Spillover effects of opioid prescribing practices: Do increased prescriptions lead to increased fatal car crashes?

Michael Betz and Lauren E. Jones

Abstract:
Objectives. To estimate the relationship between commuting zone (CZ)-level opioid prescription rates and CZ-level car crash fatality outcomes.

Methods. We link data on CZ-level opioid prescription rates to data from the Fatality Analysis and Reporting System on all fatal crashes in the United States between 2007 and 2016. We estimate models that link the numbers of fatal crashes, deaths, and crashes where the driver was impaired in each CZ-year to the local opioid prescription rate. We include year- and CZ-level fixed effects in all our models.

Results. We find that fatal car crashes and car crash fatalities are about 1.03 times more likely when the opioid prescription rate in a commuting zone increases by one standard deviation (SD). We also find that the number of drivers involved in fatal crashes who test positive for narcotics increases by about 1.30 times. We find no significant relationship between opioid prescription rates and alcohol use among drivers involved in fatal crashes.

Conclusion: Local opioid prescribing practices led to increases in opioid use among drivers involved in fatal crashes. Prescriptions also led to increases in annual traffic fatalities.
Drug overdoses are now the leading killer of Americans under age 55.\textsuperscript{1} Researchers have shown that local prescribing behavior has resulted in increased overdose deaths, and has contributed to the ongoing crisis.\textsuperscript{2} However, widespread prescriptions for opioids may have indirect public health consequences on other outcomes such as car crash fatalities. Opioids cause users to experience sedation and drowsiness, and the FDA requires that prescriptions for opioids be accompanied by a warning against driving or operating heavy machinery.\textsuperscript{3-5} Recent research demonstrates that in some areas of the United States, the probability that prescription opioids are detected in drivers who die in fatal crashes has increased by 700 percent over the past several decades.\textsuperscript{6-7}

These studies show that among drivers who die in fatal car crashes, opioid use has become increasingly common. However, they do not test whether opioid prescribing practices have \textit{caused} increased traffic fatalities. Increased prevalence of opioids among fatally injured drivers may simply reflect the fact that more Americans now take prescription opioids: over 17 percent of Americans filled a prescription for an opioid in 2017.\textsuperscript{1} Thus, higher opioid use nationwide could lead mechanically to higher use rates among deceased drivers. This may be especially true if underlying demographic characteristics of prescription opioid users are also associated with involvement in fatal crashes.\textsuperscript{8-11}

Furthermore, if prescription opioids act as a substitute for other intoxicants like alcohol, higher rates of opioid use among drivers could reflect lower rates of drunk driving. If drivers are substituting away from more dangerous intoxicants, then increased prescription opioid use may perversely lead to fewer fatal crashes. If this is true, then apparent increases in the rate of drugged driving in fatal crashes could be driven in part by a reduction in the number of fatal crashes. In this case, past estimates of the prevalence of drugged driving may be substantially
inflated. Recent work has demonstrated that the number of drug-positive deceased drivers overtook the number of alcohol-positive drivers in Milwaukee.\textsuperscript{12}

Further complicating the issue is the fact that there is not a consensus on the extent to which opioid use impairs driving ability. Some experimental studies have shown that in higher doses, prescription opioid use can impair driving ability.\textsuperscript{13-14} Many others have shown that at therapeutic doses, prescription opioids do not affect driving ability.\textsuperscript{15-16} A recent study showed limited evidence of driving impairment due to methadone and buprenorphine, which are opioid partial agonists used to treat opioid use disorder.\textsuperscript{17} These results confuse the question of whether we should expect to find a positive relationship between local opioid prescription rates and car crash fatalities. We address this gap in the literature by estimating the relationship between the prevalence of local opioid prescriptions and local traffic fatality outcomes. Using data on all fatal car accidents in the US between 2007 and 2016, we estimate fixed effects models that relate changes in commuting zone (CZ)-level counts of fatal crashes, deaths and driver use of alcohol and opioids to changes in CZ-level per-capita prescriptions for opioids.

METHODS

Data

We use data from the Fatality Analysis and Reporting System (FARS), which is a census of all fatal crashes in the United States collected annually since 1975. Compiled by the National Highway Traffic Safety Administration, the data include information on all individuals and vehicles involved in fatal car crashes and are collected from police reports, as well as other driver and health administrative records. For our purposes, we use information collected on the
fatality outcomes, as well as the results of alcohol and drug tests reported in the data, for all individuals involved in fatal crashes between 2007 and 2016.

