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Education and Male-Female Differences in Later-Life Cognition: International Evidence From Latin America and the Caribbean

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Abstract This study explores the role of early-life education for differences in cognitive functioning between men and women aged 60 and older from seven major urban areas in Latin America and the Caribbean. After documenting statistically significant differences in cognitive functioning between men and women for six of the seven study sites, I assess the extent to which these differences can be explained by prevailing male-female differences in education. I decompose predicted male-female differences in cognitive functioning based on various statistical models for later-life cognition and find robust evidence that male-female differences in education are a major driving force behind cognitive functioning differences between older men and women. This study therefore suggests that early-life differences in educational attainment between boys and girls during childhood have a lasting impact on gender inequity in cognitive functioning at older ages. Increases in educational attainment and the closing of the gender gap in education in many countries in Latin America and the Caribbean may thus result in both higher levels and a more gender-equitable distribution of later-life cognition among the future elderly in those countries.

Keywords Education · Later-life cognition · Male-female differences · Oaxaca-Blinder decomposition · Latin America and the Caribbean

Introduction

Cognitive capacity is an important determinant of functioning and well-being because it affects various aspects of life, including leisure time activities (Schooler and Mulatu 2001), financial planning for retirement (Banks and Oldfield 2007), and

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adherence to complex medical treatments (Hinkin et al. 2002). Ensuring high levels of cognitive functioning throughout the life course is, therefore, a key goal of human development.

Previous research has shown that socioeconomic factors, most notably education, display a large positive association with later-life cognition in a number of international settings (Cagney and Lauderdale 2002; Lee et al. 2003; Yount 2008), including Latin America and the Caribbean (Tuong Nguyen et al. 2008). Yet, with the exception of Yount (2008), who studied gender differences in cognition between older men and women in Ismailia, Egypt, there is little empirical evidence on the role of differential access to education during childhood for gender disparities in later-life cognition.

The strong association between education and cognition at older ages may stem from a variety of sources, including ability-based selection into education, lasting acquired knowledge during the education process, and education-induced changes in brain function that buffer the effects of neuropathology and thus help maintain higher levels of cognition (Alley et al. 2007; Diamond 1988; Richards and Deary 2005). The pathways linking education and later-life cognition are commonly analyzed using the concepts of passive and active cognitive reserve (Stern 2002). Education is thereby instrumental in avoiding functional impairment, either through higher initial levels of cognition relative to clinical manifestation of impairment (passive models) or through improved compensatory use of brain networks, which are less (or not at all) susceptible to disruption (active models). In these neuropsychological models, a positive association between education and larger levels of reserve thus represents a combination of both causal and noncausal mechanisms (Richards and Sacker 2003; Richards et al. 2004).¹ Over and above any direct effects of education on later-life cognition, some of the beneficial cognitive effects of education may be indirect, mediated through its relationship with other cognitive resources through the life course, such as occupation, health, or lifestyle factors (Christensen et al. 1994, 1996; Finkel et al. 2009; Tuong Nguyen et al. 2008).

Both theory and empirical evidence regarding the link between education and later-life cognition suggests that historically prevailing gender differences in access to education play a key role in explaining gender disparities in later-life cognition. Consistent with this hypothesis, Yount (2008) showed that older men in Ismailia have higher levels of cognitive functioning than older women, with differences in education explaining most of the estimated cognition gap.

In this article, I examine the robustness of Yount's findings across seven urban areas in Latin America and the Caribbean. In doing so, I complement previous research of Tuong Nguyen et al. (2008) on the life-course determinants of later-life cognition in Latin America and the Caribbean by analyzing the implications of gender differences in resource allocations over the life course for potential gender differences in later-life cognition, highlighting the prominent role of education in this relationship.

¹ See also Yount (2008) for a more extensive description of a conceptual model relating differential life-course resource allocation between men and women to gender differences in later-life cognition using the concept of cognitive reserve.

Data

I use data from the Survey on Health, Well-being, and Aging in Latin America and the Caribbean, 2000 (SABE), which gathers information on numerous aspects of health and aging for representative samples of persons aged 60 and older residing in private households in Bridgetown (Barbados), Buenos Aires (Argentina), Mexico City (Mexico), Havana (Cuba), Montevideo (Uruguay), Santiago (Chile), and São Paulo (Brazil) (Pelaez et al. 2005).² SABE interviews were conducted between winter 1999 and fall 2000 in the official language of each country. The seven urban samples were selected using multistage sampling methods based on existing household surveys, census data, or national election registries, with some survey sites oversampling respondents aged 75 and older or 80 and older to ensure sufficient sample representation of the oldest-old. Response rates across study sites ranged from 62.5% (Buenos Aires) to 95.3% (Havana). All data used in the analyses were weighted to be representative of the underlying study populations using sampling weights that account for site-specific aspects of the sampling process as well as nonresponse by primary sampling units.

