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The authors investigate the changing role of marketing communication over the life cycle of a new product category. They postulate two effects of marketing communication on consumers' choices: an "indirect effect" through reduction of uncertainty about product quality and a "direct effect" (i.e., more is better). The authors expect that the indirect effect is relatively larger in the early, postlaunch stages. They develop a structural model of demand that allows for such temporal differences in the roles of marketing communication. They use a random coefficients discrete choice model with a Bayesian learning process to model physician learning about new drugs and market-level data for the prescription antihistamines category. They find that marketing communication has a primarily indirect effect 6–14 months after introduction but that the direct effect subsequently dominates. The results suggest that firms should follow a pattern of heavier communication at the introduction phase followed by lower levels.

Temporal Differences in the Role of Marketing Communication in New Product Categories

Marketing communication and product experience play significant roles in influencing consumer preferences and behavior in experience good categories that have intangible product characteristics. There is little research that documents the exact role of marketing communication in the evolution of consumers' preferences in such categories that are new to consumers. In this research, we use a modeling approach that enables us to distinguish and examine the evolution of the two major effects of marketing communication since the inception of the category. The two effects we consider are based on existing theories of the role of marketing communication. The first effect refers to marketing communication that enables consumers to update their prior beliefs and reduce uncertainty about the true quality of the new product through a Bayesian learning process. Because marketing communication affects consumer utility indirectly through perceived product quality, we refer to it as the "indirect Bayesian learning effect," or simply "indirect effect." The second effect consists of all effects that are not indirect (e.g., reminder effects) that influence preferences through goodwill accumulation. Because this effect is manifest in a direct shift in consumer utility, we refer to it as the "direct goodwill effect," or "direct effect."

Our empirical analysis uses data from a category of ethical drugs. In the pharmaceutical industry, direct marketing communication with physicians is usually referred to as detailing. Detailing comprises promotional visits made to physicians by pharmaceutical representatives.[1] The main sources of information considered by physicians to inform their current diagnoses and prescription decisions are detailing, meetings and conferences, and feedback from previous prescriptions. Additional sources of information include word of mouth and journal advertising. Our main focus is the effect of detailing on the evolution of physician prefer-

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[1]For a recent multidisciplinary review of detailing in the pharmaceutical industry, see Manchanda and Honka (2005).

ences and resulting prescriptions through the two previously mentioned mechanisms.

Note that our definition of indirect and direct effects loosely maps to the "informative" (indirect) and the "persuasive" (direct)[2] effects of marketing communication that are documented by structural approaches in the economics and marketing literature.[3] Studies that have modeled one or both of these effects include those of Erdem and Keane (1996), Anand and Shachar (2001), Currie and Park (2002), Ackerberg (2003), and Byzalov and Shachar (2004). The findings on the presence of the effects in these studies are mixed. For example, Ackerberg, Currie and Park, and Erdem and Keane find evidence for a predominantly indirect effect. In contrast, Anand and Shachar find evidence for both effects (in a mature product category). Byzalov and Shachar find that risk aversion may explain the direct effect found in studies that assume risk neutrality.

This study is in line with previous research because we allow for both direct and indirect effects. Furthermore, we postulate that the role of detailing is different in the introduction stage of a drug versus its subsequent stages. At the time of a drug's introduction, a physician's experience is limited, and it is likely that he or she is uncertain and not well informed about its efficacy (an intangible characteristic that we define subsequently). Thus, detailing is postulated to have a primarily indirect effect in the introductory phase of a drug's life cycle by helping the physician identify the "true" efficacy of the drug and reducing the uncertainty about this true efficacy. Over time, as the physician learns about the drug and experience develops, the uncertainty about a drug's efficacy is substantially reduced, and the effects of detailing are likely to be more direct and to dominate the indirect effect. Behavioral research (see Feldman and Lynch 1988; Mitra and Lynch 1995) has also documented that the role of advertising is different for consumers who are familiar with a product versus those who are not. Specifically, this research shows that even after the informative role of advertising has dissipated, there is still a residual role of advertising through effects such as reminders.

As we mentioned previously, the data set for our empirical analysis is from a category of ethical drugs. Our data set consists of aggregate data on second-generation antihistamines (that treat allergies) for the total United States market. A unique feature of this data set is that we observe marketing activities and aggregate physician prescription behavior from the time of introduction of the category. We also observe it for a relatively long period after its introduction. These features of the data enable us to investigate the effects of marketing communication in both the introductory and subsequent stages of the life cycles of the brands in the category. Ethical drugs are particularly suitable to the study of the role of marketing communication on the evolution of preferences because there is substantial uncertainty about how patients respond to treatments. In addition, the majority of marketing communication dollars are targeted at the physician (Wittink 2002).

