

Killer Acquisitions*

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Firms have incentives to acquire innovative targets to discontinue the development of the targets' innovation projects and preempt future competition. We call such acquisitions "killer acquisitions." We develop a parsimonious model in which killer acquisitions arise from an incumbent's desire to prevent the profit cannibalization of existing products that are substitutes for (or overlap with) the target's innovation. We provide empirical evidence for this phenomenon by tracking detailed project-level development for more than 35,000 pharmaceutical drug projects. Acquired drug projects are less likely to be developed when the acquired project overlaps with the acquirer's portfolio of products and projects. This pattern is more pronounced when the acquirer has strong incentives to protect its market power due to weak existing competition. Alternative interpretations such as optimal project selection and human capital and technology redeployment do not explain our results. Conservative estimates indicate that about 6.4% of all acquisitions in our sample are killer acquisitions and that such acquisitions often occur just below thresholds for antitrust scrutiny.

JEL Classification: O31, L41, G34, L65

Keywords: Innovation, Mergers and Acquisitions, Drug Development, Competition

*We thank Audra Boone, Kevin Bryan, Lorenzo Caliendo, Judy Chevalier, Jason Donaldson, Jan Eeckhout, Constança Esteves-Sorenson, Jonathan Feinstein, Joachim Henkel, Scott Hemphill, Mitsuru Igami, Rob Jensen, Josh Krieger, Jim Levinsohn, Danielle Li, Louis Kaplow, Mushfiq Mobarak, John Morley, Justin Murfin, Barry Nalebuff, Micah Officer, Fiona Scott Morton, Merih Sevilir, Scott Stern, Yang Sun, Rick Townsend, Kosuke Uetake, Thomas Wollmann, Alexei Zhdanov, Jidong Zhou, Jeff Zwiebel, and seminar participants at the ALEA Annual Meeting (Boston), AOM Annual Meeting, ASU Winter Conference, Berkeley (Hass), Brandeis, Chicago Booth (Micro), Colorado Boulder (Leeds), FIRS Annual Conference (Barcelona), FSU SunTrust Conference, LBS, NBER Summer Institute Industrial Organization, NBER Entrepreneurship, NBER Organizational Economics, Northwestern (Kellogg), NYU Stern (Strategy, Finance), Queen's Organizational Economics Conference, Rising Five-star Junior Finance Workshop, Rochester, UBC (Economics, Sauder), UCL (Cass), UCLA (Law & Economics, Finance), USC (Marshall), Washington University in St. Louis, Wharton (Finance, Management), WFA Annual Meeting (Coronado), and Yale for helpful comments. James Baker provided excellent research assistance. The Cowles Foundation for Research in Economics, the Yale Center for Science and Social Science Information, and the Yale School of Medicine Library provided data access support. All errors are our own.

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Innovation drives economic growth. Innovating firms are often acquired by incumbents, typically in the early stages of product development. Economists traditionally view this positively: firms who are better at exploiting technologies acquire innovative targets to realize synergies, effectively enabling specialization and subsequently increasing innovation and overall welfare. In this paper, we propose and test a different motive for acquisitions of innovating firms. 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 eliminate potentially promising, yet likely competing, innovation.

A recent case involving the pharmaceutical firm Questcor (a subsidiary of Mallinckrodt) helps illustrate this phenomenon. In the early 2000s, Questcor enjoyed a monopoly in adrenocorticotrophic hormone (ACTH) drugs with its product Acthar, which treats rare, serious conditions, including infantile spasms. In the mid-2000s, Synacthen, a synthetic competitor to Acthar was beginning development for the U.S. market. Questcor acquired the U.S. development rights for Synacthen in 2013. Following the logic of killer acquisitions—that is, shutting down competition even before there is a marketable product—Questcor did not develop Synacthen. As the FTC argued in an antitrust complaint: “With the acquisition of Synacthen, Questcor thwarted a nascent challenge to its Acthar monopoly.”¹ In other words, Questcor acquired and eliminated competition *preemptively*.

In this paper, we theoretically and empirically study killer acquisitions. First, to motivate the empirical analysis, we build a parsimonious model that combines endogenous acquisition decisions, innovation, and product market competition. The key innovation of our theoretical framework is that we allow corporate acquisitions to occur when the target firm’s project is still under development and continued development of the project is necessary, costly, and uncertain. In our model, an incumbent firm that acquires an innovative project has weaker incentives to continue developing such a project than an independent entrepreneur if the new project overlaps with (i.e., is a substitute for) the incumbent’s existing product. This is a general, well-known result which arises when an acquirer cannibalizes (or replaces)

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

some of his own existing product's profits by developing a new product or, in other words, it arises because of "the monopolist's disincentive created by his preinvention monopoly profits" (Arrow, 1962). We show that this disincentive to innovate in order to protect existing profits from the "gale of creative destruction" of new inventions (Schumpeter, 1942) can be so strong that an incumbent firm may acquire a startup simply to shut down the startup's projects. As such, killer acquisitions prevent the development of new products that would otherwise reduce the incumbent's profits.

We show that killer acquisitions arise from Arrow's replacement effect which is present for any degree of acquirer-target product overlap. In such cases, acquirers have strictly stronger incentives to discontinue project development than independent entrepreneurs. However, we further show that competition erodes an incumbent's profits and thus diminishes the incentive to protect existing profits. As a result, both existing and future product market competition (i.e., proximate patent expiry) reduce the difference in project development decisions between acquirers and independent entrepreneurs and therefore decrease the prevalence of killer acquisitions.

In the second part of the paper, we seek to provide empirical support for our theoretical predictions. Doing so presents significant empirical challenges. An ideal setting would allow us to observe development activity at the project level. Further, we need to track projects as they move across firms. It is also crucial to accurately characterize overlap between the acquiring firm's products and the target's project and to quantify competition in the relevant product market.

Drug development within the pharmaceutical industry offers all of these features. Further, the pharmaceutical industry is highly innovative, and the successful commercialization of innovative drugs is potentially very socially valuable.² We collect detailed development information on more than 35,000 drug projects originated by more than 6,700 companies in the past two and half decades and follow each drug from initiation. We collect relevant acquisition events from comprehensive data sources. Importantly, we observe development milestones of drug projects independent of project ownership, meaning we can follow the

²R&D intensity in the pharmaceutical industry is second only to semiconductors in the U.S. manufacturing sector, at 11.3% in 2014 (US NSF, NCSSES, 2018).

same projects pre- and post-acquisition.³

To finely categorize acquirer overlap with the target's project, and thus identify potentially competing products, we use uniquely precise pharmaceutical market categories. Specifically, if the target's drug project falls both in the same therapeutic market (e.g., antihypertensives) and uses the same mechanism of action (e.g., calcium channel antagonists), within which the acquirer is developing or has launched a drug, we consider that to be an overlapping acquisition. Using this same tight measure of overlap, we characterize competition in the product market and the development pipeline of the relevant project. Using detailed pharmaceutical categorizations to measure overlap and competition is particularly desirable given the complications associated with coarse industry codes and broad product categorizations often used out of necessity in other settings.

Our key empirical test for killer acquisitions focuses on the development stage of drug projects; it compares projects acquired by overlapping incumbents to those acquired by non-overlapping incumbents, and to non-acquired projects. Decreased likelihood of development of overlapping projects post-acquisition provides supportive evidence for killer acquisitions.

The baseline regression uses a project-year panel to estimate the annual probability of a development event or milestone. The key finding is that projects acquired by an incumbent with an overlapping drug are 39.6% less likely to be continued in the development process compared to drugs that are not acquired. This estimate accounts for any differences in development life cycles using project age and vintage (initiation year) fixed effects. Reassuringly, the likelihood of a development milestone for acquired drugs, whether overlapping or not, is not statistically distinguishable from non-acquired drugs in years prior to acquisition.

Our theory also predicts that incumbents have a stronger incentive to acquire and terminate overlapping innovation when the market is less competitive. The central idea is that in a less competitive market, an incumbent has more to lose from cannibalization if the target's innovation is successfully developed. We test this idea by repeating the baseline analysis in subsamples with low and high levels of existing competition (as measured by the number of competing drugs), either in the product market or separately in the development

³For 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 track and document the development of Dom-0800 post-2006, regardless of its change in ownership.

pipeline. We find that the decrease in development probability for acquired, overlapping projects is concentrated in markets with low competition of either type. Our theory also predicts that the effects of cannibalization will be strongest when the incumbent’s drug is far from patent expiry, i.e., when the incumbent has more to lose. Accordingly, we find that the decrease in development rates is concentrated in overlapping acquisitions for which the acquirer’s overlapping drug is relatively far from patent expiry and future generic competition is more distant.

In additional empirical tests, we examine the progression of projects through the phases of clinical trials. While more limited as compared to our main analysis in the types of development milestones as well as the sample of projects, this additional analysis both mirrors prior work on drug development (Krieger, 2017; Guedj and Scharfstein, 2004) and ensures comparison of projects at precisely the same stage of development. Specifically, we focus on projects that start Phase I trials and examine their likelihood of starting Phase II. We find drug projects are 39.8% less likely to enter Phase II if they are acquired during Phase I by an acquirer with an overlapping drug. As in the main analyses, these findings are concentrated in markets with low competition.

We employ several additional tests to address potential alternative explanations. One alternative explanation for our baseline finding is optimal project selection. Specifically, for multi-project targets, the acquirer could strategically and optimally choose to continue the more promising or complementary projects while discontinuing those that are less promising. To assess this concern, we repeat our analysis for acquisitions of single-drug companies, where the acquirer cannot be employing such a strategy. Our results are robust to focusing on only this set of acquisitions. Hence, optimal project selection cannot explain our results.

We also investigate whether changes to the timing of development rather than true discontinuation might be behind our estimates. Acquiring firms might delay development (or accelerate it), or they may be slower at developing, which would result in decreased development events over the observed project life cycle post-acquisition. We find no evidence that such development timing differences are driving our main results.

Another alternative explanation is capital redeployment, in which the acquiring firm’s intention is to acquire and redeploy the core assets of the acquired target—i.e., its underlying

technology or human capital—to more productive uses. If this were the case, our results on decreased development of acquired, overlapping projects could be explained simply as a by-product. To address this, we separately consider technology and human capital redeployment. To explore technology redeployment, we track the chemical similarity of acquired drugs to pre- and post-acquisition projects of the acquirer, finding no evidence supporting the idea that acquired technologies are integrated into acquirers’ drug new development projects. To explore human capital redeployment, we examine inventor mobility and productivity around the acquisition events. We show that only 22% of inventors from target firms eventually work for the acquiring firm and further that those inventors do not become more productive post-acquisition. These results are inconsistent with explanations regarding technology or human capital redeployment.

Our conservative estimates indicate that about 6.4% of all acquisitions in our sample (or about 49 pharmaceutical acquisitions per year) are killer acquisitions. Our analysis also reveals that acquirers conducting killer acquisitions are much more likely to undertake acquisition deals that do not trigger FTC notification requirements for pre-merger review and thereby avoid antitrust scrutiny. Acquisitions of overlapping targets bunch just below the FTC acquisition transaction value threshold, while there is no such pattern for non-overlapping acquisitions. In addition, these below-threshold deals exhibit much higher termination rates and much lower launch rates. Eliminating the adverse effect on drug project development from killer acquisitions would raise the pharmaceutical industry’s aggregate drug project development rate by nearly 5%.

Overall, this paper makes three contributions. First, we shed light on a fundamental impediment to corporate innovation. Specifically, we highlight how the motive to protect existing profits, thought to discourage an incumbent’s own innovation, can also incentivize powerful incumbent firms to stifle the innovation of others. Second, we document the importance of this obstacle to innovation in the pharmaceutical industry, an industry crucial to consumer and social welfare and in which innovation is fundamental. Third, we provide new evidence relating to trends and consequences of increasing market concentration. Incumbents in already concentrated markets further reduce competition by acquiring *future* product market competitors. Such acquisitions often avoid antitrust scrutiny and pose potentially

grave concerns for consumer welfare.

Related prior literature on corporate acquisitions in corporate finance, the economics of innovation, and industrial organization has focused on agency conflicts, synergies, and existing competition. 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 may be driven by the pursuit of synergies between the acquirer and the target (Rhodes-Kropf and Robinson, 2008).⁴ In the context of technology acquisitions, Gans and Stern (2003), Arora and Gambardella (2010), and Arora et al. (2014) document that owning a related technology increases the likelihood of a successful acquisition and subsequent innovation performance. Third, M&A transactions between *existing* competitors may occur in order to increase market power (Baker and Bresnahan, 1985). This is the focus of much of U.S. (and foreign) antitrust law.⁵

The central idea of this article, however, is that incumbents have weaker incentives to pursue innovation and may even acquire potential *future* competitors to terminate innovation if such innovation threatens their existing profits.⁶ 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 whenever new and existing products are substitutes for each other (“replacement effect”).⁷ The other part has its theoretical roots in Gilbert and Newbery

⁴Mergers 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). Post-merger increases in cash flow, 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).

