Killer Acquisitions*

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This paper argues incumbent firms may acquire innovative targets solely to discontinue the target's innovation projects and preempt future competition. We call such acquisitions "killer acquisitions." We develop a parsimonious model illustrating this phenomenon. Using pharmaceutical industry data, we show that acquired drug projects are less likely to be developed when they overlap with the acquirer's existing product portfolio, especially when the acquirer's market power is large due to weak competition or distant patent expiration. Conservative estimates indicate about 6% of acquisitions in our sample are killer acquisitions. These acquisitions disproportionately occur just below thresholds for antitrust scrutiny.

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1. Introduction

Innovation drives economic growth and firm profitability. 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) illustrates this phenomenon. In the early 2000s, Questcor enjoyed a monopoly in adrenocorticotropic 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.[2]

This paper theoretically and empirically studies killer acquisitions. First, to motivate the empirical analysis, we build a parsimonious model that combines endogenous acquisition

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

[2]The attempted acquisition of Heartware by Thoratec, both medical device firms, in 2009 provides an additional example of acquisitions aimed at pre-emptively eliminating innovative competition. At the time, Thoratec had a monopoly in the U.S. market for left ventricular assist devices (LVAD), a life-sustaining technology for end-stage heart failure patients, and Heartware was running clinical trials for their own potentially competing device (the "HVAD"), but had yet to receive FDA approval. In its complaint the FTC argued that "Thoratec's proposed $282 million acquisition of Heartware threatens to eliminate the one company poised to seriously challenge Thoratec's monopoly of the U.S. LVAD market. By acquiring Heartware, Thoratec willfully seeks to maintain its LVAD monopoly, thereby denying patients the potentially life-saving benefits of competition between Thoratec and HeartWare" (FTC Administrative Complaint Docket No. 9339: https://www.ftc.gov/sites/default/files/documents/cases/2009/07/090730thorateadminccmpt.pdf).

decisions, innovation choices, and product market competition. Our model formalizes the seemingly counterintuitive phenomenon of incumbents acquiring innovative potential entrants merely to shut down the entrant's innovative endeavors. It also highlights the conditions under which killer acquisitions are particularly prevalent.

We model acquisitions that occur when the target firm's project is still under development and therefore further development is necessary and costly, and the ultimate project success is uncertain. An incumbent acquirer has weaker incentives to continue development than an entrepreneur if the new project overlaps with (i.e., potentially substitutes for) a drug in the incumbent's portfolio. This is a general, well-known result: "the monopolist's disincentive created by his preinvention monopoly profits" (Arrow, 1962). We show that this disincentive to innovate can be so strong that an incumbent firm may acquire an innovative entrepreneur simply to shut down the entrepreneur's projects and thereby stem the "gale of creative destruction" of new inventions (Schumpeter, 1942). However, both existing and future product market competition reduce the difference in project development decisions between acquirers and independent entrepreneurs and thereby diminish the incentive for killer acquisitions. Finally, we show that positive acquirer-target product overlap is necessary for the killer acquisition motive to exist.

In the second part of the paper, we provide empirical support for our theory. Doing so presents significant empirical challenges. We need to observe project-level development activity and track projects as they move across firms. It is also crucial to accurately measure overlap between the acquiring firm's products and the target's project and to quantify competition in the relevant product market.

Pharmaceutical drug development offers all of these features. Further, documenting killer acquisitions in the pharmaceutical industry is also worthwhile since the industry is highly innovative, and the successful commercialization of innovative drugs is potentially very socially valuable.[3] We collect detailed development information on more than 16,000 drug projects originated by more than 4,000 companies in the past two and half decades and follow each drug from initiation. We collect relevant acquisition events from comprehensive

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

data sources. Importantly, we observe development milestones of drug projects independent of project ownership, meaning we can follow the same projects pre- and post-acquisition.[4]

To finely categorize acquirer overlap with the target's project, and thus identify potentially competing products, we use pharmaceutical categories based on substitutability. Specifically, if the target's drug project is in the same therapeutic class (e.g., antihypertensive) and uses the same mechanism of action (e.g., calcium channel antagonist) in which the acquirer has a drug, we consider that acquisition to be an overlapping acquisition.

