# Advertising as a Signaling Device in the Swedish Pharmaceuticals Market

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August 2003

**Abstract**

The paper empirically studies whether pharmaceutical firms uses advertising as a signal for high quality drugs. A nested random effects count data hurdle model is introduced to handle the excess number of zero observations in the sample as well as nested random drug, firm and substance specific effects. The empirical study indicate that drug quality (measured as the number of side-effects) do not influence pharmaceutical firms decision to advertise or not, but do affect the number of ads in a given period. The higher quality of the drug the more ads.

**Key words:** Signaling, pharmaceutical industry, advertising, product quality, nested random effects, count data.
**JEL classification: L15, L21**

*The authors would like to thank Kurt Brännäs and the participants at a seminar at Umeå University for helpful comments and suggestions. Financial support from the Jan Wallander and Tom Hedelius foundation is also gratefully acknowledged.

# 1 Introduction

In all markets for experience goods where consumers are uncertain about product quality, producers want to signal product quality. Although much effort has been spent theoretically analyzing the relationship between advertising and pricing and product quality (see e.g. Milgrom and Roberts, 1986; Bagwell and Riordan, 1991; Zhao, 2000 and Herzendorf and Overgaard, 2001), less effort has been spent trying to empirically verify the theoretical results. The purpose of this paper is to test the descriptive validity[1] of the relationship between advertising and product quality using data from the Swedish pharmaceuticals market.

The origin of the signaling literature in advertising dates back to Nelson (1970, 1974). In his model, repeat purchases creates a positive correlation between advertising and product quality since only high quality producers are assumed to attract repeat purchases. This requires that low quality producers are unable to recoup the costs of advertising if they were to mimic the advertising strategy of a high quality producer. This idea have been formalized by Milgrom and Roberts (1986) who develop a game between a high quality producer and a low quality producer. In their model both price and advertising can be used as signals, and if the low quality producer is able to mimic the high quality producers price, the high quality producer will respond by using advertising as a signal.

Before a pharmaceutical product reaches the market, it has to be approved by the Swedish Medical Products Agency (SMPA). Thus, pharmaceutical firms will engage in R&D and product testing to the extent that they are convinced that their product will be approved, but not until they know the product quality with certainty. As such, the pharmaceutical company will still be potentially liable if their product causes side effects after the introduction into the market. However, after the testing procedures, it is reasonable to assume that the firm has information concerning product quality which is superior to the prescribing physicians. Under these circumstances the high quality producer will have an incentive to signal high quality to the prescribing physicians. This could be achieved by using advertising as a signaling device, since the price (another common signaling device) of pharmaceutical products is, in practice, regulated in the Swedish market. Formally, the firms can set prices freely. However, in order for the patient's

[1] I.e. study if empirical data support the predictions from theory.

costs to be reimbursed, the Swedish National Social Insurance Board (NSIB) must accept the prices set by the firms. In addition, a so-called reference-price system was introduced in 1993**.** The system limits the reimbursement to patients who purchase pharmaceuticals to 110 percent of the price of the least-cost identical generic alternative. If the pharmaceutical companies were to set prices high in order to signal high quality, their products would not be reimbursed under the Swedish reference price system, which would decrease the demand for that product considerably. If firms were to use a low price in order to signal quality and increase initial sales, firms would have to keep these low prices indefinitely (unless price increases could be justified to the NSIB by increased input costs). As such, price is not (in practice) a viable signaling instrument in the Swedish pharmaceuticals market, while advertising is.

Physicians know the quality of the pharmaceutical product only after having prescribed the product to his/her patients and observing the outcome. The pharmaceutical products are thus experience goods for the prescribing physicians. As this is the case, the physician should rationally ignore any *a priori* claims by producers of pharmaceutical products to be of high quality. As noted by Spence (1973), it is then not necessarily the informational content of the signal, but rather the level of signaling that affects the behavior of the individual.

This paper contributes to the literature in the following ways; Firstly, this is (to our knowledge) the first study concerning product quality signaling in pharmaceutical markets. Secondly, the use of a hurdle model makes it possible to account for informational advertising aimed at making prescribing physicians aware of the product (i.e. announcing the product to the market), and other types of informational advertising aimed at informing the physicians of observable product characteristics (i.e. package size, price of the product etc.). Thirdly, the paper introduces nested random effects in a Poisson hurdle model framework.

