Killer Acquisitions*

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This article demonstrates, using a parsimonious model, that incumbent firms have incentives to 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 then provide detailed empirical evidence using the setting of drug development, in which we are able to track detailed project-level development histories of nearly 70,000 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 and technology 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—
amquisitions to kill. We argue that an incumbent firm may acquire an innovative target and
terminate development of the target’s innovations to preempt future competition. We call
such acquisitions “killer acquisitions” as they are intended to kill potentially promising, yet
likely competing, innovation.

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

In this paper, we theoretically model and empirically demonstrate this phenomenon. Our
analysis proceeds in two steps. First, to motivate the empirical analysis, we formalize the
concept of a killer acquisition using a parsimonious model that combines product market
competition, innovation, and endogenous acquisition decisions. In our model, an incumbent
firm that acquires a startup (i.e. the target) with an innovative project has weaker incentives

to continue the project’s development compared to a non-acquired entrant. This is because the incumbent firm incurs cannibalization of their existing product portfolio or, in other words, because of “the monopolist’s disincentive created by his preinvention monopoly profits” and, hence the incumbent has less to gain from successful development of the innovative project. This is Arrow’s “replacement effect” (Arrow, 1962). We show that under some circumstances this replacement effect can be so strong that incumbent firms may choose acquire startups simply to prevent them 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 acquirer-target product overlap is high, incumbents have stronger incentives to discontinue project development for such projects. Additionally, higher product market competition already in the related market erodes the incumbents’ existing profits and reduces the negative impact of the replacement effect when project development is successful. As a result, existing 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 for the same project subsequent to 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 nearly 70,000 drug projects originated by more than 8,000 companies in the past two and half decades, accompanied by the acquisition events collected from comprehensive data sources. We are able to observe the full development cycle for each drug from the initiation to the end point of the project (either successfully launched or discontinued). 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. We use the market-technology measures to finely categorize the degree to which an acquirer overlaps with the project, and thus identify potentially competing products. Further, we are able to characterize competition in both the development space and product market of the project, by using products under development as well as already launched products. Using existing, detailed pharmaceutical categorizations to measure overlap and competition is particularly desirable, given the complications associated with coarse industry coding systems or with the wide variations in product categorizations often used out of necessity in other settings.

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 a drug market, defined as the same therapeutic market and mechanism of action, 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. In additional analyses, we examine the progression of projects through the phases of clinical trials. Similarly, we find that for projects that start Phase II, acquired projects are less likely to enter Phase III, particularly when there is overlap between the target drug and the acquirer.

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 levels of existing competition. Competition is categorized using the number of firms with competing projects in the same therapeutic market and mechanism of action, either launched in the product market or in pipelines. We find that killer acquisitions 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 those that are tangential to the goal of the acquisition. To assess this concern, we repeat our analysis in acquisitions of single-drug companies, where the acquirer cannot be employing an “optimal project selection” strategy. Our results are robust to focusing on only this set of acquisitions, and, moreover, the magnitude actually increases. Hence, “optimal project selection” cannot explain our results.
Economic forces on the acquirer side could also confound our baseline interpretation. Previous research shows that the absence of private benefits in mature firms decrease the tendency to continue development (Guedj and Scharfstein, 2004) and that complex organization structures in larger firms are detrimental to the development of innovation projects (Seru, 2014). These forces could be the driving force behind the termination or slow-down of development after a project is acquired. We guard against this concern by including fixed effects at the developing company level (i.e., the acquirer firm after the acquisition), intended to capture acquirer firm-specific development productivity that could affect project development when changing owners. We find that after controlling for the developing ability of the acquirer firm, the killing intensity becomes even larger.

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

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

Our paper is also related to a large literature in corporate finance and industrial organiza-

\[^2\]Igami (2017) empirically shows that such cannibalization makes incumbents reluctant to innovate in the hard disk drive manufacturing industry. Existing firms’ slow responses to new technologies is grounded in a large literature on competition and innovation. See Cohen (2010) for a comprehensive survey.
tion which broadly highlights three distinct motives for acquisition: agency conflicts, synergies, and market power. First, in the absence of appropriate corporate governance mechanisms and incentive design, managerial interests that diverge from shareholder interests can lead to potentially value-destroying acquisitions (Roll, 1986; Morck et al., 1990). Second, acquisitions are driven by the pursuit of synergies between the acquirer and the target (Rhodes-Kropf and Robinson, 2008). Mergers have been shown to increase industry-adjusted cash flows (Healy et al., 1992; Andrade et al., 2001) and productivity (Maksimovic and Phillips, 2001) and an active acquisition market can also spur innovation (Phillips and Zhdanov, 2013). The post-merger increases in cash flows, new products, and patents are related to the ex-ante similarity of acquirer and target (Bena and Li, 2014; Hoberg and Phillips, 2010), but are harder to realize in markets with product integration difficulty (Hoberg and Phillips, 2017). Third, M&A transactions between existing competitors may occur to increase market power. This is the focus of much of US (and foreign) antitrust law.3

