Gender Without Children*

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Abstract

What would the lives of women look like if they knew from an early age that they would not have children? Would they make different choices about human capital or early career investments? Would they behave differently in the marriage market? Would they fare better in the labor market? In this paper, we follow 152 women diagnosed with the Mayer-Rokitanski-Kuster-Hauser (MRKH) type I syndrome. This congenital condition, diagnosed at puberty, is characterised by the absence of the uterus in otherwise phenotypically normal 46, XX females. Using granular health registries matched with administrative data from Sweden, we confirm that MRKH is not associated with worse health, nor with differential pre-diagnosis characteristics, and that it has a large negative impact on the probability to ever live with a child. Relative to women from the general population, women with the condition have better educational outcomes, tend to marry and divorce at the same rate, but mate with older men, and hold significantly more progressive beliefs regarding gender roles. The condition has also very large positive effects on earnings and employment. Dynamics reveal that most of this positive effect emerges around the arrival of children in women in the general population, with little difference before. We also find that women with MRKH perform as well as men in the labor market in the long run. Results confirm that "child penalties" on the labor market trajectories of women are large and persistent and that they explain the bulk of the remaining gender gap.

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Introduction

What is the impact of children on women's life trajectories? How do children shape careers, marriage and partnership formation, education choices, and beliefs about gender roles? A large and growing literature studies the consequences of parenthood for women's labor market outcomes. A robust empirical finding, documented across countries and institutional contexts, is the existence of a substantial and persistent "child penalty" in earnings and employment following the birth of the first child (e.g. Lundberg and Rose (2000), Angelov et al. (2016), Kleven, Landais and Søgaard (2019), Kleven, Landais and Leite-Mariante (2024)). Using event-study designs and administrative data, this literature shows that gender gaps in earnings widen sharply at childbirth and remain large for many years thereafter. Instrumental-variable approaches, exploiting quasi-random variation in fertility, have largely confirmed these patterns (e.g. Gallen et al. (2023), Bensnes, Huitfeldt and Leuven (2023)).

Yet two important limitations constrain our understanding of the impact of fertility on the life trajectories of women relative to men. First, existing approaches primarily identify the *post-birth* impact of children. Event-study designs rely on comparing women before and after childbirth, under a no-anticipation assumption. Instrumental-variable strategies identify local average treatment effects around the timing of fertility shocks. Both approaches are therefore best suited to capturing the realized consequences of children once they arrive. They are less informative about *anticipatory effects*: forward-looking adjustments in education, occupation choice, partnership formation, and labor supply that may occur years before childbirth, in response to expectations about future family formation. If anticipated fertility affects expected labor supply interruptions, time constraints, or specialization within the household, it may influence choices long before children are born. Existing empirical designs are not well equipped to capture such effects. Second, long-run dynamic effects may be imperfectly identified. When fertility timing is endogenous and staggered across cohorts, dynamic treatment effects estimated out of heterogeneous exposure lengths may be confounded by dynamic selection. While recent advances in difference-in-differences methods have clarified identification under staggered adoption, they still rely on assumptions—such as parallel trends and no anticipation—that are difficult to test in this context (Kleven (2022)).

Addressing these limitations requires a different source of variation: exogenous differences in the probability of *ever* having children, known early in life and realized before major long-term investments (such as education, marriage, or early career choices) are made. Such variation would allow us to trace the full life-cycle consequences of (expected) childlessness, separately identifying anticipatory behavior and post-birth effects, and to construct a credible counterfactual trajectory of women absent children. In other words, the question we need to ask is: what would the lives of women look like absent children. What would gender mean without children?

In this paper, we study the lives of women with Mayer–Rokitansky–Küster–Hauser (MRKH) type I syndrome. MRKH type I is a rare congenital condition characterized by the absence of the uterus in otherwise phenotypically normal 46,XX females. The condition is typically diagnosed at puberty, when primary amenorrhea leads to medical investigation. Because it implies infertility that is known early in life, MRKH generates a large, plausibly exogenous, and early shift in the probability of ever having biological children. We combine detailed Swedish health registries identifying women diagnosed with MRKH with comprehensive administrative data on education, earnings, fertility, marriage, and family formation, complemented by an original survey. Our data allow us to follow women from adolescence through midlife and to compare them to women in the general population, as well as to men and to siblings.

We proceed in three steps. First, we document that MRKH is not associated with differential parental background, pre-diagnosis trajectories, or health outcomes, consistent with its congenital and largely random etiology. Women diagnosed with MRKH have dramatically lower probabilities of ever living with a child, although some adopt later in life. This confirms that the condition induces a substantial and persistent shift in expected fertility.

Second, we examine the impact of the condition on women's life trajectories, focusing on education, family formation, gender beliefs, and labor market outcomes. We find modest positive effects on overall educational attainment. Strikingly, however, women with MRKH make remarkably similar choices of field of study and early occupation as women in the general population: they do not disproportionately select more demanding programs, higher-return occupations, or male-dominated industries. In contrast, partnership formation patterns differ markedly. Women with MRKH are just as likely to marry, but they are more likely to divorce, tend to partner with older spouses, and are more likely to live in gender-egalitarian households. The condition is also associated with substantial differences in gender beliefs, with women with MRKH expressing significantly more progressive views on gender roles.Finally, we document large and economically significant labor market effects. On average, women with MRKH earn approximately 30 percent more between ages 20 and 40 than women in the general population.

Third, we go beyond an intent-to-treat framework, and use MRKH as an instrument for lifelong childlessness to revisit the identification of the child penalty. This strategy allows us to construct a counterfactual earnings trajectory of women absent children and to decompose the dynamic impact of children into pre- and post-birth components. We confirm large and persistent post-birth earnings penalties, consistent with prior event-study estimates. Importantly, we also identify significant pre-birth dynamics: earnings rise more steeply in the years preceding childbirth, consistent with endogenous timing of fertility and intertemporal substitution in labor supply.

Comparing counterfactual women without children to men yields a stark result. Absent children,

women's earnings trajectories in their thirties and forties closely resemble those of men. The vast majority of the gender earnings gap at these ages can be attributed to the impact of children. Differences in early adulthood are partly explained by education and occupation choices, but they account for only a modest share of the long-run gap.

Our findings make three contributions. First, we provide a novel design to identify both anticipatory and realized effects of children over the full life cycle. Second, we offer direct evidence that most of the persistent gender earnings gap in mid-career is driven by the child penalty, rather than by pre-existing differences in education or occupation. Third, we demonstrate the value of linking medical registry data to administrative and survey data to study rare conditions as sources of quasi-experimental variation. More broadly, our study provides a partial-equilibrium perspective on what women's lives would look like in a society where most others have children, but some women know early on that they will not. By leveraging exogenous variation in expected fertility, we shed new light on how children shape careers, partnerships, and gender inequality over the life cycle.

1 The Mayer–Rokitansky–Küster–Hauser (MRKH) Syndrome

Mayer–Rokitansky–Küster–Hauser (MRKH) syndrome is a rare congenital disorder of female reproductive development. It affects approximately one in 5,000 female live births, a prevalence that is consistently reported across clinical and population-based studies (see, among others, Herlin et al. (2020), Fontana et al. (2017)). Despite its rarity, MRKH represents one of the leading causes of absolute uterine factor infertility in women.

The condition is characterised by congenital absence or severe underdevelopment of the uterus, cervix, and the upper part of the vagina in otherwise phenotypically normal women with a 46,XX karyotype. A schematic representation of the reproductive anatomy associated with MRKH syndrome is provided in Figure 1. Importantly, external genitalia are normal, and ovarian development and function are typically preserved. As a result, secondary sexual characteristics such as breast development and pubic hair develop normally at puberty.

Clinically, MRKH syndrome is classified into two main subtypes. Type I (also referred to as the isolated or typical form) involves only the reproductive tract anomalies described above. Type II (also known as the atypical form or MURCS association) includes additional congenital malformations, most commonly affecting the renal and skeletal systems, but sometimes also involving hearing loss or cardiac defects. In this paper, we focus exclusively on women diagnosed with MRKH type I. Detailed diagnostic information allows us to identify and exclude women with type II MRKH.

From an embryological perspective, MRKH syndrome results from abnormal development of the

Müllerian ducts during early fetal life. In typical female development, the Müllerian ducts differentiate into the fallopian tubes, uterus, cervix, and upper vagina. In MRKH, this process is disrupted, leading to agenesis or hypoplasia of these structures. Existing evidence suggests that the condition arises from complex interactions between genetic and developmental pathways involved in Müllerian duct formation, regression, and differentiation (Mullen and Behringer (2014), Herlin (2024)). A comprehensive discussion of the embryology underlying MRKH is provided in Appendix Section I.1.

The genetic aetiology of MRKH syndrome is not yet fully understood, but current evidence supports a congenital origin with a strong genetic component. The disorder is widely considered to follow an autosomal dominant pattern with incomplete penetrance and variable expressivity, potentially involving multiple genes and regulatory elements (Herlin et al. (2019), Herlin (2024)). Because the condition is congenital and not linked to parental socioeconomic characteristics or behaviours, it is plausibly exogenous with respect to many economic outcomes of interest. Additional details on aetiology are discussed in Appendix Section I.2.[1]

A defining feature of MRKH syndrome is absolute uterine factor infertility. Because women with the condition lack a functional uterus, they are unable to carry a pregnancy themselves. In addition, the absence of the uterus leads to primary amenorrhea. As a result, the condition is typically diagnosed early in life, most often during adolescence when menstruation fails to occur despite normal pubertal development. This early and salient diagnosis distinguishes MRKH from many other health conditions that may remain latent or undetected until later adulthood.

Recent medical advances have begun to change the reproductive possibilities available to women with MRKH. Because ovarian function is usually intact, ovocyte retrieval followed by in vitro fertilisation and gestational surrogacy is one possible pathway to genetic parenthood in settings where surrogacy is legally permitted. In addition, uterus transplantation has emerged as a novel experimental treatment for absolute uterine factor infertility. As of 1 March 2025, more than 135 uterus transplants have been performed worldwide, resulting in over 65 healthy live births. However, these developments are not relevant for the cohorts studied in this paper. Our empirical analysis focuses on Swedish women born before the clinical availability of uterus transplantation, and surrogacy is not permitted under Swedish law. Consequently, all women with MRKH in our sample face infertility throughout their reproductive lives.

Beyond infertility, women with MRKH type I generally experience normal physiological and hormonal development. Ovarian function is preserved, and women ovulate regularly despite the ab-

[1] Historically, MRKH syndrome has been described and refined over more than two centuries, with early clinical accounts dating back to the late eighteenth and nineteenth centuries; a detailed historical overview is provided in Appendix Section I.4 (see also Patnaik et al. (2015)).

sence of menstruation. Ovulation without menstrual bleeding can nevertheless lead to cyclical pelvic or abdominal pain in some cases. Importantly, aside from the reproductive tract anomalies, women with MRKH type I do not differ systematically from the general population in terms of physical appearance or overall health. Although the upper portion of the vagina is absent or shortened at birth, effective treatments exist to enable sexual function. Non-surgical vaginal dilation or surgical vaginoplasty are routinely offered, typically in late adolescence or early adulthood. In our survey data, 83% of respondents report having undergone vaginal dilation or vaginoplasty, allowing for normal sexual intercourse.

Finally, MRKH syndrome is largely invisible to outsiders. Because affected women have normal external genitalia and secondary sexual characteristics, the condition is not observable in everyday social or professional interactions. Unless a woman chooses to disclose her diagnosis, friends, colleagues, and employers would generally be unaware of the condition. This lack of visibility is an important contextual feature for interpreting the social and economic outcomes studied in the remainder of the paper.

2 Data

For this project, we rely on Swedish health registries matched to rich administrative data containing detailed information on education, labor market outcomes, and demographics for the universe of the Swedish population.

2.1 Inpatient and Outpatient Care Data

The Swedish health care system is a universal, tax-funded system. We use information from several health registers collected by the National Board of Health and Welfare, which cover the entire Swedish population.

Our two main sources are the inpatient register (available for the period 1987 to 2015) and the outpatient register (available for the period 2001 to 2015). For each inpatient stay and outpatient visit, we observe the exact date and the associated diagnosis codes. Diagnoses may refer to either primary conditions or secondary diagnoses (i.e., coexisting conditions that, while not the main reason for admission or consultation, may affect treatment, require additional care, or be relevant for assessing the patient's overall condition). Diagnosis codes are recorded using ICD-9 classifications from 1987 to 1996 and ICD-10 classifications from 1997 onward. Access to outpatient records is particularly valuable in our context, as MRKH diagnoses are not necessarily made in hospital settings; they are often established by specialists in outpatient care.

2.2 Additional Registry Data

We complement the health registers with administrative information from a wide range of population-wide registries covering a broad set of behaviors and outcomes. This allows us to characterize life trajectories in detail for all Swedish men and women, with particular emphasis on four dimensions: education and early human capital investments; fertility and family decisions; labor market trajectories; and income and wealth accumulation.

Regarding education, we have detailed records at both the upper secondary (*Gymnasium*) and university levels for the years 1985–2015, including enrollment histories, field and major choices, grades, and degree completion. We also observe standardized test scores administered at the end of compulsory schooling (*Grundskola*). These national exams are mandatory for all students in grade 9, when students are approximately 15 years old.

We reconstruct fertility histories and family linkages using birth and adoption registers covering all biological births and adoptions from 1932 to 2015. Labor market trajectories are drawn from the LISA database (1985–2015) and matched employer–employee registers, as well as firm-level surveys covering approximately 50 percent of employees. These data provide detailed information on labor supply (participation, contracted hours, and effective hours), earnings, employers, and occupations.

2.3 Sample Construction and Descriptives

Identification of MRKH Syndrome We restrict our sample to individuals born between 1972 and 1995. This choice ensures that we can observe women around puberty—the typical age of diagnosis—and follow them as far as possible into adulthood, given the time coverage of our administrative data.

To identify women with MRKH syndrome, we rely on diagnoses indicating uterovaginal aplasia or agenesis (ICD-10 codes Q51.0: Congenital absence of uterus, and Q52.0: Congenital absence of vagina), together with evidence of a normal female karyotype and normal reproductive hormone profiles.[2]

The use of patient registry data to identify rare conditions in cohort studies remains relatively uncommon in epidemiology, partly due to concerns that ICD codes may imperfectly capture true diagnoses. In the case of MRKH, however, Herlin et al. (2016) conduct a validation study using the Danish National Patient Registry, linking ICD-coded cases to complete medical records. In 93

[2]For the period prior to 1997, diagnoses are recorded under ICD-9 codes. We rely in particular on codes 752D (Other anomalies or malformations of the uterus) and 752E (Anomalies or malformations of the cervix, vagina, and external genitalia).

percent of cases coded as Q51.0 or Q52.0, the diagnosis of MRKH was confirmed in the medical files, providing strong reassurance regarding the reliability of these codes.

To isolate MRKH type I cases, we further exclude women with extragenital anomalies (renal, skeletal, cardiac, or auditory) associated with MRKH type II, following classifications from the American National Organization for Rare Disorders and Herlin et al. (2016). Appendix Section I.5 provides the full list of excluded conditions and their corresponding ICD codes.

Table 1 reports prevalence estimates and compares them to the Danish population-based study. We identify 259 women with MRKH syndrome, corresponding to approximately one case per 6,319 female live births in Sweden. This prevalence is slightly lower than the Danish estimate (1 per 5,000) but remains within its 95 percent confidence interval.

Among identified cases, 37.1 percent present additional anomalies consistent with MRKH type II, including hearing loss (9.3 percent), heart defects (7.3 percent), kidney anomalies (16.8 percent), and skeletal malformations (16.6 percent). Women with MRKH type I—defined by the absence of such extragenital anomalies—represent 62.9 percent of cases, a proportion very close to the 56.5 percent reported in Denmark.

In the remainder of the paper, we focus on the 152 women identified with MRKH type I and compare their life trajectories to those of women in the general population born in the same cohorts. For simplicity, we refer to these women as having MRKH, with the understanding that we restrict attention to type I cases and systematically exclude type II cases.

Sample Descriptives Figure 2 Panel A presents the cohort distribution of MRKH diagnoses in our sample. We find no clear time trend in recorded prevalence, consistent with evidence from Herlin et al. (2016). The absence of trends is in line with the congenital and largely random etiology of the condition. It also suggests that, among cohorts born after 1972, there is no strong secular change in the probability of being diagnosed conditional on having the syndrome.

Our cohort restriction (1972–1995) reflects the need to observe women both around puberty—when diagnosis typically occurs—and sufficiently far into adulthood. Given that our administrative data span 1985–2015, women born in 1972 are observed from age 13 to 43, whereas those born in 1995 are observed only up to age 20. As a result, early educational outcomes are observed for all cohorts, while longer-term labor market outcomes are only available for older cohorts.

Panel B of Figure 2 displays the distribution of age at first recorded MRKH diagnosis. Three patterns emerge.

First, diagnoses before age 15 are extremely rare. This reflects clinical practice: primary amenorrhea—the main symptom of MRKH—is typically defined as the absence of menstruation by age 15, and medical consultation is generally recommended only after that age.

Second, most diagnoses occur between ages 15 and 18. In our data, 55 percent of women are diagnosed before age 18, with a clear mode at ages 17 and 18.

Third, a small tail of later diagnoses appears in the data. This likely reflects limitations in outpatient registry coverage, which begins only in 2001. Because many MRKH diagnoses are made in outpatient care, diagnoses recorded at later ages for older cohorts may correspond to secondary mentions rather than first diagnoses.

Two pieces of evidence support this interpretation. First, among cohorts born after 1986—whom we can observe in outpatient data from age 15 onward—85 percent of women are diagnosed before age 20. Second, in our survey data (see Section 2.4), the average reported age at diagnosis is 16.5 years; 91 percent report being diagnosed before age 20, and 98 percent before age 24.

Overall, the evidence indicates that nearly all women with MRKH are diagnosed during adolescence. To address potential measurement error in the precise timing of diagnosis—particularly for early cohorts—we report robustness checks using alternative definitions of MRKH exposure. Specifically, we compare specifications based on ever being diagnosed with MRKH to specifications excluding women who have not yet been observed with a diagnosis at a given point in time. We also present results focusing on a narrower subsample of women diagnosed with certainty before age 18.

2.4 Survey in MRKH Support Groups

We complement our administrative data with an original survey of women diagnosed with MRKH. Beyond providing an opportunity to validate our registry-based findings in an independent sample, the survey allows us to collect additional information that is not available in administrative records, including beliefs, norms, life satisfaction, and self-reported health. It also offers a unique window into the lived experience of the condition. Respondents report, for example, their sexual orientation, whether they underwent vaginoplasty, whether they disclose their diagnosis to friends, colleagues, or employers, and their expectations regarding romantic relationships and family life.

To recruit participants, we followed established practice in the epidemiological literature on rare conditions and contacted women through MRKH support groups in three large social media communities (see, e.g., Johnson et al. (2014) and Jacobs et al. (2016)). We obtained 131 completed responses with informed consent. Among respondents, 66 percent are diagnosed with MRKH type I, a share very close to that observed in the Swedish health registry data. As in the administrative analysis, we restrict the survey sample to women with MRKH type I syndrome.

Within this group, the average reported age at diagnosis is 16.5 years; 91 percent report having been diagnosed before age 20. Birth cohorts range from 1962 to 2004, with a median birth year of

1988. Respondents originate from a diverse set of countries, including Belgium, Canada, France, Hungary, Indonesia, Norway, Portugal, Sweden, the United States, and the United Kingdom.

Estimating the effects of MRKH on survey-based outcomes requires an appropriate comparison group representative of women from the same countries and cohorts. To this end, we designed our survey instrument to replicate as closely as possible the wording and structure of questions used in large, nationally representative surveys. For measures such as gender norms and life satisfaction, we rely on identical questions to those included in recent waves of the International Social Survey Programme (ISSP) and the World Values Survey (WVS). By pooling microdata from these sources with our survey data, we compare outcomes of women with MRKH to those of women from the same country and cohort in the ISSP and WVS samples.

A potential concern with recruitment through online support groups is selection into the survey sample. To assess the extent of selection, Appendix Table V.1 replicates, using survey data, key results on parental background, fertility, education, and labor market outcomes that can be benchmarked against the Swedish administrative data. While parental socioeconomic outcomes appear somewhat more favorable in the survey sample than in the administrative data, once we condition on parental background, we find remarkably similar effects of MRKH on fertility, educational attainment, and labor market trajectories. This comparison suggests that, conditional on parental characteristics, the surveyed MRKH women are broadly comparable to MRKH women in the population at large.

3 Pre-Diagnosis Outcomes, Health and Fertility

3.1 Pre-Diagnosis Characteristics

We begin by comparing women with MRKH to women in the general population prior to diagnosis. The objective is to assess whether women with the condition differ systematically in predetermined characteristics that could influence their lifetime trajectories, even before they are aware of their diagnosis. This exercise provides a direct test of the quasi-random distribution of the condition in the population and allows us to validate what is known about the etiology of MRKH. We focus on two sets of characteristics measured before diagnosis: parental background and early educational outcomes.

Family Background Using the birth and adoption registers, we construct intergenerational linkages that allow us to match each woman in our sample to her mother and father. We then estimate regressions of parental outcomes y_i^p, for parent $p \in \{\text{mother, father}\}$ of individual i, on cohort fixed effects and an indicator for whether the daughter is ever diagnosed with MRKH:

$$y_i^p = \alpha^p + \theta^p \cdot \mathbb{1}\left[\text{MRKH}_i = 1\right] + \sum_k \beta_k^p \cdot \mathbb{1}\left[\text{cohort}_i = k\right] + \epsilon_i^p. \tag{1}$$

We estimate this specification separately for mothers and fathers. Because any genetic mutation predisposing to MRKH would plausibly be transmitted through the paternal line, distinguishing between maternal and paternal outcomes allows us to assess whether the potential genetic channel is itself correlated with parental characteristics that could shape children's long-run outcomes.

We first examine parental education and labor market outcomes. For education, we use total years of completed schooling. For labor market outcomes, we consider labor force participation and total annual earnings, computed as averages over the years in which the child is between ages 10 and 14.

Panel A of Figure 3 reports the estimated coefficients θ^p from equation (1), scaled by the mean outcome in the general population. We find no statistically or economically significant differences in parental education, labor force participation, or earnings. Despite the limited number of MRKH cases, these null effects are estimated with reasonable precision.

Panel B of Figure 3 turns to family structure and parental fertility histories. We find no evidence that later parental age at childbirth is associated with a higher probability of MRKH: neither maternal nor paternal age at birth differs significantly between MRKH women and the general population. We also detect no relationship between MRKH incidence and parental completed fertility or birth parity. Similarly, parental marital status is not significantly correlated with the presence of the condition.

Finally, Appendix Figure II.1 shows no systematic association between MRKH and parental health. Using 12 broad categories of diagnoses, we find no significant differences in health outcomes for either mothers or fathers.

Childhood Environment and Early Educational Outcomes We next examine whether MRKH is correlated with differences in childhood environments. Because we observe individuals' place of residence, we can test whether the condition is systematically associated with variation in local socioeconomic conditions that might influence life trajectories. In practice, we find no such relationship. For example, Panel A of Appendix Figure II.2 shows no correlation between MRKH and the average income level of the municipality of residence during childhood.

We also assess whether differences emerge just prior to diagnosis. Sweden administers a nationwide standardized test at the end of *Grundskola* (compulsory schooling), when students are approximately 15 years old. Since MRKH diagnoses occur at age 15 or later, performance on this

exam provides a measure of academic achievement prior to knowledge of the condition. For each cohort, we rank students by their percentile in the national distribution of test scores and estimate specification (1), replacing the parental outcome with the individual's test score percentile.

Panel B of Appendix Figure II.2 reports the results. We find no statistically significant differences in test score percentiles between women later diagnosed with MRKH and women in the general population. These null effects remain robust after controlling for parental education, income, and marital status.

Taken together, these findings indicate that women diagnosed with MRKH do not differ from other women in terms of parental background, childhood environment, or pre-diagnosis educational performance in ways that could systematically shape their life trajectories. This evidence strongly supports the view that MRKH is distributed quasi-randomly in the population, consistent with current medical understanding of its etiology and physiology.

3.2 Health

Leveraging the rich diagnostic information available in the inpatient and outpatient registers, we now examine how MRKH shapes the health trajectories of affected women.

General Health Conditions We group diagnoses into 13 broad categories corresponding to the highest level of aggregation ("chapters") in the ICD-9 and ICD-10 classifications. For each category, we construct an indicator equal to one if an individual ever received, at any point in her life, a primary or secondary diagnosis within that category.

