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Abstract

Human antibiotic consumption is considered the main driver of antibiotic resistance. Reducing human antibiotic consumption without compromising health care quality poses one of the most important global health policy challenges. A crucial condition for designing effective policies is to identify who drives antibiotic treatment decisions, physicians or patient demand. We measure the causal effect of physician practice style on antibiotic intake and health outcomes exploiting variation in patient-physician relations due to physician exits in general practice in Denmark. We estimate that physician practice style accounts for 53 to 56 percent of between-clinic differences in all antibiotic consumption, and for 74 to 81 percent in the consumption of second-line antibiotic drugs. We find little evidence that low prescribing styles adversely affect health outcomes measured as preventable hospitalizations due to infections. Our findings suggest that policies to curb antibiotic resistance are most effective when aimed at improving physician decision-making, in particular when they target high prescribers. High prescribing practice styles are positively associated with physician age and negatively with staff size and the availability of diagnostic tools, suggesting that improvements in the quality of diagnostic information is an important path to improved decisions.

JEL-Codes: I110, J440, I120.

Keywords: antibiotic prescribing, practice styles, general practitioners.

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1 Introduction

The continued rise of bacteria resistant to antibiotic treatment is among the most pressing public health problems today. When bacteria are resistant against antibiotics, even common infections become associated with high health risks. The United Nations describe antibiotic resistance as "[...] a global crisis that risks reversing a century of progress in health", causing over 700,000 annual deaths already (IACG 2019). One of the main drivers of antibiotic resistance is the intake of antibiotic drugs in health care (e.g. Costelloe et al. 2010). Therefore, international organizations such as the World Health Organization or the European Commission have repeatedly called for prudent use of antibiotics in human medicine.¹ Many of the implemented policies aim at reducing antibiotic consumption in the patient population by targeting physicians and other health professionals.² The effectiveness and efficiency of such policies depend vitally on the extent to which the individual physician affects her patients' antibiotic intake as well as her quality of care in terms of infection-related health outcomes.

We measure the causal impact of physician practice style on antibiotic consumption and associated health outcomes by exploiting variation in patient-physician assignments due to physician exits from general practice clinics in Denmark.³ When individual physicians move or retire from their clinic or when a clinic closes due to their exit, their patients must switch to a new physician and are exposed to different practice styles. Our main identifying assumption requires that patients would be exposed to the same practice style throughout all periods were it not for the physician exit. This parallel trend assumption is backed up by our empirical setting as variation in the timing of physician exits is plausibly exogenous to antibiotic prescribing practice style and patient assignment to general practitioners is restrictive in Denmark. Moreover, we show that pre-trends are absent and provide supporting evidence against selective assignment from patients to physicians after physician exits.

Denmark provides a close to ideal setting for studying the role of practice style variation. First, Denmark's universal healthcare system ensures equal access and is based on a gatekeeper system where citizens access the primary healthcare system via assigned general practitioners. Moreover, standards in medical education are high and homogeneous.⁴ Finally, Denmark is at the forefront of antibiotic stewardship efforts and financial incentives to prescribe antibiotics are largely absent. Hence, antibiotic prescribing is comparatively low in Denmark. We find that even in such a setting antibiotic prescribing differs drastically across general practice clinics, with a mean of 0.71 (0.86)

¹See European Centre for Disease Prevention and Control (<https://ecdc.europa.eu/en/news-events/resistance-last-line-antibiotics-increasingly-established-europe>) and World Health Organization (<https://www.who.int/en/news-room/fact-sheets/detail/antibiotic-resistance>) (both accessed on 30 May 2021).

²For example, the World Health Organization offers an online course on antibiotic stewardship for practicing clinicians (<https://openwho.org/courses/AMR-competency>) (access: 13 June 2021). A number of randomized control trials in the medical literature evaluate behavioral interventions to affect physician prescribing decisions. Prominent examples include a large-scale letter-based peer comparison feedback intervention by Public Health England (Hallsworth et al. 2016), setting up poster-sized commitment letters in general practice clinics (Meeker et al. 2014), or requiring physician to enter justifications for their prescribing decisions in electronic records (Meeker et al. 2016).

³We refer to (general practice) clinics and practices interchangeably where unambiguous. We refer to prescribing practice styles, practice styles, or prescribing styles as all behaviors affecting patients' antibiotic treatment. These include preferences and indirect factors, such as diagnostic skill, that affect all patients similarly.

⁴For example, physicians begin their careers as interns distributed across the country based on a lottery system. Fadlon et al. (2020) find strong persistence in physician location based on a physician's initial draw.

prescriptions per patient per year and a standard deviation of 1.44 (1.64) in 2005 (2012).

Based on our identification strategy exploiting patient reassignments due to physician exits, we estimate the causal effect of practice style on prescribing using large-scale administrative data on antibiotics dispensed at all Danish pharmacies between 2005 and 2012. We find that physicians' practice styles determine 53 to 56 percent of the differences between clinics in terms of log antibiotic prescriptions, implying that harmonization of practice styles would reduce differences in log antibiotic consumption between general practice clinics in our sample by more than half. However, the effect varies by antibiotic subcategory and is largest, 74 to 81 percent, when we restrict our analysis to the second-line antibiotic classes macrolides, lincosamides and streptogramins, cephalosporins, and quinolones. This result is robust to a number of relaxations of our econometric assumptions. We also analyze which physician and clinic characteristics correlate with differences in prescribing styles. We find that higher prescribing style intensities are associated with physicians' age, staff size, and diagnostic availability. These correlations are primarily driven by prescribing styles for second-line antibiotics.

Finally, we study the effects of a change in antibiotic prescribing style on patient health by investigating hospitalizations due to infection-related ambulatory care sensitive conditions. We find that high prescribing practice styles do not result in lower hospitalization rates for patients. For penicillin prescribing, our results even indicate that being exposed to a more intense prescribing style increases a patient's rate of avoidable infection-related hospitalizations. This effect is primarily driven by an increase in hospitalizations for conditions commonly caused by viruses, specifically ear, nose, and throat infections, and pneumonia.

These results are important for the design of health care policy measures to curb the rise of antibiotic resistance. While the relationship between antibiotic prescribing and resistance is well established, documented correlations provide limited policy implications. Specifically, it is difficult to infer whether physicians prescribing with higher intensity unnecessarily amplify the issue of antibiotic resistance. Antibiotic treatments have sizable benefits when pathogens causing an infection are difficult to treat or when patients' overall preferences and health conditions demand higher antibiotic intake. In this case, a policy that incentivizes reductions in antibiotic prescribing could end up harming high-risk patients who require more intensive treatment. To assess the potential efficiency gains of antibiotic stewardship policies, it is necessary to determine to which degree physicians contribute to their patients' antibiotic consumption and their corresponding health outcomes. Our results suggest that incentivizing physicians to reduce antibiotic prescribing can be effective without leading to adverse health consequences for the patient population in general practice.

Our study expands on a growing literature attempting to measure the causes of the remarkable observed variation in health care provision. One strand of this literature, initiated by Finkelstein et al. (2016), uses moving patients to separate patient-driven from local factors of geographic variation in health care utilization in the United States. Variation from patient migration has been exploited to study geographic variation in exposure to prescription drugs in Denmark treating pain, anxiety, and depression (Laird and Nielsen 2016), prescription opioid abuse in the United States (Finkelstein

et al. 2018), health care utilization in the Netherlands (Moura et al. 2019), and ambulatory care in Germany (Salm and Wübker 2020).

However, using variation driven by patient migration as an empirical strategy comes with two important limitations. First, patient migration does not allow to identify physician effects because the mover’s residence, hence their social, economic, and health environment, also changes. For example, antibiotic prescriptions are often linked to community acquired infections, where some transmittable infections might be more demanding than others. As our goal is to study the extent of physician prescribing styles, we require a clean separation of physician effects from local factors. Second, although general practitioners frequently prescribe antibiotics, many patients do not receive an antibiotic prescription over long periods of time. The lack of observable variation in antibiotic consumption by moving patients results in a lack of statistical power to study practice style variation.

For the above reasons, we use individual physician migration as our main source of quasi-exogenous variation. Employing a similar design, Fadlon and Van Parys (2020) leverage exits of primary care physicians from Medicare in the United States to identify the effect of switching to a physician with high-utilization practice style on patients’ health care utilization. Using similar variation from physician migration or exits, Molitor (2018) examines how cardiologists’ practice styles evolve along the career path in heterogenous work environments in the United States. Simeonova et al. (2020) study adherence to medical treatments across primary care clinics in Denmark. Kristiansen and Sheng (2020) examine the health effects of physician-patient matching based on socioeconomic status. Chan (2021) identifies the causal effect of trainees on physician team decisions using a similar design based on frequent rotation of trainees across teams and random assignment of patients to physician teams. We identify agency in antibiotic consumption using the physician migration framework to provide evidence for the effective design of policies that can curb the growth of antibiotic resistance.

Our focus on physicians’ practice styles in antibiotic prescribing is related to Ribers and Ullrich (2020), who study heterogeneity in physicians’ preferences and their abilities to correctly diagnose bacterial infections as separate aspects of practice styles, using information from diagnostic tests for urinary tract infections. In contrast, we examine physicians’ general antibiotic prescribing practice in a broad context. Our analysis of physician practice styles contributes to a strand of health economics literature that studies within and between-region variation in physician treatment styles (e.g. Chandra and Staiger 2007; Epstein and Nicholson 2009; Currie et al. 2016). Cutler et al. (2019) measure physician incentives and patient preferences using strategic survey data. Consistent with our results, they find that patient preferences explain variation in Medicare expenditures to a much lesser extent than supply-side factors. Finally, our study complements economic research on antibiotic prescribing, a long-standing literature which has formed an important basis for public health policy design. Yet, the role of the individual physician is still not well understood. The problem of antibiotic prescribing under the threat of antibiotic resistance has been studied mostly theoretically in the economic literature (e.g. Laxminarayan et al. 2001). Empirical economic work on antibiotic prescribing has mainly focused on effects of the institutional environment or payment system (Currie et al. 2014; Bennett et al. 2015; Ellegård et al. 2018). Since the bulk of the policy

interventions to curb the rise of antibiotic resistance targets the individual physician, quantifying the physician’s role in antibiotic prescribing and mapping this role into health outcomes is crucial.

The remainder of the paper is organized as follows. Section 2 presents a model of prescribing and practice style to conceptualize the causal mechanism we aim to measure. Section 3 describes the institutional background and data and Section 4 provides descriptive evidence motivating the main analysis. Section 5 describes identification and estimation of the causal treatment effect and Section 6 presents estimation results. Section 7 provides further results on physician prescribing styles, including correlates with physician characteristics as well as the effects on health outcomes. Section 8 concludes.

2 Model of prescribing and practice style differences

Our main objective is to measure the importance of physician practice styles in determining antibiotic prescribing to patients. We model antibiotic prescribing using a framework that includes practice style as a time-invariant physician-specific component of prescription decisions. In this framework, we measure physician effects, defined by the difference in physician practice styles, relative to observed differences in antibiotic prescriptions. Some clinics are run by multiple general practitioners. For these, we measure effects over sets of general practitioners at a clinic. We refer to such a set of general practitioners as *physicians* where unambiguous.

We characterize antibiotic prescribing in a stylized model that follows Finkelstein et al. (2016). Patients obtain expected utility $u(y|\alpha_i, h_{it}) = \alpha_i y - \frac{1}{2}(y - h_{it})^2$ from consuming y amounts of antibiotic drugs, where α_i denotes individual time-invariant factors and h_{it} denotes patient health.⁵ Each patient i in each year t is matched to a set of physicians j . Physicians j make antibiotic prescribing decisions $y_{ijt}^* = \operatorname{argmax}_y \tilde{u}_j(y|\alpha_i, h_{it})$ such that their utility from treating patients is maximized. Physicians’ utility is given by $\tilde{u}_j(y|\alpha_i, h_{it}) = u(y|\alpha_i, h_{it}) + (\delta_j - c_{jt})y$, where δ_j denotes j ’s prescribing practice style and c_{jt} denotes time-varying clinic characteristics. Physicians’ utility thus captures differences between prescribing decisions that arise due to heterogeneity in the time-invariant prescribing practice styles and time-varying clinic characteristics that affect the cost of antibiotic prescribing.

Ensuing the maximization of physicians’ utility $\tilde{u}_j(y|\alpha_i, h_{it}) = \alpha_i y - \frac{1}{2}(y - h_{it})^2 + (\delta_j - c_{jt})y$, observed antibiotic prescribing by physicians j to their assigned patient i in year t can be written as:

$$y_{ijt} = \alpha_i + \delta_{j(i,t)} + x_{it}\beta + \epsilon_{it}, \tag{1}$$

where y_{ijt} denotes a measure of antibiotic prescribing, α_i denotes all time-invariant individual factors affecting patient i ’s antibiotic prescriptions, $\delta_{j(i,t)}$ denotes the antibiotic prescribing practice style of physicians j assigned to patient i in year t , and x_{it} is a vector of time-varying characteristics with β being the corresponding vector of coefficients, such that $x_{it}\beta$ subsumes patient and clinic

⁵The patient factor α_i absorbs patient-level drivers of antibiotic consumption that remain fixed over time including, for example, preferences and location-specific effects.

characteristics h_{it} and c_{jt} at the patient-year level. Finally, ϵ_{it} denotes an idiosyncratic error term.

Ignore for now the time-varying characteristics x_{it} . Differences in antibiotic prescribing y_{ijt} and $y_{kj't}$ to two patients i and k assigned to two different sets of physicians j and j' can be driven by two mechanisms. Antibiotic prescribing either differs because of differences in patient factors $\alpha_i \neq \alpha_k$ or because patients are exposed to different practice styles $\delta_j \neq \delta_{j'}$. To identify the degree to which differences in practice styles contribute to observable differences in prescribing, we require exogenous variation in patient-physician assignments. Our source of quasi-exogenous variation is based on physician exits from a clinic. After a physician exit, a patient i is either assigned to a different clinic, or she stays at the clinic but can no longer be treated by the exiting physician. In both cases, a physician exit changes the set of physicians that can treat patient i from j to j' . The change in physicians shifts the practice style patient i is exposed to from δ_j to $\delta_{j'}$.

Hence, patients exposed to a change in their assigned physicians from j to j' due to physician exits provide information about the difference in practice styles $\delta_{j'} - \delta_j$. To operationalize a measure, we define a treatment indicator that is one after a physician exit event has taken place and zero otherwise, $D_{it} = 1\{j(i, s) = j, j(i, t) = j', \text{ for some } s < t\}$, and rewrite Equation (1):

$$y_{ijt} = \alpha_i + \delta_j + (\delta_{j'} - \delta_j) \times D_{it} + x_{it}\beta + \epsilon_{it}.$$

With exogenous treatment D_{it} , the average treatment effect on the treated corresponding to the difference in practice styles $\delta_{j'} - \delta_j$ is identified. However, such treatment effects would not be directly comparable across patients because identification is in differences $\delta_{j'} - \delta_j$ rather than in levels $\delta_{j'}$ and δ_j . For example, $\delta_{j'} - \delta_j$ could be zero if treated patients are equally likely to switch to physicians with higher- and lower-prescribing practice styles than their pre-exit physicians.

To obtain a generalizable measure we scale the difference in practice styles between two sets of physicians assigned to a treated patient by the difference in their mean prescribing over untreated patients. The scaled difference in practice styles can be interpreted as the share of physician effects, as opposed to patient factors, in determining differences in mean antibiotic prescribing between physicians. We denote the scaled difference in practice styles between two sets of physicians j and j' as $\frac{\delta_{j'} - \delta_j}{y_{j'} - y_j}$ where $y_j = \mathbb{E}[y_{ijt}|D_{it} = 0]$ denotes mean prescribing by physicians j to patients unexposed to treatment.

For patients exposed to a physician exit, we denote the difference between physicians' mean prescribing as $\Delta_i = y_{j'} - y_j$. The measure Δ_i captures all differences in antibiotic prescribing between physicians j and j' , which may be due to different patient pools, physician effects, or time-varying control variables. For never-treated patients, $\Delta_i = 0$. We can now rewrite Equation (1) such that:

$$y_{it} = \alpha_i + \delta_{j_0} + \theta \times D_{it} \times \Delta_i + x_{it}\beta + \epsilon_{it}, \tag{2}$$

where the coefficient θ measures the importance of physician practice styles. Specifically, this coefficient captures the share of differences in mean prescribing that can be attributed to differences in practice styles. The coefficient is zero if variation in antibiotic prescribing is determined exclusively

by differences in physicians’ patient pools.

To conclude, the measure θ answers the question: By what share would the difference in antibiotic consumption between patients assigned to treated clinics j and patients assigned to never-treated destination clinics j' be reduced if we could harmonize physicians’ prescribing practice styles? Thus, the measure quantifies the extent of noise in antibiotic prescribing induced by heterogeneity in physician prescribing styles. After presenting the main results, we will provide further causal evidence that this noise does not reflect quality differences measured in patients’ health outcomes.

3 Institutional background and data

Denmark offers a close to ideal setting to quantify the role of practice style variation for antibiotic prescribing, which is often confounded by institutional factors. Given notable homogeneity in access to Danish health care, medical education, and centrally designed public health efforts, we would expect few factors driving variation in health care provision. Our analysis is based on linked population-level administrative data, which registers nation-wide claims filed in primary health care, individual-level socio-economic information, and all purchases of prescribed antibiotic drugs in Denmark. Information can be linked through patients’ social security numbers and their general practitioners’ clinic license numbers.

3.1 Institutional setting

We study antibiotic prescribing practice styles using administrative data covering the full population of Denmark. Denmark has a tax-funded public health insurance system that fully covers all visits and services in general practice. General practitioners act as gatekeepers; visits to most specialists as well as scheduled hospital procedures require the referral of a general practitioner in order to be covered by insurance. General practitioners are self-employed and work under nationally regulated contracts for the public funder. In order to receive reimbursement claims, each clinic has to acquire a unique license number (ydernummer). During our observation period of 2005-2012, a total of approximately 3,280 general practice clinics file claims.

Patients are assigned to a fixed general practice clinic by a list system.⁶ Switching the general practitioner is only possible if patients pay a small fee of 150 DKK (about 20 USD), and patients have to choose a general practitioner located within 15 km of their residence. Clinics cannot turn away patients selectively. However, they may close for the intake of new patients after reaching 1,600 listed patients per physician. Patients rarely switch away from their default general practitioners, except when moving, also because capacity constrained clinics limit the actual choice set of clinics.⁷

Patients require a physician prescription before they are dispensed antibiotic drugs. Prescription drugs are purchased in a pharmacy and typically associated with a small copayment. Importantly, Danish physicians are not remunerated for prescriptions. The type and number of antibiotic

⁶This section closely follows the description of regulations in Danish general practice in Simonsen et al. (2019).

⁷Kristiansen and Sheng (2020) documents that the number of patients per physician in general practice was close to or larger than the capacity limit of 1,600 patients in the majority of municipalities in 2010.

prescriptions dispensed is therefore not driven by physicians' financial incentives. In general, Denmark is a country with low antibiotic prescribing rates and conservative prescribing practices (Coenen et al. 2007).

A physician might leave a clinic over time, for example when she retires or relocates. If a physician leaves a clinic, the clinic may close or it can continue operation if at least one other physician continues to work at the clinic. When a clinic closes, all of its former patients are either assigned to new physicians who acquire the patient list (and often the physical practice) or patients are redistributed randomly to existing clinics nearby. If a clinic continues to exist after a physician leaves, the remaining physicians can reduce the number of patients assigned to their clinic by first off-listing all of their patients and, subsequently, requiring patients to re-apply for the clinic. Patients are then re-assigned to the clinic on a first-come, first-served basis. The local government is responsible to ensure access to at least two nearby clinics for all patients in a region.

3.2 Sample construction

For our main analysis, we construct a sample of patients matched to clinics for the years 2005 to 2012 in two steps. First, we identify general practice clinics and keep those in which physician exits occurred. Second, we match patients to their main general practice clinic in order to obtain a yearly panel of patient-level observations. For all patient-clinic matches, we identify patients exposed to a physician exit. Finally, we construct our main explanatory variable, the difference in average antibiotic prescribing induced by a physician exit.

In the first step, we consider all general practitioners in the Danish registry of clinics between 2005 and 2012. This registry links physicians' social security number to their clinics' license number and registers the in- and outflow of physicians to clinics. For reasons of data minimization the Danish Health Data Protection Authority provided only a non-selective portion of the license number registry. Out of all 3,280 clinics who file claims, we consider the 1,605 clinics for which registry records are complete. Next, we supplement information on the outflow of physicians by adding data from the national death registry, the employment registries, and the health claims registries. We assume a physician exited a clinic whenever we can link the personal identifier to death or retirement, or when the physician joins a new clinic after she reported her position at the old clinic. We identify clinic closures by the last year a clinic files claims. Whenever we find records of multiple exiting events, such as deaths, retirements, or clinic closures, we only consider the event that occurs first.

