# Economic and Fertility Consequences of Medical Innovation*

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**Abstract**

This paper studies the economic consequences of major positive shocks to health and life expectancy that arise from transformative medical innovation. Between 2012 and 2019, a new class of therapies, CFTR modulators, raised the life expectancy of patients with Cystic Fibrosis (CF) from the mid-30s to over 60. Based on underlying genetics, different subpopulations of CF patients benefited in different years from 2012-2019. We leverage this variation to isolate the causal effects of transformative new therapy on economic and family outcomes, drawing comparisons between patients with CF who benefit from therapy earlier, later, or never at all. Using data from the Cystic Fibrosis Foundation Patient Registry, we show first that pulmonary health improves and mortality rates fall by >50% after therapy eligibility. We then show eligibility impacts economic and family life: eligibility leads to a 7.5pp ($\sim$ 18%) increase in the probability of working full time, and a 7pp ($\sim$ 150%) increase in the probability of pregnancy. Effects are substantially larger than predicted by observed health characteristics, and are concentrated among those who benefit at younger ages. Educational attainment may progress more quickly, but does not appear to increase overall. Shocks to health and lifespan from new therapies have substantial economic impacts.

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# 1 Introduction

Health and lifespan are of fundamental economic importance (Murphy and Topel, 2006). New innovations in medical technology have made understanding the consequences of health and lifespan changes particularly pressing - for example, genetically targeted therapies have the potential to have transformative impacts, but at enormous cost. This paper explores the economic consequences of major shocks to health and lifespan from new precision medicines. We do this in the context of new, 21st century therapies for patients with Cystic Fibrosis (CF) in the United States - a paradigmatic example of a new wave of medical technologies.

Since 2012, a series of remarkable new therapies called CFTR modulators have revolutionized treatment for a subset of patients with CF - raising expected lifespan for eligible patients by over 20 years from a baseline of ∼30-35 years. Due to variations in the genetic mutations that cause CF, between 2012 and 2019, different sub-groups of CF patients became eligible for these new therapies. We leverage this staggered timing of treatment eligibility to draw comparisons between patients with CF who become eligible with patients who never become eligible or who become eligible later on.

We find that eligibility has substantial economic consequences. Eligibility for therapy increases full-time work by 18% and pregnancy by 150%. Impacts on educational attainment are very limited, but we find suggestive evidence that eligibility enables students to progress more quickly through the education system. Effects on health, labor markets, and pregnancy are largest among younger individuals, with no evidence for similar age-specific relationships for education.

These findings suggest that the economic consequences of new therapies are substantial, but also that valuing the economic returns to new therapies may be complex. Prior work has sought to understand the economic value of medical treatments, noting the value of improvements in health and lifespan (Cutler and Meara, 2000; Cutler and McClellan, 2001; Cutler, 2007; Cutler et al., 2022; Papageorge, 2016; Thirumurthy et al., 2008). However, limited empirical work has been able to comprehensively document the non-health spillovers of new medical treatments, especially for 21st-century therapies. Further, it is unclear how to value changes in characteristics like fertility.

Related literatures explore the responses of human capital investments to life expectancy. Prior research has shown that human capital investments respond substantially to changes in expectations around lifespan (Jayachandran and Lleras-Muney, 2009; Oster et al., 2013a; Karpati, 2025). Oster et al.'s work on Huntington's disease is most closely related to this paper, where they find that changes in life expectancy due to genetic testing shape human capital decision-making. This project complements that work: rather than emphasizing negative shocks to expectations, this focuses on the impacts of large improvements in health and expected longevity. Other work by Oster et al. suggests that this paper's line of inquiry may be high-yield: individuals with high risk of shortened lifespan may have optimistic expectations of their own health, and thus positive and negative shocks may not be symmetric (Oster et al., 2013b). We are also able to capture many individuals in a period critical for human capital decision-making, while conditions like Huntington's disease tend to manifest at later ages.

## 2 Cystic Fibrosis, Innovation in Treatment, and Data

Cystic Fibrosis is a genetic condition known by hallmark symptoms of mucosal buildup in the lungs, pancreatic insufficiency, and abnormally salty sweat, all caused fundamentally by failures in a protein that traffics chloride ions (Ong and Ramsey, 2023). The impacts of the disease on the lungs are the primary source of morbidity and mortality for CF. Thickened mucus in the lungs leads to obstructive lung disease, chronic infections, and chronic inflammation. This thickened mucus often leads to bacterial colonization of the lungs, with concomitant chronic infections, neutrophilic inflammation, and worsening plugging of the airways with mucus (Stoltz et al., 2015; Ong and Ramsey, 2023). Ultimately, respiratory failure has historically been the primary cause of death in CF patients (Oppenheimer and Esterly, 1975; CFF, 2023).

CF is a rare condition, affecting approximately 89,000 individuals globally, ∼35,000 of whom live in the United States (Bell et al., 2020; Ong and Ramsey, 2023; Cromwell et al., 2023). In the US, CF is primarily diagnosed as part of newborn genetic screening, though some children are diagnosed at older ages using a sweat chloride test (Farrell et al., 2017).

Inherited mutations in the CFTR gene can disrupt the process for producing functional CFTR protein in a number of ways that lead to CF. Figure 1 describes the physiologic process to produce functioning CFTR protein, and describes the different classes of mutations that prevent this process. In short, deficiencies in the ability to produce the intermediate RNA template that converts genetic code into protein, deficiencies in the production and trafficking of the protein to the cell membrane, deficiencies in the structure of the protein itself, or deficiencies in the quantity of CFTR protein produced can all lead to CF (Clancy and Jain, 2012; Mall et al., 2020). This variation in the genetic mutations and mechanistic failures that lead to CF underpins the identification strategy of this paper.

### 2.1 Innovation in treatment of Cystic Fibrosis

Starting in 2012, a series of CFTR modulators were approved for use for individuals with genotypes that would be responsive to each drug. Research has found that combinations of these drugs are often more effective than each drug alone, so we describe the approval by the marginal new drug being combined. Between 2012 and 2019, the share of individuals with CF who were eligible for a CFTR modulator increased to nearly 90% (Bell et al., 2020).

The class of CFTR modulator therapies nests both highly effective and non-highly effective modulators (Mall et al., 2020). The "highly effective" characteristic of the therapy is a function of the match between drug efficacy and the target genetic mutations (Mall et al., 2020). Ivacaftor, first approved in 2012 and expanded to cover additional mutations in 2014, is highly effective for "gating" mutations but not for "residual" function mutations. The other highly effective therapy is elexacaftor, which was approved in 2019. Additional, less-effective, modulators were developed in the interim for some sub-populations of CF patients. Approximately 10% of individuals with CF do not yet have any eligible CFTR modulator therapy. In practice, we will focus on individuals who become newly eligible for highly-effective modulators who were not previously eligible for any highly or non-highly effective modulator.