Our main empirical analyses focus on the development stage of drug projects. We compare projects acquired by overlapping incumbents to those acquired by non-overlapping incumbents, and to non-acquired projects. The baseline regression uses a project-year panel to estimate the annual probability of development. Following the logic of killer acquisitions, we expect a decreased likelihood of development of overlapping projects post-acquisition. Correspondingly, we find projects acquired by an incumbent with an overlapping drug are 28.6% less likely to be continued in the development process compared to drugs that are not acquired.

This finding is robust to controlling for a variety of economic forces. In our tightest specification, we control for drug development life cycles using therapeutic class-mechanism of action-age fixed effects, and include project fixed effects to account for any unobservable but time-invariant project characteristics. This result also holds if we only compare acquired projects within the same target firm: projects from the same target firm that overlap are more likely to be terminated than those that do not. Reassuringly, the development patterns for overlapping acquired drugs are statistically indistinguishable from non-overlapping acquired drugs and non-acquired drugs in the years prior to acquisition.

Our theory also predicts that incumbents have a stronger incentive to acquire and terminate overlapping innovation in ex-ante less competitive markets, i.e., when the incumbent has more to lose if the target's innovation is successfully developed. To examine this, we repeat the baseline analysis in subsamples with low and high levels of existing competition (as measured by the number of competing drugs in the same therapeutic class and mechanism of action), separately for the product market and the development pipeline. We find that the decrease

[4]For example, we can observe Dom-0800, an anti-CD40 ligand human domain antibody, originated by Domantis in 2005. Domantis was acquired by GlaxoSmithKline in 2006. Yet, we track and document the development of Dom-0800 post-2006, regardless of its change in ownership.

in development probability for acquired, overlapping projects is concentrated in markets with low competition. Our theory also predicts that when the incumbent's drug is far from patent expiration and thus generic competition, incumbents have a stronger incentive to acquire and terminate innovation because the loss from cannibalization is large. Accordingly, we find that the decrease in development rates is concentrated in overlapping acquisitions for which the patent on the acquirer's overlapping drug is relatively far from expiry.

In additional empirical tests, we examine the progression of projects through the phases of clinical trials. While limited in terms of the sample of projects and breadth of development milestones, this additional analysis ensures comparison of projects at precisely the same stage of development and mirrors prior work on drug development (Krieger, 2017; Guedj and Scharfstein, 2004). We focus on projects that start Phase I trials and examine their likelihood of starting Phase II. We find that drug projects are 46.6% 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.

Despite the difficulties associated with testing for strategic motives, our main analyses, combined with additional tests, collectively suggest that killer acquisitions are both strategic and intentional. First, as our model predicts, we find acquisitions are almost four times more likely when the incumbent acquirer's products overlap with the target project.[5] Second, we find 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 (Wollmann, 2018). 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.

We employ several additional tests to address potential alternative explanations for lower

[5]In our model, overlapping acquisitions do not occur because they have a positive "direct" effect on the acquiring incumbent's profits (e.g., due to synergies between acquirer and target), but because they allow the acquirer to change the behavior of the target (e.g., the overlapping project is never developed) which is beneficial for the incumbent only when there is product-project overlap. Ellison and Ellison (2011) also study incumbents' strategic motives in the pharmaceutical industry, but they focus on investment and advertising choices to deter entry. In their setting, the strategic motive is identified by the non-monotonicity of investment with respect to market size whereas in ours it is identified by the lack of development of acquired overlapping projects.

development rates of overlapping acquired drugs. One alternative explanation is optimal project selection. Specifically, for multi-project targets, the acquirer could strategically and optimally choose to continue only the most promising projects while discontinuing those that are less promising. To assess this concern, we repeat our analysis for acquisitions of single-drug companies. Our results are robust to focusing on only this set of acquisitions, which implies optimal project selection does not explain our results.

Next, we investigate whether changes to the timing of development rather than true discontinuation might be behind our estimates. Acquiring firms might purposefully delay development or simply 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 drive our main results. In fact, we find decreased development post-acquisition is driven by drugs that are never developed post-acquisition, that is by immediate and permanent terminations.