To assess the impact of MRKH on overall health, we estimate linear probability models analogous to specification (1), regressing each health-category indicator on cohort fixed effects and a dummy for ever being diagnosed with MRKH. Panel A of Figure 4 reports the results. For each condition, the dashed line represents the average prevalence in the general population, while the bar corresponds to the estimated coefficient θ capturing the association with MRKH.

Two categories, highlighted in red, exhibit significant differences. These are congenital malformations—mechanically equal to one among MRKH women—and pregnancy-, childbirth-, and puerperium-related conditions—mechanically equal to zero. These differences reflect the defining features of the syndrome. For the remaining eleven categories, we detect no systematic differences in prevalence between women with and without MRKH.

Menstrual Pain A limited medical literature documents pelvic pain and comorbid pain syndromes among individuals with MRKH (Gaikaiwari et al. (2024)). Although women with MRKH lack a uterus, they have functioning ovaries and may experience mild ovulatory pain (*mittelschmerz*). Clinical case studies also report instances of endometriosis among MRKH patients (e.g., Marsh et

al. (2013)).

To investigate menstrual pain more directly, we focus on diagnoses for premenstrual tension syndrome and dysmenorrhea (ICD-10 codes N94.3, N94.4, N94.5, N94.6; ICD-9 codes 625D, 625E). In the general female population, 3.5 percent are diagnosed with such conditions. Using a specification analogous to specification (1), we estimate that MRKH is associated with a 2.06 percentage point (0.27) reduction in the probability of receiving a menstrual pain diagnosis. Thus, women with MRKH are approximately 57 percent less likely to experience diagnosed menstrual pain, but they are not entirely exempt.

In the remainder of the paper, we show that our main results are robust to controlling for menstrual pain diagnoses or to excluding women with such diagnoses from the sample.[3]

Mental Health We next turn to mental health. In Panel A of Figure 4, we find no significant association between MRKH and the overall prevalence of mental and behavioral disorders. Panel B disaggregates mental health into eleven subcategories, each regressed on a dummy for ever being diagnosed with MRKH. Again, none of these conditions is significantly associated with the syndrome.

We also report the F-statistic from a joint test of significance of the eleven mental health indicators in a linear probability model predicting MRKH status. We reject joint significance, and this result remains unchanged after controlling for parental background and pre-diagnosis characteristics.

To examine dynamics, we assess whether mental health diagnoses differ before versus after age 15—the typical age of MRKH diagnosis. For each condition, we find no difference in the probability of diagnosis before age 15 (i.e., prior to diagnosis of MRKH) or afterward (Appendix Figure II.3). Estimating age-specific effects, we likewise find no systematic pattern suggesting a deterioration in mental health following diagnosis (Appendix Figure II.4).[4]

Reported Health and Life Satisfaction We next use our survey data to further investigate subjective health. A large majority of women with MRKH type I (92 percent) report that their health condition does not limit their daily activities. While informative, this statistic does not indicate whether subjective health differs relative to the general population.

To construct a comparison group, we pool our survey data with the World Values Survey and estimate:

[3] All available evidence suggests that our estimated labor market effects capture the consequences of infertility rather than amenorrhea per se. We return to this issue in the concluding section.

[4] Although point estimates are small throughout, statistical precision is limited; we cannot formally rule out increases of up to five percentage points.

$$y_i = \alpha + \theta \cdot \mathbb{1}\left[\text{MRKH Survey}_i = 1\right] + \mathbb{X}_i'\beta + \epsilon_i, \tag{2}$$

where $\mathbb{X}$ includes country, age, and cohort fixed effects. Figure 5 shows that women with MRKH do not report worse subjective health than women in the general population. We replicate the analysis using life satisfaction, measured on a 1–10 scale, and again find no negative association with MRKH. These results are robust to controlling for parental education.

Finally, respondents do not appear to anticipate poorer future health. When asked about perceived health risks, only two women reported concerns, both citing potential auditory problems.

3.3 Fertility and Probability of Living with a Child

We now examine the effect of MRKH on fertility. Using patient records for childbirth in the health registry data, we estimate the probability of having given birth by age a as a function of MRKH status:

$$\text{Pr(child)}_i^a = \alpha^a + \theta^a \cdot \mathbb{1}[\text{MRKH}_i = 1] + \epsilon_i^a. \tag{3}$$

Panel A.(i) of Figure 6 reports, for each age, the estimated coefficient θ^a scaled by α^a, the mean in the general population. The results confirm that MRKH is associated with a 100 percent reduction in biological fertility: none of the women with MRKH in our sample experienced childbirth. During the period covered by our data, there were no alternative pathways to biological motherhood for these women. Surrogacy was (and remains) illegal in Sweden, and uterus transplantation was not yet available.

Panel A.(ii) instead examines the probability of living with a child. Here, the negative association remains strong but is slightly smaller in magnitude. On average, women with MRKH are 87 percent less likely to live with a child between ages 20 and 40. The probability rises gradually after age 30, reflecting adoptions.

Panel B decomposes co-residence with children into three arrangements: biological children (orange), exclusively adopted children (blue), and exclusively stepchildren (green). Panel B.(i), for the general population, shows that approximately 85 percent of women live with a child by age 40; the vast majority have biological children, and most enter parenthood in their twenties.

In contrast, Panel B.(ii) shows that among women with MRKH, only 30 percent live with a child by age 40. This is driven almost entirely by adoption, which typically occurs after age 30.

3.4 Summary and Interpretations

Summary and Discussion regarding Health Effects of MRKH Taken together, the evidence presented above points to the absence of any systematic relationship between MRKH type I and overall health status. This pattern is fully consistent with current medical knowledge regarding the condition. In econometric terms, the results support an exclusion restriction: MRKH does not appear to affect women's life trajectories through direct and systematic effects on either objective or subjective health.

Three additional considerations are worth emphasizing.

First, the relatively small number of MRKH cases in our sample warrants caution. For health conditions with very low baseline prevalence, we cannot rule out moderate effects with high statistical confidence. Nevertheless, the uniformly small point estimates across all diagnostic categories, combined with the consistency of results across data sources (administrative registries and survey data) and across outcomes (objective diagnoses and subjective health), strongly suggest the absence of meaningful health-related channels.

Second, the lack of detectable effects on mental health diagnoses—and the absence of any negative association with life satisfaction—is consistent with existing medical studies based on smaller samples of MRKH patients.[5] However, these findings contrast with recent evidence by Bögl et al. (2024), who show that women experiencing temporary infertility, as revealed by unsuccessful attempts to conceive following assisted reproductive technology (ART), exhibit significantly higher rates of antidepressant and anti-anxiety prescriptions.[6]

Several factors may explain this discrepancy. First, women who fail to conceive with Assistive Reproductive Technologies (ART) will typically be undergoing infertility treatments which can cause hormonal imbalances. These, in turn, can significantly affect mental health by disrupting brain chemicals (neurotransmitters) that regulate mood, stress, and cognition, leading to symptoms like anxiety, depression, and mood swings. By contrast, MRKH is not associated with hormonal imbalances.

Second, both the learning process and nature of infertility are radically different across the two contexts. In the case of ART, infertility is revealed to individuals who want to conceive, after expectations have been set, and investments sunk towards the realization of this objective. Furthermore, couples doing fertility treatments that ultimately fail are often left with a diagnosis of

[5]For example, Weijenborg et al. (2019) compare 54 MRKH patients to 79 healthy controls using the Symptom Checklist–90 (SCL-90) and the Hospital Anxiety and Depression Scale (HADS), and find no significant differences between groups.

[6]Even accounting for the width of our confidence intervals, our estimates rule out effects as large as those documented in Bögl et al. (2024).

"unexplained infertility", opening the door to thought distortions. Such distortions are a key cause of psychological strain and can fuel emotional distress, according to the cognitive behavioral model (CBM) of mental health. Examples of such distortions are thinking that infertility is one of the partners' fault, that the couple should have tried to conceive earlier, or simply did not do something right. In line with that interpretation, Bögl et al. (2024) find that failure to conceive after ART initiation is significantly correlated with partnership strain and higher divorce rates.

In contrast, primary infertility due to MRKH is diagnosed early in life and is medically unambiguous: infertility stems from the congenital absence of a uterus. The random congenital nature of the condition leaves little scope for self-blame. Importantly, the diagnosis typically occurs before major life investments related to family formation—such as partnership choice—are undertaken. This difference in timing and clarity of diagnosis likely explains why the mental health dynamics associated with MRKH differ from those observed following ART failure.

This does not imply, however—and this is the third important point—that infertility associated with MRKH is inconsequential for well-being. The diagnosis may generate grief related to the inability to carry a child, and affected women would very likely prefer not to have the condition.[7] Our results simply indicate that such effects do not manifest in persistent, systematically detectable differences in diagnosed mental health conditions or reported life satisfaction at the population level.

Intent-to-Treat and IV Interpretations What do these findings on fertility, health, and parental background imply for identification?

Interpreting fertility as a first-stage outcome, MRKH generates a 100 percent reduction in biological fertility in our sample. The condition is diagnosed early in life (typically between ages 15 and 18) and is not associated with differences in parental background, childhood environment, or health characteristics that could independently shape life trajectories. These patterns align with the clinical and epidemiological understanding of MRKH type I as a random congenital condition.

In light of this evidence, we primarily report intent-to-treat (ITT) estimates, which can be interpreted as capturing the causal effect of primary infertility—known from adolescence—on subsequent life outcomes. The absence of systematic effects on mental health and life satisfaction further suggests that psychological well-being is unlikely to be a major mediating channel of the ITT estimates.[8]

[7] This is evidenced, for instance, by the strong interest in alternative reproductive options such as surrogacy or uterus transplantation. In Pastor et al. (2017), 91 percent of surveyed women with MRKH report that they would consider uterus transplantation.

[8] Although controlling for mental health diagnoses arguably conditions on an endogenous variable, we show that the ITT estimates are robust to such controls. We interpret this insensitivity as further evidence that the main effects of MRKH are not driven by changes in psychological health.

Because MRKH substantially reduces the probability of ever living with a child—only 25 to 30 percent of affected women adopt—we also present instrumental-variable (IV) estimates treating MRKH as an instrument for ever having children. This interpretation rests on two key assumptions. First, identification relies on standard monotonicity assumptions. Intuitively, we need to assume that individuals with the MRKH condition and who decide to have a child –an adopted child in this case– would also choose to have a child in the absence of the condition.Second, it assumes that the effects of adoption are comparable to those of biological children. This assumption can be assessed in an event-study framework and is strongly supported by the evidence in Kleven et al. (2021), which we replicate in our context.[9]

How does MRKH compare to other instruments used to study the effects of fertility?

Early contributions focused on instruments for the intensive margin of fertility, such as twin births (Rosenzweig and Wolpin (1980), Bronars and Grogger (1994)) and sibling sex composition (Angrist and Evans (1998)). More recently, Gallen et al. (2023) propose an instrument for the extensive margin based on failures of long-acting reversible contraceptives (LARCs), which generate unplanned births. Like MRKH, LARC failures produce a large and persistent first stage on the probability of having any child. However, the corresponding local average treatment effect pertains to women who had chosen not to conceive—a population distinct from that identified by MRKH. Notably, in the same Swedish context, Gallen et al. (2023) document labor market effects of comparable magnitude to those we estimate.

Another strand of the literature exploits infertility revealed through unsuccessful in-vitro fertilization (IVF) treatments (Lundborg et al. (2017), Bensnes et al. (2023)). As discussed above, infertility revealed through ART may itself affect health, income, or marital stability, raising concerns regarding the exclusion restriction.

Relative to these instruments, MRKH possesses several distinctive features that make it particularly well suited to studying the long-run dynamic effects of children. First, it combines a strong and persistent first stage on the extensive margin of fertility with credible exclusion restrictions. Identification of dynamic treatment effects requires keeping track of the full dynamics of the first-stage effect of the IV, a point emphasized in recent work (e.g., Gallen et al. (2023), Bensnes et al. (2023)). What matters is not simply how the instrument affects the probability to be treated in period 1, but also in all subsequent periods. Furthermore, the whole dynamics of the first-stage must be exogenous. With certain instruments, the difference in the probability to be treated in period 1 may be exogeneous, but not in period 2 or later, due to dynamic selection. In the MRKH setting,

[9] Appendix Figure IV.5 compares event-study estimates for adoptive mothers with MRKH to those for biological mothers in the general population. Because international adoption often requires marriage or stable partnership, we also explore specifications controlling for marital status and partner characteristics.

the probability of ever having children is exogenously shifted at all horizons.

Second, because MRKH is a random congenital condition, it does not identify a local average treatment effect for a subgroup defined by fertility preferences—such as women who sought IVF or women who deliberately avoided pregnancy. Instead, it shifts fertility prospects independently of prior intentions.

Finally, and crucially, MRKH is diagnosed early in life, before key investments in education, occupation, and partnership formation are undertaken. These investments are likely shaped by expectations about future childbearing. The MRKH setting therefore uniquely enables us to study not only the post-birth effects of children, but also the anticipatory adjustments that precede them, providing a comprehensive view of the dynamic impact of children over the life cycle.

4 Reduced-Form Effects of MRKH on Education, Family Formation, Beliefs, and Labor Market Outcomes

4.1 Education

We now document how various dimensions of women's lives are affected by primary infertility brought about by the MRKH syndrome. We start with education. We regress each educational outcome y_i on a dummy for ever having been diagnosed with MRKH and a vector $\mathbb{X}_i^p$ of controls for pre-determined characteristics.[10]

$$y_i = \theta \cdot \mathbb{1}\left[\text{MRKH}_i = 1\right] + \mathbb{X}_i^{p'} \beta + \epsilon_i \tag{4}$$

The vector $\mathbb{X}_i^p$ includes cohort fixed effects as well as a large set of pre-diagnosis characteristics capturing family background (parental education and income levels, parental marital and fertility history), childhood environment (county –*Sveriges län*– of birth, average income in municipality of residence during childhood) and pre-diagnosis educational attainment (grade percentile at age 15). While we showed in section 3 above that none of these characteristics correlates with MRKH, inclusion of these controls helps improving the statistical precision of our estimates of θ.

Results are reported in Table 2 and suggest the presence of positive but very modest effects of having the condition on educational attainment. First, we find a precise zero effect on the probability to graduate from high school.[11] This result is not surprising in light of the fact that most women

[10] Note that for each individual i, we define educational outcome y_i based on the highest education they achieved that is observable in our data.

[11] As explained above, graduation from *Grundskola*, in 9-th grade, marks the end of compulsory schooling in Sweden.

have just been diagnosed or are not diagnosed yet, at the time of finishing high school, leaving little space for behavioral adjustments. Moving to the number of years in education beyond compulsory schooling, we find a very small but insignificant correlation with MRKH.While we do not find much effect in terms of number of years of schooling, it does seem that MRKH women are slightly more likely to graduate from university (+ 2.3 pp (3.5)).

These small positive effects on education achievement are robust to the inclusion of a rich set of controls for health and psychological well-being (column (3)), and to the introduction of family fixed effects (in column (5)). A fraction of women may, of course, not learn about their condition before finishing their education. Can this explain such modest responses of educational investment? While the argument seems plausible, it is unlikely to be the main explanation, as we find similarly modest effects when restricting attention to women diagnosed before 18 (column (4)).

4.2 Marriage & Relationships

What are the implications of primary infertility on mating behaviors and family formation? To investigate this question, we follow a specification similar to (4) and regress various marriage and relationship outcomes on a dummy for having been diagnosed with the condition and a set of pre-diagnosis controls. Results, reported in Table 3, indicate interesting differences in the mating patterns of women with MRKH, patterns in line with models where reproductive capital shapes mating opportunities and returns in the marriage market as well as gender specialisation within the household (e.g. Low (2024)).

First, it does not seem that the MRKH condition is associated with a decline in marriage probabilities. If anything, women with MRKH are slightly more likely to ever marry, and there seems to be a small (although statistically insignificant) negative association with age at first marriage.[12] But women with MRKH are significantly more likely to ever get divorced. The effect size – a 5 pp increase – is quite large when compared to a mean of about 7% in the general population. Consequently, their average marriage duration is significantly shorter, by about 3 years.

In addition to having different marriage outcomes, MRKH women also choose different types of partners and have different relationship structures. In particular, they tend to be in relationships with significantly older men: the average age difference between MRKH women and their spouse is almost 5 years (against a baseline of 3.6 years in the general population). And while we do not detect any significant association with relative education within the household, we do find that MRKH women live in partnerships that are significantly more equal in terms of earnings. The

[12]Note that unfortunately, we cannot detect cohabitation patterns (i.e. unregistered partnerships) for women with MRKH who are not married. The reason is that cohabitants are defined in the administrative data as individuals of opposite sex living on the same property and having a (biological/adoptive) child together. Cohabitants who never have children together, as is the case for the vast majority of women with MRKH in our sample, cannot be identified.

effect size is extremely large: married MRKH women are almost 30pp more likely to earn strictly more than their husband relative to a baseline rate of just 17% for non-MRKH women.

4.3 Gender Beliefs

Using our dedicated survey, we can also explore to what extent the MRKH condition shapes beliefs and attitudes. Of particular interest are values regarding gender roles and the family. The survey elicited agreement with four statements that capture adherence to stereotypical gender roles: whether "*a preschool child is likely to suffer if mother works*", whether "*all in all, family life suffers when the woman has a full-time job*", whether "*Both men and women should contribute to the household income.*" and whether "*a man's job is to earn money; a woman's job is to look after the home*". These questions are present in global value surveys and responses can therefore be compared to responses in the general population, using the following linear probability model specification:

$$y_i = \theta \cdot \mathbb{1}\left[\text{MRKH Survey}_i = 1\right] + \sum_j \alpha_j \cdot \mathbb{1}\left[\text{age}_i = j\right] + \tilde{\mathbb{X}}_i^{p\prime}\beta + \epsilon_i \tag{5}$$

where y_i is a dummy for disagreeing with a particular statement. The vector $\tilde{\mathbb{X}}_i^p$ includes country fixed effects and cohort fixed effects, as well as controls for the level of parental education. Results are reported in Figure 7. In each panel, the blue bar depicts the estimated coefficient $\hat{\theta}$, while the dashed line corresponds to the counterfactual level of disagreement among MRKH women predicted from the model when omitting the contribution of $\hat{\theta}$. Across all statements, MRKH women appear much less likely to adhere to conservative gender stereotypes. They hold significantly more progressive gender attitudes and are much more likely to believe that work and family life are compatible. These results are insensitive to the inclusion of various controls, for health, for parental education or for own education. They also prove robust to the introduction of controls for having a child among women in the general population, a specification where we are in effect comparing MRKH women to women who do not have children. This suggests that these differences in values are not merely experiential: they do not simply reflect the impact of being exposed to children on gender stereotypes as in Pan et al. (2018). Instead, one may speculate that they capture adaptive preference formation, where desires or values endogenously respond to underlying opportunities (e.g. Elster (1983) or Nussbaum (2000)).

4.4 Labour Market Outcomes

We finally turn to labor market outcomes. For this analysis, observations are now at the individual-year level. We regress outcome y_{it} on the vector of controls $\mathbb{X}_i^{p'}$ defined above, to which we add a set of age fixed effects.

$$y_{it} = \theta \cdot \mathbb{1}\left[\text{MRKH}_{it} = 1\right] + \sum_j \alpha_j \cdot \mathbb{1}\left[\text{age}_{it} = j\right] + \mathbb{X}_i^{p'}\beta + \epsilon_{it} \tag{6}$$

The coefficient of interest is θ, which captures the effect of having been diagnosed with MRKH on average labor market outcomes from age 16 to 42, the age range over which our sampled cohorts are observed. Standard errors are clustered at the individual level.

Figure 8 reports the results, starting with total earnings. Note that we run specification (6) in levels, in order to include individuals with zero earnings. We then rescale the estimated coefficient $\hat{\theta}$ by the average predicted outcome among control women. We find very large positive effects on earnings. Women with MRKH earn almost 30% more on average than women in the general population. Decomposing this effect into extensive and intensive margin effects, we find that both play an important role. MRKH is correlated with a 10% increase in labor force participation, and a 15% increase in labor income conditional on participating.[13]

In short, women with MRKH fare significantly better than other women in the labor market. As we demonstrate in Figure 9, these very large positive effects of primary infertility on labor market outcomes appear very robust. First, to the definition of the "treatment" variable. Whether we focus on women who have been diagnosed, who will ever be diagnosed, or who are diagnosed before age 18, results are very similar (Panel A, Figure 9). Results are also very stable across the different sets of controls that we include (Panel B, Figure 9). In particular, they do not prove sensitive to controlling for a large vector of health diagnoses.[14] Nor are they affected by excluding women with genitourinary diagnoses indicative of premenstrual syndromes or dysmenorrhea. The results are also robust to the inclusion of family fixed-effects: it appears, if anything that comparing women with the MRKH condition to their sisters yields even larger effects on earnings.

Could these large effects of infertility on labor market outcomes be driven by marriage and relationship patterns, which, as we showed above, are significantly different for women with the

[13] Labor force participation is defined as having any positive earnings. The fact that the extensive and intensive margin percentage effects do not add up simply reflects the presence of significant positive selection into participation based on conditional earnings.

[14] In practice, for each chapter of ICD9 and ICD10 codes, we create dummies for ever being diagnosed with diseases corresponding to that chapter. We then include all dummies in specification (6).

MRKH condition? To investigate the mediating role of partnership structures, we add controls for marital outcomes and partner characteristics.[15] Estimates are hardly affected by the inclusion of these covariates, suggesting that mating patterns are not the main pathway through which infertility affects the labor market careers of women.

To uncover the mechanisms behind these large effects of MRKH on earnings, we now move to an in-depth analysis of their dynamics.

5 Dynamics, Child Penalties & Gender Gaps

5.1 Labour Market Outcomes over Time

To study how labor market effects evolve over the life cycle, we extend specification (6) by interacting age fixed effects with an indicator for having been diagnosed with MRKH:

$$y_{it} = \sum_j \theta_j \cdot \mathbb{1}[\text{MRKH}_{it} = 1] \cdot \mathbb{1}[\text{age}_{it} = j] + \sum_j \alpha_j \cdot \mathbb{1}[\text{age}_{it} = j] + \mathbb{X}_i^{p'} \boldsymbol{\beta} + \epsilon_{it}. \tag{7}$$

The coefficients of interest are the θ_j, which measure, at each age j, the difference in outcomes between women with MRKH and women in the general population.

Figure 10 plots the estimated θ_j, scaled by the counterfactual outcome for women with MRKH, defined as the model prediction absent the MRKH indicator. The figure shows that labor market outcomes are very similar across groups in the early twenties. From the mid-twenties onward, however, women with MRKH begin to outperform women in the general population, and this gap increases with age.

The timing of this divergence closely coincides with childbearing in the general population. Figure 10 also reports the age profile of the difference in the fraction of women with children between the two groups. The labor market gap widens sharply when women in the general population start having children, and subsequently levels off at later ages, when a subset of women with MRKH begin to have children through adoption.

Taken together, these patterns suggest that the positive association between infertility and labor market outcomes is largely driven by the arrival of children in the general population. This raises the question of whether the observed differences reflect anticipatory behaviors—that is, actions taken prior to childbearing that may respond to expectations about future family formation—or the direct impact of children after they are born.

[15] In practice, we introduce dummy variables for being married, and for being divorced, as well as controls for spousal education, spousal age and spousal earnings.

We address this question in two steps. First, we examine several dimensions of early-life investments, such as education and early career behaviors, and probe their responsiveness to the anticipation of childbearing. Second, we estimate a fully dynamic model of the impact of children on labor market trajectories, explicitly accounting for both pre-child and post-child effects.

5.2 Early Human Capital and Career Investments

We examine whether educational attainment and early career choices respond to primary infertility, as revealed by a diagnosis of MRKH syndrome. We begin by analysing broad educational field choices, classified according to the Swedish Standard Classification of Education.[16] For each educational field, we estimate the probability of selecting that field using a specification analogous to equation (4).

The results are reported in Figure 11, Panel A. The dashed line indicates the average probability of choosing each field in the general female population, while the blue bars report the estimated coefficient θ, capturing the effect of MRKH on the probability of selecting that field. For comparison, the figure also includes a red dashed line showing the average probability of choosing each field among men.

Educational field choices are strongly gendered. Relative to men, women are substantially less likely to choose manufacturing, engineering, and technology fields, and considerably more likely to choose health care. Against this backdrop, we find no statistically significant differences in broad field choices between women with MRKH and women in the general population. If anything, women with MRKH appear slightly more likely to select traditionally female-dominated fields such as health care or education. The only fields in which women with MRKH are somewhat less likely to enrol—thereby resembling men more closely—are the social sciences and humanities.

Table 4, Panel A, provides complementary evidence using a more granular classification of educational fields. At this level, we find small and marginally significant effects on the probability of choosing STEM fields, but no evidence that women with MRKH disproportionately select fields with a higher share of men or fields associated with higher expected lifetime returns.

