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The Impact of Physician Preferences on the Prescription of New Drugs

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Abstract

We study the role of the physician in the choice of drugs for patients with high blood pressure (hypertension) and ulcers. A prescription micro dataset from a small Swedish municipality where both patients and physicians can be followed over time is used. We test if the choice of drug is independent of the physician using a mixed logit estimator that allows us to control for the drug the patient has received in the past. The probability of prescribing a particular drug varies between physicians. Seeing a physician that has prescribed 10 percentage units more of a specific drug in the past, increases the probability of receiving that drug by 3 percentage units for a new hypertension patient and 6.5 percentage units for a new ulcer patient. Female physicians are less likely to prescribe new drugs than male physicians. The lower bound of the welfare loss of physician induced variations in drug prescriptions for new patients is 1% of drug expenditures for hypertension and 11% of drug expenditures for ulcers.

Keywords: pharmaceuticals, physician behavior, medical practice variations, practice style, mixed logit estimator.

JEL: C25, I11, L10, O33.

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1 Introduction

The expenditure on pharmaceuticals is increasing rapidly in many countries. An important explanation for the increased expenditure is the switch from old to new drugs (Gerdtham et al. 1998). According to a study by Gerdtham et al. (1998) real pharmaceutical expenditure in Sweden would have increased by only 15% rather than the observed 50% between 1990 and 1995, without this shift. An interesting question is whether the increased expenditures are well motivated. Does the switch from old and less expensive drugs to new and more expensive drugs represent genuine welfare improvements? The reason the answer to this question is not obvious lies in the special institutional arrangements on the market for pharmaceuticals: In this market the consumer is neither the sole decision maker nor responsible for a large share of the costs. To evaluate the welfare effects of pharmaceutical innovation it is crucial to understand how physician prescription decisions are made.

Yet little is known about physician prescription behavior, with the exception of some recent studies. Hellerstein (1998) and Coscelli (2000) found effects of habit persistence of physicians in the choice between prescribing generic and branded drug versions. Lundin (2000) found that the physicians take into account costs borne by the patients to a larger extent than costs reimbursed by the state, and Stern and Trajtenberg (1998) found that more concentrated prescribers are more likely to prescribe drugs with high levels of advertising, low prices and high (lagged) market shares.

From a welfare perspective it is important to disentangle the effect of the physician from the effect of the patient in the prescription decisions. If we, for instance, observe a variation in the prescription pattern between physicians, the welfare implications of these variations depend on if the patients or the physicians cause them. If the patients cause them they may represent

legitimate variations due to variations in patient preferences or patient mix between physicians. If the physicians, on the other hand, cause them, the variations lead to welfare losses. Because, if the likelihood of receiving a particular drug for a patient varies between physicians, not all physicians are adopting an optimal prescription pattern. The larger the physician induced variations, the larger the welfare losses. In the health services research field there is a large literature on the variation in the use of various medical interventions across geographical areas (Wennberg and Gittelsohn 1973, 1982; Phelps and Parente 1990; Phelps and Mooney 1992). One of the main hypotheses to explain these small area variations, the physician practice style hypothesis, argues that the variations are caused by physician uncertainty about the effects of medical interventions (Wennberg 1984). This explanation has also been used to generate estimates of welfare losses of small area variations (Phelps and Parente 1990; Phelps and Mooney 1992). A limitation of this literature, however, is that it is based on aggregate data, making it difficult to test if the variations are actually caused by the physicians.

The aim of this study is to test the importance of the physician in choice of prescription drugs. We use individual level data for two common diseases: ulcers and high blood pressure (hypertension). The data set is based on all drugs dispensed from the two pharmacies in a small Swedish municipality during 1988-1995. In the data set we can track both physicians and patients over time. The ability to follow both is of crucial importance to be able to fully control for patient heterogeneity. Which drug is prescribed today for a patient is likely to depend on which drug the patient has received in the past, and this temporal persistence in drug choice can be controlled for by following patients over time. We use the mixed logit estimator (see McFadden and Train (1997), Brownstone and Train (1999) and Revelt and Train (1998)), a

so-called simulation estimator, to estimate a dynamic discrete choice model. This estimator allows us to include a lagged dependent variable in our model and to drop the restrictive assumption of independence of irrelevant alternatives in the standard multinomial logit model.

We test if the choice of drug is independent of the physician. As a measure of physician preferences we use the fraction of the different drugs prescribed by the physician in the past (controlling for differences in patient mix between physicians). For both hypertension and ulcers we can reject the null hypothesis that the choice of drug is independent of the physician. Seeing a physician that has prescribed 10 percentage units more of a specific drug in the past, increases the probability of receiving that drug by 3 percentage units for a new hypertension patient and 6.5 percentage units for a new ulcer patient. For repeated prescriptions to the same patient the effect of the physician is statistically significant but much smaller, because the patient typically receives the same drug as prescribed last time. We also test if physician prescription behavior varies with observable physician characteristics (gender of the physician, year of graduation from medical school, and prescription volume). The only significant physician characteristic is the gender of the physician. Female physicians are less likely to prescribe new drugs than male physicians.

To assess the policy importance of the variations in drug prescriptions, we do some tentative estimations of the welfare losses associated with physician induced variations in drug prescriptions within a small geographical area (Phelps and Parente 1990). The lower bound of the welfare loss for hypertension is estimated to 1.0% of drug expenditure for new patients and 0.2% for repeated prescriptions. For ulcers the lower bound of the welfare loss is 11.4% of drug expenditure for new patients and 0.9% for repeated prescriptions.

The paper is organized as follows. In Section 2 we give a brief background to the problem at hand. The model used is specified in Section 3 and the data and variables used are described in Section 4. In Section 5 the results are presented, followed by some concluding remarks in Section 6.

2 Background

2.1 The Swedish pharmaceutical market

Before proceeding, it is important to give a brief description of the institutional arrangements on the market for pharmaceuticals in Sweden. Prices of prescription drugs are regulated in Sweden. Until 1993, pharmaceutical prices were determined in negotiations between the producers and the National Corporation of Swedish Pharmacies (a government owned company that has a monopoly on all retailing of pharmaceuticals). From 1993 a government agency, the National Social Insurance Board, has been responsible for the price regulation and negotiates with the producers. Pharmaceutical prices are set annually and are the same for all purchasers.

Another important characteristic is the high degree of third party financing of prescription drugs. The bulk of the cost of prescription drugs (about 80%) is reimbursed by the government, and financed from general taxes. At the time of our study patients paid a fixed user charge per prescription (for 3 months' use of the drug) and the remaining part of the drug cost was paid by the National Social Insurance Board (the user charge per prescription in 1988 was SEK (Swedish Kronor) 65; exchange rate 1988 \$1=SEK 6.13).¹ Due to the construction of the reimbursement system, the price faced by the patient

¹The reimbursement system has subsequently been changed and since 1997 the copayment depends on the cost of the drug up to a specific annual cost.

does not vary between drugs, giving them little incentive to take into account any price differences. Similarly, physicians do not have any pecuniary incentives to incorporate the price of drugs into prescription decisions. Physicians usually work within the County Council system, and are on salary. Hence the choice of drugs has no effect on the physician's income.

