ESSAYS IN INDUSTRIAL ORGANIZATION AND HEALTH ECONOMICS

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This dissertation is a collection of three independent essays in industrial organization and health economics. The first two essays discuss the relationship between patents and firms' incentives in the design and timing of new product introductions in the pharmaceutical industry. The third essay discusses consumer incentives generated by health insurance plans, examining whether more generous health insurance encourages people to smoke more.

Essay 1 studies when companies are doing R&D, to what extent they do it in-house, to what extent they outsource it, and how do firms' incentives in patent protection affect their integration decisions in new product development. Essay 1 asserts that a firm may want to protect its own patented products from becoming obsolete when introducing new ones in the same product area. In particular, a firm may want to invest to ensure minimal product substitutability when the firm's existing patents are of high value. Under an incomplete contract framework, my model incorporates such an incentive in patent protection, and predicts that a firm is more likely to develop a new product in-house if it already owns at least one existing patented product in the same area. This likelihood also increases both with the patent length and the expected market share of the firm's existing product(s) by the time the new one reaches the market. The pharmaceutical sector is a natural setting to test these predictions. Controlling for firm characteristics and therapeutic class heterogeneity, my empirical findings are consistent with the theory.

Essay 2 explores firms' timing strategies in new compound development in the pharmaceutical industry, particularly how firms make pipeline planning and licensing deci-

sions when their existing drugs are approaching patent expiration. Using compound development data between 1989 and 2004, I find that controlling for heterogeneity in firm characteristics, a firm is more likely to advance its pipeline compounds to a higher level of development when the firm also has drugs with expiring patents in the same therapeutic class. In contrast, this effect is weaker when the expiring patents belong to a different class. Additionally, the likelihood of a firm licensing out a late stage compound is lower when the firm has expiring patents in that compound's same class. Yet this negative association is again weaker when the expiring patents and the compound of interest are from different classes. These results are all consistent with a firm's incentive to introduce a new patented drug in a timely manner so that customers can switch from the old drug about to go off-patent to the new one.

Essay 3 uses data from the Rand Health Insurance Experiment (HIE) to study whether insurance plans create incentives for individuals to smoke. I exploit the exogenous variation of coinsurance rates in the HIE, and find that the benefit from a more generous plan has to be large for non-smokers to start smoking, while smaller benefits suffice for smokers to continue smoking. Additionally, older individuals are more prone to moral hazard in the HIE than younger ones. This difference is consistent with the notion that older individuals have a higher expected benefit from low coinsurance than younger ones in the HIE.

BIOGRAPHICAL SKETCH

Yijun Pan was born in Shanghai, China to parents who greatly value and encourage intellectual pursuits. At age seven, she accompanied her parents to Australia, where her dad completed a master's degree in education. While in Australia, Yijun attended a local elementary school, an experience which significantly broadened her horizons at a young age and inspired her to pursue her own higher education abroad later on. After attending middle and high school back in Shanghai, at age 17 she came to the United States for her undergraduate degree at the University of Miami. There, she double majored in Mathematics and Economics, and minored in Finance. She was inducted into Phi Beta Kappa in her junior year, and graduated Summa Cum Laude with Honors in May 2011. Intrigued by the economist's way of thinking, she was eager to continue her studies in economics immediately after college, and realized this goal by entering Cornell's Ph.D. program. The following three essays are the end result of her five-year training at Cornell.

To my parents, Henson Pan and Shirley Lu, and my husband, Christopher Cairns, with whom I share the road of life.

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First and foremost, I am deeply indebted to my dissertation committee, in particular my chair Professor Michael Waldman, but also Professors Sean Nicholson, George Jakubson and Catherine Barrera. Their tireless guidance and insights were invaluable both intellectually and emotionally in swimming the vast ocean of doing an economics PhD. The final product owes as much to their efforts as to my own.

I would also like to acknowledge the unceasing love and support my parents have given me for the past 26 years. Their actions and words taught me the value of hard work, persistence and courage. I am truly grateful for the precious opportunities that they have provided me.

Finally, I must single out my husband and fellow doctoral Cornellian, Christopher Cairns. He has offered tremendous support at each stage of my Ph.D. program, even reading and critiquing numerous drafts. He now knows far more about incentives in economic theory and unobservables in econometric estimation than he ever wished to. I could not have completed this process without him.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER 1:

PATENTS, IN-HOUSE PIPELINES, AND PLANNED ANTI-OBSOLESCENCE: THEORY AND EVIDENCE FROM THE PHARMACEUTICAL INDUSTRY

1.1 Introduction

Research and development (R&D) intensive firms frequently face the question of whether to conduct R&D in-house or outsource. Despite the rising trend of outsourcing, partnering with outside firms does not come free of concern. Peter Chambre, chief executive of Cambridge Antibody Technology (CAT), once remarked that "he would prefer to delay licensing products to drug companies until they are at a later stage to generate more value from the company's technology" (Mills, 2004). Jim Hall, president of life science at advisory firm Wood Mackenzie recalls "a $60-$70 million deal between a large pharma and medium-sized biotech falling apart when the biotech found that the more 'successful' the alliance became, the more money it would lose" (Lam, 2004). Indeed, incompatible objectives is ranked as the number two reason for alliance failure in 1999 and again making the top eight list in 2003 (Lam, 2004).

For industries that rely heavily on the intellectual property rights legal regime, this paper points to patent ownership as one of the sources of disparity in firm objectives and identifies an important link between patent ownership and integration decisions in development. I model a firm's choice between in-house and outsourced development on the project level by incorporating the idea of planned *anti*-obsolescence. That is, in crowded product areas with many existing goods under patent, firms may want to protect their own patented products from becoming obsolete when introducing new ones in the same product class. My model generates a set of new predictions about how the incentive to protect the patent value of a firm's existing products can affect in-house

versus outsourced development decisions for its new products. I find empirical support for these predictions in the pharmaceutical industry.

To model a firm's in-house and outsource decisions, I consider a setting where there is a cost advantage to outsourcing but development outcomes are uncertain. Firms cannot control the exact location of a new product relative to the existing ones in product space, but instead choose a mean location and an investment level for location precision. Each realization of the new product's location is associated with an error term that can be drawn from one of two distributions: both are uniform with mean zero, but one has a higher variance than the other. I assume that more investment increases the probability of drawing from the distribution with a smaller variance. The choices of mean location and investment level are examined in a four period model, where I further assume that the originator of the new product also owns an existing patented product in the same product class as the new one. The patent for the existing product can expire in the second, third or fourth period. For each scenario, my model compares the development choices made by the originator when the new product is kept in-house to those made by the licensees when the new product is outsourced.

The main intuition behind the model is that the value of an existing patent is only internalized by the firm that owns it. If a product is developed in-house, the originating firm can prevent patent devaluation for its existing products by maximally differentiating its new product from the old ones in the same product class. However, if a product is outsourced, the licensees do not have the incentive to internalize the value of the originator's existing patents. While both the originator and the licensees want to limit substitutability from the standpoint of the new product's profitability, the originator has an additional motivation to ensure maximal differentiation (through investment for location precision) from the standpoint of protecting its own existing patents. Therefore,

the originator balances the trade-off between practicing planned *anti*-obsolescence by keeping the new product in-house and savings in development cost by outsourcing the new product.

My model generates three testable predictions. First, compared to firms that do not own existing patented products, those that do are more likely to develop new products in the same product class as the existing ones in-house in order to prevent patent devaluation. Second, as the remaining patent duration for a firm's existing products increases, the probability of in-house development for new products in the same class also rises. Third, a firm is also more likely to develop a new product in-house the more market share the firm expects to have of its existing patented products in the same product area as the new one by the time the new compound reaches the market.

I test the model's predictions within the pharmaceutical sector, where the patent system is a defining feature of the industry and a large number of firms employ both in-house and outsourced development strategies. Using detailed compound-level data from the Pharmaprojects dataset, I find a positive relationship between in-house decisions and patent existence as well as patent length for other compounds in the same therapeutic class and same firm as the compound in development. The empirical results also show how the market position of a firm can affect the probability of in-house development. Holding the total number of drugs on the market fixed within a therapeutic class, the more patented drugs other firms own on the market, the less incentive the firm of interest has in protecting the patent value of its existing compounds in that therapeutic class. Furthermore, by supplementing the main data source with the IMS sales data, I find that a firm is more likely to develop a new compound in-house the more market share the firm expects to have from its same-class patented drugs by the time the new compound reaches the market. Overall, the findings are consistent with the theory of planned *anti*-

obsolescence in explaining integration decisions in development.

The following section gives a brief overview of the related literature. Section 1.3 describes a model for a firm's in-house and outsource decisions by employing the idea of planned *anti*-obsolescence. In Section 1.4, I apply the model to the pharmaceutical industry, and provide summary statistics of the data sets used in the paper. Section 1.5 tests the model's predictions and presents the empirical findings. Section 1.6 discusses alternative explanations. I conclude in Section 1.7.

1.2 Related Literature

This paper brings together literature on planned obsolescence, firm integration decisions with a focus on the organization of innovation, and empirical studies on alliances in the pharmaceutical industry.

The concept of planned *anti*-obsolescence introduced in this paper is closely related to and in fact stems from the much researched topic of planned obsolescence in the economics literature. Extant theoretical work on planned obsolescence can be divided into two streams. The first stream considers the choice of durability faced by monopolists of durable goods. Most of these papers have focused on whether firms have the incentive to reduce the durability of a good to maximize profit, and whether the level of durability is socially optimal.[1] The second stream of work largely follows from Bulow's (1986) point that "planned obsolescence is much more than a matter of durability; it is also and perhaps primarily about how often a firm will introduce a new product". In this type of model, durable goods monopolists face a time inconsistency problem: in order to max-

[1] Work that focus on the choice of durability in the planned obsolescence literature include for example Swan (1972), Bulow (1986), and Carlton and Perloff (2005).

imize current period profits, the monopolists have too high of an incentive to introduce new goods and kill off old ones from the standpoint of both firm profitability and social welfare (Waldman, 1993, 1996; Choi, 1994).[2]

A common idea in the above papers is that planned obsolescence arises because monopolists are selling instead of renting their durable goods. By selling, monopolists do not internalize how the value of goods sold in the previous period can be affected by the value of new goods sold in the current period when maximizing current period profit. If monopolists are allowed to rent, however, then they retain ownership of the goods, and thus can avoid having such a high incentive to practice planned obsolescence that their own profitability is reduced (Waldman, 1996).

In this paper, I examine a related but different situation of new product introduction in which the goods themselves are not durable, but the associated patents are. Because the only durability is in the patents, the standard argument of time inconsistency does not apply to the products. Nevertheless, the case of patent-holding firms largely resembles that of a monopolist who is renting a good in the sense that firms retain the ownership of patents even though the products are being sold on the market. Consequently, firms have an incentive to internalize the value of their existing patents in new project development — a behavior referred to as planned *anti*-obsolescence in this paper. More specifically, in order to prevent goods under patent from becoming obsolete, firms would want to control the design and nature of new products such that these new goods do not steal market share away from the firms' own patented products. The key point here is that planned *anti*-obsolescence arises precisely because firms retain patent ownership.

[2]More theoretical discussion on new product introduction in the planned obsolescence literature include papers such as Fudenberg and Tirole (1998), and Nahm (2004). Recent empirical work has tested the theory by using data from the college textbooks industry, largely because college textbooks have a well-defined used goods market (Chevalier & Goolsbee, 2009). There is evidence that publishers introduce new editions more frequently when competition from used books increases (Iizuka, 2007).

A second body of work that this paper relates to focuses on the integration decisions of a firm. Theories on the topic broadly fall into two categories: one emphasizes transaction costs across firm boundaries, and the other focuses on the provision of property rights.[3] The second approach, which is of particular relevance to my paper, uses the incomplete contract framework first proposed by Grossman and Hart (1986). In their model, two parties both make distinct ex ante investments that are non-contractible; and utility is non-transferable ex ante. Grossman and Hart (1986) show that party 1 purchases party 2 when the former's investment is more important than the latter's. A related study by Aghion and Tirole (1994) uses the incomplete contract framework to examine the organization of innovation. They suggest that when capital input is more important than intellectual input, research is more likely to be conducted under an integrated structure; *vice versa*, research is more likely to be conducted by an independent unit. Lerner and Merges (1998) find empirical evidence on the positive relationship between a firm's financial capabilities and its control rights to R&D.

This paper fits into the property rights approach in that it also employs incomplete contracting to analyze integration decisions in development. I assume that all ex ante development decisions are non-contractible as in Grossman and Hart (1986). An integration case is one in which an originating firm develops independently and is able to maintain control in shaping its new products and limiting substitutability; a non-integration case is one in which the originator outsources development from outside firms and loses some control in product design. I show that giving full control to the originator in developing the new product is optimal when it is more important to protect the patent value of the old product than to reduce the fixed cost of development for the new product.

In addition to the Grossman and Hart-like results regarding the integration decisions,

[3] See Gibbons (2005) for a recent survey on the topic.

my modeling approach is also able to shed new light on the relationship between the incentive to protect the patent value of existing goods and the size of investment in new product development. To explicitly introduce the idea of planned *anti*-obsolescence, I set up a model where the importance of the originator's investment lies entirely in its incentive to internalize the old patent value while the importance of the licensee's investment comes entirely from a reduction of fixed costs. In other words, my model differs from that of Grossman and Hart (1986), and of Aghion and Tirole (1994) in that the nature of the input — namely, the investment technology to improve location precision — is exactly the same (instead of being distinct) between the originator and the licensee. Hence, any investment discrepancy is solely attributable to whether or not each party cares about the old patent value in new product development. This leads to an important theoretical result of the paper that is new to existing work: because of the incentive to prevent patent devaluation, the investment level for location precision in the integration case is weakly higher than in the non-integration case.

Finally, most of the research on firm boundaries in the pharmaceutical industry has studied either why firms form alliances, or the outcomes of alliances. Nicholson et al. (2005) show that biotech companies send positive signals to investors by allying with larger pharmaceutical firms. Danzon et al. (2005) find that success rates of more complex phase 2 and phase 3 trials are higher for products developed in an alliance. Few papers focus on the specific characteristics of R&D projects that firms choose to do in-house or outsource. Azoulay (2004) finds that pharmaceutical firms are more likely to outsource data-intensive clinical trials while assigning knowledge-intensive trials to internal teams. This paper is the first that offers a patent protection angle to pharmaceutical alliance decisions on the project level.

1.3 Theoretical Analysis

In this section, I set up a four-period model that incorporates the idea of planned *anti*-obsolescence into a firm's decision on whether to develop a new product internally or not. The analysis consists of two parts. The first part compares the development decisions between the in-house and outsource cases. The second part examines a firm's choice of internal or external development and derives testable predictions. I discuss additional theoretical considerations at the end of this section.

1.3.1 The Model

In a four-period model with no discounting, consider a risk neutral firm (originator) that owns an existing patented product, and is thinking of developing a new one in the same product area. There is a pool of competitive risk neutral in-licensing firms (licensees). The licensees have a cost advantage in development compared to the originator. In particular, the fixed cost of development is F for the originator, and γF (where $0 < \gamma < 1$) for the licensees.[4] All firms have a marginal cost of production equal to zero. The licensees have a reservation profit of $\tilde{\pi}$. Assume that the new product's patent lasts for all four periods while the patent for the old product could expire in periods 2, 3, or 4. The originator knows the duration of the old patent when deciding whether to develop the new product in-house or not.

In the in-house case, the originator makes all the decisions related to new product development and pricing. In the outsource case, the originator writes a contract with

[4]The cost advantage that the licensees have over the originator could also be thought of as the former having less of a financial constraint than the latter. In the pharmaceutical industry, there are many instances where large pharma companies are the licensees and small biotech firms are the originators. The fixed cost of development is often lower for the large pharma companies than for the small biotech firms.

the licensee. The contract specifies a sharing rule on the profits from the new product, and an allocation of the development rights. I make a simplifying assumption on the sharing rule by having the originator charge a lump sum license fee and having the licensee bear all the consequences of its action. The contract delegates pricing decisions to the originator but development rights to the licensee. Note that in the absence of any information distortion, an optimal contract for the originator would specify exactly the actions that the licensee needs to take in development in order to ensure joint profit maximization of the originator's old and new products. However, given the possibility of an information distortion, I assume in the current setting that the development decisions are non-verifiable and hence non-contractible. I return to this issue later and discuss additional related theoretical considerations.

Following Salop's (1979) model, the product space is characterized by a unit circle. The location of the new product depends on the non-contractible development decisions. That is, the firm developing the new product (either originator or licensee) determines where to place it on the circle relative to the existing patented one. Due to uncertainties in development, the developer cannot control the exact location of the new product, but instead chooses a mean for location, $\bar{l}$, and an investment level, K, that affects the range of possible locations that the product could fall into. The clockwise distance between the new product and the existing patented one is given by $l = \bar{l} + \epsilon$, where ϵ is an error term that follows one of two possible uniform distributions, either $U[-\alpha, \alpha]$ or $U[-\beta, \beta]$ with $\alpha < \beta$. For tractability, I limit the error range to be no more than half the unit circle, $\beta \leq \frac{1}{4}$.[5] The higher the investment level, the more likely that the uniform distribution of ϵ will have the smaller of the two variances. Let $p(K)$ denote the probability that ϵ follows $U[-\alpha, \alpha]$ under investment level K. It is assumed that $p(0) = 0$, $p'(0) = \infty$, $p'(K) > 0$ and $p''(K) < 0$ for all $K \geq 0$, and $p(\infty) < 1$. All

[5]This condition ensures that after the patent for the old product expires, the pricing equilibrium is such that some people still strictly prefer the new product.

firms have the same technology in locating the new product, but the originator and the licensees could have different incentives in development, and thus different investment amounts to affect location precision.

On the demand side, there is a continuum of consumers of mass M uniformly located along the circumference of the unit circle. Consumers can buy multiple units of the same product in each location.[6] Their valuation for each additional unit decreases, and takes the form $V(q) = \overline{V} - vq$. There is a travel cost associated with each unit purchased. The cost function is $c(s) = cs$, where s is the distance between the consumer and the product on the unit circle. Assume $\overline{V} >> c$ so that the market is always fully covered. For any product price P, a consumer purchases an optimal quantity that maximizes net total utility, $\int_0^q (\overline{V} - vq)dq - q(P + cs)$. Each consumer chooses the product that gives the highest net total utility under the optimal quantity.[7]

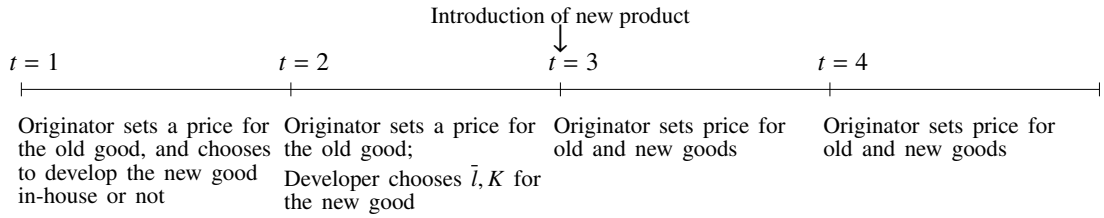
Figure 1.1 depicts the timing of the model. In the first period, the originator chooses a price for its existing patented product, and decides whether to develop the new product on its own or outsource development to a licensee. In the second period, the originator prices the old product again. The developer (either the originator or the licensee) chooses a mean for the new product location and an investment level for the product range. Development takes one period. At the beginning of the third period, the developer brings the new product on the market.[8] The originator chooses a price for both its

[6]One could also model a situation in which each consumer buys zero or one unit of a product, but there are multiple consumers in each location. That is, in each location, there is a continuum of consumers of mass M; the consumer valuations $V_i's$ are distributed uniformly over $[0, \overline{V}]$. This slightly different set up offers an alternative way of thinking about the model while all the mathematical derivations from the original set up will still follow through.

[7]Consumers buy either the old product or the new one, but never both at the same time. This is consistent with typical demand behavior in the pharmaceutical industry. For example, individuals who are being treated for depression seldom take different types of anti-depressants at the same time due to concerns for possible unwanted drug interaction.

[8]The theoretical model abstracts away from the probability that a new product could fail in development. This abstraction does not affect the model's predictions in a substantive way. The reason is that even if failure probabilities are incorporated, the misaligned incentives between the originator and the licensee still persist regarding whether to internalize the patent value of the originator's existing goods in

Figure 1.1: Time Line



old product and the new one. If the patent for the old product expires at the beginning of that period, generic entry causes the old product to sell at a price equal to marginal cost. This pricing behavior is repeated in the fourth period. Consumers observe product price in each period. They decide whether to buy the existing old product or the new one, and how much to buy given the product of their choice. I focus on subgame-perfect Nash equilibria.

1.3.2 Analysis and Testable Predictions

Both the in-house and outsource cases involve a number of actions, including product development, pricing, and consumer purchase decisions. An important point to note is that once product location is determined, firm pricing and consumer purchase decisions are all independent of where development takes place (either internally or externally). The location decision itself, however, is sensitive to whether the new product is developed in-house or not. To solve the model, I start by taking product location as fixed and derive consumer demand and firm pricing strategies. Given product demand and price as a function of location, I then derive the development decisions in the in-house and outsource cases. Appendix A gives the objective functions and the first order conditions

new product development. This being said, the section on empirical evidence from the pharmaceutical industry does use measures of patent existence and length that take into account failure probabilities.

for a firm's choice of mean product location and investment level.

This subsection focuses on analyzing the general results. First, I describe what happens when the originator develops its new product in-house and when the originator outsources development. In the second part, I discuss the trade-offs between in-house and outsourced development, and derive a set of testable predictions.

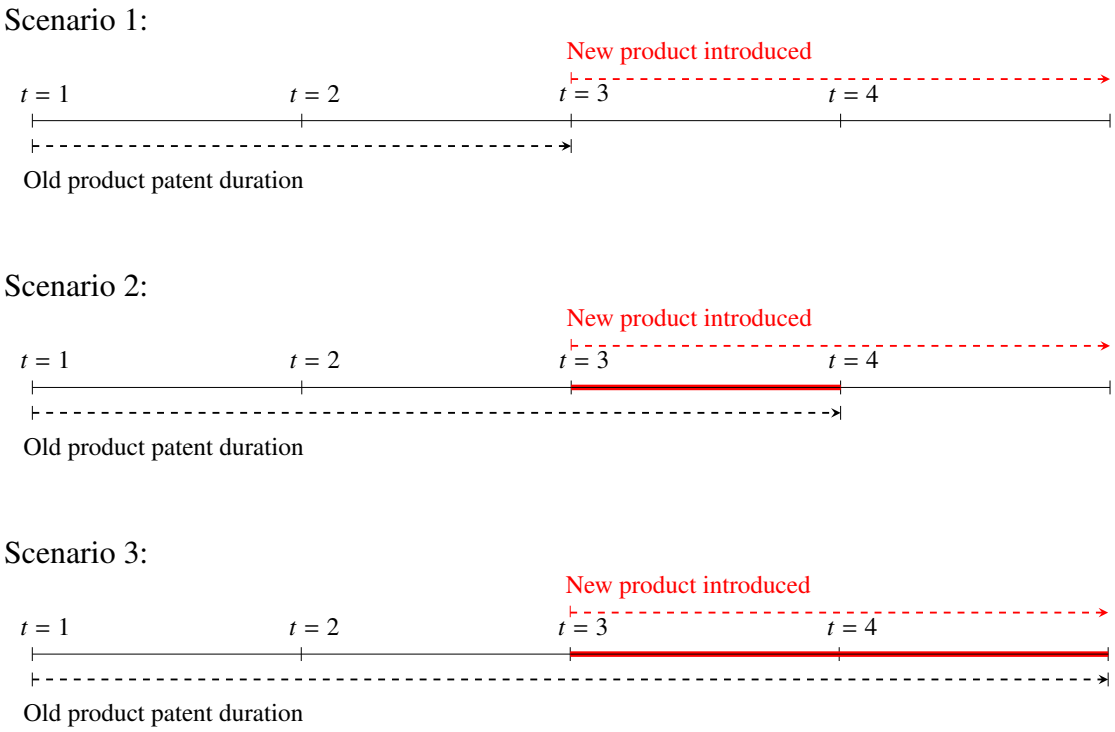
1.3.2.1 A Comparison of In-house and Outsource Development Decisions

For each of the three scenarios regarding when the old product's patent expires, either at the end of period 2, 3, or 4, I compare the development decisions between the in-house and outsource cases. The main intuition is that, because the originator owns the old product's patent, in-house development means the originator will maximize joint profit from the old and the new products. If development is instead outsourced, the licensee does not internalize the value of the originator's old product and thus only maximizes profit from the new one.

The top panel of Figure 1.2 shows the first scenario in which the patent for the old product expires at the end of the second period — before the new product is introduced. Following patent expiration, there is generic entry. As a result, the price of the old product in the third and fourth periods equals marginal cost. In other words, profit from the old product is zero in the subsequent two periods, regardless of where the new product is placed in product space. Furthermore, because the new product only appears on the market in the third period, it does not affect profit from the old product in the first two periods. As a result, the originator has in fact the same development incentives as the licensee: both maximize total profit from the new product.

In contrast to the first scenario, the new product location will affect the old product's

Figure 1.2: Comparison of Scenarios



price and profit when the old patent expires at the end of periods 3 and 4 under the second and third scenarios, respectively. As illustrated in the middle and bottom panel of Figure 1.2, in both of those scenarios, the old product's patent protection time overlaps with the new product's market time. The only difference between the two scenarios is the length of the old patent. As the old patent lasts longer, the location of the new product will affect more periods of the old product's profit. In turn, development incentives will differ between the in-house and outsource cases — unlike the licensee, the originator cares about protecting the patent value of the old product when developing the new one.

In the following proposition, I systematically compare across all three scenarios the originator's and the licensee's decisions concerning new product development. Let $\bar{l}^I(t)$ and $K^I(t)$ denote the in-house (I) choices of mean product location and investment level as functions of the old patent length t. Similarly, $\bar{l}^O(t)$ and $K^O(t)$ are the outsource (O)

counterparts.

Proposition 1. Suppose a firm owns two products in the same class and the patent of the old product lasts for t periods, $t \in \{2, 3, 4\}$. *The in-house and outsource decisions on mean product location and investment level are given by (i) through (iii):*

(i) $\bar{l}^I(t) = \bar{l}^O(t) = \frac{1}{2}, \forall t \in \{2, 3, 4\}$.

(ii) $K^I(2) = K^O(2)$; $K^I(t) > K^O(t), \forall t \in \{3, 4\}$.

(iii) Both $K^I(t)$ *and* $K^O(t)$ *are increasing in t.*

Proposition 1 shows that the mean location of the new product is always the same regardless of when the old patent expires and where development takes place. The reason is that, maximizing the new product's profit is in the interest of both the originator and the licensee. Furthermore, the mean location of the new product is at maximal distance from the old one on the unit circle. This is because more product differentiation increases the profit from the new product (the old product's profit also increases when it is under patent protection).

Additionally, investment in location precision is the same between in-house and outsourced development only when the new product's location does not affect the old product's profit. This corresponds to the scenario in which the old patent lasts for two periods (i.e., $t = 2$). When $t = 3$ or 4, the investment amount is higher in the in-house case than in the outsource case. The logic is as follows. While the originator makes investment decisions to maximize joint profit from both products, the licensee ignores the effect of its investment choice on the profitability of the originator's old product. Hence, when profit from the old product depends on the location of the new one (i.e., $t = 3, 4$), the return from investment is higher for the originator than for the licensee — the additional return for the originator is increased expected profit from the old product.

The proposition also shows that within each case (either in-house or outsourced), investment level increases with the patent length of the old product. The longer the old patent lasts, the higher the benefit from a more precise placement of the new product — for the originator, the benefit is increased expected profits from both products, while for the licensee, the benefit is increased expected profit from the new product.