We conduct a parallel analysis of early career choices, which provides further evidence on anticipatory human capital investment. We focus on labor market entry, defined as the first year in which an individual is observed working after having completed her highest level of education. We first examine broad occupational categories at labor market entry, using one-digit occupations from the Swedish Standard Classification of Occupations (SSYK). The results are shown in Figure 11,

[16] The Swedish SUN2000 (*Svensk utbildningsnomenklatur*) is the national standard classification of education, adapted from ISCED 97. It categorises education by level and field of study. Vocational programmes typically start at the upper secondary level. When the highest attained education is secondary school, the field is generally coded as general education.

Panel B. Once again, occupational choices at entry are remarkably similar for women with and without MRKH. This similarity is striking given the pronounced gender segregation in entry-level occupations: men are substantially more likely to work in manufacturing and much less likely to work in service-sector occupations.

In Table 4, Panel B, we further explore occupational sorting using a more detailed three-digit occupational classification. We first examine average working hours in the occupation, which capture differences in time intensity and the extent to which careers require continuous labor supply. We find precisely estimated zero effects: women with MRKH do not sort into occupations that demand longer working hours. We next consider average occupational wage rates and again find no evidence that women with MRKH disproportionately enter higher-paying occupations. Finally, we examine sorting into "women-friendly" workplaces, defined by the fraction of men in the occupation, and find no differential selection along this dimension.

Overall, these findings indicate that women with MRKH do not adjust early educational or occupational choices in ways predicted by standard forward-looking human capital models. Despite facing fundamentally different expectations regarding future childbearing and labor supply interruptions, they do not invest more heavily in high-return, high-intensity careers at labor market entry. This suggests that either anticipatory responses to future fertility constraints are limited at young ages, or that social norms and conformity considerations (as in, for example, Bursztyn et al. (2017)) constrain deviations from typical female early career paths, even when facing significantly lower expected lifetime career costs of family formation.

5.3 Revisiting the Child Penalty

We now revisit the child penalty through the lens of the preceding empirical evidence. Two findings are particularly salient. First, as shown in Figure 10, the labor market effects of MRKH display a distinctive dynamic pattern: the condition is associated with large and increasing positive effects that emerge precisely when women in the general population begin to have children. Second, as documented in the previous subsection, early educational and career choices—made prior to the onset of childbearing in the general population—are remarkably similar for women with and without MRKH.

Taken together, these findings suggest that the positive association between MRKH and labor market outcomes is largely driven by the absence of post-child penalties rather than by differential pre-child human capital investments. To formally characterise the impact of children on female labor market trajectories—and to decompose it into pre-child (anticipatory) and post-child components—we now move beyond intent-to-treat estimates and use MRKH more explicitly as an instrument to revisit the identification of the child penalty.

A Child Penalty Framework To clarify the discussion, we lay out a simple framework grounded in the potential outcomes approach. Let $\mathbf{E}_{jk}[Y_i]$ denote the expected labor market outcome Y at age j for a woman in the general population whose first child is born at age k. In our data, fertility is observed from ages 20 to 42; women who do not have a child by age 42 are assigned $k = \infty$. Let $\mathbf{E}_{j0}[Y_i]$ denote the potential outcome at age j in the absence of children.

We posit the following structure:

$$\frac{\mathbf{E}_{jk}[Y_i] - \mathbf{E}_{j0}[Y_i]}{\mathbf{E}_{j0}[Y_i]} = 1 + \underbrace{P_{j-k}}_{\text{“child penalty”}} + \underbrace{\gamma_k}_{\text{heterogeneity term}}. \tag{8}$$

At age j, women whose first child arrives at age k experience the $(j-k)$-th dynamic effect of childbearing, captured by P_{j-k}. We allow for unrestricted heterogeneity across women in the timing of first birth through γ_k, which captures permanent, non-child-related differences across groups defined by age at first birth. For expositional simplicity, we assume homogeneous average treatment effects of children across birth cohorts, though this assumption can be relaxed.

Let ω_k denote the probability that a woman in the general population has her first child at age k. By construction, $\sum_k \omega_k \gamma_k = 0$, implying that the weighted average of heterogeneity terms across fertility timing groups equals the population counterfactual profile absent children.

Identification Define event time $e = j - k$, corresponding to time since (or until) the birth of the first child. Under this structure, each dynamic child penalty parameter P_e is identified as a weighted average of relative outcome differences:

$$\forall e, \qquad P_e = \sum_k \omega_k \cdot \Delta_{e+k,k}, \tag{9}$$

where

$$\Delta_{jk} = \frac{\mathbf{E}_{jk}[Y_i] - \mathbf{E}_{j0}[Y_i]}{\mathbf{E}_{j0}[Y_i]}.$$

Intuitively, if a credibly exogenous control group of women who never have children is available, this allows identification of $\mathbf{E}_{j0}[Y_i]$ at all ages j. In turn, one can recover the full set of relative differences Δ_{jk} across all ages and fertility timings, and therefore identify the entire sequence of dynamic child penalty parameters $\{P_e\}$.

Connection to the Existing Literature This framework closely relates to the event-study and staggered difference-in-differences approaches commonly used to estimate child penalties. Standard identification in this literature relies on two key assumptions: parallel trends and no anticipa-

tion (see, for example, Kleven (2022)).[17]

A key advantage of our setting is that we can relax the no-anticipation assumption. When a valid counterfactual group of never-treated individuals is available, the full dynamic response to treatment—including anticipatory effects—can be identified (see Borusyak et al. (2024)). The MRKH condition provides precisely such a setting: it is a plausibly exogenous determinant of whether a woman ever has children, and it is known well before childbearing would otherwise occur in the general population.

Regarding parallel trends, equation (8) implicitly maintains a parallel trends assumption, expressed here in relative terms rather than levels. This assumption could also be relaxed, but only at the cost of imposing additional restrictions elsewhere. More generally, identification requires that the number of dynamic treatment effects and unrestricted group-specific trends not exceed the number of independent moments available in the data.

Finally, equation (9) reflects standard identification results in settings with dynamic treatments, as in Cellini et al. (2010), Giupponi and Landais (2022), Gallen et al. (2023), and Bensnes et al. (2023) in the context of child penalties. At any age, comparisons between treated groups (i.e. women who have children, but who had their first child at different age) and untreated women who do not have children mechanically combine multiple dynamic treatment effects. Proper identification therefore requires explicitly accounting for the full dynamics of treatment across groups that differ in their treatment timing.

Estimation We now turn to estimation. Building on the framework introduced above, we estimate the following specification:

$$y_{it} = \sum_j \theta_j \, \mathbb{1}[\text{age}_{it} = j] \cdot \mathbb{1}[F_i = 0] + \sum_{k>0} \sum_j \delta_{j,k} \, \mathbb{1}[\text{age}_{it} = j] \cdot \mathbb{1}[F_i = k] + \mathbb{X}_i^{p'} \boldsymbol{\beta} + \epsilon_{it}, \tag{10}$$

where F_i denotes age at first birth, with $F_i = 0$ for women who never have children.

If infertility due to MRKH implied perfect compliance—that is, if women diagnosed with MRKH had zero probability of having children—one could directly replace the indicator $\mathbb{1}[F_i = 0]$ with an indicator for MRKH diagnosis. In practice, some women with MRKH do become parents through adoption. We therefore instrument the indicator $\mathbb{1}[F_i = 0]$ using an indicator for being diagnosed with MRKH syndrome.

Under this specification, we recover $\hat{\Delta}_{j,k} = \frac{\hat{\delta}_{j,k} - \hat{\theta}_j}{\hat{\theta}_j}$, which corresponds to the relative difference at age j between women whose first child arrives at age k and women who never have children.

[17] Instrumental-variables approaches to estimating the dynamic impact of children rely on analogous assumptions: independence between treatment and dynamic potential outcomes (akin to parallel trends), and independence between the instrument and pre-treatment outcomes (akin to no anticipation).

Appendix Figure IV.1 illustrates the IV estimates of $\Delta_{j,k}$ from specification (10) using labor earnings as the outcome. Each panel corresponds to a different age at first birth k and plots $\hat{\Delta}_{j,k}$ across all ages j. The figure provides an intuitive visual representation of how the child penalty parameters are identified. In each panel, earnings decline sharply at the age of first birth relative to women who do not have children and remain persistently lower thereafter.

The child penalty at each event time e is then obtained by aggregating these effects across fertility timing groups, weighting by their population shares: $\hat{P}_e = \sum_k \omega_k \cdot \hat{\Delta}_{e+k,k}$. Standard errors for P_e are computed via bootstrap stratified across age at first child and having been diagnosed with MRKH.

Results Figure 12, Panel A, presents the estimated child penalties in total earnings.[18] Panel B compares these estimates to those obtained from a standard event-study specification and from intent-to-treat estimates.

Three main insights emerge. First, the short-run impact of childbirth is large and precisely estimated, and closely matches standard event-study estimates. This is unsurprising, as identifying assumptions underlying event-study designs are generally most credible in the immediate post-birth period.

Second, we find substantial and persistent long-run child penalties. Ten years after the birth of the first child, earnings remain 25–30% lower relative to the counterfactual of never having children. These magnitudes are remarkably close to those obtained by Gallen et al. (2023) for Sweden and Bensnes et al. (2023) for Norway, using alternative IV strategies. This consistency reinforces the conclusion that the labor market impact of children is large and highly persistent.

Third, and most novel, we uncover clear pre-child dynamics. In the years leading up to the first birth, women's earnings are not only higher but also grow at a steeper rate relative to women who never have children. The magnitude of this effect is substantial: relative earnings increase by roughly 15 percentage points in the three to four years preceding childbirth. This pattern contrasts with the common view that women anticipating motherhood begin to reduce labor market investments well before children arrive. Instead, the evidence is consistent with strong intertemporal substitution: women increase labor supply and earnings when the time cost of doing so is lower, namely prior to the arrival of children and associated constraints.

These pre-child dynamics are also consistent with endogenous fertility timing. Women typically postpone childbearing until after completing education and accumulating initial labor market experience, a phase characterized by steep earnings growth. Supporting this interpretation, Appendix

[18] Estimates for employment are reported in Appendix Figure IV.3.

Figure IV.4 shows that controlling for age at exit from education reduces the magnitude of pre-child effects by approximately one half.

Finally, these dynamics may be reinforced by institutional incentives embedded in the parental leave system. As suggested by Albrecht et al. (2018), Swedish women may increase labor supply and earnings prior to childbirth in order to qualify for more generous parental leave benefits.

5.4 Comparison with Men & Implications for Gender Gaps

So far, we have focused on comparisons between women with MRKH and women in the general population. We now broaden the perspective and ask: how do women with MRKH compare to men? More broadly, what do the child-penalty estimates identified using MRKH as an instrument imply for gender gaps in earnings?

To address these questions, we extend specification (6) to include men:

$$
\begin{aligned}
y_{it} = & \sum_j \alpha_j \, \mathbb{1}[\text{age}_{it} = j] \cdot \mathbb{1}[M_i = 1] \\
& + \sum_j \theta_j \, \mathbb{1}[\text{age}_{it} = j] \cdot \mathbb{1}[F_i = 0] \cdot \mathbb{1}[M_i = 0] \\
& + \sum_j \delta_j \, \mathbb{1}[\text{age}_{it} = j] \cdot \mathbb{1}[F_i > 0] \cdot \mathbb{1}[M_i = 0] \\
& + \mathbb{X}_i^{p\prime} \boldsymbol{\beta} + \epsilon_{it}
\end{aligned} \tag{11}
$$

where $\mathbb{1}[M_i = 1]$ indicates that individual i is male. The control vector $\mathbb{X}_i^p$ is unchanged and includes cohort fixed effects and parental background controls.

As before, we instrument the indicator $\mathbb{1}[F_i = 0]$ (never having children) with an indicator for MRKH diagnosis. This allows us to recover the predicted earnings profile of women absent children. Conceptually, this counterfactual profile is anchored in the earnings trajectory of women with MRKH, corrected for the fact that a small fraction of them become parents through adoption. Figure 14, Panel A, compares the observed earnings profile of women with MRKH to the IV-based counterfactual profile of women absent children. The counterfactual is plotted with 95% confidence intervals (standard errors clustered at the individual level). The two profiles are nearly identical, diverging slightly around age 30, when some women with MRKH begin adopting children. Panel B contrasts this counterfactual profile with the earnings trajectory of women who never have children in the data. The difference is striking: women who remain childless exhibit a much flatter earnings profile. This highlights the importance of a valid instrument. Selection into childlessness is substantial and operates in both directions. On the one hand, some women with strong

career preferences remain childless and exhibit steep earnings growth (positive selection). On the other hand, adverse shocks—such as health problems—may jointly depress fertility and earnings (negative selection), as suggested by results in Bögl et al. (2024). Overall, these selection forces distort the naive comparison between mothers and women who never have children.

We next compare these profiles to those of men. From specification (11), we recover the age-specific earnings trajectories of men and of all women in the population, net of cohort effects. These are shown in Figure 13, together with the counterfactual profile of women absent children.

The average gender earnings gap between ages 20 and 42 is approximately 30%, and it widens steadily over the life cycle. However, comparing men to counterfactual women absent children yields a markedly different picture. In the early twenties, the trajectories of all women and counterfactual women are nearly identical. Starting in the late twenties, the counterfactual profile diverges upward from the average female profile and converges toward the male trajectory. By age 40, the earnings of counterfactual women absent children are statistically indistinguishable from those of men. Between ages 32 and 42, the remaining gap averages only 3%. This implies that roughly 90% of the gender earnings gap at these ages can be attributed to the impact of children.

These results confirm that child penalties are clearly first-order in explaining gender inequality in earnings. Absent children, women's earnings trajectories would closely resemble those of men in their thirties and forties.

These findings are robust to the inclusion of family fixed effects. Women with MRKH have earnings trajectories much closer to those of their brothers than to those of their sisters. Relative to their sisters, women with MRKH begin to diverge sharply in their twenties, exhibit steeper growth throughout their thirties, and earn substantially more by their forties. The earnings gap with brothers after age 30 is approximately 4%, compared to 35% for non-MRKH sisters (see Appendix Figure IV.6).

Nevertheless, a gender gap remains in the twenties. Between ages 20 and 32, the earnings gap between men and counterfactual women absent children is 16.9%, close to the overall gender gap at those ages. Why do women who know they will not have children not exhibit steeper early-career earnings profiles?

A first explanation relates to education. Women attain more education than men and enter the labor market later. As a result, they are more likely to have zero earnings in their early twenties and accumulate less labor market experience at young ages. This mechanically generates flatter early-career earnings profiles and steeper growth later on. To quantify the role of education, we augment specification (11) with controls for completed education, years of schooling, and field of

study.[19] We then reconstruct the counterfactual earnings profile of women without children under the assumption that they had the same educational distribution as men. As shown in Figure 15, Panel A, controlling for education closes nearly half of the early-career gender gap. Nearly half of the earnings gap in the twenties is therefore attributable to differences in educational attainment and field choice.

A second channel operates through early occupational sorting. As shown earlier, women—including those with MRKH—enter different occupations than men at labor market entry. To assess the contribution of this margin, we further augment specification (11) with controls for first occupation. This amounts to constructing a counterfactual in which women without children have the same education and initial occupational allocation as men. As shown in Figure 15, Panel B, accounting for occupation reduces the early-career gap slightly further, but the quantitative impact is modest. This likely reflects the relatively compressed wage distribution and wage floors across occupations in the Swedish labor market, as in other Scandinavian countries.

A final potential explanation for the residual early-career gap is discrimination. Because MRKH is not a visible condition, employers cannot directly observe which women will remain childless. Statistical discrimination against women based on expected fertility may therefore persist even for those who will not have children. To explore this possibility, we exploit information from our survey on whether women disclosed their condition to colleagues or employers. Approximately 45% did not disclose to colleagues, and 55% did not disclose to their employer. We adapt specification (5) to include an interaction between MRKH status and disclosure to the employer. Although these results are correlational, Appendix Figure V.3 shows a large and statistically significant positive association between earnings and disclosure, consistent with the presence of statistical discrimination against women early in their careers.

Conclusion

In this study, we seek to advance understanding of the life trajectories of women diagnosed with MRKH type I. Leveraging detailed health registries linked to comprehensive administrative data, and complemented by a bespoke survey, we demonstrate the value of combining population-wide registry data with targeted survey evidence to study rare medical conditions.

Our findings confirm the current medical understanding of MRKH as a random congenital condition. Diagnosis is not associated with parental health or socioeconomic characteristics, nor with differential pre-diagnosis trajectories. We find no evidence that women with MRKH experience worse subsequent health outcomes, including mental health. Nor do we detect lower levels of

[19] Specifically, we include fixed effects for completion of education, years of education, and one-digit SUN codes for field of education.

subjective life satisfaction. In this sense, MRKH appears orthogonal to background characteristics and early-life development, making it a uniquely powerful setting for studying the consequences of infertility.

At the same time, MRKH continues to have a substantial impact on fertility outcomes. Although uterus transplantation technologies have expanded rapidly over the past decade, offering new possibilities for women with the condition, primary infertility remains the dominant reality in our sample. Women with MRKH are significantly less likely to ever live with a child. Those who do typically become parents through adoption, and at later ages—primarily in their thirties.

Against this background, we document that primary infertility has limited effects on education and human capital accumulation. In contrast, it substantially shapes family formation and partnership trajectories. While women with MRKH have similar probabilities of ever marrying, they exhibit higher divorce rates and sort differently into partnerships. The condition is also associated with distinct beliefs: women with MRKH are significantly more likely to hold progressive views on gender roles.

Most strikingly, MRKH is associated with large and persistent differences in labor market trajectories. On average, women with MRKH earn approximately 30 percent more between ages 20 and 40 than women in the general population.[20] This differential emerges primarily from the large post-birth earnings declines experienced by women in the general population.

Taken together, our results provide direct and unusually clean evidence on the central role of the "child penalty" in shaping women's labor market trajectories. Absent children, women's earnings profiles would closely resemble those of men. Indeed, we show that nearly the entire gender earnings gap at prime working ages can be attributed to the impact of children on women's careers, consistent with the mechanism highlighted by Kleven et al. (2019).

The 152 women with MRKH type I whom we follow in this study offer a rare empirical window into infertility in a society where the vast majority of women can and will have children. This setting allows us to construct counterfactual life trajectories for women absent children in a partial-equilibrium sense. These counterfactuals are conceptually distinct from the "Children of Men" scenario imagined in Alfonso Cuarón's dystopian film, in which universal infertility leads to social collapse. Our analysis is not about a world without children in general equilibrium. Rather, it examines what happens within an existing social equilibrium—where norms, institutions, and

[20]The estimated effect of MRKH on labor market outcomes captures infertility rather than amenorrhea. Results are robust to controlling for, and excluding, women diagnosed with menstrual pain. Moreover, mediation analysis shows that nearly the entire effect operates through the arrival of children in the general population, ruling out amenorrhea and supporting infertility as the key mechanism. Existing evidence suggests that any direct link between menstruation and labor market outcomes—typically operating through absenteeism—is limited (Herrmann and Rockoff (2012)).

expectations are built around parenthood—when some women know from an early age that they will not have biological children.

Precisely because the broader equilibrium remains unchanged, this perspective is highly informative. It reveals the extent to which, in contemporary societies, children fundamentally shape women's economic trajectories, family lives, and beliefs. By isolating infertility as an exogenous and congenital shock, we provide new evidence on the magnitude and mechanisms of the child penalty, and on the central role of parenthood in generating persistent gender inequality in the labor market.

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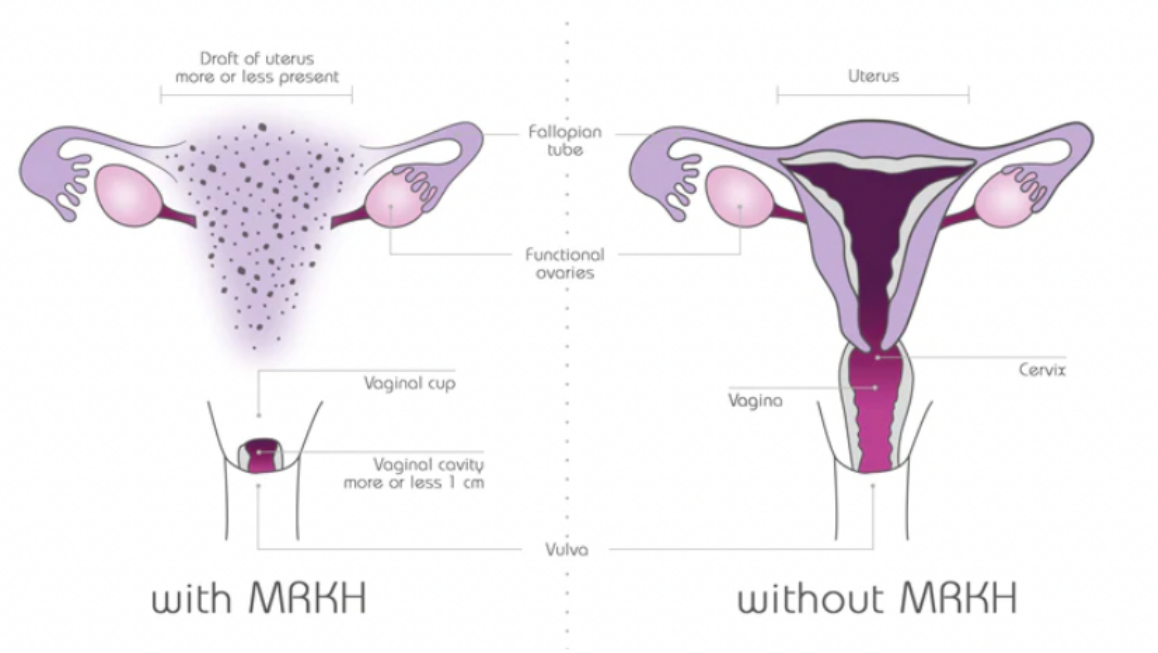
FIGURE 1: **Reproductive system: Women With & Without MRKH Syndrome**



Notes: The figure provides a schematic representation of the reproductive anatomy associated with MRKH syndrome. From an embryological perspective, MRKH syndrome results from abnormal development of the Müllerian ducts during early fetal life. In typical female development, the Müllerian ducts differentiate into the fallopian tubes, uterus, cervix, and upper vagina. In MRKH, this process is disrupted, leading to agenesis or hypoplasia of the uterus, cervix, and the upper part of the vagina in otherwise phenotypically normal women with a 46,XX karyotype. Although the upper portion of the vagina is absent or shortened at birth, effective treatments exist to enable sexual function. Non-surgical vaginal dilation or surgical vaginoplasty are routinely offered, typically in late adolescence or early adulthood. External genitalia are normal, and ovarian development and function are preserved. As a result, secondary sexual characteristics such as breast development and pubic hair develop normally at puberty.

