Killer Acquisitions*

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Firms may acquire innovative targets to discontinue the development of the targets' innovation projects in order to preempt future competition. We call such acquisitions "killer acquisitions." We develop a parsimonious model and provide empirical evidence for this phenomenon in drug development by tracking detailed project-level development histories of more than 60,000 drug projects. We show theoretically and empirically that acquired drug projects are less likely to be continued in the development process, and this result is particularly pronounced when the acquired project overlaps with the acquirer's development pipeline and when the acquirer has strong incentives to protect its market power. We also document that alternative interpretations such as optimal project selection, organizational frictions, and human capital and technology redeployment do not explain our results. Our conservative estimates indicate that about 7% of all acquisitions in our sample are killer acquisitions and that eliminating their adverse effect on drug project development would raise the pharmaceutical industry's aggregate drug project continuation rate by more than 5%. These findings have important implications for antitrust policy, startup exit, and the process of creative destruction.

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1. Introduction

This article highlights a novel, and potentially concerning, motive for corporate acquisitions—acquisitions to *kill*. We argue that an incumbent firm may acquire an innovative target and terminate development of the target's innovations to preempt future competition. We call such acquisitions "killer acquisitions" as they are intended to kill potentially promising, yet likely competing, innovation.

A recent case involving the pharmaceutical firm Mallinckrodt and its subsidiary Questcor exemplifies this phenomenon. In the early 2000s, Questcor enjoyed a monopoly in the category of adrenocorticotropic hormone (ACTH) drugs with its product Acthar. Acthar treats rare, serious conditions, including infantile spasms and nephrotic syndrome. In the mid-2000s, development began on Synacthen, a synthetic, direct competitor to Acthar. In an effort to pre-empt potential future competition, Questcor acquired the US development rights of Synacthen in 2013. Following the logic of killer acquisitions—that is, stopping competition before there is even a marketable product—Questcor did not develop Synacthen. No longer facing the prospect of other competitors, Questcor raised the price of Acthar from $40 per vial in 2001 to over $34,000 per vial by 2015. As the FTC argued in an antitrust complaint, Questcor acquired Synacthen to preempt competition: "With the acquisition of Synacthen, Questcor thwarted a nascent challenge to its Acthar monopoly."[1] In other words, unlike typical antitrust, Questcor was punished for eliminating competition *preemptively*. In January 2017, Mallinckrodt (which acquired Questcor in 2014) settled the anti-competitive acquisition case, agreeing to pay $100 million.

In this paper, we theoretically model and empirically demonstrate this phenomenon. Our analysis proceeds in two steps. First, to motivate the empirical analysis, we formalize the concept of a killer acquisition using a parsimonious model that combines endogenous acquisition decisions, innovation, and product market competition. In our model, an incumbent firm that acquires an entrant with an innovative project has weaker incentives to continue the project's development compared to a non-acquired entrant if the new project (partially) overlaps with

[1] FTC Matter/File Number: 1310172, "Complaint for Injunctive and Other Equitable Relief," https://www.ftc.gov/system/files/documents/cases/170118mallinckrodt_complaint_public.pdf

the incumbent's existing product portfolio. This is because the incumbent acquirer suffers from cannibalization of his existing product portfolio or, in other words, because of "the monopolist's disincentive created by his preinvention monopoly profits". This is Arrow's "replacement effect" (Arrow, 1962). This replacement effect can be so strong that incumbent firms may acquire startups simply to kill their projects and to prevent them from developing overlapping products that, if successful, would cannibalize the incumbent's profits (i.e., killer acquisitions).

We show that the replacement effect is present for any degree of acquirer-target product overlap and in such cases acquirers have strictly stronger incentives to discontinue project development than independent entrepreneurs. In addition, higher product market competition erodes the incumbents' profits and reduces the negative impact of the replacement effect when project development is successful. As a result, both existing competition as well as future product market competition (e.g., following patent expiry) diminish the killer acquisition motive.

In the second part of the paper, we aim to provide empirical support for our arguments. Conceptually, our empirical test for killer acquisitions is simple. We compare the development of acquired projects and those that are not acquired; we treat a lower continuation rate of acquired projects as a sign of "killer acquisitions." Importantly, we expect killer acquisitions to be more frequent when the target project overlaps with the acquirer's innovation pipelines or existing products.

The implementation of our tests, however, presents many empirical challenges. An ideal setting requires first that we observe outcomes at the project level, including, notably, continuation events. Second, we need to observe both project-level development within the target company prior to the acquisition as well as continuation and development decisions for the same project after acquisition. Further, we need be able to accurately characterize the potential product market overlap between the acquiring firm and the target's project as well as competition in the related product market.

We overcome these empirical challenges by focusing on the pharmaceutical industry and exploiting the setting of drug development. We collect detailed development information on more than 60,000 drug projects originated by more than 8,000 companies in the past two

and half decades, accompanied by the acquisition events collected from comprehensive data sources. We are able to observe the full development cycle for each drug from the initiation to the end point of the project (either successfully launched or discontinued). Importantly, we observe project development independent of acquisition events. For example, we can observe Dom-0800, an anti-CD40 ligand human domain antibody, originated by Domantis in 2005. Domantis was acquired by GlaxoSmithKline in 2006; yet, we are able to follow the development of Dom-0800 post-2006, regardless of its change in ownership.

Moreover, we collect information to characterize both the market (the intended disease) and the technology (the mechanism of action) of each drug project. We use market-technology measures to finely categorize acquirer overlap with the target's project, and thus identify potentially competing products. Further, we are able to separately characterize competition in both the development pipeline and product market of the project by distinguishing products under development and launched products. Using detailed pharmaceutical categorizations to measure overlap and competition is particularly desirable given the complications associated with coarse industry codes and wide variations in product categorizations often used out of necessity in other settings.

Armed with this database, a simple cross-sectional comparison of discontinuation rates shows that drug development projects that undergo an acquisition are on average less likely to be continued in the development process. Or equivalently, acquired projects are more likely to be "killed." Quantitatively, using all drug projects that originated from 1990 to 2011, we find that 92.11% of acquired drugs were discontinued by 2017, while the termination rate was 84.95% for non-acquired drugs. This pattern holds if we limit our sample to those that originated before 2000 (i.e., those with a more complete life-cycle record). Further, when we use a drug-year panel to characterize the annual probability of continuing a drug project, we show that post-acquisition, a drug is 22.09% less likely to be continued in the development process in each year. Overall, killer acquisitions dominate the acquisition sample.

Our key test for killer acquisitions is whether discontinuation of acquired projects is more pervasive when the target's new project could plausible compete with the acquirer's drugs. We capture this product market overlap by flagging whether the target's drug falls in a drug market, defined as the same therapeutic market and mechanism of action, in which

the acquirer is developing or has developed a drug. We show that killer acquisitions occur more often (doubling the intensity to discontinue a project) when the acquired drug overlaps with the acquirer's drugs. Further, we find that the intensity is dampened if the acquirer's overlapping drug is near patent expiry in which case the effects of cannibalization are minimal.

Acquired project terminations are also more pervasive when the acquirer has more market power and thus has more to lose if the target's new product successfully launches due to the replacement effect. We test this idea by repeating our baseline analysis in project subsamples with different levels of existing competition. We measure competition using the number of firms with competing projects in the same therapeutic market and mechanism of action, either launched in the product market or in the development pipeline. We find that killer acquisitions are concentrated in areas with low levels of product market competition. In additional analyses, we examine the progression of projects through the phases of clinical trials, and similarly find that projects that start Phase I trials are less likely to enter Phase II if they are acquired, and in particular when there is overlap between the target drug and the acquirer, and that these findings are concentrated in areas with low product market competition.

We conduct several refinements of the baseline analysis to sharpen the interpretation that acquiring firms intentionally kill targets' projects. One potential alternative explanation for our baseline finding is optimal project selection. In particular, the acquirer could strategically and optimally choose to continue the more promising or complementary projects of the target, but discontinue those that are tangential to the goal of the acquisition. To assess this concern, we repeat our analysis in acquisitions of single-drug companies, where the acquirer cannot be employing an "optimal project selection" strategy. Our results are robust to focusing on only this set of acquisitions, and, moreover, the magnitude actually increases. Hence, "optimal project selection" cannot explain our results.

Economic forces on the acquirer side could also confound our baseline interpretation. Previous research shows that the absence of private benefits in mature firms decreases the tendency to continue development (Guedj and Scharfstein, 2004) and that complex organization structures in larger firms are detrimental to the development of innovation projects (Seru, 2014). These forces could be the driving force behind the termination or

slow-down of development after a project is acquired. We guard against this concern by including fixed effects at the developing company level (i.e., the acquirer firm after the acquisition), intended to capture acquirer firm-specific development productivity that could affect project development when changing owners. We find that after controlling for the developing ability of the acquirer firm, the killing intensity increases.

Another plausible explanation is capital redeployment, i.e. post-acquisition project discontinuation is a by-product of the process of integrating and more efficiently redeploying acquired human capital and technologies to other projects. We collect detailed information on inventor mobility and productivity around the acquisition events, and information on the chemical similarity of drugs. We show that only 22% of inventors from target firms eventually work for the acquiring firm and further show that those inventors do not become more productive post-acquisition. We also find no supporting evidence that acquired technologies are integrated into acquirers' drug new development projects. These results are inconsistent with explanations regarding human capital or technology redeployment.

The central idea of this article is that incumbents have lower incentives to pursue innovation and may acquire potential future competitors to kill innovation. The first part of this idea dates back to at least Arrow (1962) who noted that the benefits of introducing a new product are smaller for incumbents than entrants, to the extent that old and new goods substitute for each other ("replacement effect").[2] The second part has its theoretical roots in Gilbert and Newbery (1982) who demonstrate that a monopolist has incentives to acquire the property rights to a new innovation to preempt entry ("efficiency effect"). Our paper combines these two forces and offers a theoretical and empirical analysis in the context of drug development.

Our paper is also related to a large literature in corporate finance and industrial organization which broadly highlights three distinct motives for acquisition: agency conflicts, synergies, and market power. First, in the absence of appropriate corporate governance mechanisms and incentive design, managerial interests that diverge from shareholder interests can lead to potentially value-destroying acquisitions (Roll, 1986; Morck et al., 1990). Second, acquisitions are driven by the pursuit of synergies between the acquirer and the target (Rhodes-Kropf

[2]Igami (2017) empirically shows that such cannibalization makes incumbents reluctant to innovate in the hard disk drive manufacturing industry. More broadly, incumbent firms' slow response to new technologies is explored in the large literature on competition and innovation. See Cohen (2010) for a comprehensive survey.

and Robinson, 2008). Mergers have been shown to increase industry-adjusted cash flows (Healy et al., 1992; Andrade et al., 2001) and productivity (Maksimovic and Phillips, 2001) and an active acquisition market can also spur innovation (Phillips and Zhdanov, 2013). The post-merger increases in cash flows, new products, and patents are related to the ex-ante similarity of acquirer and target (Bena and Li, 2014; Hoberg and Phillips, 2010), but are harder to realize in markets with product integration difficulty (Hoberg and Phillips, 2017). Third, M&A transactions between *existing* competitors may occur to increase market power. This is the focus of much of US (and foreign) antitrust law.[3]

Our analysis suggests another unique motive for acquisitions. Acquisitions of innovative entrants may be driven by the desire to preempt *future* product market competition. This preemption motive generates the same prediction as a synergistic acquisition strategy, namely that incumbent firms acquire entrants that are similar to them. However, the two motives have vastly different implications for post-acquisition behavior. While the synergy motive suggests that acquired projects should be more likely to continue in development, the preemption motive predicts the opposite. Our data provides detailed information on post-acquisition development at the project level which allows us to distinguish these motives. Our findings on the existence and relative prevalence of killer acquisitions also suggest that earlier research exclusively highlighting the importance of misaligned managerial incentives or synergies in acquisition decisions should be interpreted more cautiously. Further, killer acquisitions may constitute a form of monopolization through preemptive acquisition and their existence and prevalence raises considerable antitrust and innovation policy concerns.

Similar to the M&A literature, the markets for technology literature (Gans and Stern, 2003; Arora and Gambardella, 2010; Arora et al., 2014) typically assumes that innovation-related transactions are synergistic, and thus experience related to the technology (i.e., owning a related technology) enables evaluation and absorption, and therefore increases the likelihood of successful acquisition and innovation. However, relevant to our arguments,

[3]Kamien and Zang (1990), Kamien and Zang (1993), Gowrisankaran (1999), Segal (1999), and Gowrisankaran and Holmes (2004) theoretically study merger decisions between existing competitors and analyze eventual market structure in a setting without antitrust policy. These papers show that even without the actions of antitrust authorities an industry may not be inevitably monopolized via mergers (i.e., there are competitive forces that push against such a trend). Segal and Whinston (2007) show that more protective antitrust policy may have conflicting effects on innovation incentives, by raising the profits of new entrants, but lowering those of continuing incumbents.

some have suggested that acquisitions of small, innovative target firms may also serve to preempt competition by enabling vital technology access (Hall, 1990; Lerner and Merges, 1998; Blonigen and Taylor, 2000; Lehto and Lehtoranta, 2006; Grimpe and Hussinger, 2008).[4] This literature also investigates the conditions under which a startup firm would want to sell its technology to incumbents instead of competing with them in the product market (Gans and Stern, 2003; Gans et al., 2002). Both the presence of patents (which reduce hazard of expropriation) and incumbent ownership of development assets (which increase potential gains from trade and hence joint surplus) increase the likelihood that startups will want to (and be able to) sell their idea (Gans et al., 2002). The pharmaceutical industry is characterized by both of these features which explains why acquisition of startups are frequent and why killer acquisitions would be particularly prevalent.

