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ABSTRACT

We examine the role of network effects in the demand for pharmaceuticals at both the brand level and for a therapeutic class of drugs. These effects emerge when use of a drug by others conveys information about its efficacy and safety to patients and physicians. This can lead to herd behavior where a particular drug -- not necessarily the most efficacious or safest -- can come to dominate the market despite the availability of close substitutes, and can also affect the rate of market diffusion. Using data for H2-antagonist antiulcer drugs, we examine two aspects of these effects. First, we use hedonic price procedures to estimate how the aggregate usage of a drug affects brand valuation. Second, we estimate discrete-time diffusion models at both the industry and brand levels to measure the impact on rates of diffusion and market saturation.

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1 Introduction.

The process by which new products embodying technological advances diffuse through markets is fascinating and exceedingly complex. In his classic study of factors affecting the diffusion of hybrid seed corn in the U.S., Griliches (1957) distinguished three components of the diffusion process: origins (supply, depending on the potential profitability of entry), ceilings (demand, the long-run equilibrium profitability differential from adopting the innovation), and slopes (the rate of approach to market saturation).

In this paper we focus on the diffusion process characterizing a set of pharmaceutical innovations — H_2-antagonist antiulcer drugs, which avoid costly hospitalizations and surgeries, and also are effective in treating rather common ailments such as heartburn. We treat the origins of this innovation as predetermined and largely exogenous, since in the U.S. the ability to obtain exclusive rights to bring medical innovations to the market depends not only on successful research and development, but also on the vicissitudes of obtaining approval from the U.S. Patent Office[1] and the U.S. Food and Drug Administration, approvals that are the outcome of lengthy stochastic administrative and regulatory proceedings.[2] Thus, within the larger context of the diffusion process, we focus our attention on demand-side phenomena involving factors that affect rates of diffusion and long-run market saturation. We consider not only the overall therapeutic class, but also particular brand-name products within the class.

More specifically, we examine "network effects," i.e., the ways in which the demand for a branded pharmaceutical by patients and physicians depends on the number of other patients that have taken or are taking the drug. Unlike computer software and telecommunications systems, where network effects stem from direct external benefits, for pharmaceuticals these network effects are largely informational in nature. They emerge when the use of a drug by others influences one's perceptions about its efficacy, safety, and "acceptability," and

[1]For a discussion of factors affecting the level and rate of patent approvals, see Griliches (1990).

[2]The pharmaceutical innovations on which we focus here were patented by private sector organizations. Griliches (1958) has argued that for hybrid corn, the ideas underlying the innovations were difficult to patent, public sector research predominated, and social rates of return to R&D were much larger than private rates.

thus affects its valuation and rate of adoption. This can lead to herd behavior, where a particular drug — not necessarily the most efficacious or safest — can come to dominate the market despite the availability of close substitutes. It can also affect the rate at which a new product diffuses into the market. Even if there were no externalities affecting individuals' valuations of a product, as more people use the product, word-of-mouth communication increases, accelerating the rate at which others become aware of it.

A priori we would expect network effects of this kind to exist because the widespread use of a prescription drug may convey information to physicians and patients about safety and efficacy, and, for physicians, may imply "accepted practice" and hence greater immunity from malpractice lawsuits.[3] Thus the use of a drug by others could reflect an informational externality in which physicians and patients process data on past and current usage to assess rationally a drug's efficacy and risks. For example, the fact that a drug is currently used by, say, a million patients is evidence that it is at least somewhat efficacious relative to its side effects and risks. Or, it could reflect a physician's rational assessment that other things equal, the probability of a malpractice suit is lower when a widely used drug is prescribed, whatever the actual efficacy and risks of the drug. In either case, the result could be herd behavior that is inefficient.[4]

We distinguish between network effects that influence consumers' valuations of a drug, and those that influence the rate of diffusion in the market.[5] Consumers' valuations are affected when the use of a drug by others influences perceived efficacy and safety. One of our

[3]There is also qualitative evidence of this dependence from early sociological studies of the diffusion of new drugs and medical technologies; see, e.g., Coleman, Katz, and Menzel (1966). For a recent study of the effects of potential malpractice liability on physician behavior, see Kessler and McClellan (1996).

[4]This is analogous to inefficient herd behavior resulting from informational externalities in technology adoption and investment decisions. The inefficiency arises when agents rationally try to free ride on the information generated by the adoption decisions of others, as in the models of Banerjee (1992), Choi (1997), and Scharfstein and Stein (1990); also see Begge and Klemperer (1992) and Bhattacharyja (1994). For a discussion of these and related models, see Bikhchandani, Hirshleifer, and Welch (1998). Besen and Farrell (1994) provide a good overview of network effects and some of their implications for market structure and evolution. Goolsbee and Klenow (1998) present evidence of very similar network effects in consumers' purchase of home computers.

[5]The decision to utilize a drug can be made or influenced by both the patient and the physician, and we do not try to differentiate their roles in the adoption decision. The use of a drug by others can affect its desirability for both patients and physicians, and we include both groups when we refer to "consumers."

goals is to determine the magnitude of this effect. A second goal is to assess the importance of cumulative sales and/or market share as a determinant of the rate of product diffusion.

Pharmaceutical markets are usually bounded in terms of therapeutic classes of drugs, the members of which often are therapeutic substitutes. Hence it is also important to distinguish between network effects at two levels. The first is with respect to a therapeutic class, e.g., H_2-antagonist antiulcer drugs, SSRI antidepressants, or anticholesterol drugs. We expect that physicians may be more willing to prescribe and patients to take a drug the more the therapeutic class of which that drug is a member has been "accepted," where "acceptance" can be measured at least in part by the number of other people that have taken drugs in that class. The second level is with respect to a specific brand of drug within a therapeutic class. We might expect that physicians and patients are more willing to use Zantac (as opposed to, say, Tagamet, Axid, or Pepcid) the greater is its "acceptance," which might be measured by its market share, total sales, or cumulative sales.

Although our focus is on the demand side of pharmaceutical markets, the issues we examine have broad implications for market structure and performance. For example, herd behavior can lead to "tipping," where a small current market share advantage can give a firm a large future advantage as its product becomes the market standard. This can lead to intense competition in the early stages of market evolution as firms struggle to win a future monopoly position, and then create barriers to would-be entrants. When the willingness of consumers to buy a new product depends on the number of other consumers who have purchased the product, sales may never take off, or, if stimulated by initially low prices, might grow very rapidly. Even if there were no externalities affecting consumers' valuations of a product, an initially large market share can lead to "tipping" by affecting the rate of diffusion. Suppose there are two competing products and switching costs are high. If the rate of diffusion for each product depends positively on the number of consumers already using the product, the firm with an initial market share advantage could increase that advantage as the market saturates, and win a future near-monopoly position.

When they occur at the brand level, these effects have implications for pricing, advertising, and R&D decisions. They can create an incentive to price low initially and advertise

heavily, and later provide the owner of a dominant brand the ability to raise price above those for other brands. They also affect the reward for being the first drug in a new therapeutic category. A strong brand-specific effect creates a first-mover advantage, making it worthwhile to invest heavily to accelerate the development of a new drug. In the absence of such an effect, it may be preferable to be the second in the market, if that can provide an opportunity to develop a drug with slightly better attributes (e.g., requiring less frequent dosing or having fewer side effects) than those of the first mover.[6]

In this paper, we focus on a particular therapeutic class, namely the H_2-antagonist antiulcer drugs, which includes four competing products: Zantac (manufactured by GlaxoWellcome), Tagamet (SmithKline-Beecham), Axid (Eli Lilly), and Pepcid (Merck).[7] These four drugs comprise a well-defined market because they all work in roughly the same way — they cause the stomach to produce less hydrochloric acid than it would otherwise. They differ in terms of dosing frequency, side effects, and their interactions with other drugs, but for most patients they could readily be substituted for each other.[8] Our analysis covers the time period from 1977 (when Tagamet was first introduced) through 1993 (the last year when all four drugs were available only by prescription, and faced no generic competition).

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[6]Indeed, as we will see, this appears to be the case with H_2-antagonist antiulcer drugs. Zantac arrived second but with better attributes than first-mover Tagamet, and soon attained a dominant share of the market. For discussions of first-mover advantages in the prescription drug market, see Bond and Lean (1977), and Berndt, Bui, Reilly, and Urban (1995, 1997).