Highly effective modulators have had transformative impacts on lifespan and health for patients who are eligible. Clinical studies of CFTR modulators have found that those eligible have

had mortality rates fall greater than 50% (Merlo et al., 2024; Bessonova et al., 2018; Ramsey et al., 2011; CFF, 2023).

We can observe these effects in our data, described in detail below. Figure 2 shows the uptake of CFTR modulators among those genetically eligible for each drug. Uptake of highly effective modulators is rapid, with over 70% of eligible patients prescribed the drug within two years of approval. These drugs have transformative impacts. The time-series of mortality rates and health outcomes for those who become eligible for highly effective modulators shows substantial improvements (Figure 3). Age-standardized mortality rates for adult CF patients fall by over 50% within a year of approval, and lung function, measured using spirometry-based Forced Expiratory Volume in 1 second (FEV1), improves by 12pp. ppFEV1 is the primary biomarker used to assess lung function in CF patients, and can be interpreted directly as the lung function as a percent of what is expected for an individual of the same age, sex, and height (Cromwell et al., 2023). A 12pp increase in lung function is very large.

### 2.2 Data: The Cystic Fibrosis Foundation Patient Registry

The primary data source is the 2003-2024 Cystic Fibrosis Foundation Patient Registry (CFFPR), a longitudinal clinical registry that tracks individuals diagnosed with CF receiving care at one of over 130 CFF-accredited care centers in the United States (CFF, 2023). The registry has operated for several decades and is widely regarded as the best source of population-level data on individuals with CF in the U.S. (Knapp et al., 2016).

Registry coverage is high - approximately 90% of individuals with CF in the US are covered by the registry (Cromwell et al., 2023). The registry pairs remarkably detailed health data and treatment histories for individuals with CF with records of educational attainment, employment status, marital status, fertility outcomes, and insurance coverage for patients. These economic and family measures are assessed primarily using surveys administered to patients during clinic visits, and are updated annually. Annual participation of CFFPR adults in the additional survey measures is approximately 85%. The unit of observation is the individual-year, using annual means for biomarker measures and taking calendar-year aggregates of hospitalization and healthcare utilization.

## 3 Empirical Strategy

Identification in the primary specification is derived from the staggered timing of genetic-based eligibility for highly effective CFTR modulators. We focus on an intent-to-treat approach, avoiding selection on characteristics related to therapy uptake. Our primary specification uses a stacked difference-in-differences approach (Cengiz et al., 2019).

Using data on individual genotypes, we are able to cleanly assign the first calendar year in which an individual becomes eligible for their first CFTR modulator drug. We group people who become first-time eligible for a highly effective modulator, having never been previously eligible for any modulator, in the same calendar year into the index treatment group for a given "stack". For the same stack of treated individuals, we build a control group of individuals with CF who are either never modulator-eligible or who become eligible for their first modulator more than 5 years after the index year.

For our analyses of work and family life, we focus on prime age adults aged 22-30 at the time

of measurement, and compare individuals at age 22-30 before and after eligibility.[1] We focus on this age to emphasize a group that has primarily completed education and is in the early stages of career and family planning. Given that the consequences of CF accumulate with age and mortality rates continue to increase, we also focus on a younger group of adults to mitigate the impacts of selective mortality. For analyses of education, we examine categories of individuals at younger ages, in order to capture the dynamics of outcomes that do not vary as frequently year-to-year.

For each group, we estimate the following difference-in-differences specification and the associated event study:

$$Y_{iht} = \beta \, Eligible_{iht} + \gamma_{jh} + \delta_{ht} + \theta_{a(it),\,x(i)} + \epsilon_{iht} \tag{1}$$

where $\theta_{a(it),\,x(i)}$ are exact age($a(it)$)-by-sex($x(i)$) fixed-effects, $\delta_{ht}$ reflects stack-event-time effects, and $\gamma_{jh}$ are stack-by-treatment group fixed effects. $Eligible_{iht}$ equals one after an individual $i$ first becomes eligible for a highly effective modulator in index year $g_h$, and zero for the stack's controls. The primary difference-in-difference coefficient $\beta$ in equation 1 captures the effect of eligibility across the pre and post-treatment periods.

The setting supports an exogenous interpretation to treatment timing. The success of ivacaftor was a surprise event that came after decades of less-successful attempts at therapeutic development (Clancy and Jain, 2012; Ledford, 2012; Hug, 2013). The expansion in 2014 to other gating mutations came after additional scientific discovery for efficacy in specific genes (Boeck et al., 2014). The success of elexacaftor, in 2019, also came on the heels of two other theoretically promising but ultimately not highly effective CFTR modulator drugs that did not nearly match the initial success of ivacaftor (Jones and Barry, 2015). The development and diffusion of highly effective therapy was not a foregone conclusion.

## 4 Results

The sample comprises patients with CF in the CFFPR. Table 1 displays characteristics for the full 2003-2024 CFFPR, as well as pre-eligibility characteristics for individuals that are in the treatment (first-time eligible for high efficacy modulators) and reference (ineligible for any CFTR modulators in the event window) groups for the primary empirical specification of 22-30 year olds. In general, the population is young. The average CF patient in the overall registry is under 21 years old. Patients have poor lung function and high morbidity: the average pre-eligibility lung function for those in the analytic sample is 73-74% of expected, and individuals average one hospitalization per year. Over 10% are underweight. Employment and college completion-rates are lower than in the general population, but a substantial share of patients with CF are still living rich economic and family lives before modulator eligibility. Comparing individuals in the primary analytic sample, there are few differences of statistical or substantive importance between the treatment and control groups conditional on age and sex (column 4).

[1] An alternative specification choice would have been to use individual fixed-effects. Individual FE models are qualitatively and quantitatively similar, but mortality selection and aging of a fixed group make interpretation with 5 years of follow-up more challenging. As a result, we prefer comparisons of a fixed age group over time with fine age-by-sex controls.