The paper is organized as follows. Section 2 specify the hypotheses concerning product quality signaling to be tested in the empirical section. Section 3 presents the data and the empirical model used to test the hypotheses developed in section 2. This section also presents the results from the empirical analysis. Finally, section 4 concludes the paper.

# 2 Hypotheses development

In the Milgrom and Roberts (1986) model, firms use both price and advertising as signals. However, in their model advertising is a costly signal, and it is only used in cases when price is insufficient in order to signal high quality. In the Swedish pharmaceuticals market price is not (in practice) a viable signaling instrument, while advertising is. As mentioned above, if the pharmaceutical companies were to set prices high in order to signal high quality, their products would not be reimbursed under the Swedish pharmaceuticals insurance system, and this would decrease the demand for such products considerably. If firms were to use a low price in order to increase initial sales and signal quality, firms would have to keep these low prices indefinitely under the Swedish system.

Most of the earlier literature concerning advertising signaling models assume that the type of advertising used for signaling is purely persuasive (in the sense that the level, and not the content, of such advertising is assumed to provide information about product quality). In practice, pharmaceutical firms use two types of advertising, informative and persuasive. In this paper, it is assumed that informative advertising provides two types of information to the prescribing physician; firstly, it makes the physician aware of the product and, secondly, it makes the physician informed about observable product characteristics (i.e. package size, price etc). These types of informative advertising will be controlled for by including market size in the empirical estimations.[2]

Consider a new pharmaceutical product in the Swedish market and assume that information about the true quality of a product is not available for the prescribing physician before any of his/her patients has used the product. After the first period, where all prescribing physicians are uninformed, there are two types of physicians. First, there are physicians which have prescribed the product in question and know the quality. Second, there are physicians which have not prescribed the new product who are uninformed about product quality. Over time, as an increasing number of prescribing physicians come in contact with the new pharmaceutical product, an increasing number of physicians become aware of the true quality of the product. Manufacturers of high quality pharmaceutical products can expect more repeat sales to

[2]Butters (1977), Grossman and Shapiro (1984) and Thomas et al (1998)) all assume that the level of informational advertising can be controlled for by including market size in the empirical specifications.

prescribing physicians than low quality producers. High quality producers will thus find it more profitable to use advertising as a signal than low quality producers, ceteris paribus.This reasoning leads to the following hypotheses:

H1: Manufacturers of high quality products will advertise more frequently than low quality producers, after controlling for market size.

H2: Over time, as prescribing physicians obtain more information about the pharmaceutical product, advertising levels will decline as the signaling effect decline, after controlling for market size.

Note, as mentioned by Thomas et al (1998), that these hypothesis do not constitute a formal test of the signaling hypothesis. Firstly, Milgrom and Roberts assume purely uninformative (persuasive) advertising, while in this paper it is assumed that the level of informative advertising is a function of market size and can be controlled for statistically. Secondly, the theoretical models are all equilibrium models, while our available data does not allow for estimation of equilibrium advertising levels as a function of product quality. However, following Thomas et al (1998), it is believed that the assumptions made above more accurately reflect reality, while still allowing for an empirical study of the relationship between advertising levels and product quality in the Swedish pharmaceuticals market.

# 3 Empirical test

## 3.1 Data

The empirical estimations in this paper are based on quarterly time-series data with respect to prices and quantities for each original product and its generic substitutes from at most 1972 to 1996. Our data, which have been provided by the Swedish Medical Product Agency, refers to 26 different substances. In addition, the products used in the study refer to the dose and package size with the largest registered sales, resulting in a dataset consisting of 3362 observations. *Ads* refer to the mean and standard deviation of the dependent variable, while the other variables in Table 1 concern the mean and standard deviation of the independent variables used in the estimations below.[3]

[3]The empirical specifications also include a trend variable not included in Table 1.

