

# State Dependence in Pharmaceutical Prescription Choice: Implications for Physician Authority and Market Structure \*

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## Abstract

Exploiting a rich dataset on complete physician and patient prescription histories of anti-ulcer medication in Italy over a three-year period, we explore the extent of persistence in pharmaceutical prescription choices over time. The results have important implications along two dimensions. First, persistence in choice behavior over time and across doctors can shed light on the degree of patient versus physician authority in dictating drug prescriptions. Second, apart from authority issues, choice persistence implies *switching costs* in drug markets. The presence of switching costs can have importance effects both on competition and entry conditions in pharmaceutical markets and in the consumer welfare and (insurer) cost of the lifetime treatment of disease. [Verify:] Controlling for unobserved heterogeneity with either fixed or random effects estimators lessens but does not eliminate the strength of these findings.

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In this paper, we measure the extent of persistence in anti-ulcer pharmaceutical prescription choice over time. We interpret our results along two dimensions. Unlike most choice situations, pharmaceutical prescriptions are the outcome of a principal-agent interaction where patients solicit diagnosis and treatment from doctors. As such, this analysis falls in the framework of agency theories involving expertise, predictions of which have recently been subject to some empirical verification.<sup>1</sup> The main question here is: who has greater weight (or “authority”) in dictating the molecule that is prescribed — the doctor or the patient? Exploiting the richness of our dataset, we are able to construct both patient and doctor prescription histories and relate these to observed choices. The magnitude of temporal dependence in patient v. physician choices provides indirect [weak?] evidence of the degree of authority in the agency relationship. Focusing further on the magnitude of this dependence in patient choices *when visiting a new doctor* provides further [stronger?] evidence on this issue.

Regardless of its source, persistence in choice behavior is indicative of “switching costs”, or reluctance to experiment, at either the patient or physician level. Klemperer [7] points out that switching costs can inhibit market competition, either by fostering product differentiation (an agent substitutes less readily between two products as a result of being habituated to one of them) or raising barriers to entry (new product has to waste promotional resources to convince habituated agents to switch). Thus our results about persistence in prescription choices have important implications about competition in pharmaceutical markets even if one is not willing to subscribe to the agency theory implications described above.

The primary results indicate that both doctor and patient choice histories are very important predictors of molecular choice. <Probability Derivative LMOL, LSH summary> As can be seen, however, it is in fact past *patient* choices which has the stronger effect. <Mag. of LMOL v. LSH> We interpret this as weak evidence of greater patient authority in the agency relationship. We also find, however, that while the importance of past patient choices are greatly reduced when visiting a new doctor, the importance of past doctor choices are unchanged. We take this as a much stronger finding that doctor authority exceeds patient authority in pharmaceutical decision-making. In addition, doctor and patient habit tend to reinforce each other <Interp. LMOL\*LSH>. **WE NEED APPS for each of these results!** [Only thing left out: Age\*LMOL and Gender\*LMOL]

Since our focus is on measuring the degree of state dependence in choices, we must be

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<sup>1</sup>Examples include Hubbard [?] and Stern and Trajtenberg( [9], hereafter ST). For an overview of agency theory, see Aghion and Tirole [cite?].

sure to control for *unobserved heterogeneity* which, as Heckman [4] argues, can also lead to findings of “spurious” state dependence. Roughly speaking, we must make sure that the effects we attribute to (time-varying) patient habit don’t actually arise from time-invariant unobserved individual tastes.

Similarly, unobserved doctor tastes for molecules may be confounded with doctor “habit”. To address these issues, we outline an extension of the framework presented here to accommodate unobserved patient and doctor heterogeneity. We consider both the fixed effects logit estimators developed by Chamberlain [2] and Honore and Kyriazidou [5] and random effects estimators as considered in Keane [?].

While the discrete choice model we employ has a natural interpretation as a random utility model, we never directly address the question: are patients prescribed the molecule that is the best for them? Abstracting from issues of authority, selecting the best treatment is a *dynamic* process of matching patients which the molecule that best treats the illness, conditional on all the information about the patient’s condition and reaction to various molecule gained through previous prescriptions. In a companion paper, we are directly examining these dynamic aspects of the prescription process by estimating a structural matching model. [rough:] The issues of authority and switching costs considered here can then be embedded in that model.

