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An empirical model of learning and patient spillovers in new drug entry

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Abstract

We specify and estimate a diffusion model for the new molecule *omeprazole* into the anti-ulcer drug market. Our model is based on a Bayesian learning process whereby doctors update their beliefs about *omeprazole*'s quality relative to existing drugs after observing its effects on the patients that have been prescribed this drug. The model also accommodates informational spillovers and heterogeneity in informativeness across patients with different diagnoses. We obtain estimates of the learning process parameters using a novel panel data set tracking doctors' complete prescription histories over a 3-year period.

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1. Introduction

First-mover advantage is a well-documented phenomenon in many differentiated product markets (see Urban et al. (1986) for a survey of the evidence). Economists have tended to attribute this phenomenon to lack of information among consumers about the quality or attributes of an entrant's product; for example, Shapiro (1982, p. 7) states that

...the fundamental source of the entry barrier is an information one: consumers have better information about established brands than about new ones [...] information is the basic barrier to be overcome by a new product...

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The doctor/patient relationship is fraught with uncertainty. Doctors have incomplete information on the medical condition of a patient, and which treatment is best for the patient. Doctors learn about the quality of alternative treatments both through direct experience (actual prescriptions of the new drug), and indirect experience (such as promotional activity by pharmaceutical companies, articles in medical journals and attendance at medical conferences). This paper focuses on direct information, which accumulates slowly, and is confounded by heterogeneity across diagnoses: what works for diagnosis X may not work as well for diagnosis Y.

Using a novel panel data set of complete prescription histories for a sample of doctors in the Rome (Italy) metropolitan area, we study the diffusion process of a new anti-ulcer drug (*omeprazole*) during a 3-year period (1990–1992). The evolution of *omeprazole*'s market share over time was marked by the gradual diffusion which characterizes new product entry into many product markets: *omeprazole*'s market share (as a proportion of total prescriptions) climbed from under 5% in the latter half of 1990 to about 15% in early 1992, and eventually up to 25% by the middle of 1995.

In this paper, we gauge how well this gradual diffusion pattern can be explained by a learning model in which doctors, initially uncertain about the quality differential between *omeprazole* and the incumbent drugs, update their beliefs about this differential after observing noisy signals from patients to whom they have prescribed *omeprazole*. To that end, we specify and estimate the parameters of such a learning model. Furthermore, in order to accommodate features specific to the pharmaceutical prescription process, we extend the basic learning model to allow for spillovers across all the patients of a given doctor, as well as heterogeneity in informativeness across patients. While there are alternative explanations for the individual-level diffusion process (such as the publication of the results of post-marketing clinical trials in medical journals), we focus on a learning explanation because our data includes especially rich detail on doctors' prescription histories.

Our results suggest that the learning model does very well in generating the observed slow diffusion path of *omeprazole* in the Italian market. The parameters of the learning model quantify, in informational terms, the disadvantage that *omeprazole* suffered relative to the existing drugs upon its entry into the Italian anti-ulcer market. This informational disadvantage can arise from either doctors' initial pessimism about *omeprazole*'s quality, or risk aversion. In addition, we find that the informational spillovers are *negative* across some diagnosis groups, which tends to *retard* the speed of learning. That is, we find that a positive outcome when prescribing *omeprazole* for certain diagnoses leads doctors to regard it as less attractive for other diagnoses.

The next section provides some background on the international and Italian anti-ulcer drug markets. Section 3 describes the doctor-level learning model, Section 4 describes our panel data set of complete prescription histories and Section 5 derives the estimating equations associated with the learning model. Results from several specifications of the learning model are presented and interpreted in Section 6, and we conclude in the last section.

2. Background

Several studies have documented the existence and, more importantly, the nature of barriers to entry into pharmaceutical markets. [Bond and Lean \(1977\)](#) found evidence of substantial pioneer advantage, but they also found that products containing some therapeutic novelty managed to gain large market shares when backed by heavy promotional campaigns. [Berndt et al. \(1997\)](#) document similar effects in the anti-ulcer drug market. Their findings clearly show that technological advances do not necessarily translate into large market shares without tremendous marketing muscle.¹ As striking as the results from the two studies are, however, they never explain the causes of pioneer advantage. The availability of doctor-level prescription histories allows us a unique opportunity to assess the role of information in explaining the diffusion patterns observed in many product markets.²

This paper joins a growing empirical literature examining behavioral explanations for diffusion patterns for new products in experience good markets. Among these studies, [Akerberg \(2002\)](#) and [Erdem and Keane \(1996\)](#) estimated structural learning models to explain consumers' purchase patterns for, respectively, yogurt and laundry detergent. [Ching \(2000\)](#) has also estimated a demand model for pharmaceuticals based on a Bayesian learning procedure. Our work differs from these papers because we consider a more general learning model which allows for spillovers across all the patients of a given doctor, as well as heterogeneity in informativeness across patients. These extensions seem especially appropriate for pharmaceutical markets, since prescription drugs (and in particular anti-ulcer drugs) are usually prescribed for several different diagnoses.

