Gender disparities in heart disease: individual and provider behavior

Please click here for the most recent version

Franziska Valder*

November 22, 2024

Abstract

Ischaemic heart disease is the leading cause of death worldwide. Despite a lower (diagnosed) lifetime prevalence of heart disease, women are significantly more likely to die if hospitalized with Acute Myocardial Infarction (AMI). This is puzzling as one would expect those who are sicker and thus more likely to die from AMI to have a higher likelihood of being diagnosed in the first place. I show that healthcare providers are important drivers of this puzzle: While the mortality gap is substantially smaller for users of lipid-lowering drugs, women are typically less likely to use these drugs. Providers have substantial power over their patients' medication usage: Switching to a provider with a higher female usage rate of lipid-lowering drugs due to a clinic closure increases the uptake of these drugs and reduces mortality. Difficulties in interpreting female symptoms may drive the lower usage rate of women since providing cholesterol measurements outside the healthcare sector can reduce gender inequalities in the usage of lipid-lowering drugs - but the success of this depends on how information is transmitted. Gender differences only disappear when providers are directly informed about the results, and when it is not the patient's responsibility to report them. Lastly, I demonstrate that these gender differences also spill over to the labor market: women reduce their labor supply and increase disability benefit receipt much more than men - after AMI but also in anticipation.

*Center for Economic Behavior and Inequality, University of Copenhagen. I gratefully acknowledge financial support from the Danish National Research Foundation (grant no. DNRF134) and from the Novo Nordisk Foundation (grant no. NNF17OC0026542). I thank Marianne Benn, and Mette Gørtz for extensive support. I furthermore thank Riccardo Ciacci, Torben Heien Nielsen, Anders Munk-Nielsen, and Ida Lykke Kristiansen for helpful comments, as well as seminar participants at the Hamburg Center for Health Economics, at the UAM-UJI Workshop on the Economics of Health and Human Capital in Madrid and the Workshop in Causal Health Economics organized by Uppsala University.

1 Introduction

Gender differences in health are a widely recognized phenomenon (World Economic Forum, 2024; Cabral and Dillender, 2024), yet our understanding of the underlying causes is limited. This paper focuses on ischaemic heart disease which is not only the leading cause of death worldwide (WHO, 2020) but also a condition characterized by significant gender inequality. I show that the behavior of both, the healthcare provider and the individual contributes to the undertreatment and underdiagnosis of heart disease in women. These inequalities not only have large implications for health outcomes but are also associated with gender inequalities in the labor market.

While men are overall more likely to be diagnosed with ischaemic heart disease (7%) compared to women (5%) (Mozaffarian et al., 2015), women experience significantly higher mortality rates after acute events such as Acute Myocardial Infarction (AMI).[1] This is puzzling: those who are sicker (and thus more likely to die from AMI) should be more likely to be diagnosed in the first place. I refer to this as the *prevalence-mortality puzzle*.

There are many potential explanations for this puzzle. The medical literature has suggested differences in the disease process across gender (pathophysiology) and gender differences in symptoms, care-seeking, and care provision (Aggarwal et al., 2018; Mehta et al., 2016; Greenwood et al., 2018).[2] There is, however, no consensus on whether pathophysiological differences are indeed responsible for the higher female mortality after AMI (Aggarwal et al., 2018). Studies investigating the role of care-seeking and care provision (Bairey Merz, 2014; Hanratty et al., 2000; Karcioğlu et al., 2002) point to the importance of medical treatment choices as well as patient characteristics such as health behaviors or demographics. The most debated factor is provider behavior (Wenger, 2012), with the argument that women are often underdiagnosed and thus do not receive the necessary preventive care, e.g. in the form of medication. This claim, however, lacks causal evidence. It is also not well understood why women would be underdiagnosed. Potential reasons are difficulties in diagnosis due to women presenting with different symptoms than men, physicians' potential lack of attention, empathy, and communication skills towards women, or beliefs about the efficacy of medication for women. Another potential explanation is individual behavior, such as gender differences in care-seeking, and other health behaviors, such as preventive care and communication. Crucially, provider and individual behavior are often analyzed in isolation despite the reality of important interactions between the two. The likely reason is that it is often challenging to disentangle both.

This paper investigates gender differences in health using the case of heart disease. It

[1] This holds even when accounting for the fact that AMIs occur on average later for women than for men (Emanuel et al., 2021; Greenwood et al., 2018; Mehta et al., 2016) and despite general advances in survival following cardiovascular disease events.

[2] Pathophysiology studies "biological processes that are directly related to disease processes of physical, mental, or psychophysiological conditions and disorders" (Witthöft, 2013). This could for example mean that women experience stronger AMIs than men.

investigates the joint role of individual and provider behavior for the *prevalence-mortality puzzle* using a unique combination of Danish register data with medical examination data. I provide evidence for the important role of the general practitioner's (GP) prescription of lipid-lowering drugs - which are a cheap and easy way to reduce the gender gap in post-AMI mortality but much less used by women. Lower usage seems to a large extent affected by the GPs: switching to a GP with a higher share of female patients using lipid-lowering drugs increases uptake of lipid-lowering drugs and reduces mortality in the long run. I then show that underdiagnosis is a potential driver of low female usage of these drugs using information shocks about cholesterol. After receiving a high cholesterol measurement in a medical examination outside the healthcare sector, women are in general still less likely to use lipid-lowering drugs but the gender differences disappear if the results are directly sent to the GP and do not need to be reported by the patients. Lastly, I demonstrate that AMI has substantial negative effects on labor supply and disability benefit receipt which are significantly larger for women and emerge years prior to AMI.

I use administrative records on hospitalizations, deaths, prescription drug pick-ups, doctor visits, and labor market status. I leverage these records with survey and medical examination data from the Copenhagen City Heart Survey (CCHS).[3] The CCHS is a cardio-vascular population study that provides data on heart markers through examinations outside of the standard healthcare system. While objective health measurements are rare in general they are also in this setting not actively pursued by the patient or the provider. I use information shocks created by receiving a high cholesterol measurement for the first time to investigate whether difficulties in recognizing women's symptoms contribute to the *prevalence-mortality puzzle*.

First, I document the existence of gender disparities in heart disease for the entire Danish population. I show that while men have a higher likelihood of being diagnosed in the hospital with AMI (0.4% versus 0.2% between ages 30 and 90), women are up to 33% (3-5 p.p.) more likely to die within the first year after AMI. This gap persists also when controlling for the fact that women are typically older at first AMI. These first results confirm prior results across different populations and countries (Berger et al., 2009; Tan et al., 2016). Second, using information on individual use of prescription medicine, I show that the gender gap in mortality is much smaller for those who used lipid-lowering drugs prior to AMI. Over the life course, women however have 18% lower usage rates of lipid-lowering drugs than men. This suggests an important margin where higher use of lipid-lowering drugs for women would lower female post-AMI mortality substantially.

Therefore, I investigate why women do not use these potentially life-saving medications as much as men. Combining administrative data on healthcare use with objective health data from the CCHS, I show that women consistently seek more care, even conditional on objective health, health behaviors, and self-assessed health. Women not receiving medication because they do not seek care thus seems an unlikely explanation. I then examine whether

[3] For an overview article about the survey see Aguib and Al Suwaidi (2015).

providers drive the lower usage of these drugs. There is indeed substantial variation in female usage rates of lipid-lowering drugs across GPs. Importantly, there are more often GPs with low female user shares than with low male user shares. Exploiting clinic closures, I demonstrate large effects of providers on medication usage: changing to a GP with a higher female usage rate of lipid-lowering drugs increases the likelihood of using these drugs, and reduces mortality in the five years after the GP change.

I then examine underdiagnosis as a potential mechanism driving lower prescribing to women. Difficulties in communication or recognizing symptoms might defer GPs from administering relevant laboratory tests and examinations, thus leading women to be underdiagnosed and undertreated. I exploit the fact that such laboratory tests are performed as part of the CCHS. The test results were sent to the GP and the respondents (waves 1 and 2) or only to the respondents with the note to contact their GP. I examine whether the use of lipid-lowering drugs differs between men and women after they receive a high cholesterol measurement for the first time during the CCHS examination. The measures of high cholesterol are clear markers for heart disease and according to guidelines should be treated with lipid-lowering drugs. If lower prescribing to women is fully driven by difficulties in recognizing and interpreting female symptoms of heart disease I should not find gender differences once the cholesterol level is known. I show that even with a first high cholesterol measurement women are on average less likely to use lipid-lowering drugs than men with a first high cholesterol measurement. This difference however disappears for those waves where GPs were directly informed - suggesting an important role for information transmission, driven by either hesitancy of women to report their cholesterol results or information frictions at the GP.

Lastly, I investigate spillovers to the labor markets. If women are undertreated this will not only affect their health but could also imply important labor market limitations. I show that after AMI, both men and women are less likely to work and more likely to be on disability benefits. This labor market penalty of AMI is however much larger for women than for men. Using event studies, I show that women also reduce their labor supply and increase disability benefit receipt drastically in the years before AMI - much more than men. This is consistent with undetected health issues before AMI which negatively affect labor supply. The gender differences with respect to heart diseases thus could amplify already existing gender disparities in the labor market.

This paper adds to several strands in the literature. One strand discusses gender differences in mortality and its causes. Most of the studies however focus on the overall (increasing) female mortality advantage - i.e. the fact that women typically live longer than men. This advantage is often explained by gender-specific preferences and behavior (Schünemann et al., 2017), such as smoking (Preston and Wang, 2006) and reductions in maternal mortality (Jayachandran et al., 2010). It is however also recognized that much remains unexplained about the female mortality advantage (Goldin and Lleras-Muney, 2019). I focus on an area that is under-explored in this literature - given that the gap in mortality here favors men. Nevertheless, some of the mechanisms are also relevant in the discussion of the overall male

mortality disadvantage. Furthermore, I shift the focus to the joint behavior of providers and individuals as an explanation.

A second strand studies gender differences in healthcare use and health behavior. This literature has established that women seek more healthcare and have higher expenditures (Bertakis et al., 2000; Wardle et al., 2004; OECD, 2016). Many studies suggest that these gender differences are caused by/related to gender differences in personality (Cobb-Clark et al., 2014), preferences (Schünemann et al., 2023; Cohen and Einav, 2007), and health perceptions (Arni et al., 2021). I provide evidence that gender differences in healthcare-seeking and health behavior persist conditional on objectively measured health which emphasizes the above-mentioned role of preferences, personality, and health perceptions.

A third strand concerns provider behavior and in particular provider bias. Several papers have demonstrated the importance of providers (Ginja et al., 2022) in general but also particularly for heart disease (Currie et al., 2016; Koulayev et al., 2017; Simeonova et al., 2024). Provider beliefs often seem to outweigh patient demand, which is problematic as a considerable share of those beliefs is unsupported by clinical evidence (Cutler et al., 2019). The importance of provider behavior is accentuated by substantial heterogeneity along patient and provider characteristics. Several studies highlight the importance of general provider characteristics such as age, gender, race, experience, or practice style (Currie et al., 2016) while others emphasize concordance between patient and provider according to gender, race or SES (Greenwood et al., 2018; Cabral and Dillender, 2024; Alsan et al., 2019; Kristiansen and Sheng, 2022). These studies suggest that concordance in patient and provider characteristics might be crucial for communication. I study provider behavior jointly with patient behavior in a setting where objective health information is available but not mandated by the providers themselves. This makes it possible to dig deeper into potential mechanisms of provider and patient behavior. The external measurement of health distinguishes this paper from a closely related study by Simeonova (2013) which shows that patient responses to provider input matter more than provider behavior itself for the racial mortality gap but where patient severity is endogenously measured. Another closely related study is Kämpfen and Mosca (2024) who demonstrate stronger responses of males to blood pressure measurements than for females but cannot establish any mechanisms.

The findings of this paper contribute to a better understanding of the high female mortality after AMI. The study of AMI is particularly policy-relevant due to its high rank in the cause of death statistics but also due to the well-established treatment. High female mortality furthermore not only reduces women's lifespan but points toward poor health in the years before acute events - likely undetected. The costs for both society and the individual are also not limited to health but spill over to the labor market. A larger AMI penalty for females in the form of reductions in labor supply and increases in disability benefit receipt as well as larger anticipatory behavior show that women with heart disease struggle more to stay attached to the labor market than men with the same condition. Gender differences in heart disease have accordingly received a lot of attention, e.g. in a report by the World Economic Forum from 2024 that calls for closing the "Women's Health Gap" (World Economic Forum,

2024). In addition, if underdiagnosis is carried through until death, i.e. if many women with heart disease are never diagnosed, this will bias the underlying data used to develop widely-used risk assessment tools such as the SCORE2 (ESC Cardiovasc Risk Collaboration and SCORE2 Working Group, 2021). My findings suggest an important role of diagnostics which could be improved by policies targeting more testing or the training of physicians.

The remainder of this paper is organized as follows: Section 2 describes the institutional background and the data used. Section 3 documents the prevalence-mortality puzzle in the Danish population. Section 4 explores the drivers of the prevalence-mortality puzzle and Section 5 investigates spillovers to the labor market. Section 6 concludes.

2 Institutional Background and Data

2.1 Institutional Background

Denmark has a universal public health insurance system for all citizens funded by taxes.[4] While the national government organizes overall health policies, primary and secondary healthcare is organized by the five regions and the 98 municipalities (Christensen et al., 2016).

Primary healthcare is mainly organized by privately owned clinics. These self-employed GPs provide (subsidized) services to their patients free of charge.[5] The number of GPs was around 3,500 in 2010, organized in about 2,200 clinics, with each GP having an average of 1,600 patients affiliated with their clinic (Forde et al., 2017). Citizens are free to choose a specific GP, but the availability of GPs varies across the country. There is a (modest) fee associated with switching GP (≈US$30) unless the change is either related to the patient moving to a different municipality or a closure of the current GP's clinic. When a GP clinic closes, patients can choose among clinics that are open for intake of new patients in the area.[6] The information available to the patient when choosing a clinic is limited but includes the names, ages, and genders of GPs in the clinic.[7]

The GP acts as the primary link between their patient and the rest of the healthcare system, thus serving as a gatekeeper for specialists in hospitals and private clinics. The GP is often responsible for initial diagnostics, routine interventions, prescription of medicine, managing chronic conditions, providing preventive care, and referring patients to specialized treatment. GPs are e.g. responsible for around 85% of all outpatient prescriptions.

Patients in hospitals receive medicine free of charge while hospitalized. When discharged from the hospital, patients need to collect their medicine at a pharmacy. While there

[4] This means that access barriers are not among the mechanisms explored here. However, as the next section will show the *prevalence-mortality puzzle* exists despite free access to care.
[5] The reimbursement framework for the primary care clinics combines capitation and fee-for-service fees.
[6] Within a 15-kilometer radius of the patient's home; 5 kilometers in the Copenhagen area.
[7] Many clinics also provide information on their clinic on the clinic's website.

is an out-of-pocket cost associated with purchasing medicine, prescription drugs sold in pharmacies are highly subsidized.

2.2 Register Data

I obtain data on AMIs from the hospital register - containing all hospitalizations and outpatient hospital contacts in Denmark since 1977. I use data from 1995 onwards to align with the drug database (see below). I merge the hospital register with population registers to present evidence on full population (diagnosed) prevalence. This is combined with outpatient care data on GP and specialist visits, and with the prescription drug database from 1995 until 2019. This data provides the Anatomical Therapeutic Chemical Classification System grouping code (ATC-code) of medications picked up. In the main part of the paper, I will focus on lipid-lowering drugs (ATC-C10) and will show results for the other cardiovascular system drugs (ATC-category C) in the appendix.[8] I also construct female medication usage rates for each ATC-code on the clinic level. I supplement this data with information on labor market status.

2.3 Copenhagen City Heart Study (CCHS)

I complement the register data with objectively and independently measured health from the Copenhagen City Heart Study (*Østerbroundersøgelsen*). This enables investigating the role of diagnostics and gender differences in care-seeking conditional on objective health for the *prevalence-mortality puzzle*.

The Copenhagen City Heart Study is a cardio-vascular population study with examinations conducted in Copenhagen between 1976 and 2015, in five waves and with an ongoing follow-up. The study goal was to learn about the prevention of coronary heart disease and stroke. The study consisted of two parts, a survey about respondents' background information, health behavior, and health history, and a medical examination in which several key health parameters were measured. Most importantly, the CCHS measured respondents' cholesterol levels from a blood sample. The results of the measurements were given to the patient and in waves 1 and 2 also directly sent to the GP. From wave 3 onwards, only patients received the results with the advice to contact their GP. The values were written into a report card with clear markers for values outside the normal range (see Figure A5 in the appendix). Respondents were invited to the survey through a random draw from the Copenhagen population register, stratified by age if they lived within the ten city wards surrounding the hospital in which the survey was conducted. This means that the CCHS study population is regionally selected. The response rate ranged between 50 and 75% across waves (Schnorh, 2009). Table A2 in the appendix presents some general demographic descriptives of the invited and participating population. It shows that those who participate are overall advantaged from a health perspective: they are younger, more likely to be married, use more care, have fewer comorbidities, have higher labor income, and more

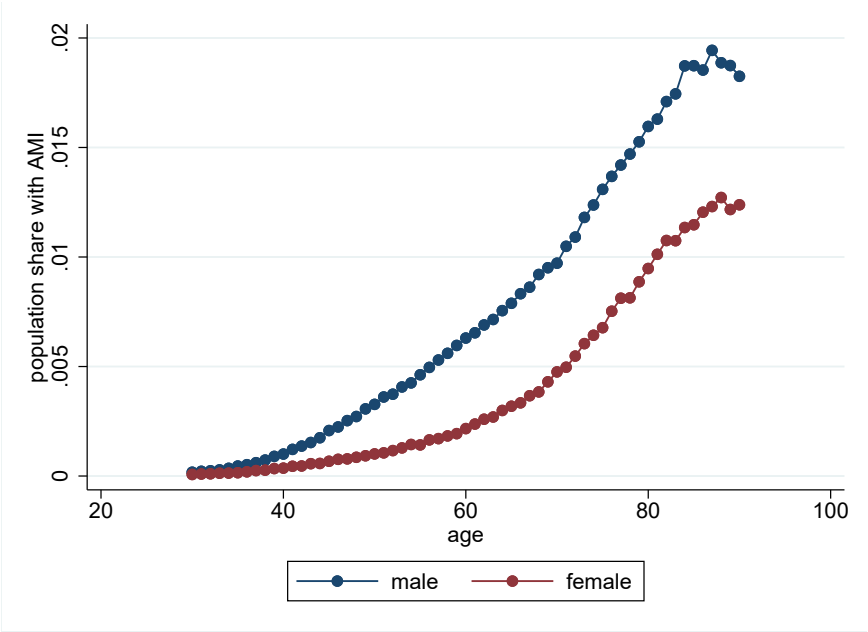
[8] See Table A1 for an explanation of the different types of drugs.

education. Issues such as undertreatment are likely less severe in such an advantaged population suggesting that the results of this paper likely present a lower bound of effects for the entire population.

3 The Prevalence-Mortality Puzzle

Figure 1 plots the prevalence of first hospitalizations with AMI in the Danish population for the ages 30 to 90. Over the entire age range, men are more likely to be diagnosed with AMI, with an average prevalence of 0.4% compared to 0.2% for women.[9] The gender gap is quite large, with the share of men with AMI being up to 0.63 percentage points higher than the share of women - equivalent to a 2.5-fold higher prevalence. This is in accordance with evidence from most other developed countries (Tan et al., 2016).

