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**Working Paper**

## The Effects of Compulsory Schooling on Health and Hospitalization over the Life Cycle

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## ABSTRACT

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# The Effects of Compulsory Schooling on Health and Hospitalization over the Life Cycle\*

This paper examines the effects of education on health and hospitalization over the life cycle. Using administrative data, we leverage a 1972 compulsory schooling reform within the United Kingdom which produced a large increase in educational attainment among affected cohorts. Our regression discontinuity design estimates suggest that the reform led to substantial reductions in hospitalization among men admitted for lifestyle-related conditions. We also report novel estimates showing that these effects vary heterogeneously over the life-cycle – with the largest health improvements occurring among men in their middle-aged years. However, we find no evidence that the reform impacted mortality during working-age years.

**JEL Classification:** I10, I12, I14, I20

**Keywords:** health, education, compulsory schooling, life cycle, gender differences

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# 1 Introduction

Research examining the relationship between education and health occupies a prominent space within the health economics literature. Indeed, a key prediction from the canonical [Grossman \(1972\)](#) model of the demand for health suggests that an increase in formal schooling can lead to improvements in health and quality-of-life through more efficient health production. Such efficiency gains can take the form of improved processing of health information and early adoption of health care innovations ([Rosenzweig and Schultz, 1983](#); [Kenkel, 1991](#); [Glied and Lleras-Muney, 2008](#)). Educational investments in childhood also have significant effects on health and health behaviors in adulthood ([Conti et al., 2010](#); [Heckman et al., 2018](#)). While an extensive empirical literature has since emerged exploring the nature of this relationship ([Grossman and Kaestner, 1997](#); [Grossman, 2000, 2006](#); [Cutler and Lleras-Muney, 2012](#)), much of this work also highlights several important challenges in estimating the causal effect of education on health. Some of the well-noted challenges to identification include endogenous time preferences ([Fuchs, 1982](#)), simultaneity, and other forms of omitted variable bias ([Cutler et al., 2011](#); [Grossman, 2015](#)).

Following a lengthy earnings literature ([Angrist and Krueger, 1991](#); [Acemoglu and Angrist, 2000](#); [Card, 2001](#); [Oreopoulos, 2006](#); [Pischke and Von Wachter, 2008](#); [Grenet, 2013](#)), a strong body of research leverages compulsory schooling reforms in an effort to obtain causal evidence concerning the effects of education on a number of health outcomes ([Adams, 2002](#); [Lleras-Muney, 2005](#); [Black et al., 2008](#); [Mazumder, 2008](#); [Albouy and Lequien, 2009](#); [Chou et al., 2010](#); [Kemptner et al., 2011](#); [McCrary and Royer, 2011](#); [Van Kippersluis et al., 2011](#); [Wilson, 2017](#)).<sup>1</sup> However, even studies invoking nearly identical identification strategies and research settings ultimately come to different conclusions regarding the effects of education on health. Several studies leveraging two notable twentieth century raising of the school leaving age (ROSLA) reforms within the United Kingdom (U.K.) find that these policies led to nearly half of the population receiving an additional year of education, but ultimately

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<sup>1</sup>See [Galama et al. \(2018\)](#) and [Xue et al. \(2021\)](#) for an excellent review of this literature.

provide contrasting evidence on the effects of these changes on health. While these particular reforms do not appear to produce any meaningful changes in self-reported health (Clark and Royer, 2013), other work finds evidence of improvements in certain lifestyle outcomes such as diabetes and obesity (Barcellos et al., 2018, 2023). Thus, findings based on subjective health measures (e.g., self-reported health and health behaviors) appear at times to be in conflict with other evidence based on more objective health measures (e.g., blood pressure and body composition measurements taken by a health professional).<sup>2</sup>

This paper reconciles some of the contrasting empirical evidence on education and health by leveraging a similar twentieth century ROSLA reform within the context of Scotland. As a constituent nation of the U.K., Scottish schools were required to raise the minimum school leaving age from 15 to 16 in September 1972. The reform exogeneously increased schooling levels by about half a year for individuals born after a cut-off birth date. We employ a regression discontinuity (RD) design in order to produce estimates of the effects of the 1972 ROSLA reform on self-reported health and health care utilization. Our RD design rests on the credible identifying assumption that the 1972 ROSLA reform serves as the sole event driving variation in educational attainment for individuals born one month apart. Our RD estimates of the effects of compulsory schooling on educational attainment are strikingly similar to those based on the experiences of England and Northern Ireland (Oreopoulos, 2006; Clark and Royer, 2013).

In addition to possessing nationally representative survey data on self-reported health measures, Scotland allows for novel insights into the education-health gradient through a nationally representative longitudinal dataset for a large (semi-) randomly selected sample containing linked information on health care utilization, cancer diagnoses (pre- and post-mortem), and drug use based on administrative records. These data not only provide us with a unique opportunity to examine both subjective and objective health outcomes within the

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<sup>2</sup>A notable exception is Davies et al. (2018) who find that the 1972 ROSLA reform affected both some self-reported (e.g. smoking status) and measured (e.g. body mass index) health outcomes—albeit for a non-nationally-representative sample from the U.K. Biobank.

same population, but also allow us to explore an important theoretical consideration scarcely discussed within the literature— the evolution of the relationship between education and health over the life cycle (Lynch, 2003; Galama and Van Kippersluis, 2019; Kaestner et al., 2020; Lleras-Muney and Moreau, 2022). If the marginal productivity of health investment changes over the life cycle, one might expect the effects of increased schooling on health and health care utilization to vary across different age profiles as well.

Our RD estimates based on administrative records indicate that the reform led to meaningful improvements in both health and health care utilization. We specifically find that an additional year of education reduces hospitalization by nearly 0.37 standard deviations. These effects are concentrated among men admitted to the hospital for conditions generally related to critical “lifestyle” behaviors— including cardiovascular disease and alcohol abuse. Individuals affected by the reform also experienced lower rates of cancer diagnoses. Similar to previous research, we find little evidence of the reform’s effects on subjective health measures such as self-reported poor health status and smoking. We show that our findings are unlikely to be explained by income effects or selective mortality.

This study also yields new insights into the evolving relationship between education and health over the life cycle. Consistent with the extended Grossman (1972) demand for health model introduced in Kaestner et al. (2020), the effects of the 1972 ROSLA reform on hospitalization indeed vary throughout the life cycle and across health conditions. For example, cumulative reductions in hospitalization for cardiovascular disease occur almost exclusively among men beginning in their late-30s and become more pronounced upon reaching the age of 55. While men also experience statistically significant reductions in injury-related admissions, these effects are relatively constant and more concentrated within the intermediate age range. Such findings are consistent with both a convergence in health stock and changes in the marginal productivity of health investment over the life cycle. Interestingly, we find no evidence that education affects mortality during one’s working age years.

Our research contributes to the existing literature on education and health in several im-

portant ways. First, the longitudinal nature of our data permits novel insights into how education affects health over the life cycle (Galama et al., 2018; Kaestner et al., 2020). We show that this dynamic relationship varies heterogeneously across disease conditions and gender. Research solely relying on aggregate estimates might not capture the significant time-varying contributions of education to health during critical periods of health stock depreciation. Our findings are also consistent with predictions from a dynamic model developed by Lleras-Muney and Moreau (2022) with the effects of education on hospitalization appearing much earlier than mortality effects which are predicted to occur later in life.

Second, we also show that education plays a critical role in reducing costly hospitalization for lifestyle-related conditions. In contrast to previous studies examining the effects of other schooling reforms on hospitalization in Nordic countries (Arendt, 2008; Meghir et al., 2018), we leverage the British 1972 ROSLA reform to study these effects at a pivotal period in the life cycle where the benefits of previous health investment are arguably most salient.

Finally, our complementary datasets allow us to study both objective and subjective health outcomes within the same population. Our findings that education produces meaningful effects on objective health measures (e.g., hospitalization and cancer incidence), but not subjective health measures (e.g., self-rated health), is consistent with previous work based on these outcomes (Clark and Royer, 2013; Davies et al., 2018; Barcellos et al., 2018, 2023). One potential explanation for these different findings could be the failure of compulsory schooling to improve perceived overall well-being later in life. Another explanation involves the susceptibility of subjective health measures to measurement error and non-random differences in responses that challenge the interpretation of the empirical estimate of interest (Bound, 1991; Mackenbach et al., 1996; Bound et al., 1999; Johnston et al., 2009).

The remainder of the paper is organized as follows. Section 2 examines some theoretical evidence on the relationship between education and health over the life cycle. Section 3 provides an overview of the institutional setting and the nature of the ROSLA reforms in Scottish schools. In this section, we also outline our identification strategy. In Section 3.2,



we introduce our data and discuss how these data differ from other sources used in this literature. We present our main results in Section 4 along with robustness checks. Section 5 explores important channels through which education might influence health while Section 6 concludes.

## 2 Health and Education over the Life Cycle

One of the most celebrated features of the canonical [Grossman \(1972\)](#) demand for health model involves its ability to account for a well-documented education-health gradient. Within this framework, education contributes to the endogenous determination of health through its effects on the marginal productivity of health investment. More educated individuals achieve greater health stock for each unit of health investment. This investment could involve a variety of inputs working to augment optimal health stock— including improved dietary regimen or more sophisticated medical care consumption.

While many studies examining the education-health gradient often hold the productivity of these investments constant, there are several reasons why one might believe that this form of human capital investment can have varying effects on health over the life cycle. First, dynamic complementarities between skill and health capital might evolve over the life cycle ([Galama and Van Kippersluis, 2019](#)). Second, the marginal productivity of health investment could differ considerably during early stages of the life cycle, often characterized by slower depreciation of health stock, and perhaps might involve a different combination of inputs altogether relative to choices made later in life. For example, strength training and nutrition could be associated with greater health improvements earlier in life while annual medical examinations could yield greater benefits among middle-aged adults. In either case, any efficiency gains associated with education are likely to vary with age.

[Kaestner et al. \(2020\)](#) make this point more explicitly by first modifying the health production function within the Grossman model to allow for education to determine health

through the productivity of health investment.

$$H_t = H \prod_{j=k}^{t-1} (1 - \delta_j) + \alpha_0 I_0 \prod_{j=k+1}^{t-1} (1 - \delta_j) + \dots + \alpha_{t-1}(E) I_{t-1}(E) \quad (1)$$

where  $H_t$  is health at age  $t$ ,  $I_{t-1}$  is gross health investment at age  $t - 1$ ,  $\delta$  is depreciation of health stock, and  $\alpha$  is the productivity of health investment. If education positively affects health through greater health investment productivity, equation (1) suggests that the effects of education are age-specific and will differ with changes in  $\delta$ . They further show that the cumulative effect of education on health is then given by:

$$\frac{\partial H_t}{\partial E} = \sum_k^{t-2} \left[ \left( \frac{\partial \alpha_k}{\partial E} I_k + \alpha_k \frac{\partial I_k}{\partial E} \right) \prod_{j=k+1}^{t-1} (1 - \delta_j) \right] + \frac{\partial \alpha_{t-1}}{\partial E} I_{t-1} + \alpha_{t-1} \frac{\partial I_{t-1}}{\partial E} \quad (2)$$

Equation 2 suggests that the total effect of education at age  $t$  is given by the sum of its effects on both the productivity and quantity of health investment. The authors go on to show that while education only yields small effects on mortality through the age of 60, before reducing the hazard rate of death, its effects on morbidity are greatest between the ages of 45 and 60.

This extension of the Grossman model offers several compelling explanations for some of the contrasting findings on the effects of compulsory schooling reforms on health. Studies producing aggregate estimates across cohorts would capture neither age-specific changes in investment productivity  $\alpha_t$  nor the subsequent changes in quantity of investment. For example, the effects of an additional year of formal schooling on health care utilization could be small at earlier ages only to become more pronounced later in life when health stock depreciation is more salient. Differences in follow-up periods across study samples could also yield conflicting evidence on the education-health gradient if investment productivity varies over the life cycle— with several studies based on the U.K. ROSLA reforms leveraging variation among similar populations albeit at different points in life (Clark and Royer, 2013; Davies et al., 2018; Barcellos et al., 2018, 2023). Finally, one might also expect for the

effects of education on health to vary across the health conditions and behaviors under consideration. Each of these explanations suggest that varying investment productivity over the life cycle is critical to understanding the relationship between education and health.

### **3 Empirical Strategy**

Our empirical analyses leverage a prominent 1972 compulsory schooling reform that produced a historic increase in educational attainment among affected U.K. birth cohorts. In order to lend further context for this natural experiment, we first provide a brief overview of our institutional setting before describing our data sources and estimation methods. We primarily use two data sources in this study. Both datasets contain information on month and year of birth and thus allow us to identify individuals affected by the 1972 ROSLA reform. The Scottish Longitudinal Study (SLS) contains linked administrative records for inpatient hospitalization for a nationally representative sample, but lacks information on years of education. We also use data from the Scottish Health Survey (SHeS) which possesses information on school-leaving age for a nationally representative sample, but only includes self-reported health measures. As we describe in greater detail below, our analyses use the SHeS to both establish a “first-stage” relationship (see Section 3.3) and to assess the effects of the reform on self-reported health. We use the SLS to estimate the reduced-form effects of the reform on our objective health measures in aggregate, and for our hospitalization results, over the life cycle.

#### **3.1 Minimum School Leaving Age in Scotland**

Similar to the rest of Great Britain, Scotland experienced two raising of school leaving age (ROSLA) reforms throughout the twentieth century. Through the 1944 Education Act, Scotland first raised the minimum school leaving age from 14 to 15 in 1947 and did so again from 15 to 16 in September 1972. Thus, students born on or after April 1, 1933 were

compelled to stay in school for one more year relative to their peers born before this date. Similarly, students born on or after September 1, 1957 were also compelled to stay in school for an additional year compared to other students born before this date. As noted in previous work, each of these U.K.-wide compulsory schooling reforms equally applied to England and Scotland (Buscha and Dickson, 2018). However, previous studies generally do not include data from Scotland.

Figure 1 illustrates the effects of each reform on Scottish educational attainment by quarter of birth. Since children typically start school upon reaching five years of age, the 1947 reform (indicated by the first vertical line) pushed the affected cohorts into obtaining at least 10 years of education. As a result, Figure 1 shows that the percentage of students who obtained nine years of education or less was cut by more than half. In the same vein, the 1972 reform dramatically reduced the number of students leaving high school with ten or fewer years of education.<sup>3</sup> The effects of each compulsory reform on Scottish educational attainment are strikingly similar to evidence based on other areas of the U.K. (Braakmann, 2011; Clark and Royer, 2013; Janke et al., 2020).<sup>4</sup>

We dedicate our attention to the effects of the 1972 ROSLA reform on educational attainment and health for two reasons. First, the 1947 ROSLA coincided with an ambitious educational infrastructure and teacher labor force expansion efforts after the Second World War known as the “Hutting Operation for Raising the School Leaving Age” (HORSA) program (Cowan et al., 2012). This program led to the construction of nearly 36,000 new school buildings and thousands of smaller dwellings known as “HORSA huts” by 1949. Thus, we cannot rule out the possibility that health improvements could be affected by changes in the quality of education provided through the first reform (Clark, 2022). These expansion efforts were also largely complete before the 1972 ROSLA reform. Second, attrition among

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<sup>3</sup>Given that the September 1, 1972 implementation date is contained in 1957Q3, the full effect of the 1972 reform in Figure 1 does not materialize until the fourth quarter of that year.

<sup>4</sup>At the time of the 1972 reform, differences between the Scottish and English educational systems were less distinct and featured similar educational attainment rates (Paterson, 2023). However, the contemporary Scottish educational system differs markedly today since its formal devolution from Westminster in 1998.

the 1933 birth cohort, mainly by way of mortality, makes our life cycle analyses less straightforward given that our study period ends in 2016. Given these considerations, our analyses will exclusively focus on the 1972 reform.<sup>5</sup>

## 3.2 Data

### 3.2.1 Scottish Longitudinal Study (SLS)

Our main data source is the Scottish Longitudinal Study (SLS) which links census data to administrative records. The Scottish census takes place approximately every ten years and uses the same methodology developed by the Office for National Statistics (ONS) for the rest of the U.K. The last census took place in 2011 and collected information on education, ethnic identity, religious identity, and housing for the entire Scottish population.

The SLS began in 2006 and created a longitudinal dataset for a representative subset of the population by linking these individuals across the 1991-2011 census waves. These individuals were selected by (semi-)randomly picking 20 of 366 possible birthdays. Any census participant born on one of these dates is automatically included in the SLS – yielding a 5.3% sample (approximately 270,000 individuals) of the entire Scottish population. The SLS records participants’ year and month of birth which we use to construct our running variable (birth month-year). For our analysis, we focus on SLS participants born within 7 years on either side of the September 1957 cut-off date—resulting in around 800 individuals per average month-year cell.<sup>6</sup>

A key advantage of the SLS data is that they allow us to match detailed information on each SLS participant’s National Health Service (NHS) health care utilization records. In particular, we have information on all inpatient hospital admissions and discharges since 1981. These records also describe the number of episodes corresponding to each admission. For example, a patient could be admitted as part of a consulting episode before being trans-

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<sup>5</sup>For completeness, Appendix B documents the effects of the 1947 ROSLA reform.

<sup>6</sup>As we discuss in Section 3.3, automated bandwidth selection from our regression discontinuity design will obviously result in observations that are distant from the cutoff receiving zero weight in the analysis.

ferred into a surgical unit – triggering a second episode within the same spell. We also observe the duration of each hospitalization episode in days. The SLS data contain rich information on the main and secondary diagnoses for each admission as indicated by International Classification of Diseases (ICD) codes.<sup>7</sup> These ICD codes are used to categorize each inpatient episode by disease and injury type. Hospital episodes related to pregnancies and childbirth are excluded from the analysis.<sup>8</sup> SLS participants are also linked more generally to the Scottish Cancer Registry which includes information on all cancer diagnoses (including post-mortem).

The SLS data are uniquely suited to address some outstanding questions on the causal effects of education on health for several reasons. First, the SLS data are nationally representative and avoid many of the self-selection concerns associated with other data sources based on voluntary enrollment. Second, the SLS data allow for analyses of the effects of the 1972 ROSLA reform on more objective measures of health and health care utilization. These administrative records capture genuine differences in the demand for health and health care in adulthood while sidestepping some of the issues corresponding to self-reported health measures. The age profile of the 1957 birth cohort (i.e., ages 24-59) in our study also serves as a critical period of health production. Finally, the longitudinal elements of the SLS data permit estimation of the effects of the reform on hospitalization over the life cycle. Previous work based on cross-sectional data sources generally focus on the aggregate effects of the reforms within or across cohorts.

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<sup>7</sup>Most diagnostic codes follow the 10<sup>th</sup> ICD revision (ICD-10). Cases invoking the older 9<sup>th</sup> revision (ICD-9) were converted to ICD-10. Some of the major classifications within our study include diseases of the circulatory system (I00-I99), respiratory system (J00-J99), digestive system (K00-K93), and sub-categories such as heart disease (I00-I52). We also separately analyze hospitalization related to risky health behaviors such as episodes related to alcohol poisoning (T51, X45, X65, and Y15), intoxication and harmful use (F10.0 and F10.1), alcohol dependency and withdrawal (F10.2 to F10.9), and drug abuse related episodes (T40 and T43.6; F11-F19). See Appendix C for additional details.

<sup>8</sup>Previous work suggests that maternal education can indeed have important effects on fertility choices, prenatal care, and infant health outcomes (Currie and Moretti, 2003; Black et al., 2008; McCrary and Royer, 2011). We focus on hospitalization not related to pregnancies and childbirth due to the more complicated nature of maternal health care delivery and to provide a more straightforward interpretation of our results. Understanding the effects compulsory schooling reforms on maternal health care utilization is a critical question worthy of further exploration in future research.

The SLS takes several precautions with respect to selective sample attrition and retention. For example, immigration could present important challenges to our study design. Individuals who emigrated to Scotland later in life, but by virtue of their birth month-year would appear to have been affected by the ROSLA reforms, could falsely be classified as “treated.” Fortunately, the SLS is regularly cross-checked with NHS registrations. Since all residents are required, and possess a strong incentive to register with the NHS upon relocating in order to receive free universal health care, we can reliably assess their immigration status. In other words, we do not believe that left-censoring presents a meaningful challenge to our findings. As a precaution, we also limit our sample to individuals who were either born in the U.K. or arrived in the U.K. before turning 14 years old. Similarly, selective emigration could also lead to important empirical hurdles. However, emigrants are required to notify the NHS when they move abroad and these cases will be accounted for in the SLS data. Rare cases of emigration without notification can be detected by virtue of the census. Dedicated SLS staff examine the whereabouts of all participants who show up in one census wave but not the next. Deaths of individuals who were born on one of the 20 semi-randomly selected birthdays are also transmitted to the SLS by mortality registries.

The SLS also contains information on each participant’s highest educational qualification such as O-Grades (equivalent to English O-Levels) or a university degree in addition to other basic demographic information typically surveyed within the census (e.g., age, sex, ethnicity, occupation, and post-code level of deprivation).<sup>9</sup> However, one important limitation of the SLS education measures involves the absence of specific details concerning each participant’s years of formal schooling. For this information, we instead turn to a secondary data source.

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<sup>9</sup>Any high school qualification is typically obtained through exams at the end of year 11 when students typically turn 16. We show below that the 1972 ROSLA thus lowered the probability of leaving high school without any qualification.

### 3.2.2 Scottish Health Survey (SHeS)

The Scottish Health Survey (SHeS) is a nationally representative survey and is primarily based on a personal interview. We specifically pool information from the 1995, 1998, 2003, 2008-2011, and 2012-2016 survey waves. The SHeS serves as the Scottish equivalent to the Health Survey of England (HSE) widely used elsewhere in the literature. One shared feature of the SHeS and HSE involves their very similar measures of self-reported health and health behaviors. One such question within the SHeS asks respondents to assess their health on a five-point Likert scale ranging from “very good” to “very bad.” We group the bottom three categories of “fair”, “bad”, and “very bad” health into a single dichotomous indicator of “poor health.” Survey participants also report whether they suffer from any longstanding illness, consume alcohol, or engage in any current or past smoking behaviors. We also code these subjective health indicators as dichotomous variables.

Crucially, the SHeS contains information concerning the age at which a person left full-time education. In contrast to the SLS data, the SHeS allows us to calculate years of schooling for each respondent. As shown in Figures 1 and 2, we use this information in order to construct our first-stage estimates describing the effects of the 1972 ROSLA reform on educational attainment.

In order to illustrate the representativeness of both SHeS and SLS, we compare the one common education measure contained within both the SLS and SHeS describing whether a participant left full-time education without any qualification. Appendix Figure A3 demonstrates that in both the SLS and SHeS data, the 1972 ROSLA reform reduced the percentage of the population leaving school without any formal educational qualification by roughly four percentage points. This finding is comforting and suggests that both datasets are indeed comparable.