The rest of this paper is organized as follows. In section 2, the data is described and some initial evidence of the degree of heterogeneity and time dependence in doctor and patient prescription histories is described. The logit model of prescription choice forming the foundation of the empirical analysis is described in Section 3 and the results are presented in Section 4. In Section 5, we outline our proposed approach for accommodating unobserved doctor and patient heterogeneity in prescription choice. A final section concludes.

## 1 State dependence in pharmaceutical prescriptions: possible interpretations

### 1.1 State dependence as symptom of physician or patient authority

Physicians have long been recognized as possessing an informational advantage over patients in the medical diagnosis of disease. This expertise suggests a comparable degree of authority in prescribing treatment. At the same time, however, diagnostic ability and experience with

alternative treatments varies considerably across doctors and patients have considerable latitude in selecting physicians. In addition, for many illnesses, patients control access to information about the effectiveness of a given treatment and, for recurring illnesses, may possess informational advantages over physicians about the relative efficacy of alternative treatments. This “levels the (information) playing field” and prompts a very important question in pharmaceutical decisionmaking: Is a doctor’s choice of a molecule to prescribe to a patient dictated more by a patient’s prescription history or by a doctor’s prescription history? This paper addresses this issue.

In previous work, Stern and Trajtenberg ([9], hereafter ST) have argued that highly “concentrated” doctors (i.e., doctors who are not very dispersed in the molecules they prescribe) tend to prescribe molecules with high market share. They interpret these results from the standpoint that higher prescription concentration implies less diagnostic ability and/or knowledge of appropriate treatments on the part of the doctors, and that the (largely passive) patients are “worse off” because they tend to be prescribed a molecule which is less tailored to their specific diagnosis.

These interpretation, however, depends on the assumptions that prescriptions for a given patient are *i.i.d.* over time and that patients are all passive. The latter assumption is not completely justified both for the reasons mentioned above and because in long-term relationships between doctor and patient the latter can potentially play an “active” role.

It is for this reason that we focus on state dependence in this paper: to the extent that both doctors’ and patients’ decisions are temporally related, there is a potential tension between the preferences of the doctor (formed over prescriptions to *all* his patients) and the preferences of the patient (formed over her own prescriptions only). Do the habits of the doctor or the patient dominate in the prescription process?

A strong interpretation of a finding of state dependence assumes that habit is a sign — a symptom — of authority. Doctors like to prescribe similar portfolios of drugs period after period due to something like a lack of diagnostic ability, knowledge of appropriate treatments, or (unobserved) benefits from pharmaceutical companies. Patients like to take the same drug over and over to avoid incurring (implicit) switching costs due to familiarity with a drug’s side effects and treatment regimen.<sup>2</sup> Given these assumptions, the question of whether the doctor or patient’s previous choice is the greater determinant of current choice translates directly into a statement about who has the greater authority — or “say” — in

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<sup>2</sup>The sample period considered here preceeded the “time release” revolution in pharmaceuticals, which greatly equalized treatment regimens across many different drugs.

the prescription process.

As we are not structurally modeling the nature of state dependence, this interpretation is open to critique. Alternative features of patient or doctor decision processes, unrelated to authority could induce a finding of state dependence in pharmaceutical choices. We therefore consider a more powerful test of authority in pharmaceutical choices.

Regardless of source, patients and doctors both exhibit state dependence in their decision-making. The question of authority is not, therefore, which of these is greater, but which of these is stronger? To examine this issue, we consider the prescription decisions of a particular subsample of patients: those for whom we observe previous molecule choices, but who are *seeing a new doctor for the first time*. For these patients-physician interactions, we measure the degree of authority not by the magnitude of state dependence in their (respective) decisions, but in the change in this magnitude relative to the norm. In particular, if patients are found to be less sensitive to their previous prescription choices when visiting a new doctor than when visiting an old, we claim that it is the (new) doctor who has the relative authority in prescription choice. Similarly, if doctors are found to be more sensitive to their previous prescription choices when seeing a new patient, the same conclusion holds. As these findings are robust regardless of the source of the persistence in patient or doctor choices, we have much more confidence that they reflect the degree of authority in the agency relationship.<sup>3</sup>

**Need Application/Implications of this finding!**

## 1.2 State dependence as symptom of switching costs

Regardless of the source of state dependence, if choices are persistent over time, then the prescription decisions exhibits (implicit) switching costs. This section motivates and describes the implications of such a finding.