2.2 The market for hypertension drugs

Hypertension is a silent killer. Individuals typically do not experience any symptoms of elevated blood pressure, even though the pressure is damaging arterial walls in the heart, brain and other organs leading to an increased risk of coronary heart disease and stroke. Hypertension affects a large part of the adult population and it has been estimated that about 7% of the adult population in Sweden receive drug treatment for hypertension (The Swedish Council on Technology Assessment in Health Care 1995). In Sweden four major classes of drugs are used in the treatment of hypertension: diuretics, beta-blockers, ACE-inhibitors and calcium antagonists. There has been a continuous shift towards treatment with ACE-inhibitors and calcium antagonists since these drugs were introduced in the mid 1980s. In Figure 1 we show the market shares of the four different classes of hypertension drugs between 1988 and 1994 based on our data set. The market share of diuretics decreased from 57.3% to 46.8% and the market share of beta-blockers decreased from 34.4% to 25.0%. In the same time period the market share of calcium antagonists increased more than threefold from 5.0% to 16.4% and the market share of ACE-inhibitors increased about fourfold from 3.2% to 11.9%.

The shift to ACE-inhibitors and calcium antagonists occurred even though

these drugs were much more expensive. In Table 1 we show the daily cost of treatment with the four classes of drugs in our data. The cheapest drugs are diuretics, which had a daily cost of SEK 1.07 in 1988. This can be compared to the daily cost of SEK 3.03 for beta-blockers, SEK 3.80 for calcium antagonists and SEK 4.99 for ACE-inhibitors in 1988. In spite of the shift from cheaper to more expensive drugs, the real mean daily cost of hypertension treatment decreased by about 7% from 1988 to 1994. This decrease is because the cost per day of treatment decreased in real terms over time within each drug class. That the prices of existing drugs increase less than the general inflation is commonly observed on the Swedish pharmaceutical market (Gerdtham et al. 1998), and is probably due to the price regulation. Without any shift in treatment between the different drug groups the cost per day of treatment would have decreased by 32% between 1988 and 1994 rather than the observed 7%. The shift from the old drugs to the new drugs (ACE-inhibitors and calcium antagonists) in hypertension, thus clearly has had a major impact on drug spending.

2.3 The market for ulcer drugs

The market for ulcer drugs is another major market for prescription drugs. Ulcers can be divided into gastric and duodenal ulcers, and are characterized by stomach pain and reduced quality of life. If left untreated ulcers can lead to life-threatening bleedings. It has been estimated that about 2% of the population in Sweden suffer from ulcers in a year and that 10% are affected in their lifetime (Apoteket AB 1999). Ulcer drugs are also used to treat two related diagnoses: dyspepsia (stomach pain without a verified diagnosis of ulcer) and gastroesophageal reflux disease (GERD; heartburn). In this paper we consider all four diagnoses to be part of the market for ulcer drugs (but

we control for the diagnosis in the estimations).

In Sweden two major classes of drugs are used in the treatment of ulcers: H₂-antagonists and proton pump inhibitors.² The first proton pump inhibitor (omeprazole) was introduced in 1988 in Sweden, the first year of our data. As can be seen in Figure 2, where we show the market share of the proton pump inhibitors, they have taken over the market from the H₂-antagonists in our observation period. In 1988 the market share of proton pump inhibitors was 8.9% and in 1995 this market share had increased to 71.0%. As for the hypertension market this shift occurred in spite of that the proton-pump inhibitors were substantially more expensive than the H₂-antagonists. In Table 1 we show the daily cost of treatment with the two classes of drugs based on the prescriptions in our data set and the official retail prices. In 1988 the daily cost of H₂-antagonists was SEK 10.66 and the daily cost of proton pump inhibitors was SEK 28.50. The mean daily cost of ulcer treatment increased in real terms by about 49% from 1988 to 1995. Without any shift in drug treatment between 1988 and 1995 the mean daily cost of ulcer would instead have decreased by 1%. The increase in drug spending due to the shift from old drugs to new drugs is thus even more pronounced on the ulcer market than the hypertension market.

²Sucralfate (a membrane protective substance) is a third ulcer drug that is used in Sweden. However, because it was rarely prescribed in our data set (9.7% of prescriptions) we merged these observations with the H₂-antagonists to create a distinction between old drugs (H₂-antagonists and sucralfate) and new drugs (proton pump inhibitors). The "old drugs" are referred to as H₂-antagonists in the paper.

3 Model

Assume that in each time period patients get one drug prescribed. Let U_{ikt} denote the utility to patient i of having drug k prescribed on occasion t , and specify utility as:

$$U_{ikt} = X_{it}\theta_k + \mu_i. \quad (1)$$

Here, X_{it} is a vector of observed characteristics of patient i at time t , and θ_k is a corresponding vector of coefficients capturing how these patient attributes affect the patient's utility from drug k . Age, sex and dummy variables for concomitant diseases are included in X . Beside these observables, how well a drug works for a particular patient also depends on unobservable factors, captured by μ_i in the model (e.g. the tendency for a patient to suffer side-effects from a particular drug). Since this unobserved heterogeneity can play a decisive role, it is important to account for it correctly in the estimations.

If both physicians and patients were perfectly informed and economic considerations (i.e. prices) played no role, prescription decisions would be based on comparisons of utilities as specified in expression (1). A more complete model, however, incorporates three more features of the choice of drug: First, it is not the patient alone who makes the decision, but a physician who makes the decision on behalf of the patient. Physicians most probably differ in their beliefs about the relative merits of different drugs, beliefs that will influence the decision. Second, the price of drugs may also matter. Third, there is likely to be temporal persistence in the choice of drugs over time for a patient. Persistence in drug choice over time is consistent with two different patterns of patient behavior. It could be due to *state dependence*: that having a particular drug prescribed at $t - 1$ by itself makes the patient more likely to have that drug prescribed again at t . This is a habit effect. It could also be

due to *heterogeneity*: that patients differ as to what drug is most medically appropriate for them. Having the same drug prescribed as in the previous period, may simply reflect that the patient has already been matched with the best drug. To distinguish between state dependence and heterogeneity empirically is very difficult, but by including a lagged dependent variable and also allowing for unobserved heterogeneity, it is possible to control for both.³

A more complete model that incorporates these features can be specified as follows. Let U_{ijkt} denote physician j 's assessment of the utility to individual i of having drug k prescribed on occasion t , and specify this as

$$U_{ijkt} = \rho U_{ijkt-1} + \delta p_{kt} + X_{it}\theta_k + \gamma_{jkt} + \mu_i + \epsilon_{ijkt}. \quad (2)$$

Where γ_{jkt} is physician j 's assessment at time t of drug k 's *average* therapeutical qualities, p_{kt} is the price of drug k on occasion t , and where lagged utility U_{ijkt-1} is included to allow for temporal persistence. The last term, ϵ_{ijkt} , is the random disturbance, reflecting imperfect perceptions and optimization, as well as the inability of the analyst to measure exactly all the relevant variables. The inclusion of this term puts our model in a random utility framework (McFadden, 1973).

To determine which drug to prescribe the physician will compare the assessed utility (U_{ijkt}) of all drugs to treat a specific disease and prescribe the drug with the highest assessed utility. The model suggests that the likelihood of receiving a specific drug for a patient will depend on the following four factors.

1. The assessment of the physician of the average therapeutical quality of the drugs to treat that disease.

³Heckman (1981) has shown that it can be very hard to distinguish empirically between unobserved heterogeneity and state dependence. See Keane (1997) for an ambitious attempt. See Maddala (1987) for a general discussion of dynamic discrete choice models.

2. The observable patient characteristics.
3. The price of the drugs to treat that disease.
4. The drug prescribed to the patient in the previous period.

