GLP-1 Treatment and Work Capacity*

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Abstract

Using linked Finnish administrative records and a staggered difference-in-differences design, we estimate how starting semaglutide (Ozempic) affects work capacity, measured by medically certified sickness absence. The first purchase is followed by persistent use and improved glycemic control, indicating that recorded purchases correspond to sustained and clinically active treatment. The reduction in sickness absence is near zero during months 0–5, becomes progressively larger thereafter, and is largest during months 12–18. Averaged over event months 0–18, sickness allowance receipt falls by 16.9 percent and compensated days by 14.3 percent relative to pre-treatment means. The decline in full-day sickness absence is not offset by greater reliance on unemployment or partial sickness benefits. The results show that semaglutide improves work capacity and can generate economic benefits beyond its direct effects on metabolic health.

Keywords: semaglutide, GLP-1, blood glucose, sickness absence, social insurance, difference-in-differences, type 2 diabetes

JEL Codes: I12, I18, J21, J22

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1 Introduction

GLP-1 therapies have rapidly expanded from specialized treatments for diabetes into one of the most prominent areas of pharmaceutical innovation in recent years. Evidence of substantial effects on glycemic control and weight, together with a growing range of other health outcomes, has broadened both their use and the population for whom treatment may be relevant (Drucker, 2018; Capehorn et al., 2020). Because these treatments may be used for long periods by a large and expanding population, their diffusion raises questions about consequences for patients, public budgets, and the economy beyond clinical efficacy and health care spending (Bock et al., 2026; Wing et al., 2026). One important margin is work capacity, which can change even when employment status does not. Health may affect how regularly individuals are able to work before changing whether they hold a job. Medically certified sickness absence therefore provides a more immediate, administratively verified measure of work incapacity. Improved glycemic control and weight loss may allow patients to work more consistently, whereas gastrointestinal adverse effects during treatment initiation may temporarily make work more difficult and gains in daily functioning may emerge only gradually.[1] These offsetting forces make the short-run effect on work capacity ambiguous ex ante. Whether any improvement emerges, and how quickly, is therefore an empirical question.

We examine this question using nationwide administrative data from Finland. We focus on semaglutide, the drug that has been at the center of the GLP-1 expansion, typically marketed as Ozempic for type 2 diabetes and later as Wegovy for weight management. Ozempic became available in Finland in late 2018 and entered the national reimbursement system in 2019 under uniform clinical eligibility criteria for patients with type 2 diabetes. We link reimbursed prescription purchases to repeated laboratory measurements and monthly social-insurance records from 2018 through 2021. The analysis follows 13,848 patients aged 18–62 at their first semaglutide purchase.

Our empirical strategy uses a staggered difference-in-differences design based on variation in when patients start semaglutide. Following Callaway and Sant'Anna (2021), each monthly cohort of new users is compared with eventual users who, in the corresponding calendar month, have not yet entered their own pre-purchase window. This comparison holds common calendar shocks fixed, avoids using already-treated patients as controls, and restricts the analysis to patients who ultimately satisfy the same reimbursement criteria (Goodman-Bacon, 2021). The resulting estimand is the effect of earlier semaglutide use among eventual users, relative to not yet having started semaglutide over the same horizon.

National reimbursement criteria shape access to semaglutide in Finland. By construction, patients start treatment after inadequate glycemic control under prior therapy has been documented. Treatment may therefore begin during a period of worsening health or reduced work capacity. We account for this initiation-related movement by using event month -2 as the reference period. Identification requires that, absent earlier treatment, outcomes for earlier and later purchasers

[1] Randomized trials identify nausea, diarrhea, and vomiting as common adverse effects of semaglutide, particularly during treatment initiation, and adverse events lead some patients to discontinue treatment (Ahrén et al., 2017; Aroda et al., 2023).

would have evolved similarly over the same calendar months. We find no evidence of systematic differential pre-treatment trends in either primary sickness-absence outcome.

We first document that the initial purchase marks persistent and clinically active treatment. Package coverage remains above 80 percent eighteen months later. Among patients with a recorded test, blood glucose falls by 1.95 mmol/L, or 19.6 percent of its pre-treatment mean, and HbA1c, triglycerides, ALT, and LDL cholesterol also decline. The persistence of purchases and expected metabolic response support interpreting the first reimbursed purchase as a meaningful treatment start.

Our main result is a gradual reduction in medically certified sickness absence. The estimates are close to zero during months 0–5, become negative in months 6–11, and are largest in months 12–18. On average over event months 0 through 18, the probability of receiving full-day sickness allowance falls by 0.6 percentage points and compensated days fall by 0.101 per patient-month, corresponding to reductions of 16.9 and 14.3 percent relative to their pre-treatment means. At the annual level, our estimate corresponds to 1.21 fewer compensated sickness-absence days per patient, a magnitude comparable to the reduction following the introduction of Cox-2 inhibitors documented by Bütikofer and Skira (2018). Complementary estimates show no clear increase in partial sickness allowance or unemployment benefits, providing no indication that the decline reflects a substantial shift into alternative benefit states.

The sickness-absence findings are stable when we adjust for sex, birth cohort, and region, apply the imputation estimator of Borusyak et al. (2024), and use a panel balanced over the shorter window covering event months -4 through 12. To assess whether the pattern extends within the drug class, we apply this design over the same study period to 2,016 new users of liraglutide, an earlier GLP-1 receptor agonist also developed by Novo Nordisk. The estimates suggest a similar but less precise delayed pattern: sickness allowance receipt changes little during the first six months and declines more clearly after one year; averaged over months 0–18, it falls by 0.8 percentage points, or 18.5 percent of its pre-treatment mean. We also examine whether the reduction in sickness absence differs by sex. The point estimates are negative for both men and women, and we find no statistically detectable difference between them, although the subgroup estimates are imprecise.

This paper contributes first to research on health as human capital and on how medical treatment affects labor-market outcomes (Grossman, 1972; Currie and Madrian, 1999; Dobkin et al., 2018). Pharmaceutical innovations for chronic pain, mental illness, and HIV can increase labor supply or reduce sickness absence and disability (Garthwaite, 2012; Bütikofer and Skira, 2018; Shapiro, 2022; Habyarimana et al., 2010). Related work shows that access to medical care affects earnings, absence, and disability receipt (Stephens and Toohey, 2022; Godøy et al., 2024), while Papageorge (2016) formalizes medication and labor supply as joint investments in health and work experience. We add evidence on a widely used treatment for a prevalent chronic disease and on a margin, medically certified work incapacity, that can respond before employment status changes.

Second, we contribute to the rapidly growing economics literature on GLP-1 therapies. Existing work examines treatment adoption and weight loss, whether drug costs are offset by lower medical

spending, and effects on food demand, self-reported health, employment, and family formation (Bock et al., 2026; Wing et al., 2026; Hristakeva et al., 2026; Kaestner and Schiman, 2026; Diamond, 2026). We study a distinct labor-market margin: medically certified work incapacity. This high-frequency measure captures changes in patients' ability to work that may arise before employment status or earnings adjust, providing new evidence on the broader economic returns to GLP-1 treatment.

Section 2 describes the institutional setting and data. Section 3 presents the empirical strategy. Sections 4 and 5 report the main and additional results. Section 6 concludes.

2 Institutional Setting and Data

2.1 Institutional Setting

Semaglutide is a GLP-1 receptor agonist that mimics the action of an incretin hormone released after eating. GLP-1 therapies increase glucose-dependent insulin secretion, suppress glucagon, slow gastric emptying, and reduce appetite and energy intake (Drucker, 2022; Blundell et al., 2017). Semaglutide is administered once weekly and, in head-to-head trials, produces larger average reductions in blood glucose and body weight than the earlier, once-daily GLP-1 therapy liraglutide (Capehorn et al., 2020).

Our study focuses on Finland, where access to reimbursed semaglutide during the study period was governed by a nationwide prescription-medicine reimbursement system. Administered by the Social Insurance Institution of Finland (Kela), the system applies common rules across the country. Reimbursement requires a prescription for an eligible condition and, for restricted medicines, a physician's statement documenting that the patient satisfies the relevant medical criteria (Kela, 2026b). Injectable semaglutide became eligible for restricted basic reimbursement on January 1, 2019 (Kela, 2019). During our study period, eligibility required type 2 diabetes, inadequate glycemic control under prior antidiabetic medication, and a body-mass index of at least 30 kg/m^2. The reimbursement population therefore consists of patients with established diabetes and elevated metabolic risk whose previous medication had not achieved adequate glycemic control.[2]

Kela's full-day sickness allowance replaces income during a medically certified period of incapacity for work. The allowance is income-related, with a minimum payment for residents with little or no prior earnings. It generally begins after the first day of illness and the following nine working days and can continue for up to 300 working days (Kela, 2026c). Employers typically continue paying wages during the waiting period and may receive the allowance directly once a spell becomes eligible. Coverage also extends to eligible unemployed, self-employed, and student residents. Full-day sickness allowance receipt therefore captures medically certified work incapacity across labor-market states rather than absence among employees alone. Finland also provides partial sickness allowance to people who cannot work full time but can continue on reduced hours.

[2]Basic reimbursement covered 40 percent of the reimbursable price. On February 1, 2020, semaglutide became eligible for the 65-percent special reimbursement rate (Lääkkeiden hintalautakunta, 2019; Kela, 2026a).

