THE JOURNAL OF INDUSTRIAL ECONOMICS 0022-1821
Volume LI June 2003 No. 2

CONSUMPTION EXTERNALITIES AND DIFFUSION IN PHARMACEUTICAL MARKETS: ANTIULCER DRUGS*

ERNST R. BERNDT,† ROBERT S. PINDYCK,† AND PIERRE AZOULAY‡

We examine the role of consumption externalities in the demand for pharmaceuticals at both the brand level and over a therapeutic class of drugs. Externalities emerge when use of a drug by others affects its value, and/or conveys information about efficacy and safety to patients and physicians. This can affect the rate of market diffusion for a new entrant, and can lead to dominance of one drug despite the availability of close substitutes. We use data for H_2-antagonist antiulcer drugs to estimate a dynamic demand model and quantify these effects. The model has three components: an hedonic price equation that measures how the aggregate usage of a drug, as well as conventional attributes, affect brand valuation; equations relating equilibrium market shares to quality-adjusted prices and marketing levels; and diffusion equations describing the dynamic adjustment process. We find that consumption externalities influence both valuations and rates of diffusion, and that they operate at the brand and not the therapeutic class level.

I. INTRODUCTION

We examine 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 these innovations as largely exogenous, and focus on demand-side factors that affect long-run market saturation, not only for the overall therapeutic class, but also for particular brands within the class. In particular, we examine consumption externalities, i.e., ways in which the demand for a branded pharmaceutical by

*The research leading to this paper was supported by the National Science Foundation through grants to Berndt and Pindyck, and by MIT's Program on the Pharmaceutical Industry. Our thanks to Winnie Choi and Ornsiri Rungruxsirivorn for their outstanding research assistance, and to Iain Cockburn, Clint Cummins, Neil Gandal, Austan Goolsbee, Zvi Griliches, Jerry Hausman, Andrea Shepard, and seminar participants at Columbia University, MIT, Tel Aviv University, and the NBER for helpful comments. This is a shorter version of our NBER Working Paper of the same title, and was initially prepared in honor of Zvi Griliches' 20 years as Director of the NBER Productivity Program.

†Authors' affiliations: Sloan School of Management, Massachusetts Institute of Technology, Cambridge, MA, USA.
email: erberndt@mit.edu
email: rpyndyck@mit.edu
‡Graduate School of Business, Columbia University, New York, NY, USA.
email: pierre.azoulay@columbia.edu

patients and physicians depends on the number of other patients that have taken or are taking the drug.

Consumption externalities arise when the use of a drug by others influences perceptions about its efficacy, safety, and 'acceptability,' and thus affects its valuation and rate of adoption. Unlike computer software and telecommunications systems where consumption externalities stem from direct external benefits, in pharmaceutical markets these externalities are largely informational in nature: The widespread use of a drug may convey information about its safety and efficacy, and, for physicians, may imply 'accepted practice' and hence greater immunity to malpractice lawsuits.[1] For example, the fact that a drug has been widely used may be evidence that it is efficacious relative to its side effects and risks. Or, physicians might conclude that the probability of a malpractice suit is lower when a widely used drug is prescribed, whatever the actual efficacy and risks of the drug.

If they are strong enough, consumption externalities could lead to the dominance of one drug — not necessarily the most efficacious or safest — despite the availability of close substitutes. Consumption externalities also affect the rate at which a new product diffuses into the market: As more people use the product, word-of-mouth communication increases, accelerating the rate at which others become aware of it. In either case, the result can be a market outcome that is inefficient.[2]

We distinguish between externalities that influence consumers' valuations of a drug, and those that influence the rate of diffusion in the market.[3] Consumers' valuations are affected when the use of a drug by others influences its perceived efficacy and safety. One of our goals is to identify and quantify the magnitude of this effect. A second goal is to assess the importance of past sales 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 are substitutes. Thus it is important to distinguish between consumption externalities at two levels. The first is

[1] There is 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). Temin (1980) has shown that physicians do not have well-organized information on the comparative effectiveness and riskiness of substitute drugs, and make decisions based largely on the customary behavior of other doctors.

[2] 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; see, e.g., Scharfstein and Stein (1990) and Bikhchandani, Hirshleifer, and Welch (1998). Goolsbee and Klenow (1999) present evidence of very similar spillover effects in consumers' purchases of home computers. Gandal, Kende, and Rob (2000) estimate a dynamic demand model of technology adoption for compact disc players and CD titles.

[3] Decisions to utilize a drug can be made or influenced by both patients and physicians. We do not try to differentiate their roles in the adoption decision, and include both when we refer to 'consumers'.

with respect to a therapeutic class, e.g., H_2-antagonist antiulcer drugs, SSRI antidepressants, or cholesterol-lowering drugs. We expect that physicians may be more willing to prescribe and patients to take a drug the more the drug's therapeutic class 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 is with respect to a specific brand of drug within a therapeutic class: Physicians and patients may be more willing to use Zantac (as opposed to Tagamet, Axid, or Pepcid) the greater is its 'acceptance,' as measured by market share or cumulative sales.

Although we focus on demand, the issues we examine have broad implications for the structure and performance of pharmaceutical markets, as well as other markets in which buyers decide whether to adopt new products or technologies. For example, consumption externalities may give firms the incentive to compete very aggressively in the early stages of market evolution, to try to win a future position with substantial market power. Even if externalities do not affect consumers' valuations of a product, an initially large market share can lead to 'tipping' by affecting the rate of diffusion: If a product's rate of diffusion depends positively on the number of consumers already using it, the firm with an initial market share advantage could increase that advantage as the market saturates.

When they occur at the brand level, consumption externalities can create an incentive to price low initially and advertise heavily, and later convey market power to the owner of a dominant brand. They also affect the reward for being first to market, making it worthwhile to invest heavily to accelerate R&D. Conversely, when they occur at the therapeutic class level, they can create second-mover advantages, whereby later entrants free-ride on the information and awareness generated by the pioneering brand. If this effect dominates, firms might find it optimal to arrive second on the market, and try to develop a drug with better attributes (e.g., fewer side effects) than those of the first mover.[4]

We focus on a particular therapeutic class, namely the H_2-antagonist antiulcer drugs: Tagamet, Zantac, Pepcid, and Axid. These drugs comprise a well-defined market because they all function in roughly the same way by causing the stomach to produce less hydrochloric acid. They differ in dosing frequency, side effects, and interactions with other drugs, but for most patients they could readily be substituted for each other. Our analysis covers the time period from 1977, when Tagamet was first introduced, through 1993, the year before Tagamet lost patent protection and two years before

[4] 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 prescription drug markets, see Bond and Lean (1977), and Berndt, Bui, Reilly, and Urban (1995, 1997). For an empirical study of pricing strategies in these markets, see Lu and Comanor (1998).

over-the-counter versions of the H_2-antagonist drugs were introduced. Prilosec, a proton-pump inhibitor used to treat similar disorders, was introduced in the United States in 1989. However, until 1995, the FDA required Prilosec to carry a warning on its label concerning safety in long-term treatment, so it was not a strong substitute for the H_2-antagonist drugs.

Because the four drugs were introduced sequentially, this data set allows us to address important issues related to brand diffusion and competition. How important, for example, is the first-mover advantage resulting from an 'installed base' of patients? How does that installed base affect a brand's rate of diffusion, and substitution across brands? What portion of a drug's value can be attributed to brand-level versus therapeutic class-level consumption externalities? We can also examine strategic issues specific to this industry. Zantac was introduced at a higher price than Tagamet and had the disadvantage of being a 'second mover,' but overtook Tagamet in sales after about four years. To what extent was this due to Zantac's better attributes and higher level of marketing?

Our model has three components. First, we estimate an hedonic price equation that accounts for the price impacts of objective attributes such as the number of side effects, dosing, etc. We also include cumulative lagged sales of a brand and/or the therapeutic class as additional attribute variables. This allows us to measure the importance of a drug's past usage, as well as conventional attributes, as components of its current value.

Second, we use the quasi-residuals from this hedonic price index as a quality-adjusted price and, based on data for the last four years of our sample, estimate an equilibrium model of brand shares. During this period we can reasonably expect that all four brands have fully diffused through the market, so we can measure the equilibrium dependence of sales on relative (quality-adjusted) prices and marketing levels.

Third, we estimate a set of dynamic diffusion equations that explains the adjustment of sales to their equilibrium, or saturation, levels. These endogenous saturation levels depend on prices, advertising levels, and population, and thus change over time as these variables evolve. Rates of diffusion for the brands depend indirectly on drug attributes through the hedonic residuals, as well as on prices and marketing efforts. But rates of diffusion 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.

This approach has the virtue that it fits the data quite well: Dynamic simulations of the model yield time paths for brand sales that track the actual time paths very closely. However, this goodness of fit comes at the expense of structural assumptions that we impose to identify key parameters (e.g., that the last four years of our sample represents a period of market equilibrium). This framework is discussed in the next section. Section 3 discusses the data

and estimation methods. Estimates of the hedonic price equations, the equilibrium share model, and the dynamic diffusion equations are presented and discussed in Sections 4, 5, and 6 respectively. Section 7 presents simulation results, and Section 8 concludes.

II. MODELLING PHARMACEUTICAL DEMANDS

As explained above, 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 model, which accounts for these two mechanisms at both the therapeutic class and brand levels, is structured as follows.