We impose two sample restrictions to cleanly measure the econometric treatment. First, we drop clinics if a physician exit occurs in the first year of our sample period, keeping 1,397 clinics, and keep only clinics in our sample period, 2005 to 2012, leaving 1,196 clinics. Second, we only consider long-term changes in composition or clinic closures, dropping clinics with multiple physician exits if these changes occur in different years as well as clinics with physician entries if they do not coincide with an exit. The resulting sample contains 980 clinics. We thus simplify the analysis because clinics with multiple changes in composition do not have clearly defined pre- and post-change periods. We refer to clinics exposed to physician exits in exactly one year as *treated clinic* and the physician exit

as *treatment*. We refer to clinics never exposed to treatment as *never-treated clinics*. We refer to never-treated clinics with patient intakes from treated clinics as *destination clinics*.

Next, we match patients to their general practice clinic. We use weekly claims data and find the modal clinic for each patient in every year.⁸ We consider all patient-year observations assigned to any of the 980 general practice clinics we keep for the analysis out of all 3,280 clinics, which amounts to 25.33% of all patient-year observations. We include patients only when they switch their modal clinic at most once, dropping 15.68% of patient-years, and obtain a panel of patients assigned to a unique general practitioner in every year.

For patients who switch exactly once, we ensure that patients are exposed to at most two practice styles, defined by the pre- and the post-exit period, as follows. We exclude all patients assigned to more than one treated clinic, dropping 0.31% of observations. For patients who switch their general practitioner without being exposed to treatment, we keep observations from their modal clinic. We exclude patients for whom the mode cannot be recovered, dropping 1.64%. For patients exposed to treatment and switching from a treated clinic, we keep all observations if the switch coincides with treatment; otherwise, we keep only observations associated with the treated clinic, dropping 0.05%. For patients exposed to treatment and switching to a treated clinic, we keep only observations at the treated clinic, dropping 0.05%. We exclude a patient’s first observed year at a treated clinic if that year is the treatment year, dropping 0.15%.

We refer to patients who are at any point in time exposed to a physician exit as *treated patients* and the complementary set of patients as *never-treated patients*.⁹

Table 1: Numbers of observations

	Clinics	Patients assigned to clinics ^a	Observations
Never-treated	556	1,100,593	6,260,514
Treated	242	330,926	1,536,253
Clinic closure	211	222,517	892,677
No clinic closure	31	108,409	643,576
Total	798	1,373,109	7,796,767

^a Because patients exposed to physician exit can be observed at two clinics, the total number of patients does not equal the sum of patients assigned to never-treated and treated clinics.

Our final sample excludes clinics with insufficient prescribing to patients unexposed to treatment (fewer than 100 patient-year observations), dropping 17 clinics, and singleton observations, dropping 0.26% of the remaining patient-year observations. The final sample, described in Table 1, contains 7,796,767 patient-year observations matching 1,373,109 patients to 798 general practice clinics. Of

⁸We consider unique claim weeks by aggregating all claims filed during the same week by the same clinic. Among patients with multiple modes, we assign a patient to the modal clinic that files the most antibiotic prescriptions or, in case of a tie, the earliest claim in a given year for this patient. Some patients switch back and forth in their modal clinic over the years. In these cases, we impute their matched clinic to be the same as the one they switched back and forth (affects 1.25% of all patient-years).

⁹Never-treated patients can be assigned to a treated clinic, for example if they are assigned to that clinic strictly after the physician exit has already occurred.

these, 1,536,253 patient-year observations matched to 242 practices are exposed to physician exits. The majority of these exits occur due to clinic closures. For a smaller number of clinics, a physician exiting a clinic is replaced by a new physician.

3.3 Variable definitions

Outcomes. Our main outcome variable is the number of antibiotic prescriptions in general practice dispensed to each patient per year at Danish pharmacies. We define an antibiotic prescription as all packages dispensed on the same day to a given patient for drugs belonging to the same level 3 Anatomic Therapeutic Chemical (ATC) class in the therapeutic subgroup of antibacterials for systemic use J01. As an alternative measure, we define the total Daily Defined Dose (DDD) over antibiotic prescriptions from general practitioners for a patient within a year. A DDD expresses the average dose per day for a drug used in adults under the drug’s main indication as defined by the WHO (Coenen et al. 2007).¹⁰

We carry out our analyses for the sum over all systemic antibiotic prescriptions (ATC J01) and separately for subcategories at the ATC level 3. These subcategories are penicillin prescriptions, second-line antibiotic prescriptions, and prescriptions from other classes. Penicillins (J01 C) are the most commonly prescribed class. We consider all macrolides, lincosamides, and streptogramins (J01 F), cephalosporins (J01 D), and quinolones (J01 M) as second-line antibiotic classes. Consumption of these antibiotic classes is used as quality indicator by the European Surveillance of Antimicrobial Consumption (ESAC) as their prescribing can suggest ‘poor practice’ when combined with other evidence Coenen et al. (2007)¹¹. We aggregate the remaining classes into one subcategory.

Table 2 shows descriptive statistics by antibiotic class defined by ATC level 3. Penicillins (J01 C) make up the largest share of prescribed antibiotic drugs, followed by macrolides, lincosamides and streptogramins (J01 F), and sulfonamides and trimethoprim (J01 E). The average DDD per prescription varies strongly between antibiotic classes. For some antibiotic classes, the share of total DDD differs markedly from the share of total prescriptions. The small shares of non-zero observations indicate that the distribution of antibiotic consumption is skewed. Because both measures are non-negative and bunched at zero, we make a transformation by taking the natural logarithm of one plus the relevant outcome.

Physician exits. We define a physician’s exit from a clinic as the treatment and the period following the exiting event as post-treatment period. An exit may or may not lead to the clinic’s closure. In any case, a physician’s exit affects the patient-physician relationship by changing the set

¹⁰Our main results are based on the number of prescriptions. We show results for DDD in the Appendix.

¹¹The distinction between first- and second-line antibiotic treatments depends on the disease indication. We refer to macrolides, lincosamides, streptogramins (J01 F), cephalosporins (J01 D), and quinolones (J01 M) collectively as second-line drugs as they are labeled as such in the ESAC framework (Coenen et al. 2007). Macrolides, cephalosporins, and quinolones are also often characterized as broad-spectrum antibiotic drugs, with the exception of erythromycin (J01 FA01) (Shapiro et al. 2014; ECDC, EFSA Panel on Biological Hazards (BIOHAZ) and EMA Committee for Medicinal Products for Veterinary Use (CVMP) 2017). Broad-spectrum antibiotic drugs are active against a broad range of bacterial groups and, hence, more likely to cause multi-drug resistances. Physicians are in general advised to avoid broad-spectrum antibiotics.

Table 2: Descriptive statistics for prescriptions and Daily Defined Dose (DDD)

A: Prescription level counts by antibiotic class					
ATC 3	Pharmacological subgroup	Share of total prescriptions	Share of total DDD	Average DDD per prescription	Share of non-zero observations
J01 C	Beta-lactam antibacterials, penicillins	60.09%	56.06%	9.01	19.21%
J01 F	Macrolides, lincosamides, streptogramins	18.27%	15.67%	8.28	6.53%
J01 E	Sulfonamides and trimethoprim	9.97%	8.08%	7.83	2.92%
J01 A	Tetracyclines	4.34%	7.48%	21.23	1.03%
J01 M	Quinolone antibacterials	3.87%	2.90%	7.22	1.41%
J01 X	Other antibacterials	3.40%	9.72%	21.66	0.87%
J01 D	Other beta-lactam antibacterials	0.07%	0.09%	12.77	0.02%
All J01	Antibacterials for systemic use	100%	100%	9.66	26.52%

B: Patient-year level counts by outcome					
		Log prescriptions		Log DDD	
		Mean	St. dev.	Mean	St. dev.
J01 All	All antibiotic prescriptions	0.29	(0.484)	0.77	(1.234)
J01 C	Penicillins	0.20	(0.393)	0.57	(1.075)
J01 F, D, M	Second-line	0.07	(0.24)	0.20	(0.659)
J01 Others	Other antibiotics	0.05	(0.222)	0.12	(0.565)

Notes: Prescriptions of J01 G (Aminoglycoside antibacterials) are too few and omitted to ensure anonymity.

of physicians that can treat a patient. For clinics that do not close after an exit, we regard the pre- and the post-treatment clinic as two separate sets of physicians.

Measuring differences in mean prescribing. To construct the measure of differences in mean prescribing Δ_i defined in equation (2), we estimate mean prescribing by the average amount of antibiotic drugs that a given set of physicians prescribes to patients not or not yet exposed to treatment. We only include patients who are never or not yet exposed to treatment for these computations in order to keep the patient pools between pre- and post-treatment sets of physicians separate.¹² The difference in mean prescribing is nonzero for all patients who either switch to a different clinic after a physician exits the original clinic, or who continue to be assigned to a clinic in which the set of physicians changes.

This definition differs from Fadlon and Van Parys (2020) who use the difference in unconditional mean prescribing as scaling factor. However, scaling by the difference in unconditional mean prescribing implies that physician effects between physicians j' and j depend on the share of treated patients relative to the share of untreated patients. A larger share of treated patients relative to untreated patients then results in a larger scale factor.¹³ The advantage of scaling in the differences

¹²For example, if a clinic is exposed to physician exit, we compute an estimate of its pre-exit mean prescribing using all observations until the exiting event, and we compute an estimate of its post-exit mean prescribing based only on observations from never-treated patients who join the clinic after the exit has occurred. To reduce noise in the average prescribing estimates, we drop physicians with fewer than 100 observations from never- or not yet treated patients as well as physicians with zero average prescribing. In the main analysis, we account for estimation error in average prescribing by a parametric bootstrap procedure.

¹³To see this in a simplified setting, let all treated patients change from physicians j to j' . Denote average patient

in mean prescribing *conditional on no exposure to treatment* is that scaling is not affected by overlaps in patient pools assigned to physicians j and j' . As a result, θ does not depend on the proportions of patients exposed to treatment.

4 Descriptive evidence

We first present descriptive evidence on the considerable variation in antibiotic prescribing across general practice clinics in Denmark. To support that our sample is not systematically selected in terms of treatment, we show there are no observable differences in the summary statistics between patients assigned to general practice clinics with a physician exit and patients not assigned to such clinics. We document and discuss some observable differences between treated and never-treated clinics. Descriptive evidence shows that patient-physician reassignments due to physician exits cause a visible shift in treated patients' antibiotic prescriptions, motivating the ensuing causal analysis.

4.1 Variation in antibiotic prescribing

Figure 1 shows the distribution of clinic-level average number of antibiotic prescriptions per patient and year in 2005 and 2012, documenting persistent variation in antibiotic prescribing across general practice clinics in Denmark. The means of these two distributions are 0.71 and 0.86 with standard deviations of 1.44 and 1.64, reflecting considerable variation even for a low-prescribing country such as Denmark (Coenen et al. 2007).¹⁴

4.2 Sample summary statistics

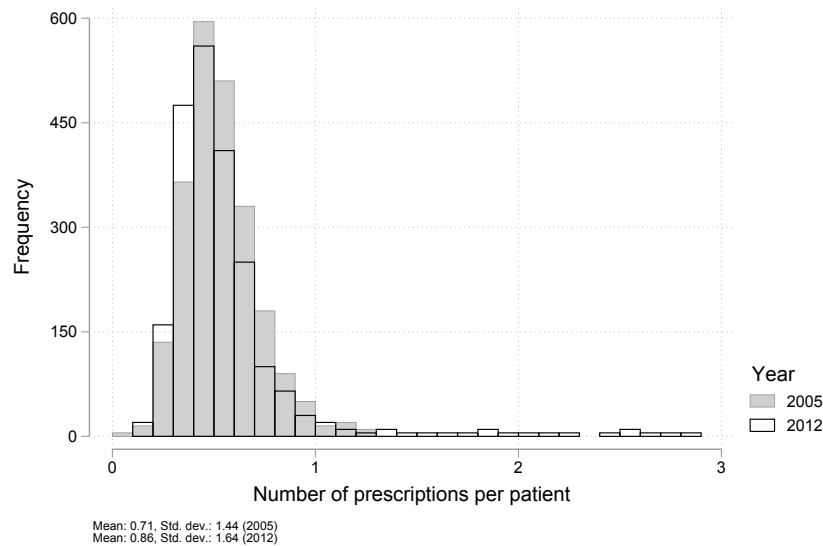
Table 3 shows descriptive statistics for treated and never-treated patients. Panel A shows averages for the main outcome variable, antibiotic prescribing. Panel B shows averages in basic demographics and health characteristics, and Panel C shows averages in family and education characteristics. Panel D shows average rates of hospitalizations for infection-related ambulatory-care sensitive conditions. Treated patients are on average older than patients never exposed to physician exit. They are also

effects in the patient pool assigned to physician j prior to treatment exposure by $\alpha^j = \mathbb{E}[\alpha_i | j(i) = j, D_{it} = 0]$. For simplicity, ignore time-varying patient characteristics x_{it} such that $\mathbb{E}[y_{ijt}] = \alpha^j + \delta_j$. Assume that the patient pools treated by j and j' prior to the physician exit differ arbitrarily, $\alpha^j \neq \alpha^{j'}$. Unconditional mean prescribing can be written as a weighted sum of mean prescribing to patients unexposed to physician exit ($D_{it} = 0$) and mean prescribing to patients already exposed to physician exit ($D_{it} = 1$). By construction, patients assigned to j have not yet been exposed to treatment. The difference in unconditional mean prescribing is $\tilde{\Delta}_i = w_D \mathbb{E}[y_{ij't}|D_{it} = 1] + (1 - w_D)(\mathbb{E}[y_{ij't}|D_{it} = 0] - \mathbb{E}[y_{ijt}|D_{it} = 0])$, where $w_D \in [0, 1]$ denotes the proportion of patients of j' who changed from j to j' .

Because patient effects are fixed over time, only physician fixed effects change once patients are exposed to physician exit. Mean prescribing to the pool of treated patients is $\mathbb{E}[y_{ijt}|D_{it} = 0] = \alpha^j + \delta_j$ prior to the exiting event, and it is $\mathbb{E}[y_{ij't}|D_{it} = 1] = \alpha^j + \delta_{j'}$ after the exiting event has taken place. Mean prescribing to the never-treated patient pool is $\mathbb{E}[y_{ij't}|D_{it} = 0] = \alpha^{j'} + \delta_{j'}$. The difference in unconditional mean prescribing can now be written as follows: $\tilde{\Delta}_i = (\delta_{j'} - \delta_j) + (1 - w_D)(\alpha^{j'} - \alpha^j)$. Scaling physician effects $\delta_{j'} - \delta_j$ by $\tilde{\Delta}_i$ implies that physician effects are weighted more the larger the proportion of treated patients w_D for a given difference in patient pools $\alpha^{j'} - \alpha^j$.

¹⁴Figure 7(a) in Appendix A.1 shows the distribution of antibiotic prescribing over clinics for all years in the sample. Figure 7(b) in Appendix A.1 shows the same patterns when prescribing is measured in DDD.

Figure 1: Distribution of the average level number of antibiotic prescriptions (ATC J01) per patient over general practice clinics



Notes: Average antibiotic prescribing per patient and year at the clinic-level. Bunched in groups of five clinics to ensure the required data anonymization. The upper five percentiles are omitted.

less likely to contact an emergency department or doctor. Other characteristics are largely similar between both groups.

Table 4 shows descriptive statistics for clinics with and without physician exits. Panel A shows averages in antibiotic prescribing per patient. Panel B shows averages in observable physician characteristics, and Panel C shows averages for clinic-level characteristics. Treated clinics are older on average, have a smaller share of female physicians, and a smaller average household size than never-treated clinics.¹⁵ They are also smaller in terms of the number of interns and the number of patients per physicians. Notably, differences in antibiotic prescribing between treated and untreated clinics and patients are small.¹⁶

¹⁵One concern could be that we simply observe patient reassignments from old to young physicians. Although Figure 8 in Appendix A.2 shows the mean age difference between pre- and post-exit physicians is slightly below zero, we observe much variation around this mean. We do not assert that antibiotic prescribing practice styles are independent of physician age or, possibly, other physician characteristics. Instead, we view practice styles as subsuming age effects and inspect the role of physician age among other physician characteristics in our analysis of practice style correlates.

¹⁶Table 7 in Appendix A.3 shows summary statistics at the patient-year level for the sample matched to clinics excluded from our analysis. Antibiotic prescribing does not differ substantially between out-of-sample and in-sample clinics. Table 8 in Appendix A.3 shows average characteristics for out-of-sample clinics. Given that we exclude clinics with multiple long-term staff changes, out-of-sample clinics have more physicians and interns than in-sample clinics.

Table 3: Averages for treatment and comparison group patients

	Never-exposed to physician exit	Exposed to physician exit
A: Antibiotic prescribing^a		
Total J01 (all antibiotics)	0.29	0.28
J01 C (penicillins)	0.20	0.20
J01 F, D or M (second-line)	0.07	0.07
Other J01 (other antibiotics)	0.05	0.05
B: Basic demographics and health		
Age	42.89	44.45
Female	0.54	0.55
Pregnant	0.02	0.02
Household size	2.61	2.58
Any visit to an emergency department	0.15	0.15
Any call to an emergency doctor	0.19	0.17
C: Family background and education		
Married couple	0.56	0.56
Cohabiting couple with children	0.07	0.06
Cohabiting couple without children	0.07	0.06
Single	0.32	0.31
First generation migrant (nordic)	0.01	0.01
First generation migrant (other country)	0.07	0.07
Second generation migrant	0.03	0.03
Missing education	0.21	0.18
School grade 7 to 10	0.26	0.28
High school or vocational training	0.32	0.33
Short higher education	0.03	0.03
Medium higher education	0.12	0.12
Long higher education	0.05	0.05
Phd education	0.003	0.003
No education	0.001	0.001
D: Any hospitalization^b		
All infection-related conditions	0.006	0.005
Cellulitis	0.001	0.001
Ear, nose and throat infections	0.001	0.001
Perforated or bleeding ulcer	0.001	0.001
Urinary tract infection	0.002	0.001
Pneumonia	0.001	0.001
Total observations (patient-years)	6,225,636	1,572,131

^a Measured by $\log(1 + \text{number of antibiotic prescriptions})$.

^b Hospitalizations for acute ambulatory care-sensitive conditions (ACSC) commonly caused by bacterial and non-bacterial infections (see Appendix D for a complete list of ICD-10 codes). Referrals from general practitioners and delayed internal hospital referrals are excluded.

Table 4: Averages for treatment and comparison group clinics

	Without physician exit	With physician exit
A: Average antibiotic prescribing per patient^a		
Total J01 (all antibiotics)	0.29	0.28
J01 C (penicillins)	0.20	0.19
J01 F, D or M (second-line)	0.07	0.07
Other J01 (other antibiotics)	0.05	0.05
B: Average physician characteristics		
Age	55.41	59.28
Household size	2.63	2.21
Female	0.35	0.28
Single	0.17	0.17
First generation migrant (nordic)	0.00	0.01
First generation migrant (other country)	0.03	0.03
Second generation migrant	0.01	0.01
Phd education	0.01	0.01
C: Size and equipment		
Number of physicians	1.42	1.49
Number of interns	0.23	0.15
Number of patients per physician	1987.81	1748.62
Diagnostic culture available	1.00	0.99
Diagnostic microscopy available	0.76	0.74
Total observations (clinic-years)	4,023	1,254

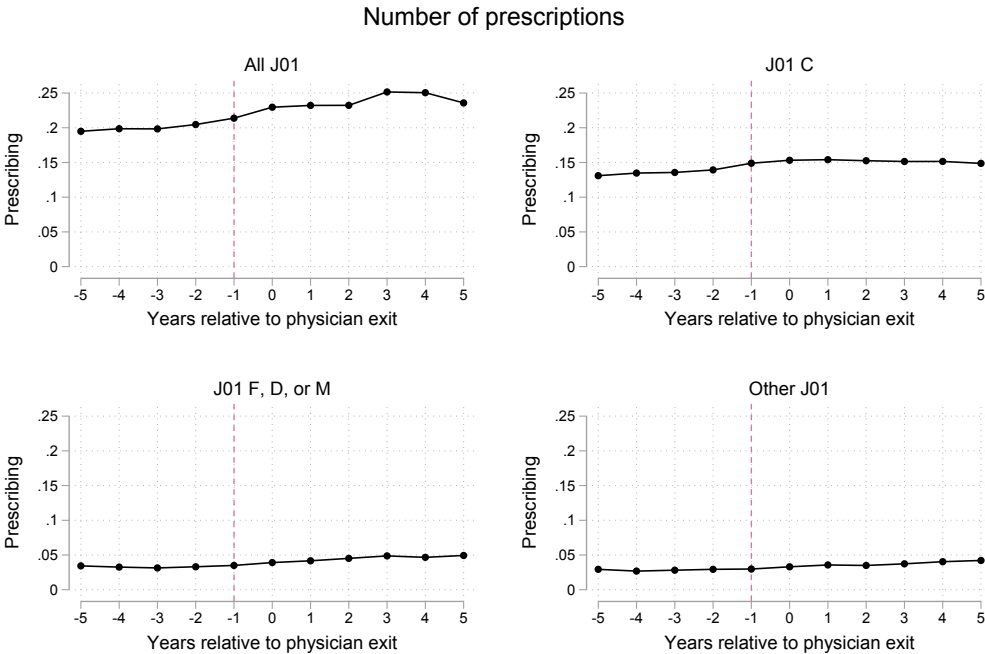
^a Measured by $\log(1 + \text{number of antibiotic prescriptions})$.