[7]Tagamet (the chemical compound cimetidine) went off patent in May 1994, and Zantac (ranitidine) in July 1997. More recently, the market was enlarged by the introduction of Prilosec, a proton pump inhibitor, which in 1996 became the world's top-selling drug. Here we confine our attention to the period prior to Tagamet patent expiration.

[8]There are many other examples of well-defined pharmaceutical markets. Anti-cholesterol drugs are one, with four major products: Mevacor (Merck), with about half of the market, Pravachol (Bristol-Myers-Squibb) and Zocor (Merck) each with about 20 percent, and Lescol (Sandoz) with about 10 percent. These drugs all do much the same thing (reduce blood cholesterol levels) in much the same way, and while their side effects and interactions differ somewhat, they are all therapeutic substitutes. Sometimes pharmaceutical market boundaries are more ambiguous. An example is painkillers; there are many types, some are more efficacious for certain types of pain than others, and side effects differ. Examples include aspirin, acetaminophen, ibuprofin, and NSAID's, which include naproxin (sold by prescription and over the counter under the brand name Aleve) and Voltaren. While some types of painkillers are used more frequently than others for certain symptoms or conditions, there is considerable spillover. For example, depending on the severity of the pain and the pain tolerance of the patient, a toothache might be treated with any of the painkillers listed above. Hence the boundaries of a "painkiller market" can be difficult to define.

To estimate the strength of category- and brand-specific network effects, we proceed in steps. We first estimate an hedonic price equation that adjusts prices for quality by accounting for the price impacts of objective attributes such as the number of side effects, dosing, etc. We also include lagged sales of a brand and/or a drug type as additional attribute variables. This allows us to measure the importance of a drug's aggregate usage as a component of its value.

We then use the residuals from this hedonic price index as a "quality-adjusted" price, and estimate dynamic diffusion models that explain the evolution of sales at the industry and brand levels. In these diffusion models, the adoption of any drug within the therapeutic class, and the adoption of a particular brand of drug within that class, depends indirectly on drug attributes through the hedonic residuals, as well as on prices and marketing efforts. But rates of diffusion in these models also depend directly on past sales of the therapeutic class and/or the particular brand, reflecting learning and word of mouth effects. Thus variables reflecting past sales can affect rates of diffusion and equilibrium market shares through multiple channels.

We describe our modelling approach in more detail in the next section. Sections 3 and 4 discuss the data and estimation methods. Estimates of the hedonic price equations are presented and discussed in Section 5, and in Section 6 we present the results of estimating the dynamic diffusion models, first at the industry level, and then at the brand level. Section 7 concludes.

2 Modelling Pharmaceutical Demand.

The past sales of a drug can affect its current demand by directly affecting its value to consumers, and by increasing awareness of the drug's existence and thereby accelerating its rate of diffusion. Our modelling approach distinguishes between these two channels, at both the therapeutic class and brand levels.

First, perceptions of a drug's efficacy, safety, and medical "acceptability" are essentially perceptions of its quality. Hence if the use of a drug by others affects these perceptions,

it should affect the drug's quality-adjusted price. This suggests that one could estimate the perceived value of a drug's past sales or market share from a hedonic price regression that includes such a variable in addition to other product attributes. Gandal (1994) and Brynjolfsson and Kemerer (1995) employed such an approach to estimate the magnitude and value of network effects in spreadsheet software programs. Berndt, Cockburn, and Griliches (1996), Cockburn and Anis (1998), and Suslow (1996) have estimated hedonic price indexes of pharmaceutical products, but did not test for the presence of network effects.

Second, as explained earlier, network effects can influence the rate of product diffusion and market saturation. Past sales or market share can have a direct effect on the rate of diffusion through word of mouth and related communication channels. When more people have used a drug, there will be a greater knowledge of its existence and actual attributes, and thus a more rapid response by physicians and patients who are potential adopters. In addition, if market size or market share is indeed a product attribute that affects the quality-adjusted price, there can be indirect effects on the rate of diffusion, and on the ultimate level of market saturation, through price. In particular, a greater acceptance of a drug (measured, say, through a greater level of sales) will imply that the quality-adjusted price is lower, which can make the level of sales at which the market ultimately saturates higher, and also make the rate of new product trials higher. These indirect effects are simply implications of a negative price elasticity of demand.

To isolate and measure these different effects, we first estimate a hedonic price equation for the therapeutic category, using an (unbalanced) panel of prices and attributes for the four H_2-antagonist drugs. Included among those attributes are measures of the numbers of patients that are taking the drug or have taken it during some previous time interval. Thus we can test whether variables that reflect the acceptance of a drug help to explain prices as expected, and we can estimate their relative contribution to perceived value. Also, we employ the residuals of this hedonic price regression as a quality-adjusted price in our dynamic diffusion models.

Next, we estimate dynamic diffusion models for the therapeutic category, and for the four individual brands. These models explain changes in the sales of a drug in terms of adjustment

to a saturation level (which can be estimated), where the adjustment is partly due to the influence of an "installed base" of patients that are using or have used the drug, and partly independent of that base. Furthermore, the installed base can be measured with respect to the entire therapeutic category, and/or with respect to an individual brand of drug. In this way we can estimate the relative importance of category-specific versus brand-specific network effects on the rate of diffusion.

2.1 Hedonic Price Equations.

To model the diffusion process, we require prices that take into account quality variations across products and over time. We employ the well-known hedonic price framework that relates the price of product i at time t, p_{it}, to a set of measured quality characteristics, C_{it}, a set of time dummy variables, Z_t, and a measure of product acceptance computed as a depreciated stock of cumulative patient days of therapy to time t, XS_{it}.

The theoretical literature provides little guidance on the appropriate functional form for estimating quality adjusted prices.[9] Following numerous others, we employ both linear and semi-log specifications. For the linear form, the hedonic price equation is

$$p_{it} = C'_{it}\beta + Z'_t\gamma + \omega \mathrm{XS}_{i,t-1} + \eta_{it}\,, \tag{1}$$

where β, γ, and ω contain parameters to be estimated, and η is a stochastic disturbance term. The depreciated stock of cumulative patient days of therapy to time t is computed as

$$\mathrm{XS}_{it} = \sum_{\tau=0}^{t} (1-\delta)^{t-\tau} X_{i,t-\tau}\,, \tag{2}$$

where δ is a monthly rate of depreciation and $X_{i,t-\tau}$ is sales of patient days of therapy of drug i in month $t-\tau$. In our empirical implementation, we compute XS_{it} in two alternative ways. First, we use eqn. (2) and set $\delta = .05$. Second, we simply replace $\mathrm{XS}_{i,t-1}$ with $X_{i,t-1}$, the previous month's sales.

To obtain measures of quality-adjusted prices for use in the subsequent diffusion analysis,

[9]For a discussion, see Chapter 4 in Berndt (1991) and the references cited therein.

we re-arrange eqn. (1) and compute a quasi-residual as follows:

$$P_{it} = p_{it} - C'_{it}\hat{\beta} - \hat{\omega}\mathrm{XS}_{i,t-1}\,, \tag{3}$$

where $\hat{\beta}$ and $\hat{\omega}$ are parameter estimates. Notice that variations in P_{it} over time and across products net out the impacts of quality differences, including valuations of network effects as measured by the depreciated stock of cumulative patient days of therapy, among drugs and over time.

2.2 Dynamic Diffusion Models.

Given estimates of quality-adjusted prices, we go on to describe the evolution of demand over time. We model demand — at both the industry and brand levels — as diffusion processes. We begin with a model of product diffusion for a therapeutic category, based on a modified version of the Bass (1969) model. Next we show how this model can be generalized to describe diffusion at the brand level. The specification of these dynamic diffusion models is not derived from a formal dynamic optimization model, in part because of difficulties in dealing with the impacts of moral hazard (due to insurance) and principal-agent issues (the physician-patient relationship). Nonetheless, models of this kind have been widely used in marketing studies of new product diffusion, and will allow us to distinguish among alternative sources of sales growth.[10]

2.2.1 Diffusion of a Therapeutic Class.

We work with models that are based on a differential equation for total sales of a drug that has the following general form:

$$\frac{dX_t}{dt} = \alpha f(X_t^* - X_t) + \beta X_t \cdot g(X_t^* - X_t)\,, \tag{4}$$

with f', $g' > 0$, and $f(0) = g(0) = 0$. Here X_t is total monthly H_2-antagonist patients at time t, and X_t^* is the market saturation level, i.e., the level of sales reached in equilibrium, and α

[10]For an overview of diffusion models of this type and their application, see Mahajan and Muller (1979) and Mahajan, Muller, and Bass (1990).

and β could in turn be functions of prices and advertising levels. Total H_2-antagonist sales X_t is simply the sum of the X_{it}. The first term on the right-hand side of eqn. (4) represents sales growth (towards the saturation level) that is independent of usage of the drug by others. (It may be due purely to advertising, a willingness by physicians to experiment with a new drug, etc.) The second term in these equations represents sales growth that is due to the influence of current sales. Note that the saturation level X_t^* can depend on prices, demographics (such as changing disease prevalence), and "events" such as the approval of a drug for treatment of some condition, and hence will likely vary over time.