First, perceptions of a drug's efficacy, safety, and medical 'acceptability' are essentially perceptions of its quality, so if past sales of a drug affect these perceptions, they should affect the drug's quality-adjusted price. This suggests that one could estimate the perceived value of a drug's past sales from an hedonic price regression that includes a variable such as past sales in addition to other product attributes. Of course, one could argue that the significance of measures of past sales reflects switching costs rather than consumption externalities. Based on discussions with physicians, however, it is our understanding that during the 1977–1993 time frame of our data, most patients were prescribed H_2 drugs for less than a year. Thus the growth in sales reflects a growing influx of new rather than long-term continuing patients. Given the preponderance of short-term treatments, it is unlikely that an observed dependence of price on past sales is due largely to switching costs.

Thus we begin by estimating an hedonic price equation using an (unbalanced) panel of prices and attributes for the four H_2-antagonist drugs. Included among the attributes are measures of the numbers of patients that are taking or have taken the drug in the past.[5] We thereby test whether variables that reflect the acceptance of a drug help to explain prices as expected, and we estimate their contribution to perceived value. Also, we employ the quasi-residuals of this hedonic regression as a quality-adjusted price in the other two components of our model.

Second, using the hedonic quasi-residuals, along with data on brand advertising, we estimate equations for the equilibrium market shares of the four brands. To do this, we use data only for the last 4 years of our sample, when the market was mature and adjustment to equilibrium was largely complete. Because the number of drugs on the market changed during the years prior to this period, we use a multinomial logit model. This restricts the

[5] Gandal (1994) and Brynjolfsson and Kemerer (1995) employed such an approach to estimate magnitude and value of network effects in spreadsheet software programs. Berndt, Cockburn, and Griliches (1996), Cockburn and Anis (2001), and Suslow (1996) have estimated hedonic price indexes of pharmaceutical products, but did not test for the presence of consumption externalities.

equilibrium cross-price elasticities to be the same for drugs with equal shares, but yields partial (i.e., subject to a constant total industry demand) own-price and advertising elasticities that depend on market shares but not on the number of drugs in the market. Using these equilibrium share equations, we calculate *fitted equilibrium shares* for the entire sample period. For example, we calculate what the Tagamet and Zantac equilibrium shares would have been in, say, 1985, when these were the only drugs on the market.

An alternative and less restrictive approach would involve estimating a share model with unadjusted prices and all the attributes. We investigated this alternative, but found that during the equilibrium time period there is simply not enough variability in the attributes to identify parameters. Moreover, we also used a nested model to test whether adding attributes to our baseline specification adds explanatory power, and found that it does not.

Third, we estimate a set of dynamic diffusion equations for the four individual brands. These equations explain changes in the sales of a particular brand in terms of adjustment to that brand's equilibrium share of an industry saturation level (which is 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 is measured both with respect to the entire therapeutic category and with respect to the individual brand. In this way we estimate the relative importance of category-specific versus brand-specific spillover effects on the rate of diffusion.

This three-step approach has the distinct disadvantage that it imposes strong structural assumptions — most notably that we can identify a period of market equilibrium. An alternative approach would be to substitute functional expressions for the brands' equilibrium shares directly into the diffusion equations, and then estimate those equations over the entire sample. We have pursued that approach, but found that it is not possible to precisely identify key parameters. By imposing identifying assumptions, our three-step approach has a number of advantages. First, it lets us measure the importance of spillover effects as a component of perceived value, and in terms of its influence on the rate of product diffusion. Second, we can model the structure of inter-brand competition in a parsimonious way, without the usual problem of having to sacrifice the dynamic aspects of demand. Third, the three parts of the model each provide information regarding a different aspect of demand, and allow us to address questions raised in the Introduction, such as the extent to which Zantac's performance can be attributed to its better attributes and higher rate of advertising.

(i). *Hedonic Price Equation*

We estimate hedonic price equations that relate 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, D_t, and two measures of product acceptance: the depreciated stock of cumulative patient days of therapy of brand i up to time t, XS_{it}, and the corresponding depreciated stock for the therapeutic class as a whole, XS_t. We employ both linear and semi-log specifications. The linear specification is

$$P_{it} = C'_{it}\beta + D'_t\gamma + \omega_1 \mathrm{XS}_{i,\,t-1} + \omega_2 \mathrm{XS}_{t-1} + \eta_{it}, \tag{1}$$

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 is computed as

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

and similarly for XS_t, but using $X_t = \Sigma_i X_{it}$. Here, δ is a monthly depreciation rate and $X_{i,t-\tau}$ is sales of patient days of therapy of drug i in month $t-\tau$. As discussed in Section 3, we set $\delta = .05$.

To obtain measures of quality-adjusted prices, we compute the quasi-residual:

$$P_{it} = p_{it} - C'_{it}\hat{\beta} - \hat{\omega}_1 \mathrm{XS}_{i,t-1} - \hat{\omega}_2 \mathrm{XS}_{t-1}. \tag{3}$$

Note that variations in P_{it} over time and across products net out the impacts of quality differences, including valuations of past sales as measured by $\mathrm{XS}_{i,t}$ and XS_t.

(ii). *Equilibrium Shares*

We use a simple multinomial logit model to describe equilibrium brand shares. Denoting the quantity share of brand i at time t by s^*_{it}, equilibrium shares are given by

$$\log\left(\frac{s^*_{it}}{s^*_{T,t}}\right) = \lambda_{i0} + \lambda_1(p_{it} - p_{T,t}) + \lambda_2(X_{it} - X_{T,t}) + \lambda_3(\mathrm{MINSTK}_{it} - \mathrm{MINSTK}_{T,t}) + \epsilon_{it}, \tag{4}$$

where $i = \mathrm{Z}$ (Zantac), A (Axid), and P (Pepcid), $s^*_{T,t}$ is the equilibrium share of Tagamet, p_{it} is the drug price (the dependent variable in eqn. (1)), X_{it} is the set of drug attributes included in the hedonic equation, and MINSTK_{it} is the depreciated stock of detailing minutes, our measure of marketing. Variation in patient and physician 'tastes' occurs through the error term ϵ_{it}.

Since this demand system includes brand-specific effects λ_{i0}, the λ_2 coefficients have to be identified off longitudinal variation. Unfortunately, there is not enough time-series variation to identify these coefficients during

the equilibrium period. As a result, we estimate the more restricted model:

$$\log\left(\frac{s_{it}^*}{s_{T,t}^*}\right) = a_{i0} + a_1(P_{it} - P_{T,t}) + a_3(\text{MINSTK}_{it} - \text{MINSTK}_{T,t}) + \epsilon_{it}, \tag{5}$$

where P_{it} is the quasi-residual from eqn. (3). Under the assumption that consumers' valuation of attributes do not change over the sample period, then eqn. (5) reduces to eqn. (4).

This parsimonious model imposes restrictions on equilibrium demands — for any two drugs, cross-price elasticities with respect to a third drug can differ only to the extent that the first two have different market shares. In our case these restrictions are less problematic: There are only four products in the market, and they are close substitutes. The difference between any two H_2 drugs is far smaller than the difference between, say, a Ford Escort and a Lexus, so there is less need to use the more complex approach of Berry, Levinsohn and Pakes (1995). Also, note that this demand model does not include an outside good. The reason is that our dynamic diffusion equations explain the adjustment to an *endogenous* saturation level X_t^* (see below), and thus account for consumers' outside treatment options.

(iii). *Saturation Levels*

Given estimates of equilibrium shares, we can determine saturation levels for each brand, i.e., the level of sales that a brand would reach once in equilibrium. These saturation levels vary over time for two reasons. First, the equilibrium market shares on which they depend change as relative quality-adjusted prices and advertising levels evolve. Note that even if nominal prices, attributes, and advertising levels were fixed, equilibrium shares could still change because changing cumulative sales affects quality-adjusted prices. Second, brand saturation levels also depend on the saturation level for sales of the overall therapeutic category, which varies as the population grows and as the average industry price changes.

We denote the industry saturation level by X_t^*, and we model it as a function of the average industry quality-adjusted price $\bar{P}_t$, the total stock of depreciated detailing minutes for the industry MINSTKTOT_t, and population POP_t:

$$\log X_t^* = b_0 + b_1 \log \bar{P}_t + b_2 \log \text{POP}_t + b_3 \log \text{MINSTKTOT}_t \tag{6}$$

Given the equilibrium shares s_{it}^* and this industry saturation level, the saturation level for each brand is just $X_{it}{}^* = s_{it}{}^* X_t{}^*$.

(iv). *Dynamic Diffusion Equations*

The third part of our model is a set of equations describing product diffusion at the brand level, i.e., how sales of each brand, X_{it}, approach the saturation level $X_{it}^* = s_{it}^* X_t^*$. These equations are not derived from a formal dynamic optimization model, in part because of difficulties of dealing with moral hazard (due to insurance) and principal-agent issues (the physician-patient relationship) without transaction-level data. Instead, we adapt a set of models that have been widely used in marketing studies of new product diffusion, in a way that allows us to distinguish among alternative sources of sales growth.[6] Specifically, we work with versions of the generalized logistic equation:

$$\frac{dX_t}{dt} = \alpha(X_t^* - X_t) + \beta X_t(X_t^* - X_t), \tag{7}$$

and the generalized Gompertz equation:

$$\frac{dX_t}{dt} = \alpha(\log X_t^* - \log X_t) + \beta X_t(\log X_t^* - \log X_t). \tag{8}$$

The first term on the right-hand side of eqns. (7) and (8) represents sales growth (towards the saturation level) that is independent of usage of the drug by others. (It may be due to advertising, a willingness of physicians to experiment with a new drug, etc.) The second term represents sales growth that is due to the influence of current sales (and in our specification below, past sales). As discussed above, the saturation level X_t^* will vary over time as prices, demographics, and levels of marketing activity change.