In summary, our paper highlights why and when firms conduct killer acquisitions to prevent future competition. The remainder of the paper proceeds as follows. Section 2 outlines our theoretical framework and develops testable hypotheses. Section 3 describes data and institutional background. Section 4 presents our main empirical results. Section 5 rules out a number of alternative explanations and provides robustness checks. Section 6 discusses implications for antitrust and social welfare and quantifies the industry-wide impact of killer acquisitions. Section 7 offers concluding remarks.

2. Theoretical Framework

In this section we propose a simple theoretical model of acquisition, innovation, and product market competition decisions which we use to investigate the project development of entrepreneurial companies and incumbent firms and to show what factors increase the likelihood of killer acquisitions.

[4] Gans and Stern (2000) theoretically analyze R&D competition between entrants and incumbents in the shadow of acquisition and show how the acquisition price depends on the possibility of the entrant to enter the product market.

2.1. Setup

The model has the following time line. In $t = 0$, an entrepreneurial company E with a single project is born. E is the originating company of the project. There are $n \geq 1$ incumbent firms which each already possess an existing (and potentially overlapping) product. One of these n incumbents which we call the (potential) acquirer A decides whether to acquire the new firm at a endogenously determined takeover price P.

In $t = 1$, the owner of the project—the acquirer A if the project has been acquired, or the entrepreneur E if it remains independent in $t = 0$—decides whether to continue developing the project. The owner assesses the probability ρ that the project will ultimately be successful, and that she would want to continue or terminate the project. Let k be the cost of continuing development of the project and L the liquidation value of the project if the firm does not continue to develop the project at $t = 1$. To denote the two potential situations that the owner faces when deciding to continue development of the project in $t = 1$:

- acq, the originating firm was acquired in $t = 0$ (A owns the project)
- $\neg acq$, the originating firm was not acquired in $t = 0$ (E owns the project)

Finally, in $t = 2$, uncertainty about the success of the project is resolved and all the firms engage in differentiated Bertrand product market competition.[5] We assume that if the project is successfully developed in $t = 2$, the drug has a payoff of π which depends on the degree of competition (i.e., the number of active firms in the market) and product differentiation in the market. If the project is unsuccessful, the payoff is zero. There are no informational asymmetries or agency problems in this model as we assume that the values of π, ρ, k, and L are commonly known in $t = 0$ and identical for all the involved parties.

2.2. Product Market Competition ($t = 2$)

2.2.1. Consumer Demand. We follow Vives (2000) and Häckner (2000) and consider an industry with n products that are produced at 0 marginal cost. We derive demand from the

[5] We choose to model competition using differentiated Bertrand competition because price-setting behavior by firms captures the form of competition in the branded drug market. However, our results are not sensitive to this particular form of competition. They also hold for Cournot competition as we show in Appendix A.A.2.

behavior of a representative consumer with the following quadratic utility function:

$$U(q) = \alpha \sum_{i=1}^{n} q_i - \frac{1}{2}\left(\beta \sum_{i=1}^{n} q_i^2 + 2\gamma \sum_{i \neq j} q_i q_j\right) \tag{1}$$

where q_i is the quantity of product i, $\alpha > 0$ represents overall product quality, $\beta > 0$ measures the concavity of the utility function, and γ represents the degree of substitutability between products i and j. $\beta > \gamma > 0$ ensures that the products are (imperfect) substitutes. The higher the γ, the more alike are the products. The resulting consumer maximization problem yields linear inverse demand for each product i given by $p_i = \alpha - \beta q_i - \gamma \sum_{j \neq i}^{n} q_j$ where p_i is the price of product i.

2.2.2. No Acquisition. Consider first the product market choices of an entrepreneur that is not acquired ($\neg acq$) in $t = 0$. If the project is successful (S), the resulting newly developed product competes against n other single-product incumbent firms. The entrepreneur's objective function is equal to

$$\max_{p_E} p_E q_E \tag{2}$$

Given that all $n+1$ single-product firms are symmetric we solve for the symmetric equilibrium which yields the following profits

$$\pi^E_{\neg acq,S} = \frac{\alpha^2(\beta - \gamma)(\beta + (n-1)\gamma)}{(2\beta + (n-2)\gamma)^2(\beta + n\gamma)} = \pi^A_{\neg acq,S} \tag{3}$$

Note that the product market profits for the entrepreneur and the n incumbent firms, including the acquirer, are identical.

If the new project fails (F), the entrepreneur does not have any product to sell in $t = 2$ and thus her profit is equal to $\pi^E_{\neg acq,F} = 0$. The n incumbent firms each have a single existing product to sell and thus their profit is equal to

$$\pi^A_{\neg acq,F} = \frac{\alpha^2(\beta - \gamma)(\beta + (n-2)\gamma)}{(2\beta + (n-3)\gamma)^2(\beta + (n-1)\gamma)}. \tag{4}$$

2.2.3. Acquisition. Next consider the product market choices in case of an acquisition (acq) by the acquirer. If the project is successful the acquirer becomes a 2-product oligopolist which optimally chooses quantities for its new and its old product and competes against $n-1$ other single-product incumbents. Its objective function is

$$\max_{p_1,p_2} p_1 q_1 + p_2 q_2 \tag{5}$$

while the remaining $n-1$ other single-product firms maximize single-product profits. Given our symmetry assumptions, in equilibrium, $p_1^* = p_2^* = q^A$ and $p_i^* = p^I$ for any $i \neq 1, 2$.

The resulting profit of the multi-product incumbent acquirer is

$$\pi^A_{acq,S} = \frac{\alpha^2(\beta-\gamma)(\beta+(n-2)\gamma)(2\beta+\gamma(2n-1))^2}{2(\beta+n\gamma)(2\beta^2+(3n-4)\beta\gamma+(1+(n-3)n)\gamma^2)^2}. \tag{6}$$

If the project is unsuccessful, the acquirer can still sell the existing product in $t=2$ and only has to compete against $n-1$ other single-product incumbents. In this case the resulting profit for the acquirer is

$$\pi^A_{acq,F} = \frac{\alpha^2(\beta-\gamma)(\beta+(n-2)\gamma)}{(2\beta+(n-3)\gamma)^2(\beta+(n-1)\gamma)}. \tag{7}$$

Comparing the six different profit expressions immediately establishes the following profit ranking

$$\pi^A_{acq,S} > \pi^A_{acq,F} = \pi^A_{\neg acq,F} > \pi^A_{\neg acq,S} = \pi^E_{\neg acq,S} > \pi^E_{\neg acq,F} = 0. \tag{8}$$

The product market profits gained by the acquirer are always at least as large as those of the entrepreneur. This is because the acquirer can sell two products rather than just one if the newly acquired project is successful and it can mitigate the amount of substitution between its two products by pricing less aggressively thus resulting in profit $\pi^A_{acq,S}$. Even if development is not successful the incumbent can fall back on selling its existing product for which it faces only $n-1$ competitors and gain $\pi^A_{acq,F}$ while a successful entrepreneur would face n competitors and gain only $\pi^E_{\neg acq,S}$.

2.3. Continuation Decision ($t = 1$)

We now investigate the development continuation decision in $t = 1$.

2.3.1. The "Replacement Effect". What matters for the development decision in $t = 1$ are the difference between $\pi^A_{acq,S}$ and $\pi^A_{acq,F}$ for the incumbent and the difference between $\pi^E_{\neg acq,S}$ and $\pi^E_{\neg acq,F}$ for the entrepreneur. It is straightforward to show that for all imperfect substitutes $\beta > \gamma > 0$ we have

$$\Delta^E \equiv \pi^E_{\neg acq,S} - \pi^E_{\neg acq,F} > \pi^A_{acq,S} - \pi^A_{acq,F} \equiv \Delta^A \tag{9}$$

This is a very general result with a simple, well-known intuition. As long as product differentiation is not so large that products are independent goods ($\gamma = 0$) or perfect substitutes ($\gamma = \beta$) the acquirer gains strictly less from developing a new product than an entrepreneur would. This is because the new product cannibalizes some of the profits of the acquirer's existing product. In contrast, an entrepreneur has no product to sell and hence no profit if she does not successfully develop the project. This is Arrow's famous "replacement effect" (Arrow, 1962). If $\gamma = 0$, the incentives to innovate are actually identical for the incumbent and the entrepreneur because in that case bringing a new product to market does not cannibalize the profits of any existing product the incumbent already owns.

2.3.2. Product Market Overlap. The entrepreneur and the acquirer obtain different benefits from continuing development of their respective projects. When a firm is acquired its project becomes part of the greater drug development portfolio of the acquiring incumbent. This acquirer may have a portfolio of entirely different drugs or the portfolio may have some overlap with the acquired company's project. This overlap is governed by the product homogeneity γ in the product market competition in $t = 2$. In contrast, an entrepreneurial company's portfolio consists, by assumption, of only a single product.

Consider first the continuation decision of an entrepreneur, $d^E = \{0, 1\}$. The decision rule to continue with the development of the project is such that the entrepreneurial company

continues development $d^E = 1$ if

$$\rho\Delta^E - k \geq L \tag{10}$$

The acquirer gains $\pi^A_{acq,S}$ from successful development of the project, but also foregoes the profit $\pi^A_{acq,F}$ he would have earned otherwise. The decision to continue development of a project of an incumbent which potentially has some product market overlap with the acquired firm's product portfolio is $d^A = 1$ if

$$\rho\Delta^A - k \geq L \tag{11}$$

Rewriting the two inequalities for the continuation decisions given by (10) and (11) shows the different success probability thresholds used by the entrepreneur and the acquirer above which the firms continue development. We denote these thresholds by ρ^E and ρ^A which are

$$\rho^E = \frac{L+k}{\Delta^E}, \quad \rho^A = \frac{L+k}{\Delta^A} \tag{12}$$

Comparison of the above thresholds shows that $\rho^E < \rho^A$ for any (imperfect) substitutes ($\beta > \gamma > 0$) which immediately yields our first prediction because in that case $\Delta^E > \Delta^A$ as discussed above. Any form of product market overlap with existing drugs in the acquirer's portfolio reduces the acquirer's propensity to continue development of the acquired project relative to the case in which the project remains independent.

Proposition 1 (Project Killing and Market Overlap)**.** *An incumbent firm that acquires a project continues development if $\rho \geq \rho^A$ while an independent entrepreneur continues if $\rho \geq \rho^E$. For any positive product market overlap $\beta > \gamma > 0$, we have $\Delta^E > \Delta^A$ and hence $\rho^E < \rho^A$.*

The difference in continuation behavior between incumbent acquirer and entrepreneur occurs when ρ is in the intermediate range between ρ^E and ρ^A. This region exists for any degree of product substitutability $\beta > \gamma > 0$ and its size depends on the difference between the entrepreneur's and the acquirer's development gains Δ^E and Δ^A. If Δ^E is much larger than Δ^A then the entrepreneur's continuation incentives are much larger than the acquirer's.

But when Δ^E is only a little larger than Δ^A, for example when the two products are close to independent goods ($\gamma \approx 0$) then the two continuation policies are quite similar.

2.3.3. Existing Competition. The degree of existing competition as measured by the number of incumbents n plays an important role in determining the relative size of Δ^E and Δ^A. In particular, the difference between Δ^E and Δ^A is decreasing in n.

Proposition 2 (Project Killing and Competition)**.** *For any positive product market overlap $\beta > \gamma > 0$, the difference $\rho^A - \rho^E$ is positive and strictly decreasing in n.*

The intuition for this result is quite simple. Successfully developing a new product equally draws consumer demand away from existing products and thus hurts the profits of all incumbent firms. When the acquiring incumbent is a monopolist he is particularly hesitant to develop an overlapping product because the demand for this new product is being drawn away entirely from his own existing product. But when he already faces many other existing competitors introducing a new product draws demand away from all existing products, only one of which is his own. In other words, when there are many existing competitors the cannibalization losses from the successful development of a new product are spread over a large number of firms. In the limit in which the number of existing products n goes to infinity, the acquiring incumbent has a vanishingly small share of the existing market and thus all of the cannibalization losses fall on the other competitors. As a result, for $n \to \infty$, Δ^E and Δ^A are the same and hence the continuation policies of the entrepreneur and the acquirer are identical.

2.3.4. Patent Life and Future Competition. Until now, we have only considered the impact of competition with imperfect substitutes. In terms of our empirical setting this captures the competition between branded drugs. However, another important aspect is competition by undifferentiated generic drugs that enter the market when a branded product's patent expires. Denote the number of years of remaining patent life of the entrepreneur's new project by T^E and those of the acquiring incumbent's existing product by $T^A < T^E$. Assume further that the firms earn the static game profits every year.

It is natural to assume that as soon as a product's patent expires an identical, undifferentiated product (e.g., a generic drug) enters the market. Bertrand competition with undifferentiated products then implies that prices and profits for that product drop to zero. Thus, for the T^A years in which his existing product's patent is still valid the acquirer either earns $\pi^A_{acq,S}$ (successful development of new project) or $\pi^A_{acq,F}$ (unsuccessful development) each year. This yields the same development gain Δ^A as before multiplied by the number of years T^A. Similarly, the entrepreneur's development gain over that time span is $T^A\Delta^E$. Thereafter, the profits for the acquirer's existing product drop to 0 and hence his incentives to develop coincide with those of the entrepreneur. Denote the development gains for the entrepreneur and the acquirer in the presence of undifferentiated generic competition after the expiry of the acquirer's existing product's patent in T^A years by $\Delta_{gen} = \Delta^E_{gen} = \Delta^A_{gen}$.[6] The reason why these development gains after generic entry are the same for the acquirer and the entrepreneur is that when the incumbent's patent on his existing product expires he no longer has to be concerned about a new product cannibalizing the profits of his existing product: generic competition has already destroyed all those profits. As a result, after T^A years it is as if the acquiring incumbent did not have any existing overlapping product.