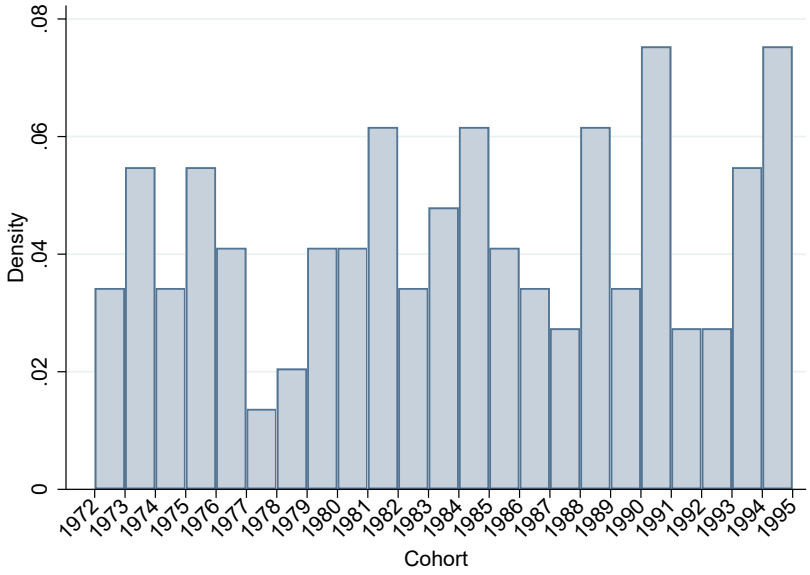
TABLE 1: **Epidemiology of MRKH**

	Medical study (Herlin et al.(2016))	Swedish data
Prevalence of MRKH	1 in 4,982	1 in 6,319 (259 women)
Age at first diagnosis	17.5	18
Typical (Type I) MRKH syndrome (%)	56.5	62.9
Atypical (Type II) MRKH syndrome (%)	43.5	37.1
Hearing loss (%)	*1.8*	*9.3*
Heart defects (%)	*3.6*	*7.3*
Kidney anomalies (%)	*34.2*	*16.8*
Skeletal malformations (%)	*12.5*	*16.6*

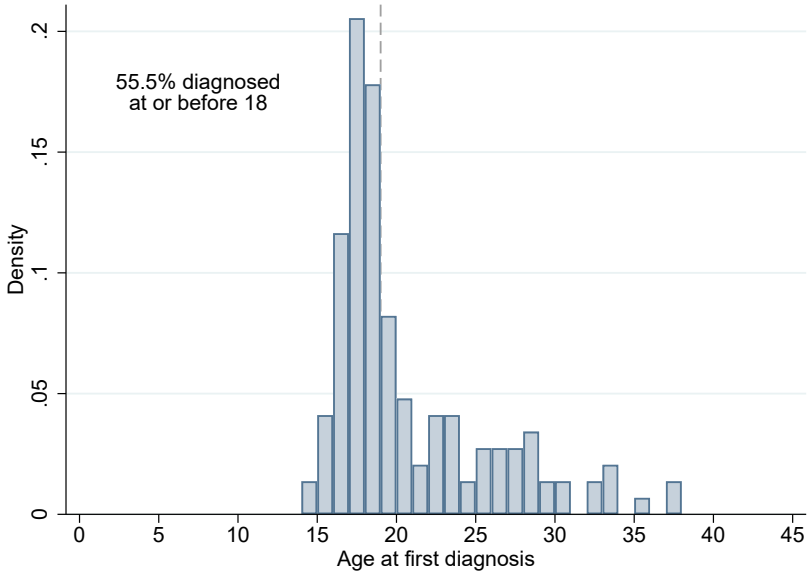
Notes: The table provides details about the prevalence of the MRKH syndrome in our data, and offers a comparison to results from Herlin et al. (2016), the only other population-wide prevalence study of MRKH, in Denmark. Our sample is restricted to individuals born between 1972 and 1995. We identify 259 women with MRKH syndrome, corresponding to one case for 6,319 female live births in Sweden. This prevalence is a bit smaller than the prevalence found in Denmark (1/5,000) but still lies in the 95% confidence interval of the Danish study. We also find that a significant portion of women (37.1%) are subject to additional conditions (such as hearing loss (9.3%), heart defects (7.3%), kidney anomalies (16.8%) and skeletal malformations (16.6%)), which are indicative of the presence of the type II MRKH syndrome, as listed by the American National Organisation for Rare Disorders and Herlin et al. (2016). Appendix Section I.5 provides a list of all these conditions, with their corresponding ICD codes. Women with type I syndrome, who experience none of these conditions, represent 62.9% of the population of MRKH women, a proportion very close to results from the Danish cohort study (56.5%). In the rest of the paper, we focus on these 152 women identified with MRKH type I syndrome, and compare their lives to the lives of all women in the general population, born in the same cohorts.

FIGURE 2: **Sample Descriptives**

A. Distribution of Women with MRKH by Cohort



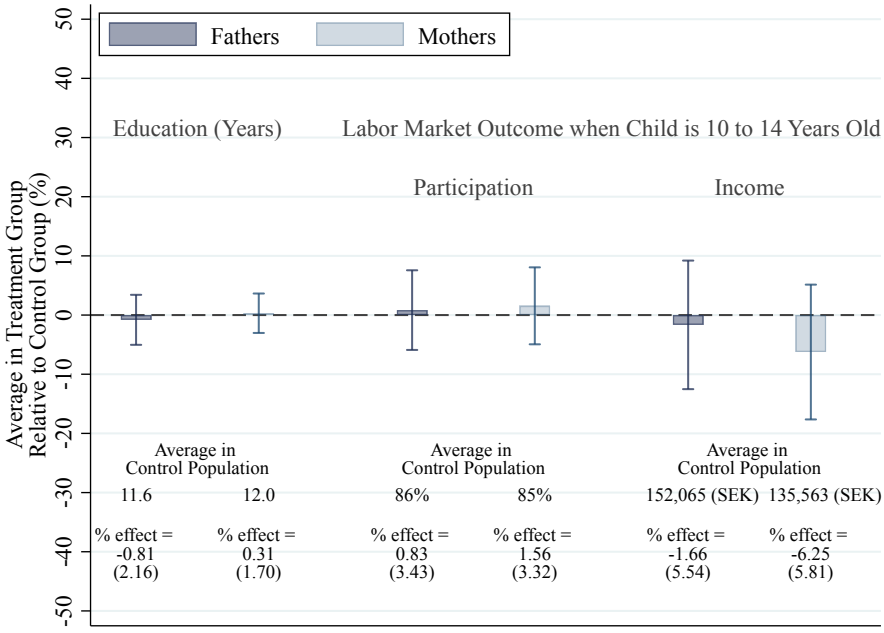
B. Distribution of Age at First MRKH Medical Record



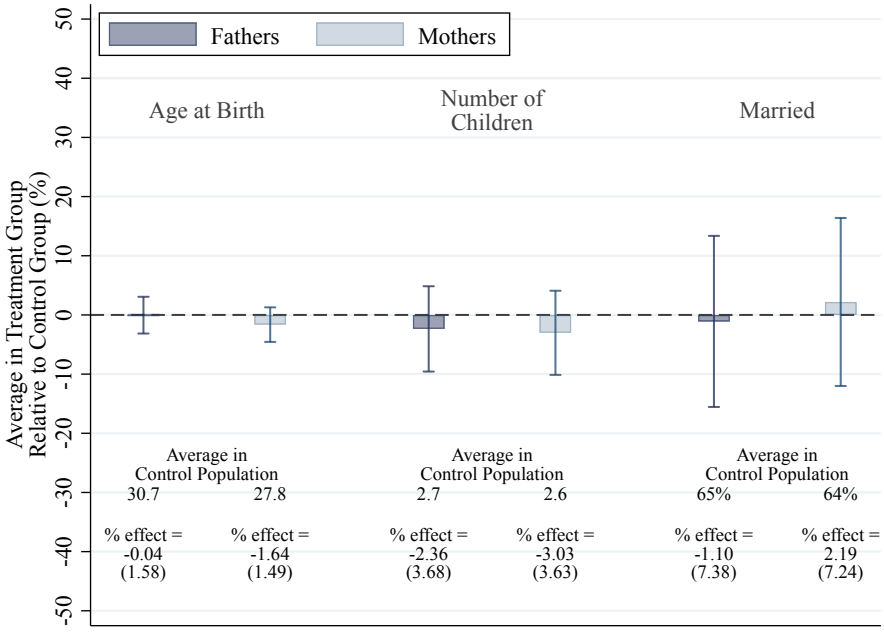
Notes: Panel A shows the cohort distribution of women with MRKH in our sample. Panel B displays the distribution of age at which the first MRKH diagnosis is observed in our health registry data. Diagnoses made before age 15 are extremely rare. This is because the Swedish health care system recommends seeing health care providers for primary amenorrhea only after adolescent girls reach the age of 15. The majority of diagnoses is made between 15 and 18. 55% of women with MRKH appear to be diagnosed before 18, with a large mode of diagnoses around 17 and 18. The small tail of late diagnoses is an artefact of the specific time coverage of our health registry data as a large fraction of women are diagnosed in the outpatient care system for which our coverage unfortunately only starts in 2001. As a result, for earlier cohorts, an observed MRKH diagnosis in the outpatient care system at a later age may not correspond to the first time a woman was diagnosed, but rather to a secondary diagnosis. See text for more details and evidence indicating that almost all women with the MRKH condition are diagnosed in their teens.

FIGURE 3: **Parental Outcomes and Family History**

A. Parental Education and Labor Market Outcomes

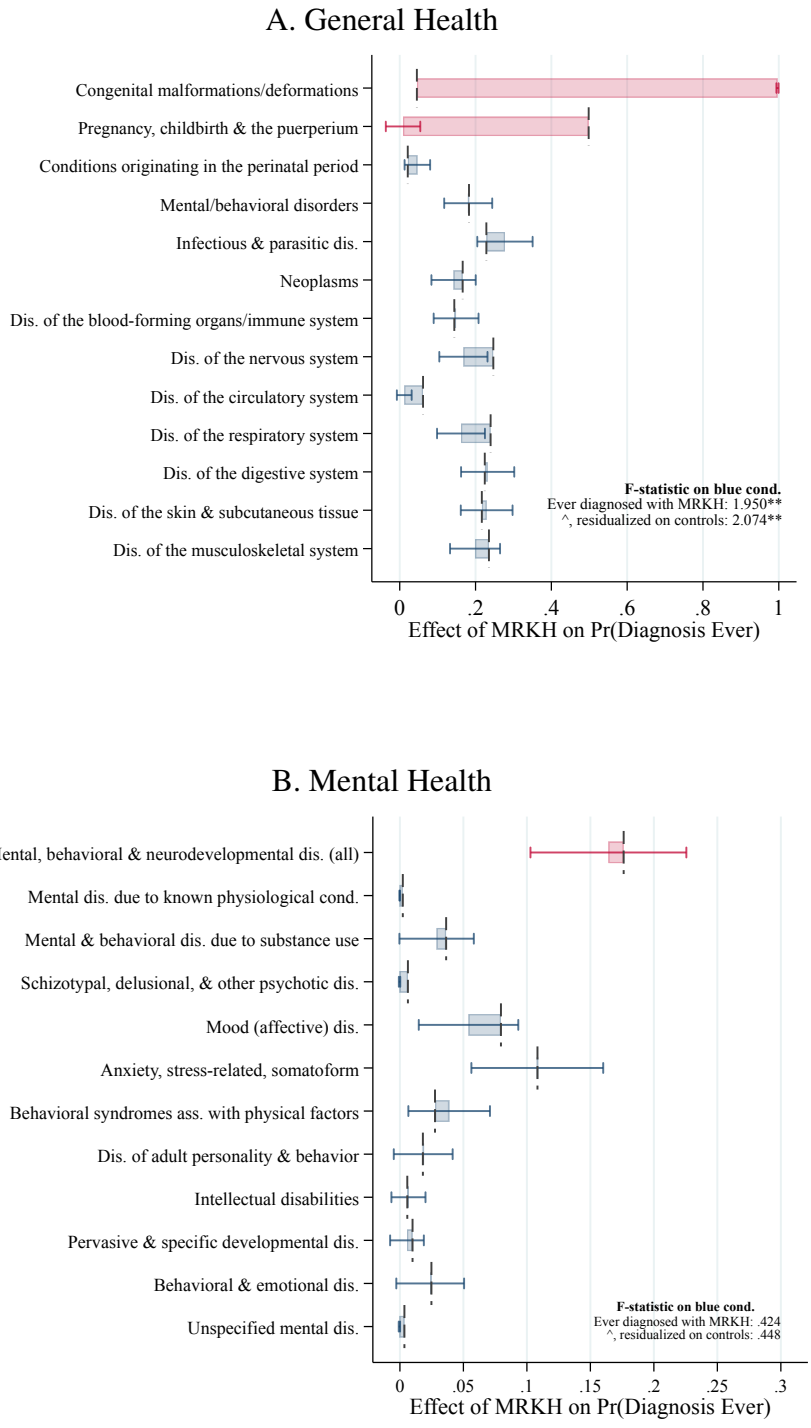


B. Family History



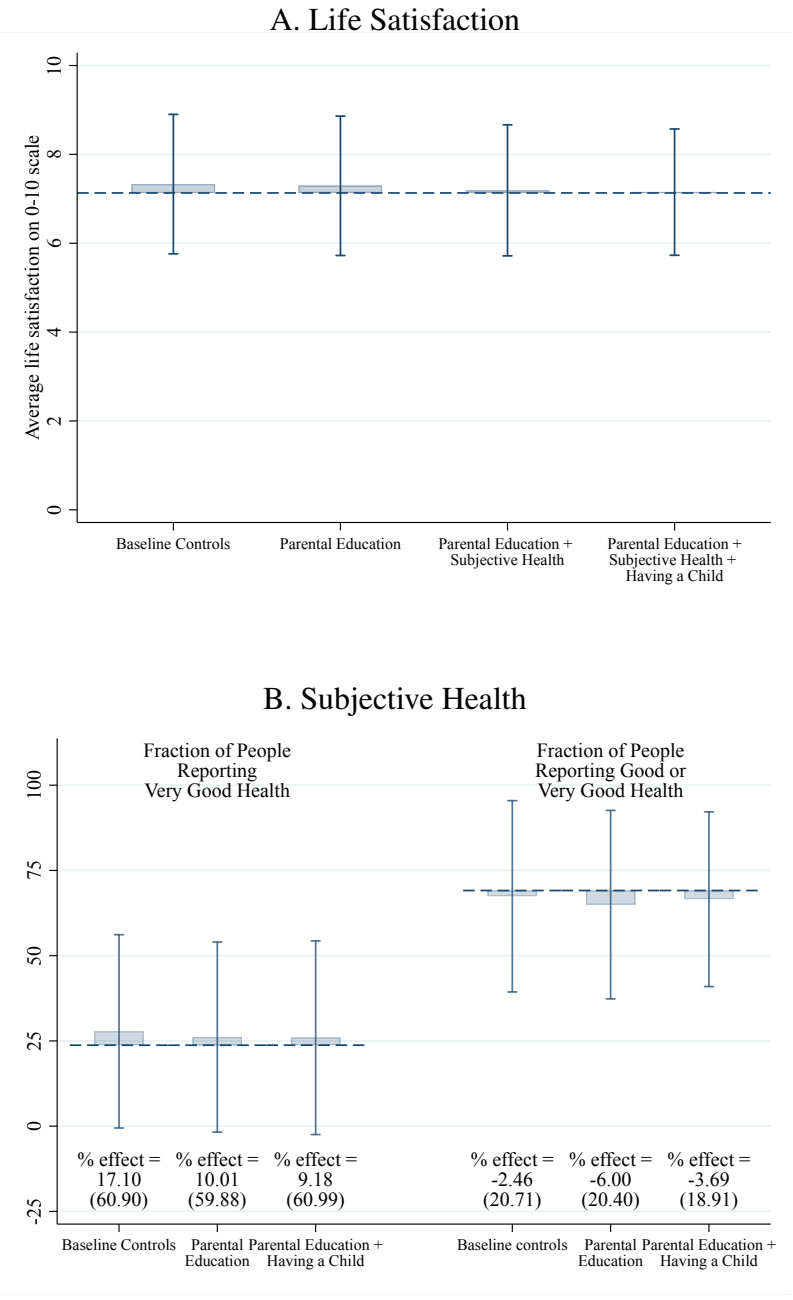
Notes: The figure tests for correlation between parental outcomes and the prevalence of the MRKH condition. Each bar corresponds to the estimated θ^p coefficient from specification (1) scaled by the average in the general population of women. Panel A focuses on education and labor market outcomes of parents (defined when the child is between 10 and 14 years old, that is prior to MRKH diagnosis). Panel B focuses on family structure and parental fertility history. Age at birth corresponds to the age of the parent when the child is born. Number of children is the total number of biological children that the parent had over their reproductive life.

FIGURE 4: **Impact of MRKH on the prevalence of health conditions**



Notes: The figure investigates correlation between general health and MRKH condition. In panel A, we group health diagnoses in 13 *chapters* –the highest level of aggregation in ICD codes. We regress separately 13 dummy variables for ever being diagnosed with these conditions on cohort fixed-effects and an indicator for ever being diagnosed with the MRKH syndrome using a linear probability model similar to specification (1). For each condition, the dashed line indicates the average prevalence in the general population, while the bar represents the estimated coefficient θ capturing the effect of MRKH (with its 95% confidence interval). In panel B, we disaggregate mental health conditions into eleven subcategories that we correlate with a dummy for ever being diagnosed with the MRKH condition.

FIGURE 5: **Life Satisfaction & Subjective Health**

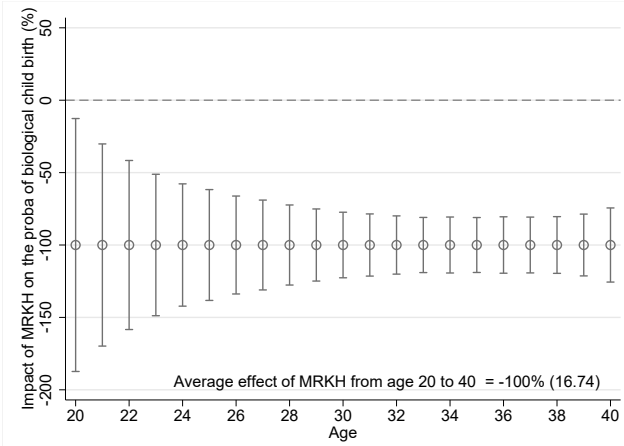


Notes: The figure investigates the relationship between MRKH and subjective health and life satisfaction using the survey that we disseminated in MRKH support groups pooled with the World Value Survey. The dashed line indicates the average prevalence in the general population, while the bar represents the estimated coefficient θ from specification (2) capturing the effect of MRKH (with its 95% confidence interval). The vector of baseline controls $\mathbb{X}$ includes country, age and cohort fixed-effects.

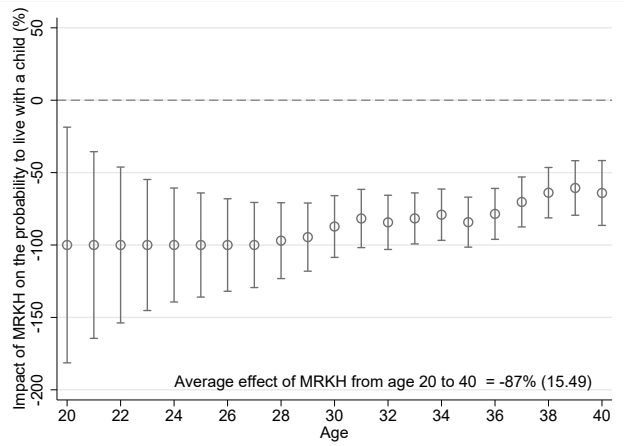
FIGURE 6: **MRKH Condition & Fertility**

A. Impact of MRKH on:

(i) Patient Record for Childbirth

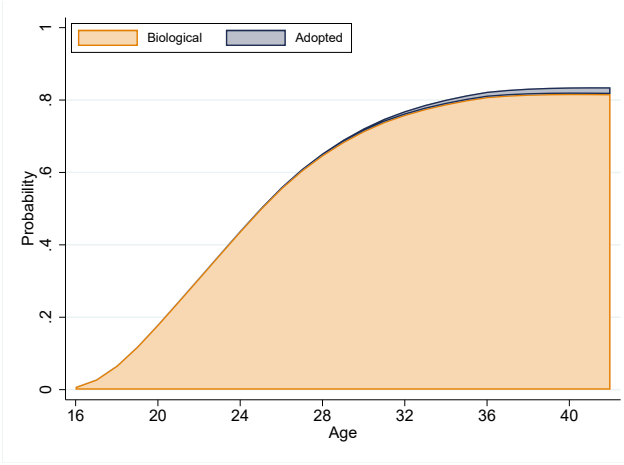


(ii) Probability to Live With a Child

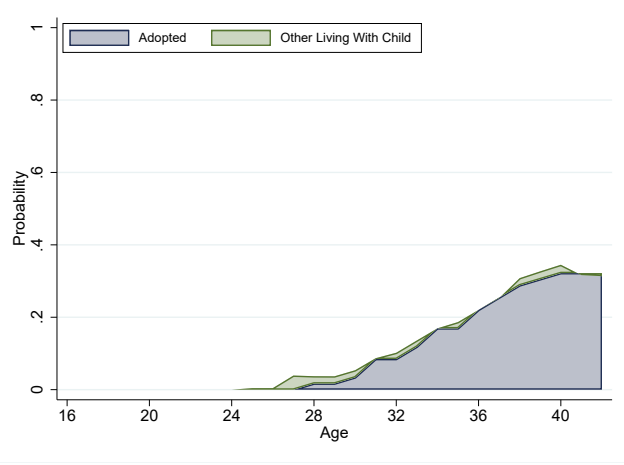


B. Decomposition of Probability to Live With a Child by Age

(i) Women in General Population



(ii) MRKH Women



Notes: The figure reports the fertility impacts of the MRKH condition. First, we look at patient records for child birth in the health registry data. In panel A.(i) we report, for each age a, the estimated coefficient θ^a from specification (3), scaled by α^a, the mean in the general population of women. The graph confirms that the MRKH condition correlates with 100% biological infertility in our sample. In our context and time frame, it is important to note there are no alternative pathways to having biological children for MRKH women. Surrogacy was (and is still) illegal in the Swedish healthcare system, while uterus transplants were not yet possible during the period covered by our data. In Panel A.(ii), we focus instead on the probability to be living with a child. There, we also find a strong negative association with the MRKH condition, but the correlation is slightly smaller in magnitude as the probability for MRKH women to live with children rises gradually after they reach their thirties, a pattern explained by adoptions. In panel B we decompose the probability to be living with a child according to three different arrangements: in orange, the probability to have biological children, in blue the probability to have exclusively adopted children, and in green the probability to have exclusively stepchildren. Panel B.(i) shows the results for the general population of women while panel B.(ii) shows the results among women with MRKH.

TABLE 2: **Effects of MRKH on Educational Achievements**

	(1) Ever diagnosed w/ MRKH	(2) Ever diagnosed w/ MRKH	(3) Ever diagnosed w/ MRKH	(4) Diagnosed before 18	(5) Ever diagnosed w/ MRKH
Proba. to graduate from high school	0.006	-0.008	-0.009	-0.002	-0.010
	(0.024)	(0.023)	(0.023)	(0.028)	(0.047)
Years of education post compulsory	0.113	0.020	0.068	0.096	0.123
	(0.178)	(0.147)	(0.147)	(0.187)	(0.302)
Proba. to graduate from university	0.019	0.023	0.025	0.060	0.075
	(0.041)	(0.035)	(0.035)	(0.046)	(0.081)
Controls					
Pre-diagnosis Controls		✓	✓	✓	✓
Health Controls			✓	✓	✓
Family F-E					✓

Notes: The table reports the estimated coefficient $\hat{\theta}$ from specification 4 for three different educational outcomes: the probability to graduate from high school (average level in the control population = 73%), the number of years of education post compulsory schooling (average level in the control population = 2.8 yrs), and the probability to graduate from university (average level in the control population = 24%). In column (2), the vector $\mathbb{X}_i^p$ of controls is introduced. It contains cohort fixed effects as well as a large set of pre-diagnosis characteristics capturing family background (parental education and income levels, parental marital and fertility history), childhood environment (county of birth, average income in municipality of residence during childhood) and pre-diagnosis educational attainment (grade percentile at age 15). In column (3), health controls are introduced: for each chapter of ICD9 and ICD10 codes, we create dummies for ever being diagnosed with diseases corresponding to that chapter. We then include all dummies in specification 6. Column (4) restricts attention to women whose age at MRKH diagnosis can be observed before 18 with certainty. In column (5), family fixed effects are introduced, so that women with MRKH are compared to their sisters.