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

3Kamien and Zang (1990), Kamien and Zang (1993), Gowrisankaran (1999), Segal (1999), and Gowrisankaran and Holmes (2004) theoretically study merger decisions between existing competitors and analyze eventual market structure in a setting without antitrust policy. These papers show that even without the actions of antitrust authorities an industry may not be inevitably monopolized via mergers (i.e., there are competitive forces that push against such a trend). Segal and Whinston (2007) show that more protective antitrust policy may have conflicting effects on innovation incentives, by raising the profits of new entrants, but lowering those of continuing incumbents.
Similar to the M&A literature, the markets for technology literature (Gans and Stern, 2003; Arora and Gambardella, 2010; Arora et al., 2014) typically assumes that innovation-related transactions are synergistic, and thus experience related to the technology (i.e., owning a related technology) enables evaluation and absorption and therefore increases the likelihood of successful acquisition and innovation. However, relevant to our arguments, some have suggested that acquisitions of small, innovative target firms may also serve to preempt competition by enabling vital technology access (Hall, 1990; Lerner and Merges, 1998; Blonigen and Taylor, 2000; Lehto and Lehtoranta, 2006; Grimpe and Hussinger, 2008).4

The markets for technology literature also provides insight into the conditions under which a startup firm would want to sell their technology to incumbents instead of competing with them in the product market (Gans and Stern, 2003; Gans et al., 2002). Both the presence of patents (which reduce hazard of expropriation) and incumbent ownership of development assets (which increase potential gains from trade and hence joint surplus) and increase the likelihood that startups will want to and be able to sell their idea (Gans et al., 2002). The pharmaceutical industry is characterized by both of these features, which highlights why the industry is characterized by acquisition outcomes for startups, and further why killer acquisitions would be particularly feasible in our setting.

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

2. 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

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

2.1. Setup

The model has the following time line. In $t = 0$, an entrepreneurial company ($E$) with a single project is born ($E$ is the originating company of the project), 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 $\rho$ 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 $\pi$ 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 $\pi$, $\rho$, $k$, and $L$ are commonly known in $t = 0$.

2.2. 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

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

The resulting familiar first order condition is

$$A - 2bq_E - a \sum_{i \neq E} q_i - c = 0$$

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

$$\pi_{E_{acq,S}} = \frac{b(A - c)^2}{(2b + a(n))^2} = \pi_{I_{acq,S}}$$

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 $\pi_{E_{acq,F}} = 0$. The $n$ incumbent firms each have a single existing product to sell and thus their profit is equal to

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

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

$$\max_{q_1,q_2} (p_1 - c)q_1 + (p_2 - c)q_2$$
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 \( q_i^* = q^*_n \) for any \( i \neq 1, 2 \), and thus the resulting first order conditions can be rewritten as

\[
A - c - a(n - 1)q^*_n - 2(a + b)q^* = 0 \quad (6)
\]

\[
A - c - [2b + a(n - 2)]q^*_n - 2aq^* = 0 \quad (7)
\]

The resulting profit is

\[
\pi_{acq,S}^I = \frac{(2b - a)^2(a + b)(A - c)^2}{2(2b^2 + abn - a^2)^2} \quad (8)
\]

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

\[
\pi_{acq,F}^I = \frac{b(A - c)^2}{(2b + a(n - 1))^2} \quad (9)
\]

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

\[
\pi_{acq,S}^I \geq \pi_{acq,F}^I = \pi_{acq.I}^I \geq \pi_{acq.I}^E = \pi_{acq.S}^E > \pi_{acq.F}^E = 0. \quad (10)
\]

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 \( \pi_{acq,S}^I \). 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 \( \pi_{acq,F}^I \) while a successful entrepreneur would face \( n \) competitors and gain only \( \pi_{acq.S}^E \).

**2.2.1. The “Replacement Effect”**. However, what matters for the development decision in \( t = 1 \) are the difference between \( \pi_{acq,S}^I \) and \( \pi_{acq,F}^I \) for the incumbent and the difference between \( \pi_{acq,S}^E \) and \( \pi_{acq.F}^E \) for the entrepreneur. It is straightforward to show that

\[
\Delta^E \equiv \pi_{acq,I}^E - \pi_{acq,F}^E \geq \pi_{acq,S}^I - \pi_{acq,F}^I \equiv \Delta^I \quad (11)
\]
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 \((\pi^E_{\text{acq,F}} = 0)\) if she does not successfully develop the project. This is Arrow’s famous “replacement 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.

2.3. 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

\[
\rho(\pi^E_{\text{acq,S}} - \pi^E_{\text{acq,F}}) - k \geq L
\]

An incumbent gains \( \pi^I_{\text{acq,S}} \) from successful development of the project, but also foregoes the profit \( \pi^I_{\text{acq,F}} \) 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

\[
\rho(\pi_{acq,S} - \pi_{acq,F}) - k \geq L \quad (13)
\]

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 \( \rho^*_E \) and \( \rho^*_I \) and they are given by

\[
\rho^E = \frac{L + k}{\pi_{acq,S} - \pi_{acq,F}^E}, \quad \rho^I = \frac{L + k}{\pi_{acq,S} - \pi_{acq,F}^I} \quad (14)
\]

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 \( \rho \) is in the intermediate range between \( \rho^E \) and \( \rho^I \). 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 \( \rho^E \) and \( \rho^I \) 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 $\rho$. 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 $n$.