⁵In theory, even without the actions of antitrust authorities, an industry may not be inevitably monopolized via mergers (Kamien and Zang, 1990, 1993; Gowrisankaran, 1999; Segal, 1999; Gowrisankaran and Holmes, 2004). Protective antitrust policy may even have conflicting effects on innovation incentives, by raising the profits of new entrants, but lowering those of continuing incumbents (Segal and Whinston, 2007).

⁶Any corporate transaction also requires that a startup firm would want to sell its technology to incumbents instead of competing with them in the product market. Gans and Stern (2003) and Gans et al. (2002) show that both the presence of patents and incumbent ownership of development assets increase the likelihood that startups will want to sell their ideas. The pharmaceutical industry is characterized by both of these features which explains why acquisitions of startups are frequent and why killer acquisitions would be particularly prevalent.

⁷Henderson (1993) and Igami (2017) empirically show that such cannibalization makes incumbents reluctant to innovate in the photolithographic alignment equipment and the hard disk drive manufacturing industries. More broadly, the slow response to new technologies by incumbent firms is explored in the large literature on competition and innovation. See Cohen (2010) for a comprehensive survey.

(1982) who demonstrate that a monopolist has incentives to acquire the property rights to a new innovation to preempt entry (“efficiency effect”).⁸ Our paper considers both of these forces and offers a theoretical and empirical analysis in the context of drug development. Quite surprisingly, the link between (horizontal) mergers and innovation has received little attention despite its significant policy relevance.⁹ Our data provide detailed information on post-acquisition development at the project level. These detailed data allow us rule out other potential explanations for the innovation gap between acquired and independent firms, and to rule out other underlying acquisition motives as driving our results.

The remainder of the paper proceeds as follows. Section 1 outlines our theoretical framework and develops testable hypotheses. Section 2 describes the data and institutional background. Section 3 presents our main empirical results and shows how other motives cannot explain these findings. Section 4 discusses implications for antitrust and social welfare and quantifies the industry-wide impact of killer acquisitions. Section 5 offers concluding remarks.

1. Theoretical Framework

We propose a simple theoretical model of acquisition, innovation, and product market competition to investigate acquisitions and project development of entrepreneurial companies and incumbent firms. All proofs are in Appendix A.

1.1. Setup

The model has the following timeline shown in Figure 1. In $t = 0$, an entrepreneurial company E with a single project is born. E is the originating company of the project. There are $n \geq 1$ incumbent firms, each possessing an existing product. One of these n incumbents, which we call the (potential) acquirer A , can acquire the entrepreneur E at an endogenously

⁸Katz and Shapiro (1985) and Gans and Stern (2000) offer comprehensive theoretical treatments of R&D competition when cooperative arrangements (e.g., licensing, alliances, acquisitions) are feasible. Lerner and Merges (1998) provide empirical evidence for such arrangements between biotech firms and pharmaceutical corporations.

⁹Like us, Federico et al. (2017, 2018), Motta and Tarantino (2017), and Gilbert (2018) present theoretical models in which merging parties have diminished innovation incentives after the merger. In Cabral (2017) such mergers can also be used to “stand on the shoulders of dwarfs” and cement the dominance of incumbents.

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

Consider first the product market choices of the entrepreneur when her project is not acquired ($-acq$). If the project is successful (S), the resulting newly developed product competes against n other single-product incumbent firms and the entrepreneur maximizes $p_E q_E$. Given that all $n + 1$ single-product firms are symmetric we solve for the symmetric equilibrium which yields profits $\pi_{-acq,S}^E = \pi_{-acq,S}^A > 0$. Note that the product market profits for the entrepreneur and the acquirer (as well as the other $n - 1$ incumbent firms) are identical.

If the new project fails (F), the entrepreneur does not have any product to sell in $t = 2$, and thus her profit is equal to $\pi_{-acq,F}^E = 0$. The n incumbent firms each have a single existing product to sell, and thus the acquirer's profit is equal to $\pi_{-acq,F}^A$. Profits are higher $\pi_{-acq,F}^A > \pi_{-acq,S}^A$ because competition now only involves n single-product firms.

Next consider the product market choices of an acquirer in the case of an acquisition (acq). If the project is unsuccessful, the acquirer can still sell his existing product in $t = 2$. In contrast to the case of no acquisition, the acquirer only has to compete against $n - 1$ other single-product incumbents. The resulting profit for the acquirer is $\pi_{acq,F}^A$. This is the same as when no acquisition occurs and the entrepreneur fails, hence $\pi_{acq,F}^A = \pi_{-acq,F}^A$.

If the project is successful, he becomes a two-product oligopolist who optimally chooses prices for his two products and competes against $n - 1$ other single-product incumbents. The acquirer's objective function is to maximize the profits from both of his products $p_1 q_1 + p_2 q_2$, whereas the remaining $n - 1$ other single-product incumbent firms maximize single-product profits.¹³ The profit of the multi-product incumbent acquirer is $\pi_{acq,S}^A$. This profit is obviously higher than when he sells only a single product with the same $n - 1$ competitors, hence $\pi_{acq,S}^A > \pi_{-acq,F}^A$.

As a result, we obtain the following profit ranking

$$(1) \quad \pi_{acq,S}^A > \pi_{acq,F}^A = \pi_{-acq,F}^A > \pi_{-acq,S}^A = \pi_{-acq,S}^E > \pi_{-acq,F}^E = 0.$$

¹³Given our symmetry assumptions, in equilibrium, the resulting prices are $p_1^* = p_2^* = p^A$ and $p_i^* = p^I$ for any $i \neq 1, 2$.

1.3. Development Decision ($t = 1$)

1.3.1. Product Market Overlap. We now investigate the development decision in $t = 1$. This is akin to a pharmaceutical firm deciding whether to proceed with the development of a new drug. What matters for the development decision in $t = 1$ are the difference between $\pi_{acq,S}^A$ and $\pi_{acq,F}^A$ 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 for all imperfect substitutes, we have

$$(2) \quad \Delta^E \equiv \pi_{-acq,S}^E - \pi_{-acq,F}^E > \pi_{acq,S}^A - \pi_{acq,F}^A \equiv \Delta^A$$

As long as products are imperfect substitutes the acquirer gains strictly less from developing a new product than an entrepreneur would. This is because the new product cannibalizes some of the profits of the acquirer's existing product. In contrast, an entrepreneur has no product to sell and hence no profit if she does not successfully develop the project. This is a very general result with a simple, well-known intuition. It is Arrow's famous "replacement effect" (Arrow, 1962) which causes the entrepreneur and the acquirer to obtain different benefits from continuing development.¹⁴

The development decisions of the entrepreneur ($d^E = \{0, 1\}$) and the acquirer ($d^A = \{0, 1\}$) are determined by

$$(3) \quad \rho\Delta^E - k \geq L, \quad \rho\Delta^A - k \geq L.$$

Rewriting these two inequalities yields the development cost thresholds used by the entrepreneur and the acquirer

$$(4) \quad k^E = \rho\Delta^E - L, \quad k^A = \rho\Delta^A - L.$$

Comparison of these thresholds shows that $k^E > k^A$ for any imperfect substitutes because in that case $\Delta^E > \Delta^A$. This immediately yields our first prediction. Any form of product market

¹⁴If products are independent, the incentives to innovate are 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.

overlap with the existing drug in the acquirer’s portfolio reduces the acquirer’s propensity to continue development of the acquired project relative to the case in which the project remains independent.

Proposition 1 (Project Development and Market Overlap). *An incumbent firm that acquires a project continues development if $k \leq k^A$ while an independent entrepreneur continues if $k \leq k^E$. For any positive product market overlap, we have $k^E > k^A$.*

The difference in development behavior between incumbent acquirer and entrepreneur occurs when k is in the intermediate range between k^A and k^E . This region exists for any positive degree of product substitutability, and its size depends on the difference between the entrepreneur’s and the acquirer’s development gains Δ^E and Δ^A . If Δ^E is much larger than Δ^A then the entrepreneur’s development incentives are much larger than the acquirer’s. But when Δ^E is only slightly larger than Δ^A —i.e., when the two products are close to independent goods—then the two development policies are quite similar.

Finally, note the crucial role that the development cost k plays in our model. Without costly development (i.e., if $k = 0$) all firms would continue development and thus killer acquisitions would never occur. Necessary and costly ongoing development of a drug project is what generates the differential development decisions of the incumbent acquirer and the independent entrepreneur.

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

Proposition 2 (Project Development and Competition). *For any positive product market overlap, the difference $k^E - k^A$ is positive and strictly decreasing in n .*

Successfully developing a new product equally draws consumer demand away from existing products and hurts the profits of all incumbent firms. In our model when the acquiring incumbent is a monopolist, he is particularly hesitant compared to an entrepreneur to develop an overlapping product because this new product draws demand away entirely from his own existing product. But when he already faces many other existing competitors, introducing a

new product draws demand away from all existing products, only one of which is his own. In other words, when there are many existing competitors the cannibalization losses from the successful development of a new product are spread over a large number of firms. As a result, when there are more existing competitors the development decisions of the acquirer become more similar to those of the entrepreneur. Figure 2 illustrates this point by plotting the development thresholds as a function of the number of incumbents. These are closer together when there are more existing incumbents.

[Insert FIGURE 2 Here.]

1.3.3. Patent Life and Future Competition. Until now, we have only considered the impact of competition with imperfect substitutes which captures the competition between branded drugs. However, another important aspect is competition from undifferentiated generic drugs that enter the market when a branded product's patent expires. Denote the number of years of remaining patent life of the entrepreneur's new project by T^E and those of the acquiring incumbent's existing product by T^A where $T^E > T^A \geq 0$. Assume, for simplicity, that the firms earn their static game profits every year.

We assume that as soon as a product's patent expires, an identical, undifferentiated product (e.g., a generic drug) enters the market. Bertrand competition between undifferentiated products then implies that prices and profits for the acquirer's existing product drop to zero. Thus, for the T^A years in which the existing product's patent is still valid, the acquirer either earns $\pi_{acq,S}^A$ (successful development of new project) or $\pi_{acq,F}^A$ (unsuccessful development) each year. This yields the same development gain Δ^A as before multiplied by the number of years T^A . Similarly, the entrepreneur's development gain over that time span is $T^A \Delta^E$. Thereafter, the profits for the acquirer's existing product drop to 0, and hence his incentives to develop coincide with those of the entrepreneur. Denote the development gains for the entrepreneur and the acquirer in the presence of undifferentiated generic competition after the expiry of the acquirer's existing product's patent in T^A years by $\Delta_{gen} = \Delta_{gen}^E = \Delta_{gen}^A$.¹⁵

¹⁵Note that these (equal) development gains are different from the previous expressions Δ^E and Δ^A . This is because when a generic product (that is undifferentiated from the acquirer's existing product) enters, it not only drives profits of that product to zero, but due to its low price it also reduces the profits of the other products that are differentiated from it.

The reason why these development gains after generic entry are the same for the acquirer and the entrepreneur is that when the incumbent's patent on his existing product expires, he no longer has to be concerned about a new product cannibalizing the profits of his existing product: generic competition has already destroyed all those profits. As a result, after T^A years it is as if the acquiring incumbent did not have any existing overlapping product.

Thus, the development decisions of the entrepreneur d_{gen}^E and the acquiring incumbent d_{gen}^A are now determined by

$$(5) \quad \rho[T^A \Delta^E + (T^E - T^A) \Delta_{gen}] - k \geq L$$

$$(6) \quad \rho[T^A \Delta^A + (T^E - T^A) \Delta_{gen}] - k \geq L$$

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

Proposition 3 (Project Development and Patent Life). *For any positive product market overlap, the difference $k^E - k^A$ is weakly positive and strictly increasing in T^A .*

The longer the patent life T^A of the acquirer's existing product, the weaker are his incentives to continue development relative to those of the entrepreneur. When the acquirer's existing overlapping product has only little remaining patent life (T^A close to 0), his development policy for the new project is quite similar to that of the entrepreneur.¹⁶

1.4. Acquisition Decision ($t = 0$)

We now show that "killer acquisitions" are theoretically possible. Although the acquirer has weaker development incentives than an entrepreneur, he may nonetheless want to acquire the entrepreneur. This is because acquiring the project prevents (by terminating the project) or softens (through multi-product pricing) the destruction of the acquirer's existing profits. In making the acquisition decision the acquiring incumbent must weigh this benefit against

¹⁶The intuition for this result is essentially the same as that of Proposition 2. Generic entry is just a particularly intense form of competition that already destroys any profits of the acquirer's existing product and thus turns self-cannibalization from the development of a new product into an entirely moot point.

paying the purchase price P .¹⁷ Our analysis shows that even when acquirers selectively choose which projects to acquire, our theoretical predictions about differential project development decisions between acquired and non-acquired projects still apply.

