# The Drug Crisis and the Living Arrangements of Children

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**Abstract**

We examine the impact of the US drug crisis on children's living arrangements. Because factors that lead to drug use could also alter family structure, we instrument for the intensity of the drug crisis with cross-state exposure to marketing of the prescription opioid at the epicenter of the crisis. We find that the crisis increased the likelihood that a child lives away from a parent or in a household headed by a grandparent. Our results suggest that if drug use had remained at 1996 levels, 1.5 million fewer children aged 0-16 would have lived away from a parent in 2015.

Keywords: opioid crisis, drug crisis, child living arrangements, foster care

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## I. Introduction

The US is now in its third decade of a devastating drug crisis, with roughly 92,000 drug deaths in 2020 alone. Between 1999 and 2020, over 932,000 people died of drug poisonings—about the same number of US soldiers that died in all armed conflicts from the Civil War through the present day (about 990,000).[1] As the drug crisis has developed, research has documented the impact of the epidemic on employment (Aliprantis and Schweitzer, 2018; Harris et al., 2020; Currie et al., 2019; Powell, 2021a), crime (Doleac and Mukherjee, 2022; Szalavitz and Rigg, 2017; Dave et al., 2021), infant health (Ziedan and Kaestner, 2020), marriage (Kaestner and Ziedan, 2020), enrollment in disability insurance programs (Cutler et al., 2017), and intimate partner violence (Stone and Rothman, 2019).

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 (deaths/100,000) between 1999 and 2019 was highest for those between the ages of 25 and 54—an age range in which many will be parents of young children and adolescents. 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 leave the household voluntarily, or be 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 the drug use results in child abuse or neglect, 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 them from the consequences of the parent's use. Finally, a child's parent could have been one of the many people who have died because of the crisis.

There is abundant anecdotal evidence in the popular press which suggests that many children are indeed experiencing these kinds of events. A 2016 article in the Wall Street Journal reported that "Social workers say the scale of the trouble exceeds anything they saw during the crack-cocaine or methamphetamine crises of previous decades" and quoted a child welfare worker who warned 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

[1] https://fas.org/sgp/crs/natsec/RL32492.pdf

the opioid crisis—especially in states that have been hard-hit.[2] National statistics support the observations in these stories—between 1980 and 2018, the fraction of children living in a household headed by a grandparent more than doubled from 3.7 to 8.3 percent.[3]

In recent years, researchers have documented a relationship between the drug crisis and foster care. The number of children entering or living in foster care and the fraction of removals from the home related to drugs have increased (Meinhofer and Anglero-Diaz, 2019). Counties with higher opioid poisoning death rates or drug prescriptions have been shown to have more foster care entries and child removals (Radel et al., 2018; Quast, 2018; Quast et al., 2018; Quast et al., 2019; Bullinger and Ward, 2021). While some work has shown that state-mandated prescription drug monitoring programs have worked to reduce child welfare removals (Gihleb et al., 2020; Bullinger and Ward, 2021), other work suggests that recent opioid supply restrictions may have increased child maltreatment (Evans et al., 2022).

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 arrangements potentially suffer from both omitted variables bias and reverse causality. Changes in the economy or in the strength of institutions 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. (2022) 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. We use this variation in an instrumental variables framework to estimate the effect of the drug crisis on children's living arrangements.

Second, while the previous literature has focused on the relationship between the crisis and foster care admissions, we consider its effects on parental absence and on the likelihood of living in a household headed by someone other than a parent. These are important outcomes to consider, as many of the pathways we describe above would not necessarily result in a formal foster care placement. Indeed, our results reveal that moves into these more informal arrangements are much more common than moves into the foster care system. For example, at the end of 2015, there were

[2] We list some citations for popular press articles in Appendix E.

[3] Authors' calculations using the 1-percent Census Public Use Micro Samples from the 1980-2000 Census and the 2010 and 2018 American Community Surveys.

135,000 children in the foster care system with parental drug abuse listed as a contributing reason for removal; this is an upper bound on the number of removals due to the opioid crisis as it includes all drugs as well as children who would not have been removed on the basis of the drug abuse alone.[4] Meanwhile, we estimate that in that same year, 1.5 million children aged 0-16 were living away from a mother or a father, and just over 270,000 were living away from *both* parents as a direct result of the crisis. About 941,000 children are in a household with a non-parent as the household head, with 43% of these living in a household headed by a grandparent. We also provide evidence that hundreds of thousands of children are living in households with fewer resources as a result of the crisis.

To conduct this analysis, we use data from the March Annual Social and Economic Supplement (ASEC) to the monthly Current Population Survey (CPS) from 1990 through 2015. We construct age-state-year cell rates of different living arrangements for children aged 0-16, and pair these with estimates of children's exposure to the crisis that are constructed from the National Center for Health Statistics Multiple Cause of Death (MCOD) data from 1973 to 2015. 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. While our measure is based on drug deaths, the death of a parent is not the only way that a child could experience a change in their living arrangements. We view our measure as a proxy for how severe the crisis has been in their state over their lifetime. In OLS models with state, age, and year fixed-effects, we document a strong correlation between this measure and child living arrangements. Our 2SLS estimates suggest that the relationship is causal—OLS and 2SLS results are similar in magnitude and we fail to reject the null hypothesis that the 2SLS estimates are equal to their OLS counterparts for most outcomes. 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 a grandparent.

It is important to note that the variation we exploit corresponds to a particular counterfactual: a world without the drug crisis. While having a child live away from a parent who is using 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*. In

[4] Authors' calculations, 2015 Adoption and Foster Care Analysis and Reporting Systems data.

the penultimate section of the paper, we discuss research from across the social sciences which suggests that the changes in living arrangements 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

A child's living arrangement at a given point in time is a product of all of that child's experiences to that point in their life. That is, it measures the *stock* of a child's experience rather than the *flow*. A fifteen-year old in a given state and year has substantially more exposure to the drug crisis than a one-year old, and the fifteen-year old could have separated from her parent because of her exposure 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, we believe this would miss much of the relevant exposure to the crisis.

We instead construct a variable that proxies for the child's cumulative exposure to the drug crisis. We call this variable $CEXPOSURE_{ast}$, and it measures the cumulative drug death rate of likely parents. The subscripts indicate that the measure will vary 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 MCOD data and can be aggregated to construct a cumulative measure. Although drug deaths are more easily measured than drug use, they are an imperfect measure because (fortunately) not all drug use results in death. In 2017 there were about 72,000 drug deaths, but data from the National Survey on Drug Use and Health 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* from state *s* in year *t*, and divide by the number of likely parents at their birth. We define likely parents as those aged 17 to 40 in the child's year and state of birth.[5] A newborn's exposure is the drug death rate of those 17-40 years old in the child's year and state 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 those among 17-40 year olds in the previous year. More precisely, we define cumulative exposure as:

[5] Data from the 1996 Natality Detail data show that 96 (93) percent of new mothers (fathers) are in this age range.

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

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.[6] We use the restricted access MCOD data from 1973 through 2015 to calculate $CEXPOSURE_{ast}$. Because three different coding schemes were used over this time period and drug deaths were coded more consistently than opioid deaths, we use drug deaths to construct our primary exposure measure.[7]

In Figure 2, we demonstrate how $CEXPOSURE_{ast}$ varies with a child's age, state, and year. In Panel A, we report the national time series for different ages. We note two things about this figure. First, cumulative exposure by a given age increases dramatically over this time period—290 percent for children aged 0, and 255 percent for children aged 16. 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 children has grown as well.

There is also tremendous variation across states in children's cumulative exposure. In Panel B, we report the cumulative exposure over time for ten-year olds from six states: California, Illinois, Nebraska, Ohio, Virginia, and West Virginia. These states represent two states each with low, medium, and high growth in $CEXPOSURE_{ast}$. These also include two triplicate (California and Illinois) and four non-triplicate states, which are at the center of our identification strategy. Rates were similarly low for the latter in 1990, but they grew at very different rates. Nebraska and Virginia see a tripling in our measure 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

### *A. Data on Outcomes*

Our primary data on the living arrangements of children come from the IPUMS processing (Flood et al., 2020) of the ASEC Supplement to the monthly CPS. IPUMS generates variables that

[6] See Appendix D1 for more information on the effects of in- and out-migration on our analyses.

[7] We describe the MCOD data in more detail and report the codes we use to define drug deaths in Appendix B.

match a child to a mother and father in the household, which allows us to determine whether each parent is in the household. The codes do not distinguish among biological, adoptive, or step-parents. There is some noise in linking children when there are multiple subfamilies in households, and IPUMS has explicit rules to match parents to children in these situations.

We use these codes to construct indicators for whether a child is living away from their mother or their father, is missing at least one parent, is missing *both* parents, is in a household headed by anyone other than a parent, is in a household headed by a grandparent, or is in foster care. Note that these categories are not mutually exclusive. A child in a household headed by a grandparent is also living in a household headed by someone who is not the child's parent, and they may also be living away from one or both parents. It is also 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). This is due in part to the fact that relatives 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).[8] This is a non-trivial issue, as foster children have been increasingly likely to be placed with a family member as a result of the Fostering Connections to Success and Increasing Adoptions Act of 2008, which established policies to encourage the practice (Child Welfare Information Gateway, 2019).

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.

[8] 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.

*B. The Econometric Model*

Figure 2 indicates 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

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

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. The vector $X_{ast}$ contains the fraction of a cell that is female, Black (non-Hispanic), other race (non-Hispanic), or Hispanic (non-Hispanic White is the omitted category). The variables $\eta_a$, $\mu_s$, and $\lambda_t$ are age, state, and year 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, yielding 22,542 observations. Because both our outcomes and measure of exposure are expressed as the number of occurrences per 100,000 people, we can 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.

There are two potential sources of bias in our OLS estimates. The first is reverse causation. The non-marital birth rate was increasing through the 1990s and early 2000s, and while divorce rates decreased over this period, divorce remained prevalent (Buckles et al., forthcoming; Lehrer and Son, 2018). As a result, many parents are living away from their children. Case and Deaton (2015; 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 α 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. 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 α would also be overstated as the estimate would be capturing these same factors that are driving both high drug use and more parents living away from 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 use (Hollingsworth et al., 2017; Carpenter et al., 2017) *and* lower marriage rates (Schaller, 2013). To address these sources of bias, we use an instrumental variables (IV) approach that is described in the next two sections.

### C. *The Marketing of OxyContin in Non-Triplicate States*

The rise in drug deaths has been driven primarily by opioids, and the drug at the center of the rise is OxyContin ER (or simply OxyContin), a branded extended-release opioid painkiller marketed by Purdue Pharma. Its active ingredient is oxycodone, an opioid 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. After it was introduced to the market in 1996, users quickly discovered they could produce an intense high by crushing or dissolving it to access all of the drug at once, which led to addiction and abuse (Quinones, 2015).

Internal documents from Purdue Pharma related to the introduction and promotion of OxyContin are now accessible as a result of various lawsuits. The documents reveal that while OxyContin became available throughout the country in January of 1996, Purdue Pharma made strategic decisions to target certain markets and patients.[9] Pre-launch market research suggested that marketing was unlikely to be effective in "triplicate" states that require providers to use a special, serialized pad to prescribe a Schedule II opioid such as OxyContin.[10] When Purdue Pharma launched OxyContin in 1996, California, Idaho, Illinois, New York, and Texas had active triplicate programs.[11] These programs, which were precursors to modern prescription drug monitoring programs, were implemented between 1939 and 1982, well before opioid deaths began rising rapidly.

Purdue Pharma's pre-market 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

[9] As seen in Appendix Figure A1, 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.

[10] 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.

[11] Purdue Pharma's OxyContin Launch Plan (1995a) includes a reference to "nine triplicate states." While these states are not listed, we believe them to be the five states we identified, plus two states that had repealed triplicate laws by 1996 (Michigan and Indiana), and two states with duplicate programs (Hawaii and Rhode Island). Our results are robust to defining all nine states as "triplicate" states, or to including Hawaii and Rhode Island as "triplicate" states while dropping Michigan and Indiana.

evidence is not encouraging." Referring to physicians in triplicate environments, they conclude: "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 Purdue Pharma, 1995b).

Figure 3 traces out differences in per capita opioid sales across non-triplicate and triplicate states over time for specific active ingredients. These data are 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. Unfortunately, the data are only available beginning in 1997. We convert all drugs to a comparable unit, morphine equivalent grams per 100,000 people. Triplicate and non-triplicate states had somewhat similar rates of oxycodone use at the start of 1997, but as Purdue's marketing of OxyContin ramped up, so did the differences in oxycodone 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 are in the same units, the growth is negligible relative to the growth seen for oxycodone, 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 other Schedule II opioids shown in Figure 3 (all except most forms of hydrocodone) 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.[12] See Alpert et al. (2022) for further evidence that Purdue's marketing directly affected drug overdose death rates and was not simply picking up other factors including the triplicate programs themselves, opioid-related policies, changes in economic conditions, or differences between urban and rural areas.