2.4. 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 $\sigma$ 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 $\sigma$ 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 $\sigma$ are known at
t = 0. Thus, the incumbent decides to acquire at a takeover price $P$ if

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

$$d^E [\rho \pi_{-acq,S}^I + (1 - \rho) \pi_{-acq,F}^I] + (1 - d^E) \pi_{-acq,F}^I (15)$$

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

\[
P = d^E\left[\rho(\pi_{acq,S} - \pi_{acq,F}) - k\right] + (1 - d^E)L
\]  

(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

\[
\sigma \geq 0.
\]  

(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

\[
\sigma + \rho\left(\pi_{acq,F} - \pi_{acq,S}\right) \geq \left(\rho\Delta^E - k - L\right)
\]  

(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 \( \rho \), the incumbent now has to compete against \( n \) other firms thus earning a lower profit \( \pi^I_{acq,S} \). Because competition reduces profits, the incumbent’s incentive to remain unchallenged is greater than the entrepreneur’s incentive to enter. This is the “efficiency effect”, first discussed by Gilbert and Newbery (1982) of monopoly persistence due to preemption incentives, which increases the incumbent’s incentive to preempt the entrepreneur. On the other hand, if \( \rho \leq \rho^E \), the expected marginal profit for
the entrepreneur from continuing development \((d^E = 1)\) given by \(\rho \Delta^E - k\) is larger than the liquidation value \(L\) that the incumbent \((d^I = 0)\) would obtain. This is the “replacement effect” which leads to a difference in valuation for developing the project between the entrepreneur and the incumbent and decreases the incentive to acquire.

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

\[
\sigma + \rho(\pi_{acq,S}^I - \pi_{acq,F}^I) \geq \rho(\Delta^E - \Delta^I) \quad (19)
\]

As before, \(\pi_{acq,S}^I - \pi_{acq,F}^I\) is the “efficiency effect” that is the gain from preemption by acquiring the entrepreneur and using multi-product pricing to soften the impact of cannibalization of the newly introduced product. On the other hand, the difference in valuation for the product between the entrepreneur and incumbent is again driven by the “replacement effect” \((\Delta^E \geq \Delta^I)\).

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 \(\rho\) 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 \(\rho(\pi_{acq,F}^I - \pi_{acq,S}^I)\) 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 (“efficiency effect”), and differences in project development valuation between originating and acquiring firms (“replacement effect”). We consider these three effects in turn.

First, acquiring the originating firm at price \( P \) always yields synergies or integration costs \( \sigma \). As discussed before such net synergies can be either positive or negative and thus either increase or decrease the incentives for acquisition.

Second, when \( \rho \) 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 \( \pi_{acq,F}^t - \pi_{acq,S}^t \) which results when the entrepreneur successfully develops the project with probability \( \rho \). 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

\[
\pi_{acq,F}^t - \pi_{acq,S}^t = \frac{b(A-c)^2}{(2b+a(n-1))^2} - \frac{b(A-c)^2}{(2b+an)^2}
\]  \hspace{1cm} (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 \( a = b \) and thus the incumbent the particularly strong incentives to maintain its market power through preemptive acquisition of the entrepreneur. 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 due to the “replacement effect”. 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 “efficiency effect” and the “replacement effect” work in opposite directions: the former is positive and the latter is negative if and only if $a > 0$. It is straightforward to show that the “efficiency effect” dominates the “replacement effect” if market overlap is high ($a$ is large) and 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 $\sigma$ 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 $\rho$ 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 $\rho$ 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 \(\rho\) 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 “efficiency effect”. It raises the incentives for acquisition because this prevents the entrepreneur who has a higher propensity for continuing development of a project (due to the “replacement effect”) from entering and reducing the profits of the 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.

3. Empirical Setup: Background and Data

The main empirical goal of our paper is to document the phenomenon of killer acquisitions. These acquisitions occur when acquiring firms acquire targets specifically to extinguish target technologies and 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.

3.1. Drug Development Background

New pharmaceutical products, or drugs, are developed following a set of structured and sequential steps. 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. 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

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5The steps below summarize those described in detail by the FDA (US Food and Drug Administration, 2017)

6Drug 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
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 see when a project is suspended or discontinued. Observing these events at the project level is crucial to identifying killer acquisitions.

3.2. 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. 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., “Osteoporosis”) (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.]

\[\text{\footnotesize 7The raw Pharmaprojects data typically updates the firm name associated with each project when it is acquired. We therefore re-constructed the historical originator firm using text descriptions included in the dataset. More details are provided in Appendix 1.}\]
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. 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.

### 3.3. Clinical Trials

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

### 3.4. 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 Ventur-
eXpert 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.8

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.

3.5. 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

8Each 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).

3.6. 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”).
4. Main Empirical Results

4.1. 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, in particular, 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.

4.2. 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,

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

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.

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 a 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 seem 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 \(\beta\) 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.

4.3. Overlap of Research Pipelines

One direct implication of the theoretical framework of killer acquisition in Section 2 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,

$$Continuation_{i,t} = \beta_O \cdot I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i + \beta \cdot I(Acquired)_i \times I(Post)_{i,t}$$

$$+ \gamma_O \cdot I(Acquired)_i \times I(Overlap)_i + \gamma \cdot I(Acquired)_i$$

$$+ \alpha_{age} + \alpha_{vintage} + \varepsilon_{i,t}. \quad (22)$$

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 \( I(\text{Acquired})_i \times I(\text{Overlap})_i \) 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 \( \beta \) coefficient is -0.015, confirming the lower continuation probability post-acquisition. More importantly, \( \beta_O \) 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 as likely to be discontinued in the development process \( ((0.015+0.020)/0.015 = 233\%) \). The coefficient associated with \( I(\text{Acquired})_i \times I(\text{Overlap})_i \) is positive and significant. One explanation for this is that incumbent firms are more likely to acquire those companies that show more positive (continuation) news, and they appear to have the ability to identify such targets.

