Threats to Security and the Risk of Sudden Cardiac Death: The Case of Homicides in Mexico

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ABSTRACT

Ischemic heart disease (IHD) ranks as the leading cause of death worldwide. Whereas much attention focuses on eating, smoking and sedentary lifestyle as risk factors, less research examines the role of acute, ambient stressors. We hypothesize that ecological threats to security, such as the sudden rise in homicides in Mexico in the last decade, may plausibly elevate the risk of both transient ischemic events and myocardial infarctions, and correspond with increased heart disease deaths. We test this hypothesis by analyzing the whether monthly increases in ischemic heart disease deaths from 2000 to 2012 vary positively with monthly increases in homicides in Mexico. Time-series analyses find that a 1 standard deviation increase in the logged monthly count of homicides increases the odds of IHD death by 8 percent ($p<.001$). Results remain robust to control for strong temporal patterning in IHD deaths, the unemployment rate, and other rival explanations.

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Ischemic heart disease (IHD) ranks as the second leading cause of death in Mexico (Instituto Nacional de Estadística Geografía e Informática (INEGI), 2014). Extensive literature documents that individual lifestyle factors contribute to the incidence of IHD (Stamler, Vaccaro, Neaton, & Wentworth, 1993; Wilson et al., 1998; Kivimaki, 2006); Seretakis, 2006; Barrett-Connor et al., 1984). The contributing roles of smoking, diabetes, and an aging population to IHD are well documented. Less research, however, focuses on acute, ambient stressors as a potential antecedent of IHD deaths. Below we describe literature which suggests the hypothesis that IHD deaths may rise with the population homicide rate in Mexico.

In December 2006, then President Felipe Calderón declared the War on Drugs which resulted in the largest proportionate increase in homicides in the Western Hemisphere (Molzahn, Rodriguez Ferreira, & Shirk, 2013). The War on Drugs was formalized under the Merida Initiative. The $2 billion U.S.-Mexico partnership provided counternarcotics and military training, detection equipment, and weapons to the Mexican military and navy with which to confront the drug trafficking organizations (DTOs) (U.S. State Department, 2013). Their key strategy was to systematically remove the leadership and top executives (Beittel, 2013). The belief was that this would dismantle the organization. Instead, this created a power vacuum and fueled an all-out turf war within and among the remaining DTOs (Beittel, 2009; Beittel, 2013). Since December 2006, it is estimated that over 50,000 homicides have been attributed to drug violence (Molzahn, Rodriguez Ferreira, & Shirk, 2013).

As the homicides escalated, so did the level of brutality in the form of gruesome murders, kidnappings, beheadings, and public executions (Beittel, 2013). These tactics were not just intended to eliminate rival gang members but to intimidate the public. The media coverage and publicity arguably contributed to a general sense of unease and insecurity even in areas unaffected by high homicides (INEGI, 2012).

This brutal threat, or perceived threat, to the security of Mexican citizens may plausibly elicit a physiological response that elevates IHD morbidity. Both retrospective and prospective studies show that stress and stressful life events (e.g., unexpected job loss) increase blood pressure, the onset of IHD and a transient ischemic event (Crombie, Kenicer, Smith, & Tunstall-Pedoe, 1989; Rabkin & Struening, 1976; Sundquist et al., 2006). Sources of ecological stress often arise from factors outside of the individual's control. The literature further suggests that sudden and acute stressful life events (e.g., earthquakes, industrial accidents) may exacerbate the condition of those already susceptible to ischemic heart disease and make it more likely to suffer an acute cardiovascular event. Examples of ecological stressors, which occur with greater frequency than do natural disasters, include rising unemployment and violent crime. For instance, Sundquist and colleagues (2006) find that, in Sweden, persons living in neighborhoods with elevated violence or unemployment show increased ischemic heart disease. 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 neighborhoods—that is, unhealthy persons sort into less secure neighborhoods. These limitations preclude making inference about whether, and to what extent, unexpectedly high regional violence increases the risk of IHD death.

