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Reference Pricing, Competition, and
Pharmaceutical Expenditures: Theory and
Evidence from a Natural Experiment

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Reference Pricing, Competition, and Pharmaceutical Expenditures: Theory and Evidence from a Natural Experiment

Abstract

We study the impact of regulation on competition between brand-names and generics and pharmaceutical expenditures using a unique policy experiment in Norway, where reference pricing (RP) replaced price cap regulation in 2003 for a sub-sample of off-patent products. First, we construct a vertical differentiation model to analyze the impact of regulation on prices and market shares of brand-names and generics. Then, we exploit a detailed panel data set at product level covering several off-patent molecules before and after the policy reform. Off-patent drugs not subject to RP serve as our control group. We find that RP significantly reduces both brand-name and generic prices, and results in significantly lower brand-name market shares. Finally, we show that RP has a strong negative effect on average molecule prices, suggesting significant cost-savings, and that patients' copayments decrease despite the extra surcharges under RP.

JEL-Code: I11, I18, L13, L65.

Keywords: pharmaceuticals, regulation, generic competition.

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1 Introduction

In pharmaceutical markets new innovations are protected by patents that restrict competing firms from copying the innovation within a certain period. When the patent expires, competing firms may enter the market with generic products. The generic versions contain exactly the same active chemical ingredient and must prove therapeutic equivalence before they can be launched on the market. Since generics have the same therapeutic effect as the brand-name, one would expect that only relative prices matter for the consumers' (or physicians') choice of drug. Thus, generic entry should trigger fierce price competition between brand-names and generics. This is, however, not what is observed. A robust empirical regularity is that the brand-names charge a higher price than their generic versions and obtain significant market shares (e.g., Scherer, 2000).^{1,2}

The high market share of higher priced brand-names in the pharmaceutical off-patent market segment is a policy concern across Western countries. The reasoning is two-fold: first, innovation incentives should be taken care of in the patent period, so there is no argument for offering rents to brand-name producers after patent expiry.³ Second, the generic versions are supposed to provide exactly the same health benefit to consumers as the brand-names, implying that only relative prices should determine the choice of drug, although consumers might have a preference for the brand-name.⁴ Consequently, most Western countries impose regulations to control prices and expenditures, and to stimulate competition in the pharmaceutical market.^{5,6}

¹Some studies show that brand-name prices even increase when the patent expires and generics enter the market (Grabowski and Vernon, 1992, Frank and Salkever, 1997). Recent papers by Ching (2010a, 2010b) also show that brand-name prices increase as the number of generics become higher.

²Brand-name market shares decline after generic entry, but the extent seems to vary across countries, over time and across molecules (see e.g., Danzon and Chao, 2000).

³This is of course a strong assumption. It is hard for regulators to design a patent protection system that induces optimal innovation incentives, see e.g., Sakakibara and Branstetter (2001), Gallini (2002), Brekke and Straume (2009). However, it should be more efficient to address innovation incentives by refining the on-patent regulation rather than allowing for substantial rents in the off-patent period.

⁴If consumers subjectively value brand-names more than generics, high brand-name market shares should not be a policy problem. However, there are two reasons for regulation: first, due to insurance, patients do not face the full cost of choosing a high-priced brand-name instead of a cheaper generic. Second, the choice of drug is often made by physicians who might be inclined (e.g., due to marketing) to prescribe a brand-name rather than a generic despite therapeutical equivalence and price differences (see, e.g., Hellerstein, 1998).

⁵Danzon (1997) provides an excellent overview of theory and practice of price regulation in the pharmaceutical industry. See also Kanavos (2001) for a comprehensive overview of pharmaceutical regulation practices in 14 EU countries.

⁶Even in the US there are some price control mechanisms. For example, (generic) reference pricing is well-established through the "maximum allowable charge" programs used by Medicaid. The recent extension of Medicare to prescription drugs has spurred a debate of price controls also in the US (see e.g., Frank and Newhouse,

However, concerns have also been raised about potential negative effects of regulation. For example, Danzon and Chao (2000) argue that regulation drives out competition and is thus counter-productive in obtaining cost-savings.⁷ In the present study, we argue that it depends on how you regulate.

In this paper, we study the impact of the two most widely used regulatory regimes – price cap regulation and reference pricing (RP) – on competition and pharmaceutical expenditures in the off-patent market.^{8,9} Under price cap regulation the regulator curbs market power by enforcing maximum prices that firms are allowed to charge. RP, on the other hand, defines a maximum reimbursement that will be covered by the regulator. If consumers demand a product with a price above the RP, they will have to cover this extra cost out-of-pocket. Our study consists of a theoretical and an empirical part. The theoretical analysis is based on a vertical differentiation model, where we analyze price competition between brand-names and generics under the different regimes and derive empirically testable predictions. The empirical part of the paper exploits a unique policy experiment in Norway, where the government exposed a subsample of the off-patent drugs on the market to RP. The policy reform can thus be viewed as a natural experiment that provides us with a comparison group consisting of off-patent drugs subject to price cap regulation throughout the whole period. We use a rich product level panel data set covering a four-year period from 2001 to 2005 that gives us variation over time (before and after the reform) and across products that are subject to different regulatory regimes (price cap regulation or RP).

Our paper builds on Brekke et al. (2009) who exploit the same policy experiment. While they focus solely on the price responses by the pharmaceutical firms to the change in regulation, our

2008)

⁷They base their conclusion on a cross-national study using data for 1992, showing that price competition between generic competitors is stronger in unregulated or less regulated markets (United States, United Kingdom, Canada, and Germany) than in countries with strict price or reimbursement regulations (France, Italy, and Japan).

⁸Reference pricing (also called internal referencing) refers to a system where the regulator (payer) defines a maximum reimbursement based on the prices of therapeutically similar or equivalent drugs on the market (see, e.g., Danzon and Ketcham, 2004). Reference pricing and price cap regulation based on international prices (external referencing) has become increasingly popular and are now the two main regulatory schemes in Western countries. See e.g. Kanavos (2001) for a survey of pharmaceutical regulation in Europe.

⁹Reference pricing (internal referencing) is used by many European countries, but also outside Europe in countries like New Zealand, Australia, Canada (British Columbia), Brazil, etc. In the US, the "maximum allowable charge" (MAC) programs used by Medicaid can be classified as reference pricing. See e.g., Danzon and Ketcham (2004).

study differs and extends their work along several important dimensions.¹⁰ In the theoretical part we offer a much richer and more detailed analysis of RP. A main contribution here is the distinction between *endogenous and exogenous RP*, where in the former case the RP is set as a function of the market prices, so that the firms take into account that their pricing might also affect the RP. This distinction is important because, as will be shown, exogenous and endogenous RP have opposite effects on the generic firms' pricing incentives. If the RP is exogenous, generic firms have an incentive to increase their prices, which implies that RP might be counterproductive with respect to cost savings, as claimed by, for instance, Danzon and Chao (2000). However, if the RP is endogenous, we show that generic firms have a strategic incentive to lower their prices to influence the RP and increase market shares. In this case, RP would unambiguously result in cost saving. We argue that this new result has a direct policy implication with respect to the design of the RP system, more specifically the frequency with which the reference price is updated, since long (short) time intervals between each update of the reference price would correspond more closely to exogenous (endogenous) RP according to our theoretical model. Finally, our new theoretical result is also important because it is consistent with, and thus provides an explanation to, several previous empirical findings on reference pricing (see e.g. Aronsson et al., 2001, Pavcnik, 2002, Brekke et al., 2009).

In the empirical part, we extend the work by Brekke et al. (2009) along two dimensions. First, we study the impact of RP on the *competition between brand-names and generics*, measured by (brand-name) market shares. The direct effect of RP is to reduce brand-name market shares, but the following price reductions by the brand-name firms pull in the opposite direction. The overall effect on competition is therefore *a priori* ambiguous. By estimating the impact on brand-name market shares, the Norwegian policy experiment allows us to directly re-examine the concerns put forward by Danzon and Chao (2000) that regulation might drive out competition in off-patent pharmaceutical markets. Second, and perhaps more importantly, we study *welfare and policy implications* by estimating the effect of RP on overall pharmaceutical expenditures and consumer expenditures (copayments). Galloping pharmaceutical expenditures are a major policy concern in most developed countries and the main policy objective of RP is to reduce

¹⁰Brekke et al. (2009) found that the introduction of reference pricing triggered significant price reductions on both brand-names and generics. They also identified a negative cross-price effect on therapeutic substitutes of the drugs subject to the reform.

such costs by inducing more generic sales. However, RP might have the (unintended) adverse effect of simply shifting costs from the payer to the consumers, as they are now exposed to extra surcharges. It is therefore of great policy interest to examine more carefully not only the total size of cost savings but also to decompose the cost effect in terms of changes in total patient expenditures. Thus, in contrast to Brekke et al. (2009), we derive results concerning the competition between brand-names and generics and, importantly, draw more clear-cut welfare and policy implications based on the policy experiment.¹¹

Our paper provides two sets of findings. The first set is concerned with the *market outcomes* of the change in regulation. First, we find that RP leads to significant price reductions on both brand-names (33 percent) and generics (22 percent). This finding runs counter to the price convergence prediction by Danzon and Liu (1996) and Danzon and Ketcham (2004), who argue that while brand-names are likely to reduce their prices, generics will respond to RP by *increasing* their prices since demand becomes less price elastic below the RP. As explained above, we show in the theory section that their argument is correct only if the RP is (perceived to be) *exogenous* to the firms' pricing decisions. However, under an *endogenous* RP system the generic producers have a strategic incentive to lower their prices to reduce the RP and thus make the brand-name more expensive for the consumers.

This finding is in line with previous empirical studies. Pavcnik (2002) studies the introduction of (therapeutic) RP in Germany in 1989. Using data for two different therapeutic fields (oral antidiabetics and antiulcerants) for 1986 to 1996, she identifies significant price reductions of the RP system on both brand-names and generics, with the effect being stronger for brand-names. Similar results are obtained in Brekke et al. (2009), as mentioned above.¹² In Norway the RP was set as a weighted average of brand-name and generic prices and updated every third month. In fact, most countries use an RP rule that depends on firms' pricing.¹³ Thus, the robust empirical finding that RP reduces both brand-name and generic prices can be explained

¹¹In addition, we have extended our data set to cover more off-patent molecules than in Brekke et al. (2009), though excluding the on-patent molecules. We also perform several robustness checks to account for potential problems related to selection issues, endogeneity and serial correlation.

