Title: The impact of violence in Venezuelan life expectancy and lifespan inequality

ABSTRACT
Background: During the first decade of the 21st century, Venezuela had one of the world's highest mortality rates due to violence. The upsurge of violence coincided with a slowdown in life expectancy improvements. In this study, we quantified the impact of violence-related mortality and other causes of death on slowing down life expectancy gains and on lifespan inequality (the dispersion of the ages at death) from 1996 to 2013.

Methods: Corrected age-specific mortality rates were computed into yearly life tables. Changes in life expectancy and lifespan inequality by sex from 1996 to 2013 were decomposed by age and causes of death.

Results: For females, life expectancy rose by +3.8 years and lifespan inequality fell by -1.2 years in 1996-2013. For males, life expectancy increased by only 1.4 months yearly and lifespan inequality even increased by almost one year throughout the period. Deceleration in male life expectancy gains was accompanied by an increasing sex differential. Increasing violence-related deaths resulted in a loss of 1.5 years of male life expectancy, and an increase of 1.2 years of lifespan inequality, while the decline of all other causes would have produced a gain of +3.5 years in life expectancy.

Conclusions: The impact of violence-related deaths among young men (ages 15 to 39) reversed gains in male life expectancy and increased lifespan inequality. Consequently, males in Venezuela are not leaving less on average, but facing larger uncertainty in their eventual death due to premature mortality caused by the upsurge of violence.

Key words: external causes of death, decomposition analysis, homicides, young male mortality, firearm-related deaths, cardiovascular revolution.

Key messages

- The recent upsurge of violence has caused male life expectancy gains from other causes of death (e.g. circulatory, infectious and respiratory) to be reversed.
- The gap between male and female life expectancy is increasing due to the excess mortality among young men.
- Increasing inequality of lifespans due to premature mortality underlies larger uncertainty in the eventual time of death of Venezuelan males.
- Opposing the eroding effect of violence, cardiovascular mortality and under-five mortality are the main drivers of increasing life expectancy in both sexes.
- The ongoing violence in Venezuela represents an urgent priority for strategies to mitigate the impact on the Venezuelan population.

INTRODUCTION
Most Latin American countries experienced sizable improvements in health, living standards, and longevity in the second half of the twentieth century.\(^1\) In Venezuela, mortality started to decline progressively from 1930:\(^2\) Life expectancy increased from 54.9 years in 1950 to 74.2 years in 2013.\(^3,4\) These advances were driven first by a reduction in infant mortality, then by the postponement of death in adults, and finally by improvements in old-age mortality.\(^5\) However, gains in life expectancy have slowed for both sexes since the mid-1990s: while between 1950 and 1990 life expectancy increased by 3.8 years per decade, since 1990 gains have fallen to 1.8 years every 10 years.\(^3\)

This slowdown coincides with a continuous rise of violence in Venezuela; indeed, at the beginning of 1990s an “epidemic” of violence had already been identified.\(^6,7\) Homicides increased steadily and more than doubled between 1995 and 2009 (from 20.3 to 49.0 homicides per 100,000 inhabitants).\(^8\) By 2010, around 13% of overall deaths were due to violence and injuries,\(^9\) ranking Venezuela as the country with the fourth highest crude rate of mortality from external causes in the world.\(^10\)