Thus, the continuation decisions of the entrepreneur d^E_{gen} and the acquiring incumbent d^A_{gen} are now determined by

$$\rho[T^A\Delta^E + (T^E - T^A)\Delta_{gen}] - k \geq L \tag{13}$$

$$\rho[T^A\Delta^A + (T^E - T^A)\Delta_{gen}] - k \geq L \tag{14}$$

where Δ_{gen} is the development gain for the entrepreneur and the incumbent in the presence of undifferentiated generic competition after the expiry of the acquirer's existing product's patent in T^A years.

From the two continuation inequalities it is immediately obvious that the longer the patent life T^A of the acquirer's existing product the weaker are his incentives to continue

[6]Note that these (equal) development gains are different from the previous expressions Δ^E and Δ^A. This is because when a generic product (that is undifferentiated from the acquirer's existing product) enters it not only drives profits of that product to zero, but due to its low price it also reduces the profits of the other products that are differentiated from it.

development relative to those of the entrepreneur.

Proposition 3 (Project Killing and Patent Life)**.** *For any positive product market overlap $\beta > \gamma > 0$, the difference $\rho^A - \rho^E$ is positive and strictly increasing in T^A.*

In other words, when the acquirer's existing overlapping product has only little remaining patent life (T^A close to 0), his continuation policy for the new project is quite similar to that of the entrepreneur. The intuition is essentially the same as that of Proposition 2. Generic entry is just a particularly intense form of competition that already destroys any profits of the acquirer's existing product and thus turns self-cannibalization from the development of a new product into an entirely moot point.

2.4. Acquisition Decision ($t = 0$)

In $t = 0$, one of the n incumbents (the acquirer A) decides whether or not to acquire the entrepreneur. Acquiring an entrepreneurial company yields an acquirer-specific payoff σ for the acquirer. This payoff is positive when there are synergies between the two firms. However, it may also be negative when the acquisition involves significant integration costs. When considering whether or not to acquire the entrepreneur the acquiring incumbent must weigh the purchase price P, any synergies and integration costs captured by σ as well as any potential cannibalization of its existing product resulting from product overlap. Note that this cannibalization may occur because of successful development by either the acquirer himself or by the entrepreneurial company if it remains independent.

Assume that all the model parameters are known with certainty at $t = 0$. Thus, the acquirer decides to acquire at a takeover price P if

$$\sigma + d^A[\rho\pi^A_{acq,S} + (1-\rho)\pi^A_{acq,F} - k] + (1-d^A)(L + \pi^A_{acq,F}) - P \geq d^E[\rho\pi^A_{\neg acq,S} + (1-\rho)\pi^A_{\neg acq,F}] + (1-d^E)\pi^A_{\neg acq,F} \quad (15)$$

where $d^i \in \{0,1\}$ for $i = \{E, A\}$ is the continuation decision for the project taken by the firm in $t = 1$ described by inequalities (10) and (11).

How is the takeover price P determined? To compensate the entrepreneur for selling

the company the incumbent must pay a price P that is equal to the expected payoff of the project under the continuation decision given by (10). Thus, the takeover price P is given by

$$P = d^E[\rho(\pi^E_{\neg acq,S} - \pi^E_{\neg acq,F}) - k] + (1 - d^E)L \tag{16}$$

Note that this price would be the result if the acquiring incumbent makes a take-it-or-leave-it to the entrepreneur in a bilateral bargaining game, but it would also be the result of any bidding contest in which there exists an outside bidder without an existing product that cannot realize any synergies ($\sigma = 0$) from the acquisition. Such a bidder would face exactly the same continuation decision as the entrepreneur in $t = 1$.

The inequality governing the acquisition decision (15) and the takeover price (16) depend on the continuation decisions d^A and d^E. There are thus three cases to consider. First, if $\rho < \rho^E$, neither acquired nor non-acquired firms choose to terminate the project, $d^A = d^E = 0$ and thus the decision rule whether or not to acquire given by (15) reduces to

$$\sigma \geq 0. \tag{17}$$

Second, for $\rho^E \leq \rho < \rho^A$, the acquiring incumbent terminates the acquired project, $d^A = 0$, while the entrepreneur continues $d^E = 1$ and thus the entrepreneur is acquired if

$$\sigma + \underbrace{\rho(\pi^A_{acq,F} - \pi^A_{\neg acq,S})}_{\text{efficiency effect}} \geq \underbrace{(\rho\Delta^E - k - L)}_{\text{replacement effect}} \tag{18}$$

If the incumbent acquires the entrepreneur's project (acq) and shuts it down, the acquirer only competes against $n - 1$ other firms thus earning a profit equal to $\pi^A_{acq,F}$. However, if the incumbent does not acquire the entrepreneur's project ($\neg acq$) and the entrepreneur successfully develops the project with probability ρ, the incumbent now has to compete against n other firms thus earning a lower profit $\pi^A_{\neg acq,S}$. Because competition reduces profits, the incumbent's incentive to remain unchallenged is greater than the entrepreneur's incentive to enter. This is the "efficiency effect", first discussed by (Gilbert and Newbery, 1982) in the context of monopoly persistence due to preemption incentives. On the other hand, if $\rho \leq \rho^E$,

the expected marginal profit for the entrepreneur from continuing development ($d^E = 1$) given by $\rho\Delta^E - k$ is larger than the liquidation value L that the acquiring incumbent ($d^A = 0$) would obtain. This is the "replacement effect" which leads to a difference in valuation for developing the project between the entrepreneur and the incumbent and decreases the incentive to acquire.

Third, for $\rho^A \leq \rho$, both acquired and non-acquired firms continue the project. Acquisition occurs if

$$\sigma + \underbrace{\rho(\pi^A_{acq,S} - \pi^A_{acq,F})}_{\text{efficiency effect}} \geq \underbrace{\rho(\Delta_E - \Delta_A)}_{\text{replacement effect}} \tag{19}$$

As before, $\pi^A_{acq,S} - \pi^A_{acq,F}$ is the "efficiency effect" that is the gain from preemption by acquiring the entrepreneur and using multi-product pricing to soften the impact of cannibalization of the newly introduced product. On the other hand, the difference in valuation for the product between the entrepreneur and acquirer is again driven by the "replacement effect" ($\Delta_E \geq \Delta_A$).

The inequalities (17), (18), and (19) illustrate the trade-off that the potential acquirer faces when contemplating the acquisition decision. The three driving forces in this decision are synergies, potential losses from cannibalization ("efficiency effect"), and differences in project development valuation between originating and acquiring firms ("replacement effect"). First, acquiring the originating firm at price P always yields synergies or integration costs σ. As discussed before, such net synergies can be either positive or negative, thereby increasing or decreasing the incentives for acquisition. Second, when ρ is sufficiently high that the entrepreneur is willing to continue development in $t = 1$ (i.e., $\rho \geq \rho^E$), acquiring the entrepreneur yields an additional benefit thus increasing the incentives for acquisition. In particular, it avoids incurring the aforementioned profit loss of $\pi^A_{acq,F} - \pi^A_{\neg acq,S}$ which results when the entrepreneur successfully develops the project with probability ρ. Third, the entrepreneur and the potential acquirer value the project differently and an acquiring incumbent must compensate the entrepreneur with an acquisition price P. This third effect is negative and thus reduces the incentives to acquire the entrepreneur. This is because the entrepreneur is both more willing to develop the project and also gains more conditional on successful development than the incumbent due to the "replacement effect". The "efficiency

effect" and the "replacement effect" work in opposite directions: the former is positive and the latter is negative for any $\beta > \gamma > 0$.

Proposition 4 (Acquisition Decisions)**.** *The potential acquirer acquires the entrepreneur in* $t = 0$ *if*

- $\rho < \rho^E$: $\quad \sigma \geq 0$
- $\rho^E \leq \rho < \rho^A$: $\quad \sigma + \rho(\pi^A_{acq,F} - \pi^A_{\neg acq,S}) \geq \rho(\Delta^E - k - L)$
- $\rho^A \leq \rho$: $\quad \sigma + \rho(\pi^A_{acq,S} - \pi^A_{acq,F}) \geq \rho(\Delta_E - \Delta_A)$

To summarize, in our model, entrepreneurial companies are acquired for two reasons. First, a potential acquirer has more to gain from acquiring entrepreneurial companies if he can realize larger synergies from the transaction or face relatively small integration costs. Such synergies may derive from technical expertise or complementary assets. Second, a potential incumbent acquirer has more to lose if they do not acquire an entrepreneurial company with a project that is similar to the acquirer's drug portfolio. This is the "efficiency effect". It raises the incentives for acquisition because it prevents the entrepreneur who has a higher propensity for continuing development of a project (due to the "replacement effect") from entering and reducing the profits of the potential acquirer. For example, Figure 1 plots the acquirer's payoffs from different acquisition choices. Similarly, Figures 2a and 2b show the dominance regions of acquisition strategies for an incumbent monopolist and duopolist. They illustrate that the relative size of the efficiency and replacement effect which depend on the degree of product substitutability and the particular form of competition, determines whether acquisitions occur or not.

3. Empirical Setup: Background and Data

The main empirical goal of our paper is to document the phenomenon of killer acquisitions. These acquisitions occur when acquiring firms acquire targets specifically to extinguish target technologies and prevent future competition. To do so, we need a setting and dataset that includes project level outcomes, both for projects that are acquired and a comparator set of

un-acquired projects, and a clean way to characterize the overlap between acquirer and target firms. Due to its regulated and therefore highly regularized product development processes, and because of frequent acquisitions of new firms by large incumbents, the pharmaceutical industry and drug development projects provide an ideal setting.

3.1. Drug Development Background

New pharmaceutical products, or drugs, are developed following a set of structured and sequential steps.[7] First, firms identify potential drug compounds through structured discovery processes. Then, for potentially promising molecules, firms run preliminary screening in vitro and/or in vivo to explore both efficacy and toxicity prior to any in human clinical trials. Last, firms undergo three phases of clinical trials in human subject for projects they find promising during pre-clinical tests [8]. Phase I trials are small (20 and 100 healthy volunteers), short, and are intended to test safety and dosage. Phase II trials are larger (100s of affected patients), typically are randomized control trials, last up to 2 years, and are intended to test efficacy. Phase III expand from Phase II trials, involving hundreds or thousands of participants and typically lasting 1 to 4 years. Near 70% of those entering phase I move to phase II, 33% from phase II to III, and about 25% of those move on from phase III (US Food and Drug Administration, 2017). Following successful trials, firms submit the drug to the FDA as a New Drug Application (NDA), and the FDA determines if, and under what conditions, the drug should be allowed to be marketed to patients. Each step in the process is more costly than the prior one, with total costs of each phase in the tens of millions ($USD) (Morgan et al., 2011). Hence, continuation of any drug project poses significant costs. Patented drugs then have a few years to earn monopoly profits before patent expiration and generic entry (Scherer, 1993). Because of this regular structure, and multiple costly steps involved in continuing each project, we are able to observe active continuation of projects, and further to see when a project is suspended or discontinued. Observing these events at the project level is crucial to identifying killer acquisitions.

[7] The steps below summarize those described in detail by the FDA (US Food and Drug Administration, 2017)

[8] Drug developers must submit a Investigation New Drug (IND) application to the FDA prior to starting clinical trials which must include: animal study and toxicity data; manufacturing information; clinical protocols (i.e., study plans); data from any prior human research; and, information about the investigator

3.2. Drug Development and Clinical Trial Data

We build our analytical dataset at the drug project level using Pharmaprojects from Pharma intelligence. Pharmaprojects is a comprehensive dataset that tracks drug projects from a very early stage through to launch or discontinuation, and documents the originating firm associated with each drug project.[9] Pharmaprojects includes nearly universal coverage of all candidate drugs being tested for eventual sale in the U.S. market, the intended therapeutic market (e.g., "osteoporosis") and mechanism of action (e.g., "calcium channel antagonist") (Branstetter et al., 2014). The database importantly records information on product development continuation events (e.g., "new patent applications" or "target identified") as well as product suspensions and discontinuations. We collect and follow all projects initiated by firms from 1989 until 2011. We stop our sample in 2011 as to see project continuation and acquisition events for at least 5 full years from initiation.

We supplement the project level outcome data from Pharmaprojects with data on clinical trials, sourced from Trialtrove, which we link to each project. Clinical trial data is available only from 1997 onwards only. Therefore, we have detailed trial information only for a subset of all projects in our sample. For these, we identify projects that start Phase I trials and track their progression to Phase II trials and beyond, following prior studies that use clinical trial progression as a measure of project development (Krieger, 2017; Guedj and Scharfstein, 2004).

3.3. Acquisition Data

Acquisition data are collected from multiple sources. We first use the standard Merger and Acquisition data from the Thomson Reuters SDC platinum. We extract all announced and completed M&As with complete information on acquirer, target, announcement and effective dates. We focus on only friendly acquisitions and when the majority of the target is acquired by the acquirer. The second data source of acquisition information is Thomson Reuters RecapIQ (now Cortellis Deals Intelligence). RecapIQ collects detailed information

[9]The raw Pharmaprojects data typically updates the firm name associated with each project when it is acquired. We therefore re-constructed the historical originator firm using text descriptions included in the dataset. More details are provided in Appendix B.

from company press release, SEC filings, and company voluntary disclosures on various types of alliances relationships in the biotechnology industry. For the purpose of our study, we keep only "acquisition" deals. The third data source of acquisitions is the SDC VentureXpert database covering mostly more early stage research labs and biotech startups, which provides complementary information to the SDC M&A and RecapIQ. We identify entrepreneurial companies that exited via an acquisition event as indicated in VentureXpert. Since VentureXpert does not provide details on the acquirer and dates of the acquisition, we conduct a manual collecting of those information to format the database consistently.