4.3 Shifts in prescribing

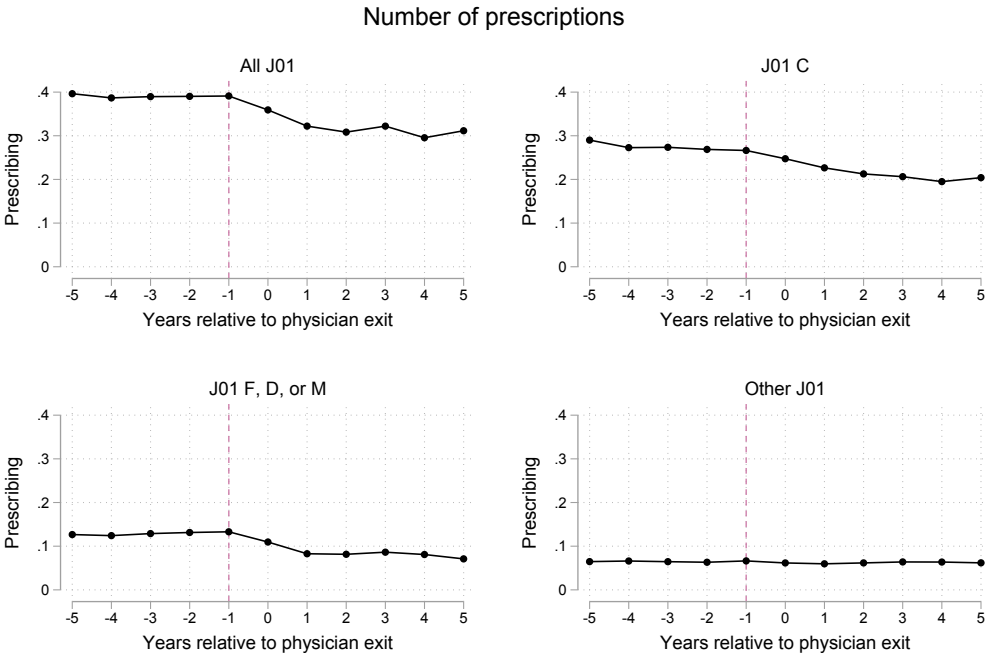
Figure 2 depicts average per-patient antibiotic prescribing to treated patients over years relative to treatment, the physician exit. Figure 2a shows how antibiotic prescribing evolves for treated patients who were assigned, pre-treatment, to clinics with average prescribing in the lower quartile. Patients who were assigned, pre-treatment, to relatively low prescribing clinics tend to consume more antibiotics post-treatment. Conversely, Figure 2b shows that antibiotic prescribing to treated patients assigned, pre-treatment, to high prescribing clinics, with average prescribing in the upper quartile, tend to consume fewer antibiotics post-treatment. In our identification strategy, changes in average prescribing at treatment onset will be attributed to changes in practice styles. The figures show a reversal to the mean in almost all prescribing outcomes. This is a first indication that antibiotic prescribing to individual patients is, to a certain degree, driven by practice styles.

Figure 2: Average antibiotic prescribing per patient assigned to treated clinic

(a) Clinics in the lower quartile of pre-exit antibiotic prescribing



(b) Clinics in the upper quartile of pre-exit antibiotic prescribing



Notes: These figures show average antibiotic prescribing by years relative to the physician exit. Relative year -1 is the last pre-exit period, relative year 0 is a transitional period, and relative year 1 is the first post-exit period. Antibiotic prescribing is measured by $\log(1 + \text{number of prescriptions})$.

5 Empirical Strategy

To measure the causal treatment effect defined in the model introduced in Section 2, we first lay out the assumptions required for identification. We then show how we estimate the parameters of interest.

5.1 Identification

We discuss the identification of physician effects in our model of prescribing in a potential outcomes framework. Let $y_{it}(1)$ denote potential antibiotic prescribing to patient i in year t when an exiting event has occurred. Let $y_{it}(0)$ denote potential prescribing when an exiting event has not occurred. Potential outcomes are determined in our model of prescribing as follows:

$$\begin{aligned} y_{it}(1) &= \alpha_i + \delta_{j'} + x_{it}\beta + \epsilon_{it} \\ y_{it}(0) &= \alpha_i + \delta_j + x_{it}\beta + \epsilon_{it}. \end{aligned}$$

Observed prescribing can be written in terms of potential outcomes:

$$y_{it} = y_{it}(0) + [y_{it}(1) - y_{it}(0)] \times D_{it},$$

where D_{it} is an indicator that is one if patient i in year t has been exposed to physician exit.

The difference in potential outcomes, $y_{it}(1) - y_{it}(0)$, equals the difference in physician practice styles $\delta_{j'} - \delta_j$. We scale $\delta_{j'} - \delta_j$ using the difference in physicians' mean prescribing $\Delta_i = y_{j'} - y_j$ in order to obtain θ , the standardized measure of physician effects. Hence, θ defines an average scaled treatment effect on the treated:

$$\theta = \mathbb{E} \left[\frac{y_{it}(1) - y_{it}(0)}{\Delta_i} \mid D_{it} = 1 \right] \quad (3)$$

Identification of θ is based on a staggered difference-in-differences design, where treatment onset - the year of a physician exit - can vary between treated patients. We discuss identification in such a design as laid out in Sun and Abraham (2020). We refer to all patients with treatment onset in the same calendar year as a cohort, and denote by variable E_i the cohort that patient i is part of, with realizations $e \in \{2006, \dots, 2012, \infty\}$. The never-treated group of patients forms its own cohort characterized by $e = \infty$. To give θ a causal interpretation based on our design, the following set of assumptions is required.

Assumption 1: The potential outcome of no exposure to exit follows parallel trends for all cohorts and time periods, $\mathbb{E}[y_{it'}(0) - y_{it}(0) \mid E_i = e] = \mathbb{E}[y_{it'}(0) - y_{it}(0)] \forall t, t'$. This assumption requires that, were it not for the physician exit, antibiotic prescribing to treated patients would have followed the same trend as prescribing to untreated patients. The parallel trends assumption implies that any change in prescribing to a treated patient i after treatment onset can be attributed to the physician

exit, rather than to underlying differences in trends between cohorts, including the never-treated group. As the timing of physician exits is arguably exogenous to underlying patient trends, we believe this assumption to be plausible. In a sensitivity analysis, we relax the parallel trends assumption to hold conditional on time-varying patient characteristics.

Assumption 2: No cohort engages in anticipatory behavior prior to the exiting event, $\mathbb{E}[y_{it}(1) - y_{it}(0) \mid E_i = e] = 0 \forall t < e$. This assumption requires that treated patients do not adjust their antibiotic consumption prior to being exposed to the physician exit. If this assumption holds for all pre-treatment periods, treated patients do not exhibit pre-trends in antibiotic consumption. In an event study specification, we allow for pre-trends that differ between treated and never-treated patients. Differential pre-trends can indicate a violation of the no anticipatory behavior assumption.

Assumption 3: The average treatment effect on the treated over cohorts is homogenous for all time periods and cohorts, $\mathbb{E} \left[\frac{y_{it}(1) - y_{it}(0)}{\Delta_i} \mid E_i = e, \Delta_i \right] = \mathbb{E} \left[\frac{y_{it}(1) - y_{it}(0)}{\Delta_i} \mid D_{it} = 1, \Delta_i \right] = \theta \forall t, e$. In an event study specification, we allow treatment effects to differ by time relative to the physician exit. However, even in the event study specification, we require that treatment effects do not differ by cohort. The homogeneity assumption is violated if treatment effects differ between early-treated patients and later-treated patients. To allow for cohort heterogeneity in treatment effects, we estimate cohort-specific treatment effects. In order to obtain the average treatment effects, we aggregate the cohort-specific treatment effects with weights depending on cohort size as proposed by Sun and Abraham (2020).

Assumption 4: Attrition of patients from our panel of patient-calendar year observations is independent of potential outcomes. Our panel is unbalanced as some patients are unobserved in the beginning or the end of the sample period, their assigned general practice clinic is not matched to our sample of clinics, or they change their clinic without being exposed to physician exit. The absence of selective attrition implies that treatment effects for patients who left the panel are not systematically different from treatment effects for patients who stay in the panel.¹⁷

Assumption 5: Patients do not sort selectively to physicians based on their potential outcomes in antibiotic prescribing, $\mathbb{E} \left[\frac{y_{it}(1) - y_{it}(0)}{\Delta_i} \mid D_{it} = 1, \Delta_i \right] = \mathbb{E} \left[\frac{y_{it}(1) - y_{it}(0)}{\Delta_i} \mid D_{it} = 1 \right] = \theta$. If for example patients with relatively high antibiotic consumption at low-prescribing pre-exit physicians systematically sort into high-prescribing post-exit physicians, we would underestimate the share of physician effects θ . We formally test for selective sorting based on observable predictors of patients' antibiotic consumption using an approach proposed by Fadlon and Van Parys (2020). This assumption is required because we scale physician effects by Δ_i in Equation (3) to measure the share of prescribing differences determined by differences in physician practice styles.

¹⁷In Table 9 of Appendix A.4 we show that there are no substantial differences in average antibiotic prescribing between our main sample and excluded observations from a subset of patients with incomplete spells. We show average descriptive statistics for excluded observations of patients for whom a clinic change does not correspond to a physician exit or the assignment pre- or post-treatment is to an out-of-sample clinic.

5.2 Estimation

Based on our identification strategy, we estimate the causal treatment effect using two-way patient and year fixed effects in a static and in an event study setup. We estimate the following static specification:

$$y_{it} = \tilde{\alpha}_i + \theta \times D_{it} \times \hat{\Delta}_i + x_{it}\beta + \epsilon_{it}, \quad (4)$$

where we measure antibiotic prescribing y_{it} in logs and assume log prescribing to be linear and additively separable in patient and physician effects, such that patient and physician effects affect level prescriptions multiplicatively.¹⁸ $\tilde{\alpha}_i = \alpha_i + \delta_j$ corresponds to patient fixed effects subsuming the initial physicians' fixed effect. We cannot identify patient fixed effects α_i separately from the initial physicians' fixed effect δ_j as our empirical strategy only identifies the difference in physician fixed effects $\delta_{j'} - \delta_j$. $\hat{\Delta}_i$ is the empirical estimate of the difference in mean prescribing between j and j' conditional on no treatment exposure. In our baseline specification x_{it} includes calendar-time fixed effects x_t , the indicator D_{it} for the post-exit period, and an indicator for the transitional year of the exit. In sensitivity analysis, we include time-varying patient and clinic characteristics in x_{it} . Our main coefficient of interest is θ , which captures the share of physician effects in determining antibiotic prescribing differences. We can interpret estimates of θ as causal effects unless the identifying assumptions stated above are violated. The identifying assumptions imply that exogeneity $\mathbb{E}[\epsilon_{it} | \tilde{\alpha}_i, D_{it} \times \hat{\Delta}_i, x_{it}] = 0$ holds.

Our second specification is a two-way fixed effects event study specification:

$$y_{it} = \tilde{\alpha}_i + \sum_{\substack{r=5 \\ r=-5, \\ r \neq -1}} \theta_r \times I_r \times \hat{\Delta}_i + x_{it}\beta + \epsilon_{it}, \quad (5)$$

where $r(i, t)$ defines the year relative to the exiting event, and $I_r = 1\{r(i, t) = r\}$ is an indicator that is one during relative year r . The omitted category is $r = -1$, the year before the exiting event. In our baseline specification x_{it} includes as control variables calendar year fixed effects x_t , the indicator D_{it} for the post-exit period, and relative year interactions outside of our effect window $I_r \times \hat{\Delta}_i$ with $r < -5, r > 5$. Under the identifying assumptions, the event study specification allows to test whether there are differential trends between the pre-exit antibiotic consumption of treated patients and the antibiotic consumption of never-treated patients, and it allows to detect dynamic treatment effects.

For inference, we use a parametric bootstrap procedure following Finkelstein et al. (2016). In a first step, we estimate for each set of physicians j average prescribing \hat{y}_j and its standard error $se(\hat{y}_j)$. We construct the asymptotic distribution of mean prescribing y_j as a normal distribution with mean \hat{y}_j and standard deviation $se(\hat{y}_j)$. In the second step, we bootstrap Equation (4) with 50

¹⁸The log specification implies that a change in physician practice style leads to larger changes in prescribing levels for patients with higher antibiotic consumption than for patients with lower antibiotic consumption (Finkelstein et al. 2016).

repetitions drawn at the patient level. Within each bootstrap repetition, we draw a realization of mean prescribing y_j for each set of physician j from its asymptotic distribution and construct the corresponding difference in mean prescribing for patients exposed to treatment, Δ_i , accordingly. The bootstrapped standard errors account for estimation error in mean prescribing y_j and therefore Δ_i .

6 Results

6.1 Physician effects in antibiotic prescribing

We estimate physician effects in antibiotic prescribing measured by the natural logarithm of 1 plus the number of antibiotic prescriptions purchased by a patient in a given year.

Panel A in Table 5 shows estimation results for Equation (4) using the log number of all antibiotic prescriptions and separated by subcategories. Our main parameter of interest is θ , the share of physician effects in determining differences in antibiotic prescribing, associated with $\hat{\Delta}_i \times D_{it}$.¹⁹ The estimation results indicate that physician effects make up an important share of the observed differences in antibiotic prescribing between clinics.²⁰ The point estimates indicate that physician effects determine about 53.8 percent of the differences in the log number of antibiotic prescriptions between clinics in our baseline specification. Physician shares in antibiotic prescribing differences are smaller, with about 46.8 percent, when only penicillins (J01 C) are considered. They are largest, with about 79.9 percent, in the case of second-line antibiotic prescriptions (J01 F, D, M).²¹

¹⁹Figure 9 in Appendix B.1 shows the distribution of estimated mean differences in antibiotic prescribing $\hat{\Delta}_i$.

²⁰Results are similar when measuring antibiotic prescribing by the natural logarithm of 1 plus Daily Defined Dose prescribed and are shown in Appendix B.2.

²¹In Appendix B.3, we estimate physician shares separately for macrolides, lincosamides, and streptogramins (J01 F), cephalosporins (J01 D), and quinolones (J01 M) and find that they are largest for macrolides, lincosamides, and streptogramins, and quinolones. We also estimate physician shares for the group of second-line prescriptions once erythromycin (J01 FA01) is excluded and find similar effect sizes as in the main analysis.

Table 5: Estimation results for the share of physician effects in antibiotic prescribing

Panel A	Number of prescriptions			
	Two-way fixed effects estimation ^a			
	J01	J01 C	J01 F, D, M	Other J01
$\hat{\Delta}_i \times D_{it}$	0.538*** (0.027)	0.468*** (0.03)	0.799*** (0.031)	0.521*** (0.055)
Event dummies ^c	yes	yes	yes	yes
Time-varying controls ^d	no	no	no	no
Observations	7,796,767	7,796,767	7,796,767	7,796,767
Groups (patients)	1,373,109	1,373,109	1,373,109	1,373,109
Panel B	Number of prescriptions			
	Two-way fixed effects estimation ^a			
	J01	J01 C	J01 F, D, M	Other J01
$\hat{\Delta}_i \times D_{it}$	0.564*** (0.028)	0.48*** (0.03)	0.809*** (0.031)	0.548*** (0.062)
Event dummies ^c	yes	yes	yes	yes
Time-varying controls ^d	yes	yes	yes	yes
Observations	7,653,853	7,653,853	7,653,853	7,653,853
Groups (patients)	1,346,414	1,346,414	1,346,414	1,346,414
Panel C	Number of prescriptions			
	Sun-Abraham interaction-weighted estimation ^b			
	J01	J01 C	J01 F, D, M	Other J01
$\hat{\Delta}_i \times D_{it}$	0.526*** (0.035)	0.544*** (0.041)	0.741*** (0.039)	0.19*** (0.039)
Event dummies ^c	yes	yes	yes	yes
Time-varying controls ^d	no	no	no	no
Observations	7,796,767	7,796,767	7,796,767	7,796,767
Groups (patients)	1,373,109	1,373,109	1,373,109	1,373,109

Notes: This table reports the average share of between-clinics difference in antibiotic prescribing attributable to physician effects, the coefficient of $\Delta_i \times D_{it}$. Δ_i denotes the difference in mean prescribing between patient i 's assigned sets of physicians and is estimated by $\hat{\Delta}_i$, the average prescribing to untreated patients. D_{it} denotes a post-treatment indicator. Antibiotic prescribing is measured by $\log(1 + \text{number of prescriptions})$. Standard errors are calculated using a parametric bootstrap to draw clinic-level mean prescribing, with 50 repetitions at the patient level. *** $p < 0.01$.

^a Two-way fixed effects estimation with calendar year fixed effects and patient fixed effects.

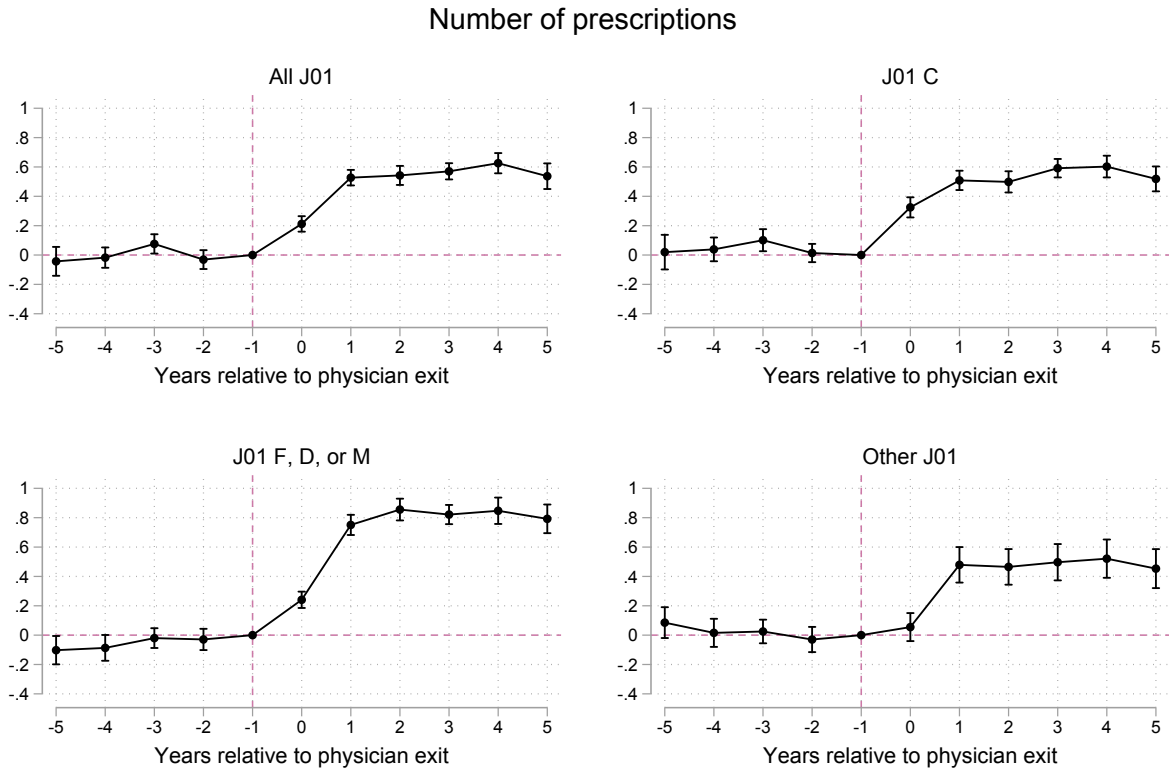
^b Interaction-weighted two-way fixed effects estimation includes calendar year and patient fixed effects, and interactions between relative year and cohort indicators. Averages of the relative-year specific treatment effects are formed to obtain the aggregate treatment effect (Sun and Abraham 2020).

^c Event dummies include an indicator for treatment onset (relative year $r = 0$) and post-treatment.

^d Control variables include Number of interns at the clinic, Age squared, Household size, Pregnant, Any visit to emergency department, and Any call to an emergency doctor.

