appendix Table B3, adding these measures has very little impact on our estimated effects.

#### *Appendix Table B4: Robustness to Omitting Treatment States*

In Appendix Table B4, we test whether any single triplicate state is driving our results. We do so by dropping each triplicate, one at a time, and rerunning the 2SLS regression. Although there are slight changes in the point estimate from one sample to the next, the evidence suggests that there was not a single triplicate state solely responsible for the estimated effects.

*Appendix Table B5: Sensitivity to Population-Related Issues*

In 1990, the triplicate states tended to have much greater population and some of the largest cities in the United States. California, New York, and Texas had the largest populations while Illinois had a larger population than all but two non-triplicate states. Clearly, the triplicate states differ from the non-triplicate states in terms of their population and tendency to have large metropolitan areas. If the changes in family structure tended to occur in less populous places (or those declining in population), our instrument might simply be picking up that difference between triplicate and non-triplicate states. In Appendix Table B5, the second column of results reports estimates from regressions in which we have included a fourth order polynomial in the states' populations of adults of child-bearing age. Three of the five estimates increase slightly in magnitude while the other two decrease very slightly. Overall, flexibly controlling for a state's population has little impact on the results.

Linked to the differences in population between triplicate and non-triplicate states, the triplicate states contain the largest cities in the United States. Instead of pure population, it might be that changes in family structure are actually caused by residing in an urban environment rather than by drug deaths. To explore this possibility, we have rerun our regressions separately for children living in metro areas and for children who are living in a non-metro area. These results are presented in columns three and four of Appendix Table B5. Although we lose a considerable amount of precision when splitting the sample in this way, our point estimates are quite similar to what we had found previously.<sup>33</sup> Moreover, the point estimates indicate that there are not large differences in our estimated impacts of the drug crisis across more and less urban areas, strongly suggesting our main results are not simply picking up differences in urban status across triplicate and non-triplicate states.

An alternative way to assess the importance of differences in population or urbanicity between triplicate and non-triplicate states is to restrict the sample of states used for the analysis to those with the largest populations. In the fifth column of Appendix Table B5, we present results in which the set of triplicate states has been restricted to California, Illinois, New York, and Texas and

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<sup>33</sup> It is worth noting that the results for metro and non-metro areas do not average up to the overall results. They do not have to do so because we are not restricting all of the other coefficients in the regression (e.g. year effects) to be the same across the two. In addition, there is a small number of individuals—approximately 1 percent—who could not be classified into either metro or non-metro areas. For these regressions, they were omitted from both groups.



the set of non-triplicate states has been restricted to Florida, Pennsylvania, Ohio, and Michigan. These were the eight most populous states in 1990. The benefit of this restriction is that our non-triplicate states are much more similar to our triplicate states in terms of population and urbanicity; the cost is a considerable loss of statistical power. Even with this severe restriction, our point estimates tend to be quite similar to those we obtain when using all non-triplicate states in the regression.

*Appendix Table B6: Heterogeneity by Race*

Because there are large baseline differences in the probability of living in different family structures for white and Black children, it is interesting to consider whether the effects of exposure to the crisis differ by race. To assess this possibility, we regress race-specific measures of family living arrangements on race-specific measures of exposure to the crisis. We report these results in Appendix Table B6. The first and third columns present means of the various living arrangement variables for white children and Black children respectively; these outcomes tend to be much more common among Black children. They are roughly twice as likely to live without a mother in the household, three times as likely to live without a father in the household, and 2.5 times as likely to live with a grandparent as the household head. The 2SLS results are presented in the second column for white children and the fourth column for Black children. We find that increased exposure to the drug crisis had substantial impacts on white children. These estimates are quite similar to our overall estimates and despite splitting the sample, retain reasonable precision. However, our estimates for Black children are far noisier, making it more difficult to reject a wide range of values. Though highly speculative, the implied total effect of the drug crisis implied by our point estimates is actually smaller in percentage terms for Black children than for white children for almost all of the outcomes.<sup>34</sup>

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<sup>34</sup> For both white and Black children, we calculate the change in exposure between 1996 and 2015, multiply that by the estimated effect of exposure, and then divide by the sample mean for the outcome, using race-specific measures at each step. The smaller effect in percentage terms for Black children is due both to the much higher rates of these outcomes experienced by Black children, but also a much lower change in exposure to the drug crisis. Between 1996 and 2015, our measure of exposure rose by roughly 17 for Black children, but by 150 for white children. We have also run the analysis for white children and Black children using the overall measure of exposure to the crisis. In those analyses, we would expect to see larger percentage impacts of the rise in the drug crisis on white children because of their larger exposure, and this is indeed what we find.