Two basic versions of this equation have permeated the literature. The first is the generalized logistic equation:

$$\frac{dX_t}{dt} = \alpha(X_t^* - X_t) + \beta X_t(X_t^* - X_t)\,, \tag{5}$$

and the second is the generalized Gompertz equation:

$$\frac{dX_t}{dt} = \alpha(\log X_t^* - \log X_t) + \beta X_t(\log X_t^* - \log X_t)\,. \tag{6}$$

If $\alpha = 0$ and X_t^* is constant, the solutions to both of these equations are S-shaped "saturation" curves, where sales begin increasing slowly, then accelerate, and finally level out as X_t approaches X^*. If $\alpha > 0$, sales can accelerate faster early on, because sales growth is not dependent solely on the current level of sales. If X_t^* is not constant, i.e., the saturation level is varying over time (perhaps in response to changing prices, medical information, or demographics), sales approach a moving target.

For estimation purposes, we need discrete-time versions of these equations. We will work with the following modified variations of these diffusion processes:

$$\Delta X_t = (X_t^* - X_{t-1})(\alpha_1 + \alpha_2 \mathrm{XN}_{t-1})\,, \tag{7}$$

and

$$\Delta X_t = (\log X_t^* - \log X_{t-1})(\alpha_1 + \alpha_2 \mathrm{XN}_{t-1})\,, \tag{8}$$

where the network variable XN_{t-1} is measured alternatively by the level or log of the previous month's sales, X_{t-1}, or by cumulative depreciated sales, XS_{t-1}. Here, α_1 captures sales

growth that is independent of current or recent usage of the drug by others, and α_2 captures sales growth driven by network effects, i.e., usage of the drug by others. Also, we generalize these models and estimate versions in which $\Delta \log X_t$ is the left-hand side variable.

We must also model the (unobserved) market saturation level, X_t^*. In eqn. (7), X_t^* appears in level form, and we model it by:

$$X_t^* = (b_0 + b_1 \text{GERD}_t + b_2 \overline{P}_t)\text{POP}_t \cdot e^{\beta t}, \tag{9}$$

where $\overline{P}_t$ is the average price for all drugs in the therapeutic category at time t, POP_t is U.S. population, and GERD_t is a dummy variable that takes on the value 1 after May 1986, when H_2-antagonist drugs were approved by the Food and Drug Administration for the treatment of gastro-esophageal reflux disease (GERD, a mild version of which is known as heartburn). In eqn. (8), X_t^* is in log form, so we model it by:

$$\log X_t^* = b_0 + b_1 \text{GERD}_t + b_2 \log \overline{P}_t + b_3 \log \text{POP}_t + b_4 \log \text{TIME}_t . \tag{10}$$

Equations (7) and (8) are estimated after substituting in eqns. (9) for X_t^* and (10) for $\log X_t^*$ respectively. Estimates of α_2 measure the importance of network effects in driving the rate of market saturation at the level of the therapeutic class.

2.2.2 Brand-Level Diffusion.

Equations (7) and (8) describe the saturation process for the H_2-antagonist therapeutic category as a whole. We can also use this framework to model the diffusion of individual brands. Potential additional sales for the therapeutic category is $X_t^* - \sum_i X_{i,t} = X_t^* - X_t$, where $X_{i,t}$ is sales of brand i at time t. Hence if network effects occur at the brand level, the logistic equation (5), for example, would be replaced by the following set of equations for the four brands:

$$\frac{dX_{i,t}}{dt} = f_i(P_{i,t}/\overline{P}_t, A_{i,t}/\overline{A}_t)(X_t^* - X_t)(1 + \phi_i \log X_{i,t-1}), \tag{11}$$

for $i = 1, ..., 4$. The function $f_i()$ describes how the rate of diffusion depends on relative prices and marketing levels. Here $P_{i,t}$ is the quality-adjusted price of brand i, $\overline{P}_t$ is the

quality-adjusted average price for all drugs in the therapeutic category (as before), and $A_{i,t}$ and $\overline{A}_t$ are the corresponding levels of marketing effort.

More generally, we could allow for network effects at both the brand and category levels:

$$\frac{dX_{i,t}}{dt} = f_i(P_{i,t}/\overline{P}_t, A_{i,t}/\overline{A}_t)(X_t^* - X_t)(1 + \phi_i \log X_{i,t-1})(1 + d_i \log X_{t-1}). \qquad (12)$$

The ϕ_i's and d_i's measure the relative importance of brand-specific and industry-level network effects respectively on the rate of diffusion of brand i. We estimate the following discrete-time version of this equation:

$$\begin{aligned} \Delta X_{it} &= (\log X_t^* - \log X_{t-1})(1 + d_0 \log X_{t-1}) \cdot \\ &\quad \left(a_i + \gamma_1 \frac{\overline{P}_t}{P_{it}} + \gamma_2 \frac{\text{MINSTK}_{it}}{\overline{\text{MINSTK}}_t}\right)(1 + \phi_i \log X_{i,t-1}) \end{aligned} \qquad (13)$$

where MINSTK_{it} is the cumulative depreciated stock of detailing minutes (marketing efforts) to physicians for drug i, and the superbar denotes arithmetic mean. In this equation, d_0 measures industry-level network effects, and ϕ_i measures the brand-specific effect for brand i. In most of the regressions presented below, we constrain the ϕ_i's to be the same across all brands. However, we also test whether the ϕ_i for Tagamet is significantly different from that for the other three brands (and we find that it is not).

To implement estimation, for each brand i we form the vectors X_i with components that begin at different time periods for each i (e.g., August 1977 for Tagamet, July 1983 for Zantac, etc.). We stack the X_i's into a vector X which comprises our unbalanced panel.

3 Measurement, Data, and Trends.

The data employed here are described in considerable detail in the Data Appendix of Berndt, Bui, Reiley, and Urban (1997). To aggregate over the various strengths and presentational formulations for each H_2-antagonist, we divide monthly sales in total milligrams of active ingredient by the recommended daily dosage, in milligrams, for duodenal ulcer treatment. This yields patient days of therapy X_{it}, expressed in millions. By 1993, monthly sales approximated 120 million patient days of therapy, which is roughly equivalent to 4 million

patients. Total revenue from sales of drug i in month t is divided by X_{it}, thereby yielding nominal price per day of patient therapy. We deflate this nominal price by the Producer Price Index for finished goods (1982 = 1.000) to obtain the real price per day of therapy for drug i, expressed in constant 1982 dollars. In 1993, the average real price of an H_2 patient day of therapy was about \$1.50. Both price and quantity measures refer to sales from wholesalers to retail drug stores.

When computing quality-adjusted average prices for the H_2 aggregate, we weight each of the products on the market at that time by the average patient-day share during the period. These average shares are computed separately for epochs when there were two, three, and four H_2 products on the market.

Marketing efforts are important in the H_2 therapeutic class. Using data from IMS America, we employ as our measure of marketing the number of minutes that physicians in the United States were "detailed" by pharmaceutical sales representatives. In the 1990s, monthly minutes of detailing ranged from about 40,000 to 250,000, varying considerably by product and over time. We construct a cumulative depreciated stock of detailing minutes, MINSTK_{it}, for each brand. This stock is expressed in millions of minutes, and is computed analogously to eqn. (2), with $\delta = .05$.[11] The average of MINSTK_{it} over all four products is computed the same way as the average price, i.e., a weighted average, where the weights are patient-day shares computed separately for epochs when there were two, three, and four H_2 products on the market.

For quality characteristics of each drug, a number of measures are available. DOSAGE is the number of tablets per day required to attain the recommended daily consumption of the active ingredient. When Zantac was introduced in 1983, it offered a twice-a-day dosage, in contrast to the incumbent Tagamet's four-times-a-day version. Lower DOSAGE is generally thought to indicate greater quality, for patient compliance is typically improved with lower daily DOSAGE. Note that the DOSAGE variable changes over time as manufacturers obtained FDA approval to market lower-dosage versions, which ultimately became available

[11]This value for δ approximates that estimated in Berndt et al. (1997) and King (1997).

in once-a-day formulations.