Life expectancy is the most widely used indicator to summarize population health and reflects the overall level of longevity of a population. This study assesses the contribution of violence to the recent slowdown in life expectancy gains in Venezuela. We further quantify the effect of homicides in an equally important dimension of health: the dispersion of the age of death distribution (referred to hereafter as lifespan inequality).\(^11\) Lifespan inequality, an indicator of how similar ages at death are, has arisen as an important public health topic, in association with the growing interest in health inequalities.\(^12\) It is interpreted as a marker of heterogeneity in age at death at the macro level\(^13\) and of survival uncertainty at the individual level.\(^14,15\) Since lifespan inequality is highly sensitive to premature mortality\(^14\) and homicides are concentrated in working ages, the net effect of the upsurge of violence is unknown. Studying life expectancy alongside lifespan inequality in the context of increasing violence gives policy makers a better understanding of the consequences of violence on population health. The combination of both indicators gives an idea of individuals’ decisions based not only on their expected lifespan, but also on the uncertainty surrounding their eventual death.\(^11\)
Homicidal mortality in Venezuela since the beginning of 1990s has been concentrated between ages 15 and 50 and affected mainly males (10 times more than females).\textsuperscript{(16)} We therefore hypothesize that it has contributed strongly to the slowing of improvements in male life expectancy and, consequently, to the increasing sex differential. As life expectancy and lifespan inequality are negatively correlated,\textsuperscript{(14)} we expect to observe a similar adverse effect on lifespan inequality reductions. To test our hypotheses we focused on the period from 1996 to 2013. In addition to the upsurge of violence, this choice of period covers major changes in the epidemiological profile of Venezuela. The stage of mortality decline from circulatory diseases\textsuperscript{(17)} and the emerging importance of cancer and diabetes\textsuperscript{(4)} set the starting point of this study.

**DATA**

We used death counts by cause, sex and age from official mortality yearbooks reported by the Venezuelan Ministry of Health from 1996 to 2013,\textsuperscript{(18)} and annual population estimates from the Venezuelan National Institute of Statistics\textsuperscript{(19)} to compute age-specific death rates. Deaths are classified according to the 10\textsuperscript{th} revision of the International Classification of Diseases (ICD-10) for the period studied. Deaths are aggregated into 5-year age groups, with deaths under one year, and over 80 in separate age groups.

Causes of death were grouped into: 1) circulatory diseases (heart diseases, hypertensive, ischemic heart diseases and cerebrovascular diseases), 2) neoplasms (C00-C97), 3) diabetes, 4) homicides and other violent causes with undetermined intention (homicides, undetermined intent and legal intervention), 5) other external causes (including traffic accidents, injuries and suicide), 6) respiratory diseases, 7) infectious diseases, 8) digestive diseases, 9) conditions originated in the perinatal period, and 10) remaining causes (ICD-10 grouping in supplementary material Table 1).

To ensure data quality, mortality estimations were adjusted for underreporting and age misreporting.\textsuperscript{(20, 21)} We applied indirect estimation methods – specifically the synthetic extinct
generation\textsuperscript{(22)} for adult mortality and Brass method in Trussell variant\textsuperscript{(23)} for infant mortality – on data from the 1990, 2001 and 2011 population censuses. We chose these methods based on data availability and their effectiveness. Synthetic extinct generation is the method that produces the fewest errors for the different adult mortality scenarios found in Latin America and the Caribbean.\textsuperscript{(24)}

A set of under-registration ratios was obtained by contrasting inter-census indirect estimation and directly estimated mortality rates. These under-registration ratios were linearly interpolated and extrapolated into the timeframe of our analysis. We assumed that the rate of under-registration does not fluctuate widely but declines smoothly at a constant pace.

**METHODS**

Annual period life tables were constructed using standard demographic methods.\textsuperscript{(25)} From these, life expectancy and lifespan inequality were calculated. Lifespan inequality was measured by the standard deviation of the age at death distribution ($\sigma$).\textsuperscript{(26)} Changes in both indicators during the study period were decomposed based on a continuous change model.\textsuperscript{(27)} Through decomposition, we dissected contributions (in years) to changes in life expectancy and lifespan inequality by each cause of death at each age.\textsuperscript{(28)} The decomposition method used in this paper is based on the line integral model.\textsuperscript{(27)} This method has the advantage of assuming that covariates change gradually along the time dimension (See supplementary material for further details).