Figure 1: Prevalence of AMI - over age and gender

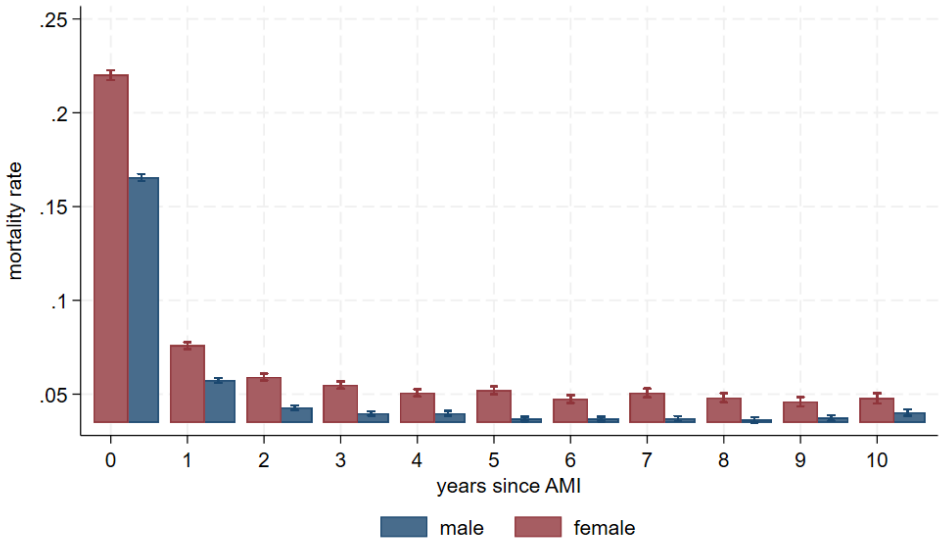


Note: Figure shows the prevalence of AMI over age, split by gender for the entire Danish population from 1995 to 2018. Restricted to ages 30-90.

When I turn to post-AMI mortality, I would expect that men are more likely to die since they are more often diagnosed and thus likely less healthy. Figure 2 however shows the opposite: in the first 10 years after AMI, women are more likely to die – between 0.7 and 5.5 percentage points. This is equivalent to 11-79% of the average mortality rate over this period (6.9%). The gap is largest in the year where AMI occurs, both in absolute and relative terms, with a 33% higher mortality rate for women. The mortality rate decreases substantially after year zero suggesting that either those who are relatively healthier survive or that those who survive can be treated effectively.

[9] While prevalence grows steadily over age there is a dip around age 90 where the curve flattens. This is because around age 90 many individuals do not make it to the hospital anymore when they have a first AMI but die right away at home/in a nursing home. Such deaths are not counted since individuals are never diagnosed in the hospital.

Figure 2: Gender gap in mortality after AMI



Notes: Figure shows the mortality rate after a first AMI for 10 years following AMI, split by gender. Restricted to ages 30-90.

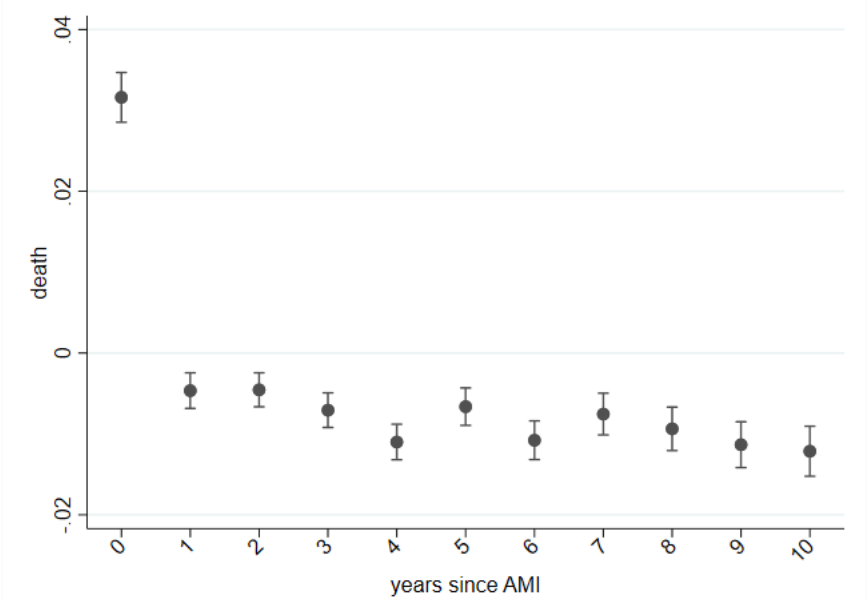
One potential explanation for the *prevalence-mortality puzzle* is that women are just older and/or sicker when they experience AMI due to their higher overall life expectancy. Figure 3 plots coefficients from a regression of death post-AMI on gender where age-fixed effects and comorbidities are taken into account. Comorbidities are measured using the well-established Charlson comorbidity index.[10] Figure 3 shows that the differences in the year of AMI shrink somewhat when controlling for age-fixed effects and comorbidities, suggesting a 3.1 percentage points higher mortality rate for women. After year zero the gap reverses, and women experience a lower mortality risk. This is on the one hand consistent with an interpretation of underdiagnosis of women and therapy effectiveness where those women who survive are finally adequately treated. It is on the other hand also consistent with more unhealthy men surviving due to earlier treatment compared to unhealthy women who die immediately.[11]

4 What drives the prevalence-mortality puzzle?

As mentioned above, underdiagnosis has been discussed as a potential driver for the *prevalence-mortality puzzle*. If women are underdiagnosed they cannot receive the necessary care

[10] The Charlson comorbidity index predicts 1-year mortality on the basis of pre-existing health conditions. I use data on hospitalizations for the year prior to AMI. I use the weights provided in Quan et al. (2011) and use the procedure for administrative data by Quan et al. (2005).
[11] Similar results are obtained when only controlling for age.

Figure 3: Gender gap in mortality after AMI - controlling for age and comorbidities



Notes: Figure shows coefficients from regressing an indicator for death on the interaction $yrs\ since\ AMI \times female$. Controlling for age-fixed effects ($\lambda_{i,t}$), year-fixed effects (μ_t), and comorbidities in the year before using the Charlson comorbidity index. $death_{i,t} = yrs\ since\ AMI \times female + yrs\ since\ AMI + charlson_{i,t-1} + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$.

in the form of check-ups or medication. Underdiagnosis seems to even occur in the acute case where women with AMI are more likely to be sent away from the hospital instead of being admitted (Pope et al., 2000) or incur much longer waiting times than men and receive less thorough examinations (Banco et al., 2022). Potential reasons for this and higher female mortality are differences in pre-existing diagnoses and treatment. In the Danish setting, I can test directly whether those women who have been treated with medication against heart disease face different mortality rates compared to women who have not been treated and compared to men. I focus on cholesterol-lowering medication. High cholesterol is next to high blood pressure the largest driver of heart disease. Compared to high blood pressure the relationship between cholesterol levels and mortality is unambiguous and there exists a clear medication treatment corridor in the form of lipid-lowering drugs.[12]

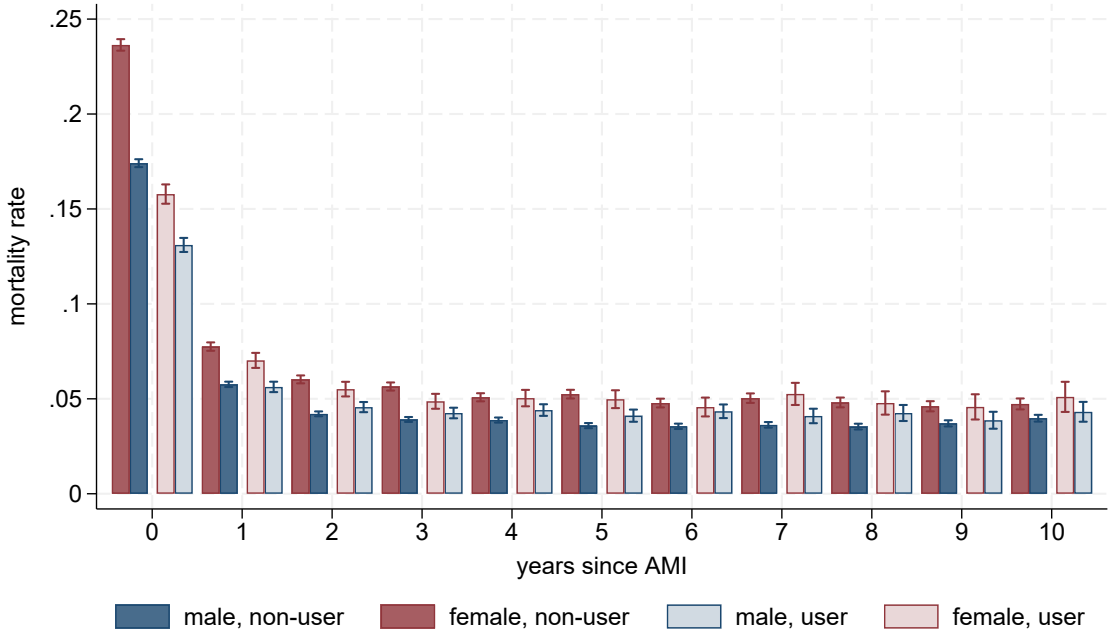
Figure 4 investigates the role of previous lipid-lowering medication descriptively. It shows the mortality rate after AMI split by gender and whether the individual has ever used lipid-

[12] Lipid-lowering drugs lower the level of total cholesterol and low-density lipoprotein (LDL) cholesterol in the blood. They are often referred to as "statins" since this is the most commonly used drug. Risk and treatment indications are less clear for high blood pressure, the second driver of heart disease. There exists a range of blood-pressure-lowering medications that are often used in combination, such that indications are not as clear as for high cholesterol.

lowering drugs before.[13] Mortality in the group treated with lipid-lowering drugs is overall smaller. However, also the gender gap in mortality is smaller in the population treated with lipid-lowering drugs (2.68 p.p., 20%) than in the untreated population (6.22 p.p., 36%) both in absolute and relative terms. This suggests that medication can reduce the gender gap in mortality by reducing female mortality to a level more similar to that of men.[14]

Figure 5 repeats this exercise controlling for age-fixed effects. Similar conclusions as from Figure 4 can be drawn here.[15]

Figure 4: Gender gap in mortality after AMI - previous use of lipid-lowering drugs



Notes: Figure shows the mortality rate after AMI for 10 years, split by gender and previous use of lipid-lowering drugs.

Now the question arises if women do not use these medications to the same extent as men. Figure 6 below suggests so. It plots the share of the population between ages 30 and 90 using lipid-lowering drugs. It shows that men have substantially larger usage rates of lipid-lowering drugs.[16] This is surprising as studies have shown that lipid-lowering drugs in the

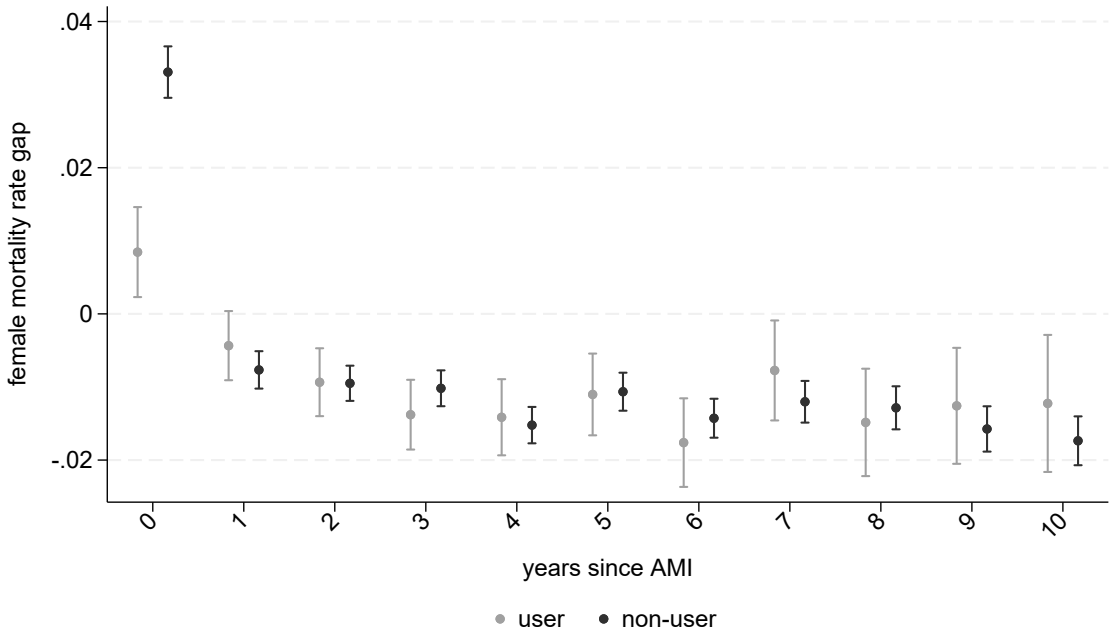
[13] I use the term "using" instead of "picking up" medication despite the data coming from prescription pick up. I elaborate on this assumption in Section 4.2.1. Figure A1 explores the role of the other category C medications. Mortality is for some ATC-codes higher for users, likely driven by higher general risk due to much poorer health when using that drug.

[14] The same holds for the other medications, see Figure A1. The exception is ATC-code C05. Here, the gender gap is bigger for treated versus untreated for ATC-code. This is likely related to the fact that C05 covers medication for treating hemorrhoids which often occur related to childbirth.

[15] This pattern also holds for other ATC-codes, most significantly for C01, C03, C07, C08, C09, and C10.

[16] Figure A3 shows that for five out of the seven remaining ATC-codes men have a higher usage share. The

Figure 5: Gender gap in mortality after AMI - previous use of lipid-lowering drugs - controlling for age



Notes: Figure shows coefficients from regressing an indicator for death on the $yrs\ since\ AMI \times female$, separately for users, and non-users of lipid-lowering drugs. Controlling for age-fixed effects ($\lambda_{i,t}$) and year-fixed effects (μ_t). Estimated separately for previous users and non-users of lipid-lowering drugs: $death_{i,t} = yrs\ since\ AMI \times female + yrs\ since\ AMI + + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$.

form of statins are equally effective in women as they are in men for secondary prevention - i.e. when heart disease has already manifested (Sever et al., 2003; Group et al., 2002; Collins, 2003). There is furthermore no evidence that statins are less safe in men than in women (Gulati and Merz, 2015). Guidelines also do not make different recommendations regarding critical cholesterol levels or treatment with statins for men versus women (Grundy et al., 2019). Once initiated, lipid-lowering drugs are typically taken for the rest of life (Wu et al., 2022) meaning that gender disparities in use are likely accentuated over the life cycle. Figure A4 in the appendix furthermore shows that the out-of-pocket price patients face is quite low, with around US$150 per year before the patent of the most popular statin (*Simavastin*) ran out in 2002, and around US$45 per year afterward.[17]

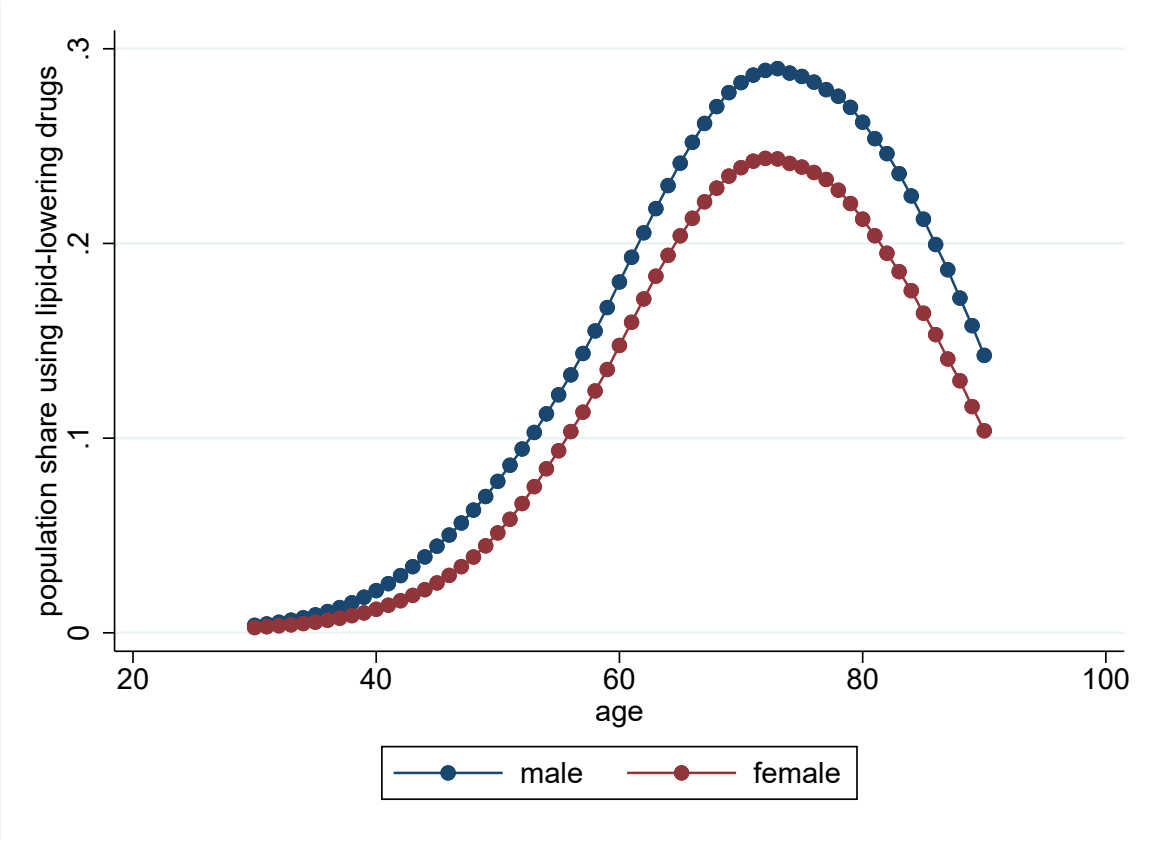
The overall lower usage of lipid-lowering drugs for women could stem from the patient or the provider side: Women could go to the doctor less, not pick up their prescription (so they

exceptions are ATC-code C03 and ATC-code C05.

[17] Usage of lipid-lowering drugs generally increases over age and often follows a hump shape at older ages, suggesting that those using lipid-lowering drugs potentially die earlier thus reducing the share of users in later age groups.

do not show up in the data), or providers could prescribe less to women.[18] The next sub-section explores reasons rooted in individual behavior and sub-section 4.2 explores provider behavior.

Figure 6: Gender differences in usage of lipid-lowering drugs



Notes: Figure shows usage rates for lipid-lowering drugs on the whole population over age, split by gender. Restricted to ages 30-90.

4.1 Gender gaps in mortality: individual behavior

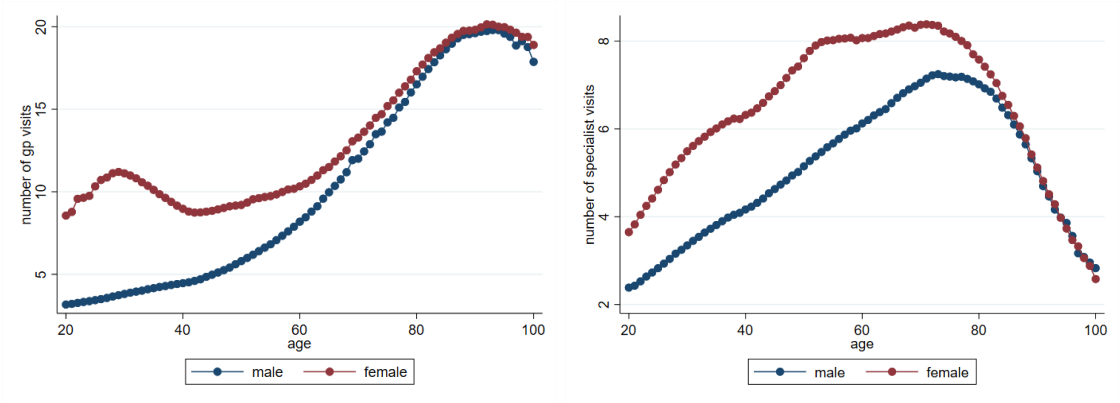
In this section, I explore the role of gender differences in healthcare-seeking behavior for the *prevalence-mortality puzzle*. I assess whether women are less likely to seek care and therefore less likely to obtain medication for heart disease. I start with some descriptive evidence on healthcare usage of the entire Danish population from the registers and then zoom in on more detailed data on health behavior and self-assessed health from the CCHS.

[18] I use the term provider when I refer to all possible physicians such as general practitioners, specialists, and doctors in the hospital. I use the term GP whenever I use explicit information about an individual's GP.