**Appendix Table B1**  
**First-Stage Estimates of Cumulative Drug Death Equations, ASEC 1990-2015**

Dependent variable	Sample mean	OLS Coefficient on <i>YearsExpNT<sub>ast</sub></i>	1 <sup>st</sup> stage F-test
Cumulative drug deaths/100K of likely mothers	75.3	7.36 (0.80) [5.78, 9.37]	84.2
Cumulative drug death/100K of likely fathers	146.1	11.1 (1.24) [8.28, 13.63]	80.3
Cumulative drug deaths/100K of likely parents	110.9	9.22 (0.96) [7.25, 11.44]	92.5

All models include fixed effects for age, year and state, plus the fraction of observations that were female, Black (non-Hispanic), other race (non-Hispanic) and Hispanic. The model includes 17 ages x 51 states x 26 years = 22,542 observations. We calculate standard errors allowing for arbitrary correlation in errors at the state level (in parentheses); clustered wild bootstrap 95% confidence intervals presented in brackets.

**Appendix Table B2**  
**2SLS Estimates of the Impact of Cumulative Drug Deaths of Likely Parents on the Living Arrangements of Children: Alternative Sample and Specification Choices**

Parameter Estimates (Standard Errors) [cluster wild bootstrap 95% CI] on  $CEXPOSURE_{i,t}$

Dependent variable	2SLS from Table 1	Use Opioid Death Rate	Restrict to Year < 2011	Restrict to Born < 1996	State-specific Time Trends
Mom not in household/100K	12.88 (3.36) [5.50, 20.33]	18.87 (4.99) [8.29, 29.35]	20.38 (4.80) [10.39, 31.14]	16.24 (9.18) [-3.05, 35.85]	10.30 (2.37) [5.61, 14.94]
Dad not in household/100K	9.68 (4.34) [-2.01, 19.12]	13.59 (6.38) [-4.29, 27.21]	16.50 (7.40) [-3.20, 32.51]	9.48 (14.95) [-35.96, 48.00]	7.51 (3.82) [0.37, 14.78]
Missing at least one parent/100K	18.29 (5.63) [2.38, 30.13]	26.11 (8.44) [2.19, 43.25]	29.47 (9.99) [2.57, 51.75]	12.18 (19.77) [-45.78, 56.36]	16.82 (4.55) [7.77, 26.30]
Missing both Mom and Dad/100K	3.64 (1.43) [0.28, 6.62]	5.20 (2.12) [0.22, 9.66]	6.53 (1.82) [2.28, 10.33]	11.80 (3.39) [4.27, 20.71]	0.63 (1.40) [-2.00, 3.34]
Grandparent head of HH/100K	6.02 (2.17) [1.60, 11.12]	8.59 (3.19) [2.34, 15.74]	6.86 (2.91) [1.28, 13.87]	6.38 (6.34) [-8.38, 26.63]	8.39 (3.37) [0.74, 16.34]
Non-parent head of HH/100k	11.45 (2.62) [6.10, 17.53]	16.35 (3.67) [9.09, 24.87]	17.37 (4.07) [8.95, 26.37]	12.35 (7.82) [-5.70, 36.80]	14.81 (4.49) [4.45, 25.89]
Foster child / 100k	-0.07 (0.48) [-1.10, 0.95]	-0.10 (0.69) [-1.61, 1.37]	0.94 (0.83) [-0.69, 2.97]	1.96 (1.47) [-0.88, 6.56]	-0.43 (0.54) [-1.53, 0.62]

Data are from the Annual Social and Economic Supplement (ASEC) of the March Current Population Survey (CPS). All models include fixed effects for age, year, and state, plus the fraction of observations that were female, Black (non-Hispanic), other race (non-Hispanic) and Hispanic. The model includes 17 ages x 51 states x 26 years = 22,542 observations. Standard errors clustered by state are reported in parentheses; 95% confidence intervals estimated via a clustered (at state) wild bootstrap reported in brackets. Baseline results reproduced in the first column. The second column uses the (potentially noisily measured) opioid death rate to construct children's exposure. The third column restricts the sample to 2010 or earlier to avoid OxyContin's reformulation. The fourth column restricts the sample to those born before OxyContin's introduction. The final column includes state-specific linear time trends.

