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Social Pensions and Mortality among Older Adults in Extreme Poverty: Evidence from Peru

Jose A. Valderrama
Universidad de Lima

Javier Olivera
LISER, PUCP
and IZA@LISER

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Social Pensions and Mortality among Older Adults in Extreme Poverty: Evidence from Peru*

Abstract

We examine the effects of Peru's non-contributory pension programme, *Pension 65*, on mortality among older adults living in extreme poverty. Using baseline survey data linked to administrative mortality records for 2012–2016, we employ a regression discontinuity design exploiting the welfare-index threshold that determines programme eligibility. We find that eligibility reduces four-year mortality by 10.7 percentage points. A complementary actuarial calculation suggests a gain of approximately one year in life expectancy at age 65 among eligible individuals. We also find that eligibility increases healthcare utilisation, but not public health insurance enrolment or preventive care, suggesting that greater use of existing healthcare services may contribute to improved survival. Our findings highlight the potential of social pensions to reduce mortality among the poorest older adults.

JEL classification

H55, J14, I38

Keywords

non-contributory pensions, mortality, regression discontinuity, old-age poverty

Corresponding author

Javier Olivera
javier.olivera@liser.lu

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1 Introduction

Social security participation is low in developing countries, mostly due to the existence of large informal labour markets and the high predominance of precarious jobs in which pension and health contributions are not compulsory. As populations are rapidly becoming older in these countries, a popular solution for governments has been the establishment of social pension programmes providing pension transfers unrelated to histories of social security contributions. These schemes, also known as non-contributory pension (NCP) programmes, provide monetary transfers targeted at the elderly poor who do not have contributory pensions and have reached retirement age. While benefits are often modest relative to average national income, they can represent a substantial addition to the resources of those who receive them (Huang and Zhang, 2021). Beyond reducing poverty, social pensions may also improve health and survival at ages when health risks are high and financial resources are particularly constrained.

A substantial literature examines the effects of social pensions, particularly on labour supply, household resources and living arrangements. Their consequences for health and survival have received comparatively less attention, despite the potential importance of additional income at older ages. Well-studied examples include programmes in South Africa (Case and Deaton, 2001; Duflo, 2000, 2003), Brazil (Barrientos et al., 2003; de Carvalho-Filho, 2008) and Mexico (Aguila et al., 2015; Juarez and Pfutze, 2015). Although the positive association between income and health is well established, credible evidence on the causal effects of income on health and mortality is harder to obtain.[1] Social pensions offer a particularly relevant setting because they provide income late in life, when even modest improvements in material conditions or access to care may affect survival.

Existing research generally points towards lower mortality following the receipt or expansion of social pensions, although some studies find no statistically detectable effect. For the United States, Balan-Cohen (2008) documents lower mortality among older men following the introduction of Old Age Assistance after 1940, while Stoian and Fishback (2010) finds no significant effect on urban mortality before 1940. In Canada, Emery and Matheson (2012) finds that the universal Old Age Security pension reduced mortality among people aged 70 and over, but detects no effect of a means-tested pension among those aged 65–69. Evidence from South Africa and the United States also points to mortality benefits from old-age income support (Mostert et al., 2022; Arno et al., 2011; Galofré-Vilà et al., 2022; Engelhardt et al., 2022). More recent studies examine social pensions in China (Cheng et al., 2018; Huang and Zhang, 2021), Chile (Miglino et al., 2023) and Mexico (Menares et al., 2026). The Chilean programme reaches the poorest 60% of the population, whereas China's New Rural Pension Scheme is available to rural residents without a means test. In Mexico, Menares et al. (2026)

[1] For evidence on the income–health relationship, see, among others, Case et al. (2002); Deaton (2002); Gerdtham and Johannesson (2004); Smith (2007); Smith and Goldman (2007); Von Gaudecker and Scholz (2007); Belloni et al. (2013). On causal identification, see Smith (1998); Deaton and Paxson (1998); Smith (1999); Lenhart (2019).

finds a mortality reduction among women but not men. These studies suggest that effects differ in magnitude and across population groups, making it important to establish which older people benefit most and through which channels. In particular, evidence remains limited for transfers directed specifically at older adults living in extreme poverty.

We address this gap by studying Peru's non-contributory pension programme, *Pension 65*. The programme provides a lifetime pension of 125 soles per month to people aged 65 or older who receive no other pension and live in households classified as extremely poor by Peru's national targeting system (SISFOH). Although modest in absolute terms (approximately USD 32 per month), the transfer was equivalent to 62% of the national extreme-poverty line in 2021. SISFOH assigns households a continuous welfare score and uses thresholds to classify them as extremely poor, poor or non-poor. Individuals on either side of the extreme-poverty threshold can thus have very similar observed welfare scores but different pension eligibility.

Our analysis uses the ESBAM survey, conducted in 2012 specifically to support a regression discontinuity design (RDD) around this threshold. We link respondents to programme administrative data and mortality records covering 2012–2016. The baseline survey also contains detailed health assessments collected by interviewers and medical technicians. Comparing individuals with welfare scores just below and just above the eligibility cut-off, we estimate the intention-to-treat (ITT) effect of pension eligibility on mortality. The identification strategy relies on the comparability of individuals close to the cut-off. We find no evidence of manipulation of the SISFOH score around the threshold, and the results are robust to a range of specification and falsification checks.

Our main finding is that eligibility reduces four-year mortality by 10.7 percentage points (pp), approximately 92% of the mortality rate among ineligible individuals at the threshold. A back-of-the-envelope actuarial extrapolation based on parametric mortality functions translates this estimate into a gain of approximately one year in life expectancy at age 65. An illustrative cost calculation also places the transfer cost per additional life-year, scaled to the length of a statistical life, at around 18% of the mean value of a statistical life for Peru.

Previous work using the 2015 ESBAM follow-up finds that *Pension 65* improves nutrition, self-reported health and cognitive functioning, reduces anaemia and symptoms of depression, and increases healthcare utilisation (Bernal et al., 2024). Several of these outcomes are established predictors of mortality, suggesting plausible pathways linking the transfer to survival. However, they do not by themselves identify the mechanism driving the mortality effect. In our analysis of the follow-up data, eligibility increases visits to healthcare centres but has no detectable effect on public health insurance enrolment, already high on both sides of the threshold, or on preventive care. This pattern points to greater use of existing health services, rather than expanded insurance coverage, as a possible mechanism.

Our study contributes to the literature in three respects. First, it provides evidence on the mortality effects of social pensions among the extreme poor, whereas programmes studied in Chile, China and Mexico serve broader populations or use less restrictive targeting. We estimate

a mortality–income elasticity of -0.82 (95% confidence interval: $[-1.53, -0.24]$), compared with estimates of approximately -0.38 for Chile and China (Miglino et al., 2023; Huang and Zhang, 2021) and -0.23 for women in Mexico (Menares et al., 2026). The underlying periods and calculations differ, and our confidence interval includes the Chilean and Chinese estimates. Nevertheless, the larger point estimate is consistent with a steeper income–mortality gradient towards the bottom of the distribution. Unlike the findings for Chile and Mexico, our mortality effect is statistically significant for both women and men. Second, the detailed baseline health information collected by ESBAM, including biomarkers and anthropometric measures, allows us to assess whether pre-existing differences in mortality risk account for the result. A predicted-mortality falsification test yields an effect whose confidence interval excludes our main ITT estimate. Third, the follow-up survey allows us to examine healthcare use and insurance coverage as potential channels, helping to distinguish greater utilisation of services from expanded access to insurance.

Our findings are local to ESBAM respondents near the eligibility threshold and should not be generalised to all potential programme beneficiaries without qualification. The remainder of this paper is organised as follows. Section 2 describes the NCP programme while Section 3 presents the data and the empirical strategy. Section 4 discusses the validity of the RDD. Section 5 analyses and discusses the results and limitations. Section 6 discusses policy impacts. Section 7 presents and discusses evidence for validation, falsification and robustness checks. Lastly, Section 8 concludes.

2 Non-contributory pensions in Peru

Introduced in late 2011, *Pension 65* is Peru's non-contributory pension programme for individuals aged 65 or older who receive no other pension and live in households classified as extremely poor by the national targeting system, SISFOH. During our study period (2012–2016), the programme paid 250 soles every two months, equivalent to 125 soles per month. It also facilitates recipients' enrolment in the public health insurance scheme, *Seguro Integral de Salud* (SIS), a feature we discuss below.

Although the pension is modest in absolute terms, it represents a substantial share of the resources available to households in extreme poverty. In 2012, the monthly equivalent was approximately US$47, or 83% of the national extreme-poverty line and 44% of the national poverty line. In rural areas, the corresponding shares were 98% and 59%.[2] The nominal benefit remained unchanged until June 2025, when the bimonthly payment increased to 350 soles. By 2021, the monthly equivalent of the original benefit was approximately US$32 and represented 62% of the extreme-poverty line and 33% of the poverty line.

The programme was initially introduced in the poorest districts of six priority departments

[2] In 2012, the monthly equivalent was also 17% of the minimum wage and 14% and 33% of average household per capita income in urban and rural areas, respectively.

and expanded in 2012 to 14 departments previously covered by a short-lived non-contributory pension pilot.[3] By 2015, *Pension 65* had reached slightly more than half a million beneficiaries. In 2021, it covered 568,599 people, approximately 19% of Peru's population aged 65 or older, at a fiscal cost of about 0.10% of GDP.

Programme eligibility depends critically on SISFOH (*Sistema de Focalización de Hogares*), the national system for assessing households' socioeconomic circumstances for social programmes. Government enumerators collect information using a standardised questionnaire, the *Ficha Socioeconómica Única* (FSU), covering housing materials, access to water, electricity and sanitation, overcrowding, education, household assets and health insurance.[4] These data are used to construct a multidimensional welfare index (the SISFOH score), which is compared with region-specific cut-offs to classify households as extremely poor, poor but not extremely poor, or non-poor. Classifications are valid for three years in urban areas and four years in rural areas.[5] A major data-collection exercise in 2012 provided the sampling frame for the survey used in our analysis.

The SISFOH score also underpins our regression discontinuity design. Households are informed of their poverty classification, but not of their numerical score or the cut-offs used to determine it. Moreover, the score is calculated from a range of household characteristics, making precise manipulation difficult. These institutional features reduce the scope for sorting around the extreme-poverty threshold; Sections 4 and 7 examine this issue empirically, including through tests of the score's density at the cut-off.

Besides being assessed during the initial data collection, households can request a SISFOH classification through their municipality. Once eligibility is confirmed, enrolment in *Pension 65* can take approximately 25 days. Programme officials also conduct information campaigns with local governments and identify potential recipients who lack a SISFOH classification or the identity document required for enrolment (Bernal et al., 2024). Households may subsequently request reassessment of their socioeconomic classification, which requires another enumerator visit, takes at least 30 working days from that visit and does not guarantee a change in status.[6] Importantly, any subsequent reassessment cannot change the 2012 SISFOH score used as the running variable in our analysis, although it may affect later programme participation.

In addition to the cash transfer, *Pension 65* facilitates registration in SIS, which covers healthcare services without direct charges for covered treatment, although out-of-pocket expenses may remain. This enrolment assistance does not create a new entitlement to insurance: individuals in households classified as poor, whether extremely poor or poor but not extremely poor, are already eligible for SIS. The programme instead provides an additional registration

[3] The *Bono Gratitud* pilot operated from October 2010 to August 2011 and reached 21,783 people across 14 departments. It paid 100 soles per month to individuals aged 75 or older living in households classified as extremely poor.

[4] The FSU questionnaire and methodological manuals are available from SISFOH at `http://www.sisfoh.gob.pe`.

[5] For details of the welfare-index methodology, see Valderrama and Pichihua (2011).

[6] See Peru's Ministry of Development and Social Inclusion (MIDIS): `https://www.gob.pe/84084`.

channel for pension recipients. Accordingly, SIS affiliation was already common on both sides of the pension-eligibility threshold before the programme reached our sample. Within the bandwidth used for the mortality analysis, 63.9% of eligible and 63.3% of ineligible individuals were affiliated at the 2012 baseline.

These institutional features suggest two potential channels through which pension eligibility may affect survival. The transfer can ease financial constraints on food, medication, transport and other health-related expenditures, while facilitated SIS registration may increase access to covered healthcare among those not yet insured. Given the high baseline rate of insurance affiliation, however, changes in healthcare utilisation need not be accompanied by changes in insurance coverage. We explore these possibilities using the 2015 ESBAM follow-up, which records SIS affiliation, healthcare visits and preventive care (Section 5.7).

3 Data and empirical strategy

3.1 Data

Our main data source is the Survey of Health and Well-being of the Elderly (ESBAM, *Encuesta de Salud y Bienestar del Adulto Mayor*), designed to evaluate *Pension 65* using a regression discontinuity design (RDD). The baseline survey was conducted between 2 November and 28 December 2012, followed by a second wave in July–September 2015. Although *Pension 65* was already operating in many districts in 2012, individuals already enrolled were excluded from the sampling frame, and enrolment of eligible sample members was scheduled for January 2013, after the baseline fieldwork (MIDIS and MEF, 2013). ESBAM collects individual- and household-level information on demographics, income, expenditure and subjective and objective health. Face-to-face interviews are complemented by measurements taken by medical technicians, including anthropometrics, blood pressure and blood samples. We link these survey data to three administrative sources: mortality records, *Pension 65* beneficiary records and the SISFOH register.

The ESBAM sampling frame covers 12 departments in which the SISFOH data collection had been completed: Amazonas, Ancash, Cajamarca, Cusco, Huánuco, Junín, La Libertad, Loreto, Pasco, Piura, Puno and the provinces of Lima. The sampling frame was restricted to households with at least one member aged 65–80 and a SISFOH score within 0.30 standard deviations on either side of the extreme-poverty threshold. This deliberately narrow window concentrates the sample around the programme eligibility cut-off. The survey used a two-stage sampling design, selecting geographical clusters and then households within them.[7] Because our estimand concerns individuals near the eligibility threshold rather than the national older

[7]Primary sampling units were urban census blocks or rural villages (*centros poblados*). Clusters were selected with probability proportional to the number of households, followed by random household selection within each cluster.

population, we do not apply survey expansion weights. Appendix Figure B–1 illustrates the concentration of ESBAM observations around the cut-off relative to the national SISFOH score distribution.

The baseline survey interviewed 4,238 individuals.[8] We match respondents to the administrative sources using the national identification number recorded in ESBAM. *Pension 65* records identify beneficiaries and the timing of transfers, while SISFOH records provide welfare scores and poverty classifications. Mortality information comes from the National Registry of Identification and Civil Status (RENIEC), which records dates of death, but not causes, during our observation period from December 2012 to December 2016.

We exclude 353 observations because of prior programme participation, receipt of other pensions, an out-of-scope poverty classification, missing or inconsistent eligibility information, or unsuccessful record linkage.[9] The resulting analytical sample contains 3,885 individuals. The SISFOH scores and linked mortality records are available only for ESBAM respondents, so we cannot extend the RDD analysis to older adults outside this survey.

Table 1 reports mortality between 2012 and 2016 by eligibility status, age and gender. Among the 2,525 eligible individuals, 209 died (8%), compared with 123 of the 1,360 ineligible individuals (9%). Mortality was lower among women than men (7% versus 10%) and increased with age, from 5% at ages 65–70 to 8% at ages 71–75 and 17% at ages 76–80. These are unadjusted differences, not RDD estimates.

[8]The original ESBAM sample size was determined using the analytical minimum detectable effect (MDE) formula for two-stage clustered regression discontinuity designs (Schochet, 2008). The design assumed an MDE of 0.15 standard deviations (Cohen, 1988), 90% power, a 5% significance level, an intraclass correlation of 0.15, an average cluster size of two and an auxiliary R^2 of 0.5625. These assumptions were set before the mortality follow-up period was defined; the MDE was not specific to mortality.

[9]The exclusions comprise 137 individuals already receiving *Pension 65* at baseline; 66 reporting contributory pensions; 59 classified as non-poor; 23 missing the SISFOH score; three aged 81 or older; one deceased at baseline; and 64 who could not be identified in the administrative records. The counts sum to 353.

Table 1: Distribution of observations in initial ESBAM sample

	Sex		Age in 2012			Overall
	Male	Female	65-70	71-75	76-80	
Overall	2,118	1,767	1,901	1,204	780	3,885
Survivor	1,904	1,649	1,801	1,106	646	3,553
Dead	214	118	100	98	134	332
Mortality rate	10%	7%	5%	8%	17%	9%
Eligible	1,406	1,119	1,206	803	516	2,525
Survivor	1,274	1,042	1,144	737	435	2,316
Dead	132	77	62	66	81	209
Mortality rate	9%	7%	5%	8%	16%	8%
Ineligible	712	648	695	401	264	1,360
Survivor	630	607	657	369	211	1,237
Dead	82	41	38	32	53	123
Mortality rate	12%	6%	5%	8%	20%	9%

Notes: The sample is composed of individuals observed in the baseline of the 2012 ESBAM survey. After dropping observations with inconsistent information or missing key information, the initial sample size is set to 3,885 individuals.

Appendix Table B–1 presents descriptive statistics for the full analytical sample; variable definitions appear in Appendix A. Appendix Table B–2 reports the corresponding statistics within the bandwidth used for the main RDD estimates, selected following Calonico et al. (2015) (see Section 4.1). In this narrower sample, four-year mortality is 7.8% among eligible individuals and 10% among ineligible individuals.

Finally, ESBAM focuses on a particularly disadvantaged older population. Appendix Figure B–2 compares the survey's 2012 household per capita income distribution with the national distribution: approximately 60% of ESBAM respondents fall below the national 20th percentile, and 70% below the 25th percentile. This concentration at the bottom of the income distribution is important when interpreting the mortality estimates and their external validity.

