Killer Acquisitions*

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This article demonstrates that incumbent firms 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 illustrate the phenomenon using a model with product market competition, innovation, and endogenous acquisition decisions. In our model incumbent firms have incentives to acquire innovative targets and then terminate the targets' project development when those products have strong replacement effects. This killer acquisition motive is stronger when the acquirer-target product overlap is high and when product market competition is low. Empirically, we exploit the setting of drug development, in which we are able to track detailed project-level development histories of more than 55,000 drug projects. We show 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 show that alternative interpretations such as optimal project selection, organizational frictions, and human capital redeployment do not explain our results. Our findings have 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 term such acquisitions "killer acquisitions" as they are intended to kill potentially promising, yet competing, innovation.

A recent case involving the pharmaceutical firm Mallinckrodt and its subsidiary Questcor illustrates the killer acquisition phenomenon. In the early 2000s, Questcor enjoyed a monopoly in the category of adrenocorticotropic hormone (ACTH) drugs with its product Acthar. Acthar treats infantile spasms (a rare but serious condition) and nephrotic syndrome (a kidney disorder) along with a few other rare conditions. In the mid-2000s, development began on a direct competitor to Acthar, a synthetic version named Synacthen. In 2013, in an effort to forestall future competition, Questcor acquired the US development rights of Synacthen. Following the logic of killer acquisitions Questcor did not pursue the development of Synacthen. In fact, it 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 January 2017, Mallinckrodt (which acquired Questcor in 2014) settled the case of an anti-competitive acquisition case, agreeing to pay $100 million.

In this paper, we model and empirically demonstrate this phenomenon. Our analysis proceeds in two steps. First, to motivate the empirical analysis, we formalize the concept of a

[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

killer acquisition using a model that combines product market competition, innovation, and endogenous acquisition decisions. In our model, an incumbent firm that acquires a startup (target) with an innovative project has weaker incentives to continue the project's development than a non-acquired entrant would. The key insight is that while both types of firms benefit from successful development, the profit will be lower for incumbent acquirers because they suffer cannibalization of their existing product portfolio. This is Arrow's replacement effect (Arrow, 1962). As a result, incumbent firms acquire startups either to realize synergies between the two firms (i.e., synergistic acquisitions) or to prevent startup from developing products that, if successful, would cannibalize the incumbent's profits (i.e., killer acquisitions).

The model also yields a rich set of predictions about the conditions under which killer acquisitions are more likely to occur. Because the replacement effect is larger when the acquirer-target product overlap is high, incumbents have stronger incentives to discontinue project development. Therefore, killer acquisitions are more likely when product overlap is high. Additionally, higher product market competition already erodes the incumbents' profits and reduces the negative impact of the replacement effect when project development is successful. As a result, product market competition diminishes 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." In addition, we expect killer acquisitions to be more frequent when the target project overlaps with the acquirer's innovation pipelines and when the competition level in the related product market is low.

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 subsequent to the acquisition. Further, we need be able to accurately characterize the potential product market overlap between the acquirer and the target project as well as competition in the related product market.

This article overcomes these empirical challenges by focusing on the pharmaceutical industry and exploiting the setting of drug development. We collect detailed development information on more than 55,000 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 launch of the research to the end point of the project (either successfully launched or discontinued). The key advantage of this setting is that it tracks project development independent of the 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 therapeutic market) and the technology (the mechanism of action) of each drug project, and therefore we can observe the degree to which an acquirer overlaps with the project. This allows us to characterize competition in both the technology space and product market of the project, and to examine products under development as well as already launched products. The relative accuracy of using existing, detailed pharmaceutical categorizations to measure overlap and

competition is particularly desirable given the complications associated with coarse industry coding systems and with the wide variations in products (Hoberg and Phillips, 2010).

Armed with this database, a simple cross-sectional comparison of survival 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 that had longer, more complete life-cycle development records.

Our baseline regression exploits a drug-year panel setting and characterizes 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 and also achieves fewer development milestones. The empirical specification controls for age and vintage (year of project origination) fixed effects. Reassuringly, the continuation probability of the acquired drugs is not statistically distinguishable from non-acquired drugs in years prior to the acquisition, and the divergence of "death rate" starts only after the event. Overall, killer acquisitions dominate the acquisition sample and lead to a disproportionate rate of innovation discontinuation events for projects acquired from targets.

To further support the interpretation of killer acquisitions, we test the model prediction that terminations of acquired projects are more pervasive when the target's new project could compete within the acquirer's existing markets. Product market overlap between the target and the acquirer is captured by whether the drug targets one of the therapeutic classes for which the acquirer is developing or has developed a project. We show that killer acquisitions

happen more (doubling the intensity) when the acquired drug overlaps with the acquirer's pipeline.

Acquired project terminations are also more pervasive when the acquirer has more monopolistic power in the market 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 competition levels. Competition levels are measured using the number of firms with competing projects, either launched in the product market or in pipelines. We find that killer acquisitions mostly concentrate in areas with low levels of product market competition.

We conduct several refinements of the baseline analysis to sharpen the interpretation that acquirers intentionally kill targets' projects. One potential explanation of the baseline finding is the optimal project selection view. In particular, the acquirer could strategically and optimally choose to continue the more promising or complementary projects of the target but discontinue others. That is, acquirer firms may kill the target projects that are tangential to the goal of the acquisition, and, if so, our project-level analysis might be misclassifying these as intentional kills. To assess this concern, we repeat our analysis in acquisitions of single-drug companies, where the acquirer cannot possibly 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 frictions from the buyer side could also confound our baseline interpretation. Previous research shows that complex organization structures in larger firms are detrimental to the development of innovation projects (Seru, 2014). These frictions 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 becomes even larger. Hence, on average, resourceful acquirers develop faster. The killer acquisition phenomenon actually becomes more pronounced when taking firm-level variation into account.

Another plausible alternative explanation of the result, as opposed to "acquiring to kill," is that the human capital behind the technologies (i.e. the research teams), rather than drug projects themselves drive pharmaceutical acquisitions. In this view, project discontinuation could be a strategically optimal step to further integrate and more efficiently redeploy acquired human capital. We test for this alternative story by collecting detailed information on inventor mobility and productivity around the acquisition events. 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. These results are inconsistent with explanations based on such acquisitions serving as a way of recruiting and retaining human capital.

All told, our paper highlights why and when firms conduct killer acquisitions to prevent future competition. First, we build a formal model of acquisition and project development choice. Then, we empirically demonstrate the phenomenon by exploiting a novel setting of drug development. Section 2 discusses related literature; Section 3 outlines our theoretical framework and develops testable hypotheses; Section 4 describes data and institutional background; Section 5 presents our main empirical results; Section 6 discusses interpretation

and robustness; and Section 7 offers concluding remarks.