**Appendix Table B3**  
**2SLS Estimates of the Impact of Cumulative Drug Deaths of Likely Parents on the**  
**Living Arrangements of Children: Additional Controls**

Parameter Estimates (Standard Errors) [cluster wild bootstrap 95% CI] on  $CEXPOSURE_{ast}$

Dependent variable	2SLS from Table 1	State Per-capita Real GDP	Unemp. Rate	Trade Shock	Rx Drug Monitoring Programs	Welfare Reform Controls
Mom not in household/100K	12.88 (3.36) [5.50, 20.33]	13.23 (3.40) [6.03, 20.60]	13.16 (3.24) [6.15, 20.26]	12.78 (3.37) [5.69, 19.95]	12.07 (3.02) [5.55, 18.72]	12.49 (3.29) [5.30, 19.53]
Dad not in household/100K	9.68 (4.34) [-2.01, 19.12]	9.03 (4.66) [-3.39, 19.03]	9.64 (4.31) [-2.76, 18.80]	9.44 (4.44) [-3.15, 18.71]	8.62 (4.02) [-1.57, 17.23]	8.88 (3.70) [-1.47, 17.00]
Missing at least one parent/100K	18.29 (5.63) [2.38, 30.13]	17.74 (5.91) [2.22, 31.30]	18.29 (5.57) [2.53, 30.45]	17.90 (5.76) [1.44, 30.61]	17.19 (5.08) [3.53, 28.84]	17.28 (4.82) [3.89, 27.64]
Missing both Mom and Dad/100K	3.64 (1.43) [0.28, 6.62]	3.71 (1.50) [0.47, 6.75]	3.82 (1.36) [0.51, 6.50]	3.67 (1.45) [0.20, 6.57]	2.85 (1.35) [-0.24, 5.72]	3.36 (1.40) [0.12, 6.22]
Grandparent head of HH/100K	6.02 (2.17) [1.60, 11.12]	5.34 (2.64) [-0.28, 11.59]	5.99 (2.19) [1.87, 11.22]	6.08 (2.22) [1.58, 11.26]	5.72 (1.97) [1.91, 10.01]	5.63 (2.04) [1.64, 10.24]
Non-parent head of HH/100k	11.45 (2.62) [6.10, 17.53]	11.40 (3.12) [4.92, 17.82]	11.50 (2.66) [6.25, 17.47]	11.61 (2.70) [6.46, 17.76]	11.38 (2.64) [5.77, 16.84]	11.05 (2.58) [5.77, 16.96]
Foster child / 100k	-0.07 (0.48) [-1.10, 0.95]	-0.06 (0.52) [-1.20, 1.06]	-0.06 (0.49) [-1.15, 1.02]	-0.06 (0.48) [-1.04, 0.91]	0.02 (0.49) [-0.96, 1.08]	-0.10 (0.49) [-1.12, 0.94]

Data are from the Annual Social and Economic Supplement (ASEC) of the March Current Population Survey (CPS). All models include fixed effects for age, year, and state, plus the fraction of observations that were female, Black (non-Hispanic), other race (non-Hispanic) and Hispanic. The model includes 17 ages x 51 states x 26 years = 22,542 observations. Standard errors clustered by state are reported in parentheses; 95% confidence intervals estimated via a clustered (at state) wild bootstrap reported in brackets. Baseline results reproduced in the first column. The second column controls for state-level per-capita real GDP. The third column controls for a state's unemployment rate. The fourth column includes a control for the 2001 trade shock interacted with year dummies. The fifth column includes controls for prescription drug monitoring programs. The final column includes controls for the welfare reforms that occurred in the 1990s.