The FARS data include alcohol test results, and – since 1991 – toxicology reports on drug use among selected individuals involved in crashes. Alcohol can be detected though blood, breath or urine samples, and drugs can be detected through blood or urine tests. Between 2007 and 2016, approximately 54 percent of drivers involved in fatal crashes were tested for alcohol, and approximately 40 percent were tested for drugs; among drivers who died in crashes, 78 percent were tested for alcohol and 62 percent were tested for drugs. The results of the alcohol tests reported in FARS indicate the blood alcohol content (BAC) of the tested individual; the results of the drug test indicate the presence of drugs by type in the tested individual. Here, we classify individuals as intoxicated if they tested positive for a BAC greater than 0, if they tested positive for any drug, or for narcotics in particular (a category that includes prescription opioids).

Data from the FARS are a select sample, since they only include observations for those involved in fatal crashes. This means that estimating models of rates of driver drug use among fatal crashes may produce misleading results. This is because the explanatory variable – the prevalence of opioid prescriptions – could affect both the likelihood of being in a fatal crash and the likelihood that the driver was intoxicated. Thus, modeling the rate of driver drug use among fatal crashes could produce misleading results since changes in the rate could be driven by changes in the likelihood of a fatal crash, changes in the likelihood of driver drug use, or both. This issue is especially troublesome if opioid prescription prevalence reduces the number of fatal crashes in an area – by reducing drunk driving, for instance. In this case, the rate of drug use among drivers involved in fatal crashes could appear inflated due to a decreasing probability of being involved in a fatal crash.
To overcome this issue, we convert the individual-level data to count data by collapsing
the FARS data at the commuting zone (CZ)-by-year level. Commuting zones are geographic
units that capture local economies where people live and work. There are 709 CZs in the US.
Because the FARS is a census, the resulting dataset captures the total number of crashes and
fatalities, as well as the total number of measured cases of drug use among drivers involved in
fatal crashes in each CZ and each year. This dataset – which measures counts rather than rates of
drug use occurrences among drivers involved in fatal crashes – will allow us to avoid the
potential pitfalls inherent in analyses that use rates.

For each CZ-year, we count the number of fatal crashes, the number of deaths in fatal
crashes, the number of driver deaths, the number of child deaths (under age 12), the number of
drivers involved in fatal crashes who were tested for drugs or alcohol, respectively, and the
number of drivers testing positive for alcohol, drugs or narcotics, respectively. We combine the
collapsed FARS data with CZ-level data on per capita prescriptions for opioid analgesic
medication. These data – which we obtained from the CDC – were collected by IQVIA
Transactional Data Warehouse (formerly IMS Health and Quintiles) from a sample of
approximately 50,000 retail pharmacies, which distribute nearly 90 percent of all retail
prescriptions in the United States.¹ We create CZ-level prescription rates by using population-
weighted averages of the annual county-level prescription rates across counties in each CZ.
Approximately 11 percent of county-year prescription rates are missing from the prescription
data. In cases where missing counties are combined with other counties into a single CZ, we
replace the missing prescription rate with the average rate of other counties in the CZ. In cases
where a CZ is composed of a single county – as is the case in rural areas – we set the CZ
prescription rate as missing if the county rate was missing. This leaves us missing about 4
percent of CZ-year prescription rates. We merge the prescription data with the FARS count data at the CZ-year level.

**Statistical Analyses**

Using our CZ-by-year data, we estimate the following poisson model:

\[
\log(O_{cy}) = \beta R_{cx_{cy}} + \theta_{cz} + \lambda_{y} + \log(pop_{cy}) + \epsilon_{cy}
\]  

Model (1) explains the log of annual CZ-level count outcomes \((O_{cy})\) using the annual CZ-level prescription rate \((R_{cx_{cy}})\). The model also includes CZ \((\theta_{cz})\) and year \((\lambda_{y})\) fixed effects. The benefit of using this approach is that we are able to control for CZ-specific characteristics and yearly trends that could affect outcomes but are unrelated to opioid prescribing patterns. One particularly worrisome issue is the question of whether drug testing practices changed over our time frame, or differ across localities. Between 2007 and 2016, the average probability that a driver involved in a fatal crash was drug tested decreased from about 40 percent in 2007 to 33 percent in 2016. There is also variation across regions in the prevalence of driver drug testing. By including CZ and year fixed effects, we are able to control for these differences that could potentially bias our estimation of the relationship between prescribing practices and crash and fatality outcomes. We also explore the relationship between opioid prescribing practices and drug testing directly by estimating a model explaining the number of tested drivers in a CZ.

