

# **IS IT POSSIBLE TO TALK ABOUT A POLARIZED - PROLONGED TRANSITION EPIDEMIOLOGICAL MODEL IN VENEZUELA?<sup>1</sup>**

An Analysis of Venezuelan Epidemiological Profile in the years 2000-2010

**Jenny García**

## **ABSTRACT**

In the attempt to build theoretical frameworks that synthesize explanations for changes in epidemiological profile throughout history, the epidemiological transition theory arises. Many critics have been made to this theory, among them, Frenk & al (1991), who point to the idea of an extended- polarized model in the case of Latin America epidemiological transition. Through the verification of the assumptions behind this model, structure of causes of death of Venezuela is analyzed with the main objective of characterizing its epidemiological profile during the years 2000 to 2010. This investigation uses partial and bivariate correlations and simple regression analysis of standardized mortality rates for the population aged 5 and older. The results indicate a clear pertinence of the model at national level analysis and some differences in the way that epidemiological profiles are being configured for women and men in the country.

**Keywords:** structure of causes of death, mortality patterns, epidemiological transition, polarized - extended model, Venezuela.

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<sup>1</sup> Translation of "¿ES VIGENTE EL MODELO POLARIZADO-PROLONGADO DE TRANSICIÓN EPIDEMIOLÓGICA EN VENEZUELA? UN ANÁLISIS DEL PERFIL EPIDEMIOLÓGICO VENEZOLANO EN LOS AÑOS 2000-2010" Master thesis resume presented in Abril 2014 as "Estructura de las causas de muerte en Venezuela, 2000-2011"

## 1. INTRODUCTION

In the attempt to construct an analytical framework that synthesizes explanations for the decline in mortality in the world and changes in the epidemiological profiles throughout history, the epidemiological transition theory arises. The term epidemiologic transition refers to the change in the patterns of causes of illness and death, as well as age in which these are manifested (Omran 1971, Frederiksen 1966, Olshansky and Ault 1986, Horiuchi 1997). There have been many inquiries made to this theory; most of them concentrate on the underlying idea of evolutionary and lineal progress of societies, splitting the particularities introduced by differential contexts (Barret et al, 1998; Gaylin, 1997; Caselli et al, 2002). Looking for more open frameworks and interdisciplinary approaches, have emerged alternative proposals to the theory of classical epidemiological transition like health transition (Caldwell, 1993; Caselli et al, 2002; Vallin & Meslé, 2004), risk transition (Smith 1990) and health care transition (Frenk et al, 1991b). With these terms are targeted the recognition of a much more complex analysis than the simple evolution of epidemiological profiles dynamics.

Frenk et al (1991) advanced a theoretical proposal that seeks to complement the different transition models presented above and generate an adaptive analytical framework to the way it has produced the decline in mortality in Latin America, its determinants and evolution of existing patterns of morbidity and mortality. The authors keep from Omran's analytic proposal (1971), the three main changes in epidemiological profile: 1) the replacement of the causes of deaths from infectious diseases, non-communicable diseases and injuries, 2) the displacement of the greater burden of morbidity and mortality from young to elderly, 3) predominance of morbidity over mortality (Frenk et al, 1991a). However, they focus in questioning the order in which such changes may occur when national level analysis is taking place. Their central point relied on the idea of a polarized- prolonged model of epidemiological transition, released of unidirectional connotations (Di Cesare, 2011). This polarized-prolonged model would be characterized by:

- An *overlap of stages* refers to the simultaneous incidence of infectious diseases and chronic degenerative diseases.
- *Reappearance* of already controlled infectious diseases such as malaria, dengue and cholera.
- A Prolonged situation of no clear resolution of the transition process, continued *stagnation in the duality* of epidemiological profiles.
- Recognition of the heterogeneity present internally in the countries. Social and geographic inequalities in the region were causing an overlapping of stage. This occurs because the poor urban dwellers and rural maintaining a different epidemiological profile to the rest of the population. Distinction introduced between urban and rural context especially for the lower strata, is what generates greater *epidemiological polarization* at national level (Frenk et al, 1991b).

In recent decades, Venezuelan epidemiological profile as a whole was characterized by increased life expectancy, lower infant mortality, augmented deaths among older adults and the proportion of deaths from heart disease, stroke and cancer. This is due to the improvements in the quality of life of the population and the expansion of coverage of the health system carried

out since the second half of the twentieth century (UNFPA, 2006; Avilan, 1998). In parallel, a significant number of deaths are still present for infectious and contagious associated with lack of access to basic sanitation, as well as deaths from external causes, especially in men between 15 and 34 years (Di Brienza et al, 2010).

Considering this background, the main objective of this paper is to analyze the structure of causes of death in an attempt to characterize the epidemiological profile of Venezuela during the first decade of the century. To do this, on the one hand, it is taken from literature the polarized- prolonged model of epidemiological transition proposed by Frenk et al (1991). On the other hand, statistic techniques used by Preston (1976) in the evaluation of the structure and change in causes of death worldwide. Preston uses partial and bivariate correlations and simple regression analysis of standardized mortality rates of specific causes of death to compare and characterize mortality patterns of different countries through time. With the combination of both approaches this paper answers the question: is the structure of causes of death in Venezuela during the years 2000 to 2010 responding to the polarized-prolonged model of epidemiological transition? And specifically, is there an overlap of stages referring to the simultaneous incidence and proportion of causes of death identified as belonging to different profiles in the classical epidemiological transition theory? Is there stagnation in the duality of epidemiological profiles? Is there an epidemiological polarization at national level set by distinction introduced between urban and rural context?

The results of the analysis come as correlations and a linear model of variation between standardized mortality rates (SMR \*) of the population aged 5 and older by specific group of causes of death. In spatial terms are considered three groups of geographical units categorized both by the proportion of urban population contained.

Data is taken from the Mortality yearbooks of the Health Ministry of the Bolivarian Republic of Venezuela, obtained from death certificates issued by hospital or forensic medicine center. All sources of data shown in this paper have been tested in terms of quality and coverage. Two problems have been identified and corrected: coverage, which has been determined and adjusted by the method of extinct generations of Bennett and Horiuchi (1981, 1984) and aged unreported deaths, which have been incorporated into the data by the proportional weights adjustment of the observed structure (Shryock and Siegel, 1980).