SABE administers a cognitive evaluation of its respondents using a reformulated and abbreviated version of Folstein's Mini-mental state examination (MMSE) as part of its mental health inventory (Folstein et al. 1975; Pelaez et al. 2005). This 19-item MMSE has been specially designed to minimize low-literacy bias and covers a number of dimensions of cognition, including orientation, memory, and executive functioning.³ With the exception of persons entirely unfit for interviewing, the MMSE was administered to all respondents at an early stage of the interview, which continued to rely on proxy respondents for persons with cognitive impairments as revealed by the MMSE.⁴

The MMSE scores take on values between 0 and 19, but their distributions are heavily left-skewed because of a large fraction of observations bunching at the maximal attainable score of 19. Such skewness is commonly encountered in survey-based cognitive assessments, which typically aim at identifying cognitively impaired respondents rather than providing a general-purpose measure of cognitive function and thus provide more differentiation at lower levels of cognition than at the upper tail of its distribution.

² The data from Mexico City also include an additional sample of women aged 50 and older, which I did not use in the analysis. A more extensive description of the SABE data, including further details on sampling frames, stratification, responses rates, and sampling weights, can be found in Pelaez et al. (2005).

³ A full description of the abbreviated MMSE used in SABE, as well as details on its scoring, can be found in Pelaez et al. (2005).

⁴ For respondents with an abbreviated MMSE score of 12 or lower, a portable functional assessment questionnaire (11-item Pfeffer scale) was administered to a personal caregiver, which resulted in a functional assessment score ranging from 0 to 33, with higher values indicating lower levels of functioning (Pfeffer et al. 1982). The full interview continued to rely on caregivers as proxies if the respondents' MMSE scores were 12 or lower and the subsequent Pfeffer scores were 6 or higher (Pelaez et al. 2005). For Santiago, missing MMSE scores appear to be coded as zeros in the data file and are therefore indistinguishable from actual scores of zero in the MMSE. Yet, because actual zeros in the MMSE are very rare at the other survey sites, I treated all zeros on the MMSE in Santiago as missing values. The results on male-female differences in cognitive functioning in Santiago are, however, largely unaffected by this particular treatment of zeros in the MMSE.

Beyond the respondents' MMSE score, I also extract information on various sociodemographic and health characteristics of the respondents—including education—that are likely to directly or indirectly impact cognitive function over the life course. The most basic benchmark model (Model 1) consists of age and education only, thus highlighting the raw association between education and age-adjusted cognitive function. I then assess the robustness of these benchmark results to alternative model specifications, which sequentially add further life-course controls pertaining to different life stages. Model 2 adds controls for early-life circumstances during the first 15 years of life, which may otherwise confound the benchmark analysis. Model 3 incorporates additional controls for cognitive resources during midlife, such as demographic circumstances and occupation. The last model (Model 4) also adds controls for later-life circumstances related to current living arrangements and health status to the analysis. All models specifications and corresponding variable coding are summarized in Table 1.

The choice of controls pertaining to each life stage is motivated by epidemiological evidence on common determinants of later-life cognition.⁵ Beyond age and education, these comprise a wide range of socioeconomic and psychosocial and health risk factors, ranging from economic and health conditions during early life (Case and Paxson 2008; Tuong Nguyen et al. 2008) to measures of mid- and later-life cognitive resources and risk factors, which may partly act as mediators in the relationship between education and cognition at older ages. The specifications of Models 3 and 4 particularly recognize existing empirical evidence on the importance of occupational complexity, activities, and social support networks for continued cognitive stimulation (Finkel et al. 2009; Fratiglioni et al. 2004; Tuong Nguyen et al. 2008), as well as studies highlighting the role of specific health behaviors and health conditions for cognitive function at older ages (Kalmijn et al. 2002; Laurin et al. 2001; Lichtenberg et al. 1995; Zelinski et al. 1998).

The initial data set contained a total of 10,597 observations for all study sites. The final estimation samples exclude observations with missing MMSE scores (552 observations). With regard to missing data for control variables, I followed a strategy similar to Yount (2008) and excluded all observations with missing education information as well as observations with missing data on items with fewer than 70 missing responses, leading to an additional deletion of 353 observations and a final analytical sample of 9,692 observations. For all remaining covariates with item nonresponse, I constructed item-specific missing value dummy variables for inclusion in the statistical analyses.