¹²Bergman and Rudholm (2003) study the effects of the Swedish RP system on brand-name (not generic) prices. Distinguishing between actual and potential competition from generics, they find that RP only reduced prices of brand-names that actually faced competition from generics firms.

¹³See, for instance, the survey by Lopez-Casasnovas and Puig-Junoy (2000).

by our theoretical model of an *endogenous* RP system.¹⁴

Secondly, we find that RP stimulates competition between brand-names and generic competition by significantly reducing the brand-names' market share (almost 15 percent). The introduction of RP makes the brand-name more expensive for consumers *for given prices*, which suggests a shift in demand towards generics. However, the brand-name firms respond to RP by substantially lowering their prices, which pull in the other direction. In our theoretical model, we show that the direct demand effect of RP tends to dominate the indirect price effect, resulting in higher generic market shares. In the empirical part we separate the two counteracting effects by controlling for relative branded-generic prices. We find, however, no significant impact of changes in relative prices on the brand-name market shares. Thus, the reduction of almost 15 percent in brand-name market shares is a *direct demand response* to the radical change in co-payments brought about by the introduction of RP, which by far outweighs any indirect effects via the price responses.

This part of our analysis is closely related to Aronsson et al. (2001) who use Swedish data to analyze the impact of relative branded-generic prices and the introduction of RP on brand-name market shares.¹⁵ The effects of both relative prices and RP are weak and often insignificant. Estimating the impact of RP on the whole sample (12 molecules) provides no significant effects on brand-name market shares. They therefore run regressions at molecule level, where they report significant, though weak, effects for 5 out of 12 molecules. While the weak effect of relative prices is in line with our results, this is not the case for RP. However, our study differs from theirs in important ways. Most importantly, in Sweden the reform was introduced for all off-patent substances, while we have a natural experiment, which improves the scope for identification. Second, we have a more detailed and extensive data set, covering 24 molecules with monthly price and volume data.

Our second set of results are concerned with *welfare and policy implications*. Danzon and Chao (2000) suggest that average molecule prices (i.e., brand-name and generic prices weighted

¹⁴A recent study by Kaiser et al. (2010) reports very similar findings to ours. Using a change in the Danish system from exogenous RP to endogenous RP, they show that prices of both brand-name and generics decline. This study also uses a structural approach, which allows for joint estimation of price and quantity effects.

¹⁵Aronsson et al. (2001) interpret relative prices as a measure of competition between brand-names and generics. This seems highly imprecise since lower relative prices might be due to higher generic prices, which would hardly be equivalent to stronger competition from generics.

by their market shares) are good proxies for changes in pharmaceutical expenditures, since overall (molecule) demand is quite price inelastic. We first test this assumption and find no significant difference in volume trends for the drugs subject to the reform compared with the drugs under PC regulation over the whole period. We then proceed by estimating the effect of RP on the average molecule prices, finding that the introduction of RP triggers a reduction of almost 30 percent, which is a substantial cost saving. There are two different effects that contribute to these savings: (i) reductions in brand-name and generic drug prices, and (ii) a shift in demand from brand-names to generics.

Finally, we take a closer look at the consumer welfare since RP offers less insurance coverage to patients. For given prices and demand, RP implies a shift of medical costs from the payer (insurer) to the patients, resulting in cost-savings for the payer and a corresponding cost-increase for the patients. The question is therefore whether the price and market share effects can offset the direct increase in patient copayments due to RP. We demonstrate that not only the generic but also the brand-name copayments become lower due to price reductions. The price reductions for brand-names more than offset the extra surcharges due to RP. These effects are reinforced by the fact that RP induces consumers to switch to generics.

The rest of the paper is organized as follows. In Section 2 we develop a theoretical model and derive testable predictions for the empirical analysis. In Section 3 we present some institutional background by describing the price cap regulation and the policy experiment with reference pricing in Norway. In Section 4 we present our data and some descriptive statistics. In Section 5 we present the empirical method and our basic results with respect to pricing and market share effects. In Section 6 we perform various robustness checks, where we test the validity of our control group and account for serial correlation and endogeneity. In Section 7 we discuss welfare and policy implications by looking at the effects of RP on overall and patient expenditures. Finally, Section 8 concludes the paper.

2 A theoretical model

Consider a therapeutic market with products offered by two firms. Firm B offers the original (off-patent) brand-name drug b , while firm G offers a generic substitute g . Consumers are

heterogeneous with respect to the gross valuation of drug treatment, represented by a parameter τ which is uniformly distributed on the interval $[0, t]$. It would be natural to think of the heterogeneity of gross valuations as reflecting differences in severity levels, but it could also be interpreted as differences in prescription practices among physicians.¹⁶ The total mass of consumers is normalized to 1. Each consumer demands either one or zero units of the most preferred drug. The utility derived from no drug consumption is zero, while a consumer who buys one unit of drug i obtains a net utility

$$U_i = \begin{cases} \theta\tau - c_b & \text{if } i = b \\ \tau - c_g & \text{if } i = g \end{cases}, \quad (1)$$

where $\theta > 1$ is the (perceived) quality difference – e.g., due to differences in advertising intensity – between the brand-name and the generic drug, and c_i is the patient copayment for drug i .¹⁷

A consumer with a positive net utility of drug consumption will choose the most preferred drug version by trading off drug quality against drug copayment. The higher the gross valuation of drug treatment, the more the consumer is willing to pay in order to purchase the (high-quality) brand-name drug. A consumer who is indifferent between the two drug versions has a gross valuation equal to $\hat{\tau}$, given by $\theta\hat{\tau} - c_b = \hat{\tau} - c_g$, yielding

$$\hat{\tau} = \frac{c_b - c_g}{\theta - 1}. \quad (2)$$

Consumers with a gross valuation higher than $\hat{\tau}$ demand the brand-name drug, while the remaining consumers demand the generic drug, as long as the net utility of drug consumption is

¹⁶For example, pharmaceutical detailing might influence a physician’s willingness to prescribe a cheaper generic substitute.

¹⁷As mentioned in the Introduction, there is strong empirical evidence that generic drugs are not perceived to be perfect substitutes to the original brand-name drug, despite being chemically identical. The findings of substantial and persistent branded-generic price differences after generic entry (see, e.g., Grabowski and Vernon, 1992, Frank and Salkever, 1997, Scott Morton, 2000, Ching 2010a, 2010b) fit well with predictions of vertical differentiation models. Two recent papers applying this approach to competition between brand-names and generics are Königbauer (2007) and Brekke et al. (2007).

non-negative. Total demand for the two drug versions are thus given by

$$D_b = \begin{cases} 0 & \text{if } c_b - c_g \geq t(\theta - 1) \\ \frac{1}{t}(t - \hat{\tau}) & \text{if } 0 < c_b - c_g < t(\theta - 1) \\ 1 & \text{if } c_b \leq c_g \end{cases} , \quad (3)$$

$$D_g = \begin{cases} 1 & \text{if } c_b - c_g \geq t(\theta - 1) \\ \frac{1}{t}(\hat{\tau} - c_g) & \text{if } 0 < c_b - c_g < t(\theta - 1) \\ 0 & \text{if } c_b \leq c_g \end{cases} . \quad (4)$$

In the following we focus on the intermediate case, where both drug versions have positive demand in equilibrium. From the above demand functions we can define the market share of the generic drug,

$$\gamma_g := \frac{D_g}{D_b + D_g}. \quad (5)$$

Assuming that marginal production costs of both drug versions are constant and equal to w , profits are given by

$$\pi_i = (p_i - w) D_i, \quad (6)$$

where p_i is the price of drug i ; $i = b, g$. Given the restrictions imposed by the regulatory regime in place, we assume that the two firms play a Bertrand game, simultaneously choosing drug prices to maximize profits.

2.1 No regulation

As a benchmark for comparison, consider the case of no regulation, where firms are free to choose drug prices and patient copayment is given by

$$c_i = f + \alpha p_i, \quad (7)$$

where $f > 0$ is a fixed fee and $\alpha \in (0, 1)$ is the coinsurance rate.¹⁸ To make sure that both firms are active in equilibrium, we impose the condition $f + \alpha w < \frac{t}{2}$.

The first-order conditions for profit maximizing drug prices yield the following best-response functions for the producers of the brand-name and generic drug, respectively:

$$p_b(p_g) = \frac{1}{2} \left[p_g + w + \frac{t(\theta - 1)}{\alpha} \right], \quad (8)$$

$$p_g(p_b) = \frac{1}{2\theta} \left[p_b + \theta w - \frac{f(\theta - 1)}{\alpha} \right]. \quad (9)$$

The best-response functions confirm that drug prices are strategic complements; a higher brand-name drug price induces a higher generic drug price, and vice versa.

Under free pricing, equilibrium drug prices are found by simultaneously solving (8)-(9), yielding

$$p_g^* = \frac{(\theta - 1)(t - 2f) + (2\theta + 1)\alpha w}{\alpha(4\theta - 1)}, \quad (10)$$

$$p_b^* = \frac{(\theta - 1)(2t\theta - f) + 3\theta\alpha w}{\alpha(4\theta - 1)}. \quad (11)$$

Since the brand-name drug is perceived to be of higher quality than the generic drug, firm B will set the higher price, $p_b^* > p_g^*$, and serve the consumers with higher gross valuation of drug treatment. The larger the degree of perceived vertical differentiation, θ , the larger the branded-generic price difference in equilibrium.