Previous research indicates that sudden ambient changes which produce negative shocks to the population (such as an economic recession) may elicit acute increases in IHD deaths in as little as a few months (Crombie, Kenicer, Smith & Tunstall-Pedoe, 1989). To date, however, no studies have directly assessed the impact of homicides on IHD mortality. The aim of this study is to determine if an increase in homicide rates coincide with unexpected increases in IHD mortality between the years 2000 and 2012. We test this hypothesis using monthly homicide and ischemic heart disease data on Mexico.

Our population-level analytic approach controls for the strong temporal patterning in IHD deaths and adjusts for the potentially confounding role of changing unemployment rates. Importantly, use of monthly values of the dependent variable minimizes the rival threat of selection into Mexico, given that it is unlikely that ill Mexicans at risk of IHD death move out of the country in months of low homicides but return when monthly homicide rates increase.

Our research contributes to the literature in two ways. First, the plausibly exogenous nature of homicide rates may lend credibility to identifying population-level antecedents of IHD death. Second, results may shed light unintended, acute, and demographically-important sequelae of the alarmingly high homicide rate in Mexico over the last decade.

METHODS

Data

We obtained IHD mortality data (ICD-10 codes I20-I25) for 156 months from January 2000 to December 2012. This time span represents the longest time series of data available to us at the time of our tests. We obtained the mortality data for IHD and homicides from Mexico's National Institute of Statistics and Geography (Instituto Nacional de Estadística Geografía e Informática [INEGI]) and Secretariat of Health's System for National Health Information (Sistema Nacional de Información en Salud [SINAIS]) (Dirección General de Información en Salud [DGIS], 2013; INEGI, 2014). 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 of all deaths in the country (Farr, 1985).

Our population-at-risk 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 risk of IHD death, we used the 2005 population projections derived from the between-Census 2005 Count of Population and Housing (SINAIS, 2014). Figure 1 plots the odds of IHD death per 10,000 adult population over the test period (odds = IHD deaths / (Population – IHD deaths)). Note that we use the "odds metric" for reasons described later in the Methods. The mean odds of IHD death is 2.727 per 10,000 persons (S.D.= 0.351). The maximum value (3.967) occurs in December 2010, and the minimum value (2.292) occurs in September 2004.

We acquired the monthly count of homicides (ICD-10 codes X85-Y09) from INEGI and SINAIS databases (DGIS, 2013; INEGI, 2014). These data are also believed to be over 90% complete (Farr, 1985). Figure 2 plots the logged unadjusted homicide rate per total population The mean monthly value for logged homicides is 7.018 per 10,000 population (S.D.= 0.430). The maximum value (7.863) occurs in May 2011 and the minimum value (6.229) occurs in February 2007. We restricted the population-at-risk to those aged 50 or over. The population-at-risk data for homicides were obtained from 2005 population projections derived from the between-Census 2005 Count of Population and Housing (SINAIS, 2014).

Unemployment may covary with homicides and independently predict IHD death via the ambient stressor hypothesis. To control for this factor, we acquired the monthly unemployment data from the Economic Information Bank on INEGI (INEGI, 2014). The unemployment data were obtained from a sample of urban unemployment in 32 cities. The data on The mean monthly unemployment rate was 4.977 percent (S.D.= 1.097). The minimum value (2.76) occurs in December 2000 and the maximum value (7.880) occurs in September 2009.

Analysis

Our test turns on whether the observed value of IHD death rises from expected values in months with an elevated homicide rate. Most statistical tests expect that the mean of all months best predicts the observed value of any month and further assume that an observed value outside an appropriate confidence interval of that mean supports the argument of a difference. IHD deaths in Mexico, however, exhibits strong seasonality and the tendency to remain elevated or depressed after high or low values.

These patterns, referred to as autocorrelation, complicate tests like ours because the statistically expected value of an autocorrelated series is not its mean.