Analytic Methods

I first estimate sex-specific means for the MMSE scores, latent cognition (as measured by adjusted MMSE scores that account for right-censoring in the raw MMSE scores), and years of education by study site, and assess the degree and

⁵ A full review of the evidence regarding the role of each listed control variable is clearly beyond the scope of this article. The interested reader may consult Brunner (2005), Lee et al. (2003), National Research Council (2000), and Whalley et al. (2004) and for an overview.

Table 1 Model specifications and variable coding

Variable	Model 1	Model 2	Model 3	Model 4
Dependent variable				
Abbreviated mini-mental state examination (score) (MMSE)	✓	✓	✓	✓
Control variables				
Baseline controls				
Age and age squared (in years)	✓	✓	✓	✓
Years of education (in years)	✓	✓	✓	✓
Early-life controls				
Economic situation of the family (poor vs. good/average vs. missing)		✓	✓	✓
Self-assessed health (poor vs. excellent/good vs. missing)		✓	✓	✓
Time of hunger/lack of food (yes vs. no vs. missing)		✓	✓	✓
Confined to bed for a month or more because of health (yes vs. no vs. missing)		✓	✓	✓
Rural residence during childhood (yes vs. no)		✓	✓	✓
Midlife controls				
Marital status (never married/partnered vs. ever married/partnered)			✓	✓
Age at first marriage <19 (yes vs. no vs. missing)			✓	✓
Number of children (0 vs. 1–3 vs. 4 or more)			✓	✓
Work status (never worked vs. worked)			✓	✓
Work type (manual vs. intellectual vs. mixed vs. missing)			✓	✓
Later-life controls				
Currently living alone (yes vs. no vs. missing)				✓
Currently working (yes vs. no vs. missing)				✓
Enough money to cover daily living necessities (yes vs. no vs. missing)				✓
Smoking (never vs. ever, but not now vs. now vs. missing)				✓
Drinking more than two drinks at least four times per week (yes vs. no)				✓
Exercise or physical activity at least three times per week (yes vs. no)				✓
Participation in handiwork/arts/crafts at least once per week (yes vs. no)				✓
Self-assessed health status (five-point scale: excellent-poor, missing)				✓
Self-reported doctor/nurse-diagnosed hypertension (yes vs. no)				✓
Self-reported doctor/nurse-diagnosed diabetes (yes vs. no)				✓
Self-reported doctor/nurse-diagnosed heart disease (yes vs. no)				✓
Self-reported doctor/nurse-diagnosed stroke (yes vs. no)				✓
Self-reported doctor/nurse-diagnosed psychiatric problem (yes vs. no)				✓
Yesavage geriatric depression scale (score, missing)				✓

Notes: All statistical models are formulated as Tobit models to incorporate potential ceiling effects in the abbreviated MMSE scores. In addition to the above coding scheme, I also included a missing value category for all variables featuring some item nonresponse.

statistical significance of male-female differences in each of these measures. The estimated means for latent cognition are computed based on sex- and site-specific Tobit models. These Tobit models assume that latent cognition Y^* coincides with the observed MMSE score only when it is strictly smaller than the maximal attainable MMSE score of 19. For individuals with higher levels of latent cognition—that is, $Y^* \geq 19$ —the MMSE measure does not provide further differentiation. Only the maximal attainable MMSE score ($\bar{Y} = 19$) is observed for those respondents, which can be interpreted as a lower bound for actual cognition. I then estimate a series of multivariate Tobit models for cognitive function using the specifications of Table 1, which sequentially add further life-course controls pertaining to different life stages to the statistical model.⁶

Formally, the Tobit models can be written as

$$Y_F^* = \mathbf{X}_F \beta_F + \varepsilon, \quad \varepsilon \sim N(0, \sigma_F) \tag{1}$$

$$Y_{iF} = \begin{cases} Y_{iF}^* & \text{if } Y_{iF}^* < \bar{Y} \\ \bar{Y} & \text{if } Y_{iF}^* \geq \bar{Y} \end{cases} \tag{2}$$

and

$$Y_M^* = \mathbf{X}_M \beta_M + \varepsilon, \quad \varepsilon \sim N(0, \sigma_M) \tag{3}$$

$$Y_{iM} = \begin{cases} Y_{iM}^* & \text{if } Y_{iM}^* < \bar{Y} \\ \bar{Y} & \text{if } Y_{iM}^* \geq \bar{Y} \end{cases} \tag{4}$$

for females and males, respectively. The parameters of these models are estimated by maximum likelihood. For brevity, I will report only the main coefficients of interest—that is, the adjusted sex-specific association between education and later-life cognition—thus contrasting the benchmark results with richer models containing further adjustments for cognitive resources over the life course.