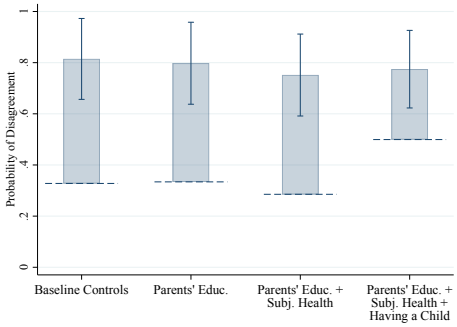
TABLE 3: **Effects of MRKH on Marriage Patterns and Partner Characteristics**

	(1) Ever diagnosed w/ MRKH	(2) Ever diagnosed w/ MRKH	(3) Ever diagnosed w/ MRKH	(4) Diagnosed before 18
	A. Marriage			
Ever married	0.015	0.045	0.062*	0.082*
(Mean = 35%)	(0.035)	(0.034)	(0.034)	(0.044)
Ever divorced	0.045*	0.052**	0.054**	0.062*
(Mean = 7%)	(0.025)	(0.025)	(0.025)	(0.034)
Age at First Marriage	-0.356	-0.508	-0.449	0.320
(Mean = 25.5 yrs old)	(0.573)	(0.558)	(0.555)	(0.889)
Average Marriage Duration	-3.140**	-3.349***	-3.340***	-2.435***
(Mean = 15 years)	(1.236)	(1.241)	(1.242)	(0.625)
	B. Partner Characteristics			
Proba. to be more educated	-0.008	-0.042	-0.030	0.005
than partner (Mean = 39%)	(0.073)	(0.070)	(0.070)	(0.089)
Proba. to earn strictly more	0.298***	0.277***	0.190***	0.298***
than partner (Mean = 17%)	(0.072)	(0.072)	(0.071)	(0.087)
Age difference w/ partner	-0.867	-1.484*	-1.373*	-1.280
(Mean = -3.6 years)	(0.795)	(0.772)	(0.773)	(1.097)
Pre-diagnosis Controls		✓	✓	✓
Health Controls			✓	✓

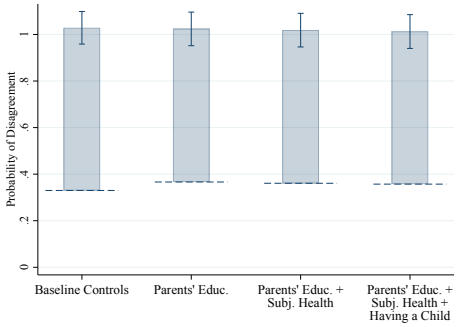
Notes: The table reports the estimated coefficient $\hat{\theta}$ from specification 4 for marriage and partnership outcomes. For the first three outcomes in Panel A, observations at the individual level, while they are at the partnership level for all other outcomes. The probability to be more educated corresponds to the probability that the woman has strictly more years of education than her partner. The probability to earn strictly more than a partner is defined as having strictly larger total earnings than one's partner over the life of the partnership. In column (2), the vector $\mathbb{X}_i^p$ of controls is introduced. It contains cohort fixed effects as well as a large set of pre-diagnosis characteristics capturing family background (parental education and income levels, parental marital and fertility history), childhood environment (county of birth, average income in municipality of residence during childhood) and pre-diagnosis educational attainment (grade percentile at age 15). In column (3), health controls are introduced: for each chapter of ICD9 and ICD10 codes, we create dummies for ever being diagnosed with diseases corresponding to that chapter. We then include all dummies in specification 6. Column (4) restricts attention to women whose age at MRKH diagnosis can be observed before 18 with certainty.

FIGURE 7: **Gender Norms & Beliefs**

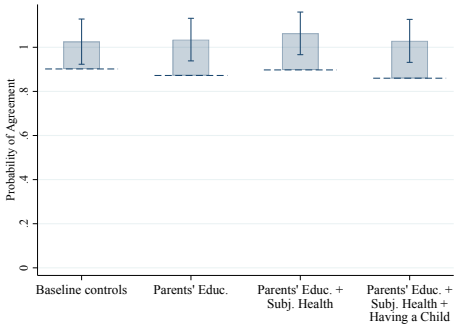
A. "*A preschool child is likely to suffer if mother works*"



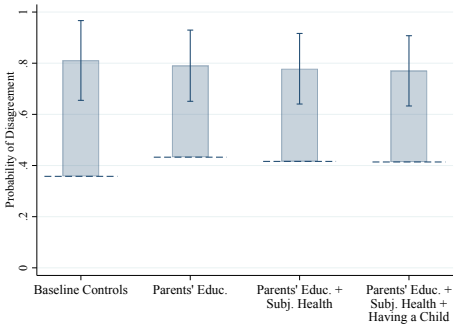
B. "*All in all, family life suffers when the woman has a full-time job*"



C. "*Both men and women should contribute to the household income*"

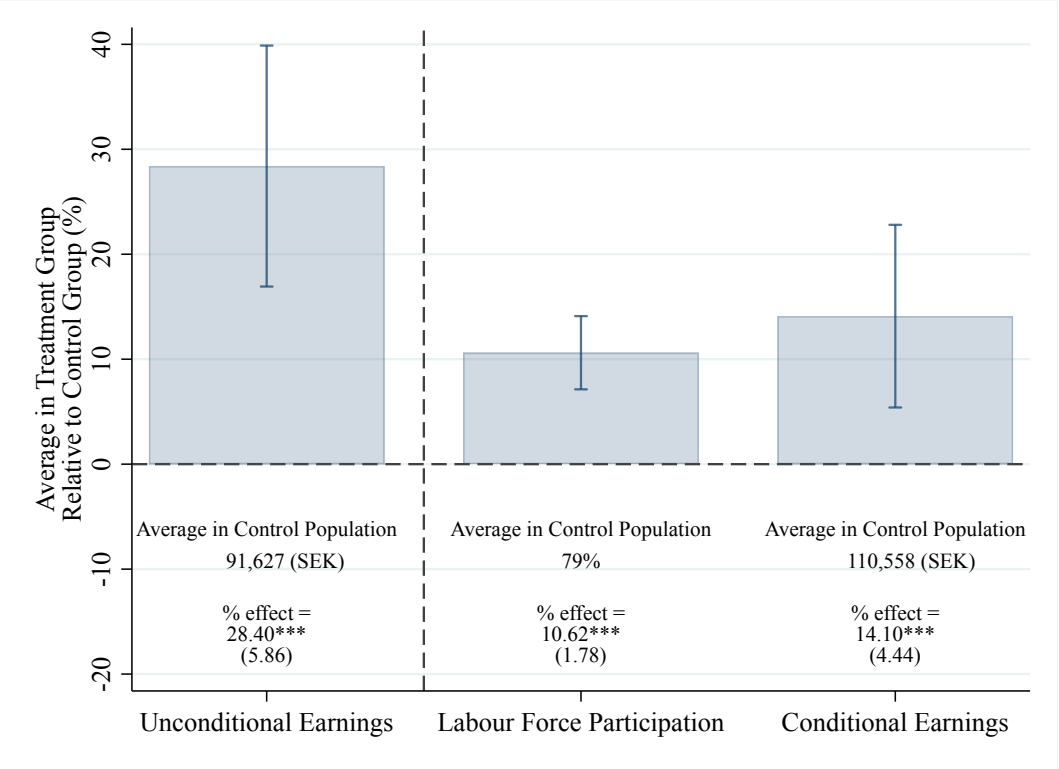


D. "*A man's job is to earn money; a woman's job is to look after the home*"



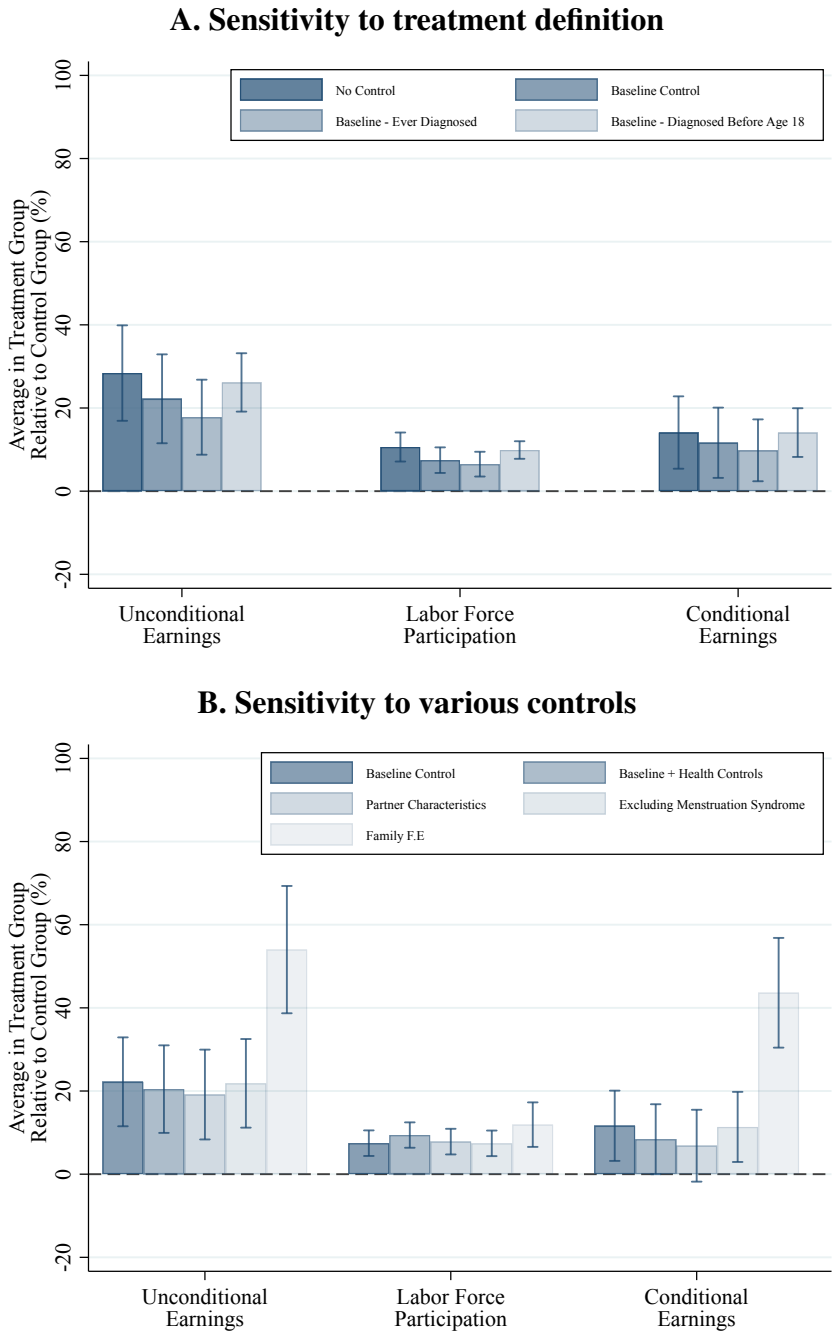
Notes: The figure reports the estimated effect of having the MRKH condition on the level of agreement with four statements on gender beliefs, using our survey pooled to global value surveys. In each panel, the blue bar depicts the estimated coefficient $\hat{\theta}$ from specification (5), capturing the effect of MRKH (with its 95% confidence interval). The dashed line corresponds to the counterfactual level of disagreement among MRKH women predicted from the model when omitting the contribution of $\hat{\theta}$. The vector of baseline controls $\mathbb{X}$ includes country, age and cohort fixed-effects.

FIGURE 8: **Effects of MRKH on Labor Market Achievements**



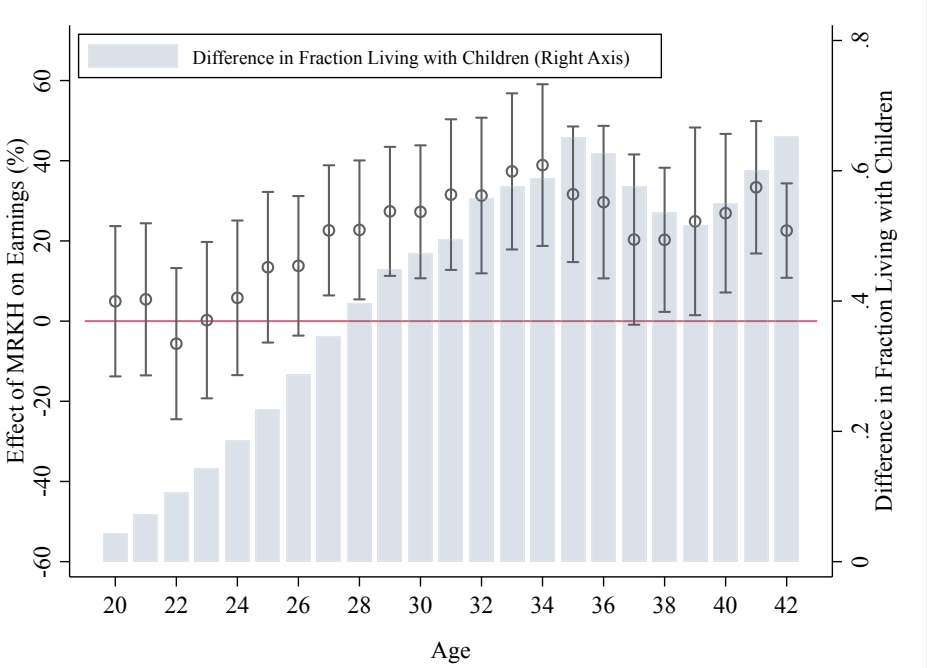
Notes: The figure reports the estimated effect of having the MRKH condition on total earnings from specification 6. The effect is then broken down between the extensive margin (labor force participation), and intensive margin (earnings conditional on participation) of labor supply. Specification 6 is run in levels, in order to include individuals with zero earnings. We then rescale the estimated coefficient $\hat{\theta}$ by the average predicted outcome among control women. Each bar corresponds to the rescaled estimated coefficient $\hat{\theta}$ from specification 6. It captures the percentage effect of having been diagnosed with MRKH on average labor market outcomes from age 16 to 42, the age range over which our sampled cohorts are observed. Standard errors are clustered at the individual level.

FIGURE 9: **Effect of MRKH on Labor Outcomes - Robustness**



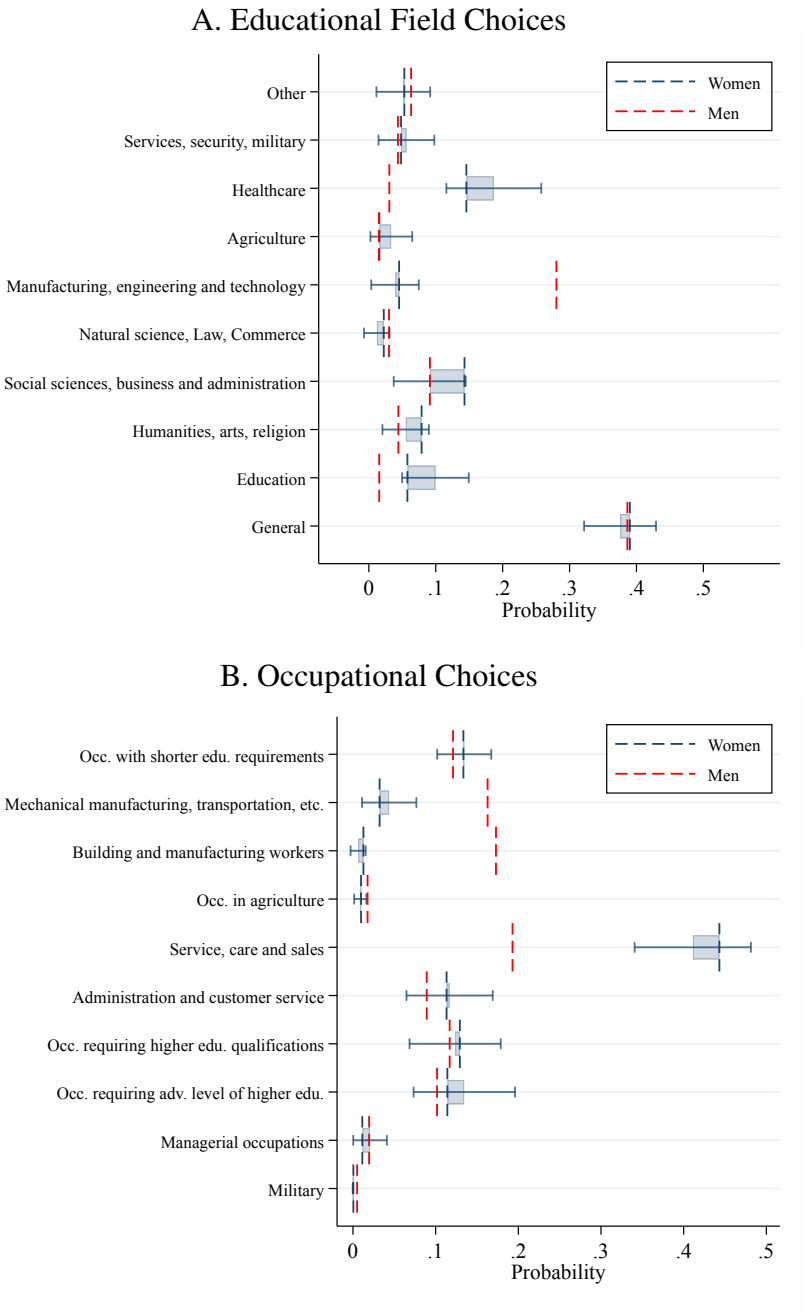
Notes: The figure reports the estimated effect of having the MRKH condition on total earnings from specification 6. The effect is then broken down between the extensive margin (labor force participation), and intensive margin (earnings conditional on participation) of labor supply. Specification 6 is run in levels, in order to include individuals with zero earnings. We then rescale the estimated coefficient $\hat{\theta}$ by the average predicted outcome among control women. Each bar corresponds to the rescaled estimated coefficient $\hat{\theta}$ from specification 6. It captures the percentage effect of having been diagnosed with MRKH on average labor market outcomes from age 16 to 42, the age range over which our sampled cohorts are observed. Standard errors are clustered at the individual level. In panel A, we compare results based on alternative definitions of the MRKH variable used in specification 6. We focus on women who have been diagnosed, who will ever be diagnosed, or who are diagnosed before age 20. In panel B, we introduce different controls to specification 6. First, the vector $\mathbb{X}_i^p$ of pre-diagnosis controls is introduced. It contains cohort fixed effects as well as a large set of pre-diagnosis characteristics capturing family background (parental education and income levels, parental marital and fertility history), childhood environment (county of birth, average income in municipality of residence during childhood) and pre-diagnosis educational attainment (grade percentile at age 15). Then, we also introduce health controls. For each chapter of ICD9 and ICD10 codes, we create dummies for ever being diagnosed with diseases corresponding to that chapter and we include all dummies in specification 6. In another specification, we exclude women with genitounitary diagnoses indicative of premenstrual syndromes or dysmenorrhea. We also investigate controlling for all marital outcomes and partner characteristics. Finally, we test for the inclusion of family fixed-effects, which amounts to comparing women with the MRKH condition to their sisters.

FIGURE 10: **Effects of MRKH on Earnings by Age**



Notes: The figure reports the estimated effect of having the MRKH condition on total earnings over the life cycle. We display the estimated coefficients θ_j from specification (7), which measure, at each age j, the difference in outcomes between women with MRKH and women in the general population. These coefficients are then scaled by the counterfactual outcome for women with MRKH, defined as the model prediction absent the MRKH indicator. We also report in the same panel the age profile of the difference in the fraction of women with children between the two groups. Standard errors are clustered at the individual level.

FIGURE 11: **Effect of MRKH on Early Career Behavior: Education and Occupation Choices**



Notes: The figure examines whether educational attainment and early career choices respond to primary infertility revealed by a diagnosis of MRKH syndrome. Panel A analyses broad educational field choices, classified according to the Swedish Standard Classification of Education. For each educational field, we estimate the probability of selecting that field using a specification analogous to equation (4). The dashed blue line indicates the average probability of choosing each field in the general female population, while the blue bars report the estimated coefficient θ, capturing the effect of MRKH on the probability of selecting that field. For comparison, the figure also includes a red dashed line showing the average probability of choosing each field among men. Panel B conducts a parallel analysis of broad occupational categories (one-digit occupations from the Swedish Standard Classification of Occupations (SSYK)) at labor market entry, defined as the first year in which an individual is observed working after having completed her highest level of education.

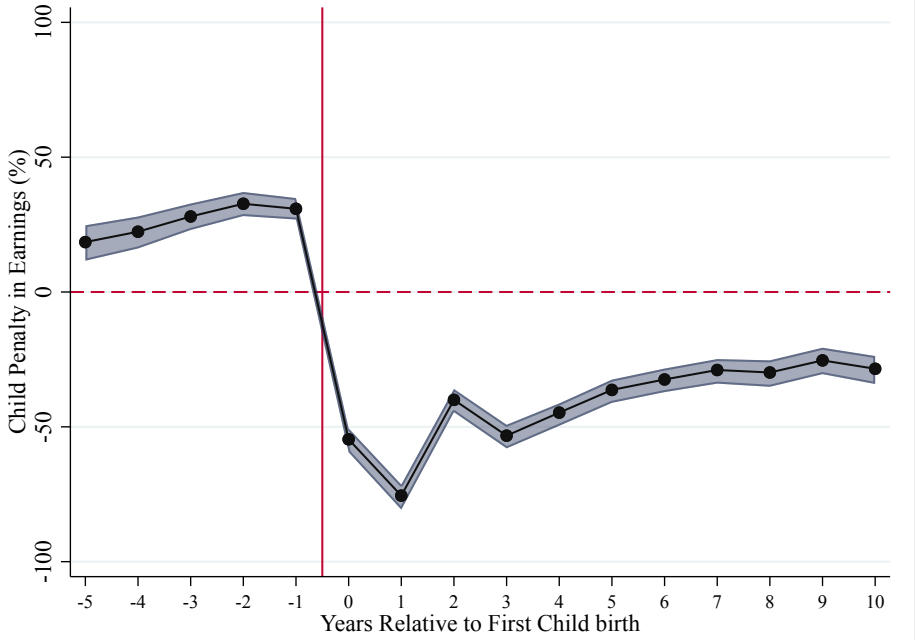
TABLE 4: **Effects of MRKH on Education and Early Career Choices**

	(1) Ever diagnosed w/ MRKH	(2) Ever diagnosed w/ MRKH	(3) Ever diagnosed w/ MRKH	(4) Diagnosed before 18
	A. Education choices			
Fraction in STEM	0.020	0.030	0.033	0.152 ***
(Mean = 39.7%)	(0.088)	(0.092)	(0.092)	(0.030)
Share of men in education field	-0.018	-0.017	-0.019	-0.023 ***
(Mean = 43.7%)	(0.016)	(0.016)	(0.016)	(0.006)
	B. Occupation choices			
Weekly hours in occupation	-0.198	-0.160	-0.145	-0.069
(Mean = 33.9 hrs)	(0.271)	(0.230)	(0.231)	(0.130)
Hourly wage in occupation	0.357	-0.439	-0.119	2.626 *
(Mean = 119.5 SEK)	(2.804)	(2.283)	(2.284)	(1.537)
Share of men in occupation	-0.007	-0.008	-0.008	-0.007
(Mean = 37.2%)	(0.013)	(0.013)	(0.013)	(0.006)
Controls				
Pre diagnosis		✓	✓	✓
Health Controls			✓	✓

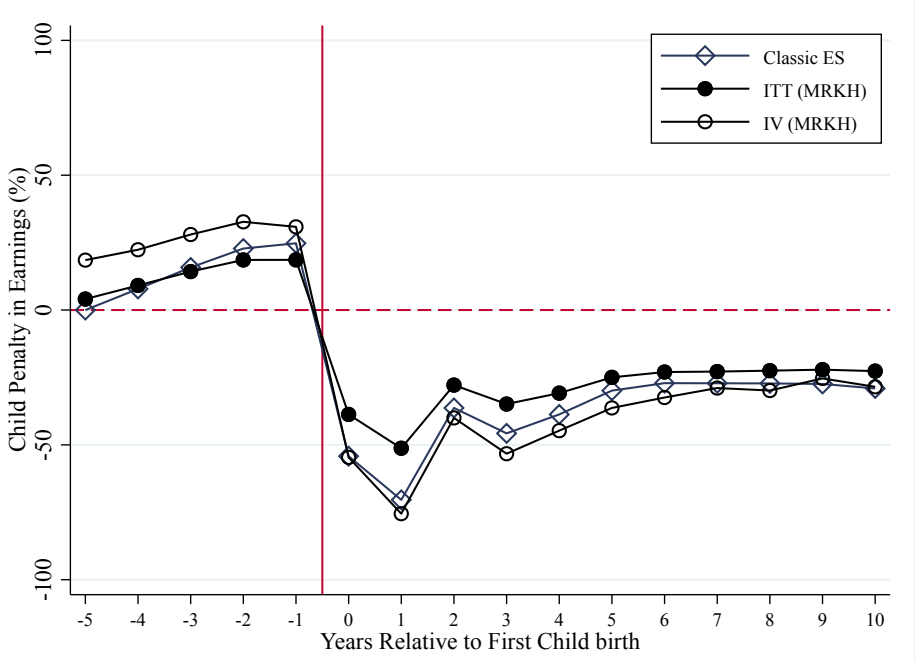
Notes: The table reports the estimated coefficient $\hat{\theta}$ from specification 4 for educational fields (at the two-digit SUN classification) in panel A and early occupation outcomes (at the three-digit SSYK occupational classification) in panel B. In column (2), the vector $\mathbb{X}_i^p$ of controls is introduced. It contains cohort fixed effects as well as a large set of pre-diagnosis characteristics capturing family background (parental education and income levels, parental marital and fertility history), childhood environment (county of birth, average income in municipality of residence during childhood) and pre-diagnosis educational attainment (grade percentile at age 15). In column (3), health controls are introduced: for each chapter of ICD9 and ICD10 codes, we create dummies for ever being diagnosed with diseases corresponding to that chapter. We then include all dummies in specification 6. Column (4) restricts attention to women whose age at MRKH diagnosis can be observed before 18 with certainty.