1.3.2.2 In-house or Outsource?

I now consider an originating firm's choice concerning in-house versus outsourced development. The firm faces a trade-off between the return from outsourcing and the return from in-house development. If the firm outsources, it saves $(1-\gamma)F$ in fixed cost; if it keeps development for the new product in-house, it avoids paying the licensee's reservation profit and possibly gains higher expected profit through more precise positioning of the new product. In other words, an important benefit associated with in-house development comes from the fact that the originator internalizes the patent value of its old product and therefore is willing to invest more in location precision for its new product than the licensee. The magnitude of this benefit depends on how investment improves location precision and when the old patent expires. I assume that as investment level K increases, the incremental increase in the probability of having a tighter location range for the new product drops quickly (i.e. $p'(K)$ is sufficiently convex). Proposition 2 summarizes the trade-off in returns between in-house and outsourced development. Let B (B') denotes the benefit from internalizing the patent value of the old product when its patent expires at the end of the third (fourth) period.

Proposition 2. Suppose a firm owns two products in the same class: one product is on the market and under patent protection while the other is in development. When $p'(K)$

is sufficiently convex,[9] *the return to internalization and the decision rules to in-house versus outsourced development are given by (i) through (iv) :*

(i) The return to internalizing the old patent value is zero when the old patent expires before the new product introduction; the return is positive and increasing the longer the old patent lasts after the new product is introduced, i.e., $B' > B > 0$.

(ii) If $(1-\gamma)F \leq \tilde{\pi}$*, then development is always kept in-house.*

(iii) If $\tilde{\pi} < (1-\gamma)F \leq \tilde{\pi} + B$*, then development is in-house (outsourced) whenever the patent for the old product expires after (before) the introduction of the new one.*

(iv) If $\tilde{\pi} + B < (1-\gamma)F \leq \tilde{\pi} + B'$*, then development is in-house (outsourced) when the patent for the old product expires (less than) two periods after the introduction of the new one.*

(v) If $(1-\gamma)F > \tilde{\pi} + B'$*, then development is always outsourced.*

Proposition 2 explains why some firms always choose in-house development, why some firms always outsource, and why some firms choose to develop some new products in-house and outsource others. Note that the benefit from outsourcing is compared to three critical values associated with the benefit from in-house development. Depending on when the old product loses patent protection, the in-house benefit could be $\tilde{\pi}$, $\tilde{\pi} + B$, or $\tilde{\pi} + B'$ in order of size. If the cost-savings from outsourcing is very low such that it is less than the licensee's reservation profit, then an originator will always develop a new product on its own and avoid paying the reservation profit. If the cost-savings from outsourcing is between $\tilde{\pi}$ and $\tilde{\pi} + B$, then an originating firm outsources when its old patent expires before the introduction of its new product and keeps development in-house otherwise. The reason is that as long as the old product's patent protection time overlaps with the new product's market time for one period, then in-house development

[9] I assume the function $p'(k)$ to be sufficiently convex around the first best value of K. This condition allows for a comparison of net return to internalization between the two scenarios where the old patent expires in periods 3 and 4, respectively.

gives an additional benefit of B (on top of savings from $\tilde{\pi}$) associated with the internalization of the old product's patent value. With the additional benefit B, the cost-savings from outsourcing is no longer greater than the return from in-house development, so in-house development is preferred. If the cost-savings from outsourcing is between $\tilde{\pi} + B$ and $\tilde{\pi} + B'$, a firm outsources even when the overlap between the old product's patent protection time and the new product's market time is one period, but the firm keeps development in-house when the overlap is two periods. In this case, the cost-savings from outsourcing is large enough that a firm will only choose in-house development when it owns an old patent that lasts for at least two periods after the introduction of the new product. The two-period overlap gives in-house development an additional benefit of B', which brings the returns from in-house development above the cost-savings from outsourcing. Finally, if the cost-savings is so large that it exceeds the highest possible return associated with in-house development, then a firm will always outsource.

I now derive two testable predictions from Proposition 2. The first prediction considers the relationship between patent existence and the choice of in-house or outsourced development. As discussed above, when the old patent expires before the introduction of the new product, there is no return to internalizing the patent value of the old product. As far as planned *anti*-obsolescence is concerned, this situation is equivalent to one where the firm has no existing patented products to begin with. In contrast, when the old patent expires after the new product is introduced, the return to internalization is positive. In other words, the gain from in-house development increases with the existence of a patented product. The loss from in-house development is forgone cost-savings from outsourcing, which is $(1 - \gamma)F$ and stays the same with or without an existing patented product. The result is that, all else equal, the probability of in-house development will be higher when the originator owns an old patented product compared to not owning any.

Testable Prediction 1. If a firm already has a patented product, this makes the firm more likely to choose in-house development for new products in the same class as the patented one.

The second prediction focuses on how patent length for existing products could affect a firm's decision on whether to develop its new product in-house or not. From Proposition 2, it is clear that the benefit from internalizing the old patent's value is larger when its remaining length after new product introduction is two periods as opposed to one, $B' > B$. Hence, the probability that the benefit from in-house development outweighs the cost-savings from outsourcing is higher the longer the old patent lasts.

Testable Prediction 2. As the remaining patent duration for a firm's existing products increases, the firm is more likely to choose in-house development for new products in the same class.

The third prediction concerns market share and integration decisions in development. While my model does not explicitly model market share, one could extend the prediction of patent length to market share understanding that both characterize the value of the existing patented products. More specifically, when a new good is introduced to a market, some of the sales from the new good may hurt the originator's existing patented products in the same market. This is a bigger problem the higher the market share the originator has of the existing patented products, and hence greater incentive for in-house development to make sure that the new product is maximally differentiated from the old ones.

Testable Prediction 3. The likelihood that a firm will develop a new product in-house increases with the market share the firm expects to have for its existing patented products

in the same class as the new one by the time the new product reaches the market.

In sum, I incorporate the idea of planned *anti*-obsolescence into a model for a firm's decision on in-house versus outsourced development. Because an originating firm retains the patent ownership of its old products, the originator internalizes its old patent value when developing new products. If development is instead outsourced, the licensee does not internalize the patent value of the originator's old products. Given the misaligned incentives between the two, a firm that owns an existing patented product is more likely to develop its new same-class products in-house. Moreover, the probability of in-house development increases with the value of the existing patented products in terms of both patent length and market share.

1.3.3 Additional Theoretical Considerations

In this subsection, I consider two alternative actions that firms might take to address the concern with outsourcing – a sales solution, and a contractual solution – and discuss the extent to which each action is limited in trying to align incentives between the originator and the licensee.

The first possibility is that when an originating firm licenses out its new product for development, the firm could also sell its old product's patent to the licensee. Once the licensee holds patent ownership of the old product, the licensee would make development decisions to maximize joint profits from both the old product and the new one. The problem with this solution is that the originating firm may not be willing to sell its old patent to the licensee in the first place due to adverse selection. Akerlof (1970) shows that asymmetric information may result in no trade occurring at all. Because the originator likely possesses private information about the quality of its patent that the licensee

does not observe, the licensee is only willing to pay a price that reflects the average quality of similar patents on the market. As a result, firms with high quality patents may be driven out of the market by firms with mediocre quality patents which may be driven out by those with low quality patents. In other words, the market may unravel in a sequence of events such that there is in fact no trade in equilibrium (Akerlof, 1970). If firms cannot take effective measures to avoid the adverse selection problem, a sales solution may often be infeasible.

The second possibility is that an originating firm could try to specify terms in a licensing agreement that would force the licensee to internalize the patent value of the originator's old products. The issue is that, the originator may not be able to easily verify that the licensee has indeed strictly followed the contractual terms. For example, a contract that states that the licensee will provide a new product maximally different from the originator's existing goods in the same market is of little value as evaluating the difference between products often comes with many dimensions of complexity. Because there is no single composite measure for how different the products are, it would be difficult to verify whether the agreement was in fact fulfilled. Along the same lines of reasoning, specifying an investment amount to ensure that the licensee internalizes the patent value of the originator's existing products may also be a futile attempt when investments include managerial effort which is non-verifiable in court (Grossman & Hart, 1986). The bottom line is that the contractual terms inevitably leave some level of flexibility, making it challenging at best impossible at worst to create an enforceable contract that would compel the licensee to care about the profitability of the originator's old products.

To conclude, strategies aimed at eliminating the incentive distortion between the originator and the licensee often come with their own complications. Rather than trying

to align incentives, my model shows that the originating firm takes into account how its incentives differ from those of the licensee, and accordingly makes integration decisions in development. In the rest of this paper, I apply my model to the pharmaceutical industry and test its predictions empirically.

1.4 Data from the Pharmaceutical Industry

The pharmaceutical industry is a fitting application of the model for four reasons. First, the industry spends a considerable amount on R&D for new chemical compounds each year. The total global pharmaceutical spending on R&D was approximately 137 billion US dollars in 2013 (Schulze et al., 2014). The fact that firms are constantly trying to bring new drugs to the market provides an interesting case to examine whether and how firms practice planned *anti*-obsolescence on their existing patented compounds when introducing new ones. Second, the patent system for pharmaceuticals allows firms to sell drugs on the market while retaining the ownership of their patents. This suggests that a firm's existing patented compounds could potentially affect the firm's development decisions for new compounds in the same therapeutic class. Third, uncertainty in pharmaceutical development matches the model's assumption that firms do not have the ability to control the exact location of their new products relative to the old ones. They could however choose a mean location and an investment level for location precision. Finally, the increasingly common phenomenon for pharmaceutical firms to develop some compounds in-house while outsourcing others suggests a possible setting in which planned *anti*-obsolescence could play a role in firms' in-house versus outsource decisions.

1.4.1 Main Data Source and Descriptive Statistics

My principal data source is the Pharmaprojects dataset. It is assembled by Informa[10], and records the development and progress of new pharmaceutical products throughout the world. The dataset contains information for the years between 1989 and 2004 on the name of a company that originated a compound, the therapeutic class, the active ingredient (AI) patent number and patent filing date if any[11], whether development is licensed out and if so the name of the licensees, the beginning and end dates of a license and the different development phases of a compound. For development phases, a substantial number of observations are right-censored. For example, some observations only show the beginning date of a compound's pre-clinical trial and have no further information on the later phases. To drop compounds that are no longer in development for a particular year, I consider a compound to have failed a phase if the time it spent in that phase is longer than 95% of the other compounds that have successfully completed the phase (Danzon et al., 2005). The failure thresholds are thus 6 years for pre-clinical, 4 years for phase I, 6 years for phase II and 7 years for phase III.

In creating the key variables for analysis, defining an indicator for in-house development requires the most care. From the model set up in Section 1.3, a compound is developed in-house if the originator makes decisions concerning product location and is outsourced if the licensees make the relevant decisions. In practice, whether a compound is developed in-house depends on whether it has ever been licensed out by the originating firm and also its stage of development when the earliest licensing deal was made.[12] Obviously, if a compound is never licensed out, the originating firm makes all

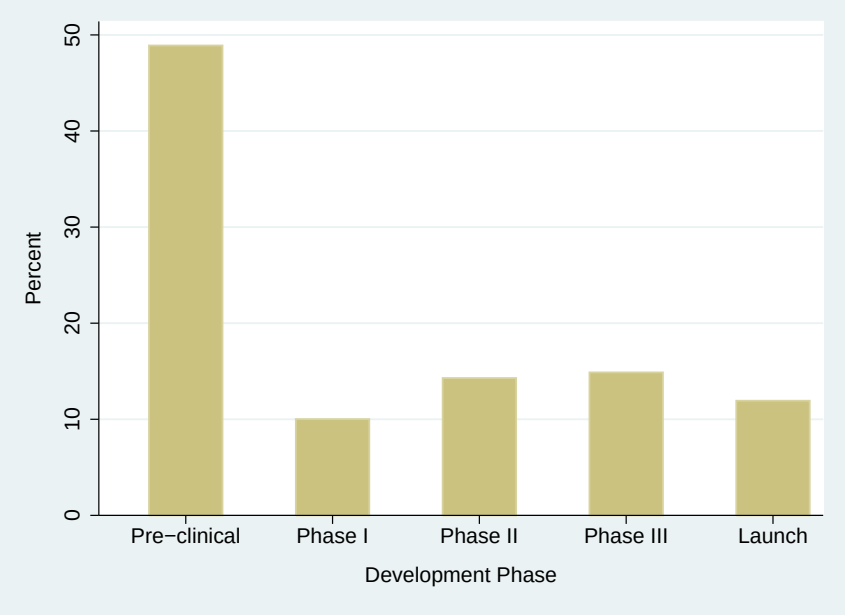
[10]The dataset is generously provided by Sean Nicholson

[11]Patents recorded in the Pharmaprojects dataset are for active ingredients (AI). While a drug could also have other non-AI patents such as method of use patents, process patents and formulation patents, Hemphill and Sampat (2012) show that AI patents are often the most important and least likely to be successfully challenged by generic companies.

[12]Note that when two firms merge, the original dataset (July 2004 release of the Pharmaprojects

the decisions and the situation is categorized under in-house development. On the other hand, if a compound is licensed out, but only at a later stage of development such that the product design can no longer be easily altered by the licensees, then the compound would still be treated as one that is developed in-house by the originator. These limitations on product design could apply to choices of target patient population, health endpoints, side effects, or more generally decisions that need to be made early on in the development process.

Figure 1.3: Distribution of the Earliest Licensing Deal by Development Phase



I take the start of phase III trials as the point in time at which changes to a drug candidate's design becomes highly limited. In other words, a compound is defined to be developed in-house if it is originated by the firm, and not licensed out before the phase III stage. Vice versa, a compound is outsourced if the originating firm licenses it out

database in LotusNotes format) always updates the company name of a compound to the acquiring firm. This means if Warner-Lambert originated compound X in 1997 and was acquired by Pfizer in 1999, compound X would only be associated with Pfizer. This is a problem as the dataset in its original format does not reflect the fact that Warner-Lambert originated and developed compound X before the merger. In the working dataset of this paper, the problem is taken care of by compiling a list of mergers and acquisitions and reassigning compounds to the acquired firm before the merger.

Table 1.1: Summary of Development Phases

Drug Development Stage	Description (taken from the FDA website)
Pre-clinical testing	Submission of investigational new drug application for the FDA to review. Companies need to show results of pre-clinical testing on laboratory animals and propose plans for human testing
Phase I trial	Usually conducted in healthy volunteers to determine the most frequent side effects, as well as how the drug is metabolized and excreted. Number of subjects range from 20 to 80. Emphasis is on safety
Phase II trial	Obtain preliminary data on whether the drug treats a certain disease or condition. Number of subjects range from a few dozen to about 300. Continues to evaluate safety and short term side effects
Phase III trial	The FDA and the sponsors meet to determine how large-scale studies in Phase III should be done. Gather more information on safety and effectiveness. Studies different populations, dosages and combined usage of other drugs. Number of subjects ranges from several hundred to about 3,000 people

Source: `http://www.fda.gov/drugs/resourcesforyou/consumers/ucm143534.htm`

before the start of phase III trials. Figure 1.3 displays the distribution of the earliest licensing deal by development phase. Of all the compounds that were ever licensed out, 48.9% were first licensed out during pre-clinical testing, 10.0% during phase I, 14.3% during phase II, 14.87% during phase III, and 11.9% were first licensed out after being launched.

Table 1.1 presents a summary of the development phases as described by the FDA, giving an idea of how each phase could affect a compound's design in terms of subject population and side effects ("The FDA's Drug Review", 2014). In the stages prior to phase III, pre-clinical consists mostly of testing on laboratory animals while phase I focuses on safety and phase II on effectiveness and side effects. Both phase I and phase II are conducted on a relatively small scale, with the former recruiting around 20 to 80

subjects and the latter between a few dozen to about 300 subjects. Phase III continues testing on safety and effectiveness within a much larger sample, usually ranging from several hundred to about 3,000. Conceivably, a developer could still affect the way a compound is marketed up until the end of phase II trials through recruitment of specific population groups and testing of specific side effects. However, once phase II is completed and the FDA meets with the developer to discuss plans for phase III, it becomes increasingly difficult for the developer to make major changes to the design and nature of a compound.

To see whether owning a patent would affect the choice of in-house compounds, I construct three measures for patent existence. The first measure, which I will call EOP1, is an indicator for the existence of other patented compounds in the same therapeutic class[13] and same firm as the compound of interest. The binary variable categorizes each compound into one of two cases: either it belongs to a firm that owns other patented compounds in the same therapeutic class or it does not. The second measure, which I will call EOP2, is the number of other patented compounds in the same class and owned by the same firm as the compound of interest. Finally, in an effort to distinguish patents in later development stages (i.e. phase III or launched) from those in earlier stages (i.e. phase I or II), a third measure EOP3 is constructed. This measure is an expected number of other patented compounds in the same class and firm that would eventually reach the market. Following Higgins and Rodriguez (2006), I obtain EOP3 through a count of same-class same-firm patented compounds weighted by their probability of becoming an approved drug conditional on the current stage of development. Based on existing research, the probabilities are 0.08 for pre-clinical, 0.20 for phase I, 0.28 for phase II,

[13] According to the Pharmaprojects Therapeutic Class Codes, there are 17 broadly defined categories. These categories are alimentary/metabolic products, blood and clotting products, cardiovascular products, dermatological products, formulations, genitourinary (including sex hormones), hormonal products (excluding sex hormones), immunological products, anti-infective products, anticancer products, musculoskeletal products, neurological products, anti-parasitic products, respiratory products, sensory products, biotechnology products, miscellaneous products

0.58 for phase III and 1 for launched drugs ("The Drug Development", n.d.).

In order to determine how the value of patents could affect a firm's in-house or outsource decisions, I also include three variables concerning patent length. The first variable LOP1 is the length of the longest patent among all other compounds in the same class and firm as the compound in question. LOP1 is constructed based on the argument that if length could serve as a proxy for value, then firms would often times want to internalize the patent with the longest remaining life in any given therapeutic class. My second length-related variable LOP2 is the sum in patent length of all other compounds within the same class and same firm as the target. Instead of focusing on the longest patent, the goal of LOP2 is to capture the value of the overall patent profile. For my third variable related to patent length, I account for the fact that compounds in earlier development stages may have longer patent life but smaller probability of reaching the market than compounds in later stages. This third variable, which I refer to as LOP3, is the weighted sum of patent length for all other same-class same-firm compounds according to their probability of reaching the market conditional on the current stage of development (Higgins & Rodriguez, 2006).

One concern when examining the relationship between a firm's patent profile and its choice of in-house versus outsourced development is that a firm may be developing compounds in areas in which it has more experience and expertise. Controlling for experience would be essential to the extent that it is also correlated with patent existence and length. I define class-specific experience as the cumulative sum of the compound-year observations within a firm's particular therapeutic class up to the current year[14].

[14]Note that another way to control for experience is to include class-specific experience by development phase (Danzon et al., 2005). Whether experience is by phase or not does not qualitatively change the estimated effect of patent profile (existence and value) on a firm's in-house or outsource decision. However, when experience is controlled for by phase, the experience coefficients for later development stages are imprecisely estimated. This is likely due to the fact that only a small number of observations in the dataset are for phase III and launched drugs. Hence, in my preferred specifications, I include the overall (not separately by phase) class-specific experience.

Hence, the constructed experience variable increases if a single compound has been developed over multiple years or if there are multiple compounds in the same class that are developed in a single year. The goal of this variable is to separate out the cost-side explanation for in-house or outsource decisions — that firms with more experience have lower development costs and in turn are more likely to keep compounds in-house — from firms' incentive to limit product substitutability and prevent patent devaluation.

In addition to experience, I also include a measure for economies of scope as another cost-side control on the firm level. Following Danzon et al. (2005), I construct a Herfindahl Hirschman Index (HHI) for each firm's therapeutic scope by summing up the squares of the percentage of compounds being developed for each therapeutic class within a firm in a given year. The bigger the HHI, the more concentrated a firm's development portfolio becomes in terms of therapeutic class, and thus the less likely that the firm will have economies of scope. One could interpret the scope measure as an indication for development costs, understanding that more therapeutic scope is often associated with lower development costs.[15]

On the market level, I construct two variables to capture potential exogenous changes to a firm's incentive in protecting the value of its patents. The first variable PDM is the number of patented drugs on the market that are in the same therapeutic class as the target compound, but from firms other than the one that owns the target compound. The second variable TDM is the total number of drugs on the market that are in the same class as the compound of interest. The purpose of constructing these two variables is to see whether an increase in the number of competing patented drugs from outside firms, holding fixed the total number of drugs on the market, could lower the target firm's incentive to internalize its patents. See Table 1.2 for a list of all the constructed

[15] A firm with more therapeutic scope may also be one that is less financially constrained. This is either because financial resources allow a firm to develop in different therapeutic areas, or because more scope reduces development costs, which may also relieve a firm of its financial constraints to some extent.

Table 1.2: Definition of Constructed Variables

Variable	Description
In-house	Indicator equals 1 if compound is never licensed out by the originating firm or if its earliest licensing deal was made after the start of Phase III trials
Existence of Patents	
EOP1	Indicator equals 1 if at least one other compound in the same therapeutic class and same firm is patented
EOP2	Number of other patented compounds in the same therapeutic class and same firm
EOP3	Expected number of other patented compounds in the same therapeutic class and same firm that would reach the market
Length of Patents	
LOP1	Length of the longest patent among other compounds in the same therapeutic class and same firm
LOP2	Sum of patent length among other compounds in the same therapeutic class and same firm
LOP3	Weighted sum of patent length among other compounds in the same therapeutic class and same firm according to their probability of becoming an approved drug
Experience	Cumulative count of compound-year observations within a firm for a therapeutic class corresponding to the compound of interest
Scope	Sum of the squares of the percentage of compounds being developed for each therapeutic class within a firm in a given year
PDM	Number of patented drugs on the market in the same therapeutic class but not the same firm as the compound of interest
TDM	Total number of drugs on the market in the same therapeutic class as the compound of interest
MSP	Market share based on sales for existing patented drugs in the same class and same firm as the compound of interest

variables along with their definitions.

Table 1.3 reports descriptive statistics for in-house development and patent profiles. The sample used in the main analysis contains 11,739 compounds originated by 610

Table 1.3: Descriptive Statistics on In-house Development and Patent Profile

Number of compounds					11739
Number of firms					610
Years covered					1989−2004
Variable	Overall Mean	Mean Outsourced Compounds	Mean In-house Compounds	Min.	Max.
	Level of Observation: compound-year (67,215 obs.)				
In-house	0.759			0	1
	(0.428)				
Existence of Patents					
EOP1	0.725	0.638	0.753	0	1
	(0.446)	(0.481)	(0.431)		
EOP2	10.504	4.567	12.393	0	101
	(17.213)	(9.521)	(18.626)		
EOP3	1.833	1.049	2.083	0	15.12
	(2.746)	(1.970)	(2.906)		
Length of Patents					
LOP1	11.605	9.587	12.248	0	20
	(7.854)	(7.952)	(7.712)		
LOP2 (x10)	10.950	4.759	12.920	0	106.20
	(17.811)	(9.717)	(19.288)		
LOP3 (x10)	1.736	1.005	1.969	0	12.38
	(2.498)	(1.796)	(2.640)		

Note: EOP1 is an indicator for at least one other patented compound in the same therapeutic class and same firm as the compound of interest. EOP2 is the number of other patented compounds in the same therapeutic class and same firm. EOP3 is the expected number of other patented compounds in the same therapeutic class and same firm that would reach the market. LOP1 is the length of the longest patent among other compounds in the same therapeutic class and same firm as the compound of interest. LOP2 is the sum of patent length among other compounds in the same therapeutic class and same firm. LOP3 is the weighted sum of patent length among other compounds in the same therapeutic class and same firm according to their probability of becoming an approved drug.

firms between 1989 and 2004. On the compound-year level, 75.9% of the observations are in-house development. The first measure for patent existence EOP1 shows that 75.3% of the observations for in-house projects and 63.8% for outsourced projects have at least one other patented compound in the same class and firm as the compound of interest. In addition, compared to outsourced projects, in-house ones have on average

a higher number of other patented compounds in the same class and firm (EOP2) as well as a higher expected number of other same-class same-firm patented compounds that would be on the market (EOP3). In terms of patent length, LOP1 shows that the length of the longest patent among other compounds in the same class and firm as the target compound is on average longer for in-house observations (12.2 years) than for outsourced ones (9.6 years). Furthermore, both the sum (LOP2) and weighted sum (LOP3) of patent length for all other same-class same-firm compounds are bigger on average for in-house observations than for outsourced ones.

Table 1.4 summarizes the control variables used in the estimation. On the compound-year level, the statistics for the development phase indicators suggest that 70.7% of the observations are pre-clinical, 7.5% are phase I, 10.4% are phase II, 7.6% are phase III, and 3.8% are launched drugs. Most of the observations are pre-clinical because a compound can fail in a development stage. As mentioned earlier, beyond a failure threshold, any compound with no further development record is dropped out of the dataset. Variables containing information on the firm of interest include therapeutic experience and scope. Experience varies substantially on the firm-class-year level, with a mean of approximately 30 development records and a standard deviation of about 70. The HHI for scope is on average 0.57 for a firm in a given year; the maximum HHI for scope is 1, indicating development in only one therapeutic class, while the minimum is 0.10, indicating a fairly diversified therapeutic pipeline. The table also displays information related to the market such as the number of patented drugs on the market in the same therapeutic class but not the same firm as the compound in development (PDM), and the total number of drugs on the market in the same class as the compound in development (TDM). These variables reflect the extent to which a market is crowded with drugs from competing firms.

Table 1.4: Descriptive Statistics on the Control Variables 1989-2004

Level of Observation	Variable	Obs.	Mean	Std. Dev.	Max.	Min.
Compound-year	Pre-clinical	67215	0.707	0.455	1	0
	Phase I	67215	0.075	0.264	1	0
	Phase II	67215	0.104	0.305	1	0
	Phase III	67215	0.076	0.265	1	0
	Launched	67215	0.038	0.192	1	0
Firm-class-year	Experience (x10)	18452	3.035	7.022	115.70	0.10
	PDM	18452	18.598	17.789	82	0
Firm-year	Scope	5911	0.574	0.314	1	0.10
Class-year	TDM	272	19.292	27.588	191	0

Note: PDM is the number of patented drugs on the market in the same therapeutic class but not the same firm as the compound of interest. TDM is the total number of drugs on the market in the same therapeutic class as the compound of interest.

In Table 1.5, I display the proportion of compounds developed in-house in each of the 17 broadly defined therapeutic classes.[16] The areas of cardiovascular and neurology have among the highest in-house development rates among all therapeutic categories (85.0% for cardiovascular and 85.3% for neurology). In these well-established markets, where firms may already own other patented products in the same area as their on-going project, positioning of the new compound becomes critical. It is thus not surprising that firms would tend to keep development in the cardiovascular and neurology areas in-house in order to better limit substitutability and prevent patent devaluation.[17]

[16]Source: Pharmaprojects Therapeutic Class Codes.
https://web.archive.org/web/20060322032407/http://support.dialog.com/searchaids/dialog/pdf/f128_therapeuticcodes.pdf

[17]It is also possible that firms keep development in-house in areas in which it has more experience and expertise. However, the focus of this paper is to bring a new perspective to why firms would or would not develop a compound in-house through the lens of patent protection. The empirical tests in section 1.5 provide further evidence of the planned *anti*-obsolescence theory in explaining integration decisions in development.