Given the estimation results from these Tobit models, Oaxaca-Blinder decompositions for each latent variable model can be used to assess the extent to which education differences between men and women explain corresponding differences in latent later-life cognition (Blinder 1973; Oaxaca 1973). As in Yount (2008), these Oaxaca-Blinder decompositions allow me to attribute the predicted differences in cognitive function between men and women to male-female differences in the prevalence of the models’ explanatory variables (explained differences or endowment effects) and male-female differences in their respective association with cognition (unexplained differences or coefficient effects).

⁶ Reflecting the right-censoring of the MMSE, the adjusted MMSE score means and predicted values from corresponding Tobit models will always be larger than the unadjusted means, with the respective difference depending on the extent of censoring and the dispersion of the MMSE scores. See, for example, Wooldridge (2001) for a more detailed discussion of the Tobit approach for dealing with data censoring.

Given the parameter estimates $\hat{\beta}_F$ and $\hat{\beta}_M$ from the preceding Tobit models, I obtain predicted levels of latent cognitive functioning $\hat{Y}_M^* = \mathbf{X}_M \hat{\beta}_M$ and $\hat{Y}_F^* = \mathbf{X}_F \hat{\beta}_F$ for men and women, respectively. The mean male-female differences in predicted levels of cognition can then be formally decomposed as

$$\overline{\hat{Y}_M} - \overline{\hat{Y}_F} = \overline{\mathbf{X}_M} \hat{\beta}_M - \overline{\mathbf{X}_F} \hat{\beta}_F \tag{5}$$

$$= \underbrace{\left(\overline{\mathbf{X}_M} - \overline{\mathbf{X}_F} \right) \left(\mathbf{W} \hat{\beta}_M - (\mathbf{I} - \mathbf{W}) \hat{\beta}_F \right)}_{= \text{Explained}} + \underbrace{\left(\overline{\mathbf{X}_M} (\mathbf{I} - \mathbf{W}) + \overline{\mathbf{X}_F} \mathbf{W} \right) \left(\hat{\beta}_M - \hat{\beta}_F \right)}_{= \text{Unexplained}} \tag{6}$$

where upper bars indicate subpopulation means. \mathbf{I} denotes the identity matrix, and \mathbf{W} denotes a weighting matrix that allows the researcher to control the benchmark model of the decomposition. To conserve space, I follow Yount (2008) and report only those decompositions that pertain to the weighting matrix $\mathbf{W} = \Omega = \left(\mathbf{X}'_M \mathbf{X}_M + \mathbf{X}'_F \mathbf{X}_F \right)^{-1} \left(\mathbf{X}'_M \mathbf{X}_M \right)$, which uses the coefficients from a pooled regression based on all observations of both sexes as a reference (Neumark 1988; Oaxaca and Ransom 1994).⁷ Beyond reporting the overall extent of explained and unexplained differences in later-life cognition, I will also present the specific contributions of education to both, highlighting its special importance.⁸

Results

Table 2 presents information on sample sizes and the number of observations with censored MMSE scores ($Y=19$), as well as site- and sex-specific population means and associated sex differences for the raw MMSE scores, latent cognition (i.e., adjusted MMSE scores that account for right-censoring), and years of education.

Site-specific estimation sample sizes vary between 982 respondents for Buenos Aires and 1,904 respondents for São Paulo. All samples contain more women than men and include a considerable fraction of right-censored observations, ranging from 13.3% of all female respondents from Mexico City to 42.7% of all male respondents from Bridgetown.

Male respondents have statistically significantly higher MMSE scores than their female counterparts in all cities except Bridgetown. A similar finding also obtains for the adjusted MMSE scores. Yet, although the estimated male-female gaps in cognitive function tend to widen after adjusting for right-censoring, the resulting gender differences are not always statistically significant, mostly because of larger standard errors associated with the adjustment for right-censoring. Finally, men have higher levels of education in all seven cities, with Bridgetown being the only place where these differences are not statistically significant.

⁷ Note, though, that the decomposition results are largely robust to common choices for the benchmark model, such as the model for men ($\mathbf{W}=\mathbf{I}$), the model for women ($\mathbf{W}=0 \cdot \mathbf{I}$), and the average coefficients from the two models ($\mathbf{W}=0.5 \cdot \mathbf{I}$) (Blinder 1973; Oaxaca 1973; Reimers 1983).