## **2. EPIDEMIOLOGICAL TRANSITION**

The main premise of the classical theory of epidemiological transition is that mortality is a key factor in the cycles of growth and decline that accompany population (Omran, 1971). It focuses on how complex changes in the patterns of health and illness have resulted from their interaction with demographic determinants, economic and sociological patterns of modernization of Western countries. (Bernabeu & Robles, 2000). Thus, knowledge about the epidemiological profiles and changes serve as a source of hypotheses to construct a theory of the population, beyond the mere description and / or explanation of morbidity and mortality in a population

Changes in epidemiological patterns are associated by Omran (1971) to stages in the process of modernization: the age of pestilence and famine, age of receding pandemics and age of degenerative or produced-by-man diseases. Each of them is related with the predominance of a group of specific causes of death and specific age group population. This has a direct impact on both overall growth and structure of populations (Bolaños, 2000), perceived in increasing in life expectancy (Omran, 1971).

In general, three categories of determinants are associated with the epidemiological transition: 1) eco-biological, related to the resistance created by the people against the agents of the diseases; 2) socioeconomic, political and cultural, identified in the lifestyles, habits, hygiene and nutrition, and finally, 3) medical and public health, including preventive and curative measures for health system; and public health (Omran, 1971). These three categories give entrance to equal number transition models in order to explain variations in pace and intensity in which populations have experienced changes in mortality. These models are exemplified by the historic changes in some countries: Classical or Western (England and Wales) Model: imply mortality's decline was gradual and occurs in large part by social, economic and environmental improvements that constitute the modernization process. Accelerated Model (Japan): the decline in mortality occurs rapidly and Late model (Chile and Ceylon) in which the greatest weight in determining what is the incorporation of health and medical advances.

Many questions have been made to the theory of epidemiological transition; most of them arise from the need of multi-causal explanations on the decline in mortality. These could be grouped into four main types: The first type of questioning is related to the methods used to measure changes in mortality patterns in the world or the internal consistency of the theory, mainly done by Mackenback (1994), Riley and Alter (1990), Barret et al (1998). The second type refers to the underlying idea of evolutionary and lineal progress of societies in the theory. The models implies that each stage of the transition is more advanced and desirable than one before, giving the optimistic projection of continuous and eventual elimination of infectious diseases, when factors contributing to its manifestation are primarily anthropogenic, so it cannot be separated from the socioeconomic inequalities of contexts (Gaylin, 1997 ; Caselli et al, 2002; Frenk & Col, 1991a).

The third types of questions here exposed go to the comparability of causes of death. (Mackenback, 1994). Finally, Omran in its initial proposal estimated that life expectancy marks the point of convergence between different populations, he provide 75 years of life expectancy as a stop, later extended by Olshansky to 85 years. In recent years, the idea of the limits of life on a specific age has been questioned by authors like Carey and Judge (2001 ) Oeppen and Vaupel (2002), who bet because they actually limits cannot be set.

Based on the criticisms and objections to the theory of epidemiological transition, as well as revisions and adaptations of it to different contexts, more comprehensive explanations led to incorporate cultural and behavioral elements of the population into the analysis on determinants of mortality, allowing contextualize differentials found in terms of race, ethnicity, religion, gender, geographic area, etc. Among these there is the health transition (Caldwell, 1993; Caselli et al, 2002 ;Vallin and Meslé, 2004), the transition risk (Smith, 1990) and the transition of care (Frenk et al, 1991). With these terms is referred the recognition of a far more

complex reality than the simple evolution of the epidemiological dynamics. The analytical frameworks presented do not differ in content but in the inclusion of its determinants.

Seeking to understand changes in Latin America epidemiological profile, Frenk et al (1991) advanced in a theoretical proposal that synthesizes various transition models presented above and generated a suitable analytical framework to explain how mortality has declined in the region, its determinants and changes in morbidity and mortality patterns. They suggested that Latin America epidemiological transition has been carried on under a prolonged-polarized model characterized by four aspects:

- An overlap of stages described by Omran in his epidemiological transition model. It refers to the simultaneous incidence and importance in proportion of occurrence of diseases identified as belonging to different stages of modernization process.

- Against-transition implies the reappearance of already controlled infectious diseases such as malaria, dengue and cholera.

- Prolonged-transition, tells about a situation of no-clear resolution of the transition process, continued stagnation in the duality or overlapping of epidemiological profiles.

- Epidemiological Polarization: refers at the recognition of the heterogeneity aggregated at the national level. The existing social and economic inequalities in the region translate themselves into different epidemiological profiles. Overlapping stages happens because poor urban strata and rural area maintain a different epidemiological profile when compared to the rest of the country. This distinction is what generates greater epidemiological polarization (Frenk & al, 1991b). Due to historical inequities on the development of the region, chances are that the first improvements occur in the most advantaged groups, increasing even more the existing differentials in mortality patterns. The bet is that these spreads tend to converge when all population may access to benefits either via social reforms, behavioral changes, health policy, etc. (Vallin and Meslé, 2004).

### **3. MORTALITY IN LATIN AMERICA**

During twentieth century in Latin America a significant decrease in mortality was recorded. This decrease was characterized by being reversible, discontinuous and dissimilar to those presented in developed countries patterns (Palloni, 1981). There is a consensus among experts to ensure that the impact of economic development on the decline in mortality in Latin America was relevant only until the early twentieth century. From 1930's when the decline is accentuated, is the application of sanitary controls based on basic notions of personal hygiene and incorporation of medical advances that have been identified as major determinants in this process (Arriaga and Davis, 1969; Preston 1976), especially in the second half of the twentieth century, when most countries applied health policies as mass vaccination campaigns and elimination of disease vectors, antibiotic distribution and large-scale construction of water system and excreta (Palloni, 2011).

Latin America as a whole is characterized by a diversity of contexts and implications behind the decline in mortality, this diversity is noted not only in levels, but also on the causes of death. for

example to the late twentieth century while in some countries there was still prevalence of infectious and parasitic illness and causes of deaths (Guatemala, Bolivia, Haití, Perú), others were dominated by chronic degenerative (Argentina, Chile, Cuba, Uruguay; Costa Rica) or external deaths (Brazil, Colombia, Ecuador, Venezuela); the internal situation of the country is not far from the regional feature (Di Cessare , 2011). In Brazil, for example, both economic growth and the application of sanitary controls have been decisive in mortality. The dominance of one over another will depend on the internal region studied (Prata, 1992).

The most significant differential found in the behavior of demographic variables in the second half of the twentieth century for Latin America has been attributed to three types of inequalities (1) those introduced by the high concentration of goods and services in cities, which has left in poverty rural areas; (2) how the effects of a rapid and unplanned urbanization, has led to a concentration of poverty belts within cities, generating a large number of marginalized urban population; (3) Finally, the segregation of population according to ethnic criteria, the most affected indigenous peoples. (Schkolnik and Chackiel, 1997).