### 3.3 Regression Discontinuity Estimation

Our identification strategy leverages exogenous variation in educational attainment through the 1972 ROSLA reform in order to estimate the effects of education on health over the life cycle. The retroactive application of the reform specifically allows for a regression discontinuity (RD) design in which students born on or after September 1, 1957 were compelled to stay in school for an additional year relative to their counterparts born before the policy cutoff date. We use the SHeS data to produce our first-stage estimates, in addition to reduced-form estimates for our subjective health measures, based on specifications of the following form:

$$Y_{ic} = \alpha_0 + \alpha_1 D_{ic} + f(Run_{ic}) + \alpha_2 \mathbf{X}_{ic} + \varepsilon_{1ic} \quad (3)$$

where  $Y_{ic}$  is years of formal schooling (i.e., the age at which an individual leaves full time education minus five) or a subjective health outcome in adulthood for student  $i$  from birth cohort  $c$  (i.e., month-year) at time  $t$ .  $D_{ic}$  is a binary indicator of being born on or after the cutoff date, and  $f(Run_{ic})$  is a function of our birth month-year running variable centered around the reform cutoff date. Estimation of (3) also includes a vector of covariates  $\mathbf{X}_{ict}$  which we keep parsimonious (e.g., sex, ethnicity and childhood religion) in order to avoid issues pertaining to collinearity. All estimates are obtained from local polynomial regression discontinuity (RD) estimation with automated bandwidth selection and heteroskedasticity-robust standard errors with nearest neighbor variance estimation (Lee and Card, 2008; Calonico et al., 2014). Our estimates are robust to both alternative bandwidth choices and polynomials of different orders.<sup>10</sup>

We employ the exact same RD methods in estimating the reduced-form effects of the reform on our objective health measures, but now use the SLS data to estimate either

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<sup>10</sup>We show in Figure A1 that alternative bandwidths that deviate from this optimal choice produce similar results.

aggregate effects or those up to specific ages  $a(t)$  over the life cycle:

$$H_{ict}^{a(t)} = \beta_0 + \beta_1^{a(t)} D_{ic} + f(Run_{ic}) + \beta_2 \mathbf{X}_{ict} + \varepsilon_{2ict} \quad (4)$$

where  $H_{ict}^{a(t)}$  is an objective health outcome. For our hospitalization results, we produce these reduced-form estimates both in aggregate and over the the life cycle. We also perform estimation separately by admission type and sex. When focusing on cancer outcomes,  $H$  is instead a binary indicator of ever receiving a cancer diagnosis across all health care settings (i.e., not only within a hospital) and we produce aggregate estimates by cancer type and sex.

As noted in Section 3.2, we are unable to directly estimate the causal effect of an additional year of schooling on  $H_{ict}^{a(t)}$  as the SHeS contains our preferred educational attainment measure while our main health outcomes are in the SLS data. Given that each of these estimates are based on nationally representative data, we instead approximate the local average treatment effect (LATE) by scaling our reduced form estimates by our first-stage (Imbens and Angrist, 1994; Angrist and Pischke, 2009). Given the high compliance rates surrounding the 1972 ROSLA reform, these LATE estimates will be close to the population average treatment effect (ATE) and lend some external validity to our results (Oreopoulos, 2006).

The causal effect of an additional year of education on health rests on the standard instrumental variables (IV) identifying assumptions. Similar to previous studies leveraging the U.K. ROSLA reforms, the natural experiment within Scotland benefited from the retroactive application of the reform precluding sorting around the cutoff potentially driven by strategic parental fertility choices and enrollment.<sup>11</sup> As shown in Section 3.1, the pronounced decline in students finishing with no more than a 10<sup>th</sup> grade education suggests a strong “first-stage”

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<sup>11</sup>We provide additional information on various predetermined characteristics and outcomes in Table A1 of the appendix. For example, Figure A2 shows no evidence of differences in the percentage of individuals who were raised Protestant. Past (and to some extent contemporary) ethnic conflict in Scotland proceeded primarily along religious lines in which Protestants of the Church of Scotland are the majority and Roman-Catholics, often of Irish descent, are a sizeable minority. We also find no evidence with respect to discontinuities in ethnic and gender composition.

relationship between the 1972 ROSLA reform and educational attainment.

Table 1 serves as the regression analog to Figure 1 and shows the effects of the 1972 reform on years of formal schooling.<sup>12</sup> Our RD estimates suggest that the 1972 reform increased average educational attainment by 0.42 years and decreased the probability of dropping out after 10 years of schooling by roughly 28.4 percentage points. These effects are similar for men and women, and more importantly, do not extend beyond the 11<sup>th</sup> grade. Figure 2 provides graphical evidence of these findings and suggests that compliance with the law was quite strong. Moreover, we can also conclude that most students affected by the reform (i.e., the “compliers”) would have dropped out of school otherwise. Both average levels of education and our first-stage results for Scotland are very consistent with findings from the existing literature based on the experiences of England and the rest of the U.K. (Clark and Royer, 2013; Buscha and Dickson, 2018; Janke et al., 2020).<sup>13</sup>

## 4 Results

### 4.1 Inpatient Hospitalization

Within the context of the Grossman model, inpatient care serves as one potential input in the production of health. However, hospitalization itself is often an acute medical event driven by prior health investment decisions and behavior. For example, educational differences in primary care visits critical to the monitoring of cardiovascular and early cancer detection could ultimately manifest in hospitalization disparities later in life (Kaestner and Sasso, 2015). In the case of allocative efficiency, additional education could also lead to individuals choosing different inputs altogether in ways that produce similar disparities. Thus, we view hospitalization as both an input for health production and a proxy for certain health

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<sup>12</sup>Note that because the optimal bandwidth slightly varies across outcomes in Table 1, the year-specific coefficients do not add up exactly to the years of education coefficient.

<sup>13</sup>First-stage estimates based on the U.K. Biobank project, in contrast, tends to over-sample more educated participants and therefore find smaller effects of the 1972 reform on educational attainment (Davies et al., 2018; Barcellos et al., 2018, 2023)

and health behaviors.<sup>14</sup>

Table 2 formally investigates the aggregate effects of the 1972 ROSLA reform on the demand for inpatient care over the 1981 to 2016 period by sex. Panel A shows that men affected by the reform, on average, experienced 1.6 fewer inpatient hospitalization episodes than cohorts born before the cutoff date. This point estimate implies a 0.16 standard deviation decline in inpatient episodes and is statistically significant at conventional levels. We also find a slightly smaller standardized effect when expressed in terms of inpatient care days. Our results are also suggestive of small declines in inpatient care utilization among women affected by the reform – although the standard errors are too large to rule out a null effect.

Note that the coefficients shown in Table 2 are reduced-form estimates. The corresponding LATE estimates can be obtained by scaling each of these reduced-form coefficients by the first-stage estimates shown in Table 1. Given that the 1972 reform led to a 0.44 year increase in formal schooling, our results indicate that an additional year of education for men reduces the number of inpatient episodes by 0.37 standard deviations— a large and economically significant reduction. Although this estimate carries some noise, the lower bound of our 95% confidence interval suggests a modest 0.13 standard deviation reduction. Interestingly, these estimates differ somewhat from two existing analyses of school reform effects on hospitalization in Denmark and Sweden ([Arendt, 2008](#); [Meghir et al., 2018](#)).<sup>15</sup>

While our flexible control for birth year-month should account for any differences in age across cohorts, we also explicitly test for age-specific effects of education by limiting our sample to individuals born between September 1951 and August 1963. These restrictions lead to the youngest person in our sample being 53 years old in 2016 when our study period

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<sup>14</sup>When treating health as a pure investment, this specific relationship between education and medical care utilization technically holds if the elasticity of the marginal efficiency of capital (MEC) curve is less than one ([Grossman, 2000](#)). We explore the extent to which this relationship holds over the life cycle in Section 5.2.

<sup>15</sup>These studies also differ from our own in terms of the type schooling reforms being entertained, identification strategies, and other important contextual factors (e.g., health care systems and underlying demographic populations).

ends and the oldest person 29 years old in 1981 when our inpatient data begin. In other words, we observe hospitalization records between the ages of 29 and 53 for every person in this sub-sample—allowing us to assess the effect of the 1972 ROSLA reform on inpatient admissions for a fixed age range. Panel B of Table 2 shows that our results are robust to these age-specific sample restrictions. The reform reduced the number of inpatient episodes and days experienced by men, between ages 29 and 53, by roughly 0.16 standard deviations. The point estimates for women are again smaller and only borderline statistically significant. Taken at face value, these results suggest that the compulsory schooling reform reduced the number of inpatient episodes between ages 29 and 53 by approximately 0.11 standard deviations for women.

Overall, our analysis of inpatient hospitalization suggests that education reduces hospitalization primarily among men. The graphical evidence in Figure 3 supports our regression results. Panels (a) and (c) show a clear drop in the number of inpatient episodes and days for men who were just affected by the 1972 reform. For women of the same cohort, on the other hand, these drops are much less clear and indicative of very small effects at best.<sup>16</sup>

## 4.2 Cancer Diagnoses

The sharp increase in educational attainment could also have important implications for cancer diagnoses later in life. Table 3 provides our reduced-form estimates of the effects of the 1972 ROSLA reform on the probability of receiving a cancer diagnosis for both men and women. We again find that these effects are primarily concentrated among men with the point estimate in column (1) suggesting a highly significant 4.6 percentage point reduction in the probability of receiving any cancer diagnosis within this group. However, evidence for some of the most prevalent cancer types, such as lung cancer, urinary (i.e., mostly issues related to the prostate) cancer, and breast cancer, is weaker. Given that our cohort of

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<sup>16</sup>Appendix Table A3 further shows that our findings cannot be explained by selective migration as the reform at best produces small, statistically insignificant effects on emigration. We also subject our principal findings to several falsification tests. Appendix Table A4 confirms the absence of any statistically significant placebo effect with respect to both education and inpatient admissions.

interest is still quite young by the end of our observed sample period, these results are best interpreted as a potential reduction in the early onset of cancer (Leuven et al., 2016).

While the reductions in cancer incidence associated with the 1972 ROSLA reform could reflect genuine health improvements, these effects could also be driven by changes in the composition of inputs employed in health production. For example, more educated individuals could demand more annual cancer screenings and check-ups (Smith, 2007; Cutler and Lleras-Muney, 2010; Lange, 2011; Palme and Simeonova, 2015). While our SLS data capture cancer diagnoses across the health system (i.e., not solely diagnoses taking place in a hospital), they unfortunately do not contain information on screening utilization such as colonoscopies. The SHeS also lack information on the use of cancer screenings and preventative care. However, higher screening rates among the treatment group would generally imply *higher rather than lower incidence* of cancer diagnoses to the right of the cutoff. Furthermore, the Scottish Cancer Registry also includes post-mortem cancer diagnoses which would, if anything, attenuate the estimates shown in Table 3.<sup>17</sup> Of course, cancer diagnoses are arguably more prone to measurement error than hospitalization so these results are best interpreted as suggestive evidence.

### 4.3 Self-Reported Health and Health Behavior

We also ask whether the 1972 ROSLA reform led to any meaningful changes in subjective health outcomes reported within the pooled SHeS data. Table 4 presents our ordinary least squares (OLS) and reduced-form estimates for several self-reported (binary) health and health behavior outcomes. Our two OLS specifications use either years of schooling or a dummy variable for having more than 11 years of schooling as measures of educational attainment. We once again provide separate estimates for both men and women.

Our OLS estimates generally confirm the familiar, positive relationship between education and health. For example, an additional year of schooling reduces the probability of

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<sup>17</sup>Post-mortem cancer diagnoses account for just 1.4% of entries. All results are robust to excluding these detections.

reporting poor health by 3.5 percentage points and the prevalence of long-standing illnesses by 2.0 percentage points. Education also reduces the probability of being a past or present smoker. Interestingly, an additional year of education increases the likelihood of current alcohol consumption— a finding which could reflect changes in social circumstances that ultimately alter drinking behavior rather than serving as a sign of alcohol abuse (Huerta and Borgonovi, 2010). These effects are similar for both men and women. Specifications based on our dummy indicator for completing more than 11 years of schooling produce qualitatively similar, statistically significant effects for each of our outcomes.

In line with the literature on education and health, our reduced-form estimates demonstrate the well-documented issues of endogeneity among the OLS estimates. None of our reduced-form RD estimates in Table 4 are statistically significant at conventional levels. Not only are these estimates smaller in magnitude relative to the corresponding OLS estimates, but some results also possess the opposite sign. For example, the first column of Table 4 suggests that the 1972 ROSLA reform *increased* the incidence of poor health by 0.4 percentage points— although we cannot rule out the possibility of no effect or meaningful reductions either. These findings are also consistent for both men and women.

Graphical evidence within Figure 4 also provides no clear evidence of changes in the incidence of poor health or long-standing illness among month-year birth cohorts close to the cutoff. As such, both our OLS and reduced-form estimates are strikingly similar those reported in Clark and Royer (2013) who invoke a nearly identical natural experiment resulting in extraordinary changes in educational attainment. It should, however, be noted that our results are based on a smaller sample and are therefore less precisely estimated. For example, the standard errors corresponding to current smoking status would lead us to fail to reject the null hypothesis that an additional year of compulsory schooling reduced the probability of being a current smoker by 10 percentage points. Our results are thus merely suggestive of a lack of an education-effect on subjective measures of health and health behaviour, even

though we cannot rule out modest benefits in a statistical sense.<sup>18</sup>

We also cautiously interpret our subjective health results due to potential concerns regarding nonresponse bias and other forms of measurement error among these outcomes (Dutz et al., 2021). A notable example of divergence in subjective and objective health measures comes from Johnston et al. (2009) who compare self-reported measures of hypertension with blood pressure measurements taken by a nurse practitioner. The authors find that 85% of their sample engaged in false negative reporting of hypertension with lower income households being significantly more likely to provide a false negative report. These concerns equally extend to health behaviors such as alcohol consumption and smoking.

#### 4.4 Education and Mortality

We also assess the effects of the 1972 reform on mortality. On the one hand, the Grossman model views the length of life as being endogenously determined and one might expect that education affects mortality through differences in health investment over time. On the other hand, we might also have concerns that selective mortality could influence the interpretation of our results. For example, higher mortality among men affected by the reform, relative to women, could also produce the documented gender differences in hospitalization. This “survivorship bias” could be driven by the least healthy men subject to the 1972 ROSLA experiencing relatively higher mortality—resulting in a less straightforward comparison. Education-induced mortality reductions could also account for differences in health outcomes across cohorts. If an additional year of education improves the longevity of a cohort’s least healthy members, we would underestimate the effects of the compulsory schooling reform on health and health care utilization (Barcellos et al., 2018, 2023).

However, death is a rare event in our sample with roughly three percent of SLS participants passing away during our study period. With these caveats in mind, we investigate the effects

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<sup>18</sup>In Appendix Table A2, we report two-stage least square estimates which also suggest no statistically significant effect for these outcomes—although these estimates are quite noisy even relative to our reduced form results.



of the 1972 reform on mortality by drawing on population data. For this purpose, we obtained data from the 1991 Scottish Census and population mortality records from National Records of Scotland. Since both data sources contain counts by month and year of birth, we can combine them into a single individual-level panel dataset in order to examine whether the 1972 ROSLA reform led to any significant changes in mortality. Following previous work (Sullivan and Von Wachter, 2009; Clark and Royer, 2013), we assess whether the reform produced a discontinuous change in mortality using a two-step estimation procedure over the 1991-2016 period. We first estimate a panel logit model in which the probability of dying in each month  $t$  for individual  $i$  of cohort  $c$  is a function of a full set of month-year cohort dummies,  $\theta_c$ , as well as age fixed effects  $\delta_a$ :

$$P(\text{Death}_{ict}|\theta_c, \delta_a) = F(\theta_c + \delta_a) \quad (5)$$

Using estimates  $\hat{\theta}_c$  from (5) as our dependent variable, the second step involves estimating a local linear model of the following form:

$$\hat{\theta}_c = \pi_0 + \pi_1 D_c + f(\text{Run}_c) + \gamma_m + \epsilon_c \quad (6)$$

where  $\gamma_m$  is a set of calendar-month fixed effects. Intuitively,  $\hat{\theta}_c$  measures the effect of being born in a particular cohort on the log odds of death. Equation (6), in turn, assesses whether a significant change in these effects takes place for cohorts born around the time of the compulsory schooling reform. Such a discontinuous change in mortality would be captured by  $\pi_1$ . We estimate equation (6) using weighted least squares where the weights are given by the inverse of the standard errors from estimation of equation (5). We also cluster our standard errors at the cohort level.

We present our findings from this analysis in Figure 5 which reveals no evidence of a discontinuous jump in the log odds of death for cohorts affected by the reform. These point estimates are economically small, -0.019 for men and -0.002 for women, and statistically

insignificant at conventional levels. The sign for each of these estimates also suggests that any mortality effects would work in the opposite direction of any potential survivorship bias in our main health estimates.<sup>19</sup>

Similar to [Clark and Royer \(2013\)](#), we fail to find any significant evidence of mortality effects associated with the 1972 ROSLA reform. Given the age of the 1957 birth cohort, it is quite possible that mortality benefits for this group could manifest only later in life. As such, our finding of fewer hospitalization without lower mortality rates is very much consistent with a dynamic model developed by [Lleras-Muney and Moreau \(2022\)](#). Their simulations predict that health effects would appear substantially earlier than any mortality effects. This is because, as outlined in Section 2, death only occurs once the stock of health falls below a critical level. In other words, any longevity improvements due to education will only become apparent later on in the life cycle when individuals' health stocks move closer to the mortality threshold.

## 5 Mechanisms

### 5.1 Lifestyle-Related Behavior

Health behavior plays a critical role in shaping the educational gradient in health ([Mokdad et al., 2004](#); [Cutler and Lleras-Muney, 2010](#)). Indeed, the health literature generally finds persistent educational differences in outcomes such as excess alcohol consumption and obesity which are often viewed as modifiable risk factors for conditions such as heart disease ([Cawley, 2015](#); [Saffer et al., 2016](#); [Whitman et al., 2017](#)). A handful of recent studies based on the same U.K. compulsory schooling reforms also find evidence of significant reductions in lifestyle-related health outcomes ([Davies et al., 2018](#); [Barcellos et al., 2023](#)). Using the 1972 ROSLA reform, [Janke et al. \(2020\)](#) find that an additional year of education resulted in a

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<sup>19</sup>As a robustness check we also deployed the same two-step approach outlined in this section to an analysis of our hospitalization outcomes. The results of this exercise are shown in Appendix Figure A5 and confirm our main result.

0.147 standard deviation reduction in a combined measure for self-reported cardiovascular disease and diabetes among U.K. adults. Similarly, [Barcellos et al. \(2018\)](#) find that the same reform led to significant reductions in unhealthy body size and lung function which are more pronounced for middle-aged U.K. adults with greater genetic predisposition to obesity.

We can formally assess the effects of the compulsory schooling reform on lifestyle-related health outcomes by disaggregating our hospitalization results by main diagnosis. Figure 6 shows the standardized reduced-form effects of the reform on inpatient care episodes across 12 major health categories. Many of the improvements in hospitalization rates that we observe are primarily driven by improvements in cardiovascular health, most notably heart disease, and internal health complications related to the digestive system (e.g., intestines, gall, biliary, pancreas, and the stomach). These effects are mostly concentrated among men. We also find evidence of significant reductions in hospitalization primarily attributable to metabolic disease (e.g., diabetes), injuries, and mental disorders. Taken together, these findings suggest that the 1972 ROSLA reform produced notable reductions in hospitalization for several lifestyle-related health outcomes.<sup>20</sup>

Excessive alcohol consumption, chronic drug use, and smoking serve as important choice variables in health production ([Grossman, 2000](#)). In many respects, hospitalization driven by these health behaviors can convey important information regarding underlying differences in gross health investment or the willingness to participate in risky behaviors such as binge drinking ([Markowitz and Ding, 2020](#)). Within the context of our study, alcohol and drug-related hospitalization outcomes go beyond self-reported participation measures and instead reveal realized differences in deleterious behaviors serious enough to require inpatient care.

Table 5 leverages our hospitalization data in order to closely investigate the nature of this relationship within the context of the 1972 ROSLA reform. We first turn to our results

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<sup>20</sup>Appendix Figure A6 provides similar hospitalization results when using inpatient days rather than episodes. These results also show that the reform produced qualitatively similar declines in hospitalization days for lifestyle-related health conditions. Interestingly, the reduced-form effects for hospitalization for metabolic disease and the musculoskeletal conditions (e.g., chronic pain conditions) are somewhat larger when expressed in inpatient days. This difference in findings could potentially be an artifact of condition-specific hospitalization intensity along the intensive and extensive margins.

for alcohol-related inpatient admissions.<sup>21</sup> Our findings suggest that the health benefits of education, in the form of lower alcohol abuse rates, again accrue primarily to men. An additional year of education produces roughly a quarter of a standard deviation reduction in the number of inpatient episodes related to excessive alcohol consumption. Again, the differences across gender are remarkable as we fail to find any such pattern for women. The graphical evidence in Figure 7 corroborates this finding. Panel (a) displays a drop in the number of alcohol-related inpatient episodes for men born around the September 1957 cutoff date whereas no such drop can be found for women. Panel (b) of Table 5 shows that our results are robust to restricting our analysis to hospitalization between ages 29 and 53.

We also analyze the effect of education on inpatient hospitalization indicative of acute drug abuse. However, we note that hospitalization for drug abuse is quite rare in these data and therefore influences the precision of our estimates. With this caveat in mind, columns (5) and (6) of Table 5 document a negative relationship between ROSLA exposure and drug-related hospitalization. While these estimates are not statistically significant, we view them as potentially suggestive evidence of a negative effect of education on drug abuse.<sup>22</sup>

## 5.2 Life Cycle Effects

The reduced-form estimates shown thus far, demonstrating the effects of the 1972 ROSLA reform on health in adulthood, reflect aggregate effects during prime working-age years. However, the marginal productivity of health investment might be age-specific as health stock continues to depreciate later in life. In order to formally address this question, we leverage the inherent panel dimension of the SLS data and estimate the effects of the compulsory schooling reform on hospitalization through each observed age (e.g., the number of inpatient episodes by age 40) over more than two decades. More formally, we use the local polynomial RD approach outlined in Section 3.3 to obtain dynamic reduced-form estimates  $\beta_1^{a(t)}$  through

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<sup>21</sup>Alcohol-related inpatient admissions include hospitalization attributable to alcohol poisoning, intoxication, harmful use, alcohol dependency, or withdrawal.