### 4.1 Impacts of CFTR modulator eligibility on individual outcomes

We begin by examining the health consequences of CFTR modulator eligibility. Figure 4 displays the estimates for the impact of highly effective CFTR modulator eligibility on health outcomes. There is little evidence for differential trends in the pre-period, and health changes sharply after eligibility for highly effective CFTR modulators. Lung function improves by 12.2 percent of predicted on a baseline of 69, and the share underweight falls by 4pp - a reduction of 37%. These are substantial changes in health, and examining the trends for the treated and control individuals shows clearly that outcomes for the control group are relatively flat and smooth for this period, while a clear trend break occurs only for treated groups (Appendix figure A.1).

We turn our attention to labor market outcomes. The primary outcomes that we assess are survey-based measures of full-time work, part-time work, or disability. Full-time work increases by 7.5pp over a base of 41%, and by 5 years after approval has increased by over 12pp - a 30% increase in full-time employment. Substantial declines in disability are also clearly observed. There is limited evidence for aggregate shifts in the share of individuals working part time, but this appears to be masking a "rippling up" effect as individuals move between non-work, part-time work, and full-time work. Appendix figure A.2 shows the difference-in-difference estimates on the propensity to transition into and out of different work statuses. The null impact of eligibility on part-time work is clearly driven by a reduction in transitions from full-time to part-time, which often happened as individual health declined, combined with an offsetting increase in transitions from non-work to part time.

We find substantial increases in pregnancy. Pregnancies increase by 7pp against a base rate of 4.6% - an increase of over 150%. A notable spike occurs in the second year after eligibility, potentially reflecting acute changes in family planning, but continues to stabilize at a higher level than the baseline.

In figure 5, we explore the extent to which baseline characteristics can explain observed results for work and pregnancy. We conduct this exercise in two steps. First, using pre-eligibility person-years, we estimate a model of work and pregnancy as a function of observed health characteristics. We estimate a fully-saturated regression of: dummy variables for a series of ppFEV1 lung function bins, a dummy variable for underweight BMI, exact age, and sex. We take these models to then predict work and pregnancy status for each individual, creating a counterfactual for predicted work and pregnancy on the basis of observed health characteristics. Finally, we compare realized work and pregnancy changes as a result of CFTR modulator eligibility with these benchmarks. We find that observed health characteristics explain 1/3-1/2 of the increase in full-time work, but functionally none of the changes in pregnancy. This suggests that changes in pregnancy are working through a mechanism other than contemporaneous health status, such as expectations of lifespan, while changes in full-time work are a combination of direct health changes and other factors.

### 4.2 Impacts of CFTR modulator eligibility on educational attainment

To examine educational attainment, we estimate the same specification as equation 1, but focusing on younger age groups that are making education choices. In order to observe dynamics, we draw comparisons among two groups - those age 19-23 and those age 24-27 at measurement.

Figure 6 shows the difference-in-difference estimates for changes in educational attainment. In

the 19-23 age group, eligibility raises high school completion and some college completion. However, at the age 24-27 period, there is no impact on any overall educational attainment. Taken together, this may suggest that in a population where educational attainment is often delayed, educational attainments like high school or some college that are often earned en route to higher education are finished more rapidly, even if overall attainment has not increased. Examining high school completion by age for ivacaftor-eligible individuals before and after eligibility is consistent with this: High school completion by ages 20 and 21 is greater among those who benefit from eligibility, but eventually re-converges by age 24 (appendix fig. A.3).

### 4.3 Age-specific impacts of CFTR modulator eligibility

In the prior section, we showed the extensive margin effects of exposure to highly effective CFTR modulator treatment at ages 22-30. We extend our analyses to examine the intensive-margin effects of highly-effective modulator eligibility. In figure 7, we estimate intensive margin slopes of becoming eligible for therapy at younger ages on outcomes between 25-30, conditional on exact age-by-sex controls, among individuals who ever become eligible for CFTR modulator therapy. As a placebo, we estimate slopes for individuals aged 30-35, for whom modulator eligibility should have no impact on outcomes before age 30.

Estimates show substantial intensive-margin effects of therapy. Those who become eligible at younger ages have better lung function and are more likely to be working full time. Linear estimates of an age-at-eligibility effect show becoming eligible an additional year earlier for CFTR modulator therapy is associated with a 1.5pp increase in the probability of later working full-time. The effect on pregnancy does not appear to be linear, rapidly increasing as individuals become treated at 25-30 and then plateauing, with limited additional marginal benefit from eligibility at younger ages.

However, there appear to be no age-specific effects on college education or more. Even among individuals who were under 18, the propensity to complete college later does not appear to be any higher, and in fact may be somewhat lower, than the completion rates of those who benefitted later. Taken with the prior extensive-margin evidence on education, it appears that highly effective CFTR modulators had no impact on overall educational attainment.

## 5 Conclusion

In this paper, we show that transformative changes in health and lifespan associated with eligibility for highly effective CF therapies lead to major improvements in labor market outcomes and striking increases in pregnancy. Effects on health and labor market outcomes are largest for those who benefit at younger ages. There is little impact on educational attainment, which may be consistent with changing consumption value of education or improved outside options in the labor market at younger ages. The results provide real-world causal evidence for substantial economic consequences from new transformative therapies and precision medicines. They also raise important questions about the value of innovation - when the quality of life is so transformed it is not necessarily clear how to value additional fertility, or new career prospects. Ongoing work explores the potential mechanisms underpinning these changes.