The impact of Purdue Pharma's marketing strategy in triplicate versus non-triplicate states can be seen in Figures 4 and 5. In Figure 4, we report trends in drug death rates for the entire

[12] The triplicate programs were replaced in 1997 (Idaho), 1999 (Texas), 2000 (Illinois), 2001 (New York), and 2004 (California).

population 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.

In Figure 5, we show trends in our cumulative exposure measure for triplicate and non-triplicate states from 1990 through 2015, for older and younger children. Prior to OxyContin's introduction in 1996, we see that cumulative exposure to drug deaths was higher for children in triplicate states, but that the trends for triplicate and non-triplicate states were similar. Because OxyContin marketing and consumption grew gradually, we would not expect an immediate jump in cumulative exposure rates right at 1996, but faster growth which leads non-triplicate states to diverge from triplicates over time. After 1996, children's cumulative exposure to drug deaths increases at a slow and steady pace in triplicate states, but accelerates in non-triplicate states. By 2001, children aged 0-5 had greater exposure to the crisis in non-triplicate states, and by 2015, their exposure was 66% higher. For children aged 11-16, the crossing point occurs later, as much of their cumulative exposure happened before 1996. But by 2015, older children in non-triplicate states had experienced about 55% more drug deaths.

### *D. The 2SLS model*

To address the potential bias in OLS estimates, we implement an instrumental variables strategy that takes advantage of the fact that Purdue Pharma marketed OxyContin much less aggressively in states with triplicate programs.[13] 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 $YearsExpNT_{ast}$, which is equal to zero before 1996, and after is constructed as:

$$(3)\quad YearsExpNT_{ast} = Min(Age_{st}, Years\ after\ 1996) * NonTriplicate_{s}$$

where $NonTriplicate_s$ 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

[13] Because our instrument is meant to capture variation in the crisis that is due to Purdue's marketing efforts, we do not include other drug policies in our instrument, such as prescription drug marketing programs (PDMPs). In Appendix Table C3, we show that our results are unaffected by controlling for the presence of a PDMP.

$YearsExpNT_{ast}$ = 10. For a 3-year-old child in the same state and year, she has only been exposed for three years and so $YearsExpNT_{ast}$ = 3. Children in triplicate states of all ages and in all years have $YearsExpNT_{ast}$ = 0. When we estimate these 2SLS models, we cluster the standard errors by state.[14]

The validity of our 2SLS model rests on two assumptions. First, it must be the case that $YearsExpNT_{ast}$ is correlated with our measure of cumulative exposure. Figures 4 and 5 suggest that this is the case, and we confirm this with the first-stage estimates reported in Table 1. Years of exposure to a non-triplicate environment is strongly correlated with all death rate measures. The results in the first row of Table 1 indicate that likely parents of a 16-year old with 17 years of exposure to a non-triplicate state had a cumulative death rate that was 157 greater per 100,000 likely parents, which is 41% larger than the sample mean. The numbers for likely mothers and fathers are death rates per 100,000 that are 125 and 189 higher respectively, or 66% and 29% above their respective sample means. The smallest F-test in the table is 80, suggesting a strong relationship between the instrument and the endogenous variable.

Second, $YearsExpNT_{ast}$ should not be correlated with the error term in equation (2). In practice, violating this assumption requires some omitted factor or process that differentially affects the family environment of older children (relative to younger children) in states that did not have a triplicate law in 1996 (relative to triplicate states) and for that effect to grow only after 1996. As always, the identifying assumption cannot be tested directly. However, we will provide a number of robustness tests which do not pinpoint any violations of this assumption. In addition, it is useful to note a few facts. First, the triplicate programs were in place well before the drug crisis, suggesting that the programs were not passed in response to anticipated changes in drug outcomes or family structures. Second, the similarity of the pre-trends for drug death risks in the two types of states in Figures 4 and 5 is reassuring. This suggests that even if triplicate and non-triplicate states are different on some dimensions, their drug environments were being affected similarly by unobserved factors in the years preceding OxyContin's introduction.[15] Finally, in Figure 6 of Alpert et al. (2022), the authors report results from an event-study model that estimates the impact of triplicate state status on drug and opioid deaths – a regression analog to our Figure 4. In that figure, there is no pre-treatment trend in the drug or opioid death rate prior to 1996 and all pre-1996 periods have

[14] We also provide wild bootstrapped 95 percent confidence intervals in the tables in Appendix C.

[15] See also Alpert et al. (2022) for more event study results that demonstrate that the two types of states had similar trends in drug outcomes before the introduction of OxyContin.

statistically insignificant coefficients, a result consistent with hypothesis that Purdue Pharma did not direct advertising to states with particular trends in drug poisoning death rates.

As a starting point, Figure 6 shows results from standard difference-in-differences, event-study specifications that do not take advantage of varying exposure across age groups. Instead, it only uses variation across triplicate and non-triplicate states, before and after the introduction of OxyContin in 1996. The model is similar to equation (1) but the variable $CEXPOSURE_{ast}$ is replaced with interactions of dummy variables for non-triplicate states with dummy variables for year effects. We create dummies for two-year intervals (1990/91, 1992/93, etc.) to enhance power, and use 1994/95 as the reference period. In the graphs, the solid lines trace out the coefficient on the interactions and the dotted vertical lines represent the 95% confidence intervals. As with other models in the paper, we weight by group size and cluster standard errors at the state level.

As seen in Panel A, children's exposure to the crisis grew differentially in non-triplicate states after 1996. The reduced-form event study results are in Panels B through G. Panels B through E show that in the years prior to and immediately following the introduction of OxyContin, children in non-triplicate states were no more likely to be living without one or both parents. However, by the end of the period, the point estimates in each of the panels are consistently above zero even if they are not individually statistically significant; joint hypothesis tests on the post-period coefficients reject the null of no effect for each of the outcomes. The results in Panels F and G also show generally positive coefficients in the period after the introduction of OxyContin, though the results are noisier. There is no visible difference in the likelihood that children in the two types of states are in foster care after 1995 in Panel H.

These event-study style figures capture differences in trends between children in triplicate and non-triplicate states. While they provide suggestive evidence that children in triplicate states were more likely to experience informal changes in their living arrangements, they do not leverage the variation in exposure due to differences in children's ages as our 2SLS strategy will. By parameterizing the cumulative exposure, we hope to provide a more precise statement of a child's exposure to the crisis and enhance the power in the analysis.

We emphasize that our 2SLS model uses cumulative drug deaths experienced by likely parents as a proxy measure for how much exposure a particular child has had to the drug crisis. If Purdue Pharma advertising is exogenous conditional on observables, the reduced-forms are unbiased estimates of the impact of this business practice on the living arrangements of children. The 2SLS estimate is a consistent estimate of the impact of this exposure measure if the impact of

Purdue Pharma advertising in non-triplicate states is only working by making the crisis more severe. There could be other avenues if, for example, grandparents or state child protective services became more aggressive over time in removing children from homes with suspected drug use because their awareness of drug use is heightened by Purdue Pharma's marketing efforts. This would cause the 2SLS estimates to overstate the impact of the crisis on outcomes. Even in this case, the 2SLS is useful as it scales the reduced-form in a meaningful way. We now turn to the results from our main empirical strategy.

## IV. Results

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

In Table 2, we report our primary results. In all models, we allow for arbitrary correlation in errors within a state and weight observations by the cell size. 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, 24 percent are living away from a father, 3 percent are living away from both parents, and 5 percent are living in a household headed by a grandparent. In the next two columns, we report the OLS and 2SLS estimates, respectively, of equation (1). In the final column, we report the p-values from a Hausman test in brackets where the null hypothesis is that the OLS and 2SLS coefficients are the same.

In the first four rows, the outcomes of interest are whether a mother, father, at least one parent, or both are missing from the household. The 2SLS results in the first row indicate that for every likely mother that dies from a drug poisoning, about 13 children are living without a mother. In the second row, we see that for every likely father's death, about 10 are living without a father. The next two rows show the effect of a death of a likely parent; approximately 18 children are missing at least one parent and about 4 are missing both parents. The OLS and 2SLS estimates in these four rows are statistically different from zero at conventional levels. The magnitudes of the OLS and 2SLS estimates are remarkably similar and the p-values on the Hausman tests suggest little omitted variables bias in the OLS models.

Although the estimates might appear large, they are consistent with evidence on the extent of the drug crisis. First, many children have been directly affected by drug-related deaths within their household. Using data from the mortality follow-back of the National Health Interview Survey (Blewett et al., 2019), we estimate that the 763,000 drug poisoning deaths of adults aged 18 and up

from 1999 through 2015 occurred in households that housed 398,000 children.[16] However, we view our measure using deaths as a proxy for a child's cumulative exposure to the drug crisis in their state. Many children likely live in households that have been affected by drugs, even if there was not a death. Bullinger and Wing (2019) estimate that 548,000 children were living with an adult with an opioid use disorder in 2017. At a community level, 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.[17] So while our estimates may appear large when compared to the number of drug deaths of likely parents, they are reasonable given that our estimates capture the effects of the severity of the drug crisis in a child's community.

In the next two rows, we model whether the child is living in a household headed by a grandparent or by a non-parent. In the 2SLS models, the death of a likely parent leads to 6 children living in a household headed by a grandparent and 11 living with a non-parent, and both coefficients are statistically different from zero at conventional levels. Again, we fail to reject the equivalence of the 2SLS and OLS models at the 5 percent level.

Finally, in the last row, we see that the estimates for foster care are negative but imprecise. This may be due in part to the fact that foster care is underreported in the ASEC data, but even if we take the upper value of the 95% confidence interval (a value of 0.52), this estimate is less than a tenth of the estimate for living in a household headed by a non-parent. We conclude that any movement of children into foster care was dwarfed by the other changes in living arrangements that we consider, and that informal arrangements appear to be much more common than formal foster care placements in this context.[18]

### *B. Robustness*

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 C. We assess the robustness of our results to 1) alternative specification and sample choices; 2) the inclusion of additional controls; 3)

[16] See Appendix D2 for more detail.

[17] Authors' calculation using the 2017 National Drug Use and Health Survey (https://www.samhsa.gov/data/sites/default/files/cbhsq-reports/NSDUHDetailedTabs2017/NSDUHDetailedTabs2017.htm#tab1-1A) and MCOD data for 2017.

[18] Because the outcomes in Table 2 could be correlated with one another, we have implemented the Romano-Wolf stepdown method to construct asymptotically valid tests for multiple hypotheses. The p-values obtained using this method show that each of the six coefficients in Table 2 that are statistically significant at the 10% level remain so, and three of the six remain significant at the 5% level (living without the mother, living without at least one parent, and living with a non-relative). The p-values for these three outcomes are 0.0840, 0.0696, and 0.0662, respectively.

omitting individual triplicate states; and 4) alternative specifications to address the difference in population between triplicate and non-triplicate states. We also report the results of a randomization inference exercise.

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 born before the introduction of OxyContin in 1996; doing so yields estimates that are less precise but consistent with our main findings.[19] 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).[20] Our results remain the same. These results are in Appendix Table C1.

Second, in Appendix Tables C2 and C3, we show that our results are not simply masking differences or changes in the economic, social, or legal environments 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 (Buchmueller and Carey, 2018) or if we control for welfare reforms which were previously shown to affect children's living arrangements (Bitler et al., 2006). Finally, our results are robust to the

[19] 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.

[20] Our results are also robust to dropping the demographic controls, and to adding either year-by-age, state-by-age, or year-by-state fixed effects.

inclusion of controls for the state's violent and property crime rates, though the point estimates are slightly smaller. This is what we would expect if one of the mechanisms behind the effects we observe is that the crisis has increased parents' criminal activity, as suggested in the introduction.

Fourth, a potential concern is that our identification strategy relies on comparing children in triplicate and non-triplicate states, and there are only five states in the former set. It is therefore possible that one of the five triplicate states could be driving the results. In Appendix Table C4, we show that our results are unchanged when we remove each of the five triplicate states, one at a time, and re-estimate the regression.

Fifth, in Appendix Table C5, 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.

As a final test, we conduct a randomization inference exercise, in which we randomly assign treatment status 10,000 times to obtain a distribution of possible point estimates and then observe the position of our estimates within that distribution. For this exercise, we use our reduced form estimates, as the first stage of the 2SLS with random treatment would be weak in most cases. We construct three separate distributions by 1) randomly assigning five states to be triplicate states for each draw; 2) randomly assigning five states and also randomly assigning years of exposure to children; and 3) randomly assigning treatment status to states to match the population size of the five actual triplicate states. Appendix Table C6 shows the position of our actual reduced form coefficients within these distributions. As we see in the table, the point estimates we obtain tend to be in the right tail of the distribution. The majority of the estimates would be significant at the ten percent level and many would be significant at the five percent level as well. These results reassure us that our estimated effects are not spurious.