<sup>22</sup>Given that hospital admissions for drug abuse are fairly rare, we were required to group men and women together for the graphical results shown in Appendix Figure A7 for disclosure control reasons.

each age  $a(t)$ . We then plot the standardized point estimates and 95% confidence intervals separately by sex.

Figure 8 presents our evidence concerning the effects of the 1972 ROSLA reform on inpatient hospitalization episodes over the life cycle in total and across various health conditions. Panel (a) shows our results for total inpatient hospitalization episodes, and while both men and women appear to benefit from earlier human capital investment through the reform, the sharpest reductions primarily occur among men beginning in their early 40s. These benefits persist through men's 40s and 50s while women enjoy substantially smaller health benefits that improve more slowly over the life cycle. Thus, Figure 8a confirms our qualitative understanding of gender differences in the education-health gradient while also articulating important age-specific effects as conceptualized by the extended Grossman model.

One might suspect that the evidence shown in Panel (a) simply reflects educational differences in inpatient care utilization rather than genuine improvements in physical health. Panels (b) through (d) of Figure 8 show that this is indeed not the case as the life cycle effects of the reform differ considerably across inpatient care diagnoses. For example, men affected by the reform experienced extraordinary reductions in hospital admissions for heart disease. These improvements in hospitalization appear as early as men's late 30s and grow to more than a -0.2 standard deviation reduction throughout their 40s and 50s. Women do not appear to share in any of these improvements over the exact same age profile. In contrast, Panel (c) provides evidence of significant declines in hospitalization associated with intestinal issues for both men and women over the life cycle— although these point estimates are larger for men upon reaching their mid-40s.

While the reduced-form effects for heart disease and intestine-related admissions evolve somewhat similarly over the life cycle, the age profile for injury-related hospitalization effects remains fairly flat. Panel (d) suggests that men affected by the 1972 ROSLA reform experienced approximately a -0.1 standard deviation decline in injury-related hospitalization. These point estimates are salient, and statistically significant, beginning in men's mid-30s

up until their early-50s.

Overall, the effects of an additional year of education on inpatient hospitalization are both age- and condition-specific. Similar to our aggregate reduced-form estimates, the health benefits of the reform are primarily concentrated among men with an exception for hospitalization related to the digestive system. Heterogeneous effect sizes and age profiles also suggest that the reform produced important changes not only in health care utilization, but also in health given that many of the well-known epidemiological characteristics of cardiovascular disease generally emerge within populations in this intermediate age range. Again, these results are very much consistent with theoretical contributions in this area. For example, [Lleras-Muney and Moreau \(2022\)](#) assume that health shocks related to accidents are distributed independently of health stock. Under this scenario, one might expect for accidents to vary over the life cycle in a manner that is quite different from disease-related shocks with more direct biological causes. The lack of a clear life cycle pattern for injury-related hospitalization, many of which are attributable to accidents or other extrinsic causes, aligns closely with predictions from their model.

### 5.3 Socioeconomic Factors

Socioeconomic outcomes, for example higher earnings, offer a further channel through which education might influence health ([Harmon and Walker, 1995](#); [Oreopoulos, 2006](#); [Oreopoulos and Salvanes, 2011](#)). Estimates based on the 1972 ROSLA reform generally suggest a 5-7% average increase in hourly wages for U.K. adults throughout adulthood. ([Grenet, 2013](#); [Buscha and Dickson, 2018](#)).<sup>23</sup> [Barcellos et al. \(2021\)](#) also find that this reform uniformly increased wages for middle-age U.K. adults, but did little to improve economic disparities.

While neither the SLS nor the SHeS contain individual earnings, the SLS records some information on neighborhood characteristics. More specifically, our data contain information

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<sup>23</sup>Earlier studies leveraging the 1947 reform point to a positive effect on earnings for men throughout the U.K. ([Oreopoulos, 2006](#); [Devereux and Hart, 2010](#)). [Clark \(2022\)](#) fails to reject null effects for the 1947 reform when considering labor market participation, unemployment, earnings, and home ownership.

on the Scottish Index of Multiple Deprivation (SIMD). To calculate the SIMD, Scotland is divided into 6,976 small areas (known as “data zones”) which are then ranked by levels of deprivation. Income is an important input for these calculations in addition to other factors such as employment, access to services, and housing. Put differently, the SIMD is a comprehensive measure of respondents’ socioeconomic environment and may well have a broader impact on health than any pure earnings measure.

Table 6 provides reduced-form evidence on the effect of the 1972 reform on SLS respondents’ socioeconomic environment later in life. We first define two distinct outcomes for neighborhood quality according to whether an individual resided in one of the 10% most deprived (i.e., “Most Deprived”) and 10% most affluent (i.e., “Least Deprived”) data zones as per the SIMD in either the 1991, 2001, or 2011 census. We also construct a measure for whether a SLS respondent resided in a data zone with below-median levels of deprivation. Using these measures, we find no meaningful evidence of changes in neighborhood quality for men. Women affected by the reform are also no more likely to reside in a very affluent or deprived neighborhood later in life, but are somewhat more likely to live in a neighbourhood with below-median levels of deprivation. Overall, the compulsory schooling reform did not produce any noteworthy changes in the probability of (re-)locating to a substantially more affluent neighborhood later in life.

The census-component of the SLS also contains information on respondent occupation. We classify respondents as “skilled” if they report employment in professional, managerial, technical, and other forms of skilled occupations. The fourth column of Table 6 shows little in the way of an effect of the ROSLA reform on occupational choice in adulthood. Finally, we also draw on self-reported household income data that are available for a sub-sample of the SHeS. The last column of Table 6 suggests a positive (household) income effect for men, but we cannot rule out null effects for men or women as these estimates are fairly imprecise.

Admittedly, our measures for socioeconomic status are too crude to fully rule out any potential income or other socioeconomic effects associated with the 1972 reform. For ex-

ample, self-reported household income generally suffers from measurement error, and in the presence of other household members with positive income, will differ from findings based on individual earnings. Even with these caveats in mind, our evidence collectively suggests that the reform produced at most limited improvements in socioeconomic status later in life.

There are several reasons why other socioeconomic factors might fail to account for a significant portion of our main findings on health and hospitalization. Most importantly, our study takes place in a context in which a critical input within the health production function, health care, is freely and universally accessible through the NHS. While some disparities certainly exist in navigating the overall U.K. health care system, these inequities are generally of limited importance when explaining educational differences in health behaviors. While we cannot completely rule out income and other socioeconomic effects as mechanisms through which education influences health, the institutional setting of the U.K. is likely to limit their importance relative to, for example, the United States.

## 6 Conclusion

We revisit a classic question within the economics literature regarding the causal effects of education on health and health care utilization. If education primarily influences health through more efficient health production, these effects will likely vary across health measures and over the life cycle. Using nationally representative administrative data, we provide new evidence on the effects of compulsory schooling reforms on both health and health care utilization. The 1972 ROSLA reform equally applied to all constituent nations throughout the U.K. and resulted in a substantial proportion of the population receiving an additional year of formal schooling.

Consistent with a growing literature based on objective health measures ([Barcellos et al., 2018](#); [Davies et al., 2018](#); [Barcellos et al., 2023](#)), we find that this reform led to significant reductions in the demand for inpatient care in adulthood. For men, our LATE estimate sug-



gests that an additional year of education reduces the number of overall inpatient episodes by 0.37 standard deviations and admissions for alcohol abuse by 0.25 standard deviations. Moreover, our estimates for overall hospital admissions among men are overwhelmingly concentrated among lifestyle-related health conditions and cannot be accounted for by changes in income or occupation.

Health conditions such as heart disease and diabetes are important drivers of costly, avoidable hospital admissions and could reflect underlying differences in the consumption of preventative health care resources (OECD, 2021; Rittiphairoj et al., 2022). Men affected by the reform also experienced significantly lower cancer incidence later in life—a finding which may reflect earlier detection and screening within this group (Cutler et al., 2011). Overall, our evidence suggests that similarly designed educational policies could serve as one potential tool in reducing inefficient hospitalization for these conditions and more effective adoption of life-saving screening practices.

Our study provides new evidence demonstrating that the effects of education on health vary considerably over the life cycle. These hospitalization effects generally possess distinct age profiles across primary diagnoses. An additional year of education does little to improve hospitalization rates for younger adults affected by the reform, but become more pronounced for this group beginning in their late-30s and persist until early-50s. Men experience sharp reductions in hospitalization for heart disease while both men and women see significant declines hospitalization for intestinal issues. Given that any income effects from the reform likely occur much earlier in life, our evidence suggests that the more educated either enjoy more efficient health production or employ distinct input mixes when confronting critical periods of health stock depreciation later in life (Kaestner et al., 2020).

We also find that an additional year of education fails to produce any meaningful reductions in the probability of reporting poor health or engaging in risky health behaviors such as smoking. These self-reported health measures inherently face some measurement error issues which in best case scenarios result in noisy estimates biased towards zero. For example,

our objective health estimates suggest that an additional year of education leads to significant reductions in hospitalization for alcohol abuse, but no meaningful changes in current alcohol consumption. While alcohol-related hospitalization possesses a more straightforward interpretation, the latter measure captures behavior ranging from “rare or occasional” consumption to “binge drinking.” Similarly, Likert scale measures of physical health may simply reflect changes in perceived overall well-being or even within-cohort health comparisons rather than tangible health improvements (Finkelstein et al., 2012; Lleras-Muney, 2022). We conclude that measurement error in these self-reported health outcomes, in addition to potential non-response survey bias (Dutz et al., 2021), could result in attenuated estimates of the effects of education on health (Bound, 1991).

We document robust and persistent gender differences in the health returns to education. Similar to other countries, the life expectancy gap at birth is roughly three years and favors women within the U.K. (Buxton, 2021)—although the exact mechanisms driving these differences are still not fully understood (Lawlor et al., 2001; Barford et al., 2006; Beltrán-Sánchez et al., 2015). An extended Grossman model offers some additional insight into these gender differences primarily through two channels. First, women might possess distinct biological advantages which allow for them to enjoy either higher levels of initial health stock or health stock which depreciates more slowly (Austad, 2006; Cullen et al., 2016). The “female advantage” could also emerge through differential responses to public health interventions or differences in other environmental exposures (Goldin and Lleras-Muney, 2019). These differences, in turn, are likely to translate into gender differences in the education-health gradient. We view these gender differences in the health returns to education as a critical area for future research.

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# Tables and Figures

Table 1: Effects of the 1972 ROSLA Reform on Education

Years of Education	$\leq 9$ years	$\leq 10$ years	$\leq 11$ years	$\leq 12$ years	$\leq 13$ years	
All (N=66,655)						
Estimate	0.417 (0.102)	-0.019 (0.005)	-0.284 (0.015)	-0.014 (0.023)	-0.023 (0.020)	-0.025 (0.020)
Outcome Mean	11.98	0.015	0.336	0.593	0.726	0.782
Bandwidth	43.25	63.16	59.66	52.92	45.39	47.68
Men (N=29,390)						
Estimate	0.443 (0.113)	-0.024 (0.008)	-0.283 (0.021)	-0.018 (0.029)	-0.029 (0.026)	-0.028 (0.021)
Outcome Mean	12.03	0.017	0.343	0.596	0.707	0.771
Bandwidth	56.25	76.97	81.49	59.40	54.59	62.39
Women (N=37,265)						
Estimate	0.435 (0.140)	-0.016 (0.006)	-0.283 (0.016)	-0.013 (0.029)	-0.026 (0.028)	-0.026 (0.026)
Outcome Mean	11.95	0.014	0.331	0.591	0.740	0.790
Bandwidth	42.42	58.85	58.57	55.05	46.26	46.76

Table 1 reports estimates for the first stage effects using local polynomial regression discontinuity estimation. Estimates are provided for years of education and five distinct levels of educational attainment, respectively, as outcomes. We also flexibly control for birth month-year, centered around the reform cut-off date. All data come from pooled waves of the Scottish Health Survey (SHeS) over the 1995-2016 period. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses.

Table 2: Effects of the 1972 ROSLA Reform on Inpatient Hospitalization

<i>Panel A: Full Sample</i>				
	Men		Women	
	Episodes	Days	Episodes	Days
Mean	5.147	13.870	6.126	14.210
[SD]	[9.792]	[57.350]	[10.180]	[48.421]
Coef (SE)	-1.614 (0.526)	-5.13 (2.184)	-0.658 (0.516)	-1.672 (3.241)
N	32,164	32,164	32,103	32,103
Bandwidth	14.23	16.24	33.49	31.63
<i>Panel B: Restricted Sample (Ages 29-53)</i>				
	Men		Women	
	Episodes	Days	Episodes	Days
Mean	2.774	6.316	3.460	7.002
[SD]	[6.471]	[40.07]	[6.168]	[24.15]
Coef (SE)	-1.009 (0.304)	-3.438 (1.501)	-0.679 (0.288)	-1.349 (1.340)
N	30,869	30,869	31,094	31,094
Bandwidth	15.31	15.04	28.40	23.65

Table 2 reports reduced-form estimates using local polynomial regression discontinuity estimation, separately for men and women. Our main outcome is the aggregate number of observed hospitalization events from 1981 to 2016—expressed in terms of hospitalization episodes and days. Panel A contains estimates for the full sample while Panel B reports these estimates for hospitalization experiences between the ages of 29 and 53. All regressions control for ethnicity and childhood religion. We flexibly control for birth month-year, centered around the reform cut-off date. All data come from the Scottish Longitudinal Study (SLS) and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses.

Table 3: Effects of the 1972 ROSLA Reform on Cancer Prevalence

		Men				Women			
		Any Cancer	Lung Cancer	Skin Cancer	Urin. Cancer	Any Cancer	Lung Cancer	Skin Cancer	Breast Cancer
Mean	0.080	0.007	0.026	0.019	0.141	0.007	0.024	0.039	
[SD]	[0.271]	[0.081]	[0.158]	[0.137]	[0.348]	[0.084]	[0.153]	[0.194]	
Coef	-0.046	0.003	-0.011	-0.007	-0.023	-0.006	-0.005	-0.004	
(SE)	(0.013)	(0.003)	(0.007)	(0.006)	(0.019)	(0.003)	(0.007)	(0.010)	
N	31,574	31,574	31,574	31,574	31,568	31,568	31,568	31,568	
Bandwidth	14.62	28.28	28.76	29.20	22.78	16.45	34.98	26.68	

		Men				Women			
		Any Cancer	Lung Cancer	Skin Cancer	Urin. Cancer	Any Cancer	Lung Cancer	Skin Cancer	Breast Cancer
Mean	0.060	0.001	0.025	0.017	0.120	0.002	0.024	0.033	
[SD]	[0.237]	[0.032]	[0.157]	[0.128]	[0.325]	[0.043]	[0.153]	[0.179]	
Coef	-0.043	0.001	-0.010	-0.010	-0.007	-0.002	-0.004	-0.008	
(SE)	(0.013)	(0.001)	(0.007)	(0.006)	(0.018)	(0.002)	(0.008)	(0.009)	
N	29,208	29,208	29,208	29,208	29,763	29,763	29,763	29,763	
Bandwidth	19.03	16.9079	33.05	27.32	24.90	32.33	37.30	33.97	

Table 3 reports regression discontinuity estimates using local polynomial regression discontinuity estimation, separately for men and women. Our main outcomes are dichotomous indicators for ever receiving a cancer diagnosis and for specific cancer types. Panel A contains estimates for the full sample while Panel B reports estimates for cancer prevalence between the ages of 29 and 53. All regressions control for ethnicity and childhood religion. We flexibly control for birth month-year, centered around the reform cut-off date. All data come from the Scottish Longitudinal Study (SLS) and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses.

Table 4: Education and Self-Reported Health

	Poor Health	Illness	Current Drinker	Current Smoker	Ever Smoked
All (N=66,655)					
Mean	0.271	0.457	0.894	0.297	0.601
[SD]	[0.444]	[0.498]	[0.308]	[0.457]	[0.489]
Reduced Form	0.005 (0.012)	0.009 (0.014)	0.017 (0.010)	-0.021 (0.017)	0.018 (0.017)
Bandwidth:	120.20	85.52	93.12	79.23	89.95
OLS (years of schooling)	-0.035 (0.001)	-0.020 (0.001)	0.010 (0.001)	-0.045 (0.001)	-0.033 (0.001)
OLS (>11 years)	-0.155 (0.003)	-0.082 (0.004)	0.046 (0.003)	-0.196 (0.004)	-0.145 (0.004)
Men (N=29,388)					
Mean	0.238	0.425	0.916	0.285	0.605
[SD]	[0.426]	[0.495]	[0.277]	[0.452]	[0.489]
Reduced Form	0.035 (0.026)	0.023 (0.025)	0.008 (0.016)	-0.017 (0.027)	0.022 (0.027)
Bandwidth:	74.91	90.86	75.88	83.69	76.24
OLS (years of schooling)	-0.033 (0.001)	-0.019 (0.001)	0.008 (0.001)	-0.040 (0.001)	-0.026 (0.001)
OLS (>11 years)	-0.148 (0.005)	-0.072 (0.006)	0.035 (0.004)	-0.174 (0.005)	-0.118 (0.006)
Women (N=37,262)					
Mean	0.296	0.480	0.877	0.306	0.599
[SD]	[0.457]	[0.500]	[0.328]	[0.461]	[0.490]
Reduced Form	-0.030 (0.019)	-0.024 (0.021)	0.025 (0.016)	-0.017 (0.019)	0.019 (0.023)
Bandwidth:	76.54	62.53	63.35	109.00	90.79
OLS (years of schooling)	-0.037 (0.001)	-0.021 (0.001)	0.011 (0.001)	-0.048 (0.001)	-0.039 (0.001)
OLS (>11 years)	-0.162 (0.005)	-0.090 (0.006)	0.054 (0.004)	-0.212 (0.005)	-0.168 (0.006)

Table 4 reports OLS and local polynomial regression discontinuity estimates. OLS estimates describe either the effect of obtaining an additional year of schooling or having more than 11 years of education. Reduced-form estimation is conducted using a local polynomial approach. Our main outcomes are dichotomous indicators for self-reported poor health, longstanding illness, current alcohol consumption, current smoking behavior, and whether the respondent ever smoked. All regressions control for sex (overall estimates), ethnicity and childhood religion. We flexibly control for birth month-year, centered around the reform cut-off date. All data come from pooled waves of the Scottish Health Survey (SHeS) over the 1995-2016 period. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses.

Table 5: Effects of the 1972 ROSLA Reform on Hospitalization for Substance Abuse

<i>Panel A: Full Sample</i>								
	Alcohol-Related Inpatient Admissions				Drug-Related Inpatient Admissions			
	Men		Women		Men		Women	
	Episodes	Days	Episodes	Days	Episodes	Days	Episodes	Days
Mean	0.310	1.023	0.129	0.412	0.034	0.096	0.290	0.070
[SD]	[2.013]	[9.583]	[1.041]	[4.947]	[0.467]	[2.119]	[0.366]	[1.572]
Coef (SE)	-0.227 (0.084)	-0.0575 (0.360)	-0.010 (0.039)	0.060 (0.169)	-0.014 (0.020)	-0.013 (0.065)	-0.002 (0.010)	0.051 (0.057)
N	32,164	32,164	32,103	32,103	32,164	32,164	32,103	32,103
Bandwidth	34.01	23.99	15.44	19.45	28.95	24.83	20.47	12.28

<i>Panel B: Restricted Sample (Ages 29-53)</i>								
	Alcohol-Related Inpatient Admissions				Drug-Related Inpatient Admissions			
	Men		Women		Men		Women	
	Episodes	Days	Episodes	Days	Episodes	Days	Episodes	Days
Mean	0.150	0.432	0.066	0.163	0.015	0.027	0.015	0.028
[SD]	[1.299]	[6.037]	[0.613]	[3.050]	[0.259]	[0.882]	[0.209]	[0.794]
Coef (SE)	-0.156 (0.051)	-0.418 (0.126)	-0.008 (0.018)	-0.009 (0.161)	-0.017 (0.010)	0.011 (0.029)	-0.004 (0.008)	-0.015 (0.025)
N	30,869	30,869	31,094	31,094	30,869	30,869	31,094	31,094
Bandwidth	21.31	11.50	16.15	27.41	26.38	25.47	20.50	20.84

Table 5 reports reduced-form estimates using local polynomial regression discontinuity estimation, separately for men and women. Our main outcome is the aggregate number of observed alcohol- and drug-related hospitalization events from 1981 to 2016—expressed in terms of hospitalization episodes and days. Panel A contains estimates for the full sample while Panel B reports these estimates for hospitalization experiences between the ages of 29 and 53. All regressions control for ethnicity and childhood religion. We flexibly control for birth month-year, centered around the reform cut-off date. All data come from the Scottish Longitudinal Study (SLS) and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses.

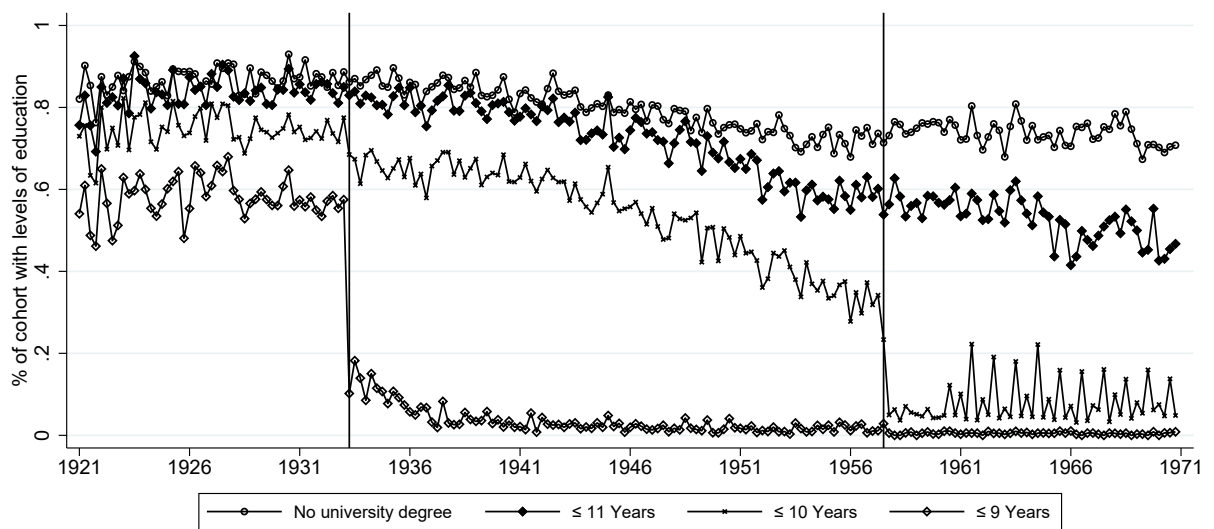


Table 6: Effects of the 1972 ROSLA Reform on Socioeconomic Outcomes

<i>Panel A: Men</i>					
	Most Deprived	Below Median	Least Deprived	Skilled Occ.	HH-Income (£)
Mean	0.095	0.489	0.108	0.755	49,800
[SD]	[0.294]	[0.500]	[0.310]	[0.430]	[45,264]
Coef (SE)	0.002 (0.012)	-0.005 (0.023)	-0.013 (0.013)	0.007 (0.020)	3,460 (3,657)
N	31,745	31,745	31,745	31,776	6,572
Bandwidth	28.73	18.54	21.76	16.67	30.84
Datasource	SLS	SLS	SLS	SLS	SHeS
<i>Panel B: Women</i>					
	Most Deprived	Below Median	Least Deprived	Skilled Occ.	HH-Income (£)
Mean	0.099	0.488	0.111	0.728	47,126
[SD]	[0.299]	[0.500]	[0.314]	[0.445]	[45,039]
Coef (SE)	-0.007 (0.008)	0.042 (0.012)	-0.010 (0.010)	0.021 (0.020)	-2,797 (4,286)
N	31,711	31,711	31,711	31,720	7,941
Bandwidth	17.25	22.49	28.82	21.75	22.98
Datasource	SLS	SLS	SLS	SLS	SHeS

Table 6 reports reduced-form estimates using local polynomial regression discontinuity estimation, separately for men (Panel A) and women (Panel B). Our main outcomes are indicators for neighborhood deprivation based on the Scottish Index of Multiple Deprivations (SMID), employment in a skilled occupation (i.e., professional, managerial, technical, or “other skilled”), and a measure of household income in 2015 £s. All regressions control for ethnicity and childhood religion. We flexibly control for birth month-year, centered around the reform cut-off date. All data for the first four columns come from the Scottish Longitudinal Study (SLS) and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date. The last column is based on data from the Scottish Health Survey (SHeS). Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses.

