Child overdoses amid the deaths-of-despair epidemic: Racial and ethnic differences in intergenerational and network diffusion

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Abstract

Since the early 2000s, contributors to the deaths-of-despair epidemic such as alcohol and drug related deaths have more than doubled. Evidence of the role of patient networks in the diffusion of prescription behaviors among physicians and of intra-household diffusion of opioids use contribute to important new questions about the population level effects of workplace- and intergenerational networks. This study responds to this need by expanding the focus from households and opioids overdose risk to examine the role of intergenerational and commuting networks in the diffusion of overdose risk from alcohol and drugs more generally among children. Analyses using negative binomial regression combined with computational statistics approaches such as cross-validation and permutation indicated that exposures to extra-local network overdose risks were associated with local adult- and child overdose deaths. These associations remained significant after controlling for multiple socioeconomic and demographic factors. The results showed that the link between network overdose risk and local child overdose deaths was accounted for in large part by intergenerational effects. Above and beyond intergenerational and spatial diffusion, network effects remained significant for Black children. High concentrations of other white residents protected white children against overdose risk but none of the minority groups. In turn, higher concentration of minority residents protected Black and Hispanic children. Higher population density increased the risk of overdose deaths among adults of all racial and ethnic groups, consistent with expectations of social and economic strain. However, it decreased the risk among children, consistent with social control expectations. Implications for future research and policy are discussed.

Introduction

Across the country, life expectancy has been declining[4], showing variations across US counties of over 20 years[5]. Important contributors to this decline are opioids and other drug and alcohol related deaths, often discussed as part of the "deaths of despair" phenomenon (Case and Deaton

4 US life expectancy: Americans are dying young at alarming rates - The

5 https://www.washingtonpost.com/news/to-your-health/wp/2017/05/08/u-s-life-expectancy-varies-by-more -than-20-years-from-county-to-county/

2015, 2017), and which have more than doubled since the early 2000 (US Congress 2019). The focus in research studies and media has largely been on adults. However, little is known about the extent to which the social forces that have been affecting adults are beginning to affect children as well. The good news is that child overdose death rates are lower than for adults and indications exist that teenagers have been using fewer illegal drugs, prescription opioids, and alcohol[6] in the past years[7]. In our analyses (Figure 1), we see a similar decline in child overdose deaths in 2017 compared to the peak years of 2007-2010. However, the bad news is that the 2017 levels still remain double compared to the 1999 level. Despite this overall growth in lethal child overdose prevalence, little is known about the extent to which the risks that have contributed to the recent, unprecedented rise in adult overdoses are beginning to diffuse and affect child overdoses as well.

Recent research has highlighted the importance of intra-household diffusion of prescription opioids use among family members and the role of increased patient demand and patient sharing networks in the diffusion of prescription behaviors among physicians (deVaam and Tobin 2019). These findings suggest that adult overdose behaviors and risk factors may diffuse to also affect children in the household. Importantly, they also point to the role of the social contexts in which adults share and learn from each other about what doctors to ask and how to ask for prescriptions. One important such social context is the work environment. Co-workers are known to be important friends and confidants in many people's lives (Marsden 1982).

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6 https://www.drugabuse.gov/publications/drugfacts/monitoring-future-survey-high-school-youth-trends

7 https://www.bloomberg.com/opinion/articles/2020-01-08/american-deaths-of-despair-aren-t-the-whole-story

Moreover, when coworkers share their employer insurance plan, it further increases their chances of using the same physician networks.

These ideas point to the possibility that, similar to intra-household diffusion, networks at work and commuting networks between work and home, may also contribute to diffusing risk for illegal or prescription drug use and overdose among adults and children. Similarly, when many coworkers drink alcohol, they may influence each other for better or worse. On the more positive side, work networks that have been successful at dealing with risky behaviors and addictions, may transmit information, attitudes, norms, and resources that may also help commuters and their families deal with risk behaviors and addictions, whether they involve drugs or alcohol.