# Tables

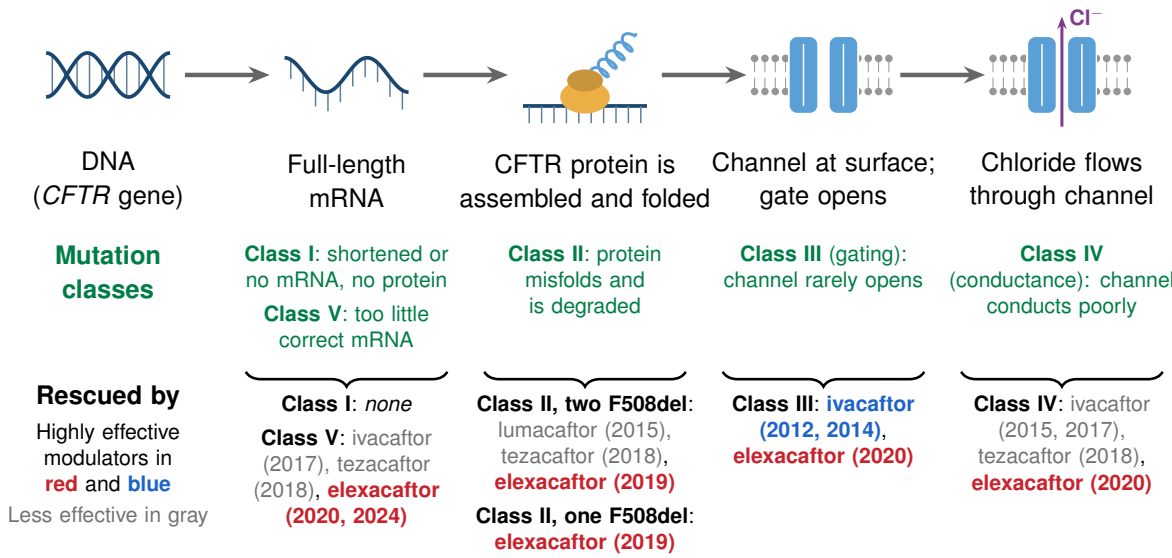
Table 1: Sample characteristics

| | Full registry (all ages) | Primary difference-in-differences sample Before eligibility | | |
|---|---|---|---|---|
| *Percent unless noted* | | Treated | Control | Adj. gap (SE) |
| | (1) | (2) | (3) | (4) |
| Age (years) | 20.5 | 23.5 | 23.0 | 0.3 (0.1) |
| Female | 48.3 | 48.1 | 46.6 | 3.0 (1.7) |
| ppFEV1 (% predicted) | 81.9 | 73.3 | 74.0 | -0.7 (0.8) |
| Underweight (BMI $<$ 18.5) | 5.9 | 11.1 | 12.6 | -1.0 (0.9) |
| Hospitalizations per year | 0.61 | 1.21 | 0.99 | 0.11 (0.05) |
| Working full time | 37.0 | 27.6 | 22.9 | 1.4 (1.1) |
| Working part time | 16.4 | 20.5 | 19.2 | 0.7 (1.0) |
| Pregnant (women) | 4.5 | 3.8 | 4.8 | -1.3 (0.7) |
| High school or more | 92.7 | 89.1 | 88.3 | 0.1 (0.8) |
| College or more | 36.3 | 24.3 | 19.9 | 3.0 (1.1) |
| Married or partnered | 42.2 | 22.8 | 20.2 | 2.0 (1.1) |
| Patients | 47,201 | 4,046 | 4,517 | |
| Person-years | 611,397 | 12,082 | 16,076 | |

*Note*: Column 1 is every observed person-year in the CF Foundation Patient Registry, all ages; employment, pregnancy, education and marital status are recorded for adults only, so those rows describe adults. Columns 2–4 are person-years in the four years before the stack's eligibility year (stacks 2012, 2014 and 2019) at ages 18–30. Outcomes are estimated at ages 22–30; the lower age floor captures pre-eligibility characteristics for individuals who reach 22 only after becoming eligible. Treated individuals first become eligible for a highly effective modulator in the stack year. Controls are pooled across stacks: individuals not eligible for any modulator through 2024, or first eligible more than five years after the stack year. Pregnancy is among women. The adjusted gap is the treated–control difference conditional on stack-by-year and exact age-by-sex fixed effects (stack-by-year only for age and female), with standard errors clustered at the individual level; it is in the units of its row. Patients are unique individuals; a control can appear in more than one stack, and control person-years are counted once per stack.

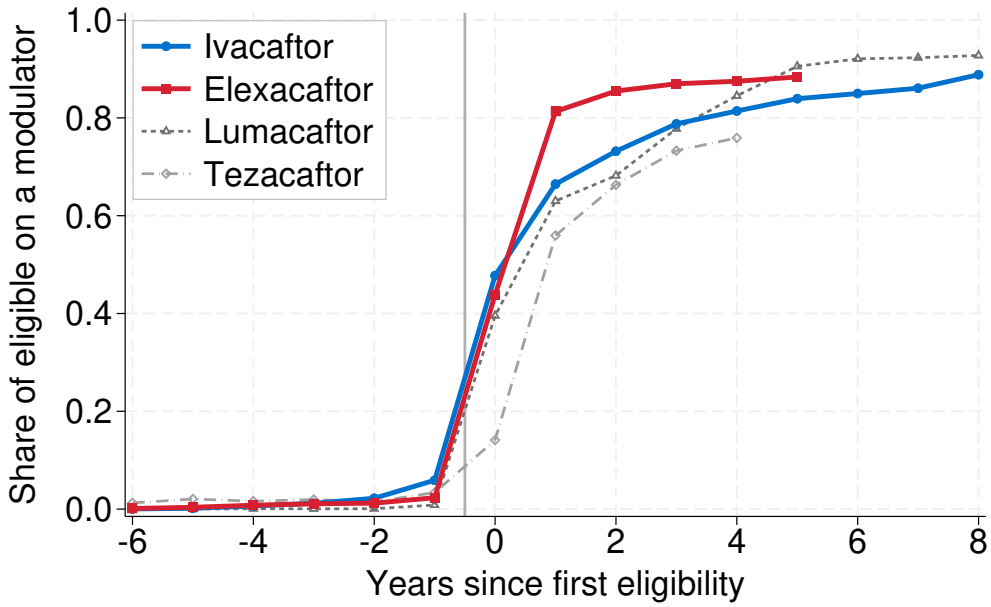
# Figures

Figure 1: CFTR function, mutation classes, and function rescue by CFTR modulators