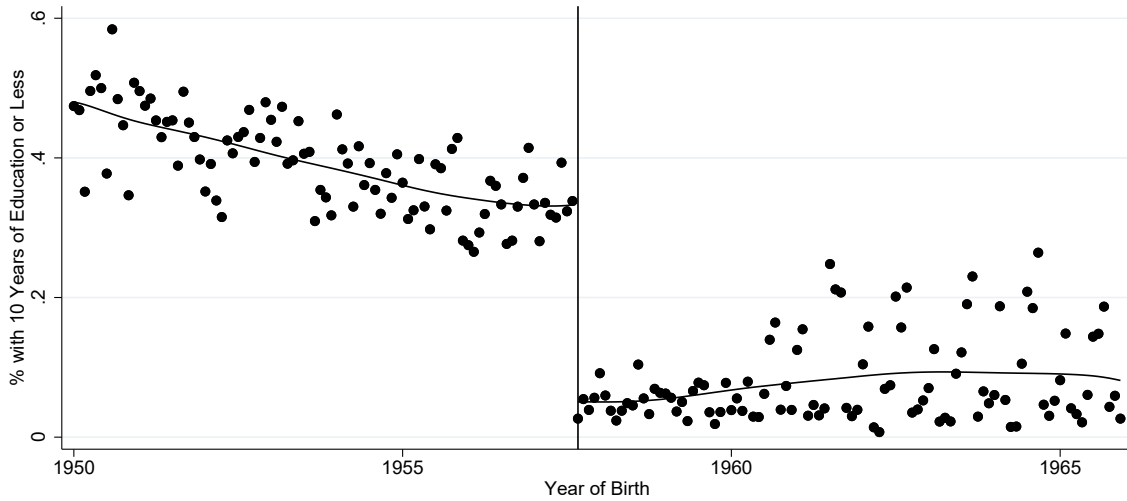
Figure 1: Years of Full-Time Education by Quarter of Birth



*Notes:* Figure 1 provides descriptive evidence of the first-stage relationship between the two twentieth century U.K. compulsory schooling reforms and educational attainment. Each dot is a quarter of birth cohort. Vertical lines are used to denote each ROSLA reform. The 1947 reform is given by the first vertical line and increased the minimum schooling leaving age from 14 to 15 for all cohorts born on or after the second quarter of 1933. The 1972 reform increased the minimum school leaving age from 15 to 16 for all cohorts born in or after September 1957. The figure is based on data from the 1995-2016 waves of the Scottish Health Survey (SHeS).

Figure 2: Effects of the 1972 ROSLA Reform on Educational Attainment

(a)  $\leq 10$  Years of Education



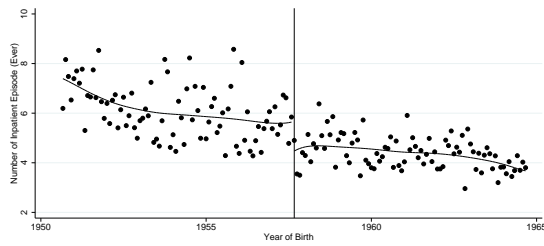
(b) Years of Education



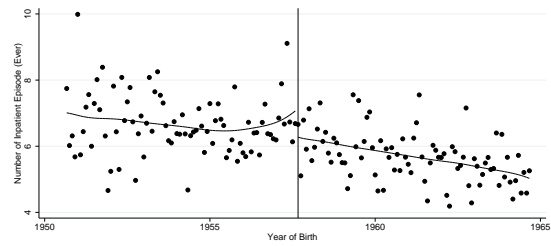
Notes: Figure 2 describes the effects of the 1972 ROSLA reform on educational attainment. Each dot is the average outcome for each month-year birth cohort. Horizontal loess lines provide a flexible fit with the vertical line denoting the 1972 ROSLA reform. Panel A describes the effect of the 1972 reform on the proportion of students with no more 10 years of education. Panel B describes the effect of the same reform on years of education. The figure is based on pooled data from the 1995-2016 waves of the Scottish Health Survey (SHeS).

Figure 3: Effects of the 1972 ROSLA Reform on Hospitalization

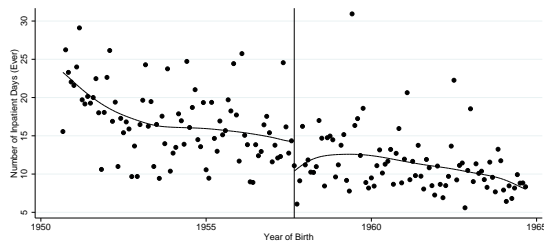
(a) Men: Inpatient Episodes



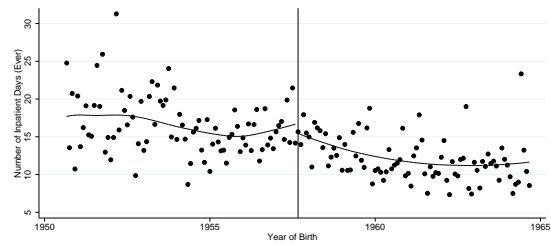
(b) Women: Inpatient Episodes



(c) Men: Inpatient Days



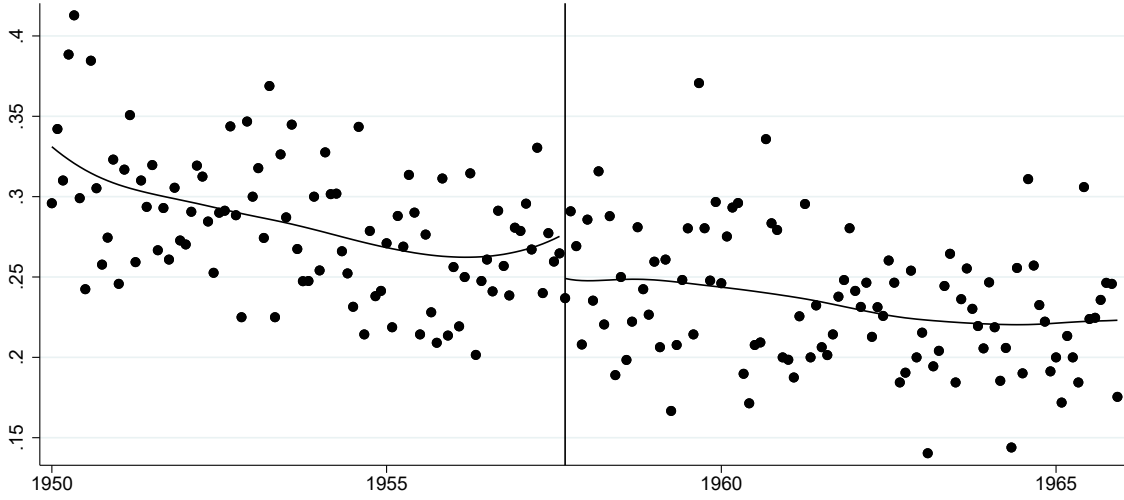
(d) Women: Inpatient Days



*Notes:* Figure 3 describes the reduced-form effects of the 1972 ROSLA reform on hospitalization. Each dot describes the average number of hospitalization events in adulthood for each month-year birth cohort. Horizontal lowess lines provide a flexible fit with the vertical line denoting the 1972 ROSLA reform. Panels (a) and (c) describe the effects of the 1972 reform on inpatient episodes and days for men, respectively. Panels (b) and (d) provide the corresponding figures for women. The figure is based on data from the Scottish Longitudinal Study (SLS) over the 1981-2016 period.

Figure 4: Effects of the 1972 ROSLA Reform on Self-Reported Health

(a) 1972 Reform: Poor Health



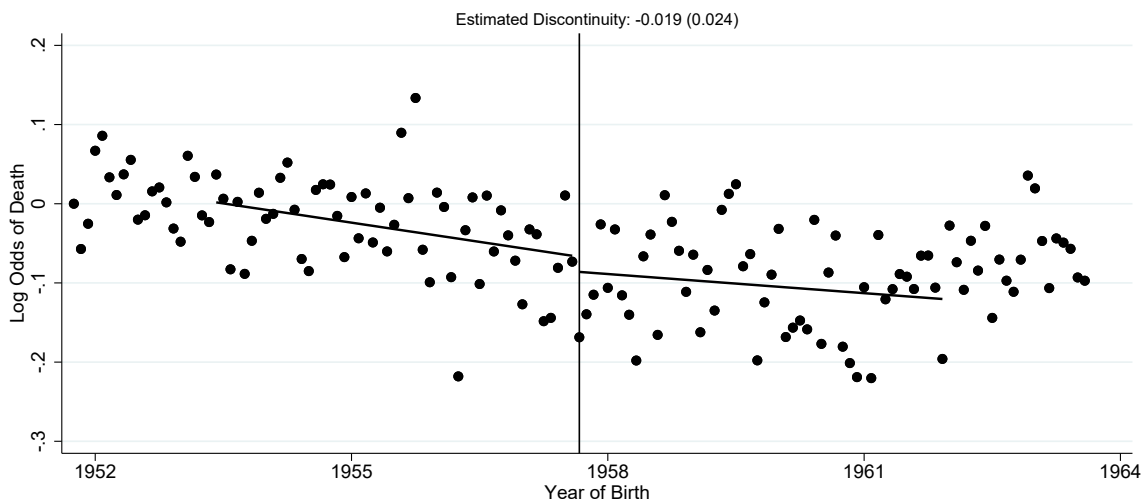
(b) 1972 Reform: Long-Standing Illness



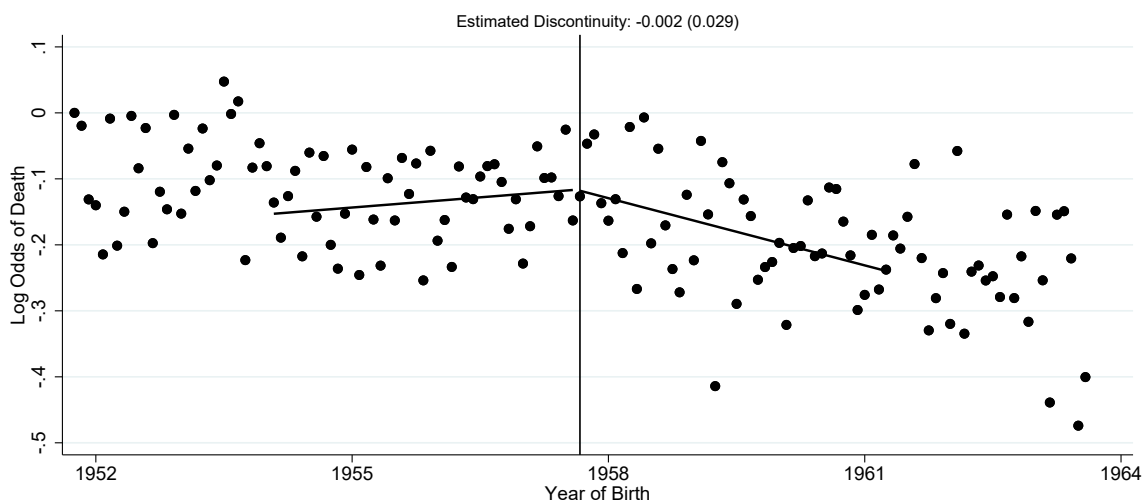
*Notes:* Figure 4 describes the reduced-form effects of the 1972 ROSLA reform on self-reported health. Each dot is the average outcome for each month-year birth cohort. Horizontal lowess lines provide a flexible fit with the vertical line denoting the 1972 ROSLA reform. Panel A describes the effect of the 1972 reform on the proportion of survey respondents reporting being in “fair,” “bad,” or “very bad” health. Panel B describes the effect of the same reform on the proportion of survey respondents who reported having a long-standing illness. The figure is based on pooled data from the 1995-2016 waves of the Scottish Health Survey (SHeS).

Figure 5: Effect of the 1972 ROSLA Reform on Mortality

(a) Men

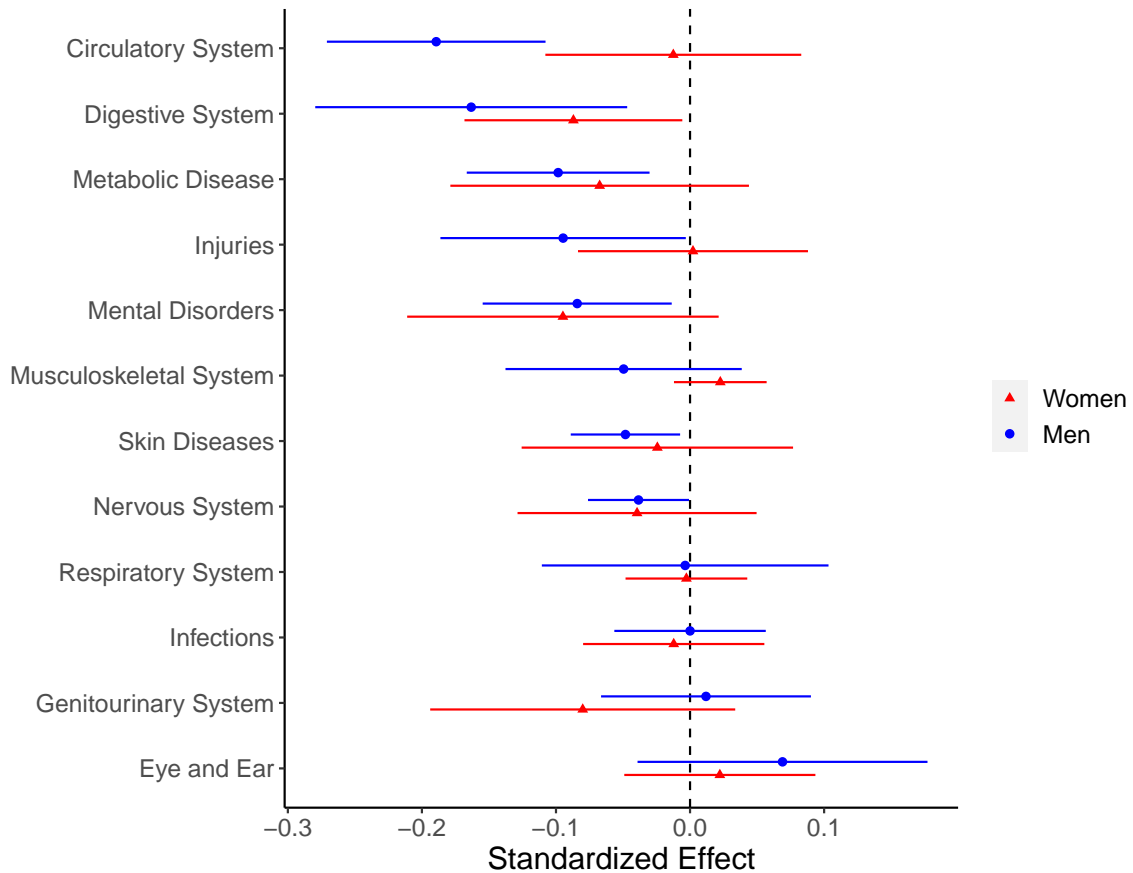


(b) Women



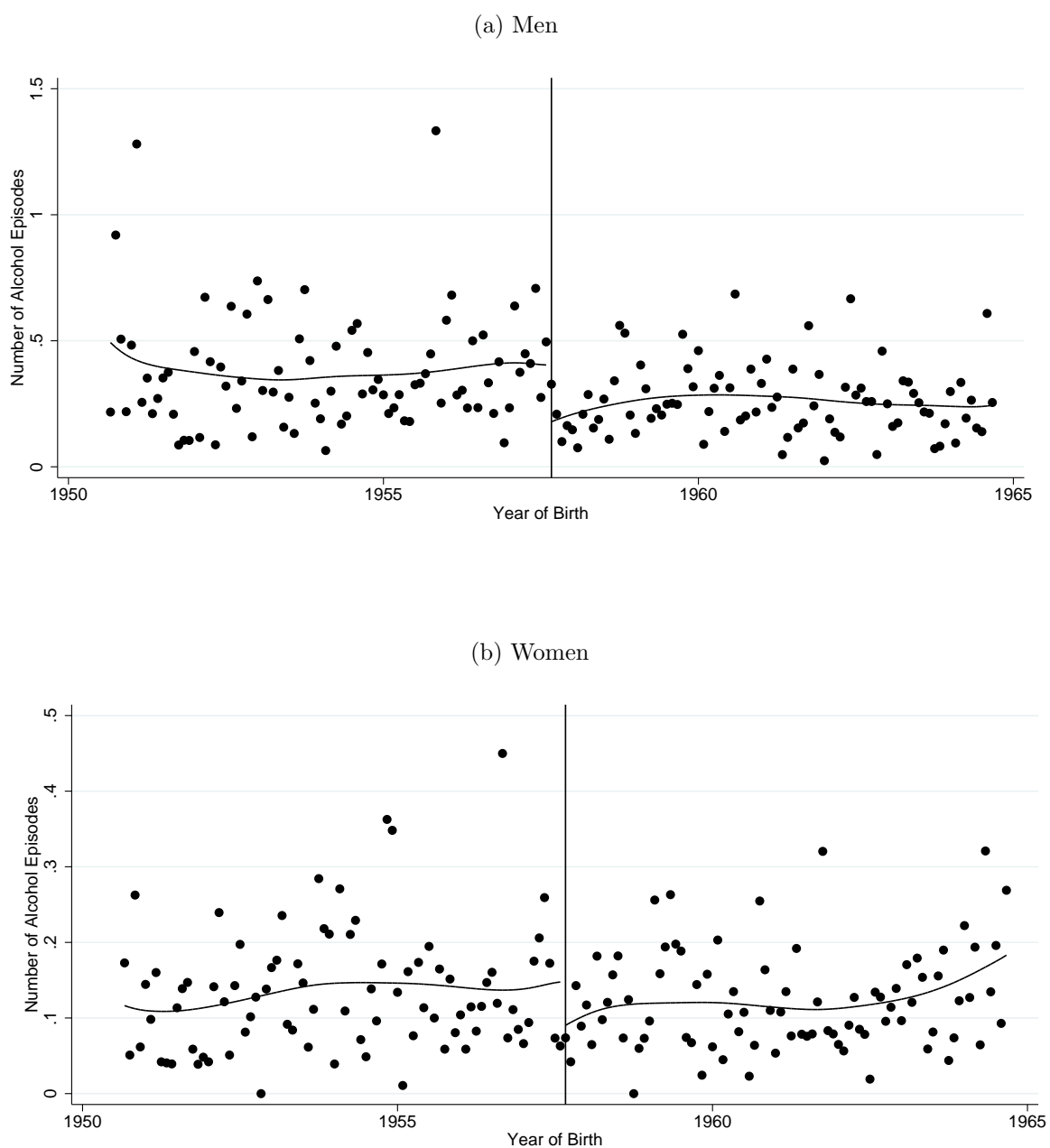
*Notes:* Figure 5 describes the relationship between the 1972 ROSLA reform and mortality. Each dot describes the log odds death ratio for each month-year birth cohort. Estimation is given by the two-step procedure outlined in equations (5)-(6) in Section 4.4. The first step involves a panel logit regression of mortality on birth month-year fixed effects. Fitted values for these fixed effects then serve as the outcome variable in a local linear regression that we use to estimate the discontinuity at the reform cutoff. Panels (a) and (b) show results based on this procedure for men and women, respectively. All estimates should be interpreted relative to the September 1950 birth cohort. The vertical line denotes the 1972 ROSLA reform. This figure is based on data from the Scottish Census and Death Registry.

Figure 6: Effects of the 1972 ROSLA Reform on Hospitalization by Diagnosis



*Notes:* Figure 6 shows a series of reduced-form estimates using local polynomial regression discontinuity estimations, eparately for men (blue circles) and women (red triangles). Our main outcome is the aggregate number of observed inpatient episodes by primary diagnosis from 1981 to 2016. Effects are rescaled to reflect changes in standard deviations. Horizontal lines are the associated 95% confidence intervals. All regressions control for ethnicity and childhood religion. We flexibly control for birth month-year, centered around the reform cut-off date. All data come from the Scottish Longitudinal Study (SLS) and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses and clustered by birth month-year.