Below we will test the importance of these four factors. Our primary hypothesis is to test if the probability of receiving a drug varies between physicians depending on the physicians' perceived quality of the drug (factor 1 above). This can be viewed as a test of the so-called practice style hypothesis in the literature on small area variations in the use of medical procedures, i.e. that the variations are caused by physician uncertainty about the effects of medical interventions (Wennberg 1984; Phelps and Parente 1990). We will refer to this as a test of physician preferences. Factor 2 (patient characteristics) and 4 (the drug prescribed in the previous period), are mainly control variables in our analysis. As a secondary hypothesis we will test if the choice of drug depends on price. Due to the third party payment system for drugs in Sweden (see above), the price paid by the patient will not vary between drugs in our analysis, suggesting that there will be a moral hazard problem. Below we describe the data and variables used to estimate equation (2).

4 Data, variables and estimation

The data set we use is administered by the Department of Public Health and Caring Sciences at Uppsala University. It contains records of all pharmaceuticals dispensed from two pharmacies in the Swedish municipality of Tierp (with 20 000 inhabitants). The data set has been used to address a host of issues in the public health field. However, with the exception of the recent study by Lundin (2000) it has not previously been used in economics. In this paper we use data from the time period 1988-1994 on all prescriptions

for the treatment of hypertension and the time period 1988-1995 on all prescriptions with ulcer drugs.⁴ Patients sometimes receive more than one drug for hypertension, but these prescriptions are excluded from our data set, i.e. we study only monotherapy.

Among other things, an observation records the identity of the prescribing physician, the identity of the patient and the date when the drug was prescribed. This allows both physicians and patients to be followed over time. The number of physicians in the hypertension sample is 67 and on average they make 119.2 prescriptions. The number of patients is 1605, receiving on average 5.0 prescriptions. In the ulcers sample the number of physicians is 273 and on average they make 16.0 prescriptions.⁵ The number of patients is 1236, receiving on average 3.5 prescriptions.

The model specified in the previous section (equation 2) focuses on the case of repeated prescriptions for existing patients. However, some prescriptions in our data set will also be for new patients, i.e. the first prescription for patients who have not previously been treated for hypertension or ulcers.⁶ Because there is reason to believe that the effect of different variables

⁴The reason that the time period differs for ulcers and hypertension is that when we ordered the hypertension data set, data for 1995 was not yet available.

⁵The reason that the number of physicians is much larger for the ulcers sample, is that prescriptions by specialists from the nearby University hospital in Uppsala are included in the ulcers sample. Some of these specialists show up very few times in the data set (for 126 physicians we only have one observation and the number of physicians with at least 10 prescriptions is 50).

⁶New patients are defined as patients who did not receive any prescription for hypertension/ulcer in the first year of our data set (1988). A prescription provides a 3-month supply of the drug, and it is therefore highly unlikely that a patient who did not receive a prescription in a particular year was on medication during that year. It is, however, possible that these patients could have been treated in previous years (although in that case it seem reasonable to treat them as new patients).

differ between new and existing patients, we estimate separate models for these groups. If for instance physicians use a patient characteristic such as gender to match patients to specific drugs, this characteristic would have an effect for new patients but not for existing patients (if the matching occurs perfectly at the first prescription). Moreover, the lagged dependent variable can of course only be included in the model for repeated prescriptions. In Tables 2 (hypertension) and 3 (ulcers) descriptive statistics for the variables included are shown.

4.1 Dependent variable

The dependent variable for hypertension is the choice between the four drug classes for the treatment of hypertension: diuretics, beta-blockers, calcium antagonists and ACE-inhibitors. For ulcers, the dependent variable is the choice between H₂-antagonists and proton pump inhibitors. In the model for repeated prescriptions we also include the lagged dependent variable (U_{ijkt-1}) as an independent variable to account for temporal persistence.

4.2 Independent variables

4.2.1 Physician prescription history and physician characteristics

To test if the choice of drug is independent of the physician we include a variable for the past prescription behavior of the physician (to test the effect of γ_{jkt} in the model above). This variable is measured as the prescription history of the physician adjusted for patient mix. Our starting point is the proportion of the different drug classes prescribed by the physician in the past. For hypertension (with 4 drug classes) we use the 20 last prescriptions

by the physician, while for ulcers (with two drug classes) we use the 10 last prescriptions of the physician.⁷⁸ The prescription pattern may differ between physicians due to that the patient mix differs between physicians. To control for this we measure the previous prescription behavior of the physician as the difference between the actual fractions of the drugs prescribed in the last 20 prescriptions (10 for ulcers) and the predicted fractions. The predictions are based on the models below that include all patient characteristics.⁹ This implies that the prescription history variable measures how much the physician has deviated from the average prescription pattern for a certain group of patients. The variable essentially measures how much the prescription pattern varies between physicians, while controlling for patient mix. This variation can be caused by either patient preferences or physician preferences (or random prescription behavior). Testing if the prescription history variable has any effect on the prescribed drug can therefore be viewed as a test of if physician preferences matter, and the size of the estimated coefficient can be used to determine how much of the variation that is caused by differences in physician preferences.

⁷For some observations less than 20 previous prescriptions (10 for ulcers) can be observed in the data set. These observations are dropped from the analysis (865 observations for hypertension and 825 observations for ulcers), to avoid that the physician preference variable is measured too imprecisely.

⁸In a sensitivity analysis we tested if the results were sensitive towards basing physician preferences on a shorter or longer prescription history than the last 20 prescriptions (10 for ulcers). Decreasing the prescription history to the last 10 prescriptions (5 for ulcers) decreased the coefficient on physician preferences, suggesting that this leads to decreased precision in the measurement of the variable. Increasing the prescription history to the last 30 prescriptions (15 for ulcers) had little effect.

⁹In these predictions we include a variable for the physician prescription history, but we set this variable to the mean value for that year for all predictions.

As we will see below physician preferences have an effect on the choice of drug. It is therefore also interesting to test why physicians differ in their prescription behavior. We therefore also test if the choice of drug is affected by various physician characteristics (in these estimations the prescription history variable is not included since it is a function of the physician characteristics).¹⁰ The gender of the physician is included to test if the prescription pattern differs between male and female physicians. The year of graduation from medical school is included to test if the prescription pattern differs between physicians with more recent training and physicians with not so recent training (the former group may for instance have more updated information on the effects of different drugs because information is available more cheaply in their more recent training). Furthermore a variable for the number of prescriptions in the previous year in our data set is included. This variable is included to test if the prescription pattern differs between low and high volume doctors (the promotion of drug companies may for instance be targeted towards high volume prescribers). For ulcers it is also relatively common for the patient to see a specialist doctor at the University hospital in Uppsala (located about 60 kilometers from Tierp) rather than a general practitioner in Tierp. The patients may for instance be referred to a specialist to carry out an endoscopy (that is used as a diagnostic test to confirm an ulcer). For ulcers we therefore also include a dummy variable for prescriptions by specialists (defined as a prescription by a doctor at the department of medicine or the department of surgery at the University hospital in Uppsala).

4.2.2 Drug prices

¹⁰In these estimations all observations are included, i.e. we do not drop observations where the observable physician prescription history is less than 20 prescriptions (10 for ulcers).

We include a variable for the price of the different drugs. As mentioned earlier, drug prices are regulated in Sweden, with the same price all over the country. Prices are set annually. In the data set we have information about the trade name of each drug prescribed. Based on this information and the official retail prices we estimated the average cost per defined daily dose (DDD) of each drug class annually for the years 1988-1995 (see Table 1). The World Health Organization (WHO) recommends the DDD system for studies of drug use (WHO Collaborating Centre for Drug Statistics Methodology 1997). A DDD is defined as the average daily dose of a drug used by an adult for treatment of the main medical indication of the drug. The cost per DDD of each drug class is used as our price variable. As can be seen in Table 1 in Section 2, the relative prices of the different drugs vary over time, which gives us the necessary price variation to test if the choice of drug is independent of price. As the price paid by the patient does not vary between drug classes, relative prices will have no effect on drug prescriptions if the physician is a perfect agent for the patient (i.e. there will be a moral hazard problem).