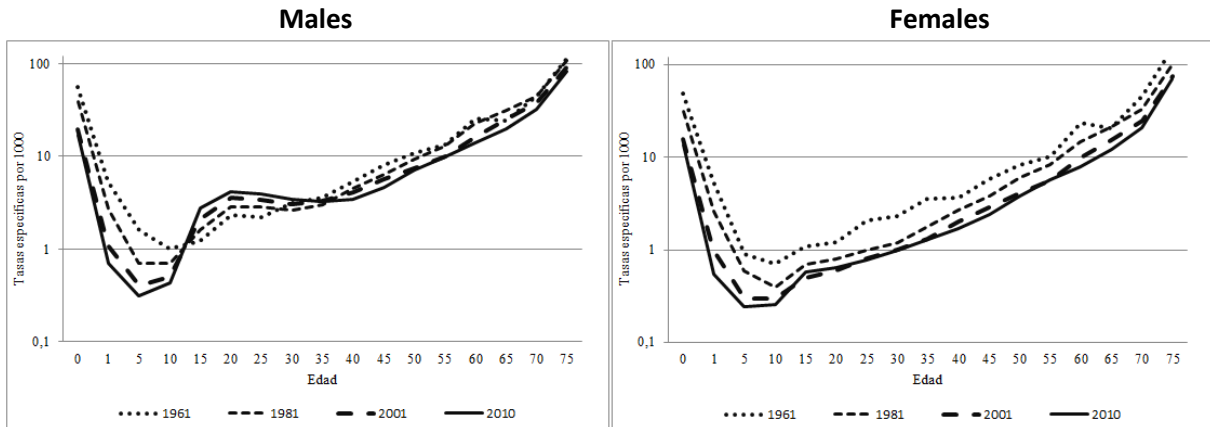
Venezuela as most of the Latin American countries has introduced accelerated demographic changes in the twentieth century. The economic and social transformations experienced marked guidelines to get better living standards. Traditional mortality indicators: life expectancy, infant mortality, maternal mortality rates, etc show significant improvements. However, acceleration in the reduction of the rates when approaches at lower levels has stopped. This slowdown in improving indicators in recent years is linked by many authors to historical inequities in the country. The diversity of internal scenarios, question not only the linear conception of a demographic and epidemiological transition nationwide (Schkolnik and Chackiel; 1997; Freitez, 2003), but also called for consideration of analysis incorporating existing internal differentials.

Changes in patterns of fertility and mortality, responsible for population growth have led to the Venezuelan population to multiply 5.8 times, from 5 to nearly 29 million in the last 60 years. Historical crude death rates fell in total 60% since the 60s until 2010, moving from 7.6 to 4.5 per 1,000 habitants. The first gains in life expectancy were concentrated in the male population, men and women having almost the same number of years of life expectancy at birth during the first half of the twentieth century (UNFPA, 2006). However, by 2010, women maintain a gain of 6 years of life expectancy at birth compared to men. This increase in life expectancy has been strongly influenced by the reduction of infant mortality (Romero, 1999).

Notable is the reduction in specific mortality rates for all age groups over time, especially in women. The age group 15-34 years is the group with the greatest difference between sexes, even from the structure presented in 1961; it captures significant male over-mortality, which is increasing due to external causes up to nowadays. During the sixties, the decline in mortality is concentrated in deaths related to infectious and parasitic diseases such as gastroenteritis, malaria, tuberculosis and malaria. Hereinafter are losing weight in total and start being displaced by those infectious and parasitic associated with postneonatal mortality, such as diarrhea, and enteritis. It is from the seventies that begin to manifest a transition in the epidemiological profile of infectious and parasitic to the growing importance of chronic degenerative cardiovascular causes. Cancer was also increasing within the structure of causes of

deaths, together with cerebrovascular diseases, diabetes and external causes complete the top five main causes of deaths since the eighties until the 2000s. (Avilan, 1998).

**Fig. 1. Venezuela. Observed Specific Mortality Rates \*\*, 1961, 1981, 2001 y 2010\*.**



Source: Freitez, A. (2003). The demographic situation in Venezuela at the beginning of the third millennium. *Revista Temas de Coyuntura* No. 47. pp. 46-92; Andrés Bello Catholic University; Institute of Economic and Social Research, Caracas, Venezuela. \* Calculations based yearbook MPPS Mortality and population projections INE, 2010. \*\* The observed rates are calculated with data from the MPPS and are presented in base 10 logarithmic scale for comparison.

In this specific context, three hypotheses related to the prolonged -polarized epidemiological transition model want to be tested in Venezuelan cause of death structure. The first hypothesis is that *epidemiological profile in Venezuela will be defined by an overlap of causes related to different phases described in the classical theory of transition*, ie both diseases chronic degenerative and infectious and parasitic, while external causes mark a significant difference between sexes. This overlap in defined stages in the classical model of epidemiological transition is given by the historical inequities. In Venezuela case, "rurality" is the variable that has historically discriminated against the living conditions of the population (Colmenares, 2005). It is in cities that have greater employment opportunities, access to education, basic services and a public health system. Therefore, the possibility of constructing homogeneous territorial conglomerates considering the urban bias could capture the structural living conditions of the population. In this sense, the second hypothesis is the *Epidemiological polarization referring to overlapping stages in Venezuela is because the rural area maintaining a distinct epidemiological profile* so that overlapping stages in the epidemiological profile of Venezuela as a whole, dissolves when urban bias is considered; since clustering territories according to urbanization level will separated two completely different epidemiological profiles. Finally, the third hypotheses points that overlapping of stages has occurred for a long duration, being able to identify *stagnation in the duality of epidemiological profiles*.

#### 4. DATA AND METHODS

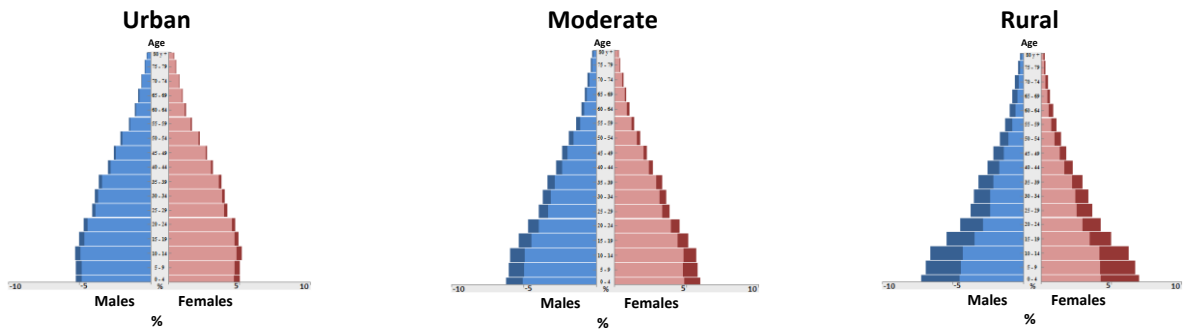
To study the structure of causes of death in Venezuela an analysis of standardized mortality rates (SMR\*) of the population aged 5 and older by specific groups causes of death will be

performed. Deaths were grouped according to their etiological similarity, using gross sections of the International Classification of Diseases in its tenth revision (ICD-10). 14 groups of causes of death are presented: Infectious and parasitic diseases (4.8%), Anemia and nutritional deficiencies (0.8%), Maternal, Heart (22.6%), Strokes (7.5%), respiratory (5.6%), Digestive and genitourinary (4.4%), accidents (7.4%), Violent (13%), Cancer (16.2%), diabetes (6.2%), genetic abnormalities (1.7%), other causes and Unknown (0.5%).