2.2 Price cap regulation

The equilibrium outcome under price cap regulation is a straightforward modification of the free pricing equilibrium derived above. If the producer of the brand-name drug faces a binding price cap, \bar{p}_b , set by a regulator, the equilibrium generic drug price is given by (9), with $p_b = \bar{p}_b$. Stricter price regulation makes the brand-name drug less expensive for consumers, inducing – all else equal – a shift in demand towards drug b . However, since prices are strategic complements, firm G will respond by lowering the price of the generic drug. An assessment of the total effect

¹⁸A copayment system with a fixed and a variable component is common for many countries (see, e.g., Kanavos, 2001). Notice, however, that the parameters α and f can be given several alternative interpretations. For example, α could be interpreted as the prescribing physician's price consciousness (see, e.g., Hellerstein, 1998), while f can be interpreted also as the (non-monetary) cost of attending a GP to obtain a prescription.

shows that the former (direct) effect dominates the latter (indirect) effect:

$$\frac{\partial \gamma_g}{\partial \bar{p}_b} = \frac{2\theta^2 \alpha (t - f - \alpha w)}{(\theta - 1) (\theta (2t - \alpha w) - f (\theta + 1) - \alpha \bar{p}_b)^2} > 0. \quad (12)$$

Proposition 1 *Under price cap regulation, a reduction in the (binding) price cap reduces the equilibrium market share of generics.*

In other words, stricter price cap regulation dampens generic competition. If price cap regulation is sufficiently strict, generic competition will be completely eliminated. The critical price cap, below which the generic producer will exit the market, is given by $\bar{p}_b^* = \frac{f(\theta-1)}{\alpha} + \theta w$. We see that the likelihood of price cap regulation driving out generic competition is increasing in the degree of perceived vertical differentiation and the fixed cost of drug consumption, while decreasing in the degree of coinsurance.

2.3 Reference pricing

Under a reference pricing (RP) system, firms are free to set drug prices, but patient copayment is based on a RP, r , that is set by a regulator.¹⁹ More specifically, if a consumer chooses a drug that is priced higher than the RP, she has to pay the full difference between the RP and the actual drug price. Usually, the RP is set at a level somewhere between the lowest and highest drug price in the market. For a RP $r \in (p_g, p_b)$, the copayment schedule is given by

$$c_i = \begin{cases} \alpha r + (p_b - r) + f & \text{if } i = b \\ \alpha p_g + f & \text{if } i = g \end{cases}. \quad (13)$$

In order to illustrate the decomposed effects of RP on drug pricing and generic competition, we will do the analysis in two steps: First, we consider the case where r is exogenous to the firms's pricing decisions, which we dub *Exogenous RP*. Subsequently, we endogenize r and make it a function of the prices set by the firms in the market. This scenario is dubbed *Endogenous RP*.

¹⁹Reference pricing is somewhat analogous to the model of yardstick competition by Shleifer (1985). However, there are several differences. First, the reference price is based on market prices rather than reported costs. Second, yardstick competition focuses on inducing cost reducing effort, while reference pricing aims are reducing prices. Finally, costs are not observable to regulators, while prices are.

2.3.1 Exogenous RP

Assume that the firms perceive the RP to be exogenously given. For $r \in (p_g, p_b)$, equilibrium prices are then given by

$$p_g^{rp}(r) = \frac{(\theta - 1)(t - 2f) + (2\theta\alpha + 1)w - r(1 - \alpha)}{\alpha(4\theta - 1)}, \quad (14)$$

$$p_b^{rp}(r) = \frac{(\theta - 1)(2t\theta - f) + \theta(2 + \alpha)w + r(2\theta - 1)(1 - \alpha)}{4\theta - 1}. \quad (15)$$

We can analyze the effects of RP by considering a marginal reduction in r . RP implies that the brand-name drug becomes relatively more expensive, and that drug demand becomes more elastic for prices above r . The resulting price responses are easily derived from (14)-(15): $\partial p_g^{rp} / \partial r < 0$ and $\partial p_b^{rp} / \partial r > 0$.

Proposition 2 *Under exogenous reference pricing, a reduction in the RP leads to a reduction (increase) in the brand-name (generic) drug price.*

This result is in line with the price convergence hypothesis²⁰: The introduction of reference pricing leads to a price convergence towards the RP; the generic drug becomes more expensive, while the brand-name drug becomes cheaper. In the case of no coinsurance, there is no incentive for the generic firm to cut price below the RP. However, the price convergence hypothesis ignores the fact that, in most reference pricing systems, the RP is determined as a function of actual drug prices and is thus *endogenous*. If the RP is frequently updated, the drug producers know that their price setting is going to affect the RP, and thereby demand and profits, in the future.

2.3.2 Endogenous RP

Assume that the RP is a weighted average of the brand-name and generic drug prices:

$$r = \beta p_g + (1 - \beta) p_b. \quad (16)$$

When the firms are able to influence the RP through their price setting, a new and counteracting incentive for the generic producer is introduced. As before, reference pricing makes the brand-

²⁰See, e.g., Danzon and Liu (1996) and Danzon and Ketcham (2004).

name drug more expensive, giving the generic producer an incentive to raise prices. However, the generic producer can make the brand-name drug even more expensive by lowering the price of the generic drug, since this automatically reduces the RP. Equilibrium prices are now given by

$$p_g^{rp} = \frac{(\theta - 1)(t - 2f) + (\alpha(2\theta + 1) + 3\beta(1 - \alpha))w}{3\beta(1 - \alpha) + \alpha(4\theta - 1)}, \quad (17)$$

$$p_b^{rp} = \frac{(\theta - 1)(\alpha(2t\theta - f) + \beta(1 - \alpha)(2t - f)) + 3(\beta(1 - \alpha) + \alpha)(\beta(1 - \alpha) + \theta\alpha)w}{(\alpha + \beta(1 - \alpha))(3\beta(1 - \alpha) + \alpha(4\theta - 1))}. \quad (18)$$

We can analyze the effects of reference pricing by considering a marginal increase in β . The equilibrium price responses of RP are given by

$$\frac{\partial p_g^{rp}}{\partial \beta} = -\frac{3(\theta - 1)(1 - \alpha)(t - 2(f + \alpha w))}{(\alpha(4\theta - 1) + 3\beta(1 - \alpha))^2} < 0, \quad (19)$$

$$\frac{\partial p_b^{rp}}{\partial \beta} = -\frac{(\theta - 1)(1 - \alpha)[2t\Omega - 3\Phi(f + \alpha w)]}{\Phi(\alpha(4\theta - 1) + 3\beta(1 - \alpha))^2} < 0, \quad (20)$$

where

$$\Omega := \alpha^2 + 3\beta^2(1 - \alpha)^2 + 2\theta\alpha^2(2\theta - 1) + 6\theta\alpha\beta(1 - \alpha),$$

$$\Phi := (\alpha + \beta(1 - \alpha))^2 < \Omega.$$

Thus, endogenizing the RP completely reverses the price response of the generic producer, implying that RP leads to price reductions for brand-name *and* generic drugs. Notably, this result holds also in the case of no coinsurance ($\alpha = 0$), as the RP is affected by generic price reductions. Since both drugs become cheaper, the effect of RP on relative prices is *a priori* uncertain. Equilibrium relative prices, defined as $\omega^{rp} := p_b^{rp}/p_g^{rp}$, are given by

$$\omega^{rp} = \frac{(\theta - 1)(\beta(1 - \alpha)(2t - f) + \alpha(2t\theta - f)) + 3(\alpha + \beta(1 - \alpha))(\beta(1 - \alpha) + \theta\alpha)w}{(\beta(1 - \alpha) + \alpha)((\theta - 1)(t - 2f) + (\alpha(2\theta + 1) + 3\beta(1 - \alpha))w)}. \quad (21)$$

It is straightforward to verify that, in our parameterized model, $\frac{\partial \omega^{rp}}{\partial \beta} < 0$, implying that the price reduction is stronger, in absolute terms, for the brand-name drug.

What is the effect of RP on the competition between brand-names and generics, measured by the generic market share? The above analysis suggests that there are two counteracting forces:

(i) For given relative drug prices, RP generally leads to an increase in the relative copayment rate, which is given by

$$\mu(p_b, p_g) := \frac{c_b(p_b, p_g)}{c_g(p_b, p_g)} = \frac{f + \alpha p_b + \beta(p_b - p_g)(1 - \alpha)}{f + \alpha p_g}. \quad (22)$$

The effect of RP is then given by

$$\frac{\partial \mu(p_b, p_g)}{\partial \beta} = \frac{(p_b - p_g)(1 - \alpha)}{f + \alpha p_g} > 0. \quad (23)$$

The strength of this effect is decreasing in both f and α . Indeed, in the absence of insurance, i.e., $\alpha \rightarrow 1$, there is obviously no effect of RP on relative copayments. Generally, though, as long as $\alpha < 1$, RP induces a shift in consumption – for given drug prices – from brand-name to generic drugs.

(ii) The positive relationship between RP and relative copayments might be, at least partly, compensated for by a reduction in relative drug prices, i.e., $\frac{\partial \omega^{rp}}{\partial \beta} < 0$, as shown above. All else equal, this effects leads to a shift of consumption from generic to brand-name drugs. The overall effect on market shares is thus a priori ambiguous.

Combining the two above mentioned effects, the overall impact of RP on competition between brand-names and their generic counterparts is

$$\frac{\partial \gamma_g^{rp}}{\partial \beta} = \frac{(f + \alpha w) \alpha (\theta - 1) (1 - \alpha) (t - 2(f + \alpha w))}{(3\alpha(t\theta - \beta(1 - \alpha)w) - \alpha(2\theta + 1)(f + \alpha w) + 3\beta(1 - \alpha)(t - f))^2} > 0. \quad (24)$$

Thus, the increase in the relative copayment rate is not outweighed by the drop in relative drug prices, implying that RP leads to an increase in the generic market share. It is also possible to confirm that the positive effect of RP on generic market shares is weaker the higher the degree of coinsurance, i.e., $\partial^2 \gamma_g^{rp} / \partial \alpha \partial \beta < 0$. This is quite intuitive, since reference pricing has a smaller impact on relative copayments for higher levels of α . In the extreme case of $\alpha = 1$, where patients pay the full drug price out-of-pocket, a reference pricing system is *de facto* irrelevant.

Proposition 3 *Assume that the RP is endogenously determined as a function of the drug prices in the market. A higher weight attached to the low-priced generic drug, implying all else equal a reduction in the RP, will then lead to (i) a reduction in both brand-name and generic drug*

prices and (ii) an increase in the market share of generic drugs.

2.4 Discussion and theoretical predictions

In our theoretical analysis, we have made the important distinction between *exogenous* and *endogenous* RP. In the Norwegian experiment, the RP was defined as a sales-weighted average of the drug prices within each therapeutical class and updated every 3 months. This suggests that *endogenous* RP – which predicts lower prices for both brand-names and generics in response to RP – is the most appropriate choice of model. Furthermore, notice that endogenizing the weights (β in our model) by market shares only reinforces our previously derived effects. To see this, observe that a reduction in p_g reduces r for a given value of β . All else equal, this shifts demand towards the generic drug. If β is endogenized by the generic market share, this will then lead to a further reduction in r , reinforcing the generic firm’s incentive to reduce prices as a response to RP.