We assume that to compensate the entrepreneur for selling the project, the acquirer must pay an endogenously determined takeover price P equal to (or greater than) the expected payoff of the project when the entrepreneur remains independent.¹⁸ Because both the acquisition decision as well as the takeover price depend on the entrepreneur's and the acquirer's development decisions, there are three cases to consider.

First, if $k > k^E$, neither the entrepreneur nor the acquirer chooses to develop the project. Both parties also have the same (liquidation) value L for the project and are indifferent as to who owns it.

Second, for $k^E \geq k > k^A$, the acquirer terminates the project, but the entrepreneur continues development. Thus, such an acquisition is a “killer acquisition” which occurs if

$$(7) \quad \underbrace{\rho(\pi_{acq,F}^A - \pi_{-acq,S}^A)}_{\text{efficiency effect}} \geq \underbrace{\rho\Delta^E - k - L}_{\text{replacement effect}}.$$

If the acquirer acquires the entrepreneur's project and shuts it down, he only competes against $n - 1$ other firms and earns a profit equal to $\pi_{acq,F}^A$. However, if the incumbent does not acquire the entrepreneur's project, the incumbent has to compete against n other firms. This yields a lower profit $\pi_{-acq,S}^A$. The difference between these (multiplied by the probability ρ with which the entrepreneur successfully develops the project) is the “efficiency effect,” first discussed by [Gilbert and Newbery \(1982\)](#) in the context of monopoly persistence due to preemption incentives. However, the expected marginal profit for the entrepreneur from

¹⁷It is straightforward to include acquirer-specific synergy components in our analysis such that acquisitions can also occur for synergistic reasons. Doing so does not qualitatively change our theoretical predictions.

¹⁸Note that this price is the same as that of an acquiring incumbent making a take-it-or-leave-it offer to the entrepreneur in a bilateral bargaining game. It is also the same price as that resulting from a bidding contest between the acquiring incumbent and an outside bidder without an overlapping existing product. Such an outside bidder would face exactly the same development decision as the entrepreneur in $t = 1$ and have the same valuation. Our takeover price assumption also means that the entrepreneur has no more incentive to innovate in the first place than it would if acquisitions were impossible. As we discuss in Section 4, in a more general model, the existence of the acquisition exit option may be valuable enough to increase ex-ante innovation incentives. Finally, although different assumptions regarding the number of potential bidders or the relative bargaining weights of acquirer and target influence the takeover price P , they do not affect whether or not the acquisition takes place.

continuing development ($d^E = 1$) given by $\rho\Delta^E - k$ is larger than the liquidation value L that the acquiring incumbent ($d^A = 0$) would obtain. This difference is the “replacement effect.” It decreases the incentive to acquire because when paying P the acquirer still needs to compensate the entrepreneur for her higher valuation.

Third, for $k \leq k^A$, both acquired and non-acquired firms develop the project. The acquisition occurs if

$$(8) \quad \underbrace{\pi_{acq,F}^A - \pi_{-acq,S}^A}_{\text{efficiency effect}} \geq \underbrace{\Delta_E - \Delta_A}_{\text{replacement effect}}$$

Here, the “replacement effect” is the difference in marginal project development gains because both parties develop the project.¹⁹

Proposition 4 (Acquisition Decisions). *In $t = 0$, the acquirer acquires the entrepreneur if*

- $k^E \geq k > k^A$: $\rho(\pi_{acq,F}^A - \pi_{-acq,S}^A) \geq \rho\Delta^E - k - L$ (“Acquire to Kill”)
- $k \leq k^A$: $\pi_{acq,F}^A - \pi_{-acq,S}^A \geq \Delta^E - \Delta^A$ (“Acquire to Continue”)

Ownership is indeterminate if $k > k^E$.

Figure 3 plots the acquirer’s payoffs from different acquisition choices for specific parameter values for which the efficiency effect is always stronger than the replacement effect. If k is above k^E , the acquirer is indifferent between “Don’t Acquire” and “Acquire to Kill,” and thus the two lines overlap. In the intermediate region where k is between k^E and k^A , it is optimal for the acquirer to “Acquire to Kill” whereas for particularly promising projects for which $k \leq k^A$, he will choose “Acquire to Continue.”

[Insert FIGURE 3 Here.]

To summarize, in our model acquisitions take place when the “efficiency effect” is sufficiently large relative to the “replacement effect.” Even though the entrepreneur generally has

¹⁹Under symmetric (differentiated) Bertrand competition, the efficiency effect is always larger than the replacement effect in this region, but this is not necessarily true under Cournot competition. In the latter case, the acquirer can have a lower valuation than the entrepreneur, and therefore the entrepreneur retains the project.

a higher propensity for developing a project (due to the “replacement effect”), acquisitions occur because they prevent the entrepreneur from reducing the existing profits of the acquirer (“efficiency effect”). Note further that even though the acquirer only has a strictly positive incentive to acquire the entrepreneur when project development is sufficiently profitable ($k \leq k^E$ so $\rho\Delta^E - k$ is positive), it is still true that the acquirer has a weaker incentive to develop the projects he acquires than the entrepreneur does with the projects she retains. This is because whenever the acquirer has a strictly positive incentive to acquire, the entrepreneur always develops any project she retains whereas the acquirer only ends up developing a subset of his acquired projects ($k \leq k^A$).

2. Background and Data

To empirically document the phenomenon of killer acquisitions, we use the setting of drug development. Adequately testing the predictions of our theoretical framework requires comprehensive data on project level outcomes, for both acquired and non-acquired projects. We also need to finely measure overlap between acquirer and target firms and to capture market and technological competition. As described in detail below, pharmaceutical project development offers all of these features.

2.1. Drug Development Background

New pharmaceutical products, or drugs, are developed following a set of structured milestones en route to commercialization. First, firms identify potential drug compounds through routinized discovery processes. Then, for any promising molecules, firms run preliminary screening in vitro and/or in vivo to explore both efficacy and toxicity prior to any clinical trials in humans. Following these pre-clinical evaluations, for promising drug projects, firms undergo three phases of clinical trials in human subjects (Phase I, II, and III).²⁰ In tandem with these regimented clinical tests, firms engage in additional commercialization activities, including patent applications, regulatory filings in the U.S. and abroad, applications

²⁰Drug developers must submit an 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.

for coverage to various public and private insurance agencies, and, finally, launching of the product in various countries around the world.

Each component of drug development represents significant expenditure; for example, clinical trials cost in the tens of millions USD (Morgan et al., 2011). Given the lengthy pre-approval process, post-approval, patented drugs usually only have a few years to earn monopoly profits before patent expiration and generic entry (Scherer, 1993). Along with the routinized nature of drug development, which allows us to observe both development and (some) termination events, these features—significant cost of development milestones with a short window to recoup said costs—allow us to credibly interpret development events as significant markers of project-level development. Observing key development milestones—or lack thereof—at the project level is crucial to identifying killer acquisitions.

2.2. Drug Development Data

To build our analytical dataset at the drug project level, we use Pharmaprojects from Pharma intelligence, which has been used by other economists studying drug development (for example, Branstetter et al. (2014)). Pharmaprojects is a comprehensive dataset that tracks drug projects from a very early stage through to launch or discontinuation. Pharmaprojects provides nearly universal coverage of all candidate drugs developed or under development for eventual sale in the U.S. market, along with the originating firm associated with each drug project.²¹

Importantly for our purposes, the dataset also includes information about each drug’s intended therapeutic market (e.g., “osteoporosis”) and mechanism of action (e.g., “calcium channel antagonist”), which we use to identify competing projects and products. Pharmaprojects also documents the occurrence and timing of key product development milestones (e.g., “new patent application”, “target identified”, “first launch”, and “additional registration for clinical trial”), including drug discontinuations. As detailed in Appendix B, we code all of the 28 types of events tracked by Pharmaprojects into three categories: development events, termination events, and neutral events that impart little information regarding the progress

²¹In the raw dataset, Pharmaprojects typically updates the “originator” firm name associated with each project when and if it is acquired. We therefore re-constructed the historical originator firm using text descriptions included in the dataset. More details are provided in Appendix B.

(or termination) of drug development. Development events reflect both research and development milestones and important steps in the commercialization process for the underlying drug project. Pharmaprojects therefore allows us to identify and capture milestones that signify development of a drug, including, but not limited to, progress through clinical trials.

[Insert TABLE 1 Here.]

Pharmaprojects provides complete development information for 55,894 projects initiated between 1989 and 2017, inclusive. From our sample we exclude projects initiated in 2011 or later so that we are able to observe project development events, discontinuations, and any acquisitions for each project in our sample for at least five full years from initiation. Our analytical sample therefore covers projects initiated between 1989 and 2010, or 35,712 drug projects, originated by 6,709 firms. Table 1 provides a tabulation of project initiation over the entire time span of our sample. Over the period of our analysis, drug project initiations increase from around 1,000 per year in the 1990s to around 2,000 projects per year in more recent periods. Table 1 also tabulates projects by broad disease groups. The largest disease areas include therapies targeting cancer (4,673 or 13% of the sample) and neurological conditions (4,049 or 11% of the sample).

Figure 4 plots the distribution of the number of new drugs originated by a company between 1989 and 2010. We find that 43% of companies originate only one drug over this period (and 60% originate two projects or fewer). These patterns align with general perceptions of drug development over this period: small innovator firms initiate innovative drug projects which are subsequently developed by large, commercialization-focused incumbent firms (Cockburn, 2004).

[Insert FIGURE 4 Here.]

We supplement the Pharmaprojects data with Pharma intelligence's Trialrove data on clinical trials, linked at the project level. Drug clinical trials comprise three main phases: Phase I trials, which are small (20 and 100 healthy volunteers), short, and are intended to test safety; Phase II trials, which are larger (100s of affected patients), typically randomized control trials lasting up to two years, and are intended to test efficacy; and Phase III trials,

which are expanded versions of Phase II trials, involving hundreds or thousands of participants, and typically lasting one to four years (US Food and Drug Administration, 2017). Following successful trials, firms may submit a New Drug Application (NDA) to the FDA, which then determines if, and under what conditions, the drug can be marketed to U.S. patients. We use Trialrove data to identify the initiation of clinical trials by phase, including the timing of trial initiation.

Notably, clinical trial phase data are widely available only from 1997 onward, when the U.S. Federal government first mandated the National Institutes of Health (NIH) to collect and make publicly available a comprehensive, clinical trials database.²² Therefore, we have comprehensive trial phase data only for a limited subset of all projects in our sample, specifically those initiated after 1997. Within this limited sample, we identify projects for which we observe the start date of Phase I trials and track their progression, following prior studies that use progression through phases of clinical trials as a measure of project development (Krieger, 2017; Guedj and Scharfstein, 2004).

2.3. Acquisition Data

We collect acquisition data from three sources. The first source is the M&A data from Thomson Reuters SDC Platinum, from which we extract all announced and completed M&As (with complete information on acquirer and target firms) and announced and effective dates. To supplement the SDC M&A data, the second data source of acquisition information we use is Thomson Reuters RecapIQ (now Cortellis Deals Intelligence). RecapIQ focuses on deals in the biotechnology industry, collecting detailed information from company press releases, SEC filings, and company voluntary disclosures. Our third source of acquisition data is the SDC VentureXpert database, which covers mainly VC-backed, early stage startups. Using VentureXpert we identify entrepreneurial companies that exited via an acquisition. However, since VentureXpert does not provide details on the acquirer and dates of the acquisition, we manually collect that information.

Armed with the original acquisition events compiled from multiple data sources, we then

²²More details on the timeline of publicly available clinical trials database can be found at <http://www.clinicaltrials.gov>.

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 on the deal dates, we drop duplicated acquisition events (possibly due to overlap of the datasets). To the best of our knowledge, this is the most comprehensive database on acquisitions in the pharmaceutical industry.²³

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

The merged drug development and acquisition data show an active acquisition market in the pharmaceutical industry, with about 24% of drug projects acquired at some point during development. As tabulated in Table 1, the rate of acquisition is lower for drugs originated more recently, which is likely a result of right truncation. That is, acquisitions typically occur a few years into development, and some acquisitions might have not been realized by the time of data construction for more recent projects.

