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Abstract

This paper examines the role of both cost-sharing schemes in health insurance systems and entry regulation for pharmaceutical R&D expenditure, drug prices, aggregate productivity, and income. The analysis suggests that both an increase in the coinsurance rate and stricter price regulations adversely affect R&D spending in the pharmaceutical sector. In contrast, entry deregulation may lead to quality-improvements of pharmaceuticals, despite reducing price-setting power of pharmaceutical companies. Extension to an endogenous growth context suggests that, when individual labor supply depends on health status, both cost-sharing and entry barriers in the pharmaceutical sector also affect aggregate productivity and wage rates.

JEL-Code: I100, L100, O300.

Keywords: aggregate productivity, cost-sharing, entry deregulation, health insurance, pharmaceutical innovation.

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1 Introduction

Dramatically rising health expenditure costs in the last decades, in particular for prescription pharmaceuticals, have triggered ongoing debates about cost-sharing between health insurers and beneficiaries.[1] For instance, in the US, a reform of Medicare (a federal program which provides health insurance for the elderly) which went into effect in 2006 (Medicare Part D) introduced coverage of prescription drug expenditure for Medicare beneficiaries. There is, however, a coinsurance rate (the fraction of expenditure on medical services paid by the insured patient) of 25 percent.[2]

It is typically argued that, compared to full coverage, cost-sharing schemes limit the growth of health insurance premiums. There is a large empirical literature on the effects of prescription drug cost-sharing on health costs and health care utilization. Empirical estimates suggest that a 10 percent increase in patients' prescription drug charge (through higher coinsurance or higher copayment) reduces prescription drug spending by 1 to 6 percent.[3]

In contrast to such short-run demand effects of prescription drugs cost-sharing, long-run supply effects on pharmaceutical innovation are underresearched. Generally, a major concern in designing health insurance systems and regulating the pharmaceutical

[1] In the EU, the average annual real growth rate of spending for pharmaceuticals was 4.7 percent (3.8 percent in Germany) between 1998 and 2008 (OECD, 2010). In the US, there was a more than fivefold increase in spending for prescription drugs between 1990 and 2006 from 40.3 to 216.7 billion USD (see "The Kaiser Family Foundation. Prescription drug trends", available at http://www.kff.org/rxdrugs/3057-03.cfm).

[2] The rate applies after some deductible, up to an initial coverage limit. After a "catastrophic" coverage limit is reached, the coinsurance rate drops to 5 percent. In Switzerland basically all health insurance contracts have a coinsurance rate of 20 percent for branded prescription drugs and 10 percent for generic drugs.

[3] Goldman, Joyce and Zheng (2007) and Gemmill, Thomson and Mossialos (2008) provide meta-studies on this expenditure elasticity. Motheral and Henderson (1999) find that demand effects are prevalent with respect to branded drugs only. Landsman et al. (2005) suggests as well that demand for prescription drugs is quite inelastic. The most convincing evidence on demand effects of cost-sharing schemes comes from the Rand Health Insurance Experiment. Based on data from this randomized experiment, Leibowitz (1985) finds that per capita prescriptions were 25 percent higher for patients with zero coinsurance than for patients who faced a 50 percent coinsurance rate, and 50 percent higher than for those who faced a 95 percent coinsurance rate. Possibly as a consequence of reduced utilization of pharmaceuticals, some studies find that increased cost-sharing results in greater use of inpatient and emergency medical services of patients with chronic diseases like congestive heart failure, lipid disorders, diabetes and schizophrenia but had little effect on health care utilization of patients with non-chronic diseases (e.g., Austvoll-Dahlgren et al., 2008).

sector is the tension between keeping prices low and ensuring that quality is high. The main issue therefore is the joint impact of cost-sharing schemes on price-setting behavior and the incentives of pharmaceutical companies to conduct R&D. As pointed out by Berndt (2002, p.45): "The resolution of this static versus dynamic efficiency conflict is likely the single most important issue facing the pharmaceutical industry".

This paper attempts to shed light on the nature of the price-quality relationship in pharmaceutical markets. It examines the role of cost-sharing and price regulations in health insurance systems for both pharmaceutical R&D and drug prices. We also explore the effects of entry deregulation, which may be seen as an attempt to lower prices of pharmaceuticals. The main issue again is whether price reductions come at the costs of less quality-improvements of pharmaceuticals.

The proposed theoretical model builds on the "ideal variety" framework, originated by Lancaster (1979). Although the framework has never been applied in the context of pharmaceutical markets and vertical R&D (to the best of my knowledge),[4] it captures well the notion that patients seek the ideal drug for their type of illness.[5] The horizontal location of a pharmaceutical firm is interpreted as the type of illness to which the drug that the firm produces is targeted to, represented as a point on the circumference of a circle. That is, pharmaceuticals are imperfect substitutes to each other.[6] Firms choose their horizontal location along with prices and R&D spending.

We show that introducing insurance coverage of prescription drug expenditure (like Medicare Part D) raises both drug prices and pharmaceutical R&D spending, whereas an increase in the coinsurance rate within an existing cost-sharing scheme has the opposite effect. Intuitively, a lower coinsurance rate makes demand for pharmaceuticals less price-sensitive and therefore allows firms to charge higher price-cost margins. This,

[4]The ideal variety model is sometimes used in the international trade literature (e.g. Helpman, 1981; Wong, 1995; Hummels and Lugovskyy, 2009).

[5]Besides realism in this respect, the ideal variety framework also has the attractive feature that the price elasticity of demand depends on the competitive environment of firms. Notably the standard version of the alternative (and far more often applied) "love of variety" model of monopolistic competition by Dixit and Stiglitz (1977) and Ethier (1982) predicts that the price elasticity of demand for a good − and thus the price mark-up − is constant. However, the empirical support for this prediction is generally weak. Under a constant price elasticity, the health insurance system could not have any effect on prices for pharmaceuticals.

[6]Examples are pain killers, antibiotics, hypertension medication, and pharmaceutical cancer therapy.

in turn, boosts the return to R&D.

By contrast, deregulation of entry may induce higher R&D spending in the pharmaceutical sector despite reducing price-setting power. The result suggests that the repeated claim by pharma lobbyists − that anything which raises drug prices and profits in the pharmaceutical sector would be conducive to R&D − is potentially erroneous. Competition policy may rather be seen as a tool to raise the quality of pharmaceuticals and limit drug expenditure at the same time. Policy measures may include encouraging entry of foreign firms, restricting marketing practices which effectively work as entry barriers, and reducing patent breadth. In fact, patent breadth has a natural representation in the proposed model, as a segment on the circumference of the circle of illnesses which includes the point targeted by a pharmaceutical firm; patent protection means that potential rivals are prohibited to locate on this segment.

We also examine the role of two kinds of price regulations for pharmaceuticals. First, as practiced in France and Italy, prices may directly be set by the government. We focus on the simple case where such price controls ignore R&D costs and show that stricter direct price regulation unambiguously reduces R&D expenditure. Second, we study the effects of a price cap − a limit amount of a patients' expenses for a drug which is reimbursed by an insurer. Such cost-sharing device is common in the public health insurance system of Germany and Japan. We show that a stricter price cap reduces both R&D spending on pharmaceuticals and drug prices. The results on the R&D expenditure effects of price regulations are consistent with a large body of empirical evidence (e.g., Scherer, 1993; Vernon, 2005; Giaccotto, Santerre and Vernon, 2005).

Finally, whereas (wage) income is exogenous in the basic model, in an extension we discuss the interplay between health status and income in a dynamic general equilibrium context with pharmaceutical R&D and endogenous innovation also outside the pharmaceutical sector. The analysis accounts for the potential dependency of effective labor supply on health status. On the one hand, higher wages boost demand for pharmaceuticals and therefore enhances R&D incentives of pharmaceutical companies. On the other hand, a better health status raises aggregate productivity and wages. We

analyze how health policy and increased entry into the pharmaceutical sector affects the interaction between health status and economic well-being.

The paper is organized as follows. Section 2 discusses the relation of our analysis to the literature. Section 3 sets up the basic model with a focus on coinsurance policy. Section 4 analyzes the equilibrium of the basic model by distinguishing the case of restricted entry where the number of pharmaceutical firms is given from the one with an endogenous number of firms. Section 5 examines the effects of price regulations for pharmaceuticals. Section 6 closes the model by endogenizing wages and allowing for R&D activity outside the pharmaceutical sector in a simple endogenous growth framework. The last section concludes.

2 Related Literature

There are only few studies on the relationship between health policy and innovation incentives of pharmaceutical firms. At the theoretical level, Garber, Jones and Romer (2006) analyze the case of a single-product monopoly firm which sells a pharmaceutical product. The drug is assumed to have heterogenous effects on the utility of ill consumers. The authors show that a coinsurance rate which ensures efficient drug utilization implies that profits of the monopoly firm may exceed consumer surplus; thus, R&D incentives may be excessive. Lakdawella and Sood (2005) analyze a similar framework and argue that a health insurance contract which sets copayment at marginal costs and where innovators are paid an ex-ante fee equal to consumer surplus may at the same time achieve two goals: it may lead to efficient drug utilization and provide efficient incentives for introducing the drug into the market. More recently, Lakdawella and Sood (2009) argue that a public health insurance system with some price-negotiation by the government is welfare-improving, particularly when coupled with an increase in patent length.

The framework proposed in this paper is different to this literature in several respects. First, it captures both horizontal and vertical differentiation of pharmaceuticals. Second, it analyzes product market competition among pharmaceutical com-

panies rather than a monopoly firm. While monopoly situations may exist in some pharmaceutical markets, the exclusive focus on these situations may be considered as a shortcoming for many other markets like those for cancer medication, hypertension medication, pain killers, and antibiotics. In such markets there is some substitutability within product groups and pharmaceutical companies engage in price competition. Third, and related, the main contribution of this paper may be to provide a unified framework which allows us to develop a differentiated view on the price-quality relationship in pharmaceutical markets by encompassing both health insurance and competition policy. The salient feature to analyze competition policy is to depart from the standard monopoly assumption. Fourth, we also provide a general equilibrium perspective which allows us to explore the link between health and income through endogenous vertical innovations within and outside the pharmaceutical sector. Finally, the focus is on a positive rather than a normative analysis.

At the empirical level, Acemoglu, Cutler, Finkelstein and Linn (2006) examine whether the first Medicare program (the "Social Security Act of 1965") had an impact on pharmaceutical innovation. They find no evidence that drug spending of the elderly (aged 65-74) relative to that of the non-elderly (55-64) went up. Similarly, there was no significant effect on the number of new molecular entity approvals, as drug spending was not covered by Medicare before 2006. Our theoretical analysis predicts that the 2006 Medicare reform spurs pharmaceutical innovation.

Importantly, the present paper contributes to the debate on the relationship between entry regulation and innovation in the pharmaceutical industry. Extending the standard ideal variety framework to a context with pharmaceutical R&D delivers predictions which are consistent with evidence that a higher intensity of competition may spur innovation (e.g., Blundell, Griffith and Van Reenen, 1999; Aghion et al., 2009). That competition may be positively linked to R&D has also been pointed out by Schumpeterian growth theory (surveyed by Aghion and Howitt, 2005, 2009), challenging predictions of standard models of endogenous technical change. In Schumpeterian growth theory, however, the possibility that competition fosters innovation rests on the feature that firms can preserve a monopoly by innovating and their incentives to

search for a superior technology rises when the entry threat is enlarged. In our theory, heterogeneity and prospect of monopoly is not needed to obtain the result that entry deregulation spurs innovative effort. Moreover, we distinguish health − and demand for pharmaceuticals which is affected by the health system − from "regular" consumption goods.