4.1.1 Descriptive evidence on gender differences on health behavior

Figure 7 shows average visits to GPs and specialists for men versus women. Overall, women visit GPs and specialists more often. Especially at younger ages, women visit GPs and specialists up to twice as much, the number of visits then converges at later ages.[19]

Figure 7: Gender differences in healthcare use



(a) GP visits

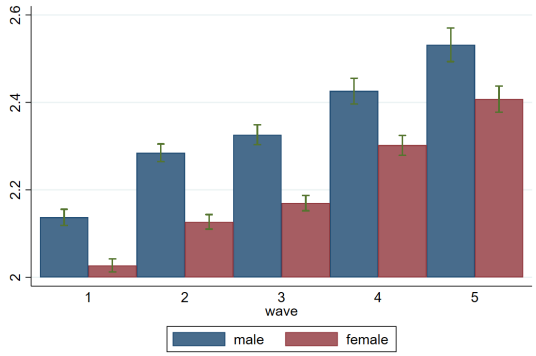
(b) Specialist visits

Notes: Figure shows averages over age. Excludes visits related to pregnancy and childbirth. For a definition of specialist visits see footnote 13.

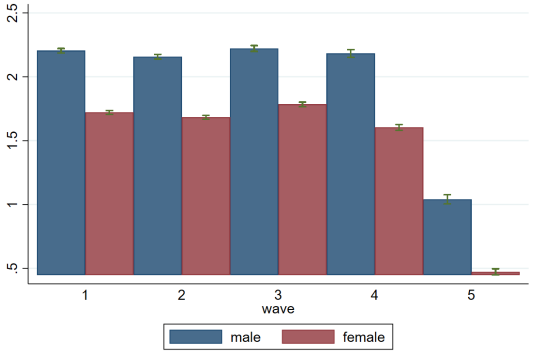
The fact that women visit GPs and specialists so much more often but still are less likely to use lipid-lowering drugs suggests that the content of those visits for men and women must be very different. One possible explanation is that providers may perceive men and women differently due to their health behaviors. Figure 8 uses data from the CCHS on self-reported health behaviors to shed some light on this. Figure 8 shows that women overall look very different from men: they smoke and drink substantially less. However, they also exercise slightly less and report worse health. These gender differences also hold conditional on objective health and age, see Table A3. This suggests that women might be perceived differently than men by their providers, due to differences in lifestyle. However, differences go in both directions: women might be perceived as healthier due to them reporting less smoking and drinking but also as less healthy due to worse self-assessed health and lower physical activity.

[19] Note that GP and specialist visits exclude visits related to pregnancy and childbirth and children's care. Specialist visits contain visits to different doctors, e.g. radiologists, internists, neurologists, psychiatrists, etc. Excludes physiotherapy, chiropractors, and gynecologists. Note that in Denmark, cardiologists do not operate in private practices but only in hospitals and will thus not show up in our measure of specialist visits.

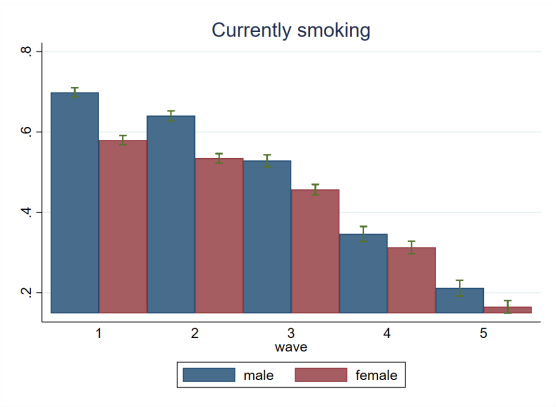
Figure 8: Gender differences in health behaviors



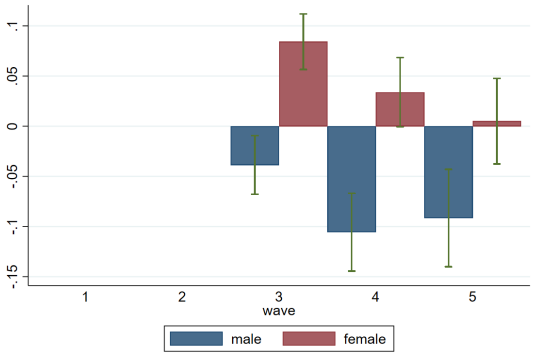
(a) Phys. activity 1-4 (passive-very active)



(b) Alcohol freq. 1-4 (never-daily)



(c) Share current smoker



(d) Self-assessed health (standardized)

4.1.2 The role of objective health for gender differences in healthcare-seeking

In this subsection, I investigate whether the higher care-seeking of women shown above is explained by poorer health or different health behaviors. Table 1 below estimates the role of gender for care-seeking in a simple OLS model with the number of GP and specialist visits as the dependent variables, using varying controls. Columns (1) and (5) show the raw gender gap in visits, accounting for year and age-fixed effects. Women have three more visits to the GP per year, and 2.4 more visits to a specialist than men. Relative to the means this suggests that women go to the GP 37% and the specialist 32% more. Columns (2) and (6) add controls for objective health, in the form of systolic and diastolic blood pressure, total cholesterol, and body mass index (BMI). The coefficients change very little and remain statistically significant. This means that also conditional on objective health, women go to the doctor more often. In the next step, instead of objective health, I include measures of self-reported health behavior (smoking, the frequency in which alcohol is consumed, the frequency of physical activity) and self-assessed health. Compared to the baseline without

controls, coefficients become smaller but remain statistically significant. In the last step, I control for both, measures of objective health and health behaviors (columns (4) and (8). Even then, the coefficients of the female dummy remain significant and still suggest 34% (of the mean) more GP visits and 30% (of the mean) more specialist visits for women compared to men. This suggests that women do consistently seek more care - regardless of their objective health and health behavior.

The evidence presented in this section suggests that a lack of women's healthcare-seeking is not the driving factor behind their lower usage of lipid-lowering drugs. I, however, find that women might be perceived very differently by the provider due to gender differences in reported health behaviors and self-assessed health, conditional on objective health. The next section therefore focuses on the role of the provider.

Table 1: Associations between gender and care-seeking, conditional on objective and subjective health

	Number of GP contacts				*Number of specialist contacts*			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mean:			*8.17*				*7.61*	
Female	3.033***	3.170***	2.644***	2.810***	2.419***	2.414***	2.316***	2.247***
	[0.124]	[0.127]	[0.129]	[0.132]	[0.145]	[0.150]	[0.155]	[0.161]
Objective health								
Systolic BP		-0.011***		-0.009**		-0.020***		-0.020***
		[0.004]		[0.004]		[0.005]		[0.005]
Diastolic BP		-0.008		-0.003		0.005		0.005
		[0.007]		[0.007]		[0.009]		[0.009]
Total cholesterol		-0.265***		-0.220***		-0.285***		-0.253***
		[0.056]		[0.055]		[0.066]		[0.067]
BMI		0.170***		0.112***		-0.029		-0.060***
		[0.016]		[0.016]		[0.018]		[0.019]
Health Behaviors								
Current smoker			-0.044	0.129			-0.833***	-0.907***
			[0.129]	[0.130]			[0.155]	[0.158]
Alcohol freq. (1-4)			-0.231***	-0.168*			0.019	0.001
			[0.086]	[0.087]			[0.104]	[0.106]
Phys. activity (1-4)			-0.224**	-0.142			0.363***	0.269**
			[0.088]	[0.088]			[0.106]	[0.108]
Self-assessed health			1.913***	1.839***			0.752***	0.793***
			[0.063]	[0.063]			[0.076]	[0.077]
Constant	4.063	3.227	6.156**	5.309*	-3.221	0.952	-3.030	2.056
	[3.213]	[3.226]	[3.104]	[3.130]	[3.747]	[3.816]	[3.741]	[3.816]
R-squared	0.071	0.078	0.129	0.130	0.075	0.079	0.082	0.087
Observations	15,726	15,516	15,330	15,128	15,726	15,516	15,330	15,128

Standard errors clustered on the individual level in brackets; *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects in all columns. Physical activity ranges from 1-4 (passive-very active), and alcohol frequency ranges from 1-4 (never-daily) as well. $\#contacts_{i,t} = female_i + \lambda_{i,t} + i.year + controls_{i,t} + \epsilon_{i,t}$

4.2 Provider Behavior

4.2.1 Variation in provider usage rates of lipid-lowering drugs

To study provider behavior, I focus on the medication usage of patients. The prescribing of medication is not only one of the key tasks of GPs but also an important dimension of the treatment of heart disease as suggested by Figures 4 and 5. There are two important assumptions related to the data. The prescription database only records medication picked up at the pharmacy and does not record prescriptions that were written but never picked up. First of all, I need to assume that those medications that are picked up are also used. Data from the CCHS reveals that around 91% of those who have picked up a prescription for lipid-lowering drugs the month before the examination, also indicate using these drugs in the survey. Only 1.6% say they do not use them, and 7.4% do not respond. Proxying medication usage with medication pick-up is a common assumption made in this setting. I will therefore use the term medication usage instead of prescription pick-up. Second, I need to assume that the pick-up of medication also reflects the prescription behavior of providers. A study from 2014 (Pottegård et al., 2014) suggests that about 9.3% of first prescriptions are not picked up with very little differences across genders. For statins, this number drops to 6.2%. However, in the most extreme scenario, there is selection of those patients who never pick up statins to specific GPs and I would largely overestimate the role of the GPs. I, therefore, exploit in the next subsection that individuals are exposed to new GPs when their clinic closes.

I first provide descriptive evidence on the variation of medication usage according to gender across GPs. I focus on lipid-lowering drugs - a drug that has a clear connection to mortality through cholesterol. I document the variation in the GP-specific rates of male and female usage of lipid-lowering drugs. The share of male patients using lipid-lowering drugs is on average higher with around 7.1% (std dev. 0.07) compared to women with around 5.6% (std dev. 0.06) of female patients. Panel (a) of Figure 9 below plots the distribution of the share of female and male patient user shares of lipid-lowering drugs.[20] The figure shows that there are more GPs with low female usage shares and high male user shares. Another way to depict gender differences in usage is to plot the gap, i.e. the difference between male and female usage share for each GP. Panel (b) of Figure 9 below shows that the distribution of this gap is clearly right-skewed reflecting more GPs with higher male than female usage shares.[21] The size of the gaps is also relatively large compared to the usage shares over age presented in Figure 6. The descriptives above suggest substantial variation in usage shares across GPs with large gaps between men and women.

[20] Figure A6 repeats this for the other ATC-codes. For ATC-codes C01, C08, and C09, I see that there are more GPs with low female usage shares and higher male usage shares. For the ATC-code C05, female shares are higher. For ATC-codes C02, shares seem similar for men and women and for ATC-codes C03 and C07, no clear pattern is visible.

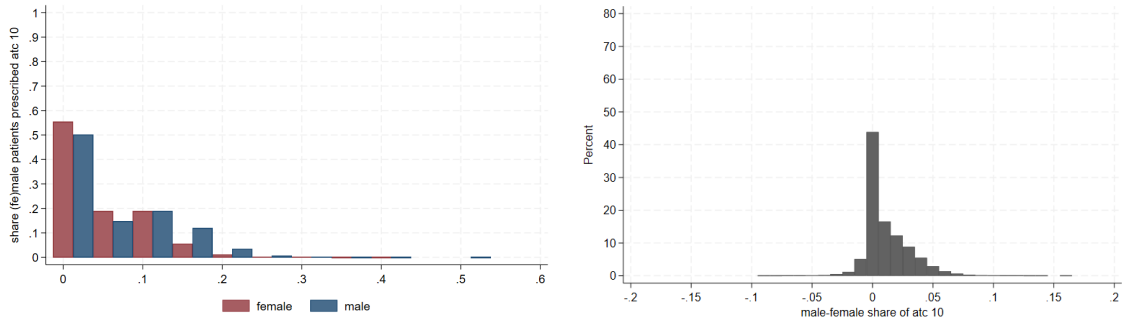
[21] Figure A7 shows the gaps for other ATC-codes.

4.2.2 The effect of provider-usage rates on uptake of lipid-lowering drugs and mortality

In order to show that medication usage is indeed a good proxy for provider prescription behavior I exploit that patients face new providers when their old clinic closes. If a provider's prescription behavior has nothing to do with individual medication use I should not find any effect of changing to a new provider on subsequent medication use. I exploit the difference in female usage rates of lipid-lowering drugs between the old (closing) and the new GP. I focus on female user rates as a proxy of a GP's prescription behavior towards females. I do however also consider outcomes of males to understand whether higher female usage rates reflect a general or a patient-gender-specific behavior.

Figure A8 in the appendix plots the distribution of the difference in female usage rates between the old and the new GP when a clinic closes. The figure shows that individuals experience different types of changes: they change to GPs with the same, lower, or higher female usage rates.[22]

Figure 9: Differences in male and female usage rates of lipid-lowering drugs



(a) Distribution of GP-level male and female usage rates of lipid-lowering drugs

(b) GP-level gap between male and female usage rates of lipid-lowering drugs

Notes: Panel (a) shows the distribution of GPs' female and male usage rates of lipid-lowering drugs. Panel (b) shows the distribution of the within-GP difference between female and male usage rates.

Next, I investigate the role of switching GPs by documenting how the difference in female usage rates of lipid-lowering drugs between the new and the old GP is related to the probability of picking up a prescription for lipid-lowering drugs relative to those individuals who switch to a GP with (roughly) the same female usage rate. Importantly, I control for the individual's 1-year lagged prescription usage. Figure 10 shows that if the difference is negative, i.e. the new provider has a lower usage rate, then the probability of picking up lipid-lowering drugs is reduced. Vice versa if the difference is positive, i.e. the new provider

[22] Note that for most ATC-codes there is a peak at 1, indicating there is a somewhat larger share of individuals who experience a doubling of the prescription rates. This might reflect time trends due to the increasing usage of medication over time. Such time trends will be accounted for in the estimations.

has a higher female usage rate then the probability of picking up a prescription is increased. It also seems that those effects are weakly linearly increasing in the size of the difference. The patterns are similar for men and women.

I then use an alternative specification where I compare the effect of closure-induced switching to a GP with a lower or higher female usage rate of lipid-lowering drugs on the change in the prescription pick-up instead of using the entire range of changes. Figure 11 shows that switching to a GP with a lower female usage share of lipid-lowering drugs reduces the probability of picking up a prescription. Switching to a higher female share GP increases the probability of picking up a prescription. Effects for men are again very similar. This points to the importance of general and not necessarily gender-specific prescription behavior.

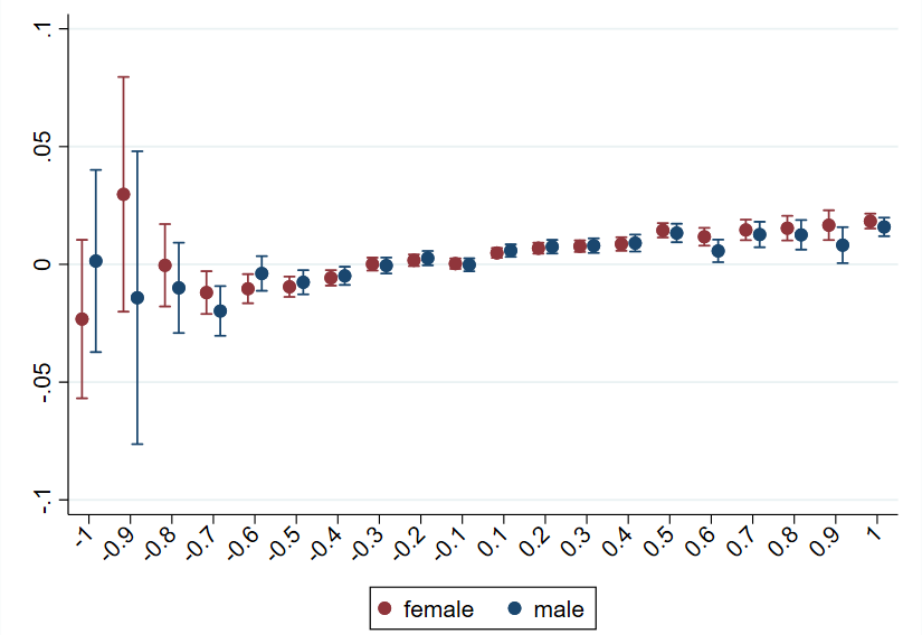
In these estimations, I control for the difference in GP practice age and gender composition as these characteristics are observed by potential patients and thus can be selected into. The effect sizes are large compared to average usage rates, ranging up to 30%. This suggests that GPs exert a substantial influence over the medication usage of their patients. One threat to this conclusion is selection. That means I need to show that after a closure women do not select to lower-female usage-rate GPs and therefore receive less medication. Figure 12 below shows that the opposite holds true. The figure depicts relative risk ratios from a multinomial logit model for the effect of the female indicator on the probability of choosing a lower or a higher female usage rate GP after a closure, relative to a similar female usage rate GP. Women have a lower relative risk ratio to choose a new provider with a lower female usage rate than men. The female indicator is also associated with a higher relative risk ratio to choose a GP that has a higher usage rate.[23]

Lastly, I investigate whether the switch to a higher or lower usage rate GP also affects mortality. Figure 13 provides suggestive evidence that the change to a lower/higher usage rate GP affects the mortality risk in the first five years after the GP change. Switching to a GP with a higher usage rate of lipid-lowering drugs is associated with a small reduction in the probability of dying in the first five years after the change. This suggests that the increase in lipid-lowering drugs induced by the doctor can be life-saving.[24]

[23] Figures A10 and A11 repeat those exercises for other ATC codes, revealing similar patterns.

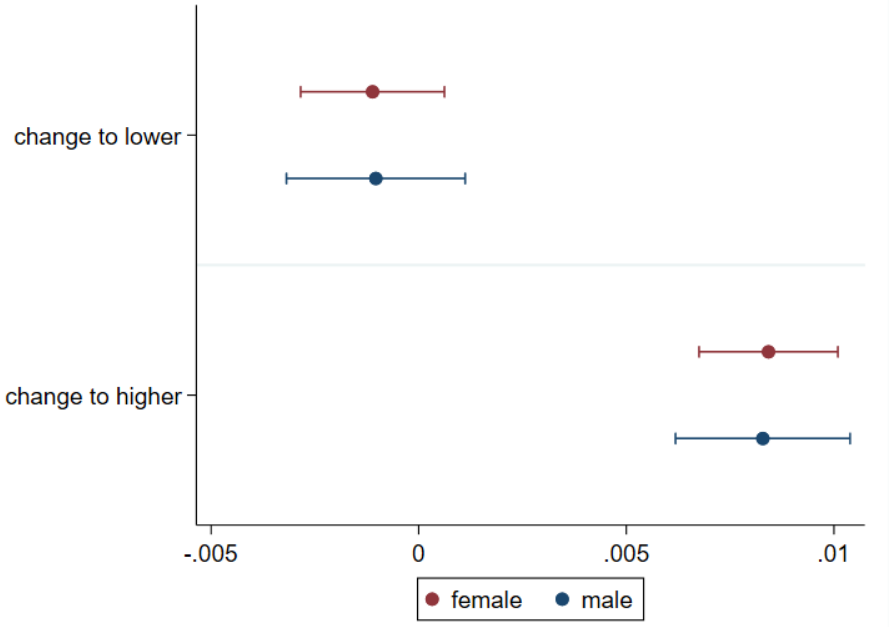
[24] Figure 13 shows the same for other ATC-codes: For ATC-code C01, C03, C05, C08, and C010, the switch to a lower usage rate GP seems to increase the probability of death, albeit mostly insignificantly. For ATC-code C02, C03, C07, and C09 switching to a higher usage rate GP is associated with a reduction in the probability of dying.