2. Related Literature

2.1. Mergers and Acquisitions

The existing literature in corporate finance and industrial organization highlights three distinct motives for acquisition. First, acquisitions may driven by agency conflicts between managers and shareholders. A long and rich literature dating back to at least Roll (1986) and Morck et al. (1990) documents that in the absence of appropriate corporate governance mechanisms and incentive design managerial interests that diverge from shareholder interests lead to potentially value-destroying acquisitions. Second, acquisitions are driven by the pursuit of synergies between the acquirer and the target, Healy et al. (1992) and Andrade et al. (2001) document increases in industry-adjusted cash flows following mergers while Maksimovic and Phillips (2001) provide evidence for increases in productivity after mergers that are related to demand shocks and acquirer skill. Rhodes-Kropf and Robinson (2008) model asset complementarity and synergies as a motive for mergers. Bena and Li (2014) and Hoberg and Phillips (2010) document evidence of synergies post merger, showing that there are increases in cash flows, new products and patents post merger that are related to ex ante similarity of acquirer and target. Hoberg and Phillips (2017) demonstrate that firms performing mergers and acquisitions in markets with high product integration difficulty experience lower ex post profitability, higher ex post expenses, and a higher propensity to divest assets. 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.[2]

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

2.2. Market For Technologies

Further, this article is related to the innovation literature on technological acquisitions in the market for technology (Gans and Stern, 2003; Arora and Gambardella, 2010; Arora et al., 2014). Similar to the M&A literature, such research typically assumes that transactions are made for synergistic reasons (i.e. gains from trade) and that related experience to the

[2]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).

technology being evaluated for acquisition (i.e. having an internal technology in that space) enables better absorption of the acquired technology and therefore increases the likelihood of successful acquisition and innovation outcomes.

One relevant and ongoing debate in this literature focuses on markets for lemons in technology, i.e. that externally bought technologies are less likely to become successful innovations than those based on internally developed technologies because licensees sell lemons. Supporting this idea, Pisano (1997) finds that internally developed technologies are more likely to succeed that those licensed in. However, Arora, Gambardella, Magazzini, and Pammolli (2009) find that licensed drugs projects are drawn from same distribution as the ones that the licensor firm kept for itself. A "killer acquisition" story reconciles these seemingly opposing results: acquired projects aren't worse ex ante than those that are not acquired, but are more likely to be discontinued than those originating in the acquiring firm, and are therefore *purposefully* less likely to succeed.

The markets for technology literature also provides insight into the conditions under which a startup firm would want to sell their technology to incumbents versus trying to compete 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) and increase the likelihood that startups will sell their ideas rather than try to compete (Gans et al., 2002). The pharmaceutical industry is characterized by both of these features, which highlights why the industry is characterized by acquisition outcomes for startups, and further whykiller acquisitions would be particularly feasible in our setting.

3. Theoretical Framework

In this section we propose a simple theoretical model of product market competition, innovation, and acquisition decisions which we use to investigate the project development choices of entrepreneurial companies and incumbent firms.

3.1. 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), and one of $n \geq 1$ incumbent firms which each already possess an existing and potentially overlapping product, decides whether to acquire the new firm at a takeover price P where P will be endogenously determined by the model.

In $t = 1$, the owner of the project—the incumbent I 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 $t = 0$
- $\neg acq$, the originating firm was not acquired in $t = 0$

Finally, in $t = 2$, uncertainty about the success of the project is resolved and all the firms

engage in differentiated Cournot product market competition. We assume that if the project is successful at $t = 2$, 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 in this model as we assume that the values of π, ρ, k, and L are commonly known in $t = 0$.

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

In $t = 2$, if the project is successful the newly developed product faces product market competition from n other existing products with linear inverse demand for each product i given by $p_i = A - bq_i - a\sum_{j\neq i}^{n} q_j$ and symmetric constant marginal cost c. Product homogeneity is captured by a where $0 \leq a \leq b$. For both the entrepreneurial company and the acquiring incumbent firm we compute the profit when the new project is successful and when it is not successful (or has been terminated in $t = 1$).

Consider first the product market choices of an entrepreneur that is not acquired in $t = 0$. If the project is successful (S), the resulting newly developed product competes against n other single-product incumbent firms including the potential acquiring firm which chose not to acquire in $t = 0$. The entrepreneur's objective function is equal to

$$max_{q_E}(p_E - c)q_E \tag{1}$$

The resulting familiar first order condition is

$$A - 2bq_E - a\sum_{i\neq E}^{n+1} q_i = 0 \tag{2}$$

and solving for the symmetric equilibrium of $n+1$ single-product firms yields

$$\pi^E_{\neg acq,S} = \frac{b(A-c)^2}{(2b+an)^2} = \pi^I_{\neg acq,S} \tag{3}$$

Note that the product market profit for the entrepreneur and the n incumbent firms is 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

$$\pi^I_{\neg acq,F} = \frac{b(A-c)^2}{(2b+a(n-1))^2} \tag{4}$$

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

$$max_{q_1,q_2}(p_1-c)q_1 + (p_2-c)q_2 \tag{5}$$

while the remaining $n-1$ other single-product firms maximize single-product profits. Given our symmetry assumptions, in equilibrium, $q_1^* = q_2^* = q^*$ and $q_i^* = q_\neg^*$ for any $i \neq 1,2$, and

thus the resulting first order conditions can be rewritten as

$$A - c - a(n-1)q^*_{\neg} - 2(a+b)q^* = 0 \tag{6}$$

$$A - c - [2b + a(n-2)]q^*_{\neg} - 2aq^* = 0 \tag{7}$$

The resulting profit is

$$\pi^I_{acq,S} = \frac{(2b-a)^2(a+b)(A-c)^2}{2(2b^2+abn-a^2)^2} \tag{8}$$

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

$$\pi^I_{acq,F} = \frac{b(A-c)^2}{(2b+a(n-1))^2} \tag{9}$$

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

$$\pi^I_{acq,S} \geq \pi^I_{acq,F} = \pi^I_{\neg acq,F} \geq \pi^I_{\neg acq,S} = \pi^E_{\neg acq,S} > \pi^E_{\neg acq,F} = 0. \tag{10}$$

Note that the inequalities are strict if $a > 0$. The product market profits gained by the incumbent are always at least as large as those of the entrepreneur. This is because the incumbent 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 producing less aggressively thus resulting in profit $\pi^I_{acq,S}$. 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 $\pi^I_{acq,F}$ while a successful entrepreneur would face n competitors and gain only $\pi^E_{\neg acq,S}$.

3.2.1. The "Replacement Effect". However, what matters for the development decision in $t = 1$ are the difference between $\pi^I_{acq,S}$ and $\pi^I_{acq,F}$ 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

$$\Delta^E \equiv \pi^E_{\neg acq,S} - \pi^E_{\neg acq,F} \geq \pi^I_{acq,S} - \pi^I_{acq,F} \equiv \Delta^I \tag{11}$$

which holds with strict inequality if $a > 0$ and with equality if $a = 0$.

