Original article

Threats to security and ischaemic heart disease deaths: the case of homicides in Mexico

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Abstract

Background: Ischaemic heart disease (IHD) ranks as the leading cause of death world-wide. Whereas much attention focuses on behavioural and lifestyle factors, less research examines the role of acute, ambient stressors. An unprecedented rise in homicides in Mexico over the past decade and the attendant media coverage and publicity have raised international concern regarding its potential health sequelae. We hypothesize that the rise in homicides in Mexico acts as an ecological threat to security and elevates the risk of both transient ischaemic events and myocardial infarctions, thereby increasing IHD deaths.

Methods: We applied time-series methods to monthly counts of IHD deaths and homicides in Mexico for 156 months spanning January 2000 to December 2012. Methods controlled for strong temporal patterns in IHD deaths, the unemployment rate and changes in the population size at risk.

Results: After controlling for trend and seasonality in IHD deaths, a 1-unit increase in the logged count of homicides coincides with a 7% increase in the odds of IHD death in that same month (95% confidence interval: 0.04 — 0.10). Inference remains robust to additional sensitivity checks, including a state-level fixed effects analysis.

Conclusions: Our findings indicate that the elevated level of homicides in Mexico serves as a population-level stressor that acutely increases the risk of IHD death. This research adds to the growing literature documenting the role of ambient threats, or perceived threats, to security on cardiovascular health.

Key words: Mexican drug war, drug violence, homicide, ischaemic heart disease

Key Messages

- We hypothesize that the unprecedented rise in homicides in Mexico over the past decade acts as a threat to security that affects population health.
- Our findings indicate that acute monthly increases in homicides serve as an antecedent of deaths from ischaemic heart disease.
- Threats to security from rising homicides, and the attendant media coverage, may induce adverse health and human costs not intuitively associated with violence.

Introduction

Ischaemic heart disease (IHD) ranks as the single leading cause of death worldwide and accounts for 13.2% of all deaths.[1] Environmental factors, as well as smoking, diabetes and an ageing population, contribute to the incidence of IHD.[2–6] Less research, however, focuses on acute, ambient stressors as an antecedent of IHD deaths. Below we describe literature which suggests the hypothesis that IHD deaths rise with homicides in Mexico.

Before the early 2000s, the Mexican government passively dealt with drug trafficking organizations (DTOs) operating within its borders.[7–9] However, on 11 December 2006, former President Felipe Calderón sent federal troops into Michoacán to reassert control over areas controlled by DTOs.[7,9] Calderón later deployed troops to 12 other states in Mexico.[10] This deployment comprised a part of a long-term anti-drug campaign, known as the Mexican Drug War. The targeted removal of cartel leadership, however, reportedly destabilized relations among existing DTOs.[11,12] As federal troops entered key drug trafficking corridors, homicides increased in magnitude.[12] Mexico's homicide rate increased 3-fold from 2007 to 2011.[13] In 2011, Mexico's annual homicide rate, moreover, was five times that of the USA in that same year.[14]

Many homicide victims in Mexico were displayed in prominent public areas with or without all of their body parts, showing clear signs of torture.[12,15] The dramatic increase in homicides and the heightened level of brutality translated into intense media coverage, complete with detailed descriptions and depictions of the murders. Therefore, the media coverage and publicity arguably contributed to a general sense of anxiety, unease and insecurity even in areas unaffected by high homicides.

The threat—real or perceived—to the security of Mexican citizens may plausibly elicit a physiological response that elevates IHD morbidity. An acute rise in overall homicides in Mexico, and the attendant media coverage, may increase transient feelings of anxiety and/or depression, both of which reportedly trigger cardiovascular events.[16,17] Research finds that acute individual (e.g. unexpected job loss) and population-level (e.g. earthquakes,

war and terrorist attacks) stressors serve as 'triggers' for a transient ischaemic event.[18–20] These triggers represent the penultimate step in the physiological process leading to severe cardiovascular events among persons already vulnerable from existing heart disease.[21] Among vulnerable persons, stress-induced triggers may elicit vasoconstriction and prothrombotic processes. These events, in turn, may cause plaque disruption and blood clots that lead to myocardial infarction or sudden cardiac death.