Table 1. Descriptives.

| Substance | $Ads$ | $Dgen$ | $Cum$ $side\text{-}eff.$ | $Nr.\ of$ $firms$ | $Ref.$ | $E(rev_{t+1})$ |
|---|---|---|---|---|---|---|
| Allopurinol | 0.29 | 0.61 | 109.34 | 3.09 | 0.26 | 630.06 |
| | (1.07) | (0.49) | (73.07) | (1.02) | (0.44) | (774.20) |
| Alprazolam | 1.06 | 0.10 | 15.00 | 1.23 | 0.33 | 590.56 |
| | (1.94) | (0.31) | (8.58) | (0.42) | (0.48) | (249.71) |
| Atenolol | 0.62 | 0.70 | 88.47 | 4.07 | 0.37 | 2667.22 |
| | (1.68) | (0.46) | (74.37) | (1.55) | (0.48) | (4433.84) |
| Azapropazone | 0.39 | 0.00 | 98.64 | 1.00 | 0.15 | 111.08 |
| | (0.90) | (0.00) | (29.82) | (0.00) | (0.36) | (158.82) |
| Buspirone | 2.35 | 0.00 | 58.26 | 1.00 | 0.65 | 4914.65 |
| | (0.00) | (0.00) | (26.92) | (0.00) | (0.49) | (2535.91) |
| Cimetedine | 0.43 | 0.71 | 145.03 | 3.56 | 0.23 | 1943.62 |
| | (1.39) | (0.46) | (77.82) | (0.73) | (0.42) | (3775.63) |
| Diklofenac | 1.04 | 0.25 | 210.50 | 1.51 | 0.30 | 2679.14 |
| | (1.78) | (0.43) | (143.12) | (0.50) | (0.46) | (1791.40) |
| Etacrynic Acid | 0.00 | 0.00 | 15.58 | 1.00 | 0.12 | 26.56 |
| | (0.00) | (0.00) | (7.68) | (0.00) | (0.32) | (49.82) |
| Furosemide | 0.78 | 0.76 | 176.71 | 4.59 | 0.26 | 1210.42 |
| | (2.30) | (0.43) | (133.59) | (1.31) | (0.44) | (1209.62) |
| Clomipramine | 0.21 | 0.14 | 104.78 | 1.30 | 0.21 | 805.16 |
| | (0.74) | (0.35) | (82.29) | (0.46) | (0.41) | (442.73) |
| Lofepramine | 1.19 | 0.00 | 74.08 | 1.00 | 0.17 | 1051.81 |
| | (1.65) | (0.00) | (48.42) | (0.00) | (0.38) | (516.16) |
| Lorazepam | 0.63 | 0.00 | 13.88 | 1.00 | 0.18 | 520.75 |
| | (1.15) | (0.00) | (6.05) | (0.00) | (0.38) | (170.14) |
| Maprotiline | 0.25 | 0.35 | 116.32 | 1.97 | 0.37 | 422.14 |
| | (1.29) | (0.48) | (104.72) | (1.00) | (0.48) | (325.91) |
| Mianserine | 0.19 | 0.26 | 139.89 | 1.74 | 0.61 | 294.04 |
| | (0.65) | (0.44) | (105.68) | (0.86) | (0.49) | (188.05) |
| Naproxen | 0.48 | 0.70 | 222.95 | 4.80 | 0.34 | 1510.93 |
| | (1.30) | (0.46) | (146.75) | (1.63) | (0.48) | (1642.79) |
| Oxazepam | 0.60 | 0.28 | 24.23 | 1.80 | 0.17 | 1481.05 |
| | (1.01) | (0.45) | (18.09) | (0.80) | (0.38) | (1189.87) |
| Pindolol | 0.41 | 0.44 | 57.21 | 2.31 | 0.21 | 777.30 |
| | (1.69) | (0.50) | (49.26) | (0.94) | (0.41) | (774.41) |
| Piroxicam | 0.62 | 0.17 | 23.53 | 1.35 | 0.40 | 610.63 |
| | (1.50) | (0.38) | (12.71) | (0.48) | (0.49) | (239.41) |

Table 1. Descriptives (continued).