This is a fairly general result with a simple, well-known intuition. As long as product differentiation is not so large that products are completely segmented ($a = 0$) an incumbent gains strictly less from introducing a new product than an entrepreneur would. This is because the new product cannibalizes some of the profits of the existing product that the incumbent already owns whereas an entrepreneur has no product to sell and hence no profit ($\pi^E_{\neg acq,F} = 0$) if she does not successfully develop the project. This is Arrow's famous "replacement effect" (or "cannibalization effect") (Arrow, 1962). When $a = 0$ 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.

3.3. Continuation Decision ($t = 1$)

Next we investigate the development continuation decision in $t = 1$. The entrepreneur and the incumbent 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 a in the product market competition in $t = 2$. In contrast, an entrepreneurial company's portfolio would consist, 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

$$\rho(\pi^E_{\neg acq,S} - \pi^E_{\neg acq,F}) - k \geq L \tag{12}$$

An incumbent gains $\pi^I_{acq,S}$ from successful development of the project, but also foregoes the profit $\pi^I_{acq,F}$ it 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 $d^I = 1$ if

$$\rho(\pi^I_{acq,S} - \pi^I_{acq,F}) - k \geq L \tag{13}$$

Rewriting the two continuation decisions given by (12) and (13) shows the different success probability thresholds used by the entrepreneurial and incumbent firms above which the firms continue development. We denote these thresholds by ρ^*_E and ρ^*_I and they are given by

$$\rho^E = \frac{L + k}{\pi^E_{\neg acq,S} - \pi^E_{\neg acq,F}}, \quad \rho^I = \frac{L + k}{\pi^I_{acq,S} - \pi^I_{acq,F}} \tag{14}$$

Comparison of these thresholds shows that $\rho^E < \rho^I$ if and only if $a > 0$ which immediately

yields our first prediction because in that case $\Delta^E > \Delta^I$ as discussed above.

Proposition 1 (Project Killing)**.** *For any positive product market overlap $a > 0$, an incumbent firm that acquires a project is less likely to continue development than an independent entrepreneur. For $\rho < \rho^E$, incumbent and entrepreneur choose to terminate the project, $d^I = d^E = 0$. For $\rho^E \leq \rho < \rho^I$, the incumbent terminates the project, $d^I = 0$, while the entrepreneur continues $d^E = 1$. For $\rho^I \leq \rho$, both continue the project, $d^I = d^E = 1$.*

Product market overlap reduces the propensity to continue development. The more similar (as captured by a) the drug project of the entrepreneurial company is to the acquiring incumbent's existing product portfolio the larger is the loss from cannibalization. The difference in continuation behavior between incumbent and entrepreneur occurs when ρ is in the intermediate range between ρ^E and ρ^I. This region grows in size the larger is the product market overlap a, but it decreases the larger is n. The latter effect is due to existing competition already competing away some of the profit that would cannibalized by the introduction of a new product.

Proposition 2 (Market Overlap and Competition)**.** *The intermediate range between ρ^E and ρ^I grows in size the larger is the product market overlap a and the smaller is the number of competitors n.*

The propensity to terminate development due to product market overlap also means that only the most promising projects will remain in development when the entrepreneurial company is acquired by an incumbent as long as there some product market overlap. Holding final product market profits and development costs fixed, a comparison of (12) and (13)

implies that independent entrepreneurs that continue with a project, will do so, on average, at lower success probabilities ρ. This generates the next proposition.

Proposition 3 (Conditional Success Rate)**.** *Conditional on continuing development a project acquired by an incumbent is more likely to successfully result in a final product than a project by an non-acquired entrepreneur. This difference in eventual success probability is increasing in product market homogeneity a and decreasing in the number of existing competitors n.*

3.4. Acquisition Decision ($t = 0$)

In $t = 0$, one of the n incumbents decides whether or not to acquire the entrepreneur. Acquiring an entrepreneurial company yields an acquirer-specific payoff σ for the incumbent. 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 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 incumbent itself or by the entrepreneurial company if it remains independent.

Assume that the degree of product homogeneity a and the net synergies σ are known at $t = 0$. Thus, the incumbent decides to acquire at a takeover price P if

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

where $d^i \in \{0,1\}$ for $i = \{E, I\}$ is the continuation decision for the project taken by the firm in $t = 1$ described by inequalities (12) and (13).

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 (12). 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 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^I 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, $d^I = d^E = 0$ and thus the decision rule whether or not to acquire given by (15) reduces to

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

Second, for $\rho^E \leq \rho < \rho^I$, the incumbent terminates the acquired project, $d^I = 0$, while

the entrepreneur continues $d^E = 1$ and thus the entrepreneur is acquired if

$$\sigma + \underbrace{\rho(\pi^I_{acq,F} - \pi^I_{\neg acq,S})}_{\text{prevent cannibalization}} \geq \underbrace{(\rho\Delta^E - k - L)}_{\text{valuation difference}} \tag{18}$$

If the incumbent acquires the entrepreneur's project (acq) and shuts it down, the incumbent only competes against $n-1$ other firms thus earning a profit equal to $\pi^I_{acq,F}$. 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 $\pi^I_{\neg acq,S}$. Note further that 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 incumbent ($d^I = 0$) would obtain, thus leading to a difference in valuation.

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

$$\sigma + \underbrace{\rho(\pi^I_{acq,S} - \pi^I_{acq,F})}_{\text{soften cannibalization}} \geq \underbrace{\rho(\Delta_E - \Delta_I)}_{\text{valuation difference}} \tag{19}$$

where $\pi^I_{acq,S} - \pi^I_{acq,F}$ is the gain from having an additional product and using multi-product pricing to soften the impact of cannibalization. The difference in valuation for the product between the entrepreneur and incumbent is again driven by the replacement effect ($\Delta_E \geq \Delta_I$).

[Insert FIGURE 1 Here.]

The three regions can be seen in Figure 1 which plots the payoff to the incumbent of each of the three possible strategies as a function of ρ for a particular set of parameter values. For sufficiently low values of $\rho < \rho^E$ it is optimal not to acquire the entrepreneurial company

because buying it would be too costly in terms of integration costs ($\sigma < 0$) and even if no acquisition occurs the entrepreneur will kill the project anyway in $t = 1$. At $\rho = \rho^E$ the payoff for "Don't Acquire" (light gray) discontinuously drops by $\rho(\pi^I_{acq,F} - \pi^I_{\neg acq,S})$ because of the cannibalization effect. As a result, in the intermediate region $\rho^E \leq \rho < \rho^I$ the incumbent's optimal strategy is "Acquire to Kill" (black). Acquiring the project prevents the entrepreneur from potentially destroying some of the incumbent's profits, but because of the replacement effect it is not sufficiently profitable for the incumbent to continue with the project. Finally, if the project is sufficiently likely to succeed $\rho \geq \rho^I$ the incumbent's optimal strategy is "Acquire to Continue" (dark gray). The incumbent thereby prevents aggressive cannibalization by the entrepreneur because even though the incumbent continues the project he softens competition through multi-product pricing. Note that the parameters used in Figure 1 are such that acquiring the entrepreneur (to kill or to continue the project) is optimal whenever $\rho \geq \rho^E$. However, for other parameter values it is possible that the purchase price P is sufficiently high that "Don't Acquire" is optimal even for $\rho \geq \rho^E$ as we explain in greater detail below.