H_2-antagonist drugs have also competed on the basis of differing medical conditions for which the product has obtained FDA marketing approval; these are called approved indications. Zantac was the first H_2-antagonist to obtain approval for the GERD (gastro-esophageal reflux disease) indication, a relatively common ailment whose symptoms vary from mild heartburn to very intense pain. Although all four H_2-antagonists had obtained approval at product launch date for active duodenal ulcer treatment, FDA approval times varied for active gastric ulcer treatment, duodenal ulcer maintenance treatment, and stress ulcer prophylaxis. We compute the SUMATT variable as the sum of the indications, other than GERD and active duodenal ulcer treatment, for which the H_2 drug had obtained FDA approval.

Finally, an important quality attribute of prescription drugs is the extent to which they might interact adversely with other medications. This is particularly important for the elderly population, who often simultaneously take several medications. The bodily absorption of Tagamet, the first H_2 entrant, involved a metabolic process that adversely affected a number of other medications, some of them used for treatment of common conditions such as those involving blood coagulation, anxiety, and asthma. For each of the four H_2-antagonists we construct a variable named INTER that sums up the number of major drugs with which that H_2-antagonist had adverse interactions, as reported in annual editions of *Physicians' Desk Reference*. By the end of our sample, in late 1993, Tagamet had registered ten adverse interactions, while Zantac, Pepcid, and Axid had either zero or one.

The construction of other variables is as follows. TIME is a time counter taking on the value of one in the first month of the sample time period, August 1977, and then proceeding with the passage of time. The U.S. population data was taken from the U.S. Census Bureau web site, www.census.go, and is expressed in millions.

Growth of H_2-antagonist industry sales was remarkably steady over the 1977–93 time period, averaging about 15 percent per year. Quantity data for the four H_2 drugs are given in Figure 1. Although Tagamet was the pioneer and only H_2 drug from 1977 until Zantac entered in July 1983, Zantac captured a significant market share very rapidly — almost 25

percent within the first year. Total industry sales continued to increase following the entry of Zantac, but soon after Zantac's entry sales of Tagamet began to fall, peaking at about 46 million patient days in April 1984. Tagamet's share continued to decline when Pepcid entered in October 1986, but Pepcid was less successful than Zantac; Pepcid's market share one year after its entry was only about 8 percent. By January 1988, Zantac sales overtook those of Tagamet, and at about the same time (April 1988), Axid entered. As the fourth entrant, however, Axid faced considerable competition, and one year after its launch, its market share was only about 4 percent. By the end of our sample in May 1993, Zantac held about 55 percent of the quantity market share, Tagamet 21 percent, Pepcid 15 percent, and Axid 9 percent.

In terms of (quality-unadjusted) prices, after original entry until it faced competition from Zantac, Tagamet gradually decreased its real price from about \$1 to \$0.80 per day. As shown in Figure 2, not only did Zantac enter with a considerable price premium over Tagamet, but thereafter prices of *both* Zantac and Tagamet rose with time, although Tagamet's price increased more rapidly. By the end of the sample, the Zantac price premium had narrowed from about 56 percent to about 25 percent. Prices of the third and fourth entrants, Pepcid and Axid, generally fell in between those of Zantac and Tagamet.

4 Estimation.

The data employed in the empirical analysis of the hedonic and brand diffusion models constitute an unbalanced panel, while those for the entire H_2 therapeutic class are a monthly time series. We estimate the parameters of the hedonic price in eqn. (1) by ordinary least squares, and compute White heteroscedasticity-robust standard errors. The diffusion models characterizing the entire H_2 therapeutic class, eqns. (7) and (8), are nonlinear in the parameters, so we estimate them using nonlinear least squares. Since the share weights of the individual drugs are constant arithmetic means within each epoch, we treat the industry average price variable as exogenous.

The brand-specific diffusion model of eqn. (13) is also nonlinear in the parameters and

variables. We treat the brand-specific price and marketing variables as endogenous, and estimate the over-identified equation by limited information maximum likelihood (LIML).[12] The excluded exogenous variables are the log of average quality-adjusted hospital price; the number of firms in the market; the producer price indexes for intermediate goods, cardiovascular, antidepressant, and antihypertension therapies; the stock of journal pages, journal expenditures, minutes, and detailing visits by the four H_2 manufacturers on all other products; age of product; ratio of average hospital price to hospital price of drug j (quality-adjusted); and industry wage rate. Details on these variables are in Berndt, Bui, Reiley, and Urban (1997). For purposes of comparison, we also estimate parameters in eqn. (13) by nonlinear least squares.

5 Hedonic Price Models.

Table 1 shows the results of estimating linear and semi-log hedonic price equations for our unbalanced panel of four drugs. All of the regressions include annual and quarterly time dummies (not shown). These dummies are highly significant, and show that real, quality-adjusted prices fell from 1977 through 1981, and then rose gradually through 1993.

We work with four basic attribute variables, whose construction and interpretation is discussed in Section 3: GERD, SUMATT, INTER, and DOSAGE. As can be seen from the table, GERD, INTER, and DOSAGE are all highly significant and have the expected signs; SUMATT is usually insignificant, and has the wrong sign.[13]

Each equation also has one or two variables that are intended to identify and measure the effects of past sales. The variables we consider include the quantity of sales for the particular drug in the previous month, the quantity of sales for the entire therapeutic category in the previous month, and the corresponding depreciated stocks of sales over the preceding two years. This last variable, XS_{it} at the brand-specific level and XS_t at the therapeutic-category level, is calculated using a monthly depreciation rate of 5 percent. As can be seen

[12]For a discussion of LIML and its advantages over other estimators, see Staiger and Stock (1997).

[13]This is not surprising in view of the fact that much prescribing is "off-label," permitted but not approved by the FDA.

from Table 1, the brand-specific variable X_i or XS_i is always positive and highly significant in both the linear and logarithmic versions. Sales or the depreciated stock of sales at the therapeutic-category level, X and XS, however, are mostly insignificant. We infer from this that the use of a drug by others affects its valuation, and that this effect operates at the brand-specific level.

To obtain some idea of the magnitude of this network effect, consider column 1 in part A of Table 1, where the coefficient on lagged quantity is about .0039. In the months prior to Zantac's introduction in August 1983, Tagamet had monthly sales of about 40 million patient days. Had this sales figure been about 10 million (25 percent) less, the contribution of the network effect to Tagamet would have fallen by about \$0.04 (i.e., $10 \times .0039$), or about 5 percent of its approximately 75-cent price at that time. This suggests a brand-specific valuation elasticity of about 0.2 (.05/.25), positive but modest.

Figure 3 shows quality-adjusted real prices for the four drugs, using column 1 of Table 1A. Note that at the time of Zantac's entry in 1983, its quality-adjusted price was close to that of Tagamet. This can help us understand the pricing of Zantac. Ignoring quality differentials, Zantac was priced higher than Tagamet by about 62 cents (in 1982 dollars). One might argue that Zantac entered the market at a higher price to signal higher quality. The drug indeed had quality advantages over Tagamet, in particular fewer interactions and less frequent dosing. However, it also had a disadvantage insofar as Tagamet's installed base gave that drug a perceived value differential. We find that the net quality adjustment to Zantac's price at its entry was about 45 to 70 cents, which accounts for most of the observed actual price difference. Note from Figure 3 that over time, Zantac gains in price advantage as its usage, and the associated component of value, grows.

6 Diffusion Models.

We now turn to the models of product quality diffusion. We begin with models for diffusion at the industry (therapeutic class) level, and then turn to brand-specific models. In both cases, we use the hedonic price quasi-residuals constructed from eqn. (3), based on parameter

estimates from column 1 in Table 1A (linear) or 1B (logarithmic). We also estimated the diffusion models using the residuals from other models in Tables 1A and 1B, and the results were qualitatively unchanged.

6.1 Industry-Level Models.

Results from estimating industry-level diffusion equations are presented in Tables 2A and 2B. In Table 2A, prior usage is measured by $\log(X_{t-1})$, whereas in Table 2B it is measured by XS_{t-1}. (We also estimated these models using $\log \mathrm{XS}_{t-1}$ and X_{t-1} as measures of prior usage and obtained similar results.) In both tables, the estimates in columns 1 and 2 are based on (7) and (9), in which the saturation X_t^* enters in level form, whereas the estimates in columns 3 through 6 are based on eqns. (8) and (10), where X_t^* enters in logarithmic form. We report results for two dependent variables, ΔX_t and $\Delta \log X_t$. In all cases, the parameter α_1 measures sales growth (towards the saturation level) that is independent of drug usage by others, and α_2 represents sales growth that is due to previous sales.