**Appendix Table B4**  
**2SLS Estimates of the Impact of Cumulative Drug Deaths of Likely Parents on the**  
**Living Arrangements of Children: Omitting Each Treatment State**

Parameter Estimates (Standard Errors) [cluster wild bootstrap 95% CI] on  $CEXPOSURE_{ast}$

Dependent variable	2SLS from Table 1	Drop Texas	Drop New York	Drop Illinois	Drop Idaho	Drop California
Mom not in household/100K	12.88 (3.36) [5.50, 20.33]	12.99 (3.49) [4.99, 21.25]	13.65 (3.58) [5.723, 21.72]	13.09 (3.50) [5.81, 20.93]	12.54 (3.36) [5.33, 20.01]	9.76 (2.20) [5.42, 13.93]
Dad not in household/100K	9.68 (4.34) [-2.01, 19.12]	12.71 (3.47) [3.84, 20.61]	10.15 (4.82) [-5.34, 19.85]	7.28 (3.85) [-3.68, 15.65]	10.07 (4.41) [-2.84, 19.24]	7.67 (5.19) [-4.55, 21.16]
Missing at least one parent/100K	18.29 (5.63) [2.38, 30.13]	21.87 (4.55) [8.85, 31.53]	19.53 (6.11) [0.03, 32.16]	15.98 (5.50) [1.48, 28.46]	18.62 (5.67) [2.47, 31.16]	14.46 (5.69) [1.64, 29.11]
Missing both Mom and Dad/100K	3.64 (1.43) [0.28, 6.62]	3.79 (1.47) [0.19, 6.68]	3.55 (1.58) [-0.28, 6.87]	3.15 (1.45) [-0.25, 6.19]	3.54 (1.45) [-0.01, 6.52]	2.60 (1.37) [-0.42, 5.28]
Grandparent head of HH/100K	6.02 (2.17) [1.60, 11.12]	4.46 (1.54) [1.61, 7.76]	5.71 (2.34) [1.24, 11.71]	5.87 (2.21) [1.35, 11.27]	6.06 (2.18) [1.68, 11.26]	7.22 (2.18) [2.65, 11.92]
Non-parent head of HH/100k	11.45 (2.62) [6.10, 17.53]	9.54 (1.80) [5.81, 13.21]	12.33 (2.69) [6.93, 18.28]	11.15 (2.65) [5.47, 17.77]	11.38 (2.63) [5.91, 17.50]	11.83 (2.98) [5.43, 18.68]
Foster child / 100k	-0.07 (0.48) [-1.10, 0.95]	-0.26 (0.49) [-1.39, 0.73]	-0.11 (0.54) [-1.54, 0.93]	-0.38 (0.42) [-1.48, 0.50]	0.05 (0.47) [-0.98, 1.02]	-0.05 (0.60) [-1.51, 1.25]

Data are from the Annual Social and Economic Supplement (ASEC) of the March Current Population Survey (CPS). All models include fixed effects for age, year, and state, plus the fraction of observations that were female, Black (non-Hispanic), other race (non-Hispanic) and Hispanic. The model includes 17 ages x 51 states x 26 years = 22,542 observations. Standard errors clustered by state are reported in parentheses; 95% confidence intervals estimated via a clustered (at state) wild bootstrap reported in brackets. Baseline results reproduced in the first column. Additional columns drop each triplicate state, one at a time.

**Appendix Table B5**  
**2SLS Estimates of the Impact of Cumulative Drug Deaths of Likely Parents on the Living Arrangements of Children: Sensitivity to Population-Related Issues**

Parameter Estimates (Standard Errors) [cluster wild bootstrap 95% CI] on  $CEXPOSURE_{ast}$