More precisely, 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, and differences in project valuation between originating and acquiring firms. We consider these three effects in turn.

First, acquiring the originating firm at price P always yields synergies or integration costs σ. As discussed before these net synergies can be either positive or negative and thus either increase or decrease 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 a aforementioned profit loss of $\pi^I_{acq,F} - \pi^I_{\neg acq,S}$ which results when the entrepreneur successfully develops the project with probability ρ. This profit loss due to entry of the entrepreneur and the resulting cannibalization of the profits of the existing product(s) in the market is equal to

$$\pi^I_{acq,F} - \pi^I_{\neg acq,S} = \frac{b(A-c)^2}{(2b + a(n-1))^2} - \frac{b(A-c)^2}{(2b+an)^2} \tag{20}$$

Straightforward inspection of this equation shows that this profit loss is equal to 0 if $a = 0$ and increasing in a. This is because cannibalization of the incumbent's profit is larger if the entrepreneurial company's product is more similar. Cannibalization is largest if the products are completely undifferentiated $a = b$. Furthermore, if $a > 0$ this profit loss is decreasing in the number of firms n. This is because when competition is already intense an additional product does not reduce the profits of existing products by much. Those profits are already competed away by the existing competition and therefore the incumbent has a lower incentive to acquire the entrepreneur.

Third, because the entrepreneur and the incumbent value the project differently and an acquiring incumbent must compensate the entrepreneur with an acquisition price P there is a third effect. 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. In the intermediate region ($\rho^E \leq \rho < \rho^I$) it occurs because the incumbent terminates the project, $d^I = 0$ while the entrepreneur would continue $d^E = 1$ and would reap an expected profit

equal to $\rho\Delta^E - k$ which is more than the incumbent's liquidation value L. In the high region ($\rho^I \leq \rho$) this project valuation effect is less negative, but it still occurs because even though both firms continue development the non-acquired firm reaps a larger net benefit $\Delta^E \geq \Delta^I$ due to a lack of self-cannibalization.

The "project valuation" and the "cannibalization" effects work in opposite directions: the project valuation effect is negative and the cannibalization effect is positive if and only if $a > 0$. It is straightforward to show that the latter dominates if competition is low (n is small). These insights combine to yield our next proposition.

Proposition 4 (Acquisition for Synergy and for Termination)**.** *An incumbent with larger synergies net of integration costs σ and higher product market overlap a is more likely to acquire the entrepreneurial company. The effect of product market overlap on acquisition propensity is largest if competition is low (n is small).*

[Insert FIGURE 2a and 2a Here.]

Figures 2a and 2b plot the regions in which "Don't Acquire" (light gray), "Acquire to Kill" (black) or "Acquire to Continue" (dark gray) are optimal for the incumbent for different combinations of the project's success probability ρ and the degree of product market overlap a between the entrepreneur's and the incumbent's product holding the other model parameters fixed at the same values as in Figure 1.

In Figure 2a the acquiring incumbent is a monopolist ($n = 1$) and thus "Acquire to Kill" is optimal when product market overlap a is high and the project's success probability ρ is high. In such a situation cannibalization by the entrepreneur is severe and likely to succeed and the incumbent finds it optimal to prevent it through acquisition. In contrast, "Don't Acquire"

is optimal when the entrepreneur's project is unlikely to succeed or when it shares little product market overlap with the incumbent. Finally, "Acquire to Continue" is optimal when the project is likely to be successful, but only has an intermediate degree of product market overlap. In that case, the incumbent finds it optimal to "Acquire to Continue" because the impact of self-cannibalization can be sufficiently dampened by multi-product pricing.

In Figure 2b the acquiring incumbent already faces competition from another existing incumbent ($n = 2$) and thus shows how the dominance regions for the incumbent's three strategies change as existing competition intensifies. The contrast between panel (a) and (b) illustrates the implication of Proposition 4. Increased existing competition erodes the profits the acquiring incumbent can protect through acquisition of the entrepreneur. As a result, the incumbent has lower incentives to acquire the entrepreneur regardless of whether it is with the intent to kill or to continue the project. In particular, in Figure 2b the region in which "Acquire to Continue" is optimal disappears entirely while the dominance region for "Acquire to Kill" shrinks. In contrast to the low competition ($n = 1$) case depicted in Figure 2a, in the high competition ($n = 2$) case the gains from acquisition do not outweigh the purchase price P for high values of ρ and a and thus "Don't Acquire" is optimal. In other words, the gains from preventing or softening cannibalization do not outweigh the valuation difference between incumbent and entrepreneur.

To summarize, in our model, entrepreneurial companies are acquired for two reasons. First, incumbents have more to gain from acquiring entrepreneurial companies if they can realize larger synergies from the transaction or face relatively small integration costs. Such synergies may derive from technical expertise or complementary assets. Second, potential incumbent acquirers have more to lose if they do not acquire an entrepreneurial company

with a project that is more similar to the potential acquirer's drug portfolio. This is the "cannibalization" effect. It raises the incentives for acquisition because an entrepreneur has a higher propensity for continuing development of a project that would reduce the profits of the incumbent. Thus, acquisitions in our model may occur for both value-enhancing (synergistic) and defensive anti-competitive reasons. Either they realize valuable synergies net of integration costs or they serve to prevent the development of projects that would otherwise hurt the profits of the acquiring incumbent if their development were ultimately successful. Note that even if there are no synergies to be realized and integration costs would otherwise deter the incumbent from buying the entrepreneur (i.e., $\sigma < 0$), the threat from continuing development, eventual project success, and cannibalization may still induce the incumbent to buy the new firm to prevent cannibalization.

4. 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 to prevent future competition. To do so, we need a setting and dataset that includes project level outcomes for companies that are acquired, 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.

4.1. Drug Development Background

New pharmaceutical products, or drugs, are developed following a set of structured and sequential steps.[3] 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 [4]. 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. About 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

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

[4]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

see when a project is suspended or discontinued. Observing these events at the project level is crucial to identifying killer acquisitions.

4.2. Drug Development 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.[5] Pharmaprojects includes nearly universal coverage of all candidate drugs being tested for eventual sale in the U.S. market, the mechanism of action (e.g., "Calcium channel antagonist"), and the intended therapeutic market (e.g., "Osteoporisis") (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 progress and acquisition events for at least 5 full years from initiation.

[Insert TABLE 1 Here.]

Table 1 provides a by-year tabulation of project coverage in our sample. Pharmaprojects provides a stable coverage from the start of the sample, with around 1,000 new drug projects per year in the 1990s. Drug development became more active since the 2000s and reached to around 2,000 projects per year after 2007. As to the ratio of acquired drugs, on average one third of drug projects were acquired at certain time point of the development life cycle.