If $\alpha = 0$ and X_t^* is constant, the solutions to both of these equations are S-shaped 'saturation' curves, where sales first increase slowly, then accelerate, and finally level out as X_t approaches X_t^*. If $\alpha > 0$, sales can accelerate faster early on, because sales growth is not dependent solely on current or past sales. If X_t^* is not constant, i.e., the saturation level is varying over time, sales pursue a moving target.[7]

We adapt these diffusion equations by noting that the saturation level for brand i is given by $s_{it}^* X_t^*$, where s_{it}^* is the equilibrium share of brand i (which in turn is a function of relative prices and advertising levels). In order to allow for consumption externalities at both the brand and the therapeutic

[6] For an overview of diffusion models of this type and their application, see Mahajan and Muller (1979), Mahajan, Muller, and Bass (1990), and Geroski (2000).

[7] As a referee pointed out, an S-shaped diffusion curve could also result if consumers' valuations were normally distributed and quality-adjusted prices were declining over time. As shown in Figure 4, however, during this time period quality-adjusted prices were generally rising.

category levels, we estimate the following two discrete-time versions of the continuous-time diffusion processes above:

$$X_{it} - X_{i,t-n} = \left[\log(\hat{s}^*_{it} X^*_t) - \log X_{i,t-n}\right] \cdot \left[C_i + \sum_{k=2}^{12} \theta_k m_{kt} + d_0 \mathrm{XS}_{t-n} + d_1 \mathrm{XS}_{i,t-n}\right] \tag{9}$$

and

$$X_{it} - X_{i,t-n} = \left[\log(\hat{s}^*_{it} X^*_t) - \log X_{i,t-n}\right] \cdot \left[C_i + \sum_{k=2}^{12} \theta_k m_{kt} + d_0 \log \mathrm{XS}_{t-n} + d_1 \log \mathrm{XS}_{i,t-n}\right] \tag{10}$$

The parameters d_0 and d_1 measure the effects of industry-level and brand-specific spillovers, respectively, on the rate of diffusion of each brand. Note that when estimating these equations, we use both the in-sample and out-of-sample *fitted* values of the equilibrium shares, $\hat{s}^*_{it}$. The industry saturation level, X^*_t, is endogenous, and is given by eqn. (6).

III. DATA AND ESTIMATION

Here we briefly summarize the construction of our data set. (Much of our data are described in more detail in the Data Appendix of Berndt, Bui, Reiley, and Urban (1997).)

To aggregate over the various strengths and presentational formulations of each drug, 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, total monthly sales was about 120 million patient days of therapy, which is roughly equivalent to 4 million patients. To obtain the nominal price per day of patient therapy, we divide total revenue from sales of drug i in month t by X_{it}. We deflate this nominal price by the Producer Price Index for finished goods (1982 = 1.00) to obtain the real price for drug i in 1982 dollars. In 1993, the average real price was about \$1.50 per patient day of therapy. Both price and quantity measures refer to sales from wholesalers to retail drug stores, as computed by IMS America.

Our measure of marketing effort is the number of minutes that physicians in the United States were 'detailed' by pharmaceutical sales representatives, obtained from IMS America. In the 1990s, monthly minutes of detailing varied from about 40,000 to 250,000 across products 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 $\delta = .05$, which is approximately the

rate estimated in Berndt *et al.* (1997) and King (1997). Also, we performed a grid search for δ by repeatedly estimating the equilibrium share equations using generalized method of moments estimation (GMM). The GMM objective function is quite flat over values of δ between 0.02 and 0.08, and has two local minima; the value of 0.05 lies midway between those minima.

To compute the quality-adjusted average price for the H_2 class, $\bar{P}_t$, 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. We also compute a *total* level of advertising for the therapeutic class, MINSTKTOT_t, by summing MINSTK_{it} over all four products.

We used several quality characteristics in our hedonic equations. The first, DOSAGE, is the number of tablets normally required per day. When Zantac appeared in 1983, it had twice-a-day dosage, in contrast to Tagamet's four-times-a-day version. Lower DOSAGE implies higher quality, because it leads to greater patient compliance. Note that the DOSAGE variable changes over time as manufacturers obtained FDA approval to market more convenient dosages, which ultimately became once-a-day formulations for all four brands.

These drugs have also differed in terms of the medical conditions for which they obtained FDA marketing approval (the 'approved indications'). Zantac was the first H_2-antagonist to obtain approval for GERD (gastro-esophageal reflux disease), a common ailment whose symptoms vary from mild heartburn to 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 SUMATT as the sum of the indications, other than GERD and active duodenal ulcer treatment, for which the drug had FDA approval.

Another important attribute of prescription drugs is the extent to which they might interact adversely with other medications. For each H_2-antagonist we construct a variable, INTER, that sums up the number of major drugs with which it had adverse interactions, as reported in annual editions of *Physicians' Desk Reference*. By late 1993, Tagamet had registered ten adverse interactions, while Zantac, Pepcid, and Axid had either zero or one.

Finally, we construct a monthly time counter, TIME, starting at one in August 1977, and take U.S. population data from the U.S. Census Bureau web site, www.census.gov (in millions of people).

Figure 1 shows monthly sales of each drug. Although Tagamet was the pioneer and only H_2-antagonist drug on the market for six years, Zantac captured market share rapidly following its entry in July 1983. Total industry sales continued to increase after Zantac's entry, but Tagamet's sales began to fall after peaking at about 46 million patient days in April 1984.

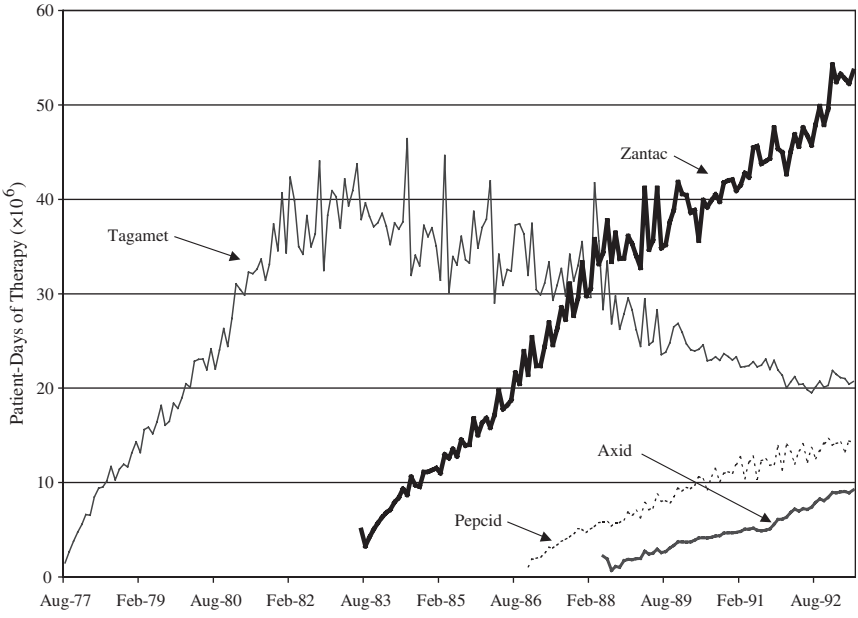

Figure 1
Monthly Sales for H_2-Antagonist Drugs

Tagamet's share continued to decline when Pepcid and Axid entered, but these drugs were far less successful than Zantac; Pepcid's share one year after entry was only about 8 percent, and Axid's 4 percent. By the end of our sample in May, 1993, Zantac held about a 55-percent market share, Tagamet 21 percent, Pepcid 15 percent, and Axid 9 percent.

Tagamet's real (quality-unadjusted) price gradually decreased from about $1 per day at entry to $0.80 per day when Zantac entered. As shown in Figure 2, Zantac entered with a large price premium over Tagamet, and thereafter the prices of *both* Zantac and Tagamet rose over time. The prices of Pepcid and Axid were between those of Zantac and Tagamet.

Finally, Figure 3 shows the depreciated stock of detailing minutes for each brand, computed using a monthly depreciation rate of 5 percent. The stocks for all four brands rose steadily most of the time that they were on the market, but Tagamet's fell during the last two years, perhaps in expectation of the imminent loss of patent protection in May 1994.