Next, we estimate treatment effects separately for each year prior or after the physician exit using the event study specification of Equation (5). This allows treatment effects to flexibly vary across time relative to treatment. Results are depicted in Figure 3. In general, we observe an absence of systematic pre-trends, estimated with narrow confidence intervals. Where pre-trends differ statistically significantly from zero, they are economically negligible. The absence of pre-trends supports our identifying assumption that there is no anticipatory behavior prior to treatment onset. The event study figures also indicate that the causal effects we find from changes in a clinic’s physician composition are persistent in the years following the changes.

Figure 3: Event study estimates of the share of physician effects



Notes: The figures display event study estimates for the share of differences in antibiotic consumption that is driven by differences in physician fixed effects. Estimations include patient fixed effects, calendar year fixed effects, and indicators for treatment onset and post-exit. Relative year -1 is the last pre-exit period, relative year 0 is a transitional period, and relative year 1 is the first post-exit period. Antibiotic prescribing is measured by $\log(1 + \text{number of prescriptions})$.

The size of physician effects varies by subcategory of antibiotic drugs. We find the largest effects for prescriptions of second-line antibiotic drugs, whereas physicians may have less leverage in prescribing penicillins. This indicates that physician practice styles matter in particular for the composition of antibiotics prescribed. This result has important implications because the effects of antibiotic intake on antibiotic resistance also differ across classes of antibiotics, among which the largest effects are caused by broad-spectrum antibiotics.

6.2 Sensitivity analyses

To investigate the robustness of our main results, we estimate a number of alternative specifications.

First, to relax the parallel trends assumption to hold conditional on patient observable characteristics, we re-estimate Equations (4) and (5) to include time-varying observable patient characteristics. Specifically, we include control variables to account for changes in a patient’s underlying health conditions that might induce changes in the amount of antibiotic consumption this patient needs. We do not include time-varying patient characteristics which are likely affected by the physician exit treatment and practice styles. For example, a patient’s diagnosed medical conditions may affect their antibiotic consumption but also depend directly on the physician’s practice style. Such mediator variables would jeopardize identification of the treatment effect. As controls for health, we include a quadratic function of age, pregnancy status, and emergency service utilization measured by any visit to the emergency department of a hospital and any claim at an on-call doctor.²² Furthermore, we include the number of interns available for less than a year at a clinic as control variable in order to account for short-term differences in the perceived cost of antibiotic prescribing that are not reflected in general prescribing practice styles. The results remain similar and are shown in Panel B of Table 5 for the static specification and in Figure 4 for the event study specification.

Second, we allow for treatment heterogeneity by year of physician exit. The specifications in Equations 4 and 5 yield unbiased estimates of θ under the assumption that the average share of physician effects θ is homogeneous over cohorts defined by the year of treatment onset. If treatment homogeneity does not hold, the two-way fixed effects estimator for θ is a weighted average of relative time specific physician effects, where weights might be negative (Goodman-Bacon 2021). We relax the treatment homogeneity assumption by estimating a cohort-saturated two-way fixed effects specification that allows for cohort-relative time specific treatment effects. To obtain estimates for the average treatment effect on the treated, we aggregate cohort-relative time specific treatment effects as proposed by Sun and Abraham (2020).²³ The results from the interaction-weighted treatment effects estimation in Panel C of Table 5 and Figure 4 are similar to our main results. The only exception is antibiotic prescribing not including macrolides, lincosamides, streptogramins, cephalosporins, or quinolones (Other J01), for which the estimated share of physician effects is lower when allowing for cohort heterogeneity.

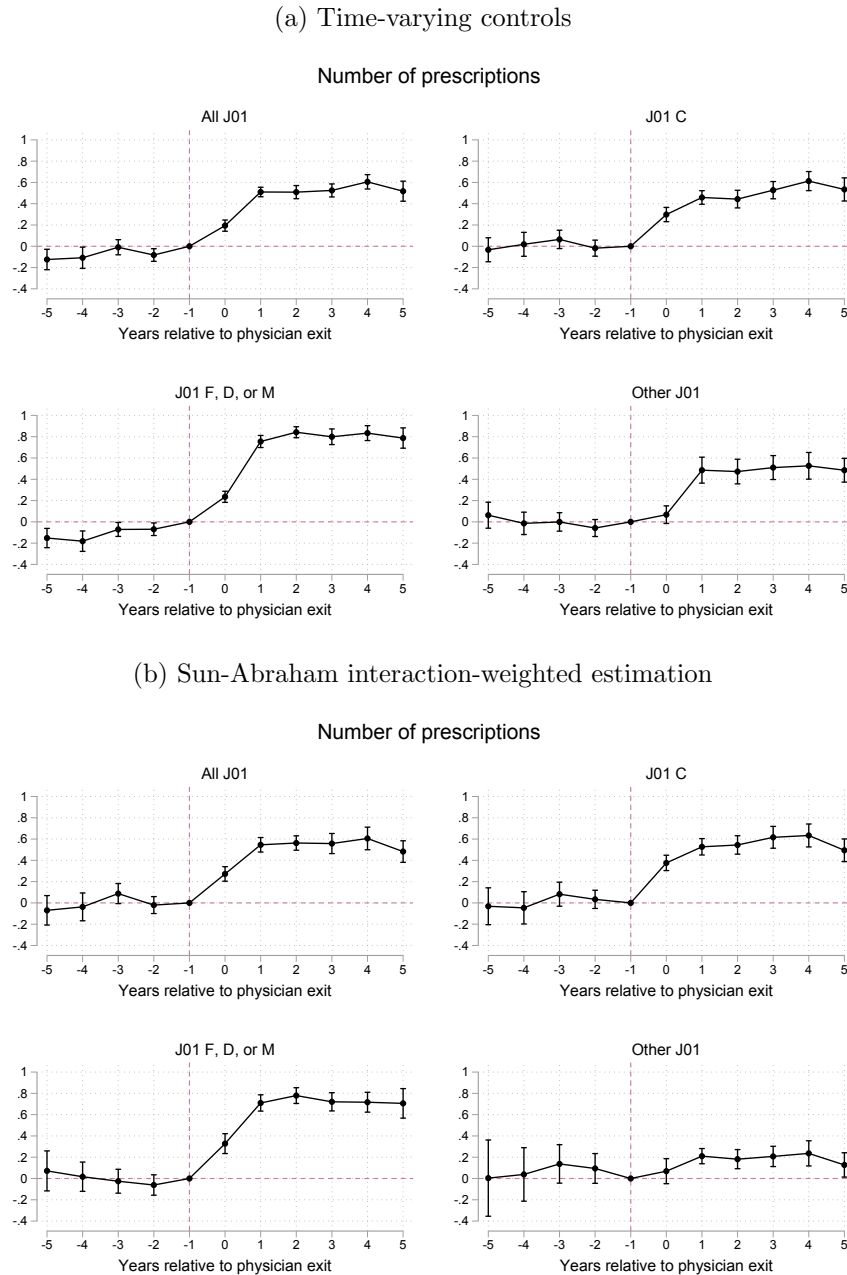
²²We code pregnancy status as a dummy variable using information from physician visit claim codes. In general, four exams with unique claim codes are performed during a pregnancy. We assume a patient is pregnant during a given year if she had at least one pregnancy exam performed during that year or if the second pregnancy exam was performed 17 weeks or less prior to the beginning of the year. The second pregnancy exam is mandatory but can be performed at any point between weeks 25 and 42.

²³In particular, we estimate the following specification:

$$y_{it} = \tilde{\alpha}_i + x_t + \sum_e \sum_r \theta_{e,r} \times I_e \times I_r \times \hat{\Delta}_i + \epsilon_{it},$$

where x_t denote the time fixed effects, cohort $e \in \{2006, \dots, 2012, \infty\}$ defines the year in which a patient is exposed to physician exit, and the remainder follows previous notation. $e = \infty$ characterizes the never-treated group, which is the omitted category. $\theta_{e,r}$ denotes cohort e -relative time r specific treatment effects. We obtain relative time specific treatment effects $\hat{\theta}_r$ by aggregating cohort-relative time specific treatment effect estimates weighting them by relative cohort size. We compute the static treatment effect estimate $\hat{\theta}$ by the average over post-treatment effects ($\hat{\theta}_r$ for $r < 0$).

Figure 4: Event study estimates of the share of physician effects, alternative specifications



Notes: Lines represent the 95% confidence intervals. Standard errors are calculated using a parametric bootstrap to draw clinic-level mean prescribing, with 50 repetitions at the patient level. Antibiotic prescribing is measured by $\log(1 + \text{number of prescriptions})$. Figure 4a displays event study estimates from estimations that include patient fixed effects, calendar year fixed effects, and indicators for treatment onset, post-exit, pregnancy, any visit to an emergency department, any call to an emergency department, any call to an emergency doctor, and as continuous variables age squared and household size. Figure 4b displays Sun-Abraham style interaction weighted estimates from fully saturated fixed effects specifications that include patient and calendar year fixed effects as well as interactions between relative period indicators and cohort indicators, where cohorts are defined by the calendar year of treatment onset. In a first step, cohort-relative year specific treatment effects are estimated. In the second step, relative year specific treatment effects are calculated as relative cohort size weighted averages by relative year.

6.3 Selective sorting in patient-physician reassignments

Selective sorting in reassignments of treated patients to physicians after physician exits can threaten our identification strategy. We investigate the presence of such sorting based on observable characteristics using a two-step procedure following the strategy in Fadlon and Van Parys (2020).

In the first step, we estimate a prediction function of the log number of antibiotic prescriptions using basic demographics and health variables (Panel B of Table 3, supplemented by squared age), as well as family background and education (Panel C of Table 3). We use all observations from never-treated patients as well as treated patients prior to when they are exposed to physician exit. In the second step, we predict post-treatment log prescriptions to treated patients after they are exposed to treatment. We regress predicted log prescriptions on the difference in average log prescriptions between post- and pre-exit physicians that treated patients are assigned to. These second step regressions include fixed effects for the pre-exit set of physicians of treated patients, as well as calendar years. We use the same parametric bootstrap procedure as above to compute standard errors. If patients sort to post-exit physicians based on their observable characteristics, and this sorting is systematic with regards to antibiotic prescribing, we expect that prescribing predicted from observable characteristics is correlated with differences in average prescribing between those physicians treated patients are assigned to.

Table 6 shows that the estimated relationship between predicted prescribing and the difference in average prescribing is small in magnitude or not statistically significant. For example, among patients with the same pre-exit physicians, if the post-exit log number of antibiotic prescriptions is predicted higher by one unit based on observable patient characteristics, this is associated with the post-exit physicians' average log prescriptions systematically higher by 0.001 units. While we cannot exclude selective sorting on unobservable characteristics such as preferences, we believe that it is unlikely for patients to choose their general practitioners primarily based on antibiotic prescribing behavior. This presumption is supported by the Danish institutional setting, where patients' choice of general practitioner is limited and, for example, 'shopping' for high prescribers of antibiotics is difficult.

Table 6: Patient-physicians selection on observable characteristics

	Difference in average prescribing ($\hat{\Delta}_i$) ^a			
	All J01 (1)	J01 C (2)	J01 F, D, M (3)	Other J01 (4)
Predicted prescribing ^b	0.0010** (0.0005)	0.0016*** (0.0004)	-0.0002 (0.0009)	-0.0005* (0.0003)
Observations	431,989	431,989	431,989	431,989

Notes: This table reports the estimated relationship between antibiotic prescribing predicted based on patient observable characteristics and the difference in mean antibiotic prescribing between post- and pre-exit physicians for treated patients. Antibiotic prescribing is measured by $\log(1 + \text{number of prescriptions})$. Two-way fixed effects estimation (calendar year and pre-exit physicians fixed effects) with observations on the patient-year level. Standard errors are calculated using a parametric bootstrap to draw clinic-level mean prescribing, with 50 repetitions at the patient level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

^a Δ_i denotes the difference in mean prescribing between patient i 's assigned sets of physicians and is estimated by $\hat{\Delta}_i$, the average prescribing to untreated patients.

^b To assess selective re-assignment between physicians and patients based on observable characteristics, we use a two-step procedure (see Fadlon and Van Parys 2020). We first predict post-treatment antibiotic prescribing to treated patients after they are exposed to treatment. To do so, we form linear predictions based on basic demographics, health, family background, and education (as in Table 3) with coefficients estimated on prescribing to treated patients prior to treatment and never-treated patients. In the second step, we regress prescribing as predicted from observable patient characteristics on the difference in average prescribing between post- and pre-exit physicians that treated patients are assigned to. The second step regressions include fixed effects for the pre-treatment set of physicians, as well as calendar years.

7 Characterizing physician effects

7.1 Correlates of practice styles

Heterogeneity in practice styles can be determined by many mechanisms. To shed light on such potential mechanisms, we characterize the correlations between practice styles and observable physician as well as clinic characteristics. Physician characteristics include information on age, household size, Ph.D. education, gender, and migration background averaged over general practitioners in a clinic. Clinic characteristics include diagnostic availability and practice size.²⁴

We proceed in two steps. First, we estimate the difference in prescribing practice styles between physicians to which treated patients are assigned *separately for each pair of physicians*. We therefore estimate physician effects separately for each pair of origin-destination physicians instead of scaling and aggregating treatment effects. Second, we regress our estimated time-invariant differences in prescribing styles on differences in standardized observable characteristics between physician pairs. Our estimation procedure is described in more detail in Appendix C.

We explore the relationships between prescribing practice styles and physician observables using two estimation approaches: either by bivariate ordinary least squares (OLS) regressions of prescribing style differences on physician observables or by a multivariate regression approach.²⁵

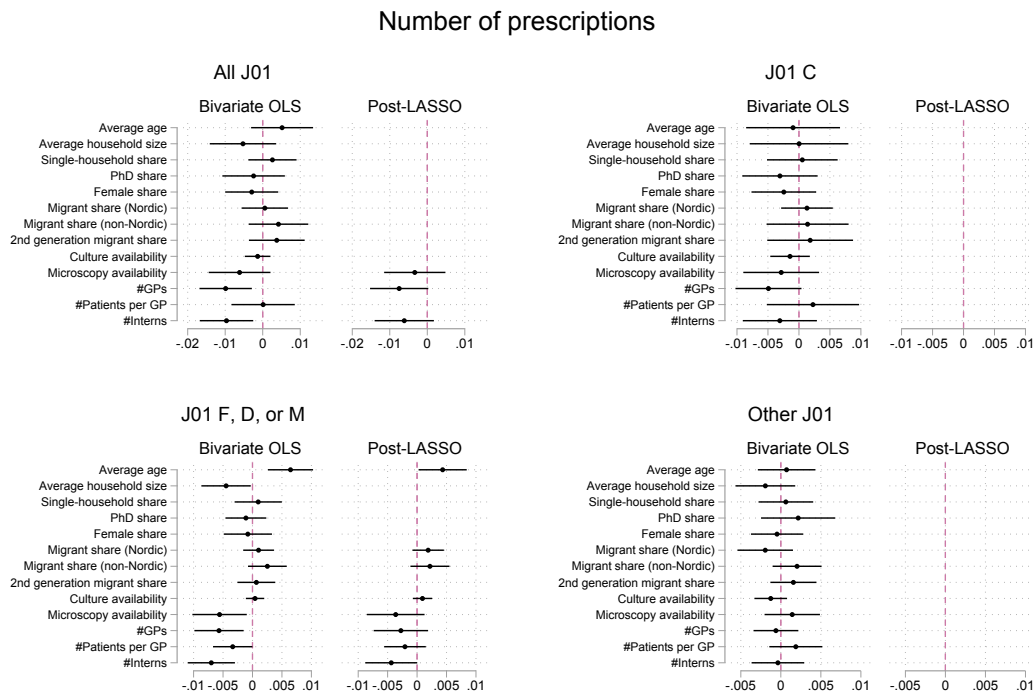
²⁴The variables included correspond to Panels B and C of Table 4. Appendix C describes the variables in detail.

²⁵Additionally, we estimate the relationships between prescribing styles and physician observables in a fixed effects regression approach in Appendix C. The results are similar to our main results.

For multivariate regressions we employ a data-driven post-LASSO OLS estimation approach that accounts for correlation between different physician characteristics (Belloni et al. 2013). A standard OLS model including all covariates might suffer from lack of precision in the estimates. The post-LASSO OLS approach first selects variables using a LASSO regression which penalizes model complexity. It then performs OLS on the reduced model including only the selected variables from the first step.

Figure 5 shows correlates of prescribing practice style differences. Each row represents the association between prescribing style differences and the difference in a physician observable estimated by OLS. The left panel shows each coefficient associated with a bivariate regression of prescribing style differences on a different variable. The right panel shows coefficients associated with a multivariate regression of prescribing style differences on variables selected by first-stage LASSO. We standardize all covariates prior to taking differences. Hence, the coefficients report the association between a one standard deviation increase in the observable characteristic and the change in the prescribing style.

Figure 5: Correlates of antibiotic prescribing practice style differences



Notes: The figure displays bivariate OLS (left panels) and post-LASSO (right panels) regression results of the estimated difference in prescribing practice style, measured by $\log(1 + \text{number of antibiotic prescriptions})$, on differences in physician characteristics between sets of pre- and post-exit physicians that treated patients are assigned to. To obtain post-LASSO estimates, we run a LASSO regression on the full set of physician characteristics, with the penalty level chosen by 10-fold cross validation to minimize mean squared error. We subsequently run an OLS regression with the estimated differences in practice styles regressed on the set of physician characteristics selected by the LASSO regression. Missing coefficients indicate that a variable has not been selected in the LASSO regression. Standard errors are calculated using a parametric bootstrap to draw differences in practices styles with 50 repetitions. Physician characteristics are standardized to have mean 0 and standard deviation 1 prior to differencing.

Figure 5 indicates that antibiotic prescribing practice style differences are associated with certain physician and clinic characteristics. Correlations with overall prescribing are driven by prescribing differences in terms of second-line antibiotic drugs. In particular, a higher average age of general practitioners is associated with a higher second-line antibiotic prescribing style. The availability of diagnostic microscopy as well as a larger clinic size in terms of the number of physicians, interns, and patients per physician are associated with a lower prescribing style. However not all effects are significantly different from zero in the post-LASSO specifications.

One interpretation of the observed correlations are changes in medical education over time that might affect younger physicians more strongly and could explain a lower second-line prescribing style in younger practices. A negative correlation in prescribing style and clinic size could be explained by differences between clinics in the weights placed on the private benefit compared to the social cost of antibiotic prescribing. While an antibiotic prescription has the private benefit of increasing chances of recovery for a patient, it comes with the externality of increasing antibiotic resistance in the community. For larger clinics, less personal patient-physician relations may lead to a lower weight placed on the private benefit of an antibiotic prescription compared to smaller clinics. Finally, clinics with a larger set of diagnostic tools might be able to target antibiotic prescriptions better.

7.2 Health effects

So far, we have focused on identifying heterogeneity in prescribing practice styles. Such heterogeneity may not be undesirable if it generates the best overall health outcomes, even if at the cost of potential increases in antibiotic resistance. However, if the effect of practice style variation is unrelated to health outcomes or if prescribing intensity adversely affects patient health, efficiency losses of such variation are difficult to reject. To inspect this further, we relate our results on antibiotic prescribing practice styles to patients' health outcomes. Using our estimates of physician effects, we investigate whether differences in prescribing styles correspond to differences in patient outcomes. We ask the following question: When patients are exogenously reassigned to physicians with more intense antibiotic prescribing styles, does this affect their rate of hospitalization?

We focus on hospitalizations for a subset of ambulatory care sensitive conditions (ACSC) typically related to infections, which are potentially preventable under sufficient primary care. Indications for ACSC are commonly used to measure ambulatory care quality.²⁶ We consider acute ACSC that are frequently caused by bacterial infections and commonly treated in general practice: Skin and soft tissue infections, ear, nose and throat infections, perforated or bleeding ulcer, urinary tract infections, and pneumonia. A complete list of ICD-10 codes is included in Appendix D. We construct an indicator variable that is one if a patient has been hospitalized for any of the infection-related ACSC we consider in a given year. We also construct indicator variables for each of the ACSC separately that indicate whether a patient has been hospitalized for a given condition as the primary diagnosis in a given year. We exclude referrals from general practitioners as well as internal hospital referrals

²⁶For example, the World Health Organization defines ACSC as "Conditions for which hospitalizations can be avoided by timely and effective care in ambulatory settings" (World Health Organization Regional Office for Europe 2016).

unless the diagnosis was made at the first day of a patient’s hospitalization spell. In Appendix D, we show basic summary statistics and results for specifications in which we use the count of hospitalizations due to ACSC as outcome variable.