4.2.3 Patient characteristics

We also include a number of patient characteristics that may be important for the choice of drug. Age and gender are included. For hypertension we also include three variables for concomitant diseases that can be expected to be important for the choice of drug: coronary heart disease (angina pectoris or a previous myocardial infarction), diabetes and congestive heart failure. ACE-inhibitors are often recommended for patients with diabetes and beta-blockers are often used in patients with coronary heart disease (Apoteket AB 1999). For patients with congestive heart failure diuretics and ACE-inhibitors

are standard therapy (Apoteket AB 1999).¹¹ For ulcers it is important to control for the diagnosis of the patient and we divide the patients into the following four diagnoses: gastric ulcers (baseline category in the estimations), duodenal ulcers, dyspepsia (stomach pain without a verified diagnosis of ulcers) and gastroesophageal reflux disease (GERD; heartburn).

4.2.4 Time trend

We also include a time trend, to control for the trend in prescription behavior that is not captured by our explanatory variables. The time trend is measured as the number of days elapsed since January 1, 1988.

4.3 Estimation

The model specified in (2) is a dynamic discrete choice model. There are some factors that will complicate estimation of this model. A first problem is that the inclusion of a lagged dependent variable as a regressor will induce correlation between the error term and the lagged dependent variable. To get unbiased estimates this correlation needs to be modelled. The way we deal with this problem is to adapt the mixed logit estimator (see McFadden and Train (1997), Brownstone and Train (1999) and Revelt and Train (1998)), a so-called simulation estimator (see Stern (1997) for an introduction). The mixed logit estimator generalizes standard logit by allowing coefficients to vary randomly across patients (mixed logit is sometimes called random parameters logit.) In general, the coefficient vector can be expressed as $\beta_i = \beta + \eta_i$, where β is the population mean and η_i is the stochastic

¹¹The congestive heart failure variable is not included in the model for first prescriptions, because only 14 of the subjects in this sample had congestive heart failure (leading to difficulties in estimating the coefficient for congestive heart failure).

deviation that represents the person's tastes relative to the average tastes in the population. The stochastic portion of utility, $\eta_i Z_{ijkt} + \epsilon_{ijkt}$, (where Z_{ijkt} represents all variables whose coefficients are allowed to vary in the population) is in general correlated over alternatives and time due to the common influence of η_i . This means that mixed logit does not exhibit the restrictive assumption of independence from irrelevant alternatives. The mixed logit estimator then estimates the average effect of a variable, as well as the standard deviation of the variation between persons, i.e. it estimates the parameters of the distribution of β_i : the mean, β , and the standard deviation, σ .

If the coefficient for lagged utility is one of the variables where the parameter is allowed to vary in the population, i.e. $\rho_i = \rho + v_i$, we have modelled the correlation between the error term and the lagged dependent variable. Now, conditional on the coefficient, ρ_i , the lagged dependent variable is uncorrelated with the error term. The conditional probability is therefore not contaminated by endogeneity bias. The unconditional probability is simply the integral of the conditional probability; taking this integral does not induce any correlation. So, the fact that we try to pick up unobserved heterogeneity by estimating a random-coefficients model means that μ_i in expression (2) is replaced by $\eta_i Z_{ijkt}$. What is important is to make sure that one part of the combined error term is truly *iid* extreme value, independent of β_i and Z_{ijkt} . In this specification that would be ϵ_{ijkt} . For specification of the likelihood function we refer to Revelt and Train (1998).

In the estimations we allow the coefficients of the three choice specific variables to vary randomly in the population: the physician prescription history, the price (the cost per day), and the lagged dependent variable.¹² In

¹²Allowing all coefficients to vary makes identification difficult (Ruud, 1996). In the ulcers sample for first prescriptions we cannot let any coefficients vary because we only

addition, we also allow the alternative specific intercepts to vary. This is like allowing for “traditional” individual specific effects (since they end up in the error term they are treated as random effects rather than as fixed effects). Introducing individual specific effects is done in order to pick up effects from unobservables that may influence the choice.¹³

A second problem in a dynamic model is the so-called initial conditions problem. If you do not observe the process from the beginning (i.e. from the first prescription of a newly diagnosed patient), then the conditional probability of the first observed choice depends on the previous choice, which is not observed. To deal with this problem we use a method suggested by Heckman (1981). The basic idea is to approximate the marginal probability of the initial state by a standard logit model including exogenous variables only, and using all observations available (the entire sequence for all patients). Then those approximations are used as the first value for the lagged dependent variable instead of the actual lagged value (but only for those individuals where the process cannot be observed from the beginning).

5 Results

5.1 The market for hypertension drugs

In Table 4 we show the estimation results for the market for hypertension drugs. Among the explanatory variables, in addition to variables that vary across choices, we also have some variables that vary across patients. To be

have two choice alternatives (proton pump inhibitor or H₂-antagonists), and only one equation is estimated for each patient, i.e. a logit rather than a multinomial logit.

¹³The alternative specific intercepts are only allowed to vary in the estimation for repeated prescriptions - for first prescriptions it is not possible because we only have one observation per patient.

able to estimate the effects from these they are interacted with choice specific dummy variables. This explains why we have three columns for each model.

The physician prescription history variable has a positive sign and is significant at the 10% level for first prescriptions and at the 1% level for repeated prescriptions. Increasing the prescription history variable by 10 percentage units on average increases the probability of receiving the drug by 3.0 percentage units for first prescriptions and 0.8 percentage units for repeated prescriptions.¹⁴ The reason for the lower impact on repeated prescriptions is that there is a strong temporal persistence in the drug choice (indicated by the highly significant lagged dependent variable: "prescribed last time").¹⁵ The probability of receiving the same drug as last time varies between 66% for ACE-inhibitors and 97% for diuretics, leaving little scope for the other variables to affect the choice of drug.

We cannot reject the null hypothesis that the choice of drug is unaffected by relative prices for either first prescriptions or repeated prescriptions, consistent with moral hazard. Of the individual characteristics age and sex are most important. Older age increases the probability of receiving diuretics and women are more likely to receive diuretics than men. The standard deviation is highly significant for the lagged dependent variable, indicating that these parameters do indeed vary in the population. This could either be because some patients have developed stronger habits for a drug or because they are

¹⁴The effect of the physician prescription history variable is estimated by increasing the physician prescription history by 10 percentage units for all observations in our data and taking the mean of the increased probability.

¹⁵The predicted effect of the prescription history variable is smaller for repeated prescriptions than first prescriptions even though the coefficient is greater. This is because on average the effect of the coefficient is evaluated at very high or very low probabilities due to the strong temporal persistence. The marginal effect in a logit model is non-linear and is smallest at high and low probabilities.

better matched with their drug than other patients are. The standard deviation for the physician prescription history variable is significant for repeated prescriptions, suggesting that allowing for heterogeneity is important for this variable as well. The standard deviation for the price variable is not significant, whereas the standard deviations for the alternative specific intercepts are significant for all three drugs, which indicates the existence of individual specific effects, i.e., that unobserved variables that are important for the choice of drug have been excluded in the model. This could, for instance, be some unobserved medical factor, making a certain drug particularly well suited for a patient.

