

A Service of



Leibniz-Informationszentrum Wirtschaft Leibniz Information Centre

Ishida, Junichiro; Takahara, Tsuyoshi

Working Paper

Should product-specific advertisement be regulated in pharmaceutical markets?

ISER Discussion Paper, No. 1182

Provided in Cooperation with:

The Institute of Social and Economic Research (ISER), Osaka University

Suggested Citation: Ishida, Junichiro; Takahara, Tsuyoshi (2022): Should product-specific advertisement be regulated in pharmaceutical markets?, ISER Discussion Paper, No. 1182, Osaka University, Institute of Social and Economic Research (ISER), Osaka

This Version is available at: https://hdl.handle.net/10419/267794

Standard-Nutzungsbedingungen:

Die Dokumente auf EconStor dürfen zu eigenen wissenschaftlichen Zwecken und zum Privatgebrauch gespeichert und kopiert werden.

Sie dürfen die Dokumente nicht für öffentliche oder kommerzielle Zwecke vervielfältigen, öffentlich ausstellen, öffentlich zugänglich machen, vertreiben oder anderweitig nutzen.

Sofern die Verfasser die Dokumente unter Open-Content-Lizenzen (insbesondere CC-Lizenzen) zur Verfügung gestellt haben sollten, gelten abweichend von diesen Nutzungsbedingungen die in der dort genannten Lizenz gewährten Nutzungsrechte.

Terms of use:

Documents in EconStor may be saved and copied for your personal and scholarly purposes.

You are not to copy documents for public or commercial purposes, to exhibit the documents publicly, to make them publicly available on the internet, or to distribute or otherwise use the documents in public.

If the documents have been made available under an Open Content Licence (especially Creative Commons Licences), you may exercise further usage rights as specified in the indicated licence.



SHOULD PRODUCT-SPECIFIC ADVERTISEMENT BE REGULATED IN PHARMACEUTICAL MARKETS?

Junichiro Ishida Tsuyoshi Takahara

July 2022

The Institute of Social and Economic Research
Osaka University
6-1 Mihogaoka, Ibaraki, Osaka 567-0047, Japan

Should Product-Specific Advertisement be Regulated in Pharmaceutical Markets?*

Junichiro Ishida[†] Tsuyoshi Takahara[‡]

July 27, 2022

Abstract

This paper examines the optimal content regulation of DTCA by comparing two forms of DTCA—product-specific and category-specific—and identifies a key tradeoff which underlies this policy debate. Our analysis suggests that the optimal form of DTCA depends crucially on the cost effectiveness of DTCA and the market-size distortion induced by DTCA. When the cost of advertisement is high, there often exists a Pareto-improving policy choice: category-specific DTCA is preferred when the market-size distortion is more severe while produce DTCA is preferred when it is less so. As the cost decreases, however, a conflict emerges between pharmaceutical firms and patients: firms are worse off under product-specific DTCA while patients are better off. We also find that the physician's reluctance to persuade misinformed patients can actually alleviate the market-size distortion and hence be welfare-enhancing.

JEL Classification Code: M37, I11, I18

Keywords: category-specific DTCA, product-specific DTCA, advertisement regulation, prescription decision, pharmaceutical market

^{*}The first author acknowledges financial support from JSPS KAKENHI (18H03640, 19H01468, 20H05631, 21H04398), and the Joint Usage/Research Center for Behavioral Economics at ISER and Osaka University for its International Joint Research Promotion Program.

[†]Institute of Social and Economic Research, Osaka University. E-Mail: jishida@iser.osaka-u.ac.jp

^{*}School of Economics, University of Toyama. E-Mail: takahara@a0.rusk.to

1. Introduction

Marketing activities, such as TV advertisement, play a major role in the pharmaceutical industry. According to Sufrin and Ross (2008), Pfizer, Merck, and Bristol-Meyer-Squibb—the three biggest pharmaceutical firms in the US—spend a greater share of expenditure on marketing than on R&D activities in 2005. Although a large fraction of advertisement is directed at physicians (prescribers), pharmaceutical firms are increasingly more involved in direct-to-consumer advertisement (DTCA), especially after the deregulation of DTCA of prescription drugs by the US Food and Drug Administration (FDA) in 1997. Owing to some unique features of the industry, however, many experts and practitioners suspect the effectiveness of DTCA in pharmaceutical markets by noting that it may distort prescription decisions in some ways. There is now a heated debate about whether DTCA should be regulated, and if so, in what ways (Ventola, 2011; Parekh and Shrank, 2018; Jacob, 2018).

A body of evidence suggests that DTCA in the pharmaceutical industry can in fact be a double-edged sword. As proponents of DTCA insist, it could serve an educational purpose by informing patients of particular medical conditions and potential treatments, thereby encouraging them to contact a health care provider at an opportune time. Opponents of DTCA argue, however, that it often misinforms patients, by selectively omitting risk factors or causes of the condition and overemphasizing drug benefits (see Section 2.6 for more discussion on this). Among those, Ventola (2011) notes:

If a patient's request for an advertised drug is clinically inappropriate and the health care provider is unable or unwilling to correct the patient's perception that it is a good choice, this situation may lead to unnecessary or harmful prescribing. An additional problem mentioned by critics is that patients may withhold information to fit a particular profile that they saw in DTC ads in an attempt to get the doctor to prescribe a drug they want but that might not be appropriate for them.

To sum up, giving information to patients is beneficial to the extent that it brings them to clinics in search of medical treatment, but anything above that could just disturb the physician-patient relationship and cause unnecessary complications.

¹The most notable example may be the case of Rofecoxib which is a nonsteroidal anti-inflammatory drug marketed by Merck. While approved in 1999, it was later withdrawn over safety concerns after disclosures that Merck withheld information about the drug's risks from physicians and patients for over five years.

Although the concern expressed by Ventola (2011) and others seems to reflect the general perception of the medical community and policymakers, it is not necessarily straightforward to understand this assertion in light of economic reasoning. Even if a patient with some symptoms is misinformed about a particular product, it is always the physician who can make the final prescription decision. As long as the physician can correct and educate any misinformed patient, the outcome should be the same regardless of the patient's prior knowledge: the physician diagnoses and prescribes the best available alternative no matter what the patient knows beforehand. To the extent that this argument holds true, therefore, the problem simply disappears and the current debate loses its ground. It is presumably for this reason that this issue has attracted only sparse attention from economic theorists, despite the heated debate among practitioners and policymakers.

A close look at reality reveals, however, that the issue is not that simple. There is abundance of evidence to show that giving information directly to patients can result in substantial prescription distortions (see section 2.6 for more detail). Our stance is that to understand and analyze this current debate in the right frame, we need to look at the human side of the physician-patient relationship and consider some psychological or behavioral factors that could potentially harm prescription decisions. More precisely, we focus on two psychological factors that are particularly eminent in this problem:

- Informed patients may develop a biased preference for a particular product, depending on the extent of exposure to DTCA. Given this biased attachment, some patients may obstinately ask for a specific brand which may not be the best alternative.
- Persuasion is costly under any circumstance, and this is especially so for physicians who tend to have a high opportunity cost. The cost is even higher when the patient is ill-informed to begin with and has developed a biased attachment.

Under these conditions, we may in fact have a situation where DTCA can be "too informative," and there may arise a scope for welfare improvement by regulating the content of DTCA. One way to achieve this is to require that DTCA only describe the presence of a disease and its typical symptoms and treatments but without providing any information about particular brand names or products; throughout the analysis, we refer to this type of advertisement as *category-specific*. This is precisely what has been done in many countries outside of the US where DTCA is permitted only if it is category-specific in nature.²

²Outside of the US, New Zealand is the only OECD country which allows DTCA that includes product

At a glance, category-specific DTCA can be an effective remedy for the distortion caused by DTCA in pharmaceutical markets because it only provides minimal information to patients while still serving the educational purpose. However, the welfare impact of such a regulation is not necessarily so straightforward, especially in pharmaceutical markets that are typically oligopolistic and dominated by a few major firms, as it alters the strategic nature of market competition. In particular, under category-specific DTCA, advertisement effectively becomes a public good, which diminishes each firm's incentive to engage in marketing activities. This can be welfare-reducing as it may leave a large fraction of potential patients uninformed and receiving no treatment at all in the end.

In this paper, we investigate how the content regulation of DTCA (hereafter, simply the content regulation) affects pharmaceutical firms' expenditures on advertisement and welfare to contribute to the current debate surrounding the pharmaceutical industry. To this end, we analyze a market of prescription-only drugs for a particular disease with two horizontally differentiated firms (pharmaceutical suppliers). The two firms decide how much to advertise to reach out potential patients who are initially unaware of the disease and need to be informed via advertisement to receive any medical treatment before it becomes too serious. Within this framework, we consider two DTCA regulation policies: in one case, the firms are allowed to promote their own specific brand names (product-specific DTCA); in the other, they can only provide general information about a disease and its typical symptoms without specifying their own brand names (category-specific DTCA).

The difference in the nature of DTCA gives different incentives to the pharmaceutical firms and induces different responses from potential patients. Under category-specific DCTA, no specific information about a particular product is available, and all informed patients are largely neutral with respect to the two available products. Under product-specific DTCA, on the other hand, informed patients may develop a biased attachment to a particular product when exposed to DTCA. Borrowing from Brekke and Kuhn (2006), we call those patients *selective* and *captive* throughout the analysis: patients are called selective when they are exposed to both firms' products and captive when exposed only to one of them. In either case, patients are informed and hence visit a (representative) physician in search of medical treatment, which captures the demand-expansion effect of DTCA (Grossman and Shapiro, 1984). We assume, however, that only selective patients can ask the physician to compare the two products and be prescribed a suitable drug while

names. In other countries, there are some regulations on the extent that a firm can promote its own brands or products, virtually prohibiting product-specific DTCA.

captive patients, with a biased attachment, insist on the product they have in mind and needs to be persuaded if an alternative measure is to be taken.

Faced with a captive patient, the physician needs to persuade him but as noted above, this persuasion process is, broadly speaking, costly for the physician. The presence of captive patients may then create a welfare loss by leading to inappropriate prescribing if the physician deems the persuasion cost too much to bear. A subtle point to note is that the presence of the persuasion cost, which imposes an extra burden on the physician, creates two forms of market distortion under product-specific DTCA: on one hand, a fraction of captive patients may receive a suboptimal outside treatment; on the other hand, they may also be prescribed the suboptimal product. To make clear distinction between them, we refer to the former, which limits the equilibrium industry demand below its efficient level, the *market-size distortion*, and to the latter as the *prescription distortion*. These two forms of distortion depend on various parameters in a somewhat complicated way and play a major role in our welfare analysis.

Main results: We obtain several results regarding the costs and benefits of product-specific DTCA vis-a-vis category-specific DTCA. Our argument centers around the interactions between the two forms of market distortion mentioned above and the strategic investment incentives that are crucially shaped by the context regulation in effect.