We estimate a difference-in-differences specification where the treatment is based on the estimated differences in prescribing practice styles between physicians which patients exposed to physician exit are assigned to. Our treatment variable is the interaction between an indicator for having been exposed to physician exit and the estimated difference in physicians’ prescribing styles. The treatment effect corresponds to the effect of an exogenous change in the antibiotic prescribing style a patient is exposed to onto hospitalization rates due to ACSC. We estimate the following baseline specification:

$$h_{it} = \tilde{\alpha}_i + \eta \times D_{it} \times (\widehat{\delta_{j'} - \delta_j}) + x_{it}\beta + \omega_{it}, \quad (6)$$

where h_{it} is an indicator variable for patient i being hospitalized for an infection-related ACSC in year t , α_i denotes patient-fixed effects, x_{it} includes calendar-year fixed effects, the post-exit indicator D_{it} , and an indicator for the year of the exit, and ω_{it} is an error term. Our coefficient of interest is η associated with the interaction between the post-exit indicator D_{it} and the estimated difference in prescribing styles $(\widehat{\delta_{j'} - \delta_j})$. We can interpret estimates of η as causal effects if the exogeneity assumption $\mathbb{E}[\omega_{it} | \tilde{\alpha}_i, D_{it} \times (\widehat{\delta_{j'} - \delta_j}), x_{it}] = 0$ holds.

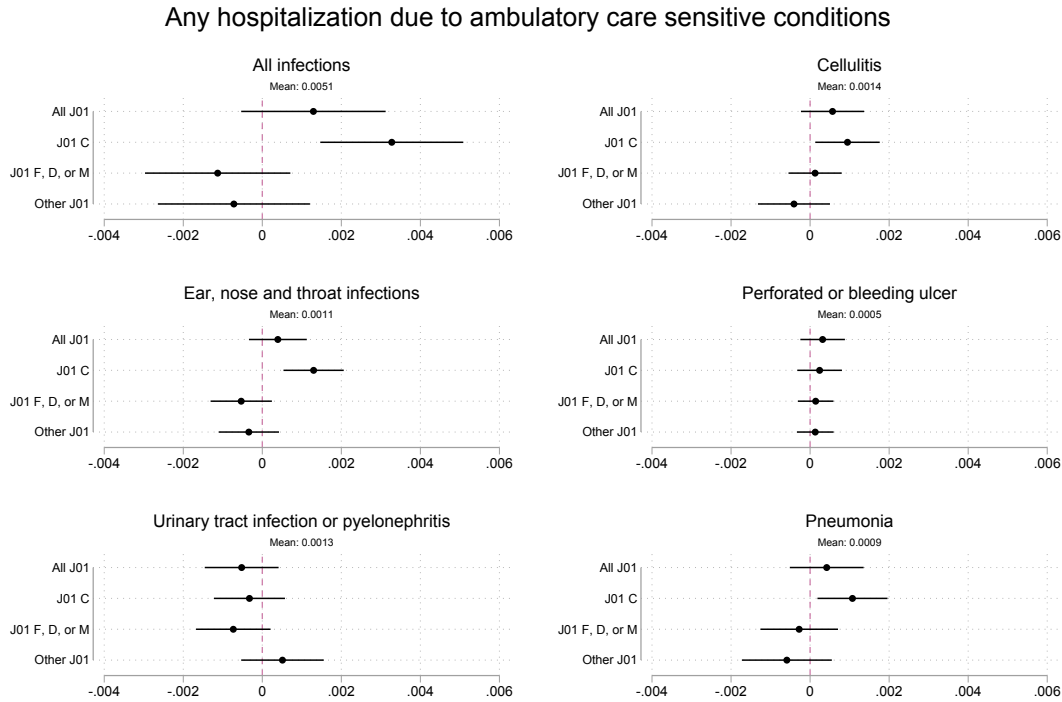
Our baseline results are shown in Figure 6.²⁷ For easier interpretation, we report scaled coefficients.²⁸ The scaled coefficients capture the effect of an increase in prescribing style by one standard deviation of overall prescribing on the probability of any hospitalization due to infection-related ACSC.

The results show that a more intense prescribing style points toward a higher rate of infection-related hospitalization, although this effect is not statistically significant. However, inspecting the antibiotic subcategories reveals that the aggregate effect is driven by the effect of a more intense penicillin prescribing style. An increase in the penicillin prescribing style by one standard deviation increases the probability of hospitalization due to infection-related ACSC significantly at the 5 percent level. This effect is driven primarily by hospitalizations for ear, nose and throat infections, pneumonia, and to a smaller degree by hospitalizations for cellulitis. The point estimates indicate that an increase in a physician’s penicillin prescribing style by one standard deviation of log prescribing increases the probability of hospitalizations for an infection-related ACSC by 0.38 percentage points. A one standard deviation increase of penicillin prescribing style has economically relevant effects as the average rate of hospitalization due to infection-related ACSC is 0.59 percent. Some of our

²⁷In Appendix D we show estimation results for a number of alternative specifications: We define the outcome as the number of hospitalizations due to ACSC, allow for an extended set of control variables, define treatment in a binary fashion as being exposed to an increase (decrease) in prescribing style, and estimate the treatment effect η as an aggregate of cohort-specific treatment effects in a Sun-Abraham-style framework.

²⁸In particular we report coefficients $\tilde{\eta} = \eta * \sigma(y_{it})$, where $\sigma(y_{it})$ is the standard deviation in log antibiotic prescriptions. η is the original coefficient of interest estimated from Equation (6). The confidence intervals have been scaled correspondingly using rescaled standard errors $se(\tilde{\eta}) = se(\eta) * \sigma(y_{it})$, where $se(\eta)$ are the original standard errors. We estimate the original standard errors $se(\eta)$ using a bootstrap with 50 repetitions on the patient level.

Figure 6: Effect of an increase in prescribing style by one standard deviation of log prescriptions on hospitalizations for ambulatory care sensitive conditions



Notes: The figure displays estimation results for the effect of antibiotic prescribing practice style differences on hospitalizations for infection-related ambulatory care sensitive conditions. We obtain the effect of practice style differences as the coefficient associated with the interaction between the estimated difference in prescribing practice styles measured by $\log(1 + \text{number of antibiotic prescriptions})$, and a post-exit indicator. Estimations include patient fixed effects, calendar year fixed effects, and indicators for treatment onset and post-exit. Lines represent the 95% confidence intervals. Standard errors are calculated using a parametric bootstrap to draw practice style differences, with 50 repetitions at the patient level.

sensitivity checks indicate a smaller or statistically insignificant effect of an increase in penicillin prescribing style, even if the direction of the coefficients remains unchanged.

Some of our sensitivity specifications indicate that a higher antibiotic prescribing style decreases the rate of hospitalization for urinary tract infections. While not statistically significant, the coefficient estimates in our main specification point toward the same direction for hospitalizations for urinary tract infections.

One possible explanation for the positive effect of penicillin prescribing on patient hospitalization rates might be that a large share of respiratory tract infections are caused by viruses rather than bacteria (Fleming-Dutra et al. 2016). While penicillins constitute the largest share of antibiotic prescriptions, antibiotic treatments are ineffective against viral infections. When treated insufficiently, upper respiratory infections in ear, nose or throat can cause more severe pneumonia. Patients assigned to physicians with more intense penicillin prescribing styles might be more likely to be hospitalized for respiratory viral infections if for example their physicians' antibiotic prescribing crowds out

treatments for alternative diagnoses. A related possible explanation is that antibiotic resistance renders standard antibiotic treatments such as penicillins ineffective. Appropriate treatment in both cases requires diagnostic information on the organism causing the infection.

8 Conclusion

For policies aimed at increasing efficiency in antibiotic prescribing, it is crucial to identify who drives prescription decisions. We assess the extent to which physicians determine differences in antibiotic prescribing between general practice clinics. To identify physician effects on prescribing separately from patient effects, we leverage quasi-experimental reassignments between patients and physicians from physician exits from clinics.

Our results indicate that physician effects determine antibiotic prescribing to a substantial degree. Our results for total antibiotic prescribing imply that harmonization of practice styles would reduce differences in log antibiotic consumption at general practice clinics in our sample by more than half. Physicians' practice styles determine 53% to 56% of the differences between clinics in terms of total antibiotic prescriptions, and the effect is much larger, 74% to 81%, when we restrict our analysis to the antibiotic classes macrolides, lincosamides and streptogramins, cephalosporins, and quinolones. These classes are considered second-line treatments as they have higher costs than alternative antibiotic drugs compared to limited evidence of clinical benefits, and are more likely to cause antibiotic resistance. Reducing prescriptions of second-line treatments from general practitioners by targeting the individual physicians could substantially curb antibiotic consumption and the rise of antibiotic resistance.

Additionally, we investigate the differences in antibiotic prescribing styles by studying their correlations with physician and clinic characteristics. We find a positive relation between prescribing intensity and the physicians' age, as well as a negative relation between prescribing intensity and the availability of diagnostic microscopy and clinic size. These correlations could for example be driven by changes in medical education and awareness over time, better use of diagnostic tools, or more flexible patient-physician treatment assignments.

Lastly, we tackle the question whether the observed differences in antibiotic prescribing practice styles affect patient health. We analyze avoidable ambulatory care sensitive conditions that can be caused by bacterial infections. For the majority of conditions, we find that a reduction in antibiotic prescribing style has no effect on a patient's hospitalization rate or even reduces it.

Our study indicates that a reduction in overall and, in particular, second-line antibiotic consumption can be achieved by targeting the individual physician. In general, decreasing antibiotic prescribing intensity in physicians' practice styles does not affect patient health. Instead, our results indicate that some conditions are treated better by physicians with less intense antibiotic prescribing style. Our results also indicate that better diagnostic accuracy might assist or improve upon outcomes achievable through across the board reductions in antibiotic prescribing.

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Appendix

A Further descriptives

A.1 Distribution of antibiotic prescribing over clinics

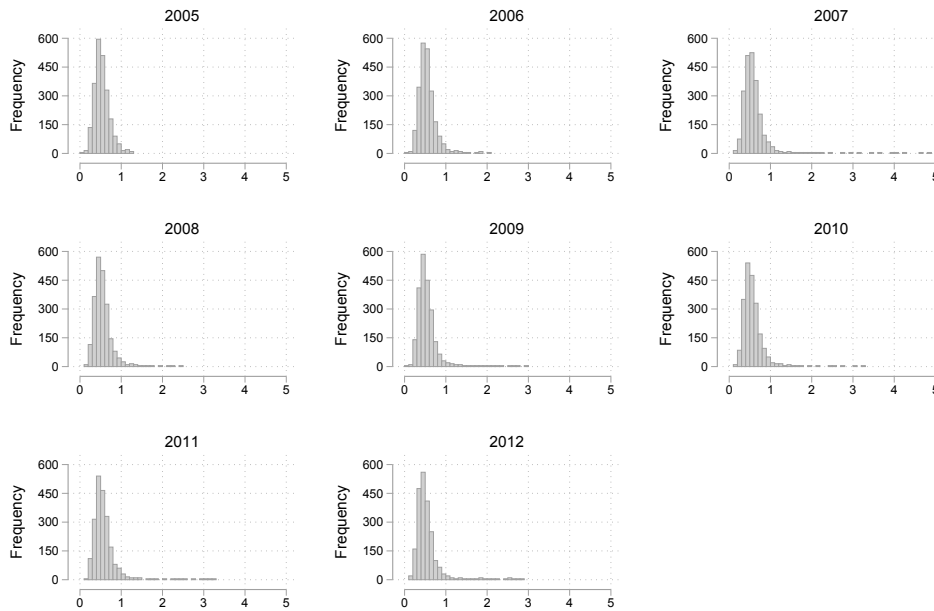
Figure 7 shows the distribution of average antibiotic prescribing per patient over general practice clinics in Denmark for each year of our sample period from 2005 to 2012. While average prescribing in Denmark is low, there is substantial and persistent heterogeneity between clinics.

A.2 Physician age

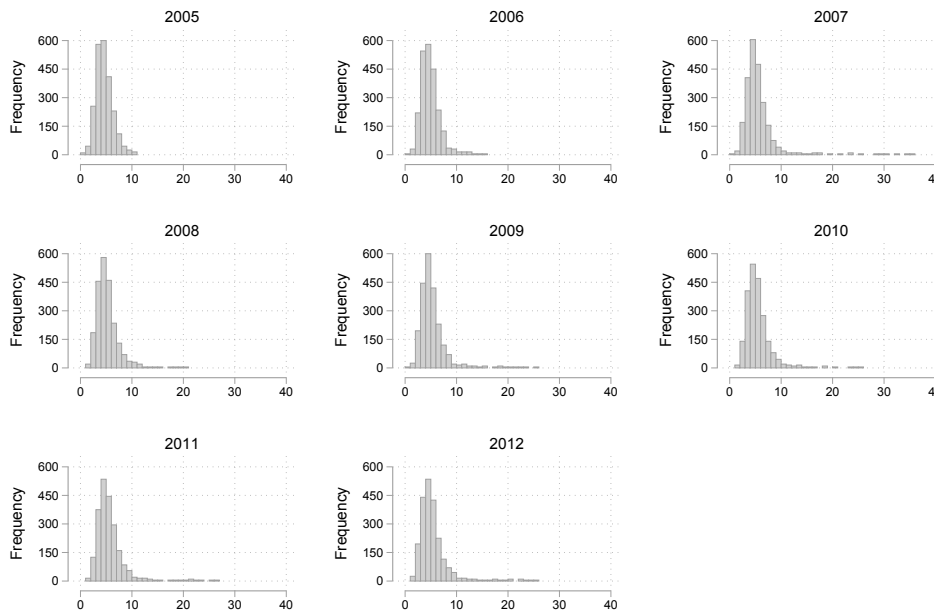
Figure 8 shows that, while the majority of reassignments for treated patients is from older to younger physicians, we also observe reassignments from younger to older physicians. Moreover, on average the age difference between physicians is not substantial.

Figure 7: Distribution of average antibiotic prescribing (ATC J01) per patient over general practice clinics

(a) Number of antibiotic prescriptions (levels)



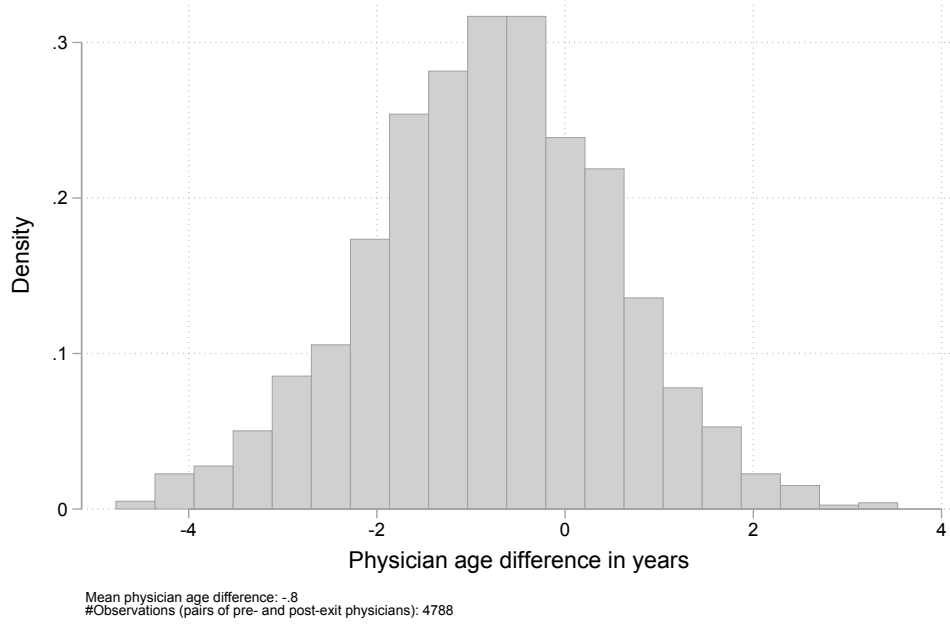
(b) Daily Defined Dose (levels)



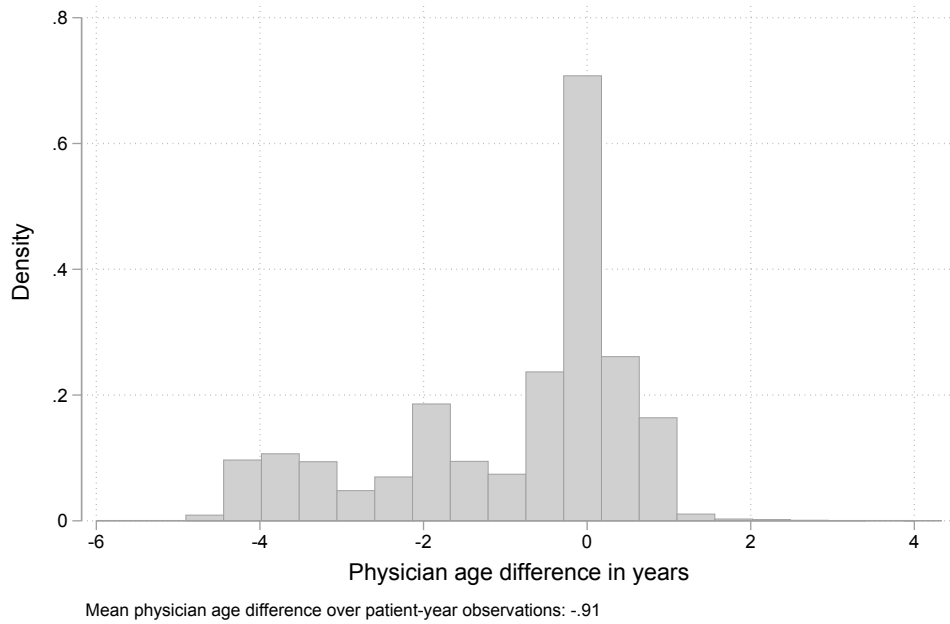
Notes: Average antibiotic prescriptions dispensed per patient and year at the clinic-level. Bunched in groups of five clinics to ensure the required data anonymization. The upper five percentiles are omitted.

Figure 8: Difference in average age between post- and pre-exit physicians

(a) Distribution over pairs of pre- and post-exit physicians



(b) Distribution over treated patients weighted by years observed



Notes: In Figure 8a, values are bunched for groups of five physicians with similar age difference due to data anonymization. In Figure 8b, values are bunched for groups of five patient-year observations.

A.3 Descriptive statistics for out-of-sample clinics and patients

We can compute average characteristics for out-of-sample clinics which are listed in the Danish registry of clinics but which we have excluded from our analyses, as well as for patients assigned to those clinics. In particular, these are general practice clinics that underwent physician entries or multiple long-term staff changes over our period of observation from 2005 to 2012.

Table 7 shows averages for patient-year observations and Table 8 shows average clinic-level characteristics. Most noticeably, staff sizes for out-of-sample clinics are larger than for in-sample clinics. This is not surprising, as larger clinics may have more fluctuation in staff. Moreover, patients assigned to out-of-sample clinics tend to be younger. However, antibiotic prescribing does not differ substantially from our main sample.

Table 7: Averages for out-of-sample observations

	Out of sample
A: Antibiotic prescribing^a	
Total J01 (all antibiotics)	0.28
J01 C (penicillins)	0.20
J01 F, D or M (second-line)	0.07
Other J01 (other antibiotics)	0.04
B: Basic demographics and health	
Age	41.21
Female	0.54
Pregnant	0.02
Household size	2.61
Any visit to an emergency department	0.15
Any call to an emergency doctor	0.18
C: Family background and education	
Married couple	0.54
Cohabiting couple with children	0.07
Cohabiting couple without children	0.07
Single	0.32
First generation migrant (nordic)	0.01
First generation migrant (other country)	0.06
Second generation migrant	0.03
Missing education	0.21
School grade 7 to 10	0.27
High school or vocational training	0.32
Short higher education	0.03
Medium higher education	0.12
Long higher education	0.05
Phd education	0.003
No education	0.001
D: Any hospitalization^b	
All infection-related conditions	0.005

Cellulitis	0.001
Ear, nose and throat infections	0.001
Perforated or bleeding ulcer	0.001
Urinary tract infection	0.001
Pneumonia	0.001
<hr/>	
Total observations (patient-years)	15,461,226
<hr/>	

^a Measured by $\log(1 + \text{number of antibiotic prescriptions})$.

^b Hospitalizations for acute ambulatory care-sensitive conditions (ACSC) commonly caused by bacterial and non-bacterial infections considered (see Appendix D for a complete list of ICD-10 codes). Referrals from general practitioners and delayed internal hospital referrals are excluded.

Table 8: Averages for out-of-sample clinics

	Out of sample
<hr/>	
A: Average antibiotic prescribing per patient^a	
Total J01 (all antibiotics)	0.28
J01 C (penicillins)	0.19
J01 F, D or M (second-line)	0.07
Other J01 (other antibiotics)	0.05
B: Average physician characteristics	
Age	55.64
Household size	2.62
Female	0.36
Single	0.16
First generation migrant (nordic)	0.01
First generation migrant (other country)	0.03
Second generation migrant	0.01
Phd education	0.01
C: Size and equipment	
Number of physicians	1.89
Number of interns	0.25
Number of patients per physician	1878.28
Diagnostic culture available	1.00
Diagnostic microscopy available	0.80
<hr/>	
Total observations (clinic-years)	6,861
<hr/>	

^a Measured by $\log(1 + \text{number of antibiotic prescriptions})$

A.4 Descriptive statistics for excluded patient-year observations

We can compute average characteristics for observations which are associated with patients in our sample but which we have excluded from the final panel of patient-years. We exclude patient-year observations in order to ensure that any switch in a patient’s general practice clinic is associated with the treatment, a physician exit, and we exclude observations when treated patients are assigned to out-of-sample clinics. For a subset of never-treated and treated patients in our sample, we thus drop observations and end up with an unbalanced panel.