3.2 Empirical strategy

We exploit the discontinuity in *Pension 65* eligibility at the SISFOH extreme-poverty threshold. As described in Section 2, SISFOH assigns households a welfare score and compares it with region-specific cut-offs. We define the running variable as Z_i, the household's SISFOH score minus the applicable extreme-poverty cut-off. Thus, $Z_i < 0$ identifies eligible individuals and $Z_i \geq 0$ identifies ineligible individuals. The running variable is measured in welfare-index points, not standard deviations; for example, $Z_i = -0.10$ indicates a score 0.10 points below the regional threshold (Valderrama and Pichihua, 2011). The main estimation bandwidth of ± 0.150 is expressed in these same units; it should not be confused with the sampling-frame restriction of ± 0.30 standard deviations described in Section 3.1.

Our regression discontinuity design (RDD) compares individuals immediately on either side of the cut-off. Identification rests on the assumption that, in the absence of eligibility, potential mortality outcomes would vary continuously with the score. The index is calculated administratively and the eligibility decision is based on the recorded SISFOH score, which is also our observed assignment variable. Consequently, imperfect measurement of underlying household welfare need not imply an error in the assignment rule itself. Nevertheless, precise sorting around the threshold or discontinuities in other determinants of mortality could compromise the design; we examine these concerns in Section 4.

Let Y_i denote the outcome and $D_i = 1[Z_i < 0]$ indicate eligibility. The discontinuity in expected outcomes at the threshold identifies the local effect of eligibility:

$$\tau = \lim_{z \to 0^-} E[Y_i \mid Z_i = z] - \lim_{z \to 0^+} E[Y_i \mid Z_i = z]. \tag{1}$$

Since crossing the threshold changes eligibility but does not ensure receipt of the pension, τ is an intention-to-treat (ITT) effect, not the effect of actual programme participation. This distinction is important because some eligible individuals may not enrol in the programme. Estimation based simply on the difference between average outcomes within a window around the cut-off could be misleading if mortality varies with the score on either side. We therefore estimate separate local linear relationships, using the following pooled specification:

$$E[Y_i \mid Z_i] = \beta_0 + \beta_1 D_i + \beta_2 Z_i + \beta_3 Z_i D_i. \tag{2}$$

Here β_1 measures the eligibility discontinuity at $Z_i = 0$, β_2 is the slope for ineligible individuals and $\beta_2 + \beta_3$ is the slope for eligible individuals (Imbens and Lemieux, 2008). Local linear estimation accounts for changes in the conditional expectation of the outcome on either side of the cut-off, allowing us to estimate the discontinuity at the threshold rather than differences in average outcomes within the bandwidth.

Our primary outcome is a binary indicator equal to one if an individual died over the four-year period ending in December 2016 and zero if the individual survived. We also estimate effects for shorter observation periods to examine how the mortality difference evolves over time. We fit the local linear models using a triangular kernel, which assigns greater weight to observations closer to the cut-off (Calonico et al., 2014). Because the RDD is identified at the threshold, covariate adjustment is not required for identification under the continuity assumption. Nevertheless, we add demographic characteristics and, importantly, baseline health measures in supplementary specifications to assess the sensitivity and precision of the estimates. Standard errors are clustered at the survey's primary sampling unit (PSU), consistent with ESBAM's two-stage sampling design.[10] Bandwidth selection and sensitivity to alternative

[10]PSU clustering accounts for within-cluster dependence and the survey design (Abadie et al., 2020). In the headline specification, PSU-clustered, heteroskedasticity-robust and conventional standard errors for the ITT estimate of -0.107 are 0.033, 0.033 and 0.029, respectively; the estimate remains significant at the 1% level under all three.

specifications are discussed in Section 5.1 and Section 7.

To investigate heterogeneity, we interact the eligibility indicator and the running-variable terms with subgroup membership:

$$E[Y_i \mid Z_i, S_{is}] = \beta_0 + \beta_1 D_i + \beta_2 Z_i + \beta_3 Z_i D_i + S_{is}\left(\beta_4 + \beta_5 D_i + \beta_6 Z_i + \beta_7 Z_i D_i\right), \tag{3}$$

where S_{is} indicates membership in subgroup s. The ITT is β_1 for $S_{is} = 0$ and $\beta_1 + \beta_5$ for $S_{is} = 1$; β_5 captures the difference between subgroup effects. We examine heterogeneity by sex, urban or rural residence, schooling (none versus some) and age (65–70 versus 71–80).

The binary-outcome analysis captures whether a death occurred by the end of a given interval, but not when it occurred. We therefore complement it with survival models that exploit the recorded dates of death during 2012–2016. As discussed by Bor et al. (2014), RDD methods can also be applied to survival outcomes. In this setting, we specify the instantaneous mortality hazard as a function of eligibility and the centred SISFOH score, allowing the score gradient to differ on either side of the cut-off:

$$\log h_i(t) = \log h_0(t) + \theta_1 D_i + \theta_2 Z_i + \theta_3 Z_i D_i. \tag{4}$$

Here $h_i(t)$ is the instantaneous hazard of death at duration t, while $h_0(t)$ is the baseline hazard. The coefficient θ_1 measures the discontinuity in the log hazard at the eligibility cut-off, and $\exp(\theta_1)$ is the corresponding hazard ratio under the proportional-hazards assumption. We estimate the model by maximum likelihood, taking $h_0(t)$ to follow a Gompertz specification, as commonly used in mortality research (Chetty et al., 2016; Dodd et al., 2018; Olivera, 2019; Castellares et al., 2020; Kulinskaya et al., 2020). We also assess robustness to Weibull and exponential baseline hazards.[11]

Figure 1 documents the jump in programme participation at the eligibility threshold. Although this jump confirms that the threshold predicts pension receipt, our analysis focuses on the reduced-form effect of eligibility, rather than rescaling it into an effect of participation. Section 4 assesses the identifying assumptions of the RDD; Section 7 presents additional falsification and robustness checks. Together, these analyses inform the credibility of a causal interpretation without treating any single diagnostic as definitive.

4 RD validity

4.1 First stage

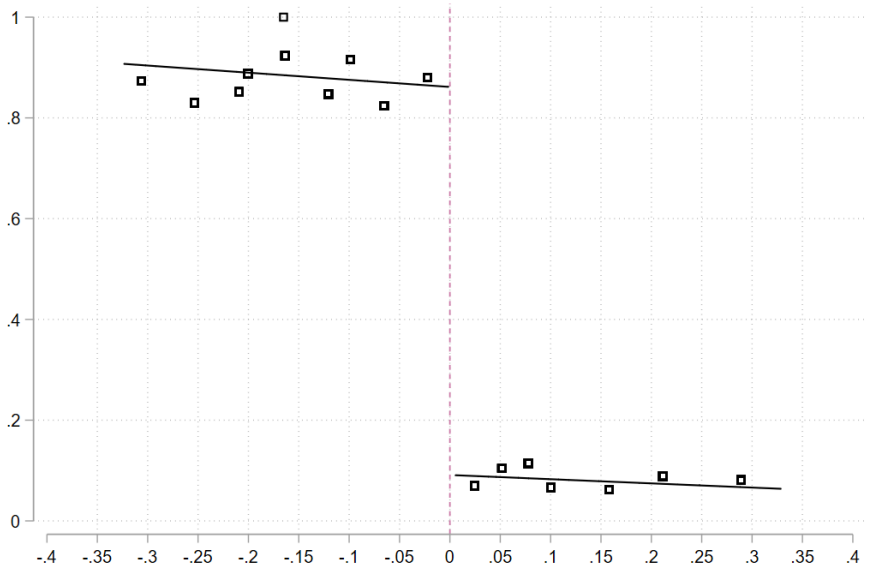
Figure 1 plots the probability of receiving *Pension 65* at any point during 2012–2016 against the centred SISFOH score. Programme receipt increases sharply at the eligibility threshold: the

[11] Duration t is measured from 31 October 2012, immediately before the ESBAM baseline fieldwork.

estimated probability is 85% immediately below the cut-off and 9% immediately above it, a discontinuity of approximately 76 pp. The threshold therefore provides a strong first stage for programme participation, although eligibility does not perfectly determine receipt, as expected in a fuzzy RDD (Hahn et al., 2001). Our main estimates nevertheless focus on the intention-to-treat effect of eligibility rather than the effect of actual receipt.

The strength of this first stage varies with the period considered. Appendix Table B–3 and Figure B–3 show that the discontinuity in receipt reaches approximately 85 pp in 2015, three years after baseline.[12]

Figure 1: *Pension 65* participation at the eligibility threshold



Notes: The figure plots the probability of receiving *Pension 65* at any point during 2012–2016 against the centred SISFOH score (running variable). Square markers represent mean programme participation within non-overlapping bins, with the number and location of bins selected using the integrated mean squared error criterion and quantile-spaced methods of Calonico et al. (2015). Solid lines show separate linear fits on either side of the eligibility threshold (vertical dashed line). Individuals to the left (right) of the threshold are eligible (ineligible) for the programme.

4.2 Continuity of the running variable and predetermined covariates

The credibility of the RDD rests on the absence of precise sorting around the eligibility cut-off and the continuity of potential outcomes at that threshold (Lee and Lemieux, 2010). Several institutional features make manipulation of the SISFOH score unlikely. As documented by Bernal et al. (2024), the underlying information was collected through a door-to-door enumeration conducted by INEI between January and July 2012 to update the national household register used across social programmes, rather than specifically for *Pension 65*. Moreover, neither the scoring

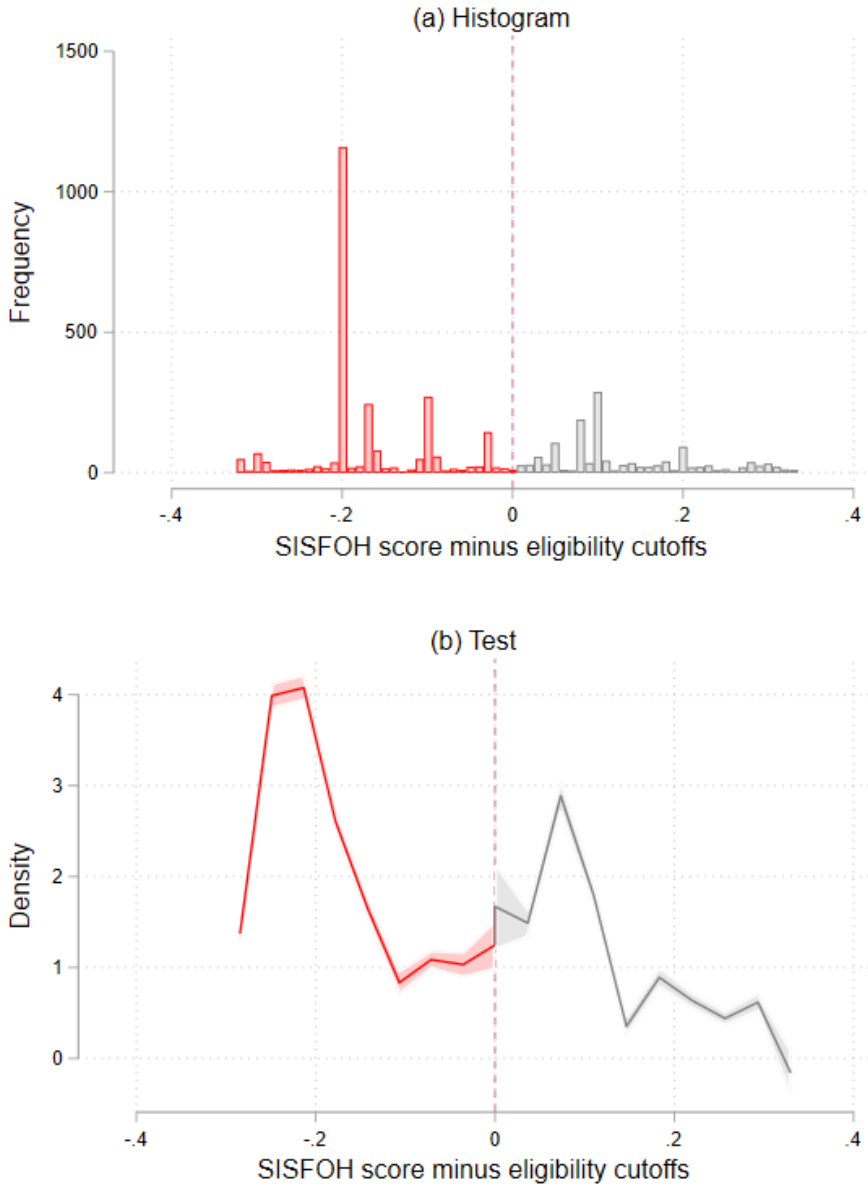
[12] A change in SISFOH eligibility conditions in late 2016 facilitated programme access for some individuals previously classified as ineligible but close to the cut-off. The share of recipients among those ineligible at baseline consequently increased after 2016. This subsequent change does not affect our analysis of mortality over 2012–2016.

algorithm nor the regional eligibility thresholds were publicly known, and government enumerators collected information in person, including verifiable dwelling characteristics. Together, these features limit households' ability to manipulate their position around the eligibility threshold. We assess the plausibility of this assumption through density and predetermined-covariate continuity tests.

First, we test for a discontinuity in the density of the running variable using the approaches of McCrary (2008) and Cattaneo et al. (2018). Figure 2 shows no clear break at the threshold, and the formal test does not reject density continuity ($p = 0.132$). This finding is consistent with the institutional barriers to precise manipulation of eligibility.

Second, following Cattaneo et al. (2020), we estimate equation 2 using each of the 15 baseline covariates as the outcome. Figure 3 reports the estimated discontinuities, while Appendix Figure D–1 plots the corresponding relationships with the running variable. Only alcohol use is statistically significant at the conventional 5% level before adjusting for multiple testing. Its unadjusted p-value of 0.005 exceeds the Bonferroni threshold of $0.05/15 = 0.0033$, and none of the 15 covariates remains significant under the Benjamini–Hochberg procedure at a 5% false discovery rate.

Figure 2: Histogram and manipulation test based on density discontinuity



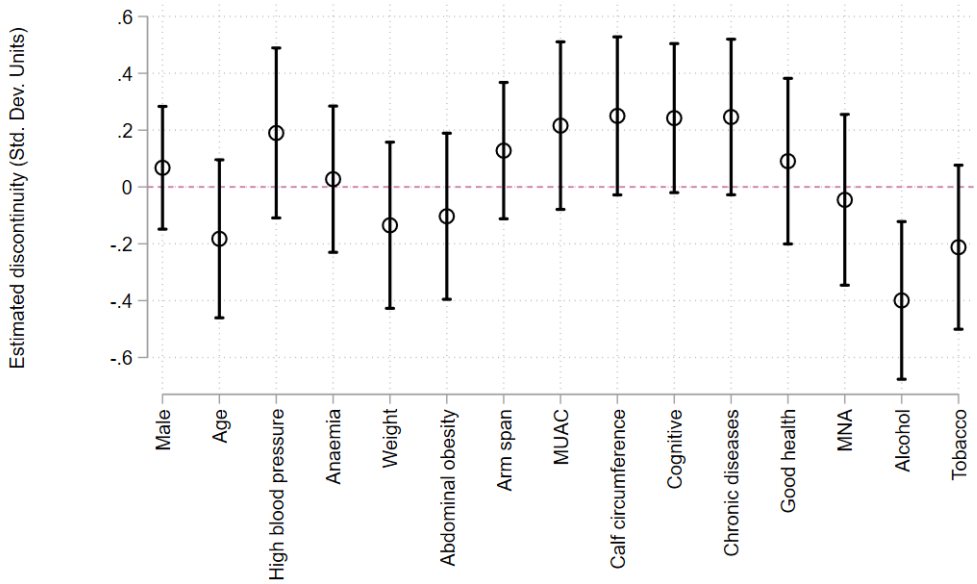
Notes: The figure displays the histogram and estimated density of the centred SISFOH score around the eligibility threshold. The density discontinuity test follows Cattaneo et al. (2018), using local cubic polynomials with bias correction and bandwidths of 0.16 and 0.10 to the left and right of the cut-off, respectively. We do not reject the null hypothesis of density continuity ($p = 0.1323$). A single value of the running variable is shared by 1,126 of the 3,885 observations, distributed across 313 primary sampling units (with at most 19 observations per unit). This concentration is consistent with the discrete inputs of the SISFOH scoring algorithm rather than localised manipulation. Importantly, this value lies outside the bandwidths used for the density test and the RD point estimates.

Statistical insignificance alone, however, does not establish covariate balance. Six of the 15 covariates have estimated discontinuities of at least 0.2 standard deviations: chronic diseases, calf circumference, cognitive functioning, mid-upper arm circumference, alcohol use and tobacco use. The largest is for alcohol use, at -0.40 standard deviations. Several of these characteristics are associated with mortality, so we also examine whether adjusting for them

changes the estimated programme effect. In Table 3, including all 15 covariates reduces the ITT estimate from -0.107 to -0.089 (approximately 17%), but the effect remains negative and statistically significant.

Taken together, the institutional setting, density test and covariate checks support the plausibility of the RDD assumptions, while the non-negligible covariate differences warrant caution. The covariate-adjusted estimates and additional checks in Section 7 help assess whether these differences materially affect our mortality findings.

Figure 3: Balance of covariates



Notes: This figure plots the ITT estimates of equation 2 using the listed covariates as dependent variables instead of mortality. Variables are standardised to facilitate comparison. All the estimated models use the triangular kernel, local linear polynomial, and the optimal bandwidth for point estimation as suggested by Calonico et al. (2015) (obtained in Table 2). The vertical lines indicate 95% confidence intervals.