Table 5 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.

[Insert TABLE 6 Here.]

To further explore how overlap relates to killing intensity, we tested how the time remaining on acquirer patents for the drugs that overlap with the target drugs conditions the results we see in Table 5. To do so, we identified patents linked to approved drugs (from FDA Orange Book data via Pharmaprojects) and merged in USPTO data on patent timelines. Following the logic above whereby killing intensity intensifies when the acquirer has a close competitor, we expect the negative relationship between overlap and continuation effects to be most pronounced among acquirers with patents that have a long remaining life. Table 6 presents the results among acquisitions with overlapping acquirers. We find that if the acquirer patents are within 5 years of expiry, the killing intensity is mitigated.
4.4. Clinical Trials

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

Table 7 presents regression results on the subsample for which we have information about Phase II start dates. We limit the sample to those projects started before 2010 (and control for vintage) to ensure projects have time to enter Phase III. We find that, compared to projects that aren’t acquired in Phase II, those that are acquired are less likely to move on to Phase III trials, and this relationship is stronger when the acquirer has overlapping projects. In terms of economic magnitude, in column (2), the decreased probability of -0.051 is 17.6% of the base rate of entering Phase III of 28.9%. Being acquired by an acquirer with overlapping products more than doubles this intensity.

4.5. 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).  

Table 8 presents the regression results to examine the intensity of killer acquisitions under 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).

5. Alternative Explanations

Results thus far, though consistent with the killer acquisition interpretation, raise the concern that they could be mechanical or subject to alternative interpretations due to the 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.

9Note 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. That is to say, if a project falls into two technology-markets, one with 0 pipeline competitors and one with 5, we use 5.
5.1. Optimal Project Selection

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

Our approach to investigating this concern is to examine only the deals with single-drug targets—that is, we try to identify the post-acquisition continuation probability only for the cases in which the target owns one and only one drug at the time of acquisition 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 9 Here.]

We report the analysis in Table 9 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.

5.2. Organizational Frictions in Acquirers

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

We assess the validity of this alternative interpretation by introducing fixed effects at the developer level (equivalently, the owner or acquirer level). To be clear, the acquired drug will be assigned 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 $\beta$ can be interpreted net of the influence from the average developer trend.

Column (2) of Table 9 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.

5.3. Discontinuation Decision

In column (3) of Table 9, 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.
5.4. Redeployment of Technologies

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

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

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

To measure chemical similarity, we follow the literature on the chemical informatics literature, in which the Tanimoto distance is the most commonly used method (Nikolova and Jaworska, 2003; Krieger, Li and Papanikolaou, 2017). The idea behind the calculation is to compute the proportion of chemical features shared by any two chemicals when divided by the union of the two. This similarity measure is bounded between 0 and 1, with 0 indicating the pair share no common chemical fragments.

[Insert TABLE 10 Here.]

In Table 10, we examine whether drugs initiated in the acquirer post-acquisition become more chemically similar to the acquired drug. If post-acquisition technology integration is pronounced, one would expect that drugs in acquirer firms to incorporate chemical components from the acquired technology and become similar to the acquired project. However, in our simple framework, we find that if anything, drugs developed in acquirer firms post the acquisition of a drug become less similar. The economic magnitude of -0.001 is indeed
negligible compared to the global similarity mean of 13.3%. Overall, this does not support the view that technology redeployment is a prominent phenomenon which explains killer acquisitions.

5.5. Redeployment of Human Capital

By now, our analyses and interpretations have been focusing on the project or technology side of the acquisition. However, it could be the case that the key motivation behind these acquisitions are human capital such as the research team, 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. In particular, we analyze how many of the inventors are retained in the combined firm and whether they are efficiently redeployed in the new firm

[Insert TABLE 11 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 11. 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.\textsuperscript{10} As a result, it is difficult to accurately assign each inventor to the specific drug project that she or he is involved in. As a result, we are not able to identify whether the leaving or staying inventors are from projects that are eventually killed. In untabulated results where we focus on cases with a single-drug target, we find that a even larger proportion of investors leave the combined firm after the acquisition.

6. Discussions

6.1. 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

\begin{footnote}{Those information are typically disclosed toward the later stage in the drug development stage when FDA requires systematic reporting.}

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$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 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 12 Here.]

In Table 12 we implement this analysis. We collect the acquisitions that are right below the FTC review threshold \([-10\%, 0]\) and those just above that \([0, 10\%]\). First, we find higher number of deals just below the threshold than just above the threshold (70% higher). Second, the survival rate of below-threshold deals is lower than those right above the threshold. Similarly, we find the launching rate is much lower (1.8% versus 9.1%) and the discontinuation rate is much higher (94.6% versus 83.3%). While this analysis is simple and purely descriptive, overall these patterns are consistent with acquirers conducting more killer acquisitions when they can expect to avoid FTC review.