Sources of anxiety induced by acute stressors, described above, often arise from factors outside the individual's control. Examples of ecological stressors, which occur with greater frequency than do natural and man-made disasters, include rising violent crime. Sundquist and colleagues[20] find that, in Sweden, persons living in neighbourhoods with elevated violence or unemployment show increased IHD relative to persons living in less violent neighborhoods. This work, albeit provocative, is limited because it does not examine acute IHD events or death, is cross-sectional in nature and cannot rule out the strong rival explanation of selection into neighbourhoods—that is, unhealthy persons sort into less secure neighbourhoods. These limitations preclude making inferences about whether unexpectedly high regional violence increases the risk of IHD death.

Negative shocks to populations that live geographically distant from an ambient shock (e.g. persons outside New York after 9/11) may elicit, within a few days, increases in severe cardiovascular events.[22] To date, however, no study directly assesses the relation between homicides and IHD mortality. Mexico provides an unusual opportunity to test whether an increase in homicides coincides with unexpected increases in IHD mortality. We test this hypothesis for the entire country of Mexico.

Methods

We outline our five research steps. First, we hypothesize that the risk of IHD death in Mexico rises above its expected value in months in which the log-count of homicides is high. Second, we use the longest series available to us to test the hypothesis. Third, we apply well-established

time-series analytical routines[23,24] consistent with the logic of correlational tests, to identify and remove patterns in the dependent variable. Fourth, we add our independent variable of interest (log-count of homicides) and a potential confounder (the unemployment rate) to the time-series regression equation to determine whether the coefficients differ from zero. Fifth, if results support the hypothesis, we conduct additional sensitivity checks to identify whether country results appear robust to choice of scale used, to IHD deaths but not diabetes deaths and to a state-level negative binomial regression that focuses on states with a high level of homicides.

Data

Whereas non-fatal measures of violence (e.g. kidnappings) may more sensitively gauge threats to security than do homicides, these indicators are not reliably or consistently measured over the test period. We therefore used homicides as the independent variable. We obtained IHD mortality (ICD-10 codes I20-I25), homicide (ICD-10 codes X85-Y09) and unemployment data for 156 months from January 2000 to December 2012. We obtained the mortality data for IHD and homicides from Mexico's Secretariat of Health's System for National Health Information.[25] The data are collected with internally consistent procedures designed to ensure comparability over time. Other literature reports high accuracy of cause-of-death codes and estimates the death file to have over 90% completeness.[26] We used date of occurrence, rather than date of registration, to assign month of death. The population-at-risk for IHD was limited to persons aged 50 or over, given that they account for 92.8% of all IHD deaths in Mexico. To derive

the population at risk of IHD death, we used 2005 population projections.[25]

The monthly plot of IHD death counts over the test period (Figure 1) shows that the maximum value (7310) occurs in December 2010 and the minimum value (2961) occurs in June 2000. Figure 1 also plots the monthly count of homicides. The maximum value (2599) occurs in May 2011 and the minimum value (507) occurs in February 2007. Unemployment may co-vary with homicides and independently predict IHD death. To control for this factor, we acquired unemployment data from the Economic Information Bank.[26] The mean monthly unemployment rate was 4.98% [standard deviation (S.D.) = 1.10]. The maximum value (7.9%) occurs in September 2009, which corresponds with the peak of the unemployment rate in the USA during the Great Recession.