Table 1.5: Distribution of In-house Development Across Broad Therapeutic Areas 1989-2004

	mean	sd	count
alimentary/metabolic	0.804	0.397	3173
anti-infective	0.823	0.381	8447
anticancer	0.781	0.414	7343
antiparasitic	0.963	0.190	267
biotechnology	0.616	0.486	12683
blood and clotting	0.814	0.389	2291
cardiovascular	0.850	0.357	5198
dermatological	0.731	0.444	1099
formulations	0.661	0.473	7573
genitourinary	0.797	0.403	1244
hormonal products	0.689	0.463	588
immunological	0.765	0.424	1609
miscellaneous	0.595	0.491	800
musculoskeletal	0.822	0.383	3601
neurological	0.853	0.354	8622
respiratory	0.820	0.384	2270
sensory	0.769	0.422	407
Total			67215

Note: The table presents the 17 broadly defined therapeutic classes in the Pharmaprojects dataset.
Source: Pharmaprojects Therapeutic Class Codes.
`https://web.archive.org/web/20060322032407/http://support.dialog.com/searchaids/dialog/pdf/f128_therapeuticcodes.pdf`

1.4.2 Secondary Data source and Descriptive Statistics

To supplement the analysis, I also use the IMS dataset to create a market share measure based on drug sales. The IMS data includes a list of all drugs and their annual sales in the US between 1992 and 2004. The sales data are merged into the principal dataset from Pharmaprojects based on the name of the drug, its therapeutic class, as well as whether the drug is branded or not.[18] Because the IMS dataset classifies drugs according to the Anatomical Therapeutic Chemical (ATC) system, which slightly differs from what is

[18]Of the drugs that are launched in the Pharmaprojects dataset, 57% are matched to those in the IMS dataset.

used in the Pharmaprojects dataset, the supplemental analysis only focuses on the drug classes that are common to both systems. I calculate for each compound in the principal dataset, the summation of market shares for other same-class same-firm patented drugs in each year. This variable is later referred to as MSP, and tests for how market share based on sales affects a firm's incentive to practice planned *anti*-obsolescence and in turn its in-house versus outsource development decisions.

Table 1.6(a): Market Share for Same-Class Same-Firm Drugs 1992-2004

	mean	sd	min	max	count
alimentary/metabolic	0.27	1.19	0.00	13.03	2946
anti-infective	0.40	0.80	0.00	4.06	7758
antiparasitic	0.00	0.00	0.00	0.00	242
blood and clotting	0.67	2.40	0.00	14.33	2121
cardiovascular	1.60	4.19	0.00	20.83	4662
dermatological	0.06	0.31	0.00	5.90	1021
genitourinary	0.52	2.29	0.00	13.02	1185
hormonal products	0.05	0.47	0.00	7.95	502
musculoskeletal	1.15	5.17	0.00	33.53	3389
neurological	0.29	0.82	0.00	6.52	7897
respiratory	0.44	1.42	0.00	11.84	2058
sensory	0.74	2.06	0.00	8.66	362
Total					60723

Note: Market share is in percentage terms. The table presents the 13 therapeutic classes common to both the Pharmaprojects dataset and the IMS sales data. The IMS sales data is only available for years between 1992 and 2004.

Table 1.6(a) shows the descriptive statistics for MSP by therapeutic class between 1992 and 2004. Compounds in the cardiovascular and musculoskeletal areas are on average associated with the highest market share for other same-class same-firm patented drugs. Recall from Table 1.5 that compounds in these two areas are also more frequently developed in-house than compounds in most of the other therapeutic categories. The pattern is suggestive that planned *anti*-obsolescence may be one of the reasons for in-house development. Furthermore, there is considerable heterogeneity within each therapeutic

class. Taking the above mentioned areas as examples again, the market share for other same-class same-firm patented drugs is on average 1.60% and 1.15% for a compound in the cardiovascular and musculoskeletal classes, respectively, while the maximum is 20.83% and 33.53%. The maximum MSP is nearly 5 standard deviations above the mean in the cardiovascular area and about 6 standard deviations above the mean in the musculoskeletal area. Table 1.6(b) displays a similar pattern of within- and across- therapeutic class variations in MSP conditioning on firms having a launched drug in the corresponding therapeutic class.

Table 1.6(b): Market Share for Same-Class Same-Firm Drugs 1992-2004 Conditional on Firms Having a Drug in the Corresponding Therapeutic Class

	mean	sd	min	max	count
alimentary/metabolic	0.49	1.60	0.00	13.03	1514
anti-infective	0.68	0.96	0.00	4.06	4475
antiparasitic	0.00	0.00	0.00	0.00	60
blood and clotting	1.67	3.60	0.00	14.33	835
cardiovascular	2.40	4.97	0.00	20.83	3078
dermatological	0.30	0.68	0.00	5.90	183
genitourinary	1.52	3.76	0.00	13.02	396
hormonal products	0.04	0.25	0.00	2.85	229
musculoskeletal	2.67	7.61	0.00	33.53	1464
neurological	0.44	0.98	0.00	6.52	5154
respiratory	0.87	1.90	0.00	11.84	1038
sensory	1.27	2.67	0.00	8.66	196
Total	0.65	2.62	0.00	33.53	31700
Total					31291

Note: Market share is in percentage terms. The table presents the 13 therapeutic classes common to both the Pharmaprojects dataset and the IMS sales data. The IMS sales data is only available for years between 1992 and 2004.

1.5 Empirical Tests

In this section, I empirically test the theoretical predictions regarding the role of planned *anti-* obsolescence in pharmaceutical firms' in-house versus outsourced development decisions. I first focus on whether each of two core components of my model—patent existence and length for old products—is an important factor in the probability of in-house development for new compounds in the same firm and same therapeutic area. I then investigate how a firm's position in the market in terms of both the number of competitors and the market share for the firm's existing patented products could affect the probability of in-house development. Robustness checks to the findings are discussed in the last part of the empirical analysis.

1.5.1 Existence of Patents

To model whether having patents affect firms' decision to develop a compound in-house or not, I estimate the following logit specification:

$$Prob(Y_{ijkt} = 1) = \Lambda(\alpha_0 + \alpha_1 EOP_{ijkt} + \alpha_2' X_{ijkt} + \alpha_3 Z_{jkt} + \alpha_4 W_{jt} + C_k + T_t + F_j), \quad (1.1)$$

where $\Lambda(\cdot) \equiv \frac{\exp(\cdot)}{1+\exp(\cdot)}$ is the standard Logistic CDF. The subscripts i, j, k, and t index compound, firm, therapeutic class, and year. Y_{ijkt} is an indicator for in-house development. EOP is a measure of patent existence. It could be an indicator (EOP1) or a count (EOP2) of other patented compounds in the same therapeutic class and belonging to the same firm as the compound of interest; or it could be an expected number of other patented compounds that would eventually reach the market (EOP3). X is a vector of development phase indicators. Note that because of how in-house and outsourced projects are defined — that a compound is outsourced if its originator licenses it out before the phase

III trial, only the decisions made prior to phase III matter effectively. Hence, the analysis uses pre-clinical testing as the omitted comparison group, and controls for phase I and phase II trials. Z is a firm's development experience in the therapeutic class that contains the compound of interest. W is a firm's therapeutic scope. Equation (1.1) also includes therapeutic-class fixed effects (C_k) to control for unobserved class characteristics that affect both the existence of patents and the integration decisions in development. Year fixed effects (T_t) control for across-time differences in firms' preferences towards internal or external development. Some specifications also include firm fixed effects (F_j) to absorb any fixed firm-level characteristics that affect the likelihood of in-house development. From Testable Prediction 1, I expect α_1 to be positive: firms with existing patents are more likely to develop the next compound in the same therapeutic area in-house than firms that do not own any existing patents.

Table 1.7 reports the estimation results. Each measure of existence of patents (either EOP1, EOP2, or EOP3) is estimated under three model specifications. The first version looks at how the probability of in-house development relates to patent existence when a firm's therapeutic experience and scope are omitted from the regression analysis. The second version adds in the experience and scope measures to control for firms potentially capitalizing on past specific investments. In the third version, I include firm fixed effects to exploit within-firm variation over time in patent ownership and in-house decisions to identify the relationship between the two. All specifications use robust standard errors to account for heteroskedasticity. Furthermore, the standard errors are clustered on the compound level to account for potential correlation of in-house or outsource decisions for a particular compound across observations.

Table 1.7: Logit Models of In-house Development: Existence of Patents

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Existence of Patents									
EOP1	0.757***	0.357***	0.309*						
	(0.107)	(0.117)	(0.178)						
EOP2				0.072***	0.050***	0.025*			
				(0.013)	(0.016)	(0.013)			
EOP3							0.291***	0.115*	0.094
							(0.052)	(0.070)	(0.068)
Experience		0.033***	0.002		0.013	−0.008		0.027***	−0.009
		(0.007)	(0.008)		(0.011)	(0.010)		(0.010)	(0.011)
Scope		−0.702***	−0.440		−0.671***	−0.345		−0.704***	−0.350
		(0.199)	(0.508)		(0.196)	(0.509)		(0.198)	(0.510)
Phase I	−1.036***	−1.011***	−0.937***	−0.983***	−0.987***	−0.942***	−1.009***	−1.005***	−0.939***
	(0.143)	(0.142)	(0.165)	(0.142)	(0.142)	(0.165)	(0.142)	(0.142)	(0.165)
Phase II	−1.170***	−1.168***	−1.249***	−1.123***	−1.136***	−1.252***	−1.161***	−1.164***	−1.250***
	(0.128)	(0.128)	(0.156)	(0.128)	(0.129)	(0.156)	(0.128)	(0.128)	(0.156)
Constant	5.280***	5.770***	3.945***	5.446***	5.755***	3.873***	5.439***	5.845***	3.926***
	(0.712)	(0.728)	(1.213)	(0.715)	(0.729)	(1.231)	(0.713)	(0.730)	(1.235)
Observations	47831	47831	38170	47831	47831	38170	47831	47831	38170
Firm controls	No	No	Yes	No	No	Yes	No	No	Yes

Note: Dependent variable is one if a compound is developed in-house, and zero otherwise. All specifications include a full set of therapeutic category and year indicators. EOP1 is an indicator for at least one other patented compound in the same therapeutic class and same firm as the compound of interest. EOP2 is the number of other patented compounds in the same therapeutic class and same firm. EOP3 is the expected number of other patented compounds in the same therapeutic class and same firm that would reach the market.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are heteroskedasticity-robust and clustered on the compound level.

The results are in line with the model's prediction (Testable Prediction 1). Column (1) shows a strong, positive, and statistically significant association between in-house development and EOP1. Column (2) shows that after controlling for a firm's therapeutic experience and scope, there is still a positive relationship between in-house development and EOP1. The estimated logit coefficient on EOP1 is smaller compared to that in column (1), but still significantly different from zero at the 1% level. This result is reassuring in that the experience and scope variables are indeed capturing the cost-side factors that may be confounding the effect of patent protection on the likelihood of internal development; leaving out these controls biases the estimate upward. Column (3) shows that even with firm fixed effects, this likelihood is still positively associated with EOP1 and the effect is significant at the 10% level.

Using either EOP2 or EOP3 as alternative measures of patent existence, I find similar results to those for EOP1. In general, the estimate of patent existence is largest in the first specification, and smallest in the third.[19] However, just as the first specification likely overestimates the effect of patent existence on in-house decisions, the third one may well underestimate this effect. In other words, the third specification (with firm fixed effects) may be controlling for too much of the variability in in-house decisions. It is possible that more common in-house development—due to a firm's incentive to protect its existing patented compounds—may also improve development efficiency through learning-by-doing, which in turn makes in-house decisions for future compounds more attractive. This means that over time, firms that historically have more incentive to practice planned *anti*-obsolescence may also, as a result, be the ones that are more efficient. Adding firm fixed effects may control for the level of efficiency in development, and hence eliminate some theoretically meaningful variation in the model estimation.

[19]For EOP1, Table 1.7 shows that the estimate in column (3) with firm fixed effects is roughly on the same order of magnitude as that in column (2) without firm fixed effects; the standard error is larger in column (3) than in column (2), possibly due to smaller sampling variation from including firm fixed effects.

Choosing the second version of the model as my preferred specification, I compute how much less licensing occurs prior to Phase III due to patent existence. The baseline probability that a compound is licensed out in any given year prior to Phase III is 9.5%. Using the estimate of EOP1, a compound is 3.4% less likely to be licensed out per year (compared to the baseline) when the originator owns at least one other patented compound in the same therapeutic area than when the originator owns none. This translates into a 20.2% cumulative decrease from the baseline over the years that the compound spends in development prior to Phase III.[20] In addition, increasing the number of other patented compounds in the same class and firm (EOP2) by one standard deviation decreases the probability of outsourced development for the compound of interest by 7.4% per year from the baseline , or cumulatively a 44.2% decrease over the course of the compound's development before Phase III. Increasing the expected number of other same-class same-firm compounds that would be on the market (EOP3) by a standard deviation decreases the probability of outsourced development by 2.8% per year, or a cumulative decrease of 17.1% over the total years of development before Phase III. EOP3 has a smaller effect than EOP2 likely because EOP3 puts a smaller weight on existing patented compounds in earlier stages of development, with the consideration that earlier stage compounds are less likely to reach the market. It is worthwhile to note, however, that if these compounds are successfully launched, they will also be more likely, compared to the later stage compounds, to still have patent protection by the time the compound of interest is introduced to the market. In such cases, downplaying earlier stage patented compounds may result in an underestimation of the effect of patent existence on the probability of in-house versus outsourced development.

Table 1.7 also shows that the coefficients on the control variables do not qualitatively change under different measures of patent existence. Regardless of which patent

[20]The average time a compound takes from pre-clinical testing to the start of Phase III trials is 6 years (DiMasi et al., 2003).

existence measure is used, firm experience on the therapeutic class level is positively associated with in-house decisions as expected in specifications without firm fixed effects, and is generally significant at the 1% level. The experience measure is imprecisely estimated when firm fixed effects are included. A possible explanation is that a firm's therapeutic class experience is highly correlated with the ability of the firm's expert team. Once differences across firms in expert ability is controlled for, experience becomes insignificant. The HHI measure for therapeutic scope is negatively correlated with the probability of in-house development. This is not surprising given that firms with a low HHI are more likely to achieve economies of scope and have a low cost in development. With regard to the indicators for development stages, the estimated coefficients consistently suggest that compounds are less likely kept in-house and more likely outsourced in later stages of development.

1.5.2 Length of Patents

To examine how in-house decisions relate to the patent length of other compounds in the same therapeutic class and same firm as the compound in question, I use a logit set up similar to that in the previous section and model the probability of in-house decision with the following equation:

$$Prob(Y_{ijkt} = 1) = \Lambda(\beta_0 + \beta_1 LOP_{ijkt} + \beta_2' X_{ijkt} + \beta_3 Z_{jkt} + \beta_4 W_{jt} + C_k + T_t + F_j). \quad (1.2)$$

LOP is one of three measures of patent length. Recall that LOP1 is the length of the longest patent among other compounds in the same class and firm as the compound of interest. LOP2 and LOP3 are, respectively, the sum and the weight sum of patent length among other same-class same-firm compounds. The control variables for development phase (X_{ijkt}), firm therapeutic experience (Z_{jkt}), scope (W_{jt}), and the fixed effects for

therapeutic category (C_k), year (T_t), and firm (F_j) are the same as those in equation (1.1). For each measure of patent length, I estimate equation (1.2) with subsets of these control variables as well as a fully specified version with all controls included. According to Testable Prediction 2, the estimated β_1 is expected to be positive — the probability of in-house development increases with the time left on patent protection for other compounds in the same class and firm as the compound of interest.

Table 1.8 shows the results are consistent with Testable Prediction 2. Patent length, measured by either LOP1, LOP2 or LOP3, is always positively associated with the likelihood that a firm will develop a compound on its own. Moreover, all three measures of patent length display similar patterns of results under different model specifications. Mirroring the earlier analysis on patent existence, excluding a firm's therapeutic experience and scope from the regression produces a relatively large estimate of patent length, but the estimate may also be contaminated by cost-side factors such as expertise and economies of scope. Including the experience and scope controls reduces the estimate on patent length. However, the estimate is still positive and significant at the 1% level. Moreover, the positive effect of patent length on in-house decisions should now be primarily capturing a firm's incentive to prevent patent devaluation of its existing compounds in new project development. Further, including firm fixed effects reduces the patent length estimate some more, but the effect is still positive. As discussed earlier, these specifications may control for the efficiency gain through learning-by-doing that is induced by the patent protection incentive, hence not reflecting the full effect of planned *anti*-obsolescence on in-house decisions. That said, two out of the three patent length measures, LOP2 and LOP3, are still significant at the 10% level when controlling for firm fixed effects. Overall, the results suggest that in order to protect the patent value of existing compounds, firms that own more patent protection time in a particular therapeutic class are more likely to develop subsequent compounds in that class in-house.

Table 1.8: Logit Models of In-house Development: Length of Patents

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Length of Patents									
LOP1	0.044***	0.019***	0.010						
	(0.007)	(0.007)	(0.010)						
LOP2				0.066***	0.043***	0.022*			
				(0.012)	(0.014)	(0.012)			
LOP3							0.306***	0.140**	0.114*
							(0.052)	(0.062)	(0.065)
Experience		0.034***	0.003		0.018*	−0.004		0.027***	−0.007
		(0.008)	(0.008)		(0.009)	(0.009)		(0.009)	(0.009)
Scope		−0.718***	−0.404		−0.687***	−0.347		−0.708***	−0.339
		(0.198)	(0.508)		(0.195)	(0.508)		(0.197)	(0.509)
Phase I	−1.018***	−1.007***	−0.936***	−0.973***	−0.988***	−0.943***	−0.989***	−1.003***	−0.942***
	(0.141)	(0.142)	(0.165)	(0.141)	(0.142)	(0.165)	(0.141)	(0.142)	(0.165)
Phase II	−1.138***	−1.160***	−1.250***	−1.102***	−1.134***	−1.252***	−1.128***	−1.160***	−1.253***
	(0.128)	(0.128)	(0.156)	(0.128)	(0.129)	(0.156)	(0.128)	(0.128)	(0.156)
Constant	4.920***	5.778***	3.989***	5.042***	5.757***	3.890***	4.952***	5.813***	3.899***
	(0.567)	(0.729)	(1.223)	(0.569)	(0.728)	(1.231)	(0.567)	(0.729)	(1.237)
Observations	47831	47831	38170	47831	47831	38170	47831	47831	38170
Firm controls	No	No	Yes	No	No	Yes	No	No	Yes

Note: Dependent variable is one if a compound is developed in-house, and zero otherwise. All specifications include a full set of therapeutic category and year indicators. LOP1 is the length of the longest patent among other compounds in the same therapeutic class and same firm as the compound of interest. LOP2 is the sum of patent length among other compounds in the same therapeutic class and same firm. LOP3 is the weighted sum of patent length among other compounds in the same therapeutic class and same firm according to their probability of becoming an approved drug.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are heteroskedasticity-robust and clustered on the compound level.

I use the model specification with experience and scope controls but no firm fixed effects to compute how much less licensing occurs prior to Phase III due to patent length. A one standard deviation increase in the length of the longest patent among other compounds in the same class and firm as the compound of interest (LOP1) is associated with a 1.1% decrease per year from the baseline probability that a firm will license out a compound prior to Phase III.[21] This translate into a cumulative decrease of 6.3% from the baseline over the years the compound spends in development prior to Phase III. Furthermore, increasing the sum (LOP2) and the weight sum (LOP3) of patent length among other same-class same-firm compounds by a standard deviation reduces the probability of outsourced development for the compound of interest by 6.3% and 3.2% per year, respectively (or cumulatively, a 37.9% and a 18.9% decrease from the baseline). The difference in magnitude between LOP2 and LOP3 is mainly driven by the fact that LOP3 discounts the patent length of compounds in early stages of development with a small probability of reaching the market. However, because these compounds have more patent protection time left than compounds in later stages of development, it is possible that LOP3 underestimates the effect of patent length on a firm's in-house versus outsourced development decisions when the early stage compounds are later successfully brought to market.

1.5.3 Market Position of a Firm

This subsection provides further support for the planned *anti*-obsolescence theory by examining the implications concerning the market position of a firm. I conduct additional empirical tests from two perspectives. One perspective continues to use the principal dataset from Pharmaprojects to look at how the number of competing drugs on

[21] As mentioned in the previous section, the baseline probability that a firm will license out a compound prior to Phase III in any given year is 9.5%.

the market from outside firms can affect a firm's incentive to prevent patent devaluation. The other perspective incorporates the secondary dataset with IMS sales to look at how a firm's own market share for patented products can affect the probability of in-house development for new projects.

1.5.3.1 Number of Other Firms' Patented Drugs

For the first approach, if the patent protection interpretation of the relationship between in-house decisions and patent profile (existence and length) is legitimate, this association should be less likely to hold when the market is crowded with patented drugs from other firms. The reason is that when most patented drugs on the market belong to other firms, an introduction of a new drug will likely have more of a negative impact on the other firms, and less so on the firm in question.

In the following logit model for in-house decisions, I interact the patent existence measure (EOP) with the number of other firms' patented drugs on the market that are in the same therapeutic class as the compound of interest (PDM). The interaction term is denoted by EOP X PDM. The equation that I estimate takes the form:

$$Prob(Y_{ijkt} = 1) = \Lambda(\gamma_0 + \gamma_1 EOP_{ijkt} + \gamma_2 EOP_{ijkt} \times PDM_{jkt} + \gamma_3 PDM_{jkt} + \gamma_4 TDM_{kt} + \gamma_5' X_{ijkt} + \gamma_6 Z_{jkt} + \gamma_7 W_{jt} + C_k + T_t + F_j). \quad (1.3)$$

TDM is the total number of drugs on the market that are in the same therapeutic class as the compound in question. Controlling for TDM, a firm's incentive to internalize its existing patents is expected to decrease as the number of competing patents on the market from other firms increases (i.e., γ_2 should be negative). All the other control variables are exactly the same as those specified in equation (1.1).

Table 1.9: Logit Models of In-house Development: Existence of Patents with Interaction Effect

	(1)	(2)	(3)	(4)
Existence of Patents				
EOP1	0.874***	0.513**		
	(0.188)	(0.256)		
EOP1 × PDM	−0.020***	−0.008		
	(0.006)	(0.008)		
EOP2			0.081***	
			(0.026)	
EOP2 × PDM			−0.002**	
			(0.001)	
EOP3				0.324***
				(0.098)
EOP3 × PDM				−0.007***
				(0.002)
PDM	0.017	0.014	0.008	0.004
	(0.028)	(0.028)	(0.026)	(0.027)
TDM	−0.003	−0.005	−0.003	0.001
	(0.013)	(0.013)	(0.012)	(0.013)
Experience	0.035***	0.006	0.023**	0.030***
	(0.007)	(0.008)	(0.011)	(0.011)
Scope	−0.596***	−0.439	−0.622***	−0.652***
	(0.202)	(0.509)	(0.198)	(0.198)
Phase I	−1.001***	−0.935***	−0.984***	−1.007***
	(0.143)	(0.164)	(0.142)	(0.143)
Phase II	−1.157***	−1.253***	−1.126***	−1.157***
	(0.128)	(0.156)	(0.129)	(0.128)
Constant	5.489***	3.893***	5.626***	5.698***
	(0.733)	(1.216)	(0.736)	(0.734)
Observations	47831	38170	47831	47831
Firm controls	No	Yes	No	No

Note: Dependent variable is one if a compound is developed in-house, and zero otherwise. All specifications include a full set of therapeutic category and year indicators.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are heteroskedasticity-robust and clustered on the compound level.

Table 1.9 displays the estimation results for equation (1.3). Column (1) shows that the main effect of having at least one other patented compound in the same therapeutic class as the compound of interest (EOP1) on in-house decisions is positive and significant at the 1% level; while the interaction effect between EOP1 and PDM is negative and significant at the 1% level. When firm fixed effects are added in column (2), the main effect is still positive and the interaction effect still negative despite the fact that some coefficients are estimated less precisely. In columns (3) and (4), I use the count of other patented compounds in the same class (EOP2) and the expected number of other patented compounds that would reach the market (EOP3) as alternative measures of patent existence under my preferred specification with no firm fixed effects, and the results are qualitatively the same as those in column (1). Overall, the findings suggest that firms have the incentive to internalize their existing patented products by developing new compounds in the same therapeutic area as the patented ones in-house, but that this incentive becomes smaller as the amount of competing patents on the market from other firms increases.

Furthermore, I examine how the effect of patent length (LOP) on in-house decisions varies with the share of competing patented drugs on the market from other firms. I estimate a logit model similar to equation (1.3) except that now the explanatory variable of interest is a measure for patent length instead of patent existence. The model is specified as the following:

$$Prob(Y_{ijkt} = 1) = \Lambda(\delta_0 + \delta_1 LOP_{ijkt} + \delta_2 LOP_{ijkt} \times PDM_{jkt} + \delta_3 PDM_{jkt} + \delta_4 TDM_{kt} + \delta_5' X_{ijkt} + \delta_6 Z_{jkt} + \delta_7 W_{jt} + C_k + T_t + F_j). \quad (1.4)$$

Based on the logic of the planned *anti*-obsolescence theory, a firm has a strong incentive to internalize its existing patents of long duration, but that this incentive is weakened when the market is crowded with competing patents from other firms. Hence, the patent

Table 1.10: Logit Models of In-house Development: Length of Patents with Interaction Effect

	(1)	(2)	(3)	(4)	(5)
Length of Patents					
LOP1	0.049***	0.025			
	(0.011)	(0.015)			
LOP1 × PDM	−0.001***	−0.001			
	(0.000)	(0.000)			
𝟙(LOP1>10)			0.633***		
			(0.187)		
𝟙(LOP1>10) × PDM			−0.014***		
			(0.005)		
LOP2				0.062***	
				(0.023)	
LOP2 × PDM				−0.001*	
				(0.001)	
LOP3					0.324***
					(0.098)
LOP3 × PDM					−0.007***
					(0.002)
PDM	0.017	0.015	0.013	0.008	0.009
	(0.028)	(0.028)	(0.028)	(0.027)	(0.027)
TDM	−0.004	−0.006	−0.003	−0.004	−0.002
	(0.013)	(0.013)	(0.013)	(0.013)	(0.012)
Experience	0.035***	0.007	0.036***	0.022**	0.030***
	(0.008)	(0.008)	(0.008)	(0.009)	(0.010)
Scope	−0.608***	−0.418	−0.645***	−0.642***	−0.651***
	(0.203)	(0.509)	(0.203)	(0.199)	(0.197)
Phase I	−0.997***	−0.932***	−0.998***	−0.984***	−1.006***
	(0.143)	(0.164)	(0.143)	(0.142)	(0.142)
Phase II	−1.149***	−1.256***	−1.154***	−1.125***	−1.152***
	(0.128)	(0.156)	(0.128)	(0.129)	(0.128)
Constant	5.471***	3.894***	5.601***	5.638***	5.659***
	(0.734)	(1.227)	(0.735)	(0.738)	(0.734)
Observations	47831	38170	47831	47831	47831
Firm controls	No	Yes	No	No	No

Note: Dependent variable is one if a compound is developed in-house, and zero otherwise. All specifications include a full set of therapeutic category and year indicators.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are heteroskedasticity-robust and clustered on the compound level.

length measure LOP is expected to have a positive estimated coefficient (i.e., $\delta_1 > 0$) and the interaction term LOP X PDM is expected to have a negative estimated coefficient (i.e., $\delta_2 < 0$).

Table 1.10 presents the logit coefficients from estimating equation (1.4). Regardless of which measure of patent length is used, the results are consistent with the planned *anti*-obsolescence theory. Column (1) shows that the main effect of LOP1 on in-house decisions is positive and significant at the 1% level while the interaction effect between LOP1 and PDM is negative and significant at the 1% level. Column (2) adds in firm fixed effects, and the results continue to show a positive main effect and a negative interaction effect despite have less significant estimates. In column (3), I revert back to my preferred specification with no firm fixed effects, but instead of using a continuous measure for LOP1, I use an indicator for whether LOP1 has more than 10 years of patent protection time left. The results do not qualitatively change from those in column (1). Other measures of patent length such as LOP2 in column (4) and LOP3 in column (5) all give similar findings. The probability of in-house development for a compound increases with the amount of patent protection time left on other compounds in the same class and same firm as the compound of interest. Additionally, the increase in probability becomes smaller as the number of competing patented drugs on the market from other firms increases, holding fixed the total number of launched drugs in the same therapeutic area as the compound of interest.

1.5.3.2 Sales-based Market Share of Patented Drugs

In the second approach, I look at how a firm's own sales-based market share of patented drugs can affect in-house decisions for new compounds. This approach is limited to a smaller study sample compared to the first due to the availability of the IMS sales data

which only covers years between 1992 and 2004. Moreover, as discussed earlier in the data section, the analysis using the secondary dataset containing sales only focuses on the 12 therapeutic classes listed in Table 1.6(a).