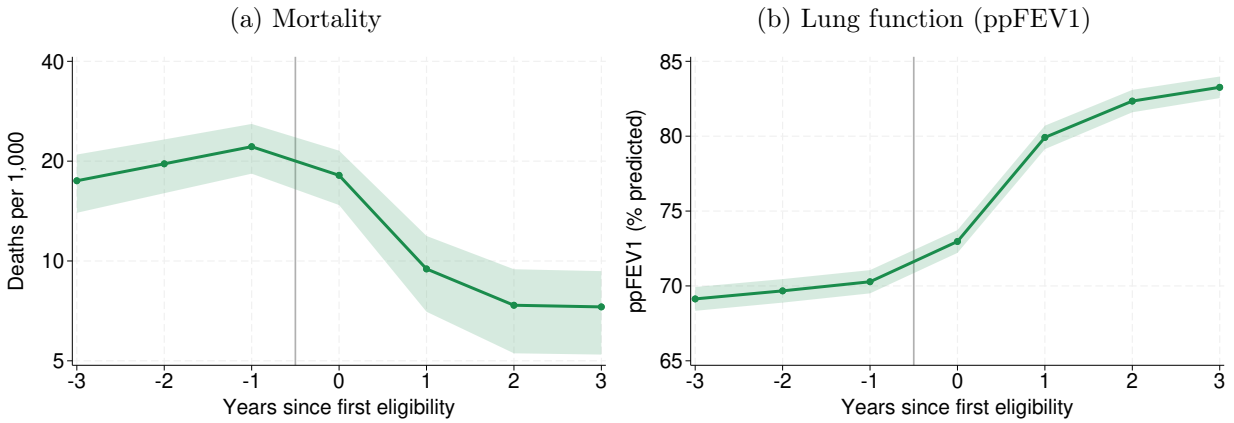

*Note*: The figure shows the process by which the CFTR protein is trafficked and functions. The second-row text describes the mutation classes that affect each step of the process. The third-row text describes the CFTR modulators developed for each different class of mutations, and when they were approved for that group. Combination modulators are described by the new drug in the combination (e.g., elexacaftor/tezacaftor/ivacaftor is described as elexacaftor). Highly effective modulators are shown in red and blue, while less effective modulators are shown in gray. This paper's primary design uses the highly-effective modulators that were approved for mutation classes without prior, less-effective, modulator eligibility. These include the 2012 and 2014 approvals of ivacaftor for gating mutations (class III), and the 2019 approval of elexacaftor/tezacaftor/ivacaftor for individuals with one copy of F508del (class II).

Figure 2: Modulator uptake by years since first eligibility



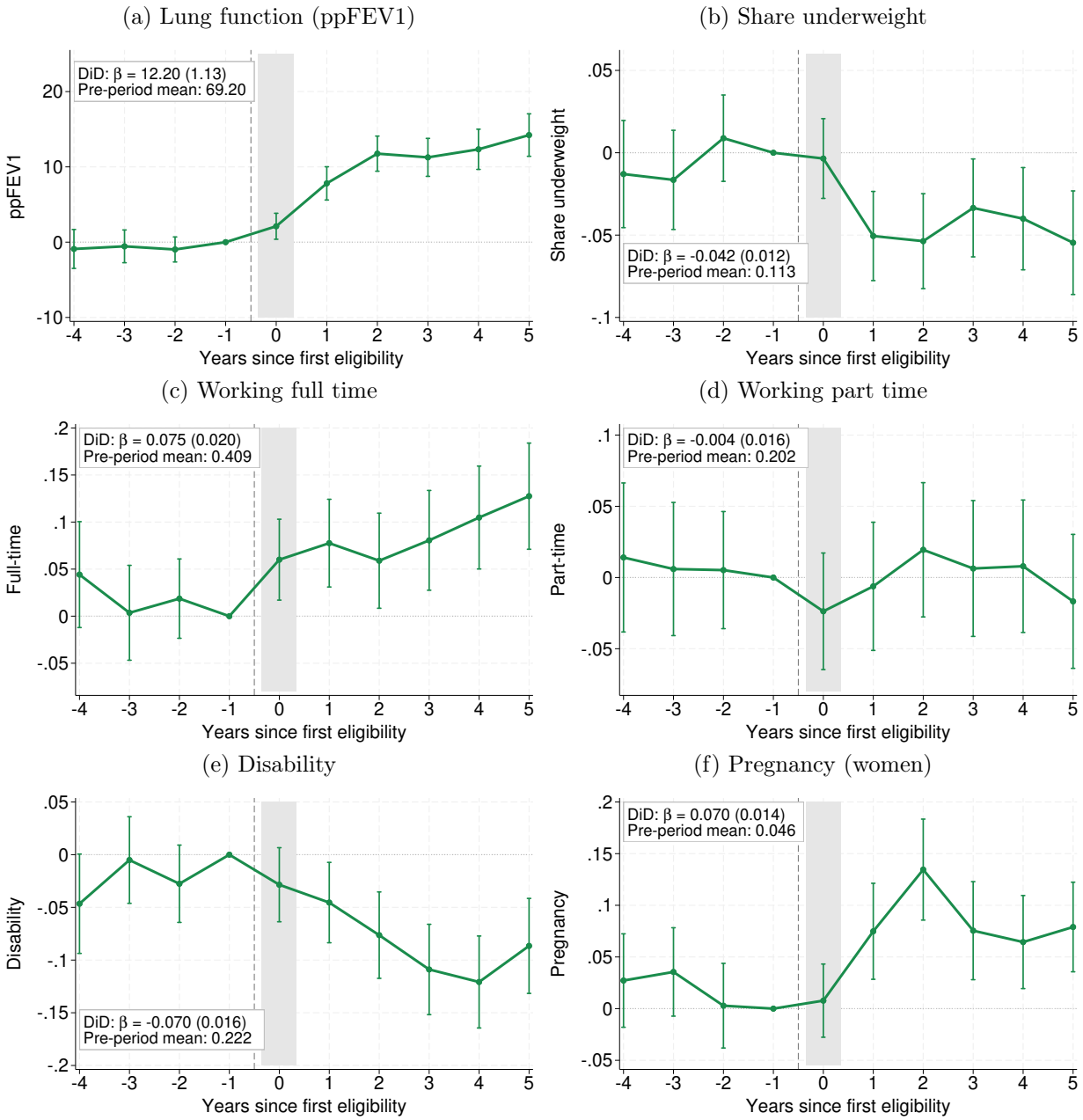

*Note*: This figure shows the share of individuals in the CFFPR registry who become genetically eligible at time=0 who are taking CFTR modulators. Highly effective modulators are marked in red and in blue, while less-effective modulators are marked in gray. Therapies are often combination drugs, but are labeled by the new molecule contained in each (e.g. elexacaftor/tezacaftor/ivacaftor is labelled simply as elexacaftor). Follow-up time differences reflect differences in the year of approval.