Using aggregate market share data, [Azoulay et al. \(2003\)](#) estimate a diffusion model to study the importance of consumption externalities in explaining the diffusion patterns of H_2 -antagonist drugs into the anti-ulcer drug market. Our analysis extends their work by using a novel micro-data set to quantify the extent of network-type spillovers across patients belonging to the same doctor.³

3. The learning model

In this section, we describe the behavioral model which forms the basis of our empirical analysis. In what follows, we index doctors by the subscript i , and assume that patients are heterogeneous in their diagnoses, which we subscript by j . We begin

¹ Using a similar data set, [Azoulay \(2002\)](#) investigates how promotional activity and scientific information arising from clinical trials affect the diffusion of competing molecules in the anti-ulcer drug market. [King \(2000\)](#) focuses on the role of marketing in increasing the perceived product differentiation (i.e., degree of substitutability) between competing anti-ulcer drugs.

² A related literature ([Stern, 1996](#); [Ellison et al., 1997](#)) has investigated the extent of competition in pharmaceutical markets by estimating cross-price elasticities between the competing drugs in a market. Unlike these papers, we abstract away from competition between existing anti-ulcer drugs.

³ Finally, there has been a long interest in diffusion models in the marketing literature. See [Bass et al. \(1990\)](#) for a review of this largely theoretical and macro-level empirical literature. [Chandrashekar and Sinha \(1995\)](#) is one of the few papers in this literature which are formulated at the micro-level.

by describing a baseline version of the learning model in which doctors are assumed to be risk neutral. At the end of this section, we discuss an alternative model which allows for risk aversion.

Consider a given patient k , from diagnosis group j , who visits doctor i during period t . We assume that doctor i distinguishes between two treatment alternatives: the new molecule, *omeprazole* (alternative 1), and *any* of the other molecules (alternative 0). The utilities for a given patient k with diagnosis j during period t from each alternative are:

$$U_{1jkt}^i = \alpha_1^* x^i + \beta p_{1t} + \zeta_1^*(t) + \delta_{1j}^* + \varepsilon_{1jkt}^i \quad \text{if take omeprazole,} \quad (3.1)$$

$$U_{0jkt}^i = \alpha_0^* x^i + \beta p_{0t} + \zeta_0^*(t) + \delta_{0j}^* + \varepsilon_{0jkt}^i \quad \text{otherwise,} \quad (3.2)$$

where

- p_{1t} and p_{0t} are, respectively, the price of *omeprazole* and a weighted average of the prices of the incumbent drugs weighted by their market shares at time t . The vector x^i contains observed doctors' characteristics.
- δ_{1j}^* and δ_{0j}^* parameterize the “unobserved quality” of *omeprazole* and the incumbent drugs when treating diagnosis j . These are unobserved by the econometrician. Doctors, however, are presumed to know δ_{0j}^* , and have imperfect information about δ_{1j}^* . As described below, doctors learn about δ_{1j}^* by prescribing *omeprazole* to their patients.
- $\zeta_1^*(t)$ and $\zeta_0^*(t)$ are flexible functions of time, which parameterize period t factors which affect the attractiveness of, respectively, *omeprazole* and the incumbent drugs. These are the same over all doctors, patients, and diagnoses. In particular, the function $\zeta_1^*(t)$ proxies for aspects of the learning process which we do not explicitly model, such as word of mouth, medical congresses, and articles in medical journals.
- ε_{1jkt}^i and ε_{0jkt}^i are i.i.d. (over doctors, patients, diagnoses, and time periods) shocks associated with, respectively, *omeprazole* and the incumbent drugs. They are observed by the doctors, but not by the econometrician.