We estimate poisson models because our outcome variables are all expressed as counts, and are therefore zero-censored and, to some extent, zero-inflated. We standardize the prescription rates and report the incidence rate ratios (IRR). The coefficient estimates of \(\beta\) capture the relative change in CZ-level crash rates when the local opioid prescription rate
increases by one standard deviation (SD). A one SD increase in the prescription rate is equivalent to adding approximately 34 prescriptions per 100 people per CZ. Finally, we also include an offset term for the CZ-level population \((\log(\text{pop}_c))\) to account for the fact that CZs with more people will mechanically have more traffic fatality events. In all cases, we cluster standard errors at the CZ-level to account for the fact that the error terms are correlated across time within CZs.

RESULTS

Figure 1 shows the trends in average prescription rates and the average number of fatal crashes per CZ. The figure reveals that from 2007 until 2012, prescription rates increased from about 89 prescriptions per 100 people to 100 prescriptions per 100 people in 2012. Average prescriptions then decreased to about 79 per 100 people in 2016. The average number of fatal crashes per CZ exhibits the opposite trend, decreasing from an average of 52 crashes per CZ in 2007 to only 42 in 2011, and then increasing to an average of 48 crashes per CZ in 2016.

In Figure 2, we again show the average CZ-level prescription rate over time in the solid line, along with various drug and alcohol related fatal crash outcomes. In panel a, we plot the average number of drivers tested for drugs per CZ (dash line) and – on its own axis – the number of drivers who tested positive for opioids per CZ (dotted line). This figure shows that between 2007 and 2016, there was a 20 percent reduction in the average number of drivers who were tested for drugs (from 43 drivers per CZ to 34 drivers). Over this same period, however, there was a 50 percent increase in the average annual number of drivers per CZ who tested positive for opioids (from 1.32 drivers per CZ in 2007 to 2.01 in 2015). In 2016, the average number of drivers per CZ who tested positive for opioids decreased slightly to 1.86.
Finally, in panel b of Figure 2, we show the trends in alcohol-related fatal crash outcomes. We again show the average CZ-level prescription rate (solid line), along with the average number of drivers who were tested for alcohol per CZ (dashed line), and – on its own axis – the average number of drivers who tested positive for alcohol per CZ (dotted line). There is a 25 percent reduction over our study period in the number of drivers tested for alcohol. Unlike opioids, however, there is also a reduction in the average number of drivers per CZ who test positive for alcohol (from 15 in 2007, to only 10 in 2016).

While these trends are suggestive, they do not reveal causal relationships. While the average number of crashes per CZ and the average CZ-level prescription rates follow opposing trends – suggesting, possibly, that more prescriptions lead to fewer crashes – our study timeframe also includes the financial crisis and recovery. Past work has shown that fatalities from car crashes are countercyclical and drug use is procyclical,\textsuperscript{18-19} and thus the patterns revealed here may just as easily reflect economic factors. To investigate this more fully, we report results from our fixed effects regression models that control for secular time trends that affected all areas similarly (i.e. financial crisis), as well as time-invariant CZ-level characteristics that might lead to some areas having more or fewer crashes regardless of year.

Tables 1 and 2 show results of these analyses. In table 1, we report estimates of the relationship between the annual local prescription rate and fatality outcomes. Each coefficient estimate comes from estimating model (1) on one of our fatality outcomes. A one standard deviation increase in the local opioid prescription rate is associated with 1.03 (95% CI: 1.01, 1.06) times more fatal traffic accidents in a CZ in a year. On average, there are about 45 annual fatal crashes per CZ. Thus, we can conclude that, at the mean, a one standard deviation increase in the local prescription rate leads to about 1.4 more fatal crashes per year.
Our estimates also show that approximately one person is killed in each additional fatal traffic accident, and that it is generally the driver. We estimate that the number of yearly traffic fatalities is about 1.03 (95% CI: 1.01, 1.06) times higher when a CZ-level prescription rate increases by one SD; the number of annual driver fatalities is 1.05 (95% CI: 1.02, 1.08) times higher. Interpreting these coefficients at the sample means suggest that a one standard deviation increase in the local prescription rate is associated with about 1.5 more annual traffic fatalities per year, and about 1.5 more driver deaths in each CZ. We do not estimate a statistically significant relationship between the local opioid prescription rate and the number of children killed in traffic accidents.