5 Results

5.1 Linear ITT effects

Table 2 reports the intention-to-treat (ITT) effect of *Pension 65* eligibility on mortality between 2012 and 2016. The outcome equals one if an individual died during this four-year period and zero otherwise. Using the full sample (column 1), eligibility reduces mortality by 6.0 pp, against an estimated mortality rate of 11.5% among ineligible individuals at the cut-off. Narrowing the bandwidth to ± 0.200 (column 2), which retains approximately half of the sample, increases the estimated reduction to 8.3 pp; the corresponding ineligible-group mortality rate is 11.3%.

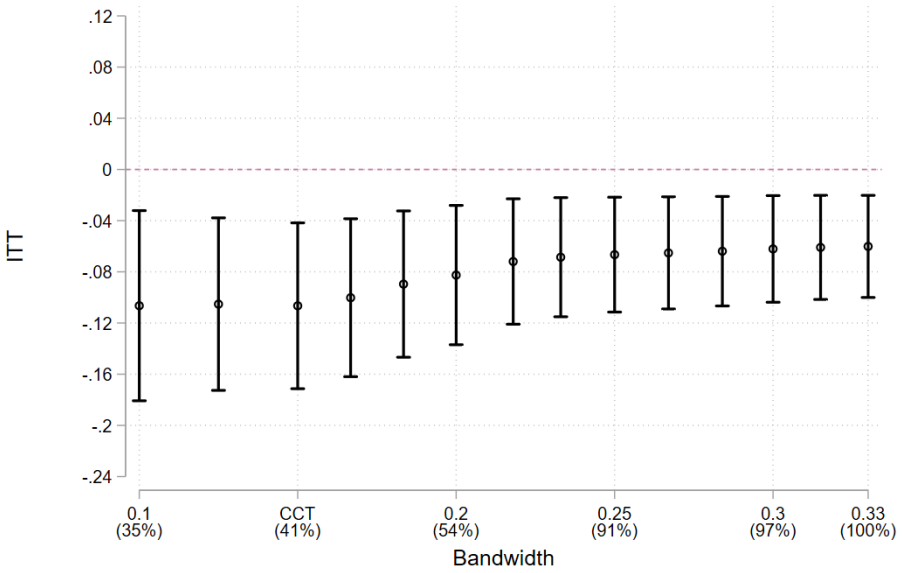
Table 2: Effect of *Pension 65* on mortality rate

	(1)	(2)	(3)
Intention-to-treat ($\hat{\beta}_1$)	-0.060	-0.083	-0.107
	(0.020)	(0.028)	(0.033)
	[0.003]	[0.003]	[0.001]
Constant ($\hat{\beta}_0$)	0.115	0.113	0.116
	(0.017)	(0.025)	(0.028)
	[0.000]	[0.000]	[0.000]
Bandwidth	+/- 0.330	+/- 0.2	+/- 0.150
Observations	3,885	2,104	1,598
Percentage Sample	Full sample	54%	41%

Notes: The table reports the ITT estimates for mortality observed in 4 years (Equation 2). The models use triangular kernel and local linear polynomial. The first model uses the full sample, which has a bandwidth of +/- 0.330. The second uses about half of the sample size, which has a bandwidth of +/- 0.2. The third uses the optimal bandwidth for point estimation as suggested by Calonico et al. (2015). The standard errors are clustered by the Primary Sampling Unit (PSU) of the sampling framing and are indicated in parentheses. P-values are reported in brackets.

Our preferred specification uses the data-driven bandwidth selector of Calonico et al. (2015), yielding ± 0.150 (column 3). The estimated ITT is -0.107, a reduction of 10.7 pp relative to a mortality rate of 11.6% for ineligible individuals at the threshold. This corresponds to approximately 92% of the latter rate. Figure 4 examines bandwidths from ± 0.100 to ± 0.330, the full-sample range. Estimates remain negative and statistically significant throughout, with magnitudes generally attenuating as the bandwidth widens. This pattern underscores the importance of the local comparison but does not, by itself, establish the absence of bias.

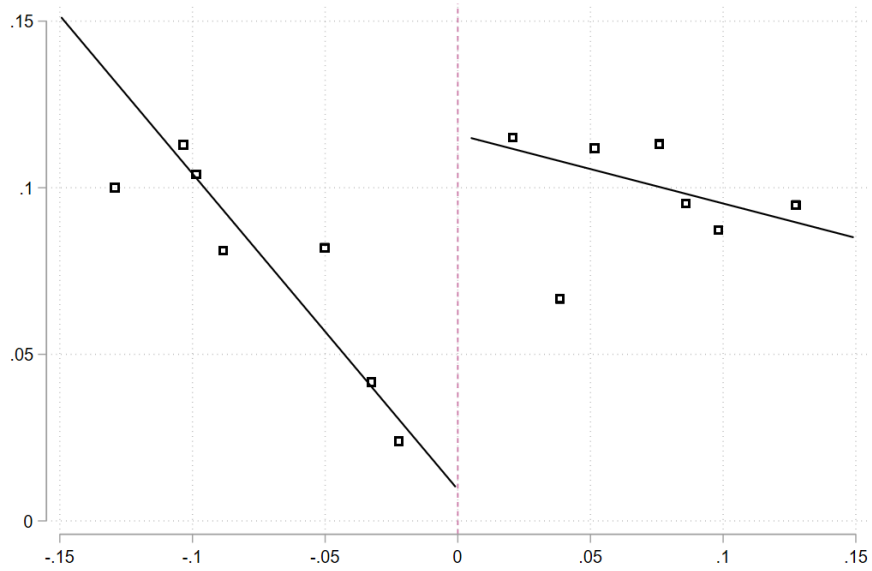
Figure 4: ITT effects by alternative bandwidths



Notes: The figure reports ITT estimates from equation 2 across alternative bandwidths, using local linear regressions and a triangular kernel. The horizontal axis indicates the percentage of the sample included in each specification. CCT identifies the bandwidth selected using Calonico et al. (2015). Vertical bars denote 95% confidence intervals.

Figure 5 illustrates the mortality discontinuity at the eligibility threshold using the preferred bandwidth. The fitted relationships on either side of the cut-off show a pronounced drop in mortality for eligible individuals. We examine the sensitivity of this result to alternative specifications and mortality windows in Section 7.

Figure 5: Intention-to-treat effects on mortality



Notes: The figure plots four-year mortality against the centred SISFOH score. Points show mean mortality within non-overlapping bins, selected using the integrated mean squared error criterion and quantile-spaced bins following Calonico et al. (2015). Solid lines are fitted local linear regressions with a triangular kernel on either side of the cut-off. Individuals to the left (right) of the dashed vertical line are eligible (ineligible).

5.2 Local average treatment effects (LATE)

Eligibility does not guarantee programme receipt, and some individuals classified as ineligible at baseline subsequently receive the pension. Defining take-up as any receipt during 2012–2016, we estimate a first-stage discontinuity of 76.2 pp at the preferred bandwidth of ± 0.150 (Appendix H, Table H–1, Panel A). Dividing the ITT by this first stage gives a Wald estimate of -0.140 (Imbens and Lemieux, 2008). The corresponding fuzzy RD estimate from `rdrobust` has a conventional 95% confidence interval of $[-0.223, -0.056]$, but a robust bias-corrected interval of $[-0.258, 0.013]$, which includes zero. The point estimate is -0.108 at ± 0.200 and -0.078 in the full sample.

The magnitude of the preferred Wald estimate also highlights a limitation of linear probability models: adding -0.140 to the estimated mortality rate of 11.6% among ineligible individuals at the cut-off gives an implausible negative probability of -2.4 pp. The analogous implied probabilities are positive at wider bandwidths (0.5 pp at ± 0.200 and 3.7 pp in the full sample). As a bounded-response check, Section 7.5 estimates a recursive bivariate probit, obtaining a treatment-effect estimate of -0.149. The nonlinear specification respects the bounds on predicted mortality probabilities, although its estimated effect is not directly interchangeable with

the linear Wald contrast.

There is also a timing issue in interpreting the Wald ratio. Because receipt is defined over the same four-year window as mortality, death can prevent subsequent take-up. Thus, although baseline eligibility cannot be caused by later mortality, observed receipt over follow-up may itself depend on survival. We therefore treat the fuzzy RD estimates as supplementary to the reduced-form ITT rather than as an unqualified causal effect of receipt. For the same reason, we do not regress mortality on cumulative years of pension receipt: individuals who die early necessarily have less time to accumulate benefits, creating a risk of immortal time bias (Suissa, 2008). Modelling the timing of take-up and subsequent mortality jointly would require a separate time-varying treatment framework.

5.3 Role of covariates

Although the RDD identifies the effect at the cut-off under the continuity assumption, controlling for predetermined characteristics provides a useful check on the sensitivity of the mortality estimate. ESBAM includes demographics, interviewer- and technician-measured health indicators, self-reported health and nutrition measures, and behavioural risk factors. Table 3 adds these variables in blocks to the preferred specification; their definitions appear in Appendix Tables A–1 and A–2.

Table 3: Effect of *Pension 65* on mortality rate, including covariates

	(1)	(2)	(3)	(4)	(5)
ITT	-0.107***	-0.099***	-0.077**	-0.095***	-0.089***
	(0.033)	(0.033)	(0.032)	(0.031)	(0.032)
Male		0.027*	0.051**	0.059***	0.053**
		(0.015)	(0.020)	(0.020)	(0.021)
Age		0.010***	0.007***	0.007***	0.007***
		(0.002)	(0.002)	(0.002)	(0.002)
High blood pressure			0.041**	0.034*	0.036*
			(0.018)	(0.018)	(0.019)
Anaemia			0.036**	0.038**	0.038**
			(0.018)	(0.017)	(0.017)
Weight			0.003**	0.004**	0.003**
			(0.002)	(0.002)	(0.002)
Abdominal obesity			0.027	0.029	0.031
			(0.020)	(0.020)	(0.020)
Arm span			-0.000	-0.001	-0.001
			(0.001)	(0.001)	(0.001)
Mid-upper arm circ. (MUAC)			-0.013***	-0.011***	-0.010**
			(0.004)	(0.004)	(0.004)
Calf circumference (CC)			-0.009**	-0.008*	-0.007*
			(0.004)	(0.004)	(0.004)
Cognitive functioning			-0.015***	-0.012***	-0.012***
			(0.004)	(0.004)	(0.004)
Chronic diseases				0.007	0.007
				(0.006)	(0.006)
Health today				-0.026	-0.026
				(0.017)	(0.017)
Nutrition score (MNA)				-0.006*	-0.007*
				(0.004)	(0.004)
Alcohol					0.037*
					(0.022)
Tobacco					0.003
					(0.021)
Constant	0.116***	-0.645***	0.177	0.251	0.226
	(0.028)	(0.133)	(0.233)	(0.230)	(0.225)
Observations	1,598	1,598	1,529	1,499	1,497

Notes: The table reports the ITT estimates for mortality observed over 4 years (equation 2) including covariates related to the mortality risk. The models use triangular kernel, local linear polynomial and the optimal bandwidth for point estimation as suggested by Calonico et al. (2015) (obtained in Table 2). The standard errors are clustered by the Primary Sampling Unit (PSU) of the sampling framing and are indicated in parenthesis. $*p < 0.10$, $**p < 0.05$, and $***p < 0.01$ indicate statistical significance levels.

Adding age and sex reduces the ITT estimate from -0.107 to -0.099 (column 2). The estimate is -0.077 when objectively measured health indicators are included (column 3), -0.095 with self-reported health and nutrition measures (column 4), and -0.089 after additionally accounting for alcohol and tobacco use (column 5). All remain statistically significant at conventional levels, although precision varies across specifications. The sample is not constant: it contains 1,598 observations in columns 1–2 and falls to 1,529, 1,499 and 1,497 in columns 3–5 because of missing covariate values. Thus, changes across columns reflect both adjustment and differences in the estimation sample.[13]

[13]Age and sex enter linearly in columns 2 and 5. Using single-year age indicators (ages 65–80) interacted

A further check allows the bandwidth selector to account for the covariates. The selected bandwidth is ± 0.141, close to our preferred ± 0.150, and the estimated ITT is -0.088 using conventional estimation (-0.102 with robust bias correction). Overall, the negative mortality effect persists after adjustment for baseline characteristics, consistent with the validity checks in Section 4.

The coefficients on the baseline covariates are also informative about mortality risk in this population, although they should not be interpreted causally. Older age, male sex, elevated blood pressure and higher weight are associated with greater mortality, in line with evidence on cardiovascular risk (Arima et al., 2011; Lev-Ari et al., 2021; Lee et al., 2022). Abdominal obesity, defined using Latin American thresholds (ALAD, 2010; Pajuelo-Ramirez et al., 2019), has a positive but statistically insignificant coefficient. By contrast, greater mid-upper arm and calf circumference, proxies for muscle mass and nutritional status, predict lower mortality; a one-centimetre difference has an association comparable in magnitude to being one or two years younger (Bernal et al., 2024). Better cognitive functioning, measured using an abbreviated Mini-Mental State Examination (Folstein et al., 1975; Leist et al., 2020), and better nutrition, measured by the Mini Nutritional Assessment (Guigoz, 2006; Harris and Haboubi, 2005; Vellas et al., 1999), are also associated with lower mortality.

5.4 Heterogeneous effects

We next examine whether the mortality effect differs by sex, age, area of residence and education using equation 3. Figure 6 reports subgroup ITT estimates. Eligibility is associated with statistically significant mortality reductions among women and men, rural residents, individuals with no schooling, and those aged 71–80. Estimates for urban residents, individuals with some schooling and those aged 65–70 are not statistically distinguishable from zero. These differences in subgroup significance do not, on their own, establish statistically significant differences between groups.[14]

Because a binary age split can conceal a gradual pattern, we also interact eligibility with age measured continuously. The interaction is -0.0067 ($p = 0.055$), corresponding to an additional mortality reduction of about 0.7 pp per year of age. The implied ITT rises in magnitude from -6.0 pp at age 65 to -16.1 pp at age 80. This pattern is suggestive of larger effects at older ages, although the interaction falls just short of conventional 5% significance.

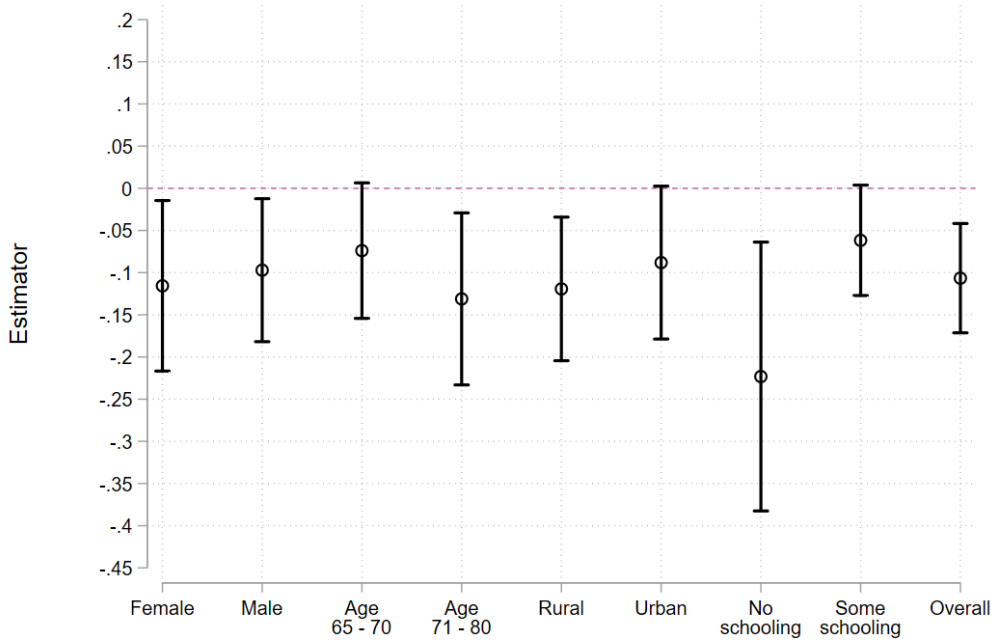
We also assess heterogeneity by household assets, using an index constructed from baseline housing characteristics and durable-goods ownership following Filmer and Pritchett (2001). Because the SISFOH score itself determines eligibility, we use this separate principal-component measure rather than treating the running variable as a wealth moderator. The first component

with sex instead produces estimates of -0.094 rather than -0.099 without other controls and -0.084 rather than -0.089 with the full set.

[14] Educational attainment is low in ESBAM: 27% of respondents have no schooling, 52% have incomplete primary schooling and 14% completed primary education.

explains only 11% of total variation, reflecting the limited dispersion of housing conditions in this highly disadvantaged sample. Appendix Table I–1 (Appendix I) shows no detectable interaction with the continuous index (0.0004; $p = 0.977$). A median split yields ITTs of -13.1 pp and -9.1 pp for lower- and higher-asset households, respectively, with overlapping confidence intervals. The evidence does not support differential effects by this measure within the ESBAM sample, but the index's limited variation constrains what can be inferred about broader wealth gradients.

Figure 6: Heterogeneous effects on mortality rate



Notes: The figure reports subgroup ITT estimates from equation 3, alongside the overall estimate. Age is split at 70 years; the text also reports a specification with age interacted continuously with eligibility. Vertical bars denote 95% confidence intervals.

Finally, we find no statistically detectable heterogeneity by whether the household receives transfers from *Juntos*, Peru's conditional cash transfer programme, or by the presence of more than one potentially eligible older adult in the household.[15]

5.5 Exposure to health campaigns

The Ministry of Health and *Pension 65* jointly organise local healthcare campaigns. Although these campaigns are directed towards pension recipients, older adults affiliated with the public health insurance scheme (SIS) can also attend, regardless of pension eligibility. Individual

[15] *Juntos* supports households with children or pregnant women subject to health and education conditions. Its poverty eligibility criteria include both extreme-poor and non-extreme-poor households, so individuals on either side of our cut-off may qualify. In unreported estimates, the mortality ITT is -11.9 pp for households with one potentially eligible older adult and -8.9 pp for those with two.

attendance is not observed, but administrative records identify campaigns by district and month. We use these data to calculate the average annual number of campaigns in each individual's district during 2013–2016.