In the current study, we investigate the role of intergenerational and extra-local commuting networks in diffusing child overdose risk across all US counties. Specifically, we ask the following research questions:

1. Are network exposures to child overdose risk related to variations across the US in county rates of child overdose deaths, independent of demographic and socioeconomic factors or selection processes?

2. To what extent is the association explained by local adults' overdoses or spatial spillovers?

3. What variations, if any, are there in network and intergenerational spillovers across racial and ethnic groups?

Theoretical and Empirical Background

People who commute to areas of high child overdose risk may be affected in ways that negatively shape their home climate and their children's risks. On one side, a culture of drug and alcohol use in their work environments may normalize perceptions of substance use among commuters and increase their own substance use. As commuters' substance use increases, they may become dysfunctional role models and less effective at monitoring their children at home. Moreover, their new attitudes and behaviors may normalize risky behavior for friends and family, who in turn may themselves become dysfunctional role models and ineffective at parenting and supervising their children. Additionally, the home environments of such commuters may become riskier, increasing also risk exposures for their own children as well. The more alcohol, drugs, and prescription medicine are to be found around homes and in the community, the more likely it is that children will find them and try them, some unfortunately, with fatal consequences (Crum, Lillie-Blanton, and Anthony 1996; Kendler et al. 2016).

On the positive side, exposures to network areas of low overdose risk may be associated with work area exposures to resources, social support and programs that can help prevent or deal with such problems as soon as they show up, connecting commuters with the same resources and thus helping them and their home environments, decreasing children's risk at home. Overall, these ideas lead to the expectation that *a community's exposure to higher levels of overdose deaths in its commuting network would contribute to higher levels of local child overdose deaths* (H1a)

It is also possible that exposures to high child overdose risk on one's commuting network may provide warnings and caution tales for commuters who may then work harder to protect their children at home, contributing to lower child overdose risk in their home communities. This would suggest an alternative expectation of a *negative link between network and local child overdose deaths* (H1b).

Selection processes. Processes of socioeconomic, demographic, and employment selection, related to residents and employers preferences and exclusion, are likely to increase the chances that communities of similar disadvantage level be connected through commuting (Graif et al 2017). To the extent that exposures to disadvantage in the commuting network affect network child overdose as well as local disadvantage risks including child overdose, an observed connection between network child overdose and local child overdose may be confounded by sociodemographic selection processes. Highly disadvantaged areas have been shown to be more connected to other highly disadvantaged areas. This phenomenon has been observed often in spatial proximity analyses (REF). An emerging strand of research has also identified examples that go beyond geographic proximity to suggest patterns of homophily in inter-neighborhood connections (i.e., connections are more likely and stronger between areas that are similar in disadvantage levels) based on social ties such as transportation networks, co-offending ties, residential mobility and commuting (Graif et al 2017; Scheffer 2012; Papachristos xxx). Based on this body of work, *we expect that exposures to network disadvantage may explain at least in part the network overdose effect on child overdose (H2)*.

Possible mechanisms. Different possible mechanism pathways may be underlying the connection between network overdose and local child overdose deaths. In this study, we

investigate two of such possible pathways, spatial spillovers and local adults' overdose risk. Proximity between two areas likely increases the chances of commuting links between them. High levels of child overdose risk in geographically proximate areas may spillover to affect local child overdose deaths. Similar spatial processes have often been observed for homicide and other violence (Baller et al. 2001; Light and Harris 2012; Messner et al. 1999); it is possible then, that network spillovers may largely operate through spatial spillovers. In sum, we expect that *spatial proximity to areas of high child overdose death levels may explain at least in part the connection between network child overdose and local child overdose deaths (H3).*

Adults are typically the majority of the working population across communities and the larger commuting group among workers. Commuting exposures to overdose deaths would thus likely impact overdose deaths among local working adults before they would impact children. The more local adults suffered from overdose deaths in a county, the more children would also be affected by overdose deaths due to intergenerational transmission of risk, or due to effects of family and community contexts on child outcomes. In sum, we expect that *local adult overdose deaths may explain in part, or in full, the connection between network overdose and local child overdose deaths (H4)*.