[5]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 1.

The acquisition ratio is lower in recent years—one driving force behind such trend is the right-truncation of the sample. That is, as acquisition happens typically after a few years of development and such events of later projects might have not been realized by 2017.

4.3. 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 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.[6]

This acquisition database is further combined with the Pharmaprojects drug development data through a fuzzy matching algorithm accompanied with 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 (new owner/developer), the timing, and the development history under the this new owner.

[Insert FIGURE 3 Here.]

Figure 3 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. We find that 45% of companies originated only one drug in their whole life cycle.

4.4. USPTO Patent and Human Capital Data

The main drug development and acquisition database is augmented using patent database from the United States Patent and Trademark Office (USPTO). We access the National Bureau of Economic Research (NBER) USPTO patent database as of 2013 to obtain annual

[6]Each of the three data sources, SDC M&A Database, RecapIQ, and VentureXpert, contributes at least 10% of the innovation cases in the final database, suggesting a potential incompleteness that could arise if using one of them alone.

patent-level information from 1991 to 2006. The relevant variables include information on the patent assignee (the entity, such as the firm, which owns the patent), the number of citations received by the patent, the technology class of the patent, and the patents application and grant year. Bhaven Sampat's USPTO patent and citation database allows us to extend the NBER patent database up to 2012. We merge the USPTO data with drug development and acquisition data using a matching algorithm similar to Ma (2017), and details of this algorithm are provided in .Appendix 2.

In addition to general patenting activities, we are further interested in measuring the reallocation of human capital subsequent to acquisition events and the productivity changes. We track inventor mobility using the Harvard Business School (HBS) patent and inventor database. This database provides the names of the inventors (the individuals who receive credit for producing a patent) and their affiliations with the assignees, thus enabling us to track their mobility (see Lai, D'Amour, and Fleming (2009) for details).

4.5. 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 by categorizing them into twenty-eight categories, from as early as "new product," to as late as "first launch" of a product or reporting "suspended product."

[Insert TABLE 2 Here.]

We code these events into 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. This system of categorization is provided in Table 2. In general, continuation events reflect efforts to commercialize the underlying drug project (such as "Additional Launches," "Additional Registrations," "New Licensees"), or the progress in the research and development process (such as "Compounds Identified," "Mechanism Identified," "Target Identified").

5. Empirical Results

5.1. Univariate Results on Post-Acquisition Survival

Our empirical analysis starts from univariate survival tests on drugs that went through an acquisition during the development process and those that did not. Specifically, we examine the rates of being active, being discontinued, and being fully launched 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 (the average duration between drug origination and acquisition, if any, is about five years), we focus on drug projects originated before 2011.

[Insert TABLE 3 Here.]

The results are reported in Table 3. We report the rate of being active, being discontinued, and being fully launched separately 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. We find that non-acquired drugs are significantly more likely to be kept active, with a survival rate of 12.69%, while the acquired drugs are much less likely to survive, with an active rate of 5.24%. Meanwhile, the rate of discontinuation is significantly lower in the non-acquired sample (84.95%) than in the acquired sample (92.11%).

The unconditional launch rates of drugs are similar across the two samples (2.36% vs. 2.65%). This means, however, conditional on continuation (or in other words, not being discontinued), the rate of successful launching is higher in the acquired sample. Specifically, the conditional launching probability in the acquired sample is 2.65%/(2.65%+5.24%) = 33.59%, while the conditional probability in the non-acquired sample is 15.68%.

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, i.e., drugs originated pre-2006, and those originated pre-2000. We find similar patterns in both those two subsamples. Overall, the simple uni-variate survival tests on post-acquisition performance confirms the existence of killer acquisitions proposed in Proposition 1—acquired drugs are less likely to be continued in the development process, and conditional on continuation, the acquired drugs are more likely to be launched, since the drugs not being killed are typically the ones of higher quality.

5.2. Baseline Regression Results

Our main test uses a panel data of drug development. A drug is included in a sample from the origination year, and is removed after the termination. The empirical specification

is conducted as follows,

$$Continuation_{i,t} = \beta \cdot I(Acquired)_i \times I(Post)_{i,t} + \gamma \cdot I(Acquired)_i + \alpha_{age} + \alpha_{vintage} + \varepsilon_{i,t}, \tag{21}$$

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.

[Insert TABLE 4 Here.]

Results are reported in Table 4, and 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 column (1), we find that acquired drugs are 1.9% less likely to have an continuation update during the year post-acquisition. The unconditional probability of having a continuation update in the sample is 8.6%, leading the economic magnitude of the post-acquisition "killing" intensity to be 1.9%/8.6% = 22.09%. Reassuringly, the dummy variable $I(Acquired)$ does not carry any load in the regressions, meaning that the acquired drugs do not seems to have different continuation probability unconditionally.

In column (2) we incorporate drug-level fixed effects in the regression analysis. In this way, unobservable drug-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 4 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" idea.

In column (7) of Table 4, we replace the dependent variable dummy $Continuation_{i,t}$ with a counting variable that counts the total number of continuation events regarding to drug i in year t. Through this counting variables, we are able to capture the speed or intensity of the development of each drug. Using a similar empirical specification as in (21), we find similar results.

5.3. Overlap of Research Pipelines

One direct implication of the theoretical framework of killer acquisition in Section 3 is that such intention is closely governed by the extent to which the acquirer has overlapping drug development projects with the target. The more overlap a drug has with the acquirer, the more likely that the acquirer is motivated to preempt the competition by acquire and terminate the project.

We measure overlap between a drug project and the acquiring firm based on therapeutic class. In the Pharmaprojects database, each drug project is assigned to one or more therapeutic classes, which is based on the condition the therapy targets (e.g. Antihypertensive, Antidiabetic, etc.). If the acquiring firm has an active project in the same therapeutic class 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 overlap with the

acquirer's pipeline or not. We estimate the following model,

$$\begin{aligned} Continuation_{i,t} = \beta_O \cdot I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i + \beta \cdot I(Acquired)_i \times I(Post)_{i,t} \\ + \gamma_O \cdot I(Acquired)_i \times I(Overlap)_i + \gamma \cdot I(Acquired)_i \\ + \alpha_{age} + \alpha_{vintage} + \varepsilon_{i,t}. \end{aligned} \tag{22}$$

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 5 Here.]

Table 5 presents the results. In column (1), the β coefficient is -0.015, confirming the lower continuation probability post-acquisition. More importantly, β_O estimate of -0.020 is also statistically significant, meaning that projects acquired by buyers that have an overlapping project in the therapeutic class are more than twice likely to be discontinued in the development process ((0.015+0.020)/0.015 = 233%). The coefficient associated with $I(Acquired)_i \times I(Overlap)_i$ is positive and significant—what does this mean? One explanation is that incumbent firms are more likely to acquire those companies that show more positive (continuation) news, and they appear to have the ability to identify such targets.