The data used to estimate the hedonic and brand diffusion equations form an unbalanced panel, while those used for the equilibrium share equations form a balanced panel. We estimate the parameters of the hedonic price equation by ordinary least squares, and compute heteroscedasticity-consistent and ARMA(2,2) serial correlation-consistent standard errors. We estimate the logit equations for the equilibrium brand shares three ways:

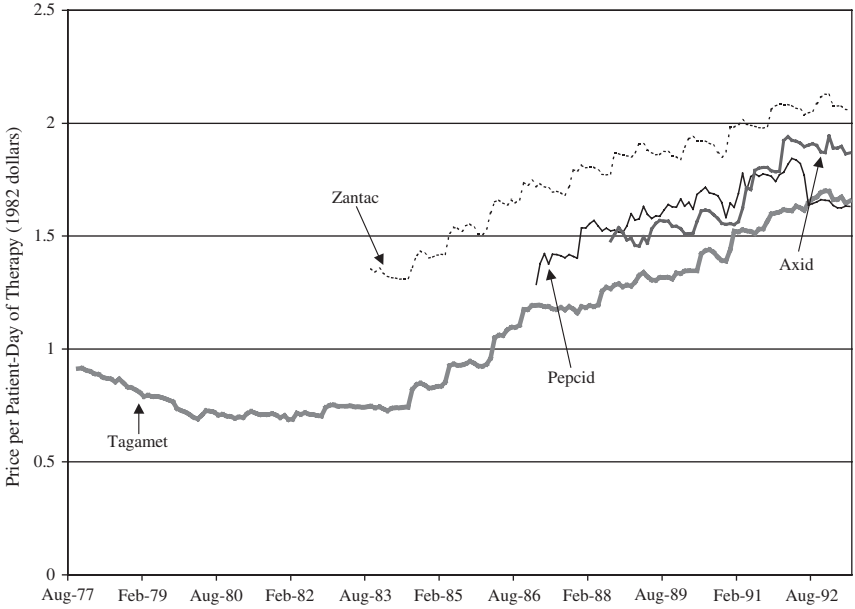

Figure 2
Real Prices of H_2-Antagonist Drugs

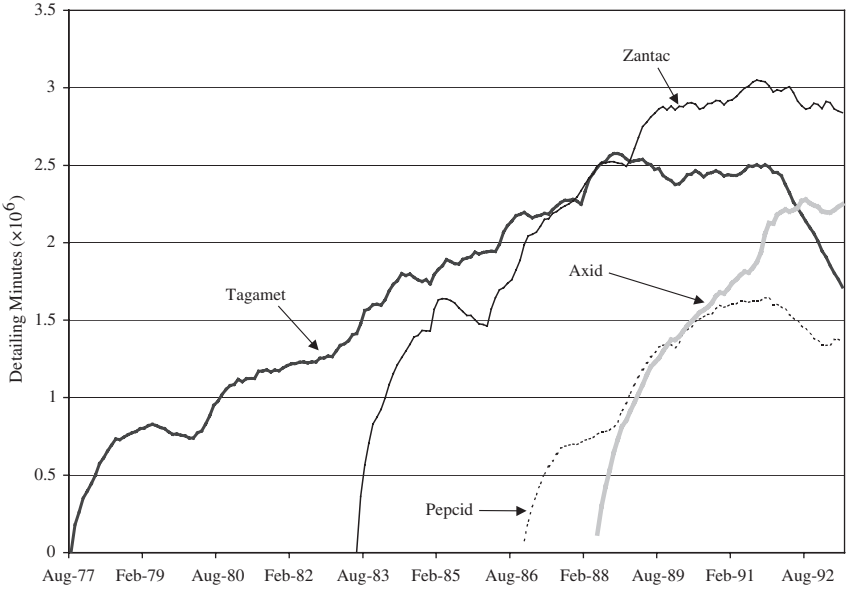

Figure 3
Detailing Stocks for H_2-Antagonist Drugs ($\delta = 5\%$)

as a seemingly unrelated regression (SUR), by three-stage least squares (3SLS), and by generalized method of moments (GMM). The brand diffusion equations 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 as exogenous. 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.

Even with our structural assumptions, we must confront issues of unobserved heterogeneity in the hedonic equation and endogeneity in the equilibrium share model. To separate out the effects of consumption externalities from unobserved drug characteristics, we use *hospital* prices and sales as identifying instruments. In the case of H_2 drugs, the hospital and drugstore segments can be considered independent. Hospitals administer these drugs intravenously to emergency room patients in order to reduce acid secretion induced by severe trauma. By contrast, drugstores sell oral preparations to outpatients suffering from a wide range of stomach-related conditions. Since both markets experience common manufacturing cost shocks, hospital prices and sales are likely to be strongly correlated with drugstore prices and sales, but uncorrelated with unobserved determinants of drugstore demand.

Table 1 shows summary statistics for the variables used in the model. Part A includes aggregate industry variables, and Parts B to E include brand-specific variables. Part F shows summary statistics for each brand's market share, price, and marketing for the last 53 months of our sample (the 'equilibrium' period).

IV. HEDONIC PRICE EQUATIONS

Table 2 presents the results of estimating linear and semi-log hedonic price equations for our unbalanced panel of four drugs. We first estimate the hedonic price equation by OLS. There may be serial correlation in the residuals, but we have no basis for making assumptions about its structure. We therefore re-estimate the model using GMM, with an instrument set composed of all of the right hand side variables. The point estimates do not change, but the t-statistics are robust to the presence of heteroscedasticity and ARMA(2,2)-serial correlation in the residuals. 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 was discussed in Section 3: GERD, SUMATT, INTER, and DOSAGE. As can be seen from the table, GERD, INTER, and DOSAGE

TABLE I
SUMMARY STATISTICS

	Mean	Std Dev	Minimum	Maximum
A. *Industry Variables (Nobs = 188)*				
X_t (patient-days/month, $\times 10^6$)	51.75	25.57	3.773	99.05
XS_t (cumul. patient-days, $\times 10^6$)	1028.4	619.7	7.715	2099.3
$\bar{P}_t$ (quality-adjusted avg. price)	1.562	0.215	1.155	1.914
$\bar{p}_t$ (real avg. price, 1982$)	1.206	0.441	0.688	1.930
POP_t (U.S. population, $\times 10^6$)	237.3	10.34	219.4	256.2
MIN_t (detailing minutes, $\times 10^6$)	0.249	0.171	0.019	0.604
$MINSTKTOT_t$ (ind. tot. stock min., $\times 10^6$)	4.173	3.107	0.263	9.268
B. *Tagamet (Nobs = 188)*				
X_{it} (patient-days/month, $\times 10^6$)	27.52	9.087	3.773	46.42
XS_{it} (cumul. patient-days, $\times 10^6$)	615.0	259.0	7.715	872.6
P_{it} (quality-adjusted price)	1.562	0.232	1.155	2.004
p_{it} (real price, 1982$)	1.056	0.328	0.688	1.700
MIN_{it} (detailing minutes, $\times 10^6$)	0.094	0.036	0.019	0.199
$MINSTK_{it}$ (stock of minutes, $\times 10^6$)	1.727	0.656	0.263	2.576
$GERD_{it}$ (GERD dummy)	0.133	0.340	0.000	1.000
$SUMATT_{it}$ (other approved indications)	1.612	0.873	0.000	3.000
$INTER_{it}$ (# adverse drug interactions)	7.096	3.617	0.000	10.00
$DOSAGE_{it}$ (daily dosing frequency)	2.516	1.164	1.000	4.000
C. *Zantac (Nobs = 117)*				
X_{it} (patient-days/month, $\times 10^6$)	30.42	14.20	4.190	54.27
XS_{it} (cumul. patient-days, $\times 10^6$)	537.8	337.9	11.92	1093.2
P_{it} (quality-adjusted price)	1.733	0.108	1.533	1.961
p_{it} (real price, 1982$)	1.770	0.239	1.309	2.129
MIN_{it} (detailing minutes, $\times 10^6$)	0.133	0.036	0.048	0.212
$MINSTK_{it}$ (stock of minutes, $\times 10^6$)	2.289	0.667	0.704	3.049
$GERD_{it}$ (GERD dummy)	0.718	0.452	0.000	1.000
$SUMATT_{it}$ (other approved indications)	1.530	0.794	0.000	2.000
$INTER_{it}$ (# adverse drug interactions)	0.145	0.354	0.000	1.000
$DOSAGE_{it}$ (daily dosing frequency)	1.342	0.476	1.000	2.000
D. *Pepcid (Nobs = 77)*				
X_{it} (patient-days/month, $\times 10^6$)	9.173	3.693	1.947	14.61
XS_{it} (cumul. patient-days, $\times 10^6$)	141.7	86.62	4.740	284.0
P_{it} (quality-adjusted price)	1.695	0.177	0.417	1.892
p_{it} (real price, 1982$)	1.616	0.123	1.286	1.844
MIN_{it} (detailing minutes, $\times 10^6$)	0.075	0.023	0.031	0.131
$MINSTK_{it}$ (stock of minutes, $\times 10^6$)	1.211	0.389	0.295	1.645
$GERD_{it}$ (GERD dummy)	0.234	0.426	0.000	1.000
$SUMATT_{it}$ (other approved indications)	1.727	0.448	1.000	2.000
$INTER_{it}$ (# adverse drug interactions)	0.000	0.000	0.000	0.000
$DOSAGE_{it}$ (daily dosing frequency)	1.000	0.000	1.000	1.000
E. *Axid (Nobs = 59)*				
X_{it} (patient-days/month, $\times 10^6$)	4.926	2.344	0.704	9.207
XS_{it} (cumul. patient-days, $\times 10^6$)	65.16	41.80	4.568	146.6
P_{it} (quality-adjusted price)	1.778	0.090	1.630	1.964
p_{it} (real price, 1982$)	1.680	0.169	1.456	1.943
MIN_{it} (detailing minutes, $\times 10^6$)	0.114	0.024	0.069	0.217
$MINSTK_{it}$ (stock of minutes, $\times 10^6$)	1.647	0.517	0.427	2.277
$GERD_{it}$ (GERD dummy)	0.390	0.492	0.000	1.000
$SUMATT_{it}$ (other approved indications)	1.000	0.000	1.000	1.000
$INTER_{it}$ (# adverse drug interactions)	1.000	0.000	1.000	1.000
$DOSAGE_{it}$ (daily dosing frequency)	1.000	1.000	1.000	1.000
F. *Balanced Panel, 1989–1993 (Nobs = 53)*				
S_{it}–Tagamet	0.293	0.059	0.212	0.407
S_{it}–Zantac	0.519	0.018	0.478	0.550
S_{it}–Pepcid	0.130	0.023	0.079	0.169