When assessing the effect of RP on competition between brand-names and generics, we have, by considering marginal changes in β , implicitly compared the outcome with the free pricing equilibrium, since this equilibrium coincides with the RP equilibrium in the limit $\beta \rightarrow 0$. However, notice that, since a binding price cap reduces generic competition (compared with free pricing), the positive effect of RP on generic market shares would be even larger if we compared with a price cap equilibrium. The drug pricing responses of replacing price cap regulation with RP are less clear, and depends on the strictness of price cap regulation. If the price cap is sufficiently low, we cannot rule out the possibility that replacing this regulatory system with RP will increase drug prices. However, the fact that we observe generic competition in markets with price cap regulation suggests that, in reality, the price cap is generally set well above marginal production costs. Furthermore, the descriptive data from the policy experiment we exploit in the subsequent empirical analysis does not suggest that this is a relevant case.

In our theoretical model, we have also made the simplifying assumption that there is only one generic competitor to the brand-name drug. How is the presence of more than one generic competitor likely to affect the results derived from the basic model? If generic drugs are perfect substitutes in demand, it only takes two generic drug producers to induce marginal cost pricing for generics. Since drug prices are strategic complements (cf. (8) and (9)), it is straightforward

to show that this would reduce the equilibrium price of the brand-name drug in our model. Thus, more generic competitors should intuitively lead to lower drug prices. Of course, marginal cost pricing of generics with two or more generic competitors is a somewhat extreme case. In reality, there are likely to be demand frictions that will lead to drug prices in excess of marginal production costs also for generics, even if a higher number of generic competitors has a dampening effect on drug prices.²¹

One limitation of our model is that it does not take into account the slow diffusion of generics. Ching (2010a, 2010b) offers a model where consumers learn the generic product quality and differ in their price sensitivity, showing that generics would enter the market slowly, gradually capturing price sensitive consumers. Consequently, brand-names respond by increasing their prices as more generics enter the market. He also shows that such a model is consistent with data using a structural estimation approach. Also in our model there is an incentive for the brand-name firm to increase its price when generic firms capture the price sensitive consumers. However, the strategic effect of competition from generics always dominates the consumer selection effect (i.e., the marginal consumer becomes less price sensitive).

Moreover, we would like to mention that the market size of a given molecule would affect the number of generic entrants and thus the degree of price competition (at least among the generic firms). As shown by Saha et al. (2006), brand-name drugs with a large market size prior to generic entry (blockbuster drugs) experience significantly more generic entrants, price erosion and generic penetration than other drugs. However, our focus is on the effect of RP rather than determinants of generic entry and competition. As our theoretical model demonstrates, the effects of RP on drug prices and market shares go mainly through the change in relative copayment rates between brand-name and generics and should therefore not depend qualitatively on the number of generics. In any case, we will account for generic competition in the empirical section.

Based on the above theoretical analysis and discussion, we postulate the following hypotheses for the empirical analysis: Switching from price cap regulation to reference pricing leads to

(i) a reduction in brand-name and generic prices (given that price cap regulation is not exces-

²¹ Such frictions could be due to imperfect information, exclusive dealing of generics or imperfect agency between prescribing physicians and patients.

sively strict),

(ii) and an increase (decrease) in generic (brand-name) market shares.

3 Institutional background

The Norwegian pharmaceutical market is, as most other Western pharmaceutical markets, extensively regulated. The regulatory body is the Norwegian Ministry of Health and Care Services and its agency called the Norwegian Medicines Agency. Norway has adopted the European patent law system to a large extent, implying that all new chemical entities are subject to patent protection for a given period. To launch their products on the Norwegian market, pharmaceutical firms need a government approval. The approval is based on (clinical) evidence showing that the drug is not dangerous and has a positive health effect. To get the drug listed for reimbursement ("blue list"), the pharmaceutical firms must in addition provide evidence of a positive cost-benefit analysis.

All prescription drugs (reimbursable or not) are subject to price control. The current system introduced in 2000 is a *price cap* scheme based on international reference pricing, also called external referencing. This scheme has become widely used across countries.²² This system covers all prescription drugs, both on-patent and off-patent, except for those included in the RP system. Under price cap regulation, producers have to report *foreign* prices in a defined set of "comparable" countries.²³ The price cap, which is the maximum *domestic* price a producer can charge for its product, is then set equal to the average of the three lowest reported foreign prices of this drug. Generic versions get the same price cap as the brand-names, but the price cap rarely binds as they are typically priced lower than the brand-name. The price cap is imposed at the wholesale level. The government then defines the maximum mark-up the pharmacies can charge, which in turn determines the price cap at the retail level for each product.

The RP system, called "index pricing", was introduced in March 2003 for a subsample of off-patent pharmaceuticals facing generic competition. Initially, the index price system covered

²²Price cap regulation based on international price comparisons is widely used in Europe, see Kanavos (2001), but also outside Europe (e.g., Brazil, Canada, Japan, Korea, Taiwan).

²³The Norwegian basket of "comparable" countries consists of Austria, Belgium, Danmark, Finland, Germany, Irland, the Netherlands, Sweden and the UK. Southern and Eastern European countries, as well as France and Switzerland, are excluded. If the product is not yet launched in any of the countries in the basket, the price cap will be determined by negotiations between the producer and the regulator.

six chemical substances: Citalopram (depression), Omeprazol (antiulcer), Cetirizin (allergy), Loratadin (allergy), Enalapril (high blood pressure) and Lisinopril (high blood pressure). The system was later extended with two additional substances; Simvastatin (high cholesterol) and Amlodipin (high blood pressure). The choice of drugs were based on two criteria: first, they should cover a wide set of diseases and not be concentrated within one particular disease type; second, the selected drugs should be high-volume drugs.²⁴

In calculating the index (reference) price, the regulator first clustered drugs with the same chemical substance. Within each substance group, drugs were classified into subgroups depending on package size and dosage in order to adjust for cost variation. The regulator then calculated the index price, defined as the sales weighted average of brand-name and generic prices, for each subgroup. For the six chemical substances initially included, there were 16 index prices in total. The regulator repeated this exercise every three months, resulting in a revised index price for every quarter of a year. Thus, if generics increase their market share and/or there is a reduction in brand-name or generic prices, this would induce a lower index price for the next period. In other words, the index price system can be classified as an *endogenous* RP system, as explained in Section 2.²⁵

The index price system also provided incentives for generic substitution at pharmacy level. The pharmacies obtained the positive margin of selling a (generic) drug priced lower than the RP. However, they also faced the negative margin of selling a (brand-name) drug priced higher than the RP. Thus, we expect that the pharmacies would always suggest a generic substitute to patients, except for the case when the physician had made a reservation against generic substitution on the prescription.²⁶

Patients in Norway are required to pay coinsurance for all reimbursable prescription drugs. The coinsurance rate is 36 percent of the price of the drug. However, for the drugs included in

²⁴The first criterion is helpful for identification purposes since it provides us with a proper control group. The second criterion could potentially be a problem if the selected drugs differ from the non-selected drugs. In Section 6, we therefore perform a pre-reform test, showing no significant differences in prices and market shares for the treatment (reference priced drugs) and the control (price capped drugs) group.

²⁵The initial reference price was based on drug prices that were set before the introduction of the RP system was announced. Thus, it was in principle not possible for the drug producers to game the regulator by increasing prices in the period before RP was introduced (see Miraldo, 2009).

²⁶The RP reform in Norway did not include incentives on the physician-side. Physicians were as usual encouraged to prescribe cheaper generics when possible, but there were no financial incentives like in, for instance, Germany (physician budgets).

the RP system, patients had, in addition, to pay the full price difference between the RP and the high-priced brand-name, if they refused generic substitution. There are patient expenditure caps, which for the period of our study were 400 NOK per script and 1,350 NOK per year. However, these caps did not apply to the extra copayment under reference pricing if the patient refused generic substitution. Thus, consumers had to fully cover the price difference between the brand-name and the RP even if they were already at the expenditure cap level.

The RP system in Norway was modified in 2005. Under the new regime (called "Trinnpris"), the RP is set as a discount on the brand-name price and comes into effect once generics enter after patent expiration. The argument for this regime is that it involves less administrative costs than the previous one, where the RP is set as the average price of brand-name and generics and calculated every quarter.

4 Data and descriptive statistics

In the empirical analysis we use data from Farmastat.²⁷ Their database includes information on sales value and volume for each package of drugs sold at the Norwegian pharmaceutical market. Values are in pharmacy purchase prices and volumes in defined daily doses (DDD) for the active substance according to the ATC-code system.²⁸ The database also provides detailed information about product name, manufacturer, launch date, package size, dosage, etc.

From this database we have information on all off-patent prescription drugs within the 40 largest ATC groups (in terms of sales volume) over a four year period from 1st of January 2001 to 31st of December 2004. All drugs in our sample are on the government's reimbursement list.

Our empirical strategy relies on a comparison of drugs subject to RP with drugs under price cap regulation. Since most of the brand-name drugs in the index price system faced competition from generics for a relatively short period before they came subject to the reform, we only include molecules with generic entry after 1st of January 1998 in our sample. This leaves us with 24 ATC groups. Table 1 lists the main characteristics of these molecules:

²⁷Farmastat is a company specialised in provision of pharmaceutical statistics. The company is owned by the Norwegian Association of Pharmaceutical Manufacturers.

²⁸The ATC-code system is used by the World Health Organization to classify pharmaceutical substances according to their chemical, pharmacological and therapeutic properties. Pharmaceuticals sharing the same seven-digit (five-level) ATC-code have the same ingredients and are considered equivalent in the treatment of a given disease.