We know little about how network exposure processes may shape racial and ethnic disparities in child overdose deaths but a large literature has documented such disparities in access to health care and in the quality of health services (e.g., Trivedi et al. 2006). If minority groups were on average at a disadvantage on this dimension alone, it would predict that *networks may be more influential in shaping health outcomes like overdose deaths for them and their children than they would be for whites* (H5a).

On the other hand, access to health care has been associated with abuse of prescription drugs like opioids (Wright et al. 2014). This may suggest that whites, who, on average have better access to health care, may be at higher risk than minority groups to suffer negative outcomes from prescription drug abuse, including overdose deaths. Commuting networks in this case may function as possible channels of transmission of information about how to abuse the healthcare system to get desired, addictive prescription drugs. They may also operate potentially as black market channels for such drugs. These processes lead to an alternative expectation that *networks may be more influential in shaping overdose deaths for whites than for minority groups (H5b)*.

Methods

Relevant, county-level data were drawn from several national databases including CDC WONDER, the American Community Surveys, and the Longitudinal Employer-Household Dynamics (LEHD) Origin-Destination Employment Statistics (LODES) database. Table 1 shows descriptive statistics for all relevant variables for the entire sample and disaggregated by time period.

Dependent Variables

Death data are drawn from the CDC WONDER mortality database (Multiple Cause of Death 1999-2019). We obtained counts of the number of deaths due to drug and alcohol overdose for children (under the age of 18) and adults (18 and over) for three, consecutive, 5-year time periods: 2005-2009, 2010-2014, and 2015-2019. We obtained counts summed over all racial/ethnic groups and disaggregated to non-Hispanic White (NHW), non-Hispanic Black

(NHB), and Hispanic. As such, the following analyses focus on estimating child and adult deaths for all races, NHW, NHB, and Hispanic deaths.

Socioeconomic and demographic variables

Local disadvantage levels, population density, location in urban vs rural areas, and Appalachian location, may all contribute to important variations in adult and child overdoses. We include in our analyses the following measures. County *disadvantage* data were derived from the 5-year county estimates from the American Communities Survey (ACS) corresponding to the 5 year periods over which deaths were aggregated. Disadvantage measures include unemployment rate for residents between the ages of 20 and 64, percent of residents living in poverty, the median income, percent of vacant housing units, and percent of residents 25 and older who have at least a bachelor's degree. These disadvantage measures were combined using principal components analysis (separate PCA for each time period) and the first principal component transformation (which had positive factor loadings for unemployment, poverty, and vacant housing, and negative factor loadings for median income and college education) is used in subsequent analyses (eigenvalues = ranged from _ to _, PCA details are available upon request) to capture community disadvantage. *Population density,* log transformed to address the highly skewed distribution, was also drawn from ACS and census data. Measures for the *racial and ethnic* make-up of counties were also obtained from the ACS and used in subsequent models. Using measures for percentage NHW, NHB, and Hispanic, binary variables were constructed indicating whether a county's percentage of each racial group falls within the top tercile of all counties, within each time period. A binary indicator of whether counties fall within the

Appalachian region was also included, based on the definition from the Appalachian Regional Commission (arc.gov).

Spatial and extra-local networks: data and measures

Spatially lagged measures of adult OD rate were created using a spatial weight matrix based on a row-normalized, queen county contiguity matrix. Network-lagged measures were also constructed using origin-destination commuting data from the LODES dataset. Each network lagged measure for a given county (e.g. County X) is a weighted sum of that variable for all counties connected to County X by outgoing commuters. The sum is weighted by the proportion of all outgoing commuters for County X which comprise a given commuting tie. These relational measures are described in Equations 1 and 2.

[Insert Equations 1 and 2 here]

Table 1 shows descriptive statistics for each variable, disaggregated by time-period and race/ethnicity.