6.2. Ex-ante Innovation Incentives and Welfare

Our theoretical and empirical analysis focuses on the acquisition and project development incentives of incumbents and entrepreneurs. In our setting, killer acquisitions have an unambiguously negative effect on welfare even though the entrepreneur is indifferent (due to his lack of bargaining power) and the acquiring incumbent (and other incumbents) are strictly better off when acquisitions are allowed. Consumers are hurt both by the lack of competition and the elimination of innovative new products. Killer acquisitions benefit incumbents, leave entrepreneurs indifferent, but disproportionately hurt consumers.
A comprehensive welfare analysis of the impact of killer acquisitions is, however, more difficult given the many different forces involved in the innovation process. It is possible that the presence of an acquisition channel also has a positive effect on welfare that is not accounted for in our analysis. In particular, the prospect of entrepreneurial exit through acquisition (by an incumbent) may spur ex-ante innovation as in Phillips and Zhdanov (2013). Whereas in our model entrepreneurs are born with a project and thus do not have to exert effort to come up with an idea, it is plausible that the prospect of later acquisition may motivate the origination of entrepreneurial ideas in the first place. However, it is important to note that killer acquisitions will only spur such idea origination if the entrepreneur receives some of the surplus that accrues to the incumbent through the acquisition.\footnote{For a model along these lines see Phillips and Zhdanov (2013) who show that increased takeover activity spurs innovation by small firms because this allows them to capture a larger share of the benefits of innovation.} If the entrepreneur is left with no surplus relative to standalone value of his project he will be unaffected by acquisitions and hence will not respond by increasing his innovation efforts. If killer acquisitions do increase ex-ante innovation, this potential welfare gain will have to be weighed against the ex-post efficiency loss due to reduced competition. Whether the former positive or the latter negative effect dominates will depend on the elasticity of the entrepreneur’s innovation response.

Furthermore, acquisitions may not only influence the intensity of entrepreneurial project generation, but they may also affect its direction. If entrepreneurs can choose between originating projects that overlap with existing products or those that do not, increased takeover activity and killer acquisitions by incumbents may spur innovation of very similar ‘me-too’ drugs at the expense of the origination of truly novel products (Arcidiacono et al., 2013). This response to the prospect of acquisitions would add to the negative welfare impact of killer acquisitions.\footnote{Rasmusen (1988) considers a theoretical model in this vein in which entrants can blackmail the incumbent by threatening to keep prices low, and buyout can make entry profitable which otherwise would not be.}

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 or technologies 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, and even though technology acquisitions can offer opportunities for synergy and gains from trade, acquisitions may also have potentially destructive consequences. In other words, as opposed to interpreting the acquisition of nascent technologies as incumbents’ effort to incorporate entrepreneurial innovation and maximize joint surplus, a significant driver fueling this trend may be killer acquisitions and creator destruction (i.e., killing the threat of creative destruction).

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

Third, our findings suggest 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.

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


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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 $\rho$. 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 acquisition strategies for different combinations of $\rho$ 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.

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

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

<table>
<thead>
<tr>
<th></th>
<th>Non-acquired</th>
<th>Acquired</th>
<th>Diff</th>
<th>T-statistics</th>
<th>Stat Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Panel A: Originated before 2011</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Active</td>
<td>12.69%</td>
<td>5.24%</td>
<td>7.45%</td>
<td>17.65841</td>
<td>***</td>
</tr>
<tr>
<td>Launched</td>
<td>2.36%</td>
<td>2.65%</td>
<td>-0.29%</td>
<td>-1.404187</td>
<td></td>
</tr>
<tr>
<td>Discontinued</td>
<td>84.95%</td>
<td>92.11%</td>
<td>-7.15%</td>
<td>-15.5506</td>
<td>***</td>
</tr>
<tr>
<td><strong>Panel B: Originated before 2006</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Active</td>
<td>7.45%</td>
<td>3.55%</td>
<td>3.89%</td>
<td>10.54502</td>
<td>***</td>
</tr>
<tr>
<td>Launched</td>
<td>2.76%</td>
<td>2.94%</td>
<td>-0.18%</td>
<td>-0.7045388</td>
<td></td>
</tr>
<tr>
<td>Discontinued</td>
<td>89.80%</td>
<td>93.51%</td>
<td>-3.72%</td>
<td>-8.477665</td>
<td>***</td>
</tr>
<tr>
<td><strong>Panel C: Originated before 2000</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Active</td>
<td>4.12%</td>
<td>2.16%</td>
<td>1.96%</td>
<td>5.617519</td>
<td>***</td>
</tr>
<tr>
<td>Launched</td>
<td>3.89%</td>
<td>3.45%</td>
<td>0.44%</td>
<td>1.193817</td>
<td></td>
</tr>
<tr>
<td>Discontinued</td>
<td>91.99%</td>
<td>94.39%</td>
<td>-2.39%</td>
<td>-4.835903</td>
<td>***</td>
</tr>
</tbody>
</table>
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,