Armed with the original acquisitions compiled from multiple data sources, we conduct a multi-step cleaning process. We first standardize company (both acquirers and targets) names and collect demographic information for each company. Second, since a same firm could appear in different databases with slightly different names, we create a unique firm identifier by linking firms with close standardized names and demographic marks (such as location). Third, based on cleaned names of acquirers and targets and the deal dates, we drop duplicated acquisition events possibly due to overlapping of the datasets. To the best of our knowledge, this is the most comprehensive database on acquisitions in the pharmaceutical industry.[10]

We combine the acquisition database with the Pharmaprojects drug development data through a fuzzy matching algorithm and a large scale, manual check. We consider a drug project acquired if the originator firm is acquired. In the end, for each drug in our database, we are able to identify whether it went through any acquisition event through its development life cycle; if yes, the acquirer, the timing of acquisition, and development events pre- and post-acquisition.

[Insert FIGURE 3 Here.]

To provide some descriptive information on our sample, we plot the distribution of the number of new drugs originated by a company between 1989 and 2011 in Figure 3. We find 45% of companies originate only one drug over this period. Further, Table A1 provides a by-year tabulation of project coverage in our sample. Pharmaprojects provides stable

[10]Each of the three data sources, SDC M&A Database, RecapIQ, and VentureXpert, contributes at least 10% of cases in the final database.

coverage throughout our sample period, with around 1,000 new drug projects per year in the 1990s increasing to around 2,000 projects per year after 2007. On average, one third of drug projects were acquired at some point in their development. The acquisition rate is lower for drugs originated more recently, which is likely a result of right truncation. Acquisitions typically occur a few years into development and therefore might have not been realized by 2017 for more recent projects.

3.4. Coding the Continuation of Drug Development

To be consistent with the model proposition on the continuation of a project, we define "continuation" events using development milestone events extracted from Pharmaprojects. Pharmaprojects lists development milestones in twenty-eight categories, from as early as "new product," to as late as "first launch" of a product or reporting "suspended product."

We code these events into three categories: continuation events, discontinuation events, and neutral events that have little information regarding the progress of drug development, as listed in Table A2. In general, continuation events reflect research and development milestones (such as "Compounds Identified," "Mechanism Identified," "Target Identified") or efforts to commercialize the underlying drug project (such as "Additional Launches," "Additional Registrations," "New Licensees").

4. Main Analysis

4.1. Post-Acquisition Survival of Drug Development Projects

Our empirical analysis starts with univariate survival tests on drugs that went through an acquisition during the development process and those that did not. Specifically, we compare the discontinuation rates between acquired and non-acquired drugs, where discontinuation is measured as of June 2017. To ensure that we leave adequate room for acquisitions to happen (the average duration between drug origination and acquisition, if any, is about five years), we focus on drug projects originated before 2011.

[Insert TABLE 1 Here.]

The results are reported in Table 1. T-test of the sample means and the significance levels are reported. We find that the rate of discontinuation is significantly lower in the non-acquired sample (84.95%) than in the acquired sample (92.11%). To better control for the right-truncation problem of not observing the acquisition events for the later sample, we repeat the analysis using samples from earlier time periods, in particular, drugs originated pre-2006, and those originated pre-2000. We find similar patterns in both those two subsamples.

We then use a panel data of drug development to conduct regression tests on post-acquisition drug development progress. A drug is included in a sample from the origination year, and is removed after termination. The empirical specification is conducted as follows,

$$\begin{aligned} Continuation_{i,t} = \beta \cdot I(Acquired)_i \times I(Post)_{i,t} + \gamma \cdot I(Acquired)_i \\ + \alpha_{age} + \alpha_{vintage} + \varepsilon_{i,t}, \end{aligned} \tag{20}$$

where the dependent variable $Continuation_{i,t}$ is a dummy variable indicating whether drug i has an active continuation event in year t. $I(Acquired)_i$ indicates whether drug i undergoes an acquisition event, $I(Post)_{i,t}$ indicates whether the drug-year (i,t) observation is after the drug is acquired. We control for the potential effects of age and vintage (the year of origination) using fixed effects, and cluster standard errors at the drug level.

We report these results in Table 1. We separately report three subsamples: pre-2011 drugs in columns (1) and (2), pre-2006 drugs in columns (3) and (4), and pre-2000 drugs in columns (5) and (6). In column (1), we find that acquired drugs are 2.0% less likely to have an continuation event during the year post-acquisition. The unconditional probability of having a continuation event in the sample is 8.5%, leading the economic magnitude of the post-acquisition "killing" intensity to be 2.0%/8.5% = 23.5%. Reassuringly, the dummy variable $I(Acquired)$ does not carry any load in the regressions, meaning that the acquired drugs do not appear to have a different unconditional continuation probability.

In column (2) we incorporate drug-level fixed effects in the regression analysis. In this way, unobservable drug-project-specific characteristics are absorbed by these fixed effects. We find that the estimate of β is statistically significant and has similar economic magnitude as in column (1). Columns (3) to (6) suggest that the result produced using earlier subsamples,

guarding against the concern that the results are biased because of the right truncation of the panel. Overall, Table 1 means that on average, acquired drug development projects are less likely to be continued under the possession of the acquirer, consistent with the "killer acquisition" logic.

Overall, these simple univariate survival tests on post-acquisition performance provide evidence for the existence of killer acquisitions proposed in Proposition 1. That is, acquired drugs are less likely to be continued in the development process.

Note that this univariate analysis is a broader version of the existence test of killer acquisitions suggested by our theory. In fact, our model does not predict that continuation should be less likely for all acquisitions, but only for acquired projects that overlap with the acquirer's existing portfolio. In theory, the set of acquired drugs could instead predominantly contain synergistic acquisitions. We would then expect acquired projects to be more likely to be developed (or less likely to be killed) than independent projects. However, Table 1 suggests that killer acquisitions not only exist, but also that they are pervasive and the dominant force in shaping average development choices in our acquisition sample.

4.2. Overlap of Research Pipelines

The main implication of the theoretical framework of killer acquisition in Section 2 is that the motive to kill is driven by the extent to which the acquirer has overlapping drug development projects with the target. If the target project closely overlaps with projects and/or drugs marketed by acquirer, the acquirer is motivated to preempt the potential competition the target represents, and therefore to acquire and stop development of the project.

We measure overlap between a drug project and the acquiring firm based on the market and technology of the focal product. To categorize a drug project's "market", we use its therapeutic class, which is the disease or condition the therapy targets (e.g., antihypertensive). To categorize a drug project's "technology," we use its mechanism of action, which describes the biological interaction involved in the drug achieving its desired end, and which usually describes both the molecular target (e.g., beta adrenoreceptor, angiotensin I converting enzyme) and the intended effect (e.g., agonist, antagonist, reducer, inhibitor).

If the acquiring firm has an active project in the same market using the same technology as that of the acquired drug project, we consider that the project overlaps with the acquirer, and vice versa. We incorporate this dummy variable into the baseline specification to estimate whether the killer acquisitions are more likely to occur on drugs that tightly overlap with the acquirer's pipeline or not. We estimate the following model,

$$\begin{aligned} Continuation_{i,t} = \beta_O \cdot I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i + \beta \cdot I(Acquired)_i \times I(Post)_{i,t} \\ + \gamma_O \cdot I(Acquired)_i \times I(Overlap)_i + \gamma \cdot I(Acquired)_i \\ + \alpha_{age} + \alpha_{vintage} + \varepsilon_{i,t}. \end{aligned} \tag{21}$$

In this specification, the triple interaction term $I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i$ captures the extra continuation probability in acquisition cases when the target and the acquirer overlap in their development pipeline. The term $I(Acquired)_i \times I(Overlap)_i$ captures the overall development conditions for drugs acquired by overlapping buyers in years before the acquisition.

[Insert TABLE 2 Here.]

Table 2 presents the results. In column (1), the β coefficient is -0.017, confirming the lower continuation probability post-acquisition. More importantly, β_O estimate of -0.019 is also statistically significant, meaning that projects acquired by buyers that have an overlapping project are more than twice as likely to be discontinued in the development process ((0.017+0.019)/0.017 = 212%). The coefficient associated with $I(Acquired)_i \times I(Overlap)_i$ is positive and significant. One explanation for this is that incumbent firms are more likely to acquire those companies that show more positive promise (continuation), and they appear to have the ability to identify such targets.

Table 2 has an additional important implication. From our baseline results in Table 1 one may worry that the "killer acquisition" result could be due to buyer's inability to identify profitable projects and to integrate them internally. If this were the case, then we should expect the "killing" intensity to mitigate, rather than intensify, in the overlapping acquisition cases, because overlapping knowledge should at least partially resolve information asymmetries between the acquirer and the target.

4.3. Market Competition

We measure competition as the count of firms who are developing or currently market a drug that overlap with the target product. Specifically, these are firms with projects under development in the same market using the same technology (our measure of "pipeline" competition), or firms who have a launched product in the same market of the focal project using the same technology (our measure of "existing product" competition).[11]

[Insert TABLE 3 Here.]

Table 3 presents the regression results to examine the intensity of killer acquisitions under different competition environments. Drug development projects are categorized into high and low competition by the competition measures described above. In columns (1) to (4), the competition measure is calculated using existing launched products while inn columns (5) to (8) the measure is calculated using the acquirer's pipeline. The results suggest that the decreased continuation probability during the post-acquisition period for overlapping projects largely concentrates in product markets with relatively low competition. Indeed, we find little evidence that killer acquisitions are a big concern in high-competition subsamples.

4.4. Patent Expiry

To further explore how overlap relates to killing intensity, we tested how the time remaining on acquirer patents for the drugs that overlap with the target drugs conditions the results we see in Table 2. To do so, we identified patents linked to approved drugs (from FDA Orange Book data via Pharmaprojects) and merged in United States Patent and Trademark Office (USPTO) data on patent filing and timelines for the relevant acquirer firm patents. Following the logic of Proposition 3 which predicts that killing intensity declines as the acquirer's patent nears expiry when the expected remaining profits are relatively small, we expect the negative

[11]Note that each drug product can fall into multiple technologies (mechanisms of action) and multiple intended markets (therapeutic classes). In the PP dataset, drug projects have on average 1.3 mechanisms of action (median 1; 81% have 1) and on average 1.9 therapeutic classes (median 2; 46% have 1). In constructing our aggregate counts of competitors, we count each project in all possible technology-markets in which it falls. For our measures of competition for the focal projects, we use the market-technology with the most competition. That is, if a project falls into two market-technologies, one with 0 pipeline competitors and one with 5, we use 5.

relationship between overlap and continuation effects to be most pronounced among acquirers with patents that have a long remaining life. Table 4 presents the results among acquisitions with overlapping acquirers. Consistent with our predictions, we find that if the acquirer patents are near expiry (i.e., within 5 years), killing intensity is mitigated.

[Insert TABLE 4 Here.]

4.5. Clinical Trials

To supplement the preceding analyses on continuation events, we also examined the likelihood that a project continues in the clinical trials process. Specifically, following the literature (Guedj and Scharfstein, 2004; Krieger, 2017), we focus on whether drugs that start Phase I clinical trials and acquired are more or less likely to subsequently start Phase II trials. In this analysis, each observation is a drug project that initiated Phase II clinical trials. The key variable is $I(Acquired\,PI)$, which indicates whether the drug is acquired during the period of Phase I trials. $I(Overlap)$, as before, indicates whether the acquisition is made by an acquirer with projects in the same therapeutic market and with the same mechanism of action.

[Insert TABLE 5 Here.]

Table 5 presents regression results on the subsample for which we have information about Phase I start dates. As with earlier analyses, we limit the sample to those projects started before 2011 (and control for vintage) to ensure projects have time to enter Phase II (and sufficient time to observe an acquisition). We find that, compared to projects that aren't acquired in Phase I, those that are acquired are less likely to move forward into Phase II trials, and this relationship is stronger when the acquirer has overlapping projects. In terms of economic magnitude, in column (2), the decreased probability of -0.254 is 48.7% of the base rate of entering Phase II of 52.1%. Being acquired by an acquirer with overlapping products decreases this even further (coefficient is -0.144).

5. Alternative Explanations

Results thus far, though consistent with the killer acquisition interpretation, raise the concern that they could be mechanical or subject to alternative interpretations due to the simple empirical design and/or sample selection. In this section we attempt to sharpen the empirical analysis and investigate potential alternative explanations for our results.

5.1. Optimal Project Selection

One concern when trying to interpret the results as that acquirers "kill" acquired products for preemptive intentions is that the discontinuation of certain drug products may result from (optimal) selection criteria—for example, the acquirer firms could be targeting one of the several projects in the target firm and choose to continue only the one(s) that could generate the most value for the combined firm. This alternative story is difficult to test directly as we do not observe the potential strategic value that each of the target's projects could generate for the acquirer.

Our approach to investigating this concern is to examine only the deals with single-drug targets—that is, we try to identify the post-acquisition continuation probability only for the cases in which the target owns one and only one drug at the time of acquisition. If optimal project selection is driving our results, we should expect that our focal patterns are much less prevalent among single-project acquisitions.

[Insert TABLE 6 Here.]