Based on a physician's assessment and agreement with the employer, recipients reduce their hours to 40–60 percent of full-time hours and receive half the regular sickness allowance.[3]

Because the allowance begins only after the waiting period and requires medical certification, it does not capture all illness or every day absent from work. We therefore interpret it as a measure of compensated work incapacity, not morbidity or total absence.[4]

Unemployment benefits provide a useful complementary outcome. Eligibility requires registration as a jobseeker and the capacity and availability to work. The system includes basic support administered by Kela and earnings-related allowances administered by unemployment-insurance funds. Full-day sickness allowance and unemployment benefits thus represent distinct states: certified incapacity for work versus availability for work and active job search. The programs cannot compensate the same day, although an individual may move between the two states within a month. Examining both outcomes indicates whether lower compensated sickness absence is accompanied by greater unemployment benefit receipt.

2.2 Data

We construct an individual-level monthly panel covering 2018–2021 from FinRegistry, a nationwide resource that links Finnish health and administrative registers (Viippola et al., 2023). Using pseudonymized individual identifiers, we combine four sources: the Kela register of reimbursed pharmacy purchases; the unpaid-periods register of the Finnish Centre for Pensions (ETK), which records compensated sickness and unemployment days reported by Kela and unemployment-insurance funds; the Kanta laboratory register; and the Population Information System of the Digital and Population Data Services Agency (DVV). Together, these sources measure treatment, work incapacity, unemployment benefit receipt, metabolic health, and demographic characteristics in the same population.

Treatment begins in the month of the first reimbursed semaglutide purchase observed in the Kela data. Prescriptions or dispensations outside the reimbursement system are therefore not observed as starts. During most of the study period, reimbursed semaglutide was supplied principally as Ozempic, an injectable once-weekly pen. A standard package contains four weekly doses and provides approximately one month of treatment. Between January 2019 and December 2021, 28,942 individuals made a first reimbursed purchase. We restrict the analysis to patients aged 18–62 at that purchase, leaving 13,848 patients: 7,298 men and 6,550 women, with a mean age of 52. This restriction fixes a common sample across outcomes and keeps patients within working age throughout the panel. The outcome panel begins in January 2018, providing pre-treatment observations for every cohort, and ends in December 2021.

Our primary outcomes come from compensated benefit days in the ETK unpaid-periods register, which classifies spells by benefit type. We use benefit-type code 120 (sickness daily allowance) to

[3] See the Social Insurance Institution of Finland, `https://www.kela.fi/partial-sickness-allowance`.

[4] Previous evidence from Finland shows that sickness absence responds to sick-pay incentives (Böckerman et al., 2018).

construct the indicator for any full-day sickness allowance and its number of compensated days, and code 121 (partial sickness daily allowance) to construct the corresponding receipt and compensated-day measures.[5] We analyze partial sickness allowance separately to determine whether lower full-day absence coincides with partially compensated work. For unemployment benefits, we combine codes 150 and 151, the basic unemployment allowance and labor-market subsidy administered by Kela, with codes 210 and 220, the earnings-related allowances for employees and self-employed workers.

The Kanta laboratory register provides repeated measures of metabolic health among tested patients. Our primary clinical marker is blood glucose, which measures glycemia at the time of the test and aligns directly with the monthly timing of the event study, although it is sensitive to meals, test timing, and short-run fluctuations. We also examine glycated hemoglobin (HbA1c), which summarizes average glycemic control over the preceding two to three months, as well as LDL cholesterol, triglycerides, and alanine aminotransferase (ALT). For each marker, we use the most recent measurement recorded within a calendar month, harmonize reported units, and remove physiologically implausible values.[6]

Laboratory outcomes are observed only when a test is recorded. Within the event window, 79.7 percent of the laboratory-linked sample have at least one usable blood-glucose measurement and 89.2 percent have at least one usable HbA1c measurement. Measurements on both sides of the first purchase are available for 42.5 percent of patients for blood glucose and 61.5 percent for HbA1c. Blood glucose is recorded in 10.4 percent of pre-treatment person-months and 8.3 percent of post-treatment person-months; the corresponding rates for HbA1c are 13.4 and 12.2 percent. Because testing occurs periodically as part of clinical care, the laboratory estimates describe the clinical response among patients tested in a given month rather than an unconditional population mean. Measurement frequencies for LDL cholesterol, triglycerides, and ALT follow similar patterns.

We also collect a small set of predetermined individual characteristics: sex, birth cohort, and region of residence. Appendix Table A1 reports descriptive statistics for the labor-market and laboratory outcomes, overall and by sex.

3 Empirical Strategy

To estimate the causal effect of semaglutide initiation on work capacity, the ideal experiment would randomly assign eligible patients either to start semaglutide or to continue their previous diabetes care over the same calendar months. We approximate this comparison using variation in the timing of first purchases among eventual users. For each cohort of new users, patients who purchase later form the comparison group while they remain on prior care. Restricting the analysis to eventual users makes the groups more comparable in clinical eligibility, access to a prescriber, and willingness to use the drug. Our estimand is therefore the effect of starting semaglutide at a given date rather

[5] The benefit-type codes are documented in the register description published by the Finnish Centre for Pensions.

[6] HbA1c is reported in both percent and mmol/mol. We convert percentage readings to mmol/mol using the master equation $\text{IFCC} = 10.93 \times \text{NGSP} - 23.5$ (Hoelzel et al., 2004) and retain the harmonized mmol/mol series.

than remaining on prior diabetes care through the same horizon, among patients who eventually initiate treatment.

Following Callaway and Sant'Anna (2021), we implement this delayed-start design in a group-time framework with later purchasers serving as not-yet-treated controls.[7] The estimator constructs a separate comparison for each first-purchase cohort g and calendar month t. It never uses already-treated patients as controls and allows effects to vary across cohorts and treatment horizons (Goodman-Bacon, 2021; de Chaisemartin and D'Haultfœuille, 2020).

For each cohort and horizon, we implement the delayed-start comparison as a two-group, two-period difference-in-differences. At a post-treatment date $t = g + e$, the comparison group contains patients whose first purchase occurs after $t+1$ and who thus remain outside their own pre-purchase window. For example, the six-month effect for the January 2020 cohort compares its change between November 2019 and July 2020 with the change over those same months among patients who first purchase in September 2020 or later. For $e \geq 0$, the cohort-specific comparison, where G_i denotes individual i's month of first purchase, is

$$\begin{aligned} \mathrm{DID}_{g,e} = &\{E[Y_{i,g+e} \mid G_i = g] - E[Y_{i,g-2} \mid G_i = g]\} \\ &- \{E[Y_{i,g+e} \mid G_i > g + e + 1] - E[Y_{i,g-2} \mid G_i > g + e + 1]\}. \end{aligned} \tag{1}$$

The first bracket is the outcome change for patients who start in g; the second is the change over the same calendar months among patients who remain outside their own pre-purchase window. Because both changes use the same calendar interval, the comparison absorbs national shocks shared by the groups, including changes in health care, social-insurance receipt, and the COVID-19 pandemic.

The reimbursement process motivates our choice of reference period. Eligibility requires documented inadequate glycemic control under previous medication, clinical reassessment, a physician statement, and a prescription, whereas the drug register records treatment only from the first pharmacy purchase. The clinical and administrative steps leading to treatment therefore begin before the observed purchase date and may already affect outcomes in month $g - 1$. For example, worsening health may both increase sickness absence and prompt the reassessment or documentation required for reimbursement. We use $g - 2$ as the reference period, leaving the immediate pre-purchase movement in $g - 1$ visible in the event study while excluding it from the post-treatment contrast.

Restricting the comparison to eventual users defines a population that ultimately qualifies for reimbursement, obtains a prescription, and purchases semaglutide, excluding patients who may never meet the clinical criteria or proceed to treatment. Within this population, identification comes from differences in when treatment starts. The clinical eligibility criteria are defined nationwide and do not mechanically depend on the work-capacity outcomes we study, but later purchasers may not yet satisfy them when they serve as controls, and differences in clinical progression can therefore

[7] Recent GLP-1 studies use related delayed-start comparisons. Wing et al. (2026) combines cohort-specific comparisons in a stacked difference-in-differences design, while Diamond (2026) compares women who start a GLP-1 for weight loss with matched women who would like to start but have not yet done so. We estimate group-time effects for semaglutide and use all later purchasers who remain outside their own pre-purchase window at the relevant horizon.

affect treatment timing. Identification requires that these differences do not generate divergent counterfactual changes in sickness absence. Under this parallel-trends restriction, $\text{DID}_{g,e}$ identifies the effect of starting treatment earlier for cohort g at horizon e. Cohorts may differ permanently in outcome levels, but not in their counterfactual changes after the common reference month, and comparisons over the same calendar months hold common policy, health care, and pandemic shocks fixed. The unadjusted specification imposes parallel trends on average, while the adjusted specification imposes it conditional on predetermined characteristics. We assess the restriction using the event-study coefficients for months -12 through -3, covariate-adjusted specifications, and the stability of the results across alternative estimators and samples. We additionally assume that treatment does not affect the outcomes of other patients.

We aggregate the group-time effects by event month using cohort-size weights. The event study spans months -12 through 18, with -2 omitted. Coefficients for months -12 through -3 assess differential movements before the initiation process, the coefficient at -1 displays the immediate pre-purchase movement, and the post-treatment averages cover the 19 event months from 0 through 18. The baseline specification is unadjusted. A second specification uses the doubly robust inverse-probability-weighted procedure of Sant'Anna and Zhao (2020) to condition on predetermined sex, birth cohort, and region.