FIGURE 12: **Child penalty estimates: Labor Earnings**

A. IV estimates

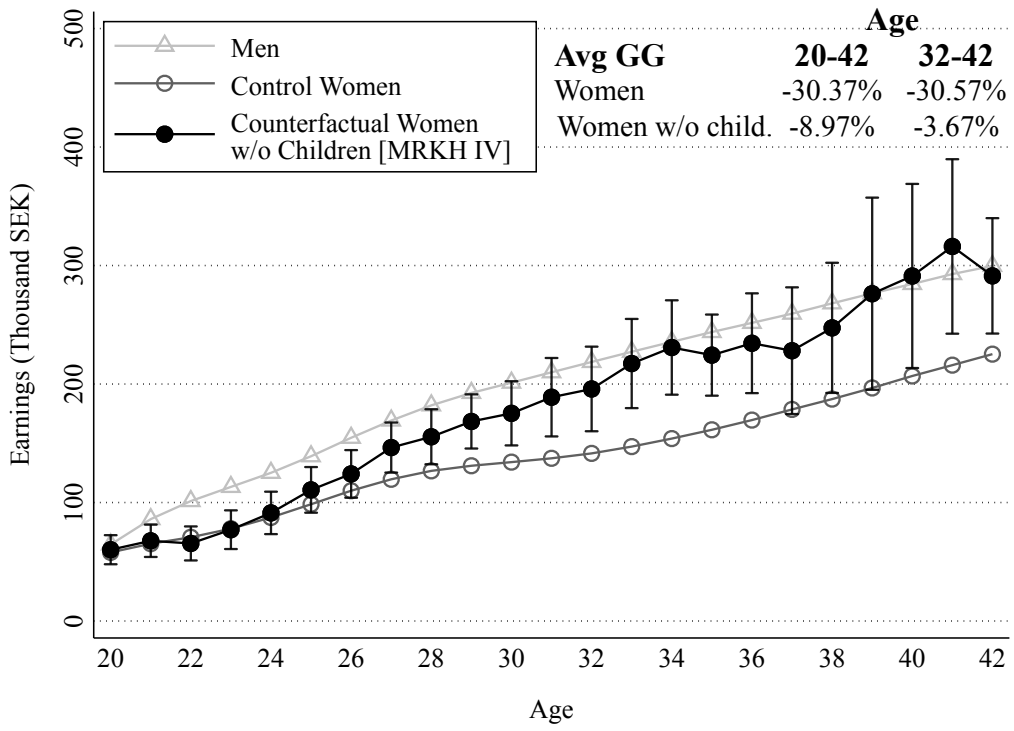


B. Comparison of ITT, IV and Event-Study Estimates



Notes: The figure reports the estimated child penalty estimates $\hat{P}_e = \sum_k \omega_k \cdot \hat{\Delta}_{e+k,k}$ for each event time e. Standard errors for P_e are computed via bootstrap stratified across age at first child and having been diagnosed with MRKH. The coefficients $\hat{\Delta}_{j,k}$, which correspond to the relative difference at age j between women whose first child arrives at age k and women who never have children are obtained from the IV specification 10, where we instrument the indicator $\mathbb{1}[F_i = 0]$ using an indicator for being diagnosed with MRKH syndrome. See text for details on the identification framework. Panel B compares these child penalty estimates to those obtained from a standard event-study specification and from intent-to-treat estimates where, instead of instrumenting the indicator $\mathbb{1}[F_i = 0]$, we simply replace it by an indicator for being diagnosed with MRKH syndrome.

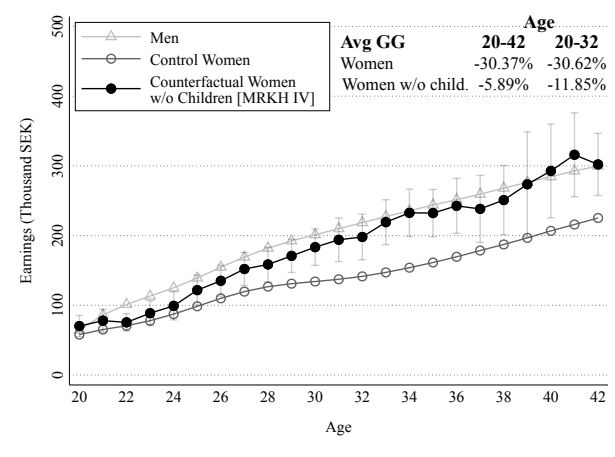
FIGURE 13: **Earnings profiles of men, women and counterfactual women absent children**



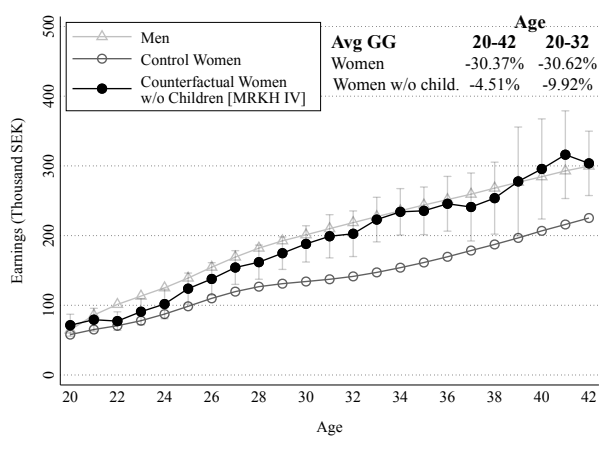
Notes: The figure reports the age-specific earnings trajectories of all men and women in the population, and the counterfactual profile of women absent children. These earnings trajectories are computed from specification (11) and are residualized on the vector $\mathbb{X}_i^p$. It contains cohort fixed effects as well as a large set of pre-diagnosis characteristics capturing family background (parental education and income levels, parental marital and fertility history), childhood environment (county of birth and average income in the municipality of residence during childhood), and pre-diagnosis educational attainment (grade percentile at age 15). The predicted earnings profile of women absent children is obtained from instrumenting the indicator $\mathbb{1}[F_i = 0]$ (never having children) with an indicator for MRKH diagnosis.This counterfactual profile is therefore strongly anchored in the earnings trajectory of women with MRKH, corrected for the fact that a small fraction of them become parents through adoption (see Figure 14, Panel A). 95% confidence intervals are computed from standard errors clustered at the individual level. Average gender gaps for women, and counterfactual women absent children are also displayed for different age groups.

FIGURE 15: **Earnings profiles of men vs counterfactual women without children: role of education and early career**

A. Controlling for education



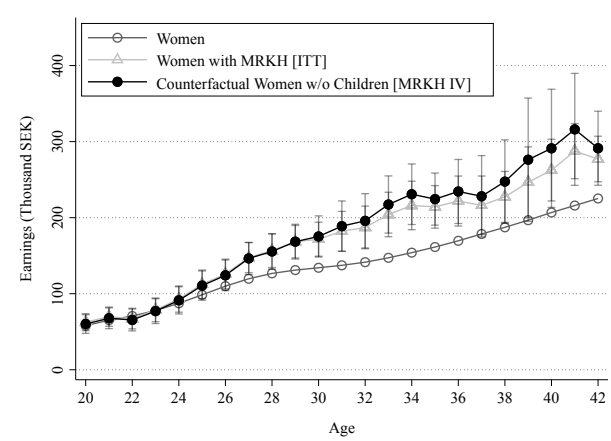
B. Controlling for education & early career choices



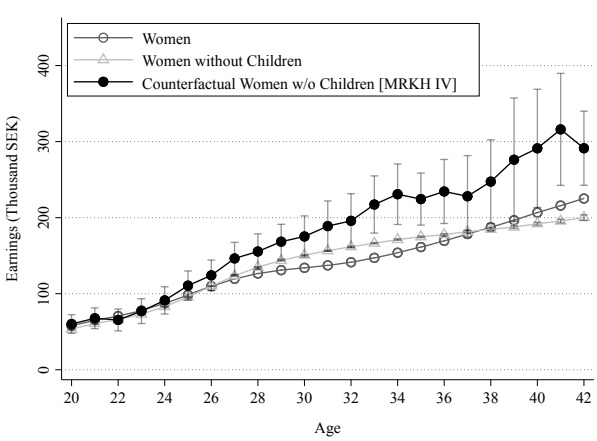
Notes: The figure explores the role of education and early career choices in explaining the gender differences in early earnings trajectories even absent children. Both panels report the age-specific earnings trajectories of all men and women in the population, and the counterfactual profile of women absent children as in Figure 13. But in panel A, we add education controls (a dummy for completed education, years of education, and field of education) in specification (11). The counterfactual profile of women absent children in this panel is then constructed by giving to these counterfactual women the same education as men at each age. In panel B, we also add first occupation controls (3-digit occupation codes fixed-effects). The counterfactual profile of women absent children in this panel is constructed by giving to these counterfactual women the same education and distribution of first occupation as men. 95% confidence intervals are computed from standard errors clustered at the individual level. Average gender gaps for women, and counterfactual women absent children are also displayed for different age groups.

FIGURE 14: **Counterfactual earnings profiles absent children: comparing MRKH women vs all women without children**

A. Women with MRKH & Counterfactual absent children



B. Women with and without children in general population



Notes: Panel A reports the observed earnings profile of women with MRKH alongside the IV-based counterfactual profile of women absent children. The counterfactual is plotted with 95% confidence intervals (standard errors clustered at the individual level). The ITT estimates are obtained by replacing the indicator $\mathbb{1}[F_i = 0]$ (never having children) with an indicator for MRKH diagnosis in specification (11). Panel B reports the observed earnings profile of women with MRKH alongside the IV-based counterfactual profile of women absent children and the earnings trajectory of women who never have children in the data. The estimates for women who never have children come from specification (11), but without instrumenting the indicator $\mathbb{1}[F_i = 0]$. The vector $\mathbb{X}_i^p$ of controls is included. It contains cohort fixed effects as well as a large set of pre-diagnosis characteristics capturing family background (parental education and income levels, parental marital and fertility history), childhood environment (county of birth and average income in the municipality of residence during childhood), and pre-diagnosis educational attainment (grade percentile at age 15).

APPENDIX

for "Gender Without Kids"

by Camille LANDAIS, Petter LUNDBORG, Tatiana PAZEM,
Erik PLUG and Johan VIKSTROM

Contents

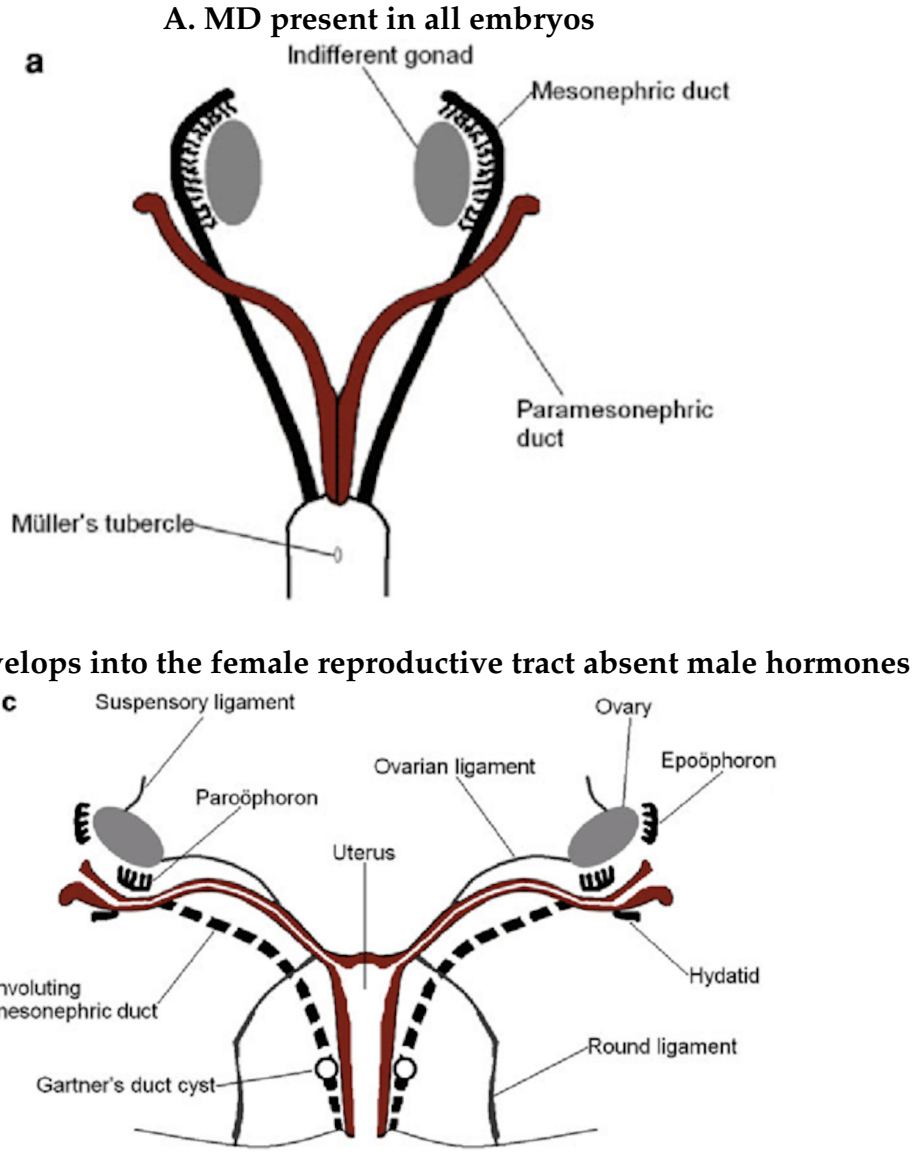
I. Further Details on MRKH Condition

I.1. Embryology of MRKH condition

The female internal reproductive tract develops from the Müllerian (paramesonephric) ducts, a pair of embryonic structures arising as bilateral invaginations of the coelomic epithelium at around five weeks post-gestation. Closely guided by the adjacent Wolffian ducts, the Müllerian ducts extend caudally toward the urogenital sinus. In the absence of anti-Müllerian hormone — which drives Müllerian duct regression in male embryos — they persist and differentiate. Between approximately the eighth and twelfth weeks of gestation, this process gives rise to the fallopian tubes, uterus, cervix, and upper two-thirds of the vagina (Herlin (2024); Mullen and Behringer (2014)). The lower third of the vagina and the external genitalia develop separately from the urogenital sinus, and the ovaries arise from an entirely distinct pathway in the intermediate mesoderm. Both are therefore unaffected when Müllerian duct development fails.

MRKH syndrome arises from an arrest of normal Müllerian duct development between the fifth and eighth weeks of gestation, resulting in agenesis or hypoplasia of the structures the ducts would otherwise form. Rather than undergoing their normal process of caudal elongation, midline fusion, and differentiation into the uterus, cervix, and upper vagina, the Müllerian ducts in affected individuals fail to develop, producing the defining anatomical features of the condition: congenital absence or severe hypoplasia of the uterus, cervix, and upper vagina. Current evidence points toward a heterogeneous genetic aetiology, with disruptions to multiple developmental pathways implicated, including genes such as WNT4, LHX1, GREB1L, TBX6, and members of the HOXA cluster (Herlin (2024); Mullen and Behringer (2014)). Because the ovaries, external genitalia, and secondary sexual characteristics develop independently of the Müllerian ducts, women with MRKH type I are phenotypically indistinguishable from the general population in all outwardly observable respects. Primary amenorrhea — the failure to begin menstruation — is thus the first clinical indication of the syndrome, typically presenting around age 15, and leading to diagnosis most commonly between ages 15 and 18 (Herlin et al. (2016); Herlin et al. (2020)).

Figure I.1: **Differentiation of the Müllerian ducts**



Notes: The figure provides a schematic representation of Müllerian duct differentiation during embryonic development. Panel A illustrates the indifferent stage, present in all embryos, in which both the mesonephric (Wolffian) and paramesonephric (Müllerian) ducts coexist alongside the indifferent gonad. Panel B illustrates the outcome in female embryos in the absence of male hormones: the Müllerian ducts persist and differentiate into the female internal reproductive tract, giving rise to the uterus and associated structures, while the mesonephric ducts involute. In MRKH syndrome, the developmental process depicted in Panel B is arrested, resulting in agenesis or hypoplasia of the uterus, cervix, and upper vagina.

I.2. Aetiology of MRKH condition

The aetiology of MRKH syndrome remains incompletely understood. Current evidence supports a heterogeneous origin, with both genetic and non-genetic factors implicated to

varying degrees across patients.

A substantial body of clinical evidence points to a heritable component in at least a subset of cases. Elevated rates of the condition have been documented among relatives, siblings, and other family members of affected women across multiple independent cohorts. This familial clustering is particularly pronounced when uterovaginal aplasia co-occurs within families alongside renal malformations — a pattern reflecting the shared embryological origin of both organ systems in the intermediate mesoderm. The inheritance pattern observed in such families — variable expression, incomplete penetrance, and transmission through both parental lines — is most consistent with autosomal dominant inheritance with incomplete penetrance and variable expressivity.

Despite this evidence for familial aggregation, the majority of MRKH cases are sporadic, arising in individuals with no family history of the condition. Two lines of evidence are particularly informative in this regard. First, multiple reports have documented monozygotic twin pairs discordant for the MRKH phenotype: since identical twins share virtually the same germline sequence, phenotypic discordance implies that the condition cannot be explained by germline genetic variation alone, and points instead toward post-zygotic events — including somatic mutations, tissue-specific mosaicism, or epigenetic modifications — as contributors in at least some individuals . Second, surveys of surrogate pregnancies involving the biological offspring of women with MRKH have generally found no genital malformations in female children, consistent with low rates of Mendelian transmission of the full phenotype (Fontana et al. (2016)). Teratogenic exposures during fetal development — including thalidomide, diethylstilbestrol, organotins, and phthalates — have been proposed as candidate non-genetic mechanisms, but systematic analyses of maternal pregnancy histories have not established a reliable association with any specific exposure (Fontana et al. (2016); Herlin et al. (2020)).

Taken together, the evidence is most consistent with a multifactorial aetiology: a subset of cases involve autosomal dominant susceptibility variants transmitted with incomplete penetrance, while the majority arise through mechanisms that likely include de novo mutations, post-zygotic events, or multifactorial interactions between genetic predisposition and developmental environment. Since the condition is predominantly sporadic, not linked to any identified parental behaviour or socioeconomic characteristic, and consistent with a largely random distribution in the general population, MRKH type I constitutes a plausibly exogenous shock to fertility.

I.3. MRKH diagnosis and treatment

MRKH syndrome is typically diagnosed at puberty, most commonly between ages 15 and 18, when primary amenorrhea prompts medical investigation despite otherwise normal pubertal development. In some cases, cyclic pelvic pain arising from ovulation in the absence of menstrual outflow provides an additional presenting complaint. Because the ovaries are unaffected and external genitalia appear entirely normal, the condition is not detectable prior to puberty in the absence of targeted screening. As documented in Section XX of the main text, the median age at first diagnosis in our Swedish registry data is 18, and survey evidence indicates an average age at diagnosis of 16.5, with 91% of respondents diagnosed before age 20.

The diagnostic workup involves a combination of clinical examination, imaging, and laboratory analysis. Physical examination confirms normal secondary sexual characteristics and identifies the shortened or absent vaginal canal. Pelvic ultrasound is typically the first-line imaging modality, confirming absence or hypoplasia of the uterus and the presence of normal ovaries; pelvic MRI is considered the gold standard for definitive diagnosis, providing superior resolution of uterovaginal anatomy, characterisation of rudimentary uterine buds, and assessment of associated extragenital anomalies, particularly renal malformations present in up to 40% of type II cases (Herlin et al. (2020)). Hormonal analysis — including FSH, LH, oestradiol, and testosterone — confirms normal ovarian function and excludes endocrine causes of amenorrhea and hyperandrogenism. Karyotyping establishes the 46,XX genotype and rules out differences of sex development involving the Y chromosome, most importantly androgen insensitivity syndrome, which presents similarly but with a 46,XY karyotype.

Clinical management focuses on enabling normal sexual function through creation or elongation of a functional vagina, deferred until the patient is emotionally mature and always preceded by counselling. Non-surgical vaginal dilation is the recommended first-line approach (Herlin et al. (2020)). The Frank method — progressive self-application of graduated dilators to the vaginal dimple — and coital dilation both achieve vaginal elongation through sustained mechanical pressure on existing tissue, and are associated with substantially lower complication rates than surgical alternatives. In our survey data, 83% of respondents with MRKH type I report having undergone dilation or vaginoplasty, allowing for normal sexual intercourse.

Surgical vaginoplasty is reserved for cases where dilation is unsuccessful or not feasible. The most widely used procedure, the McIndoe technique, involves lining a surgically created rectovesical space with a split-thickness skin graft; alternative approaches include

the laparoscopic Vecchietti procedure, which uses traction to invaginate the vaginal dimple, and the Davydov procedure, which uses a peritoneal flap (Herlin et al. (2020)). All techniques can achieve functional outcomes for the majority of patients, though complication profiles and operative complexity differ considerably.

I.4. History of MRKH condition

Reports of congenital defects of the female reproductive organs extend across more than two millennia. Among the earliest notable accounts is that of Hippocrates (c. 460–377 B.C.), who in On the Nature of Women described anomalies of the female reproductive system consistent with what would later be recognised as uterovaginal aplasia (Patnaik et al. (2015)). The medieval Arab physician Albucasis similarly documented cases of absent or obstructed vaginal structures. The first anatomically precise description is attributed to the Italian anatomist Realdo Colombo, who in De Re Anatomica (1562) described a woman with no womb or vagina who complained of pain during coitus — an entity he termed vulva rara (Herlin et al. (2020)).

The modern definition of the condition emerged from four independent contributions spanning approximately 130 years (Herlin et al. (2020); Patnaik et al. (2015)). In 1829, German anatomist August Franz Josef Karl Mayer published a typology of uterine duplication anomalies based on post-mortem observations, describing cases of a rudimentary divided uterus — uterus bipartitus — alongside other congenital malformations of the female genital tract. Austrian anatomist Carl von Rokitansky independently enriched this picture in 1838, reporting autopsy cases of women with a shortened blind-ending vagina and rudimentary uterine remnants alongside entirely normal ovaries and fallopian tubes — the anatomical combination he described as uterus bipartitus solidus cum vagina solida. His original illustration of this morphology remains a landmark in the condition's history. Both Mayer and Rokitansky worked exclusively from post-mortem specimens. German gynaecologist Hermann Küster was the first, in 1910, to describe the condition in a living patient — surgically removing her pain-causing uterine remnants — and to document its association with extragenital anomalies including skeletal deformations and urological defects such as renal ectopy and vaginal agenesis (Patnaik et al. (2015)). Finally, Swiss gynaecologist Georges André Hauser, in a series of papers published in 1961 reviewing 21 cases, introduced sex-chromatin analysis to demonstrate that affected women had a normal female karyotype, decisively distinguishing the condition from testicular feminisation syndrome (androgen insensitivity syndrome) and completing its clinical and chromosomal definition (Herlin (2024); Herlin et al. (2020)). It was in

these 1961 papers that the condition first received the name "Mayer-Rokitansky-Küster syndrome", subsequently extended to include Hauser's name.

These four contributions all described isolated uterovaginal agenesis — what contemporary literature classifies as type I MRKH. The formal recognition of an atypical form associated with renal, skeletal, auditory, and cardiac anomalies (type II MRKH, or the MURCS association) came in 1977, when Schmid-Tannwald and Hauser described seven cases with associated renal malformations, establishing the two-subtype classification used today (Herlin et al. (2020)).

Contemporary research has shifted from clinical description toward aetiological investigation, enabled by high-resolution genomic technologies including chromosomal microarray analysis and whole-exome and whole-genome sequencing. These approaches have identified a growing number of candidate genetic variants, including recurrent deletions at chromosomal regions 17q12 and 16p11.2, and variants in genes such as GREB1L, LHX1, TBX6, and PAX8 (Herlin (2024); Fontana et al. (2016)). On the clinical side, the first successful uterus transplantation in a woman with MRKH syndrome — performed by a Swedish team and resulting in a live birth in Gothenburg in September 2014 — has opened a new frontier in fertility treatment for the condition, though one that postdates the cohorts studied in the present paper. The precise aetiology of MRKH in the majority of cases nonetheless remains unknown, and understanding its genetic architecture and developing effective treatments continue to be the central focus of current research.

I.5. Identification of women with MRKH Type II Syndrome

As described in Section 2.3 of the main text, our analysis is restricted to women diagnosed with MRKH type I — that is, isolated uterovaginal aplasia with no associated extragenital malformations. Women with MRKH type II are systematically identified and excluded from the sample. This section details the criteria and ICD codes used to implement that exclusion.