First, it is intuitively clear that the firms invest more in advertisement under product-specific DTCA than under category-specific DTCA. In fact, the advertisement competition is excessively strong under product-specific DTCA because of the potential of business stealing, as a consequence of which the firms overinvest in advertisement compared to the efficient level. Under category-specific DTCA, on the other hand, the incentives to inform patients via advertisement are substantially weaker due to its public-good nature, and the firms indeed underinvest compared to the efficient level. Despite the apparent distortion in prescription decisions induced by product-specific DTCA, the welfare effect of the content regulation is thus ambiguous because the stronger, though excessive, incentives to invest in advertisement reduce the number of uninformed patients, ultimately helping them receive treatment in time.

With this result as a building block, we proceed to characterize the welfare impact of the content regulation. When the cost of advertisement is relatively high, the interests of the firms and potential patients are better aligned, and there often exists a Paretoimproving policy choice. The optimal policy in this case is largely determined by the extent of the market-size distortion induced by product-specific DTCA: category-specific DTCA is preferred when the market-size distortion is more severe while product-specific DTCA is preferred when it is less so. As the cost of advertisement decreases, however, the over-investment problem becomes more of a factor, and a serious conflict arises between the firms and potential patients as a consequence: while the overinvestment problem is purely detrimental to the firms, it is beneficial from the viewpoint of patients as it reduces the likelihood of being left out and helps them seek treatment in time. In general, patients are made better off under product-specific DTCA as the cost gets smaller while the firms are made worse off.

It is also worth emphasizing that although the presence of the persuasion cost is the source of market distortion in our setup, its welfare impact is not entirely negative. This is because an increase in the persuasion cost affects the two forms of distortion in different ways: while it aggravates the prescription distortion, it reduces the market-size distortion by allowing each firm to capture patients who would otherwise be prescribed the rival firm's product. The latter effect provides the firms an extra incentive to invest in advertisement, which can potentially be welfare-enhancing by reducing the number of uninformed patients. We find that this "distortions as incentives" mechanism can indeed be strong enough to compensate for the loss from the prescription distortion and benefit not only the firms but also patients under some conditions.

Finally, although most of our key insight can be illustrated under the assumption of regulated prices, we also extend the baseline model to incorporate the firms' price setting behavior to evaluate the impact of free market pricing in this context. We find that when the regulated price is set low enough to begin with, free market pricing not only benefits the firms but also patients despite the higher equilibrium price. This suggests that the two forms of regulation are often complementary to each other in that a less stringent advertisement regulation (i.e., product-specific DTCA) tends to be compatible with a less stringent price regulation—the type of policy mix adopted in the US—and hence should be evaluated jointly.

Literature: Our model is most closely related to and draws on Brekke and Kuhn (2006) who investigate the strategic relationship between DTCA and detailing (marketing to physicians). Their focus is on how much information to disseminate to physicians via detailing while their DTCA is category-specific in nature as its only role is to bring them to clin-

ics.³ Detailing in their model plays a similar role to product-specific DTCA in that it is an attempt to affect the market share, but is fundamentally different as it is targeted to a different segment of market participants, i.e., physicians; DTCA and detailing are thus complementary to each other, with no inherent conflict between them. This draws clear contrast to our model where product and category-specific DTCA are two alternative ways of reaching out to potential patients. We show that these two forms of DTCA yield different welfare implications, thereby calling for active policy intervention under some conditions.

There are several studies in the fields of management and marketing which investigate the issue of product-specific advertisement versus category-specific advertisement. Bala and Bhardwaj (2010) consider two forms of DTCA, called constructive and combative advertising, in conjunction with detailing. Combative advertising in their model is similar to product-specific DTCA although it has no market-expansion effect. In their model, each firm mixes two forms of DTCA and examines the optimal share of DTCA. Amaldoss and He (2009) consider a duopoly setting where each firm chooses the brand specificity of DTCA. They consider an aggregate demand model and conduct comparative statics with respect to the brand specificity. Both of these studies are entirely positive and provide no welfare analysis; most importantly, patients are not horizontally differentiated in their settings, so that there is no explicit measure of product-patient mismatch (and prescription distortion) which lies at the core of our welfare analysis.

Our work also has an obvious connection to analyses of advertisement in industrial organization. The effect of brand specificity in DTCA has been examined in various contexts outside of the pharmaceutical industry (Krishnamurthy, 2000; Jørgensen and Sigué, 2015; Amir et al., 2021; Shin and Yu, 2021). In conventional analyses of advertisement, consumers precisely know their preferences (or payoff functions). Here, the situation we deal with is somewhat special in that physicians, with their expertise, are better informed about what patients need than patients themselves: specifically, we assume that patients are lined up on a Hotelling line without knowing their exact location which only the attending physician can tell. However, even though physicians are informationally superior, it is still patients who must make an initial move and contact a physician on their own. This unique industry structure gives rise to the possibility of "misinformation" and a scope for welfare improvement by policy intervention. Although we cast our analysis in a specific context of advertisement competition, we believe that this problem can provide a broader perspective as it is a realistic and pervasive issue in the medical industry at large to what

³In their model, there are captive and selective physicians, but no such distinction is made for patients.

extent patients should be informed.

2. The Model

2.1. Environment

We consider a market of prescription-only drugs for a particular symptom that comprises of two pharmaceutical firms, potential patients and a representative physician. In this market, potential patients with unit mass are horizontally differentiated and uniformly distributed on the Hotelling line extended on [0,1]. Each patient is identified with his location x which captures his "type" (e.g., disease type and/or physical constitution). The pharmaceutical products are supplied by the two firms, each indexed by i=0,1, where firm 0 is located at the left end of the Hotelling line while firm 1 is located at the right end. The two firms supply pharmaceutical products for the same symptom although their products are horizontally differentiated by their chemical compounds and consequent treatment effects.

As a motivating example of our setting, we take the market of antidepressants which offers a variety of drugs for major depressive disorder (MDD).⁴ Those drugs are categorized into several distinct classes such as selective serotonin reuptake inhibitor (SSRI), serotonin norepinephrine reuptake inhibitor (SNRI), and noradrenergic antagonist-specific serotonin antagonist (NaSSA). Drugs from different classes have different mechanisms of action and hence produce different effects, or side effects, on different patients. Moreover, even within the same class, there are some subtle yet notable pharmacological differences. As a consequence, there are no "one-size-fits-all" drugs that are equally effective for all MDD patients (Santarsieri and Schwartz, 2015). Finding a right drug for each patient generally requires a high level of expertise, giving rise to a unique feature of the market where buyers (patients) often do not know the best product for them, and must rely on outside experts (physicians) to identify what is best suited for them.

2.2. Pharmaceutical firms

In the first stage of the game, the two firms simultaneously choose the levels of DTCA, which directly affect each potential patient's decision of whether or not to seek medical treatment by visiting a physician. Let θ_i denote the fraction of patients who are exposed

⁴Incidentally, it is also one of the most DTCA-intensive markets. See Rosenthal et al. (2002) and Shapiro (2020).

to firm *i*'s advertisement. The demand for firm *i*'s product (product *i*) is assumed to be a function of (θ_i, θ_{-i}) , $i \neq -i$, and denoted as $Q_i(\theta_i, \theta_{-i})$. Firm *i*'s profit is then given by

$$\pi_i = p_i Q_i(\theta_i, \theta_{-i}) - \frac{c}{2} \theta_i^2,$$

where p_i denotes the price of product i, and c measures the cost of advertisement. To focus our attention, we abstract away from detailing and take the intensity of detailing as exogenous.⁵

As most of our key insight can be illustrated under regulated prices, we assume for now that the pharmaceutical prices are fixed exogenously at $p = p_0 = p_1$ based on factors that are not directly related to the issue at hand. The assumption of regulated prices is a relevant one in most countries outside of the US; we will extend our analysis to allow the firms to set prices endogenously in section 5. To ensure the existence of an interior solution, we also assume that c is large relative to p.

Assumption 1. 2c > p.

2.3. Patients

A patient receives medical treatment when he visits a physician. The utility derived by patient x from consuming a unit of product i is given by

$$u(x,i) = v - \tau |x - i| - \lambda p,$$

where $v \in (0, \infty)$ denotes the gross effectiveness of a pharmaceutical product, $\lambda \in [0, 1]$ the copayment rate, and $\tau \in (0, \infty)$ the "transport cost." The transport cost τ measures the degree of substitutability between the two products: for instance, τ is larger for two antidepressants that belong to different classes than for those in the same class. The utility of patients who receive no treatment is normalized to 0.

The key aspect of our model is that a patient's type x can be observed only by the physician but not by the patient himself—a defining characteristic of the pharmaceutical industry—as assumed in Brekke and Kuhn (2006). The two products have the same effectiveness but differ in their treatment effects across patients, depending on the specific product-patient type match. We assume that each product is effective enough to ensure that it is beneficial even for the most "distant" patient (at x = 0.5).

⁵This is by no means to deny the importance of detailing. It is rather the opposite: we look at the case where detailing is so pervasive that almost all physicians have already been exposed to the two products (as we assume here), and detailing can hardly make a difference.

Assumption 2. $2(v - \lambda p) > \tau$.

The assumption suggests that there are some patient types for whom multiple products are potentially beneficial, as is typically the case. Moreover, it is important to note that the choice of advertising regulation increasingly becomes both irrelevant and insignificant as the value of each product, measured by $v - \lambda p$, gets smaller. For the purpose of this study, we thus focus on the case where the value is relatively large.

2.4. Advertising regulation

We consider two regulatory regimes—product-specific and category-specific—on the content of DTCA. Under product-specific DTCA, a pharmaceutical firm is allowed to provide information about a specific product, including its brand name, usage and safety.⁶ Then, when the firms are allowed to engage in product-specific DTCA, patients are classified into three (publicly observable) informational types, depending on the extent of exposure to the firms' DTCA:

- A patient is *selective* if he is exposed to advertisement from both firms, which occurs with probability $\theta_i \theta_{-i}$.
- A patient is *captive of product i* if he is exposed to advertisement from only one of the firms, which occurs with probability $\theta_i(1-\theta_{-i})$.
- A patient is *uninformed* if he is exposed to advertisement from neither, which occurs with probability $(1 \theta_i)(1 \theta_{-i})$.

In contrast, when the firms are restricted to category-specific DTCA, they can only provide general information about the presence of a disease and its symptoms without specifying particular brand names or products. Under category-specific DTCA, therefore, there are no captive patients. As such, a patient becomes selective with probability $\theta_i + \theta_{-i} - \theta_i \theta_{-i}$ and uninformed with $(1 - \theta_i)(1 - \theta_{-i})$.

⁶An important premise of our analysis is that product-specific DTCA is effective in raising the demand for its own product (via business stealing) and indeed generates different effects from category-specific DTCA. Of course, if product-specific and category-specific DTCA make no difference, the whole debate over DTCA becomes groundless; the current debate over DTCA reflects physicians' perception that product-specific DTCA does make a difference at some level. Although empirical evidence on this issue may not be conclusive, there is in fact some evidence in support of this claim (Kalyanaram, 2008, 2009; Liu and Gupta, 2011; Dave and Saffer, 2012; Sinkinson and Starc, 2018). Most notably, Sinkinson and Starc (2018) measure the impact of DTCA by pharmaceutical firms and find substantial evidence of business stealing.