| Substance | $Ads$ | $Dgen$ | $Cum.$ $side\text{-}eff.$ | $Nr.\ of$ $firms$ | $Ref.$ | $E(rev_{t+1})$ |
|---|---|---|---|---|---|---|
| Protriptyline | 0.00 | 0.00 | 9.61 | 1.00 | 0.12 | 25.66 |
| | (0.00) | (0.00) | (4.48) | (0.00) | (0.32) | (54.62) |
| Sukralfate | 1.82 | 0.36 | 18.08 | 1.73 | 0.16 | 1340.74 |
| | (2.35) | (0.48) | (9.85) | (0.45) | (0.37) | (1119.15) |
| Sulindac | 0.92 | 0.00 | 254.00 | 1.00 | 0.21 | 1624.97 |
| | (1.70) | (0.00) | (94.98) | (0.00) | (0.41) | (512.66) |
| Estradiol | 0.47 | 0.16 | 67.27 | 1.50 | 0.45 | 557.45 |
| | (1.29) | (0.37) | (47.77) | (0.83) | (0.50) | (391.40) |
| Diazepam | 0.47 | 0.73 | 77.09 | 3.29 | 0.18 | 517.47 |
| | (1.45) | (0.44) | (41.59) | (0.46) | (0.38) | (401.83) |
| Indometacine | 1.00 | 0.61 | 252.42 | 2.85 | 0.21 | 354.13 |
| | (2.37) | (0.49) | (178.29) | (0.92) | (0.40) | (323.02) |
| Propranolol | 0.25 | 0.57 | 50.30 | 2.90 | 0.31 | 943.49 |
| | (1.01) | (0.50) | (42.30) | (1.03) | (0.46) | (1216.13) |
| Timolol | 0.01 | 0.51 | 41.68 | 2.81 | 0.35 | 2005.81 |
| | (0.17) | (0.50) | (34.60) | (1.22) | (0.48) | (2290.47) |
| TOTAL | 0.55 | 0.49 | 111.07 | 2.83 | 0.26 | 1095.54 |
| | (1.60) | (0.50) | (116.97) | (1.56) | (0.44) | (1922.74) |

The independent variables are: a dummy variable for generic firms ($dgen$), the cumulative number of side-effects ($cum\ side\text{-}eff.$), the number of firms (brand-name and generic) selling a specific substance ($Nr.\ of\ firms$) and a dummy variable for the introduction of the reference price system ($ref$). In addition, the expected future revenue one period ahead, $E(rev_{t+1})$, is predicted by using the parameter estimates from the following model

$$rev_{i,t} = \alpha + \beta_i rev_{it-1} + t_{it-1} + \varepsilon_{it}.$$

## 3.2 Econometric specification

Since the amount of advertising is measured as the number of ads per quarter[4], i.e. a non-negative integer-valued observation, and since the sample contain a large number of zeroes (85 percent of the observations are zeros, an excess of zeros not consistent with commonly used count data models) a

[4]The number of ads is used as a proxy for the advertisment cost.

count data hurdle modelling approach is chosen (e.g., Mullahy, 1986). The hurdle model has an interpretation as a two-part model. The first part is a binary outcome model, modelling a zero or a positive outcome, and the second part is a zero truncated count data model conditional on the positive outcome. In a given period of time the pharmaceutical firms are assumed to first decide whether to advertise for a particular pharmaceutical or not. Conditional on deciding to advertise they then choose the number of ads for that period.

In panel data analysis it is customary to control for unobserved individual (drug) specific effects (fixed or random). In the present analysis it also seem reasonable, in addition to drug specific unobserved effects, to control for unobserved effects at the substance, as well as, at the firm level. The motivation for unobserved firm specific effects is that firms marketing strategies may differ in an unobserved manner. Pharmaceutical firms may devote different (unobserved) attention to marketing through official journals/papers contra direct marketing activities towards physicians. The motivation to control for substance specific unobserved heterogeneity is that the value of signaling a high quality may differ among different fields of use (unobserved by the researcher) for the substance. To account for these unobserved specific effects a nested-error component structure[5] is introduced for the Poisson hurdle panel regression model.

Let $\mathbf{y} = \{y_{ijkt}\}$ be the number of ads for drug $i = 1, ..., n$ across time periods $t = 1, ..., T$. Assume that $y_{ijk} = (y_{ijk1}, ..., y_{ijkT})$ is Poisson distributed conditionally on the parameter vector $\boldsymbol{\beta}$ and an unobserved heterogeneity term $u_{ijk}$, i.e. $y_{ijk}|\boldsymbol{\beta}, u_{ijk} \sim$Poisson$(\theta_{ijk})$. To allow for heterogeneity at the drug, firm and substance level, the heterogeneity term is specified as