Our hypothesis turns on whether the risk of IHD death rises above expected values in months with an elevated number of homicides. The risk of IHD death, however, exhibits seasonality and other patterns. Such patterning appears commonplace in health time series.[28] These patterns, referred to as autocorrelation, complicate observational tests because the expected value of the patterned series is not its mean. One analytical solution involves identifying this autocorrelation and expressing it as an effect of earlier values of the dependent variable itself.[23,29,30] This data-driven approach, as outlined by Box and Jenkins,[31] identifies autocorrelation from the dependent variable series and removes autocorrelation such that the expected value of the residuals is zero and the monthly observations are statistically independent of one another. In our test, removing autocorrelation from the risk of IHD death before testing the relation with homicides also minimises the risk of a
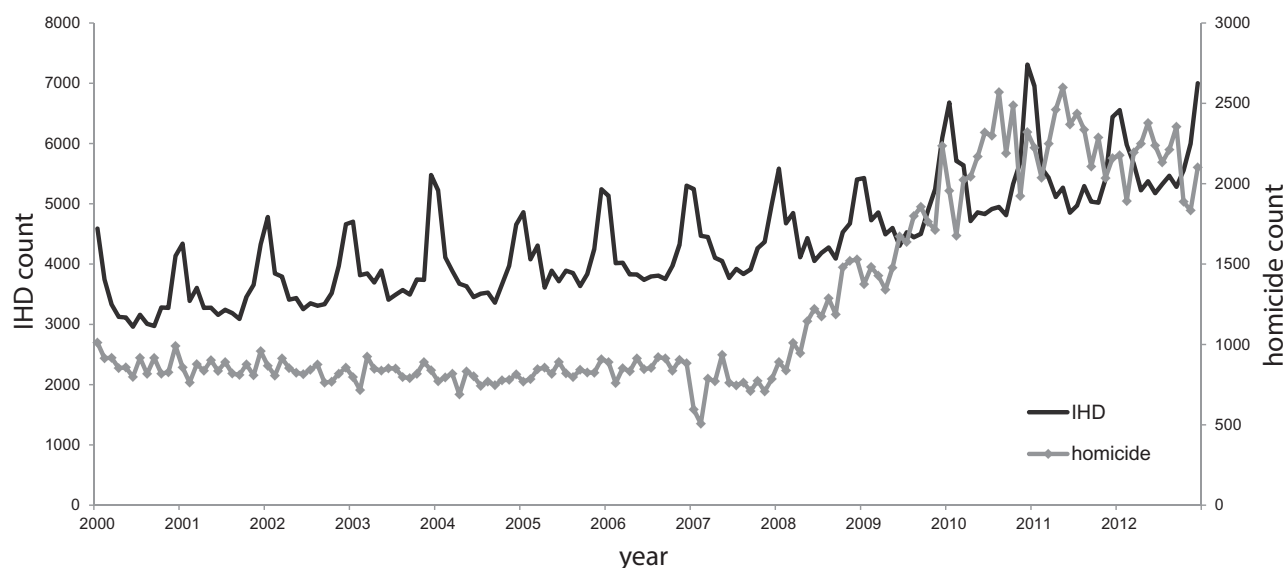
Figure 1. Counts of IHD and homicide deaths spanning 156 months from January 2000 to December 2012.

spurious association that could arise from a shared pattern between homicides and high or low values of IHD deaths.

To implement this approach, we used the strategies devised by Dickey and Fuller[32] as well as Box and Jenkins,[31] to identify and model patterns in the monthly counts of the risk of IHD death. The augmented Dickey-Fuller test detects non-stationarity exhibited by IHD deaths. Box and Jenkins routines model these patterns and the tendency of a series to remain elevated or depressed after high or low values. Box-Jenkins methods model trends by differencing a series (i.e. subtracting the values of each month from those of the next month). If we detect seasonal cycles, we similarly use the differencing operator but at 12 (e.g. subtracting values for February 2005 from that of February 2006). The Box and Jenkins approach uses 'autoregressive' and 'moving average' parameters to model other forms of autocorrelation. Autoregressive parameters best describe patterns that persist for relatively long periods, whereas moving average parameters parsimoniously describe less persistent patterns.