[Insert Table 1 about here]

Analytic strategy

Mixed effects negative binomial regression models

We estimate mixed effects negative binomial regression models to account for panel structure of our data (time periods nested in counties). For all models, the exposure term is the total

population of individuals for whom death due to OD would be counted in the outcome (e.g., for the model predicting NHW child OD, exposure equals the population of NHW children), making model coefficients interpretable as changes to population death rates (see Osgood, 2000). We begin with a series of nested models exploring the predictors of total adult and child OD deaths Key predictors are the network and spatially lagged OD terms. In the models predicting child OD, we also include for local adult OD rate as a predictor, accounting for the possibility of drug and alcohol abuse diffusing adults to children, perhaps within households (e.g., de Vaan and Stuart, 2019). Next, we disaggregate the outcomes by race and estimate separate models for non-Hispanic White, non-Hispanic Black, and Hispanic deaths.

Permutation-based assessment of predictor significance

Our research questions focus on the processes through which counties influence one another with regard to child OD. However, our focus on network ties explicitly highlights that observations are not independent of one another, violating the independence assumption of regression techniques. Consequently, estimated standard errors obtained using traditional methods may be biased. As an alternative means of evaluating the significance of each predictor, we perform a series of permutation tests, randomly permuting each predictor and fitting the model 1000 times, producing a distribution of model errors (measured as mean absolute error, see Equation 3) under the null hypothesis that the permuted predictor has no relationship with the outcome. Each model's observed MAE is compared to this distribution, and the proportion of permuted models in which the MAE was lower than the observed MAE (indicating higher model accuracy) is reported. A low proportion indicates that relatively few permuted models outperformed the unpermuted model, suggesting that the permuted variable is

important to model fit. Because these significance tests are derived from simulation rather than statistical theory, they do not rest on the assumption of observation independence and may be used to assess the significance of individual predictors for our dataset of interconnected counties.

[Insert Equation 3 here]

Results

Table 2 shows coefficient and standard error estimates from mixed effects negative binomial regression models predicting OD deaths for adults and children. Models 1-3 predict total adult OD deaths, while Models 4-7 predict total child OD deaths. Turning first to adult OD deaths, Model 1 includes only sociodemographic controls, Model 2 adds the spatial lag term, and Model 3 adds the network lag term. As shown, many of the initially included controls are significant predictors of total adult OD deaths. As shown, disadvantage, population density, Appalachian status, and later time period (compared to the 2005-2009 period) are all positively associated with deaths. On the other hand, concentrations of NHW and NHB populations are negatively associated with deaths. The spatially lagged term (added in Model 2) shows a significant, positive association with deaths. This suggests that total adult OD deaths tend to be clustered in space, either because of behavioral "spillovers" (i.e., one county's OD rate directly influences its neighbors) or because of unmeasured, exogenous influences which are spatially clustered and not captured by the other control terms. Importantly, the network lagged term (added in Model 3) is also positively associated with OD deaths, even after controlling for these spatial effects. The addition of the commuting network lag also explains a portion of the spatial

effect (note the diminished spatial lag coefficient from Model 2 to Model 3). Finally, a comparison of model fit statistics (AIC and BIC) reveals that including the network lag contributes to model fit above and beyond the spatial lag. All of these results suggest that commuting networks matter to adult OD deaths in a way which is independent of spatial contiguity and local structural correlates of OD.

Turning next to Models 4-7, these show coefficient and standard error estimates from similar mixed effects negative binomial regression models predicting child OD deaths. As shown, most sociodemographic predictors have similar associations with total child OD deaths as they do with total adult OD deaths. The only substantive differences are that population density now has a negative association with deaths, and later time periods have fewer deaths, on average (compared to the 2005-2009 period). In Models 5 and 6, space and network lagged terms are again shown to be positive predictors. However, these associations are largely explained by local adult OD (added in Model 7). For total child OD deaths, the effects of spatial and network spillovers seem to be primarily mediated by local adult deaths.