This result has an additional important implication. From our baseline results in Table 4 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 have resolved information asymmetries between the acquirer and the target.

5.4. Market Competition

We measure competition both in the pipeline and existing competition in the market. For both measures, we count the number of firms with a drug or drug project that is in the same technology-market as the focal product. 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). To categorize a drug project's "market", we use its therapeutic class as defined above.

We measure competition as the count of firms who are: developing a drug that targets the same market using the same technology (our measure of "pipeline" competition), or who already have a drug in the same market of the focal project using the same technology (our measure of "existing product" competition).[7]

[Insert TABLE 6 Here.]

Table 6 presents the regression results to examine the intensity of killer acquisitions under

[7]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 technology-market with the most competition, i.e. if a project falls into two technology-markets, one with 0 pipeline competitors and one with 5, we use 5.

different competition environments. Drug development projects are categorized into terciles—high, medium, and low competition—by the competition measures described above. In the upper panel the competition measure is calculated using existing launched products while in the bottom panel the measure is calculated using the pipeline. The results suggest that the decreased continuation probability during the post-acquisition period largely concentrates in projects where the competition is not too high. Indeed, we find little evidence that killer acquisitions are a big concern in high-competition subsamples. Interestingly, the unconditional project continuation probability, as captured in the constant terms, presents an inverted-U pattern, similar to that identified in Aghion, Bloom, Blundell, Griffith, and Howitt (2005).

6. Alternative Explanations and Discussions

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 very simple empirical design and sample selection. In this section we attempt to sharpen the empirical approach and we discuss potential alternative explanations for our results.

6.1. 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 and thus the acquirer does not need to pick among multiple newly acquired drugs. If optimal project selection is driving our results, we should expect that the killing phenomenon does not exist in this analysis.

[Insert TABLE 7 Here.]

We report the analysis in Table 7 column (1). If anything, 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 update. This doubling of magnitude not only confirms that the identified results in Table 4 is likely due to the intention of "killing," but also suggests that those single-drug companies are the most vulnerable to the threat of such preemptive competitive strategies implemented by incumbent larger competitors.

6.2. 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). 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 a new developer (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 influence from the average developer trend.

Column (2) of Table 7 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 the 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 bottomline is that the interpretation of our main finding does not seem to be affected by the organizational frictions in the acquiring firm.

6.3. Discontinuation Decision

In column (3) of Table 7, 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 2 for detailed definitions of such event). We find that the likelihood of termination is significantly higher in years post-acquisition.

6.4. 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, key inventors, among others (Ouimet and Zarutskie, 2011). Under this view, the termination of acquired projects is a by-product of acquiring and efficiently redeploying valuable human capital in the acquired companies.

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

Nevertheless, we empirically assess this concern by using inventor level information

extracted from the USPTO records and HBS Inventor Database, following a similar approach as (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—i.e., how many of the inventors are retained in the combined firm, and are they efficiently redeployed in the new firm?

[Insert TABLE 8 Here.]

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 redeployed human capital to become more productive in the combined firm.

We show the analysis results in Table 8. 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 the data is that it is difficult to link each patent to a specific drug project for those early-stage projects.[8] As a result, it is difficult to accurately assign each

[8]Those information are typically disclosed toward the later stage in the drug development stage when

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.

6.5. Antitrust and the FTC Review Threshold

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 the paper, many of such acquisitions are made when the technology or project is still at a nascent stage and thus might not satisfy the 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.

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

FDA requires systematic reporting.

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 9 Here.]

In Table 9 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.

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 a 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 is more incentivized to protect its market power. We also show that alternative interpretations such as optimal project selection, organizational frictions, and the intent to redeploy human capital do not explain our results.

We want to add a few concluding remarks to link our finding 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,[9] 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 incorporate 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 can only be harmed as a result. Our results on the killer acquisition phenomenon around the FTC review thresholds, highlighting the phenomenon is more prevalent for acquisitions that are too small to scrutinize, exacerbates this concern.

Third, our findings suggest the Schumpeterian creative destruction process—whereby startups inventions can topple entrenched and less innovative incumbents—may be even more challenging 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.

[9]For example, TechCrunch shows 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 ($A = 100$, $c = 5$, $b = 4$, $k = 20$, $L = 20$, $\sigma = -15$, and $n = 1$).