We report the analysis in Table 6 column (1). We find the post-acquisition discontinuation probability is much higher in cases involving single-drug targets. The estimate, -0.035, almost doubles that for the full sample. This means that those targets are 3.5% less likely to receive a continuation event. This doubling of magnitude not only confirms that the identified results in Table 1 is unlikely due to the project selection and instead supports the "killing" hypothesis, but also suggests that those single-drug companies are more vulnerable to the threat of such preemptive competitive strategies implemented by incumbent competitors.

5.2. Organizational Frictions in Acquirers

Recent literature documents the effect of acquisition on the productivity of the combined firm (and the target as a division), and finds acquired divisions could be of lower productivity after the event due to the inefficient functioning of the internal organization of the larger acquirer (Seru, 2014). Relatedly, larger firms may be less willing to continue drug development than smaller firms (Guedj and Scharfstein, 2004). Under this line of economic reasoning, the post-acquisition discontinuation, or slow development in general of target technologies could be driven by the fact that an acquired entrepreneurial project (as compared to an non-acquired one) is now being managed by a more slow-moving organization facing organizational frictions in making investment decisions.

We assess the validity of this alternative interpretation by introducing fixed effects at the developer level (equivalently, the owner or acquirer level). To be clear, the acquired drug will be assigned to the acquirer after the acquisition event. Any productivity change or investment patterns that can be attributed to the organizational environment should be absorbed by these fixed effects, and the estimate of β can be interpreted net of the average influence from the developer.

Column (2) of Table 6 reports the results. We find that the point estimate, -0.108, is statistically significant and economically large. The size is much larger than in other specifications, meaning that after netting out the effect of the developer, the post-acquisition continuation becomes even less likely. This directional move of the point estimate means that fixed effects of the acquirers (typically larger firms) are typically positive, suggesting that larger pharmaceutical companies are in general better at developing than the smaller ones. This is not surprising given previous studies documenting the advantages of bigger drug firms in research, regulation, and commercialization-related resources. The bottom line is that the interpretation of our main finding does not seem to be affected by the organizational frictions in the acquiring firm.

5.3. Discontinuation Decision

In column (3) of Table 6, we conduct an additional test to investigate the discontinuation decision for a given drug. The rationale behind this check is to make sure that the results reported thus far are not driven by any reporting bias regarding drug development progress. For the dependent variables, we use a dummy variable indicating whether the drug is discontinued (see Table A2 for detailed definitions of such event). We find that the likelihood of termination is significantly higher in years post-acquisition.

5.4. Redeployment of Technologies

In order to more convincingly show that those innovative projects are terminated for competition preemption purposes, now we turn to address the possibility that technologies of terminated projects are redeployed by the acquirer firm.

When an acquired project is killed from the development process, there could be three different scenarios that follow: the technology could be shelved (in other words, hibernated), the technology could be redeployed in projects that are less competitive with the firm's other product, or the technology could be redeployed in a potentially better project in the original market. If it is the last case, then the termination of acquired product should not be interpreted as killing.

We assess whether and how the technologies of terminated projects are redeployed by exploiting molecule-level information for each project. Specifically, we collect information of the chemical structure underlying each drug project, and track whether acquirer firms initiate projects that incorporate acquired technologies using chemical similarities post-acquisition. If acquired drugs are indeed likely to be redeployed, one would expect new projects in acquirer firms to become more similar to the acquired project.

To measure chemical similarity, we follow the literature on the chemical informatics literature, in which the Tanimoto distance is the most commonly used method (Nikolova and Jaworska, 2003; Krieger, Li and Papanikolaou, 2017). The idea behind the calculation is to compute the proportion of chemical features shared by any two chemicals when divided by the union of the two. This similarity measure is bounded between 0 and 1, with 0 indicating

the pair share no common chemical fragments.

[Insert TABLE 7 Here.]

In Table 7 Panel A, we examine whether drugs initiated in the acquirer post-acquisition become more chemically similar to the acquired drug. If post-acquisition technology integration is pronounced, one would expect that drugs in acquirer firms to incorporate chemical components from the acquired technology and become similar to the acquired project. However, in our simple framework, we find that if anything, drugs developed in acquirer firms post the acquisition of a drug become less similar. The economic magnitude of -0.001 is indeed negligible compared to the global similarity mean of 13.3%. Overall, this does not support the view that technology redeployment is a prominent phenomenon which explains killer acquisitions.

5.5. Redeployment of Human Capital

By now, our analyses and interpretations have been focusing on the project or technology side of the acquisition. However, it could be the case that the key motivation behind these acquisitions are human capital such as the research team or other key individuals (Ouimet and Zarutskie, 2011). Under this view, the termination of acquired projects could be simply a by-product of acquiring and efficiently redeploying valuable human capital within the acquired company.

Before addressing this concern below, it is worth highlighting that the acquiring "for-team" motivation might not be as pervasive in the pharmaceutical industry as in other industries. The pharmaceutical industry is typically project-driven, and technological expertise may not be easily transferable to other projects (Gompers, Gornall, Kaplan and Strebulaev, 2016). As a result, acquiring a company solely for its human capital without continuing the project itself may not be a viably profitable strategy.

To measure the reallocation of human capital subsequent to acquisition events and any changes in inventor productivity associated with acquisition, we track inventor mobility using the Harvard Business School patent and inventor database. This database provides the names of the inventors (the individuals credited with producing a patent) and their affiliations with

the assignees, enabling us to track their mobility and patenting over time (see Lai, D'Amour and Fleming (2009) for details). We follow a similar approach to Bernstein (2015); Brav et al. (2017). Specifically, we construct a list of pre-acquisition inventors by identifying those who filed at one patent within the five-year window prior to the acquisition event. We then track the mobility and productivity of those inventors, analyzing how many of the inventors are retained in the acquiring firm and whether they are efficiently redeployed in the new firm.

Under the human capital acquisition view, a significant proportion of pre-acquisition inventors in the target firm should be retained and redeployed even after the projects are terminated. Moreover, since the acquirer firms intend to put the acquired human capital to use on more valuable projects, we should expect the inventors to become more productive in their new roles.

We show the analysis results in Table 7 Panel B. Only 22% of pre-acquisition inventors move to the acquirer after the acquisition while 78% for move to other firms. Those two sets of inventors are statistically comparable before the acquisition event, patenting for roughly 4.35 to 4.57 times for the target within the five years leading up to the acquisition. Post-acquisition, we find little evidence that the retained inventors became more productive in the combined firm. In fact, their average patenting quantity drops by 30% from 4.57 to 3.16 patents in five years. In contrast, regarding inventors who move to other firms, the productivity drop is milder ($< 10\%$).

One limitation of this analysis is that it is difficult to link each patent to a specific drug project for those early-stage projects.[12] As a result, it is difficult to accurately assign each inventor to the specific drug project that she or he is involved in. As a result, we are not able to identify whether the leaving or staying inventors are from projects that are eventually killed. In untabulated results where we focus on cases with a single-drug target, we find that a even larger proportion of investors leave the combined firm after the acquisition.

[12]That information is typically disclosed late in drug development stage when FDA requires systematic reporting.

6. Discussion

6.1. Antitrust and FTC Review Thresholds

In principle, the killer acquisition phenomenon is detrimental to market competition and should be scrutinized by the Federal Trade Commission (FTC). However, as shown in our paper, many such acquisitions are made when the technology or project is still at a nascent stage and thus are exempted from the pre-merger review rule of the FTC under the "Hart-Scott-Rodino (HSR) Antitrust Improvements Act." Under HSR, deals under $50 million (annually adjusted) do not need to submit filings for pre-acquisition review. For deals between $50 million and $200 million (annually adjusted), the size-of-the-person test is conducted, and if the larger party has lower than $100 million in assets or sales and the smaller party has lower than $10 million in assets, the deal does not need to be reviewed by the FTC. Since the size-of-the-person test is typically not satisfied for smaller pharmaceutical companies, effectively acquisitions below $200 million will typically not be investigated. Wollmann (2018) shows that these review exemptions can result in stealth consolidation: anticompetitive acquisitions whose small size enables them to escape regulatory scrutiny but whose cumulative effect is large.

Do acquirers conducting killer acquisitions attempt to avoid FTC review by making acquisition deals that do not trigger FTC reporting requirements under HSR? We answer this question by examining acquisitions around the HSR threshold and comparing the project development decisions of the above and below-threshold deals. If firms perform killer acquisitions intentionally under the radar of the FTC, we should expect to see, first, a bunching of acquisition deals just below the threshold and second, a higher killing rate (and lower launching rate) in the below-threshold deals.

[Insert TABLE 8 Here.]

In Table 8 we implement this analysis. We collect the acquisitions that are right below the FTC review threshold $[-10\%, 0]$ and those just above that $[0, 10\%]$. First, we find higher number of deals just below the threshold than just above the threshold (70% higher). Second, the survival rate of below-threshold deals is lower than those right above the threshold.

Similarly, we find the launching rate is much lower (1.8% versus 9.1%) and the discontinuation rate is much higher (94.6% versus 83.3%). While this analysis is simple and purely descriptive, overall these patterns are consistent with acquirers conducting more killer acquisitions when they can expect to avoid FTC review.

6.2. Ex-ante Innovation Incentives and Welfare

Our theoretical and empirical analysis focuses on the acquisition and project development incentives of incumbents and entrepreneurs. In our setting, killer acquisitions have an unambiguously negative effect on welfare even though the entrepreneur is indifferent (due to his lack of bargaining power) and the acquiring incumbent (and other incumbents) are strictly better off when acquisitions are allowed. Consumers are hurt both by the lack of competition and the elimination of innovative new products. Killer acquisitions benefit incumbents, leave entrepreneurs indifferent, but disproportionately hurt consumers.

A comprehensive welfare analysis of the impact of killer acquisitions is, however, more difficult given the many different forces involved in the innovation process. It is possible that the presence of an acquisition channel also has a positive effect on welfare that is not accounted for in our analysis. In particular, the prospect of entrepreneurial exit through acquisition (by an incumbent) may spur ex-ante innovation as in Phillips and Zhdanov (2013). Whereas in our model entrepreneurs are born with a project and thus do not have to exert effort to come up with an idea, it is plausible that the prospect of later acquisition may motivate the origination of entrepreneurial ideas in the first place. However, it is important to note that killer acquisitions will only spur such idea origination if the entrepreneur receives some of the surplus that accrues to the incumbent through the acquisition.[13] If the entrepreneur is left with no surplus relative to standalone value of his project he will be unaffected by acquisitions and hence will not respond by increasing his innovation efforts. If killer acquisitions do increase ex-ante innovation, this potential welfare gain will have to be weighed against the ex-post efficiency loss due to reduced competition. Whether the former positive or the latter negative effect dominates will depend on the elasticity of the entrepreneur's innovation

[13]For a model along these lines see Phillips and Zhdanov (2013) who show that increased takeover activity spurs innovation by small firms because this allows them to capture a larger share of the benefits of innovation.

response.

Furthermore, acquisitions may not only influence the intensity of entrepreneurial project generation, but they may also affect its direction. If entrepreneurs can choose between originating projects that overlap with existing products or those that do not, increased takeover activity and killer acquisitions by incumbents may spur innovation of very similar 'me-too' drugs at the expense of the origination of truly novel products (Arcidiacono et al., 2013). This response to the prospect of acquisitions would add to the negative welfare impact of killer acquisitions.[14]

6.3. Frequency and Importance of Killer Acquisitions

Our empirical estimates document large and significant effects of acquisitions that overlap with acquirers' existing product portfolios on project continuation rates. Our findings on differential project continuation rates also allow us to roughly calculate the pervasiveness of killer acquisitions as well as their impact on industry-wide development decisions.

In particular, we documented that when an acquired project overlaps with a product in the acquirer's existing product portfolio the project is less likely to be continued: acquired projects with overlap (25.5% of acquired projects) continue at a rate of 5.8% while acquired projects without overlap (74.5% of acquired projects) continue development at a rate of 6.8%. Given the reduction in continuation rate, it is natural to ask how many of these acquisitions of overlapping projects are purely killer acquisitions. To roughly calculate this number assume that there are two types of acquisitions that fall into the acquired with overlap category: killer acquisitions which are purely intended to shut down future competitors (and thus have a continuation rate of 0%) and acquisitions that have the same continuation rate as acquisitions without overlap (6.8%). Based on these numbers we estimate that 7.1% $(= (1 - \frac{0.058}{0.076}) \times 0.255)$ of all acquisitions or about 54 $(= 0.071 \times 758)$ acquisitions every year are killer acquisitions. Given that our back-of-the-envelope calculation assumes that killer acquisitions lead to immediate termination and that there are no additional synergies in the development of overlapping drugs this is a lower bound on the actual number of killer

[14] Rasmusen (1988) considers a theoretical model in this vein in which entrants can blackmail the incumbent by threatening to keep prices low, and buyout can make entry profitable which otherwise would not be.

acquisitions.

Having quantified the approximate frequency of killer acquisitions it is natural to ask what this means in terms of innovation and antitrust policy (i.e., how overall development rates in the pharmaceutical industry would be affected if antitrust policy directly targeted such killer acquisitions). The average continuation rate in our sample is 7.5%. Consider first the case in which acquisitions of overlapping projects are no longer allowed and that all such projects instead have the same continuation rate (8.5%) as non-acquired projects (56% of all projects). In that case, the number of total drug projects for which development continues, would increase by 5.3% ($=\frac{0.085-0.049}{0.0754} \times (1-0.561) \times 0.255$) or by about 7 ($= 0.0754 \times 0.0534 \times 1727$) drug projects per year.