With the exception of Model (6), both α_1 and α_2 are statistically significant, and α_2 is positive, implying that past sales indeed affect the continued growth of sales. Of course we cannot determine from this whether the network effect is operating at the industry and/or brand-specific levels since we are fitting aggregate industry sales. Also, note that this network effect relates to the rate of diffusion, and is distinct from the brand-specific effect on valuation that we observed in our hedonic price equations.

The parameter estimates also provide evidence on the long-run price elasticity of demand for the therapeutic category. This elasticity is found by setting $\Delta X_t = 0$, i.e., from the parameters of the equation for the saturation level, X_t^*. For Models (1) and (2), we compute the elasticity at the point of means, and in Models (3) – (6), the elasticity is given by the coefficient b_2. We find that at the industry level demand is inelastic, with the quality-adjusted price elasticity in the range of –0.10 to –0.55.

Estimates on the GERD indicator variable are in most cases positive but not significant. Since GERD was also an explanatory variable in the hedonic equation (where it was significantly positive), its effects may be captured primarily by $\overline{P}_t$. In Models 3 and 4, the

population elasticity is implausibly high (elasticities ranging from about 8 to 12), and not surprisingly this elasticity falls dramatically in Models 5 and 6 when log(TIME) is added as a regressor.

To assess the ability of these models to replicate the evolution of industry sales, we ran dynamic simulations, in which quality-adjusted price, population, and approval for GERD are all exogenous, and the sales level, X_t, is solved recursively from month to month. Figure 4 plots the results of this dynamic simulation for Model (1) in Table 2B. It shows the actual level of sales in millions of patients (the fluctuating line), the simulated saturation level (the relatively smooth curve), and the simulated level of sales. Note that the model predicts saturation to occur in about three years, whereas actual sales took about four years to saturate (assuming our estimates of the saturation level are correct). Simulations of the other models typically resulted in similar rapid rates of saturation. To assess the importance of α_2, we set it to one-half its estimated value, and then performed another dynamic simulation. Note from Figure 4 that the resulting simulated rate of saturation is slower.

As can be seen from Figure 4, the estimated saturation level X_t^* grows four-fold from 1977 to 1993, i.e., more than 9 percent per year, much faster than the underlying population growth rate. What might account for this? One possibility is that the perceived long-term safety of using an H_2 drug depends not only on how many people have taken the drug, but also on *how long* the drug has been in use, particularly for patients with chronic conditions requiring maintenance therapy. Hence longer usage increases the perceived safety for a large potential population, and thereby increases the saturation level.

6.2 Brand-Level Models.

Tables 3A and 3B show the results of estimating our brand-level diffusion models, by non-linear least squares and limited information maximum likelihood (LIML), respectively. These models are all multi-product variations of a generalized Gompertz equation. The models differ in terms of whether time is included as an explanatory variable for the saturation level, and whether the parameter for the brand-level network effect is constrained to be the same across all brands. Hence all these models are variations of equation (13).

Note that in these models the rate of diffusion depends on potential sales growth up to the saturation level ($\log X_t^* - \log X_{t-1}$), on previous sales of all drugs in the therapeutic category ($d_0 \log X_{t-1}$), and on previous sales of the particular brand ($\phi_j \log X_{j,t-1}$). Also, the rate of saturation can depend on relative prices and on relative marketing levels. The magnitudes of these latter effects are measured by the parameters γ_1 and γ_2, respectively. In all these models, prices affect sales in two ways. First, as in the industry-level diffusion models discussed earlier, individual brand prices affect the average quality-adjusted price for the therapeutic category, $\overline{P}_t$, which in turn affects the saturation level. Second, *relative* prices can affect the rate of diffusion, as can relative marketing efforts. We would expect that if the price of a particular brand is higher than the average price, this would reduce the rate of trials for that brand. We would also expect that the higher the rate of marketing (in this case measured through minutes of detailing), the greater would be the rate of trials for the brand.

The NLS and LIML estimates are very similar. We again find network effects associated with the rate of diffusion to be significant. Now we can separately identify significant network effects at both the therapeutic category and brand levels — the parameters d_0 and ϕ_j are all positive and significant. Unlike the purely brand-specific network valuation effects observed in the hedonic price equations, the rate of diffusion for a brand is affected by prior sales of both the brand and the entire therapeutic category. A comparison of Model 1 with Model 3, and Model 2 with Model 4, shows that the brand-level network effect is the same for Tagamet as for Zantac, Pepcid, and Axid; each of these ϕ_j estimates is about 0.4. Estimates of the impact of the H_2 therapeutic class network effect on brand diffusion range from about 0.3 (when TIME is included) to 0.5 – 0.9 (TIME excluded).

We find relative marketing to be significant (γ_2 is always positive and significant), but not relative price (γ_1 is positive as it should be, but always statistically insignificant). We can also determine industry average price elasticities of demand from these brand-level models; that elasticity is given by the coefficient b_2. Observe that it is about –0.35, which is in the range of estimates that we obtained for the industry-level models.

LIML estimates displayed in Table 3B are broadly comparable to those of Table 3A,

with the exception of γ_2, which, while still positive, is no longer significant. The fit of the equations for the three other endogenous variables (the advertising and price ratios and the log of average quality-adjusted price) was high (R^2 ranging from 0.70 to 0.97). Finally, Hausman specification tests could not reject the null hypothesis of exogenous regressors.

7 Conclusions.

Our purpose in this paper has been to identify the distinct ways in which network effects influence the demands for prescription pharmaceuticals, and to obtain empirical estimates of their importance. We have focused on the case of H_2 antagonists employed for antiulcer/heartburn treatments, but our approach could be applied to other well-defined therapeutic categories of prescription drugs. It could also be applied to other products for which information about efficacy and safety is conveyed by the usage of others.

Our research is ongoing, and results to date are therefore best viewed as preliminary. We find that distinct network effects appear to operate at both the brand-specific and the entire H_2-class level, although the latter is only relevant for the rate of diffusion. When we take our estimated models and simulate them by solving them dynamically beginning at different starting dates, we obtain results implying that demand reaches saturation levels within three years, a finding we view with caution.

The contrast between the results of Tables 1 and 3 has important strategic implications. Our hedonic price equations suggest that pioneering firms benefit (in terms of consumer valuation) by being first to market and establishing a large installed base before another firm enters. On the other hand, our results from estimating the brand-level diffusion models suggest that rates of diffusion can be accelerated by both the brand-specific and therapeutic category installed bases, and that later entrants can reduce their disadvantage by using prices and advertising to accelerate diffusion. A firm may even choose to delay entry if that provides an opportunity to introduce a product embodying better quality attributes.

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Table 1. Hedonic Price Equation

A. Dependent Variable = P_{jt}

	(1)	(2)	(3)	(4)	(5)	(6)
Const.	1.3876	1.3688	1.3841	1.3704	1.3924	1.3867
	(28.39)	(29.52)	(28.85)	(28.87)	(24.75)	(24.56)
GERD	0.1649	0.2278	0.1634	0.1776	0.2280	0.1778
	(14.96)	(20.71)	(14.94)	(16.24)	(20.78)	(16.21)
SUMATT	–0.0195	0.0157	–0.0211	–0.0157	0.0159	–0.0156
	(–1.82)	(1.61)	(–1.94)	(–1.48)	(1.64)	(–1.47)
INTER	–0.0407	–0.0378	–0.0407	–0.0443	–0.0378	–0.0443
	(–27.48)	(–22.57)	(–27.54)	(–26.20)	(–22.57)	(–26.14)
DOSAGE	–0.1279	–0.1183	–0.1277	–0.1196	–0.1184	–0.1197
	(–11.37)	(–11.20)	(–11.57)	(-10.91)	(–11.14)	(–10.91)
$X_i(-1)$	$.3947\times10^{-2}$		$.4072\times10^{-2}$			
	(8.96)		(9.15)			
XS_i				$.2040\times10^{-3}$		$.2037\times10^{-3}$
				(8.35)		(8.33)
$X(-1)$		$-.7140\times10^{-3}$	$-.2349\times10^{-2}$			
		(–0.65)	(–2.18)			
XS					$.3202\times10^{-3}$	$.2338\times10^{-3}$
					(0.80)	(0.60)
R^2	.967	.960	.967	.966	.960	.966
Zantac Advan.	\$0.443	\$0.614	\$0.424	\$0.705	\$0.569	\$0.421

Note: All regressions include annual and quarterly time dummies; NOB = 441; t-statistics (from heteroscedasticity-consistent standard errors) in parentheses. Zantac Advan. is the estimated price advantage of Zantac at its entry in July 1983 explained by attribute differences with Tagamet. (The actual deflated price difference was \$0.615.)

