A Life Course Approach to Mortality in Mexico

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Introduction:

While much aging research addresses the relationship between socioeconomic status (SES) in adulthood and health, less has examined how early life risk factors including SES and education shape late life outcomes and less has done so in developing countries such as Mexico. Indeed, aging is a lifelong process which, as Kierkegaard once stated "can only be understood backwards, but...lived forwards" (Settersten 2003). Research has shown early life SES to be associated with mental (Gilman, Kawachi & Fitzmaurice et al. 2002) and cardiovascular health (Pollitt, Rose & Kaufman 2005). Also, lack of education has been demonstrated to be an important risk factor for mortality in multiple countries (Kunst & Mackenbach 1994).

Understanding how factors such as early life SES and education are patterned across the life course and influence mortality risk are important steps towards increasing life expectancy. However, it is insufficient to address these issues without regards to large scale demographic changes in Mexico. Mexico is experiencing an epidemiologic transition, evidenced by decreases in communicable disease and increases in mortality from myocardial infarctions, diabetes and hypertension (Rivera, Barquera & Campirano et al. 2002). Studies of late life mortality risk are becoming increasingly important because the Mexican population is rapidly aging (CONAPO 2005). In addition, urbanization and increases in education (Wong & Palloni 2009) have occurred in Mexico over the previous century. Given these changes, major differences may exist between successive cohorts that must be explicitly modeled.

Life course epidemiology suggests multiple frameworks connecting early life SES with late life outcomes including critical period approaches in which conditions in a specific period by themselves have lifelong effects on later outcomes. In contrast, other researchers allow risk factors to be patterned across the life course (Kuh & Ben-Shlomo 2004). Using this model, early life factors are hypothesized to predict future risk factors which predict outcomes, creating a "chain of risk." This analysis uses the latter approach by suggesting a theoretical model: early life SES will predict education which will predict late life health behaviors, SES and access to health care which will predict mortality. Given demographic changes in Mexico, factors such as education may have different relationships with mortality across cohorts. Given urbanization, younger cohorts may have greater ability to take advantage of their education leading to an increased importance of education. Alternatively, increased education may lead to more homogenous populations creating equalizing effects of education in younger cohorts.

The aim of this analysis is to determine how life course predictors of mortality (early life SES, education, health behaviors, late life SES and access to health care) are associated with mortality and patterned over the life course and to determine how these relationships differ across cohorts of older Mexicans. Understanding late life mortality risk will be increasingly important given the rapid aging of the Mexican population.

Method:

Data for this analysis comes from all waves of the Mexican Health & Aging Study (MAHS). The MHAS is a longitudinal, household based sample of older Mexicans (age 50+) and spouses with an oversample of high out migration states. The first wave of the MHAS was completed in 2001, the second in 2003 and third in 2012 providing eleven years of follow-up.

While attrition is a major concern in longitudinal studies, the MHAS had remarkably low attrition with a response rate of 88% in 2012, nine years after the previous wave (ENASEM 2013). The analysis presented in this abstract uses preliminary 2012 data and the final paper will be updated with official release data. Due to the focus on older adults in this analysis, only respondents and spouses age 50+ are kept. Respondents were eliminated if ineligible for interviews, could not determine a month of mortality, had no follow up time or had missing data on key variables. After exclusions the analytic sample size was 11,275 with 2,603 mortality events.

While traditional analyses of mortality have used vital registration, an innovative feature of this paper is the use of longitudinal data with long term follow up. This provides the opportunity to take advantage of the rich supply of detailed covariates necessary for life course research and not available in many mortality analyses. Covariates are from baseline surveys and include age, sex, rural/urban location, education, marital status, United States migration history, smoking behaviors, binge drinking, physical activity, health service access, pharmacist consultations, self reported financial standing, self-reported doctor diagnosed diabetes and hypertension, obesity and depressive symptoms. Early life SES is proxied as self reported presence of a toilet in the household prior to age 10. Education is categorized as none, 1-5 years, 6 years or 7 or more years. Late life socioeconomic status is self-reported financial standing. The sample is divided into a cohort born before 1940 and one after 1940.