Finally, Acemoglu and Johnson (2007) and Aghion, Howitt and Murtin (2010) examine the causal effect of higher life expectancy on per capita income growth. They construct a country-varying instrument for life expectancy by exploiting country differences in the date when global medical innovations (like antibiotics) where introduced. Whereas Acemoglu and Johnson (2007) find no effect, Aghion et al. (2010) find a positive impact. There are also interesting theoretical papers on endogenous life expectancy and economic growth. Van Zona and Muysken (2001) propose a Lucas-type growth model, extended to include the production of health services and longevity, which is capable to explain productivity slowdowns by low productivity of the health-sector. Sanso and Aísa (2006) show that the long-run growth rate of the economy critically depends on the rate at which the efficacy of the resources devoted to health decreases with biological age. These papers do not consider policy issues, however. The present paper does not explicitly model life expectancy but shows how health policy and entry deregulation jointly affects the quality of pharmaceuticals and aggregate productivity in a simple endogenous growth framework.

3 The Basic Model

There is a unit mass of individuals, indexed by j. Individuals draw utility $U(j)$ from consumption of a homogenous (numeraire) good, $C(j)$, and their health status, $H(j)$, according to utility function

$$U(j) = u(C(j), H(j)), \tag{1}$$

with partial derivatives $u_C > 0$, $u_H > 0$, $u_{CC} < 0$, $u_{HH} \leq 0$, and $u_{CH} \geq 0$ (i.e., the marginal utility from consumption is non-decreasing in the health level).

An individual becomes ill with probability s. Illness has two consequences. First, whereas the health level without illness is normalized to unity, it drops below one when ill; health can be improved by consuming a pharmaceutical. Second, we allow labor supply to positively depend on health status.[7] Formally, an individual with health level H inelastically supplies $g(H)$ units of labor, with $g' \geq 0$, $g'' \leq 0$, and $g(1) = 1$; that is, labor supply is unity if an individual stays healthy. The wage rate per unit of labor, w, is exogenous in the basic model and will be endogenized in section 6.

There are n pharmaceutical firms, indexed by i. Each firm produces one drug with identical technology in a monopolistically competitive environment. Firms cannot engage in price-discrimination. Marginal production costs are constant and denoted by c; that is, to produce one unit of any pharmaceutical product requires c units of the numeraire.

We distinguish the case where the number of firms n is exogenous (restricted entry) and the case where pharmaceutical companies can enter the market by incurring $f > 0$ units of the numeraire (i.e., n is endogenous). For simplicity, suppose that in the case where entry is restricted and firms earn positive profits, profits accrue to investors outside the economy.

Pharmaceuticals differ in one horizontal dimension of attributes. Each variety is targeted to a certain type of illness. Illnesses are represented by points on the circumference of a circle with unit length. Ill individuals are characterized by their location on the circumference and are uniformly distributed on it. Firms choose to which illness their drug is targeted to (i.e., choose a location on the circumference of the circle). Different kinds of drugs are imperfectly substitutable. For instance, some pain killers that help well for some kinds of headache work less for other types but still have an effect, some work better for rheumatism than for headache, and so on. A certain kind of chemotherapy may improve the health status for various forms of cancer but particular substances may be particularly well-suited for a specific type of cancer. The same is true for illnesses caused by bacteria, which can be treated with various kinds of

[7] Empirical support for this assumption is provided by Cai, Mavromaras and Oguzoglu (2008). They find that individuals who experience health shocks respond by incremental reductions in labor supply rather than by leaving the labor force.

antibiotics. Typically, a specific kind of antibiotic kills or prevents breeding of a rather wide spectrum of bacteria but is more effective against certain types of bacteria than others. As a final example, there are several classes of medication against hypertension. Products are quite substitutable, targeting different sources of high blood pressure and differing with respect to side effects. Thus, the structure of the pharmaceutical market is represented here by oligopolistic competition on prices for differentiated goods.

Price setting power arises because pharmaceutical products cannot be imitated, e.g. because of patent protection. Patent breadth has a natural representation in the model. It is defined as the sum of the lengths of the segments on the circumference of the circle of illnesses to the left and right of the location of firm i (representing the closest substitutes to product i) where rivals are not allowed to locate. Consider a symmetric situation where the distance between the location of each firm on circumference of the circle (with unit length) is $1/n$. This is also the size of the segment on the circle of each firm ($0.5/n$ on both sides of a firm's location) which is protected by patent law. Thus, if the patent breadth is at least $1/n$, then no additional firm is allowed to enter. The restricted entry case may therefore be interpreted as a situation where no firm can enter despite positive profits because it would infringe a patent. An increase in the firm number n may thus reflect a change in the patent law which reduces the patent breadth such that more firms can enter. An alternative competition policy which raises n in the restricted entry case would be to encourage entry of foreign firms.

In the case of an endogenous number of firms, it is assumed that the patent breadth is smaller than the equilibrium value of $1/n$. A decrease in f in the endogenous entry case may capture, for instance, lower administrative costs associated with weaker entry regulation (for examples and measurement, see Djankov, La Porta, Lopez-de-Silanes and Shleifer, 2002). Also extensive marketing effort of pharma firms for branded prescription drugs via sales representatives (who directly contact physicians) erect entry barriers for potential rivals. Such entry barriers could be reduced (again, captured by a decrease in f) by regulating the activities of sales representatives in the pharmaceutical sector like restraining gift-giving to physicians. Also prohibiting drug makers to use doctors' prescribing data to develop marketing strategies could lead to a decrease in f.

Pharmaceutical firms can affect the "quality" (i.e. the vertical dimension) of drugs by incurring R&D costs. Higher quality means that the health status improves for a given type of illness to which the drug is targeted to and possibly also for related illnesses. To capture both the horizontal and vertical dimension of pharmaceuticals, suppose that health status of an ill individual j when consuming one unit of drug i is

$$H(j) = h(\delta_i(j), Q_i), \tag{2}$$

where $\delta_i(j)$ is the shorter (arc) distance between the illness of consumer j and the horizontal location of firm i's product on the circumference of the circle of illnesses; Q_i is the quality of drug i. We assume that function h has partial derivatives $h_\delta < 0$ (i.e., the health level is lower when the drug is less suited), $h_Q > 0$ with $\lim_{Q\to\infty} h(0, Q) \leq 1$ (recall that unity is the upper limit of the health level by definition), and $h_{QQ} \leq 0$ (i.e., the marginal gain in health from a quality-improvement is non-increasing in the quality level); moreover, suppose $h_{\delta\delta} \leq 0$ and $h_{\delta Q} < 0$. Property $h_{\delta Q} < 0$ implies a ranking of the impact of higher R&D on health improvement for different patients. For instance, consider a drug which contains antibiotics. Suppose the drug is best suited to fight (a specific form of) pneumonia but also works against some other illnesses caused by bacteria. However, also suppose the bacteria which cause pneumonia have developed some antibiotic resistance. Then $h_{\delta Q} < 0$ means that an increase in R&D spending directed to overcome antibiotic resistance of bacteria which cause pneumonia has a larger effect on health for patients with pneumonia than for patients with other bacterial infections.[8]

To supply a drug with quality Q_i, firm i has to incur R&D costs $B(Q_i)$ which are strictly convex in Q_i, $B' > 0$, $B'' > 0$. Following the "endogenous sunk cost" approach (e.g., Sutton, 1991, 1998) and "quality ladder" models of endogenous growth (e.g., Grossman and Helpman, 1991), R&D costs are not reflected in marginal production costs.[9]

[8] See The Economist (2011) for a discussion on the efforts to tackle the resistance of antibiotics, e.g. via R&D.

[9] We abstract from uncertainty in the R&D process. Nothing would change, however, if firms are successful in innovating and entering the market only with some probability, as long as there are many

Illnesses are assumed to be perfectly and costlessly detectable by diagnostic tests. Moreover, individuals know the horizontal and vertical location of each firm, as well as function h, and therefore are capable of choosing the product which maximizes their utility. Alternatively, one may assume that physicians choose on behalf and in the interest of patients. To abstract from informational constraints greatly simplifies the analysis.[10]

To distinguish pharmaceuticals from "regular" consumption goods, we assume that more is not better. More precisely, ill individuals do not gain from consuming more than one dose of a drug. For simplicity, they also do not gain from consuming different drugs.[11]

For reasons of tractability, we follow the common assumption in ideal variety models that firms simultaneously choose price and their "location" on the circumference of the product circle to maximize profits. In the present context, they also choose the quality Q_i of a drug at the same time.[12]

Finally, suppose that there exists a health insurance system which covers the risk of needing drug treatment. However, patients themselves have to pay a fraction $\tau \in [0, 1]$

potential innovators which are risk-neutral. In this case, neither supply of R&D funds is affected by uncertainty (due to the law of large numbers) nor is demand.

[10] A priori, it seems unclear whether and how the nature of price competition in the pharmaceutical market would change under asymmetric information between physicians and patients and/or under limited information of both. These are challenging issues which are beyond the scope of the present paper.

[11] For some diseases treatment is more effective when several drugs are combined, like for attacking HIV, the virus that causes AIDS. In our context, this would be captured by defining a drug as a combination of active pharmaceutical ingredients in one dose. Many drugs contain several active ingredients, so it does not matter if those are combined in, say, one injection/pill or provided via several different injections/pills.

[12] Assuming that prices and quality are chosen simultaneously draws on the seminal paper on R&D choice under imperfect competition in Dasgupta and Stiglitz (1980). It is shown in Appendix C that assuming a two-stage decision process may not change results. There we suppose that firms choose the type of horizontal differentiation along with the vertical quality component at stage 1, whereas at stage 2 they choose prices (product market competition). If at stage 1 firms take prices of other firms as given (along with product quality and horizontal location), then the behavior of firms is shown to be exactly the same as in the case where there is just one decision stage. That is, nothing changes compared to the analysis presented in the main body of the paper if we assume that firms take the effect of their product differentiation choices on their own price setting power at stage 2 into account but do not account for the possible equilibrium price adjustments of and due to rivals at stage 2. Unfortunately, relaxing the latter assumption would complicate the analysis to the point of intractability.

of the price of medication − the coinsurance rate.[13] Health insurance is assumed to be fair, i.e., the insurance premium, T, is equal to the expected reimbursement of patients' medication expenses from the insurance. In the next section we examine the effect of an increase in the coinsurance rate τ on the R&D expenditure of pharmaceutical firms and on prices of their products.

4 Equilibrium Analysis

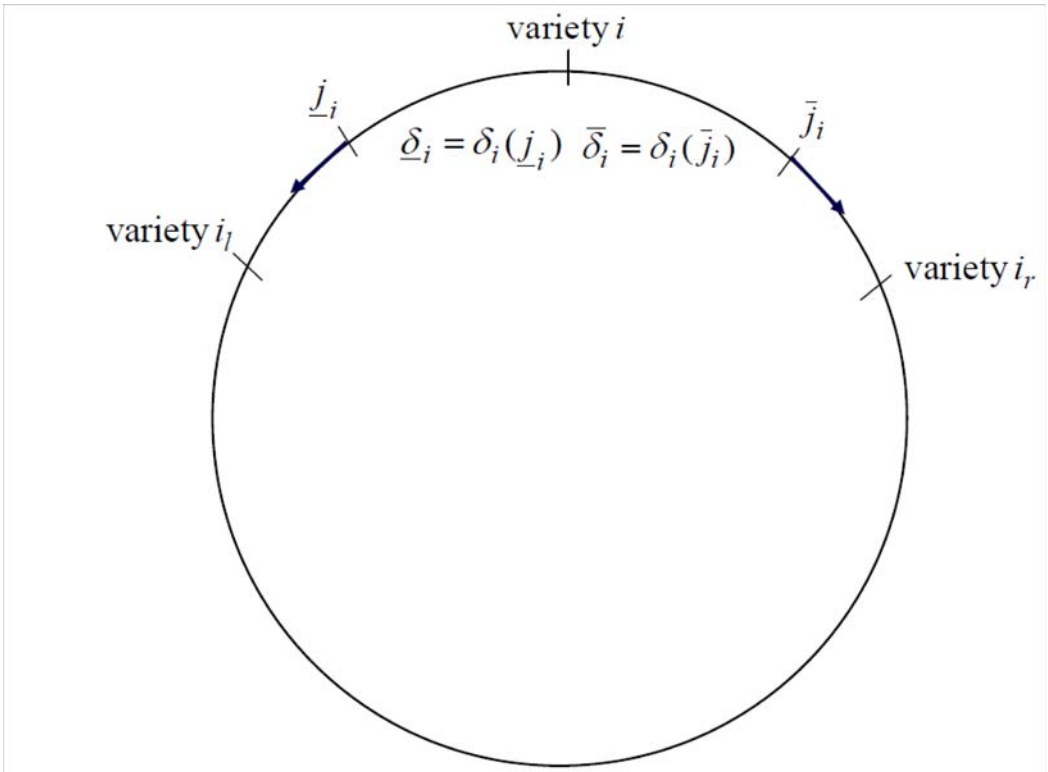

Figure 1: The product circle of differentiated pharmaceutical goods and the impact of quality-improvements (indicated by arrows).