Figure 10: Effect of change in female usage rate on own usage of lipid-lowering drugs



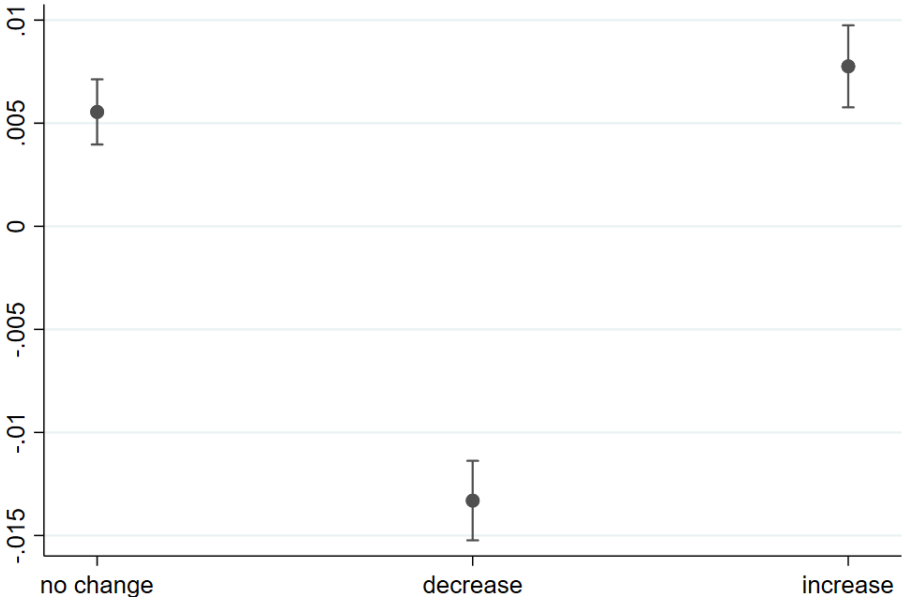
Notes: Figure shows the effect of the change in female usage rates after closure for the probability of receiving medication. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects as well as the change in age and gender composition between the closing and the new clinic. $P(ATC10_{i,t}) = changerate_ATC10_{i,t} + ATC10_{i,t-1} + change\ docgender_{i,t} + change\ docage_{i,t} + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$.

Figure 11: Effect of change in female usage rates on change in probability to use lipid-lowering drugs



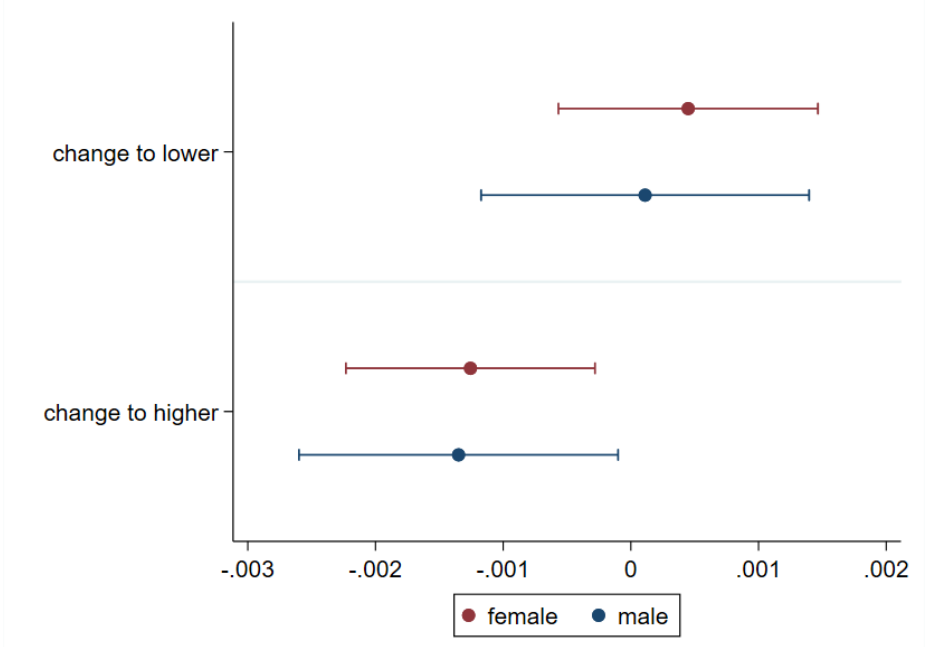
Notes: Figure shows the effect of the change in female usage rates after closure for the probability of receiving medication. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects as well as the change in age and gender composition between the closing and the new clinic. $ATC10_{i,t} = negchange_ATC10_{i,t} \times closure + poschange_ATC10_{i,t} \times closure + closure + ATC10_{i,t-1} + change\ docgender_{i,t} + change\ docage_{i,t} + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$.

Figure 12: Selection into provider with different female usage rates of lipid-lowering drugs, relative risk ratios



Notes: Figure shows relative risk ratios for the type of change in female usage rate individuals experience after a closure. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects as well as the change in age and gender composition between the closing and the new clinic. $changecat_ATC10_{i,t} = female + change\ docgender_{i,t} + change\ docage_{i,t} + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$.

Figure 13: Effect of change in female usage rates on probability of death



Notes: Figure shows the effect of the change in female usage rates after closure for the probability of death during the first five years after closure. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects as well as the change in age and gender composition between the closing and the new clinic. $death_{i,t} = negchange_ATC10_{i,t} \times closure + poschange_ATC10_{i,t} \times closure + closure + change\ docgender_{i,t} + change\ docage_{i,t} + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$.

4.2.3 Mechanisms

The previous section demonstrated substantial influence of GPs on medication usage. This suggests that the low female usage rates of medications to a large extent reflect GP prescription behavior. However, it remains unclear why women are prescribed less than men. There is a multitude of potential reasons. I will discuss diagnostics as the most prominent explanation here but acknowledge that there might be many more. With diagnostics, I mean that GPs could have difficulties in assessing women's symptoms given that many guidelines and clinical trials are based on men (Mauvais-Jarvis et al., 2020). Also, women might report the same symptoms differently than men. It is challenging to investigate this when health is often latent or measured by the (potentially biased) GP.

I therefore combine the register data with cholesterol measurements from the CCHS. This enables me to rely on objectively and independently measured health outcomes since GPs are only presented with the results of the measurements from the CCHS and cannot influence whether the tests are carried out. The measurements were sent to the GP and the respondents for waves 1 and 2, and from wave 3 onwards only to the respondents with the advice to consult their GP. This means that except for the first two waves, where GPs were informed directly, any potential prescription by the GP is largely affected by whether the respondent informed their GP of their results. In a robustness test, I focus on waves 1 and 2 exclusively to avoid the influence of patients not reporting to their GP. I focus on the measurement of total cholesterol. Following guidelines, I define high cholesterol as cholesterol exceeding a threshold of 6.2 millimoles per liter (mmol/L), a threshold also used in Arni et al. (2021) and recommended by the expert panel of the National Cholesterol Education Program in 1988 (Waters, 2019). In the CCHS itself, thresholds were defined as well. These thresholds varied across waves and in waves 1-2 also across age and gender.[25] This was a design choice based on the preference of the physicians who designed the CCHS but not related to guidelines. In a robustness test, I also explore the CCHS thresholds. The reports sent to GPs and patients contained both values of cholesterol and the markers for values outside the normal range, see Figure A5.

Table 2 provides an overview of the cholesterol measurements. In total, around 46,000 measurements were taken throughout the five waves. Total cholesterol is on average quite high, reflected by 24-43% of the sample having cholesterol measurements above the threshold of 6.2 mmol/L, in particular in the first waves. The reason is that during waves 1 and 2, lipid-lowering drugs as well as other cardiovascular drugs were not yet available. They were introduced during the early 90s (in wave 3).[26] I will particularly exploit first high cholesterol measurements. These are defined as a respondent having high cholesterol measured for the first time in the survey and not having used any ATC-code C medication before or having reported a hospital contact related to heart disease. I use this definition to ensure that

[25] See Table note of Table 2.

[26] Note that I do not define high cholesterol for those who already use lipid-lowering drugs as their cholesterol levels cannot be taken at face value since they are affected by the medication.

the high cholesterol measurement is likely new information - based on the data available.[27] Given those restrictions, I can also only use data from wave two onwards for this measure. The thresholds from the survey itself lead to fewer individuals being classified as having high cholesterol. I use the guideline threshold in the main analysis since it reflects the scientific state of the art at the time.

Table 2: CCHS measurements

wave	1	2	3	4	5
# measurements	13,091	11,103	8,698	4,686	2,484
mean (mmol/L)	6.09	5.86	6.10	5.50	5.62
cholesterol (guideline)					
% high cholesterol	42.51	35.48	42.30	24.93	26.97
% first high cholesterol		7.24	13.73	4.25	7.29
cholesterol (CCHS thresholds)					
% high cholesterol	6.61	4.64	21.81	9.43	9.86
% first high cholesterol		2.12	14.30	3.18	5.27

Note: Guideline: High cholesterol is defined as total cholesterol > 6.2 mmol/L. CCHS thresholds: During waves 1 and 2, women were classified to have high cholesterol if: age 20-29 and total cholesterol above 6 mmol/L, age 30-49 and total cholesterol above 7 mmol/L, and older than 50 and total cholesterol above 8 mmol/L. During waves 1 and 2, men were classified to have high cholesterol if: aged 20-29, and total cholesterol over 7 mmol/L, or older than 30, and a total cholesterol above 8 mmol/L. From wave 3 onwards, men and women were classified as having high cholesterol if total cholesterol was above 7 mmol/L. Note that respondents using lipid-lowering drugs or anti-hypertensives are excluded here.

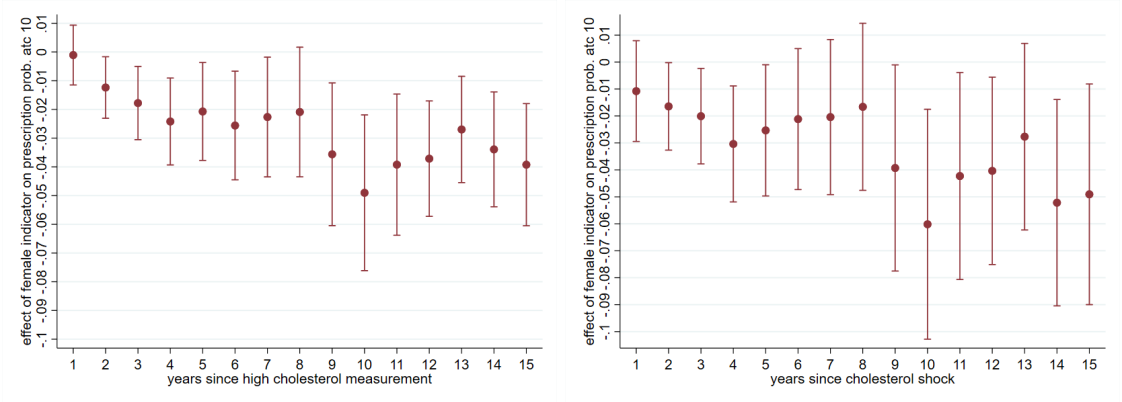
This new information about high cholesterol enables me to study one potential mechanism of the low usage rates of women. If GPs merely do not recognize women's symptoms and do not test for cholesterol, receiving a first measurement of high cholesterol from the CCHS should lead to similar uptake in lipid-lowering drugs across genders. Figure A13 in the appendix investigates the average response to (first) high cholesterol measurements jointly for both genders. It shows that both a high cholesterol measurement and a first cholesterol measurement are associated with an increase in the usage of lipid-lowering drugs over the next 15 years. Note that the effect is smaller for the information shocks, potentially due to the fact that a repeated high cholesterol measure indicates a real need for medication. Figure 14 turns to gender differences: Panel (a) shows the effect of being female on the probability of using ATC-code C10 medication, i.e. lipid-lowering drugs, in the first 15 years after a high cholesterol measurement. Not surprisingly, women have an up to 3 percentage points lower probability of using lipid-lowering drugs. Noteworthy the difference only arises in the second year after the measurement. This might be due to measurement error but also

[27] I cannot observe whether patients have received a cholesterol test at their GP before the CCHS.

because in the first years, I lack information on prescriptions for those measurements in waves two and three because they took place before the prescription register started.

When I then turn to those who have received a high cholesterol measurement for the first time in the survey examination, I would expect that this information leads to similar usage shares of men and women if GPs were just merely not recognizing symptoms or not administering blood tests. Strikingly, in Panel (b) of Figure 14 however I also see a large gender gap arising here, which is even slightly larger than in Panel (a) in the further distance.

Figure 14: Gender differences in usage of lipid-lowering drugs after (first) high cholesterol measurement



(a) high cholesterol measurement

(b) first high cholesterol measurement

Notes: Figure shows the effect of $female$ on the usage of lipid-lowering drugs after receiving (a) a high cholesterol measurement and (b) a first high cholesterol measurement. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects. $ATC10_{i,t} = female_i + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$

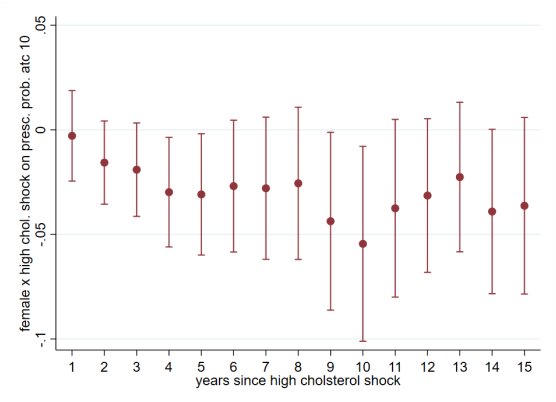
Another way of investigating the differential uptake by gender is to use an interaction specification in the full sample of measurements. Figure 15 plots the coefficients of the interaction between the female indicator and an indicator for a first high-cholesterol shock. This coefficient should be interpreted as indicating the gender difference in the effect of obtaining a high cholesterol shock for uptake. The coefficients are negative and thus confirm the picture above that women with a high cholesterol shock are less likely to use lipid-lowering drugs compared to men who incur the same shock.

In a robustness test, I repeat this exercise with the CCHS thresholds for high cholesterol. In waves 1 and 2 these thresholds also differed by age and gender, whereas from wave 3 onwards everyone with total cholesterol above 7 mmol/L was considered high cholesterol.[28] Importantly the CCHS classification sets lower thresholds for women below 50 to be classified as having high cholesterol.

Implementing these alternative thresholds shrinks the sample of individuals with high

[28] See table notes of Table 2.

Figure 15: Effect of high cholesterol shock on prescription usage probability



(a) first high cholesterol measurement

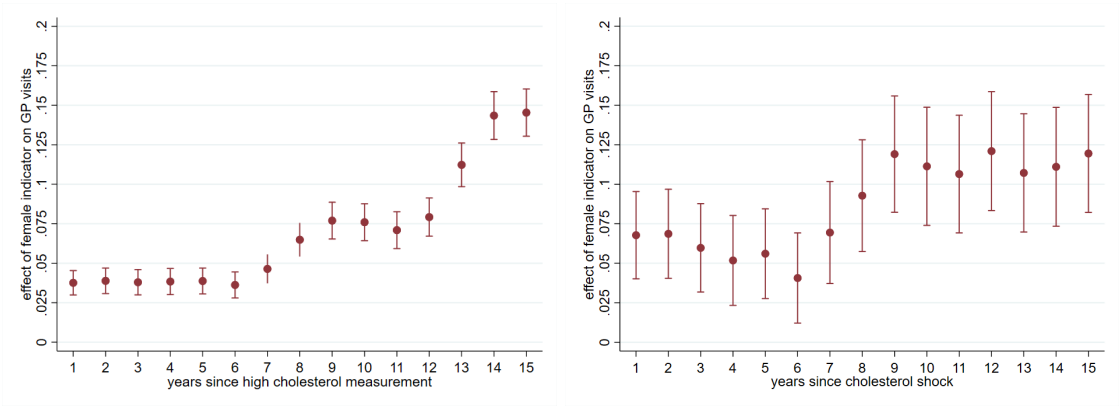
Notes: Figure shows the effect of $female \times high\ cholesterol\ shock$ on the usage of lipid-lowering drugs. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects. $ATC10_{i,t} = female_i + high\ cholesterol\ shock + female \times high\ cholesterol\ shock\lambda_{i,t} + \mu_t + \epsilon_{i,t}$

cholesterol and high cholesterol shocks given that these thresholds are stricter. Nevertheless, Figures A14 and A15 show similar patterns as Figures 14 and 15 - albeit somewhat less precisely estimated due to the reduced sample size. This shows that even when thresholds are favoring women they are less likely to use medication.

These patterns suggest that either GPs do not respond to the information, that women are not seeking care or if they seek care they do not report their high cholesterol from the CCHS to their GP (relevant only from wave 3 onwards), or that they do not want to use lipid-lowering drugs. I test whether care-seeking can explain the observed pattern. If women go to the doctor less after receiving a (first) high cholesterol measurement than men this would explain why they are less likely to use lipid-lowering drugs. Figure 16 repeats the previous exercise but uses the number of visits to the GP as the dependent variable. The figure shows that even after a (first) high cholesterol measurement women visit the GP more often than men - so this cannot explain the gender gap in lipid-lowering usage. However, using the interaction specification, Figure 17 shows that even though women see the doctor more often than men if they receive a cholesterol shock they do not disproportionally do so: the change in GP visits for women with a high cholesterol shock is even a little smaller compared to men with a high cholesterol shock.

To better understand what now drives the gender differences in the uptake of lipid-lowering drugs I exploit the fact that during waves 1 and 2 of the CCHS, results were immediately sent to the GP. This means that GPs in that case had full information and could act upon that when the patient visited the next time. Unfortunately, we can only focus on the uptake of lipid-lowering drugs at a larger time distance due to the starting date of the prescription drug database (around 12 years after wave 2). Nevertheless, it enables studying whether

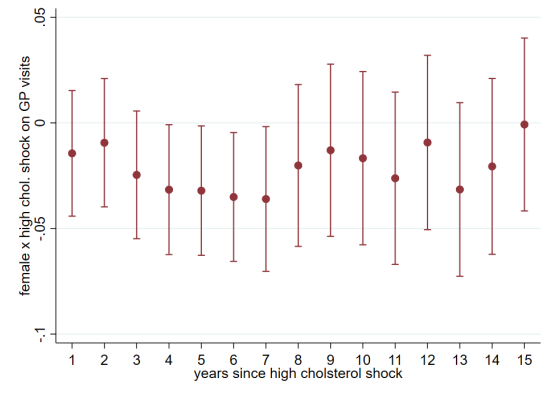
Figure 16: Gender differences in GP visits after (first) high cholesterol measurement



(a) high cholesterol measurement (b) first high cholesterol measurement

Notes: Figure shows the effect of $female$ on the probability of visiting a GP visit after receiving (a) a high cholesterol measurement and (b) a first high cholesterol measurement. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects. $GP\ visit_i, t = female_i + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$.

Figure 17: Effect of high cholesterol shock on GP visits



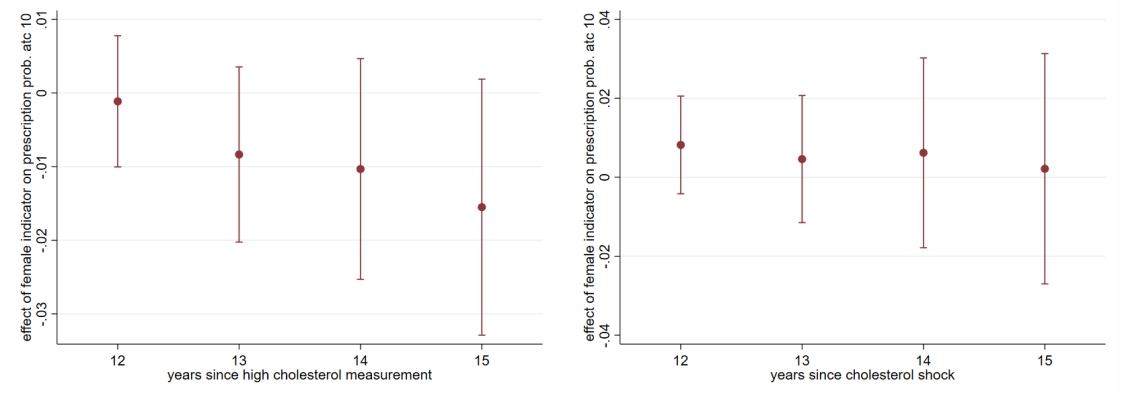
(a) first high cholesterol measurement

Notes: Figure shows the effect of $female \times high\ cholesterol\ shock$ on the probability of visiting a GP. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects. $GP\ visit_i, t = female_i + high\ cholesterol\ shock + female \times high\ cholesterol\ shock + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$

there are gender differences in the uptake of lipid-lowering drugs in the long term if GPs receive information immediately and not only through their patients. Figure 18 Panel (a) below shows that high cholesterol measurement is still associated with slightly lower uptake of women, but insignificantly. Panel (b) however shows no gender differences in uptake. Similarly, the interaction specification in Figure 19 shows no gender differences either. That means that long-term gender differences in the use of lipid-lowering drugs disappear when

information is directly sent to the GP. This indicates two potential channels underlying the existence of gender differences when results have to be shared by the patient: 1) when the responsibility to report results is shifted to women they do not report the results of the blood test or do not use statins despite seeking care more often than men or 2) there are information frictions when women have to bring the reports themselves, e.g. if they do not bring the test results on paper but rather verbally inform their GP. The first channel implies that women are unwilling to share test results or do not want to use statins. It might seem surprising that women seek more healthcare but when it comes to reporting cholesterol levels or using statins seem less active than men. It does however align well with women trying to improve health via alternatives to medication such as lifestyle changes, e.g. diet or exercise. This also is in accordance with survey evidence by Nanna et al. (2019) indicating women are more skeptical about statins. This finding is also consistent with the findings by Simeonova (2013) that patient response to provider input is more important than provider behavior for the racial mortality gap. The second channel points to information frictions related to gender, for example, driven by differential interpretations of the same cholesterol levels depending on who reports them. This is in line with Sarsons (2024) who provides evidence for gender affecting the way that information about ability is interpreted. The evidence presented here suggests that testing alone is not sufficient to reduce the gender gap in the usage of lipid-lowering drugs but that it also matters in which way this information is transmitted to the GP.