TABLE I (continued)

	Mean	Std Dev	Minimum	Maximum
S_{it}–Axid	0.058	0.023	0.011	0.094
P_{it}–Tagamet	1.854	0.093	1.709	2.004
P_{it}–Zantac	1.808	0.086	1.673	1.961
P_{it}–Pepcid	1.738	0.106	1.542	1.892
P_{it}–Axid	1.790	0.088	1.630	1.964
MINSTK_{it}–Tagamet	2.343	0.221	1.713	2.538
MINSTK_{it}–Zantac	2.895	0.099	2.535	3.049
MINSTK_{it}–Pepcid	1.450	0.154	1.030	1.645
MINSTK_{it}–Axid	1.758	0.415	0.914	2.277

are all highly significant and have the expected signs; SUMATT is usually insignificant, and has the wrong sign, which may reflect the fact that much prescribing is 'off-label,' i.e., permitted but not formally approved by the FDA.[8]

Each equation also has one or two variables that measure the effects of past sales at the brand-specific and therapeutic category levels. The first variable, $\text{XS}_{i,t-1}$, is the depreciated stock of past sales of brand i, calculated using a monthly depreciation rate of 5 percent. The second variable, XS_{t-1}, is the corresponding depreciated stock of past sales for the therapeutic category. Note in Table 2 that the brand-specific variable XS_{t-1} is always positive and highly significant in both the linear and semi-log versions, while the variable for the therapeutic category, $\text{XS}_{i,t-1}$, is insignificant. We infer from this that the use of a drug by others affects its valuation, and that this effect operates at the brand rather than the therapeutic class level.[9]

To see the magnitude of this effect, consider column (1) in Table 2, where the coefficient on $\text{XS}_{i,t-1}$ is about 0.00018. Just prior to Zantac's introduction in August 1983, Tagamet had a depreciated stock of past sales of 786 million patient days. Had this figure been about 200 million (25 percent) less, the value of Tagamet would have been reduced by \$0.036 (i.e., 200×0.00018), or about 5 percent of its approximately \$0.75 price at that

[8] It is unclear whether marketing effort should be included in the hedonic equation. One could argue that our measures of consumption externalities fully incorporate the effects of marketing and other informational investments. In our data, the simple correlation between XS_{it} and MINSTK_{it} is 0.802, 0.954, 0.849, and 0.945 for the four brands, suggesting that it would be very difficult to estimate the separate effects of marketing efforts and consumption externalities as components of the hedonic price. However, when we instead included in the hedonic equation the residual of a simple regression of the stock of detailing minutes on the stock of patient days (the brand-specific consumption externality measure), the results were little affected.

[9] To check on whether the lagged quantity variable was correlated with the hedonic disturbance term, we instrumented past sales using cumulative hospital sales. We ran a Hausman test for exogeneity of past sales and could not reject the null hypothesis for each of the six specifications. To explore possible strategic pricing, we also ran regressions adding as a regressor the number of firms competing in the market that month, initially as a single count variable, and then as three dummy variables for the duopoly, three-firm, and four-term epochs. The parameter estimates on these variables were always insignificant.

TABLE II
HEDONIC PRICE EQUATION

	A. *Dependent Variable* = P_{it}			B. *Dependent Variable* = $\log P_{it}$		
	(1)	(2)	(3)	(4)	(5)	(6)
Const.	1.3535 (18.82)	1.4152 (18.44)	1.3919 (17.64)	0.5113 (7.47)	0.5467 (7.45)	0.5387 (7.21)
$GERD_{it}$	0.1816 (10.72)	0.2320 (13.48)	0.1820 (10.71)	0.1245 (10.50)	0.1418 (13.40)	0.1247 (10.48)
$SUMATT_{it}$	−0.0159 (−0.98)	0.0163 (1.11)	−0.0156 (−0.97)	−0.0061 (−0.49)	0.0050 (0.46)	−0.0059 (−0.48)
$INTER_{it}$	−0.0452 (−16.62)	−0.0375 (−14.37)	−0.0452 (−16.59)	−0.0286 (−14.33)	−0.0259 (−14.56)	−0.0285 (−14.29)
$DOSAGE_{it}$	−0.1158 (−6.83)	−0.1194 (−7.41)	−0.1157 (−6.83)	−0.1555 (−9.63)	−0.1566 (−9.91)	−0.1554 (−9.64)
$XS_{it}(-1)$	0.1758×10^{-3} (5.49)		0.1753×10^{-3} (5.48)	0.6030×10^{-4} (2.72)		0.5998×10^{-4} (2.71)
$XS_t(-1)$		0.4804×10^{-3} (1.79)	0.4265×10^{-3} (1.50)		0.0239×10^{-3} (1.45)	0.3054×10^{-3} (1.34)
R^2	0.966	0.960	0.967	0.970	0.969	0.970
Zantac Price Premium	\$0.01	\$0.35	\$0.30	\$0.04	\$0.27	\$0.25

Note: All regressions include annual and quarterly time dummies; NOB = 441; *t*-statistics (from heteroscedasticity-consistent and ARMA(2,2) serial-correlation consistent standard errors) in parentheses. Zantac price premium is the estimated quality-adjusted price of Zantac minus that of Tagamet at the time of Zantac's entry in July 1983, based on attribute differences with Tagamet. (The actual deflated price difference was \$0.615.)

time. This implies a brand-specific valuation elasticity of 0.2 (0.05/0.25), which is positive but modest. The semi-log hedonic equation yields even smaller elasticities. In all of the calculations that follow, we use Model (1) of Table 2, i.e., the linear hedonic equation.

Figure 4 shows quality-adjusted real prices for the four drugs. The sharp movements in these prices are largely due to changes in the drugs' attributes. For example, increases in the quality-adjusted price of Tagamet during 1980–82 are due to findings of additional interactions with other drugs that reduced its effective quality. The sharp drops in the price of Tagamet in January 1985 and January 1987 are due to changes in dosing from four daily doses to two, and then to one. Zantac's quality-adjusted price also dropped in January 1987 because its daily dosing dropped from two to one. Pepcid's quality-adjusted price dropped in December 1991 when it received approval for treatment of GERD.

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 about 61 cents above Tagamet (in 1982 dollars). One might argue that this higher price was intended to signal higher quality. Zantac 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 Tagamet a perceived value premium. Our hedonic equation implies

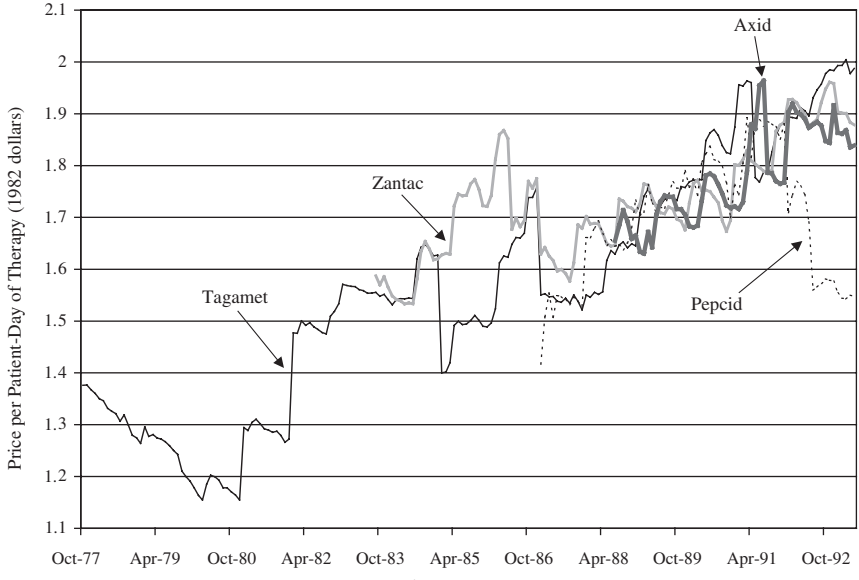

Figure 4
Quality-Adjusted Real Prices of H_2-Antagonist Drugs

that Zantac had a 72-cent advantage from its better dosing and interaction profile, and a 12-cent disadvantage from the consumption externality, implying a net price premium of only $61 - 72 + 12 = 1$ cent.

V. EQUILIBRIUM SHARE EQUATIONS

Using the hedonic equation (1) from Table 2, we construct quasi-residuals that represent quality-adjusted prices for each brand. With these quasi-residuals, along with the depreciated stock of detailing minutes for each brand, we estimate a multinomial logit model using the last 53 months of data for our sample. During this period, all four brands were well established and their efficacy and side-effects were well known. Thus it is reasonable to impose the identifying assumption that the market was in equilibrium during this period, so that any changes in market shares were due to changes in prices and marketing efforts.

Because price and marketing levels are likely to be endogenous, we need a set of instrumental variables for consistent estimation. We use four instruments: (i) the log of the wage rate in the pharmaceutical industry, (ii) the PPI for intermediate goods, (iii) the cumulative stocks of detailing minutes for each of the four firms on all their *other* products (calculated the same way as MINSTK_{it}), and (iv) quality-adjusted prices for each of the firms for H_2-antagonist drugs *sold to hospitals*.[10] Note that the first two of these instruments vary only over time, and the second two vary over time and across drugs.