### C. *An Alternative Instrument*

In this section, we compare our main results to those obtained using a different instrument that also exploits variation in the severity of the crisis that is driven by Purdue Pharma's marketing

strategy. Arteaga and Barone (2022) present evidence that another important component of Purdue Pharma's marketing strategy was to target areas with high rates of cancer pain. We refer the reader to the Arteaga and Barone paper for a more detailed description of this idea, but we summarize it here.

Up until the mid-1990s, opioid analgesics were typically used for cancer patients and for palliative care at the end of life. OxyContin was developed to replace Purdue Pharma's primary product in this market, MS Contin, as that drug's patent was scheduled to expire and Purdue Pharma expected stiff competition from generics (Purdue Pharma, 1995a). Purdue Pharma also planned to introduce OxyContin into the non-cancer market, but recognized that doing so would be met with some resistance from the medical community. As a result, Purdue Pharma planned that in the first year of release "OxyContin will be marketed for cancer pain" (Purdue Pharma, 1995a, p14). In that year, Purdue Pharma targeted 13,000 visits to the top 30% of current opioid analgesic prescribers and 7,600 visits to the top 20%. As virtually all prescriptions in this class were for cancer pain management, this meant that Purdue Pharma's early rollout was focused almost exclusively on doctors and nurses treating cancer patients, and pharmacies that currently stocked MS Contin (Purdue Pharma, 1996, p.45). Purdue Pharma then relied heavily on its existing sales reps when it entered OxyContin into the non-malignant pain market in 1997 (Purdue Pharma, 1997, p37).

Appendix Figures A2, A3, and A4 are analogs to Figures 3, 4, and 5, comparing trends in prescription drugs, cumulative drug deaths, and death rates for likely parents for high- and low-cancer states. High- (low-) cancer states are those in the top (bottom) quartile of the distribution of the cancer death rate in 1995, the year before OxyContin was released. We only include cancer deaths to those over age 56 in our measure of cancer prevalence to exclude likely parents of children aged 0-16.[21]

Figure A2 mirrors Figure 3, showing that for all other Schedule II analgesics, there is virtually no difference in use across states with high and low cancer rates in 1995, but there is a massive difference in oxycodone use. In Figure A3, we see that in the 12 years prior to the introduction of OxyContin, states with low cancer death rates in 1995 had higher drug poisoning death rates, but the two sets of states had very similar trends. After 1996, the low-cancer states continued on a steady upward trajectory, but the high-cancer states experienced a rapid increase and

[21] This choice also mirrors the variation we use in our instrumental variables strategy below. Of course, state-level cancer death rates in 1995 for those aged 57 and up are correlated with cancer death rates of likely parents. Therefore, in all models that use the instrument based on the cancer death risk, we also include the cancer death rate of likely parents (age 17-40) as a control variable. This control also helps to account for the fact that state-level cancer rates are unsurprisingly highly correlated over time, so that states with more cancer in 1995 may continue to have more cancer after OxyContin is introduced.

had a higher drug death rate by 2001. By 2015, the drug death rate in the high-cancer states was a little more than twice that of states that had low cancer rates in 1995. Last, in Figure A4, we show that cumulative death rates for likely parents of children 0-5 in high- and low-cancer states track each other well until about 2001 and diverge after that. The pre-1996 levels for likely parents of 11-16 year-olds show very similar trends pre-1996 but decidedly different paths after.

Collectively, these figures suggest that a state's 1995 cancer death rate for those age 57+ is correlated with the severity of the opioid crisis.[22] Under the assumption that these rates should not otherwise affect children's living arrangements (which we return to below), we define a second instrument that is equal to zero before 1996, and after 1996 is constructed as follows:

$$(4)\quad YearsExpCDR_{ast} = Min(Age_{st}, Years\ after\ 1996) * (1995\ Cancer\ Death\ Rate\ Ages \geq 57_s)$$

which is the interaction between the years of exposure to an environment with OxyContin and the state-level cancer death rate for people over age 56. The instrument captures the fact that holding a child's age constant, likely parents in high-cancer states will have experienced more Purdue Pharma marketing than likely parents from low-cancer states. Likewise, holding the state constant, we expect 15-year-olds in 2010 to have more aggregate exposure to Purdue Pharma marketing than 5-year olds.

We report the 2SLS results using this instrument in Table 3. For comparison, in column (1) of the table we reproduce the 2SLS estimates from our main instrument that relies on states' triplicate prescription laws. Column 2 shows the estimates using the instrument based on the 1995 cancer death rate for those age 57+. The third column presents estimates from a specification that uses both instruments together; because this model is over-identified, we are able to perform an over-identification tests of the null hypothesis of valid instruments. The p-values from this test are in column 4.

The magnitudes of the estimates in Table 3 are remarkably similar across the 2SLS specifications. This result further supports our identification strategy, as any threat to identification is unlikely to apply to both instruments. For example, if one is concerned that an omitted factor drives both cancer rates and child living arrangements, that factor would also need to be correlated with triplicate state status for the two estimates to yield such similar results. Alternatively, a weakness of the triplicate instrument is that it relies on a set of five triplicate states for identification, and it could be that there is something unique about this set that led to different trends in child living arrangements over this period—though the similarity of the pre-trends in Figures 3-5 and 6A, and

[22] We discuss these figures in more detail and show estimates for the first stage in Appendix A.

the exercise in Appendix Table C4, provide reassurance on this front. Nevertheless, the cancer instrument relies on a continuous measure that varies across all states, and is therefore not subject to this same concern. Column 4 shows the results of the over-identification test, and for six of the seven outcomes, we fail to reject the null hypothesis of valid instruments.

While the results in Table 3 support the validity of both instruments, we acknowledge a potential violation of the exclusion restriction for the cancer rate IV, if many cancer deaths among those age 57+ in 1995 affect the set of adults available to care for the child (even after controlling for cancer deaths among likely parents). This is most relevant for the outcome of living with a grandparent—in states with many cancer deaths for older people, there may be fewer grandparents available to care for the child. This would bias estimates using this instrument toward zero for this outcome, which is indeed what we see, and this is the only outcome where there is a meaningful difference in the magnitudes of the estimates with different instruments. Because of this potential issue, we continue to treat the 2SLS results using states' triplicate status as the instrument as our main results. We take the striking similarity of the estimates with the two different IVs and the reassuring results of the over-identification tests as further support for the validity of this strategy.

### D. *Heterogeneity by Age and Race*

An interesting question is whether the age at which a child is exposed to the drug crisis matters for their likelihood of entering an alternative living arrangement. To examine this issue, note that our instrument—cumulative years of exposure to a non-triplicate regime—can be expressed as the sums of exposure at different ages. To see this, let $YearsExp_{ast}$ be the integer component of the instrument that we define in equation 3. This variable can be calculated as the sum of the exposure at ages 0-5, 6-11, and 12-16, as in the following equation:

$$(5) \quad YearsExp_{ast} = YearsExp(0\text{-}5)_{ast} + YearsExp(6\text{-}11)_{ast} + YearsExp(12\text{-}16)_{ast}$$

We estimate the reduced-form model in this context, as a 2SLS model would use all three instruments when estimating the coefficient on cumulative exposure on different ages. The reduced-form provides a direct estimate of how exposure to a non-triplicate state impacts living arrangements at different ages.

The basic results are reported in Table 4. In the first column (model 1), we report reduced-form results using the regular instrument, for comparison. In the next three columns (model 2), we replace that instrument with its components at different ages. By construction, $YearsExp(6\text{-}11)_{ast}$ is zero for anyone under 6 and $YearsExp(12\text{-}16)_{ast}$ is zero for anyone under 12. These persistent effects

will be captured by our age effect controls in the model. In the final column, we report the p-value on the test that the three coefficients on the exposure variables are the same value.

For all outcomes except foster care, the results show a large pronounced effect of exposure during ages 6-11 (as with our main estimates, all coefficients for foster care are statistically insignificant at conventional levels). For these six outcomes, the coefficients on the effect of exposure in this age range are statistically significant, and the point estimates are much larger than in model 1 where we use just the one instrument. The results for exposure during ages 0-5 and 12-16 are almost all positive, but the coefficients are not statistically significant at conventional levels. Despite the imprecision of some of the estimates, in four of the models, we can reject the null that the coefficients across all three variables are the same. Thus, these reduced form estimates provide evidence that exposure to the crisis during ages 6-11 has the largest impact on the chance that a child is living away from a parent.

We have also explored the possibility of heterogeneous effects across racial groups. The opioid crisis was more acute among White Americans—86% of likely parents who died from drug use between 1996 and 2015 were White. At the same time, there are much higher baseline probabilities that Black children are living away from a parent. Given these stark differences, in Appendix Table C7, we explore whether the effects of the crisis vary by race.[23] The results for White children correspond to the aggregate results for all races in Table 2, but the results for Black children are too imprecise to make definitive conclusions. This is not surprising given the much smaller samples and the substantially weaker first stage for this group, a result consistent with Powell (2021b) who shows that exposure to non-triplicate states produced a substantially larger impact for White adults.

## VI. Changes in Family Structure and Child Well-Being

### *A. Further Evidence from the Current Population Survey*

The results in the previous section demonstrate that the drug crisis has had large impacts on the living arrangements of children. We stress that our analysis does not address the important question of whether, *conditional on having a drug-using parent*, it is better for the child to live away from the parent. Instead, the counterfactual we consider is one in which the drug crisis was much less severe due to less aggressive marketing of OxyContin. Furthermore, our identification strategy does

[23] 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.

not allow us to isolate the effects of these changes in living arrangements on a child's well-being, as it will capture effects of the crisis through other channels as well (e.g. from changes in parents' behavior or from a child's own drug use). Indeed, Arteaga and Barone (2022) show that the crisis increased the demand for social safety net programs, including SNAP.

Nevertheless, we can use our estimation strategy to estimate the impacts of the crisis as a whole on the characteristics of the child's home environment and health status. We estimate the same OLS and 2SLS models as before, but with three different measures of a household's economic status as the dependent variable: indicators that the household is in poverty, on SNAP, or without health insurance. For our measure of child health, we use a question from the ASEC data that asks the respondent to evaluate the health status of each household member on a five-point scale (poor, fair, good, very good, or excellent).[24] Our dependent variable is binary variable equal to one if the child is reported to be in fair or poor health, and zero otherwise.

The results are presented in Table 5. The structure of the table follows that of Table 2 where each row contains a different outcome. The 2SLS coefficients for the three measures of household resources are all positive, and are statistically significant for two of the three dependent variables. These 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. Additionally, we find that each additional drug death of a likely parent causes 2.56 more children to be in poor or fair health. For all four dependent variables in the table, the OLS and 2SLS coefficients are similar and we fail to reject the null hypothesis that they are equal.

### *B. Evidence from the Literature*

While our empirical analysis using the CPS suggests that children's living environments and outcomes have been worsened by the crisis, they do not isolate the effect of changes in living arrangements. However, there is an abundance of research from across the social sciences that suggests that changes in living arrangements like those we observe likely contribute to worse outcomes. Child psychologists have long proposed that parent-child separations have adverse effects on children's mental and emotional health and on the development of their personalities (Rutter, 1971). 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

[24] Self-reported health status is first reported in the ASES starting in 1996. The data is reported by the respondent for each household member. This sample then has 20 years for 51 states and 17 ages or 17,340 obesrvations.

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.[25] 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.[26] 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). Children who have experienced trauma have worse adult outcomes such as higher rates of mortality (Felitti et al., 1998), substance abuse and mental health incidence (Anda et al., 2006; Dube at 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).

Furthermore, while we know of no studies that have identified the causal effect of parental separation as a result of drug use specifically, prior work has done so for parental divorce (Gruber, 2004), death (Chen et al., 2009), and absence due to military deployments (Lyle, 2006). Each of these papers shows that parental separation leads to worse outcomes for children, including lower educational attainment, lower income, and risky behaviors.[27] Causal estimates of the effects of separation due to parental incarceration are more mixed (Dobbie et al., 2018; Arteaga, forthcoming; Norris et al., 2021; Billings, 2018). Finally, Doyle (2007; 2008) 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. Thus, the existing evidence suggests that many of the millions of children whose living arrangements have been affected by the crisis will likely have adverse outcomes as a result.

[25] Because we only see the child's current living arrangement, we will not observe a family structure transition for children who were separated from a parent in the past but were later reunified. Our estimates therefore provide a lower bound on the number of children whose lives have been disrupted by the crisis.