In Table 2 we show results of the same models estimated on driver intoxication outcomes. Again, each reported coefficient comes from estimating model (1) on one of our driver intoxication outcomes. We find that drivers in fatal accidents are more likely to be drug tested when local opioid prescription rates increase. A one standard deviation increase in the local prescription rate is associated with 1.07 (95% CI: 1.01, 1.13) times more tested drivers. This translates into approximately 2.3 additional drivers tested for drugs each year in each CZ. We also find that when prescription rates increase, more drivers involved in fatal crashes test positive for drugs, and for opioids in particular. We find that the number of drivers involved in fatal crashes who test positive for drugs is 1.13 (95% CI: 1.05, 1.22) times higher when a CZ increases its opioid prescription rate by one SD; the number of drivers testing positive for narcotics is 1.30 (95% CI: 1.16, 1.47) times higher. While these estimates are large in relative terms, they are smaller in absolute terms. They suggest that when a CZ increases its opioid prescription rate by on SD, about 1 additional driver tests positive for drugs and about 0.5 additional drivers test positive for narcotics in particular. In the final two columns of table 2, we
report the results of the models explaining the alcohol variables. We do not estimate significant relationships between the local opioid prescription rate and any of our alcohol involvement measures.

DISCUSSION

Our results reveal significant and positive relationships between local opioid prescription rates and drug involvement in fatal crashes. We estimate that a one standard deviation increase in local opioid prescriptions per 100 people leads to 1.3 times more drivers involved in fatal crashes who test positive for narcotics. While we also find that more prevalent opioid prescriptions are linked to more drug testing, we find a larger proportional increase in the likelihood of testing positive for opioids than in the likelihood of being drug tested at all. This implies that the large increase in positive opioid tests is not simply driven by more prevalent testing of drivers.

The effect of local prescribing practices on drugged driving translates into additional traffic fatalities. We estimate that a one standard deviation increase in the average local opioid prescription rate is associated with 1 to 2 additional annual fatal traffic accidents per CZ. Using predicted values from our model, we can conclude that if each commuting zone had reduced their annual opioid prescription rate by one standard deviation, approximately 20 fewer individuals would have died in fatal crashes per year across the United States.

Because our models control for CZ and year fixed effects, the estimated effects reflect relationships between shifts in local prescribing behavior and car crash outcomes, net of secular trends in crashes or prescribing behavior that are common across the country, and net of time-invariant local factors that may affect both prescriptions and fatalities. If we assume that there are not time-varying factors within commuting zones that affect both the number of crashes and
prescription rates similarly, then we can interpret the current estimates as reflecting the causal effect of increased prescriptions in a community on traffic fatality and drug use outcomes.

We do not uncover any effect of local prescriptions on alcohol outcomes. There is no effect of additional local opioid prescriptions on the number of drivers tested for alcohol, or the number of drivers with alcohol in their system. At least among those who drive while intoxicated and are eventually involved in a fatal crash, additional opioids prescriptions are not associated with less alcohol use. This would suggest there is no substitution effect between opioids and alcohol among drivers in fatal crashes and the increase in fatal crashes involving a drugged driver are not offset by decreases in fatal crashes involving drunk drivers.

Limitations

Our results should be interpreted in light of several caveats. First, the estimated effects only reflect the causal effect of additional prescriptions on traffic fatality outcomes under the assumption that, after controlling for CZ and year fixed effects, no additional factors affect both prescription rates and traffic conditions simultaneously. One potential confounding factor that could affect both variables is the economic crisis, which came to a head during the study timeframe. If bad economic conditions caused individuals to demand more prescription drugs and to drive more recklessly, then the observed relationship between prescribing practices and fatal crashes could reflect local economic conditions rather than the causal effect of prescriptions on crashes. However, past research has shown that bad economic conditions tend to reduce traffic fatalities (Ruhm 2000). Therefore, if economic conditions are a confounding variable in the present study, they likely bias us against the results we uncover. We further investigate this potential source of bias by estimating our models after including the annual CZ-level unemployment rate as a control. The results of this analysis (reported in the online appendix)
show that our main estimates are virtually unchanged after controlling for the unemployment rate. Thus, it is unlikely that our results are driven by unobserved economic conditions.

Our study is also limited by the fact that we are only able to estimate intent-to-treat effects. The estimated effects we report capture the effects of local prescription rates on community-level outcomes. They do not capture the effect of additional prescriptions on the probability that an individual is involved in a fatal crash. Because we estimate effects on localities, we are unable to account for the fact that some prescriptions obtained in one CZ may actually be used elsewhere, and may therefore affect traffic fatalities in a different CZ. Furthermore, we are unable to capture the effects of additional opioid usage that is not reflected in prescription rates. This includes illegally trafficked prescription drugs, fentanyl and fentanyl analogs, or non-prescription opioids, such as heroin. In this sense, our estimate is lower bound of the total effect of the opioid crisis on fatal car crashes.