Table 9 shows averages in observations which we drop for in-sample patients. In the case of never-treated patients, most noticeably the average age is lower than in our main sample. We might observe a lower average age because these observations are dropped when never-treated patients switch clinics for reasons unrelated to a physician exit as we then only keep observations at the modal clinic. Presumably, switching clinics is more common among younger patients, who could for example be more likely to move geographically or be more selective about choosing their physicians. In the case of treated patients, average characteristics are similar to the main sample. In both cases, antibiotic prescribing in the excluded observations does not differ substantially from our main sample, alleviating concerns about selective attrition based on antibiotic prescribing.

Table 9: Averages for excluded observations of in-sample patients

	Never-exposed to physician exit	Exposed to physician exit
A: Antibiotic prescribing^a		
Total J01 (all antibiotics)	0.28	0.27
J01 C (penicillins)	0.19	0.19
J01 F, D or M (broad-spectrum)	0.06	0.06
Other J01 (other antibiotics)	0.05	0.05
B: Basic demographics and health		
Age	36.42	43.61
Female	0.57	0.57
Pregnant	0.04	0.03
Household size	2.56	2.56
Any visit to an emergency department	0.17	0.15
Any call to an emergency doctor	0.22	0.22
C: Family background and education		
Married couple	0.45	0.53
Cohabiting couple with children	0.08	0.06
Cohabiting couple without children	0.11	0.08
Single	0.36	0.34
First generation migrant (nordic)	0.01	0.01
First generation migrant (other country)	0.07	0.07
Second generation migrant	0.03	0.03
Missing education	0.20	0.17
School grade 7 to 10	0.25	0.26

High school or vocational training	0.33	0.34
Short higher education	0.03	0.03
Medium higher education	0.13	0.13
Long higher education	0.06	0.06
Phd education	0.003	0.003
No education	0.001	0.001
D: Any hospitalization^b		
All infection-related conditions	0.005	0.006
Cellulitis	0.001	0.001
Ear, nose and throat infections	0.001	0.001
Perforated or bleeding ulcer	0.000	0.000
Urinary tract infection	0.001	0.002
Pneumonia	0.001	0.001
<hr/>		
Total observations (patient-years)	246,417	72,828
<hr/>		

^a Measured by $\log(1 + \text{number of antibiotic prescriptions})$.

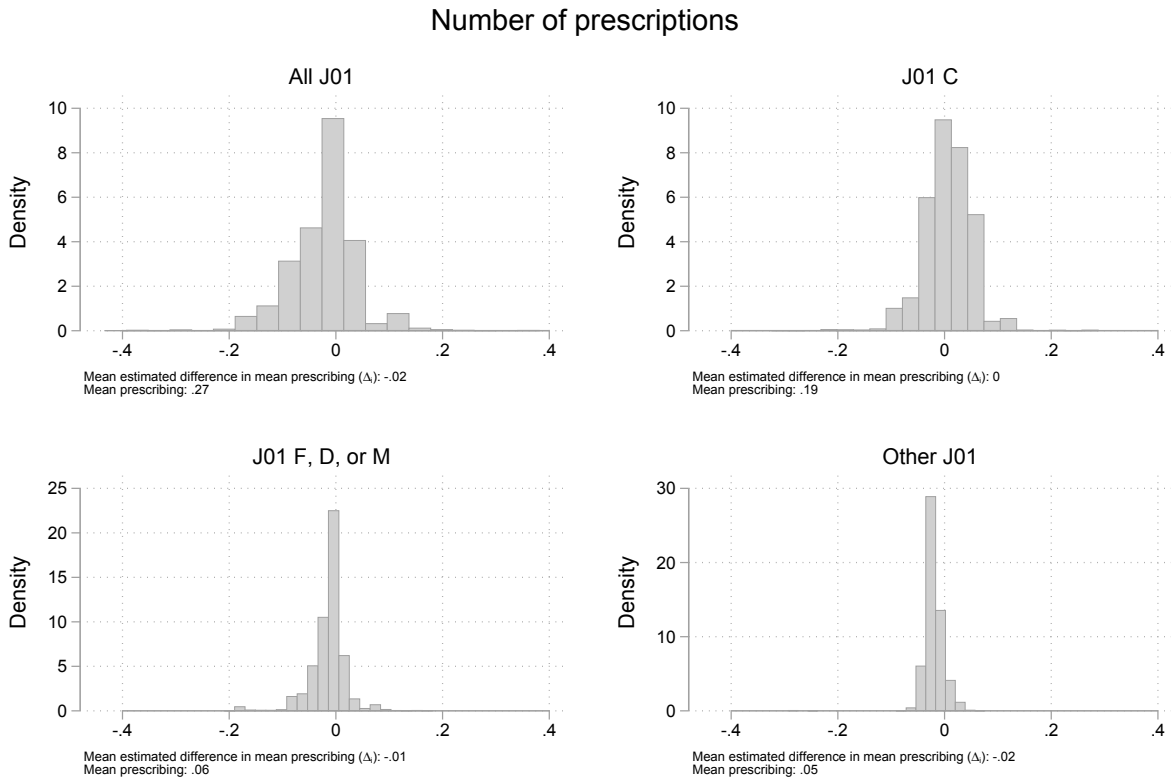
^b Hospitalizations for acute ambulatory care-sensitive conditions (ACSC) commonly caused by bacterial and non-bacterial infections (see Appendix D for a complete list of ICD-10 codes). Referrals from general practitioners and delayed internal hospital referrals are excluded.

B Further Results

B.1 Differences in mean prescriptions

Figure 9 shows a histogram of the difference in average log prescribing between treated clinics and never-treated destination clinics assigned to patients exposed to a physician exit ($\hat{\Delta}_i$). Our identification strategy uses these differences to obtain a standardized measure of the treatment effect.

Figure 9: Distribution over the difference in average log prescribing between treated patients' pre- and post-exit physicians ($\hat{\Delta}_i$)



Notes: Values are bunched for groups of five patients with similar estimated mean difference in average prescribing to ensure the required data anonymization.

B.2 Analysis based on Daily Defined Dose

Table 10, Figure 10, and Figure 11 show our main analyses when we measure antibiotic prescribing by $\log(1 + \text{Daily Defined Dose})$. The results are similar to our main results.

Table 10: Estimation results for the share of physician effects in antibiotic prescribing

Panel A	Daily Defined Dose			
	Two-way fixed effects estimation ^a			
	J01	J01 C	J01 F, D, M	Other J01
$\hat{\Delta}_i \times D_{it}$	0.546*** (0.026)	0.521*** (0.032)	0.783*** (0.031)	0.466*** (0.049)
Event dummies ^c	yes	yes	yes	yes
Time-varying controls ^d	no	no	no	no
Observations	7,796,767	7,796,767	7,796,767	7,796,767
Groups (patients)	1,373,109	1,373,109	1,373,109	1,373,109
Panel B	Daily Defined Dose			
	Two-way fixed effects estimation ^a			
	J01	J01 C	J01 F, D, M	Other J01
$\hat{\Delta}_i \times D_{it}$	0.567*** (0.027)	0.533*** (0.031)	0.791*** (0.03)	0.494*** (0.053)
Event dummies ^c	yes	yes	yes	yes
Time-varying controls ^d	yes	yes	yes	yes
Observations	7,653,853	7,653,853	7,653,853	7,653,853
Groups (patients)	1,346,414	1,346,414	1,346,414	1,346,414
Panel C	Daily Defined Dose			
	Sun-Abraham interaction-weighted estimation ^b			
	J01	J01 C	J01 F, D, M	Other J01
$\hat{\Delta}_i \times D_{it}$	0.454*** (0.031)	0.496*** (0.042)	0.728*** (0.036)	0.181*** (0.038)
Event dummies ^c	yes	yes	yes	yes
Time-varying controls ^d	no	no	no	no
Observations	7,796,767	7,796,767	7,796,767	7,796,767
Groups (patients)	1,373,109	1,373,109	1,373,109	1,373,109

Notes: This table reports the average share of between-clinics difference in antibiotic prescribing attributable to physician effects, the coefficient of $\Delta_i \times D_{it}$. Δ_i denotes the difference in mean prescribing between patient i 's assigned sets of physicians and is estimated by $\hat{\Delta}_i$, the average prescribing to untreated patients. D_{it} denotes a post-treatment indicator. Antibiotic prescribing is measured by $\log(1 + \text{Daily Defined Dose})$. Standard errors are calculated using a parametric bootstrap to draw clinic-level mean prescribing, with 50 repetitions at the patient level. *** $p < 0.01$.

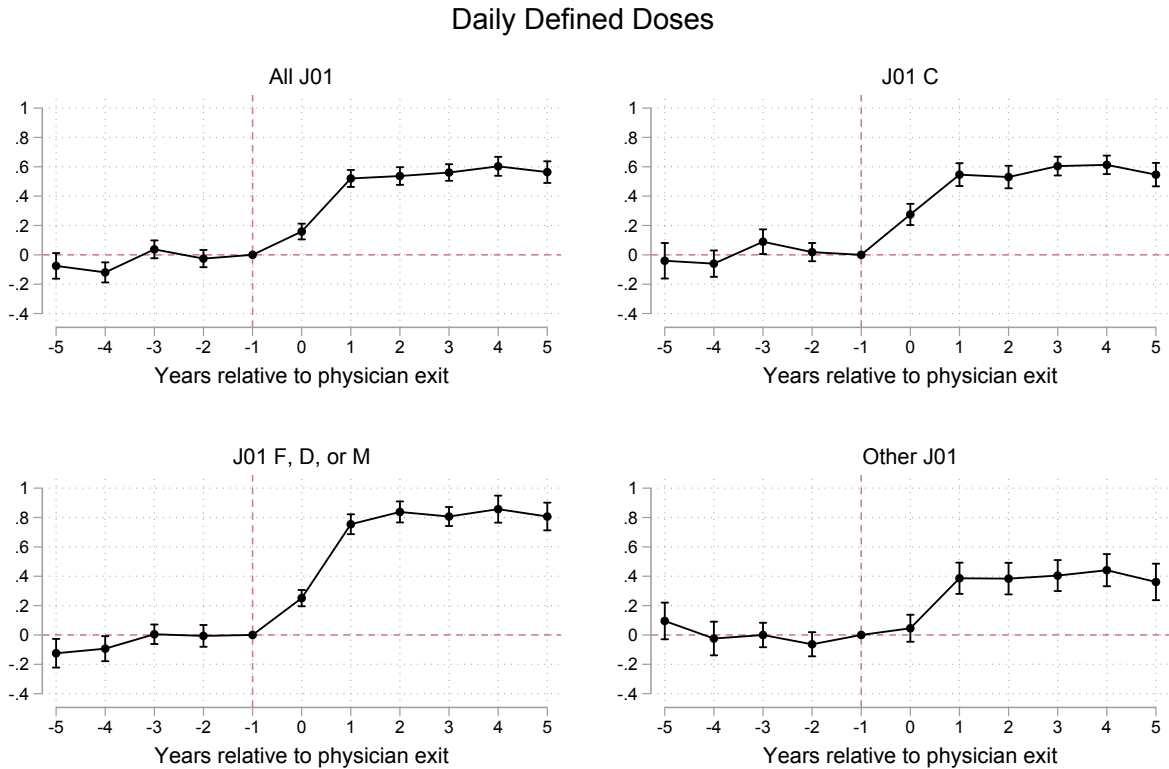
^a Two-way fixed effects estimation with calendar year fixed effects and patient fixed effects.

^b Interaction-weighted two-way fixed effects estimation includes calendar year and patient fixed effects, and interactions between relative year and cohort indicators. Averages of the relative-year specific treatment effects are formed to obtain the aggregate treatment effect (Sun and Abraham 2020).

^c Event dummies include an indicator for treatment onset (relative year $r = 0$) and post-treatment.

^d Control variables include Number of interns at the clinic, Age squared, Household size, Pregnant, Any visit to emergency department, and Any call to an emergency doctor.

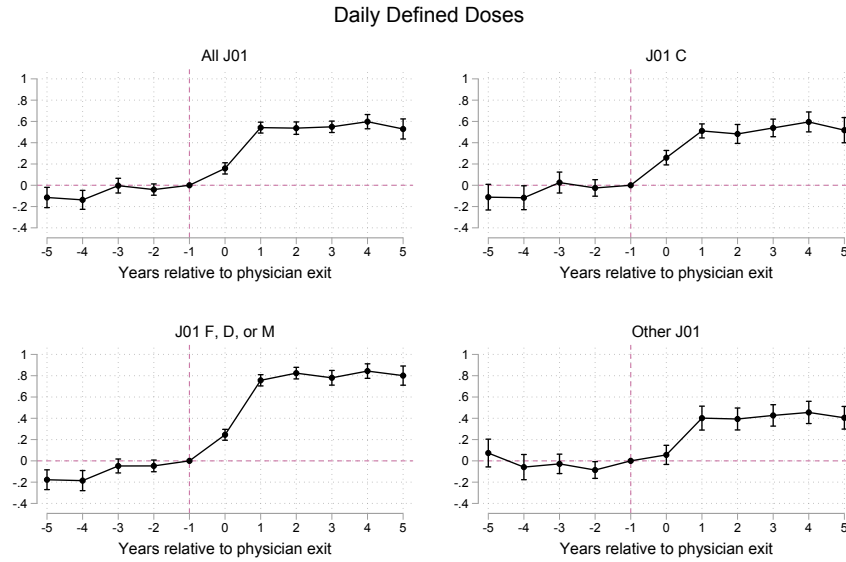
Figure 10: Event study estimates of the share of physician effects (DDD)



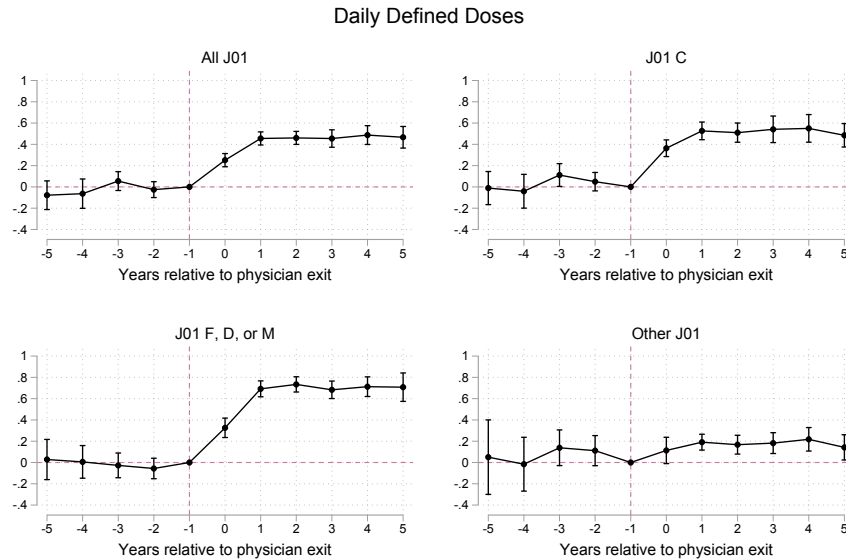
Notes: The figures display event study estimates for the share of differences in antibiotic consumption that is driven by differences in physician fixed effects. Estimations include patient fixed effects, calendar year fixed effects, and as time-varying characteristics indicators for the year of treatment onset and the post-exit period. Relative year -1 is the last pre-exit period, relative year 0 is a transitional period, and relative year 1 is the first post-exit period. Antibiotic prescribing is measured by $\log(1 + \text{Daily Defined Dose})$.

Figure 11: Event study estimates of the share of physician effects (DDD), alternative specifications

(a) Time-varying controls



(b) Sun-Abraham interaction-weighted estimation



Notes: Lines represent the 95% confidence intervals. Standard errors are calculated using a parametric bootstrap to draw clinic-level mean prescribing, with 50 repetitions at the patient level. Antibiotic prescribing is measured by $\log(1 + \text{Daily Defined Dose})$. Figure 11a displays event study estimates from estimations that include patient fixed effects, calendar year fixed effects, and indicators for treatment onset, post-exit, pregnancy, any visit to an emergency department, any call to an emergency department, any call to an emergency doctor, and as continuous variables age squared and household size. Figure 11b displays Sun-Abraham style interaction weighted estimates from fully saturated fixed effects specifications that include patient and calendar year fixed effects as well as interactions between relative period indicators and cohort indicators, where cohorts are defined by the calendar year of treatment onset. In a first step, cohort-relative year specific treatment effects are estimated. In the second step, relative year specific treatment effects are calculated as relative cohort size weighted averages by relative year.

B.3 Analyses for second-line antibiotic drugs

Table 11 shows estimation results for the share of physician effects in second-line antibiotic drugs separately for each ATC level 3 drug class: macrolides, lincosamides, and streptogramins (J01 F), cephalosporins (J01 D), and quinolones (J01 M). Columns (1) to (3) of Table 11 show baseline estimation results using as outcomes the log number of antibiotic prescriptions, Columns (4) to (6) show estimation results when we allow for time-varying control variables, and Column (7) to (9) show estimation results when we measure prescribing by Daily Defined Dose. The results indicate that the substantial share of physician effects from analyzing these classes collectively are driven by the group of macrolides, lincosamides, and streptogramins (J01 F), and the group of quinolones (J01 M). In contrast, physician effects are much smaller in the group of cephalosporins (J01 D).

Table 12 shows estimation results for the share of physician effects in broad-spectrum antibiotic drugs. Specifically, this analysis includes all macrolides, lincosamides, and streptogramins (J01 F), cephalosporins (J01 D), and quinolones (J01 M), but excludes erythromycin. Broad-spectrum antibiotic drugs are active against a large range of bacterial groups. However, their excessive consumption can disrupt the native bacterial flora and enable multidrug resistances. Physicians are therefore in general advised to avoid prescribing broad-spectrum antibiotics.²⁹ The results show that the share of physician effects remain large in broad-spectrum antibiotic prescriptions. The categorization into broad- and narrow-spectrum classes is not fixed.³⁰ For example, in the ESAC framework macrolides, lincosamides, and streptogramins, excluding erythromycin (J01 F, D, M, excluding J01 FA01), are considered broad-spectrum antibiotics,³¹ whereas macrolides, lincosamides, streptogramins (J01 F) are not considered broad-spectrum antibiotics by the Danish Health Data Authority (<https://medstat.dk/en>).

²⁹See Levy, Stuart B. 1998. "The Challenge of Antibiotic Resistance." *Scientific American*, 278(3): 46–53.

³⁰See Acar, Jacques. 1997. "Broad- and Narrow-Spectrum Antibiotics: An Unhelpful Categorization." *Clinical Microbiology and Infection*, 3(4): 395–396.

³¹See ECDC (European Centre for Disease Prevention and Control), EFSA BIOHAZ Panel (European Food Safety Authority Panel on Biological Hazards) and CVMP (EMA Committee for Medicinal Products for Veterinary Use), 2017. "ECDC, EFSA and EMA Joint Scientific Opinion on a List of Outcome Indicators as Regards Surveillance of Antimicrobial Resistance and Antimicrobial Consumption in Humans and Food-Producing Animals." *EFSA Journal* 2017, 15(10):5017, 70 pp.

Table 11: Estimation results for the share of physician effects in antibiotic prescribing

Panel A	Number of prescriptions		
	Two-way fixed effects estimation ^a		
	J01 F	J01 D	J01 M
$\hat{\Delta}_i \times D_{it}$	0.834*** (0.03)	0.275*** (0.089)	0.630*** (0.057)
Event dummies ^b	yes	yes	yes
Time-varying controls ^c	no	no	no
Observations	7,796,767	7,796,767	7,796,767
Groups (patients)	1,373,109	1,373,109	1,373,109
Panel B	Number of prescriptions^d		
	Two-way fixed effects estimation ^a		
	J01 F	J01 D	J01 M
$\hat{\Delta}_i \times D_{it}$	0.837*** (0.03)	0.288*** (0.091)	0.662*** (0.059)
Event dummies ^b	yes	yes	yes
Time-varying controls ^c	yes	yes	yes
Observations	7,653,853	7,653,853	7,653,853
Groups (patients)	1,346,414	1,346,414	1,346,414
Panel C	Daily Defined Dose		
	Two-way fixed effects estimation ^a		
	J01 F	J01 D	J01 M
$\hat{\Delta}_i \times D_{it}$	0.812*** (0.03)	0.244*** (0.104)	0.668*** (0.056)
Event dummies ^b	yes	yes	yes
Time-varying controls ^c	no	no	no
Observations	7,796,767	7,796,767	7,796,767
Groups (patients)	1,373,109	1,373,109	1,373,109

Notes: This table reports the average share of between-clinics difference in antibiotic prescribing attributable to physician effects, the coefficient of $\Delta_i \times D_{it}$. Δ_i denotes the difference in mean prescribing between patient i 's assigned sets of physicians and is estimated by $\hat{\Delta}_i$, the average prescribing to untreated patients. D_{it} denotes a post-treatment indicator. Antibiotic prescribing is measured by $\log(1 + \text{number of antibiotic prescriptions})$ (*Number of prescriptions*) or $\log(1 + \text{Daily Defined Dose})$ (*Daily Defined Dose*). Standard errors are calculated using a parametric bootstrap to draw clinic-level mean prescribing, with 50 repetitions at the patient level. *** $p < 0.01$.