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

where the dependent variable \(Continuation_{i,t}\) is a dummy variable indicating whether drug \(i\) has an active continuation event in year \(t\). \(I(\text{Acquired})_i\) indicates whether drug \(i\) undergoes an acquisition event, \(I(\text{Post})_{i,t}\) 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.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>I(Acquired) × I(Post)</td>
<td>-0.019***</td>
<td>-0.017***</td>
<td>-0.024***</td>
<td>-0.022***</td>
<td>-0.012***</td>
<td>-0.015***</td>
<td>-0.020***</td>
<td>(-5.755)</td>
</tr>
<tr>
<td>I(Acquired)</td>
<td>-0.003</td>
<td>-0.004</td>
<td>-0.005</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(-1.112)</td>
</tr>
<tr>
<td>Constant</td>
<td>0.095***</td>
<td>0.095***</td>
<td>0.098***</td>
<td>0.097***</td>
<td>0.081***</td>
<td>0.079***</td>
<td>0.149***</td>
<td>(97.026)</td>
</tr>
<tr>
<td>Observations</td>
<td>248,564</td>
<td>248,564</td>
<td>167,827</td>
<td>167,827</td>
<td>90,052</td>
<td>90,052</td>
<td>248,564</td>
<td></td>
</tr>
<tr>
<td>R-squared</td>
<td>0.018</td>
<td>0.248</td>
<td>0.015</td>
<td>0.250</td>
<td>0.007</td>
<td>0.241</td>
<td>0.261</td>
<td></td>
</tr>
<tr>
<td>Project FE</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>Age FE</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>Originating Year FE</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td></td>
</tr>
</tbody>
</table>
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,

\[
Continuation_{i,t} = \beta_0 \cdot I(Acquired)_{i,t} \times I(Post)_{i,t} \times I(Overlap)_{i,t} + \beta \cdot I(Acquired)_{i,t} \times I(Post)_{i,t} \\
+ \gamma_0 \cdot I(Acquired)_{i,t} \times I(Overlap)_{i,t} + \gamma \cdot I(Acquired)_{i,t} \\
+ \alpha_{age} + \alpha_{vintage} + \varepsilon_{i,t}.
\]

where the dependent variable \(Continuation_{i,t}\) is a dummy variable indicating whether drug \(i\) has an active continuation event in year \(t\). \(I(Acquired)\) 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 column (1), we control for age and vintage (the year of origination) fixed effects; in column (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.

<table>
<thead>
<tr>
<th></th>
<th>(1)</th>
<th>(2)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Continuation Event = 1</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I(Acquired) \times I(Post)</td>
<td>-0.015***</td>
<td>-0.011***</td>
</tr>
<tr>
<td></td>
<td>(-4.202)</td>
<td>(-2.837)</td>
</tr>
<tr>
<td>I(Acquired) \times I(Post) \times Overlap</td>
<td>-0.020**</td>
<td>-0.029***</td>
</tr>
<tr>
<td></td>
<td>(-2.304)</td>
<td>(-3.079)</td>
</tr>
<tr>
<td>I(Acquired)</td>
<td>-0.006**</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(-2.138)</td>
<td></td>
</tr>
<tr>
<td>I(Acquired) \times Overlap</td>
<td>0.015**</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(2.475)</td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>0.095***</td>
<td>0.094***</td>
</tr>
<tr>
<td></td>
<td>(97.029)</td>
<td>(326.571)</td>
</tr>
<tr>
<td>Observations</td>
<td>248,564</td>
<td>248,564</td>
</tr>
<tr>
<td>R-squared</td>
<td>0.018</td>
<td>0.248</td>
</tr>
<tr>
<td>Project FE</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Age FE</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Originating Year FE</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>

49
Table 6
Acquisitions and Project Continuation: Patent Life Among Overlaps

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

\[ Continuation_{i,t} = \beta_0 \cdot I(\text{Post})_{i,t} + \beta \cdot I(\text{Near Pat Expiry})_i \]
\[ + \gamma_0 \cdot I(\text{Near Pat Expiry})_i \times I(\text{Post})_{i,t} \]
\[ + \alpha_{age} + \alpha_{vintage} + \varepsilon_{i,t}. \]

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

<table>
<thead>
<tr>
<th></th>
<th>(1)</th>
<th>(2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continuation Event = 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I(\text{Post})</td>
<td>-0.089***</td>
<td>-0.055***</td>
</tr>
<tr>
<td></td>
<td>(-3.650)</td>
<td>(-2.633)</td>
</tr>
<tr>
<td>I(\text{Near Pat Expiry})</td>
<td>-0.079***</td>
<td>-0.045**</td>
</tr>
<tr>
<td></td>
<td>(-3.235)</td>
<td>(-2.151)</td>
</tr>
<tr>
<td>I(\text{Post}) \times I(\text{Near Pat Expiry})</td>
<td>0.045</td>
<td>0.068*</td>
</tr>
<tr>
<td></td>
<td>(1.640)</td>
<td>(1.674)</td>
</tr>
<tr>
<td>Observations</td>
<td>3,216</td>
<td>3,216</td>
</tr>
<tr>
<td>R-squared</td>
<td>0.041</td>
<td>0.152</td>
</tr>
<tr>
<td>Age FE</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Originating Year FE</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Acquiror FE</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>
This table presents the differing continuation rates of drug projects. The sample for this analysis is drugs that entered Phase II trials. The analysis looks at the effect of acquisition, and overlap with the acquirer, on the likelihood the project enters Phase III trials. The empirical specification uses the following model,

$$\text{PhaseIII}_i = \beta \cdot I(\text{Acquired PII})_i + \gamma_0 \cdot I(\text{Acquired PII})_i \times I(\text{Overlap})_i + \alpha_{\text{vintage}} + \varepsilon_i.$$