We develop a brand-level discrete choice model of demand that allows for category expansion. The model allows for both direct and indirect effects of detailing and also controls for the effects of other promotional activities (e.g., direct-to-consumer advertising [DTCA], meetings, events). We find evidence for both the indirect and the direct effect of detailing on physicians' prescription behavior. In addition, we find that detailing has primarily indirect effects in the introductory phase (typically 6–14 months after introduction) but that the direct effects dominate subsequent stages. The finding that the direct effects are significant may explain why firms continue to detail long after a drug is introduced. We also find that, on average, physicians are more responsive to detailing than to other promotional activities.

The key contributions of this article are the following: First, it empirically distinguishes between two different effects of marketing communication and finds evidence for both. Second, it documents the temporal aspect of these two effects of detailing (i.e., the indirect effect dominates in the introductory phase of the product life cycle, and the direct effect subsequently dominates). Third, it provides empirical estimates for the length of time for which the indirect effect dominates. Finally, it fills the gap between research that studies new product categories without accounting for the behavioral process by which preferences evolve (e.g., Heilman, Bowman, and Wright 2000) and research that accounts for this behavioral process but does not study new products or product categories (e.g., Anand and Shachar 2001; Erdem and Keane 1996).

DATA

The data we used in this study are for the antihistamines market in the United States, and we obtained them from Verispan Inc., a firm that collects data on prescriptions written by physicians and on marketing activities of pharmaceutical firms. Our data contain monthly observations from April 1993 to December 2001 for the entire United States antihistamines market. We use the data for the three main second-generation antihistamine brands: Claritin (introduced in April 1993), Zyrtec (introduced in January 1996) and Allegra (introduced in August 1996). Clarinex, which is the fourth antihistamine in the category, was introduced in January 2002, and therefore we do not include it in our analysis. For the brands we use in our study, there are a total of 242 brand–month combinations.

As we mentioned previously, a unique feature of this data set is that we observe the category from its inception. Thus, the data do not suffer from the "initial conditions" problem that is common in models of the kind we use. We also observe the data for a fairly long period and at frequent (monthly) intervals. For each brand, we have information on the number of new prescriptions (NRx's) (no refills), written in that month; the average retail price (per treatment course) for a prescription; and expenditure on detailing, DTCA, and other marketing expenditures (OMEs) such as meetings and events. Verispan collected the NRx and retail price data through a pharmacy retail audit and the data on promotional expenditures for each drug directly from the

[2]This has also been referred to as "prestige" (Ackerberg 2001) or as a "complementary" (Bagwell 2003) effect.

[3]There is another stream of literature that infers that advertising is informative (persuasive) if it increases (decreases) price sensitivity and decreases (increases) prices in equilibrium. The studies find mixed results; some find an informative effect (Leffler 1981), and others find a persuasive

Table 1 contains descriptive statistics for the data. From the table, it is clear that detailing is the primary form of promotional activity directed at physicians. Expenditure on detailing is approximately six times greater than that on OMEs directed at physicians. The expenditure on DTCA is in the same range as that on detailing. Claritin is the brand with the largest number of NRx's in the category. It is also the highest priced brand and has the highest mean DTCA expenditure. However, Allegra has the highest mean detailing and OME. Table 1 also shows the seasonal effect that exists in this category. There are substantial differences between the number of prescriptions written in the months that constitute the allergy season and the number of prescriptions written in the other months.

MODEL

Prescription Decision

Although the prescription decision is a complex multiagent process that involves the physician, the patient, and possibly intermediaries such as insurance firms and health maintenance organizations, the final decision is the physician's because the drug is dispensed only on the basis of the physician's prescription. Thus, we abstract away from this multiagent process and assume that there is a single decision maker, who we henceforth refer to as the physician.

We assume that physicians value the health of their patients and that the physicians' preferences map onto a utility function over the space of treatment options. This may be due to their sense of professional integrity and/or a desire to avoid malpractice suits in the future and to maintain their reputation. When physicians must make a decision on treatment, we assume that they choose the option that gives them the greatest utility.[4] On the basis of the medical literature (Kelley and Good 1999) and our discussions with physicians, the drugs in this category are considered substitutes, and the use of multiple drugs to treat allergies is extremely rare. Therefore, we assume that physicians make a discrete choice among available options (i.e., they choose only one of the alternatives for a particular patient). Furthermore, we assume that drugs are bundles of characteristics and that physicians have utility over those bundles. Physicians observe these characteristics, but they may be uncertain about some. Given that physicians imperfectly observe one or more of these characteristics before making a decision, they maximize the expected utility of the alternatives at the decision stage.