3. Empirical Analysis

3.1. Empirical Specification

The model in Section 1 provides several predictions for when projects will be less likely to be developed post-acquisition. The first main implication of the theoretical framework (building from Proposition 1) is that if the target project overlaps with projects or products marketed by the acquirer, the acquirer has weaker incentives to continue development. We therefore need a measure of overlap between the target’s and the acquirer’s projects to test

²³Each of the three data sources, SDC M&A Database, RecapIQ, and VentureXpert, contributes at least 10% of cases in the final database.

for differences in the likelihood of development across overlapping acquired, non-overlapping acquired, and non-acquired projects post-acquisition.

We measure overlap between a drug project and the acquiring firm based on a combination of the market and technology categorizations of the focal product. To categorize a drug project’s “market” we use its therapeutic class, which is the disease or condition the therapy targets (e.g., antihypertensive). We use Pharmaprojects therapeutic categories, which represent 230 different narrowly defined therapeutic markets. 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). There are 2,749 potential mechanisms in Pharmaprojects. If the acquiring firm has an active project in the same market using the same technology as that of the acquired drug project, we categorize the project as overlapping with the acquirer. This means that our measure is quite tightly defined. The logic for measuring overlap this narrowly is to ensure that we are capturing potential substitutes rather than complements. If we instead simply used the market (therapeutic class) criterion we would also capture drugs that are complementary as they capture different sub-markets (i.e., treat different symptoms of the same disease). As outlined in Table 1, about one fifth of acquired drug projects are overlapping with the acquirer. Table 1 also provides some information on the narrow categories we use to define overlap. For example, there are 783 market-technology (or therapeutic class - mechanism of action) combinations in the disease group of “Anti-Cancer” alone.

For our main empirical analyses, we use panel data of drug development events. We compare overlapping acquired, non-overlapping acquired, and non-acquired projects. A project is included in the sample from the origination year and is removed from the sample after a successful U.S. launch if any. The empirical specification is as follows,

(9)

$$\begin{aligned}
 Development_{i,t} = & \beta \cdot I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i + \gamma_1 \cdot I(Acquired)_i \times I(Post)_{i,t} \\
 & + \gamma_2 \cdot I(Acquired)_i \times I(Overlap)_i + \gamma_3 \cdot I(Acquired)_i \\
 & + \alpha_{age} + \alpha_{vintage} + \varepsilon_{i,t},
 \end{aligned}$$

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

In this panel specification, the interaction term $I(Acquired)_i \times I(Post)_{i,t}$ captures the change of development progress for all acquired drug projects in the years after the acquisition. The term $I(Acquired)_i \times I(Overlap)_i$ captures the overall development conditions for drugs acquired by overlapping buyers in years before the acquisition. The key term for our test is the triple interaction term $I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i$, which captures the additional change in development event probability for acquisition cases when the target and the acquirer overlap. Our model predicts a negative coefficient, consistent with the interpretation that when acquired projects overlap with the acquirer’s portfolio they are more likely to be terminated.

In an ideal setting where terminations are reported accurately and in a timely manner, we would use a survival analysis to test if and when drugs are terminated in development. However, project terminations are typically not observed, either at a specific point in time or at all. Hence, in the above main specification, we are effectively using a lack of development events to proxy for termination. We use a panel structure to test for the likelihood of observed, active development of a project. There are additional advantages to a panel structure which are not possible in a survival analysis structure, including the ability to account for differences between acquired and non-acquired projects, as well as the ability to account for pre-acquisition differences between overlapping and non-overlapping projects. Furthermore, we perform additional analysis to address the issue of accurately capturing drug terminations (as outlined in the section following our main results).

3.2. Main Results: Development of Drug Projects Post-Acquisition

Table 2 presents the regression results. In column (1), β estimate of -0.019 is statistically significant, meaning that acquired drug projects that overlap with the acquirers’ pipelines

are 1.9% less likely to have a development event during the years post-acquisition. The unconditional probability of having a development event in the sample is 9.1%, leading the economic magnitude to be roughly $(1.9\%+1.7\%)/9.1\% = 39.6\%$. That is, being acquired by a firm with an overlapping project is associated with a 39.6% lower development probability. In column (2) we incorporate drug-level fixed effects in the regression analysis to absorb variation due to unobservable drug-project-specific characteristics. We find that the estimate of β is statistically significant and of similar economic magnitude to column (1). In this setting, being acquired by a firm with an overlapping project is associated with a 28.9% decrease in development rate.

One potential concern for our analysis is right truncation, especially given the long development timelines for pharmaceuticals (e.g., two to seven years for Phase I, II, and III (US Food and Drug Administration, 2017)). To ensure this is not an issue, we re-run our main analysis on samples of projects originating before 2000 (columns (3) and (4)). Our results are robust to using the pre-2000 sample.

[Insert TABLE 2 Here.]

Beyond our main finding on overlap, Table 2 also includes several other results that warrant discussion. The γ_1 coefficient associated with $I(Acquired)_i \times I(Post)_{i,t}$ is -0.017, meaning a lower probability of development or milestone events post-acquisition. This is surprising if we were to assume, as is plausible, that typically higher-quality projects are acquired and that development synergies materialize post-acquisition (Andrade, Mitchell and Stafford, 2001). This is, however, consistent with an interpretation that differences between acquirers (which are typically larger firms) and targets (which are typically smaller) may affect development choices. For instance, Guedj and Scharfstein (2004) argue and show that larger firms more efficiently terminate projects than small firms because their managers have lower private benefits from continuing development. Alternatively, this finding is also consistent with agency problems inherent in the organization of large firms (Seru, 2014). One additional explanation could be that our measure of overlap is quite tight (same therapeutic class and same mechanism of action) and therefore some of the non-overlapping acquisitions are in fact “killer.” However, to be clear, our main test is based on the triple interaction

term: based on these estimates, projects acquired by buyers that have an overlapping project are $\frac{3}{4}$ as likely to have a development event post-acquisition ($5.7/7.6 = 75\%$) than those acquired by buyers without an overlapping project. A further reassuring result is that the dummy variables on $I(Acquired)_i \times I(Overlap)_i$ and $I(Acquired)$ do not carry any load in the regressions, meaning that acquired drugs do not appear to have a significantly different unconditional likelihood of development pre-acquisition.

Note that if we were to simply compare development for acquired and non-acquired drugs to assess “killer acquisitions,” a valid critique would be that the result could be due to the buyer’s inability to identify profitable projects and to integrate them internally, i.e., some form of informational asymmetry. However, following this logic, we would expect overlap to mitigate rather than intensify our results, as having developed a closely related project should at least partially resolve information asymmetries. In fact, acquired projects with overlap are less likely to be developed post-acquisition, inconsistent with informational asymmetry based explanations.

We interpret the decreased development rates after a project is acquired by firms with an overlapping project as attributable to the desire to preempt competition, i.e., as evidence for “killer acquisitions.” Yet, the target firms in our main analysis in Table 2 may be developing multiple projects. An alternative explanation for our results could therefore be that acquirer firms develop only the most promising of the acquired projects of multi-project targets and shut the others down. For multi-product targets, the discontinuation of overlapping projects may be a side-effect of the goal of the acquisition.

To investigate this, we only analyze deals with single-drug targets—that is, we try to identify post-acquisition development only for the cases in which the target owns one and only one drug at the time of acquisition. In doing so, we focus on the small sub-sample of drug projects that are initiated in single-project companies and examine how their development progresses are affected by acquisitions. If project selection is driving our results, we should expect that our focal patterns are much less prevalent among single-project acquisitions. As outlined in Table 2 columns (5) and (6), we find the post-acquisition development rate in cases involving single-drug targets to be similar to our main results. The baseline development rate is 19.5% in this subsample, significantly higher than 9.1% in the full sample, consistent with

Guedj and Scharfstein (2004). In the specification with both age and project fixed effects, the estimate of -0.177 means acquired single-project targets are 17.7% less likely to have a development event if they are acquired by a firm with an overlapping project.

Overall, Table 2 provides evidence that acquired drug development projects are less likely to be developed under the possession of an acquirer that has potentially competitive projects, consistent with the model prediction.

Thus far, we infer project discontinuation from decreased likelihood of development events for acquired overlapping projects. However, one concern is that this decrease could reflect different patterns of development for acquired overlapping projects instead of actual project terminations. Acquiring firms might delay development (or accelerate it), or they may be slower at developing, which would result in decreased development events over the observed project life cycle post-acquisition. Although our analysis to investigate right truncation concerns suggests that this is not an issue, we further address this concern by running two analyses of post-acquisition development that compare overlapping and non-overlapping acquisitions.

[Insert TABLE 3 Here.]

First, we look at whether a project is ever developed post-acquisition. A purposefully terminated project should incur no post-acquisition development events; hence, we would expect the likelihood of never having a post-acquisition development event to be significantly higher for overlapping acquisitions. To test this, we temporarily focus only on the sample of acquired projects and examine the possibility that they never incur any development events post-acquisition. For each acquired project, we create one pre-acquisition observation and one post-acquisition observation. The outcome variable is if the project experiences no development milestone during the pre- or post-acquisition period. Table 3 column (1) presents this analysis. We find overlapping projects are 4.9% more likely to have no development events—that is, to appear to be fully discontinued—in the post-acquisition period compared to non-overlapping projects.

Second, we aim to confirm that the main results in Table 2 are driven by acquired projects that never incur any development events post-acquisition, i.e., terminated projects. To

do so, we re-run our main analyses focusing on acquired projects that have at least one development event post-acquisition, i.e., we take out the “never-developed” projects identified in the first analysis (from Table 3 column (1)). If terminated projects are driving our main findings, after removing them from the analysis we should find no significant differences remain between acquired-overlap and acquired-non-overlap projects. In other words, a null result after we take out projects that are never developed post-acquisition is consistent with our predictions. In Table 3 column (2) we find no significant differences in likelihood of development events between acquired-overlap and acquired-non-overlap projects. Overall, then, terminated projects—those with no post-acquisition development—appear to be driving our main results.

3.3. The Role of Market Competition

To investigate the predictions of Proposition 2, we examine how our results change across different levels of competition. We measure competition as the count of drugs currently marketed or under development that overlap with the target product. Specifically, we count launched products in the same market using the same technology as the focal project (our measure of “existing product” competition) or projects under development in the same market using the same technology (our measure of “pipeline” competition).²⁴

[Insert TABLE 4 Here.]

Table 4 presents the regression results which examine whether the post-acquisition development pattern of acquired projects varies under different competition environments. We categorize drug development projects into high and low competition by the sample median of competition measures described above. In columns (1) to (4), the competition measure is calculated using existing, launched products while in columns (5) to (8), the measure is

²⁴Note that each drug product can fall into multiple technologies (mechanisms of action) and multiple intended markets (therapeutic classes). In the Pharmaproject 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-market categories in which it falls. For our measures of competition for the focal projects, we use the technology-market category with the most competition. That is, if a project falls into two technology-market categories, one with 0 pipeline competitors and one with 5, we use 5.

calculated using projects in the development pipeline.²⁵

The results suggest that the decreased likelihood of development during the post-acquisition period for overlapping projects concentrates in product markets with relatively low competition. Comparing columns (1) and (3), development of an overlapping acquired drug in the low competition environment decreases by 2.1%, while under high competition, the coefficient is -0.002, which is both insignificant and economically negligible. Similar patterns arise when we compare the parallel regressions with project fixed effects (columns (2) and (4)). The results in columns (5) to (8), which measure competition in the drug development pipeline, convey a similar message. However, the economic significance is slightly weaker in the setting with project-level fixed effects.

Thus, our analysis highlights a positive reinforcement loop that competition provides. If incumbents already face significant existing competition, acquired projects are not significantly more frequently discontinued than independent projects. Thus, in addition to well-known benefits for consumers, increased competition also deters incumbents from acquiring and terminating the projects of potential future competitors.

3.4. Heterogeneity across Patent Expiry

To further explore how overlap relates to project development and to provide empirical evidence for the theoretical predictions of Proposition 3, we investigate how the time remaining on acquirer patents influences the results in Table 2. This additional analysis focuses only on projects that are acquired and overlap with the acquirer’s project(s). For each of those projects, we identify the patents associated with the relevant (overlapping) approved drugs of the acquiring firm using FDA Orange Book data (which are linked into Pharmaprojects) and then merged with United States Patent and Trademark Office (USPTO) data on patent filing dates. Proposition 3 predicts that the likelihood of termination declines (or likelihood of development increases) as the acquirer’s patent nears expiry, i.e., when the expected remaining profits are comparatively small. Following that logic, we expect the negative

²⁵As a result of various reporting requirements tied to regulation, pharmaceutical firms can observe what other firms have in their development pipelines. Further, data providers (including Pharmaprojects) aggregate and sell pipeline data to firms. Because competitor’s pipelines are observable, we use pipeline data as a measure of future or expected competition.

relationship between overlap and development to be most pronounced among acquirers with overlapping drug patents with a long remaining patent life and to be mitigated when the acquirer’s relevant patent is near expiry.