It is also interesting to test if the choice of drug varies with observable physician characteristics. The result of this test is reported in Table 5. There is no significant effect of the year of graduation or the volume of prescriptions. The gender of the physician is, however, significant for repeated prescriptions for ACE-inhibitors. Female physicians are significantly less likely to switch patients from diuretics to ACE-inhibitors than male physicians. The probability of switching a patient from diuretics to ACE-inhibitors is 1.4% for male physicians and 0.9% for female physicians.

5.2 The market for ulcer drugs

The estimation results for the market for ulcer drugs are shown in Table 6. The physician prescription history variable has a positive sign and is significant at the 1% level for both first prescriptions and repeated prescriptions. An increase by 10 percentage units in the prescription history variable increases the probability of receiving a proton pump inhibitor by 6.5 percentage units for first prescriptions and 1.8 percentage units for repeated prescrip-

tions. As for hypertension there is a strong temporal persistence in the drug choice. The probability of receiving a proton pump inhibitor if it was prescribed last time is 82% and the corresponding probability of receiving a H₂-antagonist if it was prescribed last time is 73%.

Consistent with moral hazard, the price variable is not significant for first prescriptions or repeated prescriptions. Age is the only significant patient characteristic for first prescriptions, and older age increases the likelihood of receiving a proton pump inhibitor. For repeated prescriptions the diagnosis is important and the likelihood of being switched from an H₂-antagonist to a proton pump inhibitor is greatest for patients with gastroesophageal reflux disease. As for hypertension the standard deviation is highly significant for the lagged dependent variable and the intercept, suggesting heterogeneity in temporal persistence and the existence of unobserved variables that are important for the choice of drugs. For the physician prescription history variable and the price variable the standard deviation is not significant.

In table 6 we also test if observable physician characteristics can explain the variation in physician prescription behavior. The year of graduation from medical school and the volume of prescriptions for ulcer are not significant. The variable for seeing a specialist doctor is significant for both first and repeated prescriptions. Specialists are more likely to prescribe proton pump inhibitors for new patients and to shift patients from H₂-antagonists to proton pump inhibitors. The gender of the physician is significant at the 10% level for both first and repeated prescriptions. Female physicians are less likely to prescribe proton pump inhibitors to new patients and are less likely to switch patients from H₂-antagonists to proton pump inhibitors. The probability of prescribing a proton pump inhibitor for a new patient is 46.4% for male physicians and 41% for female physicians, and the probability of switching

a patient from an H₂-antagonist to a proton pump inhibitor is 31% for male physicians and 26% for female physicians.

5.3 Welfare losses: hypertension

To evaluate the welfare consequences of the variation in prescription pattern between physicians within a small geographical area we adopt the approach developed by Phelps and Mooney (1990) to estimate the welfare losses of medical practice variations. They derive a formula for the welfare loss of medical practice variations if the average use rate is the correct one.¹⁶ In that case the welfare loss is equal to:

$$WL = \frac{1}{2} \sum_{i=1, N} (X_i - \mu)^2 dP/dX \quad (3)$$

In equation (3), X_i is the rate of use of physician i , μ is the mean rate of use and dP/dX is the slope of the demand curve.¹⁷ To carry out an illustrative estimation of the welfare loss with this approach we use the same demand elasticity (-0.15) as used by Phelps and Parente (1990) and Phelps and Mooney (1992).¹⁸ Because this figure is highly uncertain we also vary it between -0.05 and -0.50 in a sensitivity analysis, to test how sensitive the results are to the demand elasticity. Equation (3) gives the welfare loss associated with the use of one medical procedure, and implicitly assumes that the alternative to using the procedure is no treatment. Our case differs

¹⁶Their approach is based on the one pioneered by Peltzman (1973) in his study of the welfare effects of the 1962 drug amendments.

¹⁷Phelps and Parente (1990) used the model to estimate welfare losses across geographical regions rather than physicians (so that the formula was indexed (i) over geographical areas rather than physicians in their estimations).

¹⁸This demand elasticity is used to estimate the slope of the demand curve (dP/dX) at the mean price in our data.

because we study which one of different alternative substitute drugs that will be used. This implies that we need to sum the welfare loss estimated with equation (3) across all the four different drugs (the four types of hypertension drugs), and then divide this sum by two. The reason that we need to divide by two is that each welfare loss will be counted twice (e.g. a 10 percentage unit higher rate of use of diuretics, implies a 10 percentage unit lower rate of use for one of the other drugs).¹⁹ All estimations should be viewed as lower bound estimates, because they assume that the average use rate (μ in equation (3)) is the correct one (because relative prices seem to have no effect on prescription choices, it seem plausible that the average use rate is too high of the newer and more expensive drugs).

We first estimate the welfare loss based on the total variation in the prescription rate. As a measure of the rate of use of different physicians we use the prescription history variable without any adjustment for patient mix (this variable shows the prescription rate of the physician at the time of the prescription).²⁰ The variation is measured as the prescription rate of the physician minus the mean prescription rate for all physicians in that year. The results are shown in Table 7. The coefficient of variation is 0.41 (the standard deviation in the rate of use divided by the mean rate of use) and the estimated welfare loss is 28.4% of drug expenditure for hypertension.²¹ Part of the overall variation will be due to patient mix. In the next step we therefore adjust the variation between physicians for patient mix. We

¹⁹This is correct as long as it is assumed that all patients always receive some drug treatment (and no treatment is not an option).

²⁰We only include the observations for which the physician prescription history variable is based on at least 20 observations (i.e. the same criterion as in the regression analysis in Table 4).

²¹The coefficient of variaton is estimated as the weighted average for the four drug classes.

use the physician prescription history variable—but this time adjusted for patient mix—as the measure of variation adjusted for patient mix. This decreases the coefficient of variation to 0.26 and the welfare loss to 11.6%.

Although we adjust for patient mix some of the remaining variation may be caused by patient preferences rather than physician preferences. As a final step we therefore use the estimated effects of the prescription history variable in the models in Table 4 to estimate how much of the variation that is caused by the physician. We predict the probability of receiving each drug based on the actual value of the prescription history variable and the mean value of the prescription history variable and measure the variation as the difference between these predictions (this is done for every prescription). We do this for all prescriptions as well as separately for first and repeated prescriptions. As can be seen in Table 7 this substantially reduces the variation. The coefficient of variation is 0.03 for repeated prescriptions and 0.08 for first prescriptions (0.04 overall), and the resulting welfare losses are 0.2% for repeated prescriptions and 1.0% for first prescriptions (0.3% overall). With a smaller demand elasticity (-0.05) the welfare losses increases to 0.6% for repeated prescriptions and 2.9% for first prescriptions (0.8% overall), and with a greater demand elasticity (-0.5) the welfare losses decreases to 0.06% for repeated prescriptions and 0.3% for first prescriptions (0.08% overall).

5.4 Welfare losses: ulcers

We carry out the same estimations for ulcers as for hypertension, also presented in Table 7.²² For the overall variation, the coefficient of variation is

²²We only include the observations when the physician prescription history variable is based on at least 10 observations (i.e. the same criterion as in the regression analysis in Table 6).

0.38 and the welfare loss is 22.7% of drug expenditure for ulcers. Adjusting for patient mix decreases the coefficient of variation to 0.36 and the welfare loss to 21.4%. Using the estimated coefficient for the prescription history variable reduces the variation further. The coefficient of variation is now 0.07 for repeated prescriptions and 0.28 for first prescriptions (0.14 overall), and the resulting welfare losses are 0.9% for repeated prescriptions and 11.4% for first prescriptions (3.3% overall). With a smaller demand elasticity (-0.05) the welfare losses increases to 2.6% for repeated prescriptions and 34.2% for first prescriptions (9.9% overall), and with a greater demand elasticity (-0.5) the welfare losses decreases to 0.3% for repeated prescriptions and 3.4% for first prescriptions (1.0% overall).