Table 1.11: Logit Models of In-house Development: Market Share and Existence of Patents

	(1)	(2)	(3)	(4)
Panel A				
Current MSP	5.950	4.498	6.278	1.855
	(10.135)	(9.145)	(9.993)	(6.966)
EOP1		0.592**		
		(0.234)		
EOP2			0.043	
			(0.028)	
EOP3				0.308*
				(0.171)
Observations	26688	26688	26688	26688
Panel B				
Future MSP	24.447**	20.347**	24.221**	24.064**
	(11.866)	(10.097)	(11.428)	(10.945)
EOP1		1.385***		
		(0.522)		
EOP2			0.004	
			(0.026)	
EOP3				0.024
				(0.245)
Observations	7317	7317	7317	7317

Note: Dependent variable is one if a compound is developed in-house, and zero otherwise. All specifications include experience, scope, development phase indicators, as well as a full set of therapeutic category and year indicators.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are heteroskedasticity-robust and clustered on the compound level.

According to Testable Prediction 3, there is a positive relationship between the probability of in-house development for a new compound and the projected market share of same-class same-firm patented drugs by the time the new compound reaches the market. It is worthwhile to point out that because what really matters is the overlap in market

time between the new and old drugs on patent, a firm's projected future rather than current market share of patented drugs should be of more relevance when deciding whether to develop a new compound in-house or not.

In Table 1.11, I examine how market share together with each of the three measures for existence of patents (EOP) affect in-house or outsource decisions. The top panel of the table reports logit coefficients estimated from the following equation:

$$Prob(Y_{ijkt} = 1)$$

$$= \Lambda(\zeta_0 + \zeta_1 MSP_{ijkt} + \zeta_2 EOP_{ijkt} + \zeta_3' X_{ijkt} + \zeta_4 Z_{jkt} + \zeta_5 W_{jt} + C_k + T_t). \quad (1.5)$$

For each compound i belonging to originating firm j and therapeutic class k, MSP_{ijkt} is the market share based on the current year-t sales of patented drugs in the same firm and class as the compound of interest. The rest of the regressors are defined in the same way as those in equation (1.1). In the bottom panel of the table, I estimate an equation similar to equation (1.5) except that current MSP is replaced with projected future MSP. The equation takes the form:

$$Prob(Y_{ijkt} = 1)$$

$$= \Lambda(\eta_0 + \eta_1 MSP_{ijkt+\tau} + \eta_2 EOP_{ijkt} + \eta_3' X_{ijkt} + \eta_4 Z_{jkt} + \eta_5 W_{jt} + C_k + T_t). \quad (1.6)$$

Because the analysis focuses on integration decisions made early on in the development process, the value of MSP is forwarded a certain number of years to approximately account for a firm's projected market share of its patented drugs by the time the new compound reaches the market assuming successful development. Based on the DiMasi et al. (2003) estimates on the average time a compound spends in each development phase, I forward the value of MSP by 10 years for pre-clinical and phase I compounds and by 8 years for phase II compounds. Every specification in Table 1.11 includes

therapeutic class and year fixed effects. Standard errors are robust and clustered on the compound level.

I compare the results between the top and bottom panels of Table 1.11. The top panel shows that current year MSP is positively associated with in-house decisions, but the effect is not statistically significant. This is true even when the model does not include any patent profile measure as in column (1). In comparison, the positive relationship between future MSP and in-house development is statistically significant across all specifications as shown in the bottom panel. Furthermore, the probability of in-house development always increases with all measures of EOP, but the estimated coefficients on EOP are generally more statistically significant in the top panel than in the bottom. Taken together, these findings are in line with the planned *anti*-obsolescence theory: a firm is more likely to develop a new compound in-house if the firm owns other existing patented compounds in the same therapeutic class, and particularly so the more market share these existing patented compounds are expected to have by the time the new compound reaches the market.

Replacing patent existence (EOP) with length (LOP) in equations (1.5) and (1.6), Table 1.12 present estimation results on how market share together with LOP influence integration decisions in early drug development. Similar to the results in Table 1.11, future MSP is a more important positive predictor of in-house development than current MSP. When current MSP is included in the estimation as opposed to future MSP, the positive correlation between patent length and the probability of in-house development is generally more statistically significant. Hence, while an increase in patent length of existing compounds increases the likelihood of in-house development for a new compound in the same class and firm as the existing ones, the positive relationship is even stronger between projected future MSP and the probability of in-house development.

The results provide support for Testable Prediction 3: a 1 percentage point increase in projected MSP is associated with an increase in the probability of in-house development by 9.6-12.1 percentage points under the various specifications in Tables 1.11 and 1.12.

Table 1.12: Logit Models of In-house Development: Market Share and Length of Patents

	(1)	(2)	(3)
Panel A			
Current MSP	4.649	7.111	3.838
	(9.251)	(10.349)	(8.294)
LOP1	0.039**		
	(0.015)		
LOP2		0.038	
		(0.023)	
LOP3			0.280*
			(0.158)
Observations	26688	26688	26688
Panel B			
Future MSP	18.843**	24.072**	23.185**
	(9.391)	(11.272)	(10.565)
LOP1	0.085***		
	(0.028)		
LOP2		0.004	
		(0.020)	
LOP3			0.056
			(0.191)
Observations	7317	7317	7317

Note: Dependent variable is one if a compound is developed in-house, and zero otherwise. All specifications include experience, scope, development phase indicators, as well as a full set of therapeutic category and year indicators.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are heteroskedasticity-robust and clustered on the compound level.

1.5.4 Robustness Checks

In this subsection, I examine the robustness of the main analysis in two aspects. First, I look at whether the empirical results change in any meaningful way when the definition of in-house development becomes more encompassing. In the analysis done so far, a compound is considered to be developed in-house by the originator if it satisfies one of the two conditions: (a) it has never been licensed out to an outside firm; or (b) the earliest licensing date for the compound is after the beginning of Phase III trials. One might question the justification for condition (b) by arguing that the design and nature of compounds will mostly be fixed by as early as the completion of Phase I testing. Thus, for the purpose of this section, as long as a compound is not licensed out before the end of Phase I trials, it should be seen as one that is developed in-house. Using the new definition of in-house development, Tables A1.1 and A1.2 in the Appendix reports the results from re-running the main empirical tests on patent existence and length, respectively. There is still a positive and statistically significant relationship between the probability of in-house development and the different measures of patent existence and length of other compounds in the same class and firm as the compound in question.

Second, one might be concerned that the analysis is based on a therapeutic categorization that is too coarse to represent the economic market of compounds. If a firm owns two compounds in the same therapeutic class but serve different markets by nature regardless of the development process, then in such cases, choosing to develop one compound in-house will not be due to the firm's incentive to limit substitutability and protect the patent value of the other. To address this concern, I re-define my main explanatory variables, patent existence and length of other compounds in the same therapeutic area and same firm as the compound of interest, by using a set of narrower

therapeutic classifications to approximate the economic market. For instance, whereas before anti-arrhythmic and cardiostimulant were part of the same cardiovascular class, now each is given its own separate group. After redefining the key explanatory variables related to patent profile, I re-run the main empirical tests on patent existence and length. As shown in Table A1.3 in the Appendix, the results are still consistent with the earlier estimates, providing further evidence of a firm's incentive to practice planned *anti*-obsolescence when making integration decisions in development.

1.6 Alternative Explanations

There are two major alternative explanations for why firms keep development of some products in-house while outsourcing others. One classic explanation is that there is a trade-off between research effort and financial investment in successfully bringing a new product to market. The basic argument found initially in Aghion and Tirole (1994) is that when the marginal efficiency of a research unit's effort is greater than the marginal efficiency of a customer's investment, then R&D is more likely conducted in an integrated structure. Note that there, the focus is on the successful development of a new product instead of how the positioning of the new product could affect the patent value of existing goods.

While the mechanism proposed by Aghion and Tirole (1994) may exist in the real world, it is unlikely to be the correct explanation for the empirical results that I find. According to the authors' theory, one could argue that firms with existing patents may be less financially constrained when the existing patented products bring in a stream of revenue with high profit margins. Thus, my results on the positive relationship between in-house development and the patent profile measures may be merely driven by the

theory that firms with more financial resources are more likely to conduct R&D in-house. However, the financial resources argument cannot explain why current market share of existing patented drugs is *less* important in predicting in-house development than the projected market share of existing patented drugs by the time the new drug reaches the market (as shown in Tables 1.11 and 1.12).

Another potential explanation for integration decisions in development is that firms may be choosing to develop some products in-house in order to capitalize on past specific investment. Even though the analysis controls for experience and scope, one might continue to question whether the patent profile measures are still to some extent reflecting past specific investments. This interpretation, however, fails to explain why the positive correlation between in-house development and the various measures of patent profile is weaker when the market is crowded with competing patented drugs from outside firms (as shown in Tables 1.9 and 1.10). Furthermore, the specific investment interpretation also does not explain the empirical finding that a firm is more likely to develop a new compound in-house the more projected market share the firm expects to have from its existing patented drugs by the time the new drug gets launched. In summary, neither of the major alternative explanations can account for the empirical evidence as fully as the theory of planned *anti*-obsolescence proposed in this paper.

1.7 Conclusion

My paper employs the idea of planned *anti*-obsolescence to study integration decisions in R&D. I construct a model to show that the ownership of patents for existing products directly affects the way in which firms shape their new products, and the willingness (or lack thereof) to license products to outside firms for development. Because the licensees

do not internalize the value of the originator's existing patents while the originator itself does, the originator has more incentive to limit substitutability than the licensees. The model generates a set of theoretical predictions regarding in-house development, patent existence, patent length and market share of existing patented products.

I apply the model to the pharmaceutical industry to test the model's predictions. The empirical results are mostly in line with the planned *anti*-obsolescence theory. I find that, controlling for firm characteristics and unobserved therapeutic class heterogeneity, firms with existing patents are more likely to develop a new compound in the same class in-house compared to firms with no existing patents. In addition, the probability of in-house development increases with the patent length of other compounds in the same class and same firm as the compound in development. When considering the market position of a firm, I find that the positive relationship between in-house development for a compound of interest and measures of patent profile (existence and length) is weaker the more same-class patented drugs other firms own on the market, holding the total number of same-class drugs on the market fixed. Moreover, using market share data based on drug sales, the results show that the probability of a new compound being developed in-house increases with the originating firm's projected market share of its existing same-class patented drugs by the time the new compound reaches the market.

These findings suggest the important role that planned *anti*-obsolescence plays in determining in-house or outsource development decisions. When the originator wants to internalize the value of its existing patents and better limit substitutability in terms of new product design, development is more likely kept in-house than outsourced. Focusing on the timing of new product introductions when existing patents are about to expire — as opposed to the design of the new product when existing patents are fairly new — would lead to a complementary perspective on firms' in-house versus outsourced

choices, and is thus an interesting topic for future research.

1.8 Appendix

In the following proofs, I derive the mean product location and the investment level for location precision for both in-house and outsourced development, as well as the condition for when in-house development is preferred for each of the three scenarios regarding when the old patent expires. Using backward induction, I first fix the location of the new product, and derive the equilibrium price and profit in each period by solving the Salop Model. Note that once location is determined, the equilibrium price and profit are independent of where development takes place.

Thus, given any location l, denote $P_j^a(l)$ and $P_j^b(l)$ the equilibrium price for the new and old products in any period j in which the old product is still under patent protection; and denote $\pi_j^a(l)$ and $\pi_j^b(l)$ the corresponding equilibrium profit for the new and old products. When the patent for the old product expires, generic entry leads to marginal cost pricing for the old product, which means profit from the old product is zero (since $MC = 0$). Denote $P_j^{b,ex}(l)$ and $\pi_j^{b,ex}(l)$ as the equilibrium price and profit for the new product in any period j in which the old product is no longer under patent protection. Finally, recall in section 1.3.2.1 that I denote $\bar{l}^I(t)$ and $K^I(t)$ as the in-house choices of mean product location and investment level as functions of the old product length t, and $\bar{l}^I(t)$ and $K^I(t)$ as the outsourced counterparts.

Proof of Proposition 1

I prove part (i) and part (ii) of Proposition 1 for each of the three scenarios regarding when the old patent expires. Part (iii) of the proposition is shown by systematically comparing across the scenarios.

Scenario 1: old patent expires at the end of second period

The objective function for the in-house case is

$$\max_{\bar{l},K} \pi_1^a + \pi_2^a + \sum_{j=3}^{4}[p(K)\int_{\bar{l}-\alpha}^{\bar{l}+\alpha} \pi_j^{b,ex}(l)\frac{1}{2\alpha}dl + (1-p(K))\int_{\bar{l}-\beta}^{\bar{l}+\beta} \pi_j^{b,ex}(l)\frac{1}{2\beta}dl] - K - F$$

The objective function for the outsourced case is

$$\max_{\bar{l},K} \sum_{j=3}^{4}[p(K)\int_{\bar{l}-\alpha}^{\bar{l}+\alpha} \pi_j^{b,ex}(l)\frac{1}{2\alpha}dl + (1-p(K))\int_{\bar{l}-\beta}^{\bar{l}+\beta} \pi_j^{b,ex}(l)\frac{1}{2\beta}dl] - K - \gamma F$$

Since π_1^a and π_2^a do not depend on l, the first order conditions for $\bar{l}$ and K are the same in both cases. Hence, in the scenario where the old patent lasts two periods (i.e., $t = 2$), (i) $\bar{l}^I(2) = \bar{l}^O(2)$, and (ii) $K^I(2) = K^O(2)$.

Now prove that $\bar{l}^I(2) = \bar{l}^O(2) = \bar{l} = \frac{1}{2}$,

suppose the optimal $\bar{l}$ is not $\frac{1}{2}$. Given $\bar{l}$, let $P_j^{b,ex}(\bar{l}) = \hat{P}$, then

$$\int_{\bar{l}-\alpha}^{\bar{l}+\alpha} \pi_j^{b,ex}(l)\frac{1}{2\alpha}dl = \int_{\bar{l}-\alpha}^{\bar{l}+\alpha} \hat{P}\{\frac{V-\hat{P}}{2cv}(c-2\hat{P}) - \frac{1}{8vc}[2\hat{P}^2 - 2c\hat{P} + c^2(l^2+(1-l)^2)]\}\frac{1}{2\alpha}dl$$

$$= \hat{P}\frac{V-\hat{P}}{2cv}(c-2\hat{P}) - \frac{\hat{P}}{8vc}\{2\hat{P}^2 - 2c\hat{P} + \frac{c^2}{2\alpha}[4\alpha(\bar{l}-\frac{1}{2})^2 + \frac{4}{3}\alpha^3 + \alpha]\}$$

Similarly, $\int_{\bar{l}-\beta}^{\bar{l}+\beta} \pi_j^{b,ex}(l)\frac{1}{2\beta}dl = \hat{P}\frac{V-\hat{P}}{2cv}(c-2\hat{P}) - \frac{\hat{P}}{8vc}\{2\hat{P}^2 - 2c\hat{P} + \frac{c^2}{2\beta}[4\beta(\bar{l}-\frac{1}{2})^2 + \frac{4}{3}\beta^3 + \beta]\}$

Hence, for any $\bar{l} \neq \frac{1}{2}$ and $\hat{P}$, $\pi_j^{b,ex}$ can always be increased by keeping $\hat{P}$ and moving the mean location to $\bar{l} = \frac{1}{2}$. This proves that the optimal location is $\frac{1}{2}$ for both the in-house and outsourced case under scenario 1.

Note: let $K^I(2) = K^O(2) = K^*$, then K^* needs to satisfy the first order condition:

$$2p'(K^*)(E_\alpha(\pi^{ex}) - E_\beta(\pi^{ex})) = 1 \tag{A1}$$

where $E_\alpha(\pi^{ex}) = \int_{\frac{1}{2}-\alpha}^{\frac{1}{2}+\alpha} \pi^{ex}(l)\frac{1}{2\alpha}dl$ and $E_\beta(\pi^{ex}) = \int_{\frac{1}{2}-\beta}^{\frac{1}{2}+\beta} \pi^{ex}(l)\frac{1}{2\beta}dl$

Also in this case, development for the new product will be kept in-house when

$$\begin{aligned} &2p(K^I(2))E_\alpha(\pi^{ex}) + 2(1-p(K^I(2)))E_\beta(\pi^{ex}) - K^I(2) - F \\ >&2p(K^O(2))E_\alpha(\pi^{ex}) + 2(1-p(K^O(2)))E_\beta(\pi^{ex}) - K^O(2) - \gamma F - \tilde{\pi} \end{aligned}$$

Since $K^I(2) = K^O(2)$, the above condition is equivalent to the following:

$$\tilde{\pi} > (1-\gamma)F \tag{A2}$$

Scenario 2: old patent expires at the end of third period

The objective function for the in-house case is

$$\begin{aligned} \max_{\bar{l},K}\ &\pi_1^a + \pi_2^a + p(K)\int_{\bar{l}-\alpha}^{\bar{l}+\alpha}[\pi_3^a(l) + \pi_3^b(l) + \pi_4^{b,ex}(l)]\frac{1}{2\alpha}dl \\ &+ (1-p(K))\int_{\bar{l}-\beta}^{\bar{l}+\beta}[\pi_3^a(l) + \pi_3^b(l) + \pi_4^{b,ex}(l)]\frac{1}{2\beta}dl - K - F \end{aligned}$$

The objective function for the outsourced case is

$$\max_{\bar{l},K}\ p(K)\int_{\bar{l}-\alpha}^{\bar{l}+\alpha}[\pi_3^b(l) + \pi_4^{b,ex}(l)]\frac{1}{2\alpha}dl + (1-p(K))\int_{\bar{l}-\beta}^{\bar{l}+\beta}[\pi_3^b(l) + \pi_4^{b,ex}(l)]\frac{1}{2\beta}dl - K - \gamma F$$

(i) First prove that when the old patent lasts three periods (i.e., $t = 3$), $\bar{l}^I(3) = \bar{l}^O(3) = \frac{1}{2}$. Because I have already proved in the first scenario that $\int_{\bar{l}-\alpha}^{\bar{l}+\alpha} \pi_j^{b,ex}(l)\frac{1}{2\alpha}dl$ is maximized at $\bar{l} = \frac{1}{2}$, I only need to show that $\int_{\bar{l}-\alpha}^{\bar{l}+\alpha} \pi_3(l)\frac{1}{2\alpha}dl$ $(*)$ and $\int_{\bar{l}-\beta}^{\bar{l}+\beta} \pi_3(l)\frac{1}{2\beta}dl$ $(**)$ are also maximized when $\bar{l} = \frac{1}{2}$ (the superscripts on π are suppressed as in this scenario $\pi_3^a(l) = \pi_3^b(l)$). Because $(*) = \int_{\bar{l}-\alpha}^{\bar{l}+\alpha} \frac{1}{8v}[V - \frac{1}{4c}(c^2l^2 + c^2(1-l)^2)]^2\frac{1}{2\alpha}dl$, the first order condition of $\bar{l}$ gives $(\bar{l} - \frac{1}{2})(V + \frac{c}{4}(2\bar{l}^2 - 2\bar{l} + 2\alpha^2 + 1) = 0$, which means that $(*)$ is maximized when $\bar{l} = \frac{1}{2}$. Similarly, $(**)$ is also maximized when $\bar{l} = \frac{1}{2}$.

(ii) Now prove that $K^I(3) > K^O(3)$. The first order conditions for K in the in-house and outsourced scenarios are respectively:

$$p'(K^I(3))(E_\alpha(2\pi + \pi^{ex}) - E_\beta(2\pi + \pi^{ex})) = 1 \tag{A3}$$

$$p'(K^O(3))(E_\alpha(\pi + \pi^{ex}) - E_\beta(\pi + \pi^{ex})) = 1 \tag{A4}$$

Since $E_\alpha(\pi) > E_\beta(\pi)$, and $p(\cdot)$ is increasing and concave, comparing the above two equations gives $K^I(3) > K^O(3)$.

Note: in this case, development for the new product will be kept in-house when

$$\begin{aligned} &p(K^I(3))E_\alpha(2\pi + \pi^{ex}) + (1 - p(K^I(3)))E_\beta(2\pi + \pi^{ex}) - K^I(3) - F \\ >&p(K^O(3))E_\alpha(2\pi + \pi^{ex}) + (1 - p(K^O(3)))E_\beta(2\pi + \pi^{ex}) - K^O(3) - \gamma F - \tilde{\pi} \end{aligned}$$

Or equivalently,

$$\tilde{\pi} + B > (1 - \gamma)F \tag{A5}$$

where

$$B = [p(K^I(3)) - p(K^O(3))](E_\alpha(2\pi + \pi^{ex}) - E_\beta(2\pi + \pi^{ex})) + K^O(3) - K^I(3) \tag{A6}$$

$B > 0$ because $p(K)E_\alpha(2\pi + \pi^{ex}) + (1 - p(K))E_\beta(2\pi + \pi^{ex}) - K$ is maximized when $K = K^I(3)$.

Scenario 3: old patent expires at the end of fourth period

The objective function for the in-house case is

$$\max_{\bar{l},K} \pi_1^a + \pi_2^a + p(K)\sum_{j=3}^{4}\int_{\bar{l}-\alpha}^{\bar{l}+\alpha}[\pi_j^a(l) + \pi_j^b(l)]\frac{1}{2\alpha}dl + (1 - p(K))\sum_{j=3}^{4}\int_{\bar{l}-\beta}^{\bar{l}+\beta}[\pi_j^a(l) + \pi_j^b(l)]\frac{1}{2\beta}dl - K - F$$

The objective function for the outsourced case is

$$\max_{\bar{l},K} p(K)\sum_{j=3}^{4}\int_{\bar{l}-\alpha}^{\bar{l}+\alpha}\pi_j^b(l)\frac{1}{2\alpha}dl + (1 - p(K))\sum_{j=3}^{4}\int_{\bar{l}-\beta}^{\bar{l}+\beta}\pi_j^b(l)\frac{1}{2\beta}dl - K - \gamma F$$

(i) The proof that $\bar{l}^I(4) = \bar{l}^O(4) = \frac{1}{2}$ directly follows from the logic in scenario 2.

(ii) Now prove that $K^I(4) > K^O(4)$. The first order conditions for K in the in-house and outsourced scenarios are respectively:

$$p'(K^I(4))(E_\alpha(4\pi) - E_\beta(4\pi)) = 1 \tag{A7}$$
$$p'(K^O(4))(E_\alpha(2\pi) - E_\beta(2\pi)) = 1 \tag{A8}$$

Since $E_\alpha(\pi) > E_\beta(\pi)$, and $p(\cdot)$ is increasing and concave, comparing the above two equations gives $K^I(4) > K^O(4)$.

Note: in this case, development for the new product will be kept in-house when

$$\begin{aligned} &p(K^I(4))E_\alpha(4\pi) + (1 - p(K^I(4)))E_\beta(4\pi) - K^I(4) - F \\ >&p(K^O(4))E_\alpha(4\pi) + (1 - p(K^O(4)))E_\beta(4\pi) - K^O(4) - \gamma F - \tilde{\pi} \end{aligned}$$

Or equivalently,

$$\tilde{\pi} + B' > (1 - \gamma)F \tag{A9}$$

where

$$B' = [p(K^I(4)) - p(K^O(4))](E_\alpha(4\pi) - E_\beta(4\pi) + K^O(4) - K^I(4) \tag{A10}$$

$B' > 0$ because $p(K)E_\alpha(4\pi) + (1 - p(K))E_\beta(4\pi) - K$ is maximized when $K = K^I(4)$.

Finally, proof of Proposition 1 (iii) is based on a systematic comparison across the three scenarios. Specifically, comparing equations A1, A3 and A7 directly gives $K^I(t)$ increases in t. Similarly, comparing equations A1, A4 and A8 gives $K^O(t)$ increases in t. *Q.E.D.*

Proof of Proposition 2

The above analysis, particularly equations A2, A5, and A9, have already shown the threshold point of in-house development in each scenario. I now complete the proof of Proposition II by showing that $B' > B$.

From A10 and A6,

$$\begin{aligned} B' - B = &[(P(K^I(4)) - P(K^O(4)))(E_\alpha 4\pi - E_\beta 4\pi) - (K^I(4) - K^O(4))] \\ &- [(P(K^I(3)) - P(K^O(3)))(E_\alpha(2\pi + \pi^{ex}) - E_\beta(2\pi + \pi^{ex})) - (K^I(3) - K^O(3))] \end{aligned}$$

From A7 and A3, the above equation can be transformed to

$$B' - B = [\frac{P(K^I(4)) - P(K^O(4))}{P'(K^I(4))} - (K^I(4) - K^O(4))] - [\frac{P(K^I(3)) - P(K^O(3))}{P'(K^I(3))} - (K^I(3) - K^O(3))]$$

Since $V >> c$, it can be shown that $p >> p^{ex}$, which means $\frac{\partial \pi(l)}{\partial l} = (1-2l)\frac{c}{4v}p >> (1-2l)\frac{c}{4v}p^{ex} = \frac{\partial \pi^{ex}(l)}{\partial l}$. Also, since $\pi(l) > \pi^{ex}(l)$ for any l, it then follows that $E_\alpha(2\pi + \pi^{ex}) - E_\beta(2\pi + \pi^{ex}) \simeq E_\alpha(2\pi) - E_\beta(2\pi)$, and $P'(K^I(3)) \simeq 2P'(K^I(4))$ based on A3 and A7. Thus,

$$\begin{aligned}
B' - B &\simeq \frac{1}{P'(K^I(4))}[(P(K^I(4)) - P(K^O(4))) - P'(K^I(4))(K^I(3) - K^O(4))] \\
&\quad - \frac{1}{P'(K^I(3))}[(P(K^I(3)) - P(K^O(3))) - P'(K^I(3))(K^I(3) - K^O(3))] \\
&\simeq \frac{1}{2P'(K^I(4))}\{2[(P(K^I(4)) - P(K^O(4))) - P'(K^I(4))(K^I(4) - K^O(4))] \\
&\quad - [(P(K^I(3)) - P(K^O(3))) - P'(K^I(3))(K^I(3) - K^O(3))]
\end{aligned}$$

Assuming $p'(\cdot)$ is convex and that $p'(\cdot)$ decays sufficiently fast (s.t $|p''(x)| \leq \frac{1}{2}|\frac{p'(x)-p'(y)}{x-y}|$ whenever $p'(y) \simeq 2p'(x)$), it then follows that $B' - B > 0$. *Q.E.D.*

Table A1.1: Logit Models of a Different Measure for In-house Development: Existence of Patents

	(1)	(2)	(3)	(4)	(5)	(6)
Existence of Patents						
EOP1	0.406***	0.423**				
	(0.133)	(0.207)				
EOP2			0.065***	0.033*		
			(0.021)	(0.017)		
EOP3					0.140*	0.143*
					(0.083)	(0.079)
Experience	0.031***	0.004	0.007	−0.010	0.024**	−0.014
	(0.008)	(0.009)	(0.012)	(0.012)	(0.012)	(0.013)
Scope	−0.672***	−0.298	−0.615***	−0.185	−0.659***	−0.164
	(0.222)	(0.582)	(0.217)	(0.581)	(0.220)	(0.580)
Phase I	−1.013***	−0.873***	−0.986***	−0.880***	−1.005***	−0.878***
	(0.144)	(0.173)	(0.143)	(0.173)	(0.143)	(0.173)
Constant	6.942***	5.029***	6.904***	4.922***	7.033***	4.958***
	(1.223)	(1.700)	(1.225)	(1.718)	(1.225)	(1.719)
Observations	42962	32745	42962	32745	42962	32745
Firm controls	No	Yes	No	Yes	No	Yes

Note: Dependent variable is one if a compound is developed in-house, and zero otherwise. All specifications include a full set of the original therapeutic category indicators and year indicators.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are heteroskedasticity-robust and clustered on the compound level.