Because the panel ends in December 2021, not every cohort contributes at every horizon. Longer-run estimates consequently draw on fewer treated individuals and place more weight on earlier first-purchase cohorts.[8]

We assess robustness using the covariate-adjusted Callaway–Sant'Anna specification, a panel balanced over event months -4 through 12, and the imputation estimator of Borusyak et al. (2024); see also Baker et al. (2026). For the imputation estimator, the untreated-outcome model is fit through $g-2$. Laboratory outcomes require an additional measurement assumption because they are observed only in months with a recorded test: changes in testing across cohorts must not account for the estimated biomarker response. In Section 2 we report testing frequencies and the share of patients observed on both sides of the first purchase. Finally, standard errors are clustered at the individual level throughout.

4 Main Results

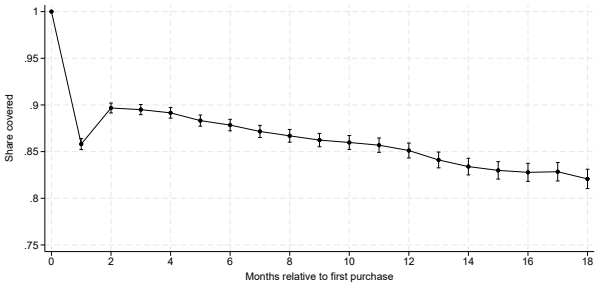
4.1 Treatment Persistence and Clinical Response

Figure 1, panel (a), reports treatment coverage after the first semaglutide purchase. Coverage equals one in month 0 by construction and remains above 80 percent in month 18. Persistence is substantially higher than that reported for broad samples of GLP-1 initiators in US commercial

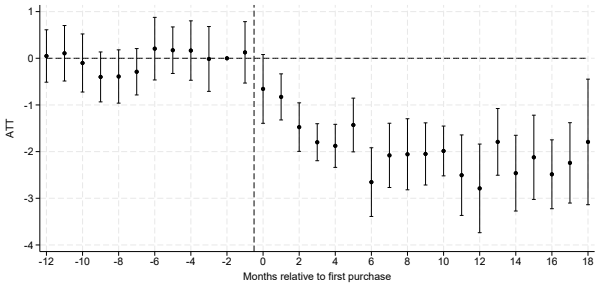
[8] Appendix Figure A1 documents this changing support. At event month 0, the estimate draws on 12,674 treated individuals from 34 monthly cohorts and 13,735 distinct eligible later purchasers; at month 18, it draws on 4,431 treated individuals from 16 cohorts and 7,994 distinct eligible later purchasers. Because the same later purchaser may enter several underlying cohort-time comparisons, the control count is not an effective denominator for the aggregated estimate.

Figure 1: Treatment persistence and glycemic response

(a) Semaglutide treatment coverage



(b) Blood glucose



Notes: Panel (a) reports the share of new users covered by purchased packages at event times 0 through 18. Panel (b) plots dynamic group-time average treatment effects on blood glucose in mmol/L, conditional on a laboratory test in the month. It compares each cohort of new semaglutide users with patients who have not yet started in the same calendar month. Event month −2 is the omitted reference. Bars are 95 percent confidence intervals based on standard errors clustered at the individual level. Appendix Table A2 summarizes the clinical estimates.

claims, although differences in populations, reimbursement, and coverage definitions prevent a direct comparison (Wing et al., 2026).

Panel (b) documents the clinical response. Blood glucose begins to fall after the first purchase and remains below its pre-treatment level through event month 18.

Appendix Table A2 summarizes the response across five laboratory markers. Over months 0–18, blood glucose falls by 1.952 mmol/L and HbA1c by 12.164 mmol/mol, equivalent to 19.6 and 19.1 percent of their respective pre-treatment means. ALT and triglycerides decline by approximately 20 percent, while LDL cholesterol falls by 5.7 percent. The size and breadth of these changes are consistent with a meaningful improvement in metabolic health among tested patients.

The pre-treatment biomarker paths clarify how patients enter treatment. Joint tests for months −12 through −3 do not reject the null of no differential pre-treatment changes for blood glucose, ALT, triglycerides, or LDL cholesterol, with p-values of 0.718, 0.324, 0.746, and 0.208. HbA1c is the exception, with a p-value of 0.001. This pattern is consistent with the eligibility process: inadequate glycemic control directly determines reimbursement, and HbA1c summarizes glucose levels over the preceding two to three months. The HbA1c movement is therefore consistent with clinical selection into treatment rather than serving as a placebo test. The other markers show no corresponding broad deterioration well before purchase.

Taken together, the persistence and laboratory response indicate that the first reimbursed purchase marks the start of sustained, clinically active treatment. This evidence does not identify the mechanism behind any change in sickness absence: glycemic control, weight loss, cardiovascular risk, adverse effects, and other health channels may all matter. Identification of the work capacity effects instead rests on comparisons in the work capacity outcomes themselves. The laboratory estimates are also conditional on a recorded test. Appendix Figure A2 plots the event studies for HbA1c, ALT, triglycerides, and LDL cholesterol.

4.2 Work Capacity

Figure 2 reports the event studies for the two primary measures of work capacity: receipt of full-day sickness allowance and the number of compensated days. Table 1 reports pre-treatment means, joint tests of the early pre-treatment coefficients, the average effects over months 0–18, and separate averages for months 0–5, 6–11, and 12–18.

Sickness absence falls on both margins. Over months 0–18, the probability of receiving any full-day sickness allowance declines by 0.6 percentage points, or 16.9 percent of its pre-treatment mean of 3.4 percent. Compensated full-day sickness allowance days decline by 0.101 per patient-month, or 14.3 percent of the pre-treatment mean of 0.708 days. The receipt estimate is statistically significant at the 5 percent level, while the estimate for compensated days is statistically significant at the 10 percent level.

The response develops gradually. For receipt, the average effects are -0.001, -0.005, and -0.010 over months 0–5, 6–11, and 12–18. The corresponding effects on compensated days are 0.008, -0.098, and -0.199. Thus, we detect little change during the first six months; the decline appears in the second six months and is largest after one year. The joint tests for months -12 through -3 have p-values of 0.164 and 0.379, and the individual coefficients show no systematic differential movements in the primary outcomes before the initiation window.

Sickness absence does rise within the initiation window itself. Compensated days increase by 0.052 in month -1 and by 0.100 in month 0, a pattern consistent with the clinical contact, documentation, and prescription that precede a reimbursed purchase. Month -1 is estimated separately and excluded from the post-treatment averages, whereas month 0 marks the first purchase and is included in them. The shape of the subsequent path is also informative. A transitory health episode around initiation would produce its largest apparent improvement in the months immediately afterwards and then fade, whereas these estimates are flat through month 5 and grow steadily from month 6 onward.

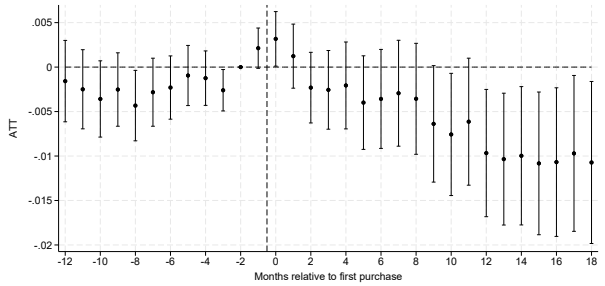
The estimates imply approximately 6 fewer recipients per 1,000 patients in a month and 1.21 fewer compensated sickness days per patient-year. The 14.3 percent decline in days is comparable to the 7–11 percent reduction associated with access to Cox-2 inhibitors in Norway (Bütikofer and Skira, 2018).

The delayed response is also economically informative. Clinical markers improve relatively soon after initiation, whereas sickness absence changes only after several months. This timing is consistent with work capacity responding to accumulated health improvements rather than immediately to a pharmacy purchase. Delayed absence responses have also been documented following antiretroviral treatment and improved access to surgery (Habyarimana et al., 2010; Godøy et al., 2024). Our data cannot determine whether glycemic control, weight loss, reduced complications, or another channel produces the decline, and the early absence response may also be muted by treatment titration or adverse effects.

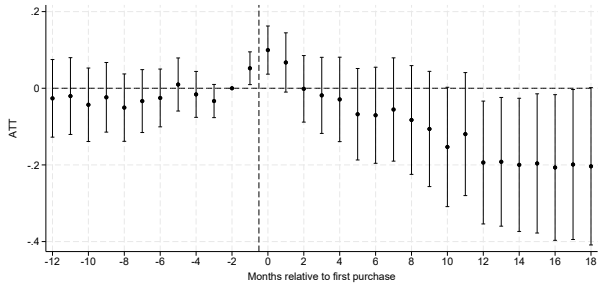
We next examine whether the decline in full-day sickness allowance is accompanied by movement into alternative benefit states. Appendix Figure A3 and Appendix Table A3 report unemployment

Figure 2: Semaglutide and medically certified sickness absence

(a) Any full-day sickness allowance



(b) Full-day sickness allowance days



Notes: Event-study estimates around the first semaglutide purchase. Panel (a) reports the probability of full-day sickness allowance receipt and panel (b) reports the number of compensated days. Both panels use the Callaway–Sant'Anna estimator with patients who start later serving as not-yet-treated controls. Event month −2 is the omitted reference. Bars are 95 percent confidence intervals based on standard errors clustered at the individual level. Appendix Table A7 reports the underlying coefficients.

benefits and partial sickness allowance together. We find no clear offsetting increase in either program. Over months 0–18, the unemployment estimates are approximately zero for receipt and −0.040 for compensated days. The partial-sickness estimates are 0.001 for receipt and 0.020 for compensated days and are statistically imprecise. Taken together, the estimates provide no indication that the reduction in full-day sickness absence reflects a substantial shift into unemployment or partially compensated work.