Consider the location of firm i on the circumference of the circle of illnesses. Denote the firm to the left of i by i_l and the firm to the right of i by i_r. The shorter (arc) distance between the location of i and i_l is denoted by $\delta_i(i_l)$ and the one between between i and i_r by $\delta_i(i_r)$. $D_i \equiv \delta_i(i_l) + \delta_i(i_r)$ is the distance between i_l and i_r. Denote

[13] We abstract from moral hazard − although sometimes being the alleged reason for implementing coinsurance schemes in the first place. This argument is unconvincing in the case of severe illness like cancer or AIDS, however. In any case, health insurance systems are exogenous in our analysis.

by $\underline{j}_i$ the patient with the ideal variety to the left of firm i's location, who is indifferent between buying from firm i and i_l. Similarly, consumer $\overline{j}_i$ is indifferent between buying from i and i_r.

As shown in Fig. 1, $\underline{\delta}_i = \delta_i(\underline{j}_i)$ is the distance between i and $\underline{j}_i$ whereas $\overline{\delta}_i = \delta_i(\overline{j}_i)$ is the distance between i and $\overline{j}_i$. Define by

$$\underline{H}_i \equiv h(\underline{\delta}_i, Q_i),\ \overline{H}_i \equiv h(\overline{\delta}_i, Q_i), \tag{3}$$

$$\underline{H}_{i_l} \equiv h(\delta_i(i_l) - \underline{\delta}_i, Q_{i_l}),\ \overline{H}_{i_r} \equiv h(\underbrace{D_i - \delta_i(i_l)}_{=\delta_i(i_r)} - \overline{\delta}_i, Q_i), \tag{4}$$

the health levels of consumer $\underline{j}_i$ and $\overline{j}_i$, respectively, when consuming drug i (eq. (3)) and the alternative drugs i_l, i_r (eq. (4)). The associated consumption levels are given by

$$\underline{C}_i \equiv wg(\underline{H}_i) - \tau p_i - T,\ \overline{C}_i \equiv wg(\overline{H}_i) - \tau p_i - T, \tag{5}$$

$$\underline{C}_{i_l} \equiv wg(\underline{H}_{i_l}) - \tau p_{i_l} - T,\ \overline{C}_{i_r} \equiv wg(\overline{H}_{i_r}) - \tau p_{i_r} - T, \tag{6}$$

where p_i, p_{i_l}, p_{i_r} denote the price of drug i, i_l, i_r. $wg(H)$ is wage income of an individual with health level H, τp_i is the coinsurance payment when consuming the drug supplied by firm i, and T is the insurance premium. Note that $(\underline{\delta}_i + \overline{\delta}_i)s$ is the mass of consumers buying from firm i (recall that a mass s of consumers is ill). Thus, with fair health insurance, the insurance premium for each individual is

$$T = (1-\tau)s\sum_{i=1}^{n}(\underline{\delta}_i + \overline{\delta}_i)p_i. \tag{7}$$

For individual $\underline{j}_i$, who is indifferent to buy from firm i and i_l, we have

$$0 = u(\underline{C}_i, \underline{H}_i) - u(\underline{C}_{i_l}, \underline{H}_{i_l}). \tag{8}$$

Substituting the respective first equations of (3)-(6) into (8) reveals that $\underline{\delta}_i$ is implicitly given as function of own price and quality, price and quality of the com-

petitor to the left, the distance to this competitor, and parameters. Write $\underline{\delta}_i = \underline{\Delta}_i(p_i, Q_i, p_{i_l}, Q_{i_l}, \delta_i(i_l), \tau, T, w)$. Similarly, for individual $\overline{j}_i$, who is indifferent to buy from firm i and i_r, we have

$$0 = u(\overline{C}_i, \overline{H}_i) - u(\overline{C}_{i_r}, \overline{H}_{i_r}). \tag{9}$$

Using the respective second equations of (3)-(6), (9) implies that we can write $\overline{\delta}_i = \overline{\Delta}_i(p_i, Q_i, p_{i_r}, Q_{i_r}, D_i - \delta_i(i_l), \tau, T, w)$.

The profit maximization problem of firm i can then be written as

$$\begin{aligned}\max_{\delta_i(i_l), Q_i, p_i} (p_i - c)s\,[&\underline{\Delta}_i(p_i, Q_i, \delta_i(i_l), p_{i_l}, Q_{i_l}, \tau, T, w) + \\ &\overline{\Delta}_i(p_i, Q_i, \underbrace{D_i - \delta_i(i_l)}_{=\delta_i(i_r)}, p_{i_r}, Q_{i_r}, \tau, T, w)] - B(Q_i) - f,\end{aligned} \tag{10}$$

taken as given p_{i_l}, Q_{i_l}, p_{i_r}, Q_{i_r}, D_i and T.

We first derive the equilibrium of the basic model for a given number of firms (restricted entry) and present comparative-static results. Then we allow for unrestricted entry, where the number of firms is endogenous, demonstrating that the main insights from the restricted entry case typically still hold.

4.1 Restricted Entry

An equilibrium in the restricted entry case is defined as locational choices, drug prices, and drug quality levels, in which firms maximize profits and ill consumers choose the drug which yields the highest utility given the choices of firms. Using expression (7) for the premium T in the first-order conditions which result from (10), we can derive the following lemma. (All proofs are relegated to Appendix A.)

Lemma 1. *In a symmetric equilibrium for a given number of firms, where* $\underline{\delta}_i = \overline{\delta}_i = \frac{1}{2n}$, $\delta_i(i_l) = \delta_i(i_r) = \frac{1}{n}$, $Q_i = Q$ *and* $p_i = p$ *for all* i, *equilibrium values* (Q^*, p^*)

are simultaneously given by[14]

$$0 = -(p-c)s\frac{h_Q\left(\frac{1}{2n},Q\right)}{h_\delta\left(\frac{1}{2n},Q\right)} - B'(Q) \equiv F(Q,p,n), \tag{11}$$

$$0 = wg'\left(h\left(\frac{1}{2n},Q\right)\right) + MRS(Q,p,n,w,\tau) - \frac{\tau n B'(Q)}{s h_Q\left(\frac{1}{2n},Q\right)} \equiv G(Q,p,n,w,\tau), \tag{12}$$

as functions of n, w, τ*, where*

$$MRS(Q,p,n,w,\tau) \equiv \frac{u_H\left(wg\left(h\left(\frac{1}{2n},Q\right)\right) - [\tau(1-s)+s]\,p, h\left(\frac{1}{2n},Q\right)\right)}{u_C\left(wg\left(h\left(\frac{1}{2n},Q\right)\right) - [\tau(1-s)+s]\,p, h\left(\frac{1}{2n},Q\right)\right)} \tag{13}$$

is the marginal rate of substitution between consumption and health under symmetry.

The first summand on the right-hand side of eq. (11) is the marginal benefit of raising quality Q which, in profit maximum, must be equal to the marginal cost of improving quality, $B'(Q)$. The marginal benefit of raising Q is higher, the higher the price-cost margin, $p-c$, the larger the total market size of the pharmaceutical market, s, the higher the effectiveness of R&D for health, h_Q, and the lower is the impact on health status of deviating from the ideal variety of a patient, $|h_\delta|$. To see intuitively that the ratio $h_Q/|h_\delta|$ matters for R&D incentives consider again the case of antibiotics. The innovation incentive is higher, the larger is for a given type of bacterium which causes an illness the effect of higher quality Q on health status, h_Q, and the wider the spectrum of bacteria and illnesses affected by the antibiotic (i.e., $|h_\delta|$ is lower).

Eq. (12) reflects that, not surprisingly, the marginal rate of substitution between consumption and health matters for price-setting behavior of firms. Prices are also affected by the marginal impact of an increase in health on wage income, wg', which determines demand for pharmaceuticals as well. Moreover, demand becomes more price-sensitive when the coinsurance rate, τ, increases.

A symmetric equilibrium exists and is unique under weak conditions. An example of sufficient conditions on the primitives of the model is spelled out in Appendix B. Uniqueness of equilibrium allows us to derive comparative-static results.

[14] Arguments s and c are suppressed in functions F, G and MRS.

Proposition 1 *(Comparative-statics in the restricted entry case) In a unique equilibrium, (a) a higher coinsurance rate τ lowers both R&D-related quality of pharmaceuticals, Q^*, and their prices, p^*. (b) A higher wage rate w raises both Q^* and p^*. (c) An increase in the number of firms, n, may raise Q^*; for instance, Q^* is increasing in n if $u_{CH} = u_{HH} = 0$,*

$$g'' = 0 \text{ and } \varepsilon(\delta, Q) \equiv -\frac{\delta h_{\delta Q}(\delta, Q)}{h_Q(\delta, Q)} \geq 1. \tag{14}$$

The intuition for the negative impact of a higher coinsurance rate, τ, on R&D spending and prices (part (a) of Proposition 1) is simple. An increase in the fraction of the drug price which a patient has to copay implies that she becomes more price-sensitive. Thus, by raising the price of its drug, a pharmaceutical company loses more customers to rivals. This induces firms to lower the prices as an equilibrium response. Consequently, also the equilibrium insurance premium, $T^* = (1 - \tau)sp^*$, is decreasing in τ. In turn, however, the reduced mark-up over marginal costs, $p - c$, lowers the marginal benefit of R&D.

By contrast, a higher wage rate, w, raises the willingness to pay for drugs of ill consumers due to two effects. First, the price sensitivity declines after an increase in w if better health raises the supplied labor units ($g' > 0$). This effect arises since the marginal impact of better health on wage income rises with w. Second, the marginal rate of substitution between consumption and health, u_H/u_C, is increasing in w. Both effects go in the same direction and explain part (b).

Part (c) of Proposition 1 can be understood as follows. Elasticity ε measures by how much the marginal benefit of higher drug quality on health (h_Q) declines if δ increases by one percent. Recall that a higher δ means that the drug is less suited to the particular illness of a patient. Now suppose that the number of pharmaceuticals increases. As a result, a firm loses customers for a given R&D spending since, on average, δ decreases. Consequently, for a given price of a drug and implied by $h_{\delta Q} < 0$, there is a higher incentive to conduct R&D in order to retain some of the customers. This effect is large if ε is high. Thus, firms may conduct more R&D despite the fact

that price-setting power is adversely affected by an increase in the number of rivals.[15]

The latter result is interesting for two reasons. First, it shows that there is not necessarily a trade-off between the goal of keeping price-setting power of pharmaceutical companies low and their R&D incentives high. To the contrary, weaker protection against entry reduces price-setting power but may foster R&D incentives. Thus, one has to distinguish whether price-setting power of pharmaceutical firms is affected by health insurance policy or by competition policy. Second, the result contributes to the recent debate on the relationship between competition and innovation. The literature has suggested that heterogeneity of firms with respect to their distance to the technology frontier is critical for the result that increased competition or entry deregulation can spur innovative effort (Aghion et al., 2005; Aghion and Howitt, 2005, 2009; Aghion et al., 2009). The basic argument runs as follows. Incumbents operating at the technology frontier can escape competition or entry, i.e., secure a monopoly position, by innovating. Increased competition means that pre-innovation profits decline whereas post-innovation profits, which are pure monopoly profits by assumption, do not depend on the number of rivals. Facilitating entry thus raises R&D expenditure. By contrast, firms below the technology frontier see the difference between post- and pre-innovation profits decline if competitive pressure rises, as they cannot escape competition. The present paper gives complementary insights on the competition-innovation relationship. It shows that the prospect of gaining pure monopoly power from innovation is not required for the result that increased entry spurs innovative effort. Rather, the result may even hold in an environment with strategically interacting firms which possess similar technology.