Although several lifespan inequality indicators exist (e.g. Gini coefficient, life years lost, variance), the high correlation between them suggest that our main results would be consistent with those obtained by another indicator.\textsuperscript{(26)} In addition, by using the standard deviation we ensure comparability with life expectancy since both are expressed in years. These indicators were chosen because they are easy to understand, to interpret, and to decompose, thereby allowing us to quantify changes in age and cause-specific mortality over time.
RESULTS

Life expectancy at birth increased continuously, although the male lag is evident. Male life expectancy increased by 2 years, from 68.6 to 70.6 years, between 1996 and 2013, while the female gain was almost double (from 75 to 78.8 years), gaining 3.8 years. Differences in mortality reductions led to an increase in the sex differential in life expectancy (from 6.4 years in 1996 to 8.2 years in 2013).

Changes in life expectancy at all ages are shown in figures 1A (Male) and 1B (Female). Specific decomposition of gained (positive) and lost (negative) years of life expectancy by changes in each cause of death group are indicated by the bars. Additionally, the sum of overall contributions by age-group is shown at the bottom and by causes of death in the legend.

Changes in life expectancy are attributable to heterogeneous contributions. Almost all age groups benefitted from increasing life expectancy, although the largest gains occurred at ages below one and above 55. Infant mortality accounts for 13% (+0.51 years) and 33% (+0.66 years) of gains in life expectancy for females and males, respectively. Gains are mostly due to conditions of the perinatal period, respiratory and infectious diseases. At older ages, circulatory diseases contributed to life expectancy gains by 1.86 years for females and 1.41 years for males. However, these gains were eroded by increasing mortality from diabetes and violence. Diabetes mortality lowered male and female life expectancy by 2.5 and 1.4 months, respectively, mostly affecting the population over 50 years old. Meanwhile, homicides had the most dramatic negative impact on male life expectancy (−1.52 years), whereas the impact in women was negligible (-0.07 years). Life expectancy losses due to homicides were concentrated in men between 15 and 50 years old.

Lifespan inequality decreased for females by -1.2 years between 1996 and 2013 (from 18.5 to 17.3 years). For males, on the contrary, inequality in lifespans increased by more than nine months (from 20.7 to 21.6 years). Figure 2 shows age and cause-contributions to these changes. This figure depicts information in a similar format to figure 1. Positive (negative) values increase (decrease) lifespan
inequality. Major contributions of under-five mortality (1.36 years in males and 1.46 years in female) due to improvements in perinatal conditions, infectious and respiratory diseases contributed to the reduction of lifespan inequality in both females and males. However, these improvements were cancelled out in males by the upsurge of homicide mortality at young ages (15-45). Homicides increased lifespan inequality by 1.31 years (+1.2 years for homicides and other violent causes of death and +0.11 for other external causes). In addition to violence, lifespan inequality increased due to improvements in cardiovascular mortality at ages above 40 years (blue bars).

**DISCUSSION**

The impact of violence has had a detrimental effect on population health in Venezuelan males. The high prevalence of violence in young men has eroded all the gains from other causes, and is responsible for a large part of the sex differential in mortality. Consequently, improvements in life expectancy have slowed down, and as we show, lifespan inequality has even increased. Males in Venezuela are not dying earlier, on average, but are facing larger uncertainty about their eventual time of death due to premature mortality by violence. In this sense, this is the first study documenting the effect of homicides on increasing lifespan inequality in tandem with the stagnation in life expectancy at the national level. The uniqueness of the Venezuelan experience could be linked to the combination of two circumstances: wealth and homicides. Unprecedented increase in national income per capita that enabled widespread distribution of wealth at individual level occurred at the same time as the most pronounced upsurge in homicide rates.\(^{(33)}\)

This excess young male mortality is a recent phenomenon. The homicide rate in Venezuela was relatively low throughout the 1980s, close to levels of countries like Costa Rica, at around 8 per 100,000 inhabitants.\(^{(34)}\) However, considerable social and economic changes during that decade brought changes in the prevalence of violence.\(^{(35)}\) Up to then, violence had been political conflict related and rarely the subject of explicit attention. Afterwards, and partly due to rapid urbanization
and rural-urban migration, violence became a phenomenon associated with urban agglomerations and their “slums”. This shift in prevalence reflects the increase in social inequality and the fragile legitimacy of the state, together with a genuine “culture of violence”\(^{(34)}\) that has developed and grown in strength in most Latin American countries.