Policy Implications

The results of the current study add to the growing literature on the effects of the opioid crisis on various public health outcomes. We show that prescribing practices have affected drug use among drivers who are involved in fatal crashes, confirming past results indicating increases in the prevalence of drugged driving. We extend this literature by providing evidence that doctor’s prescribing habits have contributed to this increase. Additional local prescriptions are also associated with additional fatal crashes, as well as additional fatalities.

Policy-makers can use the current results to help quantify the effects of the crisis. Using the Department of Transportation’s value of a statistical life estimate of 9.6 million dollars per person, we estimate that an additional 34 prescriptions per 100 people in each commuting zone
caused motor vehicle deaths valued at about 200 million USD per year. These avoidable deaths represent additional casualties of the opioid crisis and should be considered as part of the costs of the crisis. Interventions to improve prescriber behaviors, prescription monitoring systems, and patient education can further reduce prescription opioid abuse and related negative spillovers, such as fatal car crashes.
References


Figure 1. Average CZ-level prescription rates, and average CZ-level number of fatal crashes over time
Figure 2. Average CZ-level prescription rates, and average CZ-level number of tested drivers and positive tests, drugs and alcohol
<table>
<thead>
<tr>
<th>Model outcome:</th>
<th>Number of Fatal Crashes</th>
<th>Number of Car Crash Deaths</th>
<th>Number of Car Crash Driver Deaths</th>
<th>Number of Car Crash Child Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standardized Rx Rate (IRR)</td>
<td>1.03</td>
<td>1.03</td>
<td>1.05</td>
<td>1.05</td>
</tr>
<tr>
<td>(95% CI)</td>
<td>(1.01, 1.06)</td>
<td>(1.01, 1.06)</td>
<td>(1.02, 1.08)</td>
<td>(0.94, 1.17)</td>
</tr>
<tr>
<td>Sample mean</td>
<td>44.7</td>
<td>48.9</td>
<td>31.2</td>
<td>1.4</td>
</tr>
<tr>
<td>X2</td>
<td>1150.5***</td>
<td>1125.21***</td>
<td>853.90***</td>
<td>86.19***</td>
</tr>
<tr>
<td>N</td>
<td>6872</td>
<td>6872</td>
<td>6872</td>
<td>6323</td>
</tr>
</tbody>
</table>

Notes: Data from the FARS and IMS Quintiles, 2007-2016, all US commuting zones. Each column reports results from a separate Poisson regression model. Outcomes reflect the annual counts of each event among all fatal crashes at the CZ-level. Controls include CZ and year fixed effects, and population exposure. Prescription rates are standardized such that coefficient estimates should be interpreted as a change in the incidence rate ratio of the event associated with a one-standard deviation increased in the CZ-level prescription rate. Standard errors clustered at the CZ-level.

Table 1. Estimated relationships between annual CZ-level opioid prescription rates and crash fatality outcomes
Table 2. Estimated relationships between annual CZ-level opioid prescription rates and driver intoxication outcomes

<table>
<thead>
<tr>
<th>Model outcome:</th>
<th>Number of Drivers Tested for Drugs</th>
<th>Number of Drivers Testing Pos. for Drugs</th>
<th>Number of Drivers Testing Pos. for Opioids</th>
<th>Number of Drivers Tested for Alcohol</th>
<th>Number of Drivers Testing Pos. for Alcohol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standardized Rx Rate (IRR)</td>
<td>1.07 (1.01, 1.13)</td>
<td>1.13 (1.05, 1.22)</td>
<td>1.30 (1.16, 1.47)</td>
<td>1.01 (0.97, 1.05)</td>
<td>1.03 (0.98, 1.09)</td>
</tr>
<tr>
<td>Sample mean</td>
<td>37.8</td>
<td>8.2</td>
<td>1.8</td>
<td>48.1</td>
<td>11.9</td>
</tr>
<tr>
<td>X2</td>
<td>163.30***</td>
<td>92.18***</td>
<td>74.86***</td>
<td>462.87***</td>
<td>618.83***</td>
</tr>
<tr>
<td>N</td>
<td>6851</td>
<td>6733</td>
<td>6018</td>
<td>6869</td>
<td>6863</td>
</tr>
</tbody>
</table>

Notes: Data from the FARS and IMS Quintiles, 2007-2016, all US commuting zones. Each column reports results from a separate Poisson regression model. Outcomes reflect the annual counts of each event among all fatal crashes at the CZ-level. Controls include CZ and year fixed effects, and population exposure. Prescription rates are standardized such that coefficient estimates should be interpreted as a change in the incidence rate ratio of the event associated with a one-standard deviation increased in the CZ-level prescription rate. Standard errors clustered at the CZ-level.