Figure 18: Gender differences in usage of lipid-lowering drugs after (first) high cholesterol measurement - wave 1 and 2 only

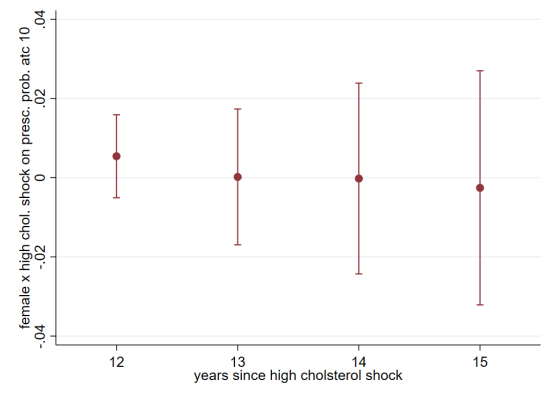


(a) high cholesterol measurement

(b) first high cholesterol measurement

Notes: Figure shows the effect of $female\times$ on the probability of using lipid-lowering drugs after receiving (a) a high cholesterol measurement (wave 1 and 2) and (b) a first high cholesterol measurement (wave 2). Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects. $ATC10_{i,t} = female_i + high\ cholesterol\ shock + female \times high\ cholesterol\ shock + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$

Figure 19: Effect of high cholesterol shock on prescription usage probability - wave 2 only



(a) first high cholesterol measurement

Notes: Figure shows the effect of $female \times high\ cholesterol\ shock$ on the probability of using lipid-lowering drugs, for wave 2 measurements. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects. $ATC10_{i,t} = female_i + high\ cholesterol\ shock + female \times high\ cholesterol\ shock + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$

5 Labor market outcomes

As mentioned above, the effects of undertreatment are likely to extend beyond health. Women with undiagnosed heart disease likely spend more time in debilitating health (Case and Paxson, 2005; World Economic Forum, 2024). This potentially has important consequences for economic productivity such as labor market outcomes. As Figure 2 shows, not everyone with AMI dies right away, and many may return to work. I, therefore, investigate whether health inequalities also spill over to the labor market. More precisely, I investigate whether having AMI has differential effects on the labor market outcomes of women versus men. I test the differential role of experiencing AMI on general labor supply and disability benefit receipt.

Table 3 uses cross-sectional data to show that women indeed face stronger labor market penalties than men when suffering from AMI.[29] While having AMI reduces labor supply, and increases disability benefit receipt, it does so more strongly for women than for men. This result also holds true if I control for comorbidities in the year before AMI - suggesting that the larger AMI penalty for women is not driven by women suffering from additional diseases. Table A5 shows similar results when conditioning on survival for the first ten years after AMI.

In addition to cross-sectional evidence, Figure 20 presents results from event studies using the imputation estimator by Borusyak et al. (2024) estimated separately for men and women. Panel (a) of Figure 20 shows two key features: First, the reduction in women's labor supply is larger than the one of men. Second, before AMI women already have larger negative pre-trends than men. That means in the years before AMI they already reduce labor supply more than men. Panel (b) of Figure 20 shows similar patterns for disability benefits: long before AMI, women have a higher likelihood of being on disability benefits than men and then have a substantial upward jump afterward, leading to even higher levels of disability benefit receipt. This supports the claim that before AMI women likely lived in more debilitating health than men. The findings of this section are in line with evidence of inequities in health spilling over to economic decisions and conditions (De Nardi et al., 2024).

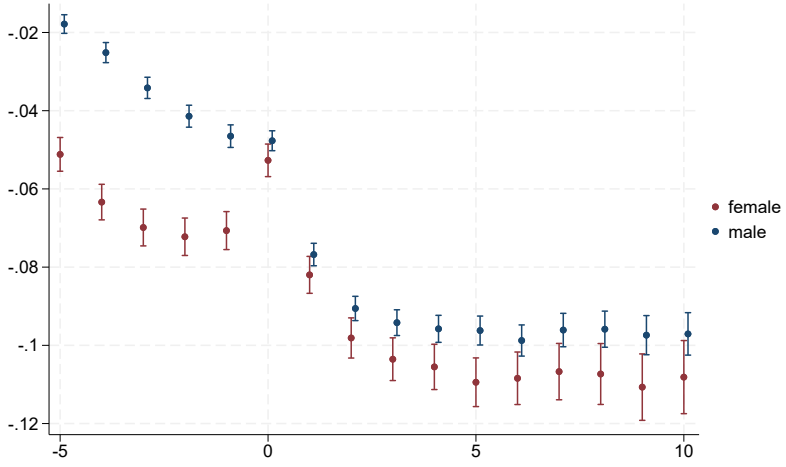
[29] Note that this focuses on all AMIs. Results for first AMIs can be found in the appendix in Table A4.

Table 3: Labor market effects of AMI

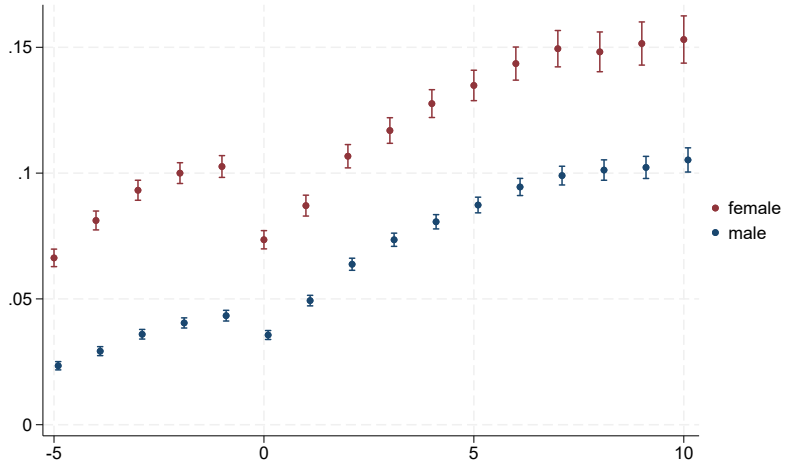
	(1) Working	(2) Disability benefits
AMI_{t-1}	-0.129***	0.073***
	[0.001]	[0.001]
Female	-0.076***	0.015***
	[0.000]	[0.000]
$AMI_{t-1} \times female$	-0.076***	0.104***
	[0.003]	[0.002]
R-squared	0.163	0.054
Observations	121,879,463	121,797,561
Controlling for comorbidity		
AMI_{t-1}	-0.015***	-0.023***
	[0.001]	[0.001]
Female	-0.076***	0.015***
	[0.000]	[0.000]
$AMI_{t-1} \times female$	-0.071***	0.100***
	[0.003]	[0.002]
R-squared	0.165	0.061
Observations	121,879,463	121,797,561

Standard errors clustered on the individual level in brackets; *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. Controls for age and year-fixed effects in all columns. $labor\ supply_{i,t} = AMI_{i,t-1} + female + AMI_{i,t-1} \times female + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$ The lower panel additionally controls for the Charlson Comorbidity Index.

Figure 20: Effect of AMI on labor market outcomes - event study



(a) Working



(b) Disability benefits

Notes: Figure shows event study results, estimated separately for men and women using the did_imputation estimator, using individual (ξi), year ($\lambda_{i,t}$), and age (μ_t) fixed effects. $labor\ supply_{i,t} = distanceAMI_{i,t} + \lambda_{i,t} + \mu_t + \xi_i + \epsilon_{i,t}$

6 Conclusion

This paper studies gender differences in health using the case of heart disease. It investigates the role of individual and provider behavior for the *prevalence-mortality puzzle* - the higher female mortality after AMI despite the higher lifetime (diagnosed) prevalence of heart disease for men. I first document the existence of a substantial mortality disadvantage for women post-AMI, confirming existing evidence. I then show that previous usage of lipid-lowering medication substantially reduces the gender gap. I investigate why women use less medication against heart disease than men. While women on average seek more healthcare and have a healthier lifestyle (less smoking and alcohol) they exercise less and are more pessimistic about their own health. Thus, I turn to the role of the provider. I first document substantial variation in GP-level medication usage rates, and that there are much more GPs with lower female usage rates. I then show that these usage rates are largely driven by GPs' prescription behavior by using information about clinic closures and changes to a new GP. I then test whether difficulties in diagnostics are a mechanism causing the low female usage rates of lipid-lowering drugs. For this, I use objective health measurements from the CCHS. On average, even after receiving a first high cholesterol measurement women are less likely to use lipid-lowering drugs than men with high cholesterol. When zooming in on wave 2, where GPs were automatically informed, gender differences in uptake disappear. This suggests that information transmission matters, either because women are unwilling to report high cholesterol or because there are information frictions when women report the results. Lastly, I show large spillovers of these health inequalities to the labor market with larger anticipation and AMI penalties for women than for men.

These results are not only important from a healthcare perspective but also in general. While population welfare is of key importance, reducing female mortality will have important general economic consequences. Whereas treatment in terms of higher medication is relatively cheap, surviving women will likely incur further health and general care costs. However, early treatment can avoid the full development of diseases. Those women who die at a higher rate after AMI are likely in worse health in the years before. A report by the World Economic Forum (2024) suggests that women spend "25% more of their lives in debilitating health" than men. Despite women in developed countries living longer than men (Goldin and Lleras-Muney, 2019), women have worse self-rated health and more hospitalization episodes than men over the life course (Case and Paxson, 2005). In addition, costs are not restricted to the health sector care but also spill over to the labor market, in the form of reduced labor market participation and higher reliance on social assistance.

Policies encouraging regular testing of cholesterol, and removing biases in doctors' beliefs on the severity of women's symptoms, through information campaigns or in medical training/education seem to be tools of first-order importance to close the gender-mortality gap in heart disease. For example, guidelines advocating for more cholesterol testing would mitigate the impact of the providers potentially having difficulties in interpreting female symptoms while at the same time also reducing the role of women's hesitancy in e.g. reporting

symptoms or information frictions for administering tests. At the same time, information campaigns related to the effectiveness of statins might remove hesitancy in women to use statins.

References

Aggarwal, N. R., Patel, H. N., Mehta, L. S., Sanghani, R. M., Lundberg, G. P., Lewis, S. J., Mendelson, M. A., Wood, M. J., Volgman, A. S., and Mieres, J. H. (2018). Sex Differences in Ischemic Heart Disease. *Circulation: Cardiovascular Quality and Outcomes*, 11(2):e004437. Publisher: American Heart Association.

Aguib, Y. and Al Suwaidi, J. (2015). The copenhagen city heart study (Østerbroundersøgelsen). *Global cardiology science & practice*, 33.

Alsan, M., Garrick, O., and Graziani, G. (2019). Does Diversity Matter for Health? Experimental Evidence from Oakland. *American Economic Review*, 109(12):4071–4111.

Arni, P., Dragone, D., Goette, L., and Ziebarth, N. R. (2021). Biased health perceptions and risky health behaviors—theory and evidence. *Journal of Health Economics*, 76:102425.

Bairey Merz, C. N. (2014). Sex, death, and the diagnosis gap. *Circulation*, 130(9):740–742.

Banco, D., Chang, J., Talmor, N., Wadhera, P., Mukhopadhyay, A., Lu, X., Dong, S., Lu, Y., Betensky, R. A., Blecker, S., et al. (2022). Sex and race differences in the evaluation and treatment of young adults presenting to the emergency department with chest pain. *Journal of the American Heart Association*, 11(10):e024199.

Berger, J. S., Elliott, L., Gallup, D., Roe, M., Granger, C. B., Armstrong, P. W., Simes, R. J., White, H. D., Van de Werf, F., Topol, E. J., et al. (2009). Sex differences in mortality following acute coronary syndromes. *Jama*, 302(8):874–882.

Bertakis, K. D., Azari, R., Helms, L. J., Callahan, E. J., and Robbins, J. A. (2000). Gender differences in the utilization of health care services. *Journal of family practice*, 49(2).

Borusyak, K., Jaravel, X., and Spiess, J. (2024). Revisiting event-study designs: robust and efficient estimation. *Review of Economic Studies*, page rdae007.

Cabral, M. and Dillender, M. (2024). Gender differences in medical evaluations: Evidence from randomly assigned doctors. *American Economic Review*, 114(2):462–499.

Case, A. and Paxson, C. (2005). Sex differences in morbidity and mortality. *Demography*, 42(2):189–214.

Christensen, B. J., Gørtz, M., and Kallestrup-Lamb, M. (2016). Medical spending in denmark. *Fiscal Studies*, 37(3-4):461–497.

Cobb-Clark, D. A., Kassenboehmer, S. C., and Schurer, S. (2014). Healthy habits: The connection between diet, exercise, and locus of control. *Journal of Economic Behavior & Organization*, 98:1–28.

Cohen, A. and Einav, L. (2007). Estimating risk preferences from deductible choice. *American economic review*, 97(3):745–788.

Collins, R. (2003). Heart protection study collaborative group: Mrc/bhf heart protection study of cholesterol-lowering with simvastatin in 5963 people with diabetes: a randomised placebo-controlled trial. *Lancet*, 361:2005–2016.

Currie, J., MacLeod, W. B., and Van Parys, J. (2016). Provider practice style and patient health outcomes: The case of heart attacks. *Journal of Health Economics*, 47:64–80.

Cutler, D. M., Skinner, J. S., Stern, A. D., and Wennberg, D. (2019). Physician Beliefs and Patient Preferences: A New Look at Regional Variation in Health Care Spending. *American Economic Journal: Economic Policy*, 11(1):192–221.

De Nardi, M., Pashchenko, S., and Porapakkarm, P. (2024). The lifetime costs of bad health. *Review of Economic Studies*, page rdae080.

Emanuel, E. J., Gudbranson, E., Van Parys, J., Gørtz, M., Helgeland, J., and Skinner, J. (2021). Comparing Health Outcomes of Privileged US Citizens With Those of Average Residents of Other Developed Countries. *JAMA Internal Medicine*, 181(3):339–344.

ESC Cardiovasc Risk Collaboration and SCORE2 Working Group (2021). Score2 risk prediction algorithms: new models to estimate 10-year risk of cardiovascular disease in europe. *European heart journal*, 42(25):2439–2454.

Forde, I., Nader, C., Socha-Dietrich, K., Oderkirk, J., and Colombo, F. (2017). Primary Care in Denmark.

Ginja, R., Riise, J., Willage, B., and Willén, A. (2022). Does your doctor matter? doctor quality and patient outcomes. *NHH Dept. of Economics Discussion Paper*, (08).

Goldin, C. and Lleras-Muney, A. (2019). "xx>xy?: The changing female advantage in life expectancy". *Journal of Health Economics*, 67:102224.

Greenwood, B. N., Carnahan, S., and Huang, L. (2018). Patient–Physician Gender Concordance and Increased Mortality among Female Heart Attack Patients. *Proceedings of the National Academy of Sciences*, 115(34):8569–8574.

Group, H. P. S. C. et al. (2002). Mrc/bhf heart protection study of cholesterol lowering with simvastatin in 20 536 high-risk individuals: a randomised placebocontrolled trial. *The Lancet*, 360(9326):7–22.

Grundy, S. M., Stone, N. J., Bailey, A. L., Beam, C., Birtcher, K. K., Blumenthal, R. S., Braun, L. T., de Ferranti, S., Faiella-Tommasino, J., Forman, D. E., Goldberg, R., Heidenreich, P. A., Hlatky, M. A., Jones, D. W., Lloyd-Jones, D., Lopez-Pajares, N., Ndumele, C. E., Orringer, C. E., Peralta, C. A., Saseen, J. J., Smith, S. C., Sperling, L., Virani, S. S., and Yeboah, J. (2019). 2018 aha/acc/aacvpr/aapa/abc/acpm/ada/ags/apha/aspc/nla/pcna guideline on the management of blood cholesterol: A report of the american college of cardiology/american heart association task force on clinical practice guidelines. *Circulation*, 139(25):e1082–e1143.

Gulati, M. and Merz, C. N. B. (2015). New cholesterol guidelines and primary prevention in women. *Trends in Cardiovascular Medicine*, 25(2):84–94.

Hanratty, B., Lawlor, D. A., Robinson, M. B., Sapsford, R. J., Greenwood, D., and Hall, A. (2000). Sex differences in risk factors, treatment and mortality after acute myocardial infarction: an observational study. *Journal of Epidemiology & Community Health*, 54(12):912–916.

Jayachandran, S., Lleras-Muney, A., and Smith, K. V. (2010). Modern medicine and the twentieth century decline in mortality: Evidence on the impact of sulfa drugs. *American Economic Journal: Applied Economics*, 2(2):118–146.

Kämpfen, F. and Mosca, I. (2024). Heterogeneous effects of blood pressure screening. *IZA Discussion Paper*, 16767.

Karcioğlu, Ö., Aslan, B. Ü., and Aslan, Ö. (2002). Gender differences in the management and survival of patients with acute myocardial infarction. *European Journal of Internal Medicine*, 13(8):474–479.

Koulayev, S., Simeonova, E., and Skipper, N. (2017). Can physicians affect patient adherence with medication? *Health Economics*, 26(6):779–794.

Kristiansen, I. L. and Sheng, S. Y. (2022). Doctor who? the effect of physician-patient match on the ses-health gradient. *The Effect of Physician-Patient Match on The SES-Health Gradient (July 8, 2022)*.

Mauvais-Jarvis, F., Merz, N. B., Barnes, P. J., Brinton, R. D., Carrero, J.-J., DeMeo, D. L., De Vries, G. J., Epperson, C. N., Govindan, R., Klein, S. L., et al. (2020). Sex and gender: modifiers of health, disease, and medicine. *The Lancet*, 396(10250):565–582.

Mehta, L. S., Beckie, T. M., DeVon, H. A., Grines, C. L., Krumholz, H. M., Johnson, M. N., Lindley, K. J., Vaccarino, V., Wang, T. Y., Watson, K. E., and Wenger, N. K. (2016). Acute myocardial infarction in women: A scientific statement from the american heart association. *Circulation*, 133.

Mozaffarian, D., Benjamin, E., Go, A., Arnett, D., Blaha, M., Cushman, M., de Ferranti, Després, J.-P., Fullerton, H., Howard, V., Huffman, M., Judd, S., Kissela, B., Lackland, D., Lichtman, J., Lisabeth, L., Liu, S., Mackey, R., Matchar, D., McGuire, D., Mohler, E., Moy, C., Muntner, P., Mussolino, M., Nasir, K., Neumar, R., Nichol, G., Palaniappan, L., Pandey, D., Reeves, M., Rodriguez, C., Sorlie, P., Stein, J., Towfighi, A., uran, T., Virani, S., Willey, J., Woo, D., Yeh, R., and Turner, M. (2015). Heart disease and stroke statistics–2015 update: a report from the american heart association. *Circulation*, 131(4).

Nanna, M. G., Wang, T. Y., Xiang, Q., Goldberg, A. C., Robinson, J. G., Roger, V. L., Virani, S. S., Wilson, P. W., Louie, M. J., Koren, A., et al. (2019). Sex differences in the use of statins in community practice: patient and provider assessment of lipid management registry. *Circulation: Cardiovascular Quality and Outcomes*, 12(8):e005562.

OECD (2016). Expenditure by disease, age and gender.