All cause mortality is determined through next of kin interviews. The next of kin provides the month of mortality which is used to calculate a time to event for mortality, defined as the time in months between the respondent's baseline interview and the month of mortality. Respondents who are lost to follow up or are still alive at the end of the study are censored at their last successful interview. Time to censor is calculated as the time in months between the baseline interview and last successful interview. Data will be analyzed using Cox proportional hazard models fit with SAS 9.2.

Preliminary Results:

Preliminary results show that in both age cohorts, lower early life SES groups had less education, less access to health care and rated their current financial standing lower. In adjusted analyses of the older cohort, early life SES is not significant after accounting for age. Education was not associated with mortality. Some support is seen for the theoretical model. Lower early life SES predicted lower education which predicted lower evaluations of the respondent's current financial standing, more obesity and less access to health care but only a lower evaluation of one's financial standing was associated with mortality. In full models, higher age, being male, lower self assessed financial situation, being diabetic, being hypertensive and being a former smoker were associated with higher mortality.

In the younger cohort, more educational equality is observed. In addition, similar mortality is seen across levels of early life SES despite a higher prevalence of risk factors (lower education, less access to health care and lower self assessed current financial situation) in the lower early life SES group. Early life SES was not statistically significant until education was added to the model when a protective effect of lower early life SES emerged. This is strengthened in full models due to similar mortality across levels of early life SES despite a higher prevalence of risk factors in the lower early life SES group. Low education was associated with higher mortality. The theoretical model operated similarly in the younger cohort but, obesity showed a protective effect against mortality in the younger cohort. In full models, higher

age, being male, lower education, lower self assessed financial situation, being diabetic, being hypertensive, not being obese and being a current smoker were associated with higher mortality. Table 1 presents preliminary regression results.

Birth Cohort:	Born Before 1940				Born After 1940			
	Model 1		Model 2		Model 3		Model 4	
	<u> HR†</u>	p‡	HR	р	HR	р	HR	р
Early Life								
Low SES	1.21	***	1.11		0.94		0.77	**
Education (Ref = 7+)							
No Education			0.97				1.34	*
1-5 Years			0.95				1.33	*
6 Years			0.86				1.22	
Late Life SES (Ref =	= Poor)							
Good			0.82	*			0.80	
Fair			0.89				0.79	*

Table 1: Cox Proportional Hazard Models Predicting Mortality by Cohort in Mexican Health & Aging Study 2001-Preliminary 2012 (Select Baseline Covariates)

* denotes p<0.05, ** denotes p<0.01, *** denotes p<0.001. Models 2 and 4 account for all covariates. †HR: Hazard Ratio. ‡ p=probably of type 1 error.

Discussion:

We find similar levels of mortality across levels of early life SES in both cohorts in the preliminary data. Lower early life SES groups were disadvantaged in education but risk factors did not behave the same across cohorts. Lower education only predicted mortality in the younger cohort. This may reflect several population level phenomena. First, selective survival may explain the differences in the relationship between education and mortality. Those with lower education may be more likely to experience mortality prior to the study, leaving only the healthiest ones, explaining why education and mortality only showed associations in the younger cohort. Alternatively, differential returns to education may explain differences in the relationship between education. Relationships between education and mortality. Urbanization may provide younger cohorts with a greater opportunity to take advantage of the benefits of higher education. Relationships between education and early life SES and mortality may also differ due to the use of all cause mortality. Epidemiologic transition literature would suggest that cohorts may die of different causes. Education and early life SES may have different relationships with different causes of death leading to the differences in the relationships that were observed. Finally, respondents may die of different causes by age.

We find mixed support for the proposed theoretical model. While early life SES, education, access to health care, late life SES and obesity seem to be patterned across the life course, late life risk factors are not consistently associated with mortality. From the proposed theoretical model the only late life risk factors that emerged as significant predictors of mortality were late life SES (in both cohorts) and non-obesity (in the younger cohort). The proposed theoretical model appears to be operating the same across cohorts with the exception of obesity.

The full paper will include validation of mortality reports with secondary data sources including vital statistics as well as further work to determine why relationships differ across cohorts using official release data. The analysis may assist those interested in early life SES, education, mortality and the life course. The longitudinal nature and availability of detailed life course variables in the MHAS provide this analysis with a unique ability to answer these questions. Efforts should be made to increase educational opportunities to children in lower SES families and areas as lower early life SES was a major predictor of educational disadvantage.

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