To put these results in context, we can compare them to policies that have attempted to encourage innovation in the pharmaceutical industry. One such policy—which is considered highly successful, but also involved high costs—is the Orphan Drug Act (ODA). The ODA gives firms substantial tax incentives to undertake clinical trials (up to 30 million USD per trial), grants, and extended market exclusivity for drugs targeted at conditions with relatively small patient pools (i.e., "orphan" diseases). There are several hundred such diseases, including many cancers. Economic analysis by Yin (2008, 2009) suggests that the ODA accounted for roughly 25 additional clinical trials per year over the period 1981 to 1994, with the effect attenuating over time. Roughly, then, eliminating killer acquisitions would result in innovation effects that are, at a lower bound, larger than a quarter of the size of the Orphan Drug Act.

7. Conclusion

This article demonstrates that incumbent firms have incentives to acquire innovative targets and terminate their innovative projects in order to preempt future competition. Empirically, we exploit the setting of drug development, in which we are able to track project development independent of acquisition deals. We show that acquired drug projects are less likely to be continued in the development process, particularly when the acquired project overlaps with the acquirer's pipeline and when the acquirer has stronger incentives to protect

his market power. We also show that alternative interpretations such as optimal project selection, organizational frictions, and the intent to redeploy human capital or technologies do not explain our results.

We want to add a few concluding remarks to link our findings to broader economic phenomena and trends. First, while acquisitions are the major outlet of startup exit and are becoming even more popular as an exit strategy over time,[15] and even though technology acquisitions can offer opportunities for synergy and gains from trade, acquisitions may also have potentially destructive consequences. In other words, as opposed to interpreting the acquisition of nascent technologies as incumbents' effort to incorporate entrepreneurial innovation and maximize joint surplus, a significant driver fueling this trend may be killer acquisitions and creator destruction (i.e., killing the threat of creative destruction).

Second, we broaden antitrust research beyond focusing on existing market competition to include acquisitions aimed at eliminating future competition by preempting the development of future innovations. If incumbent firms use killer acquisitions to preempt competitive entrants before they enter the market, market competition will be harmed. Our results on the killer acquisition phenomenon around the FTC review thresholds, which highlights the fact that the phenomenon is more prevalent for acquisitions that are too small to scrutinize, exacerbates this concern.

Third, our findings suggest that the Schumpeterian creative destruction process—whereby startups inventions can topple entrenched and less innovative incumbents—may be smaller than previously documented. That is, we see lower rates of innovation not only because incumbents hesitate to innovate, but also because incumbent firms with market power acquire innovators to terminate competition and as a consequence inhibit technological progress.

[15]For example, TechCrunch documents that more than 95% of VC-backed startup exits are through acquisitions rather than IPOs: https://techcrunch.com/2017/01/31/cb-insights-3358-tech-exits-in-2016-unicorn-births-down-68/.

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Figure 1. Strategy Payoff

This graph plots the incumbent's payoff from pursuing one of the three acquisition strategies "Don't Acquire" (light gray), "Acquire to Kill" (black), and "Acquire to Continue" (dark gray) as a function of ρ. Other parameter values are held constant ($\alpha = 100$, $\beta = 4$, $\gamma = 3$, $k = 20$, $L = 20$, $\sigma = -20$, and $n = 1$).

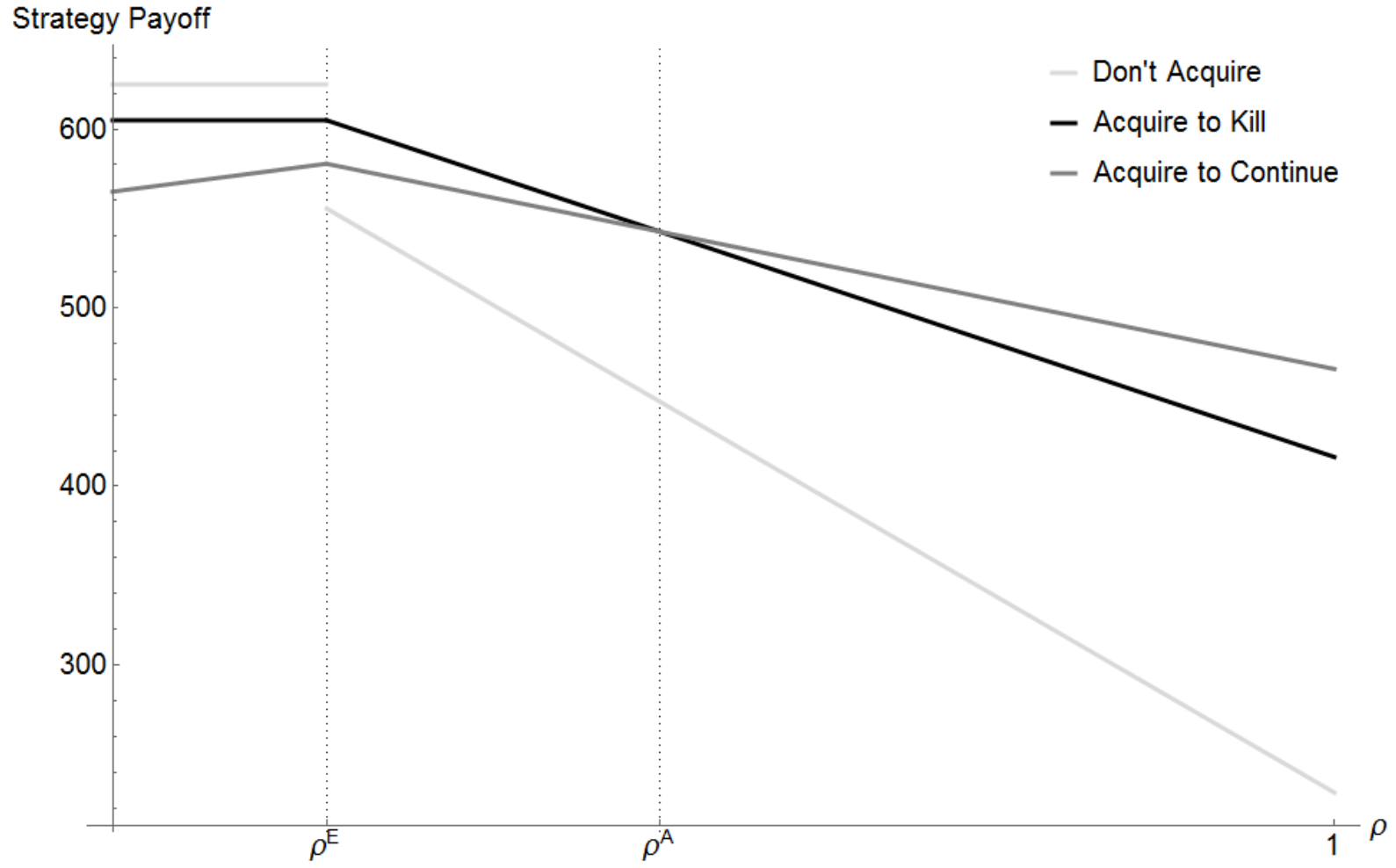
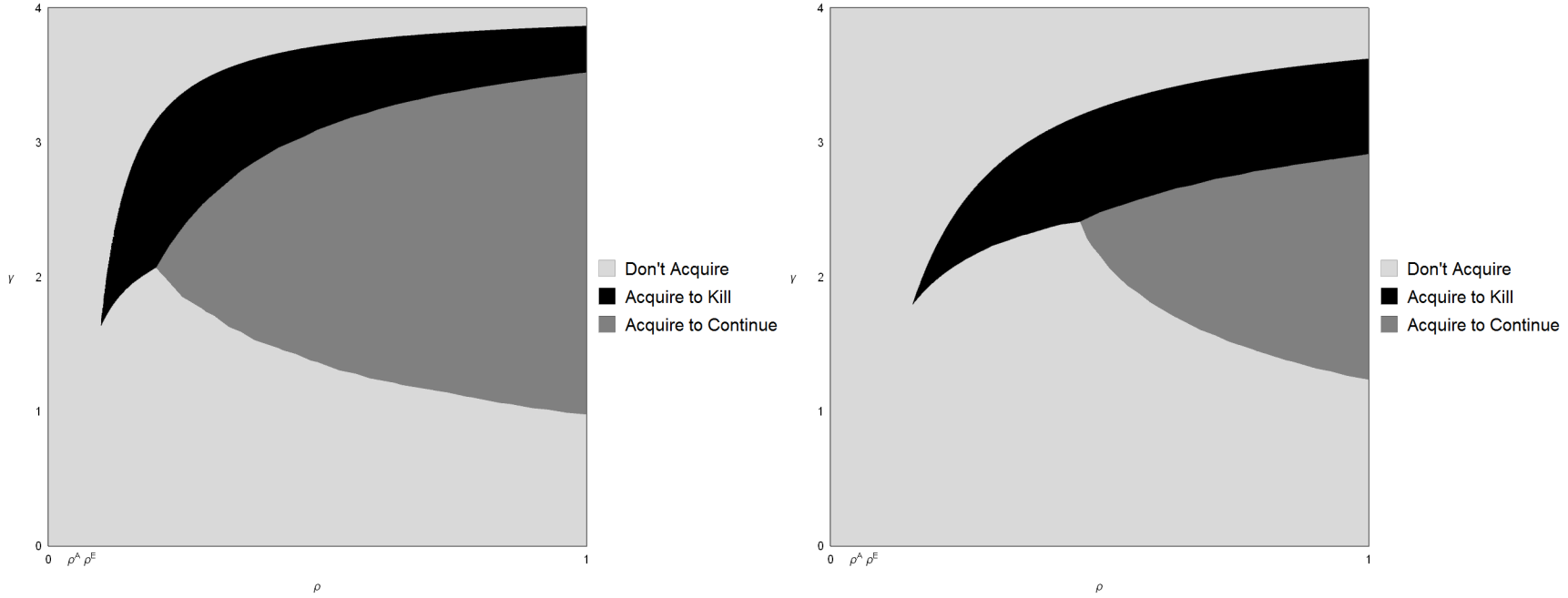


Figure 2. Optimal Acquisition Strategies

Dominance regions of the three acquisition strategies for different combinations of ρ and γ for $n = 1$ and $n = 2$. Other parameter values are held constant ($\alpha = 100$, $\beta = 4$, $k = 20$, $L = 20$, and $\sigma = -20$).



(a) $n = 1$ **(b)** $n = 2$

Figure 3. Firm Size (No. of New Drugs Originated) Distribution

This graph plots the distribution of the number of new drugs originated by a company between 1989 and 2011. We assign a drug to a company if the company was the first to own the drug development project, but not the ones that are obtained through acquisitions. The drug origination data are from the Pharmaprojects database.

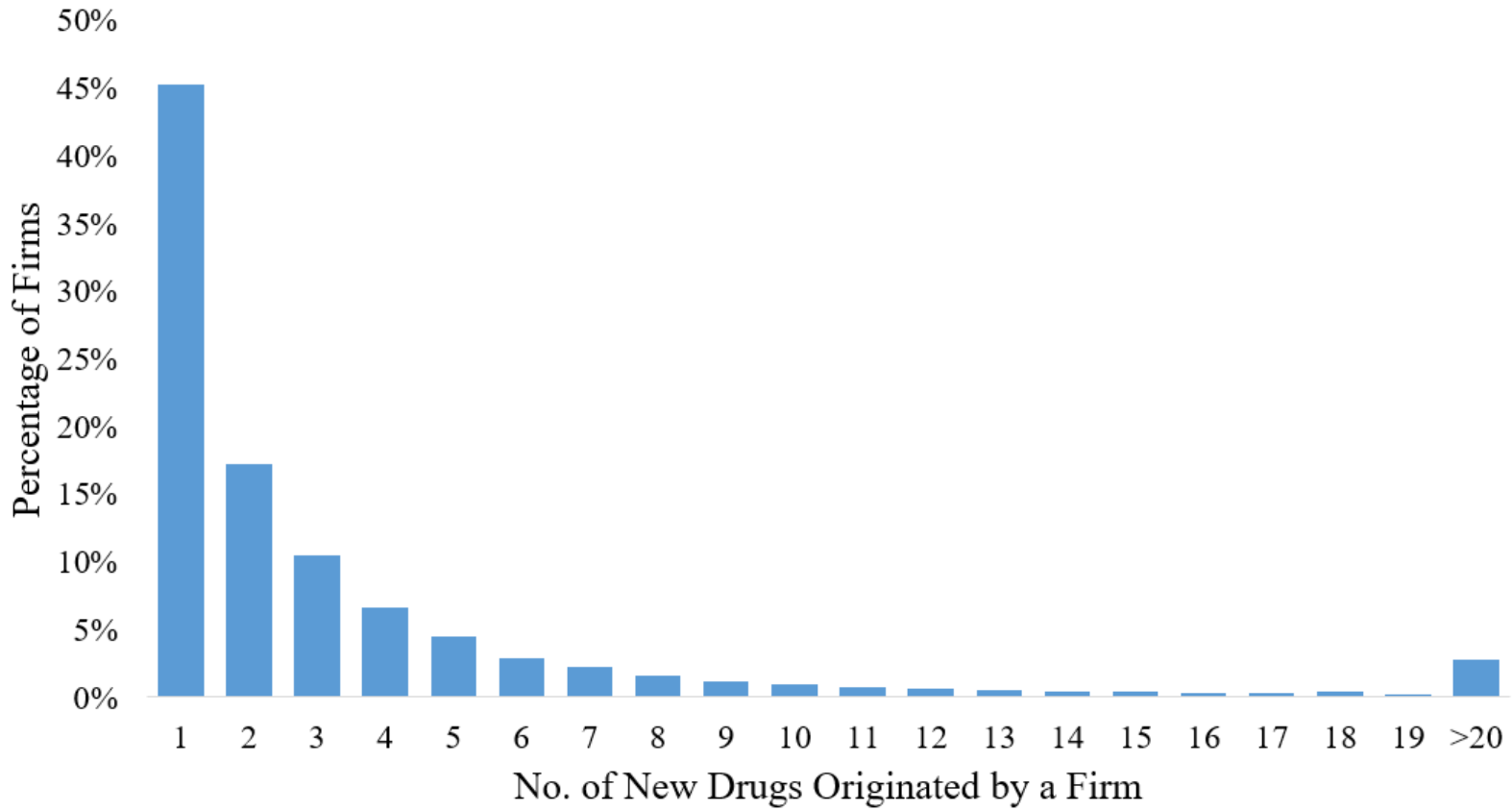


Table 1
Acquisition and Project Continuation

Panel A: Univariate Analysis

This table presents univariate survival tests on the drugs that went through an acquisition during the development process and those that do not. Specifically, we examine the rates of discontinuation among those acquired drugs and those non-acquired ones, where those development status are as of June 2017. To ensure that we leave adequate room for acquisitions to happen, we focus on drug projects originated before 2011, originated before 2006, and originated before 2000. We report the discontinuation rate for the non-acquired drug sample, the acquired sample, and the difference between the two samples. T-test of the sample means and the significance levels are reported. ***, **, and * indicate significance at the 1%, 5%, and 10% levels, respectively.