Researchers dating to Fisher (1921) have addressed this problem by identifying autocorrelation and expressing it as an effect of earlier values of the dependent variable itself. This empirical approach, as outlined by Box and Jenkins (Box et al., 1994), removes autocorrelation from the dependent variable such that the expected value of the residuals is zero. Then, the analyst can add the independent variable to the equation to determine if the coefficient differs from zero in the hypothesized direction.

The analytic strategy described above requires that we estimate the following test equation:

$$\nabla^{d} Y_{t} = \omega_{0} \nabla^{d} H_{t} + \omega_{1} \nabla^{d} U_{t} + \frac{\left(1 - \theta B^{q}\right)}{\left(1 - \varphi B^{p}\right)} a_{t} \quad [1]$$

∇^{d} is the difference operator that indicates a series was differenced at order d (i.e., values at t subtracted from values at t-d) to remove seasonality or trend detected by the Dickey-Fuller (Dickey, Hasza & Fuller ,1984) test.

Y_t is the odds of IHD death for adults aged ≥50 years in month t. We specified the odds of death to allow familiar interpretation of the coefficients as an “effect on odds” metric.

H_t is the logged homicide rate in month t.

U_t is the unemployment rate in month t.

ω_0 is the estimated parameter for the homicide rate variable.

ω_1 is the estimated parameter for the total number of births variable.

θ is the moving average parameter.

φ is the autoregressive parameter.

B^p and B^q are backshift operators that yield the value of a at month t-p for autoregressive and t-q for moving average patterns respectively.

a_t is the error term for month t.

The analyses proceeded through the following steps. First, we used Box-Jenkins methods to identify and model autocorrelation in the monthly odds of IHD deaths. Second, we added the homicide and unemployment variables to the equation and estimated their coefficients. Third, we inspected the residuals from the full equation to ensure they exhibited no autocorrelation. Fourth, we assessed whether results appeared sensitive to the removal of outliers in IHD death. Fifth, as a falsification test, we repeated steps 1-4 except that we used diabetes mortality as the dependent variable. We assumed no relation between homicide rates and diabetes and therefore expect a null result for homicides in the diabetes falsification test.

RESULTS

As shown in Figure 1, IHD deaths exhibit strong winter peaks in January and February. IHD deaths also appear to have a slightly higher mean in the second half, relative to the first half, of the series. The ARIMA method identified such strong seasonality which required us to difference the series at 12 months (i.e., values at month t subtracted from values at month t-12) to render the time series a stationary one.

The time series also exhibited “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. Figure 2 shows the results of the ARIMA model in which we identified, and removed autocorrelation in the series. We use this “residualized” series as the dependent variable for our tests, as its mean is 0 and the expected value at all months is 0. Figure 3 shows the initial drop in homicides after Calderon’s “War on Drugs” followed by a steady, and then sustained, rise. We note that the logged-scale of the Y-axis tends to suppress the sudden absolute rise in overall homicides after 2006.

Table 1 presents the results of the time-series equation that estimates the log-odds of IHD deaths (equation [1]). Consistent with our hypothesis, IHD deaths vary positively with homicides in that same month (coef: .067, standard error (SE) = .016, $p<.001$). Given that the residual plot of IHD (Figure 2) shows some strong outliers in the dependent variable, we then applied outlier detection and adjustment methods to assess whether control for these outliers affected inference (Chang, Tiao, & Chen, 1988). Outlier-adjusted results, shown on the right column of Table 1, indicate that inference for the logged homicide coefficient remains similar to the original test, although the result becomes slightly stronger. To give the reader a sense of the magnitude of the findings, we took the antilog of the outlier-adjusted coefficient to yield the familiar “odds ratio” measure. A one unit increase in log-homicides corresponds with an IHD odds ratio of 1.08, which equates to approximately an eight percent increase. A one unit change in log-homicides reflects the observed difference in the level of homicides in Mexico in 2010-2012 relative to that before the “War on Drugs” (i.e., before

December 2006). This value equates to an excess of approximately 370 IHD deaths per month statistically attributable to a one unit increase in log-homicides.