Estimation results are in Table 3. Columns (1) and (2) show the SUR and 3SLS estimates, respectively. A Hausman specification test on the 3SLS estimates fails to reject exogeneity of price and advertising; the test statistic is 6.486 ($p = 0.090$). This failure to reject exogeneity is not due to a problem of using weak instruments. In the first-stage regressions, both individual *t*-tests and joint *F*-tests on whether parameters of the overidentifying instruments are zero were decisively rejected. Column (3), our preferred model, is estimated by GMM, with *t*-statistics based on heteroscedasticity-consistent and ARMA(1,1) serial-correlation-consistent standard errors. The *J*-statistic used to test the overidentifying restrictions is 16.618; with 10 degrees of freedom (five instruments, including the constant, times three equations, minus five parameters), the p-value is 0.093.

Table 3 also shows price and detailing elasticities computed at the point of means for the 1989–1993 sample period. Focusing on column (3), note that the own-price elasticities are in the range of about –0.3 to –0.6. These elasticities are based on holding the total quantity of H_2-antagonist drugs constant when the price of a single drug changes, i.e., they only reflect

[10] We also used hospital *quantity sales* as an additional instrument, with no change in the results.

TABLE III
ESTIMATES OF EQUILIBRIUM LOGIT MARKET SHARES, 1989-1993

		(Omitted Share is Tagamet, NOBS = 53)		
		(1) SUR	(2) 3SLS	(3) GMM
Intercept–Zantac		0.1983 (10.47)	0.1937 (10.14)	0.2055 (14.89)
Intercept–Pepcid		–0.0817 (–3.33)	–0.0788 (–3.16)	–0.0957 (–4.31)
Intercept–Axid		–1.1067 (–73.38)	–1.1045 (–71.23)	–1.1047 (–87.50)
a_1		–0.2889 (–4.76)	–0.3129 (–4.90)	–0.3442 (–5.91)
a_2		0.7634 (32.08)	0.7697 (31.53)	0.7414 (43.12)
R^2		0.78/0.81/0.96	0.78/0.81/0.96	0.77/0.81/0.96
ϵ_P	Tagamet	–0.385 (–4.757)	–0.417 (–4.897)	–0.459 (–5.908)
	Zantac	–0.250 (–4.757)	–0.271 (–4.897)	–0.298 (–5.908)
	Pepcid	–0.435 (–4.757)	–0.471 (–4.897)	–0.518 (–5.908)
	Axid	–0.485 (–4.757)	–0.525 (–4.897)	–0.577 (–5.908)
ϵ_{MIN}	Tagamet	1.286 (32.078)	1.297 (31.534)	1.249 (43.119)
	Zantac	1.057 (32.078)	1.066 (31.534)	1.027 (43.119)
	Pepcid	0.958 (32.078)	0.966 (31.534)	0.930 (43.119)
	Axid	1.258 (32.078)	1.268 (31.534)	1.222 (43.119)

Note: For models (1) and (2), *t*-statistics (from heteroscedasticity-consistent standard errors) are in parentheses. For model (2), the Hausman test statistic for exogeneity of price and advertising is 6.486 (p = 0.090). For model (3), the *t*-statistics are from heteroscedasticity-consistent and ARMA(1,1) serial-correlation-consistent standard errors; the *J*-statistic for the test of overidentifying restrictions is 16.618, df = 10, p = 0.083. The price and advertising elasticities are computed at the point of means for the 1989–1993 sample period.

substitution within the therapeutic category, so the *total* own-price elasticities will be larger in magnitude. The estimated detailing elasticities are close to unity, which might seem large. After launch ramp-up, the advertising-to-sales ratio for these drugs was about 15 to 20 percent, so even if the own-price elasticities were –1, the advertising elasticity should be about 0.2 if the marginal cost of detailing were constant. (Detailing accounted for about 80 percent of total pharmaceutical marketing.) It is likely, however, that the marginal cost of detailing rises sharply as it becomes increasingly difficult for detailers to get additional minutes of physicians' time, and is much higher than the average cost. This is consistent with our large elasticity estimates.

As can be seen from Table 3, our elasticity estimates are robust to the choice of estimation method. Although not shown in the table, both the price and detailing elasticities are also robust to the monthly depreciation rate used to compute the stock of detailing minutes. Depreciation rates between 2 percent and 8 percent yield very little change in the parameter estimates or the optimized value of the GMM objective function.

Using column (3) from Table 3, we construct fitted values of equilibrium shares for the four drugs. By 'equilibrium shares,' we mean the shares that each of these drugs would have had at any point in time had the market already reached equilibrium. For the months prior to 1989, we generate out-of-sample backcasts of the equilibrium shares. For example, let k denote the

number of drugs competing in the market at a point in time. We can write the fitted shares from model (3) in Table 3 as

$$(11) \qquad \hat{s}^*_{it}(k) = \frac{\exp(\delta_{it})}{1 + \sum_{j=1}^{k} \exp(\delta_{jt})}$$

where $\delta_{it} = \hat{a}_{i0} + \hat{a}_1(P_{it} - P_{T,t}) + \hat{a}_2(\text{MINSTK}_{it} - \text{MINSTK}_{T,t})$, and the subscript T denotes Tagamet.

To check our identifying assumption that the last 53 months represents an equilibrium period, we re-estimated the share model over a period that begins six months earlier, and then six months later. The resulting elasticity estimates were essentially unchanged. Also, to test the assumption that the market was in equilibrium during this period, we examined whether the residuals of the demand system exhibited any brand-specific time trends. In a model that also included drug and year effects, we were unable to reject the hypothesis that the brand-specific time trends were individually or jointly equal to zero.

VI. DIFFUSION EQUATIONS

The third component of our model is a set of equations describing the diffusion of the brands as they approach their equilibrium levels. We estimate modified Gompertz equations (9) and (10), using the hedonic quasi-residuals and equilibrium shares described above.[11]

These equations explain the *change* in sales; at issue is how large a time interval this change should represent. In principle, we could estimate a model describing monthly changes in sales. However, it is unclear whether the accounting of sales in the data is free of lags, and there is high-frequency noise due to ordering and stocking decisions by drugstores, so we use three-month changes in sales.[12] Also, we estimate models in which the depreciated stock of past sales (of the brand and the therapeutic category) are in linear and in logarithmic form. Estimation is by NLS, combining the data for the four brands to form an unbalanced panel. The results are shown in Table 4.

Note that individual brand prices affect the average quality-adjusted price for the therapeutic category, $\bar{P}_t$, which in turn affects the industry saturation level. In addition, *relative* prices affect brand saturation levels through the equilibrium shares. Finally, the long-run own-price elasticity for the

[11] We also estimated logistic versions of the model, with little change in the results.

[12] We also estimated the model using one-month and six-month changes. The results using six-month changes are very close to those reported here, but one-month changes yield a worse fit.

therapeutic category is given by the estimated coefficient, b_1, and ranges from –0.3 to –0.9. The total own-price elasticity for each individual brand is

$$E_i^P = \frac{\partial \log s_i^*}{\partial \log P_i} + b_1 \bar{s}_{i4} = a_1(1 - s_i^*)P_i + b_1 \bar{s}_{i4}$$

Likewise, the total detailing elasticity for each individual brand is given by

$$E_i^A = \frac{\partial \log s_i^*}{\partial \log \text{MINSTK}_i} + b_3 \frac{\text{MINSTK}_i}{\text{MINSKTOT}}$$
$$= a_2(1 - s_i^*)\text{MINSTK}_i + b_3 \frac{\text{MINSTK}_i}{\text{MINSKTOT}}$$

Here, $\bar{s}_{i4}$ is the average share of drug i during the period in which all four drugs are present.

In this model, consumption externalities attributable to past sales of the therapeutic category are captured by the coefficient d_0, while those attributable to past sales of the individual brand are captured by d_1. Note that d_1 is positive and significant, while d_0 is insignificant. We infer from this that the effect of past consumption on the rate of product diffusion occurs primarily at the brand level.

TABLE IV
BRAND DIFFUSION EQUATIONS NOBS = 429

Past Sales	(1) XS_{t-3}	(2) $\log[XS_{t-3}]$		(1)	(2)
C_{Tagamet}	0.5307 (1.25)	–2.6602 (–1.76)	**Elasticity Estimates**		
C_{Zantac}	2.6309 (1.46)	–3.1004 (–1.11)	ϵ_P (Tagamet)	–0.7569 (–3.73)	–0.8202 (–2.76)
C_{Pepcid}	4.2109 (1.46)	–2.0477 (–0.68)	ϵ_P (Zantac)	–0.8266 (–2.46)	–0.9388 (–1.84)
C_{Axid}	5.1534 (1.50)	–3.9436 (–1.35)	ϵ_P (Pepcid)	–0.6490 (–5.38)	–0.6770 (–4.39)
d_0	–0.0024 (–1.04)	0.0612 (0.13)	ϵ_P (Axid)	–0.6357 (–6.08)	–0.6483 (–5.73)
d_1	0.0180 (4.04)	1.3585 (4.57)			
b_0	–26.887 (–2.41)	–49.474 (–3.05)	ϵ_{MIN} (Tagamet)	1.3104 (36.74)	1.2794 (28.45)
b_1	–1.0205 (–1.59)	–1.2366 (–1.26)	ϵ_{MIN} (Zantac)	1.1021 (31.48)	1.0639 (21.87)
b_2	5.7040 (2.73)	9.8617 (3.25)	ϵ_{MIN} (Pepcid)	0.9677 (38.60)	0.9487 (31.42)
b_3	0.2182 (2.92)	0.1069 (0.87)	ϵ_{MIN} (Axid)	1.2672 (39.29)	1.2442 (32.65)
R^2	0.293	0.200			

Note: In each model, the consumption externality is $CE_t = XS_t$ or $\log[XS_t]$, and $CE_{it} = XS_{it}$ or $\log[XS_{it}]$. We estimate the following model by nonlinear least squares, using data for the four brands, combined to form an unbalanced panel:

$$X_{it} - X_{it-3} = [\log(\hat{s}_{it}^* X_t^*) - \log X_{it-3}] \cdot \left[C_i + \sum_{k=2}^{12} \theta_k m_{kt} + d_0 \text{CE}_{t-3} + d_1 \text{CE}_{it-3} \right]$$

where $\log X_t^* = b_0 + b_1 \log \bar{P}_t + b_2 \log \text{POP}_t + b_3 \log \text{MINSTKTOT}_t$. The $\hat{s}_{it}^*$'s are the fitted equilibrium market shares from model (3) of Table 4, adjusted to account for the number of competing brands in each of the 4 epochs. The m_{kt}'s are a set of monthly time dummies whose coefficients θ_k are not reported. Numbers in parentheses are t-statistics from heteroscedasticity-consistent standard errors.