	Non-acquired	Acquired	Diff	T-statistics	Stat Significance
			Originated before 2011		
Discontinued	86.80%	92.13%	-5.33%	-13.40	***
N	27,140	8,728			
			Originated before 2006		
Discontinued	90.11%	93.14%	-3.03%	-7.80	***
N	17,508	8,728			
			Originated before 2000		
Discontinued	92.00%	93.71%	-1.71%	-3.83	***
N	9,270	5,404			

Panel B: Acquisitions and Project Continuation: Baseline Regression Results

This table presents the post-acquisition continuation rates of drug projects using a drug-year panel sample. The empirical specification uses the following model,

$$Continuation_{i,t} = \beta \cdot I(Acquired)_i \times I(Post)_{i,t} + \gamma \cdot I(Acquired)_i + \alpha_{age} + \alpha_{vintage} + \varepsilon_{i,t},$$

where the dependent variable $Continuation_{i,t}$ is a dummy variable indicating whether drug i has an active continuation event in year t. $I(Acquired)_i$ indicates whether drug i undergoes an acquisition event, $I(Post)_{i,t}$ indicates whether the drug-year (i,t) observation is after the drug is acquired. We separately report the three subsamples of pre-2011 drugs in columns (1) and (2), pre-2006 drugs in columns (3) and (4), and pre-2000 drugs in columns (5) and (6). In columns (1), (3), and (5), we control for age and vintage (the year of origination) fixed effects; in columns (2), (4), and (6) we control for age and drug fixed effects. The t-statistics based on standard errors clustered at the drug project level are displayed in parentheses. ***, **, and * indicate significance at the 1%, 5%, and 10% levels, respectively.

	Continuation Event = 1					
	(1)	(2)	(3)	(4)	(5)	(6)
	Originated before 2011		Originated before 2006		Originated before 2000	
I(Acquired) × I(Post)	-0.020***	-0.016***	-0.024***	-0.017***	-0.017***	-0.019***
	(-7.080)	(-5.011)	(-7.511)	(-5.111)	(-4.566)	(-4.652)
I(Acquired)	-0.002		-0.004		-0.003	
	(-0.908)		(-1.565)		(-1.090)	
Observations	311,501	311,501	211,444	211,444	127,910	127,910
R-squared	0.018	0.243	0.016	0.244	0.009	0.237
Project FE	No	Yes	No	Yes	No	Yes
Age FE	Yes	Yes	Yes	Yes	Yes	Yes
Vintage FE	Yes	No	Yes	No	Yes	No

Table 2
Acquisitions, Product Overlap, and Project Continuation

This table presents the post-acquisition continuation rates of drug projects using a drug-year panel sample. The empirical specification uses the following model,

$$\begin{aligned} Continuation_{i,t} = {} & \beta_O \cdot I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i + \beta \cdot I(Acquired)_i \times I(Post)_{i,t} \\ & + \gamma_O \cdot I(Acquired)_i \times I(Overlap)_i + \gamma \cdot I(Acquired)_i \\ & + \alpha_{age} + \alpha_{vintage} + \varepsilon_{i,t}. \end{aligned}$$

where the dependent variable $Continuation_{i,t}$ is a dummy variable indicating whether drug i has an active continuation event in year t. $I(Acquired)_i$ indicates whether drug i undergoes an acquisition event, $I(Post)_{i,t}$ indicates whether the drug-year (i,t) observation is after the drug is acquired. $I(Overlap)$ is a dummy variable indicating whether the acquired drug overlaps with the pipeline of the acquirer. In columns (1), (3), and (5), we control for age and vintage (the year of origination) fixed effects; in columns (2), (4), and (6), we control for age and drug fixed effects. The t-statistics based on standard errors clustered at the drug project level are displayed in parentheses. ***, **, and * indicate significance at the 1%, 5%, and 10% levels, respectively.

	Continuation Event = 1					
	(1)	(2)	(3)	(4)	(5)	(6)
	Originated before 2011		Originated before 2006		Originated before 2000	
I(Acquired) × I(Post) × Overlap	-0.019***	-0.013*	-0.019***	-0.007	-0.030***	-0.017*
	(-2.894)	(-1.747)	(-2.652)	(-0.929)	(-3.508)	(-1.791)
I(Acquired) × I(Post)	-0.017***	-0.013***	-0.020***	-0.016***	-0.013***	-0.016***
	(-5.239)	(-3.684)	(-5.845)	(-4.165)	(-3.050)	(-3.471)
I(Acquired) × Overlap	-0.001		-0.000		-0.000	
	(-0.178)		(-0.078)		(-0.061)	
I(Acquired)	-0.002		-0.004		-0.003	
	(-0.720)		(-1.373)		(-0.955)	
Observations	311,501	311,501	211,444	211,444	127,910	127,910
R-squared	0.018	0.243	0.016	0.244	0.009	0.237
Project FE	No	Yes	No	Yes	No	Yes
Age FE	Yes	Yes	Yes	Yes	Yes	Yes
Vintage FE	Yes	No	Yes	No	Yes	No

Table 3
Acquisitions and Project Continuation: Market Competition

This table presents the post-acquisition continuation rates of drug projects using a drug-year panel sample. The empirical specification uses the following model,

$$Continuation_{i,t} = \beta \cdot I(Acquired)_i \times I(Post)_{i,t} + \gamma \cdot I(Acquired)_i + \alpha_{age} + \alpha_{vintage} + \varepsilon_{i,t},$$

where the dependent variable $Continuation_{i,t}$ is a dummy variable indicating whether drug i has an active continuation event in year t. $I(Acquired)_i$ indicates whether drug i undergoes an acquisition event, $I(Post)_{i,t}$ indicates whether the drug-year (i,t) observation is after the drug is acquired. Drug development projects are categorized into terciles—high, medium, and low competition—by the competition measures described above. We count the number of firms with a drug or drug project that is in the same technology-market as the focal product. In columns (1) to (4) the competition measure is calculated using existing launched products while in columns (5) to (8) the measure is calculated using the pipeline. In columns (1), (3), (5) and (7), we control for age and vintage (the year of origination) fixed effects; in columns (2), (4), (6) and (8), we control for age and drug fixed effects. The t-statistics based on standard errors clustered at the drug project level are displayed in parentheses. ***, **, and * indicate significance at the 1%, 5%, and 10% levels, respectively.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Continuation Event = 1							
	Low Competition		High Competition		Low Competition		High Competition	
I(Acquired) × I(Post) × Overlap	-0.021***	-0.018**	-0.002	0.027	-0.030***	-0.016	-0.013	-0.012
	(-3.156)	(-2.288)	(-0.118)	(1.254)	(-3.082)	(-1.396)	(-1.549)	(-1.257)
I(Acquired) × I(Post)	-0.016***	-0.013***	-0.023*	-0.014	-0.016***	-0.016***	-0.017***	-0.004
	(-5.349)	(-3.557)	(-1.826)	(-0.892)	(-3.853)	(-3.405)	(-3.760)	(-0.717)
Competition Measure	Existing Product				Pipeline			
Project FE	No	Yes	No	Yes	No	Yes	No	Yes
Age FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Originating Year FE	Yes	No	Yes	No	Yes	No	Yes	No

Table 4
Acquisitions and Project Continuation: Patent Life Among Overlaps

This table presents the differing post-acquisition continuation rates of drug projects using a drug-year panel sample. The sample for this analysis is acquired projects where the acquirer has overlap with the target firm. The analysis looks at how remaining patent term length conditions effect of acquisition on continuation rates. The empirical specification uses the following model,

$$\begin{aligned} Continuation_{i,t} = \beta_O \cdot I(Post)_{i,t} + \beta \cdot I(NearPatExpiry)_i \\ + \gamma_O \cdot I(NearPatExpiry)_i \times I(Post)_{i,t} \\ + \alpha_{age} + \alpha_{vintage} + \varepsilon_{i,t}. \end{aligned}$$

where the dependent variable $Continuation_{i,t}$ is a dummy variable indicating whether drug i has an active continuation event in year t. $I(Post)_{i,t}$ indicates whether the drug-year (i,t) observation is after the drug is acquired. $I(NeatPatExpire)$ is a dummy variable indicating whether the overlapping acquirer drug is within 5 years of patent expiry. We control for age and vintage (the year of origination) fixed effects and age fixed. Column (2) also includes acquiror firm FE. The t-statistics based on standard errors clustered at the drug project level are displayed in parentheses. ***, **, and * indicate significance at the 1%, 5%, and 10% levels, respectively.

	(1)	(2)
	Continuation Event = 1	
I(Post) × I(Near Patent Expiry)	0.045	0.068*
	(-1.640)	(-1.674)
I(Near Patent Expiry)	-0.079***	-0.045***
	(-3.235)	(-2.151)
I(Post)	-0.089***	-0.055***
	(-3.650)	(-2.633)
Observations	3,216	3,216
R-squared	0.041	0.152
Age FE	Yes	Yes
Originating Year FE	Yes	Yes
Acquiror FE	No	Yes

Table 5
Acquisitions and Project Continuation: Clinical Trials

This table presents the differing continuation rates of drug projects. The sample for this analysis is drugs that entered Phase I trials (for which we have detailed trial data). The analysis looks at the effect of acquisition, and overlap with the acquirer, on the likelihood the project enters Phase II trials. The empirical specification uses the following model,

$$PhaseII_i = \beta \cdot I(Acquired\ PI)_i + \gamma_O \cdot I(Acquired\ PI)_i \times I(Overlap)_i + \alpha_{vintage} + \varepsilon_i.$$

where the dependent variable $PhaseII_i$ is a dummy variable indicating whether drug i enters Phase II. $I(Acquired\ PI)_i$ indicates whether the drug (i) is acquired in Phase I. $I(Overlap)$ is a dummy variable indicating whether the acquired drug overlaps with the pipeline of the acquirer. In Column (2) we control for vintage (the year of origination) fixed effects. ***, **, and * indicate significance at the 1%, 5%, and 10% levels, respectively.

	(1)	(2)	(3)	(4)	(5)	(6)
	Phase II = 1		Phase II = 1		Phase II = 1	
			Low Competition	High Competition	Low Competition	High Competition
I(Acquired PI) × Overlap		-0.144***	-0.146**	-0.046	-0.185*	-0.062
		(-8.253)	(-1.989)	(-0.327)	(-1.678)	(-0.770)
I(Acquired PI)	-0.254***	-0.226***	-0.220***	-0.242***	-0.222***	-0.238***
	(-10.223)	(-8.253)	(-7.129)	(-2.519)	(-5.749)	(-5.298)
Competition Measure			Existing Product		Pipeline	
Observations	4,171	4,171	3,146	436	1,938	1,644
R-squared	0.077	0.079	0.069	0.229	0.083	0.243
Vintage FE	Yes	Yes	Yes	Yes	Yes	Yes
Phase Start Year FE	Yes	Yes	Yes	Yes	Yes	Yes

Table 6
Empirical Explorations on Alternative Interpretations

This table presents the post-acquisition continuation rates of drug projects using a drug-year panel sample. The empirical specification uses the following model,

$$Continuation_{i,t} = \beta \cdot I(Acquired)_i \times I(Post)_{i,t} + \gamma \cdot I(Acquired)_i + \alpha_{age} + \alpha_{vintage} + \varepsilon_{i,t},$$

where the dependent variable $Continuation_{i,t}$ is a dummy variable indicating whether drug i has an active continuation event in year t. $I(Acquired)_i$ indicates whether drug i undergoes an acquisition event, $I(Post)_{i,t}$ indicates whether the drug-year (i,t) observation is after the drug is acquired. We use pre-2011 drugs in all regressions. In column (1) the acquisition sample is restricted to cases where the target has only one drug. In column (2) we control for developer FE to account for the unobservable developer quality. In column (3) the dependent variable is the dummy variable indicating the termination event of a drug. In all regression we control for age and drug fixed effects. The t-statistics based on standard errors clustered at the drug project level are displayed in parentheses. ***, **, and * indicate significance at the 1%, 5%, and 10% levels, respectively.