Appendix Table D–3 shows that greater district-level campaign exposure is associated with lower mortality, while the estimated ITT of pension eligibility remains close to the baseline result (-11.3 pp). One additional campaign per year is associated with a 1.0 pp reduction in mortality. Interacting campaign exposure with eligibility yields a small and statistically insignificant coefficient (0.0002; standard error 0.004), so we retain the additive model. The corresponding interaction in the survival analysis is also insignificant (-0.423; standard error 0.381; Appendix Table D–4). Because campaign placement is not randomly assigned, these associations should not be interpreted as causal effects of the campaigns. Nor do the estimates establish whether individual recipients attended them.

5.6 ITT effects with survival models

The preceding estimates concern the probability of dying within four years. Survival models additionally use the timing of deaths. Following equation 4, we estimate the discontinuity in the mortality hazard using a Gompertz proportional-hazards model. Table 4 reports the results. At the preferred bandwidth, the estimated log-hazard effect is -1.680, corresponding to a hazard ratio of $\exp(-1.680) \approx 0.19$. Thus, eligible individuals have an estimated instantaneous mortality hazard approximately 81% lower than that of ineligible individuals at the cut-off, under the model's assumptions. This hazard-ratio reduction should not be interpreted as an 81% reduction in four-year mortality probability.

Table 4: Effect of *Pension 65* on log of mortality hazard rate

	(1)	(2)	(3)
Intention-to-treat ($\hat{\beta}_1$)	-0.739	-1.109	-1.680
	(0.259)	(0.356)	(0.525)
	[0.004]	[0.002]	[0.001]
Constant ($\hat{\beta}_0$)	-3.771	-3.845	-3.878
	(0.203)	(0.283)	(0.319)
	[0.001]	[0.001]	[0.001]
Bandwidth	+/- 0.330	+/- 0.2	+/- 0.150
Observations	3,885	2,104	1,598
Percentage Sample	Full sample	54%	41%

Notes: The table reports the ITT estimates for the log of mortality hazard rate observed during 4 years (equation 4). The estimator corresponds to a Gompertz-type model. The models use triangular kernel, local linear polynomial and the optimal bandwidth for point estimation as suggested by Calonico et al. (2015) (obtained in the last model of Table 2). The standard errors are indicated in parenthesis. P-values are reported in brackets.

The estimates remain qualitatively similar under exponential, Weibull and Cox specifications (Appendix Table C–1). Appendix Figure C–1 illustrates the survival discontinuity, while

Figure C–2, Figure C–3 and Table C–2 report bandwidth, subgroup and covariate checks. We also estimate separate linear RDs for each follow-up month to examine the evolution of mortality without imposing the Gompertz hazard shape (Section 7).

5.7 Potential mechanisms

The mortality reduction may reflect improvements in nutrition, health or access to care. Using the 2015 ESBAM follow-up, Bernal et al. (2024) finds that *Pension 65* reduces anaemia and depressive symptoms while improving dietary quality, cognitive functioning, self-reported health, healthcare utilisation and anthropometric markers associated with mortality. We extend this analysis using the same follow-up to examine which outcomes respond most consistently to eligibility, including health insurance and preventive care.

Of the 3,885 baseline respondents, 3,512 were reinterviewed in 2015 (90.4%). Among the 373 not reinterviewed, 208 had died and 165 could not be located, implying a loss to follow-up other than death of 4.2% of the baseline sample. We recover more matched observations than Bernal et al. (2024) (3,512 versus 3,351) by manually identifying respondents with initially missing SISFOH scores. Importantly, the main mortality analysis uses administrative records for the baseline cohort and does not depend on reinterview; however, analyses of follow-up outcomes necessarily condition on survival and survey response.

Appendix Table G–1 presents three specifications: estimates using the full follow-up sample, local linear estimates using the ± 0.150 bandwidth and triangular kernel employed for mortality, and outcome-specific local polynomial estimates with MSE-optimal bandwidths following Calonico et al. (2015). The full-sample results are broadly consistent with Bernal et al. (2024). At our preferred mortality bandwidth, eligibility is associated with better cognitive functioning and self-reported health, more reported chronic conditions and greater use of healthcare centres, alongside lower household food expenditure per capita. The last finding should be interpreted cautiously: per capita expenditure depends on household size, and the programme is also associated with an increase in the number of household members. Under outcome-specific bandwidths, healthcare-centre attendance remains significant at the 5% level, while reductions in hypertension and abdominal obesity and improvements in mid-upper arm circumference and self-reported health are significant only at the 10% level. Food expenditure per capita also falls weakly ($p = 0.082$), but total household food expenditure does not show a detectable response under this specification.

Healthcare-centre attendance is the most consistent result across the three approaches, and self-reported health relative to the previous year also improves in each. By contrast, we find no detectable increase in SIS affiliation or preventive care. At ± 0.150, the standardised ITT for affiliation is 0.027 (standard error 0.161; $N = 1,444$); expressed in percentage points, the estimated discontinuity in 2015 affiliation is 1.0 (standard error 6.0), and among individuals uninsured at baseline it is -4.3 (standard error 12.1). Preventive care, measured using recent

screenings, check-ups and influenza vaccination, has an estimated standardised effect of 0.007 (standard error 0.176; $N = 1,444$).[16]

The follow-up also suggests lower labour supply in the full sample: working hours and the probability of working fall by 0.298 and 0.311 standard deviations, respectively (both significant at the 1% level). Neither remains statistically significant at the preferred mortality bandwidth (-0.109 and -0.096 standard deviations, respectively), so we do not regard this as a robust channel. Overall, the evidence points most consistently to greater healthcare utilisation, rather than expanded insurance enrolment or preventive care. Since SIS was available on both sides of the threshold, the transfer may have helped recipients cover transport, medication or other costs of using existing services. These results are suggestive rather than a formal mediation analysis, particularly because follow-up health outcomes are observed only for survivors.

5.8 Limitations

Several limitations qualify the interpretation of our findings. First, the validity checks in Section 4 reveal economically meaningful differences in some predetermined characteristics, despite the absence of significant discontinuities after multiple-testing corrections. Covariate adjustment does not eliminate the mortality effect, but the remaining imbalances warrant caution. Second, the RDD identifies a local effect for ESBAM respondents close to the extreme-poverty threshold and cannot be generalised without qualification to all potential beneficiaries. Third, the 2015 follow-up outcomes are observed only for surviving, reinterviewed individuals, and the available data do not identify distinct causal pathways from income to mortality. Food expenditure per capita is additionally affected by household composition. Finally, the fuzzy RD treatment-receipt estimates depend on take-up measured during the same period as mortality and are subject to the timing concern discussed in Section 5.2. For these reasons, we place greatest weight on the reduced-form effect of baseline eligibility on mortality.

6 Policy implications

6.1 Life expectancy gains

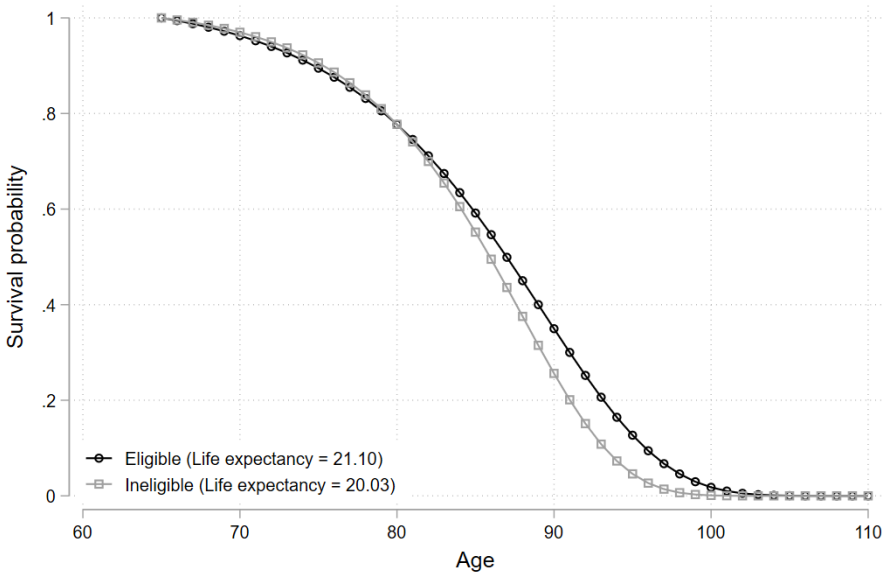
To place the mortality estimates in a longer-term perspective, we use an actuarial exercise to approximate differences in remaining life expectancy between eligible and ineligible individuals.

[16] SIS affiliation rose between baseline and follow-up on both sides of the cut-off: from 63.3% to 76.3% among ineligible individuals and from 63.9% to 85.4% among eligible individuals within the bandwidth. Although baseline shares are similar, the 2012 local linear discontinuity is 13.2 pp (standard error 7.3; $p = 0.070$). Controlling for baseline affiliation changes the main mortality ITT from -0.107 to -0.111 (standard error 0.033; $N = 1,596$). Baseline affiliation itself is positively associated with mortality (0.032; standard error 0.015), consistent with selection into insurance among individuals in poorer health. The full-follow-up-sample effect on SIS affiliation is 0.171 standard deviations, significant only at the 10% level, and does not persist at the mortality bandwidth or in the outcome-specific specification.

We calculate annual mortality rates by attained age from ESBAM observations during 2012–2016, pooling information across birth cohorts for each age. We then fit Gompertz mortality functions and derive survival curves from age 65 for the two groups. Because estimating age-specific mortality requires sufficient observations, this exercise uses the full ESBAM sample rather than the narrower bandwidth of our RDD.[17]

Figure 7 shows that the fitted survival curve lies above that of the ineligible group for eligible individuals. The implied remaining life expectancy at age 65 is 21.10 years for eligible individuals and 20.03 years for ineligible individuals, a difference of 1.07 years. For context, official life tables for Peru's Private Pension System (SPP) report 23.43 years of remaining life expectancy at age 65 for both sexes combined, although that population is not directly comparable to ESBAM respondents.

Figure 7: Estimated survival curves by *Pension 65* eligibility



Notes: Survival curves are fitted separately for eligible and ineligible individuals using the full ESBAM sample and mortality observed during 2012–2016. The estimates assume a Gompertz mortality function and extrapolate survival beyond the observation period. They are descriptive actuarial projections, not RDD estimates.

This life expectancy difference should not be interpreted as a causal effect of pension eligibility. Unlike the four-year ITT and the hazard ratio estimated at the eligibility cut-off (Table 4), the actuarial projections compare the full eligible and ineligible samples and extrapolate mortality beyond the observed period. Differences in baseline characteristics may therefore contribute to the estimated gap. In particular, the eligible group is poorer by construction and may face higher underlying mortality risk, which complicates the comparison even if it works against observing a survival advantage. The estimated 81% reduction in the hazard at the threshold

[17]For example, mortality between ages 70 and 71 is estimated by pooling observations reaching those ages in different calendar years. The procedure combines information from the birth cohorts represented in the survey (approximately 1932–1947), implicitly treating their age-specific mortality experiences as comparable.

and the 1.07-year life expectancy gap thus refer to different populations, time horizons and estimands. The actuarial calculation is informative about the potential magnitude of longevity differences, but it does not directly convert the local RDD effect into years of life gained.

6.2 Illustrative cost comparison

We next compare the transfer cost associated with the actuarial life expectancy difference with published valuations of mortality-risk reductions. The value of a statistical life (VSL) measures willingness to pay for small reductions in the risk of death and is commonly used in policy appraisal (Viscusi, 1994; Robinson et al., 2019). Appendix Table E–1 reports estimates and extrapolations for Peru in 2012 ranging from US$0.36 million to US$3.35 million, with a mean of US$0.98 million. Because these valuations are not estimated directly from the ESBAM population, their application to poor older adults in Peru also requires caution.

Following the illustrative approach of Miglino et al. (2023), we calculate the discounted present value of pension payments over 21.10 years of expected remaining life at age 65, assuming continuous receipt and using a 3% discount rate, and divide it by the estimated 1.07-year difference in life expectancy. The result is US$8,343 (in 2012 dollars) per additional year of life. Scaling this amount by the 21.10 years of remaining life expectancy yields approximately US$176,000, equivalent to 18% of the mean and 28% of the median VSL estimates for Peru.[18]

This comparison is necessarily approximate. In particular, scaling a cost per additional life-year by remaining life expectancy does not provide a directly identified cost per statistical life saved, and the underlying longevity difference is not a causal RDD estimate. Moreover, the calculation includes pension payments but not the potential additional public cost of healthcare use (Table G–1), administrative costs or the marginal cost of public funds. A fuller welfare appraisal would also need to value the redistribution generated by the pension and account for fiscal externalities (see Hendren and Sprung-Keyser, 2020). We therefore interpret the calculation as an illustrative benchmark, not a complete cost-benefit analysis.

6.3 Mortality–income elasticity

To compare the magnitude of our findings with those of other social pension programmes, we calculate an illustrative mortality–income elasticity: the percentage difference in mortality divided by the percentage increase in income associated with the transfer. Within the preferred bandwidth (± 0.150), we use triangular-kernel-weighted means and augment the baseline monetary income of household members aged 65 or older by the monthly pension amount (125 soles) for eligible individuals. This focuses on the income resources of older household members, rather than those of the entire household, and assumes that eligible individuals receive

[18] Our actuarial method differs from that of Miglino et al. (2023), who assume a common mortality profile for treatment and control groups after their four-year observation period; we fit separate mortality functions for the two groups.

the transfer while ineligible individuals do not. On this basis, average income rises by 46.7%, while the corresponding weighted mortality rate is 38.1% lower. Their ratio gives an elasticity of -0.82, with a 95% confidence interval of $[-1.53, -0.24]$ from a bootstrap clustered at the primary sampling unit.[19]

Two choices matter for interpreting this figure. First, we use income received by older household members rather than total household income, reflecting the fact that the pension is paid to older individuals and improving comparability with studies based on applicants' or recipients' own income (Miglino et al., 2023; Menares et al., 2026). Using total household income instead yields a larger and much less precise elasticity of -1.80.[20]

Second, our calculation uses weighted group means within the bandwidth rather than the discontinuity in income and the fitted mortality mean at the cut-off, as in Miglino et al. (2023). The estimated income discontinuity at the threshold is imprecise (97.3 soles; standard error 53.7), which makes a ratio based on that estimate unstable. Threshold-based elasticities range from -3.1 to -5.3, depending on how transfer receipt is measured, and their bootstrap intervals include zero. By contrast, replacing the assumed transfer with payments actually received while retaining the group-means approach gives -1.20 (95% interval $[-2.76, -0.45]$).[21] We retain -0.82 as a transparent, comparatively stable summary of the mortality and income differences within the bandwidth. Because it is constructed from group means, however, it should not be read as a separately identified causal income elasticity from the RDD.

For additional context, Appendix Figure D–3 (Appendix D) plots the cross-sectional relationship between baseline income and four-year mortality across income deciles in ESBAM. The corresponding regression slope is -0.000036 in the probability of death per sol (standard error 0.000013; $p = 0.005$; $N = 3{,}885$), compared with -0.0011 obtained by dividing the local ITT mortality estimate by the estimated income discontinuity of 97.3 soles. These figures capture different relationships: the former is an unconditional association across the full sample, potentially affected by confounding and measurement error in reported income, while the latter combines a local eligibility effect with an imprecisely estimated income change. Neither comparison identifies a separate causal effect of income, and their difference should not be interpreted as direct evidence of the degree of confounding or attenuation in the cross-sectional gradient.

Our preferred elasticity is larger in magnitude than estimates for Chile's *Pensión Básica*

[19] The weighted income comparison is 275.2 versus 403.7 soles, and the corresponding four-year mortality rates are 10.3% versus 6.4%. The confidence interval is the percentile interval from 1,000 bootstrap replications resampling primary sampling units. The income increase is constructed by adding the pension amount; it is not an independently estimated causal effect of eligibility on observed post-transfer income.

[20] With total household monetary income, the implied income increase is 21.2%; the bootstrap standard error of the resulting elasticity is 6.05, and its 95% percentile interval is $[-12.0, -0.42]$ (1,000 PSU-clustered replications). Other studies use household or area-level income measures (Huang and Zhang, 2021; Barham and Rowberry, 2013; Jensen and Richter, 2004).

[21] The threshold-based elasticity is -3.1 when the pension amount is added mechanically (95% percentile interval $[-19.7, 10.1]$) and -5.3 when income reflects payments received over the observation period (interval $[-78.3, 54.9]$); both intervals are based on 1,000 PSU-clustered bootstrap replications.

Solidaria (−0.386 for four-year mortality; Miglino et al., 2023) and China's New Rural Pension Scheme (−0.38 for one-year mortality; Huang and Zhang, 2021). For Mexico's *70 y Más*, Menares et al. (2026) report an elasticity of −0.23 among women, for whom the programme reduces annual mortality by 5.5%, with no detectable effect among men. Appendix Figure F–1 (Appendix F) also includes estimates derived from studies of Union Army veterans' pensions, Mexico's *Progresa* and the US Social Security Notch (Salm, 2011; Barham and Rowberry, 2013; Snyder and Evans, 2006); the last reports higher male mortality associated with higher income.