Figure 3: Trends in mortality and lung function around eligibility for highly effective modulators



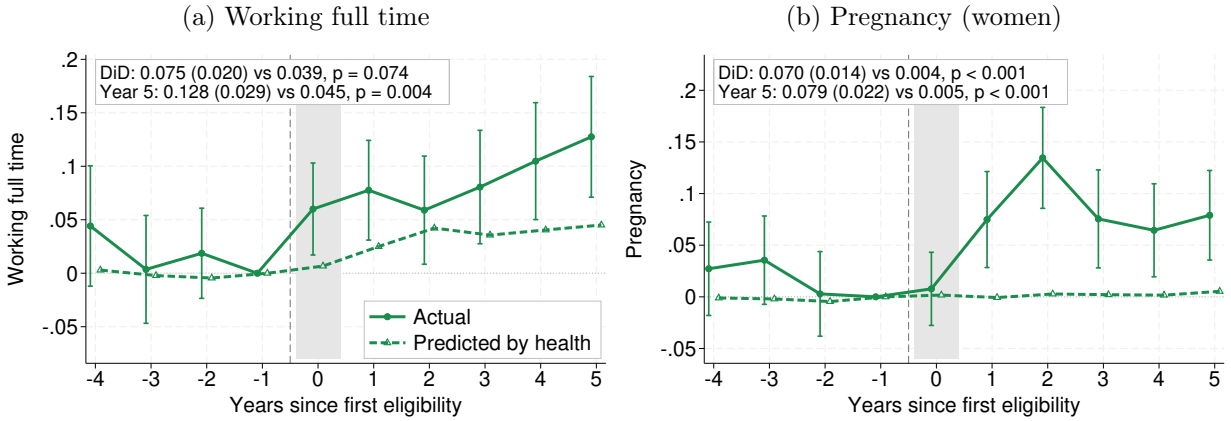

*Note*: Age-standardized annual mortality rates and lung function assessed via ppFEV1 are displayed for adults 18-45 who become initially eligible for highly effective CFTR modulators. 95% confidence intervals clustered at the individual level are shaded.

Figure 4: Event-study estimates: health and employment



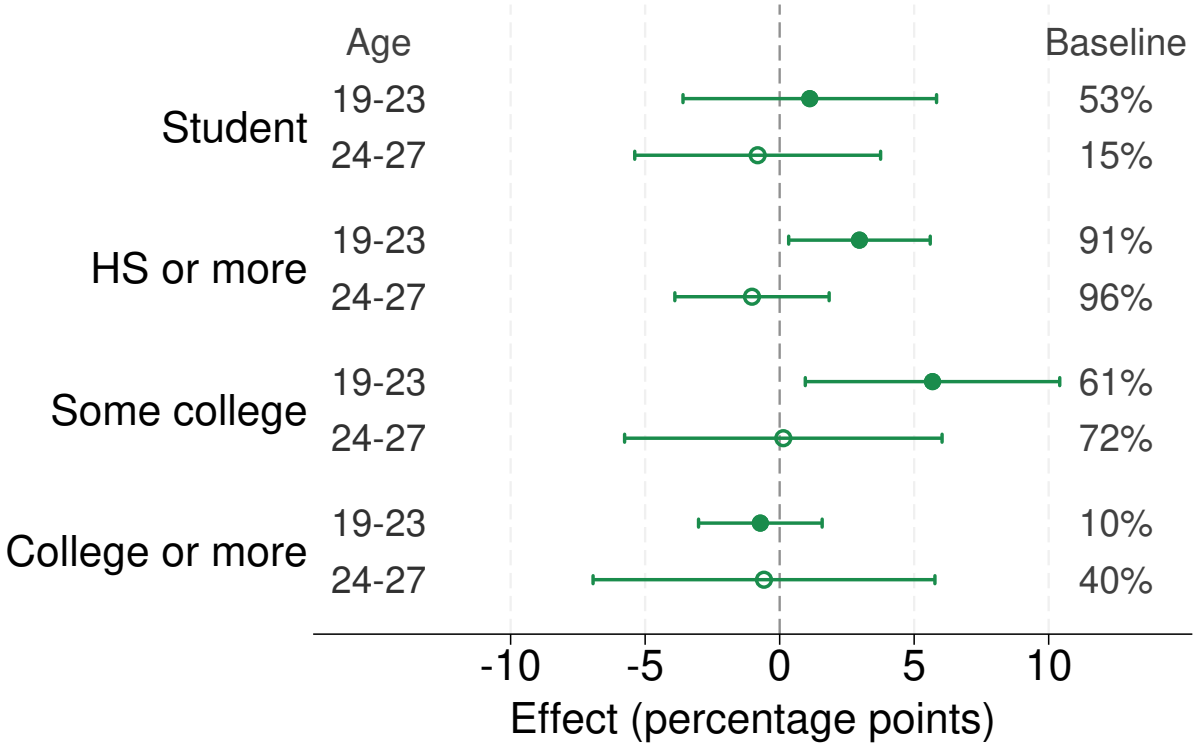

*Note*: Event study estimates of the health, economic, and family impacts of CFTR modulators. Estimates from a stacked difference-in-differences estimate including those age 22-30 at the time of measurement, exact age-by-sex controls, and stack-event-time fixed effects. Difference-in-differences estimates and pre-period means displayed in the box. Standard errors clustered at the individual level.