The analyses proceeded through the following steps. First, we converted the dependent variable of IHD death to the log-odds of IHD death for adults aged ≥ 50 years in month t. We specified the log-odds of death to control for changes over time in the population over 50 years at risk of IHD death in Mexico, and to allow familiar epidemiological interpretation of the antilog of independent variable coefficients as an 'effect on odds' metric. Second, we identified and removed autocorrelation in the monthly log-odds of IHD death. Third, consistent with the epidemiological literature, we added the homicide and unemployment variables to the equation and estimated their coefficients.[23,28] We used the natural log of homicides to render the post-2007 series more comparable in scale to the level of homicides before 2007. Fourth, we inspected the residual autocorrelation and partial autocorrelation functions from the final model to ensure they exhibited no autocorrelation. Fifth, we assessed sensitivity of results to the choice of scale for the dependent variable. Sixth, as a falsification test, we repeated steps 1–4 except that we used diabetes mortality as the dependent variable. We assumed no relation between homicides and diabetes and therefore expect a null result for homicides in the diabetes falsification test. Seventh, we assessed whether a state-level analysis using regions with high homicides supported inference from the country-level test.

Results

IHD deaths exhibit strong winter peaks in January and February and appear to have a slightly higher mean in the second half, relative to the first half, of the series.

Inspection of the autocorrelation and partial autocorrelation function (ACF and PACF; see Appendix Table 1, available as Supplementary data at *IJE* online) and results from the Dickey-Fuller test identified strong seasonality which required us to difference the IHD series at 12 months (i.e. values at month t subtracted from values at month t+12) to render the time series mean-stationary. We, per convention, similarly applied this difference operator to all independent variables, including log-homicides and the unemployment rate.[30]

Further inspection of the ACF and PACF of the IHD series indicated 'echoes' at lag 1 month such that high or low values of IHD deaths were 'remembered' with similarly high or low values in the subsequent month, albeit in diminishing amounts (see ACF and PACF at lag 1 and 12 months, Appendix Table 1). In standard ARIMA(p,d,q) notation, we identified an ARIMA(1,12,12) model. We arrived at this model by using the ACF/PACF diagnostics as well as the equation with the smallest AIC value.[30] Figure 2 plots the monthly log-odds of IHD death after removal of autocorrelation. We use this series as the dependent variable for our tests, as its mean and expected value at all months is zero (see Appendix Table 2 which shows autocorrelation removed, available as Supplementary data at *IJE* online).

Consistent with our hypothesis, the log-odds of IHD death varies positively with log-homicides in that same month [Table 1, homicide coefficient: 0.067; 95% confidence interval (CI) = 0.04 — 0.10]. Inference remains essentially the same if we remove the unemployment rate from the model. To assist with interpretation, a 1% increase in the monthly differences of homicides predicts a 0.07% increase in the monthly differences in the odds of a heart disease death.