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, first, 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' new drug development projects. We also do not find that acquirers are more likely to cite acquired and terminated projects' patents. 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% of all acquisitions in our sample (or about 45 pharmaceutical acquisitions per year) are killer acquisitions. Eliminating the adverse effect on drug project development from killer acquisitions would raise the pharmaceutical

industry's aggregate drug project development rate by about 4%. However, despite the ex-post inefficiencies of killer acquisitions and their adverse effect on consumer surplus, the overall effect on social welfare is ambiguous because these acquisitions may also increase ex-ante incentives for the creation of new drug projects.[6]

Overall, this paper makes three contributions. First, we shed new light on a fundamental impediment to corporate innovation. Specifically, we highlight how the motive to protect existing profits, known to discourage an incumbent's own innovation, can also incentivize powerful incumbents to stifle the innovation of other firms. Second, we document the importance of this obstacle to innovation in the pharmaceutical industry, an innovation-focused industry crucial to consumer and social welfare. 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. We show that such acquisitions often avoid antitrust scrutiny and may therefore pose concerns for consumer welfare.

The prior literature on motives for corporate acquisitions has focused on agency conflicts (Roll, 1986; Morck et al., 1990), synergies (Bena and Li, 2014; Maksimovic and Phillips, 2001), and increasing existing market power (Baker and Bresnahan, 1985). This paper adds to this literature in two ways. First, in our model, acquisitions are not driven by synergies or by incentives to increase current market power. Instead, we argue that incumbents acquire innovative targets to terminate nascent innovation that may threaten their profits in the future. This new mechanism combines two classic effects in the innovation literature: the "replacement effect" (Arrow, 1962), which reduces the incentives of an incumbent to introduce new products that are substitutes for existing products,[7] and the "efficiency effect" (Gilbert and Newbery, 1982), which gives an incumbent strong incentives to acquire the property rights to a new innovation to preempt entry.[8] Second, we focus on the implications of

[6]Protective antitrust policy may have conflicting effects on innovation incentives, by raising the profits of new entrants, but lowering those of continuing incumbents in settings with continual innovation and "winner-take-all" competition (Segal and Whinston, 2007), even under cooperative entrepreneurial commercialization choices such as licensing or acquisitions (Gans, 2017).

[7]Henderson (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.

[8]Katz and Shapiro (1985) and Gans and Stern (2000) offer comprehensive theoretical treatments of R&D

acquisitions and increasing concentration on innovation. Quite surprisingly, the link between (horizontal) mergers and innovation has received little attention despite its significant policy relevance.[9] Our paper provides a theoretical and empirical analysis of a new channel through which acquisitions impact innovation. By using detailed project-level data on acquisition and development decisions we are able to rule out other potential explanations for the observed acquisition patterns and the innovation gap between acquired and independent firms.

We also contribute to the literature on innovation and competition in the pharmaceutical industry. A number of papers have documented the tradeoffs involved in promoting competition while fostering innovation, through investigating the product market interactions between patented and generic drugs (Caves et al., 1991; Grabowski and Vernon, 1992; Scott Morton, 2000; Ellison and Ellison, 2011), the role of pricing (Howard et al., 2015), and internal R&D policies (Pisano, 1990; Cockburn and Henderson, 1994). Our paper complements this literature by presenting evidence that the market for corporate control plays a crucial role in shaping competition and innovation in drug development. Even though acquisitions can create value, incumbents may abuse this mechanism and thereby impede innovative competition.

The remainder of the paper proceeds as follows. Section 2 outlines our theoretical framework and develops testable hypotheses. Section 3 describes the data and institutional background. Section 4 presents our main empirical results. Section 5 discusses how other motives cannot explain our main findings, implications for antitrust and social welfare, and quantifies the industry-wide impact of killer acquisitions. Section 6 offers concluding remarks.

2. Theoretical Framework

To guide our empirical strategy we propose a simple theoretical model of acquisition, innovation, and product market competition. The model provides four distinct empirical predictions about development and acquisition choices and how they are affected by product overlap and existing and future competition. All proofs are in Appendix A.