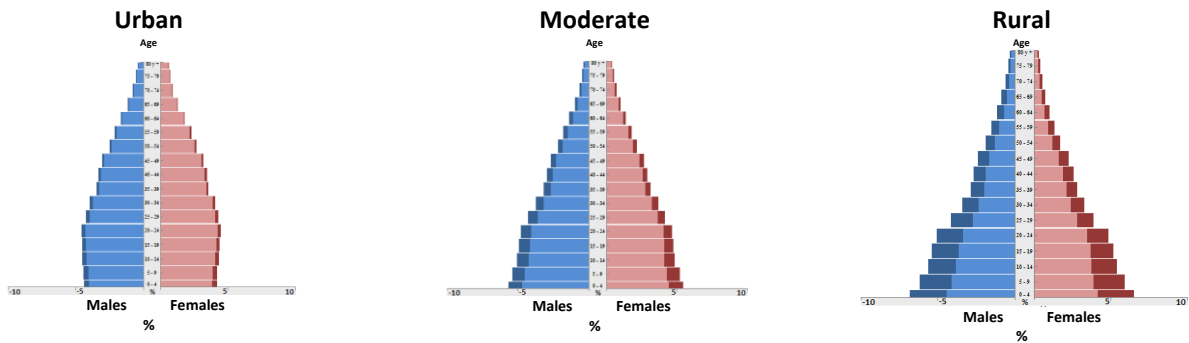
Moreover, spatial scale is considered in federal states. In Venezuela, there are 24 federal states; in this case they have been grouped according to the proportion of the urban population presented in 2001. These groups are not necessarily homogeneous, actually within the group of states considered urban, rural populations are also found in the same way as in the group of rural, urban population is contained. Selection is made in terms of predominance, due to not be possible split cities and pure rural areas because of the way that mortality database is constructed.

**Fig. 2. States groups, 2001 and 2010. Population structure**

**2001:**



**2010:**



Urban      Rural

Other important element to be considered is how medical resources are distributed, even when in all groups is possible to find an important proportion of urban population, the distribution of medical personal and infrastructure is bigger when it comes to the urban group as it can be seen in table 1.



**Table 1. Venezuela, 2003. Health resources distribution\***

Groups	first attention centers	Hospitals	physicians
Urbans	8,1	0,90	2,2
Moderates	20,5	1,36	1,6
Rurals	42,3	0,22	1,3
<b>Venezuela</b>	<b>15,89</b>	<b>1,08</b>	<b>1,95</b>

Source: Venezuelan National Statistic Institute, 2005. <http://www.ine.gov.ve/> . \*Indicators by 100.000 inhabitants

#### 4.1 Data: Quality and coverage

The data source used in this paper comes from mortality registration published by the Venezuela Ministry of Health; the institution is responsible for issuing death certificates when death occurred. The quality of the information contained in this data has been evaluated through basic indicators; the results are shown below in Table 1.

**Table 2. Venezuela and states groups. Quality assessment information, 2000-2010.**

<b>Males</b>										
<b>States Groups</b>	<b>Myers</b>		<b>Whipple</b>		<b>Overestimated Age</b>		<b>%Unknown Age</b>		<b>% Ill-defined causes</b>	
	min	max	min	max	min	max	min	max	min	max
Urban	-0,7	1,1	102,3	107,4	4,6	5,9	0,02	0,1	0,3	0,5
Moderate	-0,8	1,4	99,1	104,5	5,2	7,0	0,05	0,1	0,2	0,6
Rural	-1,1	1,9	102,4	112,4	5,5	7,9	0,06	0,3	0,9	0,2
<b>Venezuela</b>	<b>-0,6</b>	<b>1,3</b>	<b>101</b>	<b>105,8</b>	<b>4,9</b>	<b>6,6</b>	<b>0,04</b>	<b>0,23</b>	<b>0,41</b>	<b>0,61</b>

<b>Females</b>										
<b>States Groups</b>	<b>Myers</b>		<b>Whipple</b>		<b>Overestimated Age</b>		<b>%Unknown Age</b>		<b>% Ill-defined causes</b>	
	min	max	min	max	min	max	min	max	min	max
Urban	-0,5	0,8	98,8	104,3	9,4	12,5	0,01	0,05	0,3	0,1
Moderate	-0,6	0,9	98,6	105,9	8,8	11,9	0,03	0,07	0,3	0,1
Rural	-1,3	1,6	92,7	110,8	9,4	13,3	0,03	0,09	0,1	0,2
<b>Venezuela</b>	<b>-0,7</b>	<b>1,3</b>	<b>99,6</b>	<b>104,9</b>	<b>9,1</b>	<b>12,5</b>	<b>0,01</b>	<b>0,07</b>	<b>0,13</b>	<b>0,19</b>