4.2 Endogenous Number of Firms

We now show that the basic insights of Proposition 1 are not critically affected by allowing for an endogenous number of firms. With unrestricted entry, in equilibrium,

[15] One can also show that higher market size, s, typically raises R&D incentives. This result is consistent with empirical evidence by Acemoglu and Linn (2004). According to their study, an increase in potential market size for drugs - measured by exploiting demographic trends in the US - has fostered pharmaceutical innovation.

profits of firms must be zero, as firms enter the market as long as profits are positive.

In a symmetric situation the zero-profit condition holds if

$$0 = \frac{(p-c)s}{n} - B(Q) - f \equiv Z(Q, p, n, f), \tag{15}$$

according to profit function (10). The equilibrium quality, price, and number of firms, denoted by (Q^{**}, p^{**}, n^{**}), are simultaneously given by equation system $F = G = Z = 0$. We define matrix

$$\mathbf{M} \equiv \begin{pmatrix} F_p & F_Q & F_n \\ G_p & G_Q & G_n \\ Z_p & Z_Q & Z_n \end{pmatrix}. \tag{16}$$

As shown in the proof of the following proposition, the determinant of $\mathbf{M}$ is positive under weak conditions, implying uniqueness of equilibrium.

Proposition 2 *(Comparative-statics with an endogenous firm number) Suppose that* $\det(\mathbf{M}) > 0$. *Then, similar to the restricted entry case, (a) a higher coinsurance rate,* τ, *lowers both equilibrium quality,* Q^{**}, *and equilibrium prices of drugs,* p^{**}. *(b) A higher wage rate,* w, *has the opposite effects. (c) Entry deregulation (decrease in* f*) tends to promote entry (*n^{**} *increases) and raises* Q^{**} *if (14) holds.*

Proposition 2 shows that the impact of an increase in the coinsurance rate and in the wage rate on R&D spending and on prices of pharmaceuticals is robust to allowing for endogenous entry of firms.

Moreover, not surprisingly, the number of firms typically declines if entry costs go up. Consistent with the effects of a change in the number of firms under restricted entry (part (c) of Proposition 1), retarding entry by higher fixed costs tends to be associated with reduced R&D spending if ε is high.

5 Price Regulations

This section examines the effects of price regulations. We distinguish direct price controls and price caps on drug expenditure reimbursement. In its simplest form,

on which we focus in this section, a price control means that the government sets a fixed, maximum drug price in a regime where health insurance does not cover drug expenses. A decrease in this price, which we denote by $p_{\max}$, captures stricter direct price regulation. By contrast, a price cap is a cost-sharing scheme which imposes a limit amount on the costs incurred by an insured patient which is reimbursed. Like coinsurance schemes, a price cap intends to keep insurance premiums low. The limit price is denoted by $\bar{p}$. We relate to a decrease in $\bar{p}$ as stricter price cap.

5.1 Price Control

Suppose there is no health insurance, i.e., the coinsurance rate is 100 percent ($\tau = 1$). A binding direct price control means that the price set by the government is below the equilibrium price. We therefore implicitly assume in this section that, for $\tau = 1$, $p_{\max} < p^*$ and $p_{\max} < p^{**}$ hold. We focus on the simplest case where the government ignores R&D costs.[16]

With restricted entry, the equilibrium drug quality, Q^*, is given by

$$F(Q^*, p_{\max}, n) = 0, \tag{17}$$

where function F was defined in (11). To see this, note that (11) reflects the first-order condition with respect to the R&D decision of firms and firms cannot set prices under direct price controls. Under free entry, which implies that firms' equilibrium profits are zero, equilibrium drug quality, Q^{**}, and the number of firms, n^{**}, are simultaneously given by

$$F(Q^{**}, p_{\max}, n^{**}) = Z(Q^{**}, p_{\max}, n^{**}, f) = 0, \tag{18}$$

where function Z was defined by (15).

Proposition 3 *(Price controls) Suppose there is a binding direct price control. (a)*

[16] Price controls follow a redistributive goal, aiming to reduce the financial burden of the ill vis-à-vis the healthy. Pharmaceutical prices in this regime are typically negotiated between pharmaceutical companies and the government. Critics of price controls argue that negotiated sales prices insufficiently account for R&D costs. The analysis would become more complicated if R&D costs and the effectiveness of drugs played a role in the setting $p_{\max}$.

Stricter price regulation (decrease in $p_{\max}$) lowers the equilibrium quality of pharmaceuticals; with unrestricted entry, it also reduces the number of firms, n^{**}. (b) Under restricted entry, an increase in the number of firms, n, unambiguously raises the quality of drugs, Q^*. With unrestricted entry, entry deregulation (decrease in f) raises both the quality of drugs, Q^{**}, and n^{**}.

A stricter price control limits the price-cost margin, $p_{\max} - c$, and therefore retards R&D incentives. The profit squeeze also retards entry. (part (a) of Proposition 3). Deregulation of entry, which allows for a larger number of competitors, unambiguously raises R&D expenditure under direct price controls (part (b)). Similar to the discussion of the last result in Proposition 1, an increased number of drugs induces pharmaceuticals companies to retain some of its customers by raising R&D. As there is no counteracting effect on R&D incentives through reduced price-setting power, the result is unambiguous.

5.2 Price Caps

A health system which combines coverage of prescription drug expenses with a price cap $\bar{p} > 0$ on reimbursement typically raises demand for drugs vis-à-vis a free market without any insurance. To see this, first note that the fair insurance premium under a binding price cap (i.e. one which is lower than the equilibrium price with full insurance) is given by $T = s\bar{p}$. Thus, total health expenditures for a customer of firm i is $p_i - \bar{p} + T = p_i - (1-s)\bar{p} < p_i$. Hence, a stricter price cap is not an intervention in a free market but restricts the drug expenditure subsidy to beneficiaries. Demand faced by pharmaceutical companies is lowered by a stricter price cap, since a decrease in $\bar{p}$ lowers the marginal rate of substitution, $MRS = u_H/u_C$, in equilibrium with symmetric firms. This can be seen as follows. A customer of firm i with health status H has a consumption level of $wg(H) - p_i + (1-s)\bar{p}$. Thus,

$$MRS = \frac{u_H(wg(H) - p_i + (1-s)\bar{p}, H)}{u_C(wg(H) - p_i + (1-s)\bar{p}, H)}. \tag{19}$$

The right-hand side of (19) is increasing in $\bar{p}$. We find the following result.

Proposition 4 *(Price caps) A stricter price cap (decrease in $\bar{p}$) lowers both the price and quality of pharmaceuticals in symmetric equilibrium. Entry regulations have similar effects as in the basic model.*

Since the marginal rate of substitution decreases with a stricter price cap, firms have less price setting power which in turn is associated with a decrease in R&D spending. Regarding entry regulations, the same discussion as for Proposition 1 and 2 applies.

6 General Equilibrium with Endogenous Growth

In this section we extend the basic model (with a coinsurance scheme) to a simple dynamic general equilibrium framework with endogenous income growth. We examine the interaction between R&D spending of pharmaceutical companies and aggregate productivity in the consumption goods sector, endogenizing the wage rate, w. One key feature of the analysis is to allow for individual labor supply to depend on health status. Consequently, illness and its pharmaceutical treatment may have effects on the scale of the economy and, thus, on productivity and wages.

6.1 Dynamic Set Up

Suppose that individuals inelastically supply their labor to a perfect labor market. Output Y of the numeraire consumption good at time $t = 1, 2, ...$ is produced under perfect competition, according to

$$Y_t = (L_t^Y)^{1-\alpha} \int_0^{N_t} A_t(k)^{1-\alpha} x_t(k)^{\alpha} \mathrm{d}k + c_t R_t, \tag{20}$$

$0 < \alpha < 1$. $x(k)$ denotes the quantity of intermediate input $k \in [0, N]$ and $A(k)$ is a productivity measure of input k.[17] $A_t(k)$ can be affected by in-house R&D of single-product firm k in period t. L^Y is labor input in final goods production. R is the input of a resource which is available in fixed supply, $\bar{R}$, and has productivity c. We assume that the associated factor market is competitive, i.e., the price of the fixed factor is c.

[17] Time index t is omitted whenever this does not lead to confusion.

The pharmaceutical sector is similar to the basic model. Suppose, however, that pharmaceutical companies use the fixed resource (rather than the numeraire) as input in both the R&D process and the production of pharmaceuticals. One unit of the fixed resource can be transformed into one unit of the drug, i.e., unit costs are c, as in the partial equilibrium analysis. Also suppose that quality level Q of a drug requires $b(Q)$ units of the fixed factor, where $b' > 0$, $b'' > 0$;[18] thus, R&D costs of pharmaceutical firms are given by $B(Q) = cb(Q)$. We assume that c is proportional to the average productivity of the intermediate goods sector, i.e.,

$$c_t = \frac{1}{N_t} \int_0^{N_t} A_t(k) \mathrm{d}k \equiv \bar{A}_t, \tag{21}$$

capturing intersectoral spillover effects. The introduction of the fixed factor together with (21) implies that costs in the pharmaceutical sector grow with the same rate as $\bar{A}$. This implies the existence of a balanced growth equilibrium (BGE). A BGE is defined as a long run equilibrium in which all variables grow at a constant rate.

Productivity of an intermediate good k evolves according to

$$A_t(k) = \bar{A}_{t-1} v(l_t^A(k)), \tag{22}$$

where $l^A(k)$ is labor input in the R&D process of intermediate good producer k, $v' > 0$, $v'' < 0$. Term $\bar{A}_{t-1}$ in (22) captures a standard intertemporal knowledge spillover effect, which will drive economic growth in the model.

The intermediate goods sector is monopolistic. One unit of the numeraire good can be transformed into one unit of an intermediate input. Moreover, production requires a fixed number of labor units, $\bar{l} > 0$, each period (Young, 1998). The mass of intermediate goods, N, is endogenous. That is, intermediate good firms enter as long as profits are non-negative.

We simplify the analysis by focussing on restricted entry in the pharmaceutical

[18] We implicitly assume that $\bar{R} - s - nb(Q^*) > 0$ holds. That is, in equilibrium, a positive amount of the fixed resource is used in the consumption good sector after s units of pharmaceuticals are produced and the resource is used for R&D input of the n pharmaceutical firms. As equilibrium quality of pharmaceuticals, Q^*, does not depend on $\bar{R}$, the condition holds whenever $\bar{R}$ is sufficiently large.

sector (i.e., the number of pharmaceutical firms, n, is exogenous). Moreover, we assume that each individual lives one period and specify the utility function to

$$u(C, H) = \ln C + H, \tag{23}$$

i.e., $u_{CH} = u_{HH} = 0$.