MRKH type II, also referred to as the MURCS association (Müllerian duct aplasia, Renal dysplasia, and Cervicothoracic Somite anomalies), is defined by the co-occurrence of uterovaginal aplasia with one or more extragenital malformations (Herlin et al. (2016)). The most frequently observed associated anomalies are renal malformations — including unilateral renal agenesis, renal ectopia, and horseshoe kidney — which are present in approximately 34% of atypical cases, followed by skeletal malformations, predominantly vertebral anomalies such as Klippel-Feil syndrome, scoliosis, and fused cervical vertebrae, present in approximately 13% of cases (Herlin et al. (2016)). Cardiac defects and

hearing impairment, while less common, also fall within the MURCS phenotypic spectrum. Importantly, these extragenital anomalies share their embryological origin with the Müllerian structures in the intermediate mesoderm, which explains their co-occurrence with uterovaginal aplasia and their relevance to the type I/type II classification. As noted in Section I.3, familial clustering of MRKH is more common among type II cases, suggesting a stronger genetic component in this subgroup.

To exclude women with type II syndrome from our sample, we identify all individuals in the inpatient and outpatient health registry data who carry, in addition to the MRKH diagnosis codes (ICD-10: Q51.0, Q52.0; ICD-9: 752D, 752E), any diagnosis corresponding to the extragenital conditions listed by the American National Organisation for Rare Disorders and Herlin et al. (2016) as indicative of the MURCS association. These conditions and their corresponding ICD-9 and ICD-10 codes are reported in Table I.1 They span four categories: hearing loss (including conductive and sensorineural hearing loss, structural abnormalities of the middle ear, and genital renal ear syndrome); heart malformations (including atrial septal defects, pulmonary valvular stenosis, and tetralogy of Fallot); kidney conditions (including renal dysplasia, unilateral renal agenesis, renal ectopia, and hydronephrosis); and skeletal malformations (including vertebral anomalies such as Klippel-Feil syndrome and scoliosis, as well as a range of cranial and limb defects). A woman is classified as MRKH type II and excluded from the analysis if she carries any diagnosis belonging to any of these four categories, regardless of whether the extragenital condition was recorded before or after the MRKH diagnosis itself.

This approach is consistent with that adopted in Herlin et al. (2016), the only other population-wide registry study of MRKH prevalence, which classified patients as typical or atypical based on the presence or absence of renal, skeletal, cardiac, and auditory anomalies in their medical records. Applying this procedure to our Swedish registry data, we identify 259 women with an MRKH diagnosis, of whom 107 (37.1%) carry at least one extragenital diagnosis indicative of type II syndrome and are excluded. The remaining 152 women, who present with none of these conditions, constitute our analytical sample of MRKH type I. As reported in Table 1 of the main text, the distribution of extragenital anomalies among excluded women — hearing loss (9.3%), heart defects (7.3%), kidney anomalies (16.8%), and skeletal malformations (16.6%) — is broadly consistent with the Danish prevalence study, providing reassurance about the validity of the registry-based classification procedure.

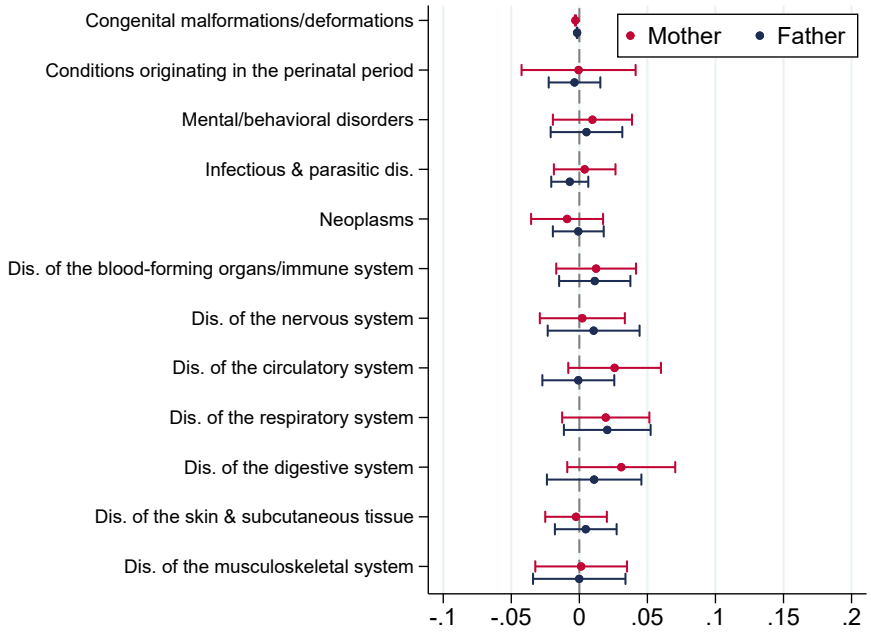
Table I.1: MRKH-related conditions and associated ICD codes

MRKH-related conditions	ICD-9 codes	ICD-10 codes
Hear loss		
- conductive hearing loss		
- structural abnormalities of the middle ear	380-389	H60-H95
- sensorineural hearing loss	744	Q16-Q17
- malformed (dysplastic)		
- Genital renal ear syndrome		
Heart malformations	393-398	I00-I52
- atrial septal defects (hole in the heart between the two upper chambers of the heart)	401-405	Q20-Q21
- pulmonary valvular stenosis: narrowing of the pulmonary valve	410-414	Q23-Q24
- tetralogy of Fallot	420-429	R00-R01
	785	
Kidney conditions		
- renal dysphasia: failure of the kidneys to develop properly		
- unilateral renal agenesis: absence of a kidney	401-405	
- renal dysplasia: malformation of one or the two kidneys	580-589	I10-I15
- underdeveloped (hypoplastic) kidneys	590-599	N00-N29
- renal ectopia: improper positioning within the body of one of both kidneys	753.0-753.4	
- kidney stones	788	
- urinary tract infections		
- hydronephrosis: abnormal accumulation of urine in the kidney due to obstruction		
Skeletal malformations		
- improper development (dysplasia) of cervical and thoracic vertebrae		
- missing and/or fused cervical vertebrae		
- limited neck motion		
- abnormally low hairline (Klippel-Feil syndrome)		M40-M54
- asymmetric, fused or wedged vertebrae	736-738	M95
- malformed or missing ribs	744	Q18
- scoliosis: abnormal sideways curvature of the spine	749	Q35-Q37
- elevation of the shoulder blade (Sprengel deformity)	755-756	Q60-Q61
- small jaw (micrognathia)	781.5	Q63
- cleft lip/palate		Q67-Q71
- underdevelopment of one side of the face causing facial asymmetry		Q76
- absence of a portion of one or more fingers or toes (ectrodactyly)		
- webbing of the fingers or toes (syndactyly)		
- duplicated thumb		
- absence of the long, thin bone of the forearm (absent radius)		

Notes: The table reports the extragenital conditions and their corresponding ICD-9 and ICD-10 codes used to identify and exclude women with MRKH type II from the analytical sample. The conditions are organized into four categories: hearing loss, heart malformations, kidney conditions, and skeletal malformations. A woman is classified as MRKH type II and excluded from the analysis if she carries any diagnosis belonging to any of these four categories in the inpatient or outpatient health registry data, regardless of whether the extragenital condition was recorded before or after the MRKH diagnosis itself.

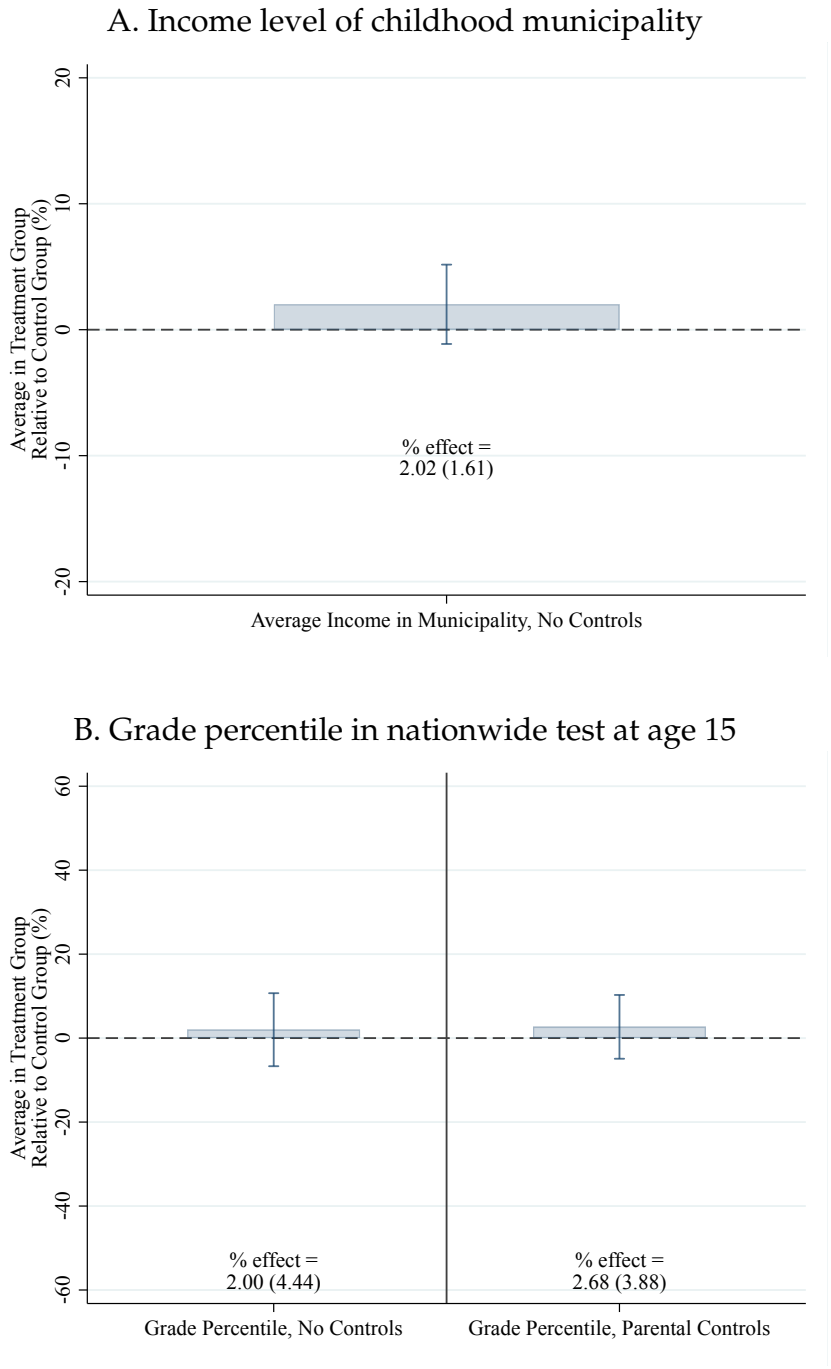
II. Additional Results from the Administrative Sample

Figure II.1: **Correlation between MRKH and parental health**



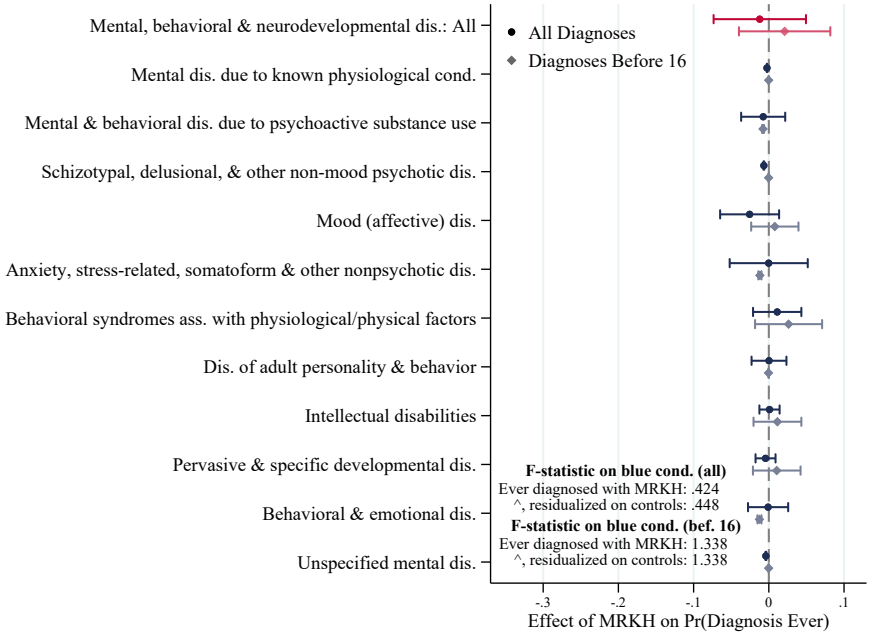
Notes: The figure investigates the health of parents of women with MRKH relative to parents of control women. For each of the 12 broad diagnostic categories shown on the y-axis, we regress a dummy for the parent having ever received a diagnosis in that category on an indicator for the child having MRKH, controlling for cohort fixed effects. Parent health is measured when the child was between 10 and 14 years old. One observation per individual is used. 95% confidence intervals are shown.

Figure II.2: **Childhood environment & early education outcomes**



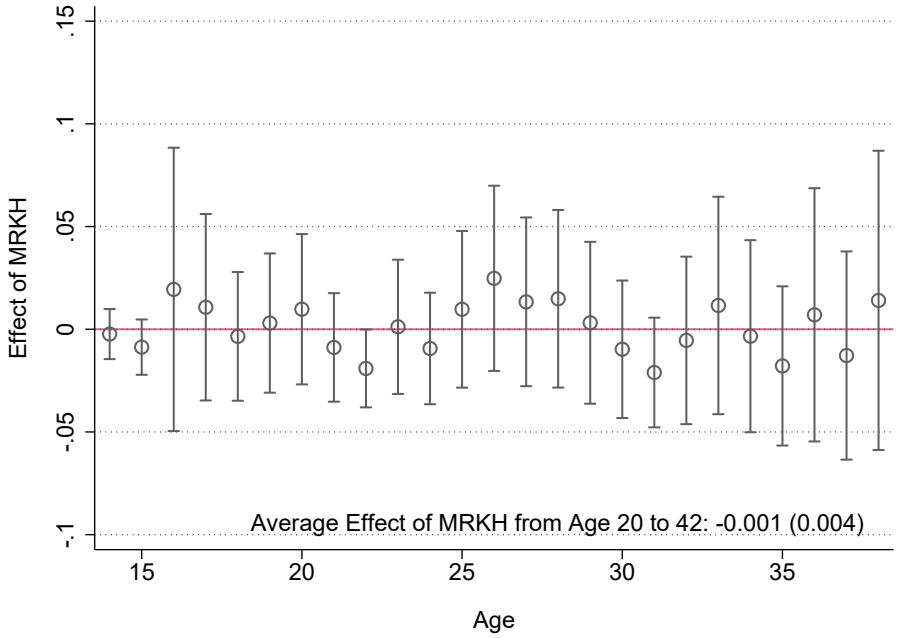
Notes: The figure investigates differences in childhood environment and pre-diagnosis educational attainment between women with MRKH and control women. Panel A shows whether women with MRKH grew up in different economic environments relative to control women. For each individual, we compute the average income in the municipality of residence between ages 10 and 14, and regress this on an indicator for ever having been diagnosed with MRKH, controlling for cohort fixed effects. The estimated coefficient is then rescaled by the average predicted outcome among control women. Panel B investigates differences in pre-diagnosis educational attainment between women with MRKH and control women. For each individual, we compute their high school grade percentile rank within their graduation year and regress this on an indicator for ever having been diagnosed with MRKH, controlling for cohort fixed effects. The estimated coefficient is then rescaled by the average predicted outcome among control women. The right bar additionally introduces a vector of pre-diagnosis controls capturing family background (parental education and income levels, parental marital and fertility history) and childhood environment (county of birth and average income in the municipality of residence during childhood).

Figure II.3: **Effect of MRKH on Probability to be Diagnosed With Mental Health Conditions at 15**



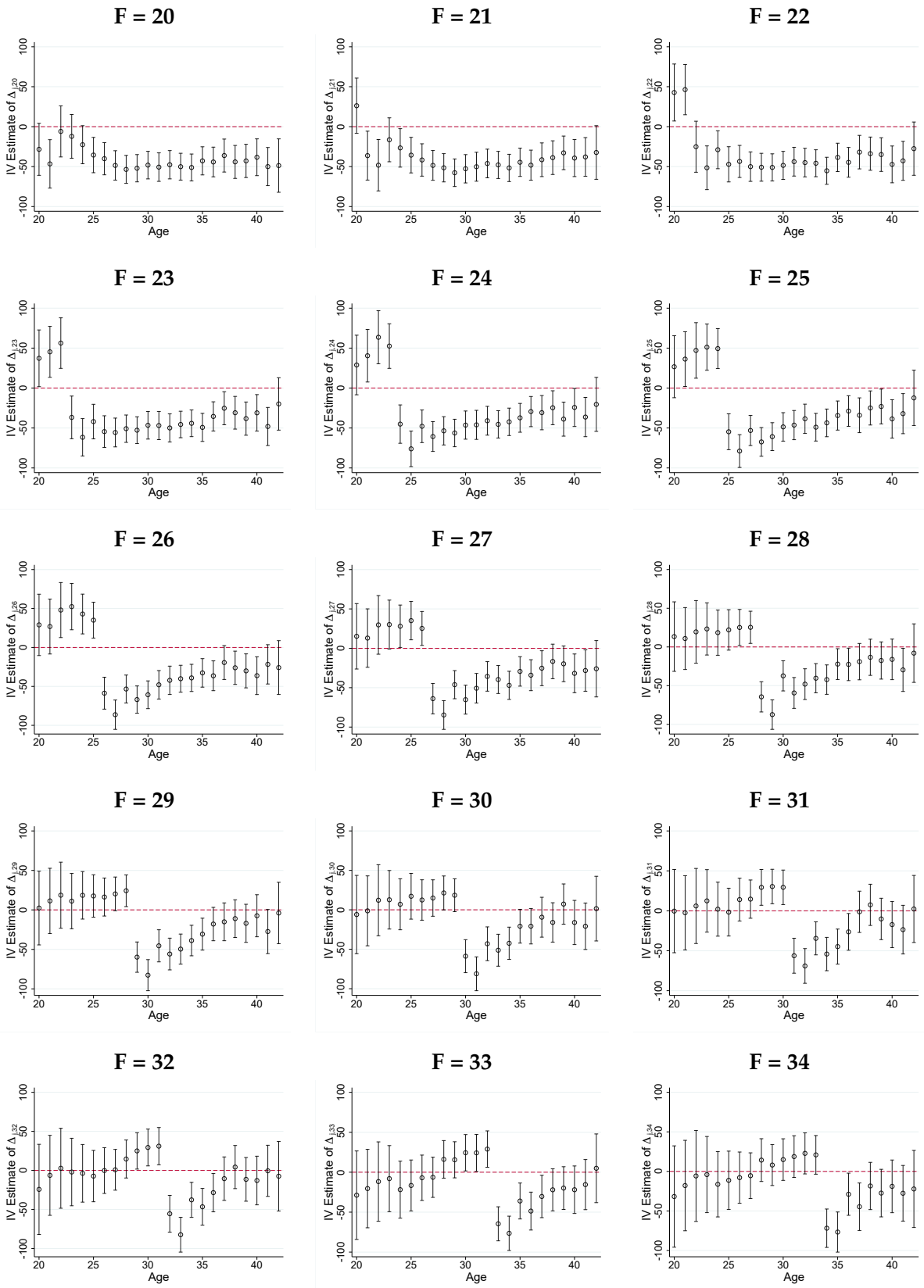
Notes: The figure investigates whether women with MRKH display different rates of mental health diagnoses prior to their MRKH diagnosis relative to control women. Mental health conditions are disaggregated into 12 subcategories. For each subcategory, we estimate two specifications using a linear probability model: one regressing a dummy for ever having received the diagnosis on an indicator for MRKH and cohort fixed effects, and one restricting the dependent variable to diagnoses made before age 16. Both specifications additionally control for cohort fixed effects, county of residence during childhood, municipality income, parental education, parental income, and parental marital status. For each condition, the bar represents the estimated coefficient with its 95% confidence interval

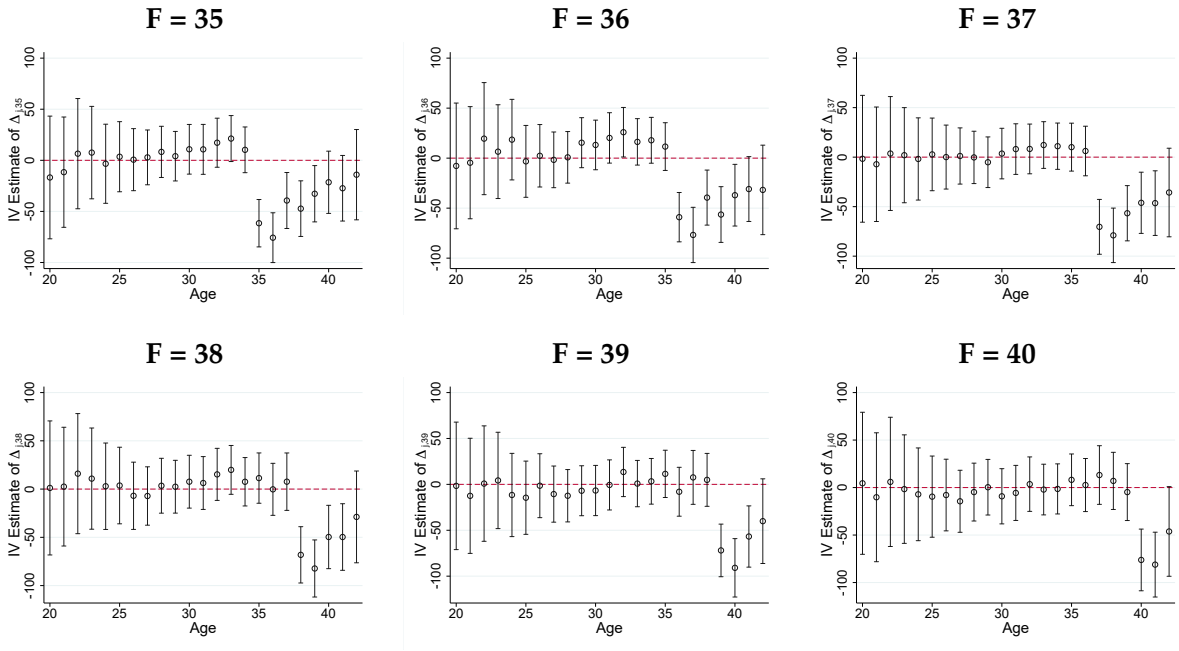
Figure II.4: **Effect of MRKH on Probability to be Diagnosed With Mental Health Conditions by Age**



Notes: The figure reports the estimated effect of the MRKH condition on the probability of being diagnosed with a mental health condition by age. The outcome is regressed on age fixed effects and their interaction with an indicator for MRKH, controlling for cohort fixed effects. The average effect over ages 20 to 42, reported in the top left corner, is obtained from a pooled specification that controls for age and cohort fixed effects. Robust standard errors are used and 95% confidence intervals are shown.