Why would patients incur costs when switching molecules? As discussed above, they may arise due to familiarity with a drug's side effects (and uncertainty about the side effects of other drugs), as well as familiarity with a particular drug's treatment regimen. If patients learn about the efficacy of drugs only through use, there will be an inclination to continue taking their chosen drug and a disinclination to take another.<sup>4</sup>

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<sup>3</sup>One of these, unobserved doctor and patient heterogeneity is explicitly considered in section ? [How do we connect this number to the unobs. het. section?]

<sup>4</sup>Anti-ulcer drugs are known for occasional side-effects, including constipation, diarrhea, and decreased

Why would doctors incur costs to switching molecule prescriptions? As described in ST, doctors face significant costs associated with informational investments in both diagnostic ability and pharmacological treatment. In addition, experimenting with alternative treatments brings greater uncertainty and possibly more repeat visits from patients dissatisfied with treatment. As such, they have a disincentive to alter their prescription patterns.<sup>5</sup>

Quantifying the extent of switching costs in pharmaceutical prescriptions sheds light on the competitiveness of drug markets. Switching costs lead to segmentation of the market, allowing manufacturers of individual drugs more market power. Furthermore, it may also constitute a barrier to entry into these markets, encouraging long-run extra-competitive profits.

**Need More on Switching Costs!**

## 2 Data

For the work in this paper, we merge two large panel datasets:

1. Complete anti-ulcer drug prescription info for 350 doctors in the Rome metropolitan area, from January 1990 to December 1992. Over 700,000 observations are recorded in this dataset.
2. Complete anti-ulcer prescriptions received by 10% of all anti-ulcer patients in Rome over the same period. About 310,000 observations are recorded in this dataset.

Our merged dataset contains the complete prescription histories for 6813 patients, which are the patients in dataset 2 above who received at least one anti-ulcer drug prescription from one of the 350 doctors in dataset 1. This merged dataset contains close to 70,000 observations. Coscelli [3] contains more details about the dataset.

**Figure 1 - Needed**

Figure 1: Summary statistics

In our analysis, we focus (as do ST) on choices between different *molecules*. We assume that all brands of a particular molecule are identical in the therapeutic value that they

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absorption of concomitantly administered medications (Warner and McIsaac [?])

<sup>5</sup>Another reason is doctors may prefer to maintain particular prescription patterns if they are compensated from pharmaceutical companies for these prescriptions.

offer a patient so that, for example, LOSEC and OMEPRAZEN are identical since they both are based on the sample molecule: *omeprazole*.<sup>6</sup> In our dataset, we observe prescription for 23 different anti-ulcer molecules. To facilitate our analysis, we aggregate the 14 smallest molecules (in terms of shares of total in-sample prescriptions) to obtain 10 alternatives available to a patient on each prescription occasion: the 9 molecules with the 9 highest market shares (measured as shares of total in-sample prescriptions), and the 10-th alternative, which is a basket composed of all the other molecules. Figure 2 contains more information on the 10 alternatives in our model.

Figure 2: Molecules in the Anti-Ulcer Market

Choice #	Molecule Name	In-Sample Mkt. Share	Major brands	Date of Entry
1	CIMETIDINE	2.23	Tagamet	1978
2	SUCRALFATE	2.33	Antepsin	1975
3	FAMOTIDINE	10.74	Famodil	1986
4	RANITIDINE	66.44	Zantac, Ranidil	1981
5	NIZATIDINE	4.33	Zanizal	1988
6	ROXATIDINE	0.96	Roxit	1992
7	OMEPRAZOLE	8.71	Losec, Omeprazen	1990
8	MISOPROSTOLE	0.71	Cytotec	1989
9	???	2.24	Gliptide	1985
10	14 OTHERS	1.31	<i>various</i>	—

### 3 Some initial evidence

This section describes the nature of persistence in pharmaceutical prescription choices over time for both patients and doctors.

**High persistence at patient level** A given patient consumes very few different molecules over time. In figure 3 we present two indicators of persistence in molecular choice: NUM-MOL, the number of different molecules tried, and HERFMOL, the herfindahl index calculated using molecular shares. If a patient were to take only a single molecule over the entire sample period, both of these magnitudes would equal 1. If their treatment were split

<sup>6</sup>Justification for this assumption? Scott's job market paper - others?

equally across two molecules, NUMMOL would equal 2 and HERFMOL would equal 0.5.<sup>7</sup> Each of these measures is calculated over different time horizons.