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.

[9] 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.

2.1. Setup

The model has the following timeline, depicted in Figure 1. In $t = 0$, an entrepreneur E (she) 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 (he), can acquire the entrepreneur E at an endogenously determined takeover price P.[10] We use the subscript acq if the entrepreneur was acquired in $t = 0$ and $\neg acq$ otherwise.

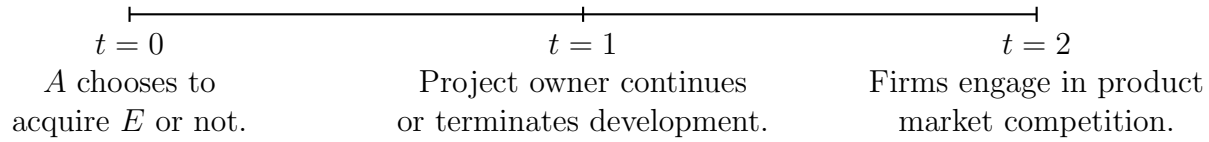

Figure 1. Model Timeline

In $t = 1$, the owner of the project—the acquirer A if the project has been acquired, or the entrepreneur E if it remains independent in $t = 0$—decides whether to develop the project. Let ρ be the probability that the project will ultimately be successful, k be the cost of developing the project, and L be the liquidation value of the project if development does not continue. This structure captures how a pharmaceutical firm decides whether to proceed with the development of a new drug. At this stage the original project idea exists and is commonly patented; however, continued development effort of the drug is necessary, very costly, and the eventual success is uncertain.

Finally, in $t = 2$, uncertainty about the success of the project is resolved and all the firms engage in product market competition with imperfect substitutes. We model competition using differentiated Bertrand competition because price-setting behavior by firms best captures strategic interactions in the branded drug market (Berndt and Newhouse, 2012).[11] We assume that if the project is successfully developed in $t = 2$, the drug has a product market payoff

[10] Our theoretical and empirical analysis focuses on an environment in which intellectual property is well protected. This allows us to abstract away from contracting difficulties in the sale of ideas as in Anton and Yao (2002).

[11] Our results are not sensitive to this particular form of competition. They also hold for Cournot competition as we show in Appendix A.2.

that depends on the degree of competition (i.e., the number of active firms/products in the market) and product differentiation in the market. If the project is unsuccessful, the payoff is zero. We assume that the values of ρ, k, and L are commonly known and identical for all the involved parties.

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

Consider first the product market choices of the entrepreneur when her project is not acquired ($\neg acq$). 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 $\pi^E_{\neg acq,S} = \pi^A_{\neg acq,S} > 0$. 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 $\pi^E_{\neg acq,F} = 0$. The n incumbent firms each have a single existing product to sell, and thus the acquirer's profit is equal to $\pi^A_{\neg acq,F}$. Profits are higher $\pi^A_{\neg acq,F} > \pi^A_{\neg acq,S}$ 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 $\pi^A_{acq,F}$. This is the same as when no acquisition occurs and the entrepreneur's project fails, hence $\pi^A_{acq,F} = \pi^A_{\neg acq,F}$.

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.[12] The profit of the multi-product incumbent acquirer is $\pi^A_{acq,S}$. This profit is obviously higher than when he sells only a single product with the same $n - 1$ competitors, hence $\pi^A_{acq,S} > \pi^A_{\neg acq,F}$.

[12] Given our symmetry assumptions, in equilibrium, the resulting prices are $p^*_1 = p^*_2 = p^A$ and $p^*_i = p^I$ for any $i \neq 1, 2$.