Table 1. Hedonic Price Equation (continued)

B. Dependent Variable = $\log P_{jt}$

	(1)	(2)	(3)	(4)	(5)	(6)
Const.	0.5233	0.5161	0.5214	0.5174	0.5280	0.5262
	(11.60)	(11.74)	(11.66)	(11.61)	(10.33)	(10.20)
GERD	0.1173	0.1388	0.1165	0.1224	0.1388	0.1226
	(14.76)	(20.39)	(14.71)	(15.96)	(20.40)	(15.90)
SUMATT	$-.7043\times10^{-2}$	$.4825\times10^{-2}$	$-.7907\times10^{-2}$	$-.5229\times10^{-2}$	$.5018\times10^{-2}$	$-.5163\times10^{-2}$
	(–0.85)	(0.67)	(–0.94)	(–0.64)	(0.70)	(–0.63)
INTER	–0.0271	–0.0261	–0.0271	–0.0282	–0.0261	–0.0282
	(–24.53)	(–22.87)	(–24.54)	(–22.95)	(–22.85)	(–22.87)
DOSAGE	–0.1595	–0.1561	–0.1594	–0.1567	–0.1563	–0.1567
	(–15.21)	(–15.27)	(–15.35)	(-15.09)	(–15.20)	(–15.08)
$X_i(-1)$	$.1343\times10^{-2}$		$.1409\times10^{-2}$			
	(4.32)		(4.49)			
XS_i				$.6600\times10^{-4}$		$.6581\times10^{-4}$
				(3.91)		(3.90)
$X(-1)$		$-.6752\times10^{-3}$	$-.1241\times10^{-2}$			
		(–0.80)	(–1.46)			
XS					$.1529\times10^{-3}$	$.1250\times10^{-3}$
					(0.51)	(0.42)
R^2	.970	.969	.970	.970	.969	.970
Zantac Advan.	\$0.545	\$0.593	\$0.598	\$0.603	\$0.556	\$0.457

Note: All regressions include annual and quarterly time dummies; NOB = 441; t-statistics (from heteroscedasticity-consistent standard errors) in parentheses. Zantac Advan. is the estimated price advantage of Zantac at its entry in July 1983 explained by attribute differences with Tagamet. (The actual deflated price difference was \$0.615.)

Table 2A. Industry Diffusion Models

	(1)	(2)	(3)	(4)	(5)	(6)
Dep. Var.	ΔX_t	$\Delta \log X_t$	ΔX_t	$\Delta \log X_t$	ΔX_t	$\Delta \log X_t$
α_1	0.7485	0.4212	0.9840	0.5096	1.6448	1.2260
	(13.61)	(11.37)	(9.08)	(8.64)	(11.79)	(17.23)
α_2	0.5361	0.0929	0.5316	0.1739	1.3900	0.0451
	(9.95)	(2.47)	(8.08)	(4.68)	(7.84)	(0.58)
b_0	0.0055	0.0046	–58.2410	–67.1355	–10.5002	–13.3757
	(10.59)	(5.26)	–7.86)	(–7.92)	(–2.71)	(–3.25)
b_1 (GERD)	$.2738\times10^{-3}$	$.2757\times10^{-3}$	0.0296	–0.0291	–0.0057	0.0135
	(1.86)	(1.27)	(0.41)	(–0.35)	(–0.20)	(0.46)
b_2 (PRICE)	–0.0014	$-.7672\times10^{-3}$	–0.1047	–0.2915	–0.4432	–0.2409
	(–2.99)	(–2.02)	(–0.33)	(–0.83)	(–3.38)	(–2.30)
b_3 (POP)			10.7425	12.3897	1.3952	1.9871
			(7.77)	(7.83)	(1.90)	(2.55)
b_4 (TIME)					0.8071	0.6978
					(22.36)	(25.35)
β	0.0074	0.0071				
	(8.82)	(5.82)				
R^2	.585	.427	.314	.313	.600	.644

Note: In each model, the dependent variable is ΔX_t or $\Delta \log X_t$. Models (1) and (2):

Dep. Var. $= (X_t^* - X_{t-1})(\alpha_1 + \alpha_2 \log X_{t-1})$ and $X_t^* = (b_0 + b_1 \text{GERD}_t + b_2 \overline{P}_t)\text{POP}_t\, e^{\beta t}$

Models (3) – (6):

Dep. Var. $= (\log X_t^* - \log X_{t-1})(\alpha_1 + \alpha_2 \log X_{t-1})$ and $\log X_t^* = b_0 + b_1 \text{GERD}_t$
$+ b_2 \log \overline{P}_t + b_3 \log \text{POP}_t + b_4 \log \text{TIME}_t$

Numbers in parentheses are t-statistics from heteroscedasticity-consistent standard errors. In models (1) and (2), the estimated long-run price elasticity of demand, computed at the point of means, is -0.459 and -0.251 respectively.

Table 2B. Industry Diffusion Models

	(1)	(2)	(3)	(4)	(5)	(6)
Dep. Var.	ΔX_t	$\Delta \log X_t$	ΔX_t	$\Delta \log X_t$	ΔX_t	$\Delta \log X_t$
α_1	0.062	0.2369	-0.0994	0.0821	0.0520	1.1203
	(0.09)	(4.16)	(-2.27)	(2.89)	(0.33)	(8.53)
α_2	0.0290	0.0069	0.0685	0.0276	0.0704	0.0041
	(11.87)	(3.87)	(15.53)	(10.02)	(12.08)	(1.02)
b_0	0.0061	0.0053	–43.2820	–43.7844	–19.6905	–13.5151
	(12.08)	(6.54)	(–11.91)	(–8.93)	(–3.93)	(–3.37)
b_1 (GERD)	$.3114\times10^{-3}$	$.3084\times10^{-3}$	0.1336	0.1287	0.0382	0.0146
	(1.90)	(1.37)	(3.93)	(2.80)	(1.15)	(0.50)
b_2 (PRICE)	–0.0017	–.0012	–0.2032	–0.1254	–0.3409	–0.2483
	(–3.80)	(–1.72)	(–1.09)	(–0.52)	(–2.14)	(–2.33)
b_3 (POP)			8.0204	8.1027	3.2433	2.0146
			(11.79)	(8.84)	(3.29)	(2.66)
b_4 (TIME)					0.5847	0.6960
					(5.99)	(26.64)
β	0.0072	0.0071				
	(8.55)	(5.87)				
R^2	.621	.443	.594	.471	.642	.646

Note: In each model, the dependent variable is ΔX_t or $\Delta \log X_t$. Models (1) and (2):

$$\text{Dep. Var.} = (X_t^* - X_{t-1})(\alpha_1 + \alpha_2 \text{XS}_{t-1}) \text{ and } X_t^* = (b_0 + b_1 \text{GERD}_t + b_2 \overline{P}_t)\text{POP}_t\, e^{\beta t}$$

Models (3) – (6):

$$\text{Dep. Var.} = (\log X_t^* - \log X_{t-1})(\alpha_1 + \alpha_2 \text{XS}_{t-1}) \text{ and } \log X_t^* = b_0 + b_1 \text{GERD}_t + b_2 \log \overline{P}_t + b_3 \log \text{POP}_t + b_4 \log \text{TIME}_t$$

Numbers in parentheses are t-statistics from heteroscedasticity-consistent standard errors. In models (1) and (2), the estimated long-run price elasticity of demand, computed at the point of means, is -0.555 and -0.396 respectively.