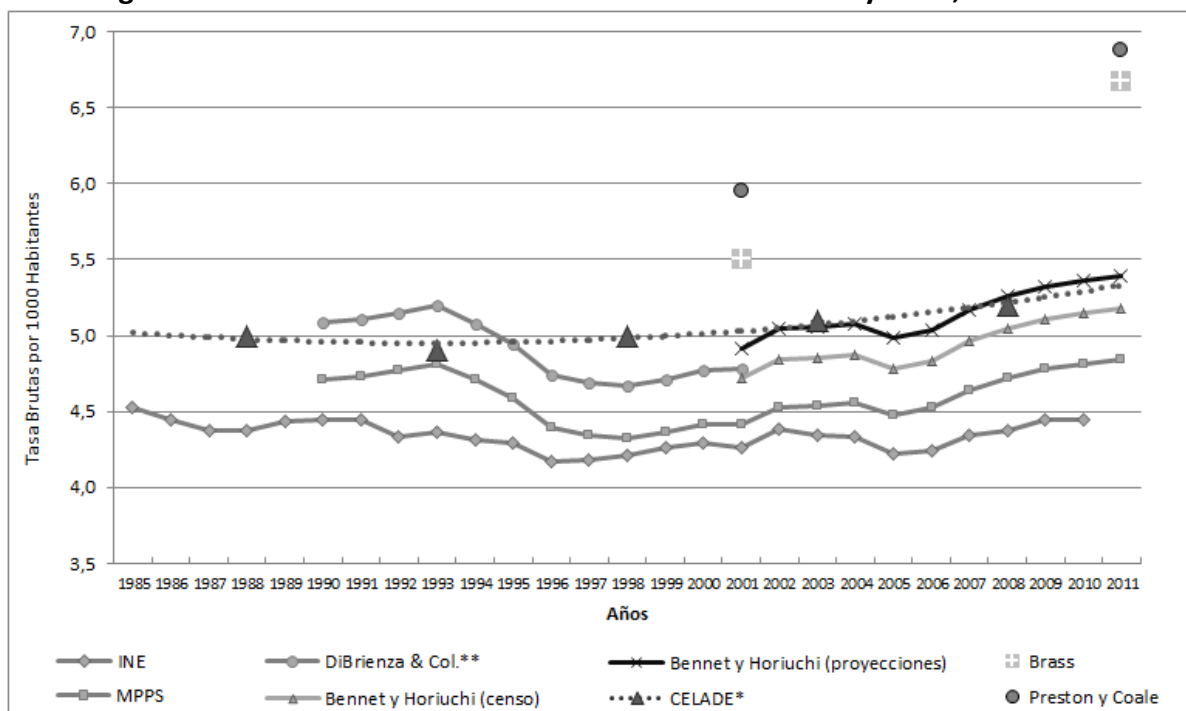
<b>Acceptable Limits</b>	<b>-5</b>	<b>5</b>	<b>100</b>	<b>110</b>	<b>M: 4,5</b>					
					<b>F: 6,5</b>					

Quality indicator seems to be inside acceptable range for performing analysis. Only adjusted in unknown age were taking, through proportional distribution, keeping the same structure presented for the known ones.

Furthermore, coverage errors found were determined and corrected through the Synthetic Extinct Generations method proposed by Bennett and Horiuchi (1981, 1984), which has already

noted Palloni and Aguirre-Pinto, is the method that produces fewer errors when the scenarios found in Latin America and the Caribbean for the period 1950-2000, (Palloni and Aguirre-Pinto, 2011); in this case United Nation population estimation are the basis for calculating rates. This correction method has been applied differently for men and women (Annexes 1) in each state group, also at national scale. Results obtained for total rates at national level are shown (see figure 3). The estimated crude rate corrected by Bennett and Horiuchi (1981, 1984) variant are the closest to those estimated by United Nations at national level (Celade, 2007), when compared with the results given by other correction methods used.

**Fig. 3. Venezuela. Observed and estimated Crude mortality rates,1985-2011.**



Source: Mortality data from the yearbook of the Ministry Health, Census of Population and Housing 2001- 2011 official population projections 2000-2020 National Institute of Statistics \* Gross five year projected rates of 1950-2050 CELADE. \*\* Gross rate corrected based on the estimated percentage of coverage Di Brienza, Freitez and Leon, 2004.

## 4.2 METHODS

The analysis in this paper follows the techniques used by Preston (1976) in the evaluation of the structure and changes in causes of death worldwide. In his analysis, he applied linear regression models to establish the contribution that specific causes of death are having to the total rate in different region and countries in the world, splitting them through inclusion on the model of a dummy variable when specific region or country want to be show. Preston (1976) also uses Partial correlations coefficients to measure possible changes over time in the contribution of specifics causes of death to the total rate. While the approach made by Preston (1976) did not aim the testing of polarized-prolonged model characteristics proposed by Frenk et al (1991),

construction and exhibition of his analysis seems to fit technical and methodological requirements.

Three hypotheses are tested on; the first refers to *epidemiological profile in Venezuela will be defined by an **overlap** of causes related to different phases described in the classical theory of transition*. To test this hypothesis bi-correlation coefficients separated by gender, between each specific causes and the combined total deaths are used. With the intention of determining, whether there is any association, these last ones are accompanied of simple regression coefficients.

Through the simple linear regression coefficients adjusted by the least squares method for each set of causes, the proportional contribution that each level has the total mortality in the case of remaining intact other causes is estimated. Thus, the sum of the intercept ( $a_{sp}$ ) of the equations obtained announces the starting point, while the sum of the slope ( $b_{sp}$ ) gives the expected change of all specific causes rates ( $r_{sp}$ ) for each variable unit of the total rate ( $r_{total}$ ). The results of the coefficient  $b_{sp}$  allow examination of the overlapping stages of classical epidemiological transition, as it marks off if exist a concentration in a significant proportion in causes of deaths identified as predominant in different stages.

The second hypothesis is provided *Epidemiological **polarization** referring to overlapping stages in Venezuela is because the rural area maintaining a distinct epidemiological profile*. Starting from this, it seeks to determine whether there are variations in the estimated model initially associated with the group of entities, such that the overlap is dissolved when urban bias is distinguished. This implies a transformation of the first model, it goes from

$$r_{sp} = a_{sp} + b_{sp} * r_{total} \quad (1)$$

to

$$r_{sp} = a_{sp} + b_{sp} * r_{total} + c_{sp} * dummy \quad (2)$$

When dummy is each states group

Coefficient  $c_{sp}$  in this model shows how average rates of death from a specific cause in a particular group of state manifest above or below specific rates of these causes throughout the country once controlled total mortality rate. If the ratio of the specific causes to the rate of all causes combined stays relatively the same behavior regardless of geographical area referred to the hypothesis of polarization cannot be maintained.

Finally, a third hypotheses is associated with the prolongation of the above polarization, indicated by the third characteristic Frenk et al (1991) as the transitional own model for Latin America. There is ***Stagnation** in the overlap of profiles shown by causes of death in Venezuela*. To test this hypothesis is considered to calculate time variations of the causes of death structure, through estimating partial correlations. A variable "time" is constructed using year of reference of data. If the correlation is strong even after controlling for the total rate and dummy

identification of states groups: extension of overlapping stages itself is rejected, since changes could be explained by rearrangement of a transitional process with mobility.

## 5. RESULTS

Clustering of causes and states conducted result in 660 standardized rates, between causes of death and its total for each sex, for 4 geographical scenarios (3 states groups and national level) in 11 years (44 possible levels of total mortality), to test the hypotheses in the polarized-prolonged epidemiological transition model. Results are presented in subsections according to the hypotheses tested.