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

<table>
<thead>
<tr>
<th></th>
<th>(1)</th>
<th>(2)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Phase III = 1</td>
<td></td>
</tr>
<tr>
<td>$I(\text{Acquired Phase II})$</td>
<td>-0.009</td>
<td>-0.051*</td>
</tr>
<tr>
<td></td>
<td>(-0.345)</td>
<td>(-1.835)</td>
</tr>
<tr>
<td>$I(\text{Acquired Phase II}) \times I(\text{Overlap})$</td>
<td>-0.124**</td>
<td>-0.114**</td>
</tr>
<tr>
<td></td>
<td>(-2.057)</td>
<td>(-1.915)</td>
</tr>
<tr>
<td>Constant</td>
<td>0.282***</td>
<td>0.289***</td>
</tr>
<tr>
<td></td>
<td>(27.274)</td>
<td>(28.146)</td>
</tr>
<tr>
<td>Observations</td>
<td>2,248</td>
<td>2,248</td>
</tr>
<tr>
<td>R-squared</td>
<td>0.003</td>
<td>0.036</td>
</tr>
<tr>
<td>Originating Year FE</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>
Table 8
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,

\[ \text{Continuation}_{i,t} = \beta \cdot I(\text{Acquired})_i \times I(\text{Post})_{i,t} + \gamma \cdot I(\text{Acquired})_i + \alpha_{\text{age}} + \alpha_{\text{vintage}} + \varepsilon_{i,t}, \]

where the dependent variable \( \text{Continuation}_{i,t} \) is a dummy variable indicating whether drug \( i \) has an active continuation event in year \( t \). \( I(\text{Acquired})_i \) indicates whether drug \( i \) undergoes an acquisition event, \( I(\text{Post})_{i,t} \) 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.

<table>
<thead>
<tr>
<th></th>
<th>(1)</th>
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<th>(3)</th>
<th>(4)</th>
<th>(5)</th>
<th>(6)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Low Competition</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( I(\text{Acquired}) \times I(\text{Post}) )</td>
<td>-0.020***</td>
<td>-0.017***</td>
<td>-0.021</td>
<td>-0.034</td>
<td>-0.001</td>
<td>-0.000</td>
</tr>
<tr>
<td></td>
<td>(-5.928)</td>
<td>(-4.322)</td>
<td>(-1.438)</td>
<td>(-1.443)</td>
<td>(-0.097)</td>
<td>(-0.013)</td>
</tr>
<tr>
<td>( I(\text{Acquired}) )</td>
<td>-0.001</td>
<td>-0.006</td>
<td>-0.009</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(-0.321)</td>
<td>(-0.513)</td>
<td>(-0.822)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Constant</strong></td>
<td>0.093***</td>
<td>0.093***</td>
<td>0.133***</td>
<td>0.133***</td>
<td>0.078***</td>
<td>0.076***</td>
</tr>
<tr>
<td></td>
<td>(94.820)</td>
<td>(316.244)</td>
<td>(35.259)</td>
<td>(63.743)</td>
<td>(21.143)</td>
<td>(47.037)</td>
</tr>
<tr>
<td><strong>Medium Competition</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( I(\text{Acquired}) \times I(\text{Post}) )</td>
<td>-0.020***</td>
<td>-0.016***</td>
<td>-0.026***</td>
<td>-0.024**</td>
<td>-0.012**</td>
<td>-0.002</td>
</tr>
<tr>
<td></td>
<td>(-3.997)</td>
<td>(-2.927)</td>
<td>(-3.521)</td>
<td>(-2.468)</td>
<td>(-2.298)</td>
<td>(-0.350)</td>
</tr>
<tr>
<td>( I(\text{Acquired}) )</td>
<td>0.005</td>
<td>-0.004</td>
<td>-0.010**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(1.240)</td>
<td>(-0.791)</td>
<td>(-2.369)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Constant</strong></td>
<td>0.095***</td>
<td>0.095***</td>
<td>0.110***</td>
<td>0.109***</td>
<td>0.088***</td>
<td>0.085***</td>
</tr>
<tr>
<td></td>
<td>(71.191)</td>
<td>(244.865)</td>
<td>(50.021)</td>
<td>(142.038)</td>
<td>(55.693)</td>
<td>(156.943)</td>
</tr>
<tr>
<td><strong>High Competition</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( I(\text{Acquired}) \times I(\text{Post}) )</td>
<td>-0.020***</td>
<td>-0.016***</td>
<td>-0.026***</td>
<td>-0.024**</td>
<td>-0.012**</td>
<td>-0.002</td>
</tr>
<tr>
<td></td>
<td>(-3.997)</td>
<td>(-2.927)</td>
<td>(-3.521)</td>
<td>(-2.468)</td>
<td>(-2.298)</td>
<td>(-0.350)</td>
</tr>
<tr>
<td>( I(\text{Acquired}) )</td>
<td>0.005</td>
<td>-0.004</td>
<td>-0.010**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(1.240)</td>
<td>(-0.791)</td>
<td>(-2.369)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Constant</strong></td>
<td>0.095***</td>
<td>0.095***</td>
<td>0.110***</td>
<td>0.109***</td>
<td>0.088***</td>
<td>0.085***</td>
</tr>
<tr>
<td></td>
<td>(71.191)</td>
<td>(244.865)</td>
<td>(50.021)</td>
<td>(142.038)</td>
<td>(55.693)</td>
<td>(156.943)</td>
</tr>
</tbody>
</table>

| 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 9

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,

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

where the dependent variable \( Continuation_{i,t} \) is a dummy variable indicating whether drug \( i \) has an active continuation event in year \( t \). \( I(\text{Acquired})_i \) indicates whether drug \( i \) undergoes an acquisition event, \( I(\text{Post})_{i,t} \) 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.