The estimates measure compensated work incapacity across the labor-market states represented among semaglutide users; they are not effects on hours or employment among employees alone. This distinction helps reconcile our findings with evidence of limited employment responses to GLP-1 use (Kaestner and Schiman, 2026). Medically certified absence is a high-frequency margin that can change without immediate job entry or exit.

4.3 Robustness

We assess the sickness-absence results using three checks reported in Appendix Figure A6 and Appendix Table A6. First, the covariate-adjusted group-time estimator conditions each comparison between earlier and later purchasers on predetermined sex, birth cohort, and region using doubly robust inverse-probability weighting. This procedure combines an outcome model for untreated changes with propensity-score reweighting, allowing parallel trends to hold conditional on these characteristics and addressing differences in observed demographic composition. Second, the imputation estimator of Borusyak et al. (2024) estimates the untreated outcome process from untreated observations, imputes the untreated potential outcome after initiation, and averages the resulting differences by event time. Agreement with this estimator shows that the dynamic pattern is preserved under a different construction of the counterfactual outcome and aggregation of treatment effects. Third, the balanced-panel specification retains only patients whose outcomes are observed at every event month from −4 through 12. It therefore estimates the medium-run path using

Table 1: Effects of starting semaglutide on work capacity

	Any full-day sickness allowance (1)	Full-day sickness allowance days (2)
ATT, months 0–18	-0.006**	-0.101*
	(0.003)	(0.059)
ATT, months 0–5	-0.001	0.008
	(0.002)	(0.042)
ATT, months 6–11	-0.005*	-0.098
	(0.003)	(0.068)
ATT, months 12–18	-0.010**	-0.199**
	(0.004)	(0.086)
Effect/mean, months 0–18 (%)	-16.9	-14.3
Pre-treatment mean	0.034	0.708
Joint pretrend test, p-value	0.164	0.379
Person-month observations	664,704	664,704
Individuals	13,848	13,848

Notes: Event month -2 is the omitted reference. The pretrend row reports the joint Wald-test p-value for event months -12 through -3. Standard errors clustered at the individual level are in parentheses. Significance levels: $^{*}p < 0.10$, $^{**}p < 0.05$, and $^{***}p < 0.01$.

cohorts that can contribute throughout a common event window, addressing changes in sample composition created by the finite follow-up period. All three specifications use month -2 as the omitted reference.

The results are stable across these alternatives. Over months 0–18, covariate adjustment gives estimates of -0.007 for receipt and -0.126 for compensated days, close to the baseline estimates of -0.006 and -0.101. The corresponding imputation estimates are -0.004 and -0.100. The balanced-panel estimates reproduce the gradual decline through month 12; because this specification ends at month 12, it does not assess the estimates for the final six months. These checks strengthen the main evidence along three dimensions—observed composition, estimation method, and event-time support—while continuing to rely on the same parallel-trends assumption.

5 Additional Results

Taken together, the main results show that persistent semaglutide use is followed by objective metabolic improvement and a delayed reduction in medically certified work incapacity. The additional analyses examine whether this pattern is also visible for an earlier GLP-1 therapy and whether it differs by sex.

5.1 Another GLP-1 Therapy: Liraglutide

In an additional analysis, we apply the same design to new users of liraglutide, an earlier, daily-dosed GLP-1 therapy observed in the same Finnish reimbursement records and over the same study period. This comparison provides within-class evidence on whether the work capacity pattern observed for semaglutide extends to another GLP-1 treatment. Randomized evidence shows that both treatments improve glycemic control and body weight, although the average clinical response is larger for semaglutide (Capehorn et al., 2020). We compare each monthly cohort of new liraglutide users with patients who purchase liraglutide later.

Appendix Figure A5 and Appendix Table A5 provide suggestive evidence of a later-emerging decline in sickness allowance receipt. Over months 0–18, receipt falls by 0.8 percentage points, or 18.5 percent of its pre-treatment mean, while compensated days fall by 0.040 per month, or 4.4 percent. The corresponding estimates for months 0–5, 6–11, and 12–18 are 0.002, −0.007, and −0.018 for receipt and 0.165, −0.005, and −0.246 for days. The full-period estimates are imprecise, and only the late receipt estimate is statistically distinguishable from zero. We therefore view the liraglutide estimates as within-class evidence rather than a replication of the semaglutide result.

5.2 Heterogeneity by Sex

We next examine whether the average effects conceal economically relevant heterogeneity by sex. This is particularly relevant because previous GLP-1 evidence on employment has emphasized gains among initially non-employed women (Diamond, 2026), while administrative evidence documents systematic differences in sickness absence between men and women that are not fully explained by objective health (Avdic and Johansson, 2017). We estimate the work capacity and clinical outcomes separately for men and women. Appendix Figure A4 plots the underlying sickness-absence paths, while Appendix Table A4 reports the post-treatment averages and tests equality between the two groups.

Averaged over months 0–18, the estimated effects on sickness allowance receipt are −0.005 for men and −0.007 for women; the corresponding effects on compensated days are −0.045 and −0.167. Both estimates for women are statistically distinguishable from zero—at the 10 percent level for receipt and the 5 percent level for compensated days—whereas the corresponding estimates for men are not. The dynamic profiles are nevertheless similar: for both sexes, the estimates are small during the first six months and become more negative at longer horizons. Moreover, the estimated differences between men and women are not statistically distinguishable from zero, with equality-test p-values of 0.776 for receipt and 0.299 for compensated days. The difference in the significance of the subgroup estimates therefore does not itself provide evidence of heterogeneous treatment effects. The equality tests for the five laboratory markers, reported in the same table, likewise show no statistically detectable differences by sex. These results complement work on employment entry among women by examining medically certified work incapacity among men and women with type 2 diabetes (Diamond, 2026).

6 Conclusions

Using Finnish administrative records and differences in treatment timing among eventual users, we estimate the effect of semaglutide on medically certified work incapacity. The first reimbursed purchase is followed by sustained treatment coverage and substantial metabolic improvements, while the sickness-absence response emerges more gradually. Estimates are close to zero during months 0–5, become negative during months 6–11, and are largest in months 12–18. Averaged over event months 0–18, full-day sickness allowance receipt falls by 16.9 percent and compensated days by 14.3 percent relative to their pre-treatment means. We find no clear offsetting movement into unemployment benefits or partial sickness allowance, and the main estimates are stable across covariate adjustment, an alternative estimator, and a balanced panel. The liraglutide results provide suggestive evidence of a similar delayed pattern, and the semaglutide estimates are negative for both men and women, although these additional estimates are less precise.

The delayed response highlights a dimension of pharmaceutical value that may be missed by analyses limited to clinical outcomes, health care spending, or employment status. Medically certified sickness absence captures a high-frequency, administratively verified margin of work capacity that can improve without an immediate change in labor-market attachment. At the same time, the design relies on comparable counterfactual changes among earlier and later purchasers. The absence of systematic differential pre-treatment movements in the two primary sickness-absence outcomes, together with the robustness exercises, supports this restriction. The data also do not isolate the underlying mechanism, measure every episode of absence, or establish effects on employment, hours, or net social value. Longer follow-up linking treatment to benefit payments, employment, earnings, and health care use is needed for a complete fiscal and welfare assessment. Our findings nevertheless indicate that the gains from effective diabetes treatment can extend beyond metabolic health to a reduction in medically certified work incapacity.