As a result, we obtain the following profit ranking

$$\pi^A_{acq,S} > \pi^A_{acq,F} = \pi^A_{\neg acq,F} > \pi^A_{\neg acq,S} = \pi^E_{\neg acq,S} > \pi^E_{\neg acq,F} = 0. \tag{1}$$

2.3. Development Decision ($t = 1$)

2.3.1. 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 $\pi^A_{acq,S}$ and $\pi^A_{acq,F}$ for the incumbent and the difference between $\pi^E_{\neg acq,S}$ and $\pi^E_{\neg acq,F}$ for the entrepreneur. It is straightforward to show that for all imperfect substitutes, we have

$$\Delta^E \equiv \pi^E_{\neg acq,S} - \pi^E_{\neg acq,F} > \pi^A_{acq,S} - \pi^A_{acq,F} \equiv \Delta^A \tag{2}$$

As long as products are imperfect substitutes the acquirer gains strictly less from developing a new product than an entrepreneur would. This is because of the "replacement effect" (Arrow, 1962): 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.[13]

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

$$\rho\Delta^E - k \geq L, \quad \rho\Delta^A - k \geq L. \tag{3}$$

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

$$k^E = \rho\Delta^E - L, \quad k^A = \rho\Delta^A - L. \tag{4}$$

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

[13] If 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.

market overlap (i.e., substitutability) 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 the incumbent acquirer and entrepreneur occurs when k is in the intermediate range between k^A and k^E, which exists for any positive degree of product substitutability. This also highlights the crucial role of the development cost k. 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 coupled with product overlap is what generates the differential development decisions of the incumbent acquirer and the independent entrepreneur.

2.3.2. 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 draws consumer demand and profits away equally from all existing products. An acquiring incumbent is hurt more by such cannibalization when he is a monopolist (i.e., the new product draws demand away only from his own existing product) than when he already faces many other existing competitors (i.e., cannibalization losses are spread over many firms). As a result, as the number of existing competitors increases, the replacement effect decreases and the acquirer's development decisions become more similar to those of the entrepreneur.

Figure 2 illustrates this point by plotting the development thresholds k^E and k^A as a function of the number of incumbents. These are closer together when there are more existing incumbents.

[**Insert FIGURE 2 Here.**]

2.3.3. Patent Life and Future Competition. Until now, we have only considered the impact of competition with imperfect substitutes, capturing the competition among branded drugs. However, another important aspect of the pharmaceutical industry 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 $T^E > T^A \geq 0$. We assume, for simplicity, that the firms earn their static game profits every year.

We also 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 $\pi^A_{acq,S}$ (successful development of new project) or $\pi^A_{acq,F}$ (unsuccessful development) each year. This yields the same development gain Δ^A as before, multiplied by the number of years T^A. 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 due to undifferentiated generic competition, the acquirer faces no more cannibalization losses from development, and hence his incentives to develop coincide with those of the entrepreneur. Specifically, the entrepreneur's and acquirer's development gains after the expiration of the acquirer's existing product's patent in T^A years are $\Delta_{gen} = \Delta^E_{gen} = \Delta^A_{gen}$.[14]

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

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

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

where Δ_{gen} is the development gain for the entrepreneur and the incumbent in the presence of

[14]This development gain is 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.

undifferentiated generic competition after the expiration 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. The intuition for this result is essentially the same as that of Proposition 2. Generic entry is just a particularly intense form of competition that destroys all profits of the acquirer's existing product and thus completely eliminates cannibalization losses from new product development.

2.4. Acquisition Decision ($t = 0$)

We now show that "killer acquisitions" can only occur when the entrepreneur's project overlaps with the acquirer's existing product. 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.[15] 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

[15]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 than it would if acquisitions were impossible. As we discuss in Section 5, 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.

continues development. Such an acquisition ("Acquire to Kill") occurs if

$$\underbrace{\rho(\pi^A_{acq,F} - \pi^A_{\neg acq,S})}_{\text{efficiency effect}} \geq \underbrace{\rho\Delta^E - k - L}_{\text{replacement effect}}. \tag{7}$$

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 $\pi^A_{acq,F}$. However, if the incumbent does not acquire the entrepreneur's project, the incumbent potentially has to compete against n other firms. This yields a lower profit $\pi^A_{\neg acq,S}$. The difference between these (multiplied by the probability ρ with which the entrepreneur successfully develops the project) is the "efficiency effect." However, the expected marginal profit for the entrepreneur from continuing development ($d^E = 1$) given by $\rho\Delta^E - k$ is larger than the liquidation value L that the acquiring incumbent ($d^A = 0$) would obtain. This 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. Such an acquisition ("Acquire to Continue") occurs if

$$\underbrace{\pi^A_{acq,F} - \pi^A_{\neg acq,S}}_{\text{efficiency effect}} \geq \underbrace{\Delta_E - \Delta_A}_{\text{replacement effect}}. \tag{8}$$

Here, the "replacement effect" is the difference in marginal project development gains because both parties develop the project.[16] Despite developing the project the acquirer still benefits from reducing competition through (less aggressive) multi-product pricing.