2.5. Physician

We assume that there is a representative physician to whom all potential patients visit when they become informed. The physician can prescribe either product 0 or product 1, or else can choose to provide an "outside treatment," which invariably provides a utility of 0 for all patient types. Given our focus on DTCA, we interpret that products 0 and 1 refer to drugs that are relatively new to the market (and hence need to be advertised) while an outside treatment refers to an aggregate category of "everything else," including all other conventionally accepted drugs and medical practices: in the case of MDD, for instance, they include older brand-name drugs, psychotherapy and various lifestyle changes (such as eating right, getting enough sleep, doing regular exercise, etc). The physician is partly altruistic in that she cares about the welfare of patients net of the persuasion cost. The utility of the physician depends on the patient's informational type.

First, if a patient is selective, he is neutral and simply accepts the physician's advice as it is. In this case, the physician incurs no persuasion cost and her utility from prescribing product i is given by u(x, i). The physician thus simply prescribes the more suitable product for the patient.⁹

Second, if a patient is captive, he has developed a biased preference in favor of the product he has been exposed to and makes a specific request for it. We assume in this case that the physician either prescribes the product as requested or provides an outside treatment. If the physician takes the option of outside treatment, she must incur a persuasion cost d which includes time, effort, and psychological discomfort of persuading a patient, as well as the risk of disrupting the future relationship. As such, the physician prescribes the requested product i if and only if $u(x,i) \ge -d$.

The assumption that the physician provides an outside treatment, rather than the other product, when the persuasion cost is small enough merits a further remark. This specification effectively implies that under product-specific DTCA, patients who are captive of

⁷More precisely, the utility gain of an outside treatment is independent of the patient type which is defined with respect to the relative effectiveness of the two products in question.

⁸What constitutes an outside treatment obviously depends on the context. For instance, since promotional activities typically decrease after generic entry (Huskamp et al., 2008), we can take brand-name drugs with expired patents as "outside of our model." Therefore, if we look at the market of antidepressants in the last decade or so, brand-name drugs such as Prozac, Zoloft, Paxil and Celexa—drugs that had seen generic entry by the early 2000s—can be regarded as constituting part of an outside treatment.

⁹Under Assumption 2, it is never optimal to provide an outside treatment in this case.

i can only be prescribed product *i*, suggesting that product-specific DTCA generates no spillover benefits to the rival firm. Note, however, that it is not our intention to argue that product-specific DTCA generates no spillovers;¹⁰ what is essential in this specification is rather that product-specific DTCA generates *less spillovers* than category-specific DTCA. Here, we focus on an extreme case where product-specific DTCA generates no spillovers in order to capture this aspect in the simplest manner.

2.6. On prescription distortion

The key premise of our analysis is that product-specific DTCA may disturb the physician-patient relationship and distort prescription decisions to some extent. We believe, at some intuitive level, that this is not only possible but also almost inevitable. On the one hand, it is hardly surprising that patients sometimes develop a biased preference for a particular product: first, it is very difficult, if not impossible, for patients to understand all the possible consequences of prescription drugs; at the same time, health-related problems are always emotionally arousing, and it is very difficult to stay perfectly calm and rational throughout the decision process. On the other hand, physicians are also humans with limited resources and persuasion is never free. A combination of these two factors necessarily gives rise to some distortion in prescription decisions.

To motivate our analysis further, there is also ample evidence which suggests that giving detailed information to patients via product-specific DTCA creates seemingly unnecessary complications and psychological pressures to physicians. Murray et al. (2003) report that many physicians feel that DTCA worsens the time efficiency of the visit and have a negative response to the recent increase in DTCA. Similarly, Robinson et al. (2004) report that "few (physicians) believe that these advertisements are a positive trend in health care." Moreover, it is well documented that many physicians have prescribed inappropriate drugs which are requested by patients. ¹¹ Kravitz et al. (2005) also report that patients' requests

¹⁰This may or may not be true and is purely a matter of empirical investigation. For instance, Shapiro (2020) finds a spillover effect of brand-specific advertisement on other products. However, given that a physician has a wealth of alternative options when faced with a captive patient, it is natural to assume that she provides an outside treatment, which includes "everything other than the rival product," at least on some occasions. This naturally limits the extent of spillovers compared to category-specific DTCA. It is of course straightforward to extend our setting to incorporate some spillovers of product-specific DTCA, at the expense of an extra notation.

¹¹See Frosch et al. (2010) and Ventola (2011) for surveys on the effect of DTCA on the physicians' prescription decision. Frosch et al. (2010) argue that the request for the advertised product by the patients increases

have a profound effect on prescription decisions in major depression and adjustment disorder. These results lend support for the view that physicians do indeed face some persuasion costs when patients are exposed to product-specific DTCA. Additionally, the magnitude of the persuasion cost seems to differ across symptoms. For instance, Kravitz et al. (2005) find that the influence of patients, especially brand-specific requests, on the prescription of antidepressants is higher for adjustment disorder than for major depression and argue that DTCA may stimulate prescribing more for questionable than for clear indications. This line of argument is again consistent with the existence of persuasion costs because it is natural to think that persuasion is more costly in the absence of clear and verifiable scientific evidence.

3. Analysis

3.1. Prescription decisions

We solve the model backward, starting with the final stage where the representative physician decides whether to prescribe a drug and, if so, which one. Given that a patient visits the physician, he must be either selective or captive. The physician's prescription decision clearly depends on the patient's informational type as we describe below.

Suppose first that a patient is selective (exposed to both products). Under Assumption 2, the physician recommends product 0 (product 1) if $x \le 0.5$ (x > 0.5) and the patient simply accepts the advice. As such, the two firms split the market evenly in this contingency. Since the two firms compete for selective patients, this is the competitive segment of the market.

If a patient is captive (exposed only to one of the products), on the other hand, the physician chooses either to prescribe the product he requests or to provide an outside treatment. The market is now monopolized by one firm, i.e., this is the monopolistic segment of the market. The physician prescribes a requested product if and only if the benefit exceeds the persuasion cost, i.e.,

$$v - \lambda p - \tau |x - i| \ge -d$$
.

both appropriate and inappropriate prescribing, though it is unclear which effect is greater. Weissman et al. (2004) show that physicians who consider DTCA positively prescribe more DTCA drug than those who consider DTCA negatively. Dai et al. (2005) also argue that the marketing activities by Merck had contributed to the widespread use of Vioxx, which was discontinued due to fatal side effects.

The demand for each product in the monopolistic segment is then given by

$$M:=\min\left\{\frac{\nu-\lambda p+d}{\tau},1\right\}.$$

By Assumption 2, M > 0.5, so that each firm can capture a larger market share.

Note that the presence of the persuasion cost generates two forms of distortion in prescription decisions. First, there is a fraction of captive patients who end up receiving an outside treatment. For the sake of exposition, we call this the *market-size distortion*, the size of which is captured by M, as it limits the industry demand below its efficient level. We say that the market-size distortion is more severe when M is small and close to 0.5, while it is less so when M is large and close to 1. Second, there is also a fraction of captive patients who are prescribed the suboptimal product. This form of distortion is called the *prescription distortion*, which we will discuss in depth when we analyze the welfare of patients in section 4.3.

3.2. Advertisement expenditures

Each firm decides how much to invest in advertisement, taking the other firm's choice as given. First, under product-specific DTCA, a patient is selective with probability $\theta_i\theta_{-i}$ and captive of i with probability $\theta_i(1-\theta_{-i})$. The demand for product i under product-specific DTCA is then obtained as

$$Q_i^p = \frac{\theta_i \theta_{-i}}{2} + \theta_i (1 - \theta_{-i}) M,$$

where the superscript P stands for product-specific DTCA. It follows from this that firm *i*'s problem under product-specific DTCA can be written as

$$\max_{\theta_i \in [0,1]} pQ_i^P - \frac{c}{2}\theta_i^2 = p\left[\frac{\theta_i \theta_{-i}}{2} + \theta_i (1 - \theta_{-i})M\right] - \frac{c}{2}\theta_i^2.$$

The best-response function is given by

$$\theta_i = \frac{p}{2c} [2M - \theta_{-i}(2M - 1)].$$

Solving this, the equilibrium expenditure, denoted by θ^P , can be obtained as

$$\theta^P = \frac{2Mp}{2c + p(2M - 1)}.$$

Note that $\theta^P < 1$ by Assumption 1.

Second, there are no captive patients under category-specific DTCA. The demand under category-specific DTCA is hence given by

$$Q_i^E = \frac{\theta_i + \theta_{-i} - \theta_i \theta_{-i}}{2},$$

where, as above, the superscript E stands for category-specific DTCA. 12 As such, firm i's problem under category-specific DTCA can be written as

$$\max_{\theta_i \in [0,1]} pQ_i^{\mathrm{E}} - \frac{c}{2}\theta_i^2 = p\frac{\theta_i + \theta_{-i} - \theta_i\theta_{-i}}{2} - \frac{c}{2}\theta_i^2.$$

We can then obtain the best-response function as

$$\theta_i = \frac{p(1-\theta_{-i})}{2c}.$$

It follows from this that the equilibrium expenditure, denoted by $\theta_i^{\rm E}$, can be obtained as

$$\theta^E = \frac{p}{2c + p}.$$

It is intuitively clear that the firms have more incentive to invest in advertisement when they are allowed to engage in product-specific DTCA. To see this, observe that

$$\theta^{P} > \theta^{E} \iff \frac{2Mp}{2c + p(2M - 1)} > \frac{p}{2c + p}.$$

This is further reduced to

$$2M(2c + p) > 2c + p(2M - 1),$$

which holds for any $M \in (0.5, 1]$. In words, there is a private gain from DTCA when the firms are allowed to promote their own products, giving them more incentive to invest in advertisement. Moreover, we can show that the difference in the advertisement expenditures widens as c gets smaller.

We summarize these findings below. Throughout the analysis, all the proofs are relegated to the Appendix unless otherwise noted.

¹² Here, the demand size for each firm is simply one half of all informed patients because of Assumption 2. If this assumption fails and M < 0.5, on the other hand, the demand size becomes $M(\theta_i + \theta_{-i} - \theta_i \theta_{-i})$. This means that the optimal investment under either regime tends to 0 as $M \to 0$. When M is relatively small, therefore, the comparison of regulatory regimes becomes increasingly irrelevant and also insignificant in absolute terms.

Proposition 1. The advertisement expenditure is higher under product-specific DTCA than under category-specific DTCA. Moreover, the difference between θ^P and θ^E is strictly decreasing in c.

Given our incentive structure, it is intuitively clear that the firms invest more in advertisement under product-specific DTCA than under category-specific DTCA; in fact, similar results have been obtained in the literature (Brekke and Kuhn, 2006; Bala and Bhardwaj, 2010). In this paper, we use this fact as a building block and investigate how this incentive structure interacts with the two forms of market distortion—market-size distortion and prescription distortion—induced by the physician's persuasion cost and affects welfare.