These comparisons warrant caution. Studies differ in the definition of income (individual, household or area averages), the population covered, the mortality horizon (ranging from one to five years) and the method used to construct the elasticity. In particular, the mortality and income effects in Huang and Zhang (2021) are estimated using different samples and age groups. Moreover, our confidence interval includes the Chilean and Chinese point estimates. For the other studies, the confidence intervals reported in Appendix Figure F–1 account for sampling uncertainty in mortality effects but hold income changes fixed, unlike our PSU-clustered bootstrap interval.

Nevertheless, the difference in targeting offers one plausible interpretation. Chile's pension reaches the poorest 60% of the population, whereas *Pension 65* is restricted to older adults living in extreme poverty. If the income–mortality gradient is steeper at very low income levels (Case et al., 2002; Deaton, 2002), a transfer may have a larger effect on survival in our population. Lifetime disadvantages in nutrition, education and access to healthcare could reinforce this pattern. The greater attendance at healthcare centres documented in Section 5.7, without corresponding increases in SIS enrolment or preventive care, suggests one possible channel, although the available evidence does not establish mediation or explain the cross-country differences. The comparison is therefore consistent with, but cannot establish, a stronger mortality response to income among the poorest older adults.

7 Falsification and robustness checks

Building on the validity tests in Section 4, we conduct additional falsification and robustness exercises. We examine placebo cut-offs, exclude observations closest to the eligibility threshold, and assess sensitivity to alternative model specifications and mortality follow-up periods. We also investigate whether baseline covariate imbalances affect our findings and complement the LATE estimates with a non-linear specification that constrains predicted mortality probabilities to the unit interval.

7.1 Placebo cut-offs

We first test for discontinuities in mortality at six placebo cut-offs, symmetrically located around the actual threshold: -0.25, -0.15, -0.075, 0.075, 0.15 and 0.25. Following Cattaneo et al. (2020), each test uses only observations on the same side of the true threshold as the corresponding placebo cut-off, so that the estimation window does not contain the actual eligibility discontinuity. Appendix Table D–1 reports the results for the linear and survival models. None of the placebo estimates is significant at the 5% level. The only estimate significant at the 10% level is from the survival model at -0.075 ($p = 0.074$, $N = 462$); its linear counterpart is not significant ($p = 0.140$). These results provide no systematic evidence of mortality discontinuities away from the programme eligibility threshold.

7.2 Sensitivity to observations near the cut-off

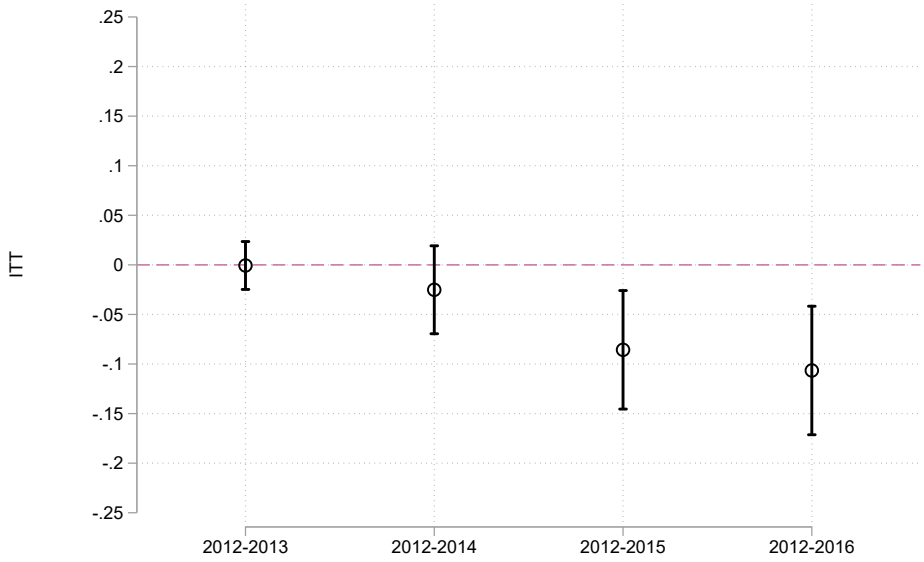
We next assess whether the estimates are driven by observations immediately adjacent to the cut-off. Following the donut-hole approach discussed by Cattaneo et al. (2020), we re-estimate the models after excluding observations with absolute centred SISFOH scores below 0.002, 0.004, 0.006, 0.008 and 0.010. Such exclusions also probe the sensitivity of local polynomial estimates to observations with high influence near the point of extrapolation. Appendix Table D–2 shows that the mortality effects remain negative and the substantive conclusions are unchanged in both the linear and survival specifications.

7.3 Alternative specifications and follow-up periods

The main findings are also robust to alternative bandwidths. Figure 4 presents the linear ITT estimates, while Appendix Figure C–2 reports the corresponding survival-model estimates. Across the bandwidths examined, effects remain negative and statistically significant, although their magnitude generally declines as the estimation window widens.

We also examine how the estimated effect evolves with the length of follow-up. Figure 8 reports the ITT on cumulative mortality from the 2012 baseline to December of each subsequent year, using the preferred bandwidth of ± 0.150. Effects are not statistically distinguishable from zero over the first two years but become statistically significant after three years. The estimates provide no clear evidence of a further change in the effect between the third and fourth years.

Figure 8: ITT effects on cumulative mortality by follow-up period

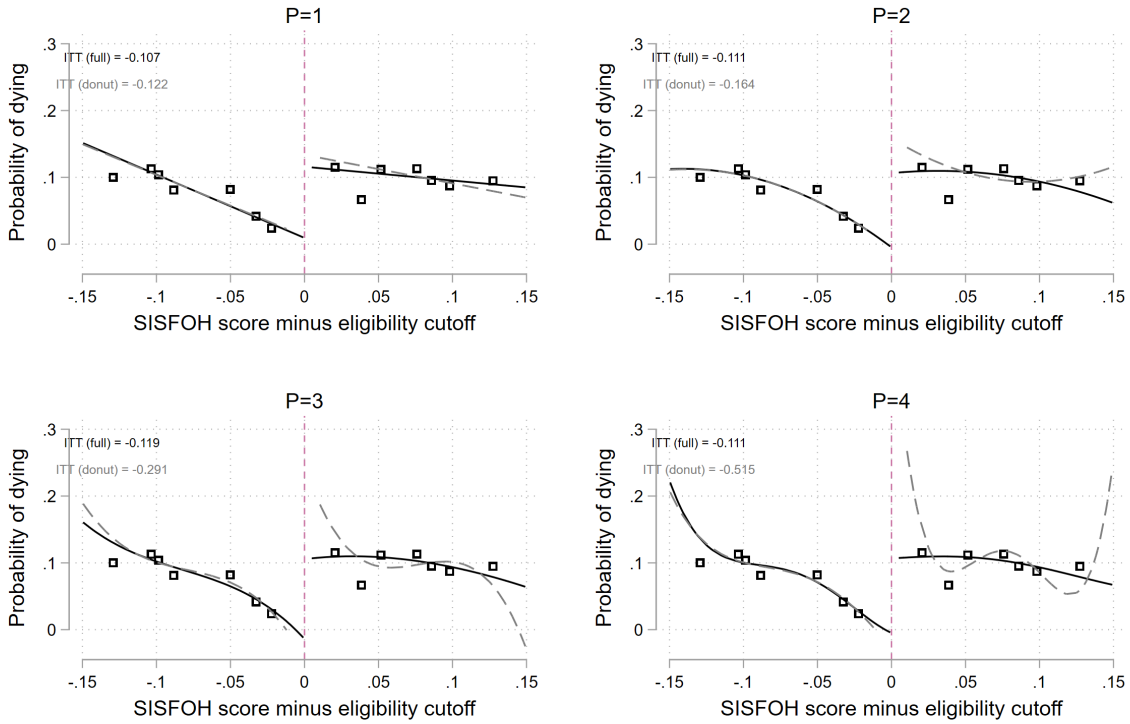


Notes: The figure reports ITT estimates for cumulative mortality from the 2012 baseline survey to December of each subsequent year. Estimates use equation 2, local linear regressions, a triangular kernel and the preferred bandwidth of ±0.150 selected for four-year mortality (Table 2). Vertical lines denote 95% confidence intervals.

Appendix Figure D–2 provides a finer description by estimating the cumulative mortality effect at each month of follow-up. Estimates are not significant through month 33, become significant at month 34, and remain significant through month 50 (December 2016), when the coefficient reaches -0.107. Because outcomes are cumulative and the monthly tests are highly related, the first significant month should not be interpreted as the precise onset of the programme's mortality effect.

Finally, Figure 9 compares polynomial orders one through four within the preferred bandwidth, both with and without observations within 0.010 of the cut-off. The local linear estimate changes only modestly, from -0.107 to -0.122, when these observations are excluded. Higher-order fits are considerably less stable, with estimates reaching -0.164, -0.291 and -0.515 for polynomial orders two, three and four, respectively. This sensitivity is consistent with the concerns raised by Gelman and Imbens (2019) about higher-order polynomial specifications in RDD applications and supports our reliance on local linear estimation.

Figure 9: Sensitivity of ITT estimates to polynomial order and donut-hole exclusion



Notes: The figure plots estimates from equation 2 using polynomial orders one to four and a bandwidth of ±0.150. Solid black lines include all observations within this bandwidth; dashed grey lines exclude observations within 0.010 of the threshold. Squares show binned mortality means, with bin selection following Calonico et al. (2015). Estimated discontinuities are reported in each panel. Individuals to the left (right) of the vertical dashed line are eligible (ineligible) for *Pension 65*.

7.4 Predicted-mortality falsification test

Section 4 documents some sizeable, though generally statistically insignificant, differences in predetermined characteristics at the cut-off. To assess whether these observed differences could account for the mortality result, we first predict mortality using baseline covariates and then use the predicted probability as the outcome in equation 2. If the observed mortality discontinuity were explained by these measured characteristics, we would expect a corresponding discontinuity in predicted mortality. Table 5 instead reports an estimate close to zero, with a confidence interval that excludes the main ITT estimate of −0.107. The result is similar whether the prediction model includes all available covariates or only those selected by a stepwise procedure.[22]

[22]These exercises test the implications of observed covariate differences under the fitted prediction models; they cannot exclude bias from unobserved determinants of mortality.

Table 5: Testing the effect of *Pension 65* on actual and predicted mortality

	(1)	(2)	(3)
Intention-to-treat ($\hat{\beta}_1$)	-0.107	-0.013	-0.006
	(0.033)	(0.010)	(0.012)
	[0.001]	[0.205]	[0.586]
95% Conf. Interval	(-0.171, -0.042)	(-0.033, 0.007)	(-0.030, 0.017)
Bandwidth	+/- 0.150	+/- 0.105	+/- 0.093
Observations	1,598	1,337	763

Notes: The table reports the ITT estimates for mortality observed in 4 years (Equation 2). The models use triangular kernel and local linear polynomial. The first column reports the ITT effect of our main model on actual mortality (column 3 of Table 2), while columns 2 and 3 report the ITT effect on predicted mortality. In the second column, the mortality rate has been predicted using all the predetermined covariates that were employed in the balance test. In the third column, the mortality rate has been predicted using only those covariates that were statistically significant (using a stepwise regression). The optimal bandwidth for point estimation is used, as suggested by Calonico et al. (2015). The standard errors are clustered by the Primary Sampling Unit (PSU) of the sampling framing and are indicated in parentheses. P-values are reported in brackets.

7.5 Bounded LATE

The linear Wald estimate in Section 5.2 is -0.140 at the preferred bandwidth. Adding this estimate to the fitted mortality rate for ineligible individuals at the cut-off (11.6 pp) produces a negative value of -2.4 pp. This is not, strictly speaking, an estimate of mortality among treated compliers, whose counterfactual mortality need not equal the overall ineligible-group rate. Nevertheless, it motivates a complementary specification that respects probability bounds. We estimate a recursive bivariate probit model that jointly specifies programme receipt and mortality, with eligibility entering the receipt equation but not the mortality equation. This model constrains predicted mortality probabilities to the $[0, 1]$ interval.

At the cut-off, the model predicts mortality of 17.5 pp without the pension and 2.7 pp with it, yielding an estimated effect of -14.9 pp (SE 4.3; 95% CI $[-23.3, -6.4]$). Allowing the running-variable slope in the mortality equation to differ at the cut-off gives an estimate of -16.3 pp (SE 4.5). Appendix H describes the model and reports the results in Table H–1, Panel B. These estimates show that the negative effect is not specific to a linear probability model, although the non-linear specification requires additional assumptions and does not remove the challenges of measuring take-up over the same period as mortality. For this reason, we continue to emphasise the reduced-form ITT estimates.

8 Conclusions

We examine the effects of Peru's non-contributory pension programme, *Pension 65*, on mortality among older adults living in extreme poverty. Exploiting a discontinuity in the welfare index determining programme eligibility, we find that eligibility reduces four-year mortality by 10.7 percentage points at the threshold. Our analysis combines a baseline survey specifically designed for a regression discontinuity design with administrative programme and mortality

records for 2012–2016. The result remains robust to alternative bandwidths, adjustments for baseline health and nutritional characteristics, and survival-model specifications, and is supported by several falsification tests.

The economic significance of this finding is substantial. An illustrative actuarial calculation suggests a difference of approximately one year in remaining life expectancy at age 65 between eligible and ineligible individuals. We also estimate a mortality–income elasticity of -0.82, larger in magnitude than estimates reported for comparable programmes in Chile and China. Although these estimates are not directly comparable, the difference is consistent with a stronger mortality response to income transfers among the poorest older adults, who face particularly severe economic and health constraints.

Our findings also provide suggestive evidence on the mechanisms underlying the mortality reduction. Eligibility increases visits to healthcare centres but has no detectable effect on public health insurance enrolment or preventive care. Since public insurance was already widely available on both sides of the eligibility threshold, the transfer may have enabled recipients to make greater use of existing healthcare services by easing financial constraints associated with accessing care. However, our analysis does not establish the extent to which increased healthcare utilisation accounts for the mortality effect.

Overall, our findings highlight the potential of even modest social pensions to improve survival among older adults facing severe economic deprivation. Our estimates are local to individuals near the eligibility threshold in the ESBAM sample and may not generalise to all potential beneficiaries. A fuller assessment of the programme's welfare implications would also need to account for the public costs of additional healthcare utilisation and the marginal cost of public funds. More broadly, our results are consistent with the hypothesis that the mortality benefits of social pensions may be greater among the poorest older adults, a proposition that future research could test across programmes with different targeting criteria.

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Appendix

A Definition of variables

Table A–1: Definition of variables

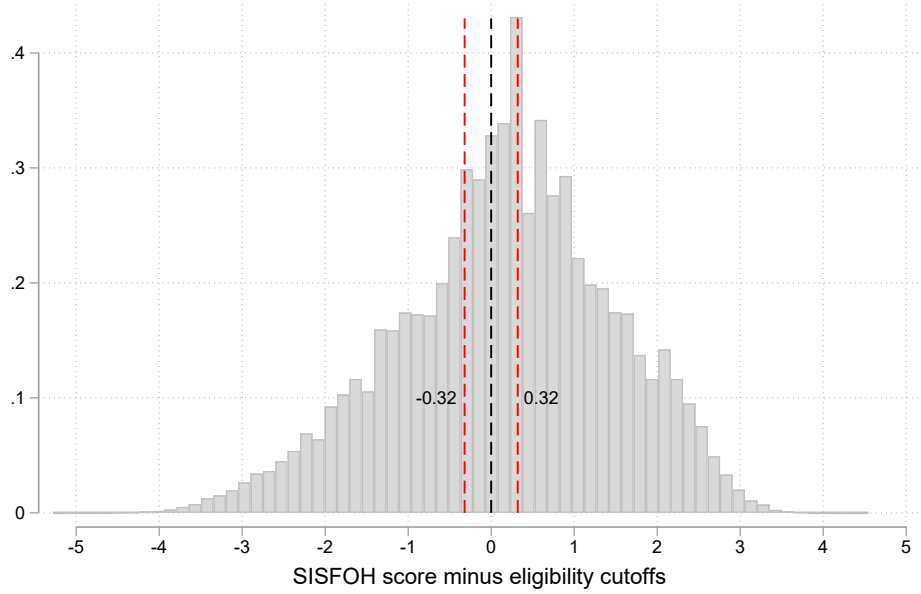
Variable	Definition
Male	It takes value 1 if the individual is male, and 0 if female.
Age	Age in years in 2012.
High blood pressure	It takes value 1 if the systolic blood pressure is greater than or equal to 140 (mm Hg) or if diastolic blood pressure is greater than or equal to 90 (mm Hg), and 0 otherwise.
Anaemia	It takes value 1 if the individual has anaemia, and 0 otherwise. The anaemia condition is determined according to haemoglobin levels analysed from blood samplings taken during the interview.
Weight	Body weight in kilograms.
Abdominal obesity	It takes value 1 if the waist measure is larger than the cut-offs that indicate obesity according to the norms set up by the Latin American Diabetes Association (see ALAD 2010 and Pajuelo-Ramirez et al. 2019), and 0 otherwise. These cut-offs are 94 cm and 88 cm for men and women, respectively.
Arm span	Arm span in centimetres.
Mid-upper arm circumference (MUAC)	Upper middle arm circumference in centimeters.
Calf circumference (CC)	Calf circumference in centimetres.
Cognitive functioning	Is a score (0-13) computed from the points assigned to correct answers for four questions. *Orientation*: day of the week, day of the month, month, and year. *Immediate memory*: recall of three words read by the interviewer. *Delayed memory*: recall of the exact words again later in the interview. *Command*: three actions that the respondent must complete in order: "I will give you a piece of paper. Take this in your right hand, fold it in half with both hands and place it on your legs".
Chronic diseases	It is the total number of chronic medically diagnosed diseases reported by the individual from a list of 13 diseases.
Health today	It takes value 1 if the individual rates her health as good or very good from a 1-4 Likert scale, and 0 otherwise.
Health compared to last year	It takes value 1 if the individual rates her health as the same, better or much better with respect to last year from a 1-5 Likert scale, and 0 if she rates her health as worst or much worst.
Health compared to others	It takes value 1 if the individual rates her health as good or very good with respect to other people of similar age from a 1-4 Likert scale, and 0 otherwise.
Subjective health index	It is an index computed with the three subjective health questions mentioned before. First, we standardised the original variables containing their Likert-scale values (mean and standard deviation equal to 0 and 1) and sum up them. Second, we re-scale this value to obtain an index ranging between 0 an 1. Thus, larger values imply better perceived health.