Figure 3: Persistence indicators at patient level

Period length	NOBS	Avg. NUMMOL	Avg. HERFMOL
Month	49472	1.065 (0.564)	0.976 (0.106)
Quarter	30034	1.113 (0.521)	0.956 (0.137)
Halfyear	20329	1.175 (0.557)	0.936 (0.160)
Year	15948	1.236 (0.613)	0.919 (0.176)
3 Years	6814	1.552 (0.883)	0.849 (0.218)

The NUMMOL figures indicate that most patients try only one molecule regardless of period length, and the HERFMOL figures indicate that even when a second molecule has been tried its share of total prescriptions is often very small. Only at the 3 year time horizon is there an upward jump both in NUMMOL as well as HERFMOL.

This finding of persistence at the patient level can also be seen from the following “switching” matrix, which tabulates the ordered pairs  $(a, b)$  where molecule  $b$  was consumed in a month after molecule  $a$  was consumed. Only 8 (out of the 23) molecules are included (the largest ones). The top element in the  $(a, b)$ th cell represents the number of “switches” from molecule  $a$  to molecule  $b$ , while the bottom element is the percentage of all switches away from brand  $a$ . Obviously the large numbers on the diagonal indicate that patients rarely switch. On the other hand, the large number in the fourth column indicate that if switches occur, they occur towards the market leader *ranitidine* (which includes Glaxo’s Zantac, which was the top anti-ulcer drug in Italy — and indeed, in the world — during the 1990-1992 sample period).

**Persistence at doctor level** In contrast to the patient level, there is less persistence at the doctor level: doctors do not prescribe the same drug to everybody. Figure 5 is the doctor-level equivalent of figure 3. The striking result in this figure is that while the number of molecules that the average doctor prescribes more than doubles when we lengthen the time period from 1 month to 3 years, the herfindahl index hardly changes. While doctors try different drugs over time, they tend to maintain the same level of concentration. Note, however, that HERFMOL doesn’t capture the extent to which doctors tend to remain

<sup>7</sup>For a given individual, HERFMOL equals the sum of the squares of that individual’s “molecule market share”.

Figure 4: Switching matrix at patient level

To: From:	1	2	3	4	5	6	7	8	9	10
1	1154 0.7181	16 0.0100	25 0.0156	260 0.1618	17 0.0106	12 0.0075	54 0.0336	15 0.0093	40 0.0249	14 0.0087
2	15 0.0082	811 0.4439	103 0.0564	615 0.3366	89 0.0487	17 0.0093	118 0.0646	9 0.0049	28 0.0153	22 0.0120
3	27 0.0043	109 0.0173	5124 0.8111	633 0.1002	42 0.0066	25 0.0040	184 0.0291	25 0.0040	96 0.0152	52 0.0082
4	197 0.0051	573 0.0149	522 0.0136	34584 0.8978	238 0.0062	151 0.0039	1247 0.0324	170 0.0044	576 0.0150	264 0.0069
5	29 0.0104	72 0.0258	46 0.0165	342 0.1225	2065 0.7399	17 0.0061	124 0.0444	16 0.0057	49 0.0176	31 0.0111
6	5 0.0111	7 0.0155	8 0.0177	65 0.1441	4 0.0089	317 0.7029	32 0.0710	0 0.0000	13 0.0288	0 0.0000
7	26 0.0055	100 0.0212	97 0.0205	881 0.1863	66 0.0140	65 0.0137	3252 0.6878	22 0.0047	151 0.0319	68 0.0144
8	10 0.0185	10 0.0185	26 0.0480	183 0.3376	15 0.0277	6 0.0111	42 0.0775	227 0.4188	16 0.0295	7 0.0129
9	35 0.0194	29 0.0161	103 0.0572	607 0.3370	47 0.0261	20 0.0111	189 0.1049	15 0.0083	721 0.4003	35 0.0194
10	20 0.0194	26 0.0252	57 0.0552	298 0.2885	35 0.0339	5 0.0048	87 0.0842	5 0.0048	35 0.0339	465 0.4501

Molecule numbers: 1-cimetidine 2-sucralfate 3-famotidine 4-ranitidine 5-nizatidine 6-roxatidine  
7-omeprazole 8-misoprostale 9-??? 10-all others

Figure 5: Persistence indicators at doctor level

Period length	NOBS	Avg. NUMMOL	Avg. HERFMOL
Month	12379	6.157 (1.696)	0.489 (0.137)
Quarter	4148	8.180 (1.679)	0.478 (0.120)
Halfyear	2083	9.472 (1.729)	0.474 (0.115)
Year	1384	10.138 (1.828)	0.475 (0.113)
3 Years	350	13.626 (1.693)	0.464 (0.103)

concentrated in the same drugs over time (i.e., how stable molecular shares at the doctor level are over time).