[26] Authors' calculations, 2017 Adoption and Foster Care Analysis and Reporting Systems data.

[27] See McLanahan et al. (2013) for a review of the literature on the causal effects of father absence.

## VII. Discussion

In this paper, we provide causal estimates of the effects of the United States' recent drug crisis on the living arrangements of children. 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.

To quantify the extent to which the drug crisis has upended the lives of children, we conduct a simple simulation that calculates the number of children living in a different family structure as a result of the crisis. To do this, we calculate the change in cumulative exposure at each age (0-16) over the 20-year period 1996 to 2015, and multiply this by the 2SLS coefficients in Table 2. We then multiply this by the number of children of each age in 2015 as measured in the SEER population data. These simulations suggest that because of the drug crisis that followed the introduction of OxyContin in 1996, about 862,000 children were living away from a mother, 954,000 were living away from a father, and over 1.5 million were living away from at least one parent in 2015. Recall that in this same year, 135,000 children were in foster care as a result of parents' abuse of *any drug*, supporting our claim that informal changes in living arrangements were many times more common that moves into formal foster care.[28] Perhaps the most heartbreaking fact is that 302,000 children were living away from both parents. Finally, we estimate that 950,000 were living in a household headed by someone other than their parent and over half of these were living in a household headed by a grandparent. Our results support the popular press accounts that grandparents are shouldering a large portion of the costs generated by the drug crisis.

It is worth emphasizing that for the majority of our estimates, the OLS and 2SLS regressions yield similar coefficients, and we cannot reject the null hypothesis that they are the same. This suggests that the OLS estimates do not suffer from severe omitted variables bias, as we would expect if, for example, diminishing economic opportunities were playing a large role in both drug use and family separations. Similarly, the equivalence of OLS and 2SLS is inconsistent with a story of reverse causality in which declining family institutions are causing drug use. Instead, it appears that supply-side factors—including the marketing efforts of Purdue Pharma—are behind much of the increase in drug use and its consequences for children.

[28] Further, when using the upper bound of the confidence interval for our foster care estimates, we calculate that approximately 43,000 children would have been in foster care in 2015 as a result of the crisis. This is a large and worrying number to be sure, but it is orders of magnitude smaller than our estimates of the number of children living away from parents or in a household headed by a non-parent.

Our study has implications for public policy aimed at ending the opioid crisis and helping its victims. Our evidence highlighting the importance of supply-side factors can help explain why policies that improve labor market outcomes have not worked to reduce drug deaths (Dow et al., 2020). Ruhm (2019) predicted this result, and argued that targeting the drug environment is a better strategy for reducing deaths. However, policies that do so have been shown to have limited impacts when users are able to switch to substitutes like heroin (Kim, 2021; Evans et al., 2019), and may even lead to worse outcomes for children (Evans et al., 2022). An alternative is drug treatment programs for those already addicted, which have been shown to reduce drug-related mortality and morbidity (Swensen, 2015; Corredor-Waldron and Currie, 2022). While these programs are costly, our findings show that by helping adult victims of the crisis, they may also help their children by reducing disruptions to their living arrangements.

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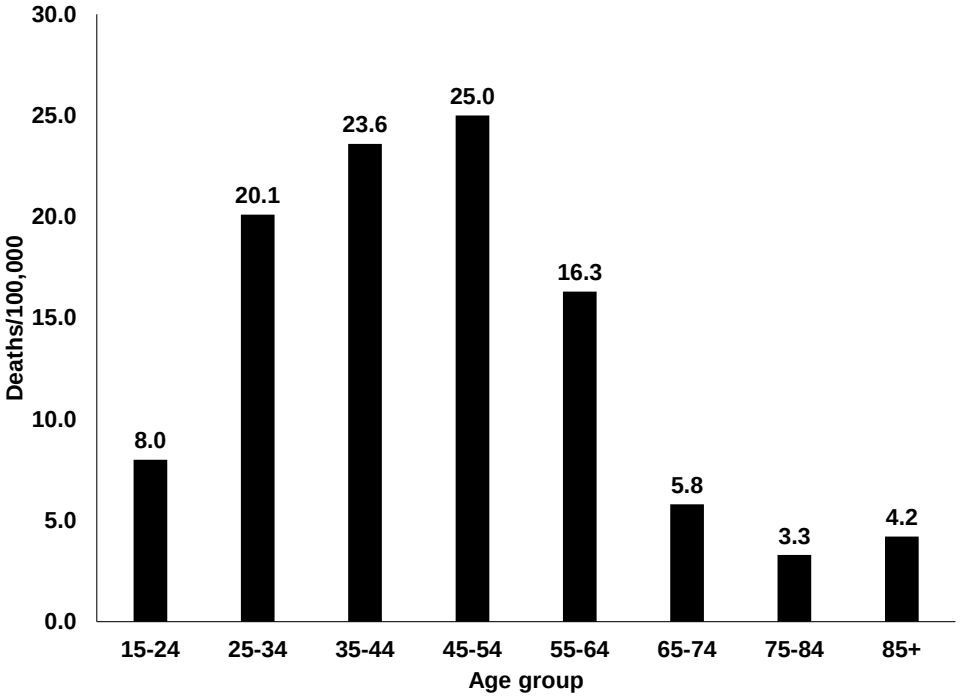
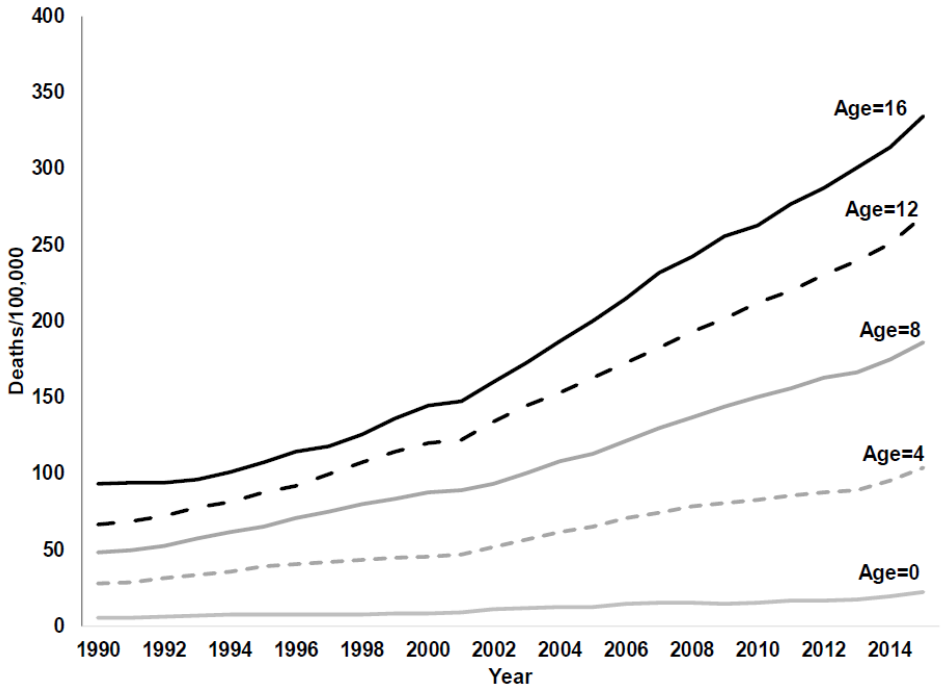
**Figure 1: Drug Death Rate by Age in the United States, 1999-2019**




Data are from the 1999-2019 Multiple Cause of Death Files.

**Figure 2: Cumulative Drug Death Rate of Likely Parents, 1990-2015**
**Panel A: By Age of Child**




**Panel B: By State of Residence, for Children Aged 10**

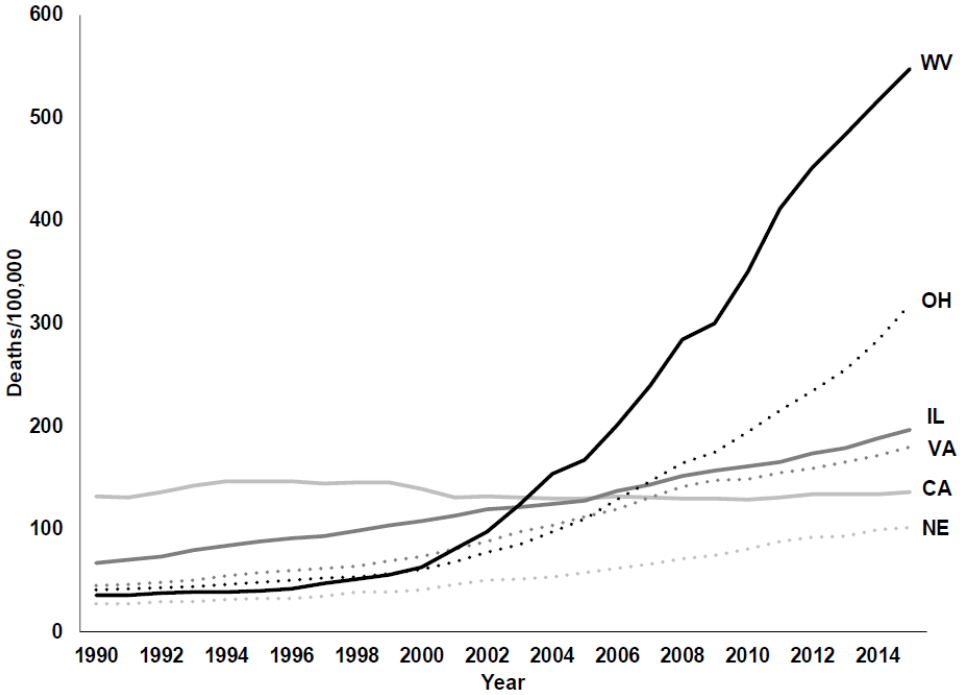
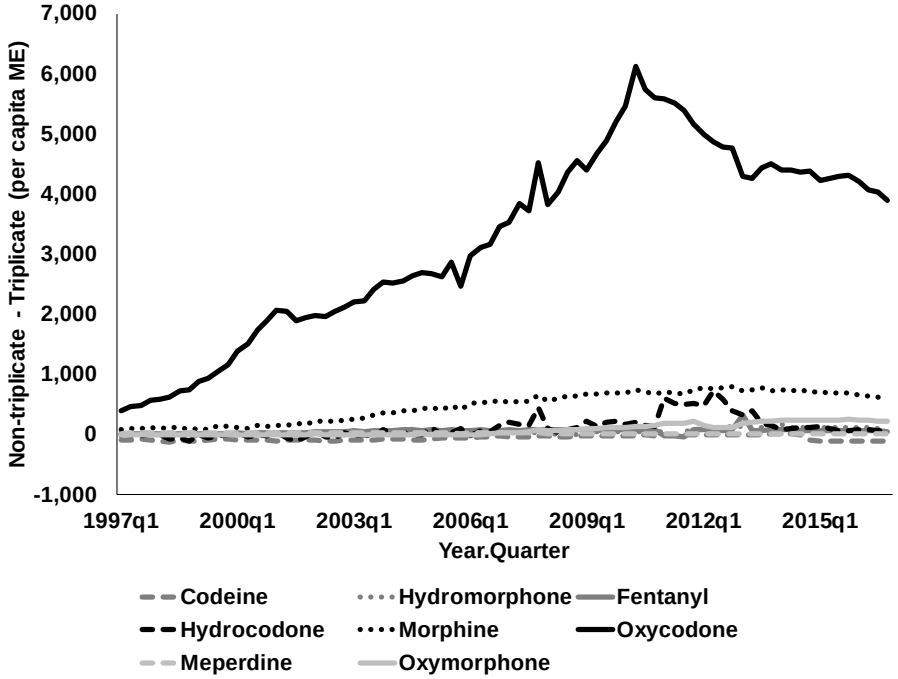


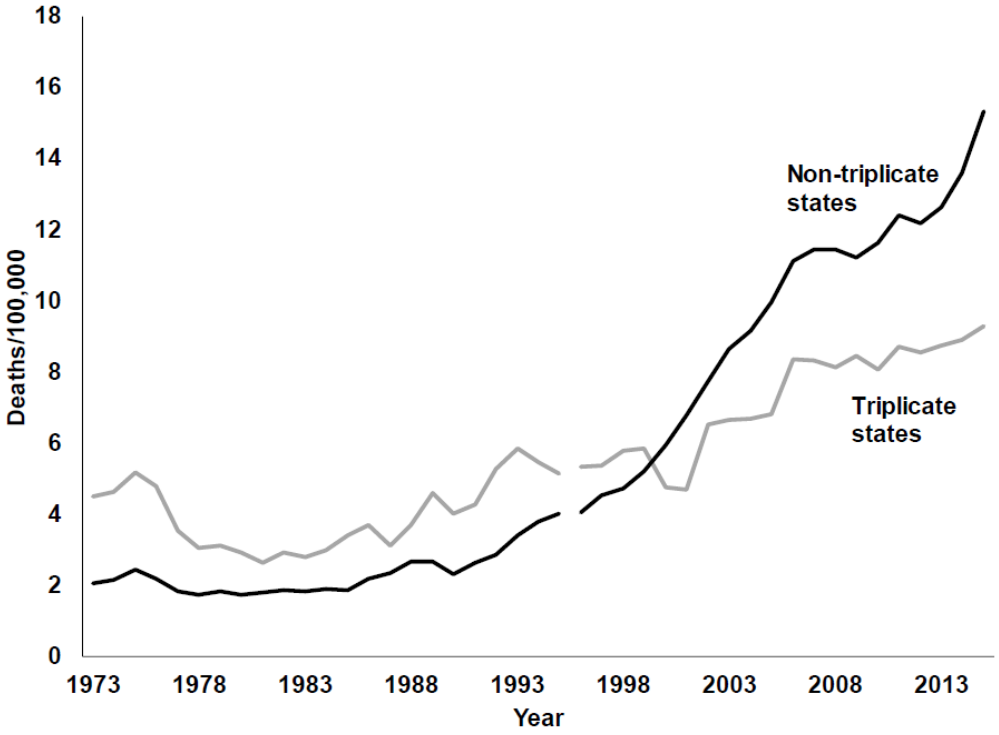

Figure shows trends in our measure of children's exposure to the drug crisis, $CEXPOSURE_{ast}$; see the text for details on how this measure is constructed. Data are from the Multiple Cause of Death Files.