6.2 Balanced Growth Equilibrium

We first look at the pharmaceutical sector by recalling the partial equilibrium analysis in section 4. First define $\hat{p}_t \equiv p_t/\bar{A}_t$ and $\hat{w}_t \equiv w_t/\bar{A}_t$. In BGE, $\hat{p}$ and $\hat{w}$ turn out to be constant, i.e., the equilibrium price of pharmaceuticals and the wage rate grow with the same rate as average productivity $\bar{A}$. Moreover, note that $b(Q) = B(Q)/\bar{A}$, according to (21). Thus, dividing (11) from Lemma 1 by $\bar{A}$ implies that

$$\hat{p}_t = 1 - \frac{h_\delta\left(\frac{1}{2n}, Q_t\right)}{h_Q\left(\frac{1}{2n}, Q_t\right)} \frac{b'(Q_t)}{s} \equiv \hat{P}(Q_t). \tag{24}$$

Note that $\hat{P}'(Q) > 0$, i.e., there is a positive relationship between productivity-adjusted prices and the quality of pharmaceuticals. Moreover, rewriting (12) by using (13) and (23), dividing by $\bar{A}$, and substituting (24) leads to

$$\hat{w}_t = \frac{\tau(1-s) + s + \frac{b'(Q_t)}{sh_Q\left(\frac{1}{2n}, Q_t\right)}\left(\tau n - \left[\tau(1-s)+s\right] h_\delta\left(\frac{1}{2n}, Q_t\right)\right)}{\Theta(Q_t)} \tag{25}$$

$$\equiv W(Q_t, \tau, n), \text{ where } \Theta(Q) \equiv g'\left(h\left(\frac{1}{2n}, Q\right)\right) + g\left(h\left(\frac{1}{2n}, Q\right)\right). \tag{26}$$

Part (b) of Proposition 1 suggests that, under weak conditions, the relationship between $\hat{w}_t$ and Q_t is positive. In fact, the numerator on the right-hand side of (25) is increasing in Q. The denominator $\Theta(Q)$ is non-increasing in Q if

$$g' \leq |g''|, \tag{A1}$$

i.e., if the impact of better health on individual labor supply is small and/or decreasing fast, as plausible. Thus, assumption A1 is sufficient (but not necessary) for $W_Q > 0$.

Now we look outside the pharmaceutical sector and at the labor market equilibrium. Total labor supply L^S in the economy is the sum of labor supply of the healthy, $1-s$, and of ill individuals:

$$L_t^S = 1 - s + sg\left(h\left(\frac{1}{2n}, Q_t\right)\right). \tag{27}$$

Lemma 2. *Outside the pharmaceutical sector, we find the following relationship between the productivity-adjusted wage and the quality of pharmaceuticals:*

$$\hat{w}_t = \frac{(1-\alpha)\alpha^{\frac{2\alpha}{1-\alpha}} v'(l^{A*})}{1+\alpha} \underbrace{\left[1 - s + sg\left(h\left(\frac{1}{2n}, Q_t\right)\right)\right]}_{=L_t^S} \equiv \tilde{W}(Q, n), \tag{28}$$

where l^{A*} is the (time-invariant) equilibrium R&D labor input of each intermediate goods producer, given by

$$\frac{v(l^{A*})}{v'(l^{A*})} - l^{A*} - \bar{l} = 0. \tag{29}$$

The growth rate of $\bar{A}$ is given by $v(l^{A*}) - 1$ at all times and does neither depend on the coinsurance rate, τ, nor on the number of pharmaceutical firms, n.

Lemma 2 shows that the (adjusted) wage rate is positively related to aggregate labor supply in the economy. Thus, a better quality of pharmaceutical products is positively associated with the marginal product of labor in the final consumption sector, $w = \partial Y/\partial L^Y$ ($\tilde{W}_Q > 0$) whenever individual labor supply depends on health status ($g' > 0$). Lemma 2 reflects a scale effect which is typical in models with endogenous technical change. The reason for the scale effect can be seen as follows. Denote by X the expenses in terms of the numeraire for the production of intermediate goods, i.e., we have $x(k) = X/N$ for all k under symmetry. Moreover, note that $A(k) = \bar{A}$ for all k. Thus, (20) implies $Y = X^\alpha (N\bar{A}L^Y)^{1-\alpha} + \bar{A}R$. Hence, the efficiency of labor, $N\bar{A}$, and thus aggregate productivity in the economy is increasing in the number of intermediate good firms, N. This property captures specialization gains. It has been used extensively in both trade theory (e.g., Ethier, 1982) and endogenous growth theory

(e.g., Romer, 1990). Since equilibrium R&D labor input per intermediate good firm, l^{A*}, is independent of labor supply, there is no scale effect in the growth rate of $\bar{A}$, consistent with empirical evidence (Jones, 1995). However, an increase in aggregate labor supply, L^S, raises market size in the intermediate goods sector. Consequently, as formally shown in the proof of Lemma 2, the equilibrium number of intermediate good firms, N, is proportional to labor supply, L^S. It follows that better health which results from quality-improvements of pharmaceuticals raises the marginal product of labor. Apart from its specific microfoundation in the model, it is the generally plausible health-productivity relationship which – together with the demand side reflected by (25) – gives rise to the insights of this section.

Lemma 3. (a) *There are no transitional dynamics.(b) Possibly, there are multiple BGE. (c) A BGE exists and is unique, if (i)* $W(0,\tau,n) < \tilde{W}(0,n)$*, (ii) assumption A1 holds, and (iii)* $W(Q,\tau,n)$ *is convex as a function of* Q*.*

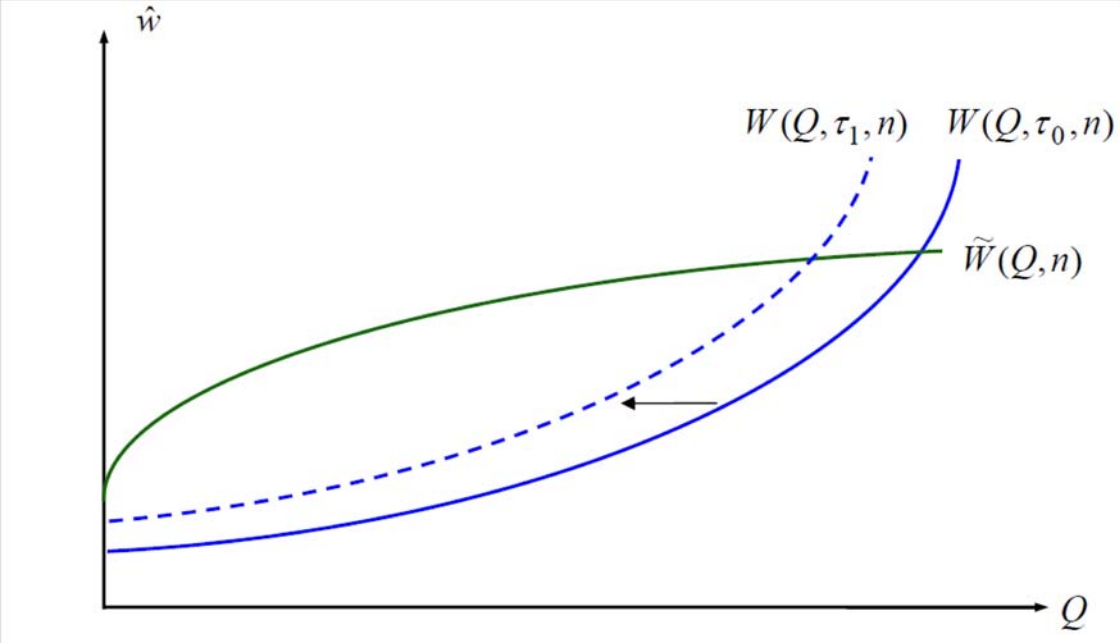

Figure 2: Unique balanced growth equilibrium and the effect of an increase in the coinsurance rate from τ_0 to τ_1.

Transitional dynamics are absent (part (a) of Lemma 3) as a consequence of the time-invariance of the R&D labor input per firm outside the pharmaceutical sector (Lemma 2), l^{A*}, and the linear spillover effect in R&D technology (22).

Part (b) of Lemma 3 is an implication of the positive wage-health relationships (25) and (28). On the one hand, according to (25), and as argued in the equilibrium analysis of the basic model, a higher wage rate makes demand for pharmaceuticals less price-sensitive, which is associated with higher R&D incentives ($W_Q > 0$). On the other hand, according to (25), better health through medical innovations raises the wage rate due to the scale effect explained above, whenever $g' > 0$ ($\tilde{W}_Q > 0$). This positive interaction is the source of potential multiplicity of equilibrium.

Fig. 2 shows a situation with a unique BGE, which fulfills presumptions (i)-(iii) in Lemma 3. Moreover, $\tilde{W}(Q, n)$ is increasing in Q (i.e., $g' > 0$ is assumed). We can employ Fig. 2 to show the following.

Proposition 5 *(Comparative-statics in general equilibrium) Suppose a unique BGE exists. Then, (a) an increase in the coinsurance rate, τ, reduces both the quality of pharmaceuticals (Q^*) and their (productivity-adjusted) prices ($\hat{p}^*$) in BGE; if $g' > 0$, it also reduces the (productivity-adjusted) wage rate ($\hat{w}^*$). (b) An increase in the number of pharmaceutical firms, n, raises Q^* and, if $g' > 0$, also raises $\hat{w}^*$ if (14) holds. (c) The growth rate of both the wage rate and prices of pharmaceuticals in BGE are independent of both τ and n.*

As there are no transitional dynamics, a change in the coinsurance rate (τ) or in the number of pharmaceutical companies (n) leads to a jump towards the new BGE. Part (a) of Proposition 5 can easily be understood. Recall that $W_\tau > 0$, according to (26). Thus, the $W-$curve in $Q - \hat{w}-$space shifts leftward when the coinsurance rate increases, say, from τ_0 to $\tau_1 > \tau_0$ (see Fig. 2). As explained after Proposition 1, an increase in τ makes demand for pharmaceuticals more price-sensitive. In turn, R&D spending in the pharmaceutical sector declines for a given wage rate. In general equilibrium, this effect is associated with a lower wage rate whenever labor supply depends on health status.

To show that part (b) holds, recall from Proposition 1 that there may be a positive relationship between the number of pharmaceutical firms and pharmaceutical R&D spending per firm. For instance, if (14) holds, such that $W_n < 0$, the $W-$curve

shifts rightward when the number of firms in the pharmaceutical sector, n, increases. Moreover, an increased choice set of pharmaceuticals improves average health status for a given drug quality Q. Thus, the $\tilde{W}-$curve shifts upwards if n increases, whenever $g' > 0$, according to (28). This reinforces the positive impact of higher entry on both $\hat{w}$ and Q.

Finally, regarding part (c) of Proposition 5, recall that there is no scale effect with respect to the growth rate of average productivity $\bar{A}$, $v(l^{A*}) - 1$. Thus, both w and p grow with the same rate as $\bar{A}$. Consequently, both wage and price growth are independent of policy measures towards the pharmaceutical sector.

In addition to examining the effects of coinsurance schemes on wages and health, the analysis in this section contributes from the theoretical side to the recent debate on the impact of higher life expectancy through medical innovations on economic outcomes (e.g., Acemoglu and Johnson, 1997; Aghion et al., 2010). It suggests that the effect is positive, in line with Aghion et al. (2010), if labor supply depends on health status.

7 Conclusion

This paper has examined the role of cost-sharing schemes in health insurance systems for prices of pharmaceuticals, R&D expenditure of pharmaceutical companies, aggregate productivity, and wages. The analysis suggests that extending coinsurance or applying stricter price regulations typically adversely affects pharmaceutical R&D spending while lowering drug prices. By contrast, lifting entry barriers may spur pharmaceutical innovations despite reducing price setting power. This happens when better suitability of a drug for patients, resulting from increased variety, leads to a sufficient increase in the effectiveness of R&D on health. In this case, stronger competition implies that firms attempt to retain some of their customers by quality-improvements in response to loss in market share. This calls into question the standard argument of pharma lobbyists that securing price-setting power − and therefore high profits − of pharmaceutical companies via entry regulation leads to high R&D spending. Examples for appropriate entry deregulation policies in the pharmaceutical sector would be to

limit non-informative marketing expenses, to promote access of foreign pharmaceutical companies to domestic markets, or to reduce patent breadth. Thus, the analysis provides a differentiated view on the often debated conflict in health policy between saving costs in health insurance systems and providing R&D incentives of firms. The results suggest that such a trade-off exists with respect to the analyzed aspects of health insurance systems but not necessarily with respect to competition policy. More generally, extending the ideal variety framework to allow for vertical R&D seems valuable for industrial policy design also beyond the present context of pharmaceutical markets, e.g., for health services.