Pope, J. H., Aufderheide, T. P., Ruthazer, R., Woolard, R. H., Feldman, J. A., Beshansky, J. R., Griffith, J. L., and Selker, H. P. (2000). Missed diagnoses of acute cardiac ischemia in the emergency department. *New England Journal of Medicine*, 342(16):1163–1170.

Pottegård, A., Christensen, R. d., Houji, A., Christiansen, C. B., Paulsen, M. S., Thomsen, J. L., and Hallas, J. (2014). Primary non-adherence in general practice: a danish register study. *European journal of clinical pharmacology*, 70:757–763.

Preston, S. H. and Wang, H. (2006). Sex mortality differences in the united states: The role of cohort smoking patterns. *Demography*, 43(4):631–646.

Quan, H., Li, B., Couris, C. M., Fushimi, K., Graham, P., Hider, P., Januel, J.-M., and Sundararajan, V. (2011). Updating and validating the charlson comorbidity index and score for risk adjustment in hospital discharge abstracts using data from 6 countries. *American journal of epidemiology*, 173(6):676–682.

Quan, H., Sundararajan, V., Halfon, P., Fong, A., Burnand, B., Luthi, J.-C., Saunders, L. D., Beck, C. A., Feasby, T. E., and Ghali, W. A. (2005). Coding algorithms for defining comorbidities in icd-9-cm and icd-10 administrative data. *Medical care*, 43(11):1130–1139.

Sarsons, H. (2024). Interpreting signals in the labor market: evidence from medical referrals. *Working Paper*, pages 141–145.

Schnorh, P. (2009). Physical activity in leisure time: impact on mortality. *Danis Medical Bulletin*, 56:40–71.

Schünemann, J., Strulik, H., and Trimborn, T. (2017). The gender gap in mortality: How much is explained by behavior? *Journal of health economics*, 54:79–90.

Schünemann, J., Strulik, H., and Trimborn, T. (2023). Anticipation of deteriorating health and information avoidance. *Journal of Health Economics*, 89:102755.

Sever, P. S., Dahlöf, B., Poulter, N. R., Wedel, H., Beevers, G., Caulfield, M., Collins, R., Kjeldsen, S. E., Kristinsson, A., McInnes, G. T., et al. (2003). Prevention of coronary and stroke events with atorvastatin in hypertensive patients who have average or lower-than-average cholesterol concentrations, in the anglo-scandinavian cardiac outcomes trial—lipid lowering arm (ascot-lla): a multicentre randomised controlled trial. *The Lancet*, 361(9364):1149–1158.

Simeonova, E. (2013). Doctors, patients and the racial mortality gap. *Journal of Health Economics*, 32(5):895–908.

Simeonova, E., Skipper, N., and Thingholm, P. R. (2024). Physician health management skills and patient outcomes. *Journal of Human Resources*, 59(3):777–809.

Tan, Y. C., Sinclair, H., Ghoorah, K., Teoh, X., Mehran, R., and Kunadian, V. (2016).

Gender differences in outcomes in patients with acute coronary syndrome in the current era: A review. *European Heart Journal: Acute Cardiovascular Care*, 5(7):51–60.

Wardle, J., Haase, A. M., Steptoe, A., Nillapun, M., Jonwutiwes, K., and Bellisie, F. (2004). Gender differences in food choice: the contribution of health beliefs and dieting. *Annals of behavioral medicine*, 27:107–116.

Waters, D. D. (2019). Cholesterol lowering guidelines: From whence we came and where we are now. *Canadian Journal of Cardiology*, 35(5):590–597.

Wenger, N. K. (2012). Women and coronary heart disease: a century after herrick: understudied, underdiagnosed, and undertreated. *Circulation*, 126(5):604–611.

WHO (2020). Global Health Estimates 2020: Deaths by Cause, Age, Sex, by Country and by Region, 2000-2019.

Witthöft, M. (2013). *Pathophysiology*, pages 1443–1445. Springer New York, New York, NY.

World Economic Forum (2024). Closing the Women's Health Gap: A $1 Trillion Opportunity to Improve Lives and Economies. Technical report, World Economic Forum in collaboration with the McKinsey Health Institute.

Wu, R., Williams, C., Zhou, J., Schlackow, I., Emberson, J., Reith, C., Keech, A., Robson, J., Wilkinson, K., Armitage, J., et al. (2022). Benefit accrual with cardiovascular disease prevention and effects of discontinuation: a modelling study. *European Heart Journal*, 43(Supplement_2):ehac544–2850.

Appendix

Table A1: ATC-codes category C (cardiovascular system drugs)

Code	Medical term	Layman's term
C01	Cardiac therapy	Treats cardiac arrhythmia angina pectoris
C03	Diuretics	Increases diuresis (increase in urine passed by the body), reduces fluid accumulation in the body
C02	Antihypertensives	Lowers blood-pressure
C04	Peripheral vasodilators	Causes dilation of peripheral arteries re-distributing blood from central arteries to peripheral arteries relieving and protecting the heart from the excess pressure of blood
C05	Vasoprotectives	Stabilising venous vessels
C08	Calcium channel blockers	Lower blood pressure by stopping calcium from entering the cells of the heart and arteries
C07	Beta-blocking agents	Lowers blood pressure and heart rate by blocking receptors normally used for increasing blood pressure and heart rate
C09	Agents acting on the renin-angiotensin system	Act on a hormone system that helps to control blood pressure and the amount of fluid in the body.
C10	Lipid modifying agents	Lower the level of total cholesterol and low-density lipoprotein (LDL) cholesterol in the blood. Often referred to as statins as this is the most commonly used drug.

Table A2: CCHS population - demographics

	invited	participating	p-value
Age	59.13	55.82	0.000
	(16.67)	(13.93)	
Married	0.52	0.70	0.000
	(0.50)	(0.46)	
Number of GP visits	6.17	7.09	0.000
	(10.14)	(8.71)	
Number of specialist visits	3.65	6.86	0.000
	(8.53)	(10.14)	
Charlson Comorbidity Index	0.19	0.09	0.000
	(0.66)	(0.45)	
Income (DKK)	56,013.52	85,348.280	0.000
	(115,241.650)	(123,807.10)	
Education (1-6,	2.30	2.54	0.000
primary-Master/PhD)	(1.64)	(1.63)	

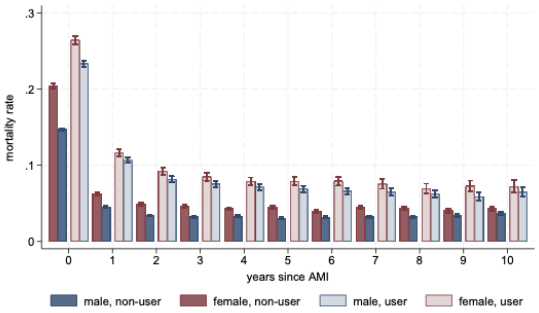
Note: Table shows means and standard deviations (in brackets). I exclude wave 5 due to insufficient information about invitations. Note that information on marital status is only available from wave 3 onwards, and that information on HP and specialist visits is available from wave 4 onwards. Information on the Charlson comorbidity index and income is available from wave 2 onwards.

Table A3: Health behavior differences across gender - conditional on objective health

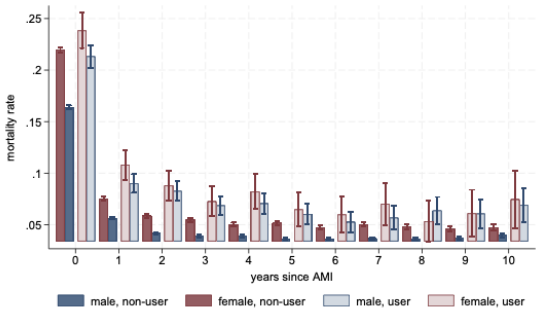
	(1) Current Smoker	(2) Alcohol freq	(3) Phys. activity	(4) Self-assessed health
Female	–0.114***	-0.479***	-0.145***	0.121***
	[0.005]	[0.007]	[0.007]	[0.016]
Constant	1.062***	2.045***	2.803***	-0.827**
	[0.120]	[0.174]	[0.180]	[0.414]
R-squared	0.104	0.245	0.068	0.050
Observations	39,353	39,316	39,251	15,325

Standard errors clustered on the individual level in brackets; $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$. Controls for age and year-fixed effects in all columns. Includes controls for objective health: systolic and diastolic blood pressure, total cholesterol, and BMI. $current\ smoker = female + i.age + i.year + objective\ health_i + \epsilon_i$

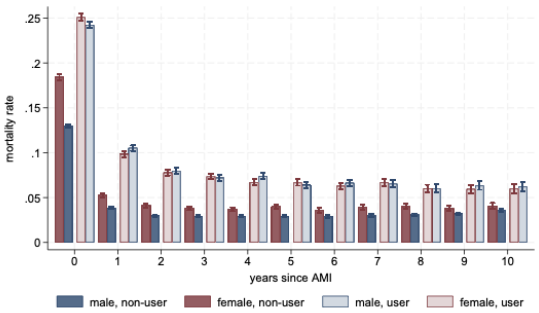
Figure A1: Gender gap in mortality after AMI - previous use



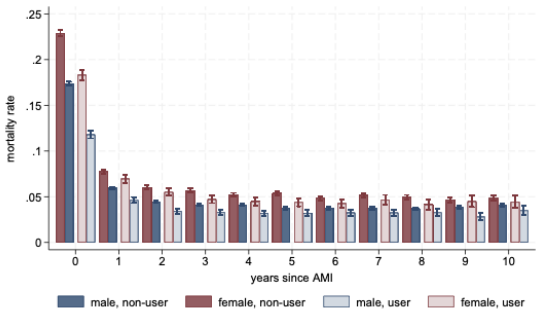
(a) C01 (Cardiac therapy)



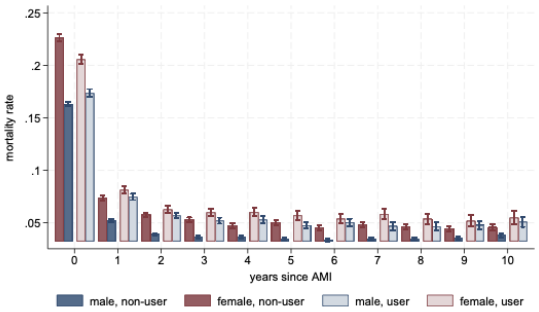
(b) C02 (Antihypertensives)



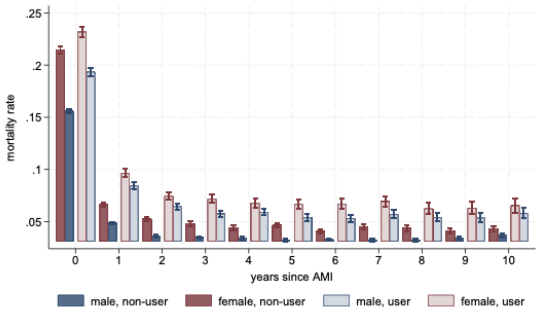
(c) C03 (Diuretics)



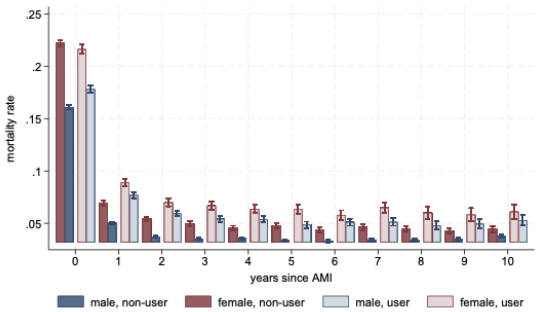
(d) C05 (Vasoprotectives)



(e) C07 (Beta blocking agents)



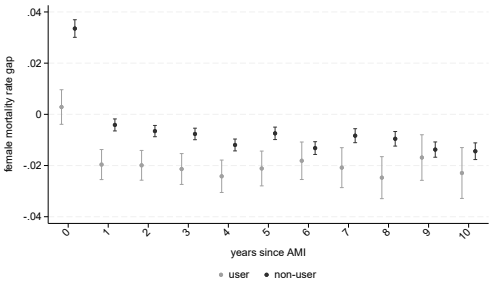
(f) C08 (Calcium channel blockers)



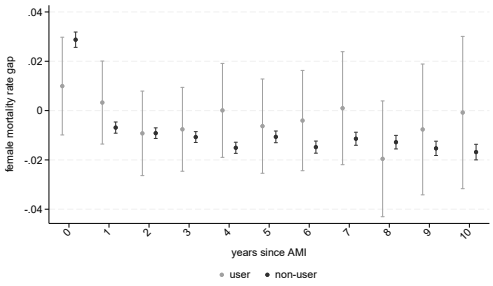
(g) C09 (agents acting on the renin-angiotensin system)

Note: Figure shows the mortality rate after AMI for 10 years, split by gender and previous use.

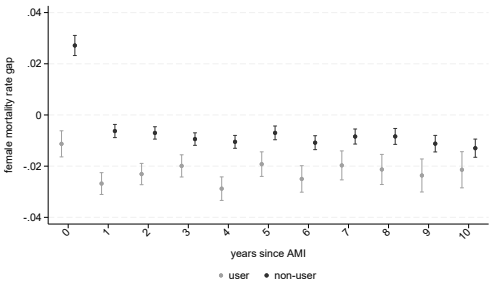
Figure A2: Gender gap in mortality after AMI - previous use - controlling for age



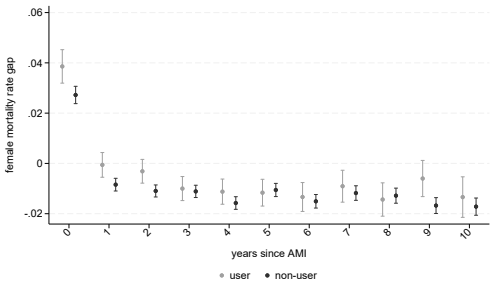
(a) C01 (Cardiac therapy)



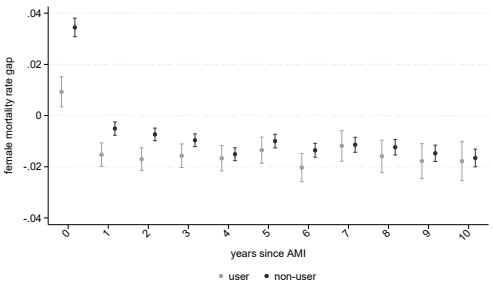
(b) C02 (Antihypertensives)



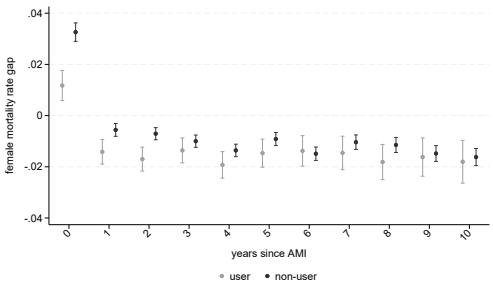
(c) C03 (Diuretics)



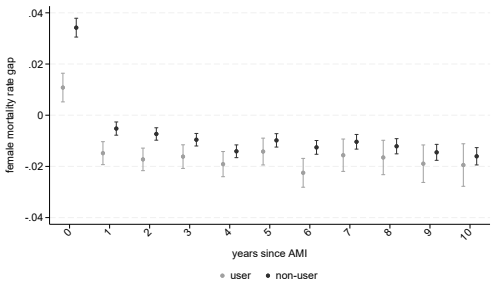
(d) C05 (Vasoprotectives)



(e) C07 (Beta blocking agents)



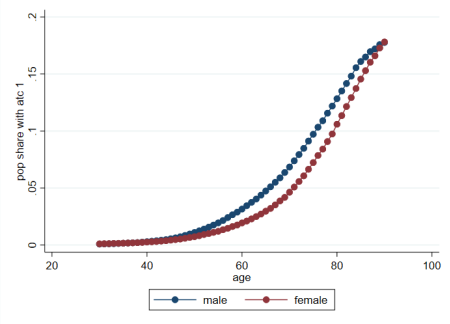
(f) C08 (Calcium channel blockers)



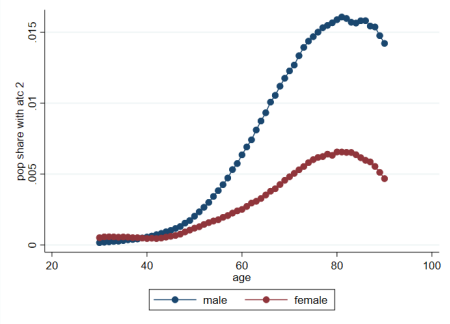
(g) C09 (agents acting on the renin-angiotensin system)

Notes: Figure shows coefficients from regressing an indicator for death on the $yrs\ since\ AMI \times female$, separately for users, and non-users of the respective ATC-c codes. Controlling for age-fixed effects ($\lambda_{i,t}$) and year-fixed effects (μ_t). Estimated separately for previous users and non-users of lipid-lowering drugs: $death_{i,t} = yrs\ since\ AMI \times female + yrs\ since\ AMI + +\lambda_{i,t} + \mu_t + \epsilon_{i,t}$.

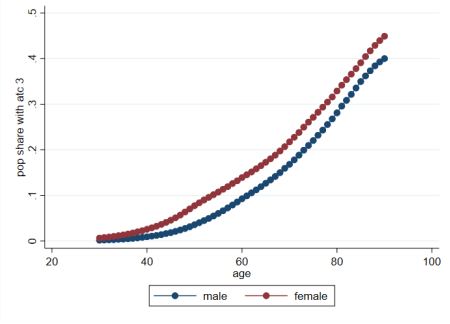
Figure A3: Gender differences in usage



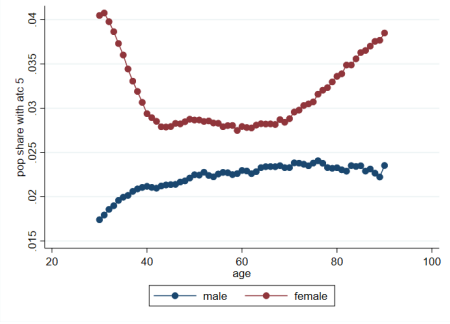
(a) C01 (Cardiac therapy)



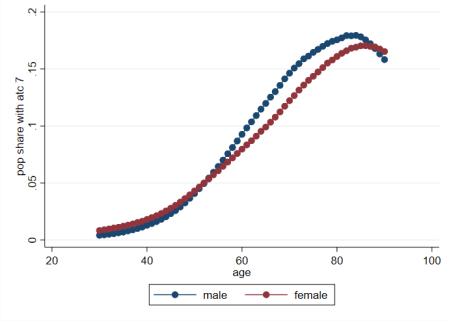
(b) C02 (Antihypertensives)



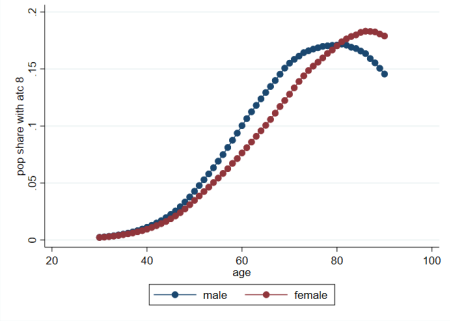
(c) C03 (Diuretics)



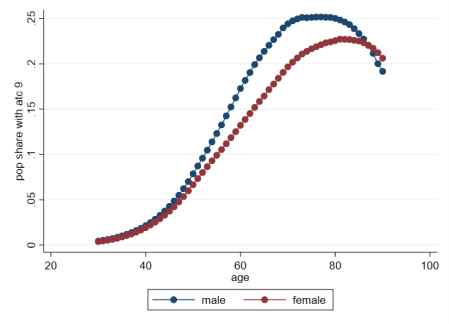
(d) C05 (Vasoprotectives)



(e) C07 (Beta blocking agents)



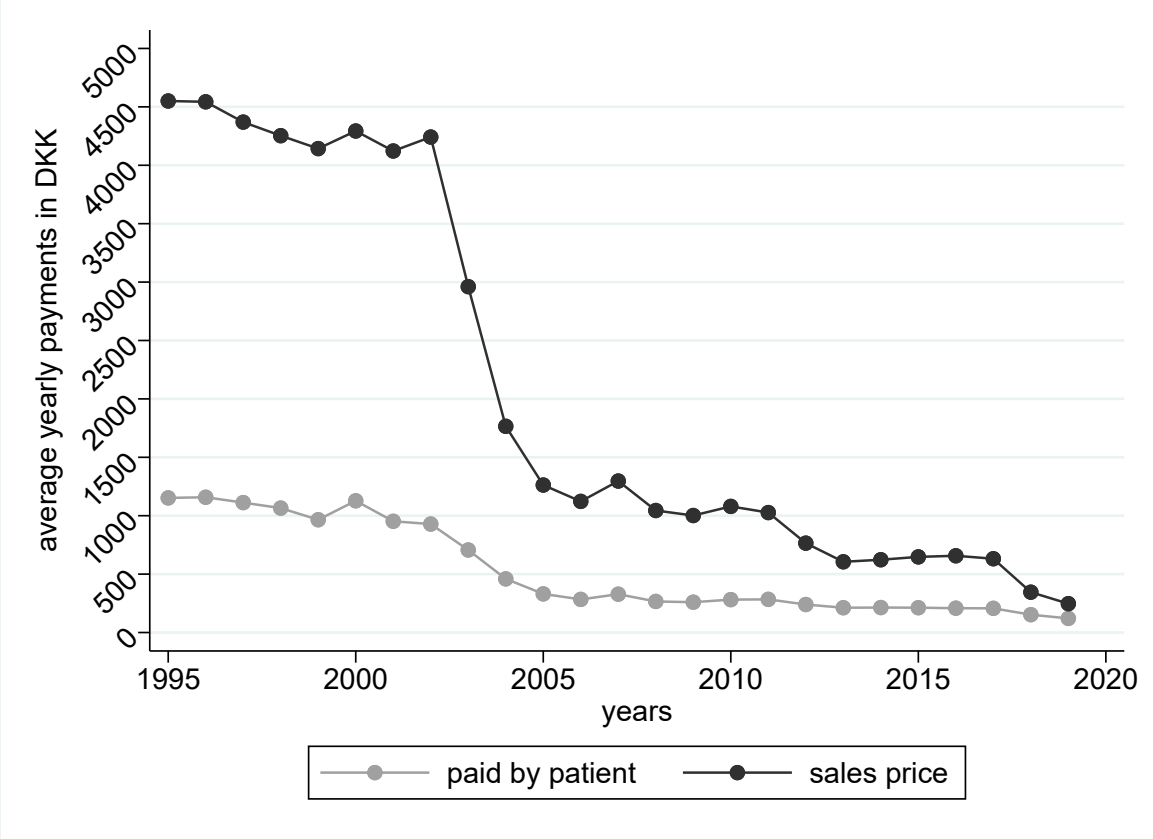
(f) C08 (Calcium channel blockers)



(g) C09 (agents acting on the renin-angiotensin system)

Note: Figure shows usage rates for different ATC-codes on the whole population over age, split by gender.