For our purposes, we assume that physicians have imperfect knowledge about the mean efficacy of the drug. The underlying dimensions that constitute the efficacy of a drug are, among others, how well it treats the condition for which it is prescribed, the severity of the side effects that patients experience, the time it takes to treat the condition, and the patient's posttreatment state of health. Thus, the physician has a belief about the mean efficacy, but this is in the form of a distribution. This belief is updated as the physician learns about the drug through prescription experience and through information received from pharmaceutical firms.

Table 1
DATA DESCRIPTIVES

Variable	Brand	Mean	Standard Deviation
Monthly NRx's (in thousands of units): spring allergy season (March–June)[a]	Allegra	823	395
	Claritin	1482	811
	Zyrtec	590	309
Monthly NRx's (in thousands of units): autumn allergy season September–October)[a]	Allegra	628	382
	Claritin	1343	630
	Zyrtec	558	231
Monthly NRx's (in thousands of units): nonseasonal months (January–February, July–August, November–December)[a]	Allegra	532	305
	Claritin	1101	522
	Zyrtec	432	208
Average retail price[b] ($)	Allegra	39	4
	Claritin	48	6
	Zyrtec	41	1
Monthly detailing expenditure (in thousands of dollars)	Allegra	7334	2906
	Claritin	6802	2473
	Zyrtec	6240	2020
Monthly DTCA expenditure (in thousands of dollars)	Allegra	5263	4187
	Claritin	5531	6423
	Zyrtec	4542	4554
Monthly OME (in thousands of dollars)	Allegra	1242	821
	Claritin	1034	1071
	Zyrtec	1123	879

[a]NRx refers to the total number of new prescriptions in the United States.

[b]We report the average retail price for the entire course of a prescription. Prices and all marketing expenditures are deflated by the Consumer Price Index with January 1991 as the base. We obtained the Consumer Price Index data from the Bureau of Labor Statistics at http://www.bls.gov.

Learning Process

We assume that physicians begin with an initial prior belief about the mean efficacy of the drug when it is first introduced. Note that because physicians are uncertain about the mean efficacy of the drug, this initial prior belief is represented by a distribution. At each time period, physicians use three sources of information to update their prior beliefs about the mean efficacy of each drug in a Bayesian fashion. This information set consists of (1) the feedback received from patients who were prescribed the drug in the last period,[5] (2) information that pharmaceutical firms provide through detailing, and (3) OMEs directed at physicians. We refer to these as feedback, detailing, and OME signals, respectively.

The following is our list of assumptions about the learning process: First, given that we are not aware of any data source that informs us about the nature of patient feedback (e.g., proportion, frequency, timing), we assume that the number of feedback signals is equal to the number of pre-

[4]However, recent research has shown that for chronic conditions, the physician's preferences on the course of therapy may be different from those of the patients (see Fraenkel et al. [2004] and the references therein).

[5]Although we do not have (free) samples in our data, the effect of sampling is captured through the previous prescriptions. Thus, we must assume that feedback from a regular prescription is not systematically different from a sample-based prescription. We capture any residual effect of samples in the econometric error term.

scriptions written in the previous period.[6] Note that this is a conservative assumption because the mean number of patient trips made annually in this category is less than one (National Center for Health Statistics 2000). Second, we also assume that the numbers of detailing and OME signals in a time period are equal to the number of dollars spent on detailing and OME in that period.[7] Third, the aggregate nature of our data (i.e., we observe only the total amount spent on detailing or OME by a firm for each time period) imposes the assumption that all physicians receive the same number of detailing and OME signals.[8] However, we allow the signal content to differ across physicians. Fourth, the nature of our data also imposes the assumption that all signals (detailing and OME) are received at the beginning of the time period of our data. That is, all the information in the details or OME in a particular period is available to the physician at the beginning of the period. Similarly, the feedback signals of the prescriptions written in the previous period are also available at the beginning of each period. Finally, at each stage, we assume that physicians update their beliefs about the efficacy of the drugs in a Bayesian manner; that is, they have a set of prior beliefs based on the information set that is available up to the previous period, and they update this with the information set of the current period to form a set of posterior beliefs. Physicians then use this set of posterior beliefs to make decisions in the current period. This set of posterior beliefs forms the set of prior beliefs for the next period.[9]

Heterogeneity

We assume that physicians are heterogeneous in their responses to various linear characteristics in their utility function (e.g., price). This is likely because each physician can potentially treat a different set of patients. For example, the distribution of the price coefficient could represent the distribution of the mean price coefficient for the patients of different physicians.