⁸ All estimations are performed using STATA 10 SE software. The decompositions are performed using Ben Jann’s STATA routine “oaxaca8” combined with the STATA command “tobit” (Jann 2008a, b).

Table 2 Mean levels of cognition and education and corresponding sex differences

		Sample Frequency		Variable Means and Corresponding Sex Differences		
		Total	Censored	MMSE Score	Adjusted MMSE Score	Years of Education
Bridgetown (N = 1,421)	Men	550	235	17.136 (0.116)	18.480 (0.195)	5.380 (0.154)
	Women	871	344	17.176 (0.088)	18.280 (0.136)	5.219 (0.109)
	Difference: Men – Women	—	—	-0.040 (0.146)	0.200 (0.238)	0.161 (0.189)
Buenos Aires (N = 982)	Men	357	81	17.010 (0.105)	17.348 (0.135)	8.080 (0.269)
	Women	625	126	16.662 (0.096)	17.011 (0.119)	6.711 (0.181)
	Difference: Men – Women	—	—	0.348* (0.142)	0.337† (0.180)	1.369*** (0.324)
Havana (N = 1,740)	Men	666	174	16.973 (0.087)	17.446 (0.117)	7.786 (0.161)
	Women	1,074	256	16.660 (0.076)	17.104 (0.095)	7.011 (0.124)
	Difference: Men – Women	—	—	0.313** (0.116)	0.342* (0.151)	0.775 (0.203)
Mexico City (N = 1,102)	Men	457	76	15.919 (0.125)	16.200 (0.145)	5.442 (0.251)
	Women	645	86	15.472 (0.115)	15.702 (0.129)	4.261 (0.168)
	Difference: Men – Women	—	—	0.447** (0.170)	0.498** (0.194)	1.181*** (0.302)
Montevideo (N = 1,378)	Men	502	152	17.049 (0.093)	17.495 (0.133)	6.409 (0.207)
	Women	876	255	16.695 (0.087)	17.215 (0.116)	5.739 (0.137)
	Difference: Men – Women	—	—	0.354** (0.127)	0.280 (0.176)	0.670** (0.248)
Santiago (N = 1,165)	Men	396	83	16.454 (0.231)	17.070 (0.282)	6.800 (0.330)
	Women	769	120	15.990 (0.140)	16.394 (0.163)	5.872 (0.228)
	Difference: Men – Women	—	—	0.464† (0.270)	0.676* (0.326)	0.928* (0.401)
São Paulo (N = 1,904)	Men	766	173	16.648 (0.105)	17.148 (0.135)	4.073 (0.177)
	Women	1,138	236	16.422 (0.088)	16.922 (0.109)	3.150 (0.103)
	Difference: Men – Women	—	—	0.226† (0.137)	0.226 (0.174)	0.923*** (0.205)

Notes: All calculations are based on weighted data. Numbers in parentheses are standard errors. The means of adjusted MMSE scores are estimated under the assumption that the MMSE scores are normally distributed but right-censored. The number of right-censored observations by survey site and sex are given in the “Censored” column.

* $p < .10$; ** $p < .05$; *** $p < .001$

Table 3 presents the estimation results based on multivariate Tobit models for age-adjusted MMSE scores on years of education (Model 1) and expanded models incorporating additional controls for early-life conditions (Model 2), early- and midlife conditions (Model 3), and a complete set of life-course controls (Model 4).

Regardless of study site and sex, age-adjusted means of cognitive function are consistently higher among respondents with more schooling (Model 1). For men, one additional year of schooling is thereby associated with a 0.144 (Montevideo) to 0.275 (Bridgetown) increase in the mean adjusted MMSE score, while the corresponding association for women ranges from 0.184 (Bridgetown) to 0.446 (São Paulo). Interestingly, the age-adjusted association between schooling and cognitive function is larger for women than for men whenever there are statistically significant sex differences.

These patterns are largely robust to the sequential inclusion of additional controls for cognitive resources and constraints pertaining to different life stages (Models 2–4). Respondents with higher levels of education also have statistically significantly higher levels of old-age cognition across almost all models and study sites, with Models 3 and 4 for females in Bridgetown being the only two exceptions. Moreover, while the estimated coefficients of schooling tend to become smaller through the inclusion of additional (potentially mediating) mid- and later-life circumstances, the estimated differences in coefficients between men and women remain mostly stable and are statistically significant across all model specifications for Mexico City and São Paulo.