Figure A4: Payment for lipid-lowering drugs over time



Note: Figure shows average yearly payments by the patient and average yearly sales prices of lipid-lowering drugs. The payments paid by patients are much lower than sales prices due to the generous subsidies.

Figure A5: CCHS measurements report card

Østerbroundersøgelsen
Bispebjerg og Frederiksberg Hospital
Nordre Fasanvej 57-59
Hovedvejen, Indgang 5 st
2000 Frederiksberg
Mail: Oebus6@regionh.dk
Telefon: 38 16 36 70

ØBUS 6 nr. Hermed sender vi dine resultater fra d. . . . .. . / . . . . . .

Ikke fastende blodprøver		Normal	Bør kontrolleres hos egen læge indenfor 1 måned	Ej udført
Hæmoglobin (blodprocent)	. . . . . mMol/L	☐	☐	☐
TSH (stofskifte)	. . . . . mIU/L	☐	☐	☐
ALAT (leverfunktion)	. . . . . U/L	☐	☐	☐
Kolesterol	. . . . . mMol/L	☐	☐	☐
LDL-kolesterol	. . . . . mMol/L	☐	☐	☐
Kreatinin (nyre funktion)	. . . . µMol/L	☐	☐	☐
Blodsukker	. . . . . mMol/L	☐	☐	☐
Triglycerid	. . . . . mMol/L	☐	☐	☐

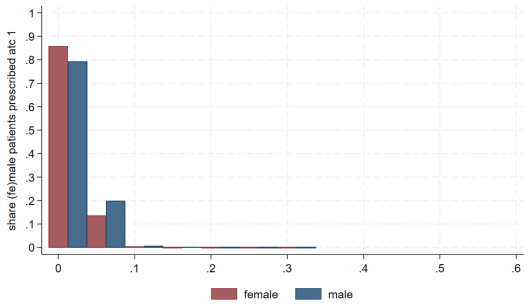
Hjerte og kredsløb				
Blodtryk	. . . . . / . . . . mmHg	☐	☐	☐
EKG		☐	☐	☐

Lungefunktion	Normal	Let nedsat	Kontroller hos læge	Ej udført
	☐	☐	☐	☐

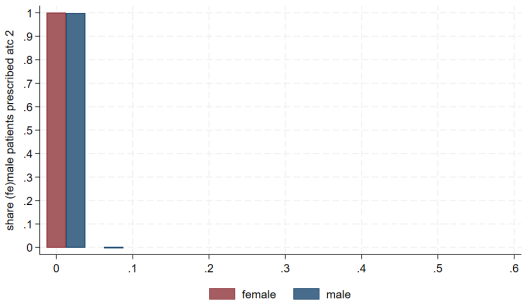
Med venlig hilsen og tak fordi du deltog
Østerbroundersøgelsen

D.16.11.2021 Version 1

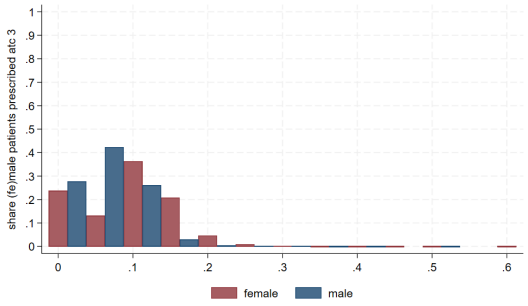
Figure A6: Distribution of GP-level male and female usage rates



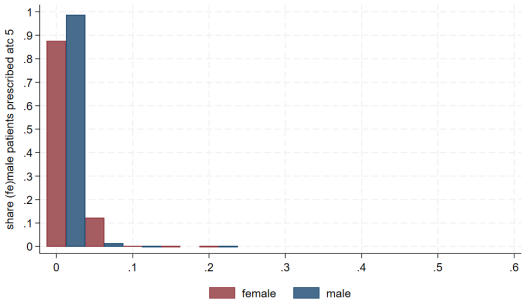
(a) C01 (Cardiac therapy)



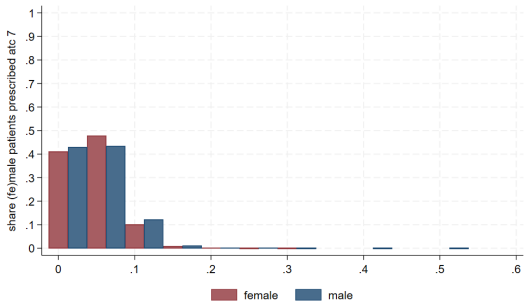
(b) C02 (Antihypertensives)



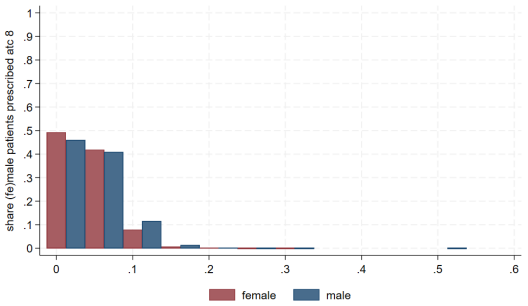
(c) C03 (Diuretics)



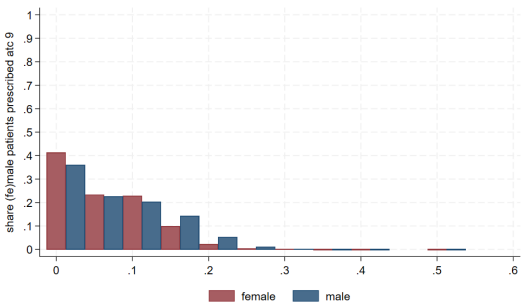
(d) C05 (Vasoprotectives)



(e) C07 (Beta blocking agents)



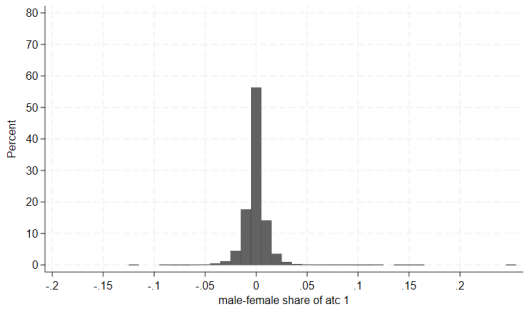
(f) C08 (Calcium channel blockers)



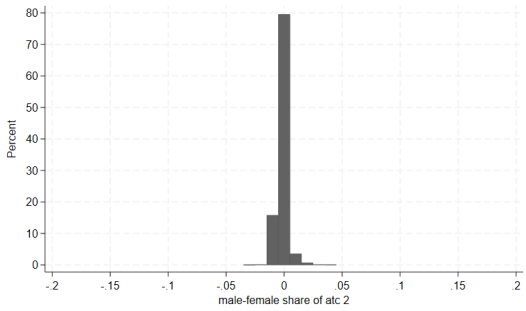
(g) C09 (agents acting on the renin-angiotensin system)

Note: Figure shows the distribution of GPs' female and male usage share.

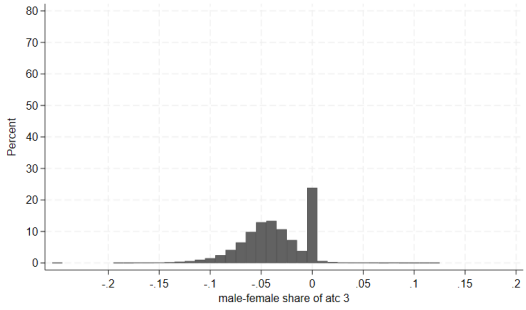
Figure A7: GP-level gap between male and female usage rates



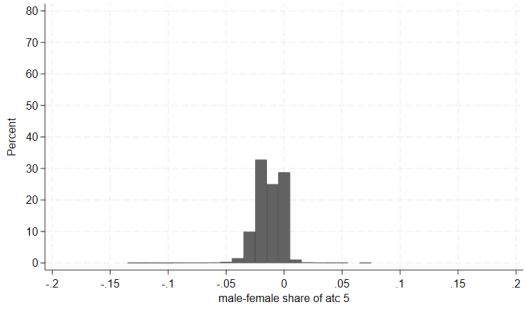
(a) C01 (Cardiac therapy)



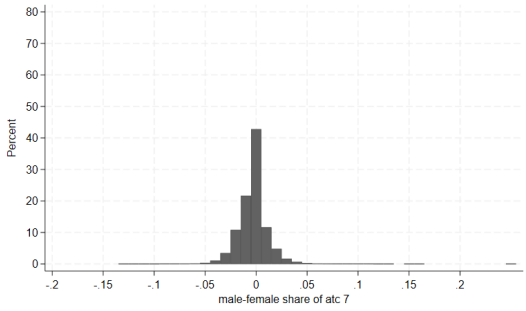
(b) C02 (Antihypertensives)



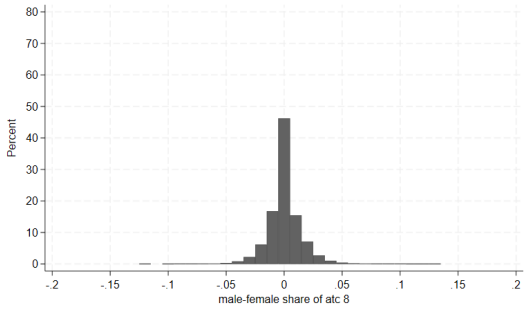
(c) C03 (Diuretics)



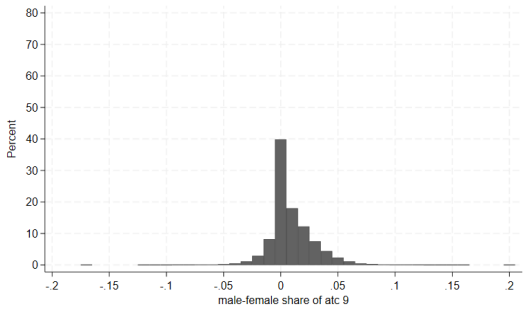
(d) C05 (Vasoprotectives)



(e) C07 (Beta blocking agents)



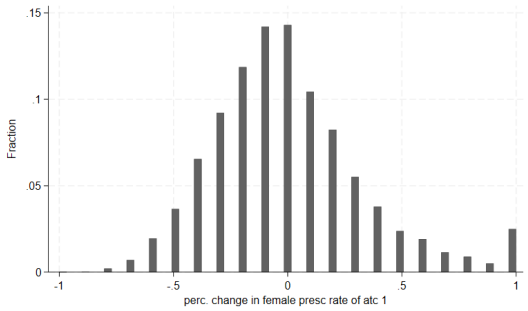
(f) C08 (Calcium channel blockers)



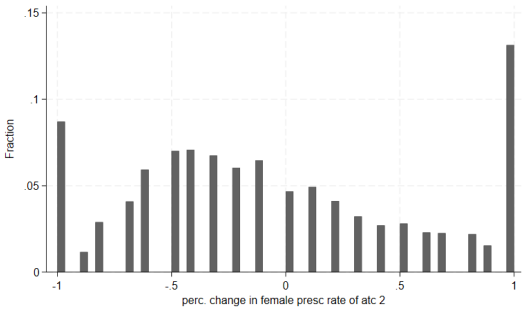
(g) C09 (agents acting on the renin-angiotensin system)

Note: Figure shows the distribution of the within-GP difference between female and male usage shares.

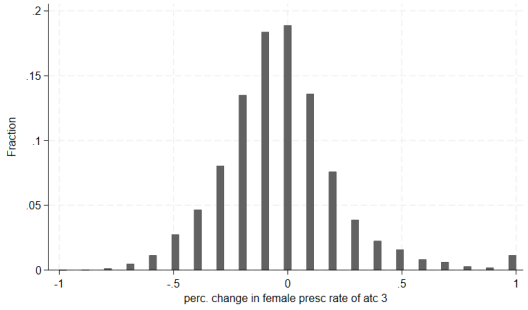
Figure A8: Distribution of change in female usage rates after closure



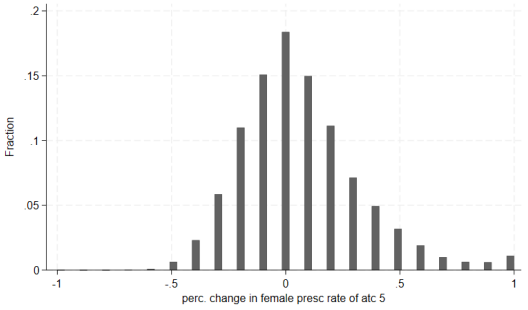
(a) C01 (Cardiac therapy)



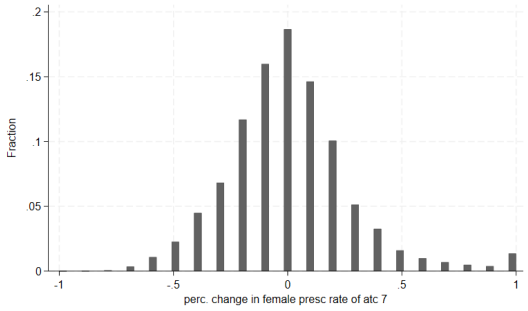
(b) C02 (Antihypertensives)



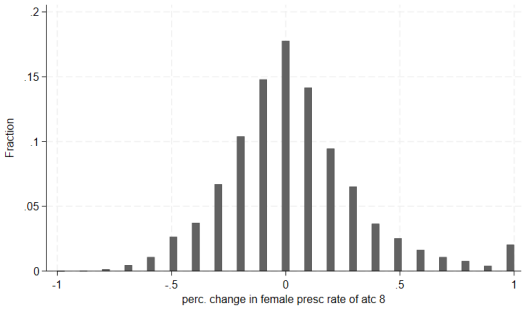
(c) C03 (Diuretics)



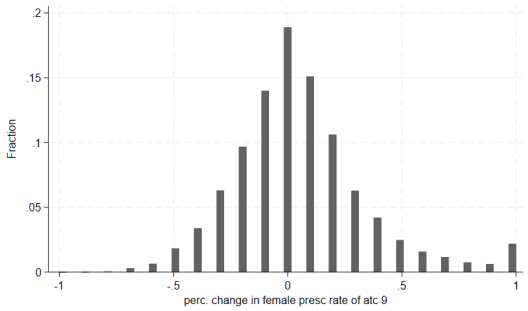
(d) C05 (Vasoprotectives)



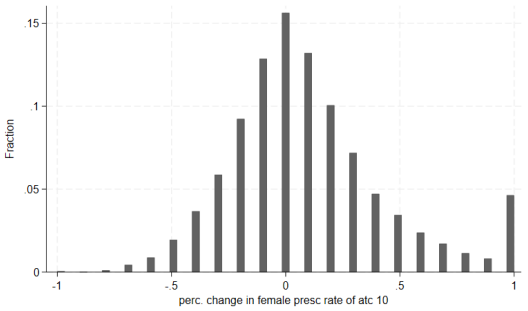
(e) C07 (Beta blocking agents)



(f) C08 (Calcium channel blockers)



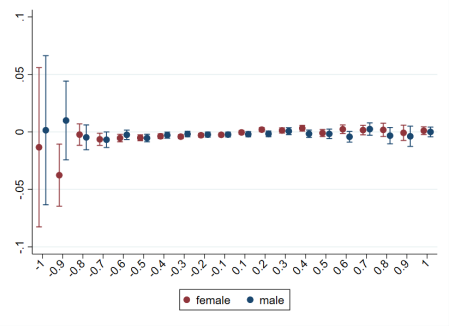
(g) C09 (agents acting on the renin-angiotensin system)



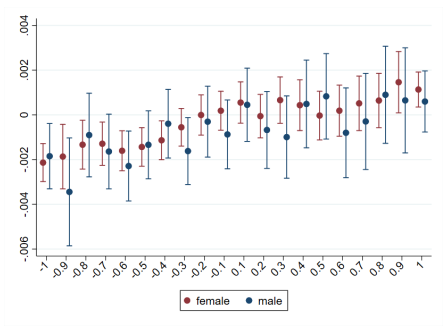
(h) C10 (lipid-lowering agents)

Note: Figure shows the distribution of the change in female usage rates after a closure.