Table A1.2: Logit Models of a Different Measure for In-house Development: Length of Patents

	(1)	(2)	(3)	(4)	(5)	(6)
Length of Patents						
LOP1	0.025***	0.017				
	(0.008)	(0.012)				
LOP2			0.058***	0.031*		
			(0.018)	(0.017)		
LOP3					0.189**	0.176**
					(0.076)	(0.079)
Experience	0.031***	0.004	0.012	−0.005	0.022**	−0.011
	(0.008)	(0.009)	(0.011)	(0.011)	(0.010)	(0.011)
Scope	−0.687***	−0.269	−0.637***	−0.190	−0.663***	−0.147
	(0.221)	(0.580)	(0.215)	(0.579)	(0.218)	(0.577)
Phase I	−1.009***	−0.871***	−0.986***	−0.881***	−1.003***	−0.882***
	(0.144)	(0.173)	(0.143)	(0.173)	(0.143)	(0.173)
Constant	6.933***	5.076***	6.898***	4.939***	6.985***	4.932***
	(1.223)	(1.706)	(1.225)	(1.718)	(1.225)	(1.718)
Observations	42962	32745	42962	32745	42962	32745
Firm controls	No	Yes	No	Yes	No	Yes

Note: Dependent variable is one if a compound is developed in-house, and zero otherwise. All specifications include a full set of the original therapeutic category indicators and year indicators.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are heteroskedasticity-robust and clustered on the compound level.

Table A1.3: Patent Profile Variables Defined on Finer Therapeutic Classifications

	(1) EOP1	(2) EOP2	(3) EOP3	(4) LOP1	(5) LOP2	(6) LOP3
Existence of Patents (EOP)	0.284**	0.072*	0.044			
	(0.120)	(0.038)	(0.133)			
Length of Patents (LOP)				0.018**	0.058*	0.078
				(0.008)	(0.030)	(0.123)
Experience	0.036***	0.034***	0.041***	0.037***	0.035***	0.040***
	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)
Scope	−0.733***	−0.738***	−0.733***	−0.747***	−0.747***	−0.739***
	(0.198)	(0.197)	(0.199)	(0.197)	(0.197)	(0.199)
Phase I	−1.007***	−0.998***	−1.005***	−1.005***	−0.997***	−1.005***
	(0.142)	(0.142)	(0.142)	(0.142)	(0.142)	(0.142)
Phase II	−1.169***	−1.158***	−1.167***	−1.162***	−1.155***	−1.167***
	(0.128)	(0.128)	(0.128)	(0.128)	(0.128)	(0.128)
Constant	5.838***	5.819***	5.934***	5.835***	5.835***	5.910***
	(0.730)	(0.733)	(0.749)	(0.731)	(0.732)	(0.742)
Observations	47831	47831	47831	47831	47831	47831

Note: Dependent variable is one if a compound is developed in-house, and zero otherwise. All specifications include a full set of the original therapeutic category indicators and year indicators.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are heteroskedasticity-robust and clustered on the compound level.

CHAPTER 2:

WHAT HAPPENS WHEN PATENTS APPROACH EXPIRATION: TIMING AND LICENSING DECISIONS OF NEW PHARMACEUTICALS

2.1 Introduction

A significant body of economic research has examined brand companies' behavior prior to patent expiration in the pharmaceutical industry. Existing work has largely focused on entry deterrence motives towards generic drugs. Some of the most widely studied strategies include lower levels of advertising (Caves et al., 1991; Ellison & Ellison, 2011), authorized generic entry by brand companies (Hollis, 2002; Reiffen & Ward, 2007; Berndt et al., 2007; Appelt, 2015), and evergreening through patent and product line extensions (Danzon & Furukawa, 2011; Hemphill & Sampat, 2012).[1] While these papers shed some light on firms' strategic decisions to change future competitive conditions, relatively little is known concerning the extent to which pharmaceutical firms control the timing of new patented product introductions in response to patent expiration of their existing drugs.

In this paper, I argue that an originating firm faces urgency in the introduction of new drugs when its old ones are about to go off patent. This urgency comes from the firm's desire to prevent physicians from switching to a competitor's drug, as it is more costly to gain a new customer than to keep an existing one (Blattberg & Deighton, 1996; Valentin Ngobo, 2004). If the originator introduces a new product long after patent expiration, promoting it may be more difficult because by then, physicians may be pressured either by insurers to prescribe cheaper generic alternatives (Garattini & Tediosi, 2000; Sarpatwari et al., 2015), or by sales representatives from other companies

[1] Evergreening in the pharmaceutical industry refers to firms' strategies of acquiring additional patents for an existing drug or for variations of an original drug in order to delay or prevent generic competition.

to use those companies' own brand-name substitutes. That said, the firm also seeks to avoid introducing the new drug long before patent expiration, as in this case it may cannibalize the value of its currently patented drugs.

Echoing the above intuition, I develop a set of hypotheses concerning a firm's pipeline planning and licensing decisions in new compound development when the firm's patents for existing drugs are about to expire. First, an originator plans its pipeline progress such that at least one compound is well along in development in a therapeutic class when the originator has expiring patents for existing drugs in that class. Furthermore, because compounds in other therapeutic classes are unlikely to serve as replacements, the effect of expiring patents on the pipeline progress of those compounds should be weaker.

In terms of an originator's licensing decision, I hypothesize that due to its desire to control the timing of new patented product introductions, an originator is less likely to license out a late stage compound when it has expiring patents in the same therapeutic class. Licensing contracts rarely specify dates as to when a drug should be launched, likely due to the high degree of uncertainty in the R&D process, which makes contracting on dates too costly and infeasible. If left to a licensee, introduction may be delayed when the licensee's incentives are not perfectly aligned with the originator's – for example, when the licensee has a drug of its own that needs to be protected from cannibalization. However, the reduction in the probability of out licensing a compound should be smaller when the expiring patents and the compound are *not* in the same therapeutic class. This is because the control of timing is less important when the compound is unlikely to be a replacement for the drug that will go off patent soon.

To test these hypotheses, I use detailed compound development data between 1989 and 2004 from the Pharmaprojects dataset. My empirical analysis consists of two parts.

In the first part, I employ an ordered logit model to examine the relationship between a firm's pipeline progress and its expiring patents. The analysis is conducted on the firm therapeutic class level. Results confirm my hypothesis: I find that controlling for firm heterogeneity, a firm is more likely to reach a more advanced stage of development for its pipeline products in a therapeutic class when the firm also has expiring patents in that class. Further supporting my hypothesis, the effect is less pronounced when the expiring patents belong to a different class.

The second part of the empirical analysis focuses on the compound level. I use a logit model to study how a firm's licensing decisions in new compound development relate to its expiring patents for existing drugs. I find that controlling for differences in firm characteristics, the likelihood of an originator out licensing a late stage compound is negatively associated with the originator's expiring patents (both existence and count) within the same therapeutic class. This association is statistically insignificant, however, when the expiring patents are *not* in the same therapeutic class as the compound of interest. Overall, the findings are all consistent with a firm's desire to control the timing of new patented drug introductions so that a replacement is readily available once the firm's patent for an old drug expires.

The rest of the paper proceeds as follows. Section 2.2 reviews relevant literature on firm strategic behavior prior to patent expiration. It also develops a set of hypotheses concerning a firm's pipeline planning and licensing decisions in new compound development when the firm's existing drugs are pending patent expiration. Section 2.3 describes the dataset used in the paper and presents summary statistics. In Section 2.4, I discuss the empirical methods used in testing the hypotheses and present the estimation results. I conclude in Section 2.5.

2.2 Firm behavior facing patent expiration

2.2.1 Literature review

This paper joins together two distinct strands of literature on economic issues in the pharmaceutical industry. One strand focuses on firms' defensive strategies prior to patent expiration, while the other looks at firms' integration decisions in new product development.

The literature on firm strategic behavior in response to patent expiration has largely centered around whether brand companies can deter generic entry through advertising, authorized generic entry, and evergreening. Caves et al. (1991) point out that an originator's entry deterrence motivation only lowers its advertising level on the margin. The overall picture still seems to suggest that the originator takes generic entry after patent expiration as a given and optimizes accordingly. More recent work by Ellison and Ellison (2011) also shows limited evidence that firms engage in entry-deterring behavior through lower levels of advertising as patents approach expiration. In addition, findings with regard to how authorized generics from brand companies can affect the entry decisions of independent generic companies are mixed. Hollis (2002) and Reiffen and Ward (2007) show that brand companies introduce authorized generics to crowd out independent generic entrants; however, Berndt et al. (2007) and Appelt (2015) find that the effect is insignificant. Finally, there is little evidence that evergreening through acquiring additional patents for a drug increases its effective market life (Hemphill & Sampat, 2012). Nor is there evidence that product line extensions through new formulations can reduce the probability of generic entry or the number of generic entrants (Danzon & Furukawa, 2011).

While the above papers all focus on evaluating the potential entry deterrence strategies of an originator facing patent expiration, none pays close attention to the originator's timing strategy regarding new pharmaceuticals. Given that a considerable amount of previous work suggests that brand companies are unsuccessful in preventing or delaying generic entry, this paper assumes from the beginning that the originator anticipates entry by generic manufacturers following patent expiration.

More importantly, the paper argues that in order to maintain its customer base, an originator with expiring patents faces urgency in the introduction of new patented drugs. This urgency exists because once the originator's old drug goes off-patent, insurance companies may pressure physicians to prescribe generic versions of the old treatment. In addition, sales representatives from other firms may see an opportunity to market their brand-name drugs and encourage the physicians to switch over. Once the originator loses its customers to generic entrants or other brand competitors, getting these customers back in the future may be more costly (Blattberg & Deighton, 1996; Valentin Ngobo, 2004). To prevent this from happening, the originating firm thus has the incentive to control the timing of its new pharmaceuticals so that it can convince the physicians to start prescribing the new treatment either just before or soon after the old drug goes off patent. By focusing on the urgency of new product introduction, this paper explores the effects of expiring patents on a firm's pipeline planning and licensing decisions in new compound development, and in turn contributes to the literature on firm strategic behavior prior to patent expiration.

Another strand of literature that the paper relates to is on a firm's integration decisions. A number of papers use the incomplete contract framework first proposed by Grossman and Hart (1986) to study the organization of innovation and product development (e.g. Aghion & Tirole, 1994; Lerner & Merges, 1998; Azoulay, 2004). Aghion and

Tirole (1994) show that firms are more likely to conduct R&D under an integrated structure when capital input is more important than intellectual input. Lerner and Merges (1998) empirically support this theory using alliance data from the biotechnology industry. Azoulay (2004) finds empirical evidence that pharmaceutical firms are more likely to outsource data-intensive clinical projects while keeping knowledge-intensive ones in-house. However, little research has been done on how patent expiration can change the relative importance of management control between the originating company and the potential licensing partner, and in turn affect the originating company's decision to license out a compound or not. This study takes contract incompleteness as a starting point, and provides empirical analysis on the impact of patent expiration on a firm's integration decisions in new drug development.

Only a few papers thus far have offered insights about mergers and patent expiration in the pharmaceutical industry. Higgins and Rodriguez (2006) show that a firm with a high desperation index is more likely to engage in acquisition to replenish its pipeline.[2] Similarly, Danzon et al. (2007) find that for a large firm, a higher percentage of its drugs near the end of their patent life is associated with a higher probability that the firm will acquire another company. Both of these papers study patent expiration at the firm level, and do not differentiate between therapeutic classes. This paper uses detailed compound-level data to examine a firm's licensing decisions, and furthermore, distinguishes between whether the expiring patent is in the same therapeutic class as the compound of interest or not.

[2]Higgins and Rodriguez (2006) define a firm's desperation index based on its pipeline productivity and the length of patent protection years for the firm's products. The authors assign a high desperation index to firms that have both a decreasing expected number of pipeline products that will reach the market and decreasing total patent protection years in the firms' portfolio.

2.2.2 Hypotheses on timing and licensing decisions

In order to analyze the pipeline planning and licensing decisions of an originating firm, I argue that an originator has a desire to control the timing of the introduction of new patented pharmaceuticals when its patents for existing drugs in the same therapeutic class are about to expire. The reason is that the cost of acquiring a customer is lower when the customer is already using one of the firm's products than it would be if the customer were using a competitor's product (Blattberg & Deighton, 1996; Valentin Ngobo, 2004). What this means is that the originating firm will want to have a replacement drug in time so that its sales representatives can encourage physicians to make the switch from its patented drug that will expire soon to the new one. If the replacement drug is not ready in time, some patients may start using substitute drugs (generic or brand-name), thereby making it more difficult for the originating firm to get these patients back in the future.

It is worthwhile to note that this switching cost argument is also similar to the issue of therapeutic category specific human capital. Pharmaceutical companies often have large marketing and sales forces that focus on particular therapeutic classes, allowing the companies to maintain or expand their customer bases. If the sales representatives have no new drug to work with, companies may either have to fire them and re-establish relationships with physicians later, or just pay the sales forces to do nothing, neither of which is optimal from a profit-maximizing standpoint (Danzon et al., 2007).

As a result, an originator may plan its pipeline such that at least one compound is well along in the development process when an existing patent in the same therapeutic class is approaching expiration. Moreover, the originator may be less likely to license out that compound for two related reasons. First, the originator and the potential licensee may have misaligned incentives in the timing of the new product. While the originator

wants to prevent its customers from switching over to a rival firm's drugs, the licensee may have other considerations – such as the cannibalization of its own products – that may delay the introduction of the originator's new drug. Second, licensing contracts in the pharmaceutical industry rarely specify dates for when a drug should be brought to market, likely because the R&D process is sufficiently uncertain that it may be too costly to specify a launch date. Hence, if the control of timing is crucial, an originator may be more reluctant to license out development to an outside firm.

In this paper, I test the following hypotheses with respect to the effect of expiring patents on a firm's timing and licensing decisions in new product development.

(1) A firm with a drug going off-patent soon is more likely to have a compound in the same therapeutic class that is at a relatively later stage of development.

(1b) The level of advancement that a firm reaches for its pipeline compounds in a therapeutic class is less affected by expiring patents from other classes.

(2) The probability of a firm licensing out a late-stage compound is lower when the firm has a drug with an expiring patent in the same therapeutic class.

(2b) An expiring patent has less of an effect on the originating firm's licensing decisions regarding new compounds when the expiring patent is not in the same therapeutic class as the new compounds in development.

2.3 Data

This paper uses the Pharmaprojects dataset, which tracks the development of all compounds in active R&D throughout the world. For each compound, the dataset provides

information on the names of the compound, its originating company, its therapeutic categories, development phases, patent status and filing dates if any, whether the compound has ever been licensed out, and if so, the names of the licensees and the corresponding licensing dates. In the data used in this paper, there are 13,645 unique compounds and 630 originating firms for the period between 1989 and 2004.

The dataset contains a considerable number of right-censored observations. This is because some compounds may not have any further information regarding the success or failure of development beyond a certain clinical trial phase (Danzon et al., 2005). Following Danzon et al. (2005), I consider a compound with right-censored observations to have failed in a development phase if the time it spent in that phase is longer than 95% of the compounds that have successfully completed that phase. The failure thresholds in my data are 6 years for pre-clinical, 4 years for phase I, 6 years for phase II, and 7 years for phase III. I drop the observations that are beyond a failure threshold for a compound with no further development information.

The two main outcome variables that I examine are the development phase and the licensing status of a pipeline product. Specifically, the first outcome measure, which I refer to as Furthest Along, is a categorical variable that describes the development phase of the compound that is the furthest along in development compared to other compounds in the same therapeutic class and firm. I aggregate the data to the firm therapeutic class level to construct this variable. This measure takes on the value 1 when a firm only has compounds in the pre-clinical stage for a therapeutic class and no compounds in the same class beyond that stage; Similarly, it takes on values 2,3,4, or 5 when the most advanced development status a firm has reached for that particular therapeutic class is phase I, phase II, phase III, or launched, respectively. This variable's purpose is to capture whether a firm is engaged in pipeline planning in anticipation of an expiring

patent.

My second outcome variable concerns a firm's licensing decision regarding compounds in development (License Out). It is an indicator on the compound level that equals 1 when an originating firm licenses out a compound for development and 0 otherwise. This variable is designed to capture whether the impact of expiring patents on a firm's licensing decisions is consistent with the firm's desire to control the timing of new pharmaceuticals during this crucial period.

In terms of my explanatory variables, I have four measures that characterize expiring patents. For each compound, I construct an indicator and a count of on-the-market drugs in the same therapeutic class and firm that are within 5 years of patent expiration.[3] I also construct an additional set of indicator and count variables for when the expiring patents are *not* in the same therapeutic class but in the same firm as the compound in development. This is to explicitly examine whether the effects of expiring patents on a firm's timing and licensing decisions of new compounds are therapeutic class-specific or not.

To control for differences in firm capabilities, I create a number of firm-level variables for the empirical analysis. Licensing propensity is the proportion of compounds that are licensed out within a therapeutic class for a firm in a given year. Therapeutic experience is a cumulative count of development records (i.e. compound-year observations) that a firm has for a therapeutic class up to the year of interest.[4] Total experience is a cumulative count of a firm's total development records across all therapeutic classes.

[3] A 5-year window period strikes a reasonable balance between having enough observations that are exposed to situations where there is an expiring patent and still being qualified as just before patent expiration. Also, as a motivating example, AstraZeneca, the originator of the blockbuster heartburn drug Prolisec, started to develop a replacement for Prolisec 6 years before its patent expired (Harris, 2002).

[4] The therapeutic experience variable gives more credit to the compound that goes further in development, and also gives more credit to the compound that takes longer in development. What this variable captures is that the more time a company spends in a therapeutic category, the more likely it has the capabilities to develop a drug in that area.

Table 2.1: Definition of Constructed Variables

Variable	Description
Furthest Along	Categorical variable for the highest level of development a firm has achieved for its pipeline compounds in a therapeutic class
License Out	Indicator equals 1 if a compound is licensed out for development
1(Same-class expiring patent)	Indicator equals 1 if there exists an expiring patent in the same therapeutic class as the compound in question
# Same-class expiring patent	Number of expiring patents in the same therapeutic class as the compound in question
1(Different-class expiring patent)	Indicator equals 1 if there exists an expiring patent in a different therapeutic class than the compound in question
# Different-class expiring patent	Number of expiring patents in a different therapeutic class than the compound in question
Out-licensing Propensity	The percentage of compounds a firm licensed out within a therapeutic class in a given year
Therapeutic experience	A cumulative count of development records a firm has within a therapeutic class in a given year
Total experience	A cumulative count of total development records a firm has in a given year
Scope	HHI index calculated by the sum of squares of the percentage of compounds developed for each therapeutic class within a firm in a given year
Small to medium sized firms	Indicator equals 1 if a firm is small to medium sized

To quantify how diverse a firm's pipeline products are, I measure a firm's therapeutic scope with a Herfindahl-Hirschman Index calculated based on the sum of squares of the percentage of compounds that are developed for each therapeutic class in a given year (Danzon et al., 2005). Finally, I capture firm size with an indicator variable – it takes on the value 1 for small-to-medium sized firms, i.e., those with less than 500 development records between the study period of 1989 and 2004, and takes on the value 0 for the 22

Table 2.2: Summary Statistics for Compound Level Variables

Variable	N	Mean	Std. Dev.	Min	Max
License Out	71.218	0.264	0.441	0	1
1(Same-class expiring patent)	71.218	0.145	0.352	0	1
# Same-class expiring patent	71.218	0.198	0.548	0	7
1(Different-class expiring patent)	71.218	0.360	0.480	0	1
# Different-class expiring patent	71.218	1.300	2.351	0	13
Pre-clinical	71.218	0.723	0.447	0	1
Phase I	71.218	0.071	0.257	0	1
Phase II	71.218	0.098	0.297	0	1
Phase III	71.218	0.071	0.258	0	1
Launch	71.218	0.036	0.186	0	1
Therapeutic categories					
Alimentary/metabolic	71.218	0.047	0.211	0	1
Anticancer	71.218	0.108	0.311	0	1
Anti-infective	71.218	0.125	0.331	0	1
Anti-parasitic	71.218	0.004	0.064	0	1
Biotechnology	71.218	0.187	0.390	0	1
Blood and clotting	71.218	0.034	0.181	0	1
Cardiovascular	71.218	0.080	0.271	0	1
Dermatological	71.218	0.016	0.127	0	1
Formulations	71.218	0.115	0.319	0	1
Genitourinary	71.218	0.018	0.135	0	1
Hormonal products	71.218	0.009	0.096	0	1
Immunological	71.218	0.023	0.151	0	1
Miscellaneous	71.218	0.012	0.108	0	1
Musculoskeletal	71.218	0.053	0.224	0	1
Neurological	71.218	0.128	0.334	0	1
Respiratory	71.218	0.034	0.180	0	1
Sensory	71.218	0.006	0.078	0	1

large firms that had 500 development records or more. Table 2.1 provides a list of all the constructed variables.

Table 2.2 presents summary statistics for the compound-level variables. Approximately 26% of the observations are developments that are licensed out. In any given year, about 15% of the compounds are in the same therapeutic class and same firm as an expiring patent. The mean number of expiring patents in the same therapeutic class and

Table 2.3: Summary Statistics for Firm Level Variables

Variable	N	Mean	Std. Dev.	Min	Max
Furthest Along=Pre-clinical	19.090	0.491	0.500	0	1
Furthest Along=Phase I	19.090	0.085	0.279	0	1
Furthest Along=Phase II	19.090	0.167	0.373	0	1
Furthest Along=Phase III	19.090	0.162	0.368	0	1
Furthest Along=Just Launched	19.090	0.093	0.290	0	1
Out-licensing propensity	19.090	0.327	0.397	0	1
Therapeutic experience	19.090	30	69	1	1.157
Total experience	6.046	103	359	1	5.972
Scope	6.046	0.577	0.315	0	1
Small to medium sized firm	630	0.967	0.180	0	1

firm as a given compound is 0.20. On the other hand, 36% of the compounds belong to a firm that has an expiring patent in a therapeutic class *different* from that of the compound in question. Furthermore, a compound is on average associated with 1 launched drug that is in the same firm but a *different* therapeutic class, and that will go off patent soon. The table also contains information on the development stages of a compound and the therapeutic categories that this paper focuses on. Not surprisingly, the pre-clinical testing stage has far more observations than the later development stages as compounds may fail in a clinical trial and be discontinued in development.

In Table 2.3, I show summary statistics for the firm-level variables. Looking at a firm's pipeline of compounds in development, 49% of the firms have pre-clinical testing as their furthest development stage for a given therapeutic class and year. The corresponding proportions for firms with phase I, II and III as their highest levels of testing are 9%, 16%, and 17%, respectively. 9% of the firms have at least one compound that is just launched from the pipeline for a given therapeutic class and year. The table also shows that averaging across therapeutic categories and years, a firm licenses out about 33% of its compounds in development. Both a firm's therapeutic experience and total

experience are heavily skewed to the right, so I log-transform them in the regression analysis. On average, firms are fairly concentrated in their scope of development – the mean HHI for a firm's therapeutic scope is 0.58. Finally, most firms in the dataset are considered small-to-medium sized, with the exception of the 22 large firms that have more than 500 development records between the study period.

2.4 Regression analysis

My analysis consists of two parts. The first is on the firm therapeutic class level, focusing on the effect of expiring patents on a firm's pipeline planning decision.[5] The second is on the compound level, looking at the relationship between expiring patents and a firm's licensing decision regarding new compounds.

I begin by exploring how a firm's pipeline progress for a therapeutic class – in terms of the highest level of development in its pipeline – relates to its expiring patents, distinguishing between whether the expiring patents are in the therapeutic class of interest or not. To accommodate the discrete nature of development level, I use an ordered logit to first estimate the following latent equation:

$$Furthest\_Along^*_{jkt} = \alpha_0 + \alpha_1 expiring\_patents_{jkt} + \alpha_2 Z_{jkt} + C_k + T_t + \epsilon_{jkt}. \quad (2.1)$$

The subscripts j, k, and t index firm, therapeutic class, and year, respectively. $Furthest\_Along^*$ is a latent continuous variable. In reality, rather than observing $Furthest\_Along^*$, I observe the development level itself:

$$Furthest\_Along_{jkt} = d, \text{ if } \mu_{d-1} < Furthest\_Along^*_{jkt} \leq \mu_d$$

[5] I examine in any given year, whether a firm's most advanced development stage for its pipeline products in a therapeutic class is pre-clinical, Phase I,Phase III, Phase III, or just launched.

where $d = 1, 2, 3, 4, 5$ corresponds to the development stages: pre-clinical testing, Phase I, Phase II, Phase III, and just launched. The μ's are unobserved thresholds,[6] and are estimated together with the coefficients (β's).

The key explanatory variable in equation (2.1), *expiring_patents*, is either an indicator or a count of a firm's marketed drugs that are within 5 years of patent expiration in the therapeutic class of interest. Z is a vector of firm-level characteristics such as therapeutic experience, total experience, development scope and firm size. The equation also includes a set of therapeutic class indicators (C_k) and year indicators (T_t). The random error term (ϵ) is assumed to follow a logistic distribution. All standard errors are robust and clustered on the firm therapeutic class level.

To further examine whether the effect of patent expiration is therapeutic class specific or not, I also estimate a model very similar to equation (2.1) except that now the key explanatory variable is an indicator or a count of a firm's soon-to-be off-patent drugs that are *not* in the therapeutic class of interest. If firms are primarily concerned with having a replacement drug, then the relationship between a firm's expiring patents and its pipeline progress should be weaker when the two do *not* pertain to the same therapeutic class.[7]

In the second part of my analysis, I investigate the association between patent expiration and a firm's licensing decisions of new compounds. I use a logit specification to define the probability that compound i in firm j and therapeutic class k is licensed out

[6] Note that $\mu_0 = -\infty$ and $\mu_5 = \infty$.

[7] Alternatively, one could examine how a firm's pipeline progress for a therapeutic class relates to the existence and count of expiring patents in the firm's *overall* portfolio (including both the expiring patents that belong to the therapeutic class of interest and those that do not belong). In Table A2.1 of the Appendix, I find that consistent with my argument, the effect of expiring patents in a firm's overall portfolio on the firm's pipeline progress for a particular therapeutic class is weaker than the effect of expiring patents in the therapeutic class of interest.

for development in year t:

$$\text{Prob}(License\_Out_{ijkt} = 1) = \Lambda(\beta_0 + \beta_1 expiring\_patents_{ijkt} + \beta_2 \tilde{Z}_{ijkt} + C_k + T_t). \quad (2.2)$$

$\Lambda(\cdot)$ is a standard logistic cumulative distribution function. The key explanatory variable in equation (2.2), $expiring\_patents_{ijkt}$, can be an indicator or a count of on-the-market drugs that are within 5 years of patent expiration and that are in the same therapeutic class as the compound of interest. The vector of firm level characteristics, $\tilde{Z}$, is similarly defined as in equation (2.1). The only difference is that it now also includes a firm's licensing propensity within a therapeutic class to capture heterogeneous firm preferences in internal or external development.

The equation is estimated separately for early development (i.e. pre-clinical and phase I) and late development (i.e. phase II and phase III) compounds. This is because late stage compounds stand the most chance of being brought to market in time to replace the drug with an expiring patent. Accordingly, a firm may be more interested in controlling the timing of a late stage compound and be more reluctant to license it out compared to an early stage one, pending patent expiration. To account for potential correlation of licensing decisions for a particular compound across years, I use robust standard errors that are clustered on the compound level.