**Figure 3: Differences in Opioid Use between Non-Triplicate and 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 4: Drug Death Rates by Triplicate State Status, 1973-2015**




Data are from the Multiple Cause of Death Data, 1973-2013. Series break point is 1996, the year Purdue Pharma released OxyContin.

**Figure 5: Cumulative Drug Death Rates for Likely Parents of Children Aged 0-5 and 11-16, 1990-2015**

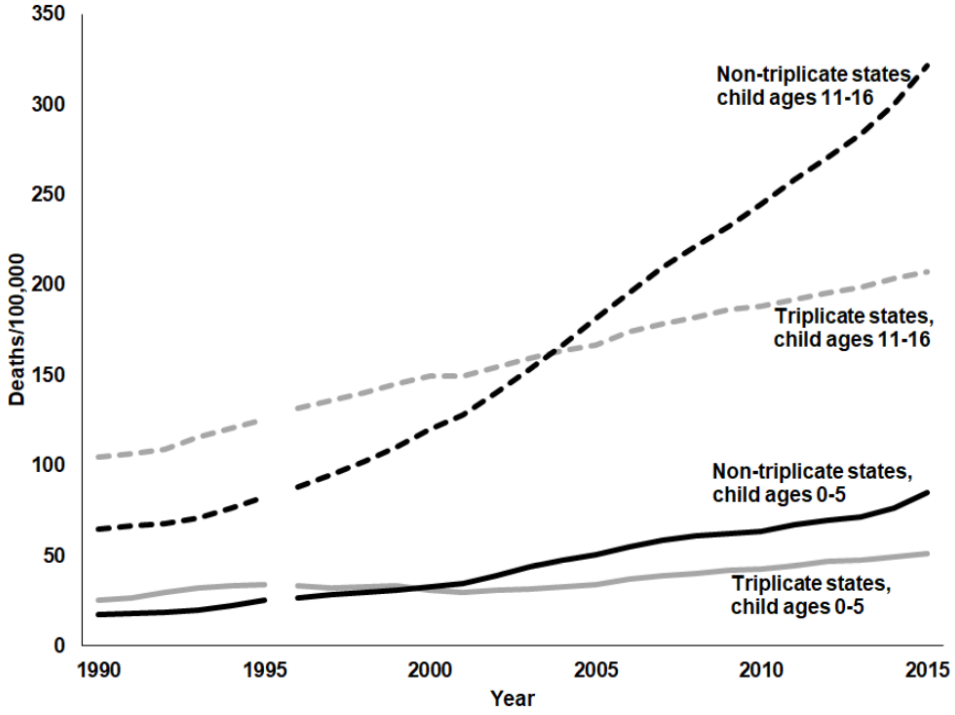
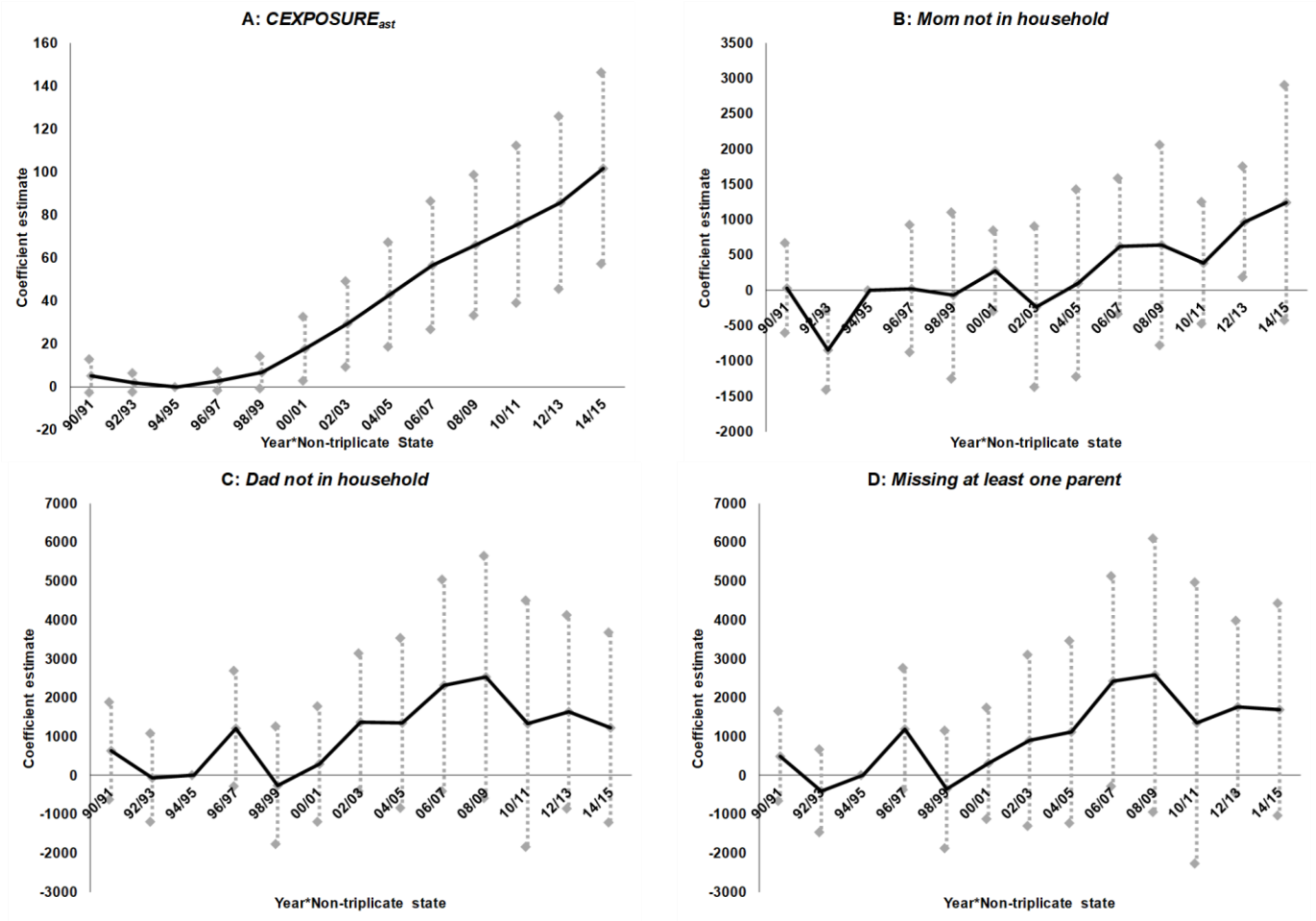
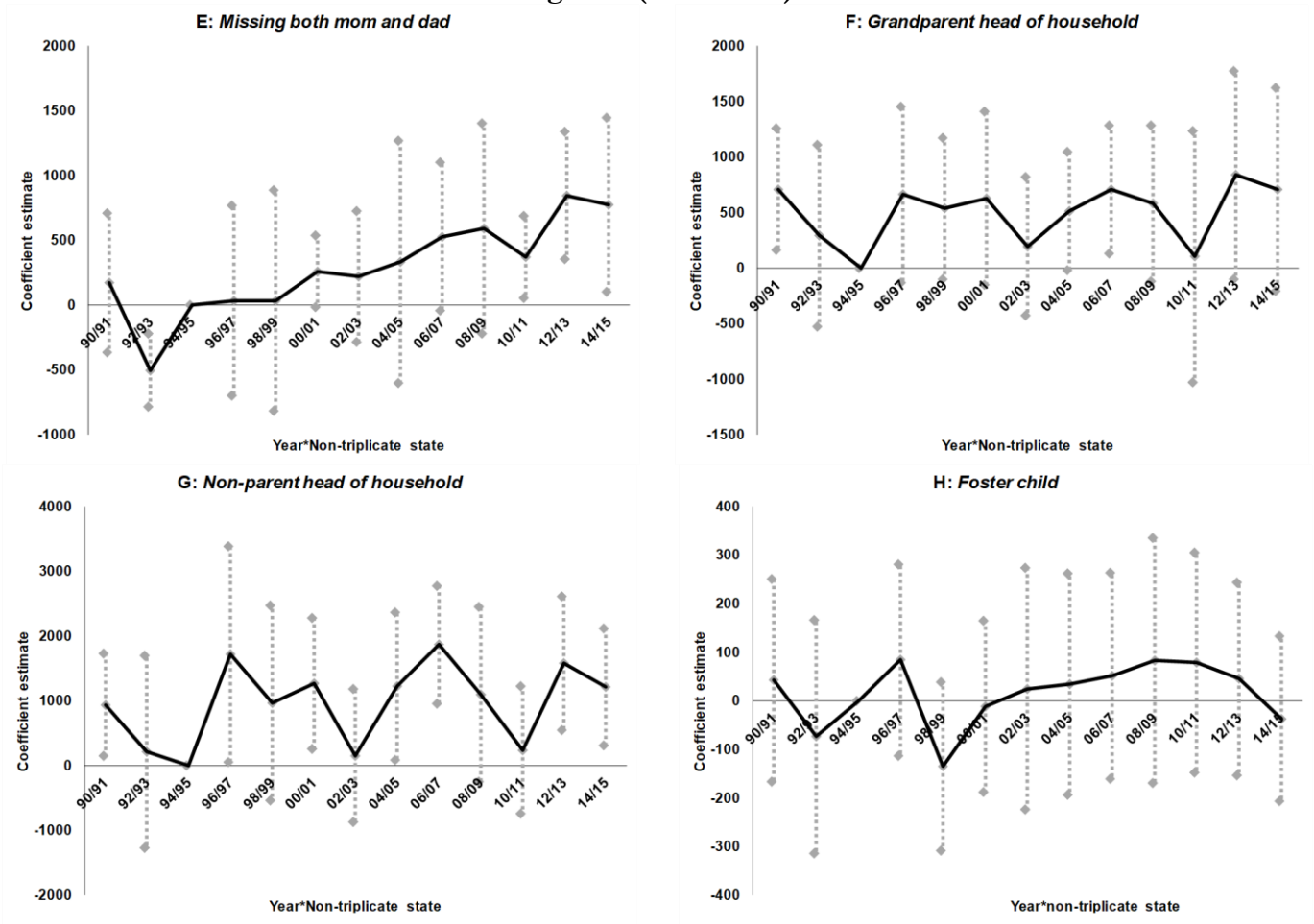



Figure shows trends in our measure of children's exposure to the drug crisis, $CEXPOSURE_{ast,}$ for children of different ages in states with and without triplicate prescription pad laws. See the text for details on how the $CEXPOSURE_{ast}$ measure is constructed. Data are from the Multiple Cause of Death Files. The series break point is 1996, the year Purdue Pharma released OxyContin.

**Figure 6: Event Studies Comparing Childrens' Experiences in Non-Triplicate and Triplicate States, 1990-2015**



## Figure 6 (Continued)




Figures 6A-6H show the coefficient estimates from event study regressions. The model is similar to that suggested by equation (1) but the replace the variable $CEXPOSURE_{ast}$ with interactions of dummy variables for non-triplicate with dummy variables for year effects. We use two-year bins to reduce noise in the estimates and omit the 1994-95 period. The dotted lines indicate 95% confidence intervals. The coefficient estimates represent changes in the number of instances per 100,000 children; note that the scale of the vertical axis changes across figures because some outcomes are more common than others. The data for Panel A are from the Multiple Cause of Death Files; data for all other figures are from the Annual Social and Economic Supplement (ASEC) of the March Current Population Survey (CPS).