In summary, the “aggregate” numbers presented here suggest that intermolecular choice in the anti-ulcer market is quite possibly dictated by patients’ tastes: patients prefer to take only one drug and have strong tastes for that drug over a fairly long time horizon. This suggests that molecular share at the doctor level is largely driven by patient mix. The next step will be to see whether these conclusions can be drawn at the micro-level, after accounting for heterogeneity (both observed and unobserved) across patients, across doctors, and over time.

## 4 The empirical model

The basic empirical model will be a discrete choice model, with variables included to capture persistence in choices at both the doctor- and patient-level. Some attempt will also be made to control for *time-invariant* unobserved heterogeneity at the (doctor-molecule) as well as (patient-molecule) level, to make sure that the persistence variables in fact capture the *time-varying* effects of consumer and doctor tastes.

The dataset contains complete prescription histories for doctors  $j$ , as well as complete prescription histories for patients  $i$ . To estimate the model, we will only use observations involving doctors and patients for whom we observe complete prescription histories, since these histories are what allows us to define the (partially out-of-sample) doctor- and patient-specific variables.

We assume that for observation  $(i, j, t)$ , doctor  $j$  and patient  $i$  jointly choose the molecule  $m$  which yields maximal utility for them both during period  $t$ , where

**END CURRENT gsc EDITS - 6/5/98 - Save “macro”-type notes in bold.**

**Need to change habit to something else**

$$U_{mijt} = X_j\beta_1 + X_i\beta_2 + X_m\beta_3 + H_{mjt}\beta_4 + H_{mit}\beta_5 + \theta_{im} + \gamma_{jm} + \delta_{mt} + \kappa_m + \epsilon_{mijt} : \quad (1)$$

- $X_j$  are covariates for doctor  $j$
- $X_i$  are covariates for patient  $i$

- $X_m$  are covariates for molecule  $m$
- $H_{mjt}$  measures doctor  $j$ 's "habit" with molecule  $m$  at time  $t$
- $H_{mit}$  measures patient  $i$ 's "habit" with molecule  $m$  at time  $t$
- $\theta_{im}$  is patient  $i$ 's time-invariant unobserved heterogeneity factor for molecule  $m$
- $\gamma_{jm}$  is doctor  $j$ 's time-invariant unobserved heterogeneity factor for molecule  $m$
- $\delta_{mt}$  is a period  $t$ -specific shock for molecule  $m$
- $\kappa_m$  is a time-invariant factor for molecule  $m$  (analogous to Berry's [1] "unobserved quality")
- $\epsilon_{mijt}$  is an iid (across  $t, i, j$ ) error term which captures aspects of molecule  $m$ 's utility not observed by the econometrician; can possibly allow for correlation across  $m$ , for a given  $(i, j, t)$ . If  $\epsilon$ 's take on extreme value distribution, then resulting choice probabilities take on logit form.

The parameters  $\beta_3$  and  $\beta_4$  are the most important for this analysis, measuring the influence of doctors' and patients' previous choices, respectively, on current prescription choice.

**Wait for final results before putting up Suggest dropping S-T type regressions as this isn't really the point. If we wanted to, we could write a "note" to whatever journal they (ever) get their stuff published in to show how you can generate their results by ignoring state dependence.**

## 5 Results

### 5 different specifications

**Model A** Constants only (maybe too simple)

**Model B** A S-T type regression - Herf, Herf\*nvis, constant effects across mols.

**Model C** A S-T type regression - Herf, Herf\*nvis, let herf effect vary across mols.

**Model D** Same as Model B, but with LMOL, LSH. Constant effects across mols.

**Model E** Same as Model D, but with effects that vary across mols. This is the (al)most general model.

Reason to estimate models B and C: do S-T results carry through? S-T results may not carry through due to differences in American and Italian medical systems. In the US, physician-patient relationship is perhaps not as long-lasting as in Italy, making room for more agency problems. On the other hand, the more short-term nature of this relationship also makes the market for physician services more competitive, imposing some discipline on doctors' behavior. In short, institutional differences between US and Italy offer no clear prediction of whether agency problems should be more or less prevalent. Furthermore, ST focus on markets for different drugs than we do.