^a Two-way fixed effects estimation with calendar year fixed effects and patient fixed effects.

^b Event dummies include an indicator for treatment onset (relative year $r = 0$) and post-treatment.

^c Control variables include Number of interns at the clinic, Age squared, Household size, Pregnant, Any visit to emergency department, and Any call to an emergency doctor.

Table 12: Estimation results for the share of physician effects in antibiotic prescribing

Panel A	Number of prescriptions	
	Two-way fixed effects estimation ^a	
	J01 F, D, M, excl. J01 FA01 (1)	J01 F, D, M, excl. J01 FA01 (2)
$\hat{\Delta}_i \times D_{it}$	0.813*** (0.03)	0.827*** (0.032)
Event dummies ^b	yes	yes
Time-varying controls ^c	no	yes
Observations	7,796,767	7,653,853
Groups (patients)	1,373,109	1,346,414
Panel B	Daily Defined Dose	
	Two-way fixed effects estimation ^c	
	J01 F, D, M, excl. J01 FA01 (1)	J01 F, D, M, excl. J01 FA01 (2)
$\hat{\Delta}_i \times D_{it}$	0.793*** (0.029)	0.806*** (0.032)
Event dummies ^a	yes	yes
Time-varying controls ^b	no	yes
Observations	7,796,767	7,653,853
Groups (patients)	1,373,109	1,346,414

Notes: This table reports the average share of between-clinics difference in antibiotic prescribing attributable to physician effects, the coefficient of $\Delta_i \times D_{it}$. Δ_i denotes the difference in mean prescribing between patient i 's assigned sets of physicians and is estimated by $\hat{\Delta}_i$, the average prescribing to untreated patients. D_{it} denotes a post-treatment indicator. Antibiotic prescribing is measured by $\log(1 + \text{number of antibiotic prescriptions})$ (*Number of prescriptions*) or $\log(1 + \text{Daily Defined Dose})$ (*Daily Defined Dose*). Standard errors are calculated using a parametric bootstrap to draw clinic-level mean prescribing, with 50 repetitions at the patient level. *** $p < 0.01$.

^a Two-way fixed effects estimation with calendar year fixed effects and patient fixed effects.

^b Event dummies include an indicator for treatment onset (relative year $r = 0$) and post-treatment.

^c Control variables include Number of interns at the clinic, Age squared, Household size, Pregnant, Any visit to emergency department, and Any call to an emergency doctor.

C Estimation of practice style correlates

C.1 Estimation details

We estimate the association between prescribing practice styles and observable physician characteristics in two steps. In the first step, we obtain the pair-specific differences in practice styles by estimating the following equation:

$$y_{it} = \tilde{\alpha}_i + (\delta_{j'} - \delta_j) \times D_{it} \times I_{j(i,t < r_0(i))=j, j(i,t > r_0(i))=j'} + x_{it}\beta + \nu_{it}, \quad (7)$$

where $\tilde{\alpha}_i, \beta$ are parameters and D_{it}, x_{it} are variables as defined above. ν_{it} is the error term. $r_0(i)$ denotes the calendar year in which patient i is exposed to a physician exit. $I_{j(i,t < r_0(i))=j, j(i,t > r_0(i))=j'}$ is an indicator which is one if a treated patient i is assigned to the set of physicians j before the physician exit occurs in $t = r_0(i)$ and to a different set of physicians j' after the physician exit has occurred. $\delta_{j'} - \delta_j$ denote the difference in prescribing practice styles between the two sets of physicians j' and j . Estimating Equation (7) fully specified in all possible pairs of sets of physicians $\{j, j'\}$ yields estimates for the pair-specific differences in practice styles. Figure 12 shows histograms of all estimated pair-specific differences in practice styles.

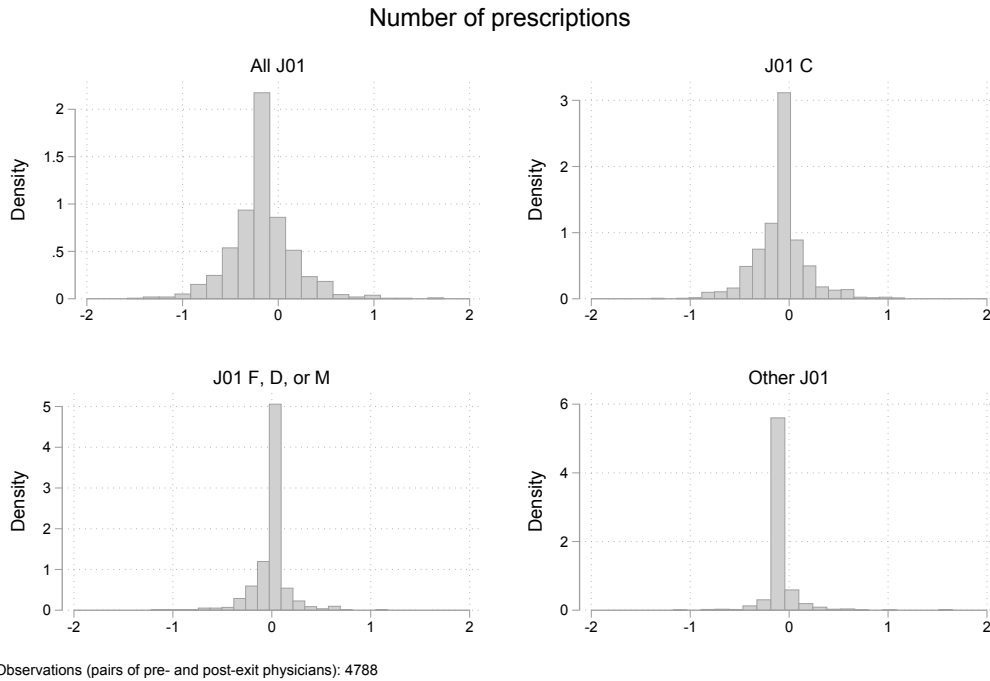
Note that our empirical strategy only allows identification of practice style differences. We therefore also construct pair-wise differences in physician observables when estimating the correlates of practice style differences. For each unique set of physicians, we construct the average over years for each observable characteristic. We then standardize each variable to have mean 0 and standard deviation 1. To obtain the covariates for our second-step regressions, we take the pair-wise difference in the standardized and average observable characteristics for each pair of sets of physicians.

In the second step, we perform either bivariate OLS regressions or multivariate post-LASSO OLS regressions. For the bivariate specifications, we regress the difference in practice styles on the differences in standardized physician characteristics. For the post-LASSO specifications, we regress the difference in practice styles on the differences in all standardized physician characteristics that have been selected by a first-step LASSO regression. The unit of observation is a pair of sets of physicians to which at least one treated patient is assigned.

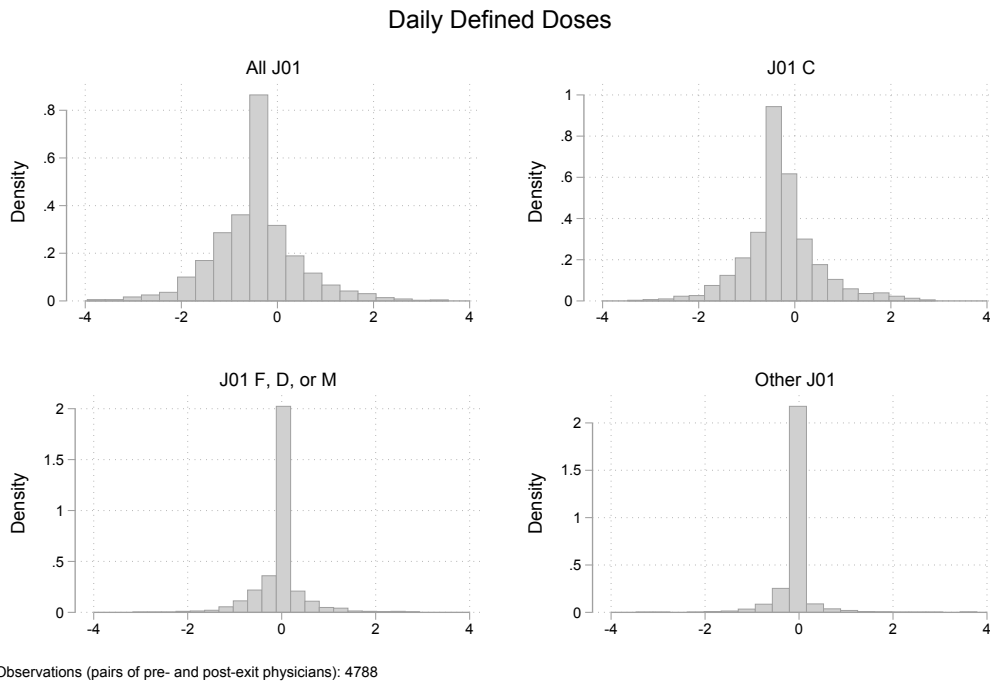
To obtain standard errors we perform a parametric bootstrap with 50 repetitions. In each repetition, we draw the difference in practice styles for each pair of sets of physicians $\{j, j'\}$ from a normal distribution with mean $\widehat{\delta_{j'} - \delta_j}$ and standard deviation $se(\widehat{\delta_{j'} - \delta_j})$ estimated from our first-step estimation of practice style differences, where se denotes the standard error. The bootstrapped standard errors account for estimation error from our first step estimation.

Figure 12: Histograms of differences in antibiotic prescribing practice styles between pairs of sets of physicians

(a) Log number of prescriptions



(b) Log Daily Defined Dose



Notes: The figures show pairwise differences in practice styles between pre- and post-exit physicians. Pairwise differences in practice styles correspond to treatment effects from our main analysis, estimated separately for all pairs of pre- and post-physicians treated patients are assigned to. Antibiotic prescribing is measured by $\log(1 + \text{number of prescriptions})$. Values are bunched for groups of five patients with similar estimated mean difference in average prescribing due to data anonymization.

C.2 Physician characteristics

Below we describe how the variables in our practices style correlates analysis are defined.

Physician personal characteristics. We construct physician individual-level characteristics and aggregate them over all physicians in a given set of physicians. We have to aggregate the individual-level characteristics as we can only observe the identity of the clinic in a given year for each prescription, but not the identity of the prescribing physician.

As physician personal characteristics we consider for all given sets of physicians the average age, the average household size, the share of general practitioners living in single households, the share of general practitioners with a PhD degree, the share of female general practitioners, and the share of general practitioners with migration backgrounds. We separate migration backgrounds by Nordic origin country (Finland, Iceland, Norway and Sweden), non-Nordic origin country, and second generation migration background.

Clinic-level characteristics. We further include a set of variables to describe diagnostic availability and personnel size at a general practice clinic. From claims data, we construct dummy variables that indicate whether microscopy and cultivation were available. We assume that either diagnostic method was available in a given year if any of the corresponding claim code were used at least once in a given year.³² We also impute diagnostic methods as available if both in the previous and the following year any of the corresponding claim codes have been used. To describe personnel size, we include the maximum number of general practitioners, the number of unique patients per general practitioner, and the maximum number of short-term medical staff working at the same time in a clinic in a year. We construct the number of unique patients at a clinic as the total number of unique social security numbers in a clinic's claims records. The number of short-term medical staff covers all recorded stays of up to a year. We refer to those short-term medical staff as interns.

C.3 Correlates with antibiotic prescribing practice style differences measured by Daily Defined Dose

Figure 13 repeats the same regressions as in the main analyses with differences in antibiotic prescribing practice styles measured by $\log(1 + \text{Daily Defined Dose})$ instead of $\log(1 + \text{number of antibiotic prescriptions})$. The results remain similar to our main results.

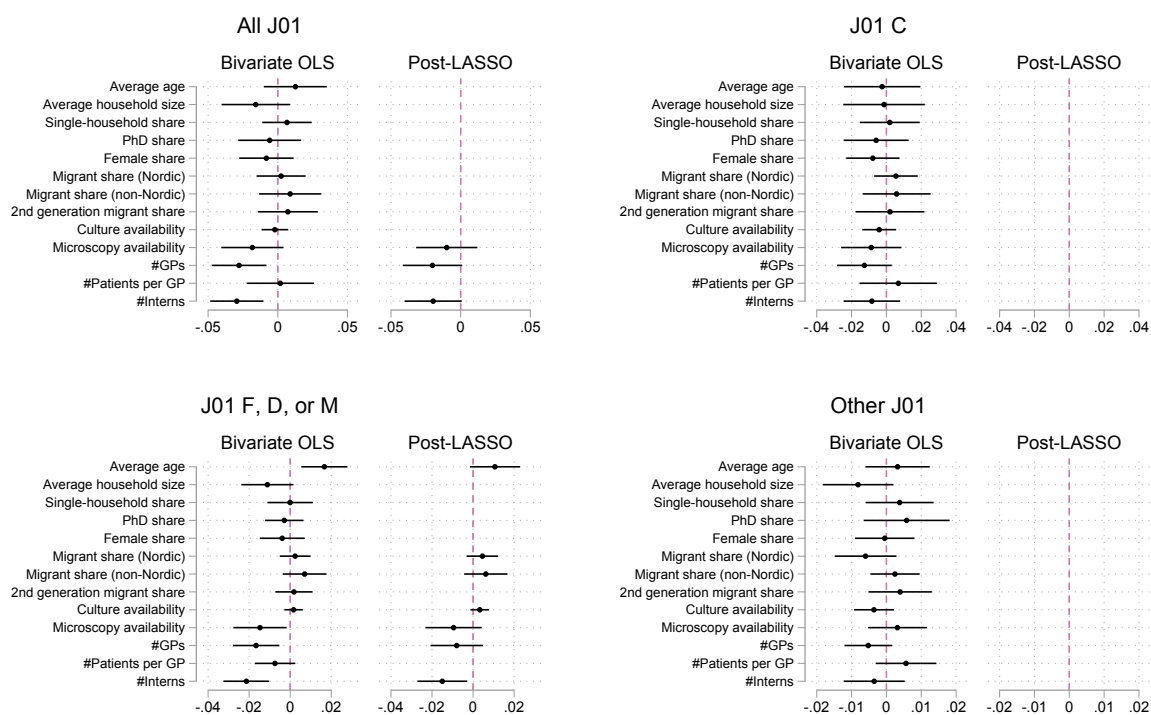
C.4 Correlates with practice style differences estimated by fixed effects

Figure 14 shows coefficients estimates from bivariate fixed effects regressions, with fixed effects for the initial pre-exit set of physicians that treated patients are assigned to. The fixed effects regressions rely on variation in the difference in physician characteristics that result from patients being assigned to different destination set of physicians after being exposed to an exit at the same initial set of physicians. The results are similar to our main results.

³²For microscopy availability, we consider the codes 802113, 807102, 807103, 807104, 807122, 807123, 807124, 807169, 808156, 808157, 808158, 808165, 808166, and 808167. For culture availability, we consider the codes 802133, 807105, 807106, 807107, 807179, and 808152.

Figure 13: Correlates of practice style differences (DDD)

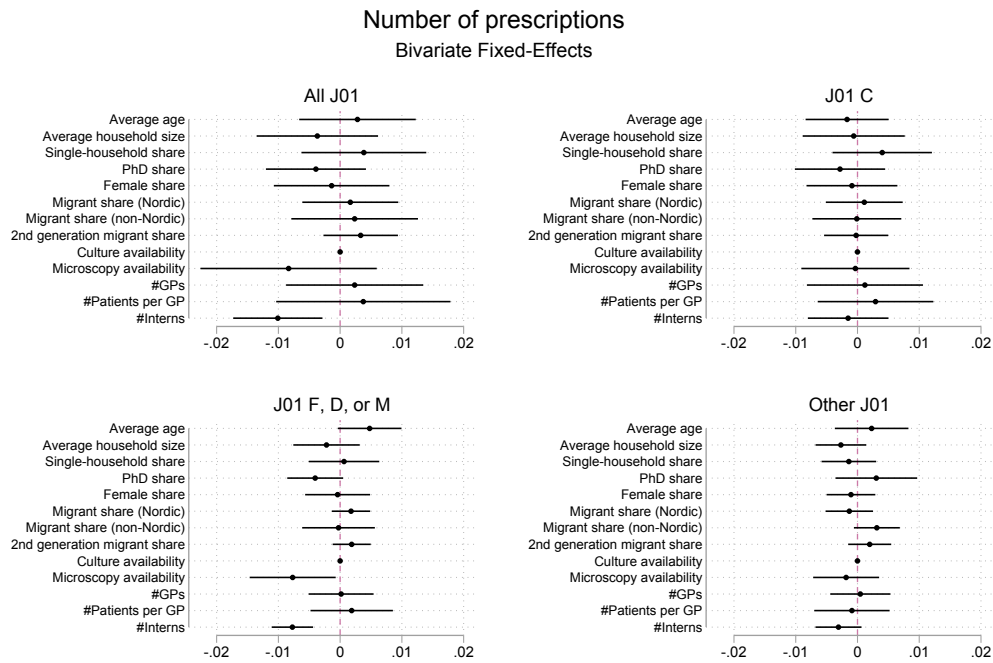
Daily Defined Doses



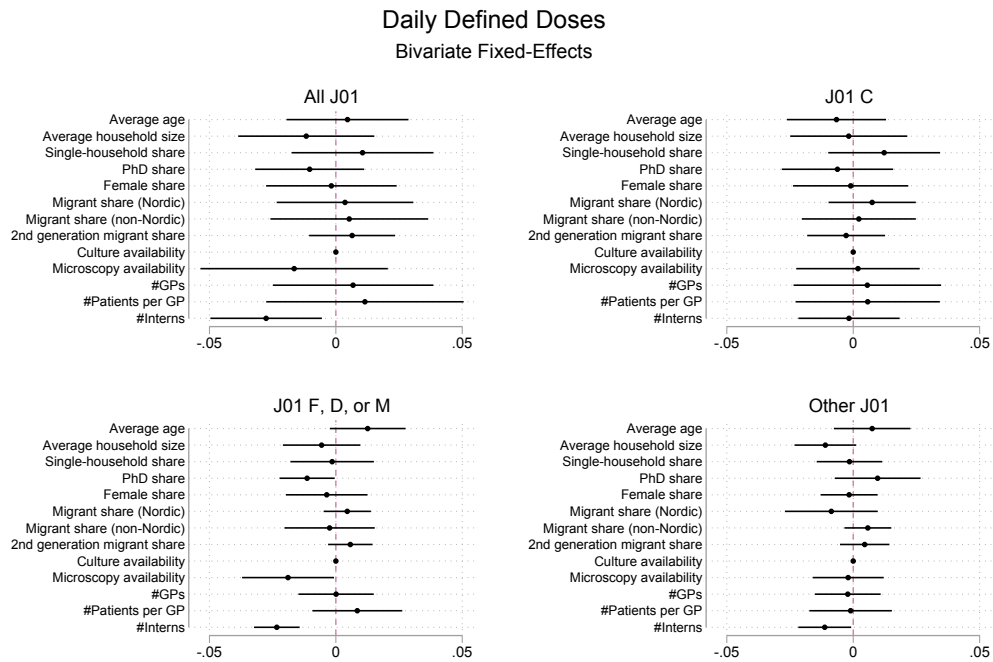
Notes: The figure displays bivariate OLS (left panels) and post-LASSO (right panels) regression results of the estimated difference in prescribing practice style, measured by $\log(1 + \text{number of antibiotic prescriptions})$, on differences in physician characteristics between sets of pre- and post-exit physicians that treated patients are assigned to. To obtain post-LASSO estimates, we run a LASSO regression on the full set of physician characteristics, with the penalty level chosen by 10-fold cross validation to minimize mean squared error. We subsequently run an OLS regression with the estimated differences in practice styles regressed on the set of physician characteristics selected by the LASSO regression. Missing coefficients indicate that a variable has not been selected in the LASSO regression. Standard errors are calculated using a parametric bootstrap to draw differences in practices styles with 50 repetitions. All physician characteristics have been standardized to have mean 0 and standard deviation 1 prior to differencing.