Figure 5: Actual and health-predicted event-study estimates



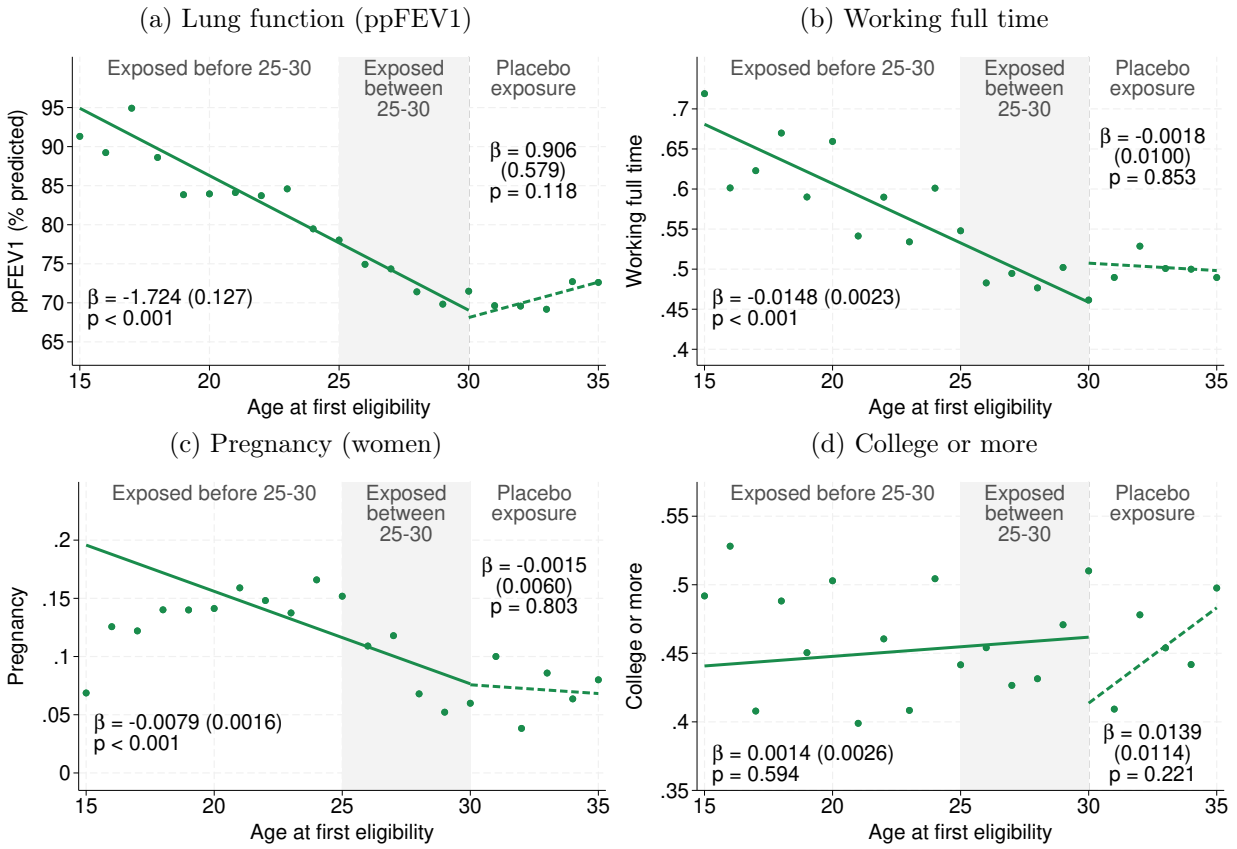

*Note*: Solid lines in the figure re-print event-study estimates from figure 4, with 95% confidence intervals clustered at the individual level. Dashed lines reflect estimates on changes in predicted outcomes from health status, estimated using a fully saturated regression on pre-eligibility person-years of outcomes as a function of 10-point ppFEV1 bins × exact age × sex × underweight status (BMI<18.5). Comparisons between changes in actual outcomes and changes in health-status-predicted outcomes are conducted for the pooled difference-in-differences and at the 5th year post-eligibility and the $p$-values are displayed.

Figure 6: Difference-in-differences estimates: educational attainment




*Note*: Difference-in-difference coefficients on the impacts of eligibility on educational attainment are presented for two age groups. Estimates from a stacked difference-in-differences model with five years of follow up are shown with 95% confidence intervals calculated with standard errors clustered at the individual level. Pre-eligibility means are shown on the right.

Figure 7: Outcomes at ages 25–30, conditional on age-by-sex controls, by age at first eligibility




*Note*: The sample is individuals who become first eligible for a highly effective modulator (ivacaftor in 2012 or 2014, elexacaftor in 2019), with outcomes measured at ages 25-30. Dots reflect binscatters of the mean outcome among individuals who are first eligible at that age, conditional on exact age-by-sex. The shaded band marks individuals first eligible during the measurement window (ages 25–30); those first eligible before 25 are exposed throughout it. Plotted lines and the printed estimates display linear regression estimates of the marginal impact of age-at-eligibility on outcomes; estimates for the solid line are at bottom left and for the dashed line in the placebo band. Solid lines are fitted for individuals first eligible at or before age 30. Dashed lines are slopes for individuals first eligible between ages 30 and 35, treated as a placebo group for whom marginal years of exposure should not impact outcomes assessed before age 30. $\beta$ is the change in the outcome per additional year of age at first eligibility, so a negative $\beta$ means earlier eligibility raises the outcome. Standard errors in parentheses are clustered at the individual level.