[Insert TABLE 5 Here.]

Table 5 presents the results on development outcomes among acquisitions with overlapping acquirers. The key result is $I(Post) \times I(NearPatentExpiry)$ which contrasts those with patents near expiry (i.e., within five years) with those with longer remaining patent life. Consistent with our predictions, we find that if the relevant acquirer patents are near expiry, the decrease in development appears to be mitigated. That is, for projects that overlap with acquirer drugs, those for which the acquirer patents are near expiry are more likely to have development events post-acquisition compared to projects that overlap with acquirer drugs and with patents relatively far from expiration. In other words, the decrease in development post-acquisition is concentrated among overlapping projects acquired by firms with relatively long life left on the overlapping patents.

3.5. Evidence from Clinical Trials

To supplement the preceding analyses of development events, we also examine the likelihood that a project continues in the clinical trials process. In addition to providing robustness, analyzing progression through the stages of clinical trials is useful because it ties closely with related research on drug development (Guedj and Scharfstein, 2004; Krieger, 2017) and because it allows us to focus, albeit narrowly, on drugs at the same stage of development, i.e., moving forward from one particular phase to another. Focusing in this way helps to alleviate concerns that our main results are driven by differences in stage of development across projects that might remain after controlling for age and vintage. Because our main analyses include a much larger sample of projects and also include many additional key development events besides trial starts (e.g., patent applications, launches), we use the clinical trial analysis as supplementary material to ensure robustness.

In this analysis, we focus on whether drugs that start Phase I clinical trials and are acquired by firms with overlapping projects are less likely to subsequently start Phase II

trials, by examining the following specification,

$$(10) \text{ PhaseII}_i = \beta \cdot I(\text{Acquired PI})_i + \gamma \cdot I(\text{Acquired PI})_i \times I(\text{Overlap})_i + \alpha_{\text{vintage}} + \varepsilon_i.$$

In this analysis, each observation is a drug project which we observed initiate Phase I clinical trials. The key variables are $I(\text{Acquired PI})$, which indicates whether the drug is acquired during Phase I trials, and $I(\text{Acquired PI}) \times I(\text{Overlap})$, which, as before, indicates if the acquisition was made by an acquirer with overlapping projects (same therapeutic market and same mechanism of action). The analysis is performed on the subsample for which information about Phase I start dates is available. As in our previous analyses, we limit the sample to those projects started before 2011 to ensure sufficient time to observe an acquisition and, specific to this analysis, to give the analyzed projects sufficient time to enter Phase II trials.

[Insert TABLE 6 Here.]

Table 6 presents the clinical trial based regression results. Compared to projects that aren't acquired in Phase I, those that are acquired are less likely to move forward into Phase II trials. Further, this relationship is stronger when the acquirer has overlapping projects. In terms of economic magnitude, in column (2), the decreased probability of -0.254 (or 25.4%) is 48.7% of the base rate (for firms that performed Phase I trials) of entering Phase II of 52.1%. Further, being acquired by an acquirer with overlapping products decreases the likelihood of starting Phase II trials by an additional 14.4%. Corresponding to the analysis on competition in Table 4, columns (3) through (6) examine how competition conditions the results. Similar to development events, we find that our results for clinical trials are concentrated in markets with low competition.

3.6. Alternative Explanations

In this section we sharpen our empirical analysis by addressing several potential alternative explanations. Importantly, a plausible alternative explanation would have to explain not just why acquired drug projects are more likely to be terminated, but why in particular

overlapping acquired drug projects are more likely to be terminated than non-acquired or non-overlapping acquired drug projects.

3.6.1. Information Asymmetry in the Acquisition Market. Focusing on overlapping projects means that asymmetric information or “market for lemons” type arguments are an implausible candidate explanation. While an acquiring firm likely knows less than the target about the quality of the target’s projects and may therefore sometimes buy lemons, this asymmetry should be much lower when the acquirer has its own overlapping projects and therefore has knowledge of both the therapeutic category and the mechanism of action of the drug candidate. Our main results are therefore unlikely to be a simple market for lemons story.

3.6.2. Optimal Project Selection. Given that some targets are multi-project, our results could reflect acquirer firms choosing optimally to develop only the most promising projects and to shut down the rest, in particular those that overlap with their own projects. However, when we investigated single-project acquisitions (in Table 2), we found results similar to those in the full sample. Our main results are therefore unlikely to be driven by optimal project selection.

3.6.3. Redeployment of Technologies. A remaining alternative explanation for our results is that firms acquire targets not for their projects but for their technologies. Under such circumstances, acquirers would shut down the target’s projects and redeploy the technologies to more productive ends, i.e., to start newer, more promising drug projects. The possibility of technology redeployment as a motive poses a concern for us since it is consistent (or at least not inconsistent) with our findings on overlap. That is, overlapping projects are the most likely to represent useful and redeployable technologies.

We assess whether and how the technologies of acquired projects are redeployed by exploiting molecule-level information for each drug candidate. To do so, we use each drug’s chemical structure and compare the structure of acquired projects to those developed by the acquirer pre- and post-acquisition. We assess whether acquirer firms’ projects post-acquisition are more similar to the acquired project than their pre-acquisition drugs. To

measure similarity, we follow recent research in economics by [Krieger et al. \(2017\)](#) and use the Tanimoto distance. The Tanimoto distance between two chemical structures is the most commonly used method to measure similarity in the chemical informatics literature ([Nikolova and Jaworska, 2003](#)). It is defined as the proportion of chemical fragments shared by any two chemicals divided by their union and it is bounded by 0 and 1, with 0 indicating the pair share no common chemical fragments. If acquired drugs are redeployed, one would expect new, post-acquisition projects in acquirer firms to be more similar to the acquired project than to their pre-acquisition projects.

[Insert TABLE 7 Here.]

Table 7 Panel A outlines chemical similarities to the acquired drug for drugs initiated by the acquirer post-acquisition compared to pre-acquisition drugs. In this analysis, each observation is a pair consisting of an acquired drug and a drug that was initiated by the acquirer within the 10-year window (i.e., ± 5 years) around the acquisition. We are particularly interested in the coefficient associated with $I(Post) \times Overlap$. Contrary to a redeployment explanation, we find that drugs developed in acquirer firms post the acquisition of a drug actually are not more similar to the acquired overlapping drug. The economic magnitude of 0.001 is also negligible compared to the global similarity mean of 0.133. Overall, these results do not support a technology redeployment explanation.

3.6.4. Redeployment of Human Capital. Similar to technology redeployment, the key motivation behind acquisitions could be human capital such as the research team or other key individuals ([Ouimet and Zarutskie, 2011](#)). Under this view, the lack of development of acquired, overlapping projects could be a by-product of acquiring and efficiently redeploying valuable human capital post-acquisition within the acquired company. Again, we would expect that human capital underpinning overlapping projects would be the most useful for the acquiring firm, and so this alternative explanation applies to our main analyses.

Before empirically addressing human capital, it is worth highlighting that acquisition-as-hiring might not be as common in the pharmaceutical industry as in other industries. The pharmaceutical industry is typically and uniquely project-driven ([Gompers et al., 2016](#)),

compared to other industries where startups are valued based more on human capital. Further, human capital and technological are closely linked in drug development. Since we didn't find any evidence of for technology redeployment, it suggests human capital is unlikely to be driving our main results. Yet, to further investigate human capital redeployment independent of technology redeployment, we explore inventor mobility and productivity post-acquisition.

To measure the reallocation of human capital subsequent to acquisition events and any changes in inventor productivity associated with acquisition, we use the target firm inventors patenting patterns. We track inventors across firms using the Harvard Patent Dataverse (see [Lai et al. \(2009\)](#) for details). This database includes disambiguated inventor names and organizational affiliations (via patent assignees), enabling us to track individuals over time and across organizations following a similar approach to [Bernstein \(2015\)](#) and [Brav et al. \(2017\)](#). Specifically, we construct a list of target firm pre-acquisition inventors by identifying those who filed at least one patent within the five-year window pre-acquisition. We track how many of the target firm inventors remain in the acquiring firm and whether there is any evidence that those who remain are efficiently redeployed. Human capital redeployment would predict both that a significant proportion of pre-acquisition inventors in the target firm should be retained and that the inventors that stay should become more productive as they are moved from the terminated projects.

Table 7 Panel B shows the human capital results. First, just 22% of pre-acquisition inventors move to the acquirer after the acquisition, while 78% move to other firms. Second, while those two sets of inventors are statistically comparable before the acquisition event, patenting roughly 4.5 times for the target within the 5 years leading up to the acquisition, post-acquisition, we find little evidence that the retained inventors become 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, inventors who move to other firms have a smaller productivity drop ($< 10\%$).

One word of caution about these results is that we cannot link target firm patents to a specific drug project because of their early stage.²⁶ As a result, we are not able to identify

²⁶That information is typically disclosed late in the drug development stage when the FDA requires systematic reporting. Patents linked at a product level are available systematically in the FDA Orange Book only for approved drugs.

whether inventors are associated with projects that are shut down. However, if we focus on cases with a single-drug target, we find that an even larger proportion of inventors leave the combined firm after the acquisition (although the sample becomes quite small).

4. Discussion

4.1. Antitrust and FTC Review Thresholds

Killer acquisitions significantly decrease the number of developed drugs and thus contribute to lower product market competition, potentially causing consumer harm. As a result, the anticompetitive effect of killer acquisitions should make them subject to antitrust scrutiny. However, as shown in our paper, many such acquisitions occur when the technology or project is still at a nascent stage and thus many such acquisitions are exempted from the pre-merger review rule of the FTC under the Hart-Scott-Rodino (HSR) Antitrust Improvements Act of 1976. In 2000, Congress amended the HSR statute to require the annual adjustment of these thresholds based on the change in gross national product. As a result, reportability under the act changes from year to year as the statutory thresholds adjust. Under HSR, deals under \$50 million (all amounts referenced here are annually adjusted) are not required to submit filings for pre-merger review. For deals between \$50 million and \$200 million, the size-of-the-person test is conducted, and if the larger party has less than \$100 million in assets or sales and the smaller party has less than \$10 million in assets, the deal does not need to be reviewed by the FTC. Since the size-of-the-person test is typically not satisfied for smaller pharmaceutical companies, acquisitions below \$200 million will usually not be investigated. [Wollmann \(2018\)](#) shows that these review exemptions can result in stealth consolidation: anticompetitive acquisitions whose small size enables them to escape regulatory scrutiny but whose cumulative effect is large.

Do acquirers conducting killer acquisitions attempt to avoid FTC review by making acquisition deals that do not trigger FTC reporting requirements under HSR? We answer this question by examining acquisitions around the HSR review threshold and comparing the project development decisions of transactions above and below the threshold. If firms conduct killer acquisitions intentionally under the radar of the FTC, we would expect to

see two empirical patterns. First, there should be bunching of acquisitions, particularly acquisitions of overlapping targets, just below the threshold. Second, for below-threshold deals, the project termination rate should be higher and the launch rate lower.

[Insert **FIGURE 5** Here.]

In Figure 5 we plot the distribution of acquisition sizes for a narrow window around the HSR review threshold, specifically, $[-5\%, 0]$ and just above it $([0, 5\%]$. The size of the acquisition is proxied using the acquisition deal amount. We also categorize acquisitions into acquisitions of non-overlapping targets (left panel) and acquisitions of overlapping targets (right panel). We observe a clear bunching pattern of deals clustering right below the review threshold, but this pattern is only detected for deals in which the target has projects that overlap with the acquirer, i.e., “killer acquisition” suspects.

[Insert **TABLE 8** Here.]

In Table 8, we test the termination and launching rates of acquisitions around the threshold. We construct two buckets which include all acquisitions with a transaction value just below the FTC review threshold. The survival rate of below-threshold acquisitions is drastically lower than those right above the threshold. Specifically, we find that the eventual product launch rate is much lower (1.8% versus 9.1%) and the discontinuation rate is much higher (94.6% versus 83.3%). Although this analysis is simple and purely descriptive, these patterns are consistent with acquirers conducting more killer acquisitions in situations in which they can expect to avoid FTC scrutiny.