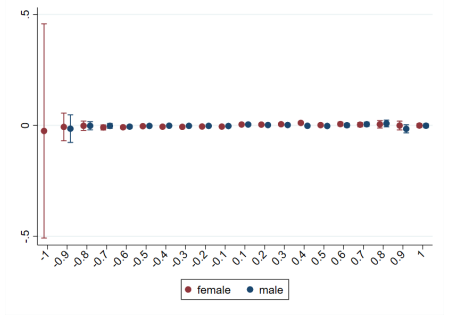
Figure A9: Effect of change in female usage rate on own usage



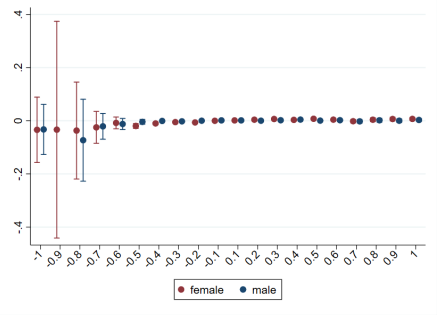
(a) C01 (Cardiac therapy)



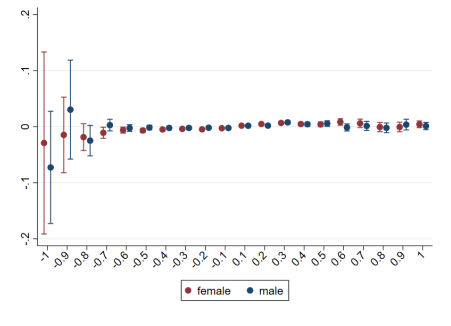
(b) C02 (Antihypertensives)



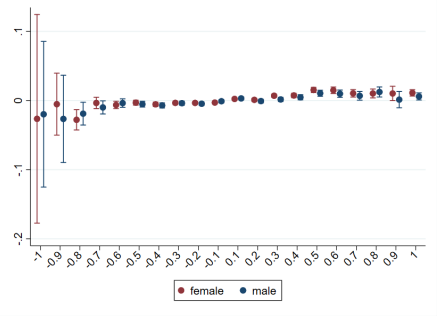
(c) C03 (Diuretics)



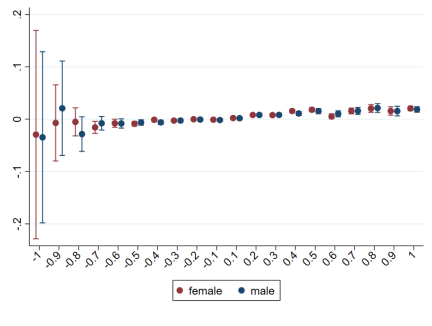
(d) C05 (Vasoprotectives)



(e) C07 (Beta blocking agents)



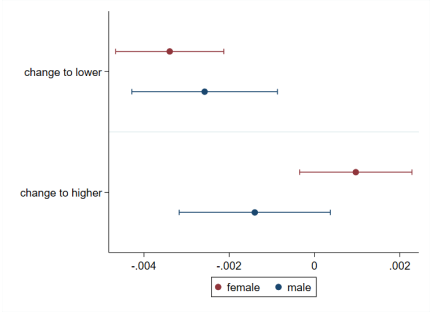
(f) C08 (Calcium channel blockers)



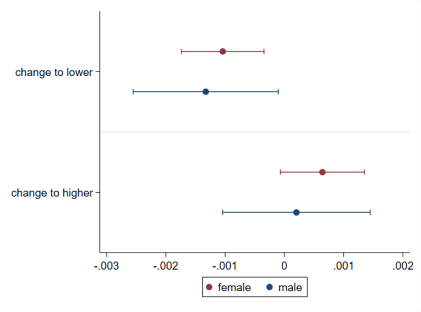
(g) C09 (agents acting on the renin-angiotensin system)

Notes: Figure shows the effect of the change in female usage rates after closure for the probability of using medication. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects and the change in age and gender composition between the closing and the new clinic. $ATC_{i,t} = changerate_ATC_{i,t} + ATC_{i,t-1} + change\ docgender_{i,t} + change\ docage_{i,t} + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$.

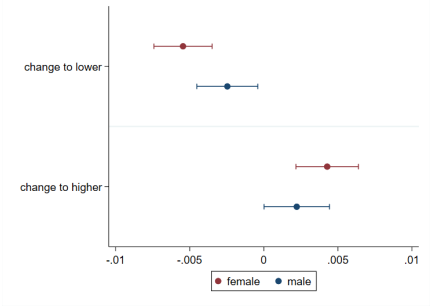
Figure A10: Effect of change in female usage rate on change in probability to use medication



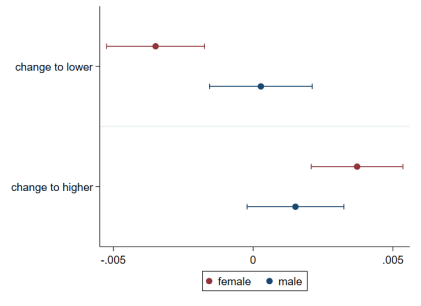
(a) C01 (Cardiac therapy)



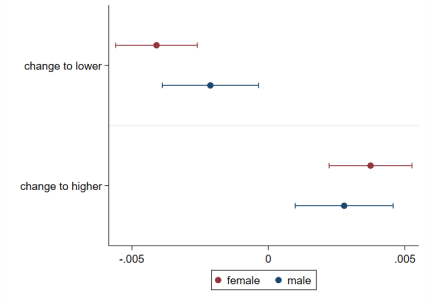
(b) C02 (Antihypertensives)



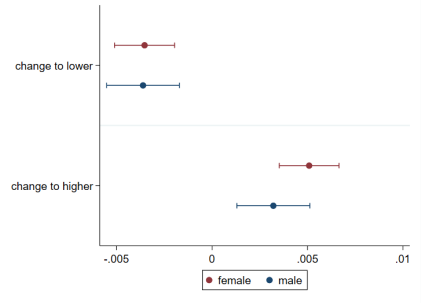
(c) C03 (Diuretics)



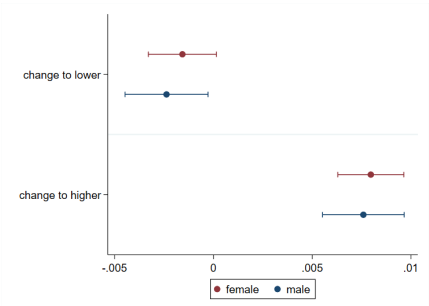
(d) C05 (Vasoprotectives)



(e) C07 (Beta blocking agents)



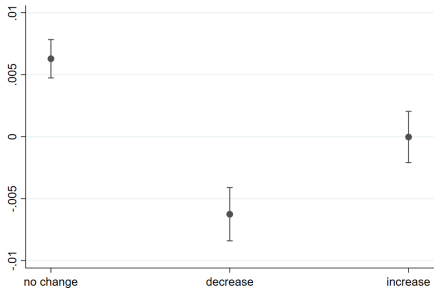
(f) C08 (Calcium channel blockers)



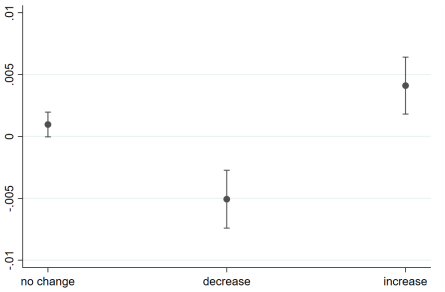
(g) C09 (agents acting on the renin-angiotensin system)

Notes: Figure shows the effect of the change in female usage rates after closure for the probability of using medication. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects as well as the change in age and gender composition between the closing and the new clinic. $ATC_{i,t} = negchange_ATC_{i,t} \times closure + poschange_ATC10_{i,t} \times closure + closure + ATC_{i,t-1} + change\ docgender_{i,t} + change\ docage_{i,t} + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$.

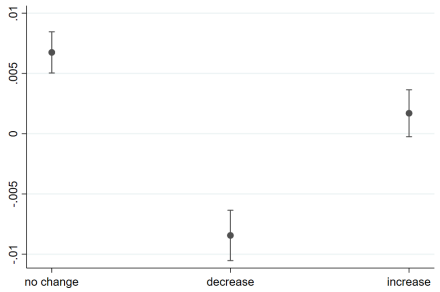
Figure A11: Selection into provider with different female usage rates



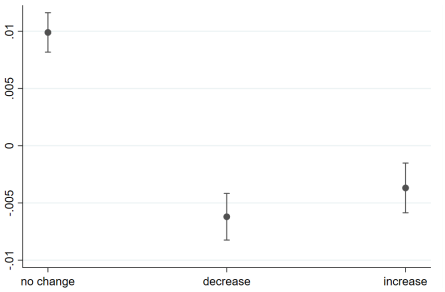
(a) C01 (Cardiac therapy)



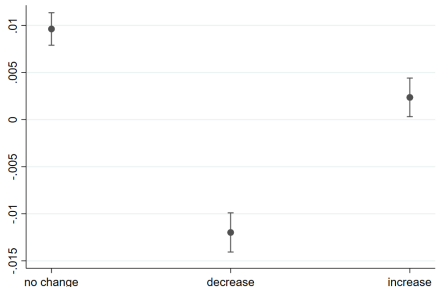
(b) C02 (Antihypertensives)



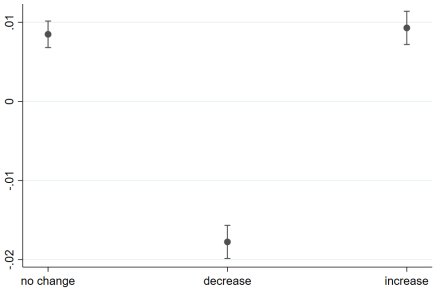
(c) C03 (Diuretics)



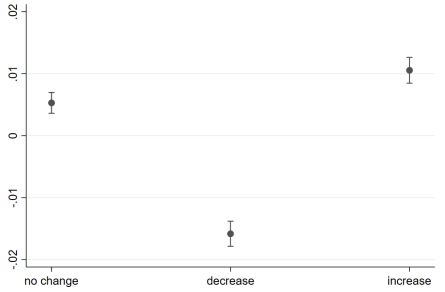
(d) C05 (Vasoprotectives)



(e) C07 (Beta blocking agents)



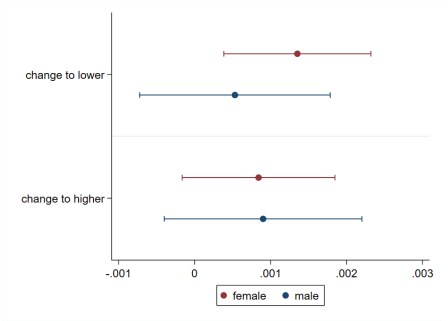
(f) C08 (Calcium channel blockers)



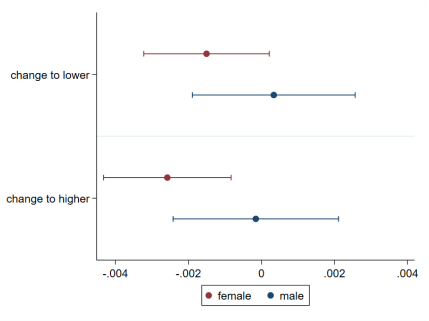
(g) C09 (agents acting on the renin-angiotensin system)

Notes: Figure shows relative risk ratios for the type of change in female usage rate individuals experience after a closure. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects as well as the change in age and gender composition between the closing and the new clinic. $changecat_ATC_{i,t} = female + change\ docgender_{i,t} + change\ docage_{i,t} + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$.

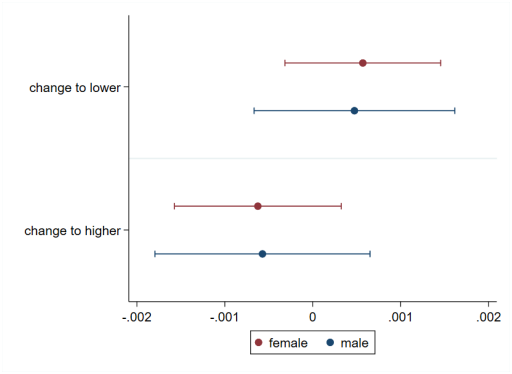
Figure A12: Effect of change in female usage rates on probability of death



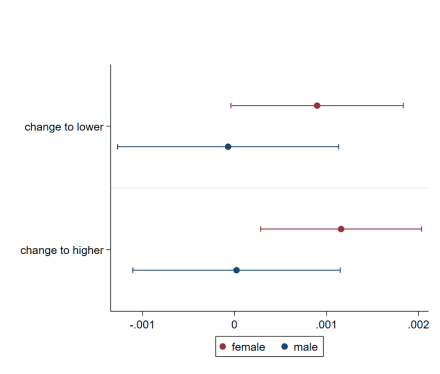
(a) C01 (Cardiac therapy)



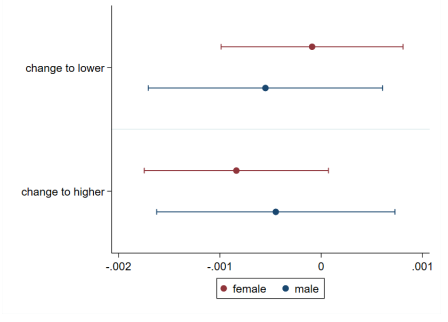
(b) C02 (Antihypertensives)



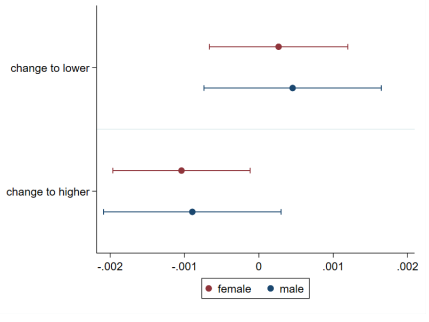
(c) C03 (Diuretics)



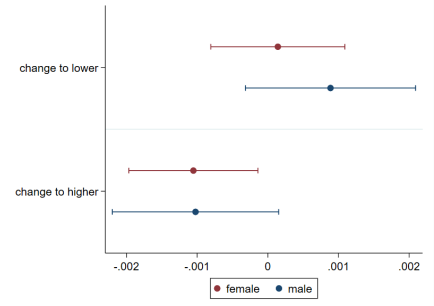
(d) C05 (Vasoprotectives)



(e) C07 (Beta blocking agents)



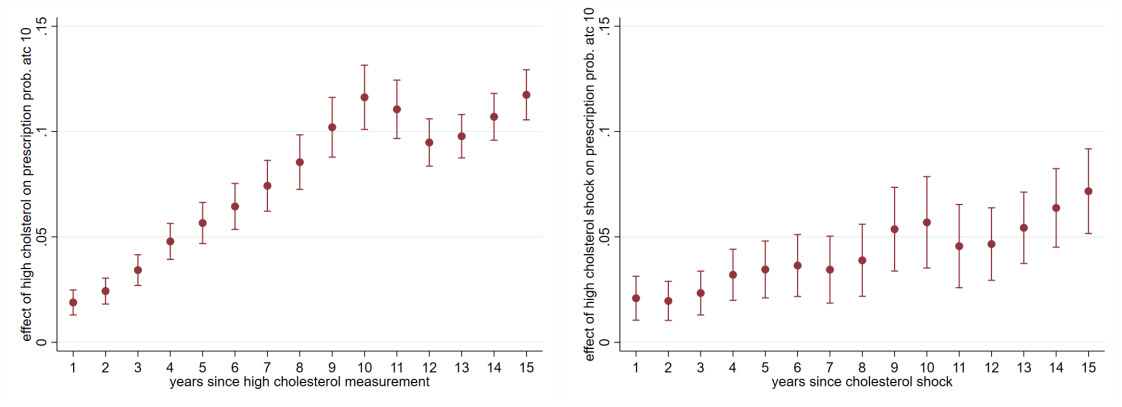
(f) C08 (Calcium channel blockers)



(g) C09 (agents acting on the renin-angiotensin system)

Notes: Figure shows the effect of the change in female usage rates after closure for the probability of death during the first five years after closure. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects and the change in age and gender composition between the closing and the new clinic. $death_{i,t} = negchange_ATC_{i,t} \times closure + poschange_ATC_{i,t} \times closure + closure + change\ docgender_{i,t} + change\ docage_{i,t} + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$.

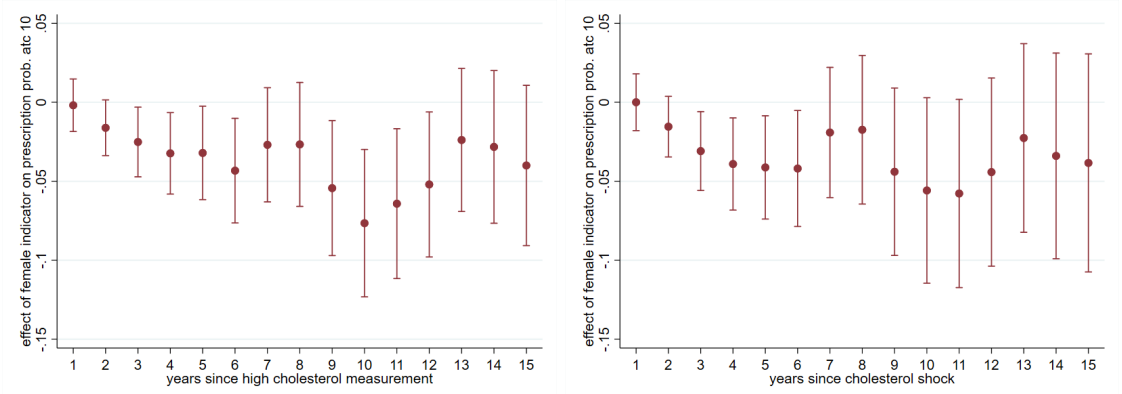
Figure A13: Usage of lipid-lowering drugs after (first) high cholesterol measurement



(a) high cholesterol measurement (b) first high cholesterol measurement

Notes: Figure shows the effect of receiving a (first) high cholesterol measurement on the probability of using lipid-lowering drugs. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects. (a) $ATC_{i,t} = high\ cholesterol_{i,t} + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$ (b) $ATC_{i,t} = high\ cholesterolshock_{i,t} + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$

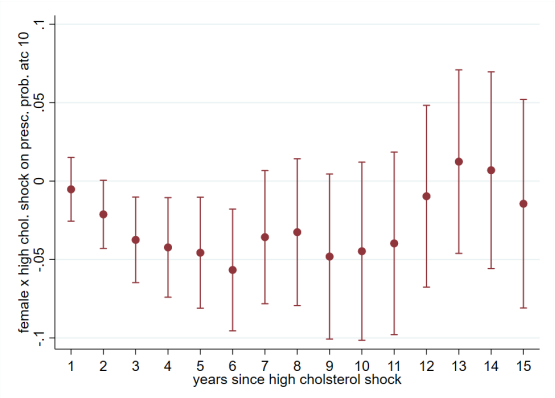
Figure A14: Gender differences in usage of lipid-lowering drugs after (first) high cholesterol measurement, CCHS thresholds



(a) high cholesterol measurement (b) first high cholesterol measurement

Notes: Figure shows the effect of $female$ on the usage of lipid-lowering drugs after receiving (a) a high cholesterol measurement and (b) a first high cholesterol measurement. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects. $ATC10_{i,t} = female_i + \lambda_{i,t} + \mu_t + \epsilon_{i,t}$

Figure A15: Effect of high cholesterol shock on prescription usage probability, CCHS tresholds



(a) first high cholesterol measurement

Notes: Figure shows the effect of $female \times high\ cholesterol\ shock$ on the usage of lipid-lowering drugs. Controls for age ($\lambda_{i,t}$) and year-fixed (μ_t) effects. $ATC10_{i,t} = female_i + high\ cholesterol\ shock + female \times high\ cholesterol\ shock\lambda_{i,t} + \mu_t + \epsilon_{i,t}$

Table A4: Labor market effects of AMI - first AMI

	(1) Working	(2) Disability Benefits
AMI_{t-1}	-0.112***	0.058***
	[0.002]	[0.001]
Female	-0.076***	0.015***
	[0.000]	[0.000]
$AMI_{t-1} \times female$	-0.084***	0.106***
	[0.003]	[0.002]
R-squared	0.162	0.054
Observations	121,709,196	121,627,573
Controlling for comorbidity		
AMI_{t-1}	-0.002	-0.036***
	[0.002]	[0.001]
Female	-0.076***	0.015***
	[0.000]	[0.000]
$AMI_{t-1} \times female$	-0.079***	0.102***
	[0.003]	[0.002]
R-squared	0.165	0.060
Observations	121,709,196	121,627,573

Standard errors clustered on the individual level in brackets; *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. Controls for age and year-fixed effects in all columns. $labor\ supply_{i,t} = heart_attack_{i,t-1} + female + heart_attack_{i,t-1} \times female + i.age + i.year + \epsilon_i$ Lower panel additionally controls for Charlson Comorbidity Index.

Table A5: Labor market effects of AMI - conditional on survival

	(1)	(2)
	Working	Disability Benefits
AMI_{t-1}	-0.124***	0.070***
	[0.001]	[0.001]
Female	-0.077***	0.015***
	[0.000]	[0.000]
$AMI_{t-1} \times female$	-0.074***	0.102***
	[0.003]	[0.002]
R-squared	0.160	0.051
Observations	119,453,841	119,453,841
first AMI		
AMI_{t-1}	-0.106***	0.054***
	[0.002]	[0.001]
Female	-0.077***	0.015***
	[0.000]	[0.000]
$AMI_{t-1} \times female$	-0.082***	0.103***
	[0.003]	[0.002]
R-squared	0.159	0.051
Observations	119,284,434	119,284,434

Standard errors clustered on the individual level in brackets; *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. Controls for age and year-fixed effects in all columns. $labor\ supply_{i,t} = heart_attack_{i,t-1} + female + heart_attack_{i,t-1} \times female + i.age + i.year + \epsilon_i$