	(1) Single-Drug Company	(2) Control Developer FE	(3) Termination Events
I(Acquired) × I(Post)	-0.035*	-0.108***	0.007***
	(-1.842)	(-11.216)	(4.695)
Constant	0.097***	0.156***	0.014***
	(985.373)	(84.372)	(113.305)
Observations	201,161	248,564	248,564
R-squared	0.248	0.084	0.305
Project FE	Yes	Yes	Yes
Age FE	Yes	Yes	Yes

Table 7
Acquisitions and Assets Redeployment

Panel A: Acquisitions and Project Similarities to Acquired Drugs

This table studies chemical similarities of drug projects between acquired drugs and drugs originated by the acquirer firm. Each observation in the sample is a drug-pair between an acquired drug and a drug from the acquirer originated within the five-year windows around the acquisition event. The key independent variable, $I(Post)$, indicates whether the acquirer drug was initiated after the acquisition event, and takes value one if so. To measure chemical similarity we use the Tanimoto distance (Nikolova and Jaworska, 2003; Krieger, Li and Papanikolaou, 2017). In column (1), we do not control for fixed effects; in column (2), we control for acquirer firm fixed effects; in column (3), we control for case-specific fixed effects. The t-statistics based on standard errors clustered at the drug project level are displayed in parentheses. ***, **, and * indicate significance at the 1%, 5%, and 10% levels, respectively.

	(1)	(2)	(3)
	Chemical Similarity		
I(Post)	-0.001*	-0.001	-0.002***
	(-1.673)	(-1.274)	(-4.208)
Constant	0.133***	0.132***	0.133***
	(94.840)	(94.827)	(576.005)
Observations	154,896	154,896	154,896
R-squared	0.000	0.013	0.361
Acquiror FE	No	Yes	No
Case FE	No	No	Yes

Panel B: Inventor Productivity (Number of New Patents) Within Five-year Window

This table presents inventor mobility and productivity around acquisition events of drug projects. We construct a list of pre-acquisition inventors by identifying those who filed at one patent within the five-year window prior to the acquisition event from the HBS inventor database. We show the number of new patent applications in the five-year window before the acquisition and the five-year window after the acquisition, for subsamples of inventors who moved to the acquirer and those who moved to other firms. T-test for subsample differences, and ***, **, and * indicate significance at the 1%, 5%, and 10% levels, respectively.

	Before Acquisition	After Acquisition	Difference
Those Who Move to Acquiror After Acquisition (22%)	4.572	3.160	-1.412***
Those Who Move to Other Firms After Acquisition (78%)	4.357	4.089	-0.267*
Difference	-0.215	0.929***	1.144***

Table 8
The Intensity of Project Discontinuation around FTC Review Threshold

This table presents univariate survival tests on the drugs that are acquired just below $[-10\%, 0]$ and just above $[0, 10\%]$ the FTC review threshold. Specifically, we examine the rates of being active, being discontinued, being fully launched, where those development status are as of June 2017. To ensure that we leave adequate room for acquisitions to happen, we focus on drug projects originated before 2011. We report the rate of being active, being discontinued, and being fully launched separately for the two samples, and the difference between them. T-test of the sample means and the significance levels are reported. ***, **, and * indicate significance at the 1%, 5%, and 10% levels, respectively.

	10% Below Threshold	10% Above Threshold	Diff	T-statistics	Stat Significance
Active	3.57%	7.58%	-4.00%	-1.175713	
Launched	1.79%	9.09%	-7.31%	-2.292933	**
Discontinued	94.64%	83.33%	11.31%	2.509447	**
N	112	66			

Appendix (Not For Publication)

A. Omitted Proofs

A.1. Bertrand Competition

In this section, we present the proofs of the main model of Bertrand competition that are omitted from the main text.

A.1.1. Product Market Overlap.

Proof of Proposition 1. From inequalities (10) and (11) it immediately follows that an incumbent firm acquires a project continues development if $\rho \geq \rho^A$ and that an independent entrepreneur continues if $\rho \geq \rho^E$. Equation (12) shows that the thresholds ρ^E and rho^A are identical if and only if $\Delta^E = \Delta^A$. Thus, it remains to show that for any positive product market overlap $\beta > \gamma > 0$, we have $\Delta^E > \Delta^A$ and hence $\rho^E < \rho^A$.

Recall $\Delta^E \equiv \pi^E_{\neg acq,S} - \pi^E_{\neg acq,F}$ and $\Delta^A \equiv \pi^A_{acq,S} - \pi^A_{acq,F}$. It is immediately apparent that for $\rho = 0$ and $\rho = \beta$ we have $\Delta^E = \Delta^A$. Rewriting the inequality $\Delta^E > \Delta^A$ to solve for γ and β establishes that $\beta > \gamma > 0$ is necessary and sufficient for this inequality to hold. □

A.1.2. Competition.

Proof of Proposition 2. Proposition 1 establishes that $\rho^A - \rho^E > 0$ for any $\beta > \gamma > 0$ and that the difference $\rho^A - \rho^E > 0$ is inversely related to the difference $\Delta^A - \Delta^E < 0$. Thus, it remains to show that the difference $\Delta^E - \Delta^A$ is decreasing in n. Straightforward differentiation of $\Delta^E - \Delta^A$ with respect to n establishes this result. Furthermore, we have $\lim_{n\to\infty}(\Delta^E - \Delta^A) = 0$. □

A.1.3. Patent Life and Future Competition.

Proof of Proposition 3. Due to Bertrand competition profits of the incumbent drop to zero after T^A years. Thus, his development gain until then is $T^A\Delta^A$. The entrepreneur's development gain over that time span is $T^A\Delta^E$.

Denote the development gains for the entrepreneur and the acquirer in the presence of undifferentiated generic competition after the expiry of the acquirer's existing product's patent in T^A years by $\Delta_{gen} = \Delta^E_{gen} = \Delta^A_{gen}$. These (equal) development gains are different from the previous expressions Δ^E and Δ^A. This is because when a generic product (that is undifferentiated from the acquirer's existing product) enters it not only drives profits of that product to zero, but due to its low price it also reduces the profits of the other products that are differentiated from it. Thereafter, the profits for the acquirer's existing product drop to 0 and hence his incentives to develop coincide with those of the entrepreneur.

Thus, the continuation decisions of the entrepreneur d^E_{gen} and the acquiring incumbent d^A_{gen} are given by inequalities (13) and (14). The proposition straightforwardly from these inequalities. □

A.2. Cournot Competition

Consider the same setting as in our main model, but assume that firms compete in quantities in the competition stage in $t = 2$.

If the entrepreneur remains independent in $t = 0$ the payoffs in $t = 2$ are

$$\begin{aligned}
\pi^E_{\neg acq,F} &= 0 \\
\pi^A_{\neg acq,F} &= \frac{\beta\alpha^2}{(2\beta + \gamma(n-1))^2} \\
\pi^E_{\neg acq,S} &= \frac{\beta\alpha^2}{(2\beta + \gamma n)^2} \\
\pi^A_{\neg acq,S} &= \frac{\beta\alpha^2}{(2\beta + \gamma n)^2}
\end{aligned}$$

If the incumbent acquires the entrepreneur in $t = 0$ the payoffs in $t = 2$ are

$$\begin{aligned}
\pi^E_{acq,F} &= \frac{\beta\alpha^2}{(2\beta + \gamma(n-1))^2} \\
\pi^A_{acq,S} &= \frac{(2\beta - \gamma)^2(\beta + \gamma)\alpha^2}{2(2\beta^2 + \beta\gamma n - \gamma^2)^2}
\end{aligned}$$

Defining Δ^E and Δ^A with these new payoffs establishes all the same results as in our main model.

B. Cleaning Pharmaprojects Data

In this section, we describe the process involved in cleaning the Pharmaprojects data for analysis. To begin, we extracted all available projects (as of June 1, 2017) from the Pharmaprojects database of 62,500 projects in total.

Our first challenge in using Pharmaprojects data for our analyses was that all projects initiated prior to 2012 were subject to possible updating of the "originator" field that contains the firm associated with the project. For example, if the project was acquired, the acquiring firm is typically erroneously listed as the "originator" of the project. We therefore needed to re-construct the original "originator" firm in such cases. To do so, we used two additional fields in the dataset: the "overview" field which often includes the name of the original firm associated with the project in case of acquisitions, and the "latest change" field which also would often contain details of acquisition events, including the associated firm names.

To extract the original "originator" firm from these fields, we used regular expressions and phrases such as "X acquired by Y" or "developed by X". Employing Stata, we algorithmically created a list of original originators and the acquiring firms, and checked these flags against our M&A datasets from SDC and Recap IQ.

Once we had a dependable measure of the true originator firms, our second challenge in using Pharmaprojects was to standardize originator firm names for matching with other datasets, including M&A events. Aided by the Stata program "stnd_compname" (Wasi and Flaaen 2014), we isolate the stem name for each originator firm associated with each project in Pharmaprojects.

C. Merging Drug Development and Acquisition Data with Patent Databases

In this section, we describe the process to merge drug development and acquisition data with USPTO patent databases, through matching company names with assignee names in the USPTO patent database. To minimize potential problems introduced by the minor discrepancy between different versions of the USPTO database, we use both NBER and Harvard Business School (HBS) patent databases to provide patent assignee information. After this step, each company in the drug development and acquisition database will have its original name, standardized name and a stem name; similar for USPTO assignees.

C.1. Name Standardization

We begin by standardizing company names in the drug development and acquisition database (drug data hereafter) and assignee names from NBER and HBS patent database, using the name standardization algorithm developed by the NBER Patent Data Project. This algorithm standardizes common company prefixes and suffixes, strips names of punctuation and capitalization; it also isolates a company's stem name (the main body of the company name) excluding these prefixes and suffixes.

C.2. The Matching Procedure

With these standardized and stem company (assignee) names and demographic information provided by both the drug data and the USPTO, we merge the databases following the matching procedures below:

1. Each standardized drug originator and owner name is matched with standardized names from the NBER data and HBS data.
   (a) If an exact match is identified, we consider this as a "*successful match*." The company is removed from the set of names waiting to be matched on both sides.
   (b) Otherwise, next step.

2. Each stem drug originator and owner name is matched with stem names from the NBER data and HBS data.

   (a) If an exact match of stem names if identified, and the two companies are located in the same city and state OR the two comapnies are located in the same state and the earliest patenting year in NBER and HBS databases is later than the founding year in the drug data, we consider this as a "*successful match*." The company is removed from the set of names waiting to be matched on both sides.

   (b) If an exact match of stem names is identified, but the two companies do not satisfy the location and chronology criterions above, we consider this as a "*potential match*." The company is moved to a pool of firms waiting for manual checks.

   (c) Otherwise, next step.

3. For the remaining companies, each stem originator and owner name is matched with up to 3 close stem names from the USPTO data using a fuzzy-matching method based on the Levenshtein edit distance.[16] The criterion is based on the length of the strings and the Levenshtein distance, and the threshold is determined through a random sampling procedure.

   (a) If the fuzzy-matched pair is located in the same city and state OR the two comapnies are located in the same state and the earliest patenting year in NBER and HBS databases is later than the founding year in the drug data, I consider this as a "*potential match*."

   (b) Otherwise, the companies are categorized as "*failed to match*."

4. The "*potential matches*" set identified in the procedures above are reviewed by hand, incorporating information from both data sources, including full patent abstracts, and company business descriptions.

   (a) Pairs confirmed as successful matches through the manual check are moved to the "*successful match*" set.

[16] The Levenshtein edit distance measures the degree of proximity between two strings, and corresponds to the number of substitutions, deletions or insertions needed to transform one string into the other one (and vice versa).

D. Additional Results

Table A1
Drug Development Projects Originated by Year

This table provides descriptive statistics on number of drugs originated by year, between 1989 and 2011. New drug projects are identified from the Pharmaprojects database. Percentage of drugs that were acquired is constructed by augmenting the Pharmaprojects data with acquisition information collected from SDC M&A database, RecapIQ, and VentureXpert.

Year	# New Drug Originations	% Acquired
1989	638	38.87%
1990	776	37.63%
1991	892	38.68%
1992	1,061	41.28%
1993	1,111	42.30%
1994	854	43.56%
1995	1,036	34.85%
1996	1,030	34.95%
1997	1,066	33.40%
1998	1,159	32.96%
1999	1,041	30.74%
2000	1,000	31.30%
2001	1,273	30.87%
2002	1,285	26.07%
2003	1,437	25.47%
2004	1,691	19.40%
2005	1,455	18.42%
2006	1,353	16.04%
2007	2,244	11.45%
2008	2,278	9.70%
2009	2,144	6.86%
2010	1,914	6.53%
2011	2,396	5.43%

Table A2
Definition of Drug Development Continuation

This table presents a list of events recorded in Pharmaprojects to track the development process of each drug. The events are listed in the alphabetical order. Each of those events are coded into one of the three categories, the continuation events, the dis-continuation events, as well as the neutral events that have little information regarding the progress on the drug development (denoted as "–" in the table).

Events	**Development Continuation Event?**
Additional Launches	Yes
Additional Registrations	Yes
Change in Disease Status	–
Change in Global Status	–
Change in Licensee Status	–
Compounds Identified	Yes
Development Continuing	Yes
Discontinued Products	No
First Launches	Yes
First Registrations	–
Global Status Reversion	–
Licences Discontinued	–
Licensing Opportunities	–
Mechanism Identified	Yes
Names Granted	Yes
New Chemical Structure	Yes
New Disease	Yes
New Licensees	Yes
New Patent Applications	Yes
New Product	–
New Therapeutic Activity	Yes
No Development Reported	–
Novel Target Reported	Yes
Orphan Drug Status Granted	Yes
Registration Submissions	–
Suspended Products	No
Target Identified	Yes
Withdrawn Products	No