< **WOULD BE GREAT TO FIND SOME STATISTICS ON HOW LONG PARTICULAR PATIENTS HAVE BEEN WITH DOCTORS IN US AND IN ITALY** >

Why estimate models D and E? Look for implications of physician vs. patient authority in looking at habit. Implicit assumption: doctors are lazy  $\rightarrow$  habit persistent, and patients are averse to switching (this rules out sampling or matching models, which is focus of another project). Extension to ST in the following sense: ST look for symptoms of physician authority — or rather, *fiat* — along one dimension of observed doctor heterogeneity: differences in dispersion of molecules prescribed. Model D and E look for symptoms of both physician and patient authority by focusing on the importance of habit.

**Results from different specifications**    **Model A** results ....

Coefficients correspond to actual (in-sample) market shares.    Order of  
 market leaders is 4       @ 60%  
                                   3,7    @ 10% each  
                                   5       @ 4%  
                                   1,2    @ 2% each

The **Model B** results already exhibit many of the trends which remain robust across the specifications. Price coefficient is significant and *positive* There are several potential reasons for this. (1) Price endogeneity, perhaps less likely since prices are set by Italian *Commissione Unica del farmaco* (the equivalent of the Food and Drug Administration); in this case, prices will be endogenous only if NHS sets price as a function of a drug's "unobserved quality", which is observed by all agents (doctors and patients) but not by econometrician. (2) Given that drug expenditures are paid for by the national health service (except for a small co-payment paid by patients), not surprising that doctors and/or patient exhibit

Figure 6: Specifications without habit

	Model A		Model B		Model C	
	Estim.	StdErr	Estim.	StdErr	Estim.	StdErr
MOLECULE CHARACTERISTICS						
Price	no		0.626	0.054	0.434	0.101
#VIS (advertising)	no		-0.0266	0.0013	0.0030	0.0023
DOCTOR AND PATIENT CHARACTERISTICS						
Doctor HERF	no		-0.727	0.440	—	—
Doctor Lagged Share (LSH)	no		no		no	
Patient Lagged MOL (LMOL)	no		no		no	
HERF*Price	no		-0.494	0.116	-0.065	0.225
HERF*#VIS	no		0.0534	0.0026	-0.0130	0.0050
HERF*LMOL	no		no		no	
HERF*LSH	no		no		no	
LSH*LMOL	no		no		no	
MOLECULE-SPECIFIC COEFFICIENTS						
Dummies	yes		yes		yes	
AGE	no		yes		yes	
GENDER	no		yes		yes	
Doctor HERF	no		no		yes	
Log-likelihood fxn value						
	-74768.3		-74547.4		-73566.7	

Figure 7: Specifications with habit (time-varying observed heterogeneity)

	<b>Model D</b>		<b>Model E</b>	
	Estim.	StdErr	Estim.	StdErr
<b>MOLECULE CHARACTERISTICS</b>				
Price	0.362	0.070	0.246	0.109
#VIS (advertising)	0.0004	0.0020	0.0104	0.0032
<b>DOCTOR AND PATIENT CHARACTERISTICS</b>				
Doctor HERF	-0.272	0.150	—	—
Doctor Lagged Share (LSH)	2.267	0.181	2.447	0.295
Patient Lagged MOL (LMOL)	3.411	0.062	3.392	0.063
HERF*Price	-0.272	0.150	-0.009	0.241
HERF*#VIS	-0.0047	-0.0043	-0.0181	0.0072
HERF*LMOL	1.055	0.141	1.079	0.143
HERF*LSH	-0.562	0.306	0.045	0.697
LSH*LMOL	-2.000	0.064	-1.986	0.064
<b>MOLECULE-SPECIFIC COEFFICIENTS</b>				
Dummies	yes		yes	
AGE	yes		yes	
GENDER	yes		yes	
Doctor HERF	no		yes	
Log-likelihood fxn value				
	-33515.7		-33469.7	

little price sensitivity (and, according to point estimate, even marginal *disutility* of income!). Furthermore, advertising coefficient is negative, and significant — again, this could be due to advertising endogeneity, if firms set advertising as a function of “unobserved quality”.