Table A–2: Definition of variables

Variable	Definition
Mini Nutritional Assessment (MNA) score	Is a score measuring the quality of diet and the risks of under-nutrition and malnutrition among old individuals. The scores originally ranged from 0 to 30, but the available information in ESBAM allows us to compute a score ranging between 0 and 19. The information to compute the MNA includes variables indicating whether the individual i) eats three or more meals per day; ii) eats dairy products at least once a day; iii) eats fruits and vegetables at least twice a day; iv) drinks less than three glasses of water per day; v) eating eggs, beans or legumes at least once a week; vi) eats meat, fish or poultry at least three times a week.
Alcohol	It takes value 1 if the individual drank alcohol at least once during the three months previous to the interview, and 0 otherwise. It is measured in the baseline ESBAM survey, but not on the follow-up survey.
Tobacco	It takes value 1 if the individual smoked during the month previous to the interview (or before), and 0 otherwise. It is measured in the baseline ESBAM survey, bu not on the follow-up survey.
Depression symptoms	It is the number of depression symptoms (score 0-9) measured with the geriatric depression scale from Sheikh and Yesavage (1986). This was measured only in the follow-up survey.
Juntos	It takes value 1 if the individual declared the household is the recipient of the conditional transfer program *Juntos*, and 0 otherwise.
Attended health centre	It takes value 1 if the individual who had any disease or symptom in the last month went to a health centre to treat them, and 0 otherwise. The value is set to missing for people who had no any disease in the last month.
Individual health expenditure	Expenditure (Soles per month) used by the individual in health services.
Individual medicine expenditure	Expenditure (Soles per month) used by the individual to buy medicines.
Working hours	Total number of hours worked in the previous week, including main and secondary occupations.
Working	It takes value 1 if the individual worked the previous week, and 0 otherwise.
Household expenditure	Total household expenditure (Soles per month).
Household food expenditure	Household expenditure on food (Soles per month).
Number of household members	It is the number of members residing permanently in the household.

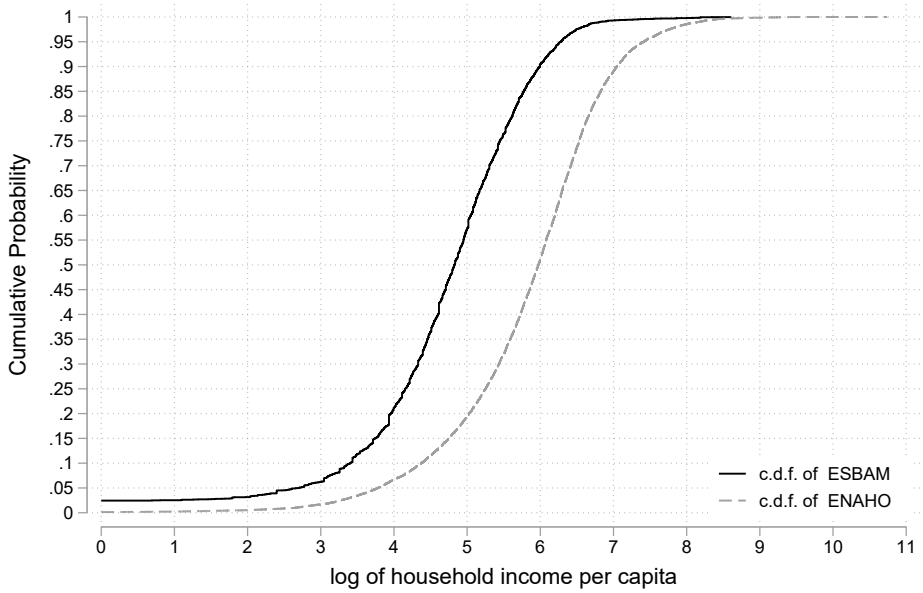
B Descriptive tables and figures

Figure B–1: National distribution of the centred SISFOH score



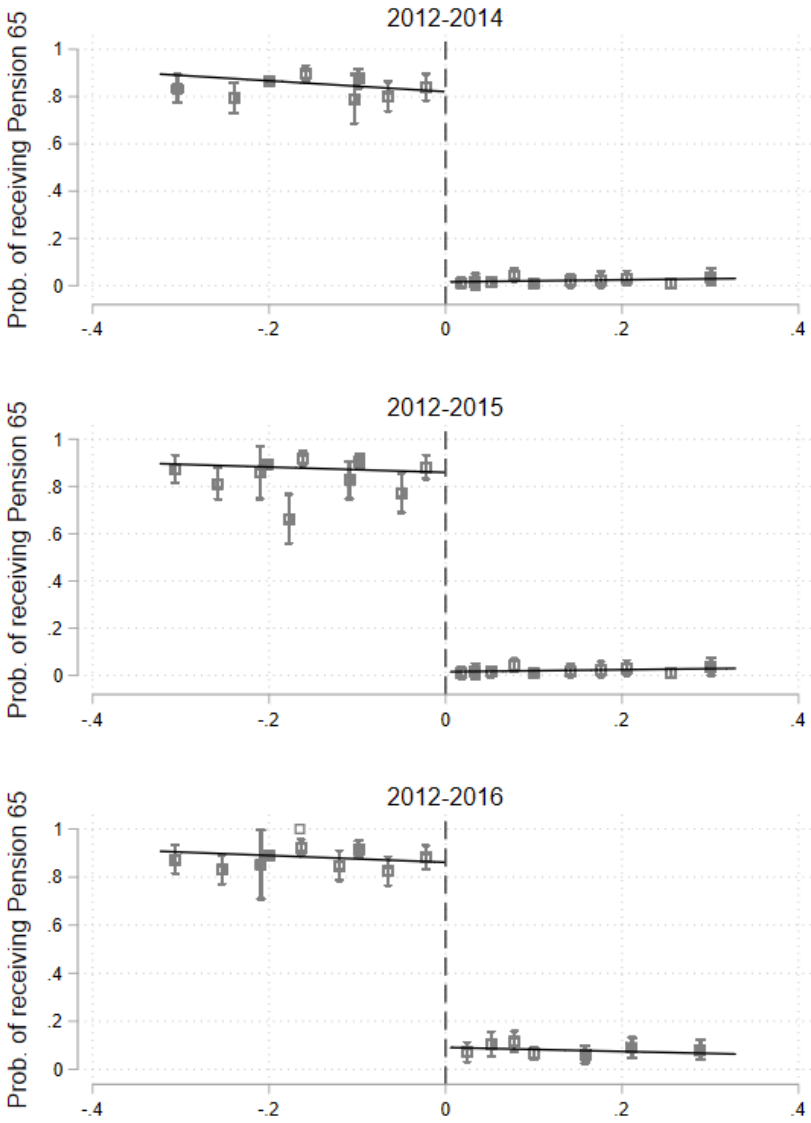
Notes: This figure plots the national distribution of the running variable, that is the SISFOH score minus eligibility cut-offs (histogram bars). The vertical red lines indicate the maximum and minimum values (bandwidth) found for the running variable in the ESBAM sample. The sampling framework correspond to observations located within this bandwidth. The data come from the SISFOH census of 2012/2013.

Figure B–2: Cumulative distribution of household income per capita in Peru and ESBAM



Notes: The figure plots the cumulative distribution of monetary gross household income per capita in Peru and the ESBAM Sample. The national income distribution is computed by exploiting the National Household Survey (ENAHO) collected in 2012. Income is transformed as log(1+income) for visualisation purposes.

Figure B–3: Probability of being a *Pension 65* recipient at different evaluation periods



Notes: The graphs plots the probability of receiving *Pension 65* anytime in the indicated period as a function of the running variable (SISFOH score minus eligibility cut-offs). The support of the running variable has been partitioned into exclusive bins. The number of bins is selected optimally to minimise the integrated mean square error of the underlying regression function, and the location is based on quantile spaced method using spacings estimators as suggested in Calonico et al. (2015). The square points indicate the local mean of the outcome at the mid-point of each bin. The bars represent the 95% confidence intervals of the local means. The solid lines are linear regressions that fit separately on each side of the threshold. Observations to the left (right) of the vertical dashed line are eligible (ineligible) to the programme.

Table B–1: Distribution of observations in sample

	Overall			Eligible			Ineligible		
	Mean	**SD**	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**	**N**
Key variable									
Dead (0/1)	0.085	0.280	3,885	0.083	0.276	2,525	0.090	0.287	1,360
Running variable	-0.066	0.161	3,885	-0.171	0.069	2,525	0.129	0.082	1,360
Treated (0/1)	0.603	0.489	3,885	0.883	0.321	2,525	0.082	0.275	1,360
Covariates									
Male (0/1)	0.545	0.498	3,885	0.557	0.497	2,525	0.524	0.500	1,360
Age	71.646	4.396	3,885	71.723	4.365	2,525	71.503	4.453	1,360
High blood pressure (0/1)	0.338	0.473	3,835	0.347	0.476	2,500	0.320	0.467	1,335
Anaemia (0/1)	0.315	0.465	3,818	0.319	0.466	2,488	0.307	0.461	1,330
Weight (Kg.)	55.560	10.518	3,826	55.004	10.345	2,495	56.603	10.762	1,331
Abdominal obesity (0/1)	0.329	0.470	3,820	0.304	0.460	2,490	0.377	0.485	1,330
Arm span (cm.)	155.932	10.787	3,838	155.801	10.871	2,501	156.179	10.628	1,337
Mid-upper arm circum. (cm.)	26.220	3.242	3,838	26.085	3.149	2,500	26.471	3.398	1,338
Calf circum. (cm.)	31.764	3.053	3,832	31.680	2.991	2,497	31.920	3.162	1,335
Cognitive functioning (0-13)	10.740	2.035	3,817	10.767	2.044	2,480	10.692	2.019	1,337
Chronic diseases (0-13)	1.040	1.322	3,885	1.028	1.315	2,525	1.062	1.334	1,360
Health today (0/1)	0.570	0.495	3,866	0.564	0.496	2,512	0.582	0.493	1,354
MNA score (0-19)	11.809	2.712	3,760	11.722	2.652	2,451	11.973	2.814	1,309
Alcohol (0/1)	0.195	0.396	3,881	0.183	0.387	2,522	0.217	0.412	1,359
Tobacco (0/1)	0.200	0.400	3,869	0.207	0.405	2,515	0.186	0.389	1,354
Juntos (0/1)	0.088	0.284	3,885	0.080	0.271	2,525	0.104	0.305	1,360

Notes: The sample is composed of individuals observed in the baseline of the 2012 ESBAM survey. After dropping observations with inconsistent or missing key information, the sample size is set to 3,885 individuals.

Table B–2: Distribution of observations in optimal selected sample

	Overall			Eligible			Ineligible		
	Mean	**SD**	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**	**N**
Key variable									
Dead (0/1)	0.091	0.287	1,598	0.078	0.269	688	0.100	0.300	910
Running variable	0.012	0.083	1,598	-0.075	0.037	688	0.078	0.033	910
Treated (0/1)	0.424	0.494	1,598	0.875	0.331	688	0.084	0.277	910
Covariates									
Male (0/1)	0.544	0.498	1,598	0.557	0.497	688	0.534	0.499	910
Age	71.468	4.353	1,598	71.506	4.261	688	71.440	4.424	910
High blood pressure (0/1)	0.340	0.474	1,579	0.403	0.491	683	0.292	0.455	896
Anaemia (0/1)	0.302	0.459	1,568	0.284	0.451	675	0.316	0.465	893
Weight (Kg.)	56.124	10.538	1,574	56.939	10.766	679	55.505	10.324	895
Abdominal obesity (0/1)	0.349	0.477	1,571	0.375	0.485	677	0.329	0.470	894
Arm span (cm.)	156.460	10.390	1,580	156.832	10.660	681	156.178	10.177	899
Mid-upper arm circum. (cm.)	26.408	3.257	1,580	26.916	3.158	681	25.022	3.279	899
Calf circum. (cm.)	31.891	3.087	1,575	32.283	3.111	679	31.628	3.045	896
Cognitive functioning (0-13)	10.752	2.037	1,574	10.994	1.940	679	10.569	2.090	895
Chronic diseases (0-13)	0.999	1.307	1,598	1.097	1.369	688	0.924	1.252	910
Health today (0/1)	0.577	0.494	1,594	0.588	0.492	687	0.568	0.496	907
MNA score (0-19)	11.918	2.755	1,541	12.162	2.539	661	11.735	2.896	880
Alcohol (0/1)	0.197	0.398	1,596	0.181	0.385	687	0.210	0.408	909
Tobacco (0/1)	0.196	0.397	1,594	0.186	0.390	687	0.204	0.403	907
Juntos (0/1)	0.116	0.320	1,598	0.122	0.328	688	0.111	0.314	910

Notes: The sample is composed of individuals observed in the baseline of the 2012 ESBAM survey within the optimal bandwidth of +/-0.150 around the eligibility threshold, resulting in a sample size of 1,598 individuals.The optimal bandwidth is obtained from the point estimation model as suggested by Calonico et al. (2015)

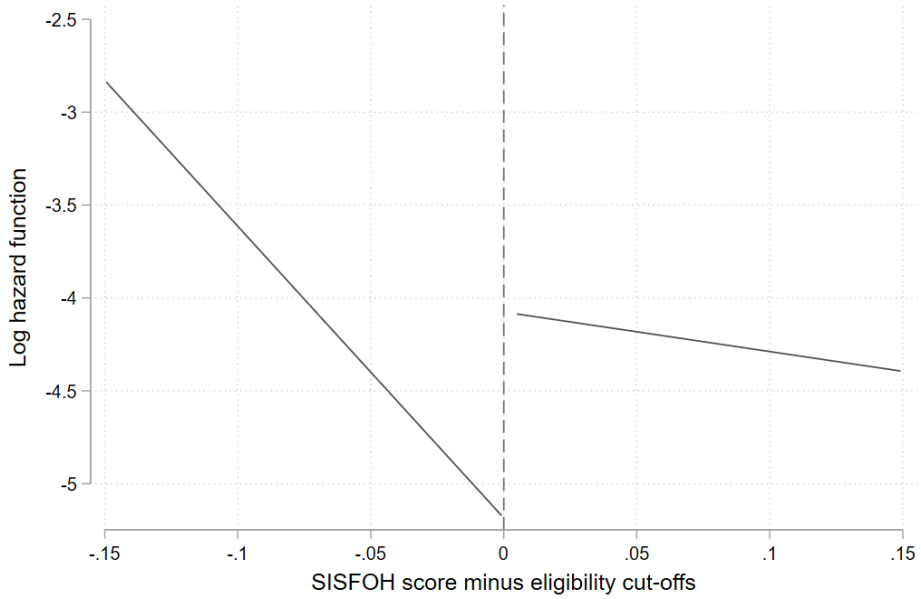
Table B–3: Cumulative number of *Pension 65* recipients

Year	Survived eligibles	Survived ineligibles	Num. of recipients	RD Estim.	S.E.	95% C.I.	
2013	2,475	1,338	1,963	0.689	0.029	0.632	0.745
2014	2,427	1,307	2,191	0.822	0.023	0.777	0.866
2015	2,370	1,272	2,243	0.852	0.020	0.812	0.891
2016	2,316	1,237	2,342	0.787	0.025	0.738	0.836

Notes: The first and second columns indicate the number of individuals who have survived until December of each year by the eligibility condition measured at the baseline (i.e. according to the SISFOH rules of 2012). The third column shows the accumulated number of individuals each year who have received the transfer from the programme, regardless of their survival or vital condition. The RD estimator is computed as the change in the intercept of two estimated linear regressions that fit separately on each side of the eligibility cutoff.