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Online Appendix

A Summary Statistics

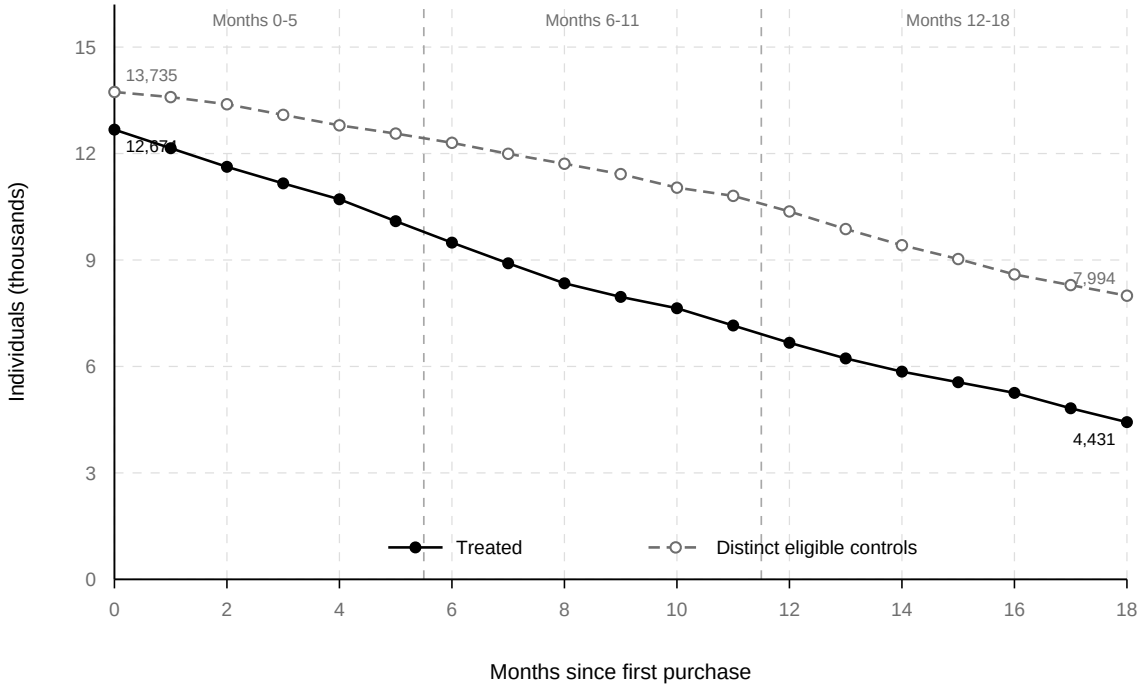
Table A1: Summary statistics

	Mean (standard deviation)			Nonmissing observations	
Variable	All	Men	Women	Person-months	Individuals
Panel A. Labor and social-insurance outcomes					
Any full-day sickness allowance	0.033	0.034	0.032	664,704	13,848
	(0.179)	(0.182)	(0.175)		
Full-day sickness allowance days	0.690	0.726	0.649	664,704	13,848
	(3.982)	(4.093)	(3.855)		
Any partial sickness allowance	0.003	0.002	0.005	664,704	13,848
	(0.057)	(0.042)	(0.070)		
Partial sickness allowance days	0.064	0.035	0.097	664,704	13,848
	(1.202)	(0.889)	(1.474)		
Any unemployment benefit	0.151	0.151	0.151	664,704	13,848
	(0.358)	(0.359)	(0.358)		
Unemployment benefit days	3.050	3.035	3.066	664,704	13,848
	(7.445)	(7.422)	(7.470)		
Panel B. Clinical outcomes					
HbA1c (mmol/mol)	59.758	60.826	58.639	71,935	12,951
	(16.145)	(16.173)	(16.040)		
Blood glucose (mmol/L)	9.280	9.451	9.106	54,801	12,383
	(3.379)	(3.455)	(3.292)		
ALT (U/L)	47.043	51.117	42.983	68,925	13,174
	(40.733)	(45.488)	(34.894)		
Triglycerides (mmol/L)	2.703	2.924	2.448	40,041	11,989
	(2.885)	(3.003)	(2.720)		
LDL cholesterol (mmol/L)	2.578	2.437	2.735	46,781	12,758
	(0.989)	(0.950)	(1.008)		
Panel C. Demographic characteristics					
Age at first purchase	52.781	53.309	52.194	664,704	13,848
	(7.888)	(7.444)	(8.314)		
Female	0.473	–	–	664,704	13,848
	(0.499)				

Notes: Standard deviations are reported in parentheses. Laboratory statistics are conditional on a usable measurement. Person-months and individuals report nonmissing observations in the full sample; sex-specific sample sizes are 350,304 person-months and 7,298 men, and 314,400 person-months and 6,550 women.

B Sample Support for the Event-Study Estimates

Figure A1: Event-study support by event month



Notes: The solid line reports treated individuals in monthly first-purchase cohorts that contribute to the unadjusted event-time estimate at each horizon. The dashed line reports distinct later purchasers eligible as not-yet-treated controls in at least one underlying cohort-time comparison. The Callaway–Sant'Anna estimator constructs separate $ATT(g,t)$ comparisons whose control sets vary across treatment cohorts, so the same later purchaser may enter several comparisons. The dashed series is therefore neither the number of control observations nor an effective denominator for the aggregated estimate, and the two series should not be added or interpreted as a treated-to-control ratio. The decline over event time reflects the finite 2018–2021 observation window and the requirement that controls remain outside their own one-month pre-purchase window, rather than attrition from the administrative panel. The figure documents sample support; it does not assess covariate overlap or the parallel-trends assumption.

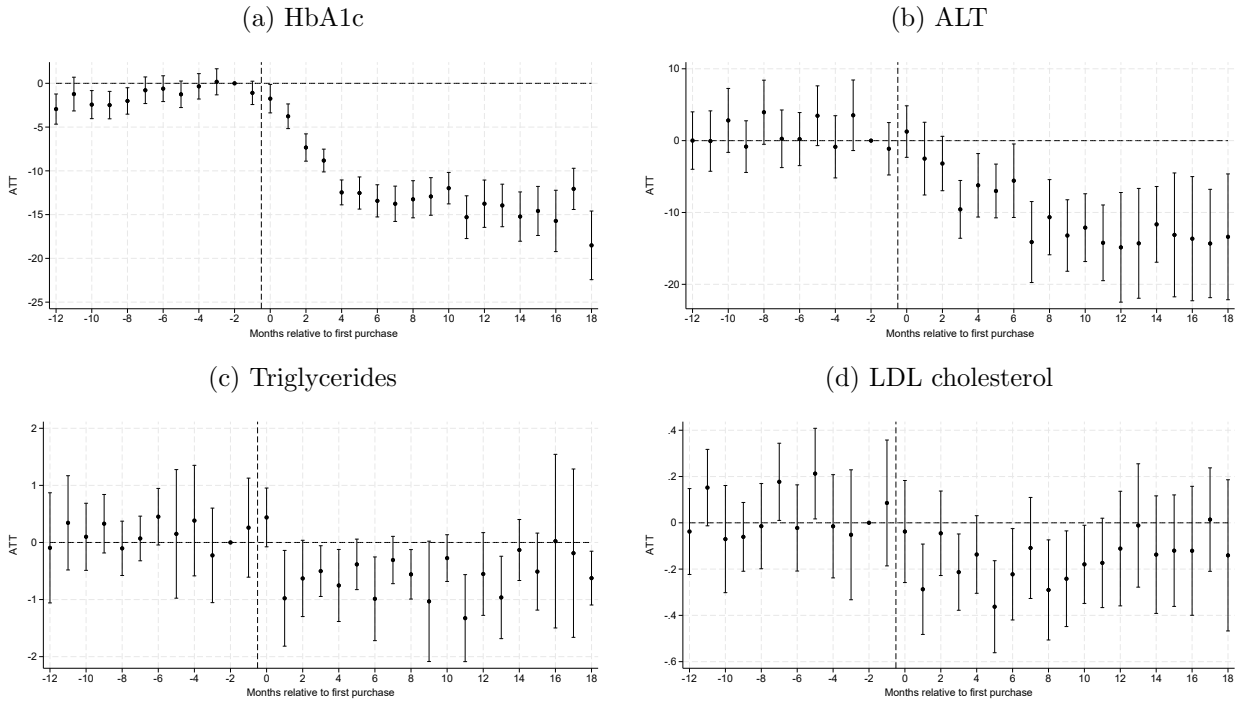
C Health Markers

Table A2: Effects of starting semaglutide on laboratory markers

	HbA1c (mmol/mol) (1)	Blood glucose (mmol/L) (2)	ALT (U/L) (3)	Triglycerides (mmol/L) (4)	LDL (mmol/L) (5)
ATT, months 0–18	-12.164***	-1.952***	-10.126***	-0.539***	-0.154***
	(0.439)	(0.134)	(1.447)	(0.121)	(0.034)
ATT, months 0–5	-7.778***	-1.344***	-4.536***	-0.468***	-0.180***
	(0.407)	(0.136)	(1.237)	(0.149)	(0.043)
ATT, months 6–11	-13.440***	-2.222***	-11.650***	-0.747***	-0.203***
	(0.552)	(0.181)	(1.540)	(0.159)	(0.047)
ATT, months 12–18	-14.829***	-2.240***	-13.611***	-0.421**	-0.090
	(0.714)	(0.217)	(2.466)	(0.200)	(0.056)
Effect/mean, months 0–18 (%)	-19.1	-19.6	-20.3	-18.4	-5.7
Pre-treatment mean	63.568	9.938	49.957	2.931	2.680
Joint pretrend test, p-value	0.001	0.718	0.324	0.746	0.208
Person-month observations	71,935	54,801	68,925	40,041	46,781
Individuals	12,951	12,383	13,174	11,989	12,758

Notes: Laboratory outcomes are conditional on a usable measurement in the month. Event month -2 is the omitted reference. The pretrend row reports the joint Wald-test p-value for event months -12 through -3. Standard errors clustered at the individual level are in parentheses. Significance levels: $^{*}p < 0.10$, $^{**}p < 0.05$, and $^{***}p < 0.01$.

Figure A2: Laboratory markers around semaglutide initiation

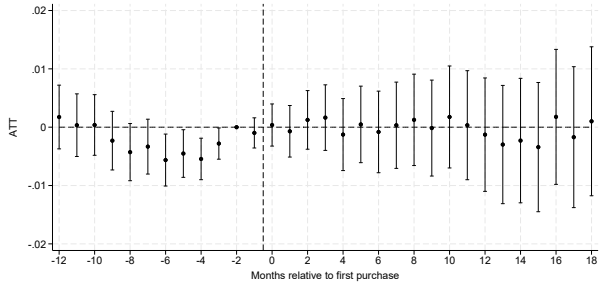


Notes: Event-study estimates for HbA1c (mmol/mol), ALT (U/L), triglycerides (mmol/L), and LDL cholesterol (mmol/L) around the first semaglutide purchase. Event month -2 is the omitted reference. Bars are 95 percent confidence intervals based on standard errors clustered at the individual level. Laboratory estimates are conditional on a recorded measurement; coverage by marker is reported in Section 2.