4.2. Frequency and Importance of Killer Acquisitions

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

In particular, we document that when an acquired project overlaps with a product in the acquirer’s existing product portfolio, the project is less likely to be continued: acquired

projects with overlap (25.5% of acquired projects) continue at an adjusted rate of 5.7%, while acquired projects without overlap (74.5% of acquired projects) continue development at an adjusted rate of 7.6%. Given the reduction in likelihood of development, it is natural to ask how many of these acquisitions of overlapping projects are purely killer acquisitions. To roughly calculate this number, assume that there are two types of acquisitions that fall into the acquired with overlap category: killer acquisitions which are purely intended to shut down future competitors (and thus have a 0% likelihood of development) and acquisitions that have the same development likelihood as acquisitions without overlap (7.6%). Given these assumptions and estimates, what would the fraction ν of pure killer acquisitions among transactions with overlap have to be to result in the lower development of acquisitions with overlap (5.7%)? Specifically, we solve the equation $5.7\% = \nu \times 0 + (1 - \nu) \times 7.6\%$ for ν which yields $\nu = 25.0\%$. Therefore, we estimate that 6.4% ($= \nu \times 25.0\%$) of all acquisitions or about 49 ($= 6.4\% \times 758$) acquisitions every year are killer acquisitions.

Note that these back-of-the-envelope calculations provide a lower bound for the actual number of killer acquisitions. This is because they assume that killer acquisitions lead to immediate termination and that there are no additional synergies in the development of overlapping drugs. If pure killer acquisitions had a smaller, but positive, likelihood of development, the implied fraction ν of killer acquisitions would have to be even higher to be consistent with our empirical results. Similarly, if there are synergies in the development of overlapping drugs, they would provide a countervailing positive force that masks the observed negative effects on development of acquired projects with overlap relative to those transactions without overlap.

Having quantified the approximate frequency of killer acquisitions, it is natural to ask what this means in terms of innovation and antitrust policy. How would overall development rates in the pharmaceutical industry be affected if antitrust policy directly targeted such killer acquisitions? The average development likelihood in our sample is 8.3%. Consider first the case in which acquisitions of overlapping projects are no longer allowed and that all such projects instead have the same development likelihood (9.3%) as non-acquired projects (56% of all projects). In that case, the number of total drug projects for which development continues would increase by 4.8% ($= \frac{9.3\% - 5.7\%}{8.3\%} \times (1 - 56.1\%) \times 25.5\%$) or by about 7 drug

projects per year ($= 8.3\% \times 4.8\% \times 1,727$) where 1,727 is the yearly average number of projects).

To give some sense of the magnitude of these results, we can compare them to estimates of the effects of targeted innovation policies in the pharmaceutical industry. One policy—considered successful, but also high cost—is the Orphan Drug Act. The policy is focused on encouraging development of drugs for conditions with small patient pools (i.e., “orphan” diseases) by giving firms substantial tax breaks on clinical trials (up to 30 million USD per trial), grants, and extended market exclusivity. There are several hundred relevant diseases, including many cancers. Economic analysis by Yin (2008, 2009) suggests the policy resulted in roughly 25 additional clinical trials per year for 1981 to 1994, with the effect attenuating over time. Eliminating killer acquisitions would result in innovation effects that are, at a lower bound, larger than a quarter of the size of the Orphan Drug Act.

4.3. Ex-ante Innovation Incentives and Welfare

Our theoretical and empirical analysis focuses on the acquisition and project development incentives of incumbents and entrepreneurs. Killer acquisitions have an unambiguously negative effect on consumer surplus if, as in our model, they leave the ex-ante incentives to originate projects unaffected. Both the entrepreneur and the acquiring incumbent, as well as all the other incumbents, are better off when such acquisitions are allowed. But consumers are hurt both by the lack of competition and the elimination of innovative new products. In other words, patients suffer because there are fewer drugs, and the drugs that are developed and brought to market are sold at higher prices.²⁷

A comprehensive welfare analysis of the impact of killer acquisitions is, however, much

²⁷Although killer acquisitions generally reduce consumer surplus, they need not reduce social surplus under a welfare standard that weights consumer surplus and producer surplus equally. This can occur if the entrepreneur’s product does not provide a sufficiently large increase in consumer surplus to fully compensate the loss in producer surplus of the existing incumbents. In Appendix A we derive a sufficient condition under which the loss in consumer surplus resulting from killer acquisitions outweighs the producer surplus gains and thus reduces social welfare overall. As long as there are few existing incumbents and the entrepreneur’s drug project is not too similar to the existing drugs of the incumbents, killer acquisitions reduce not only consumer surplus but also total welfare. Put differently, killer acquisitions of “me-too” drugs (drugs that are very close substitutes) in markets in which there is more than a single incumbent need not be welfare-reducing because they destroy producer surplus of existing incumbents by more than they increase consumer surplus. However, as we show in Appendix A it is precisely in cases in which killer acquisitions do not harm welfare that they are also unlikely to take place.

more difficult given the many different forces involved in the innovation process. In particular, such an analysis would have to quantify, among other factors, the impact on patient mortality, consumer surplus, technological spillovers from innovation, and ex-ante incentives to generate new ideas. As a result, a formal welfare analysis is well beyond the scope of the present paper.

That said, patient mortality, consumer surplus, and technological spillovers are all likely negatively affected by killer acquisitions. At the same time, it is possible that the presence of an acquisition channel also has a positive effect on welfare if the prospect of entrepreneurial exit through acquisition (by an incumbent) spurs 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. Yet, it is important to note that killer acquisitions will motivate such idea origination only if the entrepreneur receives some of the surplus that accrues to the incumbent through the acquisition.²⁸ If the entrepreneur is left with no surplus relative to the standalone value of her project, she will be unaffected by acquisitions and hence will not respond by increasing her innovation efforts. If, on the other hand, 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 acquisition would add to the negative welfare impact of killer acquisitions.²⁹

²⁸For 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.

²⁹[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 when it otherwise would not be.

Because killer acquisitions may motivate ex-ante innovation the overall effect of such acquisitions on social welfare remains unclear. However, we doubt that this acquisition channel which generates significant ex-post inefficiencies resulting from the protection of market power, is indeed the most effective way to motivate ex-ante innovation. This is particularly so because our analysis emphasizes the interplay between competition and innovation and the positive reinforcement loop that competition provides. Recall that killer acquisitions are less likely to occur when incumbents already face significant existing competition. Thus, raising the level of existing competition not only has the well-known immediate benefits for social welfare, but it also deters incumbents from engaging in killer acquisitions of future competitors.

5. Conclusion

In this article we documented that incumbent firms acquire innovative targets and terminate their innovative projects in order to preempt future competition. Empirically, we exploited the setting of drug development, in which we were able to track project development before and after acquisitions. We showed that acquired drugs are less likely to be developed, particularly when they overlap with the acquirer's product portfolio and when the acquirer has strong incentives to protect his existing market power. We also showed that alternative interpretations such as optimal project selection and the redeployment of human or technological capital do not explain our results.

Although our analyses focus on the pharmaceutical sector, the core insights extend beyond that specific setting. Acquisitions are the primary form of startup exit and have become increasingly popular as an exit strategy over time across various industries,³⁰ suggesting that the potentially damaging consequences reach beyond pharmaceuticals. Our results caution against interpreting acquisitions of nascent technologies solely as incumbents' efforts to integrate and foster entrepreneurial innovation. Instead, a significant source fueling this trend may actually be killer acquisitions that harm innovation.

Our results also suggest that antitrust policy should continue to closely scrutinize the

³⁰For 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/>.

impact of acquisitions on corporate innovation, in particular when such acquisitions plausibly prevent the development of future competing products and technologies. The fact that killer acquisitions routinely avoid regulatory scrutiny by acquiring entrepreneurial ventures at transaction values below the HSR review thresholds exacerbates the concern that some anticompetitive behavior currently goes unchecked.

Finally, the magnitude of the Schumpeterian gale of creative destruction—whereby startups’ inventions topple entrenched and less innovative incumbents—may be smaller than previously documented. Rates of innovation may be lower not only because incumbents are reluctant to innovate, but also because incumbent firms with market power acquire innovators to eliminate future competition and thereby inhibit technological progress.

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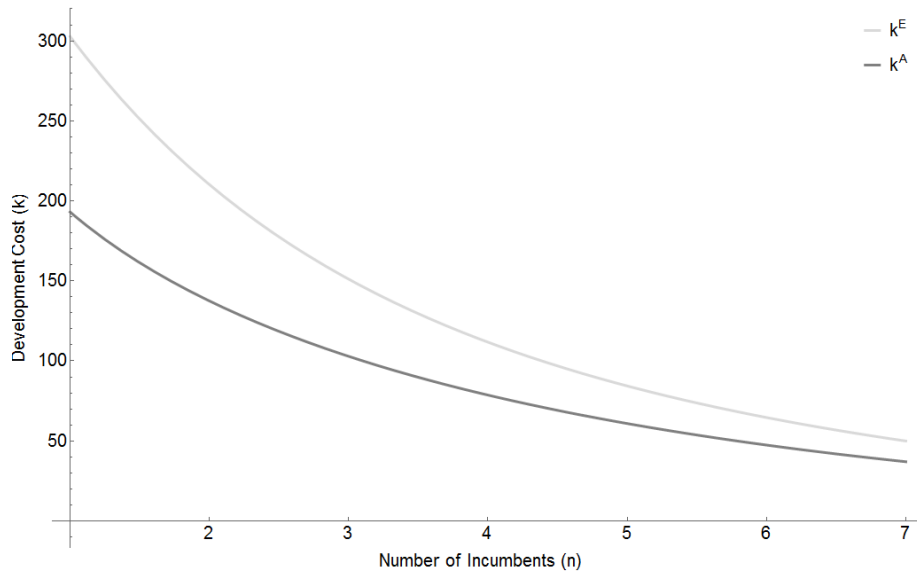
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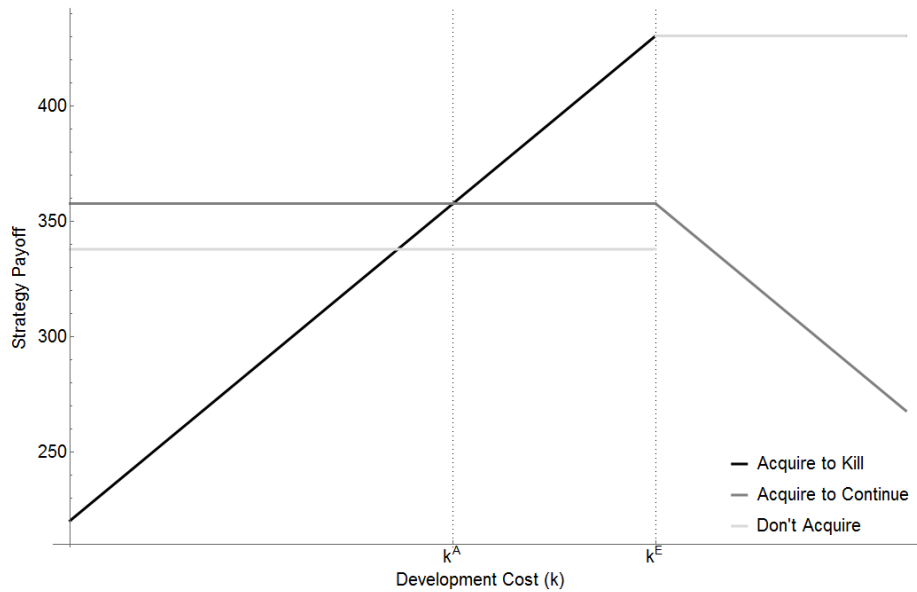
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Figure 2. Development Cost Thresholds and Competition



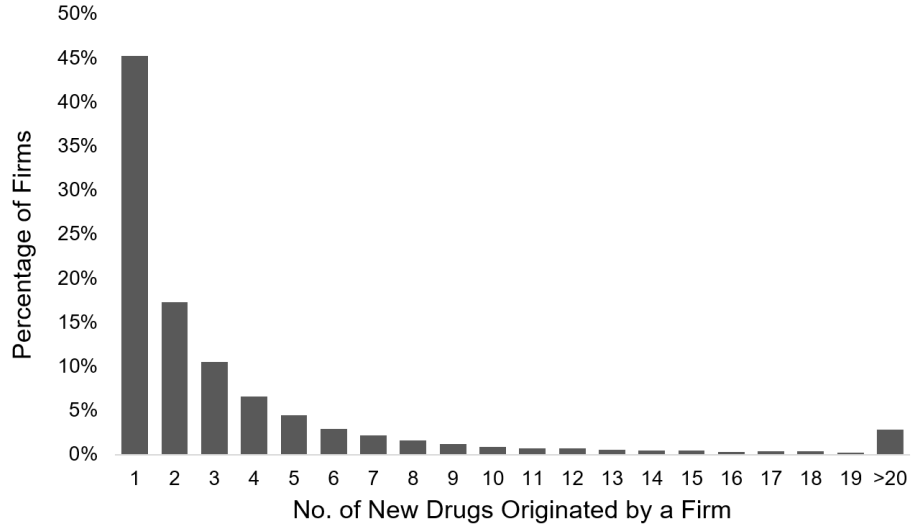
This graph plots the optimal development cost thresholds of the entrepreneur (k^E , light gray) and the acquirer (k^A , dark gray) as a function of the number of incumbents n . Other parameter values are held constant ($\alpha = 100$, $\beta = 4$, $\gamma = 1.5$, $\rho = 0.75$, and $L = 20$).