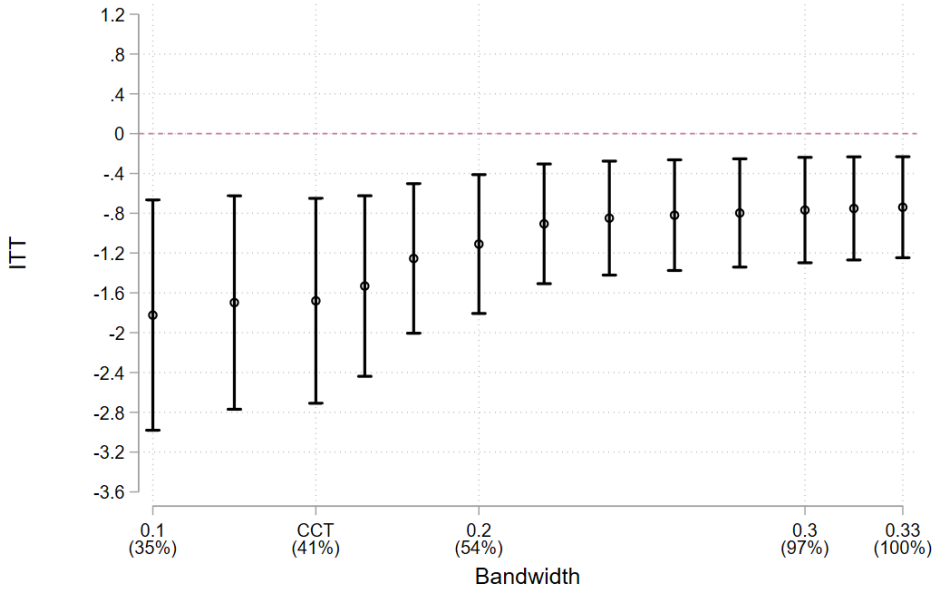
C Tables and figures of survival models

Figure C–1: Intention-to-treat in survival model



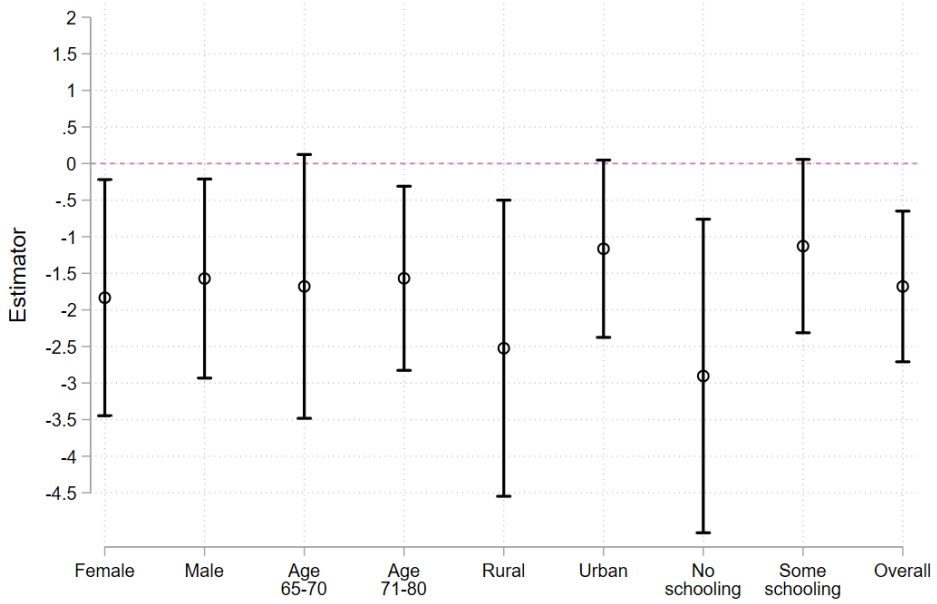
Notes: The graph plots the log of the hazard ratio of mortality observed over 4 years as a function of the running variable (SISFOH score minus eligibility cut-offs). The parametric model follows a Gompertz distribution. The lines are linear regressions that fit separately on each threshold side. Observations to the left (right) of the vertical dashed line are eligible (ineligible) to the programme.

Figure C–2: ITT effects on the log of mortality hazard ratio, by alternative bandwidths



Notes: This figure plots the ITT effects estimated for equation 4 for alternative bandwidths; CCT corresponds to the optimally estimated bandwidth proposed by Calonico et al. (2015). All models use triangular kernel and local linear polynomial. Vertical lines indicate 95% confidence intervals.

Figure C–3: Heterogeneous effects on the log of mortality hazard rate



Notes: The graph plots the estimated ITT coefficients from equation 4 for four distinctive demographic groups (by sex, age, area, and education) and the overall effect. The vertical lines indicate 95% confidence intervals.

Table C–1: Effect of *Pension 65* on log of mortality hazard rate under alternative functions

Model	ITT Estimator	95% C.I.	
Cox	-1.679	-2.710	-0.649
Exponential	-1.671	-2.698	-0.645
Weibull	-1.680	-2.711	-0.648

Notes: The table reports the ITT estimates for the log of mortality hazard rate observed over 4 years (equation 4). The estimators correspond to a non-parametric version (Cox model) and two parametric models (Exponential and Weibull). The models use triangular kernel, local linear polynomial and the optimal bandwidth for point estimation as suggested by Calonico et al. (2015) (obtained in the Table 2).

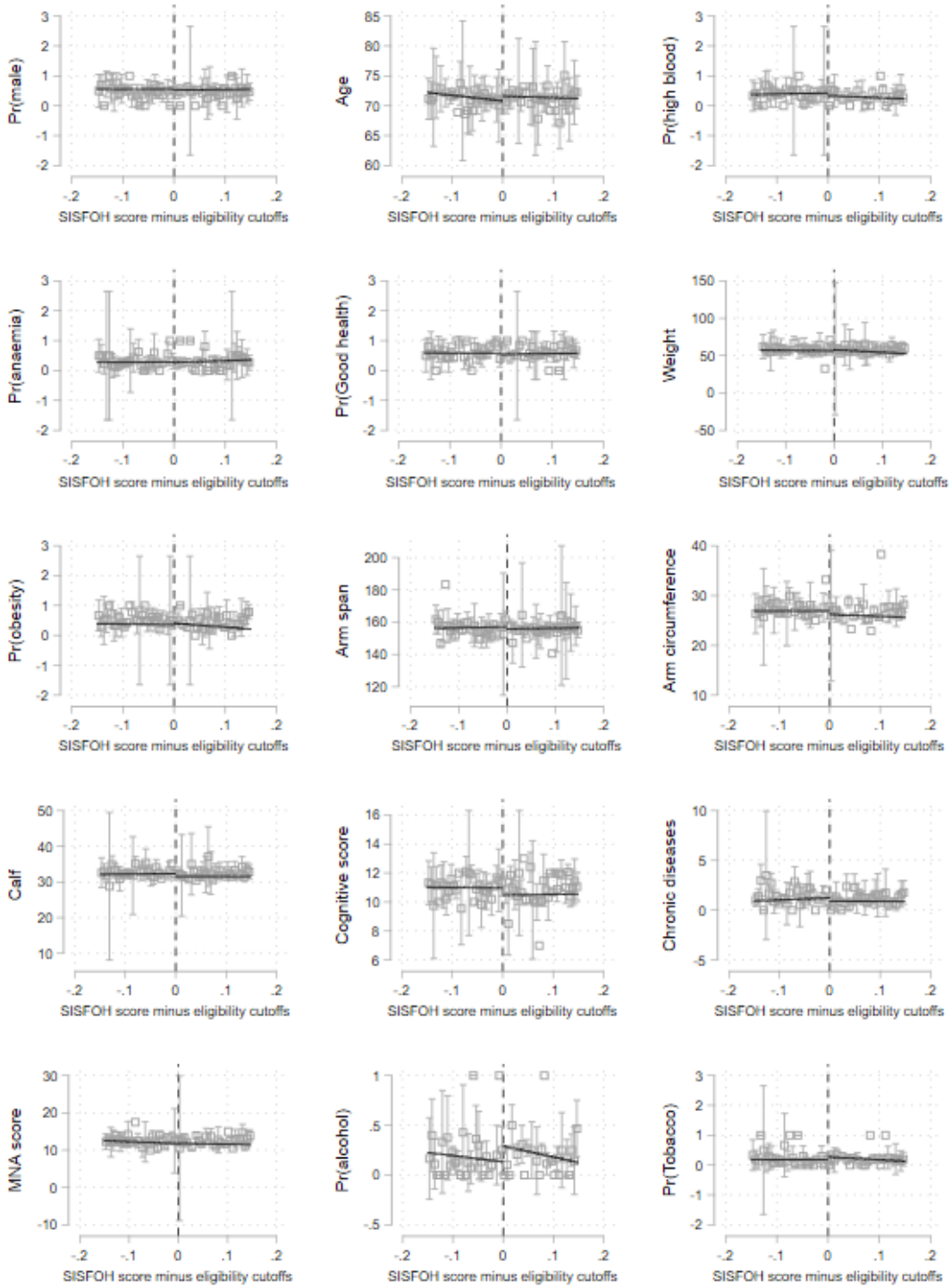
Table C–2: Effect of *Pension 65* on log of mortality hazard rate including covariates

	(1)	(2)	(3)	(4)	(5)
ITT	-1.680***	-1.595***	-1.293**	-1.740***	-1.674***
	(0.525)	(0.529)	(0.547)	(0.577)	(0.592)
Male		0.373*	0.645***	0.712***	0.670***
		(0.195)	(0.248)	(0.251)	(0.253)
Age		0.124***	0.089***	0.086***	0.092***
		(0.021)	(0.021)	(0.022)	(0.022)
High blood pressure			0.467**	0.427*	0.445*
			(0.226)	(0.234)	(0.230)
Anaemia			0.414**	0.465**	0.496**
			(0.196)	(0.197)	(0.198)
Weight			0.041**	0.045**	0.042**
			(0.021)	(0.021)	(0.021)
Abdominal obesity			0.409	0.428	0.522*
			(0.272)	(0.277)	(0.279)
Arm span			-0.003	-0.011	-0.012
			(0.014)	(0.015)	(0.015)
Mid-upper arm circ. (MUAC)			-0.188***	-0.170***	-0.167***
			(0.060)	(0.060)	(0.060)
Calf circumference (CC)			-0.096**	-0.086**	-0.084**
			(0.043)	(0.044)	(0.041)
Cognitive functioning			-0.150***	-0.117***	-0.119***
			(0.040)	(0.040)	(0.041)
Chronic diseases				0.077	0.079
				(0.064)	(0.065)
Health today				-0.357	-0.373
				(0.235)	(0.237)
Nutrition score (MNA)				-0.062	-0.068
				(0.051)	(0.052)
Alcohol					0.529**
					(0.237)
Tobacco					-0.030
					(0.236)
Constant	-3.878***	-13.099***	-3.761	-2.946	-3.249
	(0.319)	(1.625)	(2.772)	(2.903)	(2.874)
Observations	1598	1598	1529	1499	1497

Notes: The table reports the ITT estimates for the log of mortality hazard rate observed over 4 years (equation 4), including covariates related to the mortality risk. The estimator corresponds to a Gompertz-type model. The models use triangular kernel, local linear polynomial and the optimal bandwidth for point estimation as suggested by Calonico et al. (2015) (obtained in Table 2). The standard errors are indicated in parentheses. $*p < 0.10$, $**p < 0.05$, and $***p < 0.01$ indicate statistically significance levels.

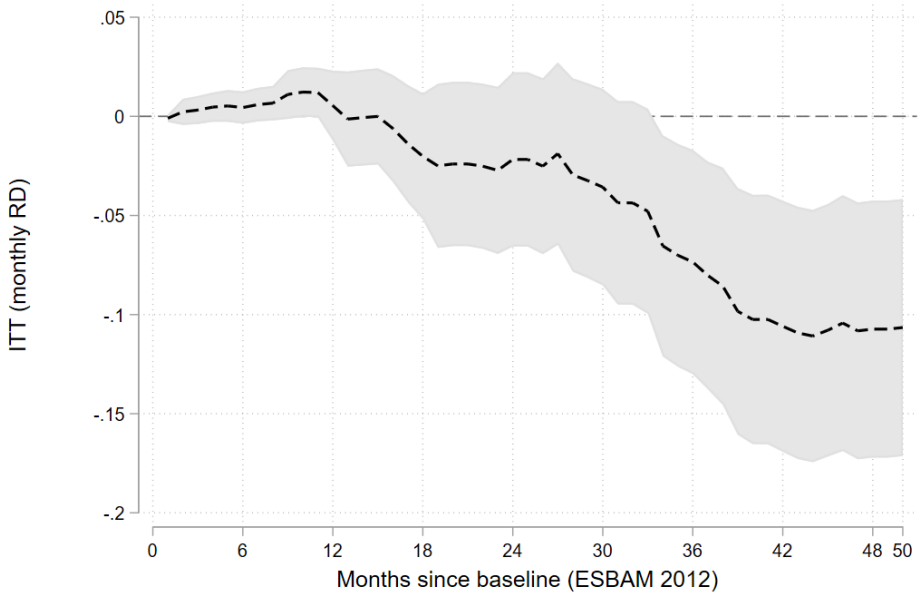
D Sensitivity and robustness checks

Figure D–1: Continuity in observables



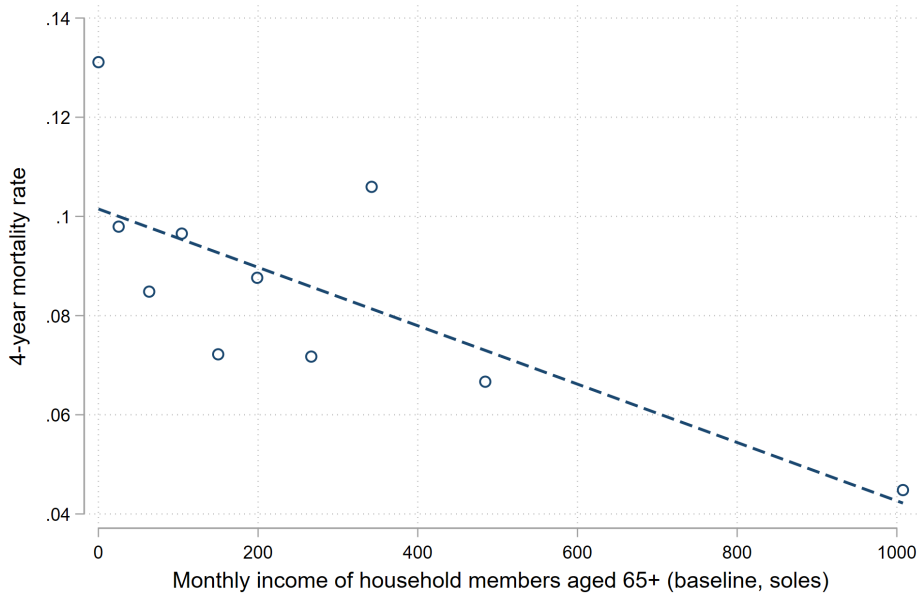
Notes: The graph plots the listed covariates as a function of the running variable (SISFOH score minus eligibility cut-offs). The support of the running variable has been partitioned into exclusive bins. The number of bins is selected optimally to minimise the integrated mean square error of the underlying regression function, and the location is based on quantile spaced method using spacings estimators as suggested in Calonico et al. (2015). The square points indicate the local mean of the outcome at the mid-point of each bin. The bars represent the 95% confidence intervals of the local means. The solid lines are linear regressions that fit separately on each side of the threshold. Observations to the left (right) of the vertical dashed line are eligible (ineligible) to the programme.

Figure D–2: Monthly ITT effects



Notes: This figure plots a separate linear RD estimate of equation 2 for each month of the follow-up period, using the dependent variable "died in or before month m" for $m = 1, \ldots, 50$. All models use the triangular kernel and the optimal bandwidth for point estimation as suggested by Calonico et al. (2015) (obtained in Table 2). The shaded area indicates 95% confidence intervals.

Figure D–3: Income-mortality gradient in the full ESBAM sample



Notes: The figure plots the mean 4-year mortality rate against mean baseline household income for each decile of the income distribution, using the full ESBAM sample (eligible and ineligible individuals, $N = 3{,}885$). The line is a linear fit across the ten decile means.

Table D–1: ITT effects under alternative cutoffs

Alternative cut-offs (x100)	Optimal bandwidth	Obs.	OLS model		Survival model	
			ITT	P-value	ITT	P-value
-25	0.026	86	-0.171	0.114	n.e.	n.e.
-15	0.043	426	0.030	0.581	0.301	0.737
-7.5	0.041	462	-0.195	0.140	-2.340	0.074
0	0.150	1,598	-0.107	0.001	-1.680	0.001
7.5	0.069	893	0.019	0.750	0.176	0.750
15	0.090	922	-0.011	0.850	-0.141	0.827
25	0.033	126	0.087	0.396	1.781	0.461

Notes: The table reports the ITT effects estimated for equations 2 and 4 at alternative cutoffs. The models use triangular kernel and local linear polynomial, and use the optimal bandwidth for point estimation as suggested by Calonico et al. (2015) obtained in linear regression around each placebo cutoff. Following Cattaneo et al. (2020), each placebo is estimated only with observations on its own side of the true threshold (eligible individuals for negative cutoffs, ineligible individuals for positive ones), so that no estimation window contains the actual discontinuity. The row for the cutoff at zero reports the main specification (Tables 2 and 4) for reference. n.e.: the survival model cannot be estimated at -0.25, where the window contains three deaths. The standard errors are clustered by the Primary Sampling Unit (PSU) of the sampling framing.

Table D–2: Sensitivity to observations near the cut-off (donut hole approach)

Donut-hole Radius (x1000)	Excluded Obs.		OLS Model		Survival Model	
	Left	Right	ITT	p-value	ITT	p-value
0	0	0	-0.107	0.001	-1.680	0.001
2	7	0	-0.106	0.002	-1.633	0.002
4	11	0	-0.105	0.002	-1.606	0.003
6	16	18	-0.118	0.001	-1.694	0.002
8	16	20	-0.121	0.001	-1.723	0.002
10	17	21	-0.122	0.001	-1.731	0.002

Notes: The table reports the ITT effects estimated for equations 2 and 4 excluding observations around cut-off. All the estimated models use triangular kernel, and the optimal bandwidth for point estimation as suggested by Calonico et al. (2015) (obtained in the Table 2). The standard errors are clustered by the Primary Sampling Unit (PSU) of the sampling framing.

Table D–3: Effect of *Pension 65* on mortality rate

	(1)	(2)	(3)
Intention-to-treat ($\hat{\beta}_1$)	-0.107	-0.113	-0.113
	(0.033)	(0.034)	(0.037)
Health campaigns in the district		-0.010	-0.010
		(0.002)	(0.002)
Health campaigns × eligible			0.0002
			(0.004)
Constant	0.116	0.144	0.144
	(0.028)	(0.031)	(0.032)
Observations	1,598	1,598	1,598

Notes: The table reports the ITT estimates for mortality observed in 4 years (Equation 2). The models use triangular kernel and local linear polynomial. The first column shows our main model. The second model includes the average number of yearly health campaigns conducted by the Ministry of Health in the individual's district of residence, to which the individual was exposed during the period 2013-2016. The third model adds the interaction of this variable with programme eligibility, to test whether the campaign effect differs on either side of the threshold. The optimal bandwidth for point estimation is used, as suggested by Calonico et al. (2015). The standard errors are clustered by the Primary Sampling Unit (PSU) of the sampling framing and are indicated in parentheses.