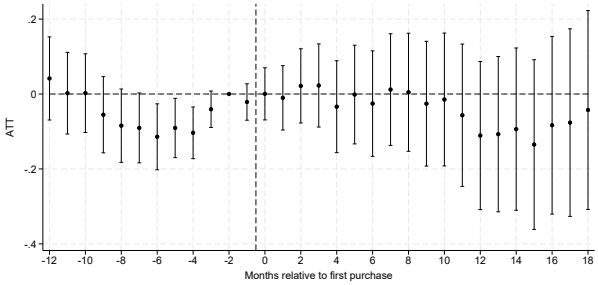
D Alternative Benefit States

Figure A3: Alternative benefit states after starting semaglutide

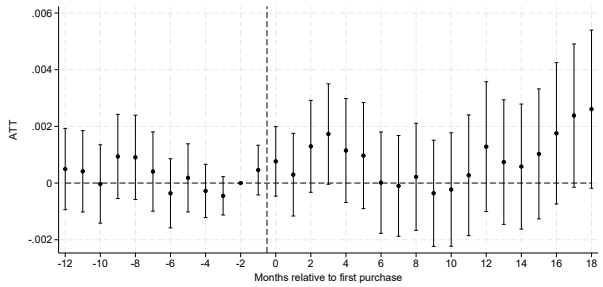
(a) Any unemployment benefit



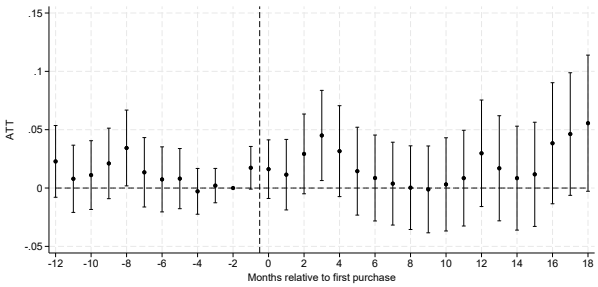
(b) Unemployment benefit days



(c) Any partial sickness allowance



(d) Partial sickness allowance days



Notes: Panels (a) and (b) report unemployment benefit receipt and compensated days, combining the basic and earnings-related schemes described in Section 2. Panels (c) and (d) report partial sickness allowance receipt and compensated days. Event month -2 is the omitted reference. Bars are 95 percent confidence intervals based on standard errors clustered at the individual level. Appendix Table A7 reports the underlying coefficients.

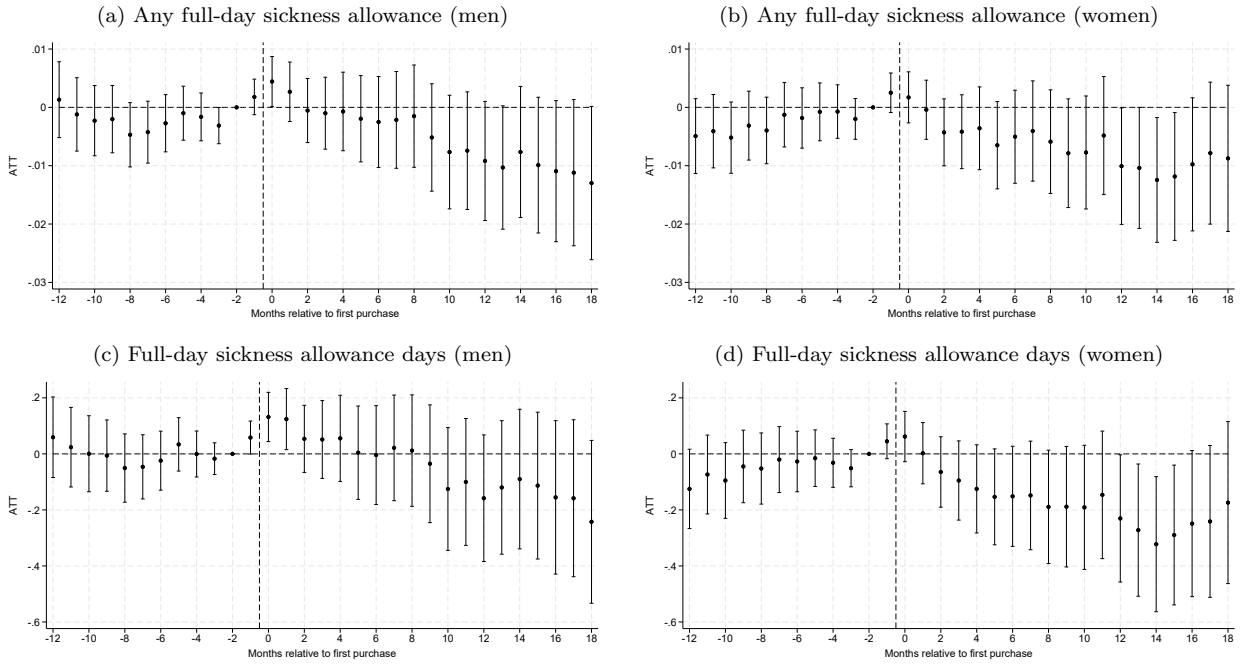
Table A3: Alternative benefit states after starting semaglutide

	Any unemployment benefit (1)	Unemployment benefit days (2)	Any partial sickness allowance (3)	Partial sickness allowance days (4)
ATT, months 0–18	0.000	-0.040	0.001	0.020
	(0.003)	(0.070)	(0.001)	(0.015)
ATT, months 0–5	0.000	0.000	0.001	0.025*
	(0.002)	(0.046)	(0.001)	(0.015)
ATT, months 6–11	0.000	-0.018	0.000	0.004
	(0.004)	(0.077)	(0.001)	(0.017)
ATT, months 12–18	-0.001	-0.093	0.001	0.030
	(0.005)	(0.108)	(0.001)	(0.021)
Effect/mean, months 0–18 (%)	-0.2	-1.3	20.5	30.8
Pre-treatment mean	0.154	3.101	0.003	0.065
Joint pretrend test, p-value	0.026	0.026	0.131	0.127
Person-month observations	664,704	664,704	664,704	664,704
Individuals	13,848	13,848	13,848	13,848

Notes: Columns (1) and (2) report unemployment benefits, combining the basic and earnings-related schemes described in Section 2. Columns (3) and (4) report partial sickness allowance. Event month -2 is the omitted reference. The pretrend row reports the joint Wald-test p-value for event months -12 through -3. Standard errors clustered at the individual level are in parentheses. Significance levels: $^{*}p < 0.10$, $^{**}p < 0.05$, and $^{***}p < 0.01$.

E Heterogeneity by Sex

Figure A4: Semaglutide and work capacity by sex



Notes: Event-study estimates for full-day sickness allowance around the first semaglutide purchase, estimated separately for men (left column) and women (right column). All panels use the Callaway–Sant'Anna estimator with patients who start later serving as not-yet-treated controls over event months −12 through 18. Event month −2 is the omitted reference. Bars are 95 percent confidence intervals based on standard errors clustered at the individual level.

Table A4: Semaglutide estimates by sex

Panel A. Work Capacity Outcomes				
	Men	Women	Men − women	Equality test p-value
Any full-day sickness allowance				
ATT, months 0–18	-0.005	-0.007*	0.001	0.776
	(0.004)	(0.004)	(0.005)	
ATT, months 0–5	0.000	-0.003	0.003	0.369
	(0.003)	(0.003)	(0.004)	
ATT, months 6–11	-0.004	-0.006	0.001	0.802
	(0.004)	(0.004)	(0.006)	
ATT, months 12–18	-0.010*	-0.010**	0.000	0.984
	(0.006)	(0.005)	(0.008)	
Full-day sickness allowance days				
ATT, months 0–18	-0.045	-0.167**	0.122	0.299
	(0.084)	(0.082)	(0.118)	
ATT, months 0–5	0.070	-0.062	0.132	0.114
	(0.059)	(0.060)	(0.084)	
ATT, months 6–11	-0.039	-0.169*	0.130	0.341
	(0.097)	(0.097)	(0.137)	
ATT, months 12–18	-0.148	-0.254**	0.106	0.538
	(0.124)	(0.119)	(0.172)	

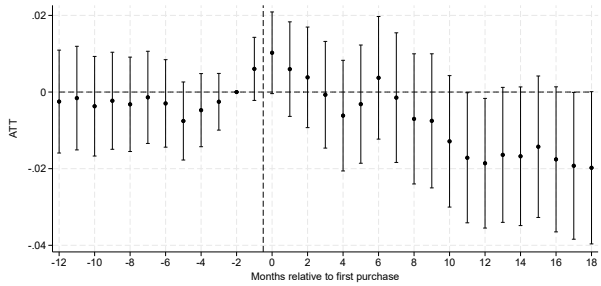
Panel B. Laboratory markers					
	HbA1c	Blood glucose	ALT	Triglycerides	LDL
	(1)	(2)	(3)	(4)	(5)
Men	-12.790***	-2.092***	-8.952***	-0.543***	-0.165***
	(0.678)	(0.223)	(2.058)	(0.157)	(0.048)
Women	-11.542***	-1.816***	-11.154***	-0.583***	-0.109**
	(0.558)	(0.156)	(1.980)	(0.186)	(0.049)
Men − women	-1.248	-0.277	2.202	0.040	-0.056
	(0.878)	(0.272)	(2.856)	(0.244)	(0.068)
Equality test, p-value	0.155	0.308	0.441	0.870	0.412

Notes: Panel A reports average treatment effects over the indicated event-month intervals. Panel B reports average treatment effects over event months 0–18. Estimates use the Callaway–Sant'Anna estimator separately by sex, with patients who start later serving as not-yet-treated controls. Event month −2 is the omitted reference. Standard errors clustered at the individual level are in parentheses. Equality tests use the difference between estimates from the nonoverlapping male and female samples. Significance levels: $^{*}p < 0.10$, $^{**}p < 0.05$, and $^{***}p < 0.01$.