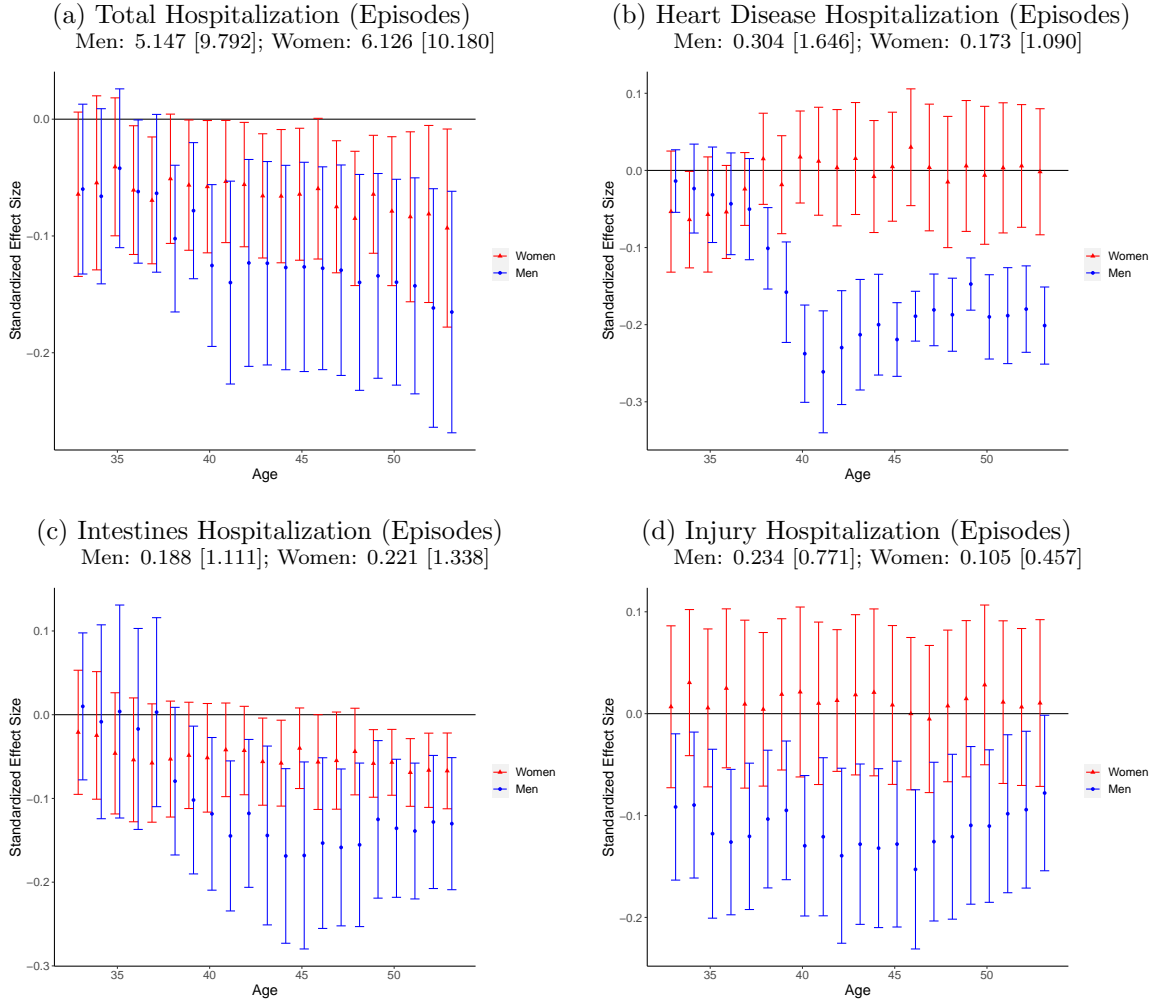
Figure 7: Effects of the 1972 ROSLA Reform on Alcohol-Related Hospitalization



*Notes:* Figure 7 describes the reduced-form effects of the 1972 ROSLA reform on alcohol-related hospitalization. Each dot describes the average number of alcohol-related inpatient episodes in adulthood for each month-year birth cohort. Alcohol-related admissions include episodes characterized as alcohol poisoning, intoxication, harmful use, or dependency/withdrawal. Horizontal lowess lines provide a flexible fit with the vertical line denoting the 1972 ROSLA reform. Panels (a) and (b) describe the effects of the 1972 reform on alcohol-related inpatient episodes for men and women, respectively. The figure is based on data from the Scottish Longitudinal Study (SLS) over the 1981-2016 period.



Figure 8: Life Cycle Effects of the 1972 ROSLA Reform on Hospitalization



*Notes:* Figure 8 shows cumulative reduced-form estimates by age  $a(t)$  using local polynomial regression discontinuity estimation, separately for men (blue circles) and women (red triangles). Our main outcome is the cumulative number of observed inpatient episodes by a particular age from 1981 to 2016 for total hospitalization and select conditions. Each panel also contains the gender-specific mean and standard deviation (in brackets) for each specified outcome over the full life cycle. Effects are re-scaled to reflect changes in standard deviations. Horizontal lines are the associated 95% confidence intervals. All regressions control for ethnicity and childhood religion. We flexibly control for birth month-year, centered around the reform cut-off date. All data come from the Scottish Longitudinal Study (SLS) and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses and clustered by birth month-year.

# The Effects of Compulsory Schooling on Health and Hospitalization over the Life Cycle

## Online Appendix

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May 26, 2023

### **Abstract**

This online appendix provides supplementary results and documentation supporting our main findings on the causal effects of the 1972 U.K. ROSLA reform on health and health care utilization in adulthood. Appendix A contains several supplementary figures and tables based on the 1972 reform at the heart of our analyses. Appendix B shows complementary findings for cohorts affected by the 1947 U.K. ROSLA reform. As noted throughout the paper, we report remarkably similar first-stage estimates describing the effect of the 1947 reform on educational attainment. However, we find weaker evidence of the reform's effects on health and health care utilization—consistent with both theory and more recent findings on the unique circumstances surrounding this reform. Finally, Appendix C provides additional documentation for the ICD-10 codes and classifications used for all of our hospitalization analyses.

# Appendix A: Supplemental Tables and Figures

Table A1: SLS Sample Means

	Men		Women	
	Sep. 1950 - August 1957	Sep. 1957 - August 1964	Sep. 1950 - August 1957	Sep. 1957 - August 1964
<b>Pre-Defined Characteristics</b>				
% White	0.998	0.997	0.998	0.998
% Non-White	0.002	0.003	0.002	0.002
% Catholic (Raised)	0.139	0.149	0.16	0.173
% Church of Scotland (Raised)	0.447	0.386	0.475	0.427
<b>Education Outcomes</b>				
% No Qualification	0.371	0.296	0.377	0.269
% At Least O-Grades	0.629	0.704	0.623	0.731
% Degree	0.245	0.239	0.256	0.255
<b>Post-Defined Characteristics</b>				
% Unskilled	0.193	0.240	0.259	0.242
% Skilled	0.447	0.474	0.418	0.457
% Professional	0.36	0.287	0.324	0.301
% Ever Married	0.843	0.665	0.894	0.759
Deprivation Index	-0.486	-0.155	-0.332	0.024
N	14,641	17,135	14,774	16,946

*Notes:* Table A1 is based on data from the Scottish Longitudinal Study (SLS) and provides means for all observable demographic characteristics among SLS participants born within seven years of the 1972 U.K. ROSLA reform.

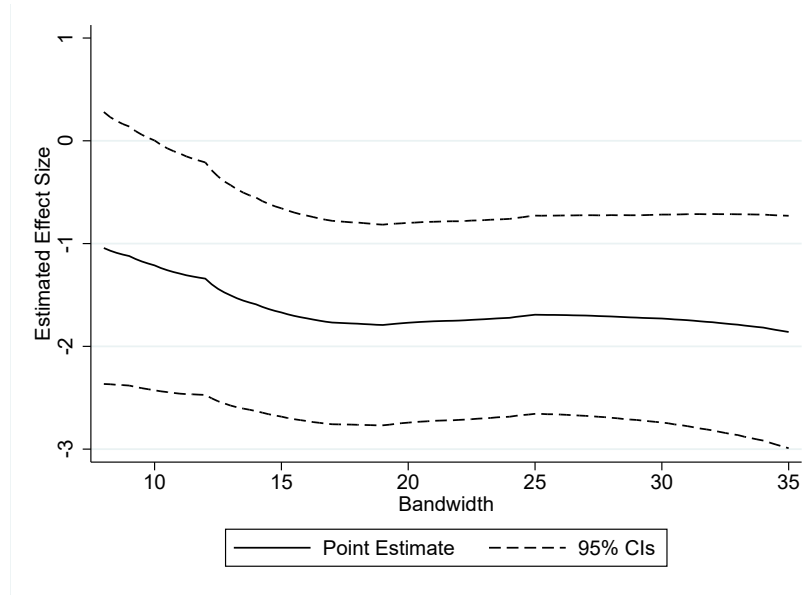
Table A2: Second-Stage Results: Education and Self-Reported Health

	Poor Health	Illness	Current Drinker	Current Smoker	Ever Smoked
	All (N=66,655)				
Mean	0.271	0.457	0.894	0.297	0.601
[SD]	[0.444]	[0.498]	[0.308]	[0.457]	[0.489]
IV (2nd Stage)	-0.018 (0.056)	0.047 (0.083)	0.057 (0.051)	-0.044 (0.072)	0.065 (0.072)
Bandwidth:	68.1	103.4	101.0	105.1	85.6

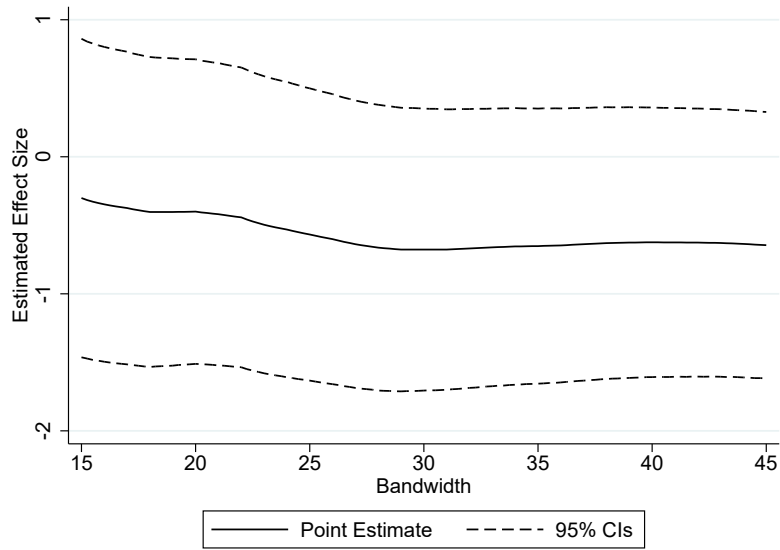
This table reports the 2nd stage estimates of an instrumental variable ("fuzzy RDD") estimation. Main outcomes are dichotomous indicators for self-reported poor health, longstanding illness, current alcohol consumption, current smoking behavior, and whether the respondent ever smoked. The endogenous (instrumented) variable is a continuous measure for years of education. All coefficients are obtained using a non-parametric regression discontinuity estimation with automated bandwidth selection. The running variable is birth month-year centered around the September 1972 cutoff. All data come from pooled waves of the Scottish Health Survey (SHES) over the 1995-2016 period. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses.

Figure A1: Main Effects Estimation by Bandwidth

(a) Men: Inpatient Episodes



(b) Women: Inpatient Episodes



Notes: Figure A1 shows the coefficients and 95% confidence intervals for our main analysis of inpatient hospitalization episodes. In Table 2, we reported reduced-form estimates using local polynomial regression discontinuity estimation with the optimal, MSE-minimizing bandwidth. In this Figure, we show the same reduced-form estimates (y-axis) alongside confidence intervals for a variety of bandwidths (x-axis)

Table A3: Effects of the 1972 ROSLA Reform on Emigration

	Men	Women
Mean	0.048	0.039
[SD]	[0.213]	[0.193]
Coef	0.009	-0.008
(SE)	(0.008)	(0.009)
N	35,228	34,831
Bandwidth	22.63	14.36

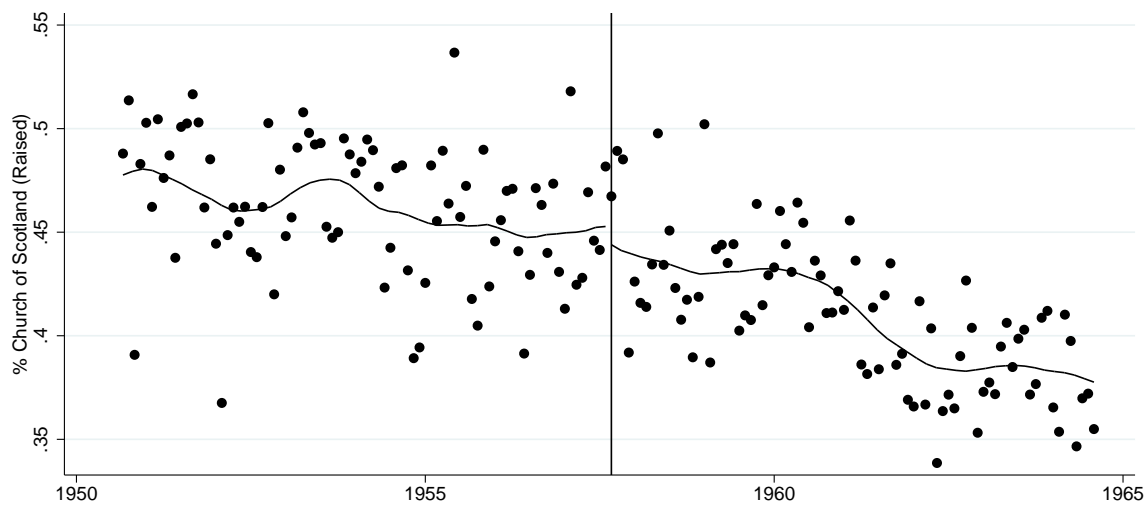
Table A3 reports reduced-form estimates using local polynomial regression discontinuity estimation. Our main outcome is observed emigration. All regressions control for ethnicity and childhood religion. We flexibly control for birth month-year, centered around the reform cut-off date. All data come from the Scottish Longitudinal Study (SLS) and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses.

Table A4: Placebo Regressions: 1958 to 1972 Birth Cohorts

	No Qual.	Inpatient Epis. (all)	Inpatient Epis. (Digest. Syst.)	Alcohol- related Epis.
<i>Panel A: Men</i>				
Mean	0.287	3.876	0.750	0.244
[SD]	[0.452]	[7.438]	[2.440]	[1.756]
Coef (SE)	-0.019 (0.016)	-0.022 (0.284)	-0.066 (0.082)	0.038 (0.068)
N	33,451	33,794	33,794	33,794
Bandwidth	26.66	25.60	26.88	26.34
<i>Panel B: Women</i>				
Mean	0.244	5.080	0.841	0.112
[SD]	[0.430]	[8.716]	[2.541]	[1.000]
Coef (SE)	-0.008 (0.028)	-0.527 (0.363)	0.003 (0.085)	-0.007 (0.044)
N	33,564	33,802	33,802	33,802
Bandwidth	15.58	18.98	27.50	20.15

Table A4 reports placebo estimates using local polynomial regression discontinuity estimation for our main outcomes of interest. More specifically, this table calculates reduced-form effects for a hypothetical ROSLA reform affecting everyone born after September 1965. Panels (A) and (B) contain separate estimates for men and women (respectively) for respondents born between August 1958 and September 1972. All regressions control for ethnicity and childhood religion. We flexibly control for birth month-year, centered around the reform cut-off date. All data come from the Scottish Longitudinal Study (SLS). Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses.

Figure A2: Balanced Covariates: Fraction Church of Scotland



*Notes:* Figure A2 provides evidence of covariate balance for the 1972 ROSLA reform. Each dot describes the proportion raised Protestant in childhood for each month-year birth cohort. Horizontal loess lines provide a flexible fit with the vertical line denoting the 1972 ROSLA reform. The figure is based on data from the Scottish Longitudinal Study (SLS) and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date.

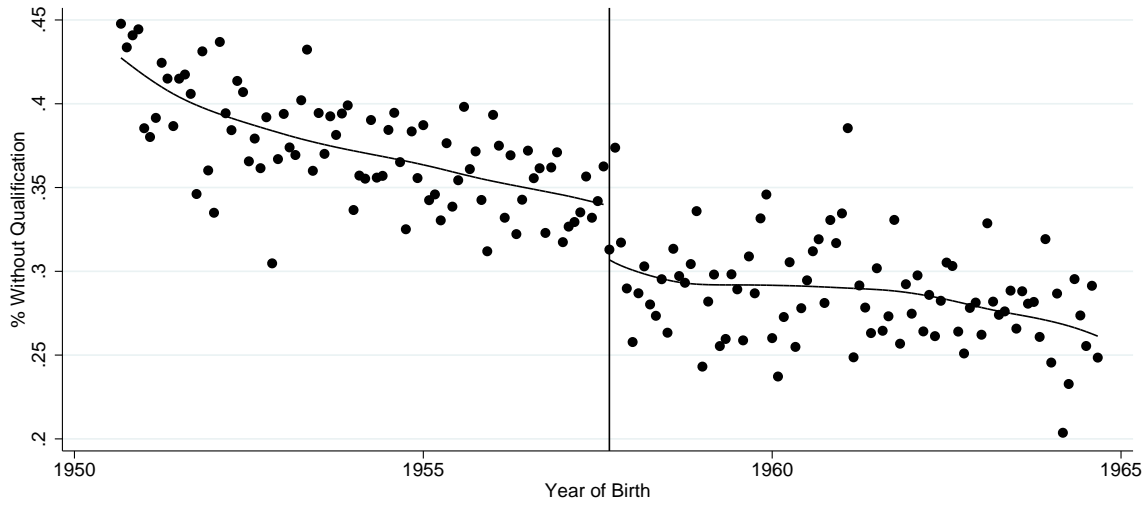


Figure A3: Comparison of Educational Measures in the SHeS and SLS Data

(a) SHeS: % No Qualification



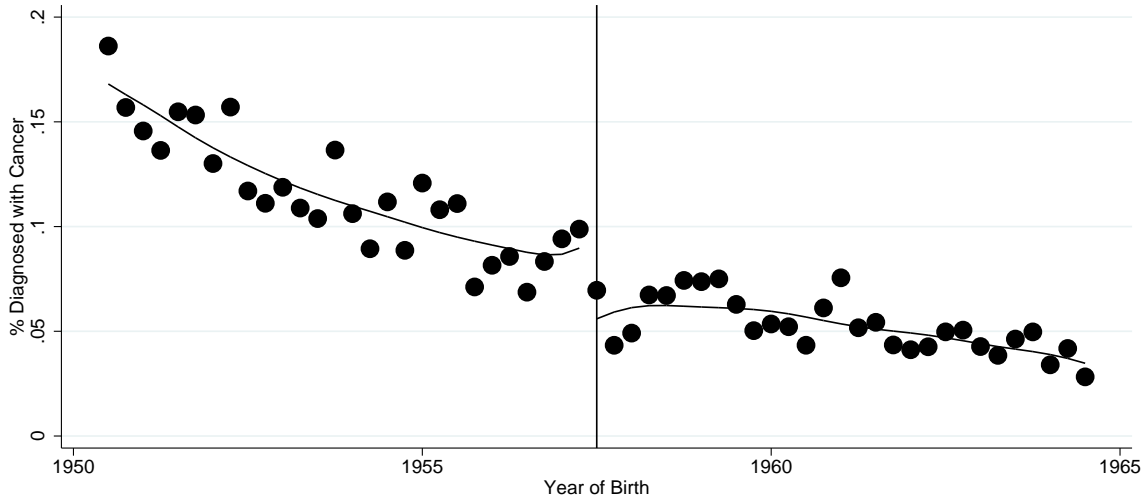
(b) SLS: % No Qualification



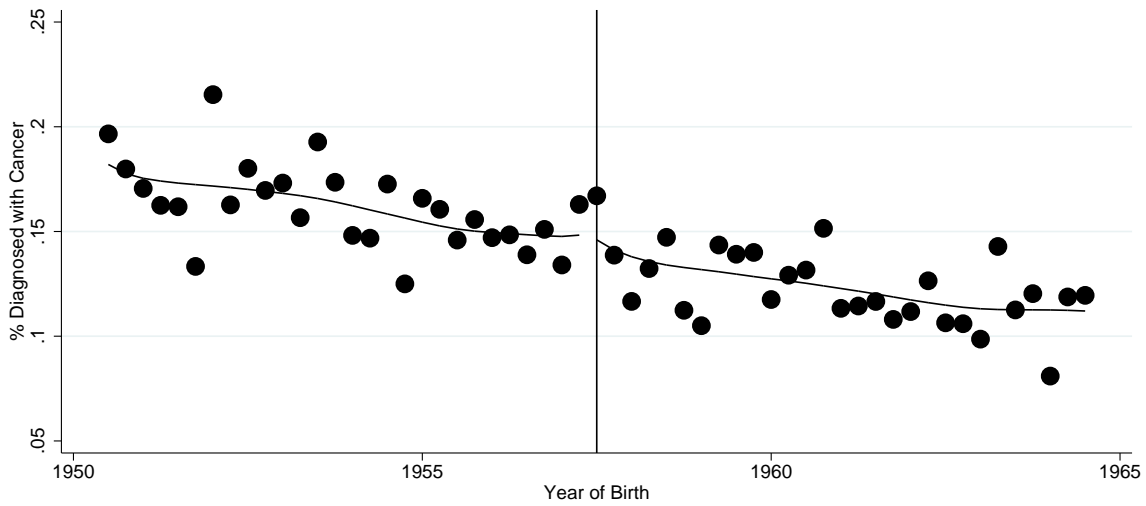
*Notes:* Figure A3 compares educational measures across the two main data sources used within this paper. Each dot describes the proportion without formal qualification ( $\approx$  high school dropouts) for each month-year birth cohort. Horizontal lowest lines provide a flexible fit with the vertical line denoting the 1972 ROSLA reform. Panel (a) shows results using information from pooled waves of the Scottish Health Survey (SHeS) over the 1995-2016 period. Panel (b) shows similar results using the most recent information for each respondent within the Scottish Longitudinal Study (SLS).