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 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 the Methods. Results indicate no relation between log homicides and the log-odds of diabetes deaths in the same month (coef: -.039, SE = .029, p = .19; for the outlier-adjusted model, coef: .003, SE = .021, p = .90). This null finding indicates that homicides cannot predict other leading causes of death that are considered unrelated to acute stressors.

DISCUSSION

Mexico's sharp rise in its homicide rate following 2006 has raised international concern. 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 an eight percent increase in risk of IHD deaths with a one unit increase in log-homicides. Results remain robust to strong patterning in IHD deaths, control for the unemployment rate, and other sensitivity checks. Our time-series analysis supports the hypothesis that population-level homicides serve as an ambient, acute stressor that may increase deaths from IHD.

Strengths of our analysis include the near completeness of all homicide and IHD deaths in Mexico. Availability of monthly data, moreover, permitted careful testing of the acute temporal relation between the population stressor and IHD deaths that is not possible with annual aggregation or with cross-sectional data. Findings also cannot arise from shared patterns in IHD and homicides over time since we removed all autocorrelation from IHD deaths before evaluating its relation with homicide. Furthermore, we minimize the rival 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.

Limitations involve that we had no information on the nature of the IHD death. Our hypothesis pertains to IHD deaths after events (e.g., transient ischemic event, acute myocardial infarctions) that may plausibly respond to population threats or heightened anxiety pertaining to safety and security. We expect that future investigation might allow refined testing of IHD deaths precipitated by these acute cardiac events. We also did not have precise estimates of small-area level homicide rates and IHD deaths. Whereas Mexico provides such mortality counts by municipality level, we discovered a large amount of missing data. This circumstance precluded examination of area-level “hot-spots” in homicides. We, therefore, caution against using our country-level results to infer associations about particular municipalities or regions.

A possible explanation for these findings include that this population may respond acutely to a large increase in monthly homicide by individuals. Prior to March 2013, multiple media outlets in Mexico reported weekly homicides from organized crime (Jackson, 2013). The government maintained statistics for crimes alleged to have been

related to drug trafficking organizations (Molzahn, Rodriguez Ferreira, & Shirk, 2013). Therefore, the media frenzy and extensive coverage of this issue may have contributed to a sense of insecurity and stress.

Discussion of the War on Drugs in Mexico is often quantified in dollars spent combatting drug trafficking organizations, kilos of illicit substances seized and the number of dead bodies. Media outlets maintain that the increase in homicides was a result of the drug turf war and consist mostly of rival gang members killing each other despite anecdotal evidence of the contrary (The British Broadcasting Corporation [BBC], 2012; Daily Mail, 2011). While it is difficult to quantify how many homicides are drug-related, our findings represent a first step in quantifying its relation to the public health burden from increased IHD deaths. If our results are corroborated by additional research, the increase in IHD suggests that there is an (as of yet) unmeasured health cost to the drug war.

Figure 1: Odds of IHD death per 10,000 in Mexico over 156 months spanning January 2000 to December 2012.