Table 1. Sample characteristics, means and standard deviances in parentheses

| ATC-code | Average brand-name prices | Average generic prices | Brand-name market shares | Reference pricing | Number of generics | Relative prices | Number of therapeutic competitors | Number of obs. |
|----------|---------------------------|------------------------|--------------------------|-------------------|--------------------|-----------------|-----------------------------------|----------------|
| A02BC01 | 10.71 (2.54) | 8.89 (1.81) | 75.58 (15.51) | Yes | 1.00 (0) | 1.28 (0.09) | 8.21 (0.41) | 38 |
| A10BA02 | 1.51 (0.18) | 1.32 (0.14) | 81.84 (4.72) | No | 3.56 (0.98) | 1.23 (0.13) | 8.06 (0.38) | 48 |
| A10BB01 | 1.88 (0.41) | 1.57 (0.22) | 95.27 (7.81) | No | 1.00 (0) | 1.26 (0.11) | 8.05 (0.37) | 43 |
| C08CA01 | 3.14 (0.62) | 2.62 (0.57) | 50.39 (15.14) | Yes | 3.80 (0.77) | 1.35 (0.15) | 1.00 (0) | 10 |
| C09AA02 | 2.34 (0.68) | 2.05 (0.53) | 73.18 (23.01) | Yes | 4.52 (0.85) | 1.54 (0.16) | 2.19 (1.24) | 48 |
| C09AA03 | 3.42 (0.77) | 2.56 (0.45) | 72.42 (22.18) | Yes | 3.19 (1.32) | 1.54 (0.14) | 2.19 (1.24) | 48 |
| C09BA02 | 4.45 (0.47) | 3.44 (0.39) | 59.58 (11.72) | No | 1.71 (0.46) | 1.32 (0.05) | 1.00 (0) | 24 |
| C10AA01 | 5.52 (1.90) | 4.67 (1.77) | 58.63 (17.66) | Yes | 3.81 (0.74) | 1.28 (0.12) | 3.90 (0.29) | 21 |
| C10AA02 | 9.21 (0.41) | 7.31 (0.83) | 61.23 (20.18) | No | 1.00 (0) | 1.36 (0.15) | 4.00 (0) | 17 |
| H02AB02 | 2.51 (0.02) | 3.00 (1.45) | 30.25 (5.78) | No | 1.92 (0.28) | 0.98 (0.09) | 7.25 (0) | 12 |
| J01FA01 | 13.66 (0.96) | 7.78 (0.40) | 75.74 (4.19) | No | 1.00 (0) | 1.76 (0.10) | 0 (0) | 48 |
| J01MA02 | 26.42 (3.86) | 23.55 (3.21) | 95.93 (8.28) | No | 1.31 (0.47) | 1.18 (0.12) | 0 (0) | 26 |
| M01AB05 | 3.49 (1.92) | 2.52 (0.12) | 86.25 (4.90) | No | 2.75 (0.44) | 1.24 (0.75) | 11.33 (1.43) | 48 |
| N03AF02 | 10.61 (0.20) | 5.48 (1.48) | 98.74 (1.34) | No | 1.00 (0) | 2.06 (0.49) | 13.67 (0.48) | 12 |
| N05AH02 | 15.51 (0.67) | 13.35 (0.52) | 73.86 (14.04) | No | 1.00 (0) | 1.31 (0.03) | 3.75 (1.31) | 48 |
| N05BA12 | 1.63 (0.01) | 1.15 (0.07) | 97.67 (1.01) | No | 1.00 (0) | 1.59 (0.10) | 3.00 (0) | 17 |
| N05BE01 | 14.76 (0.86) | 11.79 (0.93) | 59.56 (15.82) | No | 2.33 (0.47) | 1.37 (0.05) | 3.75 (1.31) | 48 |
| N05CF02 | 2.76 (0.13) | 2.11 (0.19) | 71.99 (13.02) | No | 1.53 (0.50) | 1.87 (0.21) | 3.00 (0) | 32 |
| N06AB03 | 6.10 (0.90) | 4.59 (0.40) | 77.69 (21.37) | No | 1.00 (0) | 1.30 (0.16) | 14.39 (0.49) | 36 |
| N06AB04 | 6.42 (0.87) | 5.46 (0.44) | 67.45 (29.89) | Yes | 3.38 (1.18) | 1.23 (0.16) | 14.44 (0.50) | 32 |
| N06AB05 | 6.94 (0.38) | 5.93 (0.54) | 69.86 (27.51) | No | 2.00 (1.07) | 1.18 (0.07) | 14.39 (0.49) | 18 |
| N06AG02 | 7.35 (0.09) | 5.45 (0.29) | 73.77 (13.12) | No | 1.22 (0.42) | 1.41 (0.04) | 14.48 (0.50) | 27 |
| R06AE07 | 2.24 (0.47) | 1.90 (0.24) | 53.07 (20.37) | Yes | 4.38 (1.21) | 1.19 (0.12) | 9.85 (0.78) | 34 |
| R06AX13 | 2.57 (0.30) | 2.44 (0.33) | 76.48 (19.04) | Yes | 3.17 (1.91) | 1.13 (0.08) | 11.44 (2.87) | 48 |

The table first provides information about our dependent variables, i.e., average prices of brand-names and generics and brand-name market shares. The average brand-name and generic prices are in NOK per DDD. Brand-name market share is the proportion of sales of brand-names compared to sales of generics within each ATC group. All prices are deflated using the consumer price index.

The table also provides information about our main explanatory variables. First, we list the average number of generic competitors within each of the 24 substances. From our theoretical discussion, we expect this to have a negative impact on brand-name and generic prices, as well as on brand-name market shares. Second, we use a variable capturing the degree of therapeutic competition, which is measured by the number of ATC groups having the same three first digits in their ATC code. We expect that more therapeutic competitors also contributes to lower

brand-name and/or generic prices. This is in line with Ellison et al. (1998) and Brekke et al. (2009) who report negative cross-price elasticities for therapeutic substitutes. Brekke et al. (2007) provide a theoretical foundation.

In explaining market shares, we also control for relative brand-name and generic prices. This enables us to decompose the direct demand effect of RP due to the changes in copayment structure from the indirect demand effect due to price responses by the firms. The "Relative price" variable is calculated as brand-name prices divided by the quantity weighted average of generic drug prices for each substance. In the analysis we divide time into one month periods. Substances where brand-names face competition from generics over the total sample period are therefore represented with 48 observations in the data set. Finally, there is a column indicating whether or not the substance is exposed to RP.²⁹

The main objective of the empirical analysis is to test the hypotheses derived in Section 2. Our first hypothesis postulates that (given that the price cap is not excessively strict) a switch from PC regulation to (endogenous) RP leads to a price reduction for both brand-names and generics. The descriptive statistics in Table 2 support our first hypothesis. Prices of brand-names subject to RP are reduced by almost 23 percent after the reform, while the reduction is only 8.5 percent for the brand-names under price cap regulation over the same period. Moreover, generics subject to the reform face a price reduction of almost 13 percent, while there is no price change for generics under price cap regulation for the whole period.

Table 2. Market shares and average prices before and during the reference pricing period.

| | Drugs subject to Reference pricing | | Drugs not subject to Reference pricing | |
|------------------------------|---|---|---|---|
| | Before the reference pricing period | During the reference pricing period | Before the reference pricing period | During the reference pricing period |
| Brand-name market shares | 90.50 (7.02) | 50.97 (13.96) | 83.06 (16.08) | 71.57 (18.58) |
| Average brand-name prices | 5.07 (3.89) | 3.91 (2.78) | 8.64 (6.75) | 7.91 (6.58) |
| Average generic prices | 3.92 (2.81) | 3.42 (2.31) | 6.39 (5.43) | 6.40 (5.75) |

Our second hypothesis postulates that a switch from price cap regulation to RP should reduce (increase) brand-name (generic) market shares. In Table 2 we compare (average) brand-name

²⁹Notice that the ATC groups C10AA01 (Simvastatin) and C08CA01 (Amlodipin) were included in the reference price system in June and September 2004, respectively, while the rest of the ATC groups subject to reference pricing were included when the reform was initiated in March 2003.

market shares before and after the RP reform with (average) market shares for brand-names under price cap regulation over the same period. From the table we see that while there has been a decrease in brand-name market shares for both groups, the decrease is substantially larger for the drugs subject to RP.

To study the effect of RP in more detail, we investigate how prices and market shares development over time. Figure 1 covers the drugs that become subject to RP during our period, while Figure 2 covers the drugs in the control group.³⁰

Figure 1. Average prices and market shares of drugs subject to reference pricing. All substances.

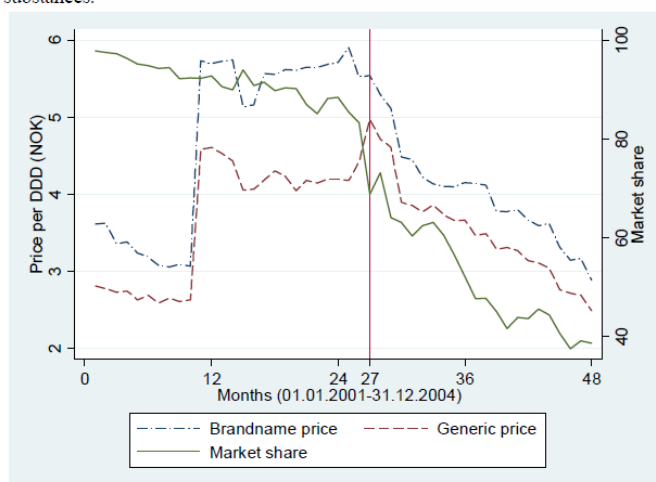
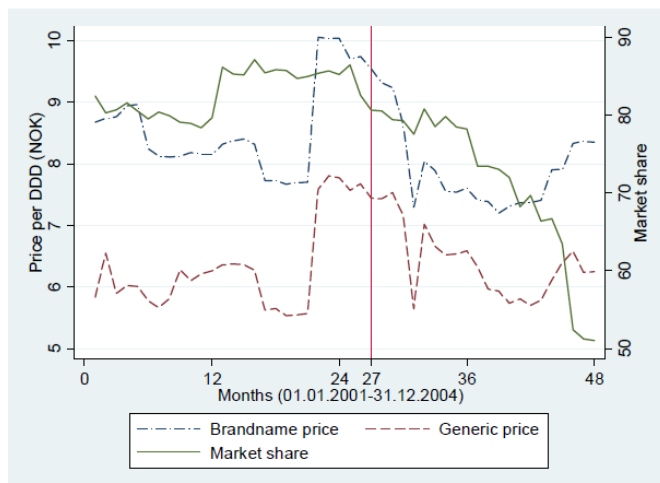


Figure 2. Average prices and market shares of drugs not subject to reference pricing. All substances.



³⁰The large shifts in the price and market share graphs are due to new products entering (or old products exiting) the market. Figure A1 and A2 in the Appendix show the same variables, but only for drugs that are present in all periods.