Figure A4: Effects of the 1972 ROSLA Reform on Cancer Prevalence

(a) Men: Any Cancer Diagnosis



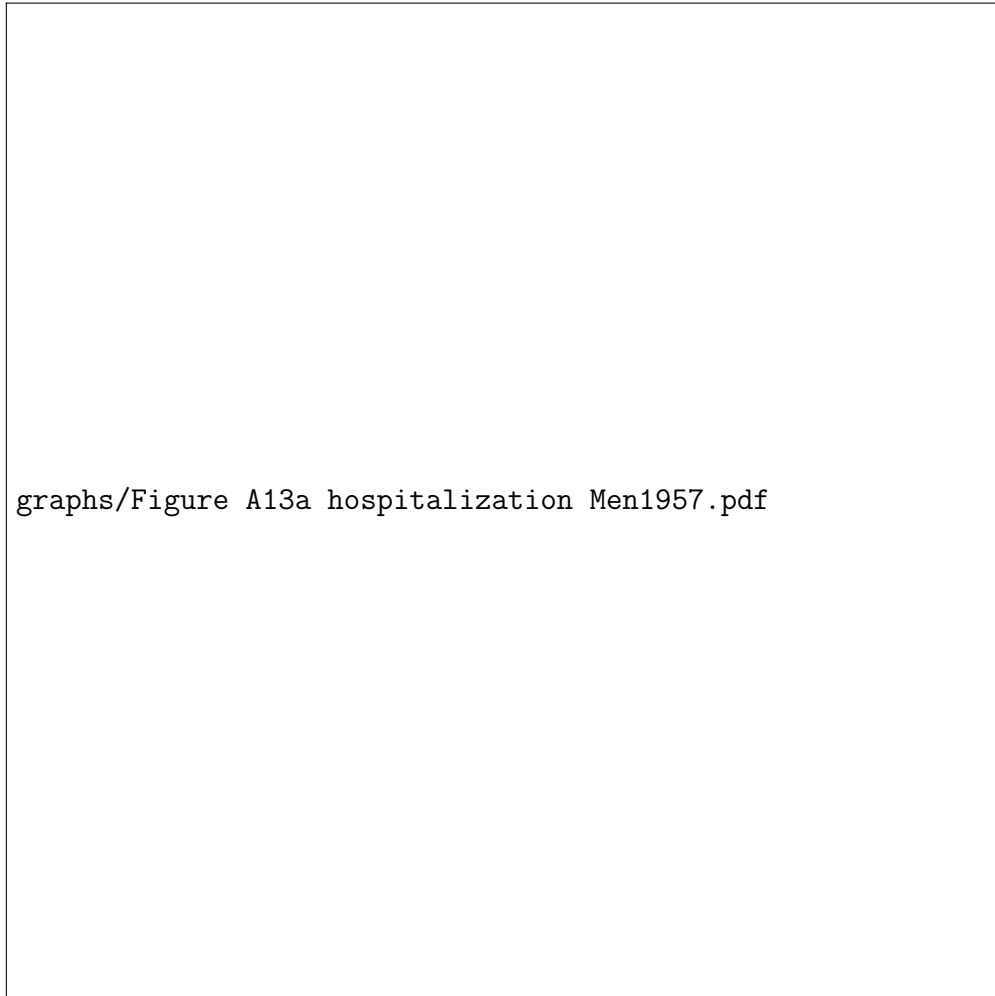
(b) Women: Any Cancer Diagnosis



*Notes:* Figure A4 describes the reduced-form effects of the 1972 ROSLA reform on cancer diagnoses. Each dot describes the proportion diagnosed with any cancer in adulthood for each month-year birth cohort. Since cancer diagnoses are rare within these groups, we instead show quarterly aggregates for later cohorts for confidentiality reasons. Horizontal lowess lines provide a flexible fit with the vertical line denoting the 1972 ROSLA reform. Panels (a) and (b) describe the effects of the 1972 reform on alcohol-related inpatient episodes for men and women, respectively. The figure is based on data from the Scottish Longitudinal Study (SLS) over the 1981-2016 period.

Figure A5: Main Effects Estimation Using Two-Step Approach

(a) Hospitalization Episodes - Men



(b) Hospitalization Episodes - Women

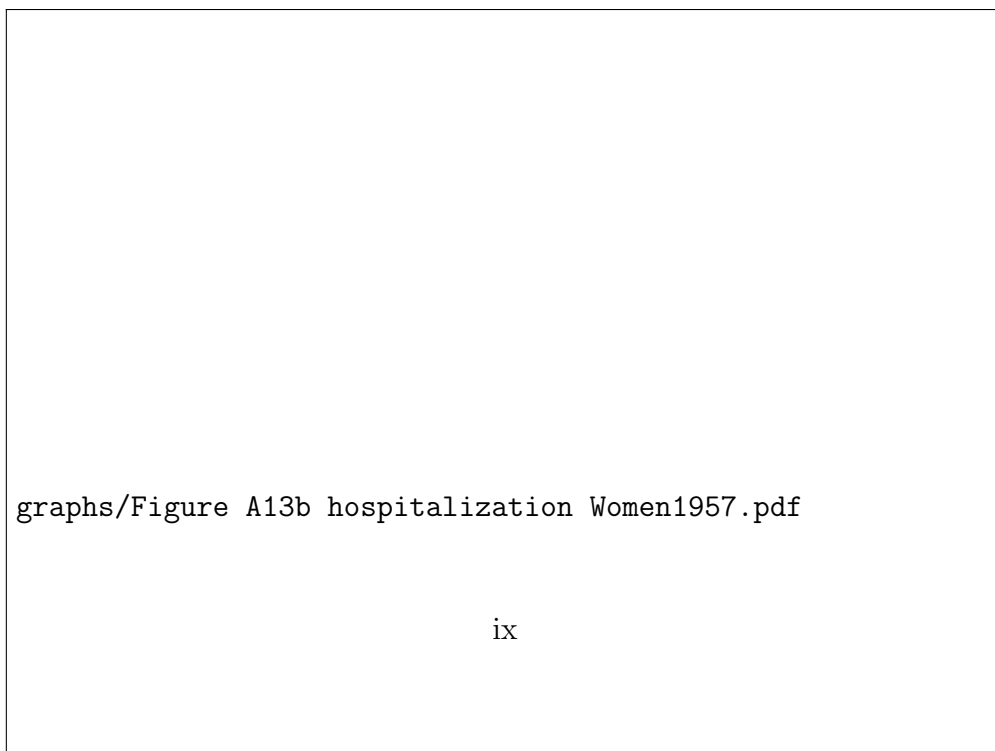
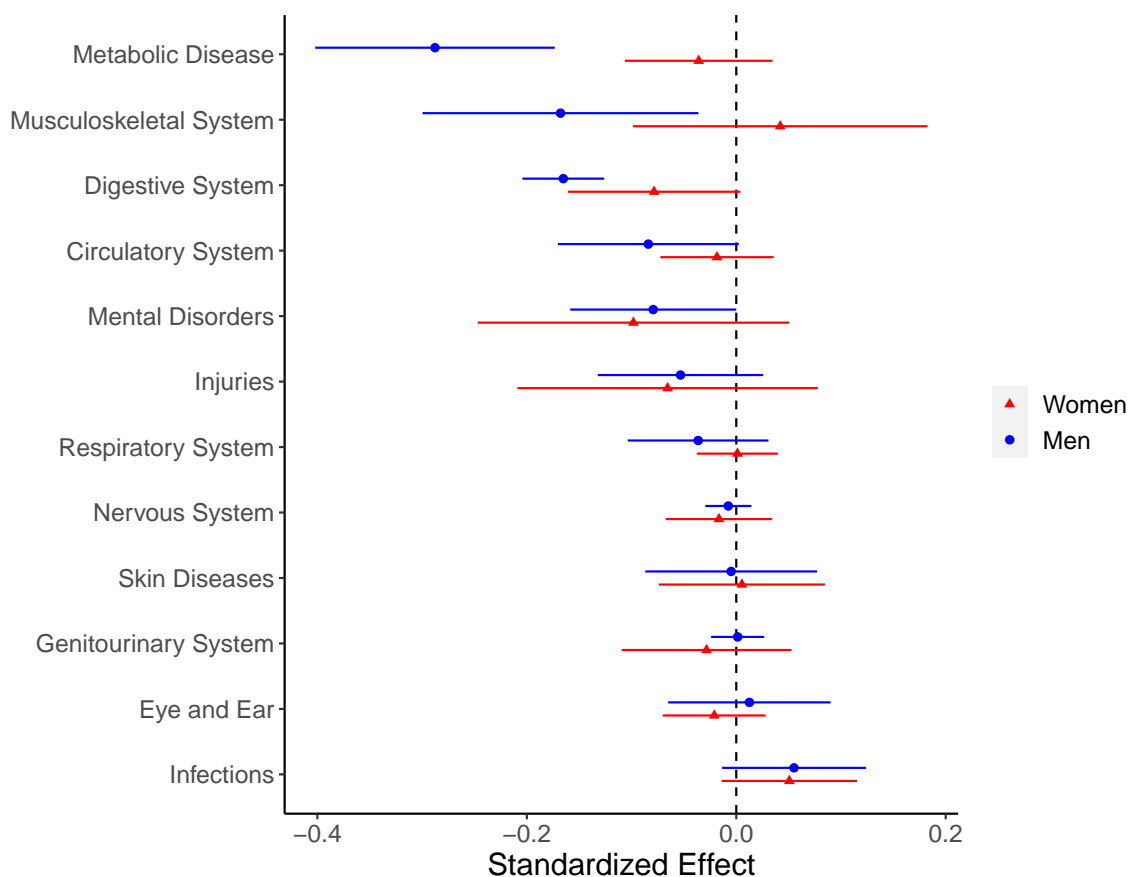
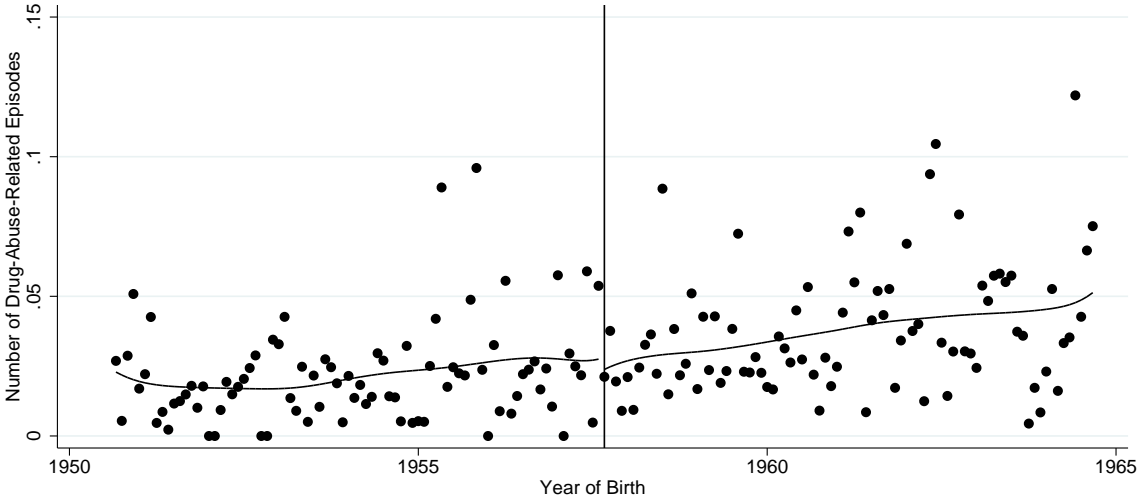


Figure A6: Effects of the 1972 ROSLA Reform on Hospitalization (Days) by Diagnosis



*Notes:* Figure A6 shows a series of reduced-form estimates using local polynomial regression discontinuity estimation, separately for men (blue circles) and women (red triangles). Our main outcome is the aggregate number of observed inpatient days by primary diagnosis from 1981 to 2016. Effects are re-scaled to reflect changes in standard deviations. Horizontal lines are the associated 95% confidence intervals. All regressions control for ethnicity, childhood religion, and are centered around the reform cutoff date. We also flexibly control for birth month-year. All data come from the Scottish Longitudinal Study (SLS) and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses and clustered by birth month-year.

Figure A7: Effects of the 1972 ROSLA Reform on Drug Abuse-Related Hospitalization



Notes: Figure A7 describes the reduced-form effects of the 1972 ROSLA reform on drug abuse-related hospitalization. Each dot describes average drug-related inpatient episodes in adulthood for each month-year birth cohort. Horizontal lowess lines provide a flexible fit with the vertical line denoting the 1972 ROSLA reform. The figure is based on data from the Scottish Longitudinal Study (SLS) over the 1981-2016 period and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date.

## Appendix B: 1947 ROSLA Reform Results

Table B1: SLS Sample Means: 1947 ROSLA Reform

<i>Panel A: Sample for 1947 ROSLA</i>	Men		Women	
	April 1926 - March 1933	April 1933 - March 1940	April 1926 - March 1933	April 1933 - March 1940
<b>Pre-Defined Characteristics</b>				
% White	0.998	0.997	0.996	0.997
% Non-White	0.002	0.003	0.004	0.003
% Catholic (Raised)	0.089	0.115	0.104	0.131
% Church of Scotland (Raised)	0.450	0.496	0.469	0.502
<b>Education Outcomes</b>				
% No Qualification	0.746	0.663	0.764	0.677
% At Least O-Grades	0.254	0.337	0.236	0.323
% Degree	0.115	0.140	0.104	0.128
<b>Post-Defined Characteristics</b>				
% Unskilled	0.273	0.243	0.350	0.338
% Skilled	0.439	0.443	0.398	0.395
% Professional	0.288	0.315	0.252	0.267
% Ever Married	0.914	0.912	0.912	0.937
Deprivation Index	-0.056	-0.146	-0.027	-0.150
N	9,910	11,240	11,408	11,801

*Notes:* Table B1 is based on data from the Scottish Longitudinal Study (SLS) and provides means for all observable demographic characteristics among SLS participants born within seven years of the 1947 U.K. ROSLA reform.

Table B2: Effects of the 1947 ROSLA Reform on Education

	Years of Education	≤9 years	≤10 years	≤11 years	≤12 years	≤13 years
All (N=37,078; bandwidth = 57 months)						
Estimate	0.530 (0.104)	-0.413 (0.024)	-0.073 (0.025)	-0.017 (0.019)	0.013 (0.017)	-0.007 (0.015)
Outcome Mean	10.17	0.571	0.749	0.838	0.891	0.925
Men (N=16,500; bandwidth = 49 months)						
Estimate	0.369 (0.164)	-0.407 (0.038)	-0.011 (0.034)	0.031 (0.034)	0.020 (0.026)	0.004 (0.024)
Outcome Mean	10.34	0.555	0.712	0.806	0.866	0.908
Women (N=20,578; bandwidth = 57 months)						
Estimate	0.653 (0.133)	-0.412 (0.030)	-0.112 (0.034)	-0.042 (0.027)	-0.036 (0.022)	-0.015 (0.018)
Outcome Mean	10.05	0.584	0.777	0.863	0.910	0.938

Table B2 reports estimates based on the first-stage equation using local polynomial regression discontinuity estimation. Estimates are provided both using years of education and for five distinct levels of educational attainment. All regressions control for sex (overall estimates), ethnicity, childhood religion, and are centered around the reform cutoff date. We also flexibly control for birth month-year. All data come from pooled waves of the Scottish Health Survey (SHeS) over the 1995-2016 period. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses.

Table B3: Effects of the 1947 ROSLA Reform on Inpatient Hospitalization

<i>Panel A: Full Sample</i>				
	Men		Women	
	Episodes	Days	Episodes	Days
Mean	11.260	57.324	10.540	58.717
[SD]	[13.110]	[111.737]	[11.998]	[111.845]
Coef (SE)	-1.700 (0.908)	-7.020 (7.349)	0.234 (0.702)	4.205 (5.346)
N	21,311	21,311	23,407	23,407
Bandwidth	21.29	23.94	29.66	24.50
<i>Panel B: Restricted Sample (Ages 53-77)</i>				
	Men		Women	
	Episodes	Days	Episodes	Days
Mean	6.305	21.426	5.305	20.601
[SD]	[8.885]	[56.78]	[7.521]	[53.214]
Coef (SE)	-1.104 (0.768)	-3.096 (3.899)	0.040 (0.513)	-1.853 (3.771)
N	12,813	12,813	16,796	16,796
Bandwidth	20.30	26.98	30.83	23.15

Table B3 reports reduced-form estimates using local polynomial regression discontinuity estimation, separately for men and women. Our main outcome is the aggregate number of observed hospitalization events from 1981 to 2016—expressed in terms of hospitalization episodes and days. Panel A contains estimates for the full sample while Panel B reports these estimates for hospitalization experiences between the ages of 53 and 77. All regressions control for ethnicity, childhood religion, and are centered around the reform cutoff date. We also flexibly control for birth month-year. All data come from the Scottish Longitudinal Study (SLS) and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses.



Table B4: Effects of the 1947 ROSLA Reform on Cancer Prevalence

		Men				Women			
		Any Cancer	Lung Cancer	Skin Cancer	Urin. Cancer	Any Cancer	Lung Cancer	Skin Cancer	Breast Cancer
Mean	0.395	0.067	0.123	0.107	0.333	0.049	0.097	0.069	
[SD]	[0.489]	[0.250]	[0.328]	[0.310]	[0.253]	[0.216]	[0.296]	[0.176]	
Coef	-0.000	0.019	0.009	-0.047	0.010	0.013	0.007	-0.006	
(SE)	(0.029)	(0.019)	(0.019)	(0.022)	(0.027)	(0.013)	(0.018)	(0.014)	
N	19,404	19,404	19,404	19,404	21,999	21,999	21,999	21,999	
Bandwidth	36.37	23.80	35.83	24.92	36.43	26.80	29.91	24.85	

		Men				Women			
		Any Cancer	Lung Cancer	Skin Cancer	Urin. Cancer	Any Cancer	Lung Cancer	Skin Cancer	Breast Cancer
Mean	0.265	0.005	0.146	0.0862	0.221	0.005	0.106	0.0580	
[SD]	[0.441]	[0.067]	[0.354]	[0.281]	[0.415]	[0.074]	[0.308]	[0.234]	
Coef	-0.041	-0.007	-0.003	-0.068	0.016	0.005	0.014	0.007	
(SE)	(0.049)	(0.009)	(0.034)	(0.033)	(0.034)	(0.003)	(0.029)	(0.022)	
N	8,501	8,501	8,501	8,501	11,936	11,936	11,936	11,936	
Bandwidth	27.46	31.84	36.00	28.11	33.79	19.63	23.31	23.87	

Table B4 reports regression discontinuity estimates using local polynomial regression discontinuity estimation, separately for men and women. Our main outcomes are dichotomous indicators for ever receiving a cancer diagnosis and for specific cancer types. Panel A contains estimates for the full sample while Panel B reports estimates for cancer prevalence between the ages of 53 and 77. All regressions control for ethnicity, childhood religion, and are centered around the reform cutoff date. We also flexibly control for birth month-year. All data come from the Scottish Longitudinal Study (SLS) and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses.

Table B5: Education and Self-Reported Health

	Poor Health	Illness	Current Drinker	Current Smoker	Ever Smoked
All (N=37,071; bandwidth = 51 months)					
Mean	0.422	0.660	0.766	0.179	0.633
[SD]	[0.494]	[0.474]	[0.424]	[0.383]	[0.482]
Reduced Form	0.031 (0.029)	0.010 (0.026)	0.006 (0.020)	-0.005 (0.021)	-0.005 (0.027)
OLS (years of schooling)	-0.042 (0.001)	-0.018 (0.001)	0.017 (0.001)	-0.032 (0.001)	-0.021 (0.001)
OLS (>11 years)	-0.179 (0.005)	-0.087 (0.006)	0.078 (0.004)	-0.130 (0.005)	-0.096 (0.006)
Men (N=16,498; bandwidth = 56 months)					
Mean	0.427	0.660	0.840	0.165	0.741
[SD]	[0.495]	[0.474]	[0.367]	[0.372]	[0.439]
Reduced Form	0.042 (0.043)	-0.011 (0.040)	-0.014 (0.028)	-0.001 (0.028)	-0.019 (0.035)
OLS (years of schooling)	-0.040 (0.001)	-0.018 (0.002)	0.012 (0.001)	-0.030 (0.001)	-0.021 (0.002)
OLS (>11 years)	-0.178 (0.008)	-0.090 (0.008)	0.056 (0.004)	-0.125 (0.007)	-0.091 (0.008)
Women (N=20,573; bandwidth = 62 months)					
Mean	0.419	0.659	0.709	0.189	0.552
[SD]	[0.494]	[0.474]	[0.455]	[0.392]	[0.498]
Reduced Form	0.021 (0.036)	0.031 (0.034)	0.019 (0.030)	-0.005 (0.028)	0.002 (0.037)
OLS (years of schooling)	-0.043 (0.001)	-0.018 (0.002)	0.022 (0.001)	-0.034 (0.001)	-0.021 (0.002)
OLS (>11 years)	-0.180 (0.007)	-0.085 (0.007)	0.096 (0.006)	-0.134 (0.007)	-0.100 (0.008)

Table B5 reports OLS and local polynomial regression discontinuity estimates, separately for men and women. OLS describe either the effect of obtaining an additional year of schooling or having more than 11 years of education. Reduced-form estimates reflect the effect of being exposed to the 1972 ROSLA. Our main outcomes are dichotomous indicators for self-reported poor health, longstanding illness, current alcohol consumption, current smoking behavior, and whether the respondent ever smoked. All regressions control for sex (overall estimates), ethnicity, childhood religion, and are centered around the reform cutoff date. We also flexibly control for birth month-year. All data come from pooled waves of the Scottish Health Survey (SHeS) over the 1995-2016 period. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses.

Table B6: Effects of the 1947 ROSLA Reform on Hospitalization for Substance Abuse

<i>Panel A: Full Sample</i>								
	Alcohol-Related Inpatient Admissions				Drug-Related Inpatient Admissions			
	Men		Women		Men		Women	
	Episodes	Days	Episodes	Days	Episodes	Days	Episodes	Days
Mean	0.283	2.183	0.127	0.968	0.007	0.033	0.008	0.049
(SD)	(1.324)	(15.78)	(0.842)	(8.788)	(0.113)	(0.980)	(0.134)	(1.224)
Coef	0.028	-0.742	0.109	0.829	0.005	0.044	0.014	0.041
(SE)	(0.084)	(0.934)	(0.046)	(0.366)	(0.005)	(0.038)	(0.010)	(0.028)
N	21,311	21,311	23,407	23,407	21,311	21,311	23,407	23,407
Bandwidth	25.07	37.73	14.25	17.67	16.77	17.94	20.55	16.29

<i>Panel B: Restricted Sample (Ages 53-77)</i>								
	Alcohol-Related Inpatient Admissions				Drug-Related Inpatient Admissions			
	Men		Women		Men		Women	
	Episodes	Days	Episodes	Days	Episodes	Days	Episodes	Days
Mean	0.113	0.713	0.051	0.427	0.005	0.022	0.004	0.023
(SD)	(0.786)	(9.258)	(0.616)	(7.102)	(0.088)	(0.780)	(0.091)	(0.750)
Coef	-0.008	-0.030	0.017	-0.147	0.007	0.092	0.001	-0.011
(SE)	(0.046)	(0.447)	(0.030)	(0.228)	(0.004)	(0.060)	(0.005)	(0.022)
N	12,717	12,717	16,651	16,651	12,717	12,717	16,651	16,651
Bandwidth	27.30	18.18	18.18	30.77	21.90	31.38	42.59	30.05

Table B6 reports reduced-form estimates using local polynomial regression discontinuity estimation, separately for men and women. Our main outcome is the aggregate number of observed alcohol- and drug-related hospitalization events from 1981 to 2016—expressed in terms of hospitalization episodes and days. Panel A contains estimates for the full sample while Panel B reports these estimates for hospitalization experiences between the ages of 53 and 77. All regressions control for ethnicity, childhood religion, and are centered around the reform cutoff date. We also flexibly control for birth month-year. All data come from the Scottish Longitudinal Study (SLS) and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses.