Finally, I examine whether an expiring patent in one therapeutic class can affect a firm's decision to license out a compound in another class. I estimate a model similar to equation (2.2) except that now the key explanatory variable is either an indicator or a count of marketed drugs that are within 5 years of patent expiration and that are *not* in the same therapeutic class as the compound of interest. If firms are interested in controlling the timing of new product introduction because they want patients to switch from an old drug at the end of its patent life to a new one, then an expiring patent in one therapeutic class should not discourage a firm's licensing behavior of new compounds

in other classes.[8]

2.4.1 Firm-level analysis: pipeline planning

I first discuss results for how a firm's pipeline planning decision for a therapeutic class relates to its expiring patents in that class. Table 2.4 reports the estimates from equation (2.1). The first column shows that a firm is more likely to have a compound further along in development for a therapeutic class when the firm also has an expiring patent in that class. Furthermore, the positive coefficient in front of the patent expiration indicator is statistically significant at the 5% level. In column (2), the level of a firm's pipeline progress in a therapeutic class is also positively associated with the number of expiring patents in that class, and the relationship between the two is statistically significant at the 1% level. Controlling for heterogeneity across firm capabilities, both columns provide evidence in line with hypothesis (1) that firms engage in pipeline planning in anticipation of patent expiration.

Additionally, based on the estimates from Table 2.4, I compute the average marginal effects for the patent expiration variables. I find that if a firm has an expiring patent in a therapeutic class, this will reduce the probability of having pre-clinical testing as the firm's most advanced development stage for its pipeline compounds in that class by 5.3 percentage points, or a 10.8% decrease from baseline.[9] On the other hand, the probabilities that the firm reaches phase III as its highest level of development for a given year

[8] Similar analyses were performed to investigate how expiring patents in a firm's *overall* portfolio affect the firm's licensing decisions concerning new compounds. Consistent with the argument that expiring patents in one therapeutic class should not discourage a firm's decision to license out a compound in another class, results from Table A2.2 in the Appendix show that expiring patents in a firm's *overall* portfolio do not discourage its licensing decision as much as expiring patents in the *particular* therapeutic class that the compound of interest belongs to.

[9] See Table 2.3 for the baseline probabilities of having a certain testing stage as a firm's highest level of development

Table 2.4: Same Therapeutic Class, Expiring Patents and Pipeline Progress

Ordered Logistic Regressions Dependent Variable: Furthest Along	(1)	(2)
1(Same-class expiring patent)	0.294**	
	(0.140)	
# Same-class expiring patent		0.373***
		(0.105)
Ln(Therapeutic experience)	1.236***	1.228***
	(0.054)	(0.053)
Ln(Total experience)	−0.262***	−0.260***
	(0.053)	(0.053)
Scope	−0.098	−0.093
	(0.178)	(0.177)
Small to medium sized firm	0.194	0.203
	(0.140)	(0.140)
Cutoff point 1	−3.018***	−0.761**
	(0.365)	(0.383)
Cutoff point 2	−2.532***	−0.277
	(0.365)	(0.383)
Cutoff point 3	−1.492***	0.764**
	(0.363)	(0.384)
Cutoff point 4	0.041	2.302***
	(0.362)	(0.386)
N	19042	19042

Note: The unit of observation is firm-therapeutic class-year. The dependent variable is the most advanced development stage a firm has achieved for its pipeline compounds in a therapeutic class for a given year. All specifications include therapeutic class and year indicators.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Robust standard errors (in parentheses) are clustered on the firm therapeutic class level.

or just launches a drug from its pipeline both increase by about 2.1 percentage points, translating into a 13.0% or a 22.6% increase from the respective baselines. Moreover, I also find that with each additional drug near the end of its patent life that a firm has for a therapeutic class, the probability of the firm only progressing as far as the pre-clinical stage for its pipeline compounds in that class declines by 13.7% from baseline. The probability of the firm progressing as far as phase III increases by 16.7%, while the

probability of launching a drug increases by 28.0%.

Next, I consider whether a firm's pipeline progress for a therapeutic class is affected by expiring patents in *other* classes. While I argue that with patent expiration approaching, firms have the incentive to bring out a new drug quickly so that they can switch patients to the new one and maintain their market position, it could also be that firms are interested in having a continuous stream of profits in order to fund their R&D activities (Ravenscraft & Long, 2000; Vernon, 2005). If the latter were the case, then a firm faced with patent expiration would want to have a compound well along in development or ready to be launched in any therapeutic class, and not just the class that the expiring patent belonged to.

Table 2.5 reports the results from an ordered logistic regression where the dependent variable is the highest level of development a firm has achieved for its pipeline compounds in a therapeutic class and the key explanatory variable is expiring patents in *other* classes. Column (1) shows that the indicator for the existence of expiring patents in *other* classes has a positive coefficient. However, the point estimate is less statistically significant and smaller than that of the same-class expiring patents indicator in Table 2.4.[10] Furthermore, in column (2) of Table 2.5, the estimated coefficient for the count of other-class expiring patents is negative and statistically insignificant. Taken together, these results are consistent with hypothesis (1b) – the relationship between a firm's pipeline progress and its expiring patents is weaker when the two do not pertain to the same therapeutic class. The results also suggest that the steady profit flow argument is unlikely to be the main factor that is driving the findings in Table 2.4. Overall, analysis on the firm level provides supporting evidence that a firm faced with an expiring

[10]The point estimate for the existence of *same-class* expiring patents in Table 2.4 is 0.294 and significant at the 5% level, while the point estimate for the existence of *different-class* expiring patents in Table 2.5 is 0.179 and significant at the 10% level. The difference in magnitude between the two estimates is not statistically significant.

patent engages in pipeline planning in order to have a replacement drug in time for its customers to switch.

Table 2.5: Different Therapeutic Class, Expiring Patents and Pipeline Progress

Ordered Logistic Regressions Dependent Variable: Furthest Along	(1)	(2)
1(Different-class expiring patent)	0.179*	
	(0.094)	
# Different-class expiring patent		−0.006
		(0.023)
Ln(Therapeutic experience)	1.251***	1.249***
	(0.054)	(0.054)
Ln(Total experience)	−0.281***	−0.262***
	(0.053)	(0.053)
Scope	−0.121	−0.103
	(0.178)	(0.178)
Small to medium sized firm	0.234	0.174
	(0.144)	(0.144)
Cutoff point 1	−0.799**	−0.787**
	(0.382)	(0.383)
Cutoff point 2	−0.313	−0.302
	(0.383)	(0.383)
Cutoff point 3	0.727*	0.739*
	(0.384)	(0.385)
Cutoff point 4	2.257***	2.267***
	(0.385)	(0.386)
N	19042	19042

Note: The unit of observation is firm-therapeutic class-year. The dependent variable is the most advanced development stage a firm has achieved for its pipeline compounds in a therapeutic class for a given year. All specifications include therapeutic class and year indicators.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Robust standard errors (in parentheses) are clustered on the firm therapeutic class level.

2.4.2 Compound-level analysis: licensing decisions

I now examine the effect of expiring patents on a firm's licensing decisions regarding new compounds. Table 2.6 displays the logit estimation results from equation (2.2) where the dependent variable is an indicator for whether a compound is licensed out for development. Columns (1) and (2) show that when looking at the licensing decisions for early stage compounds (i.e. pre-clinical and phase I), the effect of a same-class expiring patent (measured by existence or number) is not statistically significant. However, this is not the case for late stage compounds (i.e. phases II and III). Consistent with hypothesis (2), column (3) shows that a firm is less likely to license out a late stage compound when the firm has an expiring patent in the same therapeutic class as the compound of interest. Column (4) shows that this likelihood is also decreasing with the number of same-class expiring patents.

As discussed earlier, compared to early stage compounds, the ones further along in development stand the most chance of being brought to market in time to serve as replacements. Thus, facing patent expiration of its existing drug, an originating firm will especially want to control the timing for the late stage compounds by not licensing them out. Otherwise, the originator runs the risk of failing to introduce a replacement drug in time; its customers may switch to a competitor's drug, at which point it may be more difficult to get these customers back.

Based on the estimates in Table 2.6, I calculate the average marginal effect of a firm's expiring patents on its probability of out licensing a late stage compound within the same therapeutic class. I find that having at least one expiring patent reduces this probability by 1.2 percentage points, equivalent to a 4.5% decrease from baseline. Furthermore, this probability also decreases by about the same magnitude with every additional drug in the firm's portfolio that will go off patent soon.

Table 2.6: Same Therapeutic Class, Expiring Patents and Licensing Decision

Logit Regressions	(1)	(2)	(3)	(4)
Dependent Variable: License out	Early Development		Late Development	
1(Same-class expiring patent)	0.113		−0.629*	
	(0.225)		(0.370)	
# Same-class expiring patent		0.009		−0.506*
		(0.148)		(0.290)
Ln(Therapeutic experience)	−0.435***	−0.429***	−0.12	−0.113
	(0.075)	(0.075)	(0.140)	(0.141)
Ln(Total experience)	−0.008	−0.01	−0.096	−0.103
	(0.089)	(0.089)	(0.146)	(0.146)
Scope	0.365	0.353	0.18	0.17
	(0.326)	(0.325)	(0.519)	(0.519)
Propensity to license out	7.347***	7.353***	6.633***	6.622***
	(0.220)	(0.220)	(0.323)	(0.322)
Small to medium sized firm	−0.643***	−0.657***	−0.616**	−0.620**
	(0.200)	(0.201)	(0.297)	(0.296)
Constant	−7.574***	−7.568***	−6.201***	−6.163***
	(0.877)	(0.878)	(1.002)	(1.002)
N	45731	45731	6695	6695

Note: The unit of observation is compound-year. The dependent variable is whether a compound is licensed out for development in a given year. All specifications include therapeutic class and year indicators.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Robust standard errors (in parentheses) are clustered on the compound level.

To see whether expiring patents have the same effect on an originator's licensing decision when the expiring patents are in a different therapeutic class than the compound of interest, I run a set of logit models similar to those in Table 2.6. The dependent variable is still an indicator of whether a compound is licensed out for development. However, the key explanatory variable is now either an indicator or a count of different-class expiring patents.

Table 2.7 presents the estimation results. For a firm's licensing decision regarding early stage compounds, column (1) shows that the effect of having at least one different-

Table 2.7: Different Therapeutic Class, Expiring Patents and Licensing Decision

Logit Regressions	(1)	(2)	(3)	(4)
Dependent Variable: License out	Early Development		Late Development	
1(Different-class expiring patent)	0.034		−0.327	
	(0.205)		(0.303)	
# Different-class expiring patent		0.095**		−0.068
		(0.042)		(0.07)
Ln(Therapeutic experience)	−0.427***	−0.413***	−0.188	−0.181
	(0.074)	(0.075)	(0.138)	(0.137)
Ln(Total experience)	−0.014	−0.064	−0.036	−0.04
	(0.092)	(0.093)	(0.153)	(0.154)
Scope	0.348	0.285	0.257	0.281
	(0.327)	(0.330)	(0.515)	(0.514)
Propensity to license out	7.353***	7.363***	6.606***	6.622***
	(0.221)	(0.220)	(0.322)	(0.323)
Small to medium sized firm	−0.649***	−0.568***	−0.613**	−0.607**
	(0.210)	(0.211)	(0.297)	(0.293)
Constant	−7.559***	−7.494***	−6.382***	−6.398***
	(0.879)	(0.896)	(0.995)	(0.989)
N	45731	45731	6695	6695

Note: The unit of observation is compound-year. The dependent variable is whether a compound is licensed out for development in a given year. All specifications include therapeutic class and year indicators.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Robust standard errors (in parentheses) are clustered on the compound level.

class expiring patent is positive but statistically insignificant. Column (2) shows that when the count of different-class expiring patents is used as the key explanatory variable, the effect is positive and significant at the 5% level. One possible explanation is that firms are resource constrained – as the number of expiring patents in other therapeutic classes increases, firms are more likely to develop compounds in those classes internally; with limited firm resources, this would in turn mean a higher probability of licensing out the compound of interest.[11] For the licensing decisions concerning late

[11] In Table A2.3 of the Appendix, I test the constrained resources explanation by including an additional covariate to column (2) of Table 2.7: the percentage of compounds that are developed internally and from a different therapeutic class than the compound of interest. As expected, the estimated coefficient for

stage compounds, columns (3) and (4) show that the effect of expiring patents in other therapeutic classes is negative but not statistically significant.

Overall, the findings in Tables 2.6 and 2.7 support hypotheses (2) and (2b). The probability of licensing out a late stage compound is lower when the originator has expiring patents in the same therapeutic class as the compound of interest. However, this effect is weaker when the expiring patents belong to a different therapeutic class. These results are consistent with a firm's incentive to introduce a replacement drug in time to prevent losing customers to competing firms.

2.5 Conclusion

This paper performs an empirical analysis of originating firms' behavior pending patent expiration – specifically, their timing and licensing decisions in new compound development. I argue that an originator has an incentive to control the timing of new patented drug introductions so as to maintain its customer base. Hence, the originator plans its pipeline progress such that at least one compound is well along in development as a potential replacement when the originator's patent for an existing drug is about to expire. Also, when faced with patent expiration of its existing drugs, an originator is less willing to license out late stage compounds in the same therapeutic class. Holding onto the timing control of these compounds is important because: (1) they stand the most chance of being introduced in time for the customers to switch to, and (2) if left to a licensee, there may be a delayed introduction as the licensee's incentive may not be aligned with the originator's.

this covariate is positive, meaning when a firm devotes more internal resources to compounds in other therapeutic classes, the firm is more likely to license out the compound of interest. The covariate, however, is imprecisely estimated, likely due to the fact that it is moderately correlated with the count of expiring patents in therapeutic classes different from the compound of interest's (correlation of 0.33).

Using detailed drug development data between 1989 and 2004, I find empirical evidence consistent with the above arguments. Controlling for heterogeneity in firm capability, an originator is more likely to achieve a higher level of development for its pipeline compounds in a therapeutic class when the originator has marketed drugs with expiring patents in the same class. The effect is less pronounced when the expiring patents belong to a different class. Furthermore, the originator's expiring patents reduce its likelihood of licensing out a late stage compound within the same therapeutic class, but the effect is weaker when the two pertain to different classes. These results all suggest that when firms face patent expiration of their own drugs, maintaining a customer base through timely introduction of new drugs as potential replacements is an important consideration in firms' pipeline planning and licensing decisions.

2.6 Appendix

Table A2.1: Any Therapeutic Class, Expiring Patents and Pipeline Progress

Ordered Logistic Regressions Dependent Variable: Furthest Along	(1)	(2)
1(Any-class expiring patent)	0.190**	
	(0.091)	
# Any-class expiring patent		0.009
		(0.022)
Ln(Therapeutic experience)	1.248***	1.248***
	(0.054)	(0.054)
Ln(Total experience)	−0.281***	−0.266***
	(0.053)	(0.053)
Scope	−0.119	−0.110
	(0.178)	(0.178)
Small to medium sized firm	0.236*	0.190
	(0.143)	(0.144)
Cutoff point 1	−3.023***	−3.049***
	(0.364)	(0.366)
Cutoff point 2	−2.537***	−2.564***
	(0.364)	(0.365)
Cutoff point 3	−1.496***	−1.524***
	(0.363)	(0.364)
Cutoff point 4	0.034	0.005
	(0.362)	(0.364)
N	19042	19042

Note: The unit of observation is firm-therapeutic class-year. The dependent variable is the most advanced development stage a firm has achieved for its pipeline compounds in a therapeutic class for a given year. All specifications include therapeutic class and year indicators.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Robust standard errors (in parentheses) are clustered on the firm therapeutic class level.

Table A2.2: Any Therapeutic Class, Expiring Patents and Licensing Decision

Logit Regressions	(1)	(2)	(3)	(4)
Dependent Variable: License out	Early Development		Late Development	
1(Any-class expiring patent)	0.085		−0.346	
	(0.186)		(0.286)	
# Any-class expiring patent		0.080**		−0.087
		(0.039)		(0.066)
Ln(Therapeutic experience)	−0.427***	−0.424***	−0.172	−0.173
	(0.074)	(0.075)	(0.138)	(0.138)
Ln(Total experience)	−0.018	−0.053	−0.044	−0.026
	(0.091)	(0.092)	(0.150)	(0.152)
Scope	0.351	0.312	0.224	0.282
	(0.325)	(0.328)	(0.515)	(0.513)
Propensity to license out	7.352***	7.357***	6.608***	6.633***
	(0.221)	(0.220)	(0.321)	(0.323)
Small to medium sized firm	−0.631***	−0.564***	−0.621**	−0.630**
	(0.206)	(0.211)	(0.298)	(0.291)
Constant	−8.580***	−8.616***	−6.846***	−6.870***
	(0.848)	(0.861)	(0.906)	(0.900)
N	45731	45731	6695	6695

Note: The unit of observation is compound-year. The dependent variable is whether a compound is licensed out for development in a given year. All specifications include therapeutic class and year indicators.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Robust standard errors (in parentheses) are clustered on the compound level.

Table A2.3: Test for Constrained Resources Explanation

Logit Regressions	(1)
Dependent Variable: License out	Early Development
# Different-class expiring patent	0.094**
	(0.042)
% Compounds from different class and developed internally	0.150
	(0.438)
Ln(Therapeutic experience)	−0.400***
	(0.080)
Ln(Total experience)	−0.077
	(0.098)
Scope	0.358
	(0.395)
Propensity to license out	7.371***
	(0.222)
Small to medium sized firm	−0.552**
	(0.217)
Constant	−8.680***
	(0.893)
N	45731

Note: The unit of observation is compound-year. The dependent variable is whether a compound is licensed out for development in a given year. All specifications include therapeutic class and year indicators.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Robust standard errors (in parentheses) are clustered on the compound level.

CHAPTER 3:

DOES HEALTH INSURANCE ENCOURAGE SMOKING? EVIDENCE FROM THE RAND HEALTH INSURANCE EXPERIMENT

3.1 Introduction

This paper evaluates the moral hazard effect of health insurance on smoking behavior. More precisely it estimates the elasticity of cigarette consumption to the expected benefit of health insurance for initial non-smokers and smokers, as well as for older and younger individuals. Insurance policies allow individuals to transfer their risk of economic loss resulting from adverse health events into an insurance pool. Basic economic theory suggests that when individuals do not fully bear the cost of getting sick, they may be more willing to take on risky health behavior.

I use data from the RAND Health Insurance Experiment (HIE) to ask: (a) how do coinsurance rates (out-of-pocket percentage) affect the probability of transitioning into or out of smoking, and (b) how is the intensity of cigarette consumption affected? In the HIE, insurance plans did not have a premium. Participants were randomly assigned to either 3-year or 5-year contracts that differed primarily by coinsurance rates and maximum dollar expenditure. Participants were given health questionnaires both at the beginning and at the end of the experiment, and asked to report information on current smoking status and amount. Because enrollment into plans was randomized, my estimation for the effect of coinsurance on smoking avoids the typical selection problems in health insurance contracts.

From the empirical results, I find that individuals are more likely to initiate smoking only when their insurance plans change from paying almost nothing (95% coinsurance) to paying everything (0% coinsurance). On the other hand, smokers have a higher prob-

ability of continuing smoking as long as their insurance plans pay some amount of medical expense as opposed to not paying anything. This effect is more significant for the older individuals than for the younger ones. Additionally, for the initial non-smokers who start smoking, a more generous insurance plan makes them smoke more. Such an effect is again stronger for older individuals than for younger ones. These results yield two important implications. First, the benefit from a more generous insurance plan has to be large for non-smokers to start smoking, while smaller benefits suffice for smokers to continue smoking. Second, older individuals are more prone to moral hazard in the HIE, possibly due to their higher expectation of benefiting from a more generous plan by utilizing medical services during the experiment.

There are two goals for this paper. First, by using data from the HIE, the paper exploits the exogenous variation in coinsurance rates in the randomized experiment, and provides a clean identification strategy on how individuals' incentives in smoking respond to changes in the generosity of insurance plans. Second, the paper separately analyzes the moral hazard effect of health insurance on smoking for baseline non-smokers and smokers, as well as for younger and older age groups. The findings suggest the importance of considering potential heterogeneous treatment effects across population subgroups.

This study relates to two strands of empirical literature. First, it contributes to empirical work on health insurance and smoking behavior. Recent papers on the topic have mostly used observational data from Medicare. Dave and Kaestner (2009) use a difference-in-difference model to compare the change in smoking behavior pre- and post- age 65 between those who were uninsured before age 65and those who were insured. They find that obtaining Medicare at age 65 increases smoking among the elderly. In contrast, de Preux (2011) finds no clear effect of receiving Medicare or anticipating

receiving it on smoking behavior. These quasi-experimental studies are applicable only to individuals around 65 years old, and have found mixed results concerning the impact of health insurance on smoking.

Second, the paper connects to empirical studies of the HIE. Over the years, data from the HIE have been used extensively to study how health insurance affects demand for medical care, medical expenditure and health outcomes. In particular, Manning et al. (1987) use the experimental results to show that cost-sharing by patients reduces the demand for medical care. Newhouse (1993) finds that more generous insurance plans do not substantially improve health outcomes. A more recent analysis of the HIE data by Aron-Dine et al. (2013) considers the challenges of measuring the elasticity of medical spending with respect to prices in non-linear health insurance plans. The authors cannot reject the null hypothesis that cost-sharing reduces medical expenditure. In addition to the above topics, the HIE data have also been used to examine whether lower coinsurance is associated with obesity (Bhattacharya et al., 2011). To the best of my knowledge, no study has used this dataset to look at the smoking-related incentives created by health insurance plans, and how the incentives might differ depending on initial smoking status and age.

The rest of the paper is organized as follows. In the next section, I describe the design of the HIE in order to show how the experiment is useful for studying insurance-induced changes in smoking behavior. Section 3.3 discusses the possible treatment effects of varying levels of coinsurance on smoking. A simple theoretical model for the moral hazard effect is also developed. Section 3.4 explains the data and provides summary statistics. In Sections 3.5 and 3.6, I use empirical models to explore the impact of coinsurance rates on changes in smoking status (extensive margin) and smoking intensity (intensive margin). Both of these sections include an explanation of methods, an

analysis of results, and a robustness check. Section 3.7 concludes and suggests future research directions.

3.2 Design of the HIE

The HIE was an experiment conducted from 1974 to 1982 across six different sites in the U.S. in an attempt to study how different cost-sharing schemes affect "medical service utilization and individual health outcomes" (Newhouse, 1993).[1] The length of the experiment was either 3 or 5 years. Those who were qualified for Medicare were not counted as eligible participants, but recruitment within six experimental sites was otherwise universal. In the selection process, low-income individuals were oversampled, since this population group was of major interest to the HIE study. The HIE matched families to insurance plans so that "groups of people... were similar to each other with respect to important [demographic] characteristics" (Ibid).

The participants were either randomly assigned to a fee-for-service (FFS) plan or an HMO plan. The FFS insurance plans varied along three dimensions: the coinsurance rate, the maximum dollar expenditure (MDE) per year, and the deductible. The coinsurance rate is the percentage of the medical bill that the patients have to pay. There were four coinsurance categories: 0%, 25%, 50% and 95%. The annual MDE had three categories: 5%, 10% and 15% of family income, with a cap of $1000 in each category. After the cap was reached, the plan paid 100%. The HIE also included a deductible plan, where the coinsurance rate was 95% for outpatient services until the deductible was met. The deductible was $150 per person or $450 per family, after which the plan paid 100%. Throughout the experiment, participants could only use the HIE insurance

[1]The six selected sites are Seattle, Wash.; Dayton, Ohio; Charleston, S.C.; Fitchburg-Leominster, Mass.; Franklin County, Mass.; Georgetown County, S.C.

plans to which they were assigned. They completed a health questionnaire at the beginning and end of the experiment, including questions on their current smoking status and daily amount of cigarette consumption. Thus, even though the experiment was originally designed to investigate how coinsurance affects the demand for medical care and health outcomes, it also contains useful information for studying the moral hazard effect of insurance on smoking.

The HIE entailed one important caveat: to ensure that participants were not financially worse off with the HIE insurance plan than with their previous insurance plans, they were offered an appropriate participation incentive, the amount of which was closely related to the MDE. Participants' maximum loss relative to their non-experimental plan occurred when they reached the MDE assigned to them in the HIE. To appropriately compensate them for this loss, their incentive was set equal to the maximum possible difference between the experimental plan, and the individual's previous plan when the latter was more generous. Overall, ceteris paribus, a higher assigned MDE usually resulted in a larger cash payment. Incentives also differed according to assigned coinsurance rates: higher rates generally led to a larger dollar transfer. In general, the less generous the experimental plan, the larger the cash incentive participants received.

3.3 Possible Treatment Effects of Different Coinsurance Rates on Smoking

In the HIE, there are two possible ways that different levels of coinsurance can affect people's smoking behavior. First, more generous coverage reduces the cost of getting sick, thus lowering the cost of smoking, and encouraging individuals to smoke more.

This is what is referred to as the moral hazard effect of health insurance on smoking. Second, because participation incentives differ substantially across coinsurance groups, those with higher coinsurance rates may have been offered larger incentives. Consequently, there may be an income effect in the sense that smokers in higher coinsurance groups may respond to the additional cash incentive by changing their health behavior. In order to isolate the moral hazard effect of health insurance on smoking, a clear understanding of the two types of treatment effects is crucial.

3.3.1 A Model of Moral Hazard in Smoking

Consider a simple theoretical model, where smokers maximize their net utility from cigarette consumption by balancing the pleasure of smoking against the displeasure of getting sick. The smokers' enjoyment from cigarettes is characterized by the function $U(S)$, with S denoting the current amount of cigarette consumption. Assume $U(\cdot)$ is twice continuously differentiable, $U'(\cdot) > 0$ and $U''(\cdot) \leq 0$. The probability that a negative health event (N) occurs due to smoking is conditioned on a person's age (A), current smoking amount (S), and smoking history (S_0, initial smoking status in the HIE). Assume that the probability is increasing in age, current smoking amount and smoking history. Furthermore, the rate of increase in the probability of getting sick from smoking rises with age and smoking history. In other words, the cross partials of the conditional probability function with respect to current smoking amount and age, as well as current smoking amount and smoking history are assumed to be positive. In the case of a negative health event related to smoking, a medical expense of M is incurred, and individuals facing a coinsurance rate of α would pay αM out of pocket.[2] Consumers

[2]Because the health insurance plans in the HIE vary primarily on coinsurance rates, I abstract away from deductibles, and other aspects of a health insurance contract for simplicity, and focus only on the coinsurance rates.

choose their optimal smoking amount, and the optimization problem is formalized as the following:

$$\max_{S} U(S) - P(N|A, S, S_0)\alpha M.$$

The optimal smoking amount satisfies the first order condition,

$$U'(S^*) - P_S(N|A, S^*, S_0)\alpha M = (\leq)\ 0 \ \text{ if } S^* > (=)\ 0.$$

Intuitively, the optimal smoking amount equates the marginal utility from smoking to the marginal disutility of getting sick. Notice that when the coinsurance rate α is very high, the cost of a smoking-related illness may be so high that individuals would choose not to smoke altogether. As the coinsurance rate decreases and the insurance plan becomes more generous, the optimal amount of cigarette consumption may become positive.

Putting into the context of the HIE, the coinsurance rate has to be low enough for non-smokers ($S^* = 0$) to start smoking. For smokers ($S^* > 0$), the effect of a change in coinsurance rates on smoking amount is obtained by taking the derivative of the first order condition with respect to α:

$$U''(S^*)\frac{\partial S^*}{\partial \alpha} - [P_S(N|A, S^*, S_0)M + \alpha P_{SS}(N|A, S^*, S_0)\frac{\partial S^*}{\partial \alpha}M] = 0$$

$$\frac{\partial S^*}{\partial \alpha} = \frac{P_S(N|A, S^*, S_0)M}{SOC(S^*)} < 0$$

By assumption, $P_S(N|A, S^*, S_0)$ is positive and the second order condition of S^* is negative, hence the partial derivative of S^* with respect to α is negative. A reduction in coinsurance rates would encourage cigarette consumption among smokers.

3.3.2 More on Moral Hazard, Health Insurance and Smoking

As is evident from the model, more generous insurance plans could induce moral hazard in smoking on both the extensive and intensive margins. On the extensive margin, moral

hazard exists if the generosity of insurance plans increases the probability of smoking initiation and decreases the probability of smoking cessation. On the intensive margin, moral hazard of health insurance could result in a larger number of cigarettes consumed.