[Insert Table 2 here]

Next, we turn to findings from the models which predicted deaths disaggregated by race/ethnicity. Table 3 shows these results. Turning first to the models predicting adult OD deaths (Models 1-3), some predictors have similar effects across all racial groups. Notably, the (race-specific) network lagged term positively predicts adult OD across all races, with a similar magnitude for White and Black OD deaths and a larger effect for Hispanic OD deaths. However, spatially lagged OD deaths are only associated with White deaths, after accounting

for the commuting network. To the extent that Black and Hispanic OD deaths "spillover" across communities, the process seems to be largely mediated by commuting networks. Disadvantage is positively associated with all adult OD deaths, but the effect is weakest for Black OD deaths. Non-Hispanic White population concentration is associated with decreased White deaths, but increased Black deaths. On the other hand, Black population concentration is associated with lower deaths for both Blacks and Hispanics (and marginally so for Whites). Hispanic population concentration is associated with higher deaths among Whites and Blacks. Overall population density is positively associated with deaths for all races, but most strongly for non-Hispanic Blacks. Appalachian status is associated with higher deaths for Whites, but lower deaths for Hispanics. All racial groups experienced increasing death rates across the three time periods, with the steepest growth for Black OD deaths.

Turning next to the models predicting child OD deaths, we see that local adult OD is a positive predictor for White, Black, and Hispanic children. Even after accounting for local adult OD, network lagged adult OD matters for Black child OD deaths, and spatially lagged adult OD matters for Hispanic child OD deaths. Disadvantage is a significant predictor of higher White child OD deaths, while White population concentration is associated with lower White child OD deaths. Black population concentration is associated with lower deaths for Black and Hispanic children, and Hispanic population concentration is associated with higher deaths for White children. In contrast to the results for adults, population density is associated with fewer child OD deaths for White and Hispanic children. Appalachian status is a marginally significant predictor of White child OD deaths, but not for Black or Hispanic OD deaths. Net of these other

factors, death rates have fallen for White and Hispanic children since the 2005-2009 time period, while remaining roughly constant for Black children.

[Insert Table 3 here]

Table 4 shows the results of the permutation tests for each key predictor in fully specified models shown in the previous tables. As shown, permutation tests for the adult OD models show the importance of the network lagged measures to model fit. Specifically, this predictor improved model performance (as measured by MAE) from models which used permuted versions of this predictor in all 100 trials. Spatially lagged measures also consistently improved model fit for the total and NHW models, but were less consistent at improving model fit for the NHB and Hispanic models. The child OD results are less clear. Although local adult OD was a strong, positive predictor of child deaths, the permutation tests reveal that the inclusion of this predictor often diminishes model fit. For the NHW models including local adult OD somewhat improved model fit (p-value = .17), but the other models tended to experience reductions in performance after this measure was included. Instead, network-lagged OD deaths were most beneficial to the performance of the NHB child OD model (p-value = .07), while spatially lagged OD deaths were most beneficial to the performance of the Hispanic child OD model (p-value = .07).

[Insert Table 4 here]

Discussion

Network levels of adult overdose deaths were found to be associated with local child overdose levels, and this association remained significant after controlling for local disadvantage, racial and ethnic diversity and population density. This connection is also not accounted for by a community's level of urbanization or Appalachian location.

An important potential selection mechanism we investigated that could potentially explain such an association was local disadvantage. To the extent that highly disadvantaged areas are more likely to be connected to other highly disadvantaged workplace areas, we expected that disadvantage could explain away the network overdose effect. Yet the results indicate that the network overdose effect remained significant and little affected in its magnitude by disadvantage.

One important mechanism of diffusion examined was adult overdoses. We hypothesized that commuting level exposures to overdose deaths would impact local working adults and the more local adults suffered from overdose deaths in a county, the more children would be affected by overdose deaths as well. The network effect seemed to be explained by local adults overdose levels in the general population of children. Still, when including adult overdose to the model, the network effect remained significant for Black children.

A second core mechanism of diffusion hypothesized was through spatial proximity to areas high in child overdose death levels. Proximity may increase the chances of commuting between any two areas and proximity to other high overdose prevalence areas may spillover to affect local child overdose deaths. However, this potential pathway did not explain away the network overdose effect, which remained significant and even slightly increased in magnitude.