Table B7: Effects of the 1947 ROSLA Reform on Emigration

	Men	Women
Mean	0.022	0.019
[SD]	[0.146]	[0.138]
Coef	0.009	0.007
(SE)	(0.005)	(0.005)
N	21,484	23,492
Bandwidth	11.18	21.38

Table B7 reports reduced-form estimates based on equation (??) using local polynomial regression discontinuity estimation, separately for men and women. Our main outcome is observed emigration. All regressions control for ethnicity, childhood religion, and are centered around the reform cutoff date. We also flexibly control for birth month-year. All data come from the Scottish Longitudinal Study (SLS) and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses.

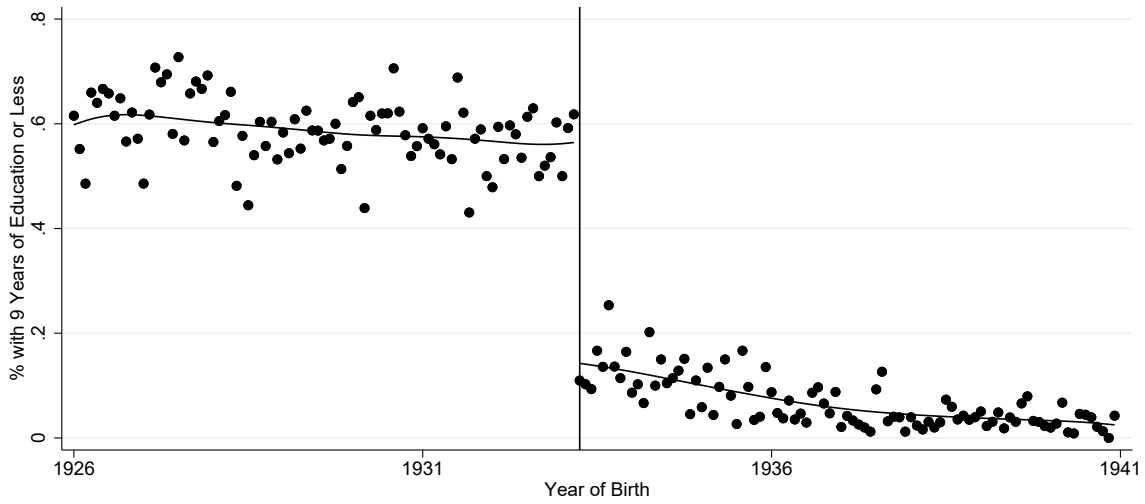
Figure B1: Balanced Covariates: Fraction Church of Scotland



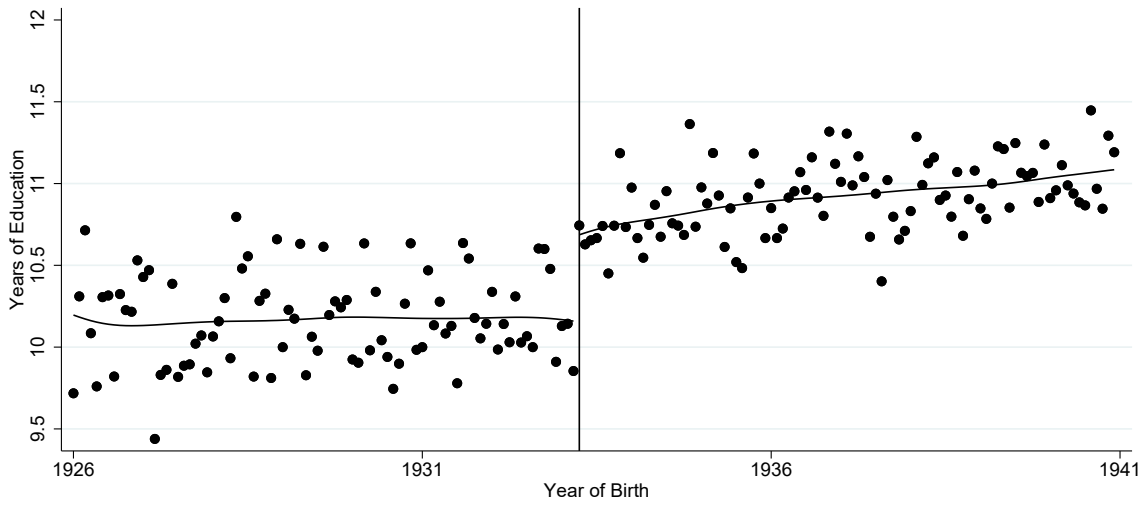
*Notes:* Figure B1 provides evidence of covariate balance for the 1947 ROSLA reform. Each dot describes the proportion raised Protestant in childhood for each month-year birth cohort. Horizontal lowess lines provide a flexible fit with the vertical line denoting the 1947 ROSLA reform. The figure is based on data from the Scottish Longitudinal Study (SLS) and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date.

Figure B2: Effects of the 1947 ROSLA Reform on Educational Attainment

(a)  $\leq 9$  Years of Education



(b) Years of Education



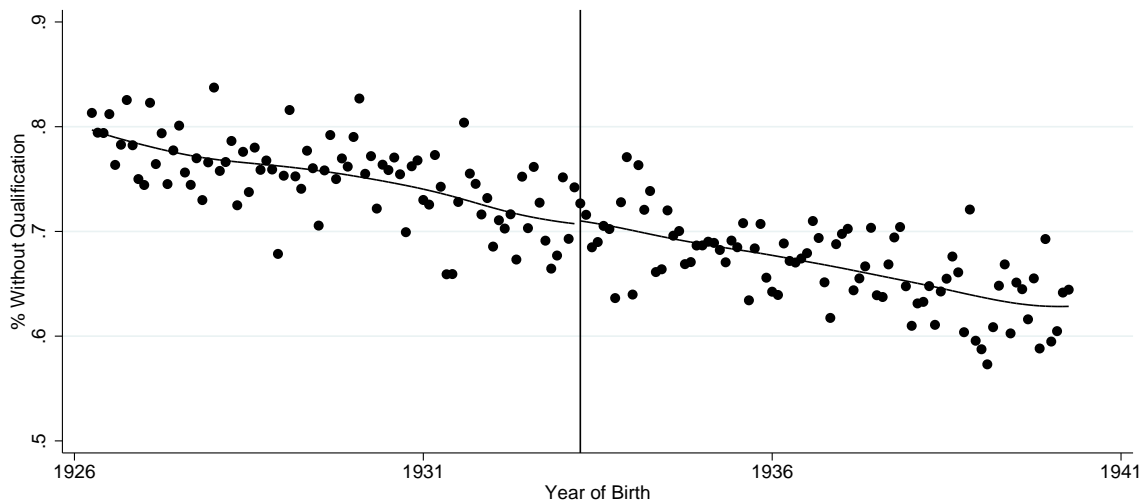
*Notes:* Figure B2 describes the effects of the 1947 ROSLA reform on educational attainment. Each dot is the average outcome for each month-year birth cohort. Horizontal lowess lines provide a flexible fit with the vertical line denoting the 1947 ROSLA reform. Panel A describes the effect of the 1947 reform on the proportion of students with no more 9 years of education. Panel B describes the effect of the same reform on years of education. The figure is based on pooled data from the 1995-2016 waves of the Scottish Health Survey (SHeS).

Figure B3: Comparison of Educational Measures in the SHeS and SLS Data

(a) SHeS: % No Qualification



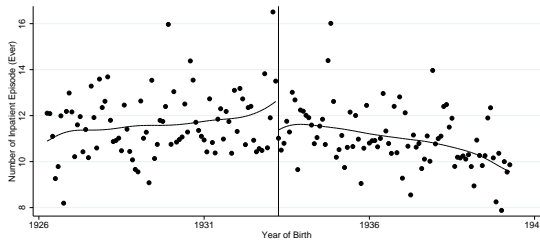
(b) SLS: % No Qualification



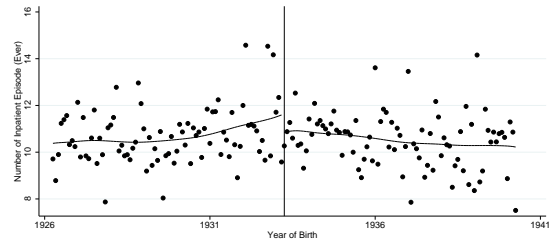
*Notes:* Figure B3 compares educational measures across the two main data sources used within this paper. Each dot describes the proportion without formal qualification ( $\approx$  high school dropouts) for each month-year birth cohort. Horizontal loess lines provide a flexible fit with the vertical line denoting the 1947 ROSLA reform. Panel (a) shows results using information from pooled waves of the Scottish Health Survey (SHeS) over the 1995-2016 period. Panel (b) shows similar results using the most recent information for each respondent within the Scottish Longitudinal Study (SLS).

Figure B4: Effects of the 1947 ROSLA Reform on Hospitalization

(a) Men: Inpatient Episodes



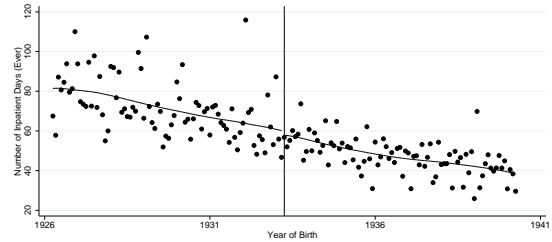
(b) Women: Inpatient Episodes



(c) Men: Inpatient Days



(d) Women: Inpatient Days

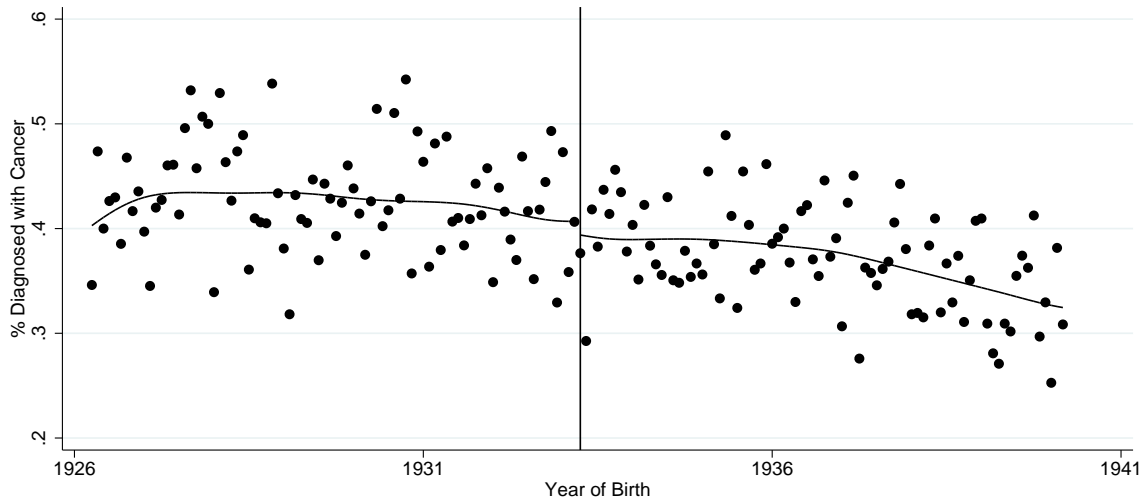


*Notes:* Figure B4 describes the reduced-form effects of the 1947 ROSLA reform on hospitalization. Each dot describes average hospitalization events in adulthood for each month-year birth cohort. Horizontal lowess lines provide a flexible fit with the vertical line denoting the 1947 ROSLA reform. Panels (a) and (c) describe the effects of the 1947 reform on inpatient episodes and days for men, respectively. Panels (b) and (d) provide the corresponding figures for women. The figure is based on data from the Scottish Longitudinal Study (SLS) over the 1981-2016 period.

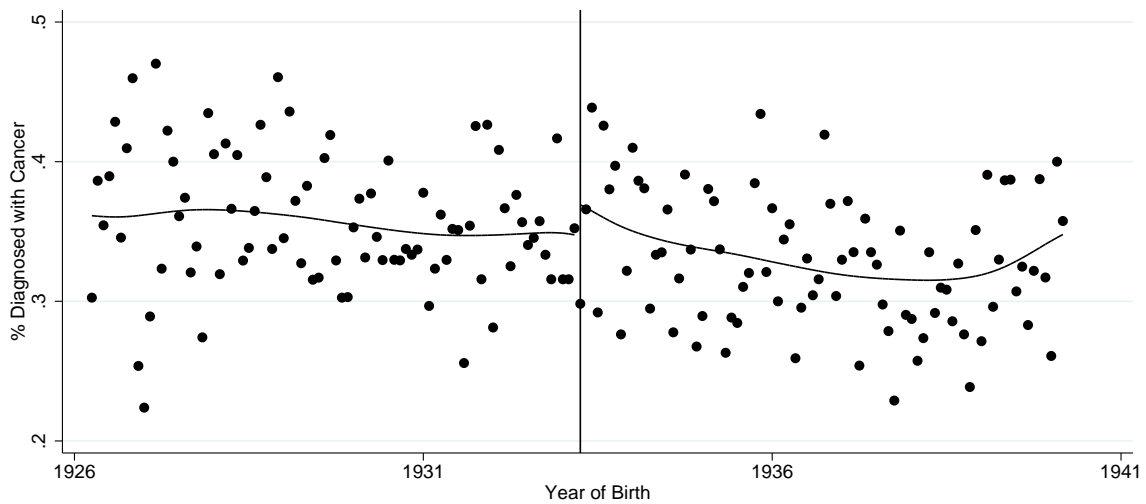


Figure B5: Effects of the 1947 ROSLA Reform on Cancer Prevalence

(a) Men: Any Cancer Diagnosis



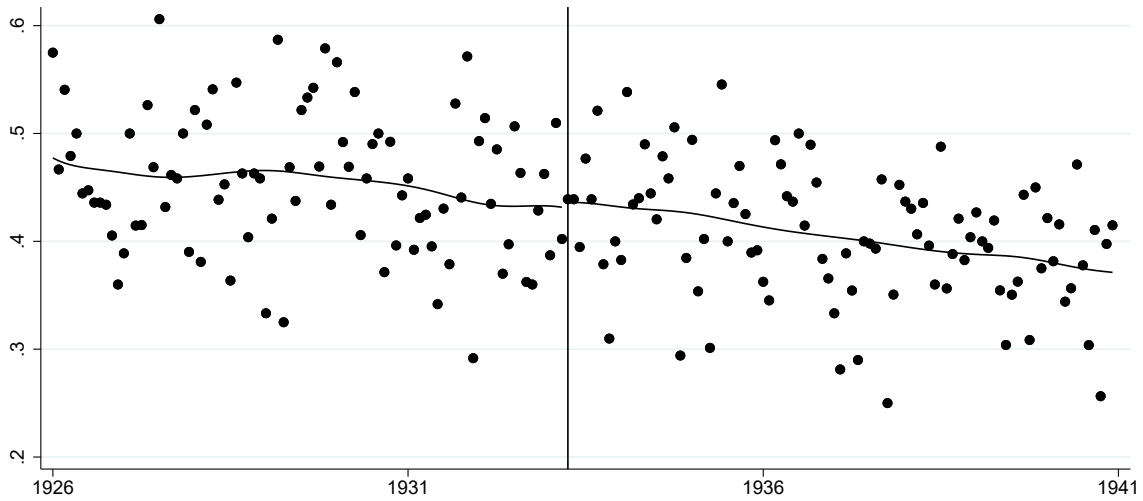
(b) Women: Any Cancer Diagnosis



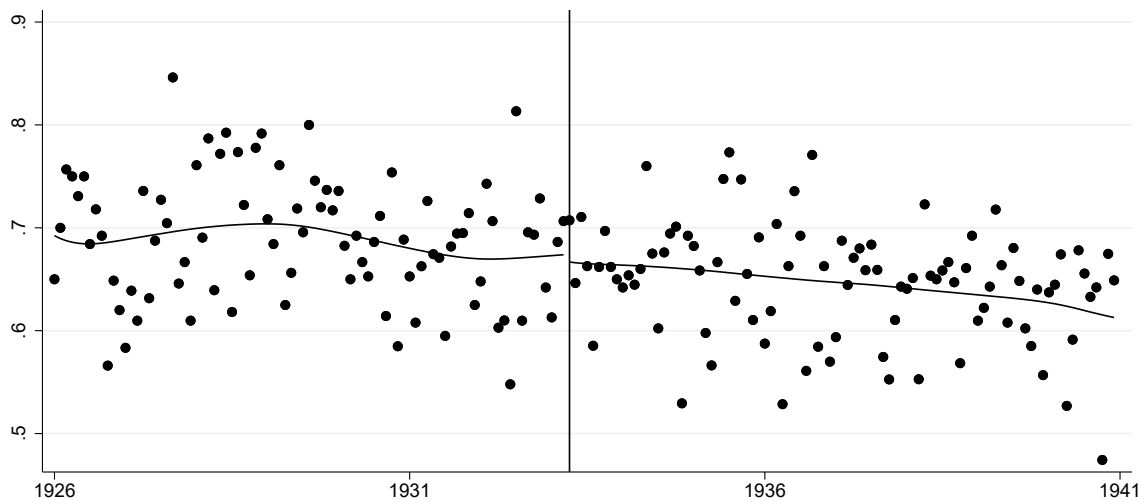
*Notes:* Figure B5 describes the reduced-form effects of the 1947 ROSLA reform on cancer diagnoses. Each dot describes the proportion diagnosed with any cancer in adulthood for each month-year birth cohort. Since cancer diagnoses are rare within these groups, we instead show quarterly aggregates for later cohorts for confidentiality reasons. Horizontal lowess lines provide a flexible fit with the vertical line denoting the 1947 ROSLA reform. Panels (a) and (b) describe the effects of the 1947 reform on alcohol-related inpatient episodes for men and women, respectively. The figure is based on data from the Scottish Longitudinal Study (SLS) over the 1981-2016 period.

Figure B6: Effects of the 1947 ROSLA Reform on Self-Reported Health

(a) 1947 Reform: Poor Health

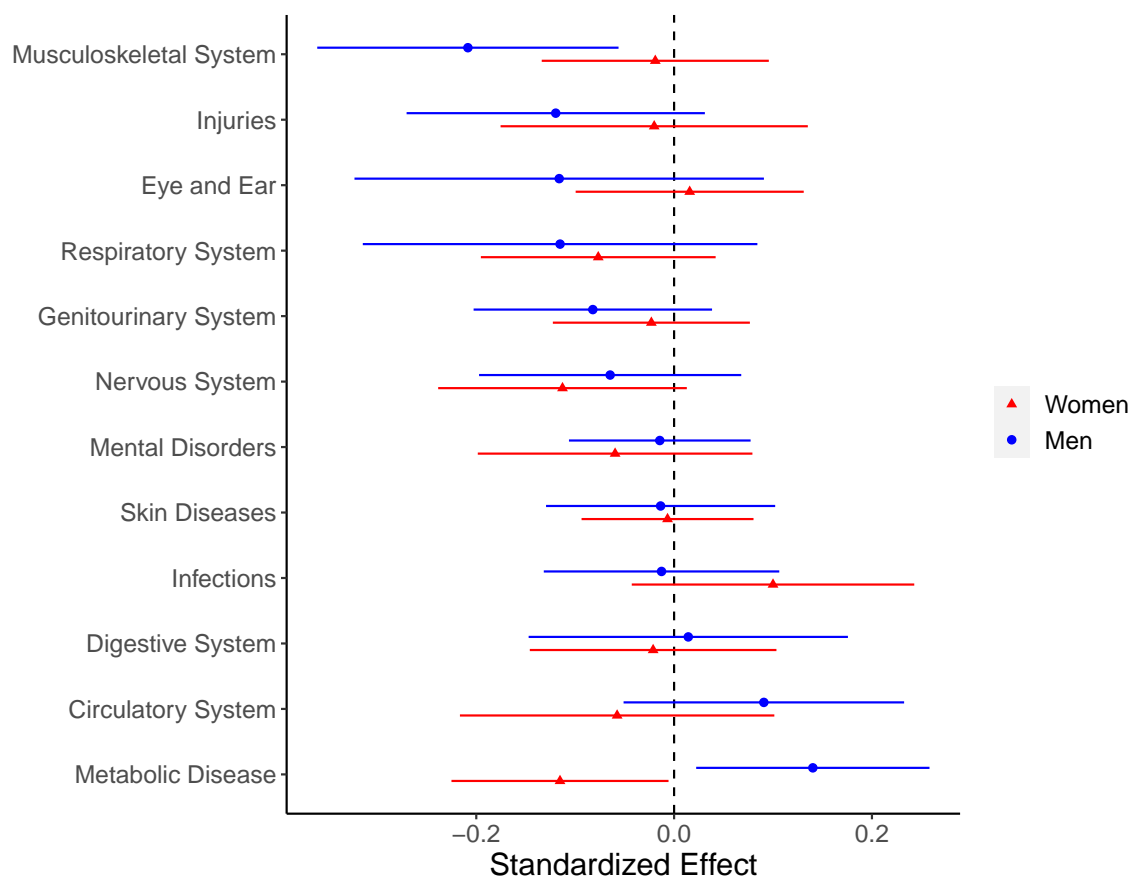


(b) 1947 Reform: Long-Standing Illness



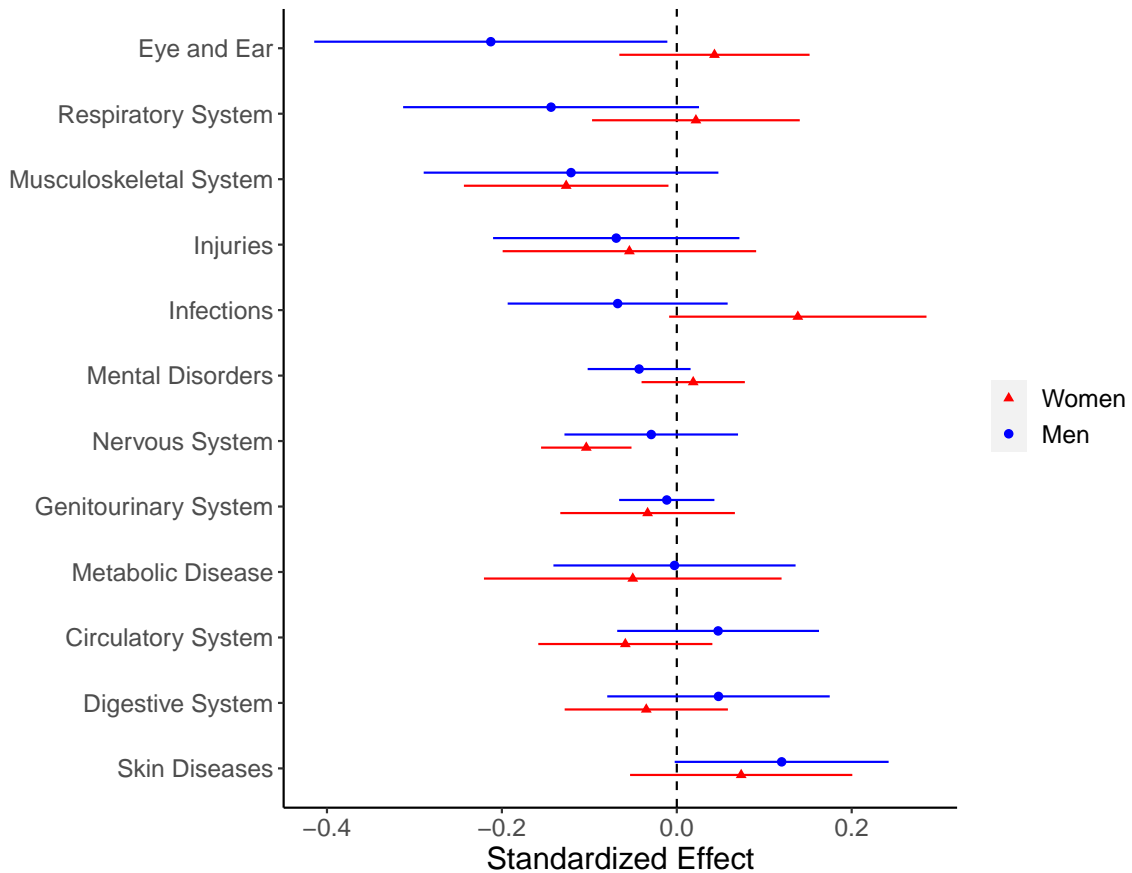
*Notes:* Figure B6 describes the reduced-form effects of the 1947 ROSLA reform on self-reported health. Each dot is the average outcome for each month-year birth cohort. Horizontal lowess lines provide a flexible fit with the vertical line denoting the 1947 ROSLA reform. Panel A describes the effect of the 1947 reform on the proportion of survey respondents reporting being in “fair,” “bad,” or “very bad” health. Panel B describes the effect of the same reform on the proportion of survey respondents who reported having a long-standing illness. The figure is based on pooled data from the 1995-2016 waves of the Scottish Health Survey (SHeS).