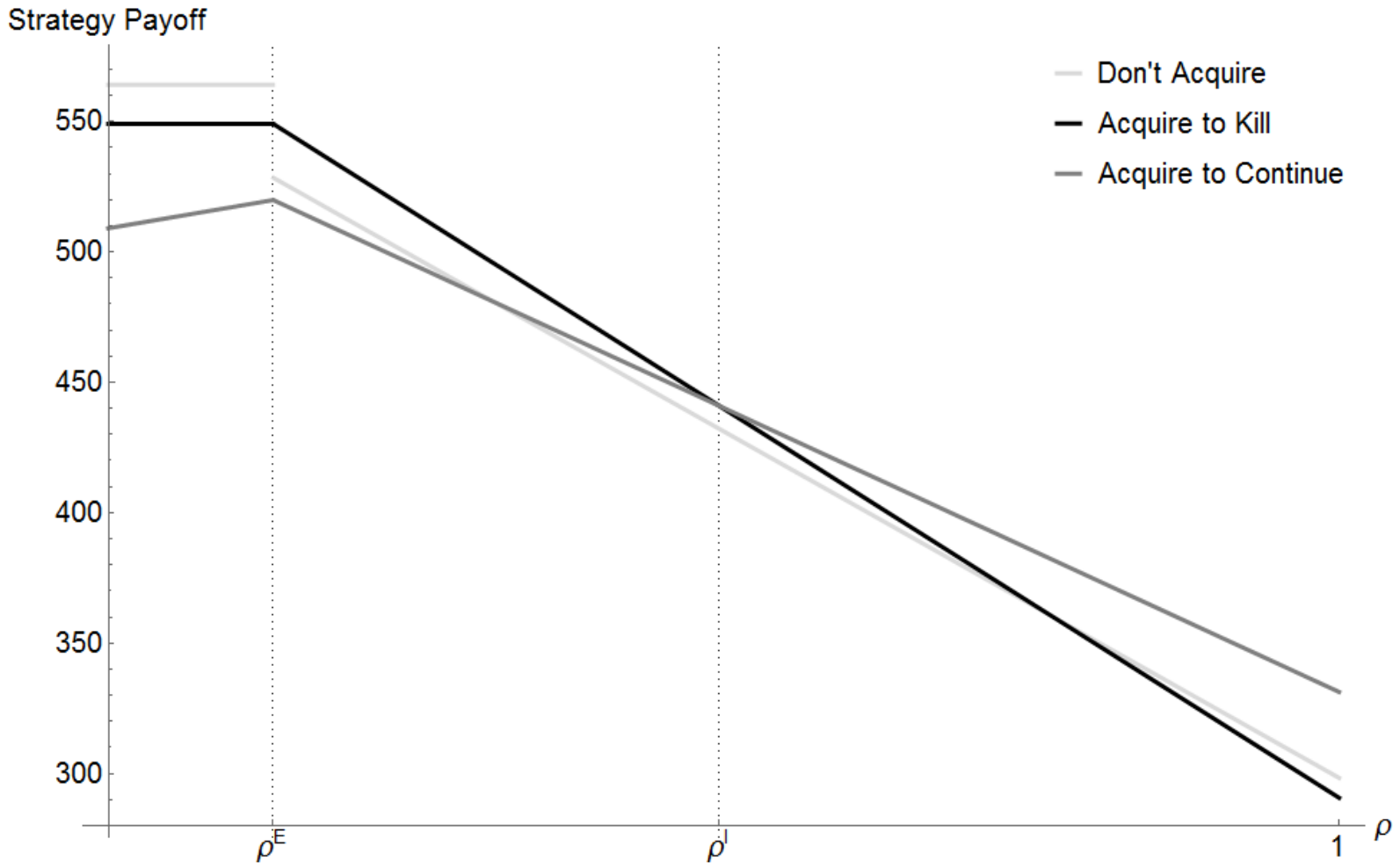
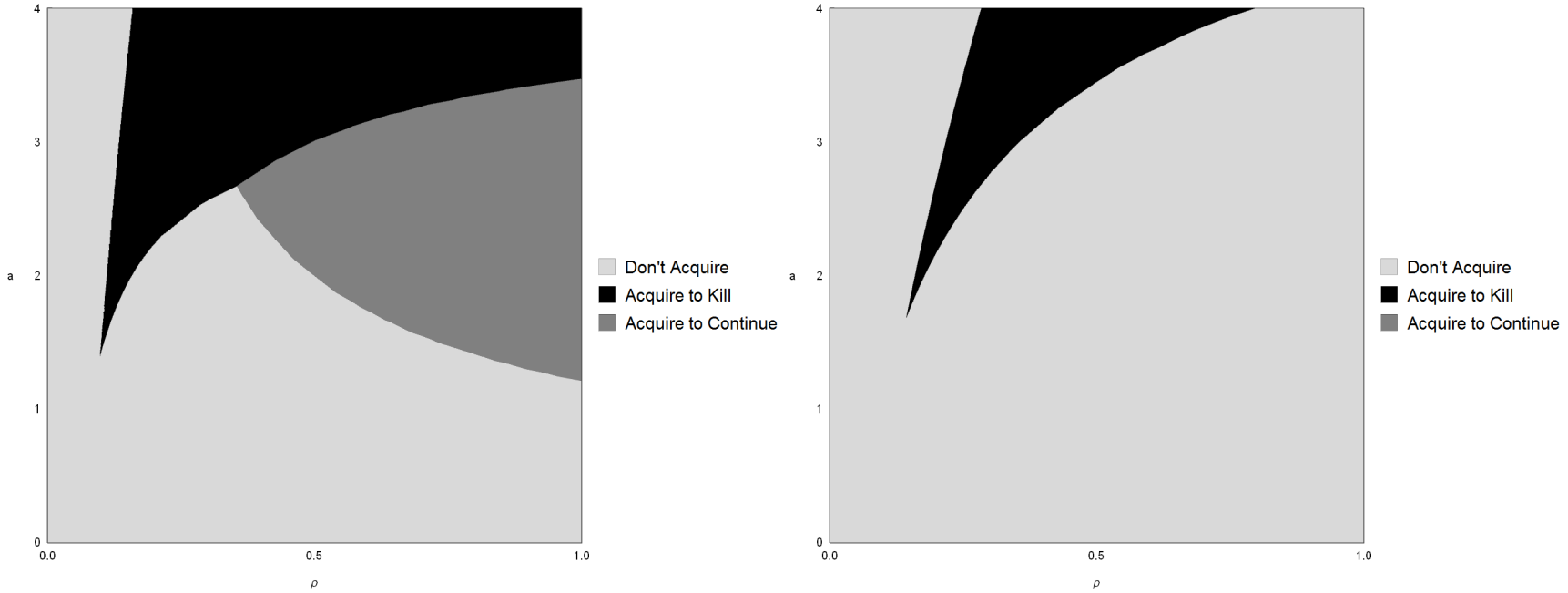


Figure 2. Optimal Acquisition Strategies

Dominance regions of the three acquistion strategies for different combinations of ρ and a for $n = 1$ and $n = 2$. Other parameter values are held constant ($A = 100$, $c = 5$, $b = 4$, $k = 20$, $L = 20$, and $\sigma = -15$).



(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.