The analysis may also be helpful to predict supply effects of the 2006 Medicare reform in the US. This reform introduced coverage of expenses for prescription drugs, effectively reducing the coinsurance rate from 100 percent to 25 percent and less. Our results obtained in the coinsurance regime thus suggest that the reform induces pharmaceutical firms to intensify their innovative effort.

The general equilibrium extension of the basic model to an endogenous growth framework gives rise to the conclusion that lower R&D expenditure in the pharmaceutical sector is associated with a reduction in aggregate productivity, in turn depressing the wage rate per unit of labor, when individual labor supply depends on health status.

Future research may build on the proposed theory to quantify welfare effects and to derive socially optimal cost-sharing schemes. For instance, limiting the coinsurance rate on drug expenditures may be warranted for a number of reasons. First, there is the standard positive welfare effect of providing health insurance to risk-averse households. Second, as focussed upon in this paper, R&D spending may be adversely related to the coinsurance rate. Thus, limiting cost-sharing may enlarge standard intertemporal "standing-on-shoulders" externalities from pharmaceutical R&D. For simplicity, such spillover effects have been ignored in this paper but should be allowed for in future research, along with other, possibly negative, R&D externalities (see e.g. Jones and Williams, 2000).

The analysis has emphasized that a better health status from the provision of higher-quality drugs may positively interact with the economic well-being of individuals. In a

welfare analysis, also such effects would have to be weighted against higher prices for pharmaceuticals which result when coinsurance rates are lowered or price caps lifted.

Finally, it may be fruitful to investigate how the nature of price competition changes in the pharmaceutical sector along with R&D incentives when physicians have superior information and pursue own interests, e.g. influenced by sales representatives.

Appendix

A: Proofs

Proof of Lemma 1: The first-order conditions associated with profit-maximization problem (10) with respect to $\delta_i(i_l)$, Q_i, p_i are

$$\frac{\partial \underline{\Delta}_i}{\partial \delta_i(i_l)} + \frac{\partial \overline{\Delta}_i}{\partial \delta_i(i_l)} = 0, \tag{30}$$

$$(p_i - c)s\left(\frac{\partial \underline{\Delta}_i}{\partial Q_i} + \frac{\partial \overline{\Delta}_i}{\partial Q_i}\right) - B'(Q_i) = 0, \tag{31}$$

$$\underline{\Delta}_i + \overline{\Delta}_i + (p_i - c)\left(\frac{\partial \underline{\Delta}_i}{\partial p_i} + \frac{\partial \overline{\Delta}_i}{\partial p_i}\right) = 0. \tag{32}$$

Using the first equations of (3)-(6) in (8) and applying the implicit function theorem, we obtain

$$\frac{\partial \underline{\Delta}_i}{\partial \delta_i(i_l)} = \frac{\left[u_C(\underline{C}_{i_l}, \underline{H}_{i_l}) w g'(\underline{H}_{i_l}) + u_H(\underline{C}_{i_l}, \underline{H}_{i_l})\right] h_\delta(\delta_i(i_l) - \underline{\delta}_i, Q_{i_l})}{\underline{\Omega}_i}, \tag{33}$$

$$\frac{\partial \underline{\Delta}_i}{\partial Q_i} = -\frac{\left[u_C(\underline{C}_i, \underline{H}_i) w g'(\underline{H}_{i_l}) + u_H(\underline{C}_i, \underline{H}_i)\right] h_Q(\underline{\delta}_i, Q_i)}{\underline{\Omega}_i}, \tag{34}$$

$$\frac{\partial \underline{\Delta}_i}{\partial p_i} = \frac{u_C(\underline{C}_i, \underline{H}_i)\tau}{\underline{\Omega}_i}, \tag{35}$$

where

$$\underline{\Omega}_i \equiv \left[u_C(\underline{C}_i, \underline{H}_i) w g'(\underline{H}_i) + u_H(\underline{C}_i, \underline{H}_i)\right] h_\delta(\underline{\delta}_i, Q_i) + \left[u_C(\underline{C}_{i_l}, \underline{H}_{i_l}) w g'(\underline{H}_{i_l}) + u_H(\underline{C}_{i_l}, \underline{H}_{i_l})\right] h_\delta(\delta_i(i_l) - \underline{\delta}_i, Q_{i_l}). \tag{36}$$

Similarly, using the second equations of (3)-(6) in (9), we obtain

$$\frac{\partial \overline{\Delta}_i}{\partial \delta_i(i_l)} = -\frac{\left[u_C(\overline{C}_{i_r}, \overline{H}_{i_r}) w g'(\overline{H}_{i_r}) + u_H(\overline{C}_{i_r}, \overline{H}_{i_r})\right] h_\delta(\delta_i(i_r) - \overline{\delta}_i, Q_{i_r})}{\overline{\Omega}_i}, \tag{37}$$

$$\frac{\partial \overline{\Delta}_i}{\partial Q_i} = -\frac{\left[u_C(\overline{C}_i, \overline{H}_i) w g'(\overline{H}_i) + u_H(\overline{C}_i, \overline{H}_i)\right] h_Q(\overline{\delta}_i, Q_i)}{\overline{\Omega}_i}, \tag{38}$$

$$\frac{\partial \overline{\Delta}_i}{\partial p_i} = \frac{u_C(\overline{C}_i, \overline{H}_i)\tau}{\overline{\Omega}_i}, \tag{39}$$

where

$$\overline{\Omega}_i \equiv \left[u_C(\overline{C}_i, \overline{H}_i) w g'(\overline{H}_i) + u_H(\overline{C}_i, \overline{H}_i)\right] h_\delta(\overline{\delta}_i, Q_i) + \left[u_C(\overline{C}_{i_r}, \overline{H}_{i_r}) w g'(\overline{H}_{i_r}) + u_H(\overline{C}_{i_r}, \overline{H}_{i_r})\right] h_\delta(\delta_i(i_r) - \underline{\delta}_i, Q_{i_r}). \tag{40}$$

In a symmetric situation, where $\underline{\delta}_i = \overline{\delta}_i = \frac{1}{2n}$, $\delta_i(i_l) = \delta_i(i_r) = \frac{1}{n}$, $Q_i = Q$ and $p_i = p$ for all i, and thus $\underline{C}_i = \overline{C}_i = \underline{C}_{i_l} = \overline{C}_{i_r} = \overline{C}$ as well as $\underline{H}_i = \overline{H}_i = \underline{H}_{i_l} = \overline{H}_{i_r} = \overline{H}$, we have $\frac{\partial \underline{\Delta}_i}{\partial \delta_i(i_l)} = 0.5$ and $\frac{\partial \overline{\Delta}_i}{\partial \delta_i(i_l)} = -0.5$, according to (33) and (37), respectively, using (36) and (40). Thus, (30) holds. Moreover, (31) leads to (11), using (34) and (38) as well as again (36) and (40). Finally, using analogously (35), (39), (36) and (40) in (32) we obtain

$$\frac{1}{n} + \frac{(p-c)\tau}{\left(w g'(\overline{H}) + \frac{u_H(\overline{C},\overline{H})}{u_C(\overline{C},\overline{H})}\right) h_\delta\left(\frac{1}{2n}, Q\right)} = 0. \tag{41}$$

Also note that $\underline{\delta}_i + \overline{\delta}_i = \frac{1}{n}$ and $p_i = p$ for all i imply that $T = (1-\tau)sp$, according to (7). Thus,

$$\overline{C} = w g\left(\overline{H}\right) - \left[\tau(1-s) + s\right] p, \text{ with } \overline{H} = h\left(\frac{1}{2n}, Q\right), \tag{42}$$

according to (5) and (3). Finally, rewriting (11) to

$$p = c - \frac{B'(Q)}{s} \frac{h_\delta\left(\frac{1}{2n}, Q\right)}{h_Q\left(\frac{1}{2n}, Q\right)}, \tag{43}$$

substituting (43) into (41) and using (42) confirms (12). ■

Proof of Proposition 1: Comparative-static results are confirmed using the implicit function theorem. Note that $F_p > 0$, according to (11). Thus, the determinant of matrix $\begin{pmatrix} F_p & F_Q \\ G_p & G_Q \end{pmatrix}$ is negative if $G_Q < F_Q G_p / F_p$, which ensures uniqueness of the equilibrium (see Appendix B for further discussion). Thus, applying the implicit function theorem,

$$sgn\left(\frac{\partial Q^*}{\partial \tau}\right) = sgn\left(F_p G_\tau - F_\tau G_p\right), \tag{44}$$

$$sgn\left(\frac{\partial p^*}{\partial \tau}\right) = sgn\left(F_\tau G_Q - F_Q G_\tau\right), \tag{45}$$

$$sgn\left(\frac{\partial Q^*}{\partial w}\right) = sgn\left(F_p G_w - F_w G_p\right), \tag{46}$$

$$sgn\left(\frac{\partial p^*}{\partial w}\right) = sgn\left(F_w G_Q - F_Q G_w\right), \tag{47}$$

$$sgn\left(\frac{\partial Q^*}{\partial n}\right) = sgn\left(F_p G_n - F_n G_p\right). \tag{48}$$

We have $F_p > 0$, $F_Q < 0$, $F_\tau = F_w = 0$, $F_n > 0$, according to (11), and $G_p < 0$, $G_\tau < 0$, $G_w > 0$, according to (12) and (13). Comparative static results regarding changes in τ and w then follow from (44)-(47), confirming parts (a) and (b).

To prove part (c) note that $\partial Q^*/\partial n > 0$ if $G_n \geq 0$, according to (48). Using (12), we have

$$G_n = -\frac{w g''(\overline{H}) h_\delta}{2n^2} + MRS_n + \frac{\tau B'(Q)}{s h_Q}\left[\varepsilon\left(\frac{1}{2n}, Q\right) - 1\right], \tag{49}$$

where

$$MRS_n = -\frac{h_\delta}{2n^2 u_C}\left[u_{CH} w g' + u_{HH} - \frac{u_H}{u_C}\left(u_{CC} w g' + u_{CH}\right)\right],$$

according to (13). Thus, $MRS_n \geq 0$ if $u_{CH} = u_{HH} = 0$. If, in addition, $g'' = 0$ and $\varepsilon \geq 1$, then $G_n \geq 0$, according to (49). This concludes the proof. ■

Proof of Proposition 2: First, note that $Z_Q < 0$, $Z_p > 0$, $Z_n < 0$, $Z_f < 0$ and $Z_\tau = Z_w = 0$, according to (15). As a remark,

$$\det(\mathbf{M}) = F_p G_Q Z_n + F_Q G_n Z_p + F_n G_p Z_Q - F_Q G_p Z_n - F_p G_n Z_Q - F_n G_Q Z_p \tag{50}$$

is positive if $G_Q < 0$ and G_n is small in magnitude. (Clearly, these are not necessary conditions for $\det(\mathbf{M}) > 0$.) For instance, $G_Q < 0$ holds if g' is zero or small such that $MRS_Q \leq 0$ (see also Appendix B). According to (13), if $g' = g'' = u_{CH} = u_{HH} = 0$ and $\varepsilon = 1$ in equilibrium, then $MRS_Q = MRS_n = G_n = 0$ and therefore $\det(\mathbf{M}) > 0$ (recall that $F_p > 0$, $F_Q < 0$, $F_n > 0$ and $G_p < 0$).