Table 4 combines the estimated site- and sex-specific variable means and model coefficients to obtain detailed decompositions of predicted male-female differences in cognitive function. The table shows that sex differences in cognitive resources across the life course, notably education, can explain substantial shares of the estimated male-female differences in cognitive function. Although the exact share of explained differences varies by model specification and survey site, the control variables typically explain more than one-half of the total cognitive functioning difference between men and women, with the explained part of the decomposition sometimes even exceeding the overall estimated cognition gap.⁹ The explained differences are also estimated fairly precisely and typically statistically significantly different from zero. Moreover, although the most comprehensive model (Model 4) tends to feature the highest levels of explained contributions, the differences across models appear fairly moderate, and the increase in the explained part toward Model 4 is not always monotonous. The overall unexplained parts, on the other hand, tend to be considerably smaller in magnitude and are often statistically insignificant, reflecting the relatively large share of explained contributions to the decompositions.

The estimations also highlight education as a key factor for decomposing cognitive functioning differences between older men and women. With the exception of Bridgetown, more than one-half of the overall explained difference in late-life cognition stems from male-female differences in educational attainment, with the exact share depending on model specification and study site. Although the specific

⁹ These cases imply a negative unexplained part of the decomposition for those models—that is, negative coefficient effects—because the sum of both decomposition parts needs to be equal to the predicted male-female difference in cognitive function.

Table 3 Coefficient estimates for years of education based on different Tobit models

	Model 1		Model 2		Model 3		Model 4	
	Coefficient	SE	Coefficient	SE	Coefficient	SE	Coefficient	SE
Bridgetown	Men	0.275*** (0.054)	0.261*** (0.053)	0.168** (0.054)	0.153** (0.054)			
	Women	0.184*** (0.041)	0.151*** (0.041)	0.079 (0.041)	0.061 (0.044)			
	Difference: Men – Women	0.091 (0.068)	0.110 (0.067)	0.089 (0.067)	0.092 (0.070)			
Buenos Aires	Men	0.157*** (0.027)	0.163*** (0.029)	0.140*** (0.029)	0.149*** (0.037)			
	Women	0.268*** (0.027)	0.255*** (0.030)	0.206*** (0.030)	0.187*** (0.035)			
	Difference: Men – Women	-0.111** (0.038)	-0.092* (0.042)	-0.066 (0.049)	-0.038 (0.051)			
Havana	Men	0.201*** (0.031)	0.198*** (0.031)	0.152*** (0.031)	0.129*** (0.031)			
	Women	0.246*** (0.024)	0.242*** (0.024)	0.210*** (0.024)	0.196*** (0.029)			
	Difference: Men – Women	-0.045 (0.039)	-0.044 (0.039)	-0.058 (0.044)	-0.067 (0.042)			
Mexico City	Men	0.200*** (0.028)	0.182*** (0.030)	0.168*** (0.030)	0.147*** (0.029)			
	Women	0.308*** (0.030)	0.301*** (0.033)	0.295*** (0.034)	0.252*** (0.035)			
	Difference: Men – Women	-0.108** (0.041)	-0.119** (0.045)	-0.127** (0.045)	-0.105* (0.045)			
Montevideo	Men	0.144*** (0.025)	0.115** (0.029)	0.086** (0.029)	0.086** (0.032)			
	Women	0.247*** (0.028)	0.219*** (0.028)	0.176*** (0.028)	0.133*** (0.033)			
	Difference: Men – Women	-0.103** (0.038)	-0.104** (0.040)	-0.090* (0.040)	-0.047 (0.046)			
Santiago	Men	0.269*** (0.058)	0.262*** (0.051)	0.210*** (0.051)	0.185*** (0.042)			
	Women	0.273*** (0.035)	0.273*** (0.038)	0.218*** (0.038)	0.207*** (0.035)			
	Difference: Men – Women	-0.004 (0.068)	-0.011 (0.064)	-0.008 (0.066)	-0.022 (0.055)			
São Paulo	Men	0.222*** (0.035)	0.215*** (0.038)	0.157*** (0.038)	0.108** (0.039)			
	Women	0.446*** (0.043)	0.428*** (0.045)	0.365*** (0.045)	0.283*** (0.042)			
	Difference: Men – Women	-0.224*** (0.055)	-0.213*** (0.059)	-0.208*** (0.065)	-0.175** (0.057)			

Notes: All calculations are based on separate Tobit models for each study site using weighted data to ensure sample representativeness. Model 1 contains controls for age and education only, while Models 2–4 sequentially add additional controls for early-life, midlife, and later-life conditions. The exact specifications of Models 1–4 are presented in Table 1.