### 5.1 Epidemiological profile in Venezuela will be defined by an overlap of causes related to different phases described in the classical theory of transition

The linear relationship between the specific causes of death and the resulting total level of the combination of all causes is assumed to create statistics generalizations to account for the contribution of each cause considered the total. The results are presented in Table 3.

**Table 3. Venezuela. Correlation coefficients of total standardized rates for each of the causes, 2000-2010**

Causes of Death	Bi-Variables Correlations		Regression Parameters					
	Males	Females	Males			Females		
			a	b	Standard Error b	a	b	Standard Error b
			Intercepto	pendiente		Intercepto	pendiente	
<b>Infectious</b>	.634**	.907**	0.123	0.024	0.015	-0.063	0.056	0.004
<b>Nutritional</b>	.654**	.899**	-0.078	0.018	0.005	-0.047	0.019	0.001
<b>Maternal</b>	-	.823**	-	-	-	-0.372	<b>0,133</b>	0.014
<b>Heart</b>	.969**	.950**	-0.111	<b>0,257</b>	0.071	-0.014	<b>0,264</b>	0.013
<b>Strokes</b>	.932**	.949**	0.114	0.052	0.027	0.019	0.096	0.005
<b>Respiratory</b>	-.716**	.673**	0.462	-0.026	0.013	0.117	0.032	0.005
<b>Digestive</b>	-0.272	.390**	0.058	0.042	0.020	0.062	0.026	0.009
<b>Accidents</b>	.860**	.863**	-2.989	<b>0,586</b>	0.105	-0.120	0.075	0.007
<b>Violents</b>	0.199	.114	-0.297	<b>0,240</b>	0.183	0.098	0.006	0.007
<b>Cancer</b>	-.741**	.955**	1.706	<b>-0,131</b>	0.028	0.401	<b>0,114</b>	0.005
<b>Diabetes</b>	-.387**	.639**	0.708	-0.062	0.020	0.111	0.056	0.010
<b>Genetics</b>	-0.104	-.018	0.032	-0.002	0.001	0.017	0.000	0.001
<b>others</b>	.292	.474**	0.662	-0.066	0.051	-0.066	0.078	0.022
<b>Unknown</b>	.797**	.818**	-0.390	0.069	0.015	-0.143	0.045	0.005

\*Correlation is significant at the 0.05 level (bilateral). \*\* Correlation is significant at the 0.01 level (bilateral).

For women, all relationships between specific causes and total are positives; it means that no cause decrease with increasing level of all causes combined. In general, among these three groups of causes were identified from the results found:

1) It includes chronic degenerative causes: heart diseases, strokes and cancer, with the highest correlation above 0.949 with all causes combined. This group is the largest contributor to changes in the level of mortality, 47.4% in each unit of total mortality.

2) Infectious and parasitic causes group: anemia and nutritional deficiencies, maternal causes, accidents and unknown. 32.8% of the change in the total rate is explained by this group of causes, also has a high correlation with total causes, though not as high as the first group (0.823 to 0.907).

3) Finally, in the third, the rest of the causes whose correlation is less than 0.823; this accounts for 19.81% of the variation on the overall level of mortality: in it are respiratory, intestinal, violent causes, diabetes, genetic abnormalities and other causes.

Roughly, about 50% of the variation in the total rate is characterized by degenerative diseases, non-transmissible (36% circulatory system and cancer 11.4%), and 50% is due to communicable diseases and external. Now, more accurately is to note the importance of maternal mortality compared to the other causes. Its occurrence contributes to variation of the total death rate, 13.3%, which is almost half the total represented by the second group of causes.

For men, the highest correlations are associated with the circulatory system (heart and stroke) and accidents. Meanwhile, considering the regression coefficients, three large groups of causes are identified also. In the first group are taken those with the greatest contribution to the change in total, certainly in the case of men external causes have a huge contribution. These account for 82.6% of the positive variation of each bound of the total (58.6% accidents and violent 24.0%).

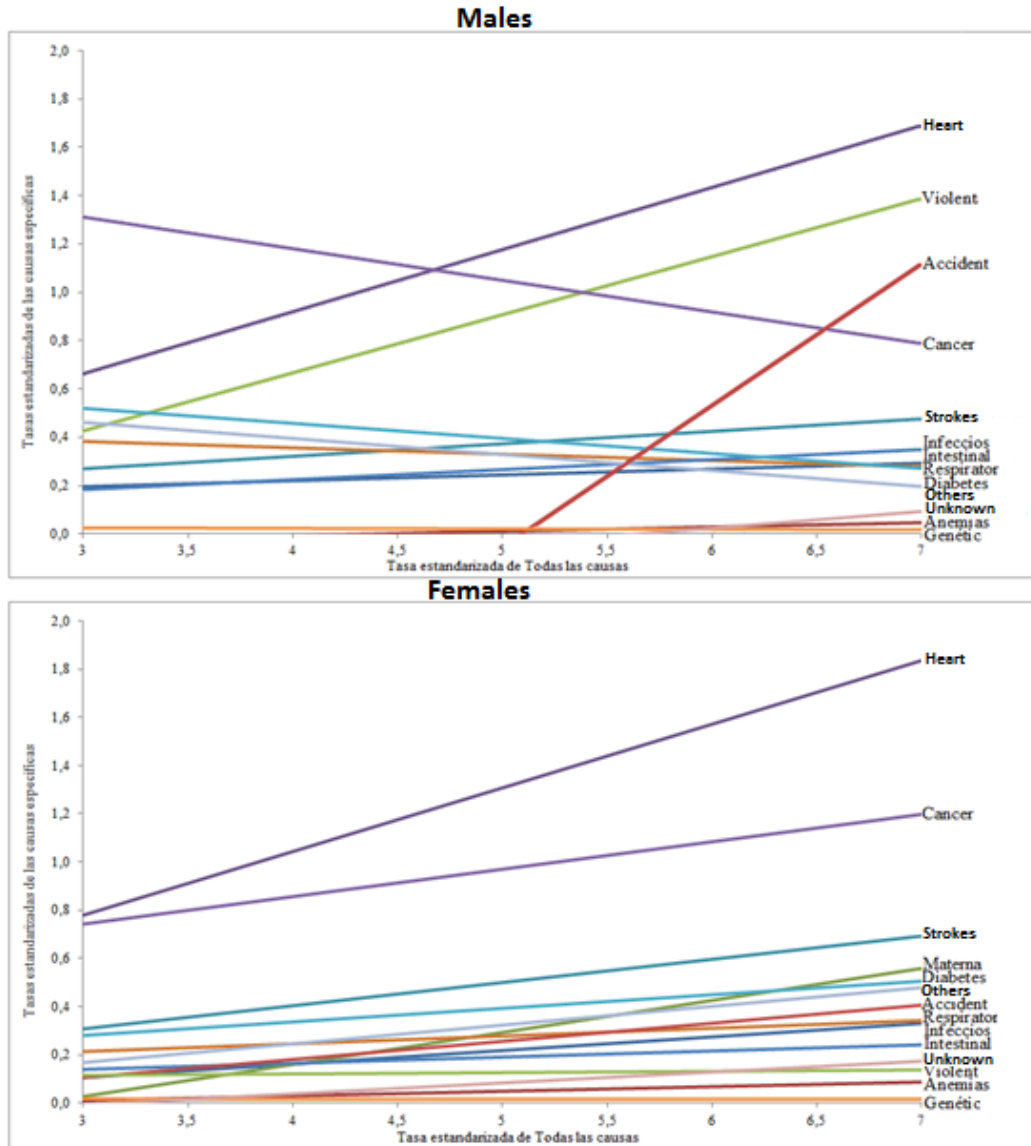
Once the group of external is separated, causes organize in opposition. One increases each time the total rate increases and others decrease. In this way a gross group of causes are taken to introduce the explanation. It is recalled that the slope coefficient  $b_{sp}$  shows the variation in each unit in overall rate occurred due to a certain specific cause, assuming that all other causes remain the same. So good, the negative force that accompanies a group of causes in itself does lower overall level while other causes not acting in the same fixed levels exposed. Is for this reason that the sum of the coefficients is not absolute number and takes into account the positive or negative slope trend. Once clarified, infectious and parasitic diseases, anemia, cardiac, cerebrovascular, intestinal and unknown causes are identified as the second group. These are responsible for 46.2% of the increase per unit of the total rate. Finally, the third leading causes respiratory component, cancer, diabetes, genetic abnormalities and other factors that have a negative relationship to varying degrees totaling 28.5% of the total rate.