Furthermore, moral hazard could be heterogeneous across the population – specifically between initial non-smokers and smokers. While many studies have analyzed the two groups separately when examining how changes in exogenous factors affect smoking behavior (Ellickson et al., 2001; DeCicca et al., 2008; Kenkel et al., 2009), it is conceivable that a reduction in coinsurance rate would also have distinct impacts on individuals' decisions to transition into or out of smoking. Likewise, initial non-smokers and smokers could respond very differently in terms of changes in the amount of cigarettes consumed when offered a more generous insurance plan. For these reasons, I separate my empirical analysis for initial non-smokers and smokers on both the extensive and intensive margins.

Another important aspect when studying health insurance and smoking behavior is that the moral hazard effect may also vary with age as smoking-related illnesses often occur later in life. Figure A3.1 in Appendix A displays a plot of the age-adjusted lung cancer incidence rates between the mid-1970s and the mid-1980s when the HIE was conducted (National Cancer Institute's Surveillance, Epidemiology, and End Results Program [SEER], 2014). It illustrates that the incidence rate begins to increase in the age bin for 20-49 year olds, continues to rise for 50-64 year olds, and peaks in the 65-74 age bin. While the division of bins is rather coarse, more recent data suggests that the rate rises sharply from around age 40 (see Figure A3.2 in Appendix A (SEER, 2014)). Consistent with the model developed in the previous subsection, the probability of falling ill from smoking increases as an individual ages.

Because the HIE was only 3 or 5 years, a temporary decrease in coinsurance rates

would have the biggest impact on those who had the highest expectation of receiving medical care. This means that older people were more likely to benefit from the low coinsurance rates in the HIE than younger people, and hence more likely to exhibit moral hazard. The intuition is straightforward: if one does not expect to benefit from a low coinsurance rate, one would not significantly increase smoking – in the event that a disease from smoking is diagnosed only *after the experiment*, one would have to bear the large medical expense under the non-HIE plan instead. Note that smoking-induced illnesses can be expensive to treat, with the first-year treatment cost for lung cancer averaging as high as $4380 or equivalently about 39% of a person's annual income in the 1980s (Patterson, 1989). Hence, low coinsurance rates in the HIE may considerably reduce the financial burden for the older aged group – the group that is more likely to experience a health complication from smoking compared to its younger counterpart. This paper captures the interaction between age and moral hazard by comparing changes in smoking behavior between older and younger HIE participants.

3.3.3 Income Effect from Direct Payment to Participants

Besides moral hazard, variations in coinsurance rates may also generate an income effect on smoking behavior due to differences in the HIE participation incentives. There are two ways that income can affect smoking: indirect, and direct. The indirect effect occurs due to the fact that health status is a normal good; people with more participation incentives (i.e., more income) demand better health (Pauly et al., 2011), and thus smoke less. In the direct effect, depending on whether people view smoking as a normal or inferior good, more income can either increase people's cigarette consumption or decrease it.

Existing evidence indicates that the income elasticity of smoking differs depending on sample period and population. While Cheng and Kenkel (2010) find that, for high income countries like the U.S., smoking is considered an inferior good; Kenkel et al. (2014) find that for low-income individuals smoking is still a normal good. According to a meta-analysis by Gallet and List (2003), the income elasticity of smoking ranges from -0.80 to 3.03 in 375 published articles. In any case, income appears to be an important factor in determining people's smoking behavior, so controlling for the income transfer (participation incentives) is essential when studying the moral hazard effect of coinsurance on smoking.

3.4 Data

I use the HIE dataset for two major reasons. First, despite having taken place over thirty years ago, HIE remains the largest and most important health insurance study in U.S. history.[3] Individuals were randomly assigned to different coinsurance groups within the six experimental sites, thus avoiding adverse selection (people with high claim rates buy more insurance) or advantageous selection (people who are more risk-averse buy more insurance) (Einav & Finkelstein, 2011).While most health insurance data contain confounding effects from both insurance selection and moral hazard, the random assignment of the experiment enables relatively clean identification when looking at the effect of insurance on smoking behavior. Second, although the dataset is somewhat old (1973-1982), this limitation is hardly a disadvantage for the purpose of this study, as it seems reasonable to assume that moral hazard in health insurance is similar over time.

Following Bhattacharya et al. (2011), I drop the HMO enrollees due to a lack of

[3] http://www.rand.org/health/projects/hie.html

information about the generosity of the HMO plan as compared to the Fee-For-Service plans. Observations with missing data for the demographic controls are also deleted. The paper focuses on adults who are 18 years and older.

3.4.1 Dependent variable and covariates

I have two measures for my dependent variable that capture individuals' smoking behavior on the extensive and intensive margins, respectively. My first measure is the change in smoking status. Participants were asked whether they "smoke cigarettes now" both at the beginning and end of the experiment. The second measure is the change in the number of cigarettes consumed per day. This variable is constructed based on the question "how many packs of cigarettes do you smoke per day?" There are four categories to choose from: less than one pack/day, about one pack/day, about two packs/day, and more than two packs/day.

My regression specifications include two sets of controls. The first set consists of indicators for the six experimental sites and indicators for the years in which participants exited the experiment. This set of covariates is used to control for any site or year effects. The second set is individual controls: demographic variables,[4] length of time in the experiment, and participation incentives offered at enrollment. Participation incentives are included to control for income effects and to minimize the potential bias from people choosing to take part in the experiment or not.

[4]Demographic variables include age, years of school completed, family size, family income in the year preceding plan enrollment, race, marital status, sex, and self-reported health status.

3.4.2 Treatment Indicators

My primary explanatory variables all measure the extent of insurance coverage. There is an indicator variable for each of the four coinsurance rates: 0%, 25%, 50%, and 95%. In addition, I create an indicator variable for whether a plan is a deductible or not. The MDE is also used to characterize the different insurance plans: except for free care, under each level of coinsurance rate, the insurance plans are further divided into three levels, having either an MDE of 5%, 10%, or 15% of family income. In all cases, if the percentage of family income exceeds $1000, the MDE caps its maximum at $1000.

3.4.3 Summary Statistics

Table 3.1 presents summary statistics for the demographic characteristics of the participants as well as the participation incentives offered to them in each coinsurance group. I use F-statistics and Chi-squared statistics to test for differences in group means for continuous and categorical variables. Except for family income, all other demographic controls (age, education, family size, marital status, sex, race, and self-reported health status) are balanced across the different insurance plans. Family income is unevenly distributed because in order to make best use of the experimental budget, the HIE followed a stratified random assignment (instead of a simple random assignment) according to "a finite selection model" (Morris, 1979; Newhouse, 1993). The F-test for family income rejects the null that the means are the same across the insurance plans at the 1% level. Finally, as expected from the specific design of the HIE (see details in Section 3.2), participation incentives are relatively different among plans. The amount of participation incentives offered ranges from $170 in the free care plan to $670 in the 95% coinsurance rate plan. Its p-value from the means test is close to 0, so I reject the null that the group

Table 3.1: Summary Statistics

	All	Free	25%	50%	95%	Deduct -ible	Means test P-value
Age	36.83	37.48	36.26	37.38	36.83	36.14	0.17
	(11.46)	(11.67)	(11.04)	(11.58)	(11.61)	(11.30)	
Education	12.31	12.15	12.40	12.43	12.41	12.32	0.41
	(2.98)	(3.16)	(2.90)	(2.98)	(2.85)	(2.88)	
Family Size	3.23	3.31	3.28	3.22	3.22	3.07	0.11
	(1.65)	(1.70)	(1.68)	(1.55)	(1.65)	(1.53)	
Marital Status	0.79	0.80	0.78	0.81	0.78	0.78	0.68
Male	0.45	0.46	0.46	0.45	0.43	0.47	0.65
Race							
White	0.88	0.88	0.88	0.92	0.86	0.87	0.37
Black	0.11	0.12	0.11	0.07	0.12	0.12	0.29
Others	0.01	0.01	0.02	0.01	0.01	0.01	0.53
Self-Reported Health Status Before the Experiment							
Excellent	0.45	0.44	0.44	0.49	0.45	0.45	0.84
Good	0.43	0.42	0.44	0.40	0.45	0.43	0.76
Fair	0.09	0.10	0.10	0.09	0.08	0.09	0.87
Poor	0.02	0.03	0.01	0.02	0.02	0.02	0.10
Missing	0.01	0.01	0.01	0.01	0.01	0.01	0.98
Family income (k)	11.32	11.09	11.47	12.65	11.61	10.76	0.00
	(5.83)	(5.76)	(5.89)	(5.99)	(5.96)	(5.61)	
Participation	0.45	0.17	0.68	0.80	0.67	0.35	0.00
Incentive (k)	(0.37)	(0.24)	(0.30)	(0.33)	(0.37)	(0.21)	
Observations	2611	862	523	180	529	517	

Note: Standard deviations in parentheses. Sample is limited to adults (age≥18).

means for participation incentives are equal.

In Table 3.2, I display the group means of smoking status and the number of cigarettes consumed per day. At the beginning of the experiment, the insurance plans are fairly balanced in the percentage of population groups who smoke. This shows that there is no systematic sorting of smokers into any particular coinsurance rate. By the end of the experiment, the percentage who smoke continues to be quite similar across insurance plans. Furthermore, there is no statistically significant difference in the per-

Table 3.2: Differences in Group Means for Smoking Status and Intensity

	(1) All	(2) Free	(3) 25%	(4) 50%	(5) 95%	(6) Deductibles
Smoker at entry	0.403	0.407	0.400	0.383	0.401	0.410
Smoker at exit	0.367	0.363	0.375	0.394	0.344	0.377
Become a smoker	0.034	0.033	0.040	0.044	0.027	0.033
Quit smoking	0.071	0.077	0.065	0.033**	0.083	0.066
Cigarette consumption	8.951	8.863	9.350	9.000	8.733	8.897
At entry (# per day)	(0.261)	(0.451)	(0.619)	(1.012)	(0.563)	(0.573)
Cigarette consumption	8.410	8.110	9.063	9.167	8.072	8.337
At exit (# per day)	(0.264)	(0.445)	(0.635)	(1.021)	(0.580)	(0.575)
Change in cigarette	−0.540	−0.754	−0.287	0.167	−0.662	−0.561
(# per day)	(0.173)	(0.309)	(0.381)	(0.647)	(0.392)	(0.374)
Observations	2611	862	523	180	529	517

Note: Standard errors in parenthesis. Difference in means relative to the 95% coinsurance plan are examined using a chi-square test for the categorical variables and a t-test for continuous variables.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

centage who start smoking between the 95% plan and the 0%, 25%, or 50% plans. For the percentage who quit smoking, the Chi-2 tests cannot reject the null that the group means for the 0% and the 25% plans have different means than the 95% plan. The only significant difference is between the 50% plan and the 95% plan, with the former having a mean of 3.3% and the latter 8.3% of population groups who quit smoking. In terms of smoking intensity, there is no significant difference compared to 95% plan in the number of daily cigarettes consumed at entry, at exit, or in the change in cigarette consumption during the experiment. A simple difference in means comparison of smoking behavior does not provide much evidence of moral hazard from health insurance.

Variations in the amount smoked before and after the experiment are small in all groups, as shown in Appendix B, where a detailed transition matrix for the level of smoking intensity is presented. No one changed from smoking less than one pack of cigarettes per day at the beginning of the experiment to smoking more than two packs per day at the end. Conversely, very few individuals went from smoking more than

two packs a day to not smoking at all. The transition matrices appear to be quite similar among the different coinsurance rate groups. However, it is important to keep in mind that all these findings are unconditional. In other words, they are suggestive, and not conclusive, regarding the existence of moral hazard because other factors such as participation incentives and demographic characteristics may be driving the results.

3.5 Extensive Margin

To control for factors that may bias the estimate for the moral hazard effect of health insurance on smoking, I use a probit model to examine whether lower coinsurance rates are associated with higher rates of smoking participation. In other words, do initial non-smokers have a higher probability of smoking initiation and do smokers have a higher probability of smoking continuation when offered more generous insurance plans?

3.5.1 Method

The probit model for the participation equation takes the following form:

$$z_i^* = \alpha + x_i'\beta + c_i'\gamma + \epsilon,\ \epsilon \sim N(0,1) \tag{3.1}$$

$$z_i = \begin{cases} 1 & \text{if } z_i^* > 0 \\ 0 & \text{otherwise,} \end{cases}$$

where z_i is a variable, indicating whether an individual is a smoker or not at the end of the experiment. It takes on the value 1 when the latent variable z_i^* is greater than 0, and 0 otherwise. x_i is a vector of explanatory variables, containing indicators for the different plan types; and c_i is a vector of covariates described in detail in Section 3.4.1.

The error term, ϵ_i , is distributed standard normal. $\Phi(\cdot)$ is the cumulative distribution function of the standard normal distribution. I separate the individuals into non-smokers and smokers at baseline, and use equation (3.1) to estimate the initiation model and the continuation model respectively. The 95% coinsurance group is omitted in the probit regression for comparison.

The probability that an individual smokes at the end of the experiment is expressed as the following:

$$\begin{aligned}\Pr(z_i = 1 \mid [1, x_i', c_i']') &= \Pr(\epsilon > -(\alpha + x_i'\beta + c_i'\gamma) \mid [1, x_i', c_i']') \\ &= \Phi(\alpha + x_i'\beta + c_i'\gamma).\end{aligned}$$

If moral hazard exists, I expect the probit coefficients for the 0%, 25%, and 50% coinsurance rates to be positive and statistically significant for both the non-smokers and smokers at baseline. Moreover, the magnitude of the coefficients would increase as the coinsurance rates get lower. For non-smokers, this means that the probability of smoking initiation rises with more generous insurance plans. For smokers, it means an increase in the probability of smoking continuation.

Considering the possible interaction between age and moral hazard, I further separate non-smokers and smokers into young and old age groups. If age plays an important role in determining how people's smoking behavior changes in response to a more generous insurance plan in the HIE, then the probit coefficients for the coinsurance rates would be significantly different between the younger and older groups.

3.5.2 Results

In general, the probability of smoking *initiation* increases as the coinsurance rates decrease, but the effect is only significant for the free care plan. The probability of smoking *continuation* is higher for participants with a coinsurance rate below 95% than those with a 95% rate. Furthermore, older smokers at baseline are more likely to continue smoking when assigned to a more generous experimental plan than younger smokers at baseline.

Table 3.3(a): Coefficient Estimates of the Extensive Margin

Probit coefficient	(1)	(2)	(3)	(4)
Smoking status at exit	Initiation (non-smokers at entry)		Continuation (smokers at entry)	
<95% Coinsurance Rate		0.256*		0.361**
		(0.153)		(0.148)
0% Coinsurance Rate	0.486*		0.299	
	(0.266)		(0.251)	
25% Coinsurance Rate	0.254		0.309*	
	(0.170)		(0.166)	
50% Coinsurance Rate	0.151		0.622**	
	(0.223)		(0.255)	
Individual Deductible	0.258	−0.104	0.374*	0.047
	(0.217)	(0.145)	(0.196)	(0.135)
Maximum $ Expenditure (k)	0.869**	0.580**	0.474	0.556**
	(0.358)	(0.254)	(0.384)	(0.277)
Participation Incentive (k)	−0.558**	−0.519*	−0.492*	−0.502*
	(0.264)	(0.266)	(0.291)	(0.285)
N	1549	1549	1044	1044
Chi-stat	1.21		1.411	
P-value	0.546		0.494	

Note: All columns include demographic controls (age, education, family income, family size, race, marital status, sex, and self-reported health status before the experiment), length in the experiment, site effects and year effects.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) clustered by family.

Table 3.3(a) reports the regression results from the participation model for non-

smokers and smokers, respectively. Column (1) displays that the probit coefficients for non-smokers at baseline are 0.49, 0.25, and 0.15 for the 0%, 25%, and 50% coinsurance rates.[5] The coefficients' positive signs and their ordering of magnitude are consistent with the moral hazard theory: non-smokers are more likely to start smoking as the coinsurance rate declines. While coefficients for the 25% and 50% groups are statistically insignificant, the 0% group has a significant coefficient at the 10% level. This suggests that moral hazard for non-smokers is most precisely estimated when individuals go from paying almost everything for their medical bill (95% coinsurance rate) to paying nothing at all (0% coinsurance rate). Moreover, a Chi-squared test for the estimates in front of the 0%, 25%, and 50% plans to be equal cannot be rejected at the 10% level. In column (2), the three levels of insurance plans are combined into one – whether the coinsurance rate is below 95% or not. The estimated probit coefficient is positive and significant, implying that non-smokers with coinsurance rates that are less than 95% are more likely to start smoking than those with the 95% rate.

For smokers at baseline, the estimated coinsurance coefficients are all positive in column (3) of Table 3.3(a). A Chi-2 test cannot reject the null hypothesis that the probit coefficients for the 0%, 25% and 50% plans are the same at the 10% level. Still, the estimates for the 25% and 50% plans are statistically significant, but the estimate for the 0% plan is not. These results show that moral hazard for smokers is estimated with precision when their insurance plans change from reimbursing almost nothing to reimbursing at least some amount for their medical expenses. Finally, column (4) indicates that smokers with coinsurance rates lower than 95% have a higher probability of smoking continuation than those with the 95% rate (the probit estimate is 0.361 and significant at 5% level).

Table 3.3(b) presents the average marginal effects for both non-smokers and smokers

[5] As mentioned earlier in Section 3.5.1, the 95% coinsurance rate is the comparison group.

Table 3.3(b): Average Marginal Effects on the Extensive Margin

Probability of being a smoker at exit	(1)	(2)	(3)	(4)
	Initiation (non-smokers at entry)		Continuation (smokers at entry)	
< 95% Coinsurance Rate		0.025*		0.095**
		(0.013)		(0.042)
0% Coinsurance Rate	0.047*		0.080	
	(0.025)		(0.069)	
25% Coinsurance Rate	0.028		0.078*	
	(0.018)		(0.042)	
50% Coinsurance Rate	0.016		0.126***	
	(0.025)		(0.044)	

Note: Average marginal effects calculated based on probit estimates from Table 3.3(a), dydx for binary variables is the discrete change between 0 and 1. The comparison group is the 95% coinsurance plan.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) clusteredy by family.

on the extensive margin. Quantitatively, *ceteris paribus*, baseline non-smokers in the 0% coinsurance plan have a 4.7 percentage point higher probability of starting smoking than those in the 95% group (column (1)). Given the baseline initiation rate of 3.4%, 4.7 percentage points translates into a 138% increase in the probability of starting smoking. While this substantial increase may be due to a large change in coinsurance rates from 95% to 0%, it also points to the possibility of relapse – baseline non-smokers who smoked at one point in their life may be particularly tempted to begin smoking again under plans with lower coinsurance rates. Column (2) shows that in a more general comparison, non-smokers with coinsurance rates less than 95% have a 2.5 percentage point higher probability of initiation (or a 74% increase from the baseline initiation rate) than those with the 95% coinsurance rate.

For baseline smokers, the effect is largest in the case of the 50% plan – a reduction of coinsurance rates from 95% to 50% increases the probability of smoking continuation by 12.6 percentage points (column (3)). This is equivalent to a 15% increase from

Table 3.4: The Effect of Coinsurance Rates on Smoking Status by Age

Probit Coefficients	(1)	(2)	(3)	(4)
Smoking Status at exit	Non-smoker at entry		Smoker at entry	
	Age<37	Age≥37	Age<37	Age≥37
< 95% Coinsurance Rate	0.261	0.255	0.268	0.461**
	(0.197)	(0.242)	(0.203)	(0.229)
Individual Deductible	0.040	−0.322	−0.025	0.204
	(0.180)	(0.270)	(0.168)	(0.235)
Maximum $ Expenditure (k)	0.105	1.210***	0.588	0.660*
	(0.345)	(0.421)	(0.368)	(0.398)
Participation Incentive (k)	−0.201	−1.053**	−0.729*	−0.340
	(0.371)	(0.438)	(0.386)	(0.404)
N	831	718	613	431

Note: All columns include demographic controls (age, education, family income, family size, race, marital status, sex, and self-reported health status before the experiment), length in the experiment, site effects and year effects.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) clustered by family.

the baseline continuation rate of 82% among smokers. Under broader plan categories, column (4) shows that baseline smokers in coinsurance plans lower than 95% are more likely to continue smoking by 9.5 percentage points (or equivalently, a 12% increase from the baseline continuation rate).

Next, I present the results for how insurance affects smoking on the extensive margin when non-smokers and smokers are further divided into young and old age groups. In Table 3.4, the age groups are simply divided at the mean (37 years old). Section 3.5.3 shows that the results are not sensitive to any particular age cut-off point. Due to concerns about the reduction in sample size after division by age, the coinsurance variables in the probit regressions are collapsed into a single indicator for whether a plan has a coinsurance rate below 95% or not. Based on findings from Table 3.3(a), collapsing the explanatory variables would not be a problem as people seem to be most affected in their smoking behavior when they switch from a 95% plan to other plans.

For non-smokers at baseline, the interaction between age and moral hazard on the extensive margin is small. The estimated probit coefficients for plans with coinsurance rates below 95% are close to identical for the younger and older age groups (Table 3.4 columns (1) & (2)). Unlike non-smokers, for smokers at baseline, the moral hazard effect clearly differs by age. The probit coefficient for plans with lower than 95% coinsurance rate is 0.27 for the younger half of the sample population, and is not statistically significant (Table 3.4 column (3)). However, for the older half, the estimate is 0.46, almost twice as big as the estimate for the younger half, and is significant at the 5% level (Table 3.4 column (4)). These results suggest that as coinsurance rates fall, smokers are more likely to continue smoking; and more importantly, the effect is mainly coming from older smokers rather than younger ones.

It is also worthwhile to note that the estimated probit coefficients for the participation incentives are negative across all specifications and statistically significant in most cases (Tables 3.3(a) & 3.4). The results suggest that there may be a negative income effect from direct incentive payments to participants on the extensive margin. Consequently, controlling for the incentive payments appears to be necessary to separately identify the moral hazard effect of health insurance on smoking.

3.5.3 Discussion and Robustness Checks

There are two major implications associated with how coinsurance rates affect smoking participation. First, the amount of financial incentive a plan must generate in order to produce observable moral hazard can be different depending on an individual's initial smoking status. For non-smokers at baseline, the reduction in coinsurance rates has to be large for an increase in the probability of smoking initiation to occur. However, for

smokers, only a small reduction is enough for them to have a higher probability of smoking continuation. Second, due to the design of the HIE, which only lasted for either 3 or 5 years depending on the experimental site, moral hazard can be heterogeneous across age. Especially for smokers at baseline, older smokers are more likely than younger smokers to continue smoking when offered a more generous insurance plan. One possible explanation is that older smokers have a higher probability of falling sick from smoking and in turn a higher expectation of using medical care for a smoking-related disease during the HIE. Because older participants are more likely to benefit from the HIE, their willingness to take on health risks from smoking increases.

Table 3.5: Robustness Checks for Different Age Cutoffs on the Extensive Margin

Probit coefficient	(1)	(2)	(3)	(4)
Smoking status at exit	Non-smokers at entry		Smokers at entry	
	Age<c	Age≥c	Age<c	Age≥c
c = 34				
< 95% coinsurance	0.243	0.272	0.256	0.449**
	(0.202)	(0.236)	(0.210)	(0.208)
N	701	848	530	514
c = 37				
< 95% coinsurance	0.261	0.255	0.268	0.461**
	(0.197)	(0.242)	(0.203)	(0.229)
N	831	718	613	431
c = 40				
< 95% coinsurance	0.298	0.218	0.291	0.596**
	(0.185)	(0.267)	(0.188)	(0.261)
N	950	514	683	361

Note: All regressions include demographic controls (age, education, family income, family size, race, marital status, sex, and self-reported health status before the experiment), length in the experiment, site effects and year effects. When estimating column (2) for c=40, 85 observations with race=0 (non-white) perfectly predict a non-smoking status at the end of the experiment, and are thus dropped.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) clustered by family.

To check that the interaction between age and moral hazard in the HIE is robust, I vary the age cutoff point in Table 3.5. Specifically, the same regression done for Table 3.4 is repeated for cutoffs at age 34 and 40. I include results for the age cutoff point at

37 years old for comparison purposes. The estimated coefficients for plans with lower than 95% coinsurance rate do not change substantively. For non-smokers at baseline, the estimates are similar between the older and the younger individuals regardless of which cutoff point is chosen (Table 3.5 columns (1) & (2)). Results for smokers at baseline are also robust to the different age cutoff points. The estimates for the below 95% coinsurance plans are consistently significant at the 5% level for older smokers, and insignificant for younger smokers. In all cases of age division, the estimate for the older group is approximately twice as big as that for the younger group (Table 3.5 columns (3) & (4)).

3.6 Intensive Margin

Having examined how coinsurance rates affect smoking behavior on the extensive margin, I focus on the intensive margin in this section. Do people in plans with lower coinsurance rates consume more cigarettes? I use both an ordered probit model and a Tobit model to estimate whether moral hazard exists on the intensive margin.

3.6.1 Methods

One way to measure effects on the intensive margin is to estimate the outcome equation separately from the participation equation. This strategy assumes that the unobservable factors in the participation equation are not correlated with the unobservable factors in the outcome equation. This method is widely used in the health economics literature (Bhattacharya et al., 2011; Madden, 2008; Manning et al., 1995). Because smoking intensity is originally recorded in the HIE as a categorical variable, the effect of coin-

surance on smoking intensity is estimated using an ordered probit model.

$$y^*_{i,1} = \alpha + \theta Y_{i,0} + x_i'\beta + c_i'\gamma + \epsilon_i,\ \epsilon_i \sim N(0,1) \tag{3.2}$$
$$Y_{i,1} = j,\ \text{if}\ \mu_{j-1} < y^*_{i,1} \leq \mu_j.$$

The dependent variable $Y_{i,1}$ is the cigarette consumption level at the end of the experiment. It takes on the value j (0, 1, 2, 3, and 4) corresponding to the different categories of smoking intensity: don't smoke, smoke less than 1 pack/day, about 1 pack/day, about 2 packs/day, and more than 2 packs/day. $Y_{i,0}$ is the cigarette consumption level at baseline. The same vectors of explanatory and control variables are employed as in equation (3.1) of Section 3.5.1, x_i and c_i. The latent variable $y^*_{i,1}$, which is a continuous random variable, relates the independent variables to $Y_{i,1}$such that whenever $y^*_{i,1}$ is in the range $(\mu_{j-1}, \mu_j]$, $Y_{i,1}$takes on the value j. Also note that $\bigcup_{j=0}^{4}(\mu_{j-1}, \mu_j] = \mathbb{R}$.