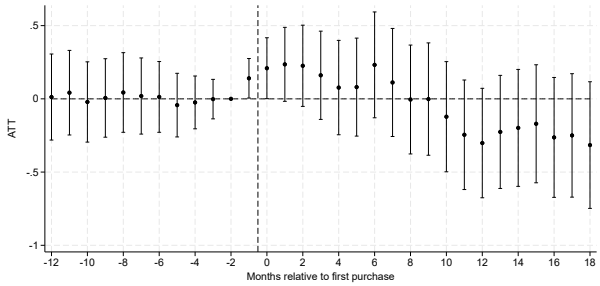
F Another GLP-1 Therapy: Liraglutide

Figure A5: Liraglutide and work capacity

(a) Any full-day sickness allowance



(b) Full-day sickness allowance days



Notes: Event-study estimates around the first liraglutide purchase. Panels (a) and (b) report the probability of receiving full-day sickness allowance and compensated full-day sickness allowance days. Both panels use the Callaway–Sant'Anna estimator with patients who start liraglutide later serving as not-yet-treated controls. Event month -2 is the omitted reference. Bars are 95 percent confidence intervals based on standard errors clustered at the individual level.

Table A5: Effects of starting liraglutide on work capacity

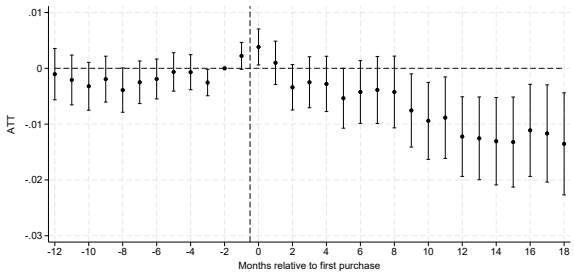
	Any full-day sickness allowance (1)	Full-day sickness allowance days (2)
ATT, months 0–18	-0.008	-0.040
	(0.007)	(0.147)
ATT, months 0–5	0.002	0.165
	(0.006)	(0.127)
ATT, months 6–11	-0.007	-0.005
	(0.008)	(0.178)
ATT, months 12–18	-0.018**	-0.246
	(0.009)	(0.193)
Effect/mean, months 0–18 (%)	-18.5	-4.4
Pre-treatment mean	0.044	0.914
Joint pretrend test, p-value	0.906	0.986
Person-month observations	96,768	96,768
Individuals	2,016	2,016

Notes: Event month -2 is the omitted reference. The pretrend row reports the joint Wald-test p-value for event months -12 through -3. Standard errors clustered at the individual level are in parentheses. Significance levels: $^{*}p < 0.10$, $^{**}p < 0.05$, and $^{***}p < 0.01$.

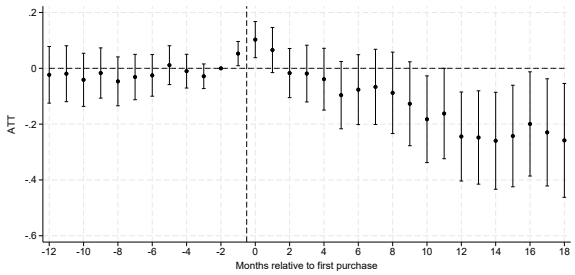
G Robustness

Figure A6: Sickness absence under alternative estimators and samples

Panel A. Covariate-adjusted Callaway–Sant'Anna

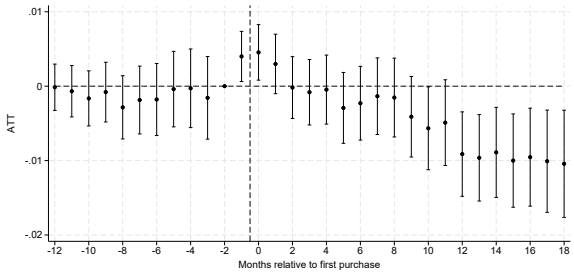


(a) Any full-day sickness allowance

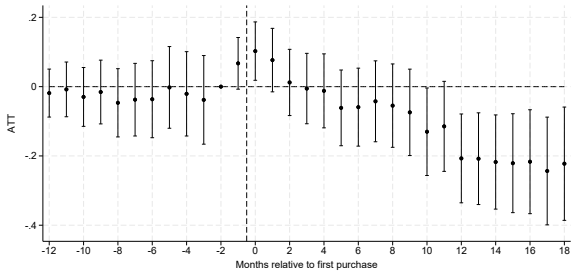


(b) Full-day sickness allowance days

Panel B. Imputation estimator

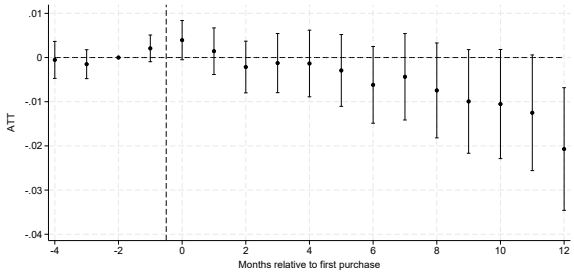


(c) Any full-day sickness allowance

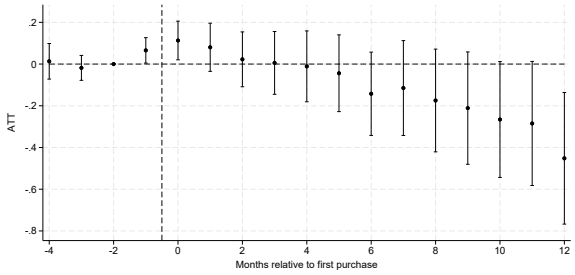


(d) Full-day sickness allowance days

Panel C. Balanced panel



(e) Any full-day sickness allowance



(f) Full-day sickness allowance days

Notes: Each row plots event-study estimates around the first semaglutide purchase. The left column reports the probability of receiving full-day sickness allowance and the right column reports compensated full-day sickness allowance days. Panel A adjusts for predetermined sex, birth cohort, and region; panel B uses the imputation estimator of Borusyak et al. (2024); and panel C uses the group-time estimator on a panel balanced over event months -4 through 12. Event month -2 is the omitted reference. Bars are 95 percent confidence intervals based on standard errors clustered at the individual level. Table A6 reports the corresponding aggregates.

Table A6: Robustness of the sickness-absence estimates

	Adjusted (1)	BJS (2)	Balanced (3)
Panel A. Any full-day sickness allowance			
ATT, months 0–18	-0.007**	-0.004**	–
	(0.003)	(0.002)	
ATT, months 0–5	-0.002	0.001	0.000
	(0.002)	(0.002)	(0.003)
ATT, months 6–11	-0.006**	-0.003	-0.008
	(0.003)	(0.002)	(0.005)
ATT, months 12–18	-0.012***	-0.010***	–
	(0.004)	(0.003)	
Pre-treatment mean	0.034	0.034	0.037
Joint pretrend test, p-value	0.155	0.506	0.615
Panel B. Full-day sickness allowance days			
ATT, months 0–18	-0.126**	-0.100**	–
	(0.058)	(0.051)	
ATT, months 0–5	0.000	0.019	0.028
	(0.042)	(0.045)	(0.064)
ATT, months 6–11	-0.117*	-0.079	-0.199*
	(0.068)	(0.057)	(0.119)
ATT, months 12–18	-0.240***	-0.219***	–
	(0.085)	(0.068)	
Pre-treatment mean	0.706	0.706	0.770
Joint pretrend test, p-value	0.424	0.512	0.525

Notes: Columns report the covariate-adjusted group-time estimator, the Borusyak–Jaravel–Spiess imputation estimator, and the group-time estimator on a balanced panel. The adjusted specification conditions on predetermined sex, birth cohort, and region. Event month -2 is the omitted reference. The balanced specification does not support the 0–18 or 12–18 aggregates. The pretrend tests use the available event months before the pre-purchase window. Standard errors clustered at the individual level are in parentheses. Significance levels: $^{*}p < 0.10$, $^{**}p < 0.05$, and $^{***}p < 0.01$.