$$u_{ijk} = v_i + \gamma_{ij} + \mu_{ik},$$

where $v_i \sim iid\ N(0, \sigma_v^2)$ is the drug specific random effect, $\gamma_{ij} \sim iid\ N(0, \sigma\gamma^2)$ is the firm specific random effect, for firm $j = 1, ..., J$, and $\mu_{ik} \sim iid\ N(0, \sigma\mu^2)$ is the substance specific random effect, for substance group $k = 1, ..., K$. The specific random effects are assumed independent of each other. The Poisson nested random effects hurdle model is obtained by letting $\theta_{1i} = \exp(\mathbf{x}_{ijk}\boldsymbol{\beta}_1 + u_{ijk})$ be the mean function for the binary choice model and $\theta_{2i} = \exp(\mathbf{x}_{ijk}\boldsymbol{\beta}_2 + u_{ijk})$ be the mean function governing the positive outcomes

[5] In the linear panel data literature this type of group and sub-group effects have been modelled by nested/hierarchical-error component models (e.g. Antweiler, 2001).

($y_{ijk} > 0$). The probabilities of observing a zero count, respectively, a one count (1, for $y_i > 0$), in the binary part of the model, is given by

$$\Pr[y_{ijk} = 0|x_{ijk}, u_{ijk}] = \int_{-\infty}^{\infty} \exp(-\theta_{1i}) dF(u_{ijk}), \tag{1}$$

$$\begin{aligned}\Pr[y_{ijk} = 1|x_{ijk}, u_{ijk}] &= \int_{-\infty}^{\infty} (1 - \Pr[y_{ijk} = 0|x_{ijk}, u_{ijk}]) dF(u_{ijk}) \\ &= \int_{-\infty}^{\infty} (1 - \exp(-\theta_{1i})) dF(u_{ijk}).\end{aligned}$$

For the second part of the hurdle model the truncated-at-zero Poisson distribution is given by

$$\Pr(y_{ijk}|\mathbf{x}_{ijk}, u_{ijk}, y_{ijk} > 0) = \int \frac{e^{-\theta_{2i}} \theta_{2i}^{y_{ijk}}}{(1 - e^{-\theta_{2i}}) y_{ijk}!} dF(u_{ijk}), \quad y_{ijk} = 1, 2, \ldots . \tag{2}$$

Since closed form analytic expressions for the integrals are not easily obtained and since numerical integration methods usually do not perform well for high-dimensional problems, simulated maximum likelihood estimation is employed. The simulation based estimation approach[6] recognizes that the distributions in equation 6 and 7 are the expected values with respect to the random term $u_{ijk}$, i.e.

$$\begin{aligned}\Pr(y_{ijk} &= 0|\mathbf{x}_{ijk}) = E_u[\Pr(y_{ijk} = 0| \exp(\mathbf{x}'_{ijk}\boldsymbol{\beta}_1 + u_{ijk}))] \\ \Pr(y_{ijk} &= 1|\mathbf{x}_{ijk}) = E_u[\Pr(y_{ijk} = 1| \exp(\mathbf{x}'_{ijk}\boldsymbol{\beta}_1 + u_{ijk}))] \\ &= E_u[1 - \Pr(y_{ijk} = 0| \exp(\mathbf{x}'_{ijk}\boldsymbol{\beta}_1 + u_{ijk}))] \\ \Pr(y_{ijk}|\mathbf{x}_{ijk}, y_{ijk} &> 0) = E_u[\Pr(y_{ijk}| \exp(\mathbf{x}'_{ijk}\boldsymbol{\beta}_2 + u_{ijk}))].\end{aligned}$$

This expectation can be approximated by drawing independently, for each observation of $(y_{ijk}, x_{ijk})$, $S$ simulated values of $u^s_{ijk}$, $s = 1, ..., S$, from the distribution of $u_{ijk}$. For convenience $u^s_{ijk}$ may be obtained by sampling $\varepsilon^{s1}_i$, $\varepsilon^{s2}_{ij}$ and $\varepsilon^{s3}_{ik}$ from a standard normal distribution and by forming $u^s_{ijk} = \sigma_v \varepsilon^{s1}_i +$

[6]For further details about simulation based estimation, see, e.g. McFadden (1989), Gouriéroux and Monfort (1991), Hajivassiliou and Ruud (1994).