The probability that an individual's smoking intensity at the end of the experiment belongs to a category j is therefore expressed in the following way:

$$\begin{aligned}
&\Pr(Y_{i,1} = j \mid [1, Y_{i,0}, x_i', c_i']') \\
&= \Pr(\mu_{j-1} < y^*_{i,1} \leq \mu_j) \\
&= \Pr(\mu_{j-1} < \alpha + Y_{i,0}\theta + x_i'\beta + c_i'\gamma + \epsilon_i \leq \mu_j) \\
&= \Phi(\mu_j - (\alpha + Y_{i,0}\theta + x_i'\beta + c_i'\gamma)) - \Phi(\mu_{j-1} - (\alpha + Y_{i,0}\theta + x_i'\beta + c_i'\gamma))
\end{aligned}$$

A better way to estimate the effect of coinsurance on smoking intensity is to relax the assumption that the unobserved factors in the participation equation are unrelated to the unobserved factors in the outcome equation. By using a Tobit model, the outcome

and participation equations are estimated in one system.

$$y^*_{i,1} = \alpha + Y_{i,0}\theta + x_i'\beta + c_i'\gamma + \epsilon_i,\ \epsilon_i \sim N(0, \sigma^2) \tag{3.3}$$

$$Y_{i,1} = \begin{cases} y^*_{i,1} & \text{if } y^*_{i,1} > 0 \\ 0 & \text{if } y^*_{i,1} \leq 0. \end{cases}$$

In the Tobit model above, the cigarette consumption level at the end of the experiment, $Y_{i,1}$ is converted from a categorical variable to a continuous one.[6] It takes on strictly positive values when the latent variable $y^*_{i,1}$ is greater than 0, and takes on the value 0 when $y^*_{i,1}$ is less than or equal to 0. The regressors in equation (3.3) are the same as the ones used in equation (3.2). The expected number of cigarettes consumed per day at the end of the experiment conditional on the regressors can then be expressed as the following:

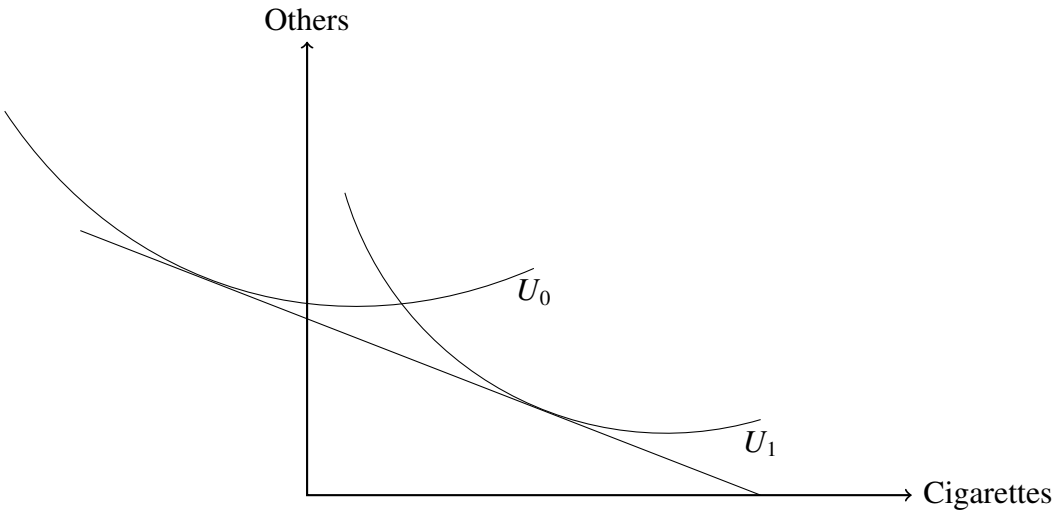
$$\begin{aligned} &E(Y_{i,1} \mid [1, Y_{i,0}, x_i', c_i']') \\ &= \Pr(Y_{i,1} > 0 \mid [1, Y_{i,0}, x_i', c_i']') E(Y_{i,1} \mid Y_{i,1} > 0, [1, Y_{i,0}, x_i', c_i']') \\ &= \Phi(\frac{\alpha + Y_{i,0}\theta + x_i'\beta + c_i'\gamma}{\sigma})(\alpha + Y_{i,0}\theta + x_i'\beta + c_i'\gamma + \sigma\lambda) \\ &\text{where } \lambda = \frac{\phi(\frac{\alpha+Y_{i,0}\theta+x_i'\beta+c_i'\gamma}{\sigma})}{\Phi(\frac{\alpha+Y_{i,0}\theta+x_i'\beta+c_i'\gamma}{\sigma})} \text{ and } \phi \text{ is the pdf of a } N(0,1). \end{aligned}$$

The key difference between the Tobit model and the ordered probit model is that the former assumes a relationship between changes in the extensive and intensive margins, while the latter does not. Figure 3.1 provides a simple illustration of how the outcome and participation equations may be determined by the same data generating process. An individual maximizes utility by deciding whether and how much to smoke. Initially, the highest utility level that could be achieved is at U_0, where the optimal choice is to not

[6]The conversion from a categorical variable to a continuous one is done by taking the mean in each category. Because a pack of cigarettes usually contains 20 cigarettes, <1 pack/day converts to 10 cigarettes/day, about 1 pack/day to 20 cigarettes/day, about 2 packs/day to 40 cigarettes/day, and more than 2 packs/day is capped at 60 cigarettes/day.

smoke. Suppose that following a change in the individual's taste preferences, the highest utility curve is now at U_1. The movement from U_0 to U_1 not only determines whether the individual smokes or not, but also determines how much the individual smokes.

Figure 3.1: A Change in Taste Preference Affects Both the Intensive and Extensive Margins



In both the ordered probit and Tobit models, I divide the analysis sample into non-smokers and smokers at baseline. Regression results where non-smokers and smokers are further divided into younger and older age groups are also presented under the Tobit model. Moral hazard exists on the intensive margin if people in lower coinsurance groups have a larger amount of cigarette consumption.

3.6.2 Results

Overall, initial non-smokers who start smoking do seem to exhibit moral hazard on the intensive margin. Especially for non-smokers assigned to the free care plan at the start of the experiment, they smoke more cigarettes per day by the end of the experiment than those assigned to the 95% coinsurance plan. Additionally, the effect differs by age,

Table 3.6: Coefficient Estimates of the Intensive Margin under the Ordered Probit Model

Ordered Probit Coefficient	(1)	(2)	(3)	(4)
Cigarette consumption at exit	Non-smokers at entry		Smokers at entry	
< 95% Coinsurance Rate		0.291**		0.098
		(0.146)		(0.108)
0% Coinsurance Rate	0.589**		0.021	
	(0.260)		(0.175)	
25% Coinsurance Rate	0.288*		0.104	
	(0.165)		(0.121)	
50% Coinsurance Rate	0.166		0.145	
	(0.216)		(0.154)	
Individual Deductible	0.294	−0.135	0.059	−0.005
	(0.211)	(0.142)	(0.143)	(0.095)
Maximum $ Expenditure (k)	0.968***	0.594**	0.362	0.458**
	(0.353)	(0.256)	(0.252)	(0.185)
Participation Incentive (k)	−0.552**	−0.503*	−0.470**	−0.481**
	(0.264)	(0.267)	(0.192)	(0.190)
Cutoff point 1	1.883***	1.692***	−3.310***	−3.265***
	(0.565)	(0.555)	(0.629)	(0.630)
Cutoff point 2	2.285***	2.091***	−2.564***	−2.519***
	(0.557)	(0.551)	(0.623)	(0.623)
Cutoff point 3	2.832***	2.634***	−1.186*	−1.142*
	(0.559)	(0.550)	(0.614)	(0.614)
Cutoff point 4			0.39	0.434
			(0.592)	(0.591)
N	1549	1549	1044	1044

Note: All regressions include number of cigarettes consumed per day at entry, demographic controls (age, education, family income, family size, race, marital status, sex, and self-reported health status before the experiment), length in the experiment, site effects and year effects.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) clustered by family.

with the older group more prone to moral hazard from the HIE than the younger group. For initial smokers who continue to smoke, coinsurance rates do not seem to affect their smoking intensity for either the older or younger participants.

Table 3.6 gives results for the ordered probit model based on equation (3.2), where the dependent variable is the level of cigarette consumption per day. It shows evidence

that moral hazard exists for *initial non-smokers* on the intensive margin. Relative to the 95% group, the estimated coefficients for the 0%, 25% and 50% coinsurance rates are all positive (column (1)). The magnitude of the estimates decreases as the coinsurance rates rise. Moreover, the estimates for the 0% and 25% groups are significant at the 5% and 10% confidence levels. The moral hazard effect is also apparent when using only an indicator for plans with coinsurance rates below 95% in column (2) – the positive and significant estimate suggest that for non-smokers who start smoking, those with rates lower than 95% are more likely to smoke at a higher intensity level than those with the 95% rate. However, there is no clear evidence that *initial smokers* increase their smoking intensity when given more generous plans. While the coinsurance coefficients in column (3) are positive, they are all statistically insignificant. The same is true in column (4), where the indicator for rates less than 95% is imprecisely estimated for *initial smokers*.

In Table 3.7(a), I re-estimate the effect of coinsurance on smoking intensity using the Tobit model (equation (3.3)). Column (1) presents results for the non-smokers at the start of the experiment. The Tobit coefficients for the 0%, 25%, and 50% coinsurance rates are 19.10, 9.62 and 5.60. The positive signs together with the decreasing pattern of the estimates suggest that more generous insurance plans are associated with more cigarette consumption at the end of the experiment. In addition, the effect is estimated with the most precision when comparing individuals in the 0% and 95% coinsurance plans. A more general comparison of initial non-smokers with coinsurance rates below 95% to those with the 95% rate in column (2) reinforces the positive relationship between the generosity of plans and the level of cigarette consumption. In contrast, the estimated coinsurance coefficients for the baseline smokers are much smaller: 0.07, 1.35, and 1.55 for the 0%, 25%, and 50% plans. None of these estimates are statistically significant. Even after combining the three binary variables for coinsurance into one indicator for rates below 95%, the estimate for the single indicator in column (4) is still small and

Table 3.7(a): Coefficient Estimates of the Intensive Margin under the Tobit Model

Tobit coefficient	(1)	(2)	(3)	(4)
Cigarette consumption at exit	Non-smokers at entry		Smokers at entry	
< 95% Coinsurance Rate		9.737*		1.341
		(5.202)		(1.411)
0% Coinsurance Rate	19.096**		0.171	
	(9.208)		(2.266)	
25% Coinsurance Rate	9.619*		1.42	
	(5.806)		(1.554)	
50% Coinsurance Rate	5.595		2.098	
	(7.511)		(2.058)	
Individual Deductible	9.635	−4.403	0.828	0.005
	(7.346)	(4.970)	(1.868)	(1.225)
Maximum $ Expenditure (k)	31.964***	20.231**	4.623	6.105**
	(12.177)	(8.620)	(3.266)	(2.401)
Participation Incentive (k)	−18.901**	−17.407*	−6.079**	−6.259**
	(8.889)	(8.994)	(2.498)	(2.472)
sigma	34.747***	34.882***	13.721***	13.725***
	(2.212)	(2.234)	(0.489)	(0.490)
N	1549	1549	1044	1044

Note: All columns include number of cigarettes consumed per day at entry, demographic controls (age, education, family income, family size, race, marital status, sex, and self-reported health status before the experiment), length in the experiment, site effects and year effects.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) clustered by family.

statistically insignificant for baseline smokers.

Table 3.7(b) reports the average marginal effects from the Tobit model. It is interesting to note that for initial non-smokers, those in the 0% coinsurance group who started smoking by the end of the experiment smoke about one cigarette more per day relative to those in the 95% group. When comparing more broadly between non-smokers with coinsurance rates less than 95% and those with the 95% rate, the former only smoke about half a cigarette more per day than the latter. Clearly, the estimated moral hazard effect on the intensive margin is mostly coming from the 0% coinsurance plan for initial non-smokers.

Table 3.7(b): Average Marginal Effects from the Tobit Model

Number of Daily Cigarette Consumption at Exit	(1)	(2)	(3)	(4)
	Non-smoker at entry		Smoker at entry	
< 95% Coinsurance Rate		0.477**		1.149
		(0.222)		(1.200)
0% Coinsurance Rate	0.973*		0.146	
	(0.514)		(1.931)	
25% Coinsurance Rate	0.547		1.233	
	(0.334)		(1.348)	
50% Coinsurance Rate	0.309		1.847	
	(0.444)		(1.820)	

Note: Average marginal effects calculated based on Table 3.7(a), dydx for binary variables is the discrete change between 0 and 1. The comparison group is the 95% coinsurance plan.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) clustered by family.

To see whether moral hazard from the HIE is heterogeneous across age on the intensive margin, I further divide the initial non-smokers and smokers into young and old age groups at the mean (37 years old). Results are shown in Table 3.8. Similar to the analysis on the extensive margin, the explanatory variables for the coinsurance rates are again collapsed into a single indicator for rates below 95%. For *initial non-smokers*, the estimated coefficient is highly significant at the 1% level for those older than age 37 (column (2)), but insignificant for the younger half (column (1)). For *initial smokers*, the estimates for the below 95% rate plans are insignificant for both the older and the younger age groups.

When looking at the control variables, it is worth pointing out that the estimated coefficients for the participation incentives are all negative and precisely estimated (Tables 3.6 through 3.8). This implies the possible existence of a negative income effect on the intensive margin resulting from a direct payment of incentives to the participants. Controlling for participation incentives is thus crucial when studying moral hazard in smoking intensity from the HIE.

Table 3.8: The Effect of Coinsurance on Smoking Intensity by Age

Tobit Coefficients	(1)	(2)	(3)	(4)
Number of Daily Cigarette	Non-smoker at entry		Smoker at entry	
Consumption at Exit	Age<37	Age≥37	Age<37	Age≥37
< 95% Coinsurance Rate	9.239	9.505***	0.603	2.322
	(6.641)	(2.003)	(1.940)	(2.014)
Individual Deductible	0.523	−10.697***	−1.345	1.968
	(6.040)	(1.593)	(1.609)	(1.892)
Maximum $ Expenditure (k)	3.713	38.103***	6.439**	6.074*
	(12.067)	(2.389)	(3.217)	(3.234)
Participation Incentive (k)	−7.51	−31.736***	−7.996**	−4.442
	(12.982)	(2.597)	(3.460)	(3.335)
sigma	35.208***	31.112***	13.762***	13.208***
	(2.868)	(1.065)	(0.616)	(0.752)
N	831	718	613	431

Note: All columns include number of cigarettes consumed per day at entry, demographic controls (age, education, family income, family size, race, marital status, sex, and self-reported health status before the experiment), length in the experiment, site effects and year effects.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) clustered by family.

3.6.3 Discussion and Robustness Checks

There are two main findings from both the ordered probit model and the Tobit model regarding the existence of moral hazard on the intensive margin: (a) the moral hazard effect is most significant for non-smokers at baseline who were assigned to a 0% coinsurance plan, and who were old; (b) smokers at baseline do not substantively change the number of cigarettes consumed per day in response to a change in the coinsurance rate. Finding (a) shows the importance of financial incentives and expectations in the HIE. When offered free care, individuals bear no financial risk (in terms of medical bills) from smoking, and hence smoke more. When individuals are older and have a higher expectation of benefiting from the HIE in the 3-year or 5-year time interval, they also smoke more. One plausible explanation for finding (b) may be that smokers at baseline who

Table 3.9: Robustness Checks for Different Age Cutoffs on the Intensive Margin

Tobit coefficient	(1)	(2)	(3)	(4)
Number of daily cigarette	Non-smokers at entry		Smokers at entry	
consumption at exit	Age<c	Age≥c	Age<c	Age≥c
c=34				
< 95% coinsurance	8.475	9.969***	−0.211	2.915
	(6.700)	(1.935)	(2.014)	(1.896)
N	791	848	530	514
c=37				
< 95% coinsurance	9.239	9.505***	0.603	2.322
	(6.641)	(2.003)	(1.940)	(2.014)
N	831	718	613	431
c=40				
< 95% coinsurance	11.043*	7.780***	1.218	1.700
	(6.282)	(2.255)	(1.824)	(2.158)
N	950	599	683	361

Note: All regressions include number of cigarettes consumed per day at entry, demographic controls (age, education, family income, family size, race, marital status, sex, and self-reported health status before the experiment), length in the experiment, site effects and year effects.

* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) clustered by family.

continue to smoke at the end of the experiment are inherently not very worried about getting sick from smoking, and are hence insensitive to a change in the coinsurance rates.

Finally, in Table 3.9 I check the robustness of the interaction between age and moral hazard in the HIE by altering the age cutoff point and re-running the Tobit model based on equation (3.3). Columns (1) and (2) show consistent evidence that the moral hazard effect on the intensive margin is more precisely estimated for the older individuals in the initial non-smoker group than for the younger ones, regardless of whether the age cutoff point is at 34, 37, or 40 years old. For smokers at baseline, the results are also robust: irrespective of how the age groups are divided, coinsurance rates have little effect on the number of daily cigarettes consumed among initial smokers who continued to smoke by the end of the HIE (columns (3) & (4)).

3.7 Conclusion

This paper studies the moral hazard effect of health insurance on smoking for both the extensive and the intensive margins. Non-smokers at baseline have a 4.7 percentage point higher probability of smoking initiation in the 0% coinsurance plan than the 95% plan. They also smoke about 1 cigarette more per day by the end of the experiment in the 0% plan relative to the 95% plan, with the effect mostly from initial non-smokers who were older. Baseline smokers are more likely to continue smoking as long as their insurance plans pay some portion of their medical bills. This effect is again more significant in terms of precision and magnitude for older individuals than for younger ones. There is little evidence that baseline smokers change their smoking intensity in response to a change in the coinsurance rates.

It is clear that moral hazard varies by the initial smoking status. A large reduction in coinsurance rates is required for baseline non-smokers to increase their probability of smoking initiation. In comparison, for baseline smokers, as long as there is some cost sharing by the health insurance plans, they are more likely to continue smoking compared to when they have to pay almost everything out-of-pocket. In terms of the number of cigarettes consumed per day at the end of the experiment, initial non-smokers are more responsive to a change in coinsurance rates than smokers. There is a positive relationship between the generosity of a plan and the smoking intensity for initial non-smokers, but the link is less clear for initial smokers.

Moral hazard is also more likely to exist among older individuals than younger ones in the HIE. Because the plans only lasted for either 3 or 5 years, and smoking-related illnesses often occur later in life, it is possible that older individuals may expect to benefit more from the generous plans than younger individuals. In other words, recognizing

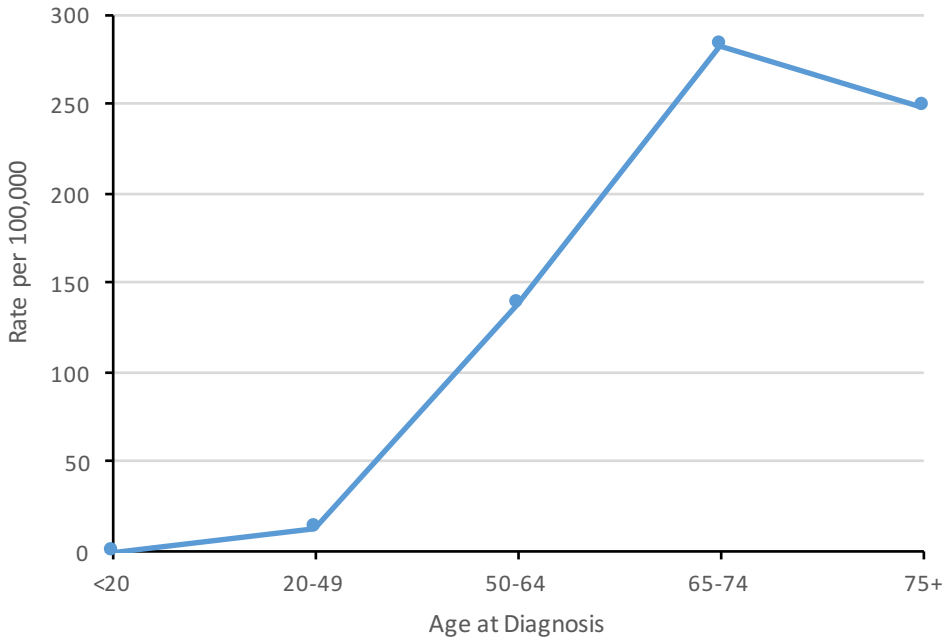
that the low coinsurance rates in the HIE are more relevant to the older participants is essential when estimating the moral hazard effect of health insurance on smoking.

As a final note, it would be interesting to extend the findings on coinsurance rates to premiums, and look at how much premiums have to increase in order to prevent any insurance-induced smoking behavior. This paper shows that a moral hazard effect for initial non-smokers is mostly precisely estimated when coinsurance rates decrease from 95% to 0%, and for initial smokers when coinsurance rates decrease from 95% to 50%. As a back of the envelope calculation, the average excess healthcare cost due to smoking is $2056 for a worker in 2010 (Berman et al., 2014). This means that holding everything else fixed, an increase in coinsurance rates from 0% to 95% would increase the out-of-pocket payment related to smoking by about $1953. The average annual premium for single worker coverage is $5049 in 2010 (Henry J. Kaiser Family Foundation, 2010), which suggests that if coinsurance rates were instead held constant, the same amount of increase in out-of-pocket payment could be achieved through a 39% ($1953/$5049) increase in premiums. Under the Patient Protection and Affordable Care Act (PPACA), insurance companies can charge as much as a 50% higher premium for smokers.[7] While my back of the envelope calculation above suggests an incremental increase in smokers' premium similar to that of the PPACA, future work could examine in greater detail the effect of health insurance premiums on smoking when such data becomes available.

[7] http://www.whitehouse.gov/healthreform/healthcare-overview

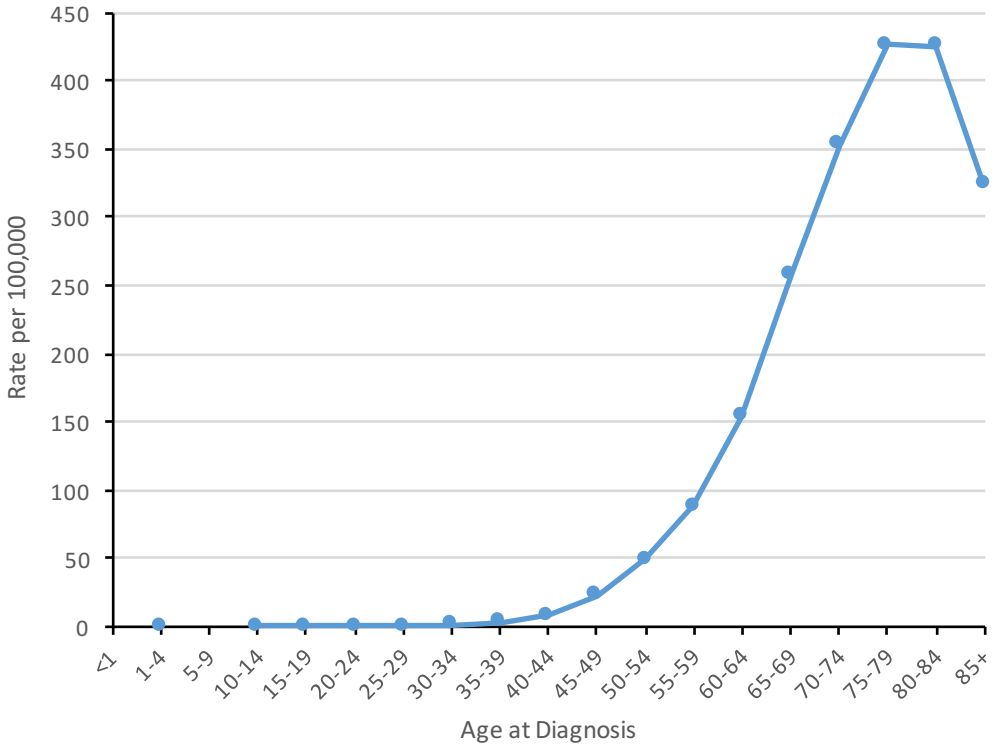
3.8 Appendix A

Figure A3.1: Age-Adjusted Lung Cancer Incidence Rates 1975-1985



Sources: National Cancer Institute's Surveillance, Epidemiology, and End Results program (SEER)

Figure A3.2: Age Specific (Crude) Lung Cancer Incidence Rates 2002-2011



Sources: National Cancer Institute's Surveillance, Epidemiology, and End Results program (SEER)

3.9 Appendix B

For the following five tables, CAMT and CAMTX stand for the packs of daily cigarette consumption at entry and at exit, respectively. There are five categories: 0-do not smoke, 1-less than one pack per day, 2-about one pack per day, 3-about two packs per day, and 4-more than two packs per day. In each table, the first row is the frequency, the second row represents the row percentage, and third row shows the column percentage.

Table A3.1: Transition Matrix of Cigarette Consumption for 0% Coinsurance

CAMTX	0	1	2	3	4	Total
CAMT						
0	483	14	9	5	0	511
	94.52	2.74	1.76	0.98	0	100
	87.98	14.74	6.29	7.35	0	59.21
1	32	57	30	2	0	121
	26.45	47.11	24.79	1.65	0	100
	5.83	60	20.98	2.94	0	14.02
2	24	22	83	17	1	147
	16.33	14.97	56.46	11.56	0.68	100
	4.37	23.16	58.04	25	12.5	17.03
3	9	2	21	42	3	77
	11.69	2.6	27.27	54.55	3.9	100
	1.64	2.11	14.69	61.76	37.5	8.92
4	1	0	0	2	4	7
	14.29	0	0	28.57	57.14	100
	0.18	0	0	2.94	50	0.81
Total	549	95	143	68	8	863
	63.62	11.01	16.57	7.88	0.93	100
	100	100	100	100	100	100

Table A3.2: Transition Matrix of Cigarette Consumption for 25% Coinsurance

CAMTX	0	1	2	3	4	Total
CAMT						
0	293	11	7	3	0	314
	93.31	3.5	2.23	0.96	0	100
	89.6	18.97	8.97	6	0	60.04
1	14	36	7	2	0	59
	23.73	61.02	11.86	3.39	0	100
	4.28	62.07	8.97	4	0	11.28
2	16	10	52	15	0	93
	17.2	10.75	55.91	16.13	0	100
	4.89	17.24	66.67	30	0	17.78
3	4	1	12	29	3	49
	8.16	2.04	24.49	59.18	6.12	100
	1.22	1.72	15.38	58	30	9.37
4	0	0	0	1	7	8
	0	0	0	12.5	87.5	100
	0	0	0	2	70	1.53
Total	327	58	78	50	10	523
	62.52	11.09	14.91	9.56	1.91	100
	100	100	100	100	100	100

Table A3.3: Transition Matrix of Cigarette Consumption for 50% Coinsurance

CAMTX	0	1	2	3	4	Total
CAMT						
0	103	4	3	1	0	111
	92.79	3.6	2.7	0.9	0	100
	94.5	21.05	9.38	5.26	0	61.67
1	2	12	2	0	0	16
	12.5	75	12.5	0	0	100
	1.83	63.16	6.25	0	0	8.89
2	3	3	21	7	0	34
	8.82	8.82	61.76	20.59	0	100
	2.75	15.79	65.63	36.84	0	18.89
3	1	0	5	11	1	18
	5.56	0	27.78	61.11	5.56	100
	0.92	0	15.63	57.89	100	10
4	0	0	1	0	0	1
	0	0	100	0	0	100
	0	0	3.13	0	0	0.56
Total	109	19	32	19	1	180
	60.56	10.56	17.78	10.56	0.56	100
	100	100	100	100	100	100

Table A3.4: Transition Matrix of Cigarette Consumption for 95% Coinsurance

CAMTX	0	1	2	3	4	Total
CAMT						
0	303	10	4	0	0	317
	95.58	3.15	1.26	0	0	100
	87.32	24.39	4.17	0	0	59.92
1	20	23	17	0	0	60
	33.33	38.33	28.33	0	0	100
	5.76	56.1	17.71	0	0	11.34
2	17	7	70	11	2	107
	15.89	6.54	65.42	10.28	1.87	100
	4.9	17.07	72.92	28.95	28.57	20.23
3	4	1	5	27	4	41
	9.76	2.44	12.2	65.85	9.76	100
	1.15	2.44	5.21	71.05	57.14	7.75
4	3	0	0	0	1	4
	75	0	0	0	25	100
	0.86	0	0	0	14.29	0.76
Total	347	41	96	38	7	529
	65.6	7.75	18.15	7.18	1.32	100
	100	100	100	100	100	100

Table A3.5: Transition Matrix of Cigarette Consumption for Deductibles

CAMTX	0	1	2	3	4	Total
CAMT						
0	288	11	5	1	0	305
	94.43	3.61	1.64	0.33	0	100
	89.44	18.03	5.75	2.33	0	58.99
1	18	31	19	0	0	68
	26.47	45.59	27.94	0	0	100
	5.59	50.82	21.84	0	0	13.15
2	8	19	53	13	1	94
	8.51	20.21	56.38	13.83	1.06	100
	2.48	31.15	60.92	30.23	25	18.18
3	8	0	10	29	1	48
	16.67	0	20.83	60.42	2.08	100
	2.48	0	11.49	67.44	25	9.28
4	0	0	0	0	2	2
	0	0	0	0	100	100
	0	0	0	0	50	0.39
Total	322	61	87	43	4	517
	62.28	11.8	16.83	8.32	0.77	100
	100	100	100	100	100	100

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