Specification: Learning About Efficacy

In this section, we describe the learning process for an individual physician. Let $\tilde{Q}_{pjt}$ denote the physician's belief about the mean efficacy of drug j at time t, where the ~ sign indicates that it is a random variable from the point of view of the physician. This is conditional on the information set of the physician up to time t. Let $\bar{Q}_{pjt}$ denote the mean of this belief (distribution) at time t, and let σ^2_{Qjt} be the variance of this belief.[10] Let nd_{jt}, ns_{jt}, and nm_{jt}, respectively denote the number of detailing, feedback, and OME signals for brand j at time t. Let Q_j denote the true mean efficacy of the drug. As we described previously, efficacy is a broad term that includes how well the drug treats the condition, its side effects, and so forth.

At time t = 0, we assume that the initial belief of the physician about the mean efficacy of Drug 1 (the only drug in the market) is normally distributed.

$$\tilde{Q}_{p,j=1,0} \sim N(Q_0, \sigma^2_{Q_0}). \tag{1}$$

Similarly, in the time period in which a new drug is introduced, we have a similar expression for the initial belief of the physician. Furthermore, for simplicity, we assume that the initial belief has the same distribution for all drugs in the category. Thus, it will have the same mean and variance as in Equation 1.

The ith feedback signal at time t for drug j, which we assume to be normally distributed, is given by

$$R_{pijt} = Q_j + \upsilon_{pijt}, \quad \upsilon_{pijt} \sim N(0, \sigma^2_{\upsilon}). \tag{2}$$

We also assume that the ith detailing signal for drug j at time t is normally distributed because there is variation across individual physician–detailer interactions. The signal is given by

$$D_{pijt} = Q_j + \omega_{pijt}, \quad \omega_{pijt} \sim N(0, \sigma^2_{\omega}). \tag{3}$$

Similarly, the ith OME signal for drug j at time t is given by

$$M_{pijt} = Q_j + \eta_{pijt}, \quad \eta_{pijt} \sim N(0, \sigma^2_{\eta}). \tag{4}$$

Thus, we assume that the detailing, OME, and feedback signals are all normally distributed around the true mean efficacy. The implicit assumption is that these signals are truthful (i.e., they are equal to the true mean efficacy of the drug in expectation). We also assume that these signals are independent, which is an assumption that could potentially be relaxed with richer data that had more variation than our current data set. The variances in Equations 1–4 are unknown parameters.

At the beginning of time t, the physicians' beliefs are formed by updating their beliefs at time (t – 1) with the feedback, OME, and detailing signals available at the start of time t. Given that the initial prior distribution (i.e., at time t = 0 for Drug 1 and the respective time periods when the other drugs are introduced) is normally distributed and all three signals are normally distributed, the self-conjugacy of the normal distribution implies that the posterior belief in any time period would also be normally distributed. Thus, the belief at the beginning of time t is given by

[6]This is not a problem as long as we are willing to assume that the ratio of feedback signals to prescriptions written remains fixed in every period. In addition, because we assume that the number of feedback signals is proportional to the number of prescriptions in the previous period, this could potentially incorporate other forms of information that are also proportional to the number of prescriptions. We are indifferent about the process through which this feedback is received.

[7]On the basis of our discussion with industry experts, it seems that the cost of a detail is similar across the three firms in our data. Thus, we need to assume a common scaling factor to go from dollars to number of calls. In our case, we set this scaling factor to one (i.e., the number of detailing signals is equal to the number of dollars spent on detailing). Similarly, for OME, we need to assume a common average cost per meeting.

[8]In effect, we must assume that each physician receives a fixed proportion of the total signals in the market and that this proportion is unchanged over time.

[9]Early studies that used a Bayesian learning process to model category-level diffusion include those of Stoneman (1981), Meyer and Sathi (1985), and Roberts and Urban (1988). More recent studies include those of Erdem and Keane (1996), Crawford and Shum (2005), Coscelli and Shum (2004), Mukherji (2002), Ching (2002), Anand and Shachar (2001), Ackerberg (2003), and Byzalov and Shachar (2004).

[10]Note that this variance does not vary by physician, because the initial prior belief is assumed to be the same for all physicians, and they receive the same number of signals in every period. As Equation 11 shows, the variance of the belief in any period is a function of the variance of the prior and of the number of signals but not their realized values.

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