3.3. Equilibrium market demand

The effect of the content regulation on the demand for each product is ambiguous with two opposing forces at work: although product-specific DTCA expands the potential market size due to higher advertisement expenditures, prescription decisions are necessarily distorted. To see this, observe that

$$\Delta Q := Q^P - Q^E = \frac{\theta^P [\theta^P + 2(1 - \theta^P)M]}{2} - \frac{\theta^E (2 - \theta^E)}{2}.$$

This shows that for any $M \in (0.5, 1]$ and $\theta^P = \theta^E$, $\Delta Q < 0$, i.e., if the equilibrium advertisement expenditures are the same between the two regimes, the demand for each product is higher under category-specific DTCA. A necessary condition for $\Delta Q > 0$ is hence that θ^P is sufficiently larger than θ^E , although this happens for a wide range of parameter values as we will see below.

To conduct comparative statics on ΔQ , we first obtain

$$\Delta Q = \frac{4M^2cp}{[2c+p(2M-1)]^2} - \frac{p(4c+p)}{2(2c+p)^2}.$$

From this, $\Delta Q > 0$ if and only if

$$\frac{8M^2c}{4c+p} > \left[\frac{2c+p(2M-1)}{2c+p} \right]^2. \tag{1}$$

Two facts are worth noting here. First, ΔQ naturally converges to 0 as c tends to infinity, because the advertisement expenditures converge to zero under both regimes. Second, observe also that (1) always holds at c = 0.5p, regardless of M, since the difference in

the advertisement expenditures widens as c gets smaller. Aside from these properties, the exact shape of ΔQ depends crucially on the value of M, as the next proposition fully characterizes.

Proposition 2. $\Delta Q > 0$ for all $c \in [0.5p, \infty)$ if $M \ge \sqrt{0.5}$. For $M \in [0.5, \sqrt{0.5})$, there exists an interior threshold $c_Q(v,d) \in (0.5p,\infty)$ such that $\Delta Q > 0$ if and only if $c_Q(v,d) > c$. Moreover, $c_Q(v,d)$ is increasing in both v and d.

3.4. Efficient advertisement

The content regulation influences the equilibrium advertisement expenditure by changing the nature of competition between the two firms. To illustrate this point, it is instructive to derive the efficient level of advertisement expenditures, defined as the one that maximizes the joint profit of the two firms, and compare it to the equilibrium level.

The efficient level under product-specific DTCA can be obtained by solving the following problem:

$$\max_{\theta \in [0,1]} p \left[\frac{\theta^2}{2} + \theta (1 - \theta) M \right] - \frac{c}{2} \theta^2$$

Let θ^{P*} denote the efficient level under product-specific DTCA, which is given by

$$\theta^{p*} = \frac{Mp}{c + p(2M - 1)}.$$

It is clear that $\theta^P > \theta^{P*}$, i.e., the firms overinvest in advertisement under product DCTA. This is due to the business-stealing effect of advertisement: a firm can "steal" a fraction of captive patients by reaching out to them and transforming them into selective.

Similarly, the problem under category-specific DTCA is defined as

$$\max_{\theta \in [0,1]} \frac{p}{2} (2\theta - \theta^2) - \frac{c}{2} \theta^2$$

As above, let θ^{E*} denote the efficient level under category-specific DTCA, which is given by

$$\theta^{E*} = \frac{p}{c+p}.$$

It follows from this that $\theta^{E*} > \theta^E$, i.e., the firms underinvest in advertisement under category-specific DTCA. Aside from the fact that there is no business-stealing effect, this is due to the fact that advertisement under category-specific DTCA is a public good which also increases the demand for the rival product as well.

We summarize these findings as follows (the proof omitted).

Proposition 3. The firms overinvest in advertisement under product-specific DTCA and underinvest under category-specific DTCA.

4. Optimal content regulation of DTCA

4.1. Welfare measures

Denote by π^k the equilibrium profit under regime k = P, E. Similarly, we denote by PS^k what we call the patient surplus (to be defined more precisely below). We consider a regulatory authority with the objective function given by

$$W^k = \omega \pi^k + (1 - \omega) P S^k, k = P, E,$$

where the parameter $\omega \in [0,1]$ reflects the authority's policy stance where it is more "pro-business" when ω is close to 1 while it is "pro-patient" when ω is close to 0.¹³

4.2. Firm profit

We first examine how the content regulation affects the firms' equilibrium profit. Letting $\Delta \pi := \pi^P - \pi^E$, $\Delta \theta := \theta^P - \theta^E$ and $\Delta C := (\theta^{P^2} - \theta^{E^2}) = \Delta \theta (\theta^P + \theta^E)$, we obtain

$$\Delta \pi = p \Delta Q - \frac{c}{2} \Delta C
= \frac{p \theta^{P} [2M - (2M - 1)\theta^{P}]}{2} - \frac{p \theta^{E} (2 - \theta^{E})}{2} - \frac{c}{2} \Delta \theta (\theta^{P} + \theta^{E})
= -p(1 - M)\theta^{E} (1 - \theta^{E}) - \frac{\Delta \theta}{2} \left[\frac{(2M - 1)p + c}{2} (\theta^{P} + \theta^{E}) - 2Mp \right].$$
(2)

The impact of the content regulation on the equilibrium profit can be decomposed into two factors. Evaluated at $\theta^P = \theta^E$, any difference in the profits can be attributed to the distortion in prescription decisions. The first term of the right-hand side of (2) captures

¹³We do not take into account the persuasion cost incurred by the physician, assuming that the number of physicians, and hence their impact on the overall welfare, are negligibly small.

this effect which is negative for any 1 > M. The second term captures the difference in the gains from demand expansion. Note that the sign of this effect is ambiguous, because the firms tend to overinvest under product-specific DTCA, diminishing their profit margins with excessive competition in advertisement.

We have already characterized ΔQ in Proposition 2. Also, from Proposition 1, we know $\Delta C \geq 0$, meaning that $\Delta Q < 0$ is a sufficient condition for $\Delta \pi < 0$. This implies that the firms are more likely to prefer category-specific DTCA to product-specific DTCA. In fact, this is always the case when M is relatively small, i.e., the market-size distortion is relatively large, as the next proposition suggests.

Proposition 4. $\Delta \pi < 0$ for all $c \in [0.5p, \infty)$ if $\sqrt{0.75} \ge M$. For $M \in (\sqrt{0.75}, 1]$, there exists an interior threshold $c_{\pi}(v, d) \in (0.5p, \infty)$ such that $\Delta \pi < 0$ if and only if $c_{\pi}(v, d) > c$. Moreover, $c_{\pi}(v, d)$ is decreasing in both v and d.

[Figure 2 about here]

4.3. Patient surplus

From the viewpoint of policymaking, the welfare of patients often carries more weight than that of firms. Since the payoff of uninformed patients is invariably zero, we only need to consider selective and captive patients. Let PS_S and PS_C denote the patient surplus for selective and captive patients, respectively.¹⁴ Also, define PS^k as the total patient surplus under regime k = P, E, where

$$PS^{P} = \theta^{P^{2}} PS_{S} + 2\theta^{P} (1 - \theta^{P}) PS_{C}$$

$$PS^{E} = \theta^{E} (2 - \theta^{E}) PS_{S}.$$

First, selective patients always end up with the most suitable product. The patient surplus for this group of patients is hence given by

$$PS_S = 2 \int_0^{\frac{1}{2}} (v - \lambda p - \tau x) dx$$
$$= v - \lambda p - \frac{\tau}{4}.$$

¹⁴In our model, some patients are misguided to demand the wrong product. There arises a conceptual issue in such a setting as to whether one should use true preferences or manipulated preferences to measure welfare. We here choose the former, provided that a patient's payoffs stem from the long-run impact on his health rather than from the joy of consuming a particular drug.

On the other hand, captive patients are prescribed the requested product as far as the patient benefit exceeds the persuasion cost. The patient surplus for this group of patients is given by

$$PS_C = \int_0^M (v - \lambda p - \tau x) dx$$
$$= (v - \lambda p)M - \tau \frac{M^2}{2}.$$

Define

$$\phi := \frac{4(\nu - \lambda p) - 2\tau M}{4(\nu - \lambda p) - \tau},$$

such that $M\phi := \frac{PS_C}{PS_S}$ which we use as a measure of the overall distortion in prescription decisions (more on this point later in section 4.4).

The welfare ranking among the three types of patient is clear: selective patients earn the highest payoff while uninformed ones earn the lowest which is zero.¹⁵ There are hence two ways to improve the patient surplus, either to increase the number of selective patients or to decrease the number of uniformed patients. More precisely, similarly as above, define $\Delta PS := PS^P - PS^E$, which is given by

$$\Delta PS = \theta^{P^{2}} PS_{S} + 2\theta^{P} (1 - \theta^{P}) PS_{C} - \theta^{E} (2 - \theta^{E}) PS_{S}$$

$$= -2\theta^{P} (1 - \theta^{P}) (PS_{S} - PS_{C}) + \Delta \theta (2 - \theta^{P} - \theta^{E}) PS_{S}.$$
(3)

Again, the impact of the content regulation can be decomposed into two factors. The first term of the right-hand side of (3) captures the loss due to the distortion in prescription decisions under product-specific DTCA: this fraction of patients would have been selective under category-specific DTCA but become captive under product-specific DTCA. Since $PS_S > PS_C$, this term is always negative. The second term captures the demand-expansion effect under product-specific DTCA, which is always positive.

To conduct comparative statics on ΔPS , we write (3) as

$$\Delta PS = \frac{4Mp[MpPS_S + (2c-p)PS_C]}{[2c+p(2M-1)]^2} - \frac{p(4c+p)PS_S}{(2c+p)^2}.$$

¹⁵Some captive patients may earn negative payoffs (when persuasion is costly) but Assumption 2 ensures that PS_C is always positive.

From this, $\Delta PS > 0$ if and only if

$$\frac{4M^{2}[p+(2c-p)\phi]}{4c+p} > \left[\frac{2c+p(2M-1)}{2c+p}\right]^{2}.$$
 (4)

Clearly, there is a close relationship between ΔPS and ΔQ ; we in fact obtain (1) if ϕ in (4) is replaced with 1. First, for the same reason as for ΔQ , ΔPS converges to 0 as c tends to infinity. Second, as the demand-expansion effect dominates, this condition holds at c=0.5p. Following the same procedure, we can establish the following claim which runs parallel to Proposition 2, except for the effect of d which we will discuss in more depth in the next subsection.

Proposition 5. $\Delta PS > 0$ for all $c \in [0.5p, \infty)$ if $M\sqrt{\phi} \ge \sqrt{0.5}$. For $M \in [0.5, \sqrt{0.5\phi^{-1}})$, there exists an interior threshold $c_{PS}(v, d)$ such that $\Delta PS > 0$ if and only if $c_{PS}(v, d) > c$. Moreover, $c_{PS}(v, d)$ is increasing in v.

The overall welfare effects of the content regulation are summarized in Figure 4. There are two crucial factors: the cost of advertisement c and the market-size distortion (which in turn depends on various parameters such as v, p and d). When the cost of advertisement is relatively high, the interests of the firms and potential patients are better aligned, and there often exists a Pareto-improving regime, depending on the extent of the market-size distortion: category-specific DTCA is preferred when the market-size distortion is more severe (M close to 0.5) while product-specific DTCA is preferred when it is less so (M close to 1). As c decreases, however, a conflict arises between them. In general, product-specific DTCA becomes the preferred choice for patients when c is relatively small, because the benefit of product-specific DTCA is maximized while the distortion in prescription decisions becomes more attenuated. However, the firms are generally worse off under product-specific DTCA because the overinvestment problem becomes more serious as the cost of advertisement gets smaller.