From the figures we see that the development of brand-name and generic average prices and market shares are fairly similar prior to the policy change. However, when RP is introduced (given by the vertical line) we see a profound change in the price pattern and the brand-name market shares for the drugs subject to RP (Figure 1), whereas the residual drugs seem to be unaffected (Figure 2).

5 Empirical method and results

In this section we analyze the impact of RP on average brand-name and generic prices and brand-name market shares. Our estimating strategy relies on a comparison of the molecules affected by RP (treatment group) to similar molecules under price cap regulation (control group). Having panel data, we are able to compare inter-temporal variation in outcomes before and after the imposition of the reform. Therefore, identification relies not only on before-after comparison, but also on comparison of variations in outcomes for molecules subject to RP with variation in outcomes for molecules not subject to this reform.

In the analyses, we estimate different versions of the following fixed effect model:

$$Y_{it} = \mathbf{X}'_{it} \boldsymbol{\beta} + a_i + \delta_t + \alpha D_{it} + \varepsilon_{it}, \quad (25)$$

where Y_{it} is one of the two outcomes described above for molecule i at time t , a_i is a molecule fixed effect, δ_t is a period specific effect common to all molecules, ε_{it} represents unobserved time varying factors that affect outcomes, \mathbf{X}'_{it} contains observable variables (the number generic competitors and the number of therapeutic competitors), and D_{it} is a variable indicating whether or not molecule i is subject to RP at time t . The effect of introducing RP is captured by α and the effect of the control variables is measured by the vector $\boldsymbol{\beta}$.

5.1 Brand-name and generic prices

We start out presenting the basic results from the regression models specified in (25). First, we analyze the effect of RP on average brand-name and generic prices by estimating the following

fixed effect model:

$$\ln P_{it} = \mathbf{X}'_{it} + a_i + \delta_t + \alpha_1 D_{it} + \alpha_2 D_{it} * B_i + \varepsilon_{it}, \quad (26)$$

where $\ln P_{it}$ is the natural logarithm of average brand-name and generic prices.³¹ By including an interaction term ($D_{it} * B_i$) between the RP indicator (D_{it}) and the brand-name indicator (B_i), we can separate the effect of RP on brand-names from the price effect on generics. The results from the regression are reported in Model 1 in Table 3.

Table 3. Estimated effects of reference pricing. Fixed effect models, robust standard errors in parentheses.

| | (1) Average brand-name and generic prices | (2) Brand-name market shares |
|--|---|------------------------------------|
| Drugs subject to reference pricing | -0.2241** (0.0224) | -14.6991** (1.4556) |
| Brand names subject to reference pricing | -0.1113** (0.0238) | - |
| Number of generics | 0.0183** (0.0049) | -2.3961** (0.4794) |
| Number of therapeutic competitors | -0.0256** (0.0051) | 2.3648** (0.3612) |
| Relative price | - | -4.9173* (1.3958) |
| Constant | 1.8430** (0.0716) | 81.9536** (4.0629) |
| Period dummies | Yes | Yes |
| Molecule dummies ¹ | Yes | Yes |
| Number of observations | 1566 | 783 |
| Number of ATC groups | 24 | 24 |
| R-squared | 0.559 | 0.788 |

¹: In model 1 we include two dummies per molecule, one for brand names and one for generics.

** : significant at the 1 percent level, * : significant at the 5 percent level.

We find that RP leads to a significant reduction in both brand-name and generic average prices, with the effect being stronger for brand-names (33 percent) than for generics (22 percent). Thus, the claim by Danzon and Liu (1996) and Danzon and Ketcham (2004) that RP results in higher generic prices is not supported. The finding is, however, in line with our theoretical prediction (Hypothesis 1), as well as previous empirical findings by Pavcnik (2002) and Brekke et al. (2009).

From Model 1 we also see that the number of generic competitors has a *positive* effect on average prices. There are two potential explanations to this relationship. First, the positive effect could be due to endogeneity problems; i.e., high priced brand-names might attract more generic

³¹Thus, we have two price observations per molecule per time period.

entry. Notice that all drugs in our sample have by definition at least one generic competitor, which means that we measure the effect of the intensity, not the existence, of competition between brand-names and generics.³² Second, the positive effect could be due to consumer learning and heterogeneity in price sensitivity, as pointed out by Ching (2010a, 2010b). He shows that generics slowly enter the market and gradually capture price sensitive consumers, which induces brand-names to raise their prices.

When estimating the effect of the reform, we pool the average brand-name and generic prices. If these prices behave very differently – as suggested by empirical studies from the US (Grabowski and Vernon, 1992, Frank and Salkever, 1997, Ching, 2010a) – our specification might be too restrictive. However, as shown in Figure 1, the average brand-name prices seem to follow the same pattern as the average generic prices. To check this more thoroughly, we estimate a SUR (Seemingly Unrelated Regression) model, where we run two separate regressions; one for the average brand-name prices and one for the average generic prices. The results, which are reported in Table A1 in the Appendix, show that the effect of the reform on average prices are almost identical to the ones reported in Table 3.

Another potential problem is that the drugs subject to RP are typically high volume drugs with large market size, which might attract more generic competition than lower volume drugs. For instance, Saha et al. (2006) show that blockbusters experience more generic entrants, price reductions and generic penetration than other drugs. Since generic firms enter the market fairly slowly, we might not capture the effect of the reform, but rather the effect of market size and the following generic competition. However, the product fixed effects in our regressions account for market size (prior to generic entry), since this is a time-constant (unobserved) product characteristics. Moreover, we also control for the number of generics, which accounts for the fact that high-volume brand-names (blockbusters) face stronger competition from generics than lower volume drugs.

However, one might argue that the price effects we estimate for the high volume drugs subject to RP would be lower for lower volume drugs because of less generic competition. Clearly, for some lower volume drugs, generic firms would not find it profitable to enter, which means that

³²Bergman and Rudholm (2003) consider a sample of off-patent molecules with and without generic competition, and find that the existence rather than the intensity of generic competition has an effect on brand-name prices.

RP would have no effect. For instance, Bergman and Rudholm (2003) show that RP had effects on brand-name prices only for substances facing actual (not potential) generic competition. On the other hand, our estimates on the effects of RP are obtained by controlling for the number of generics, as well as unobserved (time-constant) product characteristics, like market size. In section 6, we will account for endogeneity issues in various ways.

5.2 Competition between brand-names and generics

The effect of RP on the competition between brand-names and their generic counterparts is not *a priori* evident, as pointed out in Section 2. RP changes the copayment structure, making the brand-name drug relatively more expensive than the generics, which increases the generics' market share (for given prices). However, the brand-names respond to RP by lowering their prices in order to retain their market shares. The net effect on market shares is thus determined by the relative strength of these two counteracting effects.

The dependent variable in the analysis is the brand-names' market shares (as a percentage). This measure of competition between brand-names and generics has been used in previous work, for instance, Aronsson et al. (2001). In the regressions we control for molecule and period specific effects, as well as the number of generic and therapeutic competitors, and relative prices. The results are presented in Model 2 in Table 3 above.

We find that the imposition of RP leads to a significant (14.7 percent) reduction in brand-name market shares. Since we control for relative prices in the regression, we can interpret this decrease as a direct demand response to RP and the corresponding change in the copayment structure. Relative prices are, however, likely to be endogenous. While relative branded-generic prices might explain market shares, market shares might also influence firms' price setting and thus relative prices. We return to this issue in Section 6.

We also find, as expected, a negative effect of the number of generics on the brand-name market shares. The number of therapeutic competitors, on the other hand, have a positive effect on brand-name market shares. This could be explained by the fact that stronger therapeutic competition might lead to a price reduction for brand-name drugs, which in turn increases their market share.

6 Robustness checks

To check the robustness of our findings in the previous section, we conduct a number of tests. First, we check the validity of our comparison group (consisting of off-patent drugs subject to price cap regulation throughout the whole period). Second, we account for potential serial correlation in the errors. Finally, we address the possibility that endogeneity might bias our results.

6.1 Pre-reform tests

An important assumption in the analyses in Section 5 is that the error term ε_{it} is uncorrelated with the reform dummy variable D_{it} (as well as with \mathbf{X}'_{it} and δ_t). This implies that, after controlling for covariates and molecule specific effects in the pre-reform period, the trends in the dependent variables for drugs subject to RP should not differ from trends for drugs subject to price cap regulation. A test of this assumption is presented in Table 4.

Table 4. Testing for pre-reform differences in price and market share trends. Fixed effect results with robust standard errors in parentheses.

| | (1) Average brand-name and generic prices | (2) Brand-name market shares |
|---|---|------------------------------------|
| Interaction period 1 | 0.06 (0.08) | 3.26 (3.32) |
| Interaction period 2 | -0.02 (0.09) | 5.05 (4.55) |
| Interaction period 3 | -0.00 (0.07) | 4.03 (3.66) |
| Interaction period 4 | -0.01 (0.08) | 2.11 (3.13) |
| Interaction period 5 | -0.06 (0.08) | 2.09 (2.64) |
| Interaction period 6 | 0.03 (0.05) | 3.12 (3.16) |
| Interaction period 7 | 0.02 (0.05) | 1.81 (2.55) |
| Interaction period 8 | 0.01 (0.05) | 2.47 (2.50) |
| Interaction period 9 | -0.03 (0.06) | 1.27 (2.40) |
| Interaction period 10 | -0.03 (0.05) | 1.61 (2.46) |
| Interaction period 11 | -0.02 (0.05) | 2.40 (3.57) |
| Interaction period 12 | -0.03 (0.05) | 1.87 (3.15) |
| Interaction period 13 | - | - |
| Interaction period 14 | -0.01 (0.04) | 0.18 (3.28) |
| Interaction period 15 | 0.02 (0.04) | 6.76 (4.25) |
| Interaction period 16 | 0.03 (0.04) | 3.74 (3.30) |
| Interaction period 17 | 0.03 (0.04) | 3.56 (2.44) |
| Interaction period 18 | 0.04 (0.04) | 1.48 (2.61) |
| Interaction period 19 | 0.07 (0.04) | 2.37 (2.84) |
| Interaction period 20 | 0.02 (0.05) | 3.41 (2.98) |
| Interaction period 21 | 0.06 (0.04) | -0.10 (2.60) |
| Interaction period 22 | 0.04 (0.04) | -0.24 (3.05) |
| Interaction period 23 | 0.06 (0.04) | 2.76 (3.02) |
| Interaction period 24 | 0.06 (0.04) | 3.48 (2.71) |
| Interaction period 25 | 0.04 (0.04) | 0.66 (2.82) |
| Interaction period 26 | 0.06 (0.04) | 1.73 (3.01) |
| Relative prices | - | -2.061* (0.997) |
| Number of generics | -0.016* (0.008) | -0.733 (0.482) |
| Number of therapeutic competitors | -0.008 (0.006) | 0.221 (0.295) |
| Molecule dummies ¹ | Yes | Yes |
| Period dummies | Yes | Yes |
| Joint insignificance of interactions (Prob>F) | 0.608 | 0.263 |
| Number of observations | 668 | 334 |
| Number of products | 20 | 20 |
| R-squared | 0.146 | 0.366 |

¹: In model 2 we include two dummies per molecule, one for brand names and one for generics.