Using $F_\tau = F_w = F_f = G_f = Z_\tau = Z_w = 0$, if $\det(\mathbf{M}) > 0$, the implicit function theorem implies that

$$sgn\left(\frac{\partial Q^{**}}{\partial \tau}\right) = -sgn\left(F_p G_\tau Z_n - F_n G_\tau Z_p\right), \tag{51}$$

$$sgn\left(\frac{\partial p^{**}}{\partial \tau}\right) = -sgn\left(F_n G_\tau Z_Q - F_Q G_\tau Z_n\right), \tag{52}$$

$$sgn\left(\frac{\partial Q^{**}}{\partial w}\right) = -sgn\left(F_p G_w Z_n - F_n G_w Z_p\right), \tag{53}$$

$$sgn\left(\frac{\partial p^{**}}{\partial w}\right) = -sgn\left(F_n G_w Z_Q - F_Q G_w Z_n\right), \tag{54}$$

$$sgn\left(\frac{\partial Q^{**}}{\partial f}\right) = -sgn\left(F_n G_p Z_f - F_p G_n Z_f\right), \tag{55}$$

$$sgn\left(\frac{\partial n^{**}}{\partial f}\right) = -sgn\left(F_p G_Q Z_f - F_Q G_p Z_f\right). \tag{56}$$

From (51)-(54) we can confirm the impact of an increase in τ and w on Q^{**} and p^{**} (parts (a) and (b)). Concerning part (c), from (55), we find that $\partial Q^{**}/\partial f < 0$ if $G_n \geq 0$, which is fulfilled if (14) holds (see the proof of part (c) of Proposition 1). Finally, (56) implies that $\partial n^{**}/\partial f < 0$ if $G_Q \leq 0$. ■

Proof of Proposition 3: To prove the results for the restricted entry case, recall that $F_Q < 0$, $F_p > 0$, $F_n > 0$ and apply the implicit function theorem. To prove the

results in the case of unrestricted entry, recall that $Z_Q < 0$, $Z_p > 0$, $Z_n < 0$, $Z_f < 0$, $F_f < 0$. Thus, the determinant of matrix $\begin{pmatrix} F_Q & F_n \\ Z_Q & Z_n \end{pmatrix}$ is positive. Applying the implicit function theorem to (18), we thus find that

$$sgn\left(\frac{\partial Q^{**}}{\partial p_{\max}}\right) = -sgn\left(F_p Z_n - F_n Z_p\right) > 0, \tag{57}$$

$$sgn\left(\frac{\partial n^{**}}{\partial p_{\max}}\right) = -sgn\left(F_Q Z_p - F_p Z_Q\right) > 0, \tag{58}$$

$$sgn\left(\frac{\partial Q^{**}}{\partial f}\right) = sgn\left(F_n Z_f\right) < 0, \tag{59}$$

$$sgn\left(\frac{\partial n^{**}}{\partial f}\right) = -sgn\left(F_Q Z_f\right) < 0. \tag{60}$$

This concludes the proof. ■

Proof of Proposition 4: As argued in subsection 5.2, health expenditures for a customer of firm i are $p_i - \bar{p} + T$ (with $T = s\bar{p}$) rather than $\tau p_i + T$ compared to the basic model with coinsurance. Thus, (5) and (6) become

$$\underline{C}_i \equiv wg(\underline{H}_i) - p_i + (1-s)\bar{p},\ \overline{C}_i \equiv wg(\overline{H}_i) - p_i + (1-s)\bar{p}, \tag{61}$$

$$\underline{C}_{i_l} \equiv wg(\underline{H}_{i_l}) - p_{i_l} + (1-s)\bar{p},\ \overline{C}_{i_r} \equiv wg(\overline{H}_{i_r}) - p_{i_r} + (1-s)\bar{p}, \tag{62}$$

With this modification, the conditions for a profit maximum of firms under restricted entry in Lemma 1, eqs. (30)-(40), remain unchanged except that we have to set $\tau = 1$ in (35) and (39). Making use of the facts that $H = h\left(\frac{1}{2n}, Q\right)$ and $p_i = p$ hold under symmetry in (19), we find that equilibrium values (Q^*, p^*) under restricted entry are simultaneously given by

$$0 = F(Q, p, n), \tag{63}$$

$$0 = wg'\left(h\left(\frac{1}{2n}, Q\right)\right) + MRS(Q, p, n, w, \bar{p}) - \frac{nB'(Q)}{sh_Q\left(\frac{1}{2n}, Q\right)} \equiv \bar{G}(Q, p, n, w, \bar{p}), \tag{64}$$

where

$$MRS(Q,p,n,w,\bar{p}) \equiv \frac{u_H\left(wg\left(h\left(\frac{1}{2n},Q\right)\right)-p+(1-s)\bar{p},h\left(\frac{1}{2n},Q\right)\right)}{u_C\left(wg\left(h\left(\frac{1}{2n},Q\right)\right)-p+(1-s)\bar{p},h\left(\frac{1}{2n},Q\right)\right)}. \tag{65}$$

If the number of firms is endogenous, equilibrium values (Q^{**},p^{**},n^{**}) are given by

$$F(Q,p,n)=\bar{G}(Q,p,n,w,\bar{p})=Z(Q,p,n,f)=0. \tag{66}$$

Since MRS is increasing in $\bar{p}$, we have $\bar{G}_{\bar{p}}>0$. The remainder of the proof is then analogous to the proofs of Proposition 1 and 2 (where we used property $G_\tau<0$) and implies that a decrease in $\bar{p}$ has similar effects than an increase in τ in the basic model. A change in the number of firms has a similar effect on function $\bar{G}$ than on function G of the basic model. This concludes the proof. ■

Proof of Lemma 2: As the consumption good sector is competitive, it takes prices for intermediates as given. The inverse demand function for good k thus reads $\partial Y/\partial x(k) = \alpha(A(k)L^Y/x(k))^{1-\alpha} \equiv \mathfrak{p}(x(k))$. Marginal costs are unity. Each firm maximizes $(\mathfrak{p}(x)-1)x$ with respect to x which leads to an optimal price of $1/\alpha$ and therefore

$$x(k)=\alpha^{\frac{2}{1-\alpha}}A(k)L^Y, \tag{67}$$

$k\in[0,N]$. Firm k thus earns profits

$$\begin{aligned}\pi_t(k) &= \left(\frac{1}{\alpha}-1\right)x_t(k)-wl_t^A(k)-w_t\bar{l} &(68)\\ &= (1-\alpha)\alpha^{\frac{1+\alpha}{1-\alpha}}\bar{A}_{t-1}v(l_t^A(k))L^Y-w_tl_t^A(k)-w_t\bar{l}, &(69)\end{aligned}$$

where we used expression (67) for $x(k)$ and (22) for $A(k)$ in the latter equation. Maximizing (69) with respect to R&D labor input leads to first-order condition

$$(1-\alpha)\alpha^{\frac{1+\alpha}{1-\alpha}}\bar{A}_{t-1}v'(l_t^A(k))L^Y=w_t. \tag{70}$$

Free entry in the intermediate goods sector implies that $\pi(k) = 0$ for all k. Combining (70) with the zero-profit condition by using (69) confirms (29).

Next, note that the wage rate is given by

$$w = \frac{\partial Y}{\partial L^Y} = (1-\alpha)(L^Y)^{-\alpha}\int_0^N A(k)^{1-\alpha}x(k)^{\alpha}\mathrm{d}k. \tag{71}$$

Using (67), (22) and $l^A(k) = l^{A*}$ we can write

$$w_t = (1-\alpha)\alpha^{\frac{2\alpha}{1-\alpha}}N_t\bar{A}_{t-1}v(l^{A*}). \tag{72}$$

Combining the two expressions (70) and (72), again using $l^A(k) = l^{A*}$, gives us

$$\alpha N(l^{A*} + \bar{l}) = L^Y. \tag{73}$$

Labor market clearing implies that

$$L^Y = L^S - N(l^{A*} + \bar{l}). \tag{74}$$

Combining (73) and (74) we find that the number of intermediate goods firms is given by

$$N = \frac{L^S}{(1+\alpha)(l^{A*} + \bar{l})}. \tag{75}$$

Hence, as claimed in the text, N is proportional to labor supply, L^S. Dividing (72) by $\bar{A}_t$ and using from (22) that

$$A_t(k) = \bar{A}_t = \bar{A}_{t-1}v(l^{A*}) \tag{76}$$

holds for all k in BGE implies

$$\hat{w} = \frac{w}{\bar{A}} = (1-\alpha)\alpha^{\frac{2\alpha}{1-\alpha}}v(l^A)N. \tag{77}$$

Substituting (75) into (77) and using both (27) and (29) confirms (28). Finally, according to (76), we have $\bar{A}_t/\bar{A}_{t-1} = v(l^{A*})$. This concludes the proof. ■

Proof of Lemma 3: To prove part (a), note that in view of the time-invariance of equilibrium R&D labor input l^{A*}, (25) and (28) give us two equations with two unknowns, $\hat{w}$ and Q, in period t. The two variables thus jump directly to a steady state; therefore also $\hat{p}$ does, according to (24). (27), (74), (75) imply that also L^S, L^Y, N are time-invariant. Moreover, substituting (67) into (20) and using $R = \bar{R} - s - nb(Q)$ as well as (21) we find that output is given by

$$Y_t = \bar{A}_t \left(\alpha^{\frac{2\alpha}{1-\alpha}} N_t L_t^Y + \bar{R} - s - nb(Q_t) \right). \tag{78}$$

Since L^Y, N and Q are time-invariant in equilibrium, Y grows with the same rate as $\bar{A}$ from the initial period onwards. The same is true for individuals' equilibrium consumption of the final good, given by (42), as both w and p grow with the same rate as $\bar{A}$.

To confirm part (b) it suffices to note that when function W is not convex as a function of Q (which may well be the case since W_{QQ} depends on third derivatives of functions b, h and g) then the $W-$curve and $\tilde{W}-$curve may intersect more than once in $Q - \hat{w}-$space. Finally, part (c) can be confirmed by using Fig. 2 (note that the $\tilde{W}-$curve is horizontal, unlike in Fig. 2, if $g'(H) = 0$ for all H). ■

Proof of Proposition 5: See the discussion of the result in the main text. ■

B: Existence and Uniqueness of Equilibrium

Define the right-hand side of (43) as $P(Q)$ and note that $P'(Q) > 0$. To show that an equilibrium exists under weak conditions, consider the following case. Suppose that $B'(0) = 0$ (thus, $P(0) = c$) and $\lim_{Q\to\infty} B'(Q) \to \infty$. Moreover, let $\lim_{C\to 0} u_C \to \infty$ and $G_Q < F_Q G_p / F_p$. Since $u_{CC} < 0$ and $u_{CH} \geq 0$, MRS is decreasing in p, implying $G_p < 0$. Thus, $G_Q < F_Q G_p / F_p$ implies that in $p - Q-$space the $F = 0$ locus (function $P(Q)$) is always steeper than the $G = 0$ locus.

Let $\tilde{Q}$ be given by

$$wg'\left(h\left(\frac{1}{2n},\tilde{Q}\right)\right)=\frac{\tau nB'(\tilde{Q})}{sh_Q\left(\frac{1}{2n},\tilde{Q}\right)} \tag{79}$$

and note that an interior and unique level of $\tilde{Q}$ exists due to the boundary conditions on B' as well as properties $g''\leq 0$, $h_{QQ}\leq 0$ and $B''>0$. Moreover, define

$$\tilde{p}\equiv\frac{wg\left(h\left(\frac{1}{2n},\tilde{Q}\right)\right)}{\tau(1-s)+s}; \tag{80}$$

thus, at $(\tilde{Q},\tilde{p})$ the consumption level is zero and $\lim_{C\to 0}u_C\to\infty$ implies $MRS(\tilde{Q},\tilde{p},\cdot)=0$. Hence, $G(\tilde{Q},\tilde{p},\cdot)=0$, according to (12) and (79). Now suppose $\tilde{p}>P(\tilde{Q})$. This means that at $Q=\tilde{Q}$, the $G=0$ locus is above the $F=0$ locus in $p-Q-$space. Since the latter is steeper than the former, there is exactly one intersection point of function $P(Q)$ and the $G=0$ locus, i.e., the equilibrium exists and is unique.