Dependent variable	2SLS from Table 1	Polynomial in Population	Restrict to Metro Areas	Restrict to Non-metro Areas	Largest Population States
Mom not in household/100K	12.88 (3.36) [5.50, 20.33]	11.18 (2.60) [5.52, 16.94]	11.48 (3.35) [3.89, 18.18]	10.33 (3.79) [2.73, 18.48]	10.82 (7.58) [-10.13, 28.43]
Dad not in household/100K	9.68 (4.34) [-2.01, 19.12]	11.95 (4.43) [3.18, 22.10]	6.50 (4.09) [-2.91, 15.75]	8.89 (7.10) [-6.82, 23.99]	3.64 (4.99) [-9.76, 16.19]
Missing at least one parent/100K	18.29 (5.63) [2.38, 30.13]	20.78 (5.48) [9.03, 34.37]	14.66 (5.37) [1.73, 25.90]	16.12 (9.13) [-3.90, 36.73]	10.18 (8.32) [-13.74, 30.43]
Missing both Mom and Dad/100K	3.64 (1.43) [0.28, 6.62]	2.53 (1.62) [-1.34, 5.77]	2.19 (1.35) [-1.05, 4.88]	2.87 (1.65) [-0.59, 6.14]	2.14 (1.80) [-3.39, 7.06]
Grandparent head of HH/100K	6.02 (2.17) [1.60, 11.12]	8.71 (2.54) [3.08, 14.39]	3.46 (2.49) [-1.93, 9.39]	6.25 (3.12) [-0.66, 12.77]	6.09 (2.61) [-1.46, 13.66]
Non-parent head of HH/100k	11.45 (2.62) [6.10, 17.53]	14.78 (3.96) [5.76, 24.58]	7.63 (2.59) [2.35, 14.12]	11.83 (3.80) [3.85, 19.39]	8.91 (2.65) [1.47, 15.14]
Foster child / 100k	-0.07 (0.48) [-1.10, 0.95]	-0.20 (0.57) [-1.45, 0.97]	0.11 (0.53) [-0.95, 1.47]	-0.70 (0.78) [-2.40, 0.83]	-0.12 (0.70) [-1.73, 1.79]

Data are from the Annual Social and Economic Supplement (ASEC) of the March Current Population Survey (CPS). All models include fixed effects for age, year, and state, plus the fraction of observations that were female, Black (non-Hispanic), other race (non-Hispanic) and Hispanic. The model includes 17 ages x 51 states x 26 years = 22,542 observations. Standard errors clustered by state are reported in parentheses; 95% confidence intervals estimated via a clustered (at state) wild bootstrap reported in brackets. Baseline results reproduced in the first column. The second column includes a fourth order polynomial in the state's parent-aged population. The third column restricts the sample to children living in urban areas. The fourth column restricts the sample to children living in non-metro areas. The fifth column restricts the sample to the four triplicate states with large populations (California, Illinois, New York, and Texas) as well as the four non-triplicate states with the largest populations as of 1990 (Florida, Pennsylvania, Ohio, and Michigan).

**Appendix Table B6**  
**2SLS Estimates of the Impact of Cumulative Drug Deaths of Likely Parents on the**  
**Living Arrangements of Children by Race**

Parameter Estimates (Standard Errors) [cluster wild bootstrap 95% CI] on  $CEXPOSURE_{at}$

Dependent variable	Mean for White Children	Restrict to White Children	Mean for Black Children	Restrict to Black Children
Mom not in household/100K	5,494	10.83 (3.07) [4.28, 17.58]	10,363	19.52 (19.92) [-14.48, 67.13]
Dad not in household/100K	18,318	7.24 (4.71) [-6.21, 18.31]	56,477	52.42 (50.33) [-56.23, 179.70]
Missing at least one parent/100K	21,603	15.49 (6.55) [-1.83, 31.18]	60,379	61.24 (47.19) [-14.05, 226.30]
Missing both Mom and Dad/100K	2,208	1.86 (1.15) [-0.87, 4.37]	6,461	13.62 (13.98) [-8.52, 49.79]
Grandparent head of HH/100K	4,214	5.91 (1.88) [0.84, 9.77]	10,470	16.44 (22.12) [-22.91, 73.85]
Non-parent head of HH/100k	7,935	10.30 (2.62) [3.73, 16.12]	16,739	45.21 (31.44) [-3.45, 144.40]
Foster child / 100k	241	-0.50 (0.32) [-1.15, 0.11]	631	7.53 (6.84) [-4.43, 30.36]

Data are from the Annual Social and Economic Supplement (ASEC) of the March Current Population Survey (CPS). All models include fixed effects for age, year, and state, plus the fraction of observations that were female and the fraction that were Hispanic. The model for Black children includes 18,013 observations; the models for white children include 22,529. This difference is due to some age-state-year combinations having zero children in that cell. Standard errors clustered by state are reported in parentheses; 95% confidence intervals estimated via a clustered (at state) wild bootstrap reported in brackets. For the estimate of the effect on “Dad not in household” for Black children, the conventional clustered wild boot strap would not find an upper limit to the confidence interval, and so we use a subcluster wild bootstrap as suggested by MacKinnon and Webb (2018).

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