** : significant at the 1 percent level, * : significant at the 5 percent level.

Here we only use observations prior to the RP reform. In order to compare the pre-reform trends in prices and market shares for drugs in the treatment and control group, we include interactions between the period dummies and a variable indicating treated molecules (in the post-reform period). If the interactions are insignificant, this is an indication of a legitimate control group, i.e., that unobservable factors affecting prices and market shares are uncorrelated with the probability that a given molecule is in the treatment group. As evident from Table 4, all interactions are statistically insignificant in both models. In addition, F-tests suggest that the interactions are jointly insignificant. These results indicate that average brand-name and generic prices and brand-name market shares for drugs in the two different groups are following the same general trend before the RP reform was implemented. We therefore conclude that the comparison group is legitimate.

6.2 Accounting for serial correlation

Even though our comparison group is legitimate, several recent papers have pointed out that standard errors in differences-in-differences regressions are often inconsistent (e.g., Wooldridge, 2002; Bertrand et al., 2004). In the case of positive serial correlation, standard errors may be biased downward, leading to overestimation of t-statistics and significance levels. To overcome the potential problem with serial correlation, we follow the solution proposed in Bertrand et al. (2004). Using Monte Carlo simulations, they show that collapsing the data into pre- and post-periods produces consistent standard errors even when the number of observations is small. In Table 5 we give the results on our three different outcomes when we ignore the time series information:

Table 5. Fixed effect estimates on average pre and post reform data, robust standard errors in parentheses.

| | (1) Average brand-name and generic prices | (2) Brand-name market shares |
|--|---|------------------------------------|
| Products subject to reference pricing | -0.2132* | -13.2200* |
| | (0.1067) | (4.9292) |
| Brand names subject to reference pricing | -0.1207 | - |
| | (0.1064) | |
| Number of generics | 0.0300 | -1.1372 |
| | (0.0349) | (3.7974) |
| Number of therapeutic competitors | -0.0410 | 3.6675 |
| | (0.0309) | (1.9271) |
| Relative price | - | 8.0965 |
| | | (15.5232) |
| Post reform | -0.1649** | -15.8138** (3.6191) |
| | (0.0276) | |
| Constant | 1.8429** | 51.1021 |
| | (0.2690) | (31.9733) |
| Molecule dummies ¹ | Yes | Yes |
| R-squared | 0.741 | 0.900 |
| Number of observations | 88 | 44 |
| Number of ATC groups | 24 | 24 |

¹: In model 1 we include two dummies per molecule, one for brand names and one for generics.

** : significant at the 1 percent level, * : significant at the 5 percent level.

Despite the substantial reduction in the number of observations³³, we find significant effects on average brand-name and generic prices (Model 1), as well as on brand-name market shares (Model 2). As expected, the standard errors are larger than those reported in Table 3, while the estimated effects of RP are about the same magnitude. We also see from Model 1 that brand-names do not have a significantly stronger price reduction than generics. However, the

³³The reason for why the number of observations is only 44 (and not 48) is that four of the molecules (two of them subject to reference pricing) do not have generic competition prior to the reform. Excluding these molecules from the sample do not qualitatively affect the results.

results also suggest that the strong significant effects of RP on average brand-name and generic prices, brand-name market shares and average molecule prices are not driven by biased standard errors.

6.3 Accounting for endogeneity

In our basic regression models in Section 5 the number of generics and relative prices are likely to be endogenous. For instance, high prices might attract more generics, while more generics might result in lower prices. This endogeneity problem can be the explanation for the positive effect of the number of generics on average prices.

In this section we allow for endogenous explanatory variables by a GMM-IV estimator³⁴ that is robust to, and efficient in the presence of, arbitrary serial correlation and heteroskedasticity (see Baum, Schaffer and Stillman, 2007). The long-run heteroskedasticity and autocorrelation consistent covariance matrix is generated using the Bartlett kernel function with a bandwidth of 12.³⁵ Orthogonality of the instruments is tested by Hansen’s J statistic, which is consistent in the presence of heteroskedasticity and autocorrelation (the null hypothesis is that the instruments are uncorrelated with the error term). However, instrument exogeneity is only one of the two criteria necessary for instruments to be valid. If the instruments are uncorrelated, or only weakly correlated, with the endogenous variables, then sampling distributions of the IV statistics are in general non-normal, and standard IV estimates, hypothesis tests, and confidence intervals are unreliable. Hence, tests for underidentification and weak identification are reported. The underidentification test is a Lagrange multiplier (LM) test of whether the excluded instruments are correlated with the endogenous regressors (the null hypothesis is that the equation is underidentified). The weak instrument test statistic is based on the Kleibergen-Paap rk statistic. As a “rule of thumb” this F -statistic should be at least 10 for weak identification not to be considered a problem (Staiger and Stock, 1997).

As instruments we use first to third lag of the endogenous variables. In addition we also use first to third lag of the number of generics in Sweden as instruments. It is reasonable to assume

³⁴IV models were estimated using the Stata module `xtivreg2` (Schaffer, 2007).

³⁵According to Baum, Schaffer and Stillman (2007), a common choice of bandwidth for these kernels is a value related to the periodicity of the data (4 for quarterly, 12 for monthly, etc.). Since we have monthly data, we choose a bandwidth of 12.

that the number generics in Sweden are correlated with the number of generics in Norway, but not directly correlated with average prices and brand name market shares.

The results from the GMM-IV models are reported in Table 6.³⁶ We first notice that the Sargan-Hansen test of overidentifying restrictions fail to reject the null hypothesis (i.e., the instruments are uncorrelated with the error term) for both models, suggesting that the set of instruments is appropriate. Considering the underidentification test, the null (i.e., the equation is underidentified) is rejected for all models, which implies that the models are identified. The weak identification tests suggest that the correlation between the instruments and the endogenous variables is sufficiently strong for both Model 1 and 2.

Table 6. Estimated effects of reference pricing. Two-stage least squares (2SLS), heteroskedastic and autocorrelation consistent standard errors in parenthesis.

| | (1) Average brand-name and generic prices | (2) Brand-name market shares |
|--|---|------------------------------------|
| Products subject to reference pricing | -0.1773* (0.0840) | -14.2688** (2.8095) |
| Brand names subject to reference pricing | -0.1081 (0.0737) | - |
| Number of generics | -0.0128 (0.0473) | -1.5112 (0.8417) |
| Number of therapeutic competitors | -0.0222** (0.0102) | 2.7643** (0.8741) |
| Relative price | - | -5.9459* (2.8350) |
| Molecule dummies ¹ | Yes | Yes |
| Period dummies | Yes | Yes |
| R squared | 0.555 | 0.799 |
| Overidentification test | 0.413 | 0.524 |
| Hansen J statistic (p-value) | | |
| Underidentification test: | 0.001 | 0.012 |
| Kleibergen-Paap statistic (p-value) | | |
| Weak identification test: | 11.045 | 52.480 |
| Kleibergen-Paap rk Wald F statistic | | |
| Number of observations | 1404 | 702 |
| Number of ATC groups | 24 | 24 |

¹: In model 1 we include two dummies per molecule, one for brand names and one for generics.

** : significant at the 1 percent level, * : significant at the 5 percent level.

Focusing first on the effect of RP, the results from the GMM-IV estimators are consistent with the results in Section 6.2. We find that RP significantly reduces brand-name and generic prices, as well as brand-name market shares. Moreover, we also see that RP does not have a significantly stronger impact on brand-name prices than generic prices (Model 1).

From Table 6 we further see that the number of generics now has a negative, but insignificant effect on average prices. Thus, the counterintuitive effect obtained in the static model is no

³⁶First step results are available upon request.

longer present when we control for endogeneity. We also find that the number of generics has an insignificant, but negative, effect on brand-name market shares. The effects of the number of therapeutic competitors and relative prices are consistent with the results reported in Section 5.

7 Welfare and Policy Implications

A full welfare analysis would require information on patients' health conditions, as well as firms' innovation and launching incentives, which we do not have. However, in the off-patent market it is likely that these important variables are not so crucial. Patients potentially substitute brand-names with generics, which have identical chemical compounds and thus effects and side-effects. Moreover, innovation and launching incentives are more efficiently stimulated through regulation in the patent period (or more broadly patent regulations) than in the off-patent period. The main intention with RP is to reduce pharmaceutical prices and expenditures (see, e.g., Lopez-Casasnovas and Puig-Junoy, 2000). We therefore take a closer look at how the introduction of RP affects the pharmaceutical expenditures. However, we also look at changes in patients' copayments, which is not a priori evident due to the extra surcharges under RP.

7.1 Pharmaceutical expenditures

Since demand for pharmaceuticals is quite price inelastic, the impact on pharmaceutical expenditures can be measured by looking at the change in average molecule prices, i.e., the sum of brand-name and generic prices weighted by their market shares (see, for instance, Danzon and Chao, 2000). Qualitatively, the impact on average molecule prices is evident, since we have found that RP leads to lower brand-name and generic prices, as well as lower brand-name market shares. However, for policy implications it is of interest to quantify the effect. We quantify the effect of RP by using the fixed effect model specified in (25), where the dependent variable in the regression is the logarithm of the average price at molecule level. We control for molecule and time period specific effects, as well as the number of generic and therapeutic competitors. The results are reported in Table 7 below.³⁷

³⁷These results also survive the robustness checks. The test results are available upon request.

