**Title: Homicides increase variation on lifespans in Mexico and its States, 2005-2015 [intended for Demography] [Second PDR] [or a health journal]**

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**Abstract [Max 250 words]:**

**\maintext[max 8000 words]**

**Introduction**

Violence is a main public health issue in Latin America since the end of the 20th century (1). This region experiences the highest homicide rate in the world (over 16.3 per 100,000 people), and a set of countries in Central America, including Mexico, have undergone an upsurge of homicide mortality in the first years of the 21st century (2). In Mexico, homicide rates doubled between 2007 and 2012 (from 9.3 to 18.6) (3). As a result of this increase, along with the burden of diabetes, male life expectancy in Mexico stagnated in the period 2000-10 (4). At the subnational level, evidence indicates that gains in life expectancy due to causes amenable to medical service throughout 2000-10, such as infectious and respiratory diseases and birth conditions, were wiped out by the increase of homicide and diabetes mortality in each of the 32 states in Mexico, albeit with large regional variations (5).

Trends in life expectancy are important and have been studied in Mexico and its states (4-6). However, life expectancy masks substantial heterogeneity in individual mortality trajectories (7, 8), referred here as lifespan variation. This age-at-death variation expresses a fundamental inequality between individuals (9), and it has arisen as an important topic since it addresses the growing interest in health inequalities and its linkage with health behavior (10). Studying both life expectancy and lifespan variation is important because individuals take decisions based not only on their expected lifetime, but also on the uncertainty in their timing of death (11). Most studies have found a negative association between these two measures, suggesting that as life expectancy increases, variation in lifespans decreases (8, 12-14). However, at the subnational level some evidence suggest that increases in variation occur with simultaneous increases in life expectancy, mostly due to a slowdown in mortality improvements in working ages (15, 16). To date, no comprehensive study of lifespan variation has focus on the effect of the sharp increase in homicide mortality under periods of life expectancy decline or stagnation.

In this study, we focus in the Mexican case, which shows substantial mortality fluctuations and large regional variation. Given the unexpected rise in homicide mortality after 2005 that have disproportionally affected young adults and the increasing burden of diabetes mortality among adults in the new century, along with improvements in mortality due to medically amenable conditions and other causes of death, it is imperative to measure their effect on the predictability of age at death in the Mexican population. For instance, states in the Northern part of the country (e.g., Chihuahua, Durango and Sinaloa) that experienced the largest losses in life expectancy due to homicides between 2005-10 (5) may also exhibit the largest effect on lifespan variation in the country, although in recent years this impact may be larger in other states as homicides spread throughout the entire country (17). However, since the more pronounced fluctuation in age-specific mortality occurred over working ages (5), it is unclear what the net effect would be on lifespan variation but it certainly had an effect on premature mortality. On the other hand, there have been mortality improvements in the country particularly at younger ages which have been a priority in the country since the 1990s (e.g., birth-related conditions) (18, 19). These improvements could have a substantial effect on reducing variation in lifespans, particularly in historically poor states, which are mostly concentrated in the South. Thus, we analyzed how lifespan variation changed over a 20-year period, from 1995 to 2015, for females and males in Mexico and its 32 states, and determined the ages and causes of death that contributed the most to the observed change in lifespan variation.

**Study data and Methods**

Data on deaths from vital statistics files publicly available through the Mexican National Institute of Statistics and Geography were used (20). These data include information on cause of death by age at the time of death, sex, and place of occurrence from 1995 to 2015. Additionally, we used population estimates corrected for completeness, age misstatement, and international migration available from the Mexican Population Council to construct age-specific death rates by sex and state [waiting for the period 2010-15, now these years are projections] (21).

***Cause-of-death classification***

We classify deaths into eight categories according to previous studies targeting the main causes of death in Mexico (5, 22) using the concept of amenable/avoidable mortality (23, 24). This concept assumes that there are some conditions that should not cause death in presence of timely and effective medical care. Deaths due to these conditions are a proxy of the performance of health care systems (23).

The first category includes conditions amenable to medical service. It refers to mortality that could be reduced by primary or secondary prevention, and timely medical care (for example, birth conditions, infectious and respiratory diseases). We analyze separately homicide, diabetes, ischemic heart diseases (IHD), lung cancer, cirrhosis, and road traffic accidents because the first two are leading causes of death in Mexico (4), and all of them are amenable to health behavior and medical service (5). The eighth category includes the rest of causes of death labeled ‘Rest’. For details on the International Classification of Diseases [ICD] codes for each cause see the Supplemental file.

Originally, data on deaths were classified according to the International Classification of Diseases (ICD), revision 9 for years 1995-1997 and revision 10 for 1998-2015. Previous studies have checked the validity of the cause-of-death codes used in this paper and did not find cause-specific ruptures in the transition from ICD 9 to ICD 10 [reference]. In addition, to mitigate biased interpretations we focus on causes of death below age 85 since coding practices above that age are less reliable due to the presence of comorbidities [referece].

***Dispersion measure***

Several dispersion measures have been proposed to analyze lifespan variability (25). In this study, is used as a dispersion indicator and we refer to it as “life disparity”. It is defined as the average remaining life expectancy when death occurs, or life years lost due to death (13, 26). For example, if a cohort of newborns die at the same age then the value of is zero; while when death is very variable, people will die before their expected lifetime, contributing lost years to life disparity. In lifetable notation it is defined as:

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where and are the survival function, the force of mortality, life expectancy at age , and the open-aged interval, respectively. This indicator was chosen because it has an easy to understand interpretation and it is also easy to decomposa allowing us to quantify the impact of age and cause-specific mortality on changes in life disparity over time (27, 28). . An additional advantage is the high correlation between and other measures of variability in ages at death (e.g., life table entropy, coefficient of variation, or the Gini coefficient) which suggests that ourmain results would be very similar to those obtained with any of these additional measures (25).

***Demographic and statistical methods***

To mitigate random variations in cause-of-death classification, we smoothed cause-specific death rates over age using a 1-d p-spline separately by year, sex and state (29). We then rescaled the smoothed cause-specific deaths to all-cause death rates to maintain the overall mortality level by year, sex, and state. Using these mortality rates we computed period life tables for males and females for each year-state in the study period (1995 to 2015) following standard demographic methods (30). Finally, we computed life disparities in 1995, 2005 and 2015 and estimated the age- and cause-specific contributions to the difference in life disparity between each period, 1995-2005 and 2005-2015, using standard decomposition techniques (31). All the analyses were carried out using R (32) and are fully reproducible from the Supplemental file.

**Preliminary Results**

**Discussion**

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