Three major violent events preceding our timeframe have been marked as tipping points: the popular uprising against price increases known as “El Caracazo” (1989) and two attempted coups (1992 and 1993).\(^{(16, 36)}\) These events doubled the preexisting homicide rate, and since then violence has been unstoppable. To put this in perspective, the homicide rates in Venezuela in 2012 (53.7 per 100,000 inhabitants) were higher than those of Latin American countries with undeclared civil wars in recent decades,\(^{(6)}\) such as El Salvador (41.2), Guatemala (39.9) and Colombia (30.8). The impact is such that in 2010 the prevalence of violence in Caracas (80.6), claimed more victims than full-blown war did in Iraq (54.6) that same year.\(^{(10)}\) Legally declared homicides, “extra-judicial executions” by organized crime groups, police brutality and violent death of indeterminate cause were the most common causes of these violent deaths.\(^{(37)}\) Similar results have been reported for Mexico, where life expectancy stagnated due to the increase in homicide mortality related to the war on drugs.\(^{(38, 39)}\)

Venezuelan government officially deployed policies aimed at stopping violence and criminal activities. These included legal and structural reforms in all existing local police forces (2006), the creation of a unique national police force (2009), and the creation of a presidential commission to control firearms and weapons (2011).\(^{(40)}\) As our results show, all these efforts were futile. The reason is linked to the structural weakness of Venezuelan public institutions\(^{(43)}\) and their high levels of corruption, but also to the ambivalent attitude of the government towards tolerating and encouraging the use of violence.\(^{(41)}\) Examining experience in the region, during the period of our analysis, Colombia and Venezuela not only saw opposite trends but almost swapped their homicide rates. From 2000 to 2012, Colombia managed to decrease its incidence of violence, from a higher starting point (66.5 to 30.8 per 100,000 inhabitants) than Venezuela (32.9 to 53.7 per 100,000 inhabitants).\(^{(8)}\) These reverse outcomes reflect different institutional approaches to social control.\(^{(33)}\)
While Venezuela was experiencing a systematic process of institutional annihilation, Colombia strengthened the credibility of its institutional mechanisms of access to justice.\(^{(42)}\)

Our results underscore the effect of violence on longevity and on lifespan inequality in Venezuela. In fact, the country could potentially increase its overall longevity and decrease inequality of lifespans just by achieving a clear public health target of reducing homicides. To reverse the future detrimental effects of violence, new public health interventions must be applied. An obvious policy target for reducing homicides is to disarm the civil population and effectively control the legal use of arms. Most of the tallied homicides in this analysis were committed with firearms, which are widely available in the country. In 1996 1.8% of overall deaths were inflicted by firearms. This proportion more than tripled in 2013 to 6%. In parallel, between 2003 and 2007 Venezuela was the 2\(^{nd}\) country in South America and 17\(^{th}\) in the world in terms of military spending increases .\(^{(43, 44)}\) Soaring weapons supply in a context of institutional weakness enlarged the existing black market in which local police and armies have become the main weapons smugglers.\(^{(45)}\)

Opposing the effects of violence, our findings show that positive changes in life expectancy and lifespan inequality were mostly driven by improvements in cardiovascular diseases. These results are in line with expected trends during the second cycle of the health transition.\(^{(32)}\) Some additional improvements in under-5 mortality were also captured. These two processes occurred in a relatively similar manner for both sexes in Venezuela and were more beneficial for women.