6 Concluding remarks

According to our results the choice of drug is not independent of the physician. The variable for physician prescription history was significant with the expected positive sign in both the model for initial prescriptions and the model for repeated prescriptions in both markets (hypertension and ulcer). The size of the physician effect varied between new prescriptions and repeated prescriptions and between the diseases. Seeing a physician that has prescribed 10 percentage units more of a specific drug in the past increased the probability of receiving that drug by 3 percentage units for a new hypertension patient and 6.5 percentage units for a new ulcer patient. For repeated prescriptions the effect was less strong (0.8 percentage units for hypertension and 1.8 percentage units for ulcer), due to the strong temporal persistence in the prescription pattern (i.e. a patient is likely to receive the same drug as prescribed previously to the patient).

Our work is related to the literature on medical practice variations (Wennberg and Gittelsohn 1973, 1982; Phelps and Parente 1990; Phelps and Mooney 1992). It has been argued that these variations are caused by physician uncertainty about the effects of medical interventions, leading to differences in physician preferences (Wennberg 1984). This has been difficult to test using aggregate data, but our results provide a test of this so-called practice style hypothesis using individual level data. Our results are consistent with the practice style hypothesis and confirm the importance of physician preferences as a source of medical practice variations. Not all the variation in the prescription pattern in our data was explained by physician preferences, however. In terms of the coefficients of variation in Table 7, 15.6% of the variation in hypertension prescriptions and 39.4% of the variation in ulcer prescriptions was caused by the physician (estimated as the percentage of the variation after controlling for patient mix). For new patients these fractions are higher. This suggests that part of the variation may also be explained by other factors such as patient preferences and unobserved patient characteristics. To some extent the prescription behavior of the physician may also be random, in which case that variation is also caused by the physician. Our estimates of the physician-induced variations may therefore be an underestimate. Our estimate of the fraction of variation that is physician specific can also be compared to the estimate of Hellerstein (1998), who estimated that 30% of the unexplained variation in generic versus trade-name prescriptions was physician-specific rather than patient-specific.

An important distinction of our work compared to the previous work on medical practice variations is that we studied the variations within a small geographical area rather than the variation between different geographical areas. Our study shows that even within a small geographical area there is

considerable variation in practice style between physicians, and these variations have not been taken into account in previous work on medical practice variations.²³ The coefficients of variation in our data (for the overall variation) are on the same level as for many of the procedures in the estimations by Phelps and Mooney (1992) for the variations in medical admissions and surgical procedures across areas in New York state (the coefficient of variation ranged between 0.0958 and 0.6052 in their study). A problem in the estimates by Phelps and Mooney (1992) is that the physicians may not cause all of the variations in medical procedures.

Our test of if the choice of drug is independent of the physician can be viewed as a test if drug prescriptions are optimal. The larger the physician induced variations, the larger the welfare losses. Our estimated welfare losses for hypertension were low. For ulcers the welfare losses were larger, especially for new patients (11% of drug expenditures). But the estimates are lower bound estimates, because they assume that the mean rate of use is the correct one (which is probably not true, especially because there seem to be a moral hazard problem; see below). The estimated welfare losses were also sensitive to the assumed demand elasticity, and should therefore be interpreted with caution. The reason for the smaller welfare losses for repeated prescriptions is that there is a strong temporal persistence in patient drug choice, i.e. the patient is likely to receive the same drug as at the last prescription. There is thus little scope for other factors to affect the choice of drug. This strong temporal persistence could either be due to a habit effect (i.e. state dependence), or it could be due to patients having been perfectly matched already

²³One could also argue that we underestimate the variation in drug prescriptions in our data, because we do not investigate the variation between different drugs within a class of drugs (e.g. the variation among the use of different beta-blockers).

with the best drug for them (i.e. heterogeneity). We could not distinguish between these effects in our analysis, but from a welfare perspective the distinction is important. If patients are eventually matched with the best drug, this suggests that patients that initially get the wrong drug will be switched to a better match. The welfare loss of initially prescribing the wrong drug will then only carry over to future periods until the drug is switched. If on the other hand there is a strong habit effect a patient may be stuck with the wrong drug for a long time (and once the patient has been started on the wrong drug it may not even be optimal to switch to a more appropriate drug if the cost of breaking the habit is high).

Physician induced variations in drug prescriptions may provide a rationale for regulating the prescription behavior of physicians. If regulations can reduce the variation in prescription behavior they may be welfare enhancing. Several regulations have also been implemented to constrain physician prescription behavior. In the US it is common with drug formularies in state Medicaid programs and in HMOs that restricts the doctor's choice of drugs to those on a list (a "positive formulary") or to those not on a list of excluded drugs (a "negative" formulary) (Dranove 1989; Schweitzer 1997). Some managed care plans also use physician practice profiling in which prescription patterns of individual physicians are tracked to identify high-cost prescribers and then change their prescription behavior (Schweitzer 1997). In Sweden the county councils (that are responsible for the health care in Sweden) have drug formulary committees that issue recommendations of the appropriate drugs to be prescribed for various diseases (although the county councils cannot force the physicians to follow these recommendations, and they cannot limit reimbursement on drugs deemed not to be appropriate). Although there is a potential for regulations to constrain physician prescrip-

tion behavior to reduce unnecessary variations, the costs of these regulations also needs to be taken into account to assess their overall welfare implications (see Peltzman (1973) and Dranove (1989) for such evaluations).

The results presented here suggest that the potential welfare gains of regulations are greatest for new patients and may vary between diseases. That the potential welfare gains are largest for new patients is logical, because they can be expected to have less information about different treatment options than more experienced patients, leading to a larger informational asymmetry between the patient and the physician. This also indicates that the potential gains from regulations are greater for more acute conditions (i.e. conditions with less repeat prescriptions). In our case hypertension is a more chronic disease than ulcer with more repeat prescriptions, and it is also associated with lower welfare losses. The more chronic the disease the greater will also the incentive be for patients to invest in information and knowledge about treatment options. This is a potential explanation for why the physician-induced variation (and welfare losses) was smaller for hypertension than ulcers also for new patients.

We also tested if the variation in physician prescription behavior could be explained by observable physician characteristic (graduation year from medical school, gender, and volume of prescriptions). The only significant physician characteristic was the gender of the physician (that was significant for first and repeated ulcer prescriptions and repeated hypertension prescriptions). Female physicians were less likely to prescribe new drugs and to switch patients from old drugs to new drugs than male physicians. For ulcers the probability of prescribing a proton pump inhibitor for a new patient was 46% for male physicians and 41% for female physicians, and the probability of switching a patient from an H₂-antagonist to a proton pump

inhibitor was 31% for male physicians and 26% for female physicians. Female physicians thus seem more conservative concerning the effects of new drugs compared to male physicians. For ulcers we also tested if the prescription pattern differed between specialist and non-specialist doctors and found that specialists were more likely to prescribe proton pump inhibitors for new patients and to shift patients from H₂-antagonists to proton pump inhibitors. A difficulty in interpreting this result is that specialists may see patients with more severe ulcer problems, i.e. if the ulcer is severe or worsening a patient may be referred to a specialist.