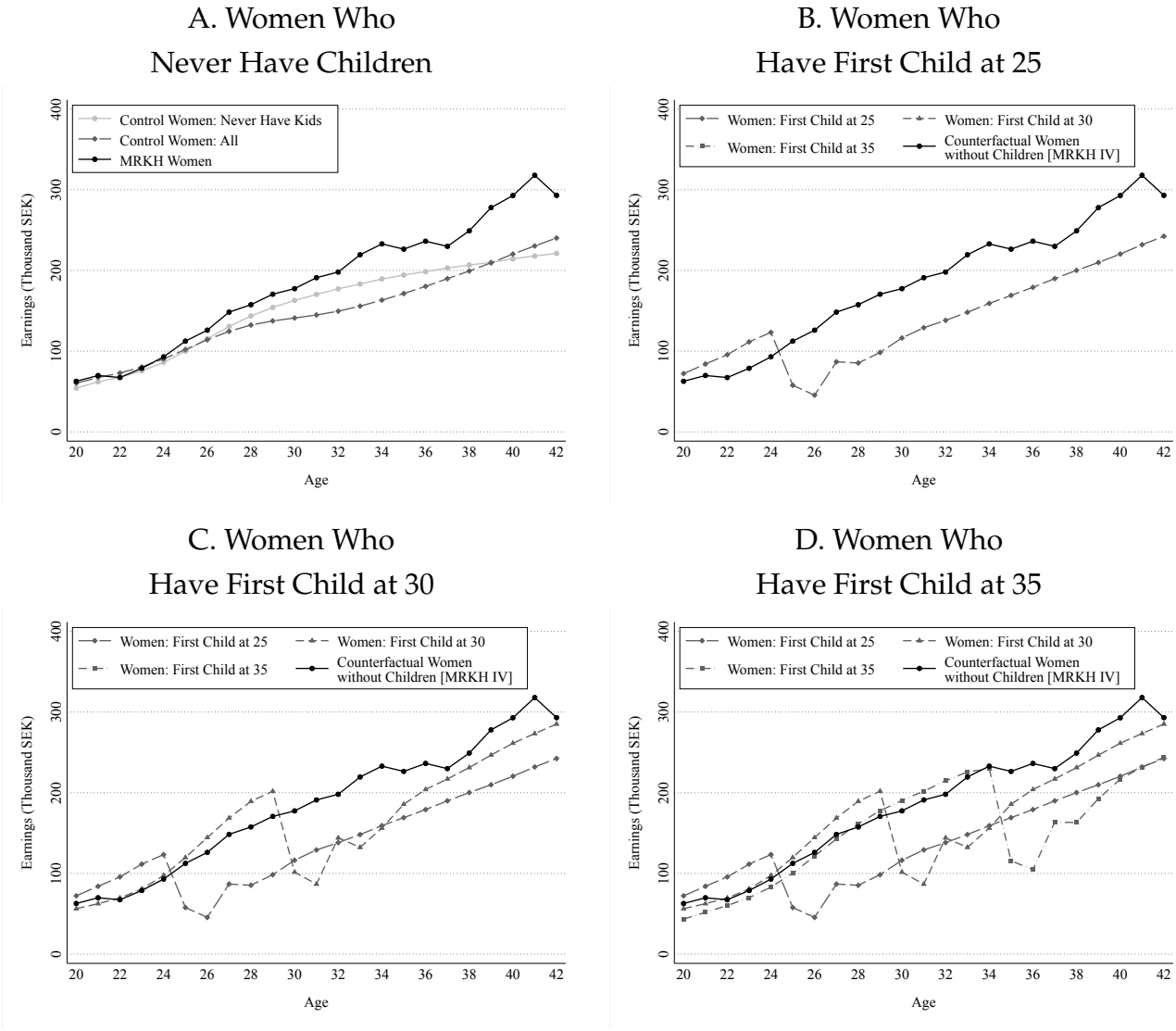
Figure IV.1: **Comparison of Earnings of MRKH Women vs Women With Kids, by Age at First Child k**





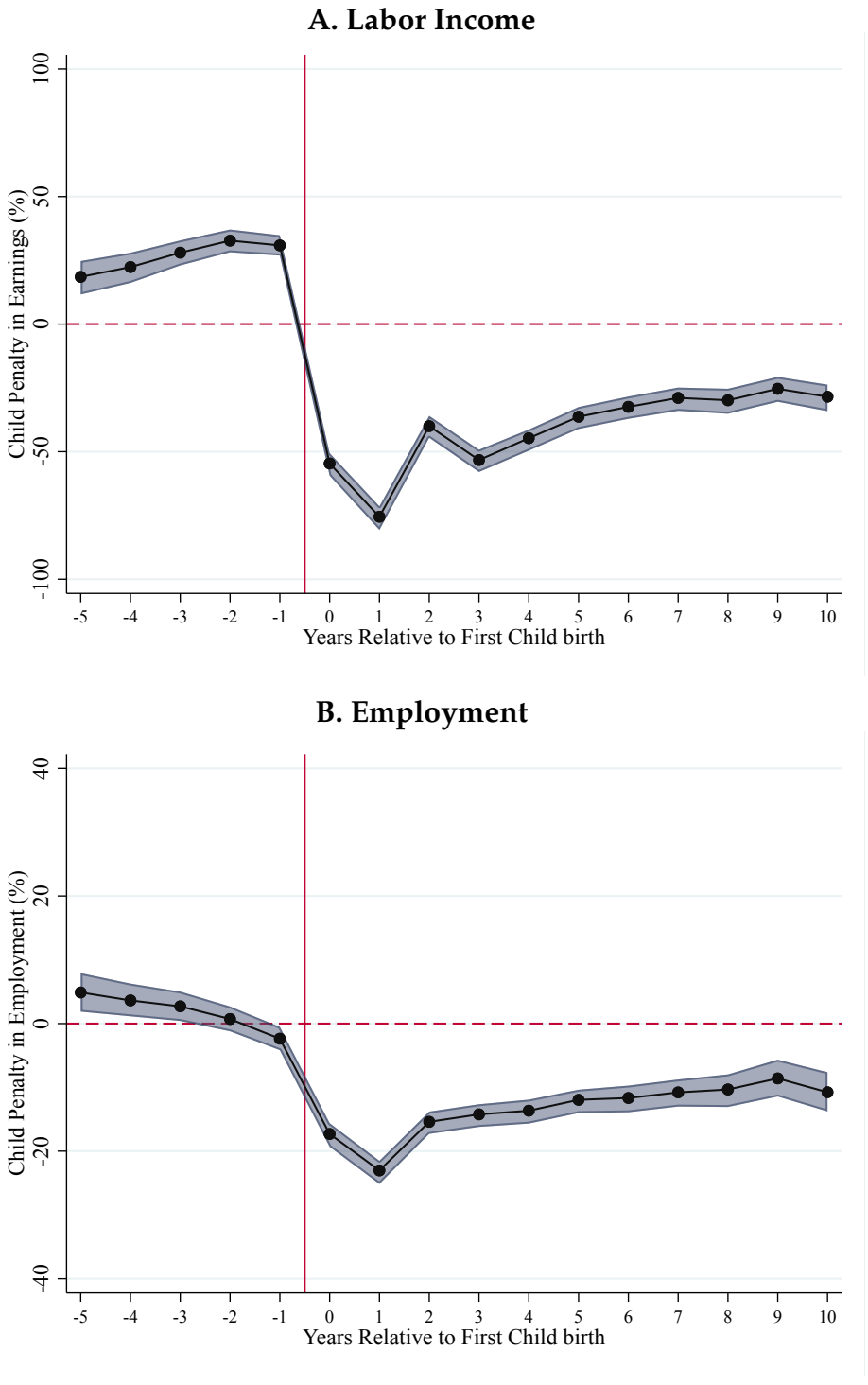
Notes: The figure illustrates the identifying variation underlying the child penalty estimates, showing that earnings drop sharply and persistently at the time of first birth relative to childless women. The figure reports IV estimates of $\Delta_{j,k}$ from specification 9 for labor earnings. Each panel focuses on a distinct age at first birth k and traces the estimated $\hat{\Delta}_{j,k}$ over all ages j. Across all panels, a sharp and persistent decline in earnings is observed at the age of first birth relative to women who remain childless, visually capturing the source of identification for the child penalty parameters.

Figure IV.2: **Comparison of Earnings Trajectories of Counterfactual Women Who Never Have Children (MRKH IV) and Control Women by Fertility Status**



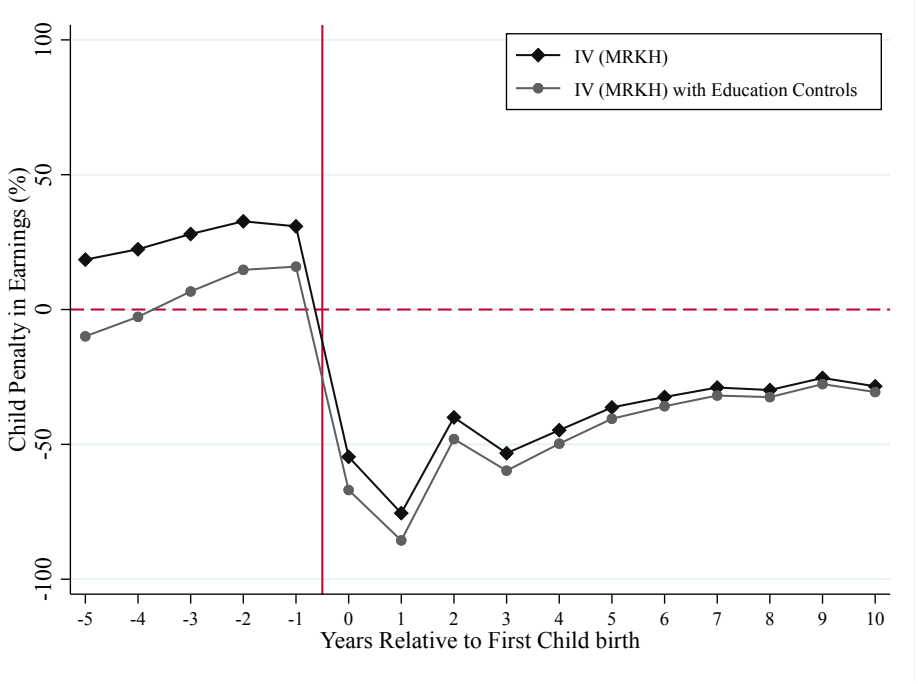
Notes: The figure investigates the role of having children in explaining the earnings differences of women with MRKH relative to control women, by comparing earnings trajectories across groups defined by fertility status. Panel A plots mean earnings profiles by age for women with MRKH, all control women, and control women who never have children. The MRKH earnings profile corresponds to the IV-based counterfactual earnings trajectory recovered by netting out the estimated child penalty at each age from the predicted earnings of women with MRKH. The profiles for control women are estimated by regressing earnings on age fixed effects and cohort fixed effects separately for each group. Panels B to D plot mean earnings profiles by age for control women who had their first child at age 25, 30, and 35 respectively, alongside the IV-based counterfactual earnings profile of women absent children, recovered using the MRKH diagnosis as an instrument. The specification controls for cohort fixed effects, county of residence during childhood, pre-diagnosis income decile, parental education and income, parental marital status, and pre-diagnosis educational attainment.

Figure IV.3: **Child Penalty: IV Estimates**



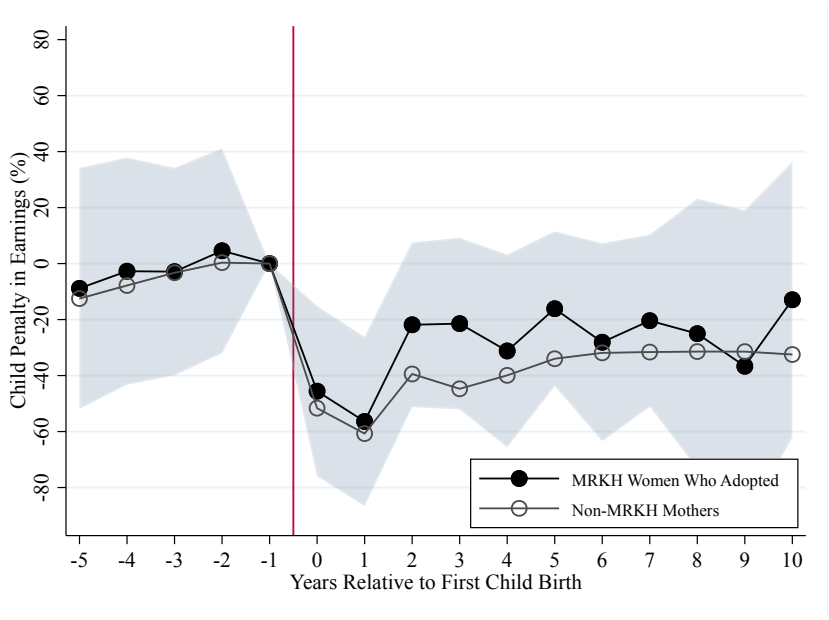
Notes: The figure reports IV estimates of the child penalty on total earnings (Panel A) and employment (Panel B). For each event time relative to first child birth, the child penalty is obtained by aggregating effects across fertility timing groups using specification 9. Standard errors are computed via bootstrap, stratified by age at first child and having being diagnosed with MRKH.

Figure IV.4: **Child Penalty in Labor Income: Mediation Analysis**



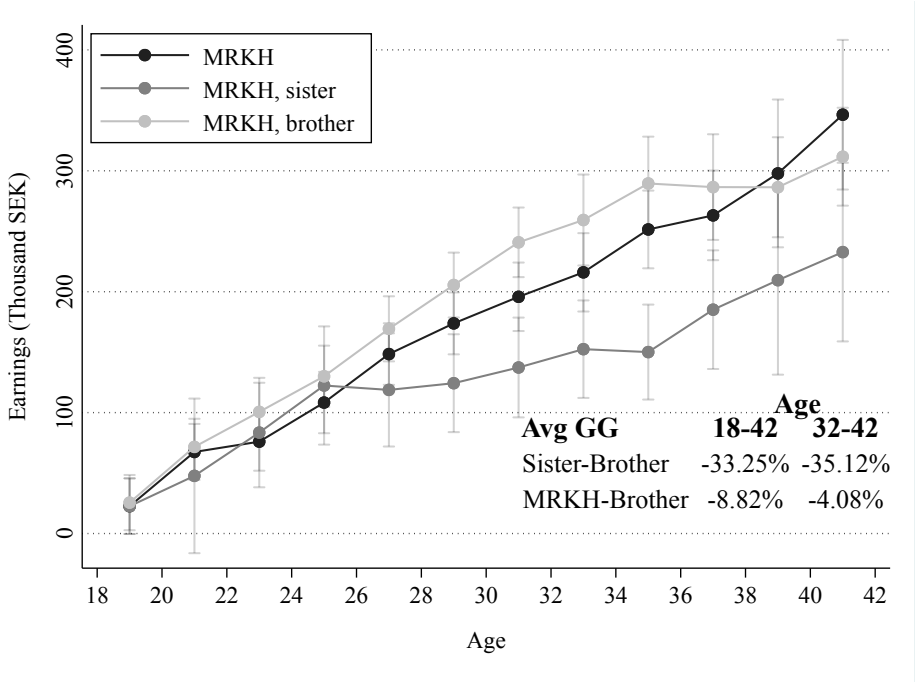
Notes: The figure explores the role of education as a mediator of the child penalty on total earnings. For each event time relative to first child birth, the child penalty is obtained by aggregating effects across fertility timing groups using specification 9, weighting by their population shares. Both specifications include a full set of controls comprising cohort fixed effects and pre-diagnosis characteristics capturing family background (parental education and income levels, parental marital and fertility history), childhood environment (county of birth and average income in the municipality of residence during childhood), and pre-diagnosis educational attainment (grade percentile at age 15). The second specification additionally controls for a dummy indicating whether the individual has completed her highest level of education at each point in time, allowing us to assess the extent to which education accounts for the observed earnings penalty.

Figure IV.5: **Event study estimates of the impact of children for adoptive women with MRKH and for biological mothers**



Notes: The figure investigates whether the child penalty differs between women with MRKH who adopted and non-MRKH mothers. For each event time relative to first child birth, the penalty is estimated separately for each group by regressing earnings on event time dummies, age fixed effects, and year fixed effects, with the period one year prior to first child birth serving as the baseline. The estimated coefficients are then rescaled by the counterfactual earnings level absent children, obtained as the predicted outcome from the regression omitting the contribution of the event time coefficients. Standard errors are robust and 95% confidence intervals are shown.

Figure IV.6: **Comparison of MRKH Women's Labor Income With Siblings**



Notes: The figure reports estimates of earnings profiles by age, comparing women with MRKH to their sisters and brothers. The sample is restricted to women with MRKH and their siblings. Specification (5) is extended to include men, allowing earnings profiles to be estimated separately for each group. The MRKH diagnosis is used as an instrument for never having children, allowing recovery of the counterfactual earnings profile of women absent children. The specification controls for age bins, year fixed effects, and family fixed effects. 95% confidence intervals are shown.

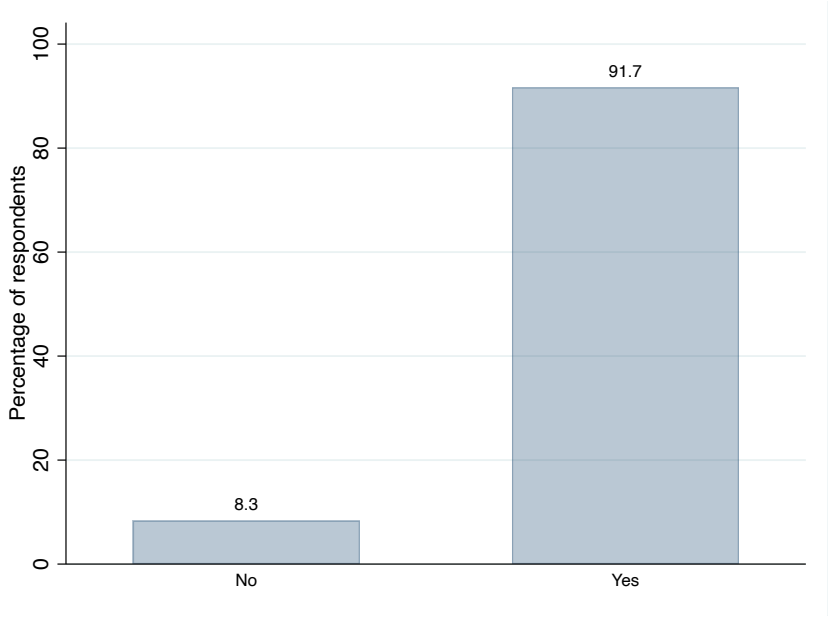
III. Additional Results Based on Survey Data

Table V.1: Effect of MRKH on Fertility, Education, and Labor Market Outcomes: Survey Evidence

	1	2	3
	A. Fertility		
Proba Having Bio Kid	-0.872***	-0.837***	-0.843***
	(0.104)	(0.107)	(0.108)
	B. Education		
Proba Graduate High School	0.156**	0.088	0.098*
	(0.076)	(0.054)	(0.052)
Proba Graduate Post Secondary	0.155	0.035	0.044
	(0.128)	(0.124)	(0.122)
	C. Labor Market		
Employment	0.151***	0.114***	0.120***
	(0.038)	(0.036)	(0.036)
Unconditional Earnings	8944.34**	8131.85**	10514.39***
	(4356.46)	(3316.71)	(3271.07)
Pre-diagnosis Controls		✓	✓
Health Controls			✓

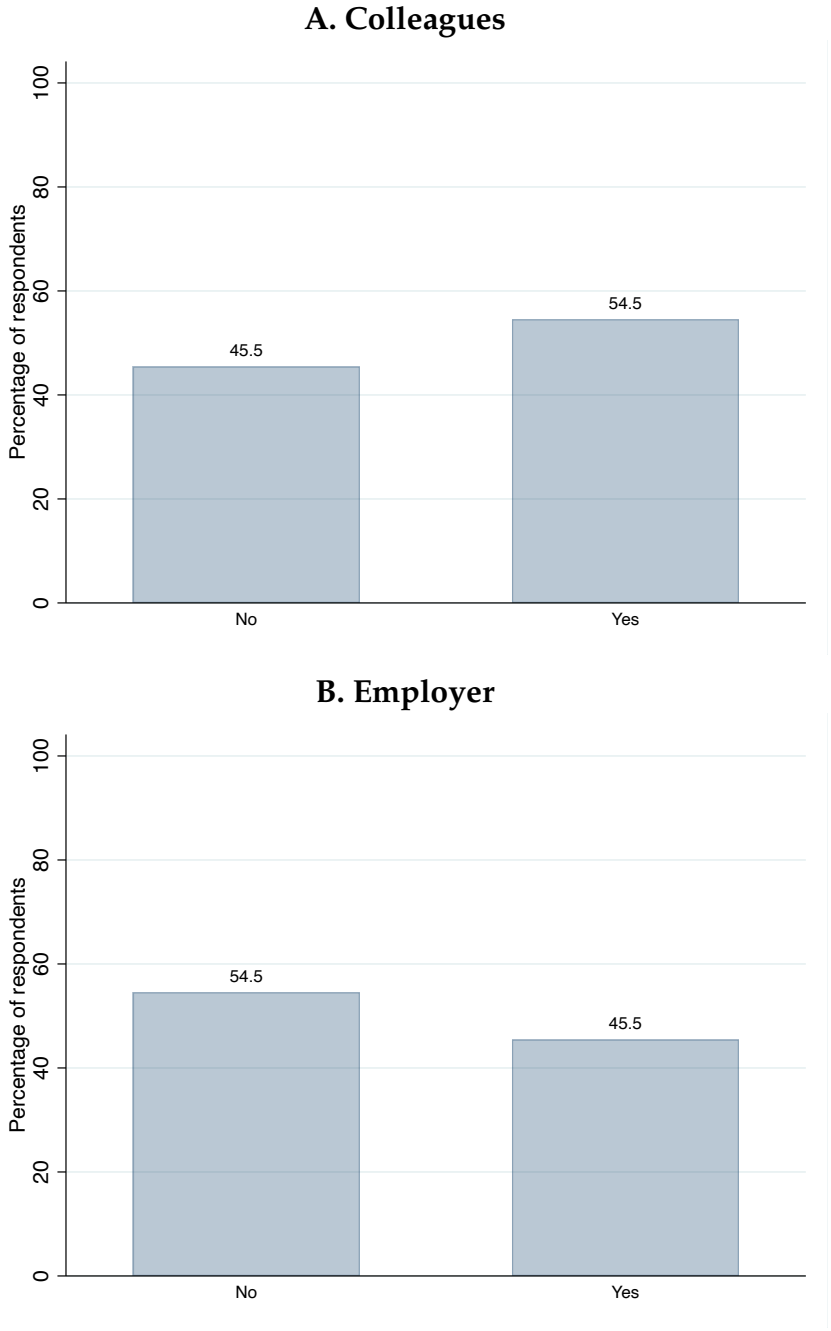
Notes: The table reports the estimated effect of the MRKH condition on fertility, education, and labor market outcomes using the survey disseminated in MRKH support groups, pooled with a control group drawn from the International Social Survey Programme (ISSP) and the World Values Survey (WVS). For each outcome, we regress an indicator for the relevant outcome on a dummy for being in the MRKH survey sample, controlling for cohort, age, and country fixed effects. The outcomes considered are the probability of having a biological child (average level in the comparison population = 59%), the probability of graduating high school (average level in the comparison population = 68%), the probability of graduating post-secondary (average level in the comparison population = 28%), employment (average level in the comparison population = 60%), and unconditional earnings (average level in the comparison population = 14,236 SEK). Column 1 includes no additional controls. Column 2 additionally controls for parental education. Column 3 further introduces both parental education and health controls. Standard errors are robust.

Figure V.1: **MRKH women tend to tell their friends about their condition**



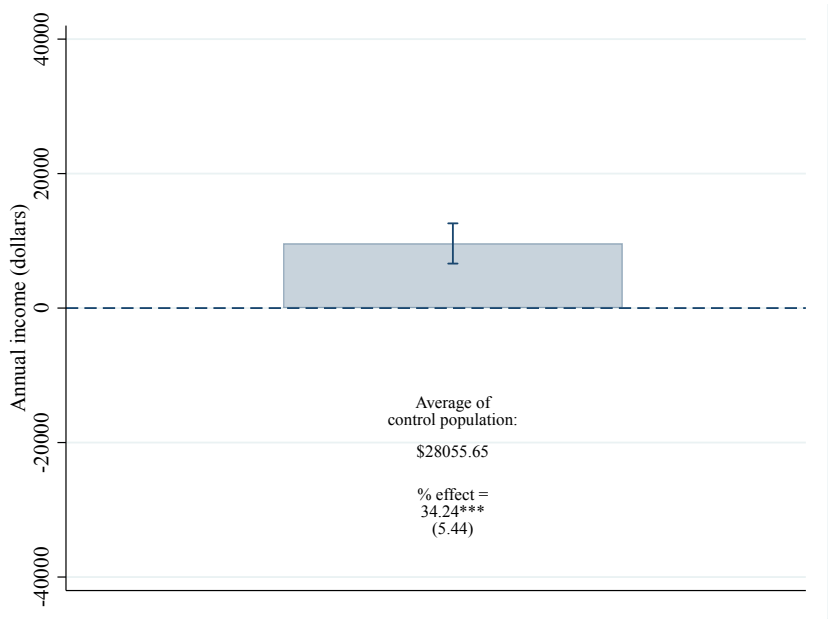
Notes: The figure displays the distribution of disclosure to friends among respondents in the MRKH survey sample, showing the percentage of women who have and have not revealed their condition to their friends.

Figure V.2: **MRKH tend not to tell their colleagues and employer**



Notes: The figure displays the distribution of workplace disclosure among respondents in the MRKH survey sample. Panel A shows the percentage of women who have and have not revealed their condition to their colleagues, and Panel B shows the percentage of women who have and have not revealed their condition to their employer.

Figure V.3: **Impact of revealing MRKH condition to one's employer**



Notes: The figure reports the estimated effect of revealing the MRKH condition to one's employer on annual income. The outcome is regressed on an interaction between an indicator for being in the MRKH survey sample and an indicator for having disclosed the condition to one's employer, controlling for cohort, age, and country fixed effects, as well as parental education and health controls. The estimated percentage effect of disclosure is reported with its standard error. Standard errors are robust.