$\sigma\gamma\varepsilon_{ij}^{s2} + \sigma\mu\varepsilon_{ik}^{s3}$. Note that $\varepsilon_i^{s1}$, $\varepsilon_{ij}^{s2}$ and $\varepsilon_{ik}^{s3}$ is constant over $t$ but vary over $i, j$ and $k$ respectively. The marginal density is approximated by forming

$$\begin{aligned}
\widehat{\Pr}(y_{ijk} &= 0|x_{ijk}) \approx \frac{1}{S}\sum_{s=1}^{S}\Pr(y_{ijk}=0|\exp(x'_{ijk}\beta_1 + u_{ijk}^s)) \\
\widehat{\Pr}(y_{ijk} &= 1|x_{ijk}) \approx 1 - \widehat{\Pr}(y_{ijk}=0|x_{ijk}) \\
\widehat{\Pr}(y_{ijk}|x_{ijk}, y_{ijk} &> 0) \approx \frac{1}{S}\sum_{s=1}^{S}\Pr(y_{ijk}|\exp(x'_{ijk}\beta_2 + u_{ijk}^s))
\end{aligned}$$

The SML estimator of $\theta_l = (\boldsymbol{\beta}_l, \sigma_{vl}, \sigma_{\gamma l}, \sigma_{\mu l})$, $l \in 1, 2$, is obtained by use of the simulated densities in the following log-likelihood functions[7]:

$$\hat{\boldsymbol{\theta}}_{1s} = \arg\max_{\boldsymbol{\theta}_1} \ \log\ L_{1s} = \sum_{h=1}^{H}(1-I_h)(\ln\widehat{\Pr}(y_{ijkh}=0) + I_h(\ln\widehat{\Pr}(y_{ijkh}=1) \tag{3}$$

for the binary model, and

$$\hat{\boldsymbol{\theta}}_{2s} = \arg\max_{\boldsymbol{\theta}_2} \ \left[\log\ L_{2s} = \sum_{h=1}^{H}\ln\widehat{\Pr}(y_{ijkh})\right],\ \forall\ y_{ijkh} > 0, \tag{4}$$

for the second part of the hurdle model. The indicator variable, $I_h = 1$ if $y_{ijkh} > 0$ and $I_h = 0$ if $y_{ijkh} = 0$. If $S, n \to \infty$ and $n/S \to 0$ or $\sqrt{n/S} \to 0$, then the SML estimator is consistent and asymptotically equivalent to the ML estimator with a limiting normal distribution (Gouriéroux and Monfort, 1991). A robust estimator of the covariance matrix is given by

$$V\left\{\sqrt{K}(\hat{\boldsymbol{\theta}}_s - \boldsymbol{\theta}_0)\right\} = \hat{\mathbf{H}}^{-1}(\boldsymbol{\theta}_0)\hat{\mathbf{I}}(\boldsymbol{\theta}_0)\hat{\mathbf{H}}^{-1}(\boldsymbol{\theta}_0)$$

where $\hat{H}(\theta_0) = -K^{-1}\partial^2 \ln\ L_s(\hat{\theta}_s)/\partial\theta\partial\theta'$ and the information matrix can be consistently estimated by

$$\hat{\mathbf{I}}(\boldsymbol{\theta}_0) = \frac{1}{K}\left[\frac{\partial \ln\ L_s(\hat{\boldsymbol{\theta}}_s)}{\partial\boldsymbol{\theta}}\frac{\partial \ln\ L_s(\hat{\boldsymbol{\theta}}_s)}{\partial\boldsymbol{\theta}'}\right].$$

[7]The full log-likelihood for the $h$ observations is given by $l = l_1 + l_2$ and is maximized by separately maximizing each component under the assumtion that that the binary part and the second part of the hurdle model are functionally independent.

## 3.3 Empirical results

Estimation results for the binary part of the model are given in Table 2. The number of side-effects, i.e. the quality variable, is not significant indicating that quality does not influence the decision to advertise or not. This indicates that, at least, part of the advertising have informational motives. In addition, the trend variable is negative, indicating that the motive for advertising (informational or persuasive) decrease over time as more physicians get informed of the product attributes by personal contact with the product.