The first challenge when studying Latin American mortality is undoubtedly data quality and coverage. Venezuela has a good quality vital statistics system compared with most Latin American countries.\(^{(29)}\) Nevertheless, continuous coverage improvements during the period of our analysis\(^{(30, 31)}\) could be introducing artificial over-estimations corresponding to wider scope of the vital statistics system and not to demographic phenomena. Adjusting estimations by incorporating under-registration dynamics has become a mandatory task. Inter/extrapolating the inter-census under-registration ratios allowed us capturing theses change in the coverage and ensured accurate comparison through time.
Future scenarios in Venezuela do not seem promising. Outbreaks of political violence have intensified in recent years and the steady militarization of public order functions could further increase the prevalence of violence. Unfortunately, official mortality data are hard to come by. The Venezuelan government has adopted a policy of total secrecy with regard to mortality counts. Official mortality databases are currently unavailable and no official mortality estimates have been published since 2013. The additional effects of the accentuated political and socio-economic breakdown on mortality trends cannot be updated at this moment. Our findings suggest that in the absence of previous wealth, male life expectancy is likely to move from stagnation to decline, and that life expectancy and lifespan inequality will be negatively correlated.

Reference
31. UNICEF. Birth registration in Latin America and the Caribbean: Closing the gaps. New York: Section Division of Data; 2016 September 2016.
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Supplementary material

Table 1. International Classification of Diseases 10th revision (ICD-10) coding grouping

<table>
<thead>
<tr>
<th>Causes of death group</th>
<th>International Classification of Diseases 10th revision coding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Circulatory diseases (cardiovascular, stroke)</td>
<td>I05-I09, I11, I13, I21-I51, I60-I69</td>
</tr>
<tr>
<td>Neoplasms</td>
<td>C00-C97</td>
</tr>
<tr>
<td>Diabetes</td>
<td>E10-E14</td>
</tr>
<tr>
<td>Homicides and other violent causes with undetermined intention</td>
<td>X85-Y09, Y10-Y34, Y35-Y36</td>
</tr>
<tr>
<td>Other external causes (including traffic accidents, injuries and suicide)</td>
<td>V01-V89, V90-X59, X60-X84</td>
</tr>
<tr>
<td>Respiratory Diseases</td>
<td>J00-J98</td>
</tr>
<tr>
<td>Infectious Diseases</td>
<td>A00-B99</td>
</tr>
<tr>
<td>Digestive Diseases</td>
<td>K00-K92</td>
</tr>
<tr>
<td>Conditions originated in the perinatal period</td>
<td>P00-P96</td>
</tr>
</tbody>
</table>

Brief description of the lifespan variation indicator

In lifetable notation, it is:

$$\sigma = \sqrt{\int_a^\omega (x-e_a)^2 f(x) \, dx}.$$  \hspace{1cm} (1)

Where $f(x)$, $e_a$, and $\omega$ denote the age at death density function, life expectancy at age $a$, and the open-aged interval (110+ in our case), respectively.

Description of the decomposition method

The decomposition method used in this paper is based on the line integral model (Horiuchi et al 2008). Suppose $f$ (e.g. $e^+$ or life expectancy) is a differentiable function of $n$ covariates (e.g. each age-cause specific mortality rate) denoted by the vector $\mathbf{A} = [x_1, x_2, \ldots, x_n]^T$. Assume that $f$ and $\mathbf{A}$ depend on the underlying dimension $t$, which is time in this case, and that we have observations available in two time points $t_1$ and $t_2$. Assuming that $\mathbf{A}$ is a differentiable function of $t$ between $t_1$ and $t_2$, the difference in $f$ between $t_1$ and $t_2$ can be expressed as follows:
\[ f_2 - f_1 = \sum_{i=1}^{n} \int_{x_i(t_1)}^{x_i(t_2)} \frac{\partial f}{\partial x_i} dx_i = \sum_{i=1}^{n} c_i, \quad (2) \]

where \( c_i \) is the total change in \( f \) (e.g. \( e^t \) or life expectancy) produced by changes in the \( i \)-th covariate, \( x_i \). The \( c_i \)'s in equation (2) were computed with numerical integration following the algorithm suggested by Horiuchi et al (2008). This method has the advantage of assuming that covariates change gradually along the time dimension.