What about the ST effects? Find strong support for ST effects: coefficient on HERF\*PRICE is negative (lazy doctors choose cheaper drugs) and HERF\*#VIS is positive (lazy doctors choose drugs they are easily informed about). What about HERF\*(Market Share)? That is explored in Model C.

The big change in **Model C** is allowing HERF’s effects to differ by molecule — in effect, we are trying to see whether HERF’s effects are larger for brands with higher market shares, which is ST’s third main result.

LHERF: Now varies cross mols

Mol4:	(+), sig.	(Right way)
Mol3, Mol7:	(+), insig.	(Right way, insig.)
Mol 5:	(-), sig.	(Wrong way)

Now HERF\*PRICE effect is insignificant, and HERF\*#VIS effect goes in the opposite way as ST predict. The #VIS coefficient becomes insignificant.

**Model D** introduces habit to the proceedings. Likelihood function rises substantially. Coefficients on both LMOL and LSH are very positive and significant, indicating large degree of habit persistence on the part of both doctors and patients.

(NEW) LHERF*LMOL -	(-), insig.	(? see next reg)
(NEW) LHERF*LSH -	(+), sig.	(Obvious)

What about ST effects? HERF\*PRICE effect goes the right way, but only marginally significant. HERF\*#VIS effect is not significant.

**Model E** is analogous to Model C, where we allow for the HERF’s effect to differ across molecule. No big changes relative to Model D. ST effects are either insignificant (HERF\*PRICE) or wrong way (HERF\*#VIS).

## 6 Caveats: Unobserved Heterogeneity

If our main task is addressing questions of habit persistence, then most important work lies ahead: controlling for *unobserved heterogeneity*, which can lead to a spurious finding of a high degree of habit persistence (see Heckman [4]).

We are interested in time-invariant patient effects which shift the *level* of utility: the  $\theta_{im}$ 's in equation (1). Advantage of fixed-effects approach (versus random effects approach): don't need to assume that unobserved heterogeneity is orthogonal to the included covariates (which is problematic when the covariates include lagged dependent variables, as here).

### 6.1 Fixed effects approach

Refer back to equation (1). Let's omit  $\gamma_{jm}$  and ignore  $\delta_{mt}$ , and simplify the notation:

$$U_{mijt} = X_{mijt}\beta + \theta_{im} + \epsilon_{mijt}. \quad (2)$$

The main assumption of the *fixed effects logit* model is that, conditional on the covariates  $X_{mijt}$  and also the fixed effects  $\theta_{im}$ , each  $\epsilon_{mijt}$  is *i.i.d.* (across  $m, i, j, t$ ) and distributed type II extreme value<sup>8</sup> so that

$$\text{Prob}(m \mid X_{mijt}, \theta_{i1}, \dots, \theta_{i10}) = \frac{\exp(X'_{mijt}\beta + \theta_{im})}{1 + \sum_{m'} \exp(X'_{m'ijt}\beta + \theta_{im'})} \quad (3)$$

It's infeasible to estimate all the fixed effects, since we have 6813 patients \* 10 molecules. Quite a sizeable literature has discussed ways to estimate the fixed logit model (Chamberlain [2] and Honore and Kyriazidou [5]). Furthermore, the methodology proposed by Berry [1] for estimating discrete-choice models can be re-interpreted as estimating a fixed-effects logit model with a cross-section of market share data and assuming time-invariant brand-specific fixed effects (what Berry calls "unobserved quality"). He explicitly eschews the (easier) random effects approach since the problem he is dealing with is price endogeneity i.e., correlation between the fixed effect and a covariate, which violates the assumption of the random effects approach.

The methodology proposed follows the Berry approach to estimating a fixed-effects logit model. In particular, it consists of two main steps, with the second nested within the first:

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<sup>8</sup>Important to emphasize that  $\epsilon$ 's are *i.i.d.* conditional on fixed effect. Rules out case where covariates which vary over  $t$  are endogenous i.e., correlation between  $X_{mijt}$  and  $\epsilon_{mijt}$  over  $t$ . In other words, the model allows the lagged "habit" indicators for a patient to be serially correlated only through the time-invariant factor  $\theta$ .