We examined whether inference for our test changed when we specified the count of IHD deaths as the dependent variable and the count of homicides as the independent variable. Results, other than the metric of the coefficient, remain essentially the same (Appendix Table 3, available as Supplementary data at *IJE* online). Given concerns about accuracy of population counts in Mexico, we also examined the count of IHD deaths as a function of the count of homicide deaths while omitting the population at risk as a control variable; inference remained unchanged (Appendix Table 4, available as Supplementary data at *IJE* online). Next, we removed the unemployment rate as a control variable from the main specification; the relation between log-homicides and log-IHD deaths became slightly stronger (Appendix Table 5, available as Supplementary data at *IJE* online). As a falsification test, we examined whether log-homicides coincide with increases in a chronic disease that research does not document as responding to acute stressors: type II diabetes deaths. We reasoned that if log-homicides moved
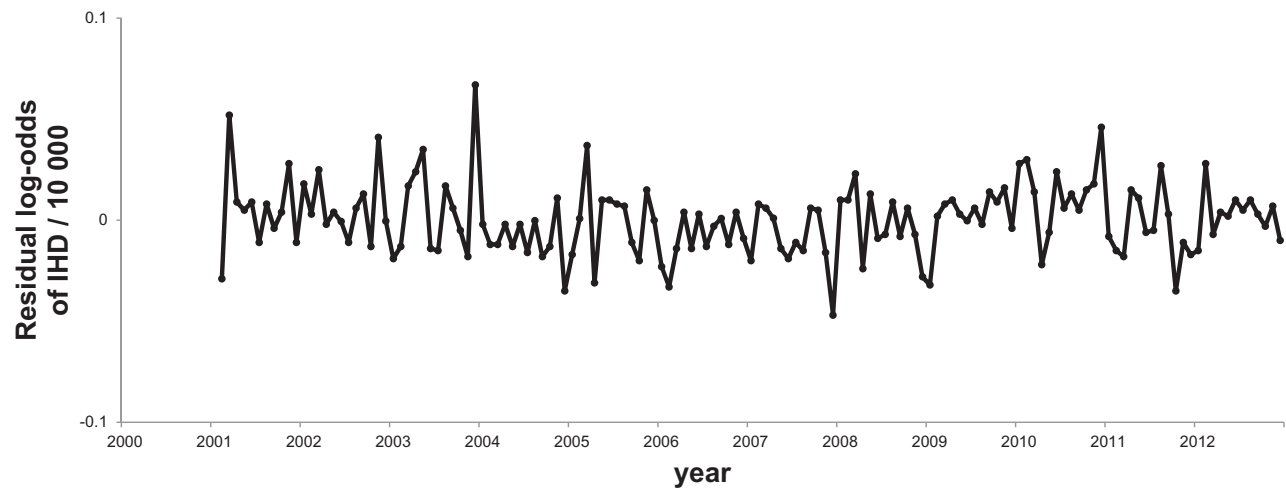
Figure 2. Residual log-odds of IHD death in Mexico over 156 months after identification and removal of autocorrelation. The mean of residuals is 0 and the series exhibits no temporal patterns. (First 14 months lost due to ARIMA modelling).

Table 1. Time series results predicting the monthly log-odds of IHD death[a] in Mexico from January 2000 to December 2012 [95% confidence intervals (CI) in parentheses]

Parameter	Coefficient (95% CI)
Logged homicide count at 0 months	0.067 (0.04 — 0.10)
Unemployment rate at 0 months	0.003 ($-$0.002 — 0.007)
Autoregressive parameter at month 1	0.401 (0.025 — 0.55)
Moving average parameter at month 12	0.784 (0.67 — 0.89)

[a]Differenced at month 12 due to strong seasonality in IHD deaths.

positively with diabetes deaths, then the relation with IHD deaths may have spuriously arisen due to an unmeasured, shared factor that corresponds with elevated mortality across several causes. We used diabetes deaths as the dependent variable and performed all time-series steps as described in Methods. Results indicate no relation between log homicides and the log-odds of diabetes deaths in the same month (coefficient: -0.039, 95% CI = -0.10 — 0.02; Appendix Table 6, available as Supplementary data at *IJE* online).

Whereas theory led us to test the homicide/IHD relation for all of Mexico, area-level 'hot-spots' in homicides may show a stronger relation than in areas with fewer homicides. Although we discovered a large amount of missing data on IHD mortality at the sub-country level—and unemployment data were not available at the state level—we conducted a negative binomial regression on the 11 states in Mexico with median monthly homicides above 1 per 100 000. We applied a state 'fixed effects' analysis that also controlled for national patterns in IHD deaths. Results in high homicide states show a positive relation between log-homicides and IHD deaths in the same month; the 95% CIs, moreover, do not contain the null value (Appendix Table 7, available as Supplementary data at *IJE* online).

Discussion

We used Mexico's rise in homicides following 2006 as a unique circumstance to test whether ambient threats to security increase the risk of IHD death. Our analysis of IHD mortality in Mexico indicates that the risk of IHD death rises above expected values in months when the mortality from homicide also increases. We find a 7% increase in risk of IHD deaths with a 1-unit increase in log-homicides. Results remain robust to adjustment for strong patterning in IHD deaths, control for the unemployment rate and other sensitivity checks. Our time-series analysis supports the hypothesis that homicides serve as an ambient, acute stressor that may trigger an increase in deaths from IHD.