<table>
<thead>
<tr>
<th></th>
<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Single-Drug Company Control Developer FE Termination Events</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I(Acquired) ( \times ) I(Post)</td>
<td>-0.035*</td>
<td>-0.108***</td>
<td>0.007***</td>
</tr>
<tr>
<td></td>
<td>(-1.842)</td>
<td>(-11.216)</td>
<td>(4.695)</td>
</tr>
<tr>
<td>Constant</td>
<td>0.097***</td>
<td>0.156***</td>
<td>0.014***</td>
</tr>
<tr>
<td></td>
<td>(985.373)</td>
<td>(84.372)</td>
<td>(113.305)</td>
</tr>
<tr>
<td>Observations</td>
<td>201,161</td>
<td>248,564</td>
<td>248,564</td>
</tr>
<tr>
<td>R-squared</td>
<td>0.248</td>
<td>0.084</td>
<td>0.305</td>
</tr>
<tr>
<td>Project FE</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Age FE</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>
This table studies chemical similarities of drug projects between acquired drugs and drugs originated by the acquirer firm. Each observation in the sample is a drug-pair between an acquired drug and a drug from the acquirer originated within the five-year windows around the acquisition event. The key independent variable, $I(\text{Post})$, indicates whether the acquirer drug was initiated after the acquisition event, and takes value one if so. To measure chemical similarity we use the Tanimoto distance (Nikolova and Jaworska, 2003; Krieger, Li and Papanikolaou, 2017). In column (1), we do not control for fixed effects; in column (2), we control for acquirer firm fixed effects; in column (3), we control for case-specific fixed effects. The t-statistics based on standard errors clustered at the drug project level are displayed in parentheses. ***, **, and * indicate significance at the 1%, 5%, and 10% levels, respectively.

<table>
<thead>
<tr>
<th></th>
<th>(1)</th>
<th>(2)</th>
<th>(3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chemical Similarity</td>
<td>-0.001*</td>
<td>-0.001</td>
<td>-0.002***</td>
</tr>
<tr>
<td></td>
<td>(-1.673)</td>
<td>(-1.274)</td>
<td>(-4.208)</td>
</tr>
<tr>
<td>Constant</td>
<td>0.133***</td>
<td>0.132***</td>
<td>0.133***</td>
</tr>
<tr>
<td></td>
<td>(94.840)</td>
<td>(94.827)</td>
<td>(576.005)</td>
</tr>
<tr>
<td>Observations</td>
<td>154,896</td>
<td>154,896</td>
<td>154,896</td>
</tr>
<tr>
<td>R-squared</td>
<td>0.000</td>
<td>0.013</td>
<td>0.361</td>
</tr>
<tr>
<td>Acquiror FE</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Case FE</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>
Table 11  
**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.

<table>
<thead>
<tr>
<th></th>
<th>Before Acquisition</th>
<th>After Acquisition</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Those Who Move to Acquiror After Acquisition (22%)</td>
<td>4.572</td>
<td>3.160</td>
<td>-1.412***</td>
</tr>
<tr>
<td>Those Who Move to Other Firms After Acquisition (78%)</td>
<td>4.357</td>
<td>4.089</td>
<td>-0.267*</td>
</tr>
<tr>
<td>Difference</td>
<td>-0.215</td>
<td>0.929***</td>
<td>1.144***</td>
</tr>
</tbody>
</table>
Table 12
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.

<table>
<thead>
<tr>
<th></th>
<th>10% Below Threshold</th>
<th>10% Above Threshold</th>
<th>Diff</th>
<th>T-statistics</th>
<th>Stat Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active</td>
<td>3.57%</td>
<td>7.58%</td>
<td>-4.00%</td>
<td>-1.175713</td>
<td></td>
</tr>
<tr>
<td>Launched</td>
<td>1.79%</td>
<td>9.09%</td>
<td>-7.31%</td>
<td>-2.292933</td>
<td>**</td>
</tr>
<tr>
<td>Discontinued</td>
<td>94.64%</td>
<td>83.33%</td>
<td>11.31%</td>
<td>2.509447</td>
<td>**</td>
</tr>
<tr>
<td>N</td>
<td>112</td>
<td>66</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
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_comname” (Wasi and Flaen 2014), we isolate the stem name for each originator firm associated with each project in Pharmaprojects.

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 is identified, and the two companies are located in the same city and state OR the two companies 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. The criterion is based on the length of the strings and the Levenshtein distance, and the threshold is determined through a random sampling procedure.

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14 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 companies 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.