**Table 1: First-Stage Estimates of Cumulative Drug Death Rate Equations, 1990-2015**

| Dependent variable | Sample mean | OLS coefficient on $YearsExpNT_{ast}$ (standard error) | First stage F-test |
|---|---|---|---|
| Cumulative drug deaths/100K of likely parents | 110.9 | 9.22<br>(0.96) | 92.7 |
| Cumulative drug deaths/100K of likely mothers | 75.3 | 7.36<br>(0.80) | 84.3 |
| Cumulative drug death/100K of likely fathers | 146.1 | 11.1<br>(1.24) | 80.4 |

Data on drug deaths are from the Multiple Cause of Death Files. 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.

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

| | | Parameter estimates (standard errors) on $CEXPOSURE_{ast}$ | | |
|---|---|---|---|---|
| Dependent variable | Sample mean | OLS | 2SLS | p-value Hausman test |
| Mom not in household/100K | 6,304 | 10.0<br>(1.99) | 12.88<br>(3.36) | 0.207 |
| Dad not in household/100K | 23,890 | 9.32<br>(1.89) | 9.68<br>(4.34) | 0.925 |
| Missing at least one parent/100K | 27,294 | 15.52<br>(3.00) | 18.29<br>(5.63) | 0.533 |
| Missing both Mom and Dad/100K | 2,900 | 4.38<br>(0.78) | 3.64<br>(1.44) | 0.532 |
| Grandparent head of HH/100K | 5,282 | 2.09<br>(1.71) | 6.02<br>(2.17) | 0.061 |
| Non-parent head of HH/100k | 9,533 | 6.41<br>(1.94) | 11.45<br>(2.62) | 0.074 |
| Foster child / 100k | 309 | 0.20<br>(0.19) | -0.07<br>(0.48) | 0.510 |

Data on living arrangements 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. We calculate standard errors allowing for arbitrary correlation in errors at the state level. The last column gives the p-value for a Hausman test, where the null hypothesis is that the 2SLS coefficient and OLS coefficients are equivalent.

**Table 3: 2SLS Estimates of the Impact of Cumulative Drug Deaths Rates of Likely Parents on Child Well-Being Using Two Different Instruments, 1990-2015 ASEC**

| Dependent variable | 2SLS $YearsExpNT_{ast}$ Instrument | 2SLS $YearsExpCDR_{ast}$ Instrument | 2SLS Both Instruments | Over-Ident. Test |
|---|---|---|---|---|
| Mom not in household/100K | 12.88 | 12.63 | 12.84 | 0.886 |
| | (3.36) | (2.16) | (2.46) | |
| | [0.207] | [0.193] | [0.103] | |
| Dad not in household/100K | 9.68 | 9.44 | 9.59 | 0.947 |
| | (4.34) | (2.94) | (3.42) | |
| | [0.925] | [0.957] | [0.921] | |
| Missing at least one parent/100K | 18.29 | 19.22 | 18.66 | 0.829 |
| | (5.63) | (3.65) | (4.46) | |
| | [0.533] | [0.189] | [0.320] | |
| Missing both Mom and Dad/100K | 3.64 | 2.66 | 3.28 | 0.176 |
| | (1.44) | (1.06) | (1.06) | |
| | [0.532] | [0.112] | [0.292] | |
| Grandparent head of HH/100K | 6.02 | 3.10 | 4.87 | 0.127 |
| | (2.17) | (1.54) | (1.69) | |
| | [0.061] | [0.556] | [0.131] | |
| Non-parent head of HH/100k | 11.45 | 11.22 | 11.36 | 0.919 |
| | (2.62) | (2.01) | (2.11) | |
| | [0.074] | [0.049] | [0.138] | |
| Foster child / 100k | -0.07 | -0.74 | -0.33 | 0.045 |
| | (0.48) | (0.44) | (0.44) | |
| | [0.510] | [0.008] | [0.056] | |

Data on living arrangements 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. Models with cancer mortality as an instrument also include a control for the cancer mortality rate of likely parents (those age 17-40). 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. The number in brackets is the p-value for a Hausman test, where the null hypothesis is that the 2SLS coefficient and OLS coefficients are equivalent. The last column shows the p-value for Hansen's overidentification test.

**Table 4: Reduced-Form Estimates of the Exposure to a Non-Triplicate Environment at Different Ages on the Living Arrangement of Children, 1990-2015 ASEC**

| | Model 1 | Model 2 | | | |
|---|---|---|---|---|---|
| Dependent Variable | NT Exposure, All ages | NT Exposure, Ages 0-5 | NT Exposure, Ages 6-11 | NT Exposure, Ages 12-16 | P-value, coefficients equal |
| Mom not in household/100K | 99.9 | 57.2 | 163.9 | 56.2 | 0.067 |
| | (21.4) | (38.6) | (32.0) | (38.5) | |
| Dad not in household/100K | 106.8 | 92.9 | 209.6 | -102.7 | 0.017 |
| | (46.2) | (90.3) | (66.0) | (83.2) | |
| Missing at least one parent/100K | 198.0 | 113.1 | 203.2 | 48.5 | 0.406 |
| | (103.5) | (85.3) | (70.6) | (97.3) | |
| Missing both parents/100K | 34.0 | 18.5 | 84.7 | -47.5 | 0.011 |
| | (12.7) | (24.2) | (18.6) | (32.8) | |
| Grandparent head of HH/100K | 60.8 | 9.89 | 126.7 | 30.5 | 0.039 |
| | (18.0) | (32.0) | (32.6) | (27.3) | |
| Non-parent head of HH/100K | 34.1 | -53.5 | 156.8 | -35.5 | 0.045 |
| | (20.7) | (60.1) | (90.2) | (94.9) | |
| Foster child/100K | -3.0 | -19.6 | -4.2 | 6.4 | 0.564 |
| | (5.1) | (16.4) | (19.4) | (18.0) | |

Data are from the Annual Social and Economic Supplement (ASEC) of the March Current Population Survey (CPS). The table shows the coefficients on our measure of exposure to a non-triplicate (NT) state environment from ages 0-16 combined (Model 1), or from ages 0-5, 6-11, and 12-16 separately (Model 2). 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 models include 17 ages x 51 states x 26 years = 22,542 observations. Standard errors clustered by state are in parentheses. The number in the last column is the p-value for the test of the null hypothesis that all three years of exposure measures are the same.

**Table 5: OLS and 2SLS Estimates of the Impact of Cumulative Drug Deaths Rates of Likely Parents on Child Well-Being, 1990-2015 ASEC**

| | | Parameter estimates (standard errors) on $CEXPOSURE_{ast}$ | | |
|---|---|---|---|---|
| Dependent variable | Sample mean | OLS | 2SLS | p-value Hausman test |
| # In poverty/100K | 19,478 | 8.49<br>(3.55) | 8.85<br>(4.54) | 0.266 |
| # On SNAP/100K | 16,064 | 8.67<br>(4.23) | 10.05<br>(5.07) | 0.592 |
| # Without health insurance/100K | 10,847 | 4.22<br>(4.66) | 4.37<br>(5.07) | 0.399 |
| # In fair or poor health/100K | 10,847 | 1.70<br>(0.53) | 2.56<br>(0.904) | 0.399 |

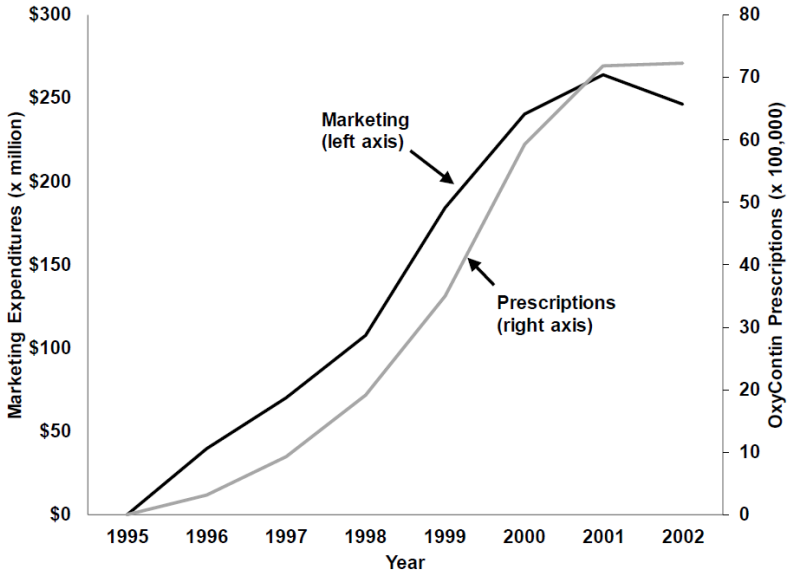
Data are from the Annual Social and Economic Supplement (ASEC) of the March Current Population Survey (CPS). All models include fixed effects for age, year and state, plus the fraction of observations that were female, Black (non-Hispanic), other race (non-Hispanic) and Hispanic. The models in the first three rows include 17 ages x 51 states x 26 years = 22,542 observations. Self-reported health status is only available from 1996 on so the last row has 17 ages x 51 states x 20 years =17,340 observations. We calculate standard errors allowing for arbitrary correlation in errors at the state level. The number in the last column is the p-value for a Hausman test, where the null hypothesis is that the 2SLS coefficient and OLS coefficients are equivalent.

# For Online Publication

## Appendix A

## Additional Detail and Evidence on the Instrumental Variables Strategies

**Figure A1: OxyContin Marketing and Prescriptions, 1995-2002**




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.

In Section V.C., we introduce an alternative instrument: a state's cancer rate in 1995, for those age 57 and over. We provide the intuition for the strategy in the main text. Here, we add additional detail and supporting evidence.

First, we show that because of Purdue Pharma's early strategy of building on their previous success in the cancer market, states' cancer rates are correlated with the severity of the drug crisis and children's exposure to it. As in Alpert et al. (2022), unfortunately there is no marketing data available by state or county in the late 1990s in the early days of OxyContin's sales, but we can document the same persistent patterns outlined for triplicate and non-triplicate states. In Appendix Figure A2 below, we repeat the structure of Figure 3 and report the difference in per-capita morphine equivalent doses between high- and low-cancer states. The patterns in this graph mirror those in Figure 3 in that for all other Schedule II analgesics, there is virtually no difference in use across states with high and low cancer rates in 1995, but there is a massive difference in oxycodone use.

In Figure A3, we show trends in drug deaths for our high- and low-cancer states (analogous to Figure 4). For the 12 years prior to the introduction of OxyContin, states with low cancer death rates in 1995 had higher drug poisoning death rates, but the two sets of states had very similar trends. After 1996, the low-cancer states continued on a steady upward trajectory, but the high-cancer states experienced a rapid increase and had a higher drug death rate by 2001. By 2015, the drug death rate in the high-cancer states was a little more than twice that of states that had low cancer rates in 1995.

Figure A4 reproduces the structure of Figure 5, but comparing our high- and low-cancer states. The cumulative death rates for likely parents of children 0-5 in both groups of states track each other well until about 2001 and diverge after that. The pre-1996 levels for likely parents of 11-16 year-olds show very similar trends pre-1996 but decidedly different paths after.

Finally, in Table A1, we show the first-stage estimates for the triplicate state IV (as reported in Table 1), the cancer-rate IV, and for both together. To evaluate the size of the first stage for the cancer-rate IV, note that the average difference in the cancer death rate from states in the top and bottom quartiles is about 200 deaths per 100,000. This means that for a 16-year old moving from a low to a higher cancer death rate state, the cumulative exposure of their likely parents increased by 38 per 100,000, and by 34 and 51 per 100,000 for mothers and fathers, respectively. The first-stage F-tests are north of 112 for all regressions in column (2). As there is some overlap between triplicate states and low cancer states in 1995, it is no surprise in column (3) that when we add both instruments, the first-stage coefficients from columns (1) and (2) are reduced in magnitude, but all coefficients in the column have very small standard errors relative to the parameter estimates and the first-stage F-tests are all greater than 99.