Table 3A. Brand-Level Diffusion Models - NLS

	(1)	(2)	(3)	(4)
b_0	−54.6497	−17.2795	−54.5717	−17.4430
	(−18.19)	(−5.07)	(−18.45)	(−4.96)
b_1	$-.6606\times10^{-3}$	0.0127	$-.7152\times10^{-3}$	0.1240
	(−0.03)	(.94)	(−0.04)	(0.92)
b_2	−0.3566	−0.3615	−0.3500	−0.3608
	(−2.08)	(−3.59)	(−2.09)	(−3.62)
b_3	10.1093	2.2377	10.1097	2.7704
	(18.05)	(4.18)	(18.31)	(4.08)
b_4		0.6694		0.6661
		(17.12)		(15.40)
d_0	0.8643	0.2908	0.8635	0.3083
	(10.85)	(2.49)	(10.80)	(2.63)
a_T	−0.9578	−0.9019	−0.9738	−0.8740
	(−1.07)	(−0.98)	(−1.31)	(−0.94)
a_Z	−0.9542	−0.9661	−0.9556	−0.9433
	(−1.05)	(−1.03)	(−1.25)	(−1.00)
a_P	-0.6106	−0.4690	−0.7349	−0.4743
	(−0.60)	(−.42)	(−0.86)	(−0.41)
a_A	-0.9275	−1.0561	−1.0716	−1.0501
	(−0.97)	(−1.05)	(−1.29)	(−0.96)
γ_1	0.5703	0.4026	0.5990	0.4047
	(0.57)	(0.35)	(0.75)	(0.36)
γ_2	.9972	1.5583	.9851	1.5242
	(3.23)	(3.52)	(3.67)	(3.62)
ϕ_1	0.4434	0.4190	0.4486	0.4481
	(14.91)	(7.65)	(13.05)	(3.53)
ϕ_2			0.3420	0.4108
			(3.08)	(6.92)
R^2	.482	.610	.482	.611

Note: The models are: $\log X_t^* = b_0 + b_1\text{GERD}_t + b_2 \log \overline{P}_t + b_3 \log \text{POP}_t + b_4 \log \text{TIME}_t$ and $\Delta X_{jt} = (\log X_t^* - \log X_{t-1})(1 + d_0 \log X_{t-1}) \cdot \text{DUM}_{jt}\left(a_j + \gamma_1(\overline{P}_t/P_{jt}) + \gamma_2(\text{MINSTK}_{jt}/\overline{\text{MINSTK}}_t)\right)(1 + \phi_j \log X_{j,t-1})$ where $j = T$ (Tagamet), Z (Zantac), P (Pepcid), and A (Axid), and DUM_{jt} is a dummy variable that equals 1 for observations on brand j. In models (1) and (2), $\phi_T = \phi_Z = \phi_P = \phi_A \equiv \phi_1$; in models (3) and (4), $\phi_Z = \phi_P = \phi_A \equiv \phi_2 \neq \phi_T$. Numbers in parentheses are t-statistics from heteroscedasticity-consistent standard errors.

Table 3B. Brand-Level Diffusion Models - LIML

	(1)	(2)	(3)	(4)
b_0	−53.6133	−17.2211	−53.4782	−17.3692
	(−19.67)	(−5.63)	(−19.56)	(−5.60)
b_1	0.1215	0.0158	0.0122	0.0154
	(0.53)	(1.05)	(0.53)	(1.03)
b_2	−0.3678	−0.3674	−0.3463	−0.3672
	(−2.33)	(−3.60)	(−2.29)	(−3.57)
b_3	9.9361	2.7280	9.9106	2.7575
	(19.43)	(4.61)	(19.33)	(4.60)
b_4		0.6688		0.6659
		(15.07)		(14.87)
d_0	0.8664	0.2945	0.8656	0.3098
	(6.68)	(1.31)	(6.69)	(1.38)
a_T	−1.1731	−0.9587	−1.1971	−0.9358
	(−0.75)	(−0.38)	(−0.80)	(−0.37)
a_Z	−1.0969	−1.0092	−1.1027	−0.9903
	(−0.74)	(−0.42)	(−0.77)	(−0.41)
a_P	−1.8795	−0.5247	−1.0118	−0.5310
	(−0.51)	(−0.19)	(−0.62)	(−0.19)
a_A	−1.1317	−1.1222	−1.2897	−1.1189
	(−0.53)	(−0.35)	(−0.74)	(−0.36)
γ_1	0.7997	0.4495	0.7955	0.4511
	(0.45)	(0.18)	(0.46)	(0.17)
γ_2	0.9612	1.5674	1.9883	1.5390
	(1.17)	(1.43)	(0.96)	(1.32)
ϕ_1	0.4430	0.4271	0.4484	0.4518
	(11.19)	(2.06)	(11.59)	(1.87)
ϕ_2			0.3148	0.4197
			(0.81)	(1.40)
LL	3165.90	3228.36	3166.36	3228.38

Note: The models are: $\log X_t^* = b_0 + b_1 \text{GERD}_t + b_2 \log \overline{P}_t + b_3 \log \text{POP}_t + b_4 \log \text{TIME}_t$ and $\Delta X_{jt} = (\log X_t^* - \log X_{t-1})(1 + d_0 \log X_{t-1}) \cdot \text{DUM}_{jt} \left(a_j + \gamma_1(\overline{P}_t/P_{jt}) + \gamma_2(\text{MINSTK}_{jt}/\overline{\text{MINSTK}}_t)\right)(1 + \phi_j \log X_{j,t-1})$ where $j = T$ (Tagamet), Z (Zantac), P (Pepcid), and A (Axid), and DUM_{jt} is a dummy variable that equals 1 for observations on brand j. In models (1) and (2), $\phi_T = \phi_Z = \phi_P = \phi_A \equiv \phi_1$; in models (3) and (4), $\phi_Z = \phi_P = \phi_A \equiv \phi_2 \neq \phi_T$. Numbers in parentheses are t-statistics from heteroscedasticity-consistent standard errors.

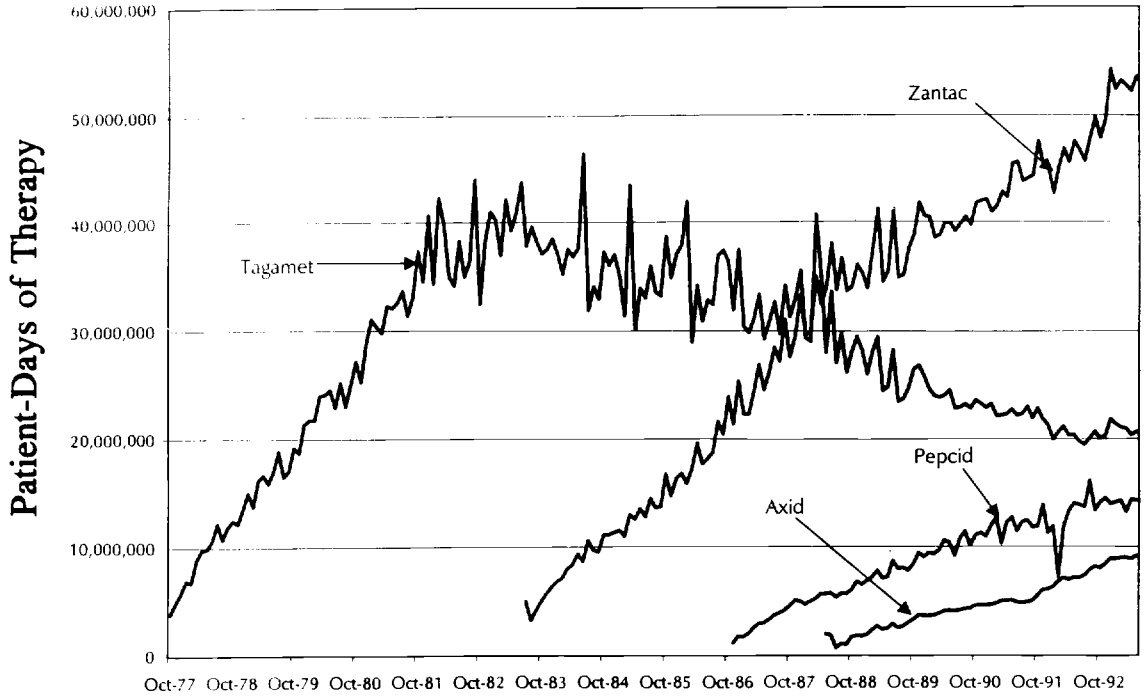

Figure 1: Monthly Sales for H_2 Blockers, 1977 – 1993 (in Patient-Days)