Figure 3. Strategy Payoffs



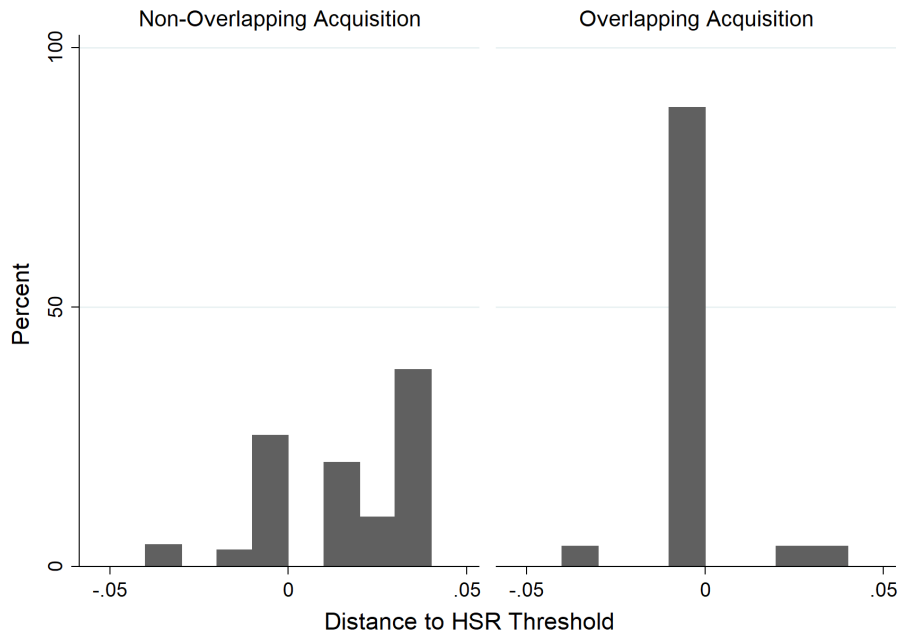
This graph plots the incumbent's payoff from pursuing one of the three acquisition strategies—"Don't Acquire" (light gray), "Acquire to Kill" (black), and "Acquire to Continue" (dark gray)—as a function of the development cost k . Other parameter values are held constant ($\alpha = 100$, $\beta = 4$, $\gamma = 1.5$, $\rho = 0.75$, $L = 20$, and $n = 2$).

Figure 4. 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 2010. We assign a drug to a company if the company was the first to own the drug development project, but we do not assign the drugs that were obtained through acquisitions. The drug origination data are from the Pharmaprojects database.

Figure 5. Acquisition Size Distributions Around HSR Review Threshold



This graph plots the distribution of acquisition size near the Hart-Scott-Rodino review threshold. Acquisitions that fall into the $[-5\%, 5\%]$ around the threshold are kept, and the horizontal axis represents the distance to the review threshold (from -5% to 5%). The non-overlapping acquisitions are reported on the left panel, and overlapping acquisitions are reported on the right panel.

Table 1
Description of Drug Development Project Acquisitions

This table provides descriptive statistics on drug projects categorized into non-acquired, acquired by non-overlap acquirers, and acquired by overlapping acquirers. The table describes the number of drugs originated over time and by consolidated disease groups, and the proportion of projects that are non-acquired, acquired by non-overlapping acquirers, as well as acquired by overlapping acquirers (i.e. acquired by an incumbent with a project in the same therapeutic class and mechanism of action as the focal project). For illustrative purposes, we present top 5 broad disease groups by number of projects (out of 16 total groups). Disease groups are high-level categorizations, and each disease group includes a large number of therapeutic classes and mechanism of action (ThC/MoA) pairs. These narrower categories are the basis for our measures of overlap and competition in the main analysis. Drug projects are identified from initial origination from the Pharmaprojects database, and acquisitions are identified from the SDC M&A database, RecapIQ, and VentureXpert.

	N	Non-Acquired	Non-overlap Acquired	Overlap Acquired
Whole Sample	35,712	76%	19%	5%
<i>By Time Period</i>				
	Beginning-1995	60%	31%	9%
	1996-2000	67%	26%	7%
	2001-2005	77%	18%	5%
	2006-2010	90%	8%	2%
<i>By High-level Disease Group (top 5)</i>				
	Anti-cancer (13 therapeutic classes; 783 ThC/MoA)	80%	15%	5%
	Neurological (27 therapeutic classes; 986 ThC/MoA)	75%	21%	4%
	Anti-infectives (28 therapeutic classes; 452 ThC/MoA)	75%	17%	8%
	Biotechnology (26 therapeutic classes; 209 ThC/MoA)	76%	18%	6%
	Alimentary/Metabolism (24 therapeutic classes; 498 ThC/MoA)	79%	18%	4%

Table 2
Acquisitions, Product Overlap, and Project Development

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

$$\begin{aligned}
 Development_{i,t} = & \beta \cdot I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i + \gamma_1 \cdot I(Acquired)_i \times I(Post)_{i,t} \\
 & + \gamma_2 \cdot I(Acquired)_i \times I(Overlap)_i + \gamma_3 \cdot I(Acquired)_i \\
 & + \alpha_{age} + \alpha_{vintage} + \varepsilon_{i,t},
 \end{aligned}$$

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

	Development Event = 1					
	(1)	(2)	(3)	(4)	(5)	(6)
	Originated before 2011		Originated before 2000		Single-Project Companies	
I(Acquired) × I(Post) × Overlap	-0.019*** (-2.894)	-0.013* (-1.747)	-0.030*** (-3.508)	-0.017* (-1.791)	-0.128** (-2.392)	-0.177*** (-2.688)
I(Acquired) × I(Post)	-0.017*** (-5.239)	-0.013*** (-3.684)	-0.013*** (-3.050)	-0.016*** (-3.471)	-0.041* (-1.747)	-0.036 (-1.376)
I(Acquired) × Overlap	-0.001 (-0.178)		-0.000 (-0.061)		0.040 (0.931)	
I(Acquired)	-0.002 (-0.720)		-0.003 (-0.955)		0.008 (0.501)	
Observations	311,501	311,501	127,910	127,910	17,780	17,780
R-squared	0.018	0.243	0.009	0.237	0.028	0.166
Project FE	No	Yes	No	Yes	No	Yes
Age FE	Yes	Yes	Yes	Yes	Yes	Yes
Vintage FE	Yes	No	Yes	No	Yes	No

Table 3
Timing of Development

This table presents the post-acquisition development likelihood of a panel of acquired drug projects. The general empirical specification is,

$$Development_{i,t} = \beta \cdot I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i + \gamma_1 \cdot I(Acquired)_i \times I(Post)_{i,t} + \gamma_2 \cdot I(Acquired)_i \times I(Overlap)_i + \alpha_{age} + \alpha_{vintage} + \varepsilon_{i,t},$$

where the dependent variable $Development_{i,t}$ is a dummy variable indicating whether or not drug i has a development event in period t . In column (1) the dependent variable=1 if the drug had NO development event in period t , where the period is either pre- or post-acquisition years. In column (2) the dependent variable=1 if the drug had a development event in year t . $I(Acquired) \times I(Post)$ indicates whether the drug-period(i, t) observation is after the drug is acquired. $I(Overlap)$ indicates whether the acquired drug overlaps with the pipeline of the acquirer. The sample in column (1) focus on all acquired drug projects. The sample in column (2) focus on acquired projects that never have a development event in the years post-acquisition (here we are examining whether the “never-developed” projects are driving our results in Table 2). In both analyses, we control for vintage (the year of origination) fixed effects; we also control for age via age at acquisition fixed effects (in column (1)), and age fixed effects (in column (2)). The t-statistics based on standard errors clustered at the drug project level are displayed in parentheses. ***, **, and * indicate significance at the 1%, 5%, and 10% levels, respectively.

	(1)	(2)
	No Development Event = 1	Development Event = 1
I(Acquired) × I(Post) × Overlap	0.049*** (2.702)	0.020 (0.609)
I(Acquired) × I(Post)	0.214*** (24.269)	-0.012 (-0.832)
I(Acquired) × Overlap	0.008 (0.686)	0.000 (0.002)
Observations	14,761	8,810
R-squared	0.084	0.016
Age FE	Yes	Yes
Vintage FE	Yes	Yes
Acquired projects only	Yes	Yes

Table 4
Acquisitions and Project Development: Market Competition

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

$$\begin{aligned} Development_{i,t} = & \beta \cdot I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i + \gamma_1 \cdot I(Acquired)_i \times I(Post)_{i,t} \\ & + \gamma_2 \cdot I(Acquired)_i \times I(Overlap)_i + \gamma_3 \cdot I(Acquired)_i \\ & + \alpha_{age} + \alpha_{vintage} + \varepsilon_{i,t}, \end{aligned}$$

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

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

Table 5
Acquisitions and Project Development: Patent Life Among Overlaps

This table presents the differing post-acquisition development likelihood 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 investigates how remaining patent term length influences the effect of acquisition on the likelihood of development. The empirical specification uses the following model,

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

where the dependent variable $Development_{i,t}$ is a dummy variable indicating whether drug i has a development event in year t . $I(Post)_{i,t}$ indicates whether the drug-year (i, t) observation is after the drug is acquired. $I(NearPatExpiry)$ 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. Column (2) also includes acquirer firm FE. The t-statistics based on standard errors clustered at the drug project level are displayed in parentheses. ***, **, and * indicate significance at the 1%, 5%, and 10% levels, respectively.

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

Table 6
Acquisitions and Project Development: Clinical Trials

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

$$PhaseII_i = \beta \cdot I(AcquiredPI)_i + \gamma \cdot I(AcquiredPI)_i \times I(Overlap)_i + \alpha_{vintage} + \varepsilon_i.$$

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

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

Table 7
Acquisitions and Assets Redeployment

Panel A: Acquisitions and Project Similarities to Acquired Drugs

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

	(1)	(2)	(3)
	Chemical Similarity		
$I(Post) \times \text{Overlap}$	0.001 (0.481)	0.000 (0.111)	0.002 (0.872)
$I(Post)$	-0.002 (-0.609)	-0.001 (-0.295)	-0.004 (-1.364)
Overlap	0.004 (1.263)	0.004 (1.206)	
Observations	154,896	154,896	154,896
R-squared	0.001	0.014	0.361
Acquirer FE	No	Yes	No
Case FE	No	No	Yes

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

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

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

Table 8

The Intensity of Project Discontinuation around FTC Review Threshold

This table presents univariate survival tests on the drugs that are acquired just below $[-5\%, 0]$ and just above $[0, 5\%]$ the FTC review threshold. Specifically, we examine the rates of being active, being discontinued, and being fully launched using the development status of each project as of June 2017. To ensure that we leave adequate room for acquisitions to occur, 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.