**Figure A2: Highest Minus Lowest Quartile States in the 1995 Cancer Death Rate Among Those Aged 57+**

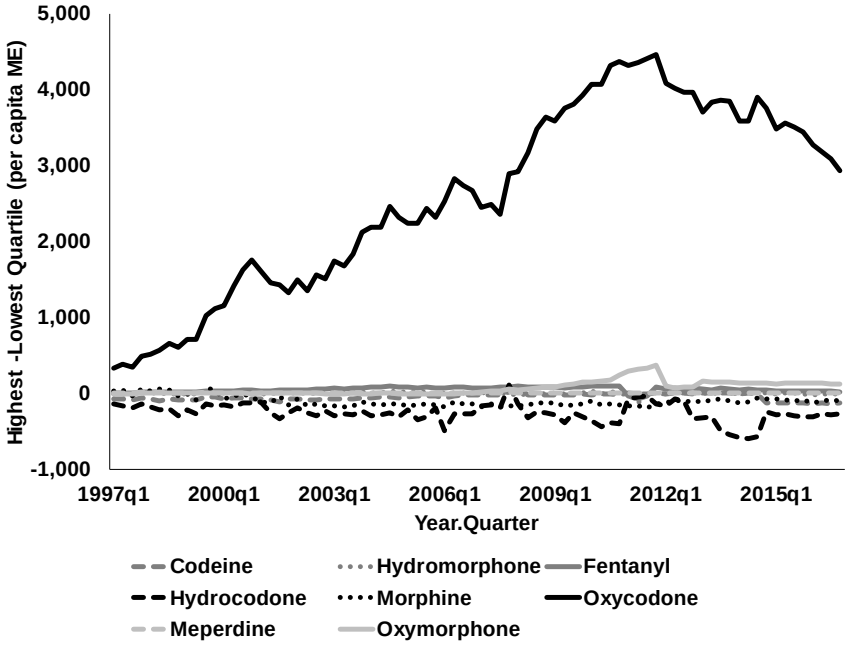
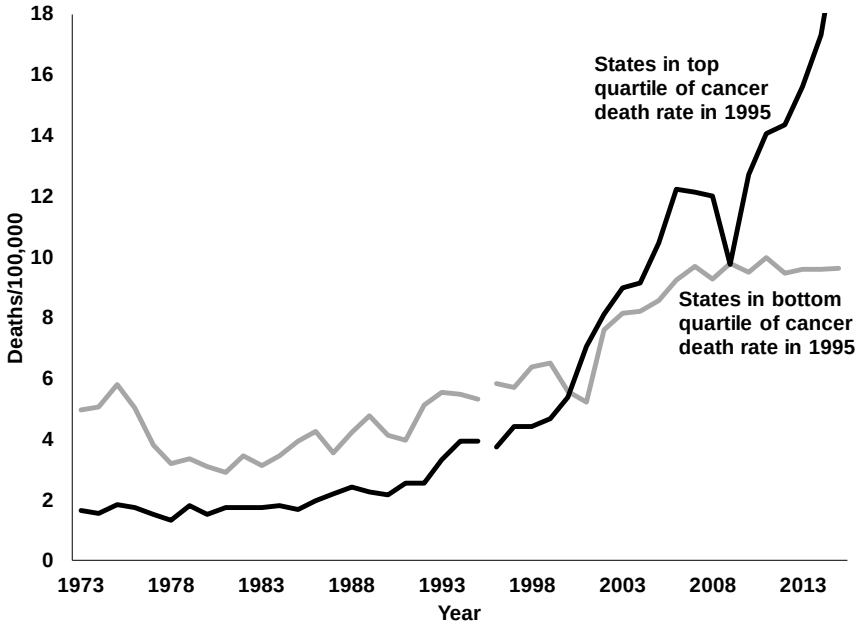



Figure shows the difference between the states in the highest and lowest quartiles of the 2005 cancer death rates for those aged 57 and above. Data are from DEA's ARCOS system. We use morphine equivalent grams per 100,000 people to put all drugs into comparable units. Codeine and hydrocodone were schedule 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 A3: Highest and Lowest Quartile States in 1995 Cancer Death Rates Among those Aged 57+**




Data are from the Multiple Cause of Death Data, 1973-2013. Series break point is 1996, the year Purdue Pharma released OxyContin.

**Figure A4: Highest and Lowest Quartile States in 1995 Cancer Death Rates Among those Aged 57+**

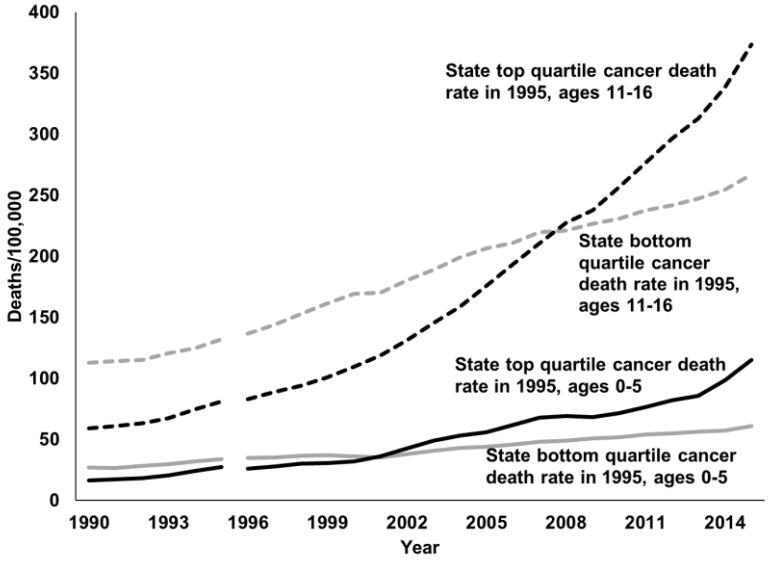



Figure shows trends in our measure of children's exposure to the drug crisis, $CEXPOSURE_{ast.,}$ for children of different ages in states in the top and bottom quartiles for age 57+ cancer mortality rates in 1995. See the text for details on how the $CEXPOSURE_{ast}$ measure is constructed. Data are from the Multiple Cause of Death Files. The series break point is 1996, the year Purdue Pharma released OxyContin.

**Table A1: First-Stage Estimates of Cumulative Drug Death Rate Equations, 1990-2015 ASEC**

| | (1) | (2) | (3) |
|---|---|---|---|
| **Panel A: Dependent variable is cumulative drug deaths/100K likely parents** | | | |
| $YearsExpNT_{ast}$ | 9.22 | | 6.392 |
| | (0.96) | | (1.407) |
| $YearsExp*CDR_{ast}$ | | 0.012 | 0.006 |
| | | (0.001) | (0.002) |
| F-statistic | 92.7 | 137.0 | 116.5 |
| Dependent Variable Mean | 110.9 | 110.9 | 110.9 |
| **Panel B: Dependent variable is cumulative drug deaths/100K likely mothers** | | | |
| $YearsExpNT_{ast}$ | 7.36 | | 5.016 |
| | (0.80) | | (1.082) |
| $YearsExp*CDR_{ast}$ | | 0.010 | 0.005 |
| | | (0.001) | (0.001) |
| F-statistic | 84.3 | 166.1 | 121.6 |
| Dependent Variable Mean | 75.3 | 75.3 | 75.3 |
| **Panel C: Dependent variable is cumulative drug deaths/100K likely fathers** | | | |
| $YearsExpNT_{ast}$ | 11.10 | | 7.870 |
| | (1.24) | | (1.990) |
| $YearsExp*CDR_{ast}$ | | 0.015 | 0.007 |
| | | (0.001) | (0.003) |
| F-statistic | 80.4 | 112.3 | 99.3 |
| Dependent Variable Mean | 146.1 | 146.1 | 146.1 |

Data on drug deaths are from the Multiple Cause of Death Files. In the first specification, the instrument is years of exposure to a non-triplicate state; in the second, the instrument is years of exposure to OxyContin interacted with the cancer mortality rate for people age 57 and over in 1995; in the third specification, both instruments are included. The second and third specifications include a control for the cancer mortality rate of likely parents (those age 17-40). 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 B

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

Given that we have ASEC data from 1990 through 2015 for children aged 0-16, we use the MCOD 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 MCOD files from 1983 to 2015, which contains state identifiers. We then supplement this with the publicly available versions of the MCOD files with state identifiers from 1973 through 1982, taken from the NBER website. In years 1973-1980, 1983-2015, the MCOD 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 MCOD 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).

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 under mental health classifications (304) that measures death due to drug dependence. 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 B1 below.

**Table B1**
**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. (2022), 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).

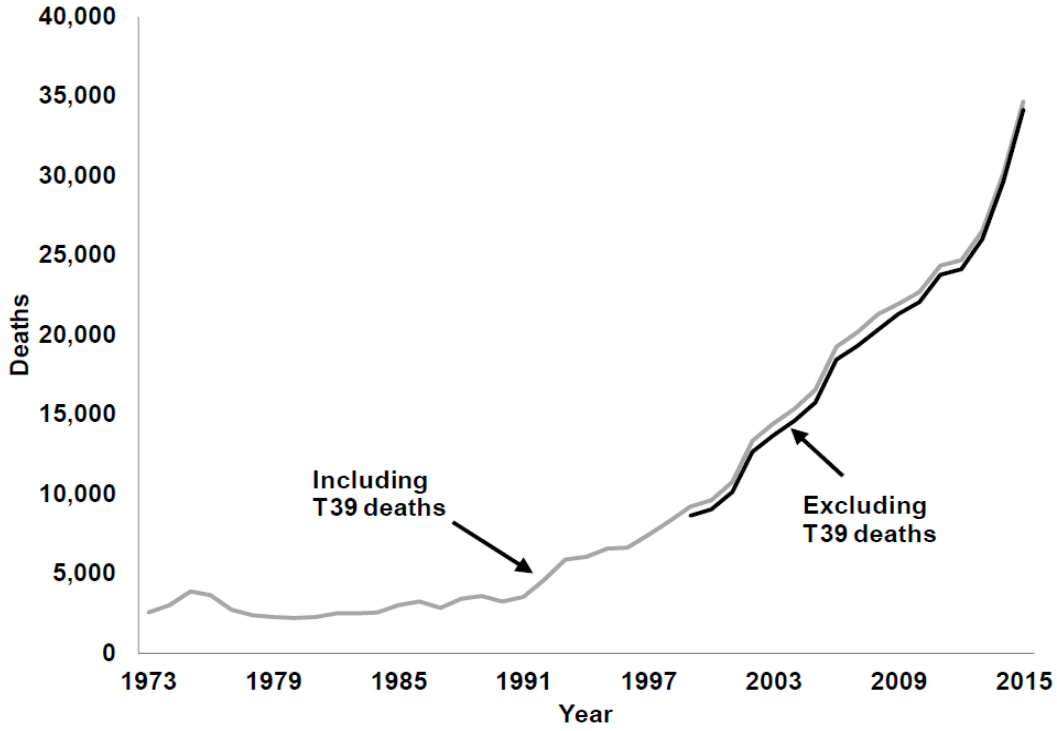
The problem we found is that in many cases during the ICD 8 and 9 era, the "965" condition codes are frequently not used when there was a drug death. In the ICD 9 era, we can identify opioids in some of the "E" codes – E850.0 (heroin), E850.1 (methadone), and E850.2 (opiates and related narcotics). Unfortunately, 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. 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 B1 below, the gray line shows the trend 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 B1**

**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 C
## Sensitivity, Robustness, and Heterogeneity

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 C1) as well as all subsequent robustness analyses (Appendix Tables C2-C5) (MacKinnon and Webb, 2018). For ease of comparison, the first column in most tables in this section contains the corresponding 2SLS results from Table 2.

*Appendix Table C1: 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 C1, 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 after 2013 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 C1, 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 C1. In most cases, 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 C2: Robustness to Additional Controls for Economic Conditions*

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 second column of results in Appendix Table C2, 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 third column, we include the state's unemployment rate. Hollingsworth et al. (2017) found a correlation between unemployment rates and drug overdose death rates, and Carpenter et al. (2017) show that the use of analgesics like Oxycodone is counter-cyclical. Again, our results are largely unaffected by this variable's inclusion. While unemployment rates might capture some of the variation in drug overdose 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.

*Appendix Table C3: Robustness to Additional Controls for the Social and Legal Environment*

In Appendix Table C3, we control for other features of states' social or legal environments that could affect both the severity of the drug crisis and children's living arrangements. First, 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. (2018)) for each state and include them in the model. 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 second column of the table, 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 third column of Appendix Table C4, adding these measures has very little impact on our estimated effects.

Another broad social change that was happening in the background as OxyContin was introduced was the dramatic reduction in crime rates. Violent crime in the US peaks in 1991 at 758/100,000 and falls by 52 percent by 2014. Over the same 1991 to 2014 period, property crime rates fell by 50 percent.[29] If crime rates were falling more or less rapidly in triplicate states, our estimates could be picking up the effects of changes in violence on children's living arrangements. In

[29] These national numbers are available in various annual reports by the FBI titled *Crime in the United States* and can be found here: https://www.fbi.gov/services/cjis/ucr/publications.

the final column of Appendix Table C3, we include controls for the state's violent crime and property crime rates. When we do so, we see the same clear pattern of results, though the coefficients are attenuated. This is what we would expect if one of the channels through which the opioid crisis affects children's living arrangements is that parents are committing crimes related to their drug use, as we suggested in the introduction.