Strengths of our analysis include the over 90% reported completeness of all homicide and IHD deaths in Mexico.[26] Availability of monthly data, moreover, permits careful testing of the acute temporal relation between the population stressor and IHD deaths that is not possible with annual data. Findings also cannot arise from shared autocorrelation in IHD and homicides over time since we removed all autocorrelation from IHD deaths before evaluating its relation with homicide. In addition, we minimize the potential rival threat of health selection into Mexico (i.e. persons with cardiac problems move into the country when homicides increase but leave when homicides decline) by examining monthly IHD data. Our falsification test using diabetes deaths further supports that our discovered association appears specific to a cause of death with acute contributing causes. Additional sensitivity checks (not shown) further indicate that potential errors in the census-reported estimates of population size do not likely explain our results.

Limitations involve that we had no information on the nature of the IHD death. Our hypothesis pertains to IHD

deaths among already vulnerable persons that may plausibly be triggered by heightened anxiety pertaining to safety and security. We expect that future investigation might allow refined testing of IHD deaths precipitated by myocardial infarction or transient ischaemic events. In addition, whereas the literature documents over 10-fold variation in homicides across municipalities in Mexico,[12] we did not have precise estimates of small-area homicides and IHD deaths. We discovered a large amount of data with missing geographical identifiers. This circumstance precluded examination of area-level 'hot-spots' in homicides. State-level analyses, although supporting our inference of a positive relation, also suffered from poor model fit (available upon request). We, therefore, caution against using our country-level results to infer associations about particular municipalities or regions. We also acknowledge that our work cannot inform potential regional heterogeneity in which some drug cartels may spread more fear and insecurity than would other cartels. In addition, our time-series study cannot rule out confounding by an unmeasured, unpatterned ecological variable that coincides with (but is not caused by) homicides and causes IHD but not diabetes deaths.

Whereas the upward IHD trend cannot explain our discovered association since we removed this and other autocorrelation from the dependent variable, it warrants further enquiry. We note that the concurrent rise in diabetes deaths over the test period may indicate population-level shifts in behavioural factors (e.g. obesity) that elevate the burden of chronic disease.[33]

One explanation for our discovered relation between homicides and IHD deaths includes heightened anxiety or insecurity during months of elevated homicides among persons not directly connected to violent events. Before March 2013, multiple media outlets in Mexico reported weekly homicides from organized crime.[34] The government maintained statistics for crimes alleged to have been related to drug trafficking organizations.[12] Therefore, extensive media coverage of this issue may have contributed to a sense of insecurity and acute anxiety. Since 2012, many major newspapers ceased publication of organized crime-related homicides and only release statistics periodically. Therefore, for ordinary citizens the narco-blogs, amateur journalism, and social media have become the dominant medium for disseminating recent information on drug-related homicides.[35] Outside Mexico, several reports find psychological and somatic sequelae after exposure to media coverage of terrorist attacks and other traumatic events.[36,37] These sequelae occur not only among persons proximate to the events, but also among geographically distant persons witnessing the events via media outlets.[38,39] Literature on the terrorist attacks of 11 September 2001 further reports severe cardiovascular events in regions outside New York in the month after the attacks.[22] This work supports the plausibility of behavioural and physiological reactions that trigger transient ischaemic events and myocardial infarctions among a vulnerable subset of the broader population.

Media discussion of the homicides in Mexico and the War on Drugs often centres on dollars spent combating drug trafficking organizations, kilograms of illicit substances seized and the number of deaths. However, recent research provides a more nuanced view of the political, economic and social factors related to homicides in Mexico.[40,41] Our findings indicate additional health sequelae of this homicide epidemic on the leading cause of death worldwide.[42–44] We encourage subsequent research on the extent to which acute changes in violence affect older, vulnerable populations not typically identified as exposed to violence.

Supplementary Data

Supplementary data are available at **IJE** online.

Conflict of interest: None declared.

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