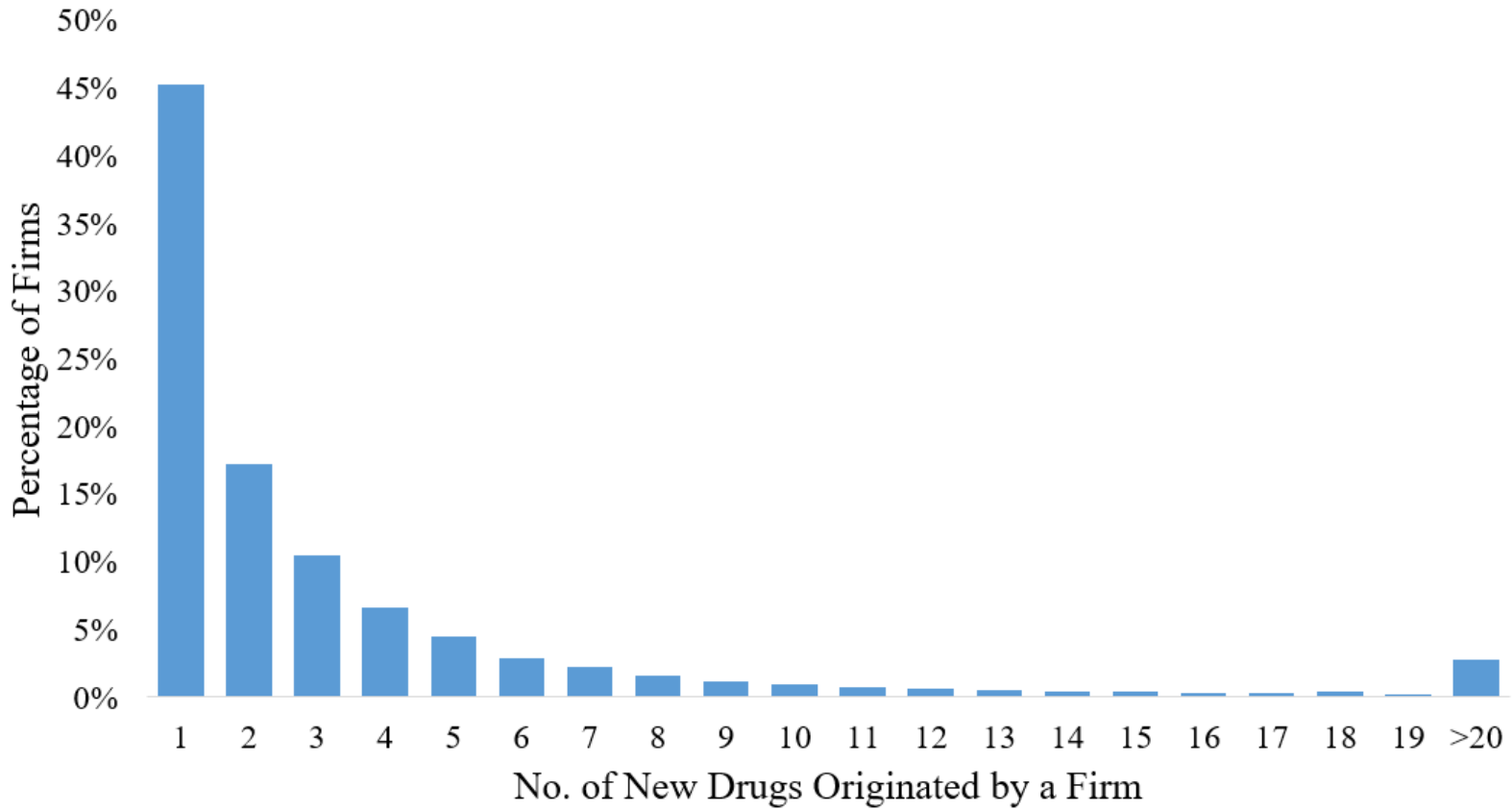


Table 1
Drug Development Projects Originated by Year

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	No. of New Drug Originations	% of Acquired	Year	No. of New Drug Originations	% of Acquired	Year	No. of New Drug Originations	% of Acquired
1989	638	38.87%	1997	1,066	33.40%	2005	1,455	18.42%
1990	776	37.63%	1998	1,159	32.96%	2006	1,353	16.04%
1991	892	38.68%	1999	1,041	30.74%	2007	2,244	11.45%
1992	1,061	41.28%	2000	1,000	31.30%	2008	2,278	9.70%
1993	1,111	42.30%	2001	1,273	30.87%	2009	2,144	6.86%
1994	854	43.56%	2002	1,285	26.07%	2010	1,914	6.53%
1995	1,036	34.85%	2003	1,437	25.47%	2011	2,396	5.43%
1996	1,030	34.95%	2004	1,691	19.40%			

Table 2
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

Table 3
Uni-variate Survival Test

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 being active, being discontinued, being fully launched 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 (Panel A), originated before 2006 (Panel B), and originated before 2000 (Panel C). We report the rate of being active, being discontinued, and being fully launched separately 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
		Panel A: Originated before 2011			
Active	12.69%	5.24%	7.45%	17.65841	***
Launched	2.36%	2.65%	-0.29%	-1.404187	
Discontinued	84.95%	92.11%	-7.15%	-15.5506	***
		Panel B: Originated before 2006			
Active	7.45%	3.55%	3.89%	10.54502	***
Launched	2.76%	2.94%	-0.18%	-0.7045388	
Discontinued	89.80%	93.51%	-3.72%	-8.477665	***
		Panel C: Originated before 2000			
Active	4.12%	2.16%	1.96%	5.617519	***
Launched	3.89%	3.45%	0.44%	1.193817	
Discontinued	91.99%	94.39%	-2.39%	-4.835903	***

Table 4
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						Intensive Margin
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Originated before 2011		Originated before 2006		Originated before 2000		Originated before 2011
I(Acquired) × I(Post)	-0.019***	-0.017***	-0.024***	-0.022***	-0.012***	-0.015***	-0.020***
	(-5.755)	(-4.577)	(-6.612)	(-5.564)	(-2.692)	(-3.006)	(-2.742)
I(Acquired)	-0.003		-0.004		-0.005		
	(-1.112)		(-1.280)		(-1.562)		
Constant	0.095***	0.095***	0.098***	0.097***	0.081***	0.079***	0.149***
	(97.026)	(325.826)	(77.834)	(259.300)	(49.572)	(183.062)	(251.991)
Observations	248,564	248,564	167,827	167,827	90,052	90,052	248,564
R-squared	0.018	0.248	0.015	0.250	0.007	0.241	0.261
Project FE	No	Yes	No	Yes	No	Yes	Yes
Age FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Originating Year FE	Yes	No	Yes	No	Yes	No	No

Table 5
Acquisitions and Project Continuation: The Effect of Product Overlap

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), we control for age and vintage (the year of origination) fixed effects; in columns (2), 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)
	Continuation Event = 1	
I(Acquired) × I(Post)	-0.015***	-0.011***
	(-4.202)	(-2.837)
I(Acquired) × I(Post) × Overlap	-0.020**	-0.029***
	(-2.304)	(-3.079)
I(Acquired)	-0.006**	
	(-2.138)	
I(Acquired) × Overlap	0.015**	
	(2.475)	
Constant	0.095***	0.094***
	(97.029)	(326.571)
Observations	248,564	248,564
R-squared	0.018	0.248
Project FE	No	Yes
Age FE	Yes	Yes
Originating Year FE	Yes	No

Table 6
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 the upper panel the competition measure is calculated using existing launched products while in the bottom panel the measure is calculated using the pipeline. 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.

	(1)	(2)	(3)	(4)	(5)	(6)
	Continuation Event = 1					
	Low Competition		Medium Competition		High Competition	
	Competition Measure = Existing Product					
I(Acquired) × I(Post)	-0.020***	-0.017***	-0.021	-0.034	-0.001	-0.000
	(-5.928)	(-4.322)	(-1.438)	(-1.443)	(-0.097)	(-0.013)
I(Acquired)	-0.001		-0.006		-0.009	
	(-0.321)		(-0.513)		(-0.822)	
Constant	0.093***	0.093***	0.133***	0.133***	0.078***	0.076***
	(94.820)	(316.244)	(35.259)	(63.743)	(21.143)	(47.037)
	Competition Measure = Pipeline					
I(Acquired) × I(Post)	-0.020***	-0.016***	-0.026***	-0.024**	-0.012**	-0.002
	(-3.997)	(-2.927)	(-3.521)	(-2.468)	(-2.298)	(-0.350)
I(Acquired)	0.005		-0.004		-0.010**	
	(1.240)		(-0.791)		(-2.369)	
Constant	0.095***	0.095***	0.110***	0.109***	0.088***	0.085***
	(71.191)	(244.865)	(50.021)	(142.038)	(55.693)	(156.943)
Project FE	No	Yes	No	Yes	No	Yes
Age FE	Yes	Yes	Yes	Yes	Yes	Yes
Originating Year FE	Yes	No	Yes	No	Yes	No

Table 7
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 8
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 9
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

Appendix 1. 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, or 55,687 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.

Appendix 2. 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.

A2.1. 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.

A2.2. 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.[10] The criterion is based on the length of the strings and the Levenshtein distance, and the threshold is determined through a random sampling procedure.

[10]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).

(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.