1. Estimating the parameters of the utility function ( $\beta$ ) by GMM using instruments that the fixed effects  $\theta$ 's are (assumed) orthogonal to.
2. At each given value of  $\beta$  — call it  $\hat{\beta}$  solving out for the fixed effects  $\theta$ 's from equations which equate observed to predicted purchase shares at the patient level. These  $\theta$ 's are therefore functions of  $\hat{\beta}$ , the covariates  $X$ , and the observed purchase shares.

I will describe the steps in reverse order.

**Step 2: Solving for the  $\theta$ 's** In Berry's original paper, as well as most subsequent applications of his approach, the fixed effects are solved out from systems of equations where observed market share is equated with predicted market share. It is important to note that no estimation is involved in this step, we solve for the  $\theta$ 's *given* the current values of the parameters  $\beta$ . < **THIS IS NOT AN ESTIMATION PROCEDURE WHICH TRIES TO "MINIMIZE THE DISTANCE" BETWEEN OBSERVED AND PREDICTED MARKET SHARES???** THAT IS JUST A WAY OF DERIVING THE  $\theta$ 's >

For our case, the fixed effects are patient- and molecule-specific. Define (observed) "purchase shares"  $s_{im}^*$  as the proportion of patient  $i$ 's total in-sample purchases which are of molecule  $m$ . Define the predicted purchase share  $s_{im}$  as the sum (over  $t$ ) of patient  $i$ 's purchase probabilities of molecule  $m$ :

$$s_{im} = \sum_{t=1}^{T_i} \frac{\exp(X'_{mijt}\beta + \theta_{im})}{1 + \sum_{m'} \exp(X'_{m'ijt}\beta + \theta_{im'})} \quad (4)$$

where  $T_i$  is the number of in-sample prescriptions for patient  $i$ .

Normalize  $\theta_{i1} = 0$ , for all  $i$  (which is ok if we include brand dummies for each brand). Then ??? the system of 9 equations

$$s_{im}^* = s_{im}, \quad m = 2, \dots, 10 \quad (5)$$

for each patient  $i$  can be solved (using some nonlinear solver) for the 9 unknowns  $\theta_{i2}, \dots, \theta_{i10}$ . < **TWO PROBLEMS: (1) ARE THESE SOLUTIONS UNIQUE? (2) FOR MOST PATIENTS, THE OBSERVED "PURCHASE SHARES" WILL BE 0 OR 1 FOR SOME DRUGS. FOR THESE, DO YOU SET (RESPECTIVELY) THE  $\theta$  TO A VERY NEGATIVE OR VERY POSITIVE NUMBER?** >

Are these systems of equations right, i.e., do solving them actually yield the  $\theta$ 's??

**Step 1: GMM Estimation of  $\beta$**  Second step is straightforward. Pick some instruments that (we assume) the  $\theta$ 's are orthogonal to, and minimize the sample orthogonality conditions:

$$\min_{\beta} \sum_i \sum_m \theta_{im} Z'_{im} (Z'Z)^{-1} Z \theta_{im} \quad (6)$$

where  $Z$  is a  $K$ -vector of  $K$  instruments.

**Remarks** Identification of coefficients on time-varying covariates will be weak, since all of the dynamics are “squished” by summing over time in the equations (5). Is this a weakness of the approach, of rather indicative of how hard it is to identify time-dependence in a fixed effects model? How “hard” is it to identify dynamics using the Chamberlain/Kyriazidou approach? Or random effects approach? Maybe interesting to compare results using fixed effects vs. random effects — my intuition says that “habit” will be much stronger using the random effects approach, since it imposes the strong (and unrealistic?) assumptions that unobserved heterogeneity is uncorrelated with covariates.

## 7 Conclusions and Extensions

Analysis so far is *static*: misses out on dynamics of the diagnosis and prescription process. Dynamics characterized by trial and error, to accommodate patient heterogeneity. Is patient-level habit a sign of switching costs or that a good match has been found? Full interpretation of results in this paper, and drawing out welfare implications, requires answer to this question. To that end, we develop a dynamic structural model of prescription process to accommodate the matching aspects of doctors' prescription behavior. Abstract away from more malevolent aspects of agency problem: consideration of long-term nature of doctor-patient relationship, as well as results in this paper, justify this modeling choice — opportunistic agency behavior doesn't define the prescription process.

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