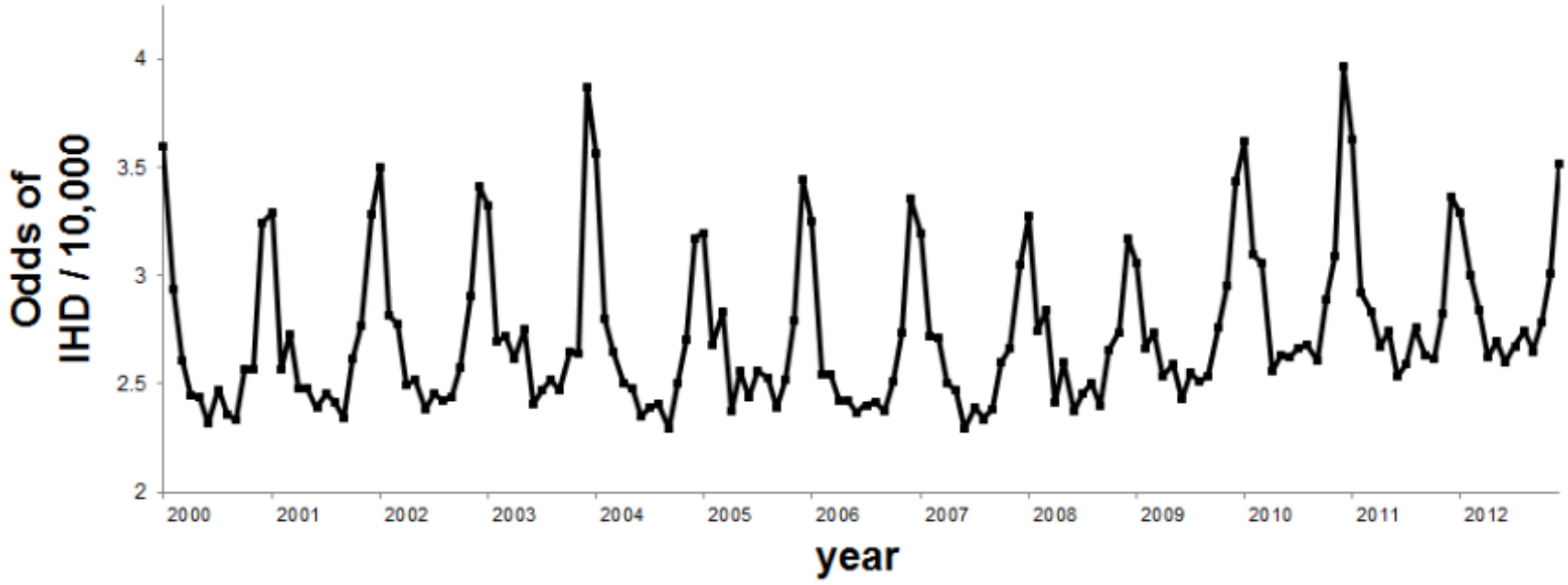

Figure 2: Residual log-odds of IHD death in Mexico over 156 months after identification and removal of autocorrelation. The mean of residuals is 0 and the series exhibits no temporal patterns. (First 14 months lost due to ARIMA modeling).