Table 7. Estimated effects of reference pricing on average molecule prices and total volume. Fixed effect model, robust standard errors in parentheses.

| | Average molecule prices | Total volume |
|------------------------------------|-------------------------|--------------------------|
| Drugs subject to reference pricing | -0.2980** (0.0241) | 50.2993 (160.6458) |
| Number of generics | 0.0076 (0.0066) | -27.9792 (42.3960) |
| Number of therapeutic competitors | -0.0396** (0.0073) | 24.2204 (20.0223) |
| Constant | 2.1325** (0.1090) | 602.5053** (179.8637) |
| Period dummies | Yes | Yes |
| Molecule dummies | Yes | Yes |
| Number of observations | 783 | 783 |
| Number of ATC groups | 24 | 24 |
| R-squared | 0.659 | 0.159 |

** : significant at the 1 percent level, * : significant at the 5 percent level.

We see that RP lowers average molecule prices by almost 30 percent. This is a substantial price reduction, especially taking into account that Norway has a relatively strict price cap regime, as explained in Section 3. It follows from the above analysis that these cost savings are explained partly by drug price reductions and partly by an increase in generic market shares. If demand is highly price inelastic, the volume effects of the price reductions following the introduction of RP should be negligible. In other words, the overall reduction in pharmaceutical expenditures should be about 30 percent, which is a substantial cost saving.

To validate this assumption, we test whether there are any changes in the volume trends for the drugs subject to the policy experiment compared to those not subject to RP. The result of this is reported in Table 7 above, where we see that there is no significant volume effect for the molecules subject to RP. We therefore conclude that the change in average molecule prices is a good proxy for the net pharmaceutical cost savings of introduction of RP, as claimed by, e.g., Danzon and Chao (2000).

7.2 Patient expenditures

RP offers less insurance coverage due to the extra surcharge where patients must pay the full price difference between the high-priced brand-name and the RP. For given prices (and market shares), RP simply shifts costs from the payer to the patients. However, the main intention of RP is to make demand more price elastic and trigger price reductions and substitution towards generics. We therefore take a closer look at the changes in patients' copayments following the introduction of RP. Unfortunately, we do not observe the patients' actual copayments. However,

we can estimate these costs by using our price and volume data.

Under PC regulation there is coinsurance where patients pay 36 percent of the price of the drug they demand. Under RP patients pay the full price difference between the high-priced brand name and the RP, in addition to 36 percent of the RP. The RP is the volume-weighted sum of the brand-name and generic prices for each molecule, which corresponds precisely to what we define as the average molecule price.

In stipulating the changes in copayments, we use the means reported in Table 2. Using the difference-in-difference approach, we first compute the before-after copayments for the drugs included in the policy experiment. We then adjust these figures by the changes in the copayments for the drugs in our control group and end up with the net changes of the reform on the patients' copayments, which are reported in Table 8 below.

Table 8. Percentage change in copayments before and during the reference pricing period.

| | Drugs subject to Reference pricing | Drugs not subject to Reference pricing | Net change |
|---------------------------|---------------------------------------|--|---------------|
| Generic copayments | -12.76 | 0.16 | -12.92 |
| Brand-name copayments | -14.82 | -8.45 | -6.37 |
| <u>Average copayments</u> | <u>-21.83</u> | <u>-9.42</u> | <u>-12.41</u> |

We see that the price reductions for generics result in a copayment reduction of about 13 percent. More interestingly, the price reductions for brand-names more than offset the extra surcharges due to RP, resulting in almost a 15 percent reduction in brand-name copayments. However, accounting for price changes that would have occurred in any case, the net reduction in copayments is about 6 percent for the brand-names. Finally, a substantial fraction of the consumers switch from brand-names to generics when RP is introduced. Accounting for the change in market shares, we find a reduction of about 12 percent in the average copayment.

These figures should be interpreted with some caution for two reasons. First, as mentioned above, we do not observe the actual copayments by the patients but rely on computations based on our price and volume data. Second, there are expenditure caps that apply to the coinsurance part of the copayments, but not the RP part. These caps weaken our results concerning the effect on patient copayments.

8 Concluding Remarks

From a simple theoretical model of competition between brand-names and their generic counterparts we have shown that *endogenous RP* reduces brand-name and generic drug prices and increases generic market shares. We have empirically confirmed these predictions by exploiting a unique policy experiment introduced in Norway in 2003, where a subsample of off-patent molecules were exposed to RP, while the residual off-molecules were still under price cap regulation. Using a detailed panel data set covering the 24 most selling off-patent molecules for the four-year period 2001-2004, we had variation over time (before and after the reform) and across products (subject to RP or price cap regulation). The magnitudes of the effects are quite striking, with the combination of price reductions (for both brand-names and generics) and increased generic market shares leading to a drop in average molecule prices of close to 30 percent. We find no significant volume effects of RP, which suggest reductions in pharmaceutical expenditures of about the same amount. Finally, the strength of the price and market share effects seem to offset the extra surcharges due to RP, resulting in lower patient copayments.

By way of conclusion, we would like to identify some aspects of pharmaceutical markets that should be taken into account when assessing our results and their implications. First, there might be unintended cross-price effects of RP to non-referenced, therapeutic substitutes, as shown – theoretically and empirically – by Brekke et al. (2007, 2009). If the therapeutic substitute is an on-patent product, RP might negatively affect the patent rent by indirectly inducing lower prices on the on-patent products.³⁸ However, any negative cross-price effects would be of less concern if the therapeutic substitutes are also off-patent, since the innovation incentives are more efficiently taken care of during the patent period rather than after patent expiration, as discussed in the Introduction.

Second, RP might also induce unintended trade-offs between patient health gains and copayments (see, e.g., Lopez-Casasnovas and Puig-Junoy, 2000). If patients trade off health gains of drug therapy against copayments, radical changes in copayments induced by RP might lead some patients to choose a less suitable and/or lower quality drug. This problem is perceived to be more severe under therapeutic than generic RP. However, Brekke et al. (2007) show, in a

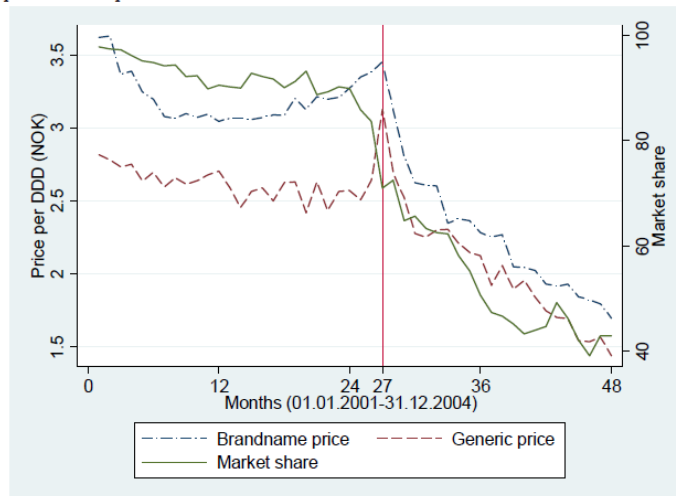
³⁸In Germany on-patent drugs were excluded from the RP system in order to limit potential negative effects on patent rent due to intensified competition caused by RP (see e.g., Danzon and Ketcham, 2004).

theoretical analysis, that this is not necessarily correct.

Finally, we should stress that effects of regulatory regimes, like PC regulation and RP, on innovation incentives and health outcomes are two very important issues that deserve to be examined much more carefully.³⁹ To obtain long-run welfare implications of regulatory regimes, it is not sufficient only to consider the price and demand (market share) effects, but also analyze the impact of the regimes on entry (and exit) of (branded and generic) drugs.⁴⁰ In the current paper, we ignore effects on drug launching and R&D, implying that we cannot make a strong recommendation about the desirability of RP versus price cap regulation in a broader sense. These issues are clearly beyond the scope of the present study, so we leave them for future research.

Appendix

Figure A1. Average prices and market shares of drugs subject to reference pricing. Substances present in all periods.



³⁹There is a recent theoretical paper by Bardey et al. (2010) on the impact of (therapeutic) reference pricing on pharmaceutical innovation.

⁴⁰Kyle (2007) analyzes strategic non-price responses by pharmaceutical firms to regulation regimes, and finds that drug launches are delayed in countries with strict price controls.

Figure A2. Average prices and market shares of drugs not subject to reference pricing. Substances present in all periods.

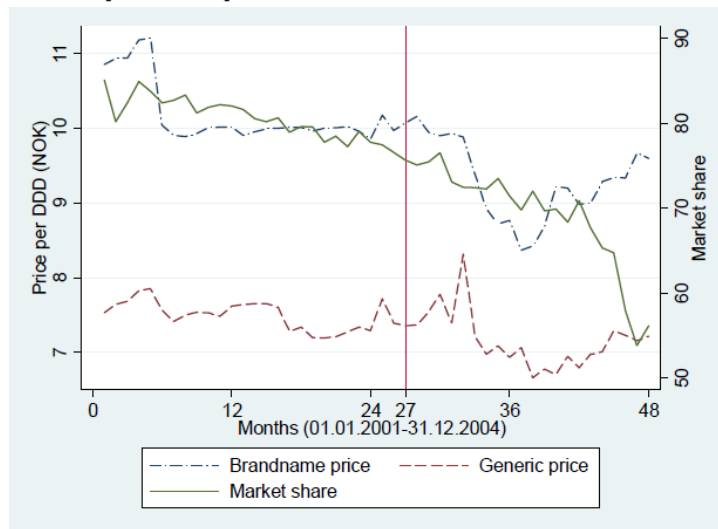


Table A1. Estimated effects of reference pricing. SUR model, standard errors in parentheses.

| | (1) Average brand-name prices | (2) Average generic prices |
|------------------------------------|-------------------------------------|----------------------------------|
| Drugs subject to reference pricing | -0.3197** (0.0229) | -0.2400** (0.0204) |
| Number of generics | 0.0107 (0.0078) | 0.0257** (0.0069) |
| Number of therapeutic competitors | -0.0448** (0.0055) | -0.0064 (0.0049) |
| Relative price | - | - |
| Constant | 3.2435** (0.0716) | 2.4228** (0.0636) |
| Period dummies | Yes | Yes |
| Molecule dummies | Yes | Yes |
| Number of observations | 783 | 783 |
| Number of ATC groups | 24 | 24 |
| R-squared | 0.979 | 0.982 |

** : significant at the 1 percent level, * : significant at the 5 percent level.

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