* $p < .10$; ** $p < .05$; *** $p < .01$; **** $p < .001$

Table 4 Decomposition summary and contributions of education to cognitive function differences between men and women

	Model 1		Model 2		Model 3		Model 4	
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE
Bridgetown								
Predicted difference	0.241	(0.241)	0.169	(0.241)	0.229	(0.252)	0.174	(0.257)
Explained difference (total)	0.178*	(0.083)	0.068	(0.100)	0.120	(0.119)	0.316*	(0.140)
Attributable to education alone	0.036	(0.043)	0.032	(0.038)	0.019	(0.024)	0.022	(0.027)
Unexplained difference (total)	0.064	(0.225)	0.101	(0.215)	0.108	(0.211)	-0.142	(0.205)
Attributable to education alone	0.482	(0.359)	0.567	(0.345)	0.429	(0.332)	0.407	(0.307)
Buenos Aires								
Predicted difference	0.334 [†]	(0.183)	0.339 [†]	(0.183)	0.351 [†]	(0.184)	0.338 [†]	(0.186)
Explained difference (total)	0.359***	(0.090)	0.328***	(0.094)	0.444***	(0.117)	0.411**	(0.138)
Attributable to education alone	0.299***	(0.076)	0.286***	(0.074)	0.239***	(0.066)	0.222***	(0.063)
Unexplained difference (total)	-0.025	(0.162)	0.011	(0.162)	-0.094	(0.143)	-0.072	(0.122)
Attributable to education alone	-0.810**	(0.281)	-0.662*	(0.303)	-0.383	(0.289)	-0.416	(0.316)
Havana								
Predicted difference	0.328*	(0.152)	0.326*	(0.153)	0.319*	(0.154)	0.325*	(0.153)
Explained difference (total)	0.240***	(0.063)	0.253***	(0.068)	0.382***	(0.088)	0.437***	(0.111)
Attributable to education alone	0.177***	(0.049)	0.173***	(0.048)	0.140***	(0.041)	0.126***	(0.037)
Unexplained difference (total)	0.088	(0.141)	0.073	(0.140)	-0.063	(0.128)	-0.112	(0.101)
Attributable to education alone	-0.337	(0.290)	-0.319	(0.285)	-0.365	(0.273)	-0.306	(0.192)
Mexico City								
Predicted difference	0.486*	(0.195)	0.489*	(0.196)	0.488*	(0.197)	0.488*	(0.199)
Explained difference (total)	0.345***	(0.094)	0.343***	(0.102)	0.524***	(0.137)	0.550***	(0.158)
Attributable to education alone	0.295***	(0.079)	0.280***	(0.076)	0.266***	(0.073)	0.230***	(0.064)
Unexplained difference (total)	0.141	(0.174)	0.146	(0.173)	-0.036	(0.148)	-0.063	(0.124)
Attributable to education alone	-0.508**	(0.192)	-0.538**	(0.202)	-0.375**	(0.133)	-0.239 [†]	(0.106)

Table 4 (continued)

	Model 1		Model 2		Model 3		Model 4	
	Estimate	SE	Estimate	SE	Estimate	SE	Estimate	SE
Montevideo								
Predicted difference	0.281	(0.177)	0.287	(0.179)	0.297 [†]	(0.179)	0.295	(0.180)
Explained difference (total)	0.172**	(0.069)	0.137*	(0.078)	0.094	(0.101)	0.147	(0.136)
Attributable to education alone	0.137**	(0.053)	0.119**	(0.046)	0.096*	(0.039)	0.076*	(0.032)
Unexplained difference (total)	0.110	(0.167)	0.150	(0.167)	0.203	(0.154)	0.148	(0.130)
Attributable to education alone	-0.627**	(0.228)	-0.625**	(0.243)	-0.424*	(0.210)	-0.155	(0.153)
Santiago								
Predicted difference	0.707*	(0.322)	0.735*	(0.326)	0.736*	(0.328)	0.556 [†]	(0.327)
Explained difference (total)	0.413**	(0.143)	0.391*	(0.154)	0.512**	(0.196)	0.366	(0.250)
Attributable to education alone	0.255*	(0.115)	0.249*	(0.112)	0.196*	(0.091)	0.191*	(0.087)
Unexplained difference (total)	0.294	(0.289)	0.345	(0.289)	0.223	(0.247)	0.190	(0.192)
Attributable to education alone	-0.022	(0.426)	-0.066	(0.399)	-0.045	(0.370)	-0.098	(0.246)
São Paulo								
Predicted difference	0.210	(0.176)	0.221	(0.177)	0.216	(0.177)	0.251	(0.173)
Explained difference (total)	0.386***	(0.078)	0.376***	(0.086)	0.575***	(0.129)	0.388**	(0.140)
Attributable to education alone	0.297***	(0.070)	0.281***	(0.068)	0.231***	(0.059)	0.177***	(0.047)
Unexplained difference (total)	-0.175	(0.163)	-0.155	(0.159)	-0.360*	(0.124)	-0.137**	(0.098)
Attributable to education alone	-0.788***	(0.196)	-0.715***	(0.201)	-0.518**	(0.164)	-0.336**	(0.112)