[Figures 3 and 4 about here]

4.4. Market distortions as incentives

As mentioned earlier, the distortion in prescription decisions is ultimately detrimental to patients for two reasons. First, the size of the market for captive patients is restricted

 $^{^{16}}$ To see this, consider an extreme case where c=0.5p and hence $\theta^P=1$. In this case, the patient surplus is maximized because patients are all selective. Patients are clearly made better off under product-specific DTCA as it causes no distortion while serving the educational purpose to the full extent.

as only a fraction M of them receive pharmaceutical treatment (the market-size distortion). Second, among those who do receive pharmaceutical treatment, some of them are prescribed the suboptimal product (the prescription distortion). The distortion in prescription decisions can be decomposed into two parts, where the ratio ϕ measures the size of the prescription distortion (corrected for the market size). Obviously, there arises no prescription distortion ($\phi=1$) when $\tau=0$, i.e., when the two products are perfect substitutes.

One crucial determinant of the overall distortion $M\phi$ is the persuasion cost d. An interesting point to note is that the effect of the persuasion cost is not entirely negative because a change in d affects M and ϕ in different ways: an increase in d raises M (less market-size distortion) but lowers ϕ (more prescription distortion). The former effect is conducive to more investment and can be welfare-improving under product-specific DTCA (as evidenced by the facts that Q^P and π^P are both increasing in d). The effect on the patient surplus is, however, more ambiguous as it also involves the prescription distortion. Below, we dissect how a change in the persuasion cost d affects the patient surplus.

Since the distortion due to the persuasion cost only arises under product-specific DTCA, we restrict our attention only to this case. Recall that the patient surplus under product-specific DTCA is given by

$$PS^{P} = \theta^{P} [\theta^{P} PS_{S} + 2(1 - \theta^{P}) PS_{C}].$$

Taking partial derivative with respect to *d* yields

$$\frac{\partial PS^{P}}{\partial d} = 2[\theta^{P}PS_{S} + (1 - 2\theta^{P})PS_{C}]\frac{\partial \theta^{P}}{\partial d} + 2\theta^{P}(1 - \theta^{P})\frac{\partial PS_{C}}{\partial d}.$$

The first term of the right-hand side reflects the indirect effect of d, which works through its impact on θ^P . Since θ^P is increasing in d (see the proof of Proposition 4), the indirect effect is unambiguously positive. The second term captures the direct effect, where we have

$$\frac{\partial PS_C}{\partial d} = -\frac{d}{\tau} < 0.$$

As such, PS^P (and hence ΔPS) increases with d if the positive indirect effect dominates the negative direct effect. The following result confirms that there is a range of situations where this holds true.

Proposition 6. Suppose $v - \lambda p > 3d$ and $v - \lambda p + d < \tau$. Then, PS^{p} increases with d if c is sufficiently large.

The proposition suggests that market distortions, in the form of a higher d, can be a blessing in this environment because they induce the firms to invest more in advertisement. Of course, this welfare improvement is at the expense of the physician who needs to incur more cost, and it will ultimately come down to how we evaluate this welfare loss (which we ignore in the current analysis).

5. A model with free market pricing

We have thus far assumed that the pharmaceutical prices are regulated and fixed exogenously at some predetermined level. Although this is a reasonable assumption in many countries, there is an important exception, namely the US, where pharmaceutical firms can set the prices relatively freely. Here, we extend the baseline analysis to incorporate price setting and illustrate how it would affect the equilibrium allocation.

5.1. Optimal pricing

We now consider a setting where each firm simultaneously chooses both the investment level θ_i and the price p_i . Given the price pair (p_0, p_1) , patient x in the competitive segment ends up with product 0 if

$$v - \tau x - \lambda p_0 > v - \tau (1 - x) - \lambda p_1 \iff \frac{\tau - \lambda (p_0 - p_1)}{\tau} > x.$$

Similarly, patient x in the monopolistic segment ends up with product i if

$$v - |x - i| - \lambda p_i > -d \iff v - \lambda p_i + d > |x - i|$$
.

The market demands for firm i are hence given by

$$Q_i^P = \theta_i \theta_{-i} \frac{\tau - \lambda (p_i - p_{-i})}{2\tau} + \theta_i (1 - \theta_{-i}) (\nu - \lambda p_i + d),$$

under product-specific DTCA and

$$Q_i^E = (\theta_i + \theta_{-i} - \theta_i \theta_{-i}) \frac{\tau - \lambda (p_i - p_{-i})}{2\tau},$$

under category-specific DTCA.

To gain some intuition for the effect of the price competition, it is helpful to look at each segment separately. First, if all patients are selective, each firm maximizes

$$p_i \frac{\tau - \lambda(p_i - p_{-i})}{2\tau},$$

and the (symmetric) optimal price is given by $p_S := \frac{\tau}{\lambda}$. Similarly, if all patients are captive, each firm maximizes

$$p_i(v - \lambda p_i + d)$$
,

and the optimal price is given by $p_C := \frac{v+d}{2\lambda}$. Naturally, the equilibrium price is bounded between p_S and p_C . This implies that free market pricing has little bite when p_C and p_S are sufficiently close to each other, in which case the equilibrium allocation can be approximated by that under price regulation.

In what follows, we assume that the price competition is intense enough so that the optimal price is lower in the competitive segment than in the monopolistic segment.

Assumption 3. $p_C > p_S \iff v + d > 2\tau$.

5.2. Advertisement expenditures

To analyze the firms' investment problems, we make additional assumptions that are analogous to Assumptions 1 and 2 to simplify the analysis and focus our attention on more relevant cases. First, c is assumed to be sufficiently large, so that the optimal investment is bounded below 1. Second, the value of each product is high enough to ensure that it is beneficial even for the most distant patient. The following assumptions provide sufficient conditions for these two properties.

Assumption 1'. $2c > p_C$.

Assumption 2′. $2(v - p_C) > 1$.

Under these assumptions, the problem faced by firm i under product-specific DTCA is formulated as

$$\max_{\theta_i \in [0,1], p_i} \ p_i Q_i^P - \frac{c}{2} \theta_i^2 = p_i \left[\theta_i \theta_{-i} \frac{\tau - \lambda (p_i - p_{-i})}{2\tau} + \theta_i (1 - \theta_{-i}) (\nu - \lambda p_i + d) \right] - \frac{c}{2} \theta_i^2.$$

This yields a pair of first-order conditions:

$$\begin{split} &\frac{p_i}{c} \left[\, \theta_{-i} \frac{\tau - \lambda (p_i - p_{-i})}{2\tau} + (1 - \theta_{-i})(\nu - \lambda p_i + d) \, \right] = \theta_i, \\ &\theta_i \theta_{-i} \frac{\tau + \lambda p_{-i}}{2\tau} + \theta_i (1 - \theta_{-i})(\nu + d) = \theta_i \theta_{-i} \frac{\lambda p_i}{\tau} + 2\lambda \theta_i (1 - \theta_{-i}) p_i. \end{split}$$

With abuse of notation, denote by θ^k and p^k the equilibrium investment and price levels, respectively, under regime k = P, E. Imposing symmetry, the equilibrium values must solve

$$\theta^{P} = \frac{2M(p^{P})p^{P}}{2c + p^{P}[2M(p^{P}) - 1]}, \ p^{P} = \frac{\theta^{P}\tau + 2\tau(1 - \theta^{P})(\nu + d)}{\lambda[\theta^{P} + 4\tau(1 - \theta^{P})]} = \frac{\theta^{P}p_{S} + 4\tau(1 - \theta^{P})p_{C}}{\theta^{P} + 4\tau(1 - \theta^{P})}, \ (5)$$

where $M(p) := \min\{v - \lambda p + d, 1\}$ to denote its dependence on p. Although the problem is now substantially complicated, we can still show that it always yields a well-defined solution.

Proposition 7. There exists a unique pair (θ^P, p^P) that satisfies (5). Moreover, θ^P is increasing in c while p^P is increasing, with $\lim_{c\to 0.5p_C} \theta^P = 1$, $\lim_{c\to 0.5p_C} p^P = p_S$, $\lim_{c\to \infty} \theta^P = 0$, $\lim_{c\to \infty} p^P = p_C$.

In contrast, the problem under category-specific DTCA is quite straightforward and defined as

$$\max_{\theta \in [0,1], p_i} p_i Q_i^E - \frac{c}{2} \theta_i^2 = p_i (\theta_i + \theta_{-i} - \theta_i \theta_{-i}) \frac{\tau - \lambda (p_i - p_{-i})}{2\tau} - \frac{c}{2} \theta_i^2.$$

The first-order conditions are given by

$$\begin{split} \frac{p_i(1-\theta_{-i})[\tau-\lambda(p_i-p_{-i})]}{2\tau c} &= \theta_i, \\ \frac{\tau+\lambda p_{-i}}{2} &= \lambda p_i. \end{split}$$

The optimal price is simple and obtained independently of (θ_i, θ_{-i}) , meaning that there is no strategic relationship between pricing and advertisement. The equilibrium values, denoted by θ^E and p^E , are given by

$$\theta^E = \frac{p^E}{2c + p^E} = \frac{\tau}{2\lambda c + \tau}, \ p^E = p_S = \frac{\tau}{\lambda}.$$

5.3. Welfare implications of free market pricing

Consider a regulatory authority which contemplates to deregulate pharmaceutical pricing by granting firms a higher degree of discretion over pricing. The welfare impact of such a reform depends obviously on the regulated price that has been imposed prior to the deregulation. For the sake of argument, suppose that the regulated price was initially set at $p = p_S$ (the lowest possible equilibrium price), as it is perhaps natural to assume

that pharmaceutical prices would rise after the deregulation. Under this circumstance, the equilibrium price jumps up to $p^P > p_S$ under product-specific DTCA while it remains the same under category-specific DTCA. Welfare implications of free market pricing can then be illustrated by examining how the two welfare measures under product-specific DTCA respond to an exogenous variation in p; below, we will hence focus on the case of product-specific DTCA to illustrate welfare consequences of free market pricing.

As in the case with price controls, it is first instructive to observe the effect of a change in *p* on the advertisement expenditure. With some computation, we obtain

$$\frac{\partial \theta^{P}}{\partial p} = \frac{4M(p)c + 2pM'(p)(2c - p)}{[2c + p(2M(p) - 1)]^{2}} = -\frac{\partial \theta^{P}}{\partial c} \frac{c}{p} + \frac{2pM'(p)(2c - p)}{[2c + p(2M(p) - 1)]^{2}},$$

which indicates that the effect of an increase in p mirrors that of a decrease in c, the only difference being that it may also change the market-size distortion under product-specific DTCA. In particular, when M(p) = 1 and hence M'(p) = 0 (no market-size distortion), an increase in p is equivalent to a decrease in c, thereby allowing us to directly apply the results obtained in the previous section. The reason for this is clear if we look at the firms' objective function: if M(p) = 1, the problem under product-specific DTCA can be normalized by p, so that only the price-cost ratio matters for the equilibrium allocation.