We also tested whether the choice of drug therapy is independent of price. We found no significant effect of price for either the market for hypertension drugs or the market for ulcer drugs. The lack of price sensitivity is not surprising given that neither patients nor physicians have any pecuniary incentive to take price into account on the Swedish pharmaceutical market. If physicians acted as perfect agents for society they would incorporate the cost of drugs into prescription decisions. Our results suggest that this is not the case. Overall we find that the role of the physician is important in prescription decisions, especially for new patients. However, further work is clearly needed to better understand the sources of why physicians differ in their prescription behavior and to further evaluate the welfare consequences of these differences.

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Table 1: Mean cost per day (Defined Daily Dose) of hypertension treatment and ulcer treatment in 1988 Swedish Kronor.

| Year | Hypertension | | | | | Ulcers | | |
|------|--------------|-------------|-----------|------------|---------------|---------------------|------------------------|---------------|
| | Diuretics | Beta-block. | Ca-antag. | ACE-inhib. | Weighted mean | Protone pump inhib. | H ₂ -inhib. | Weighted mean |
| 1988 | 1.07 | 3.03 | 3.80 | 4.99 | 2.00 | 28.50 | 10.66 | 12.25 |
| 1989 | 1.02 | 3.09 | 4.10 | 4.89 | 2.19 | 27.13 | 11.21 | 14.21 |
| 1990 | 0.97 | 2.89 | 4.11 | 4.60 | 2.12 | 25.52 | 10.91 | 16.13 |
| 1991 | 0.94 | 2.80 | 4.22 | 4.38 | 2.12 | 25.33 | 10.96 | 16.81 |
| 1992 | 0.92 | 2.74 | 4.24 | 4.24 | 2.27 | 24.44 | 11.24 | 17.17 |
| 1993 | 0.83 | 2.53 | 3.92 | 4.33 | 2.16 | 22.67 | 11.19 | 17.93 |
| 1994 | 0.74 | 1.83 | 3.76 | 3.68 | 1.86 | 21.39 | 11.15 | 17.65 |
| 1995 | – | – | – | – | – | 21.15 | 11.23 | 18.22 |

Table 2: Descriptive statistics - Hypertension.

| Variable | Repeated prescriptions | | | | First prescriptions | | | |
|-------------------------------------|------------------------|-------------|-----------|------------|---------------------|-------------|-----------|------------|
| | Diuretics | Beta-block. | Ca-antag. | ACE-inhib. | Diuretics | Beta-block. | Ca-antag. | ACE-inhib. |
| Market share (as number of prescr.) | 0.53 | 0.28 | 0.10 | 0.09 | 0.27 | 0.39 | 0.22 | 0.12 |
| Cost per day (SEK) | 1.08 | 3.17 | 4.77 | 5.22 | 1.08 | 3.22 | 4.90 | 5.26 |
| Physician prescription volume | | | 112.8 | | | | 10.8 | |
| Physician gender (1 = female) | | | 0.33 | | | | 0.30 | |
| Physician graduation year | | | 1985.4 | | | | 1985.8 | |
| Gender (patient; 1 = female) | | | 0.65 | | | | 0.60 | |
| Age (patient) | | | 70 | | | | 63 | |
| Diabetes | | | 0.14 | | | | 0.07 | |
| Coronary heart disease | | | 0.09 | | | | 0.06 | |
| Congestive heart failure | | | 0.09 | | | | 0.03 | |
| Number of obs. | | | 7446 | | | | 538 | |

Table 3: Descriptive statistics - Ulcers.

| Variable | Repeated prescriptions | First prescriptions |
|--|------------------------|---------------------|
| Market share;proton pump inhibitors (as number of prescr.) | 0.53 | 0.45 |
| Cost per day (SEK) | 17.3 | 16.5 |
| Physician prescription volume | 13.5 | 8.9 |
| Physician gender (1=female) | 0.22 | 0.24 |
| Physician graduation year | 1985.0 | 1985.6 |
| Specialist | 0.085 | 0.085 |
| Gender (patient; 1=female) | 0.56 | 0.57 |
| Age (patient) | 61.7 | 53.8 |
| Gastric ulcer | 0.07 | 0.05 |
| Duodenal ulcer | 0.20 | 0.13 |
| Gatrosophageal reflux disease (GERD) | 0.27 | 0.20 |
| Dyspepsia | 0.46 | 0.62 |
| Number of obs | 3302 | 1081 |

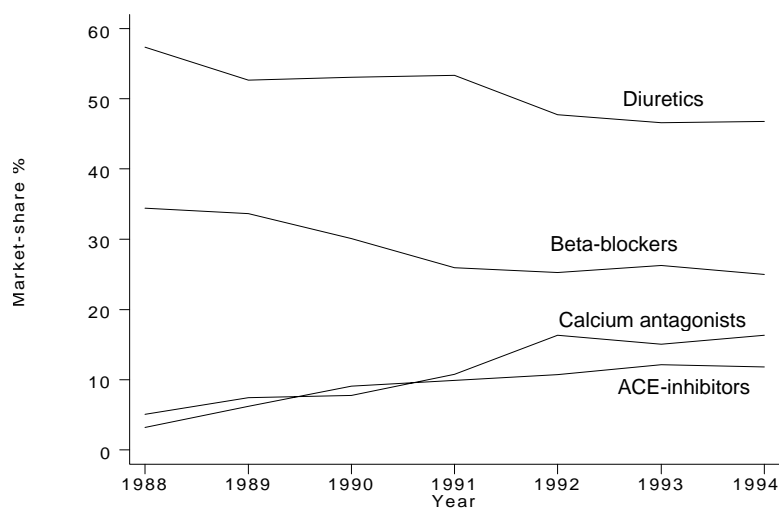


Figure 1: Market-shares, as measured by number of prescriptions filled out, from 1988 to 1994 of the four different types of hypertensive agents.

Table 4: Parameter estimates - Hypertension. Model with physician preferences (physician prescription history). Dependent variable is type of drug.

| | | Repeated prescr. | | | First prescr. | | |
|---------------------------|---------|------------------|------------|------------|---------------|-----------|------------|
| Cost per day | Mean | | -0.17 | | | 0.63 | |
| | St.dev. | | (-0.85) | | | (1.42) | |
| Prescribed last time | Mean | | 3.30 | | | | |
| | St.dev. | | (30.11)*** | | | | |
| Physician prescr. history | Mean | | 1.71 | | | 1.37 | |
| | St.dev. | | (4.17)*** | | | (1.94)* | |
| Intercept | Mean | Beta-block. | Ca-antag. | ACE-inhib. | Beta-block. | Ca-antag. | ACE-inhib. |
| | St.dev. | | | | | | |
| Gender | | | | | | | |
| Age | | | | | | | |
| Diabetes | | | | | | | |
| Coronary heart disease | | | | | | | |
| Congestive heart failure | | | | | | | |
| Time trend | | | | | | | |
| Observations | | | 6632 | | | 487 | |
| Log likelihood | | | -3125 | | | -564 | |
| Pseudo R ² | | | 0.59 | | | 0.20 | |

Notes: Numbers in parentheses are z-values.

* Significant at the 10 % level. ** Significant at the 5% level. ***Significant at the 1% level.