*Appendix Table C4: Robustness to Omitting Treatment States for the Triplicate Instrument*

In Appendix Table C4, we test whether any single triplicate state is driving our results. We do so by dropping each triplicate in models with only this instrument, 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 C5: 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 C5, 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 C5. 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.[30] 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 C5, we present results in which the set of triplicate states has been restricted to California, Illinois, New York, and Texas and 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 C6: Randomization Inference Tests*

In Appendix Table C6, we show the results of a randomization inference exercise in which we compare our reduced form coefficients to those obtained by randomly assigning treatment status 10,000 times to obtain a distribution of possible point estimates. We construct three separate distributions by 1) randomly assigning five states to be triplicate states for each draw; 2) randomly assigning five states and also randomly assigning years of exposure to children in those states; and 3) randomly assigning treatment status to states to match the population size of the five actual triplicate states. Appendix Table C6 shows the position of our actual reduced form coefficients within these distributions. As we see in the table, the point estimates we obtain tend to be in the right tail of the distribution. The majority of the estimates would be significant at the ten percent level and many would be significant at the five percent level as well. These results reassure us that our estimated effects are not spurious.

*Appendix Table C 7: Results by Race*

The opioid crisis was more acute among White Americans—86% of likely parents who died from drug use between 1996 and 2015 were White. At the same time, there are much higher baseline probabilities that Black children are living away from a parent. Given these stark differences, in

[30] 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.

Table C7, we explore whether the effects of the crisis vary by race.[31] Within each group, the first column reports the sample mean of the outcome, while the second and third report 2SLS estimates of the effect of the crisis using the triplicate state instrument. The second column uses our cumulative exposure measure for all parents as the covariate of interest, while the third column uses a race-specific cumulative exposure measure. As with Table 2, the numbers in parentheses are standard errors while in this table, the numbers in brackets are now the first-stage F-statistics. First, focusing on the results using deaths to all parents as the instrument, we see that the results for White children are close to those reported in column (3) of Table 2, though slightly smaller for most outcomes. For Black children, the coefficient is smaller for missing a mother and larger for missing a father. When using the race-specific measure of deaths, the coefficients for White children are very similar. This is not surprising, as the aggregate measure is driven by deaths of White parents. The results for Black children are too imprecise to make definitive conclusions. This is a result of the much smaller samples and the substantially weaker first stage for this group, which is consistent with Powell (2021b) who shows that exposure to non-triplicate states produced a substantially larger impact for White adults.

[31] 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.

## Appendix Table C1

### 2SLS Estimates of the Impact of Cumulative Drug Death Rates of Likely Parents on the Living Arrangements of Children: Alternative Sample and Specification Choices

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

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

Data on living arrangements 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 C2

## 2SLS Estimates of the Impact of Cumulative Drug Death Rates of Likely Parents on the Living Arrangements of Children with Controls for Economic Conditions

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 |
|---|---|---|---|---|
| Mom not in household/100K | 12.88<br>(3.36)<br>[5.50, 20.33] | 13.23<br>(3.40)<br>[6.03, 20.60] | 13.16<br>(3.24)<br>[6.15, 20.26] | 12.78<br>(3.37)<br>[5.69, 19.95] |
| Dad not in household/100K | 9.68<br>(4.34)<br>[-2.01,19.12] | 9.03<br>(4.66)<br>[-3.39, 19.03] | 9.64<br>(4.31)<br>[-2.76, 18.80] | 9.44<br>(4.44)<br>[-3.15, 18.71] |
| Missing at least one parent/100K | 18.29<br>(5.63)<br>[2.38, 30.13] | 17.74<br>(5.91)<br>[2.22, 31.30] | 18.29<br>(5.57)<br>[2.53, 30.45] | 17.90<br>(5.76)<br>[1.44, 30.61] |
| Missing both Mom and Dad/100K | 3.64<br>(1.43)<br>[0.28, 6.62] | 3.71<br>(1.50)<br>[0.47, 6.75] | 3.82<br>(1.36)<br>[0.51, 6.50] | 3.67<br>(1.45)<br>[0.20, 6.57] |
| Grandparent head of HH/100K | 6.02<br>(2.17)<br>[1.60, 11.12] | 5.34<br>(2.64)<br>[-0.28, 11.59] | 5.99<br>(2.19)<br>[1.87, 11.22] | 6.08<br>(2.22)<br>[1.58, 11.26] |
| Non-parent head of HH/100k | 11.45<br>(2.62)<br>[6.10, 17.53] | 11.40<br>(3.12)<br>[4.92, 17.82] | 11.50<br>(2.66)<br>[6.25, 17.47] | 11.61<br>(2.70)<br>[6.46, 17.76] |
| Foster child / 100k | -0.07<br>(0.48)<br>[-1.10, 0.95] | -0.06<br>(0.52)<br>[-1.20, 1.06] | -0.06<br>(0.49)<br>[-1.15, 1.02] | -0.06<br>(0.48)<br>[-1.04, 0.91] |

Data on living arrangements 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, and the cancer death rate for likely adults. 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.

## Appendix Table C3

## 2SLS Estimates of the Impact of Cumulative Drug Death Rates of Likely Parents on the Living Arrangements of Children with Controls for the Social and Legal Environment

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

| Dependent variable | 2SLS from Table 1 | Rx Drug Monitoring Programs | Welfare Reform Controls | Crime Rate Controls |
|---|---|---|---|---|
| Mom not in household/100K | 12.88<br>(3.36)<br>[5.50, 20.33] | 12.07<br>(3.02)<br>[5.55, 18.72] | 12.49<br>(3.29)<br>[5.30, 19.53] | 9.63<br>(2.47)<br>[4.33, 14.81] |
| Dad not in household/100K | 9.68<br>(4.34)<br>[-2.01,19.12] | 8.62<br>(4.02)<br>[-1.57, 17.23] | 8.88<br>(3.70)<br>[-1.47, 17.00] | 6.12<br>(4.03)<br>[-2.86, 15.22] |
| Missing at least one parent/100K | 18.29<br>(5.63)<br>[2.38, 30.13] | 17.19<br>(5.08)<br>[3.53, 28.84] | 17.28<br>(4.82)<br>[3.89, 27.64] | 13.26<br>(5.06)<br>[0.81, 24.15] |
| Missing both Mom and Dad/100K | 3.64<br>(1.43)<br>[0.28, 6.62] | 2.85<br>(1.35)<br>[-0.24, 5.72] | 3.36<br>(1.40)<br>[0.12, 6.22] | 1.79<br>(1.11)<br>[-0.52, 4.02] |
| Grandparent head of HH/100K | 6.02<br>(2.17)<br>[1.60, 11.12] | 5.72<br>(1.97)<br>[1.91, 10.01] | 5.63<br>(2.04)<br>[1.64, 10.24] | 3.40<br>(2.84)<br>[-3.03, 9.52] |
| Non-parent head of HH/100k | 11.45<br>(2.62)<br>[6.10, 17.53] | 11.38<br>(2.64)<br>[5.77, 16.84] | 11.05<br>(2.58)<br>[5.77, 16.96] | 8.05<br>(3.18)<br>[1.28, 15.08] |
| Foster child / 100k | -0.07<br>(0.48)<br>[-1.10, 0.95] | 0.02<br>(0.49)<br>[-0.96, 1.08] | -0.10<br>(0.49)<br>[-1.12, 0.94] | -0.19<br>(0.51)<br>[-1.24, 0.87] |

Data on living arrangements 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, and the cancer death rate for likely adults. 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 prescription drug monitoring programs. The third column includes controls for the welfare reforms that occurred in the 1990s. The final column includes controls for the violent crime rate and the property crime rate.

## Appendix Table C4

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

Data on living arrangements 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 C5

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

Data on living arrangements 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 C6

### Results of Randomization Inference Tests: Position of Our Reduced Form Coefficients in Simulated Distributions

| | Simulated Distribution | | |
|---|---|---|---|
| | Randomly assign 5 triplicate states | Randomly assign 5 triplicate states and years of exposure | Randomly assign treatment status to match population size |
| Mom not in household/100k | 9512 | 9636 | 9873 |
| Dad not in household/100k | 7940 | 8490 | 9127 |
| Missing at least one parent/100k | 8089 | 8802 | 9455 |
| Missing both Mom and Dad/100k | 9957 | 9970 | 9880 |
| Grandparent head of HH/100k | 9664 | 9701 | 9781 |
| Non-parent head of HH/100k | 9298 | 9669 | 9852 |
| Foster child/100k | 8746 | 9470 | 7505 |

Data on living arrangements 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. Our treatment varies in two dimensions: 1) whether a state is a triplicate state and 2) how long a child has been exposed (based on child's age and years since 1996). In the first column, we have randomly assigned triplicate status to states, but have not randomized exposure that is based on the age and years since 1996. This randomizes only the first source of variation in treatment. In the second column, we have randomized both sources of variation in the treatment. In the third column, we have only randomized the first source of variation, but instead of basing it on the number of states that were triplicates in 1995, we assign states to treatment until we have reached the same fraction of the 1995 population that was in a triplicate state. We report the number of estimated coefficients that are smaller than the estimate we obtained with our actual treatment variable.

## Appendix Table C7

### 2SLS Estimates of the Impact of Cumulative Drug Death Rates of Likely Parents on the Living Arrangements of Children by Race, 1990-2015 ASEC

Parameter Estimates (Standard Errors) [First-Stage F-Statistic] on $CEXPOSURE_{ast}$

| | White Children | | | Black Children | | |
|---|---|---|---|---|---|---|
| | | Measure of Likely Parents' Mortality | | | Measure of Likely Parents' Mortality | |
| Dependent variable | Mean | All Parents | White Only | Mean | All Parents | Black Only |
| Mom not in household/100K | 5,494 | 11.88<br>(3.24)<br>[81.51] | 10.83<br>(3.07)<br>[76.15] | 10,363 | 7.32<br>(7.39)<br>[96.92] | 19.52<br>(19.92)<br>[7.06] |
| Dad not in household/100K | 18,318 | 7.89<br>(5.07)<br>[78.50] | 7.24<br>(4.71)<br>[74.57] | 56,477 | 16.32<br>(10.46)<br>[59.01] | 52.42<br>(50.33)<br>[2.93] |
| Missing at least one parent/100K | 21,603 | 16.94<br>(6.99)<br>[89.23] | 15.49<br>(6.55)<br>[83.42] | 60,379 | 20.86<br>(12.43)<br>[78.11] | 61.23<br>(47.19)<br>[4.72] |
| Missing both Mom and Dad/100K | 2,208 | 2.03<br>(1.24)<br>[89.23] | 1.86<br>(1.15)<br>[83.42] | 6,461 | 4.64<br>(4.16)<br>[78.11] | 13.62<br>(13.98)<br>[4.72] |
| Grandparent head of HH/100K | 4,214 | 6.46<br>(2.03)<br>[89.23] | 5.91<br>(1.88)<br>[83.42] | 10,470 | 5.60<br>(6.67)<br>[78.11] | 16.44<br>(22.12)<br>[4.72] |
| Non-parent head of HH/100k | 7,935 | 11.26<br>(2.78)<br>[89.23] | 10.30<br>(2.62)<br>[83.42] | 16,739 | 15.40<br>(7.93)<br>[78.11] | 45.21<br>(31.44)<br>[4.72] |
| Foster child / 100k | 241 | -0.55<br>(0.35)<br>[89.23] | -0.50<br>(0.32)<br>[83.42] | 631 | 2.57<br>(1.62)<br>[78.11] | 7.53<br>(6.84)<br>[4.72] |

Data on living arrangements 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; the F-statistic for the first stage is reported in brackets.

## Appendix D

## Additional Details

*D1: Measurement error related to in- and out-migration*

Our measure of cumulative exposure is based on data from the ASEC files and unfortunately, the ASEC only includes child's current state of residence, not her birth state. Migration in response to the drug crisis would cause measurement error that could lead us to either under- 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.

*D2: Estimating the number of children in households with a parental drug death*

Using data from the 1986 through 2004 mortality follow-back of the National Health Interview Survey (Blewett et al., 2019), we calculate that households with an adult death related to drug poisoning in the four quarters after the household entered the survey included an average of 0.71 children.[32] Assuming this number is representative for more recent years, we can scale the 568,699 adult drug deaths from 1999 to 2018 to estimate the number of children directly affected by a parent's drug death. That number is 398,089 (rounded to 389,000 in the main text).

[32] 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.

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