We prove here the first hypothesis of this work. Indeed, it is possible to speak about the existence of overlapping causes of death identified in different stages of classical epidemiological transition, ie, the simultaneous incidence of infectious and parasitic diseases, maternal diseases and anemia with chronic degenerative causes for both sexes and different levels of mortality from all causes combined.

It simulates below (Figure 4) the behavior of all specific causes of the estimated linear regression for different levels of mortality found in Venezuela in 2000-2010 coefficients.

Obviously none of the analyzed populations meet this exact distribution of causes. Its presentation is used to facilitate understanding of the contribution of causes at different levels of total mortality by sex.

**Fig 4. Venezuela. Linear regression of total standardized rates and specific causes, 2000-2010.**



**5.2 Epidemiological polarization referring to overlapping stages in Venezuela is because rural areas maintaining a distinct epidemiological profile.**

To determine whether there are variations in the estimated model of the structure of causes of deaths associated with states groups, the linear regression model previously performed between each specific cause of death and total causes by sex is repeated, this time adding a variable dummy for each group.

Coefficients obtained c\_sp shown have acceptable levels of significance, estimated at 0.05 (see Table 3) for each group of states. 84 coefficients of linear regressions, of which 63% have acceptable levels of significance. In 53 of the 84 coefficients gotten, states groups from which data has significant value in predicting specific cause of death rates. From these coefficients with significance, 45% are in rural states group.

The spatial consideration becomes more significant in those causes of death that have greater weight within the structure, violent causes, accidents, diabetes, heart disease and stroke. Rates of violent causes are the only -controlling total mortality- rate for which all group of states for both sexes become important, especially in the case of men in which the weight of c\_sp coefficient is particularly high in urban states (above) and rural (below average rates from violence for all populations).

C\_sp coefficients for men indicate groups of opposite causes, according to the criteria of the classical theory of epidemiological transition in the structure of causes of deaths among rural and moderate states, with urban states. Besides the main causes, other causes when acquiring significance in a group does not have it in the other. Causes of death structure in urban states groups seem differ from the rest of the country population. Thus, infectious and parasitic diseases and anemia and nutritional deficiencies, and intestinal causes only become significant in rural states, which converge with that indicated by the second hypothesis for this work; being that the urban bias seems dilute overlapping profiles in male case.

**Table 4. C\_sp estimated coefficients \*\* and Significance of dummy variable in the regression states groups and sex \*, 2000-2010**

Causes of Death	Male			Female		
	Urban	Moderate	Rural	Urban	Moderate	Rural
<b>Infectious</b>			0,028	0,015	-0,014	0,016
			0.00097134	0.00168592	0.00012860	0.01179860
<b>Nutritional</b>			0,010	0,008	-0,007	0,007
			0.00037618	0.00000000	0.00000000	0.00085983
<b>Maternal</b>						0,112
						0.00000001
<b>Heart</b>	-0,155	0,114	0,145		0,037	-0,101
	0.00000029	0.00171842	0.00040217		0.00305051	0.00000008
<b>Strokes</b>	-0,055	0,028	0,070		0,015	-0,037
	0.00000224	0.04931629	0.00000114		0.00077651	0.00000010
<b>Respiratory</b>	0,015		-0,050	-0,024	0,018	
	0.02093885		0.00000000	0.00008113	0.00022922	
<b>Digestive</b>			-0,037	-0,023		0,057
			0.00202702	0.03829338		0.00004802
<b>Accidents</b>	-0,233	0,154	0,335	-0,019		0,029
	0.00000022	0.00499771	0.00000000	0.01773571		0.00624367
<b>Violents</b>	0,414	-0,241	-0,468	0,041	-0,035	0,044
	0.00000008	0.01225098	0.00000326	0.00000020	0.00000000	0.00009586
<b>Cancer</b>	0,040		-0,098			
	0.00221759		0.00000000			
<b>Diabetes</b>		0,038	-0,054	-0,051	0,041	-0,035
		0.00010289	0.00000034	0.00001210	0.00000623	0.03909711
<b>Genetics</b>						-0,004
						0.00034590
<b>others</b>		-0,056	0,065	0,078		-0,106
		0.03827322	0.03670348	0.00320639		0.00255396
<b>Unknown</b>	-0,022		0,055		-0,017	0,048
	0.00198769		0.00000000		0.00016432	0.00000000

\* All values used refer to the three-year average of the standardized rates. \*\* Significance at 0.05 Coefficients for both sexes.