When M(p) < 1, on the other hand, there is an additional effect on the market-size distortion, and the problem can no longer be normalized by the price. Since $M'(p) = -\lambda < 0$, this makes the effect of an increase in p somewhat weaker. We can still show, however, that the overall effect is positive under some mild conditions. To this end, it suffices to show that $4M(p)c > 2\lambda p(2c-p)$ for all $c \in [0.5p, \infty)$. This condition clearly holds if c is sufficiently small and close to 0.5p. As $c \to \infty$, this becomes

$$M(p) \ge \lambda p \iff \frac{v+d}{2\lambda} \ge p,$$

which generally holds because $\frac{\nu+d}{2\lambda}$ is the upperbound of the equilibrium price. This argument suggests that the equilibrium advertisement expenditure is generally increasing in p.

Given this result, we now turn to the equilibrium firm profit. Taking partial derivative with respect to p, we obtain

$$\frac{\partial \pi^P}{\partial p} = \frac{\theta^P}{2} [(2M'(p)p + 2M(p))(1 - \theta^P) + \theta^P] + [M(p)p(1 - 2\theta^P) + (p - c)\theta^P] \frac{\partial \theta^P}{\partial p}.$$
 (6)

The first term of the right-hand side is the direct effect of a price increase which is always positive. An increase in p also induces an indirect effect via an increase in θ^p . Since the firms generally overinvest, this indirect effect is always negative as any further increase in the investment level from its equilibrium level can only lower the firm profit, given the price. Despite this apparent tradeoff, we can show that the direct effect dominates, so that an increase in the price level generally benefits the firms (see Proposition 8 below).

The situation is more complicated for the patient surplus. Taking partial derivative with respect to p, we obtain

$$\frac{\partial PS^{P}}{\partial p} = \theta^{P} \left[\theta^{P} \frac{\partial PS_{S}}{\partial p} + 2(1 - \theta^{P}) \frac{\partial PS_{C}}{\partial p} \right] + \left[2\theta^{P} (PS_{S} - PS_{C}) + 2(1 - \theta^{P}) PS_{C} \right] \frac{\partial \theta^{P}}{\partial p}, \quad (7)$$

where

$$\frac{\partial PS_S}{\partial p} = -\lambda p, \ \frac{\partial PS_C}{\partial p} = -\lambda [M(p) + M'(p)p] - \tau M(p)M'(p).$$

Again, the first term captures the direct effect which is now negative. The second term is the indirect effect which stems from an increase in θ^P . In contrast to the equilibrium profit, the indirect effect is positive because more investment is always beneficial for patients. The effect of a price increase on the patient surplus is more ambiguous due to this tradeoff but is still positive when the price is relatively low to begin with.

Proposition 8. (i) π^P is increasing in p for all $p \in (0, p_C]$. (ii) PS^P is increasing in p if p is sufficiently small.

The first part of the proposition implies that the two forms of regulation are complementary in that the benefit of a less stringent advertisement regulation, i.e., product-specific DTCA, is amplified when it is combined with a less stringent price regulation. As such, in the region where $\Delta PS > 0$, product-specific DTCA and concurrent price liberalization are the right policy mix for a pro-business authority. This is, however, not necessarily true for the welfare of patients as they suffer directly from a higher equilibrium price. Still, when the price level is sufficiently low to begin with, this negative effect is more than offset by an increase in the investment level.¹⁷ The intuition behind this result is that at a price sufficiently close to zero, the firms make almost no investment, and there are hence no informed patients who directly suffer from a price increase. Although more patients become informed and are forced to incur more cost as the price increases, this effect is of second order and is dominated by the indirect effect which is of first order.

¹⁷The same reasoning suggests that the patient surplus is increasing in p if c is sufficiently large.

6. Conclusion

This paper investigates the effect of regulating the content of DTCA in a pharmaceutical market with emphasis on the distinction between product-specific and category-specific DTCA. Owing to a unique information structure of the market, the two different forms of DTCA generate different incentives to the pharmaceutical firms and induce different responses from potential patients. We argue that although product-specific DTCA may distort prescription decisions, this welfare loss is partly offset by stronger incentives to invest in advertisement, which subsequently reduce the fraction of uniformed patients.

The overall welfare impact is ambiguous and depends, among other things, on the cost effectiveness of advertisement and the market-size distortion. When the cost of advertisement is relatively high, the interests of the pharmaceutical firms and potential patients are better aligned, and there often exists a Pareto-improving policy choice: category-specific DTCA is preferred when the market-size distortion is more severe while product-specific DTCA is preferred when it is less so. As the cost of advertisement decreases, however, a conflict emerges between the firms and patients. In general, product-specific DTCA emerges as the preferred choice for patients as the prescription distortion is more attenuated while serving the educational purpose, but the firms are made worse off due to the overinvestment problem.

Throughout the analysis, we deliberately restrict our attention to the policy issue of product-specific versus category-specific. In reality, however, other policy instruments can often be utilized in conjunction with the content regulation. Pricing regulation, which we discussed in section 5, is a case in point. Another possible instrument is the cost of advertisement, which may be controlled via tax/subsidy schemes or pre-clearance procedures of advertisement. Our analysis suggests that the impact of these policy interventions should not be evaluated in isolation, because a change in underlying parameters may also affect the optimal form of content regulation. We argue that this gives rise to an additional policy dimension to consider and hope to see more work along this line in future.

References

AMALDOSS, W. AND C. HE (2009): "Direct-to-Consumer Advertising of Prescription Drugs: A Strategic Analysis," *Marketing Science*, 28, 472–487.

AMIR, R., D. MACHOWSKA, AND M. TROEGE (2021): "Advertising Patterns in a Dynamic

- Oligopolistic Growing Market with Decay," *Journal of Economic Dynamics and Control*, 131, 104229.
- BALA, R. AND P. BHARDWAJ (2010): "Detailing vs. Direct-to-Consumer Advertising in the Prescription Pharmaceutical Industry," *Management Science*, 56, 148–160.
- Brekke, K. R. and M. Kuhn (2006): "Direct to Consumer Advertising in Pharmaceutical Markets," *Journal of Health Economics*, 25, 102–130.
- DAI, C., R. S. STAFFORD, AND G. C. ALEXANDER (2005): "National Trends in Cyclooxygenase-2 Inhibitor Use Since Market Release: Nonselective Diffusion of a Selectively Cost-Effective Innovation," *Archives of Internal Medicine*, 165, 171–177.
- DAVE, D. AND H. SAFFER (2012): "Impact of Direct-to-Consumer Advertising on Pharmaceutical Prices and Demand," *Southern Economic Journal*, 79, 97–126.
- FROSCH, D. L., D. GRANDE, D. M. TARN, AND R. L. KRAVITZ (2010): "A Decade of Controversy: Balancing Policy With Evidence in the Regulation of Prescription Drug Advertising," *American Journal of Public Health*, 100, 24–32.
- GROSSMAN, G. M. AND C. SHAPIRO (1984): "Informative Advertising with Differentiated Products," *The Review of Economic Studies*, 51, 63–81.
- HUSKAMP, H. A., J. M. DONOHUE, C. KOSS, E. R. BERNDT, AND R. G. FRANK (2008): "Generic Entry, Reformulations and Promotion of SSRIs in the US," *PharmacoEconomics*, 26, 603–616.
- JACOB, N. T. (2018): "Drug Promotion Practices: A Review," *British Journal of Clinical Pharmacology*, 84, 1659–1667.
- JØRGENSEN, S. AND S.-P. SIGUÉ (2015): "Defensive, Offensive, and Generic Advertising in a Lanchester Model with Market Growth," *Dynamic Games and Applications*, 5, 523–539.
- KALYANARAM, G. (2008): "The Order of Entry Effect in Prescription (RX) and Over-The-Counter (OTC) Pharmaceutical Drugs," *International Journal of Pharmaceutical and Healthcare Marketing*, 2, 35–46.
- ——— (2009): "The Endogenous Modeling of the Effect of Direct-to-Consumer Advertising in Prescription Drugs," *International Journal of Pharmaceutical and Healthcare Marketing*, 3, 137–148.
- KRAVITZ, R. L., R. M. EPSTEIN, M. D. FELDMAN, C. E. FRANZ, R. AZARI, M. S. WILKES, L. HINTON, AND P. FRANKS (2005): "Influence of Patients' Requests for Direct-to-Consumer Ad-

- vertised Antidepressants: A Randomized Controlled Trial," *Journal of American Medical Association*, 293, 1995–2002.
- KRISHNAMURTHY, S. (2000): "Enlarging the Pie vs. Increasing One's Slice: An Analysis of the Relationship Between Generic and Brand Advertising," *Marketing Letters*, 11, 37–48.
- LIU, Q. AND S. GUPTA (2011): "The Impact of Direct-to-Consumer Advertising of Prescription Drugs on Physician Visits and Drug Requests: Empirical Findings and Public Policy Implications," *International Journal of Research in Marketing*, 28, 205–217.
- MURRAY, E., B. LO, L. POLLACK, K. DONELAN, AND K. LEE (2003): "Direct-to-Consumer Advertising: Physicians' Views of Its Effects on Quality of Care and the Doctor-Patient Relationship," *Journal of American Board of Family Medicine*, 16, 513–524.
- PAREKH, N. AND W. H. SHRANK (2018): "Dangers and Opportunities of Direct-to-Consumer Advertising," *Journal of General Internal Medicine*, 33, 586–587.
- ROBINSON, A. R., K. B. HOHMANN, J. I. RIFKIN, D. TOPP, C. M. GILROY, J. A. PICKARD, AND R. J. ANDERSON (2004): "Direct-to-Consumer Pharmaceutical Advertising: Physician and Public Opinion and Potential Effects on the Physician-Patient Relationship," *Archives of Internal Medicine*, 164, 427–432.
- ROSENTHAL, M. B., E. R. BERNDT, J. M. DONOHUE, R. G. FRANK, AND A. M. EPSTEIN (2002): "Promotion of Prescription Drugs to Consumers," *New England Journal of Medicine*, 346, 498–505.
- SANTARSIERI, D. AND T. L. SCHWARTZ (2015): "Antidepressant Efficacy and Side-Effect Burden: A Quick Guide for Clinicians," *Drugs in Context*, 4, 1–12.
- SHAPIRO, B. (2020): "Promoting Wellness or Waste? Evidence from Antidepressant Advertising," Becker Friedman Institute for Research in Economics Working Paper.
- SHIN, J. AND J. YU (2021): "Targeted Advertising and Consumer Inference," *Marketing Science*, 40, 900–922.
- SINKINSON, M. AND A. STARC (2018): "Ask Your Doctor? Direct-to-Consumer Advertising of Pharmaceuticals," *The Review of Economic Studies*, 86, 836–881.
- SUFRIN, C. B. AND J. S. ROSS (2008): "Pharmaceutical Industry Marketing: Understanding Its Impact on Women's Health," *Obstetrical and Gynecological Survey*, 63, 585–596.
- VENTOLA, C. L. (2011): "Direct-to-Consumer Pharmaceutical Advertisement: Therapeutic or Toxic?" *Pharmacy and Therapeutics*, 36, 669–684.