The study contributes to the literature by highlighting for the first time the importance of nonresidential, extra-local exposures to overdose risk in shaping local child overdose deaths. These findings build on a rapidly growing literature that has highlighted the importance of spatial spillovers and of exposure to risk in communities where people spend time for various routine activities and extend this literature by showing evidence consistent with network spillover effects on child overdose deaths across large communities. While the commuting networks inevitably overlap at least in part with spatial proximity networks, this study indicates that networks operate above and beyond contiguity processes. Moreover, the network spillovers seem to also operate for historically vulnerable minority children above and beyond factors that shape local adults' overdose risk. Much work remains to be done to investigate the sources of the observed disparities and to understand the possible mechanisms in more depth. Importantly, the current study has focused on counties, and important variations in possible mechanisms may operate on a more geographically granular level. We hope our findings contribute important motivation for future research investigating these questions on communities of different geographic and social scales.

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Tables and Figures

Table 1. Descriptive statistics by time period

	2005-2009				2010-2014				2015-2019			
	Mean	SD	Min.	Max.	Mean	SD	Min.	Max.	Mean	SD	Min.	Max.
Adult OD rate												
Total	12.50	9.15	.00	69	15.38	11.56	.00	118	20.19	14.37	.00	139
NHW	13.48	9.96	.00	78	16.68	12.14	.00	120	21.59	15.40	.00	139
NHB	9.11	52.13	.00	2381	10.20	58.08	.00	2222	19.06	59.29	.00	1852
Hispanic	5.71	21.31	.00	388	7.37	30.66	.00	1282	11.05	30.56	.00	1031
Child OD rate												
Total	.51	1.53	.00	29	.42	1.45	.00	23	.34	1.56	.00	52
NHW	.55	2.00	.00	51	.44	1.73	.00	30	.32	1.58	.00	45
NHB	.23	3.26	.00	143	.34	9.37	.00	493	.34	5.09	.00	190
Hispanic	.41	7.29	.00	319	.30	6.53	.00	337	.24	3.74	.00	132
Network-lagged adult OD rate												
Total	12.86	5.48	1.79	48	15.89	6.84	2.29	64	22.14	10.70	5.84	102
NHW	14.20	6.09	.95	49	17.93	7.24	2.19	67	24.18	11.40	4.17	103
NHB	11.09	17.88	.08	658	11.77	7.80	.12	115	23.04	14.80	1.19	128
Hispanic	5.93	6.18	.05	71	7.72	6.23	.57	140	12.79	8.43	1.07	59
Spatially-lagged adult OD rate												
Total	12.41	6.85	.00	50	15.38	8.85	.00	91	20.16	11.16	.00	87
NHW	13.38	7.42	.00	53	16.62	9.14	.00	92	21.55	11.88	.00	88
NHB	9.26	22.14	.00	476	10.01	21.30	.00	444	18.87	25.79	.00	463
Hispanic	5.58	9.12	.00	107	7.48	13.65	.00	263	11.06	12.90	.00	172
Sociodemographic controls												
Disadvantage index	-.07	.88	-4.38	3.43	-.07	.90	-4.38	3.07	-.07	.88	-3.89	3.07
Upper tercile NHW	.34		0	1	.34		0	1	.34		0	1
Upper tercile NHB	.35		0	1	.34		0	1	.34		0	1
Upper tercile Hispanic	.32		0	1	.32		0	1	.32		0	1
Population density (logged)	3.88	1.60	.04	11.16	3.87	1.63	.04	11.18	3.87	1.65	.04	11.18
Appalachian indicator	.14		0	1	.14		0	1	.14		0	1

Notes: N=9,359 US counties
Lagged measures are total adult OD rates per 100,000
Disadvantage score is based on college graduates, unemployment, poverty, median income, and vacant housing units: standardized first principal component transformation

Table 2. Mixed effects models predicting overdoses across all racial and ethnic groups