Table 5: Parameter estimates - Hypertension. Model with physician characteristics. Dependent variable is type of drug.

| | | Repeated prescr. | | | First prescr. | | |
|-----------------------------|--------|---------------------------------|--------------------------------|---------------------------------|-------------------------------|-----------------------------|------------------------------|
| Cost per day | Mean | | -0.13 (-0.69) | | 0.65 (1.52) | | |
| | St.dev | | 0.04 (0.37) | | 0.13 (0.50) | | |
| Prescribed last time | Mean | | 3.20 (33.61)*** | | | | |
| | St.dev | | 1.67 (14.61)*** | | | | |
| Intercept | Mean | Beta-block. 2.50 (2.43)** | Ca-antag. 1.29 (3.75)*** | ACE-inhib. 4.53 (5.12)*** | Beta-block. 7.30 (3.08) | Ca-antag. 4.25 (1.56) | ACE-inhib. 4.16 (1.28) |
| | St.dev | 0.65 (4.20)*** | 0.82 (5.31)*** | 1.06 (6.33)*** | | | |
| Physician gender | | -0.060 (-0.42) | -0.233 (-1.45) | -0.464 (-2.47)** | 0.31 (1.05) | 0.42 (1.26) | 0.51 (1.31) |
| Physician graduation year | | 0.017 (1.78) | 0.015 (1.34) | 0.008 (0.66) | -0.017 (-0.082) | -0.033 (-1.44) | -0.018 (-0.56) |
| Physician prescription vol. | | -0.0017 (-1.67) | 0.0014 (0.78) | -0.0023 (-1.19) | -0.0014 (-0.41) | -0.0048 (-1.24) | 0.0051 (1.13) |
| Gender | | -0.14 (-0.95) | -0.53 (-3.23)*** | -0.54 (-2.79)*** | -0.83 (-3.06)*** | -0.87 (-2.80)*** | -1.16 (-3.25)*** |
| Age | | -0.055 (-7.86)*** | -0.047 (-6.39)*** | -0.088 (-9.85)*** | -0.107 (-8.02)*** | -0.066 (-4.20)*** | -0.102 (-5.94)*** |
| Diabetes | | -0.185 (-0.91) | 0.024 (0.11) | 0.072 (0.27) | 0.58 (1.21) | -0.53 (-0.82) | 1.19 (2.14)** |
| Coronary heart disease | | 0.19 (0.081) | 0.47 (1.95)** | -0.46 (-1.27) | 0.37 (0.76) | 0.33 (0.62) | -0.11 (-0.14) |
| Congestive heart failure | | -1.17 (-3.42)*** | -0.60 (-2.00)** | -0.11 (-0.28) | | | |
| Time trend | | -0.00012 (-1.22) | 0.00052 (3.55)*** | 0.00059 (4.82)*** | 0.00047 (2.08)** | 0.00081 (2.71)*** | 0.00031 (1.08) |
| Observations | | | 7446 | | | 538 | |
| Log likelihood | | | -3536 | | | -617 | |
| Pseudo R ² | | | 0.59 | | | 0.04 | |

Notes: See Table 4.

Table 6: Parameter estimates - Ulcers. Dependent variable is type of drug (1=proton pump inhibitors).

| | | Repeated prescr. | | First prescr. | |
|-----------------------------------|---------|---------------------------|-----------------------------|---------------------------|-----------------------------|
| | | Model with phys. pref. | Model with phys. charac. | Model with phys. pref. | Model with phys. charac. |
| Cost per day (SEK) | Mean | 0.66 (0.51) | -0.05 (-0.05) | -2.66 (-1.56) | -1.18 (-0.81) |
| | St.dev. | 0.001 0.001 | 0.15 (0.86) | | |
| Prescribed last time | Mean | 2.32 (13.75)*** | 2.09 (14.20)*** | | |
| | St.dev. | 1.28 (5.56)*** | 1.27 (7.22)*** | | |
| Physician prescription history | Mean | 1.28 (3.83)*** | | 3.25 (7.63)*** | |
| | St.dev. | 0.40 (0.32) | | | |
| Constant | Mean | -4.19 (-1.22) | -1.14 (0.39) | 4.38 (0.99) | 1.34 (0.34) |
| | St.dev. | 1.09 (7.92)*** | 1.00 (5.88)*** | | |
| Physician gender (1=female) | | | -0.23 (-1.93)* | | -0.21 (-1.90)* |
| Physician grad. year | | | -0.012 (-1.38) | | 0.0074 (0.68) |
| Physician prescription volume | | | -0.00083 (-0.41) | | 0.0043 (1.61) |
| Specialist | | | 0.66 (4.10)*** | | 0.63 (2.59)*** |
| Gender (1=female) | | -0.12 (-0.77) | -0.032 (-0.22) | -0.18 (-1.18) | -0.24 (-1.79) |
| Age | | -0.0032 (-0.65) | -0.0039 (-0.87) | 0.016 (3.91)*** | 0.016 (4.42)*** |
| Duodenal ulcer | | -0.13 (-0.17) | -0.104 (-0.38) | 0.42 (0.96) | 0.41 (1.16) |
| GERD | | 0.70 (2.15)** | 0.46 (1.69)* | -0.39 (-0.95) | -0.18 (-0.55) |
| Dyspepsia | | -0.13 (-0.44) | -0.21 (-0.84) | -0.57 (-1.46) | -0.45 (-1.42) |
| Time trend | | 0.0011 (3.00)*** | 0.00099 (3.13)*** | 0.00037 (0.79) | 0.00061 (1.52) |
| Observations | | 2706 | 3302 | 852 | 1081 |
| Log likelihood | | -1284 | -1605 | -508 | -666 |
| Pseudo R ² | | 0.31 | 0.30 | 0.15 | 0.10 |

Notes: See Table 4.

Table 7: Welfare losses of variations in prescription patterns between physicians.

| | Mean cost/DDD | Coefficient of variation | Welfare loss (as % of expenditure) |
|--|------------------|-----------------------------|--|
| HYPERTENSION | | | |
| 1. Total variation | 2.49 | 0.408 | 28.4 |
| 2. Variation controlling for patient mix | 2.49 | 0.256 | 11.6 |
| 3. Variation induced by physician | 2.49 | 0.040 | 0.3 |
| 4. Variation induced by physician - Repeat. pres. | 2.43 | 0.034 | 0.2 |
| 5. Variation induced by physician - First pres. | 3.17 | 0.082 | 1.0 |
| ULCERS | | | |
| 1. Total variation | 17.61 | 0.375 | 22.7 |
| 2. Variation controlling for patient mix | 17.61 | 0.363 | 21.4 |
| 3. Variation induced by physician | 17.61 | 0.143 | 3.3 |
| 4. Variation induced by physician - Repeat. pres. | 17.86 | 0.072 | 0.9 |
| 5. Variation induced by physician - First pres. | 17.11 | 0.280 | 11.4 |

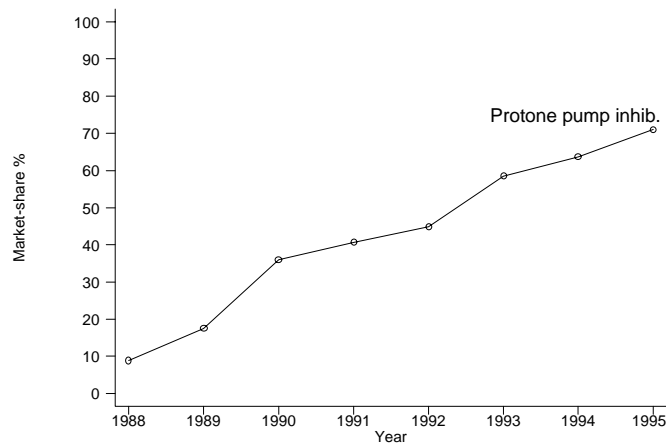


Figure 2: Market-share, as measured by number of prescriptions filled out, for protone pump inhibitors 1988-1995.