As for women, rates of infectious and parasitic causes, anemia and nutritional deficiencies and diabetes have greater significance in all states. Not the same situation as in men, which seems to differ quite the contribution of the federal states. There is in the group called moderate states in which the territorial contribution adds to reverse trends in specific rates for urban and rural states. The greatest contribution in explaining maternal causes, is located in rural states, one that is not significant for the others groups. For women not seem to be enough evidence to prove the second hypothesis of polarization since overlapping epidemiological profiles fails to be resolved through urban bias.

### 5.3 There is Stagnation in the overlap of profiles shown by causes of death in Venezuela.

It is notorious that rates considered for the analysis, as before mentioned, come from the years 2000 to 2010, a relatively short period of time to witness significant changes in trend or structure of the causes of deaths. However, small changes in the contribution to the total level of specific causes may shed light on the existence of mobility in the prevalence of cases based on the identification of specific epidemiological profiles.

For calculating time variations of the structure of the causes of death, partial correlations were estimated using a variable "time" that indicates the period of time of the data in years. Correlation coefficients for sex are presented in two columns (see Table 4): At first, partial correlation is controlled by the total causes of death. In the second column, is controlled by the total mortality rate and the reference time of each states group.

**Table 5. States Groups. Partial correlation coefficients of specific causes rates, 2000-2010.**

Causes of Death	Male		Female	
	Controlling by total rate	Controlling by total rate States Group	Controlling by total rate	Controlling by total rate States Group
Infectious	-0,786	-0,829	-,156	-,138
Nutritional	-0,644	-0,664	-,254	-,244
Maternal			,612	-,066
Heart	-0,599	-0,945	-,589	-,444
Strokes	-0,494	-0,745	-,571	-,559
Respiratory	0,226	0,237	,451	,476
Digestive	0,587	0,588	,787	,162
Accidents	-0,013	0,366	,693	,036
Violents	0,537	0,933	-,021	,406
Cancer	-0,063	-0,549	,007	,432
Diabetes	0,369	0,515	,515	,700
Genetics	0,016	-0,027	-,779	-,321
others	-0,870	-0,897	-,835	-,362
Unknown	-0,199	-0,140	,510	-,220

Correlation coefficients show significant levels for infectious and parasitic diseases, anemia, heart diseases, strokes and other causes in the period considered, and are obtained even higher



once they are controlled by states groups in men case. Negative sign of coefficients in this case, does not mean they are actually decreasing over time, but their contribution to the overall rate level has decreased. This phenomenon could be explained by the increase in the total contribution of some other causes of death; and external causes for both sexes, most emphatically in men case. Correlations raised significance to the extent that is controlled by states groups, indicating that there is mobility between causes of death to the inner states group, breaking the possibility that polarization captured through urban bias is prolonged .

For women, the behavior is contrary. Correlation coefficients vary little when controlled by states groups; fading the significance of correlations occurring between causes and time variable controlled only by the total fee; no cause has any significant variation over time. Here, although polarization stages fails to be captured by urban bias, evidences seem to indicate that overlapping profiles in women it is in fact prolonged over time.

## 7. DISCUSSION

Literature has set that gaps in Venezuelan population living standards become recognizable to the extent in which is considered the urban bias as a spatial disparities frame. (Gruson, 2008). In this sense, three hypotheses have been tested derived from criticism to classical epidemiological transition made by Frenk et al (1991) involving the relationship between epidemiological profile and the urban bias in Venezuela. In the run, It has been clearly demonstrated the simultaneous occurrence of deaths separated by Omran as belonging to different stages of the transition process, identifying the existence of **overlapping** profiles at national level (accepting first hypotheses of the study). The overlapping begin to vanish when federal states with marked rural population and still in pre-transitional epidemiological profile are separated from more urban populations federal states with post-transitional patterns of mortality, or what is equal: **Epidemiological polarization**. This characteristic could be due to the historical processes that have combined development in improving just urban life quality. This polarization as demonstrated is captured more clearly in the case of men, whose behavior on specifics causes differs significantly between groups of states and homogenized internally.

Polarization in overlapping stages however, fails to be captured in the case of women by urban bias, with the exception of deaths from maternal causes. Polarization in this regard could be obeying to more conditions related to social stratum, than to geographical differentiation; it would be advisable to investigate further more this aspect. Regarding the third hypotheses in this study, about **stagnation** in the overlap of profiles, it was proof to be more a characteristic defining female profiles rather than men. Either way, it could be said that there are enough elements to agree with Frenk et al (1991) in the identification of a Polarized-prolonged model of epidemiological transition in Venezuela as a suitable framework to analyses causes of death structures. Even do it manifests differently between men and women.

However, Both processes, overlap and prolonged polarization manifested in the study, which leads to stagnation point to the overall epidemiological profile, could be influenced by the short period of time analyzed in this study, only 11 years; it would be advisable to include longer

periods as to monitor the historical changes and thus identify variables that help understanding why the conditions of polarization varies between sexes.

Furthermore, comparing results obtained for Venezuelan case with the analysis of the structure of causes of deaths made by Preston (1976) with data from different countries around the world since the late nineteenth century until 1972, seem to correspond. Preston could identify 165 possible scenarios in framing different levels of mortality, concluding that at different levels of mortality importance that specified causes of death varies. Thus, infectious and parasitic diseases and respiratory causes contribute less and less to the total level presented by all causes combined and the decline in the contribution is further accentuated as higher the level of mortality from all causes a population has. Preston established that decline in importance of this type of case, contrary to what literature on the determinants of mortality in developing countries says, does not happen as a drastic change after the Second World War, but reduced its contribution seems to be straight and continuously over time.

Additionally, mentions the historic step in the prevalence of infectious diseases to degenerative far to be reducing the level of all causes combined, making clear that the predominant type of degenerative causes in the structure will vary significantly by sex. These variations in the structure of the causes of death by sex are related by Preston (1976) by differences in urbanization processes that introduce removals in the present rates of cancer and cardiovascular disease (the two most common types of causes of death considered within degenerative category), being that cancer tends to have a greater contribution in the case of women, while for men it has heart diseases.

Even more, some similarly with results obtained by Preston (1976) are found for the first decade of the twentieth century in Venezuela. It seems identified a structure of causes of death characterized by the prevalence of diseases of the circulatory system, for women this share high level contribute to deaths associated with cancer and diabetes. This trend is intensified in urban states (which has the lowest levels of mortality from all causes). Meanwhile in rural states, there are still some vestiges of communicable and exogenous causes (of infectious and parasitic type, maternal and nutritional deficiencies) whose contribution is on par with the causes of death related to the circulatory system.

For men, the prevalence of causes of death related to the circulatory system competes with the great contribution rates for deaths from external causes concentrated in the more urban states. By contrast in the scene of the most rural states, seems to be still the promotion process causes the circulatory system to infectious, anemia and intestinal causes.

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