Figure B7: Effects of the 1947 ROSLA Reform on Hospitalization (Episodes) by Diagnosis



*Notes:* Figure B7 shows a series of reduced-form estimates using local polynomial regression discontinuity estimation, separately for men ( blue circles) and women (red triangles). Our main outcome is the aggregate number of observed inpatient episodes by primary diagnosis from 1981 to 2016. Effects are re-scaled to reflect changes in standard deviations. Horizontal lines are the associated 95% confidence intervals. All regressions control for ethnicity, childhood religion, and are centered around the reform cutoff date. We also flexibly control for birth month-year. All data come from the Scottish Longitudinal Study (SLS) and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses and clustered by birth month-year.

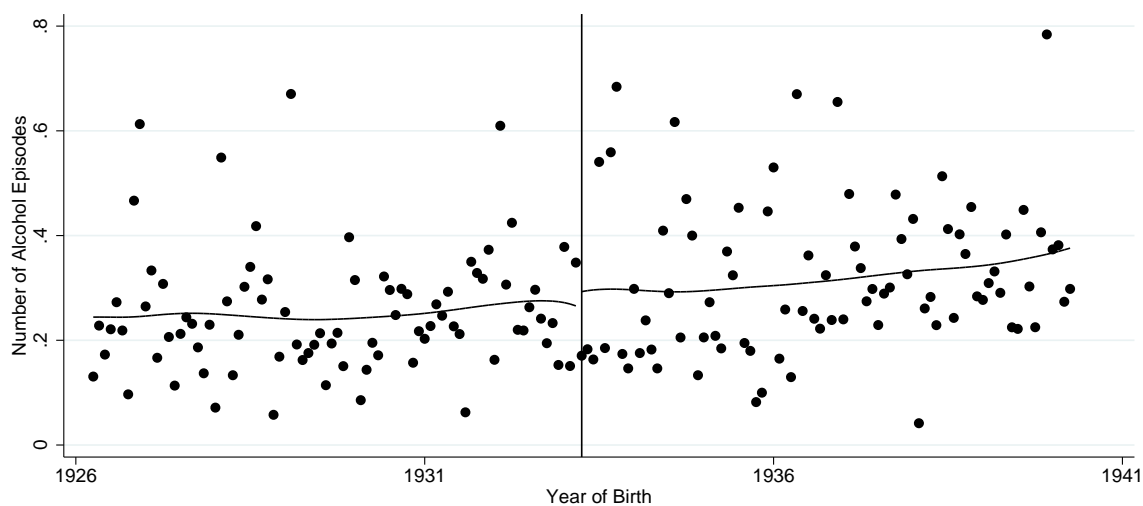
Figure B8: Effects of the 1947 ROSLA Reform on Hospitalization (Days) by Diagnosis



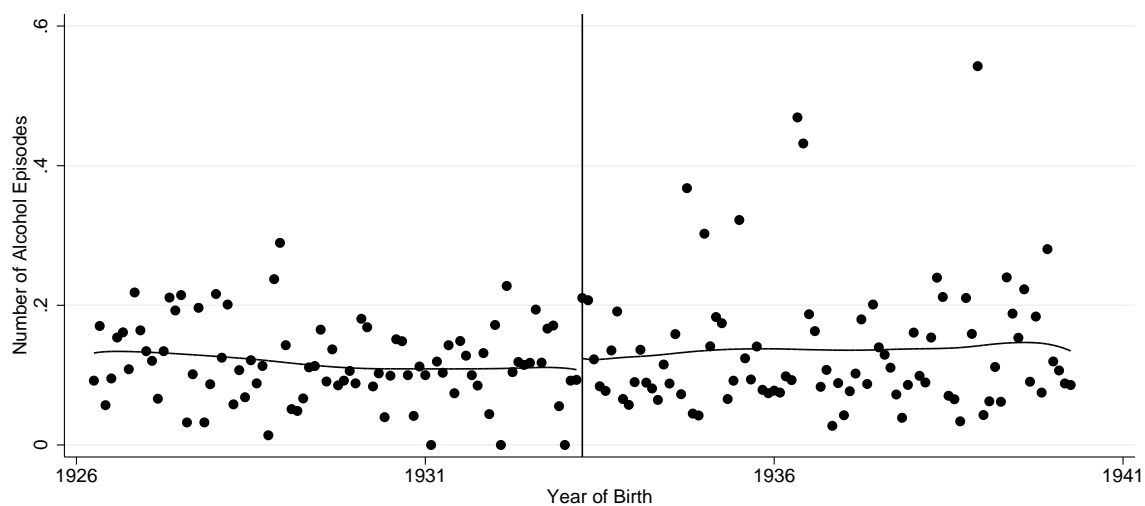
*Notes:* Figure B8 shows a series of reduced-form estimates using local polynomial regression discontinuity estimation, separately for men (blue circles) and women (red triangles). Our main outcome is the aggregate number of observed inpatient days by primary diagnosis from 1981 to 2016. Effects are re-scaled to reflect changes in standard deviations. Horizontal lines are the associated 95% confidence intervals. All regressions control for ethnicity, childhood religion, and are centered around the reform cutoff date. We also flexibly control for birth month-year. All data come from the Scottish Longitudinal Study (SLS) and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date. Heteroskedasticity-robust standard errors, obtained via nearest-neighbor variance estimation, are reported in parentheses and clustered by birth month-year.

Figure B9: Effects of the 1947 ROSLA Reform on Alcohol-Related Hospitalization

(a) Men



(b) Women



*Notes:* Figure B9 describes the reduced-form effects of the 1947 ROSLA reform on alcohol-related hospitalization. Each dot describes average alcohol-related inpatient episodes in adulthood for each month-year birth cohort. Alcohol-related admissions include episodes characterized as alcohol poisoning, intoxication, harmful use, or dependency/withdrawal. Horizontal loess lines provide a flexible fit with the vertical line denoting the 1947 ROSLA reform. Panels (a) and (b) describe the effects of the 1947 reform on alcohol-related inpatient episodes for men and women, respectively. The figure is based on data from the Scottish Longitudinal Study (SLS) over the 1981-2016 period.

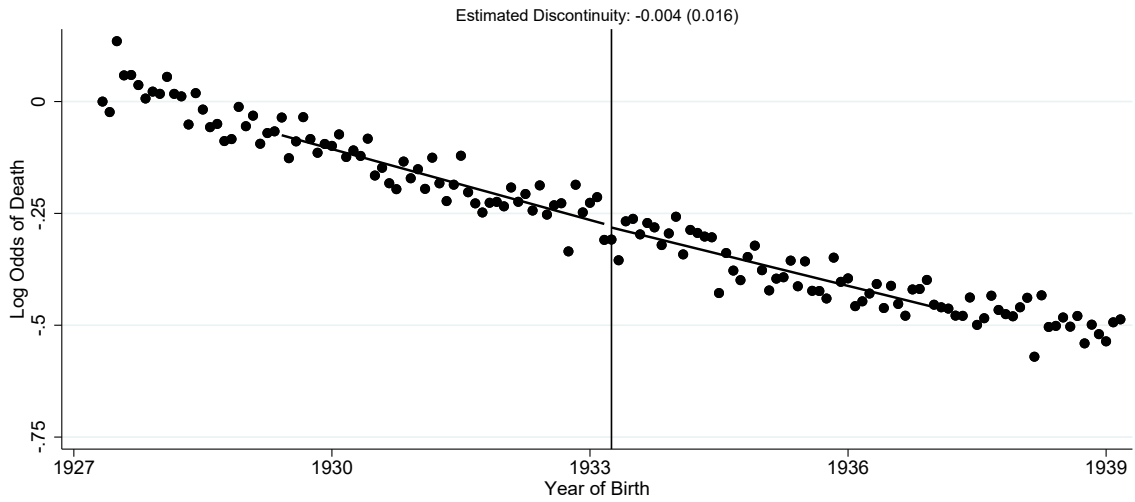
Figure B10: Effects of the 1947 ROSLA Reform on Drug Abuse-Related Hospitalization



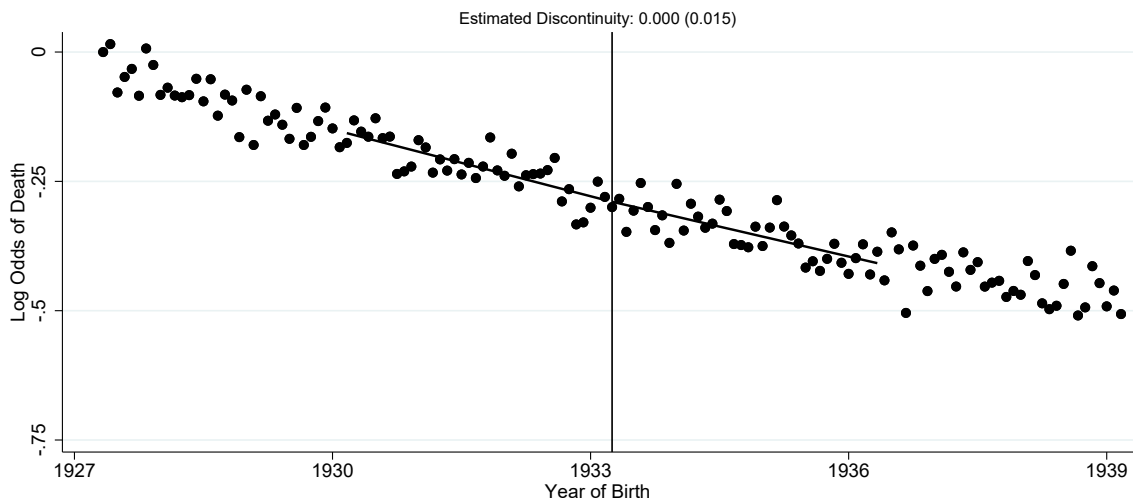
*Notes:* Figure B10 describes the reduced-form effects of the 1947 ROSLA reform on drug abuse-related hospitalization. Each dot describes average drug-related inpatient episodes in adulthood for each month-year birth cohort. Horizontal lowess lines provide a flexible fit with the vertical line denoting the 1947 ROSLA reform. The figure is based on data from the Scottish Longitudinal Study (SLS) over the 1981-2016 period and we further restrict this sample to individuals born within seven years of the ROSLA cutoff date..

Figure B11: Effects of the 1947 ROSLA Reform on Mortality

(a) Men



(b) Women



*Notes:* Figure B11 describes the relationship between the 1947 ROSLA reform and mortality. Each dot describes log odds death ratio for each month-year birth cohort. Estimation is given by the two-step procedure outlined in equations (5)-(6) in Section 4.4. The first step involves as panel logit regression of mortality on birth month-year fixed effects. Fitted values for these fixed effects then serve as the outcome variable in a local linear regression that we use to estimate the discontinuity at the reform cutoff. Panels (a) and (b) show results based on this procedure for men and women, respectively. All estimates should be interpreted relative to the September 1950 birth cohort. The vertical line denotes the 1947 ROSLA reform. This figure is based on data from the Scottish Census and Death Registry.

## Appendix C: Hospitalization Diagnosis Categories

Appendix C provides detailed definitions for each major category and subcategory associated with hospital admissions in the Scottish Longitudinal Study (SLS). Each category is based on the 2016 *International Classification of Diseases and Related Health Problems: 10th Revision, 5th Edition*. These categories appear in the following Figure 6, Figure A6, Figure B7, and Figure B8. Major categories are given with corresponding ICD-10 codes in parentheses:

### **Infectious and Parasitic Diseases (A00-B99)**

“Infections” include: Intestinal infectious diseases (A00-A09); Tuberculosis (A15-A19); Certain zoonotic bacterial diseases (A20-A28); Other bacterial diseases (A30-A49); Infections with a predominantly sexual mode of transmission (A50-A64); Other spirochetal diseases (A65-A69) Other diseases caused by chlamydiae (A70-A74); Rickettsioses (A75-A79); Viral and prion infections of the central nervous system (A80-A89); Arthropod-borne viral fevers and viral hemorrhagic fevers (A90-A99); Viral infections characterized by skin and mucous membrane lesions (B00-B09); Other human herpesviruses (B10); Viral hepatitis (B15-B19); Human immunodeficiency virus [HIV] disease (B20-B20); Other viral diseases (B25-B34); Mycoses (B35-B49); Protozoal diseases (B50-B64); Helminthiases (B65-B83); Pediculosis, acariasis and other infestations (B85-B89); Sequelae of infectious and parasitic diseases (B90-B94); Bacterial and viral infectious agents (B95-B97); Other infectious diseases (B99).

### **Endocrine, Nutritional, and Metabolic Diseases (E00-E89)**

“Metabolic Disease” include: Disorders of thyroid gland (E00-E07); Diabetes mellitus (E08-E13); Other disorders of glucose regulation and pancreatic internal secretions (E15-E16); Disorders of other endocrine gland (E20-E35); Intraoperative complications of endocrine system (E36); Malnutrition (E40-E46); Other nutritional deficiencies (E50-E64); Overweight, obesity and other hyperalimentation (E65-E68); Metabolic disorders (E70-E88); Other post-procedural endocrine and metabolic complications and disorders (E89).



## **Mental, Behavioral, and Neurodevelopmental Disorders (F01-F99)**

“Mental Disorders” include: Mental disorders due to known physiological conditions (F01-F09); Mental and behavioral disorders due to psychoactive substance use (F10-F19); Schizophrenia, schizotypal, delusional, and other non-mood psychotic disorders (F20-F29); Mood [affective] disorders (F30-F39); Anxiety, dissociative, stress-related, somatoform and other nonpsychotic mental disorders (F40-F48); Behavioral syndromes associated with physiological disturbances and physical factors (F50-F59); Disorders of adult personality and behavior (F60-F69); Intellectual Disabilities (F70-F79); Pervasive and specific developmental disorders (F80-F89); Behavioral and emotional disorders with onset usually occurring in childhood and adolescence (F90-F98); Unspecified mental disorder (F99-F99).

## **Diseases of the Nervous System (G00-G99)**

“Nervous System” include: Inflammatory diseases of the central nervous system (G00-G09); Systemic atrophies primarily affecting the central nervous system (G10-G14); Extrapyramidal and movement disorders (G20-G26); Other degenerative diseases of the nervous system (G30-G32); Demyelinating diseases of the central nervous system (G35-G37); Episodic and paroxysmal disorders (G40-G47); Nerve, nerve root and plexus disorders (G50-G59); Polyneuropathies and other disorders of the peripheral nervous system (G60-G65); Diseases of myoneural junction and muscle (G70-G73); Cerebral palsy and other paralytic syndromes (G80-G83); Other disorders of the nervous system (G89-G99).

## **Diseases of the Eye, Adnexa, Ear, and Mastoid Process (H00-H95)**

“Eye and Ear” include: Disorders of eyelid, lacrimal system and orbit (H00-H05); Disorders of conjunctiva (H10-H11); Disorders of sclera, cornea, iris and ciliary body (H15-H22); Disorders of lens (H25-H28); Disorders of choroid and retina (H30-H36); Glaucoma (H40-H42); Disorders of vitreous body and globe (H43-H44); Disorders of optic nerve and visual

pathways (H46-H47); Disorders of ocular muscles, binocular movement, accommodation and refraction (H49-H52); Visual disturbances and blindness (H53-H54); Other disorders of eye and adnexa (H55-H57); Intraoperative and postprocedural complications and other disorders of eye and adnexa (H59); Diseases of external ear (H60-H62); Diseases of middle ear and mastoid (H65-H75); Diseases of inner ear (H80-H83); Other disorders of ear (H90-H94); Other intraoperative and postprocedural complications and disorders of ear and mastoid process (H95).

### **Diseases of the Circulatory System (I00-I99)**

“Circulatory System” include: Heart Disease: Acute rheumatic fever (I00-I02); Chronic rheumatic heart diseases (I05-I09); Hypertensive diseases (I10-I16); Ischemic heart diseases (I20-I25); Pulmonary heart disease and diseases of pulmonary circulation (I26-I28); Other forms of heart disease (I30-I52); Cerebrovascular diseases (I60-I69); Diseases of arteries, arterioles and capillaries (I70-I79); Diseases of veins, lymphatic vessels and lymph nodes, not elsewhere classified (I80-I89); Other and unspecified disorders of the circulatory system (I95-I99).<sup>24</sup>

### **Diseases of the Respiratory System (J00-J99)**

“Respiratory System” include: Acute upper respiratory infections (J00-J06); Influenza and pneumonia (J09-J18); Other acute lower respiratory infections (J20-J22); Other diseases of upper respiratory tract (J30-J39); Chronic lower respiratory diseases (J40-J47); Lung diseases due to external agents (J60-J70); Other respiratory diseases principally affecting the interstitium (J80-J84); Suppurative and necrotic conditions of the lower respiratory tract (J85-J86); Other diseases of the pleura (J90-J94); Intraoperative and postprocedural complications and disorders of respiratory system, not elsewhere classified (J95); Other diseases

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<sup>24</sup>We also focus on “Heart Disease” which includes (ICD-10 codes defined as before): acute rheumatic fever, chronic rheumatic heart diseases, hypertensive diseases, ischemic heart diseases, pulmonary heart disease and diseases of pulmonary circulation, and other forms of heart disease.

of the respiratory system (J96-J99).

### **Diseases of the Digestive System (K00-K95)**

“Digestive System” include: Diseases of oral cavity and salivary glands (K00-K14); Diseases of esophagus, stomach and duodenum (K20-K3); Diseases of appendix (K35-K38); Hernia (K40-K46); Noninfective enteritis and colitis (K50-K52); Other diseases of intestines (K55-K64); Diseases of peritoneum and retroperitoneum (K65-K68); Diseases of liver (K70-K77); Disorders of gallbladder, biliary tract and pancreas (K80-K87); Other diseases of the digestive system (K90-K95).

### **Diseases of the Skin and Subcutaneous Tissue (L00-L99)**

“Skin Diseases” include: Infections of the skin and subcutaneous tissue (L00-L08); Bullous disorders (L10-L14); Dermatitis and eczema (L20-L30); Papulosquamous disorders (L40-L45); Urticaria and erythema (L49-L54); Radiation-related disorders of the skin and subcutaneous tissue (L55-L59); Disorders of skin appendages (L60-L75); Intraoperative and postprocedural complications of skin and subcutaneous tissue (L76); Other disorders of the skin and subcutaneous tissue (L80-L99).

### **Diseases of the Musculoskeletal System and Connective Tissue (M00-M99)**

“Musculoskeletal System” include: Arthropathies (M00-M25); Dentofacial anomalies [including malocclusion] and other disorders of jaw (M26-M27); Systemic connective tissue disorders (M30-M36); Dorsopathies (M40-M54); Soft tissue disorders (M60-M79); Osteopathies and chondropathie (M80-M94); ther disorders of the musculoskeletal system and connective tissue (M95); Other intraoperative and postprocedural complications and disorders of musculoskeletal system (M96); Periprosthetic fracture around internal prosthetic joint (M97); Biomechanical lesions, not elsewhere classified (M99).

### **Diseases of the Genitourinary System (N00-N99)**

“Genitourinary System” include: Glomerular diseases (N00-N08); Renal tubulo-interstitial diseases (N10-N16); Acute kidney failure and chronic kidney disease (N17-N19); Other disorders of kidney and ureter (N20-N23); Urolithiasis (N25-N29); Other diseases of the urinary system (N30-N39); Diseases of male genital organs (N40-N53); Disorders of breast (N60-N65); Inflammatory diseases of female pelvic organs (N70-N77); Noninflammatory disorders of female genital tract (N80-N98); Other intraoperative and postprocedural complications and disorders of genitourinary system (N99)

### **Injury, Poisoning, and Other Consequences of External Causes (S00-T88)**

“Injuries” include: Injuries to the head (S00-S09); Injuries to the neck (S10-S19); Injuries to the thorax (S20-S29); Injuries to the abdomen, lower back, lumbar spine, pelvis and external genitals (S30-S39); Injuries to the shoulder and upper arm (S40-S49); Injuries to the elbow and forearm (S50-S59); Injuries to the wrist, hand and fingers (S60-S69); Injuries to the hip and thigh (S70-S79); Injuries to the knee and lower leg (S80-S89); Injuries to the ankle and foot (S90-S99); Injury, poisoning and certain other consequences of external causes (T07-T88).