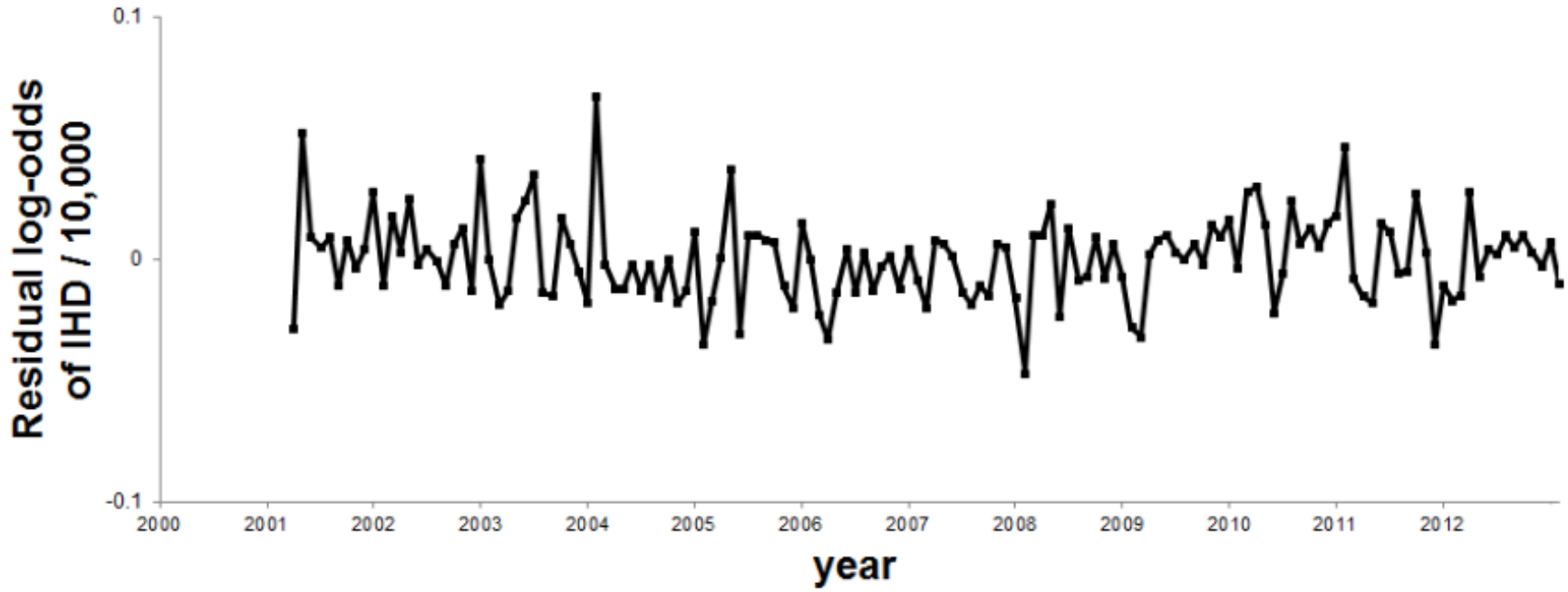

Figure 3. Logged homicide counts over 156 months spanning January 2000 to December 2012. The War on Drugs begins on December 2006 (month 84) and is indicated by the red marker.