Figure 14: Correlates of practice style differences

(a) Bivariate fixed effects estimation (Log prescriptions)



(b) Bivariate fixed effects estimation (Log DDD)



Notes: The figure displays bivariate fixed effects regression results of the estimated difference in prescribing practice style, measured by $\log(1 + \text{number of antibiotic prescriptions})$, on differences in physician characteristics between sets of pre- and post-exit physicians that treated patients are assigned to and fixed effects for the pre-exit physicians. Standard errors are calculated using a parametric bootstrap to draw differences in practices styles with 50 repetitions. All physician characteristics are standardized to have mean 0 and standard deviation 1 prior to differencing.

D Effects of practice style differences on patient health

D.1 Ambulatory care sensitive conditions

We identify ambulatory care sensitive conditions by the definitions provided in.³³ We restrict the analysis to conditions that are both frequently caused by infectious agents including bacteria, and commonly encountered in general practice: cellulitis, ear, nose and throat infections, perforated or bleeding ulcer, urinary tract infection, and pneumonia. Table 13 shows means and standard deviations for hospitalizations due to infection-related ambulatory care sensitive conditions. Table 14 lists the conditions including sub-categories and their corresponding ICD-10 codes.

Table 13: Descriptive statistics of hospitalizations due to ambulatory care sensitive conditions, patient-year level observations

	Any hospitalization		Number of hospitalizations	
	Mean	St. dev.	Mean	St. dev.
All infection-related condition	0.0051	(0.072)	0.0061	(0.091)
Cellulitis	0.0014	(0.037)	0.0017	(0.049)
Ear, nose, and throat infections	0.0011	(0.033)	0.0013	(0.040)
Perforated or bleeding ulcer	0.0005	(0.022)	0.0006	(0.029)
Urinary tract infection or pyelonephritis	0.0013	(0.036)	0.0015	(0.046)
Pneumonia	0.0009	(0.031)	0.0010	(0.035)
Observations (patient-years)	7,796,767		7,796,767	

³³See Bardsley, Martin, Ian Blunt, Sian Davies, and Jennifer Dixon. 2013. "Is Secondary Preventive Care Improving? Observational Study of 10-Year Trends in Emergency Admissions for Conditions Amenable to Ambulatory Care." *BMJ Open*, 3(1).

Table 14: List of ICD-10 codes for infection-related ambulatory care sensitive conditions

ICD-10 code	Category
Cellulitis	
L03	Cellulitis
L04	Acute lymphadenitis
L08	Other local infections of skin and subcutaneous tissue
L88	Pyoderma gangrenosum
L980	Pyogenic granuloma
L983	Eosinophilic cellulitis
Ear, nose and throat infections	
H66	Otitis media, unspecified
H67	Otitis media in diseases classified elsewhere
J02	Acute pharyngitis
J03	Acute tonsillitis
J06	Acute upper respiratory infections of multiple and unspecified sites
J312	Chronic pharyngitis
Perforated/bleeding ulcer	
K250-K252	Gastric ulcer
K254-K256	
K260-K262	Duodenal ulcer
K264-K266	
K270-K272	Peptic ulcer, site unspecified
K274-K276	
K280-K282	Gastrojejunal ulcer
K284-K286	
Urinary tract infection/Pyelonephritis	
N10	Acute tubulo-interstitial nephritis
N11	Chronic tubulo-interstitial nephritis
N12	Tubulo-interstitial nephritis, not specified as acute or chronic
N136	Pyonephrosis
N390	Urinary tract infection, site not specified
Pneumonia	
J13	Pneumonia due to Streptococcus pneumoniae
J14	Pneumonia due to Haemophilus influenzae
J153	Pneumonia due to streptococcus, group B
J154	Pneumonia due to other streptococci
J157	Pneumonia due to Mycoplasma pneumoniae
J159	Bacterial pneumonia, unspecified
J168	Pneumonia due to other specified infectious organisms
J181	Lobar pneumonia, unspecified
J188	Other pneumonia, organism unspecified

D.2 Alternative specifications

We estimate a number of alternative specifications to analyze the health effects of changes in antibiotic prescribing practice style.

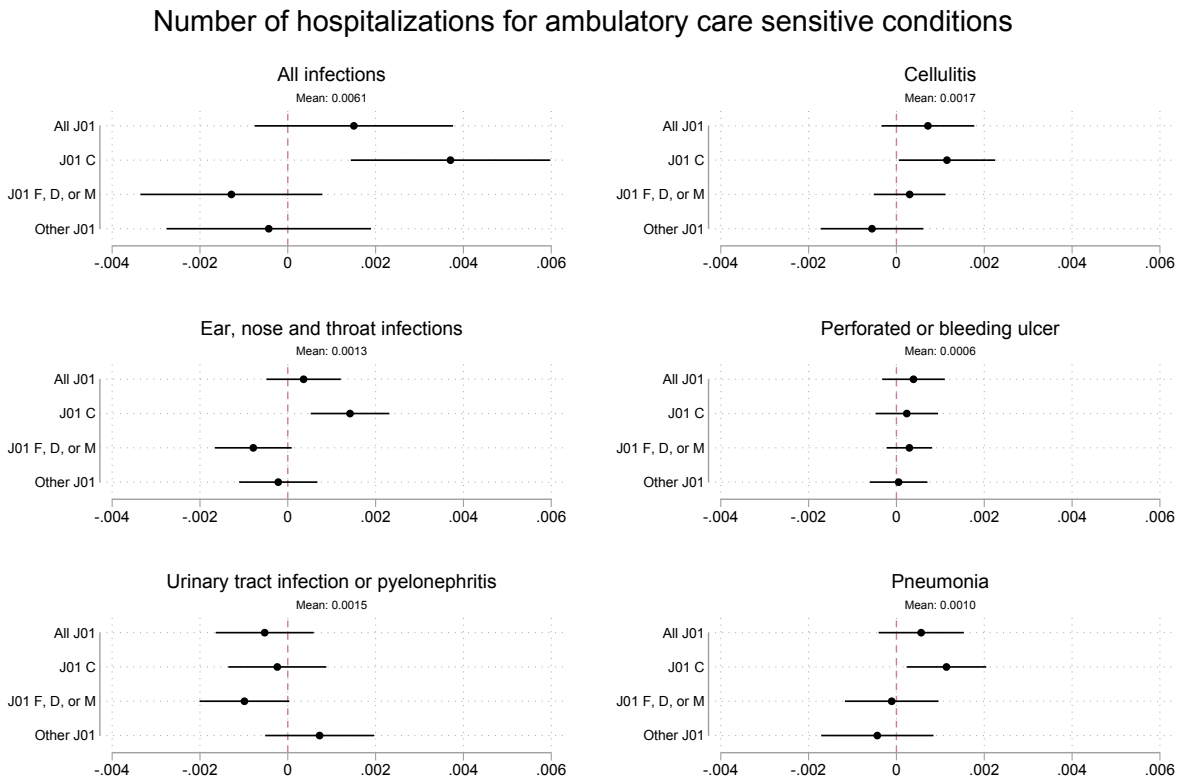
Figure 15 shows results when we define the outcome variable as the count number of hospitalizations due to ACSC. The number of hospitalizations excludes referrals from general practitioners as well as internal referrals with the same diagnostic code. The results are similar in direction and magnitude as our main results.

Figure 16 shows results for specifications in which we allow for an extended set of control variables. Results are similar as in our main specification.

Figure 17 shows results for dynamic specifications that include cohort-specific effects. We estimate the average treatment effect as a weighted aggregate of the cohort-specific effects in a Sun-Abraham-style framework. Again, for most conditions, a one-standard deviation change in prescribing style does not systematically affect the rate of hospitalization. However, in the case of hospitalizations for urinary tract infections or pyelonephritis, all point estimates are negative, indicating that an increase in prescribing practice style might lower the rate of hospitalization due to this condition. Moreover, a more intense second-line prescribing style increases the rate of hospitalization due to this condition significantly.

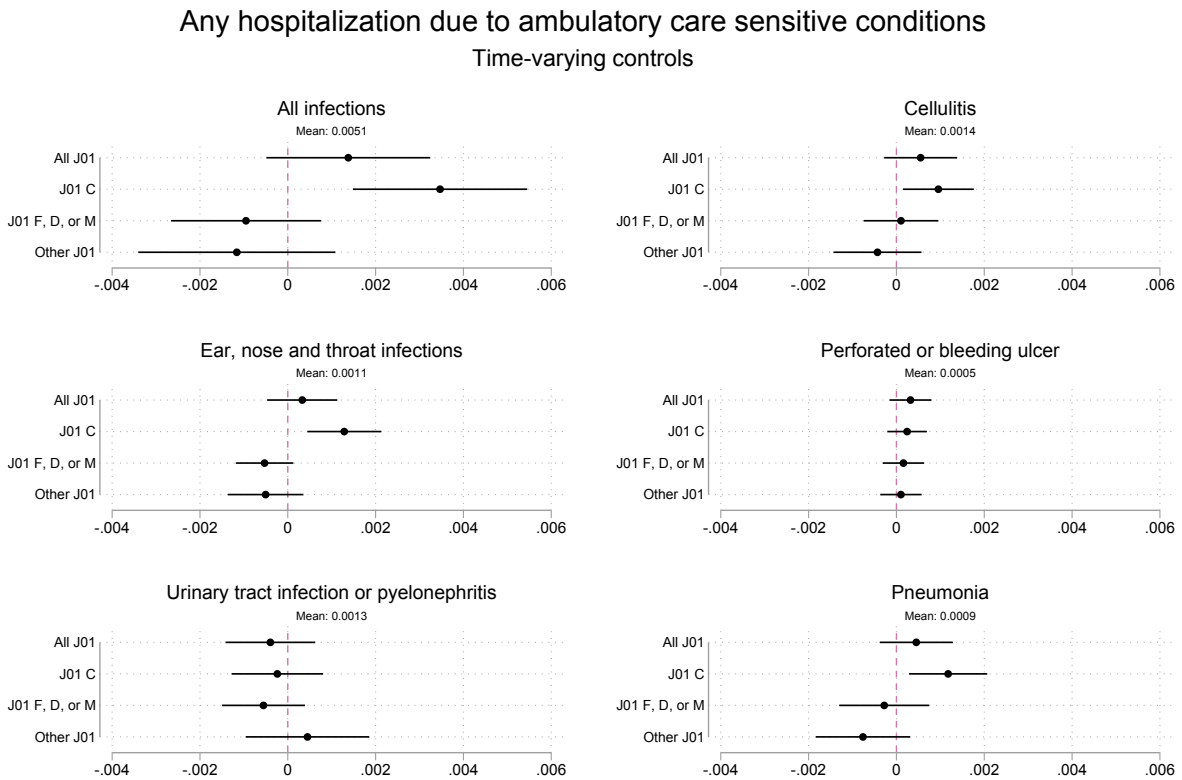
Figure 18 shows results for specifications in which we define treatment in a binary fashion. The left panels show the estimated effects of being exposed to a lower prescribing intensity. We constrain the treatment group as patients who are exposed to the lowest 25 percentile of an estimated decrease in prescribing intensity. We exclude from the sample all patients who are exposed to smaller changes or increases in prescribing intensity, such that the control group is comprised of patients unexposed to physician exit. The right panels show the estimated effects of being exposed to the upper 25 percentile of an estimated increase in prescribing intensity and we define the sample accordingly. The estimates from these specifications cannot be interpreted in the same way as in the previous specifications as treatment is defined differently. The direction of the estimates do not generally contradict results from our prior specifications. Notably, a change in antibiotic prescribing style systematically affects hospitalizations for urinary tract infections: a decrease in prescribing style lowers the rate of hospitalization for this condition, while it increases with a higher prescribing style. Additionally, the results corroborate our main results of a positive relationship between antibiotic prescribing and hospitalizations for pneumonia, and penicillin prescribing and hospitalizations for ear, nose, and throat infections.

Figure 15: Effect of an increase in prescribing style by one standard deviation of log prescriptions on the number of hospitalizations for ambulatory care sensitive conditions



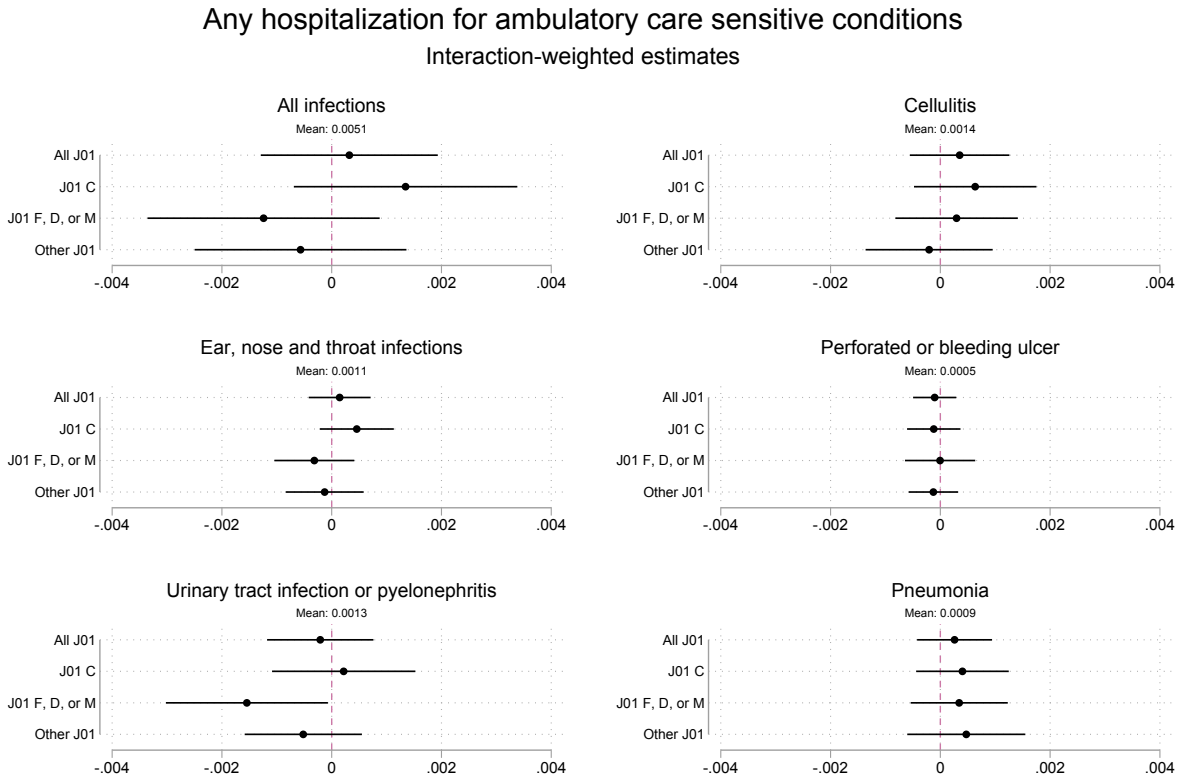
Notes: The figure displays estimation results for the effect of antibiotic prescribing practice style differences on hospitalizations for infection-related ambulatory care sensitive conditions. We obtain the effect of practice style differences as the coefficient associated with the interaction between the estimated difference in prescribing practice styles measured by $\log(1 + \text{number of antibiotic prescriptions})$, and a post-exit indicator. Estimations include patient fixed effects, calendar year fixed effects, and indicators for treatment onset and post-exit. Lines represent the 95% confidence intervals. Standard errors are calculated using a parametric bootstrap to draw practice style differences, with 50 repetitions at the patient level.

Figure 16: Effect of an increase in prescribing style by one standard deviation of log prescriptions on hospitalizations for ambulatory care sensitive conditions, allowing for time-varying control variables



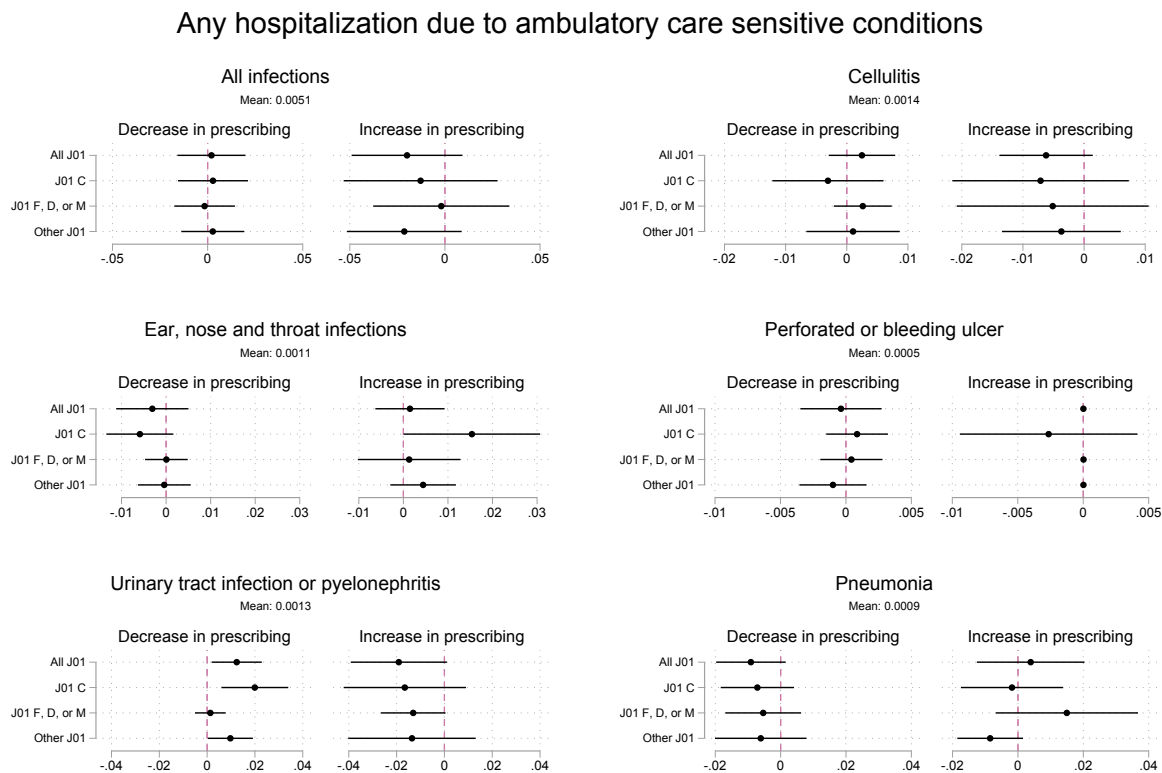
Notes: The figure displays estimation results for the effect of antibiotic prescribing practice style differences on hospitalizations for infection-related ambulatory care sensitive conditions. We obtain the effect of practice style differences as the coefficient associated with the interaction between the estimated difference in prescribing practice styles measured by $\log(1 + \text{number of antibiotic prescriptions})$, and a post-exit indicator. Estimations include patient fixed effects, calendar year fixed effects, and indicators for treatment onset, post-exit, pregnancy, and as continuous variables age squared and household size. Lines represent the 95% confidence intervals. Standard errors are calculated using a parametric bootstrap to draw practice style differences, with 50 repetitions at the patient level.

Figure 17: Cohort size and relative time weighted average effect of an increase in prescribing style by one standard deviation of log prescriptions on hospitalizations for ambulatory care sensitive conditions



Notes: The figure displays estimation results for the effect of antibiotic prescribing practice style differences on hospitalizations for infection-related ambulatory care sensitive conditions. We obtain the effect of practice style differences as the coefficient associated with the interaction between the estimated difference in prescribing practice styles measured by $\log(1 + \text{number of antibiotic prescriptions})$, and a post-exit indicator. Estimations include patient fixed effects, calendar year fixed effects, and indicators for treatment onset and post-exit. To aggregate treatment effects, we take averages of the relative-year specific treatment effects weighted by cohort-relative year size, as proposed by Sun and Abraham (2020). Lines represent the 95% confidence intervals. Standard errors are calculated using a parametric bootstrap to draw practice style differences, with 50 repetitions at the patient level.

Figure 18: Effect of an overall decrease or increase in prescribing practice style on the number of hospitalizations due to ambulatory care sensitive conditions



Notes: The figure displays estimation results for the effect of antibiotic prescribing practice style differences on hospitalizations for infection-related ambulatory care sensitive conditions. We obtain the effect of a decrease in prescribing style as the coefficient associated with the interaction between an indicator for being exposed to the lowest quartile of an estimated decrease in prescribing practice styles measured by $\log(1 + \text{number of antibiotic prescriptions})$, and a post-exit indicator. We exclude treated patients exposed to increases in estimated prescribing styles in each specification. We proceed conversely in order to obtain the effect of an increase in prescribing style. Estimations include patient fixed effects, calendar year fixed effects, and indicators for treatment onset and post-exit. Lines represent the 95% confidence intervals. Standard errors are calculated using a parametric bootstrap to draw practice style differences, with 50 repetitions at the patient level.