WEISSMAN, J. S., D. BLUMENTHAL, A. J. SILK, M. NEWMAN, K. ZAPERT, R. LEITMAN, AND S. FEIBELMANN (2004): "Physicians Report On Patient Encounters Involving Direct-To-Consumer Advertising," *Health Affairs*, 23, 219–233.

Appendix: Proof

Proof of Proposition 1. We have already seen that $\theta^P > \theta^E$ always holds. Note also that

$$\frac{\partial \theta^{P}}{\partial c} - \frac{\partial \theta^{E}}{\partial c} = -\frac{4Mp}{[2c + p(2M - 1)]^{2}} + \frac{2p}{(2c + p)^{2}}.$$

This is negative if

$$\frac{4Mp}{[2c+p(2M-1)]^2} > \frac{2p}{(2c+p)^2},$$

which can be written as

$$2M(2c+p)^2 > [2c+p(2M-1)]^2.$$

With some computation, this is reduced to

$$4c^{2}(2M-1)+4cp+p^{2}[2M-(2M-1)^{2}]>0$$

which holds for any $M \in (0.5, 1]$.

Proof of Proposition 2. Define θ^k and Q^k , k = P, E, as functions of v, p, d and c. The effects of v and d are identical. Note that Q^P is increasing in v if

$$2c + p(2M - 1) > 2Mp$$

which for any c > 0.5p. This suggests that ΔQ is increasing in ν and decreasing in d.

With respect to a change in c, observe that $\Delta Q = \frac{1}{8} > 0$ at c = 0.5p and $\lim_{c \to \infty} \Delta Q = 0$. It is also straightforward to obtain

$$\frac{\partial Q^{P}}{\partial c} = \left[\theta^{P} + (1 - 2\theta^{P})M\right] \frac{\partial \theta^{P}}{\partial c} = -\frac{4M^{2}p[2c - p(2M - 1)]}{[2c + p(2M - 1)]^{3}},$$

$$\frac{\partial Q^{E}}{\partial c} = (1 - \theta^{E}) \frac{\partial \theta^{E}}{\partial c} = -\frac{4cp}{(2c + p)^{3}}.$$

It follows from this that ΔQ is decreasing in c if

$$\frac{M^2[2c - p(2M - 1)]}{[2c + p(2M - 1)]^3} > \frac{c}{(2c + p)^3}.$$

Alternatively, the condition can be written as

$$F(c)G(c)^3 > \frac{1}{M^2},$$

where

$$F(c) := \frac{2c - p(2M - 1)}{c}, \ G(c) := \frac{2c + p}{2c + p(2M - 1)}.$$

Evaluated at c = 0.5p, the condition is reduced to

$$F(0.5p)G(0.5p)^3 = \frac{4(1-M)}{M^3} > \frac{1}{M^2} \iff \frac{4}{5} > M.$$

As $c \to \infty$, on the other hand, we have

$$\lim_{c \to \infty} F(c)G(c)^{3} = 2 > \frac{1}{M^{2}} \iff M > \sqrt{0.5}.$$

Note also that FG^3 is increasing in c if

$$F'G + 3FG' > 0, (8)$$

where

$$F'(c) = \frac{p(2M-1)}{c^2}, \ G'(c) = -\frac{4p(1-M)}{[2c+p(2M-1)]^2}.$$

It then follows that (10) can be written as

$$\frac{p(2M-1)(2c+p)}{c^2\lceil 2c+p(2M-1)\rceil} > \frac{12p(1-M)[2c-p(2M-1)]}{c\lceil 2c+p(2M-1)\rceil^2},$$

which is simplified to

$$\frac{p(2M-1)(2c+p)}{c} > \frac{12p(1-M)[2c-p(2M-1)]}{2c+p(2M-1)}.$$

Note that the left-hand side is strictly decreasing while the right-hand side is strictly increasing. This means that FG^3 may increase at the beginning but must eventually decrease as c gets sufficiently large. Combined with the fact that FG^3 must converge to 2 regardless of M, this fact allows us to pin down the shape of ΔQ . There are three possible cases we need to investigate.

Case 1 ($\sqrt{0.5} > M$): In this case, $FG^3 > M^{-2}$ when c is relatively small. At some point, FG^3 starts decreasing and converges to 2 which is lower than M^{-2} . This means that ΔQ first

decreases and then increases, ultimately converging to 0. As such, there exists a unique threshold $c_Q(v,d)$ such that $\Delta Q > 0$ if and only if $c_Q > c$. Since ΔQ is increasing in v and d, the threshold c_Q must also be increasing in v and d.

Case 2 (0.8 $\geq M \geq \sqrt{0.5}$): In this case, FG^3 must invariably be smaller larger than M^{-2} , which implies that ΔQ is monotonically decreasing with $\Delta Q > 0$ for all c > 0.5p.

Case 3 (1 > M > 0.8): In this case, FG^3 is lower than M^{-2} when c is relatively small. As c gets larger, it increases but then decreases and converges to 2 which is larger than M^{-2} . This means that ΔQ first increases and then decreases with ΔQ > 0 for all c > 0.5p.

Proof of Proposition 4. Since

$$\frac{\partial \Delta C}{\partial c} = 2\theta^{P} \frac{\partial \theta^{P}}{\partial c} - 2\theta^{E} \frac{\partial \theta^{E}}{\partial c},$$

we have

$$\frac{\partial \Delta \pi}{\partial c} = p[M - (2M - 1)\theta^{P}] \frac{\partial \theta^{P}}{\partial c} - p(1 - \theta^{E}) \frac{\partial \theta^{E}}{\partial c} - c\theta^{P} \frac{\partial \theta^{P}}{\partial c} + c\theta^{E} \frac{\partial \theta^{E}}{\partial c} - \frac{\Delta C}{2}$$
$$= (c\theta^{P} - Mp) \frac{\partial \theta^{P}}{\partial c} - c\theta^{E} \frac{\partial \theta^{E}}{\partial c} - \frac{\theta^{P^{2}} - \theta^{E^{2}}}{2}.$$

Observe that

$$\frac{\theta^{P^2}}{2} = -\frac{\partial \theta^P}{\partial c} \frac{Mp}{2}, \quad \frac{\theta^{E^2}}{2} = -\frac{\partial \theta^E}{\partial c} \frac{p}{4},$$

which gives us

$$\begin{split} \frac{\partial \Delta \pi}{\partial c} &= \left(c\theta^P - \frac{Mp}{2}\right) \frac{\partial \theta^P}{\partial c} - \left(c\theta^E + \frac{p}{4}\right) \frac{\partial \theta^E}{\partial c} \\ &= \frac{Mp[2c - p(2M - 1)]}{2[2c + p(2M - 1)]} \frac{\partial \theta^P}{\partial c} - \frac{p(6c + p)}{4(2c + p)} \frac{\partial \theta^E}{\partial c} \\ &= -\frac{2(Mp)^2[2c - p(2M - 1)]}{[2c + p(2M - 1)]^3} + \frac{p^2(6c + p)}{2(2c + p)^3}. \end{split}$$

From this, $\Delta \pi$ is increasing in *c* if

$$\frac{1}{M^2} > \frac{4[2c - p(2M - 1)]}{6c + p} \left(\frac{2c + p}{2c + p(2M - 1)}\right)^3 = H(c)G(c)^3,\tag{9}$$

where

$$H(c) := \frac{4[2c - p(2M - 1)]}{6c + p}.$$

As above, HG^3 is increasing in c if H'G + 3HG' > 0. With some computation, we obtain

$$H'(c) = \frac{16p(3M-1)}{(6c+p)^2},$$

so that this condition becomes

$$\frac{16p(3M-1)(2c+p)}{(6c+p)^2[2c+p(2M-1)]} > \frac{48p(1-M)[2c-p(2M-1)]}{[2c+p(2M-1)]^2(6c+p)},$$

which is further simplified to

$$\frac{(3M-1)(2c+p)}{(6c+p)} > \frac{3(1-M)[2c-p(2M-1)]}{[2c+p(2M-1)]}.$$
 (10)

Note that the right-hand side is decreasing in *c* while the left-hand side is increasing.

Evaluated at c = 0.5p, (10) becomes

$$\frac{3M-1}{3(1-M)} > \frac{2(1-M)}{M}.$$

This holds if and only if $M > \frac{2}{3}$. In the limit as $c \to \infty$, on the other hand, (10) becomes

$$\frac{3M-1}{3(1-M)} > 3,$$

which holds if and only if $M > \frac{5}{6}$. This means that: if $M > \frac{5}{6}$, HG^3 is increasing for all $c \in [0.5p, \infty)$; if $\frac{5}{6} > M > \frac{2}{3}$, HG^3 first increases and then decreases.

To complete the proof, we now check the boundary conditions for (9). It is easy to verify that (9) holds at c=0.5p if $M>\frac{2}{3}$, so that $\Delta\pi$ increases at the beginning. At the other end, as $c\to\infty$, this holds if $\sqrt{0.75}>M$. Note also that $\sqrt{0.75}\approx 0.866>\frac{5}{6}$. This means that for $M>\sqrt{0.75}$, $\Delta\pi$ increases at first, then decreases and converges to 0, proving that there exists a threshold $c_\pi(v,d)$ such that $\Delta\pi>0$ if and only if $c>c_\pi(v,d)$. For $\sqrt{0.75}\geq M\geq \frac{5}{6}$, on the other hand, $\Delta\pi$ is increasing for all c, and hence $\Delta\pi<0$ for all c. We can also show that $\Delta\pi$ is increasing in v. Since a decrease in v is equivalent to a decrease in M with p and d fixed, this means that $\Delta\pi<0$ for all c if $\sqrt{0.75}>M$.

Finally, since $\Delta \pi$ is increasing in ν , it is clear that c_{π} is decreasing in ν . To show that $\Delta \pi$ is increasing in d, recall that the profit under product-specific DTCA is given by

$$\pi^{P} = \frac{p\theta^{P}[2M - (2M - 1)\theta^{P}]}{2} - \frac{c}{2}\theta^{P^{2}},$$

from which we obtain

$$\frac{\partial \pi^{P}}{\partial d} = 2[Mp^{P}(1 - 2\theta^{P}) + (p^{P} - c)\theta^{P}] \frac{\partial \theta^{P}}{\partial d} + p^{P}\theta^{P}(1 - \theta^{P})$$
$$= -(2M - 1)p^{P}\theta^{P} \frac{\partial \theta^{P}}{\partial d} + p^{P}\theta^{P}(1 - \theta^{P}).$$

If $\nu - \lambda p + d > \tau$ and M = 1, we have $\frac{\partial \theta^P}{\partial d} = 0$, and π^P is increasing in d. If not, we have

$$\frac{\partial \theta^{P}}{\partial d} = \frac{2p(2c-p)}{[2c+p(2M-1)]^{2}} > 0.$$

Given this, π^P is increasing in d if

$$1 - \theta^{p} > \frac{2p(2M - 1)(2c - p)}{[2c + p(2M - 1)]^{2}} \iff 2c > p(2M - 1),$$

which holds for any c > 0.5p.