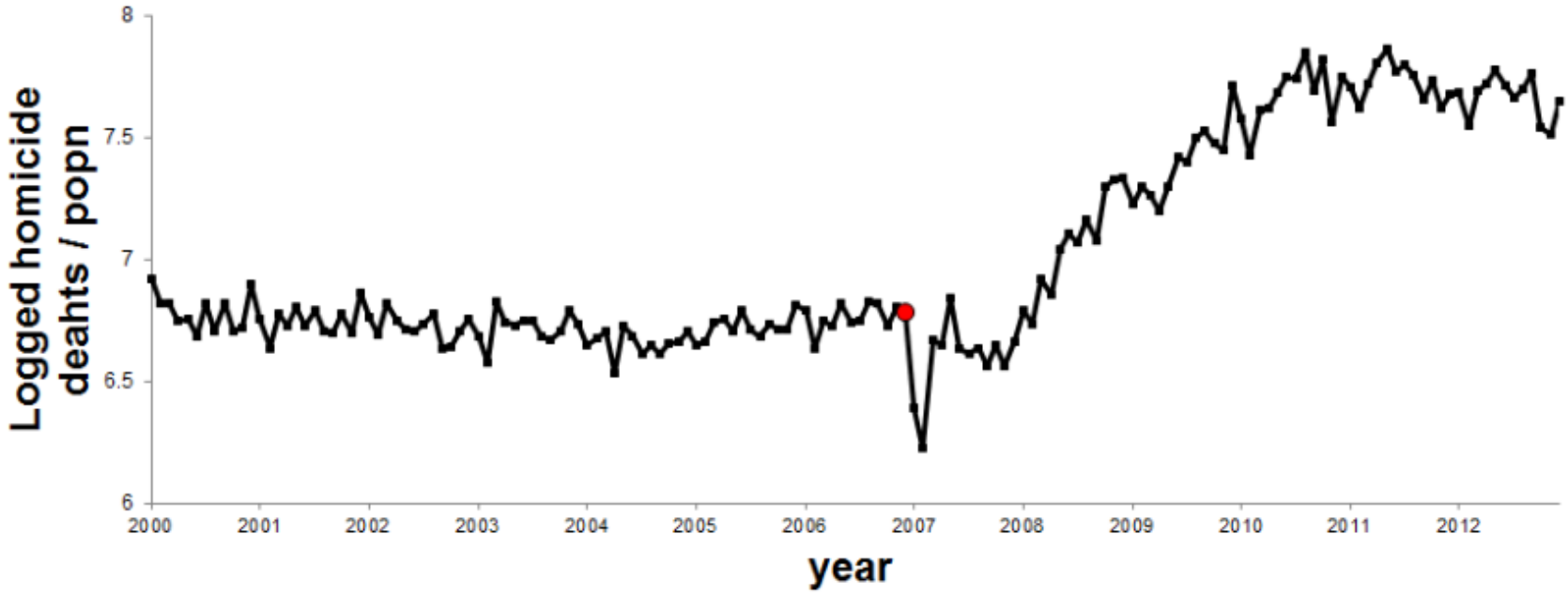

Table 1. Time series results predicting the monthly log-odds of IHD death in Mexico from January 2000 to December 2012 (standard errors in parentheses).

Parameter	Initial Model	Outlier-adjusted Model
Constant	—	—
Logged homicide rate lagged at 0 months	.067 (.016)***	.078 (.017)***
Unemployment Rate lagged at 0 months	.003 (.002)	.001 (.002)
Differenced at month:	12	12
AR parameter at month 1	.401 (.075)***	.448 (.077)***
MA parameter at month 12	.784 (.057)***	.762 (.060)***

†p<.10; *p<.05; **p<.01; ***p<.001; all tests are 2-tailed

REFERENCES

Barrett-Connor, E., Suarez, L., Khaw, K., Criqui, M. H., and Wingard, D. L. ((1984). Ischemic heart disease risk factors after age 50. *Journal of Chronic Diseases, 12*, 903-908.

Beittel, J. S. (2009). Mexico's drug-related violence. Congressional Research Service. Retrieved April 30, 2013 from http://www.fas.org/sgp/crs/row/R40582.pdf

Beittel, J. S. (2013). Mexico's drug trafficking organizations: Source and scope of the violence. Congressional Research Service. Retrieved April 30, 2013 from *www.fas.org/sgp/**crs**/row/R41576.pdf*

Box G., Jenkins G., Reinsel G. (1994). *Time series analysis: Forecasting and control* (3rd ed.). London: Prentice-Hall.

Chang, I., Tiao, G. C., and Chen, C. (1988). Estimation of time series parameters in the presence of outliers. *Technometrics, 30*(2), 193-204.

Crombie, I. K., Kenicer, M. B., Smith, W. C., and Tunstall-Pedoe, H. D. (1989). Unemployment, socioenvironmental factors, and coronary heart disease in Scotland. *British Heart Journal, 61,* 172-177.

Daily Mail. (2011, April 11). Drug cartels sink to new low as they murder children to scare off rival gangs in Mexico. *Daily Mail*. Retrieved from http://www.dailymail.co.uk/news/article-1375578/Drug-cartels-murder-children-scare-rival-gangs-Mexico.html

Dirección General de Información en Salud (DGIS) (2013). *Base de datos de mortalidad, 1985-2011*. [online]*: Sistema Nacional de Información en Salud*

(SINAIS). México: Secretaría de Salud. Retrieved October 28, 2013 from http://www.sinais.salud.gob.mx

Instituto Nacional de Estadística Geografía e Informática. (2014). Banco de Informacion Economica. Retrieved from http://www.inegi.org.mx/sistemas/bie/

Dickey, D. A., Hasza, D. P., and Fuller, W. A. (1984). Testing for unit roots in seasonal time series. *Journal of the American Statistical Association, 79*(386), 355-367.

Ebrahim, S. and Smith, G. D. (2001). Exporting failure? Coronary heart disease and stroke in developing countries. *International Journal of Epidemiology, 30*, 201-205.

Farr, W. (1985) Vital statistics: memorial volume of selections from the reports and writings, 1985. Bulletin of the World Health Organization 78: 88–95.

INEGI. (2012). Encuesta Nacional de Victimización y Percepción sobre Seguridad Pública, Retrieved from http://www3.inegi.org.mx/sistemas/tabuladosbasicos/tabgeneral.aspx?c=31914ands=est

Fisher R. A. (1921) Studies in crop variation: An examination of the yield of dressed grain. *The Journal of Agricultural Science, 11*, 107–135.