	5% Below Threshold	5% Above Threshold	Diff	T-statistics	Stat Significance
Active	3.57%	7.58%	-4.00%	-1.176	
Launched	1.79%	9.09%	-7.31%	-2.293	**
Discontinued	94.64%	83.33%	11.31%	2.509	**
N	112	66			

Appendix (Not For Publication)

A. Omitted Proofs

A.1. Bertrand Competition

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

A.1.1. Consumer Demand. We follow [Vives \(2000\)](#) and [Häckner \(2000\)](#) and consider an industry with n products that are produced at 0 marginal cost. We derive demand from the behavior of a representative consumer with a quadratic utility function

$$(11) \quad U(q) = \alpha \sum_{i=1}^n q_i - \frac{1}{2} \left(\beta \sum_{i=1}^n q_i^2 + 2\gamma \sum_{i \neq j} q_i q_j \right)$$

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

A.1.2. No Acquisition. Consider first the product market choices of an entrepreneur that is not acquired ($\neg acq$). If the project is successful (S), the resulting newly developed product competes against n other single-product incumbent firms. The entrepreneur's objective function is

$$(12) \quad \max_{p_E} p_E q_E$$

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

$$(13) \quad \pi_{-acq,S}^E = \frac{\alpha^2(\beta - \gamma)(\beta + (n - 1)\gamma)}{(2\beta + (n - 2)\gamma)^2(\beta + n\gamma)} = \pi_{-acq,S}^A.$$

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

$$(14) \quad \pi_{-acq,F}^A = \frac{\alpha^2(\beta - \gamma)(\beta + (n - 2)\gamma)}{(2\beta + (n - 3)\gamma)^2(\beta + (n - 1)\gamma)} = \pi_{-acq,F}^I.$$

A.1.3. Acquisition. Next consider the product market choices of an acquirer in the case of an acquisition (acq). If the project is successful, he becomes a two-product oligopolist who optimally chooses quantities for his new and his old product and competes against $n - 1$ other single-product incumbents. The acquirer's objective function is

$$(15) \quad \max_{p_1, p_2} p_1 q_1 + p_2 q_2$$

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

The profit of the multi-product incumbent acquirer is

$$(16) \quad \pi_{acq,S}^A = \frac{\alpha^2(\beta - \gamma)(\beta + (n - 2)\gamma)(2\beta + \gamma(2n - 1))^2}{2(\beta + n\gamma)(2\beta^2 + (3n - 4)\beta\gamma + (1 + (n - 3)n)\gamma^2)^2}.$$

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

$$(17) \quad \pi_{acq,F}^A = \frac{\alpha^2(\beta - \gamma)(\beta + (n - 2)\gamma)}{(2\beta + (n - 3)\gamma)^2(\beta + (n - 1)\gamma)}.$$

Comparing these expressions yields the following profit ranking if $\beta > \gamma > 0$

$$(18) \quad \pi_{acq,S}^A > \pi_{acq,F}^A = \pi_{-acq,F}^A > \pi_{-acq,S}^A = \pi_{-acq,S}^E > \pi_{-acq,F}^E = 0$$

as well as the following inequality

$$(19) \quad \Delta^E \equiv \pi_{-acq,S}^E - \pi_{-acq,F}^E > \pi_{acq,S}^A - \pi_{acq,F}^A \equiv \Delta^A.$$

A.1.4. Product Market Overlap.

Proof of Proposition 1. From the inequality (3) it immediately follows that an incumbent firm acquires a project and continues development if $k \leq k^A$ and that an independent entrepreneur continues if $k \leq k^E$. Equation (4) shows that the thresholds k^E and k^A are identical if and only if $\Delta^E = \Delta^A$. Thus, it remains to show that for any positive product market overlap $\beta > \gamma > 0$, we have $\Delta^E > \Delta^A$ and hence $k^E > k^A$.

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

A.1.5. Competition.

Proof of Proposition 2. Note that the difference between the thresholds is given by $k^E - k^A = \rho(\Delta^E - \Delta^A)$. Proposition 1 establishes that $\Delta^E - \Delta^A > 0$ for any $\beta > \gamma > 0$. Substituting the profit expressions $\pi_{-acq,S}^E$, $\pi_{-acq,F}^E$, $\pi_{acq,S}^A$, and $\pi_{acq,F}^A$ and differentiation of $\Delta^E - \Delta^A$ with respect to n establishes the result. Furthermore, we have $\lim_{n \rightarrow \infty} (k^E - k^A) = 0$. \square

A.1.6. Patent Life and Future Competition.

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

Denote the development gains for the entrepreneur and the acquirer in the presence of undifferentiated generic competition after the expiry of the acquirer's existing product's

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

Thus, the development decisions of the entrepreneur d_{gen}^E and the acquiring incumbent d_{gen}^A are given by inequalities (5) and (6). And therefore, the resulting difference in the development thresholds is given by $\rho T^A(\Delta^E - \Delta^A)$. This difference is increasing in T^A which establishes the proposition. \square

A.1.7. Acquisition Decision.

Proof of Proposition 4. The acquirer decides to acquire at a takeover price P if

$$(20) \quad d^A[\rho\pi_{acq,S}^A + (1 - \rho)\pi_{acq,F}^A - k] + (1 - d^A)(L + \pi_{acq,F}^A) - P \geq \\ d^E[\rho\pi_{acq,S}^A + (1 - \rho)\pi_{acq,F}^A] + (1 - d^E)\pi_{acq,F}^A$$

where $d^i \in \{0, 1\}$ for $i = \{E, A\}$ is the development decision for the owner of the project in $t = 1$.

To compensate the entrepreneur for selling the project, the acquirer must pay a price P that is equal to the expected payoff of the project when the entrepreneur remains independent. Thus,

$$(21) \quad P = d^E(\rho\Delta^E - k) + (1 - d^E)L.$$

Substituting the takeover price (21) into the inequality for the acquisition decision (20) and solving for each of the three cases of ρ establishes the proposition. \square

A.1.8. Welfare. We now show under what conditions killer acquisitions are welfare-decreasing. This is the case whenever $k^E \geq k > k^A$ and the social surplus resulting from no acquisition (and continued development) is higher than when there is no acquisition (and termination).

Under a social welfare standard which uses the unweighted sum of consumer surplus and producer surplus, this is given by the following inequality

$$(22) \quad \rho\pi_{-acq,S}^E - k + n[\rho\pi_{-acq,S}^A + (1 - \rho)\pi_{-acq,F}^A] + \rho CS_{-acq,S} + (1 - \rho)CS_{-acq,F} \geq \\ P + (\pi_{acq,F}^A + L - P) + CS_{acq,F}$$

where P is the transaction price which is just a transfer between incumbent and entrepreneur and $CS_{-acq,S}$ and $CS_{-acq,F} = CS_{acq,F}$ are the consumer surplus values under the different scenarios. Recall that $\pi_{-acq,F}^A = \pi_{acq,F}^A$ then rewriting this condition yields

$$(23) \quad (\rho\pi_{-acq,S}^E - k - L) + \rho(CS_{-acq,S} - CS_{-acq,F}) \geq n\rho(\pi_{-acq,F}^A - \pi_{-acq,S}^A).$$

The first term in brackets on the left-hand side is the entrepreneur's net expected profit gain from continuing development. This is positive in the killer acquisitions region $k^E \geq k > k^A$. The second term is the expected increase in consumer surplus due to continued development which is also positive both because there is more product variety and because prices are lower. The term on the right-hand side of the inequality is the expected loss in profit for the n incumbents, and it is also positive. We can derive a sufficient condition for killer acquisitions to be welfare-reducing by setting the first term to zero (i.e., $k = k^E$ so the entrepreneur just wants to develop it). This yields the following condition

$$(24) \quad CS_{-acq,S} - CS_{-acq,F} \geq n(\pi_{-acq,F}^A - \pi_{-acq,S}^A).$$

Hsu and Wang (2005) derive expressions for consumer surplus and total welfare under differentiated goods oligopoly. Using their expressions, we obtain the following expression for the increase in consumer surplus

$$(25) \quad CS_{-acq,S} - CS_{-acq,F} = \frac{(n+1)[\beta + \gamma n]}{2} q_{n+1}^2 - \frac{n[\beta + \gamma(n-1)]}{2} q_n^2$$

It is now straightforward to show that the sufficient condition for killer acquisitions to be welfare-reducing given by inequality (24) is always satisfied for any degree of product

substitution under differentiated Bertrand competition with a single incumbent ($n = 1$). For $n \geq 2$ the inequality is satisfied under differentiated Bertrand competition as long as the entrant's product is sufficiently differentiated (i.e., $\gamma < \gamma^W$) from the existing incumbents.

Furthermore, as n increases the threshold γ^W below which killer acquisitions decrease, welfare also decreases, thus increasing the region under which killer acquisitions do not necessarily reduce welfare. However, from Proposition 2 we know that as n increases, the region in which killer acquisitions occur shrinks. Thus, it is precisely in the cases in which killer acquisitions do not occur for a large set of parameter values that their social welfare impact is also potentially beneficial.

A.2. Cournot Competition

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

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

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

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

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

Defining Δ^E and Δ^A with these new payoffs and the same logic of proofs above establishes all the propositions as in our main model.

B. Cleaning Pharmaprojects Data

To build our analytical dataset at the drug project level, we use Pharmaprojects from Pharma intelligence. Pharmaprojects is a comprehensive dataset that tracks drug projects from a very early stage through to launch or discontinuation. Pharmaprojects provides nearly universal coverage of all candidate drugs developed or under development for eventual sale in the U.S. market, along with the originating firm associated with each drug project. In this Appendix, we describe the process involved in cleaning the data.

B.1. Identifying Originators of Drug Projects

Our first challenge in using Pharmaprojects data for our analyses was to identify the developer of each drug project at each point in time, particularly prior to and post acquisition. In the raw dataset, Pharmaprojects typically updates the “originator” firm name associated with each project when and if it is acquired. More specifically, if the project was acquired, the acquiring firm is typically erroneously listed as the “originator” of the project in raw Pharmaprojects data. We therefore needed to re-construct the original “originator” firm in such cases.

To do so, we make use of two additional fields in the dataset. The first is the “overview” field, which intends to provide background of the drug project and thus often includes the name of the original firm associated with the project in the case of acquisitions. For example, the drug Trastuzumab had the originator as “Roche” when it was initially developed by Genentech. The overview text reads “Trastuzumab is a humanized MAb to HER2, a cell surface oncoprotein which is overproduced in breast and ovarian cancers, under development by Genentech (Roche)” and hence we could use this information to extract the original originator as Genentech.

The second is the “latest change” field, which also would often contain details of acquisition events, including the associated firm names. For example, the field often read “Firm XYZ acquired by Firm ABC”, which we would use to impute the original originator name as “Firm XYZ”.

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.” We algorithmically created a list of original originators and the acquiring firms, and we checked them against our M&A datasets from SDC and Recap IQ.

B.2. Merging Pharmaprojects with Acquisition Data

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. We do so first by using the Stata program “stnd_compname” (Wasi and Flaaen, 2015), which isolated the stem name for each originator firm associated with each project in Pharmaprojects. We then checked all non-exact matching manually to confirm accuracy.

B.3. Categorizing Development Milestones

Pharmaprojects comprehensively documents the occurrence and timing of key product development milestones (e.g., “new patent application”, “target identified”, “first launch”, and “additional registration for clinical trial”), including drug discontinuations. We aggregate the 28 events tracked by Pharmaprojects into three categories: development events, termination events, and neutral events that impart little information regarding the progress (or termination) of drug development. Development events reflect both research and development milestones and important steps in the commercialization process for the underlying drug project. Pharmaprojects therefore allows us to identify and capture milestones that signify development of a drug, including, but not limited to, progress through clinical trials. The Table “Measuring Drug Development” details all events that comprise our main development milestone dependent variable.

B.4. Clinical Trials Information

We supplement the Pharmaprojects data with Pharma Intelligence’s Trialrove data on clinical trials, linked at the project level. Drug clinical trials comprise three main phases: Phase I trials, which are small (20 and 100 healthy volunteers), short, and are intended to test safety; Phase II trials, which are larger (100s of affected patients), typically randomized

control trials lasting up to two years, and are intended to test efficacy; and Phase III trials, which are expanded versions of Phase II trials, involving hundreds or thousands of participants, and typically lasting one to four years (US Food and Drug Administration, 2017). Following successful trials, firms may submit a New Drug Application (NDA) to the FDA, which then determines if, and under what conditions, the drug can be marketed to U.S. patients. We use Trialrove data to identify the initiation of clinical trials by phase, including the timing of trial initiation.

Notably, clinical trial data are widely available only from 1997 onward, when the U.S. Federal government first mandated the National Institutes of Health (NIH) to collect and make publicly available a comprehensive, clinical trials database.³¹ Therefore, we have comprehensive trial data only for a limited subset of all projects in our sample, specifically those initiated after 1997. Within this limited sample, we identify projects for which we observe the start date of the first round of Phase I trials and track their progression to future trial phases, following prior studies that use progression through phases of clinical trials as a measure of project development (Krieger, 2017; Guedj and Scharfstein, 2004).

³¹More details on the timeline of publicly available clinical trials database can be found at <http://www.clinicaltrials.gov>.

Measuring Drug Development

This table presents a list of events recorded in Pharmaprojects to track the development process of each drug. The events are listed in alphabetical order. Each of these events is coded into one of the three categories: development events, discontinuation events, and neutral events with little information regarding drug development progress (denoted as “-” in the table).

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

C. Merging Drug Development and Acquisition Data with Patent Databases

In this section, we describe the process to merge drug development and acquisition data with USPTO patent databases through matching company names with assignee names in the USPTO patent database. To minimize potential problems introduced by the minor discrepancy between different versions of the USPTO database, we use both NBER and Harvard 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; it is similar for USPTO assignees.

C.1. Name Standardization

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

C.2. The Matching Procedure

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

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

2. Each stem drug originator and owner name is matched with stem names from the NBER data and HBS data.
 - (a) If an exact match of stem names 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 criteria 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.³² The criterion is based on the length of the strings and the Levenshtein distance, and the threshold is determined through a random sampling procedure.
 - (a) If the fuzzy-matched pair is located in the same city and state OR the two 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.

³²The 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).