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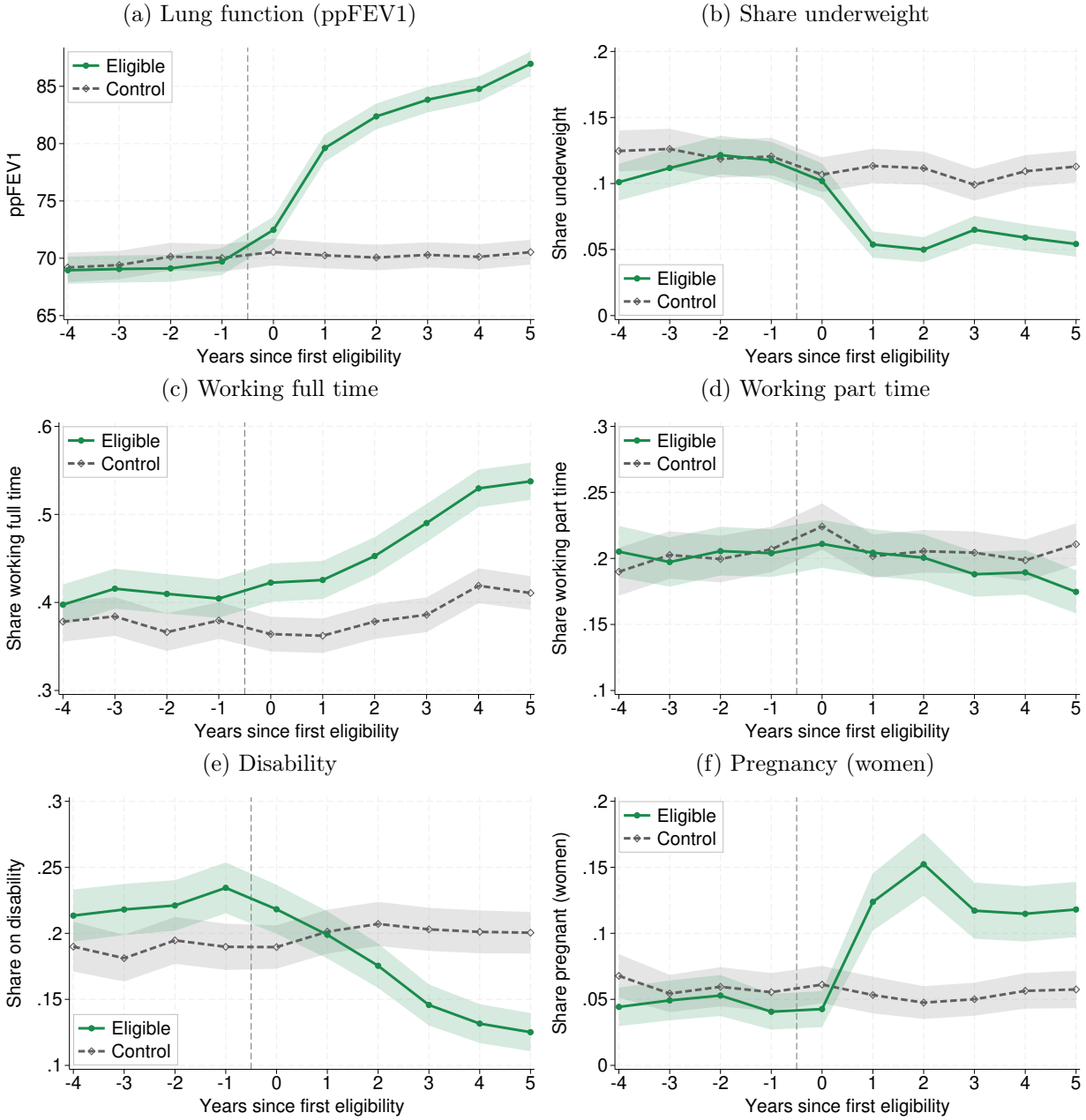
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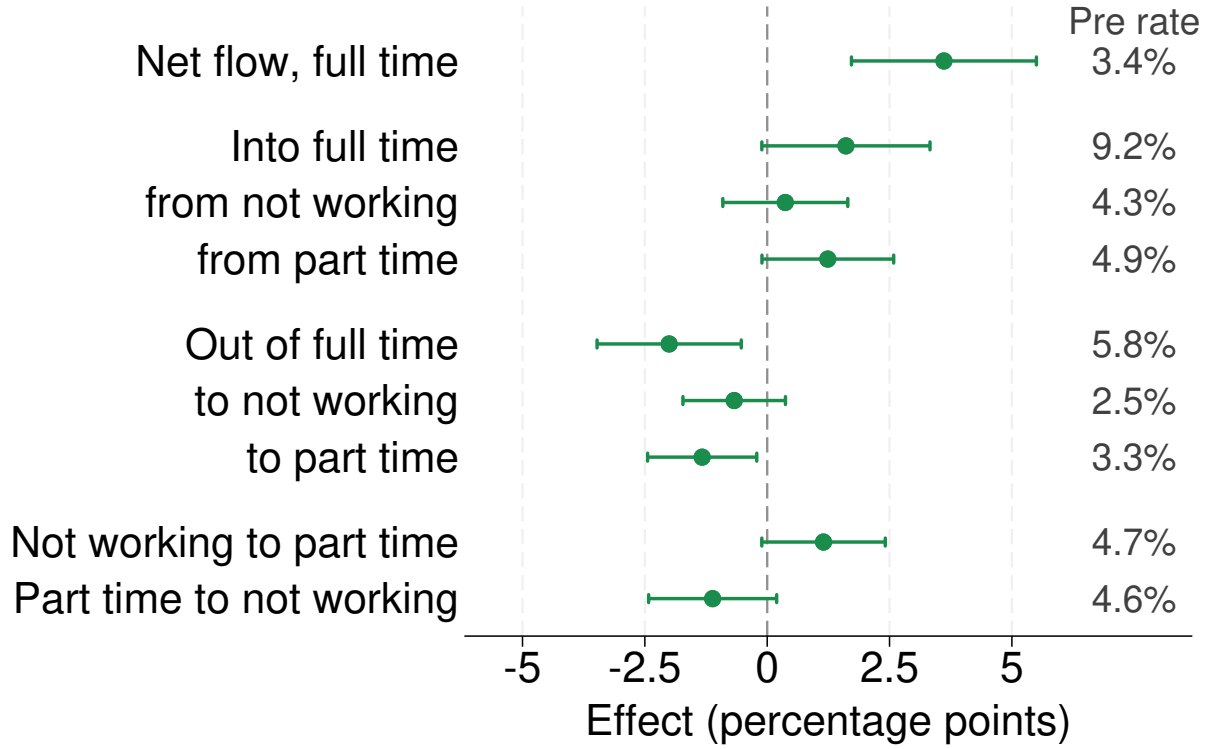
# A Appendix

Figure A.1: Eligible and control means over event time



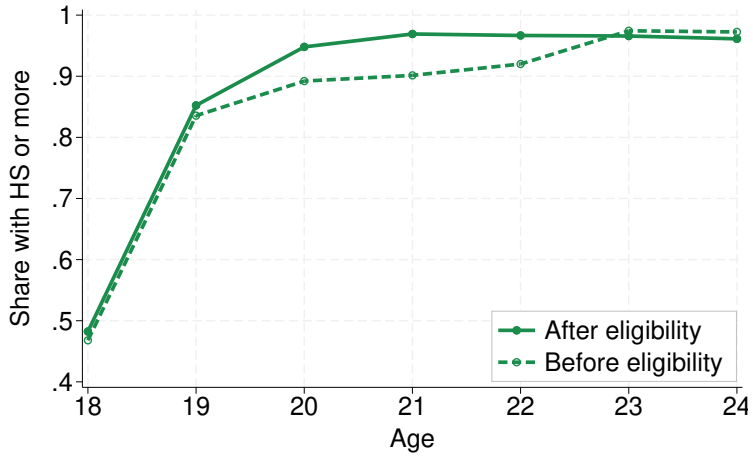

*Note*: This figure displays the trends around new eligibility for the treatment and control groups used to estimate the primary difference-in-differences specification. Individuals are age 22-30 at the time of measurement. Eligible individuals are those who become eligible for the first time for a new highly effective CFTR modulator at time 0. Control individuals either never become eligible for highly effective CFTR modulators or become eligible more than five years after the index year. 95% confidence intervals around the mean for an annual year are shaded, from standard errors clustered at the individual level.

Figure A.2: Difference-in-differences estimates: annual work transitions




*Note*: Difference-in-differences estimates for the annual probability of making a work transition are displayed for individuals aged 22-30 at the time of measurement, estimated using equation 1. Transitions between full-time, part-time, and not working are displayed, with net transitions into each domain, as well as transitions between specific domains. Eligibility leads to an increased net flow into full-time work, with reductions in transitions out of full-time work to part-time work, and positive but not statistically significant increases of transitions into full-time work from not working and part-time.

Figure A.3: Share with HS or more by age before and after eligibility: ivacaftor cohorts




*Note*: Ivacaftor-eligible patients only (gating mutations; first eligible in 2012 or 2014); no control group is shown. "Before eligibility" is the four years before first eligibility (2008–2011 for the 2012 cohort; 2010–2013 for the 2014 cohort). "After eligibility" is the year after first eligibility through 2019 (2013–2019 for the 2012 cohort; 2015–2019 for the 2014 cohort); the eligibility year itself is excluded. Each point is the share of person-years at that age (18–24) reporting HS or more, using the raw annual education reports.