Table 4 also shows estimated *total* own-price and detailing elasticities. The price elasticities are on the order of –0.65 to –0.94, and the detailing elasticities are 1 to 1.3. Given that marginal production cost for these antiulcer drugs is very small (about 10 cents to 20 cents per daily dose), we would expect the own-price elasticities to be close to –1 if producers maximize profits, so our estimated price elasticities seem somewhat low. As explained earlier, the large detailing elasticities may reflect a rising marginal cost of detailing.

VII. SIMULATIONS

Simulations of the complete model are used for two purposes. First, in-sample simulations test the model's validity: Using historical values for the attributes, prices, advertising levels, and population, we can solve for all of the other variables endogenously in a dynamic framework, and compare the results to the actual data. Second, we use the model to simulate the effects of alternative strategies for pricing, detailing, and quality improvement.

Because the model is highly nonlinear, the convergence and stability of the simulations are sensitive to initial conditions. To deal with this, we simulate the full model using the actual values of sales for each brand for the first 12 months following the entry of the brand.

Figure 5 shows the simulated and actual sales for all four brands. The simulated series comes from a dynamic simulation in which real prices,

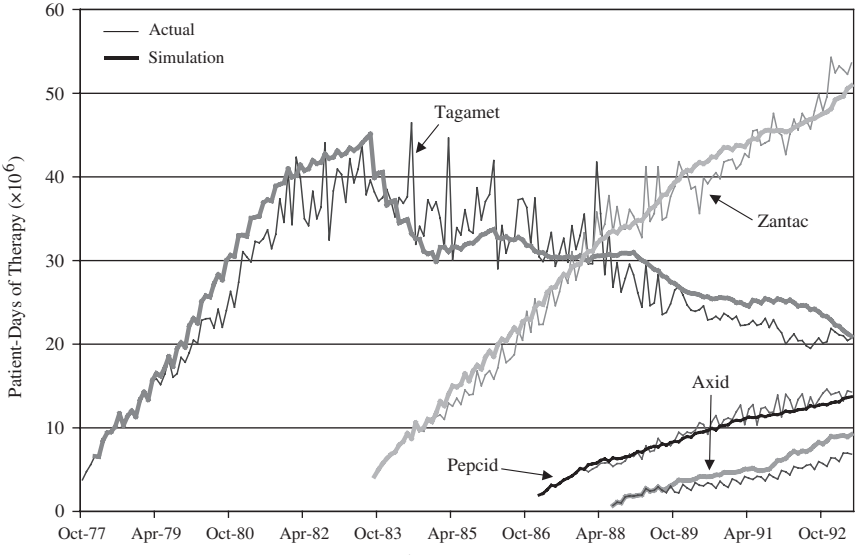

Figure 5
Full Simulation of Brand Sales versus Actual

detailing minutes, attribute levels, and population are exogenous, and all other variables (quality-adjusted prices and average price, equilibrium shares, the industry saturation level, and sales of each brand) are solved for endogenously. Note that overall, the simulated values are very close to the actual values.

We also use the model to simulate three different changes in market conditions:

- We set Zantac's nominal price in each month equal to that of Tagamet.
- We set Zantac's detailing minutes in each month equal to that of Tagamet.
- We reduced d_1, the coefficient in the brand diffusion model that determines the impact of past sales on the rate of growth of current sales, by 50 percent (from 0.018 to 0.009).

The results of these simulations are summarized in Table 5, which shows the change in sales for each brand (the experiment minus the base case) in May 1993, and the resulting change in cumulative profits for Zantac and Tagamet.

Because Zantac had better attributes than Tagamet, setting Zantac's nominal price equal to Tagamet's makes its quality-adjusted price lower than Tagamet's. The result is that Zantac's sales are about 20 percent higher, because its lower quality-adjusted price results in an increase in its equilibrium share. However, the sales of Tagamet, Pepcid, and Axid are also higher than in the base case simulation. The reason is that even though their equilibrium *shares* are lower, the average industry price is now lower, so that the industry saturation level, X_t^*, is higher, which outweighs the equilibrium share reductions.

TABLE V
SIMULATION EXPERIMENTS

Experiment	ΔX_T	ΔX_Z	ΔX_P	ΔX_A	$\Delta\Sigma\Pi_T$	$\Delta\Sigma\Pi_Z$
			May 1993			
(1)						
Zantac Price at	1.5733	10.784	0.7073	0.1710	268.21	–669.6
Tagamet Level	6.87%	22.3%	5.05%	2.79%	5.07%	–11.0%
(2)						
Zantac Advertising	5.8974	–13.548	2.9091	1.0013	306.54	–662.2
at Tagamet Level	26.0%	–27.88%	22.34%	16.25%	5.79%	–10.7%
(3)						
Coefficient d_1	3.5294	–3.5837	–1.6551	–3.1595	–106.12	–882.3
Reduced by 50%	15.7%	–7.39%	–12.8%	–52.9%	–2.02%	–14.4%

Note: ΔX_Z is the difference (absolute and percentage change) in Zantac sales between the experiment and the base case, in May 1993. Similarly, ΔX_T, ΔX_P, and ΔX_A are the differences in Tagamet, Pepcid, and Axid sales in May 1993. $\Delta\Sigma\Pi_Z$ is the aggregate change in gross profit for Zantac under the simulation experiment compared to the base case simulation, and $\Delta\Sigma\Pi_T$ is the aggregate change in gross profit for Tagamet. In Experiment (2), we use an average cost per minute of detailing, which varies from \$3.76 in 1983 to \$8.09 in 1993, to calculate the savings in reduced advertising expenditures for Zantac.

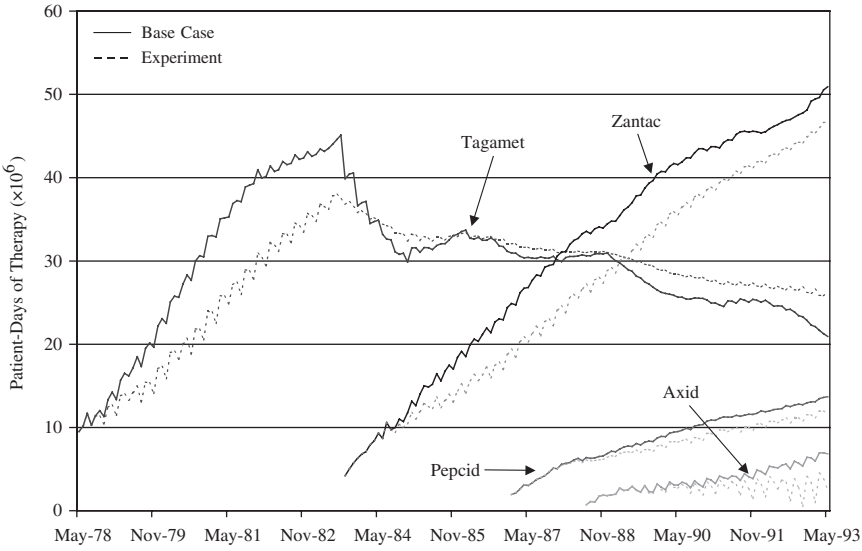

Figure 6
Coefficient d_1 Set Equal to 50 Percent of Estimated Value

Next, we set Zantac's detailing level equal to Tagamet's. Recall that Zantac detailed much more heavily than Tagamet, so in effect we are decreasing Zantac's detailing. The result is that Zantac's sales are much lower by the end of the period, and the sales of Tagamet, Axid, and Pepcid are all higher. This is due to the large estimates (about 1.0) of the advertising elasticities of demand in the equilibrium share model. Table 5 shows the impact on Zantac's cumulative gross profits, ignoring production costs (which are small), but accounting for detailing costs, which are estimated annually from aggregate pharmaceutical industry data.[13] Observe that although Zantac's detailing expenditures drop, its sales drop by so much that its cumulative profits fall by almost \$700 million.