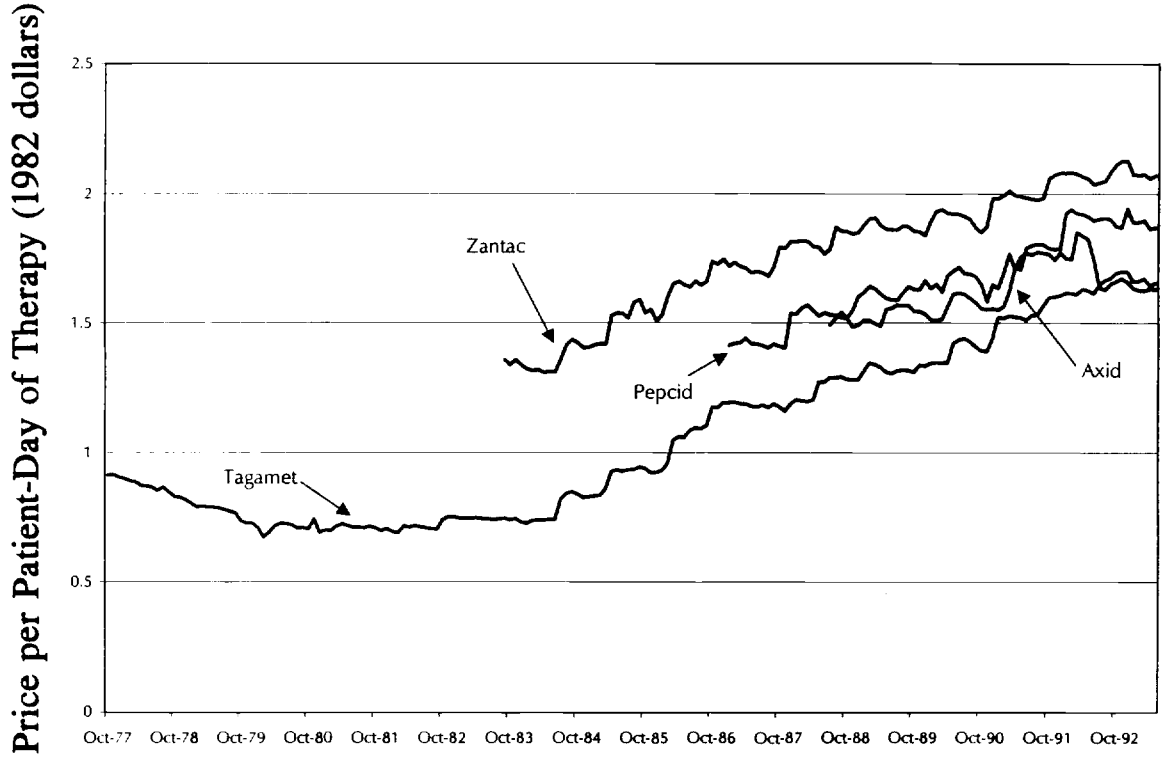

Figure 2: Real Prices of H_2 Blockers, 1977 – 1993

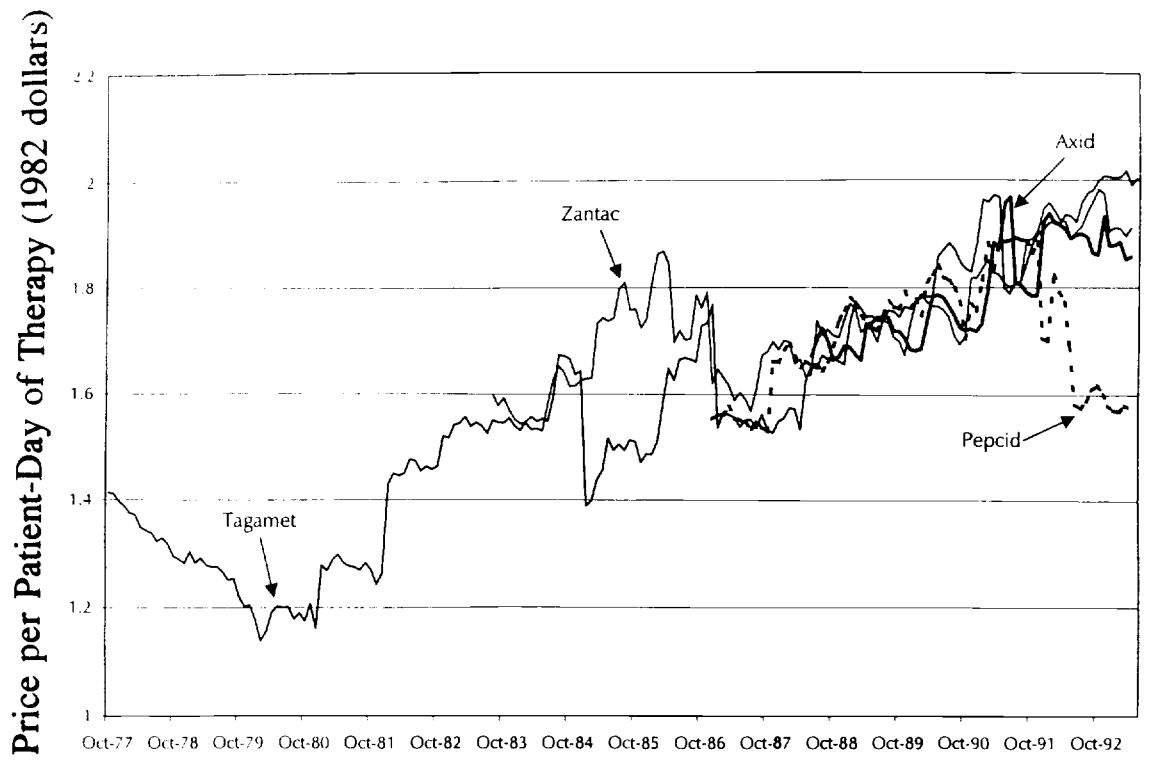

Figure 3: Quality-Adjusted Real Prices for H-$_2$ Antagonist Drugs, 1977–1993

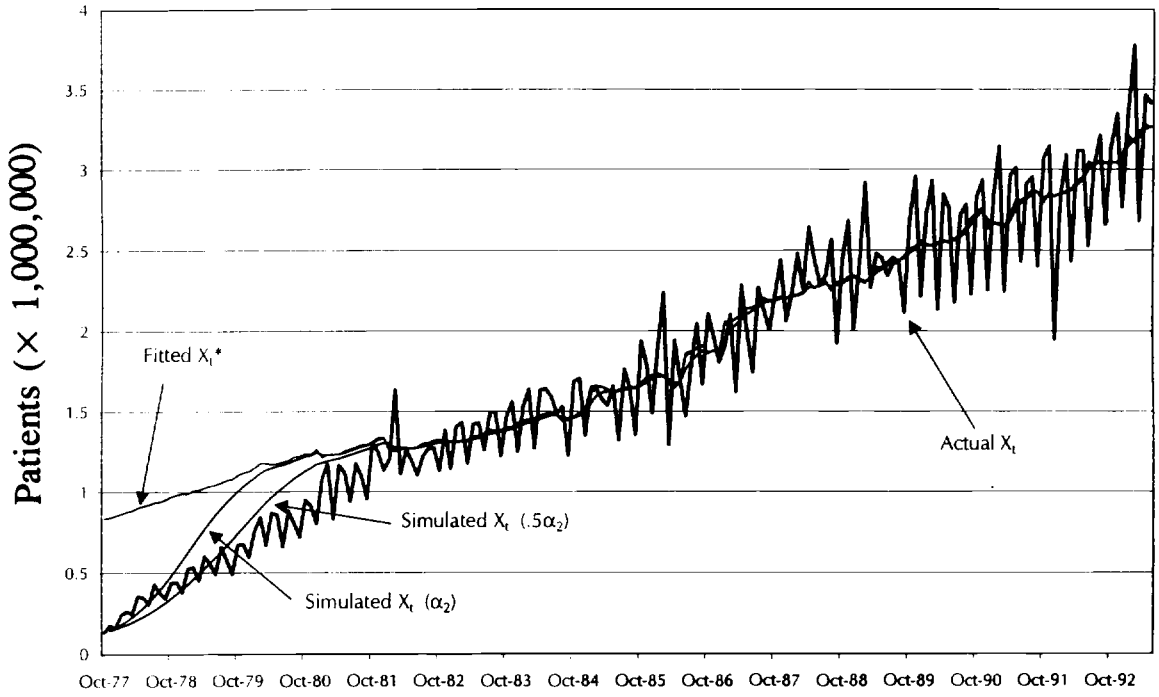

Figure 4: Simulation of Industry-Level Diffusion Model