	Adult Overdose Deaths			Child Overdose Deaths			
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Local adult OD							.02 ***
							(.002)
Network-lagged adult OD			.02 ***			.01 **	.00
			(.002)			(.005)	(.005)
Spatially-lagged adult OD		.04 ***	.02 ***		.02 ***	.01 *	-.01
		(.001)	(.001)		(.002)	(.005)	(.005)
Disadvantage index	.16 ***	.12 ***	.12 ***	.12 ***	.10 ***	.10 ***	.04
	(.012)	(.010)	(.010)	(.026)	(.026)	(.026)	(.025)
Upper tercile NHW	-.07 **	-.08 ***	-.09 ***	-.37 ***	-.37 ***	-.38 ***	-.35 ***
	(.022)	(.018)	(.018)	(.070)	(.071)	(.071)	(.070)
Upper tercile NHB	-.17 ***	-.09 ***	-.09 ***	-.16 **	-.12 *	-.12 *	-.09 *
	(.022)	(.017)	(.017)	(.049)	(.048)	(.048)	(.047)
Upper tercile Hispanic	.02	.01	.02	.06	.08 †	.10 *	.11 **
	(.020)	(.015)	(.015)	(.045)	(.044)	(.045)	(.043)
Population Density (log)	.14 ***	.08 ***	.08 ***	-.07 ***	-.10 ***	-.10 ***	-.12 ***
	(.008)	(.006)	(.006)	(.017)	(.017)	(.017)	(.015)
Appalachian status	.36 ***	.06 **	.05 *	.20 **	.06	.06	.07
	(.029)	(.021)	(.021)	(.061)	(.065)	(.066)	(.064)
Time (Ref. 2005-2009)							
2010-2014	.20 ***	.08 ***	.07 ***	-.32 ***	-.38 ***	-.39 ***	-.38 ***
	(.008)	(.007)	(.007)	(.039)	(.039)	(.039)	(.039)
2015-2019	.51 ***	.17 ***	.12 ***	-.41 ***	-.62 ***	-.67 ***	-.68 ***
	(.011)	(.010)	(.012)	(.044)	(.050)	(.053)	(.053)
Constant	-9.63 ***	-9.78 ***	-9.82 ***	-12.87 ***	-13.06 ***	-13.11 ***	-13.01 ***
	(.036)	(.028)	(.028)	(.089)	(.086)	(.087)	(.084)
AIC	65391	62249	62098	11043	10963	10959	10869
BIC	65470	62334	62191	11122	11049	11052	10969

Notes: N=9,359 US counties; ***p < .001; ** p < .01; * p < .05; † p < 0.10
Exposure = county age-specific population
Lagged measures are total adult OD rates per 100,000
Disadvantage score is based on college graduates, unemployment, poverty, median income, and vacant housing units: standardized first principal component transformation

Table 3. Mixed effects models predicting overdoses disaggregated by race and ethnicity

	Adult Overdose Deaths			Child Overdose Deaths		
	NHW	NHB	Hispanic	NHW	NHB	Hispanic
Local Adult OD				.02 ***	.003 ***	.01 ***
				(.003)	(.0004)	(.001)
Network-lagged adult OD	.02 ***	.02 ***	.05 ***	.01 †	.01 **	.01
	(.001)	(.003)	(.002)	(.005)	(.003)	(.007)
Spatially-lagged adult OD	.02 ***	.00	.00	.00	.00	.01 ***
	(.001)	(.001)	(.002)	(.005)	(.003)	(.004)
Disadvantage index	.14 ***	.07 ***	.12 ***	.09 **	.10 †	.08
	(.009)	(.021)	(.020)	(.029)	(.053)	(.058)
Upper tercile NHW	-.08 ***	.22 ***	-.03	-.26 ***	-.62	-.11
	(.017)	(.064)	(.058)	(.074)	(.452)	(.367)
Upper tercile NHB	-.03 †	-.27 ***	-.20 ***	-.04	-.32 *	-.22 *
	(.016)	(.043)	(.035)	(.055)	(.153)	(.114)
Upper tercile Hispanic	.07 ***	.09 **	-.02	.18 ***	-.04	.08
	(.015)	(.033)	(.036)	(.050)	(.104)	(.171)
Population Density (log)	.09 ***	.21 ***	.10 ***	-.06 ***	-.03	-.11 **
	(.006)	(.016)	(.013)	(.019)	(.040)	(.037)
Appalachian status	.05 *	.08	-.18 **	.13 †	-.12	-.13
	(.019)	(.053)	(.061)	(.072)	(.167)	(.301)
Time (Ref. 2005-2009)						
2010-2014	.07 ***	.05 *	.10 ***	-.37 ***	-.14	-.27 **
	(.008)	(.019)	(.021)	(.046)	(.116)	(.100)
2015-2019	.10 ***	.51 ***	.25 ***	-.76 ***	.03	-.22 †
	(.012)	(.039)	(.025)	(.062)	(.102)	(.122)
Constant	-9.82 ***	-10.60 ***	-10.46 ***	-12.10 ***	-12.30 ***	-12.33 ***
	(.028)	(.079)	(.063)	(.100)	(.256)	(.252)