Proof of Proposition 5. Observe that $\Delta PS = \frac{PS_S}{4} > 0$ at c = 0.5p and $\lim_{c \to \infty} \Delta PS = 0$. It is also straightforward to obtain

$$\begin{split} \frac{\partial PS^{P}}{\partial c} &= 2[\theta^{P}PS_{S} + (1-2\theta^{P})PS_{C}] \frac{\partial \theta^{P}}{\partial c} = -\frac{8Mp[2Mp(PS_{S} - PS_{C}) + (2c-p)PS_{C}]}{[2c+p(2M-1)]^{3}}, \\ \frac{\partial PS^{E}}{\partial c} &= 2(1-\theta^{E})PS_{S} \frac{\partial \theta^{E}}{\partial c} = -\frac{8pc}{(2c+p)^{3}}PS_{S}. \end{split}$$

It follows from this that ΔPS is decreasing in c if

$$\frac{M^2[2c\phi - p[(2M+1)\phi - 2]]}{[2c + p(2M-1)]^3} > \frac{c}{(2c+p)^3}.$$
 (11)

Here, we extend the definition of F in the proof of Proposition 2 and, with slight abuse of notation, define

$$F(c;\phi) = \frac{2c\phi - p[(2M+1)\phi - 2)}{c},$$

so that (11) is given by

$$F(c;\phi)G(c)^3 > \frac{1}{M^2}.$$

Note that F(c; 1) = F(c).

Evaluated at c = 0.5p, (11) is reduced to

$$F(0.5p;\phi)G(0.5p) = \frac{4(1-M\phi)}{M^3} > \frac{1}{M^2} \iff \frac{4}{1+4\phi} > M.$$

As $c \to \infty$, on the other hand, it converges to

$$\lim_{c \to \infty} F(c; \phi) G(c) = 2\phi > \frac{1}{M^2} \iff M > \sqrt{0.5\phi^{-1}}.$$

Moreover, as in Proposition 2, we can show that FG^3 is increasing in c if

$$\frac{p[(2M+1)\phi-2)(2c+p)}{c^2[2c+p(2M-1)]} > \frac{12p(1-M)[2c-p[(2M+1)\phi-2)]]}{c[2c+p(2M-1)]^2},$$

which is simplified to

$$\frac{p[(2M+1)\phi-2)(2c+p)}{c} > \frac{12p(1-M)[2c-p[(2M+1)\phi-2)]]}{2c+p(2M-1)},$$

If $(2M+1)\phi \leq 2$, FG^3 is weakly decreasing in c. If $(2M+1)\phi > 2$, we can essentially follow the same argument as in Proposition 2. In either case, we can conclude that: (i) if $M \geq \sqrt{0.5\phi^{-1}}$, $\Delta PS > 0$ for all $c \in (0.5p, \infty)$; (ii) if $\sqrt{0.5\phi^{-1}} > M$, there exists an interior threshold c_{PS} such that $\Delta PS > 0$ if and only if $c_{PS} > c$.

Finally, it is easy to verify that PS^P is increasing in ν while PS^E is independent of it. That c_{PS} is increasing in ν immediately follows from this fact.

Proof of Proposition 6. Observe that PS^P is increasing in d if

$$[\theta^{P} P S_{S} + (1 - 2\theta^{P}) P S_{C}] \frac{\partial \theta^{P}}{\partial d} > \theta^{P} (1 - \theta^{P}) \frac{d}{\tau}.$$

Note that if $v - \lambda p + d < \tau$,

$$\frac{\partial \theta^{P}}{\partial d} = \frac{2p(2c-p)}{[2c+p(2M-1)]^{2}}, \ \theta^{P}(1-\theta^{P}) = \frac{2Mp(2c-p)}{[2c+p(2M-1)]^{2}} = M\frac{\partial \theta^{P}}{\partial d}.$$

The condition can thus be written as

$$\theta^P P S_S + (1 - 2\theta^P) P S_C > \frac{Md}{\tau},$$

which converges to

$$PS_C = \frac{M(v - \lambda p - d)}{2\tau} > \frac{Md}{\tau} \iff v - \lambda p > 3d,$$

as c tends to infinity.

Proof of Proposition 7. Define

$$S(p) := \frac{2M(p)p}{2c + p[2M(p) - 1]} = \frac{2(\nu + d)p - 2\lambda p^2}{2c - p[2(\nu + d) - 1] - 2\lambda p^2}, \ P(\theta) := \frac{\theta p_S + 4\tau(1 - \theta)p_C}{\theta + 4\tau(1 - \theta)}.$$

It is clear that S is upward sloping in p if M(p) = 1. If M(p) < 1, we have

$$S(p) = \frac{2(\nu + d)p - 2\lambda p^2}{2c - p[2(\nu + d) - 1] - 2\lambda p^2}.$$

Given this, *S* is upward sloping in *p* if

$$S'(p) = \frac{2(\nu + d - 2\lambda p)(2c - p) + 2(\nu + d - \lambda p)p}{\left[2c - p\left[2(\nu + d) - 1\right] - 2\lambda p^2\right]^2} > 0.$$

This condition can be written as

$$(v+d-2\lambda p)(2c-p)+(v+d-\lambda p)p>0,$$

which holds for any $p \in [0, p_C]$. Similarly, P is downward sloping in θ if

$$P'(\theta) = \frac{(p_S - 4\tau p_C)[\theta + 4\tau(1 - \theta)] - (1 - 4\tau)[\theta p_S + 4\tau(1 - \theta)p_C]}{[\theta + 4\tau(1 - \theta)]^2}$$
$$= -\frac{4\tau(p_C - p_S)}{[\theta + 4\tau(1 - \theta)]^2} < 0,$$

which clearly holds. This shows that there is always a unique pair that satisfies (5).

It is also clear that S shifts downward as c increases while P is not affected. This means that θ^P is decreasing in c while p^P is increasing. Moreover, it is easy to verify that $\theta^P \to 1$ and $p^P = p_S$ as $c \to 0.5p_S$ and $\theta^P \to 0$ and $p^P = p_C$ as $c \to \infty$.

Proof of Proposition 8. By rearranging (6), we obtain

$$\frac{\partial \pi^{P}}{\partial p} = \frac{\theta^{P}}{2} \left[(2M'p + 2M)(1 - \theta^{P}) + \theta^{P} - 2Mp \frac{\partial \theta^{P}}{\partial p} \right],$$

where $M' = -\lambda$ if $v - \lambda p + d < 1$ and M' = 0 if $v - \lambda p + d \ge 1$. Since

$$\frac{\partial \theta^P}{\partial p} = \frac{4Mc + 2M'p(2c - p)}{[2c + p(2M - 1)]^2},$$

 π^P is increasing in p if

$$2M'p\left[1-\theta^{P}-\frac{2Mp(2c-p)}{[2c+p(2M-1)]^{2}}\right]+2M(1-\theta^{P})+\theta^{P}-\frac{8M^{2}pc}{[2c+p(2M-1)]^{2}}>0.$$

Substituting θ^P yields

$$2M'p\left[\frac{2c-p}{2c+p(2M-1)} - \frac{2Mp(2c-p)}{[2c+p(2M-1)]^2}\right] + \frac{4Mc}{2c+p(2M-1)} - \frac{8M^2pc}{[2c+p(2M-1)]^2} > 0,$$

which is simplified to

$$M'p(2c-p) + 2Mc > 0.$$

When M = 1, M' = 0 and this condition clearly holds. When M < 1, we have

$$2Mc > \lambda p(2c - p). \tag{12}$$

This condition holds at p = 0. Note also that the right-hand side is decreasing in p at first and then becomes increasing while the left-hand side is strictly decreasing. This means that (12) holds for all $p \in (0, p_C]$ if it holds at $p = p_C$. This is the case if

$$2\left(\nu - \lambda \frac{\nu + d}{2\lambda} + d\right)c = (\nu + d)c > \frac{\nu + d}{2}\left(2c - \frac{\nu + d}{2\lambda}\right),$$

which holds for any c.

Similarly, it follows from (7) that PS^P is increasing in p if

$$[2\theta^{P}(PS_{S}-PS_{C})+2(1-\theta^{P})PS_{C}]\frac{\partial\theta^{P}}{\partial\rho}>\theta^{P}[\lambda p\theta^{P}+2(1-\theta^{P})[\lambda(M+M'p)+\tau MM']],$$

where

$$PS_S - PS_C = (v - \lambda p)(1 - M) + \frac{\tau}{4}(2M^2 - 1).$$

With some computation, we obtain

$$\begin{split} & \big[2Mp(PS_S - PS_C) + (2c - p)PS_C \big] \frac{2Mc + M'p(2c - p)}{2c + p(2M - 1)} \\ & > Mp \big[\lambda Mp^2 + (2c - p)[\lambda(M + M'p) + \tau MM'] \big], \end{split}$$

As $p \to 0$, this condition is reduced to

$$\frac{4Mc^2PS_C}{2c + p(2M - 1)} > 0,$$

which always holds. ■

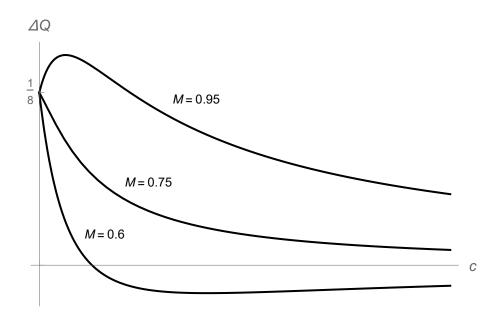


Figure 1. The difference in market demand ΔQ (p = 0.6)

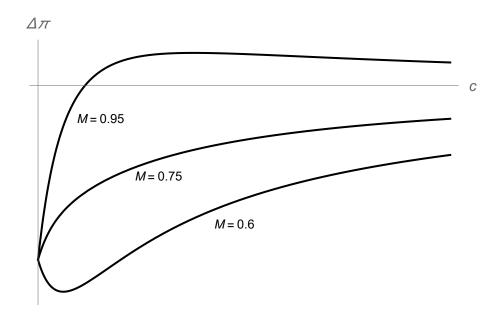


Figure 2. The difference in firm profit $\Delta\pi$ (p = 0.6)

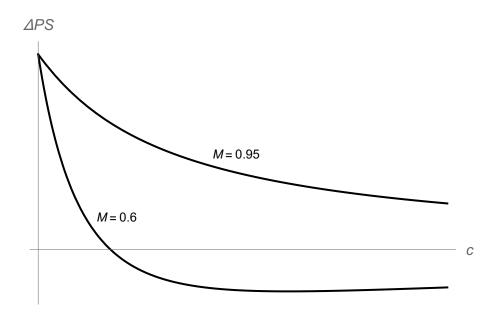


Figure 3. The difference in patient surplus ΔPS ($\nu=1.2,\ p=0.6,\ \tau=\lambda=1$)

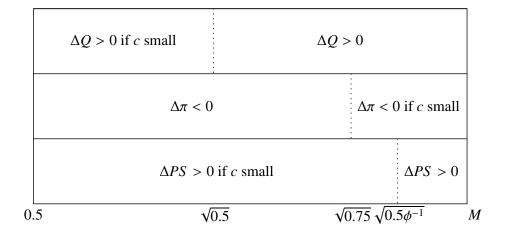


Figure 4. The welfare effects of the content regulation: summary