Notes: All calculations are based on separate models for each study site using weighted data to ensure sample representativeness. Numbers in parentheses are standard errors. Model 1 contains controls for age and education only, while Models 2–4 sequentially add additional controls for early-life, midlife, and later-life conditions. The exact specifications of Models 1–4 are presented in Table 1.

[†] $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$

contribution of education to the explained differences in cognitive functioning tends to decrease with the inclusion of additional control variables—especially mid- and later-life controls—it nonetheless remains positive and statistically significant for all model specifications and study sites, with Bridgetown again being the only exception. Notably, this result also obtains for the richest model specification (Model 4), which contains controls for cognitive resources pertaining to all life stages.

Another interesting finding relates to the contributions of education to the unexplained parts of the decompositions. Reflecting the larger association of schooling and later-life cognition for women than for men, education contributes negatively to the unexplained parts of the cognitive functioning decompositions for all study sites but Bridgetown. These negative unexplained contributions of education are often quite large in magnitude, even if they are only sometimes statistically significant, and tend to decrease with the inclusion of additional life-course controls.

Summary and Discussion

In this study, I document statistically significant differences in early-life education and later-life cognitive functioning between older men and women for several urban areas in Latin America and the Caribbean, with men having both more years of early-life education and higher levels of later-life cognitive functioning than women. I also document a strong relationship between years of education and later-life cognition, which often seems stronger for women than for men. This finding obtains for various different statistical models ranging from a basic model containing age and education only to richer models that incorporate additional early-, mid-, and later-life controls.

Combining these findings within a formal decomposition framework highlights further the key role of education for gender differences in later-life cognition. Gender differences in education explain a large share of the observed differences in later-life cognition between men and women across a range of different models, holding up the inclusion of a wide range of additional cognitive resources that may work as mediators or otherwise confound the analysis. I also find a negative unexplained effect of education on gender differences in later-life cognition. This finding suggests that the stronger association between education and later-life cognition for women relative to men tends to mitigate at least some of the endowment effects that gender differences in educational attainment have on corresponding differences in cognition at older ages.

The data presented here exhibit the same overall pattern as data for Ismailia, Egypt, presented in Yount (2008), even if the documented gender differences in both education and cognitive function are considerably smaller for my study sites. Although this finding indicates the robustness of the impact of gender discrimination over the life course on later-life cognitive performance across different geographic and cultural settings, it also suggests a monotonous dose-response relationship between the extent of gender differences in access to education and other cognitive resources across the life course and subsequent gender differences in later-life cognition. In addition, I find that the association between education on the one hand

and later-life cognition on the other tends to be larger for women than for men, which is again consistent with the evidence presented in Yount (2008). Higher returns to education for women or gender-specific selection into education based on persistent cognitive abilities could be candidate explanations for this finding, although it is difficult to directly attribute it to a single cause given the available data.

In sum, the results point to a central role of equal educational opportunities for boys and girls for closing the cognitive functioning differences between men and women at older ages. In this regard, recent changes in the educational attainment of boys and girls in Latin America and the Caribbean suggest that both levels of later-life cognition and corresponding gender differences may change over the coming decades (Duryea et al. 2007). In terms of overall levels of later-life cognition, the overall rise in education levels over the past decades may result in improved levels of later-life cognition among younger cohorts. With regard to gender differences, one may expect a closing or even reversal of gender differences in later-life cognition over the coming decades, as larger increases in educational attainment for girls relative to boys in many Latin American countries have led to a closing, and in some cases a reversal, of historical gender differences in educational attainment among younger cohorts.

The findings presented in this article document a suggestive association between education and later-life cognition and corresponding significant gender differences in both measures. Yet, the analysis does not necessarily reveal a causal relationship between these measures and thus needs to be interpreted with some caution. The analysis shows that Yount's (2008) findings also apply to different geographic and cultural settings in Latin America and the Caribbean. This study, however, is based data from urban areas only. It would be interesting to explore whether similar relationships also obtain in more rural environments in Latin America and the Caribbean, which I leave as a potential direction for future research.

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