Herrera, A. R., Ubilla, C. M. S., and Murguia, P. (2013). Sistema de informacion para la vigilancia epidemiologica de los factores de riesgo en enfermedades cronicas no transmisibles (SVEECNT). Retrieved from http://www.epidemiologia.salud.gob.mx/doctos/reuniones_sinave/rojun13/viernes21/2_SVEECNT.pdf

Instituto Nacional de Estadística Geografía e Informática (INEGI). (2014). Principales causas de mortalidad por residencia habitual, grupos de edad y sexo del fallecido.

Retrieved from http://www.inegi.org.mx/est/contenidos/proyectos/registros/vitales/consulta.asp?c=11800#

Jackson, A. (2013, March 11). Mexico: Another newspaper stops reporting on organized crime due to safety concerns. *Global Post*. Retrieved from http://www.globalpost.com/dispatch/news/regions/americas/mexico/130311/mexico-newspaper-zocalo-stops-reporting-organized-crime

Kivimaki, M., Virtaren, M., Elovainio, M., Kouvonen, A., Vaananen, A., and Vahtera, J. (2006). Work stress in the etiology of coronary heart disease –a meta-analysis. *Scandinavian Journal of Work, Environment and Health*. 32(6), 431-442.

Lopez, A. D., Mathers, C. D., Ezzati, M., Jamison, D. T., and Murray, C. J. L. (2006). Global and regional burden of disease and risk factors, 2001: systematic analysis of population health data. *The Lancet, 367*(9524), 1747-1757.

Mittleman, M. A. & Mostofsky, E. (2011). Physical, psychological and chemical triggers of acute cardiovascular events: preventive strategies. *Circulation*, 124(3), 346-54.

Molzahn, C., Rodriguez Ferreira, O., and Shirk, D. A. (2013). *Drug violence in Mexico. Transborder Institute. Retrieved January 23, 2013 from http://www.sandiego.edu/peacestudies/institutes/tbi/publications/reports.php*

Rabkin, J. G. and Struening, E. L. (1976). Life events, stress, and illness. *Science*, 194, 1013-1020.

Seretakis, D., Lagiou, P., Lipworth, L., Signorello, L. B., Rothman, K. J., and Trichopoulos, D. (1997). Changing seasonality of mortality from coronary heart disease. *Journal of the American Medical Association, 278(*12), 1012-1014.

Stamler, J., Vaccaro, O., Neaton, J. D., and Wentworth, D. (1993) Diabetes, other risk factors, and 12-yr cardiovascular mortality for men screened in the Multiple Risk Factor Intervention Trial. *Diabetes Care, 16*, 434–444.

Stevens, G., Dias, R. H., Thomas, K. J. A., Rivera, J. A., Carvalho, N., Barquera, S., Hill, K., and Ezzati, M. (2008). Characterizing the epidemiological transition in Mexico: National and subnational burden of diseases, injuries, and risk factors. *Plos Medicine, 5(*7). DOI: 10.1371/journal.pmed.0050125.

Sundquist, K., Theobald, H., Yang, M., Li, X., Johansson, S. E., and Sundquist, J. (2006). Neighborhood violent crime and unemployment increase the risk of coronary heart disease: A multilevel study in an urban setting. *Social Science and Medicine, 62*, 2061-2071.

The British Broadcasting Corporation. (2012, February 18). Mexico drug wars: Murders down in Ciudad Juarez. *BBC News*. Retrieved from http://www.bbc.co.uk/news/world-latin-america-17082002

U.S. State Department (June 2013). Merida Initiative. Retrieved on June 13, 2013 from http://www.state.gov/j/inl/merida/

Wilson, P. W. F., D'Agostino, R., Levy, D., Belanger, A. M., Silbershatz, H., and Kannel, W. B. (1998). Prediction of coronary heart disease using risk factor categories. *Circulation*, 97, 1837-1847.