Throughout, we abstract away from agency problems between the doctor and the patient, and assume the doctor maximizes the *patient's* utility from the prescription.⁴ Doctor i chooses the option with the higher *per-period* utility.⁵ The choice rule for

⁴ The reputation effects resulting from the long-term nature of many patient–doctor relationships in Italy (the National Health Service requires each enrollee to list a general practitioner) tend to minimize the divergence between doctors' and patients' objective functions which potentially form the basis of agency problems.

⁵ For computational tractability we have assumed that doctors are myopic in our model, so that in any given time period, a doctor chooses the molecule with the highest *per-period* utility based solely on her current information. If the doctor were forward-looking, she would choose the molecule with the highest *present discounted utility* and thereby take into account the information that she would gain about *omeprazole* by prescribing it this period. Ongoing work by Crawford and Shum (2000) examines issues of uncertainty and matching in pharmaceutical demand in a fully forward-looking framework. Ferreyra (1999) has recently estimated a forward-looking dynamic learning model, using the same data that we use in this paper, but without allowing for spillovers across patients.

the doctor is to prescribe *omeprazole* if $E_t(U_{1kjt}^i) > U_{0kjt}^i$. If we assume that ε_{1jkt}^i and ε_{0jkt}^i are i.i.d. with the type 1 extreme value distribution, the probability that doctor i prescribes *omeprazole* takes the familiar logit form:⁶

$$\text{Prob}(\text{prescribe } omeprazole) = \frac{\exp(\alpha x^i + \beta \Delta p_t + \zeta(t) + E_t \delta_j)}{1 + \exp(\alpha x^i + \beta \Delta p_t + \zeta(t) + E_t \delta_j)} \quad (3.3)$$

where we have substituted $\alpha \equiv \alpha_1^* - \alpha_0^*$, $\zeta(t) \equiv \zeta_1^*(t) - \zeta_0^*(t)$, and $E_t \delta_j \equiv E_t \delta_{1j}^* - \delta_{0j}$. α , β , and the $\zeta(t)$ function are to be estimated.⁷

By distinguishing between different diagnoses, we allow the entrant and incumbent anti-ulcer drugs to differ in their effectiveness and suitability across diagnoses. This accommodates “segmentation” or “horizontal differentiation” in the market on the basis of diagnosis, which we believe to be an important feature of the anti-ulcer drug market.

3.1. Bayesian updating

The main focus of the paper is to measure how well the diffusion pattern for *omeprazole* can be explained by doctors’ learning about δ . We explain this learning process in this section. Throughout, we assume that the learning processes are independent across doctors.⁸ Therefore, we describe the learning process for doctor i , omitting the superscript i in most of the equations below for expositional clarity. We assume that, at time $t = 0$ (i.e., at *omeprazole*’s entry), she (doctor i) has the following *initial* beliefs about $\vec{\delta}$, the J -dimensional vector of quality differentials between *omeprazole* and the incumbent drugs:

$$\vec{\delta} \sim N \left(\vec{\delta}_1 \equiv \begin{bmatrix} E_1 \delta_1 \\ \vdots \\ E_1 \delta_J \end{bmatrix}, \Sigma_{\delta,1} \equiv \begin{bmatrix} \sigma_{\delta,1}^2 & 0 & \dots & 0 \\ 0 & \sigma_{\delta,2}^2 & \dots & 0 \\ 0 & \dots & \dots & \vdots \\ 0 & 0 & \dots & \sigma_{\delta,J}^2 \end{bmatrix} \right). \quad (3.4)$$

Throughout, we adopt the indexing convention that the subscript t denotes the beginning of period t ; therefore, $\vec{\delta}_1$ denotes the mean of doctors’ beliefs at the beginning of period 1, corresponding to the mean of the doctors’ initial beliefs (and $\Sigma_{\delta,1}$ is similarly the initial variance–covariance matrix). The assumption that the initial variance–covariance matrix $\Sigma_{\delta,1}$ is diagonal implies that the information that doctors had about

⁶ By aggregating all the non-*omeprazole*-based drugs into one alternative, we are implicitly assuming that all these drugs are perfectly substitutable, and that an *omeprazole*-based drug substitutes equally well with all of them. We make this assumption because we want to focus on the diffusion of drugs based on *omeprazole* into the marketplace.

⁷ In most of the specifications reported below, we assume that the time function $\zeta(t)$ is a quadratic time trend. As we point out below, since the price differential Δp_t only varies over time, it would be impossible to separately identify the price coefficient β apart from a full set of time dummies.

⁸ Informational spillovers across doctors (“word of mouth”) at the aggregate level are captured by the $\zeta(t)$ ’s.

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