H Event-Study Coefficients

Table A7: Semaglutide event-study coefficients: work capacity

Event month	Any full-day sickness allowance (1)	Full-day sickness allowance days (2)	Any partial sickness allowance (3)	Partial sickness allowance days (4)	Any unemployment benefit (5)	Unemployment benefit days (6)
−12	-0.002	-0.026	0.000	0.023	0.002	0.042
	(0.002)	(0.052)	(0.001)	(0.016)	(0.003)	(0.057)
−11	-0.002	-0.020	0.000	0.008	0.000	0.002
	(0.002)	(0.051)	(0.001)	(0.015)	(0.003)	(0.056)
−10	-0.004**	-0.043	0.000	0.011	0.000	0.002
	(0.002)	(0.049)	(0.001)	(0.015)	(0.003)	(0.054)
−9	-0.003	-0.024	0.001	0.021	-0.002	-0.055
	(0.002)	(0.046)	(0.001)	(0.015)	(0.003)	(0.052)
−8	-0.004**	-0.051	0.001	0.034	-0.004	-0.085
	(0.002)	(0.045)	(0.001)	(0.017)	(0.003)	(0.055)
−7	-0.003	-0.033	0.000	0.013	-0.003	-0.090*
	(0.002)	(0.042)	(0.001)	(0.015)	(0.002)	(0.048)
−6	-0.002	-0.025	0.000	0.007	-0.006***	-0.114**
	(0.002)	(0.038)	(0.001)	(0.014)	(0.002)	(0.045)
−5	-0.001	0.010	0.000	0.008	-0.005**	-0.091**
	(0.002)	(0.035)	(0.001)	(0.013)	(0.002)	(0.040)
−4	-0.001	-0.016	0.000	-0.003	-0.005**	-0.104***
	(0.002)	(0.031)	(0.000)	(0.010)	(0.002)	(0.035)
−3	-0.003***	-0.033	0.000	0.002	-0.003***	-0.041
	(0.001)	(0.022)	(0.000)	(0.008)	(0.001)	(0.025)
−2	–	–	–	–	–	–
−1	0.002**	0.052**	0.000	0.017	-0.001	-0.021
	(0.001)	(0.022)	(0.000)	(0.009)	(0.001)	(0.025)
0	0.003	0.100***	0.001	0.016	0.000	0.000
	(0.002)	(0.032)	(0.001)	(0.013)	(0.002)	(0.035)
1	0.001	0.067*	0.000	0.011	-0.001	-0.010
	(0.002)	(0.039)	(0.001)	(0.015)	(0.002)	(0.044)
2	-0.002	-0.002	0.001	0.029	0.001	0.022
	(0.002)	(0.044)	(0.001)	(0.017)	(0.003)	(0.051)
3	-0.003	-0.019	0.002	0.045	0.002	0.023
	(0.002)	(0.051)	(0.001)	(0.020)	(0.003)	(0.057)
4	-0.002	-0.029	0.001	0.032	-0.001	-0.034
	(0.002)	(0.050)	(0.001)	(0.020)	(0.003)	(0.063)
5	-0.004	-0.068	0.001	0.014	0.000	-0.001
	(0.003)	(0.061)	(0.001)	(0.019)	(0.003)	(0.067)
6	-0.004	-0.070	0.000	0.009	-0.001	-0.026
	(0.003)	(0.064)	(0.001)	(0.019)	(0.004)	(0.072)
7	0.000	-0.055	0.000	0.004	0.000	0.012
	(0.003)	(0.069)	(0.001)	(0.018)	(0.004)	(0.076)
8	-0.004	-0.083	0.000	0.000	0.001	0.005
	(0.003)	(0.072)	(0.001)	(0.018)	(0.004)	(0.080)
9	-0.006**	-0.106	0.000	-0.001	0.000	-0.026
	(0.003)	(0.077)	(0.001)	(0.019)	(0.004)	(0.085)
10	-0.008**	-0.153*	0.000	0.003	0.002	-0.015
	(0.004)	(0.079)	(0.001)	(0.020)	(0.004)	(0.090)
11	-0.006	-0.120	0.000	0.008	0.000	-0.057
	(0.004)	(0.082)	(0.001)	(0.021)	(0.005)	(0.097)
12	-0.010**	-0.194**	0.001	0.030	-0.001	-0.111
	(0.004)	(0.082)	(0.001)	(0.023)	(0.005)	(0.101)
13	-0.010**	-0.192**	0.001	0.017	-0.003	-0.107
	(0.004)	(0.088)	(0.001)	(0.023)	(0.005)	(0.106)
14	-0.010**	-0.200**	0.001	0.008	-0.002	-0.094
	(0.004)	(0.089)	(0.001)	(0.023)	(0.005)	(0.111)
15	-0.011***	-0.196**	0.001	0.012	-0.003	-0.135
	(0.004)	(0.093)	(0.001)	(0.023)	(0.005)	(0.116)
16	-0.011***	-0.207**	0.002	0.038	0.002	-0.083
	(0.004)	(0.097)	(0.001)	(0.026)	(0.006)	(0.121)
17	-0.010**	-0.199**	0.002	0.046	-0.002	-0.076
	(0.004)	(0.100)	(0.001)	(0.027)	(0.006)	(0.128)
18	-0.011**	-0.204*	0.003	0.056	0.001	-0.043
	(0.005)	(0.105)	(0.001)	(0.030)	(0.007)	(0.135)

Notes: The table reports the coefficients plotted in the corresponding event-study figures. Event month −2 is the omitted reference. Standard errors clustered at the individual level are in parentheses. Significance levels: $^{*}p < 0.10$, $^{**}p < 0.05$, and $^{***}p < 0.01$.

Table A8: Semaglutide event-study coefficients: clinical outcomes

Event month	HbA1c	Blood glucose	ALT	Triglycerides	LDL
	(1)	(2)	(3)	(4)	(5)
−12	-2.942***	0.049	0.010	-0.094	-0.038
	(0.879)	(0.286)	(2.038)	(0.492)	(0.095)
−11	-1.229	0.107	-0.061	0.344	0.152*
	(0.983)	(0.303)	(2.143)	(0.421)	(0.084)
−10	-2.434***	-0.101	2.815	0.099	-0.070
	(0.818)	(0.317)	(2.269)	(0.300)	(0.118)
−9	-2.489***	-0.400	-0.834	0.329	-0.061
	(0.800)	(0.273)	(1.833)	(0.261)	(0.076)
−8	-2.013***	-0.390	3.955*	-0.103	-0.014
	(0.775)	(0.291)	(2.277)	(0.243)	(0.094)
−7	-0.792	-0.289	0.256	0.070	0.177**
	(0.776)	(0.253)	(2.047)	(0.200)	(0.085)
−6	-0.613	0.206	0.212	0.451*	-0.022
	(0.753)	(0.342)	(1.885)	(0.253)	(0.095)
−5	-1.254	0.173	3.464	0.150	0.213**
	(0.774)	(0.254)	(2.122)	(0.575)	(0.100)
−4	-0.344	0.165	-0.862	0.383	-0.015
	(0.740)	(0.325)	(2.209)	(0.494)	(0.114)
−3	0.171	-0.015	3.529	-0.226	-0.052
	(0.700)	(0.354)	(2.506)	(0.422)	(0.143)
−2	–	–	–	–	–
−1	-1.092	0.127	-1.135	0.260	0.086
	(0.681)	(0.336)	(1.860)	(0.443)	(0.139)
0	1.751**	0.657*	1.262	0.439*	0.038
	(0.830)	(0.376)	(1.829)	(0.263)	(0.113)
1	-3.766***	-0.828***	-2.504	-0.978**	-0.287***
	(0.719)	(0.252)	(2.582)	(0.428)	(0.100)
2	-7.335***	-1.475***	-3.186*	-0.630*	-0.045
	(0.795)	(0.265)	(1.932)	(0.341)	(0.093)
3	-8.822***	-1.799***	-9.565***	-0.502**	-0.213**
	(0.664)	(0.202)	(2.052)	(0.227)	(0.084)
4	-12.463***	-1.876***	-6.217***	-0.753**	-0.137
	(0.726)	(0.235)	(2.253)	(0.322)	(0.086)
5	-12.533***	-1.430***	-7.006***	-0.384*	-0.363***
	(0.936)	(0.294)	(1.909)	(0.226)	(0.101)
6	-13.428***	-2.654***	-5.579**	-0.987***	-0.223**
	(0.937)	(0.375)	(2.609)	(0.374)	(0.101)
7	-13.763***	-2.081***	-14.117***	-0.537**	-0.109
	(1.029)	(0.351)	(2.876)	(0.212)	(0.111)
8	-13.247***	-2.057***	-10.659***	-0.558**	-0.290***
	(1.082)	(0.388)	(2.670)	(0.221)	(0.110)
9	-12.927***	-2.050***	-13.206***	-1.031*	-0.242**
	(1.095)	(0.340)	(2.537)	(0.538)	(0.105)
10	-11.978***	-1.986***	-12.114***	-0.274	-0.179**
	(0.917)	(0.272)	(2.407)	(0.209)	(0.086)
11	-15.294***	-2.505***	-14.225***	-1.325***	-0.173*
	(1.246)	(0.440)	(2.688)	(0.389)	(0.099)
12	-13.754***	-2.788***	-14.851***	-0.552	-0.111
	(1.379)	(0.484)	(3.892)	(0.370)	(0.126)
13	-13.953***	-1.791***	-14.294***	-0.963***	-0.012
	(1.240)	(0.365)	(3.900)	(0.368)	(0.136)
14	-15.224***	-2.461***	-11.655***	-0.132	-0.138
	(1.436)	(0.414)	(2.687)	(0.273)	(0.130)
15	-14.581***	-2.122***	-13.120***	-0.510	-0.120
	(1.431)	(0.460)	(4.399)	(0.344)	(0.123)
16	-15.721***	-2.486***	-13.647***	0.023	-0.121
	(1.788)	(0.376)	(4.408)	(0.776)	(0.142)
17	-12.059***	-2.241***	-14.317***	-0.187	0.014
	(1.201)	(0.439)	(3.847)	(0.752)	(0.114)
18	-18.513***	-1.793***	-13.390***	-0.624***	-0.141
	(2.000)	(0.686)	(4.468)	(0.240)	(0.167)

Notes: The table reports the coefficients plotted in the corresponding event-study figures. Laboratory outcomes are conditional on a usable measurement in the month. Event month −2 is the omitted reference. Standard errors clustered at the individual level are in parentheses. Significance levels: $^{*}p < 0.10$, $^{**}p < 0.05$, and $^{***}p < 0.01$.