Table D–4: Effect of *Pension 65* on log mortality hazard rate

	(1)	(2)	(3)
Intention-to-treat ($\hat{\beta}_1$)	-1.680	-1.745	-1.317
	(0.525)	(0.532)	(0.574)
Health campaigns in the district		-0.273	-0.202
		(0.081)	(0.065)
Health campaigns × eligible			-0.423
			(0.381)
Constant	-3.878	-3.406	-3.505
	(0.319)	(0.351)	(0.346)
Observations	1,598	1,598	1,598

Notes: The table reports the ITT estimates for the log of the mortality hazard rate observed over 4 years (Equation 4). The first column shows our main model. The second model includes the average number of yearly health campaigns conducted by the Ministry of Health in the individual's district of residence, to which the individual was exposed during the period 2013-2016. The third model adds the interaction of this variable with programme eligibility, to test whether the campaign effect differs on either side of the threshold. The optimal bandwidth for point estimation is used, as suggested by Calonico et al. (2015). The standard errors are clustered by the Primary Sampling Unit (PSU) of the sampling framing and are indicated in parentheses.

E Value of Statistical Life (VSL)

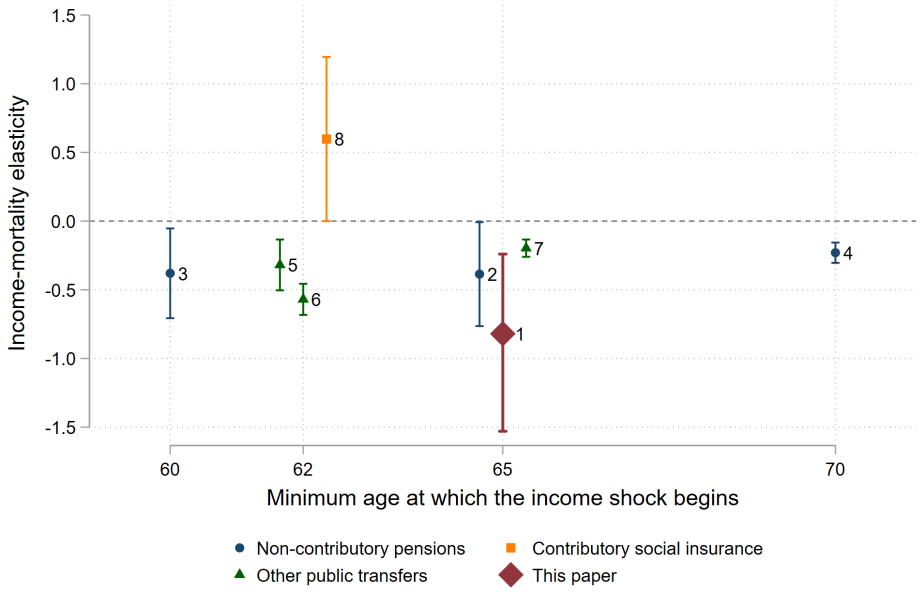
Table E–1: Value of Statistical Life (VSL) estimations for Peru (2012)

Study	Features	VSL (USD)
Robinson et al. (2019)	base=160; ε=1.5	427,711
Robinson et al. (2019)	base=100, ε=1.0	606,786
Robinson et al. (2019)	base=160, ε=1.0	970,858
Viscusi and Masterman (2017)	ε=1.0	1,044,306
Sweis (2022)	γ=0.1	3,348,777
Sweis (2022)	γ=1.0	362,030
Sweis (2022)	γ=0.5	633,552
Mardones and Riquelme (2018)	predicted from other studies	451,746

Notes: This table shows the Value of Statistical Life (VSL) estimated for Peru in different studies. All cases have been adapted to show the VSL in 2012 Dollars. Robinson et al. (2019) extrapolate VSL estimates of USD using different base values and income elasticity (ε). The base value multiplies the Gross National Income (GNI) per capita, while the ε summarises the rate at which VSL changes with income.Viscusi and Masterman (2017) uses unitary income elasticity and their US estimate of VSL to compute VSL in 189 countries. Sweis (2022) uses a consumption-health maximisation framework to measure the VSL in selected countries in order to measure the total value of loss from deaths caused by COVID-19. She presents estimates of VSL by different values of γ, which is the degree of homogeneity of the utility function (a smaller gamma implies more benefits to invest in health and survive longer), yet the author prefers γ=0.5 to measure the value of COVID-19 deaths. Mardones and Riquelme (2018) use VSL estimates from different studies to estimate the relationship between Gross Domestic Product (GDP) per capita and VSL, and then predict the VSL for selected Latin American countries.

F Comparative of mortality-income elasticities

Figure F–1: Mortality-income elasticity: comparison across studies



Notes: Each marker is the elasticity of older-adult mortality with respect to income implied by one evaluation of an old-age income transfer, plotted against the minimum age at which the transfer begins; bars are 95% confidence intervals. Circles are non-contributory social pensions, the square is contributory social insurance, triangles are other public transfers and the diamond is this paper. (1) This paper (Peru, 4-year mortality); (2) Miglino et al. (2023) (Chile, 4-year mortality); (3) Huang and Zhang (2021) (China, 1-year mortality); (4) Menares et al. (2026) (Mexico, women, annual mortality); (5) and (6) Salm (2011) (United States, Union Army veterans' pensions under the 1907 and 1912 laws, annual mortality hazard); (7) Barham and Rowberry (2013) (Mexico, *Progresa*, annual mortality rate of people aged 65 and above by municipality); (8) Snyder and Evans (2006) (United States, Social Security Notch, men, 5-year mortality after age 65). Estimates 1 to 4 are reported by the authors. We compute estimates 5 to 8 from the mortality effect and the income change each paper reports: hazard ratios of 0.885 and 0.704 for pensions equal to 36% and 52% of average non-farm earnings in (5) and (6); a 4.3% fall in the elderly mortality rate for a 22% rise in beneficiary income in (7); and 2.4% higher mortality for the cohort with about 4% higher income in (8). The authors attribute the positive sign in (8) to the larger increase in part-time work among the cohort with lower benefits rather than to income itself, and the estimate rests on an income change of only about 4%; using death records linked to the 1940 census, Noghanibehambari and Fletcher (2025) find the opposite sign for the same reform. The interval for this paper is the bootstrap interval clustered by PSU reported in the main text; for the other studies it is propagated from the standard error of the mortality effect, with the income change held fixed. Income measures and mortality windows differ across studies, so the comparison should be read with that in mind. Not shown: Jensen and Richter (2004), who do not report the baseline mortality needed to express their effect in relative terms; Cheng et al. (2018), who report only modest evidence of a mortality effect; and women in Snyder and Evans (2006), whose income the Notch barely changed.

G Mechanisms

Table G–1: ITT effects of *Pension 65* on various outcomes (2015 follow-up sample)

	All sample			Bandwidth 0.150			Non-parametric		
Outcomes	Effect	SE	N	Effect	SE	N	Effect	SE	N
High blood pressure	0.091	0.083	3,494	-0.163	0.146	1,434	-0.284*	0.171	1,235
Anaemia	-0.246***	0.082	3,454	-0.012	0.151	1,417	0.228	0.282	469
Weight	0.127	0.085	3,478	-0.110	0.158	1,426	-0.294	0.275	644
Abdominal obesity	0.147*	0.081	3,468	-0.209	0.146	1,420	-0.476*	0.269	467
Arm span	0.014	0.077	3,481	-0.065	0.134	1,429	0.265	0.248	469
Mid-upper arm circ. (MUAC)	0.322***	0.086	3,496	0.097	0.151	1,435	0.250*	0.135	1,828
Calf circumference (CC)	0.260***	0.082	3,492	-0.110	0.149	1,435	-0.093	0.236	689
Depression symptoms	-0.237***	0.086	3,511	-0.154	0.131	1,444	0.239	0.212	476
Cognitive functioning	0.333***	0.082	3,449	0.387***	0.133	1,422	-0.058	0.272	443
Chronic diseases	0.356***	0.097	3,512	0.567***	0.165	1,444	-0.052	0.257	449
Nutrition score (MNA)	0.307***	0.087	3,354	-0.001	0.164	1,374	-0.148	0.251	621
Health today	0.131	0.082	3,506	0.233*	0.133	1,441	-0.008	0.240	481
Health compared to last year	0.292***	0.080	3,503	0.334**	0.136	1,441	0.267*	0.151	1,247
Health compared to other people	0.197**	0.078	3,455	0.092	0.136	1,420	0.022	0.158	1,220
Subjective health index	0.246***	0.081	3,454	0.226*	0.136	1,419	0.100	0.209	649
Attended health centre	0.347***	0.095	2,474	0.716***	0.157	1,016	0.570**	0.260	476
Indiv. health expenditure	0.106	0.066	3,512	0.172	0.119	1,444	0.157	0.104	1,856
Indiv. medicine expenditure	0.120**	0.048	3,512	0.127	0.081	1,444	0.098	0.063	3,037
Working hours	-0.298***	0.091	3,512	-0.109	0.167	1,444	-0.102	0.188	1,322
Working	-0.311***	0.089	3,512	-0.097	0.156	1,444	-0.068	0.172	1,243
Household expenditure	0.544***	0.100	3,512	0.035	0.201	1,444	-0.071	0.237	1,322
Household food expenditure	0.486***	0.093	3,512	-0.102	0.175	1,444	-0.203	0.283	743
Expenditure per capita	0.332***	0.085	3,494	-0.221	0.183	1,436	-0.495	0.388	634
Food expenditure per capita	0.382***	0.083	3,494	-0.317**	0.150	1,436	-0.418*	0.239	654
Number of household members	0.276***	0.105	3,512	0.459***	0.177	1,444	0.396***	0.113	3,507
Affiliated to SIS	0.171*	0.092	3,511	0.027	0.161	1,444	0.365	0.334	475
Preventive care uptake (last 2 years)	0.136	0.097	3,512	0.007	0.176	1,444	-0.241	0.229	692

Notes: The table reports the ITT effects of *Pension 65* on various outcomes measured during the follow-up survey fielded between July and September 2015, together with their standard errors. All the outcomes have been standardised to show mean equal to zero and standard deviation equal to one. From the 3,885 observations of our baseline, we found 3,512 individuals surveyed in the follow-up. The first block of columns reports the ITT effects on all the 2015 sample, the second block reports the ITT effects when we use the bandwidth considered in our main analysis of mortality on the baseline sample (bandwidth equal to 0.150) and kernel weights, and the third block shows the ITT effects when we use the non-parametric approach suggested by Calonico et al. (2015) for each outcome (rdrobust with kernel weights and polynomial of degree one, and MSE-optimal bandwidths). The standard errors are clustered by the Primary Sampling Unit (PSU) of the sampling framing. $*p < 0.10$, $**p < 0.05$, and $***p < 0.01$ indicate statistically significance levels based on those standard errors.

H Local average treatment effects (LATE)

This appendix describes the recursive bivariate probit used in Section 7.5. We estimate the following system by full information maximum likelihood (Greene, 2018):

$$treated_i^* = \gamma_0 + \gamma_1 D_i + \gamma_2 (z_i - z_0) + \gamma_3 (z_i - z_0) D_i + u_i, \quad treated_i = 1[treated_i^* > 0] \tag{5}$$

$$y_i^* = \delta_0 + \delta_1 treated_i + \delta_2 (z_i - z_0) + v_i, \quad y_i = 1[y_i^* > 0] \tag{6}$$

where (u_i, v_i) are jointly normal with correlation ρ and D_i is the eligibility indicator of equation 2. Eligibility enters only the take-up equation, which is the same exclusion restriction used by the linear Wald ratio. The running variable enters the outcome equation with a common slope on both sides of the cut-off, because a separate slope would require the term $(z_i - z_0)D_i$, which is built from the excluded instrument. Under monotonicity, a threshold-crossing model of this kind identifies a LATE-type parameter (Vytlacil, 2002), so it differs from the Wald ratio in functional form, not in the parameter it targets. Unlike the linear ratio, it keeps predicted probabilities within $[0, 1]$ (Chiburis et al., 2012).[23]

Table H–1 reports the linear Wald ratio at three bandwidths (Panel A) and the bivariate probit at the preferred bandwidth (Panel B). The common slope explains why predicted mortality without the pension (17.5 pp) is higher than the linear control mean at the cut-off (11.6 pp). The estimated correlation, $\hat{\rho} = 0.206$ (standard error 0.148), is not distinguishable from zero ($\chi^2(1) = 1.83$, $p = 0.177$). Because D_i is excluded from the outcome equation, ρ is identified only through the functional form of the joint normal, so this test has little power and says little about selection on unobservables. Adding $(z_i - z_0)D_i$ to the outcome equation moves the estimate from -14.9 to -16.3 pp; we keep the specification without it because that term weakens the exclusion restriction.

[23] Following Freedman and Sekhon (2010), we estimate the two equations jointly rather than in two steps. The model uses `pweight`, because `biprobit` in Stata does not accept the `aweight` used elsewhere in the paper; the point estimates are identical under both weight types. Standard errors are clustered by PSU.

Table H–1: ITT, first stage and LATE, 4-year mortality window

	Full sample	+/- 0.200	+/- 0.150 (preferred)
Panel A: linear Wald ratio			
ITT effect	-0.060	-0.083	-0.107
	[0.003]	[0.003]	[0.001]
First stage (receipt in 2012–2016)	0.771	0.761	0.762
	[0.000]	[0.000]	[0.000]
LATE (ITT / first stage)	-0.078	-0.108	-0.140
Control mortality at the cutoff	0.115	0.113	0.116
Implied treated mortality (control+LATE)	0.037	0.005	-0.024
Panel B: recursive bivariate probit (preferred bandwidth, robustness)			
Predicted control mortality			0.175
			[0.000]
Predicted treated mortality			0.027
			[0.001]
Bounded LATE (difference)			-0.149
			[0.001]
$\hat{\rho}$ (s.e.)			0.206 (0.148)
Wald test $\rho = 0$: $\chi^2(1)$ [p-value]			1.83 [0.177]
Obs.	3,885	2,104	1,598
Clusters (PSU)	1,064	831	718

Notes: Panel A reports, for each bandwidth, the intention-to-treat (ITT) effect on 4-year mortality, the first-stage effect of eligibility on actually receiving *Pension 65* at any time between 2012 and 2016 (receipt starting after 2016 is not counted, since only individuals who survived to 2017 could start receiving the pension then), and the local average treatment effect (LATE) obtained as the linear Wald ratio of the two. Because this ratio is not bounded, adding it to the observed control-group mortality rate at the cutoff can imply a treated-group mortality rate below zero, as it does at the preferred bandwidth. Panel B re-estimates the LATE at the preferred bandwidth, as a robustness check (Section 7.5), with a recursive bivariate probit in which the eligibility discontinuity enters only the take-up equation and the running variable enters both equations additively (Greene, 2018); the reported probabilities are predictions from this model at the cutoff and are constrained to lie in $[0,1]$ by construction. The bounded LATE reported in Panel B is the difference between the two predicted margins; its standard error, 0.043 (delta-method, clustered by PSU), is obtained by posting the margins and testing this difference directly, and confirms the estimate is statistically significant at the 1% level. Interacting the running variable with eligibility in the outcome equation, as a sensitivity check, moves the bounded LATE to -0.163 (SE 0.045, *p-value*$<$0.001; predicted control mortality 0.187, predicted treated mortality 0.025), which does not change the sign or the order of magnitude of the estimate. The estimated correlation between the two equations' errors, $\hat{\rho}$, is not statistically distinguishable from zero (Wald test reported below Panel B); this is expected under the exclusion restriction used here and does not indicate that the take-up and outcome equations are unrelated. All models use triangular kernel weights and standard errors clustered by the Primary Sampling Unit (PSU) of the sampling frame. The corresponding p-values are reported in brackets.

I Heterogeneity by age and household assets

Table I–1: Heterogeneity by age and household assets, 4-year mortality window

	Preferred bandwidth (+/- 0.150)
Panel A: continuous age interaction	
Treatment (D)	0.379
	[0.123]
Age (years)	0.013
	[0.000]
D × Age	-0.007
	[0.055]
Implied effect at age 65	-0.060
Implied effect at age 80	-0.161
Panel B: household asset index (first principal component)	
Share of variance explained by first component	0.111
Treatment (D)	-0.105
	[0.001]
Asset index	0.006
	[0.659]
D × Asset index	0.000
	[0.977]
Panel C: split at the median of the asset index	
Treatment (D), higher-asset half	-0.091
	[0.039]
Treatment (D), lower-asset half	-0.131
	[0.011]
Obs. (Panels A and B)	1,598
Obs. (Panel C, higher / lower)	793 / 805
Clusters (PSU, Panels A and B)	718
Clusters (PSU, Panel C, higher / lower)	392 / 379

Notes: Panel A interacts treatment with age measured continuously in years, instead of with the binary 65–70/71–80 split used in Figure 6; the implied effect at a given age is the sum of the treatment coefficient and the interaction coefficient times that age. Panel B builds an asset index following Filmer and Pritchett, 2001, taking the first principal component of category dummies for wall, floor and roof material, water source and sanitation, together with binary ownership of a fixed telephone, cellphone, cable television and internet, all measured at the ESBAM baseline; the index is standardised to mean zero and standard deviation one, and interacted with treatment. Panel C splits the sample at the median of the asset index and re-estimates the baseline specification separately in each half. All models use triangular kernel weights and standard errors clustered by the Primary Sampling Unit (PSU) of the sampling frame. The corresponding p-values are reported in brackets.