To lead back existence and uniqueness of equilibrium to the primitives of the model, note from $F_Q<0$, $G_p<0$ and $F_p>0$ that $G_Q<F_QG_p/F_p$ always holds if $G_Q<0$. A sufficient (but not necessary) condition for $G_Q<0$ is $MRS_Q\leq 0$. For instance, $MRS_Q\leq 0$ holds if g' is small or $|g''|$ is large, according to (13). ($MRS_Q<0$ if $g'=0$.) Moreover, note from (79) and (80) that $\tilde{Q}$ and $\tilde{p}$ do not depend on marginal cost c. Thus, using (43), we have $\tilde{p}>P(\tilde{Q})$ if c is sufficiently small.

C: Two-stage Decision

Suppose that, alternatively to the analysis in the main body of the paper, firms engage in a two-stage decision process. At stage 1, they choose the type of horizontal differentiation along with the vertical quality component. At stage 2, they choose prices (product market competition). There are two ways to analyze the model in this case. First, firms foresee the Bertrand equilibrium for any vector of horizontal and vertical location of firms and take the related equilibrium responses into account at stage 1. Unfortunately, the analysis becomes intractable.[19]

[19]Lancaster (1979) and applications of the ideal variety model in the context of goods trade also

The second way to analyze the two-stage problem is to assume that at stage 1 firms take prices of other firms as given (along with product quality and horizontal location) and therefore only foresee the impact of their choices on their price setting power for given prices of rivals. In this case, the behavior of firms is exactly the same as in the case where there is just one decision stage.

To see this, note that at stage 2 the optimal price of each firm fulfills first-order condition (32), which gives us the optimal price of firm i. Recalling that $\underline{\Delta}_i$ is a function of $p_i, Q_i, \delta_i(i_l), p_{i_l}, Q_{i_l}$ and $\overline{\Delta}_i$ is a function of $p_i, Q_i, D_i - \delta_i(i_l), p_{i_r}, Q_{i_r}$, we see that (32) gives us p_i implicitly as a function of $Q_i, \delta_i(i_l), p_{i_l}, p_{i_r}, Q_{i_l}, Q_{i_r}$. Write $p_i = \tilde{P}_i(Q_i, \delta_i(i_l), p_{i_l}, p_{i_r}, Q_{i_l}, Q_{i_r})$. Now, the optimization problem at stage 1 is:

$$\max_{\delta_i(i_l), Q_i} \left[\tilde{P}(Q_i, \delta_i(i_l), \cdot) - c\right] s \left[\underline{\Delta}_i(\tilde{P}(Q_i, \delta_i(i_l), \cdot), Q_i, \delta_i(i_l), \cdot) + \overline{\Delta}_i(\tilde{P}(Q_i, \delta_i(i_l), \cdot), Q_i, D_i - \delta_i(i_l), \cdot\right] - B(Q_i) - f, \tag{81}$$

where firms take as given $p_{i_l}, p_{i_r}, Q_{i_l}, Q_{i_r}, D_i$. The first-order condition with respect to vertical differentiation Q_i is:

$$\begin{aligned} 0 &= (p_i - c)s\left(\frac{\partial \underline{\Delta}_i}{\partial Q_i} + \frac{\partial \overline{\Delta}_i}{\partial Q_i}\right) - B'(Q_i) + \\ &\quad \frac{\partial \tilde{P}_i}{\partial Q_i} s\left[\underline{\Delta}_i + \overline{\Delta}_i + (p_i - c)\left(\frac{\partial \underline{\Delta}_i}{\partial p_i} + \frac{\partial \overline{\Delta}_i}{\partial p_i}\right)\right]. \end{aligned} \tag{82}$$

Applying the envelope theorem, the term is squared brackets of (82) becomes zero, according to stage 2 first-order condition (32). Thus, (82) coincides with first-order condition (31) of the profit maximization problem (10). An analogous argument holds for the first-order condition with respect to horizontal differentiation $\delta_i(i_l)$ associated with profit maximization problem (81); it coincides with (30). This confirms the claim.

focus on simultaneous choices of horizontal location of firms and prices.

References

Acemoglu, Daron, David Cutler, Amy Finkelstein and Joshua Linn (2006). Did Medicare Induce Pharmaceutical Innovation? *American Economic Association Papers and Proceedings* 96, 103-107.

Acemoglu, Daron and Joshua Linn (2004). Market Size in Innovation: Theory and Evidence from the Pharmaceutical Industry, *Quarterly Journal of Economics* 119, 1049–1090.

Acemoglu, Daron and Simon Johnson (2007). Disease and Development: The Effect of Life Expectancy on Economic Growth, *Journal of Political Economy* 115, 925-985.

Aghion, Philippe, Nicolas Bloom, Richard Blundell, Rachel Griffith and Peter Howitt (2005). Competition and Innovation: An Inverted U Relationship, *Quarterly Journal of Economics* 120, 701-728.

Aghion, Philippe, Richard Blundell, Rachel Griffith, Peter Howitt and Susanne Prantl (2009). The Effects of Entry on Incumbent Innovation and Productivity, *Review of Economics and Statistics* 91, 20-32.

Aghion, Philippe and Peter Howitt (2005). Growth with Quality-improving Innovations: An Integrated Framework, in: P. Aghion and S. Durlauf (eds.), *Handbook of Economic Growth*, pp. 67-110, Amsterdam: North-Holland.

Aghion, Philippe and Peter Howitt (2009). The Economics of Growth, Cambridge MA, MIT Press.

Aghion, Philippe, Peter Howitt and Fabrice Murtin (2010). The Relationship between Health and Growth: When Lucas Meets Nelson-Phelps, NBER Working Paper No. 15813.

Austvoll-Dahlgren, Astrid, Morten Aaserud, Gunn Elisabeth Vist, Craig Ramsay, Andrew D Oxman, Heidrun Sturm, Jan Peter Kösters, Åsa Vernby (2008). Pharmaceutical Policies: Effects of Cap and Co-payment on Rational Drug Use, *Cochrane Database of Systematic Reviews 2008*, Issue 1. Art. No.: CD007017.

Berndt, Ernst R. (2002). Pharmaceuticals in U.S. Health Care: Determinants of Quantity and Price, *Journal of Economic Perspectives* 16, 45-66.

Blundell, Richard, Rachel Griffith and John Van Reenen (1999). Market Share, Market Value and Innovation in a Panel of British Manufacturing Firms, *Review of Economic Studies* 66, 529-554.

Cai, Lixin, Kostas Mavromaras and Umut Oguzoglu (2008). The Effects of Health and Health Shocks on Hours Worked, IZA Discussion Paper No. 3496.

Dasgupta, Partha and Stiglitz, Joseph (1980). Industrial Structure and the Nature of Innovative Activity, *Economic Journal* 90, 266-93

Dixit, Avinash and Joseph E. Stiglitz (1977). Monopolistic Competition and Optimum Product Diversity, *American Economic Review* 67, 297-308.

Djankov, Simeon, Rafael La Porta, Florenzio Lopez-de-Silanes and Andrei Shleifer (2002). The Regulation of Entry, *Quarterly Journal of Economics* 116, 1-37.

Ethier, William J. (1982). National and International Returns to Scale in the Modern Theory of International Trade, *American Economic Review* 72, 389-405.

Garber, Alan M., Charles I. Jones and Paul Romer (2006). Insurance and Incentives for Medical Innovation, *Forum for Health Economics & Policy* 9 (2), Article 4, Berkeley Electronic Press.

Gemmill, Marin C., Sarah Thomson and Elias Mossialos (2008). What Impact Do Prescription Drug Charges Have on Efficiency and Equity, *International Journal for Equity in Health* 7:12.

Giaccotto, Carmelo, Rexford E. Santerre and John A. Vernon (2005). Drug Prices and Research and Development Investment Behavior in the Pharmaceutical Industry, *Journal of Law and Economics* 48, 195-214.

Goldman, Dana P., Geoffrey F. Joyce, Yuhui Zheng (2007). Prescription Drug Cost Sharing: Associations with Medication and Medical Utilization and Spending and Health, *Journal of the American Medical Association* 298, 61-69.

Grossman, Gene M. and Elhanan Helpman (1991). Quality Ladders in the Theory of Growth, *Review of Economic Studies* 58, 43-61.

Helpman, Elhanan (1981). International Trade in the Presence of Product Differentiation, Economies of Scale and Monopolistic Competition, *Journal of International Economics* 11, 305-340.

Hummels, David and Volodymyr Lugovsky (2009). Trade in Ideal Varieties: Theory and Evidence, *Journal of Money, Credit and Banking* 41, 3-33.

Jones, Charles I. (1995). Time Series Tests of Endogenous Growth Models, *Quarterly Journal of Economics* 110, 495-525.

Jones, Charles I. and John C. Williams (2000). Too Much of a Good Thing? The Economics of Investment in R&D, *Journal of Economic Growth* 5, 65-85.

Lakdawalla, Darius and Neeraj Sood (2005). Insurance and Innovation in Health Care Markets, NBER Working Paper No. 11602.

Lakdawalla, Darius and Neeraj Sood (2009). Innovation and the Welfare Effects of Public Drug Insurance, *Journal of Public Economics* 93, 541-48.

Lancaster, Kelvin (1979). Variety, Equity, and Efficiency, Columbia University Press.

Landsman, Pamela B., Winnie Yu, XiaoFeng Liu, Steven M. Teutsch and Marc L. Berger (2005). Impact of 3-Tier Pharmacy Benefit Design and Increased Consumer Cost-sharing on Drug Utilization, *American Journal of Managed Care* 11, 621-628.

Leibowitz, Arleen, Willard G. Manning and Joseph P. Newhouse (1985). The Demand for Prescription Drugs as a Function of Cost-sharing, *Social Science and Medicine* 10, 1063-1069.

Motheral, Brenda R. and Rochelle Henderson (1999). The Effect of a Copay Increase on Pharmaceutical Utilization, Expenditures, and Treatment Continuation, *American Journal of Managed Care* 5, 1383-1394.

OECD (2010). Health at a Glance: Europe 2010, Paris.

Romer, Paul M. (1990). Endogenous Technological Change, *Journal of Political Economy* 98, 71-101.

Sanso, Marcos and Rosa M. Aísa (2006). Endogenous Longevity, Biological Deterioration and Economic Growth, *Journal of Health Economics* 25, 555-578.

Scherer, F.M. (1993). Pricing, Profits, and Technological Progress in the Pharmaceutical Industry, *Journal of Economic Perspectives* 7, 97-115.

Sutton, John (1991). Sunk Costs and Market Structure: Price Competition, Advertising, and the Evolution of Concentration, MIT Press.

Sutton, John (1998). Technology and Market Structure, MIT Press.

The Economist (2011). Resistance to Antibiotics: The Spread of Superbugs, April 2nd, 70-72.

Vernon, John A. (2005). Examining the Link between Price Regulation and Pharmaceutical R&D Investment, *Health Economics* 14, 1-16.

Van Zona, Adriaan and Joan Muysken (2001). Health and Endogenous Growth, *Journal of Health Economics* 20, 169-185.

Wong, Kar-yiu (1995). International Trade in Goods and Factor Mobility, MIT Press.

Young, Alwyn (1998). Growth without Scale Effects, *Journal of Political Economy* 106, 41–63.