Lastly, we evaluate the effect of past sales on the rate of brand diffusion. This effect is captured by the coefficient d_1 in the diffusion equations; note from column (1) of Table 4 that the estimated value for d_1 is 0.0180. We

[13] We used average 'Cost per Call' data, estimated each year for the entire pharmaceutical industry by IMS (IMS, 1996, pp. 7–47 and A-20), from a survey of manufacturers who estimate the cost of keeping a representative 'in the field'—salary, bonus, car, insurance, expenses, training, etc. IMS indicates that, on average, a call involves from two to four 'product details,' i.e., individual products discussed by the sales representative. In addition to the number of detailing minutes for each drug, we have data on the number of details per month for each drug. Aggregating these two series to the level of the H_2-antagonist class and assuming that each 'call' comprises three 'details,' we compute an average annual cost per detailing minute from 1977 to 1993. In 1982 dollars, this average cost increases from \$3.28 in 1977 to \$8.09 in 1993.

reduce this by half, i.e., to 0.0090. The results are shown in Figure 6. Because past sales of Tagamet now contribute less to the growth of sales, Tagamet's sales grow much more slowly than in the base case. The same is true, however, for Zantac, Pepcid, and Axid. As a result, by late 1984 Tagamet's sales are *higher* than in the base case simulation. As Table 5 shows, Tagamet's cumulative gross profits fall by about $25 million, but Zantac's cumulative gross profits fall by nearly $900 million. Thus past sales play a significant role in brand diffusion and profitability.

Because our estimates of total own-price elasticities are all below one in magnitude (see Table 4), simulations of unilateral or multilateral price increases will yield higher profits. It thus appears that all four firms were pricing below their optimal levels. Although prices rose over time (see Figure 2), why did they not rise faster? Pharmaceutical pricing has often been subject to political pressure, which may explain lower-than-optimal prices.

What if *all* four firms had advertised more than they did? We ran a simulation in which detailing for all firms was increased by 10 percent above actual values during the equilibrium period, January 1989 to April 1993. The cumulative percentage changes in each firm's profits over the 53 months, accounting for the cost of the added detailing, were 0.36 percent for Tagamet, 2.80 percent for Zantac, –4.05 percent for Pepcid, and –2.77 percent for Axid. Much of these differences is due to differences in market shares; note from eqn. (6) that *any* increase in detailing increases the *industry* saturation level, so firms will benefit in proportion to their shares. Part of these differences is due to different detailing elasticities, as shown in Table 4. That is why Pepcid suffers a greater loss in profits than Axid, even though its market share is larger.

VIII. CONCLUSIONS

In order to examine the ways in which consumption externalities influence the demands for prescription drugs, we have estimated a three-stage model of quality-adjusted prices, equilibrium market shares and saturation levels, and rates of brand diffusion. Consumption externalities are captured by introducing the depreciated stock of past sales, for the brand and for the therapeutic class, in both an hedonic price equation and an equation for brand diffusion. The resulting model fits the data well — dynamic simulations yield time paths for brand sales that track the actual time paths very closely. Furthermore, we are able to identify the sources of consumption externalities, and measure their magnitudes.

We find that consumption externalities operate at the brand-specific level. Although statistically significant, their economic importance is mixed. In our hedonic equations, past sales contribute to the value of a brand, but only explain a few percent of that value. However, past sales have an economically significant effect on the rate of diffusion. Our simulations

imply that had the magnitude of the effect of past sales been 50 percent smaller, Zantac would have earned $882 million less in gross profits, an amount roughly equivalent to three months of 1992 sales.

These results have 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, but that this effect is modest. On the other hand, we find that rates of diffusion can be accelerated by a larger brand-specific installed base. Thus, even if the ultimate saturation level for a second entrant is close to that of a first entrant with similar attributes, the more rapid rate of diffusion can result in much greater profits.

In the case of antiulcer drugs, consumption externalities were not large enough to prevent the second entrant from overcoming the pioneering brand. Our results shed light on Zantac's success. It derived little benefit from the information about H_2-antagonists generated by Tagamet: free-riding from inter-brand consumption externalities is negligible. Instead, delayed entry allowed Glaxo to introduce a product with better quality attributes. This, together with an unusually large amount of detailing, allowed Zantac to overcome the limited first-mover advantage that Tagamet obtained from its installed base of patients.[14]

REFERENCES

Berndt, Ernst R., *The Practice of Econometrics: Classic and Contemporary*, Reading, MA: Addison-Wesley Publishing Company, 1991.

Berndt, Ernst R., Cockburn Iain and Griliches Zvi, 'Pharmaceutical Innovations and Market Dynamics: Tracking Effects on Price Indexes for Antidepressant Drugs,' *Brookings Papers on Economic Activity: Microeconomics* 1996, 133–188.

Berndt, Ernst R., Bui Linda T., Reiley David H. and Urban Glen L., 'The Roles of Marketing, Product Quality, and Price Competition in the Growth and Composition of the U.S. Antiulcer Drug Industry,' in *The Economics of New Goods*, T. Bresnahan and R. Gordon, eds., University of Chicago Press, 1997, 277–322.

Berndt, Ernst R., Bui Linda T., Reiley David H. and Urban Glen L., 'Information, Marketing, and Pricing in the U.S. Antiulcer Drug Market,' *American Economic Review Papers and Proceedings*, May 1995, 85.

Berndt, Ernst R., Pindyck Robert S. and Azoulay Pierre, 'Consumption Externalities and Diffusion in Pharmaceutical Markets: Antiulcer Drugs,' NBER Working Paper No. 7772, June 2000.

Berry, Steven T., Levinsohn James and Ariel Pakes, 'Automobile Prices in Market Equilibrium,' *Econometrica*, 1995, 60(4), 889–917.

[14] Anecdotal evidence suggests that these features are not unique to the antiulcer drug market. In the anti-hypertensive market, for example, Merck introduced its ACE Inhibitor Vasotec later than Bristol Myers-Squibb's pioner, Capoten. As recounted by Werth (1994), p. 58: 'Merck had put scores of chemists on the task of improving [Capoten], then followed up with a withering sales campaign so effective that it ended up beating Squibb in the market even though Capoten was launched first and was much the same drug'.

Bikhchandani, Sushil, Hirshleifer David and Welch Ivo, 'Learning from the Behavior of Others: Conformity, Fads, and Informational Cascades,' *Journal of Economic Perspectives*, Summer 1998, 12, 151–170.

Bond, Ronald S. and Lean David F., *Sales, Promotion, and Product Differentiation in Two Prescription Drug Markets*, Staff Report of the Bureau of Economics of the Federal Trade Commission, February, 1977.

Brynjolfsson, Erik and Kemerer Chris F., 'Network Externalities in Microcomputer Software: An Econometric Analysis of the Spreadsheet Market,' *Management Science*, December 1996, 42, 1627–1647.

Cockburn, Iain and Anis Aslam H., 'Hedonic Analysis of Arthritis Drugs,' Chapter 11 in David M. Cutler and Ernst R. Berndt, eds., *Medical Care Output and Productivity*, Chicago: University of Chicago Press for the National Bureau of Economic Research, 2001, 439–458.

Coleman, James S., Katz Elihu and Menzel Herbert, *Medical Innovation: A Diffusion Study*, Indianapolis: Bobbs-Merrill, 1966.

Ellison, Sara Fisher, Cockburn Iain, Griliches Zvi and Hausman Jerry, 'Characteristics of Demand for Pharmaceutical Products: An Examination of Four Cephalosporins,' *RAND Journal of Economics*, Autumn, 1997, 28, 426–446.

Gandal, Neil, 'Hedonic Price Indexes for Spreadsheets and an Empirical Test for Network Externalities,' *RAND Journal of Economics*, Spring, 1994, **25**, 160–170.

Gandal, Neil, Kende Michael and Rob Rafael, 'The Dynamics of Technological Adoption in Hardware/Software Systems: The Case of Compact Disc Players,' *RAND Journal of Economics*, Spring, 2000, 31, 43–61.

Geroski, P. A., 'Models of Technology Diffusion,' *Research Policy*, 2000, **29**, 603–625

Goolsbee, Austan and Klenow Peter J., 'Evidence on Network and Learning Externalities in the Diffusion of Home Computers,' NBER Working Paper No. 7329, Sept. 1999.

IMS America, *Information Services Manual*, Philadelphia, 1996.

Kessler, Daniel and McClellan Mark, 'Do Doctors Practice Defensive Medicine?' *Quarterly Journal of Economics*, May, 1996, 20, 353–390.

King, Charles III, 'Empirical Studies of Marketing, Product Differentiation, Pricing and Competition,' Ph.D. dissertation, Massachusetts Institute of Technology, June, 1997.

Lu, Z. John and Comanor William S., 'Strategic Pricing of New Pharmaceuticals,' *Review of Economics and Statistics*, February, 1998, 80, 108–118.

Mahajan, Vijay and Muller Eitan, 'Innovation Diffusion and New Product Growth Models in Marketing,' *Journal of Marketing*, Fall, 1979, 43, 55–68.

Mahajan, Vijay, Muller Eitan and Bass Frank M., 'New Product Diffusion Models in Marketing: A Review and Directions for Research,' *Journal of Marketing*, 1990, 54, 1–26.

Scharfstein, David and Stein Jeremy, 'Herd Behavior and Investment,' *American Economic Review*, June, 1990, 80, 465–479.

Suslow, Valerie Y., 'Measuring Quality Change in the Market for Anti-Ulcer Drugs,' Chapter 4 in Robert M. Helms, ed., *Competitive Strategies in the Pharmaceutical Industry*, Washington, DC, American Enterprise Institute: AEI Press, 1996, 49–72.

Temin, Peter, *Taking Your Medicine: Drug Regulation in the United States*, Harvard University Press, 1980.

Werth, Barry, *The Billion-Dollar Molecule*, New York: Simon and Schuster, 1994.