Notes: N=9,359 US counties; ***p < .001; ** p < .01; * p < .05; † p < 0.10
Exposure = county race-specific, age-specific population
Lagged measures are race-specific adult OD rates per 100,000
Disadvantage score is based on college graduates, unemployment, poverty, median income, and vacant housing units: standardized first principal component transformation

Table 4. Estimated coefficients and permutation-based p-values for key predictors in mixed effects negative binomial models

	Total		NHW		NHB		Hispanic	
	Coef.	p-value	Coef.	p-value	Coef.	p-value	Coef.	p-value
Adult OD Models								
Network-lagged OD	.015	.00	.016	.00	.017	.00	.051	.00
Spatially-lagged OD	.023	.00	.020	.00	.000	.30	.001	.38
Child OD Models								
Local adult OD	.023	1.00	.017	.17	.003	1.00	.006	.90
Network-lagged OD	.005	.97	.010	1.00	.008	.07	.005	.63
Spatially-lagged OD	-.005	.97	-.003	.99	.003	.89	.014	.07

Notes: N=9,359 US counties; ***p < .001; ** p < .01; * p < .05; † p < 0.10
Exposure = county race-specific, age-specific population
Lagged measures are race-specific adult OD rates per 100,000

Appendix Tables:

Appendix Table 1. IDC codes

OVERDOSE
X40 (Accidental poisoning by and exposure to nonopioid analgesics, antipyretics and antirheumatics)
X41 (Accidental poisoning by and exposure to antiepileptic, sedative-hypnotic, antiparkinsonism and psychotropic drugs, not elsewhere classified)
X42 (Accidental poisoning by and exposure to narcotics and psychodysleptics [hallucinogens], not elsewhere classified)
X43 (Accidental poisoning by and exposure to other drugs acting on the autonomic nervous system)
X44 (Accidental poisoning by and exposure to other and unspecified drugs, medicaments and biological substances)
X45 (Accidental poisoning by and exposure to alcohol)
Y10 (Poisoning by and exposure to nonopioid analgesics, antipyretics and antirheumatics, undetermined intent)
Y11 (Poisoning by and exposure to antiepileptic, sedative-hypnotic, antiparkinsonism and psychotropic drugs, not elsewhere classified, undetermined intent)
Y12 (Poisoning by and exposure to narcotics and psychodysleptics [hallucinogens], not elsewhere classified, undetermined intent)
Y13 (Poisoning by and exposure to other drugs acting on the autonomic nervous system, undetermined intent)
Y14 (Poisoning by and exposure to other and unspecified drugs, medicaments and biological substances, undetermined intent)
Y15 (Poisoning by and exposure to alcohol, undetermined intent)
Y45 (Analgesics, antipyretics and anti-inflammatory drugs)
Y47 (Sedatives, hypnotics and antianxiety drugs)
Y49 (Psychotropic drugs, not elsewhere classified)