Table 2: Estimates for the binary part

| Variable | Estimate | S.e. |
|---|---|---|
| $E(rev_{t+1})$ | 0.29* | 0.07 |
| $Nr.\ of\ firms$ | -0.30* | 0.12 |
| $ref$ | -0.95* | 0.30 |
| $dgen$ | -2.68* | 0.56 |
| $cum\ side\text{-}eff$ | 0.21 | 0.13 |
| $Constant$ | -1.51* | 0.34 |
| $Trend$ | -2.19* | 0.40 |
| $v_i$ | 0.064 | 0.099 |
| $\gamma_{ij}$ | 2.56* | 0.63 |
| $\mu_{ik}$ | 2.51* | 0.75 |
| Log-likelihood | | -1208.30 |
| Observations | | 3362 |
| S | | 20 |

* Significant at the 5 percent level.

The results show that there is a positive and statistically significant relationship between advertising and estimated sales. This also indicates that, at least, some of the pharmaceutical firm's advertising consists of informational advertising (presenting package sizes, price, etc.), and that this type of advertising increase as market size increases. Note also that generic firms has a lower probability of advertising than brand name firms (Table 2), but if generic firms choose to advertise, they advertise more than brand name firms after controlling for market size (Table 3). The results from the binary part also indicate that more generic firms in the market lower the probability of advertising, and that the introduction of the reference price system in 1993 decreased the probability of a pharmaceutical firm advertising (after controlling for market size). The substance and firm specific random effects

are statistically significant indicating the importance of controlling for these effects.[8]

Estimation results for the quantity decision is given in Table 3. The results indicate that for a given pharmaceutical product more side effects (lower quality) has a negative and significant effect on the number of ads. Since both the binary part of the empirical model and the market size variable control for the informational effects of advertising, this indicates that pharmaceutical firms use the level of advertising as a signal of product quality in the Swedish pharmaceuticals market. The trend variable is negative and statistically significant at the 10 percent level, indicating that the signaling effect of advertising decline over time. This is to be expected as more physicians get informed of the true quality level trough personal experience with the product.

Table 3: Estimates for number of ads

| Variable | Estimate | S.e. |
|---|---|---|
| $E(rev_{t+1})$ | 0.043* | 0.013 |
| $Nr.\ of\ firms$ | 0.014 | 0.027 |
| $ref$ | 0.070 | 0.090 |
| $dgen$ | 0.33* | 0.068 |
| $cum\ side\text{-}eff$ | -0.089* | 0.034 |
| $Constant$ | 1.25* | 0.062 |
| $Trend$ | -0.13 | 0.072 |
| $v_i$ | 0.25* | 0.029 |
| $\gamma_{ij}$ | 6.16E-05 | 0.012 |
| $\mu_{ik}$ | 4.91E-04 | 0.018 |
| Log-likelihood | | -1101.61 |
| Observations | | 513 |
| S | | 20 |

* Significant at the 5 percent level.

Again the results show a positive and statistically significant relationship between advertising and estimated sales, indicating that the level of informational advertising increase as market size increases. The same result was found by Thomas et al (1998). Note also that neither the number of generic firms in the market, nor the introduction of the reference system affected the number of ads in Läkartidningen. The drug specific random effect is

[8] Neglecting to control for unobserved heterogeneity may render biased estimates.

statistically significant while the substance and firm-specific random effects are not.

# 4 Conclusion

The purpose of this paper has been to test the descriptive validity, i.e. study if empirical data support the predictions from theory, of the relationship between advertising and product quality using data from the Swedish pharmaceuticals market.

The use of a nested (hierarchical) random effects hurdle model in this paper makes it possible to control for the excess number of zeros concerning the number of ads. Another novelty of this econometric specification is that it makes it possible to specify the unobserved heterogeneity among drugs, pharmaceutical substances and pharmaceutical firms in a more elaborate manor than in an unnested random effects model. The empirical results concerning the (nested) random effects parameters ($v_i, \gamma_{ij}$ and $\mu_{ik}$) indicate that using a ordinary random effects model would have provided biased parameter estimates.

The results concerning advertising as a signaling device indicate that pharmaceutical firms use advertising both in an informational and an persuasive manor. Concerning the decision to advertise or not, product quality does not seem to influence this decision, indicating additional reasons for advertising other than signaling product quality. However, the amount of advertising (the number of ads), given that a firm advertises, is positively related to product quality (number of side-effects) supporting the hypothesis that pharmaceutical firms use advertising as a signaling device (see hypothesis 1, section 2). In addition, the trend variable is negative and statistically significant at the 10 percent level. This is interpreted as, at least, weak evidence that advertising levels will decline as the signaling effect of the ads decline (hypothesis 2, section 2).

# 5 References

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