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 $k \leq k^A$, he will choose "Acquire to

[16] Under 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.

Continue."

[Insert FIGURE 3 Here.]

Thus, acquisitions take place if $k \leq k^E$ and if the "efficiency effect" is sufficiently large relative to the "replacement effect." Even though the entrepreneur generally has 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").[17] Importantly, without any product market overlap, the acquirer never has a strictly positive incentive to acquire the entrepreneur, neither to "Acquire to Kill" nor to "Acquire to Continue." This is because without overlap, acquiring the project does not give the acquirer any gains resulting from reduced competition, and the two bargaining entities have exactly the same value for the project. This yields our final proposition.

Proposition 4 (Acquisition)**.** *If there is positive product market overlap, the acquirer may have strictly positive incentives to acquire the entrepreneur. If there is no product market overlap, the acquirer is always indifferent between acquiring and not acquiring the entrepreneur.*

Proposition 4 highlights that positive product market overlap is a necessary condition for (continuing or killer) acquisitions to occur. It immediately implies that acquisitions should be more likely when the acquirer's product and the entrepreneur's project overlap because the strategic acquisition motives outlined in our model are otherwise absent. Other theories of corporate acquisitions, most notably those emphasizing synergies in project development between acquirer and target, also predict that acquisitions are more likely to occur when the product portfolios of the merging parties are closely related. However, these theories produce diametrically opposed predictions for subsequent development choices: acquired projects with overlap should be more rather than less likely to be developed.

To summarize, our theoretical framework predicts that (i) following an acquisition, overlapping drug projects should be less likely to be developed; that when (ii) existing or

[17] Although 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), the acquirer has a weaker incentive to develop than the entrepreneur. 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 ($k \leq k^A$).

(iii) future competition is low, this difference in development choices between overlapping acquired drugs and their non-overlapping acquired or non-acquired counterparts should be more pronounced; and that (iv) acquisitions by incumbents should target entrepreneurial firms developing drug projects that overlap with existing drugs of the incumbent. We now explore whether these theoretical predictions are supported empirically in the pharmaceutical industry.

3. Background and Data

To empirically document the phenomenon of killer acquisitions, we use the setting of drug development. 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 measure overlap between acquirer and target firms, and market and technological competition. As described in detail below, pharmaceutical project development offers these features. Further, the pharmaceutical industry represents a significant and growing amount of healthcare spending, innovative activity, and M&A transactions, and is an economically and socially important industry of ongoing interest to economists (see Lakdawalla (2018) for a summary).

3.1. Drug Development Background

The development of innovative pharmaceutical products, often known as branded or patented drugs, involves a standard set of structured milestones en route to commercialization. First, firms identify potential drug compounds through routine discovery processes. Then, for any promising compounds, 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, drugs undergo three phases of clinical trials (Phases I, II, and III).[18] In tandem with these regimented clinical tests, firms engage in additional commercialization activities, including patent filing during the pre-clinical and/or discovery stage, regulatory filings in the U.S. and abroad, applications for coverage to various public and private insurance agencies,

[18]Drug 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.

and, finally, launching and marketing of the product in various countries around the world. Given the lengthy process prior to FDA approval and marketing, patented drugs usually have only a few years post-approval of monopoly profits before patent expiration and generic entry (Scherer, 1993).

Each component of drug development represents significant expenditure and time.[19] Because development is regulated and standardized, and reaching development milestones is typically very costly, we can interpret observed development events as credible evidence of purposeful and significant project-level development. Further, we are able to observe project-level development (or lack thereof) regardless of ownership, which is crucial to identifying killer acquisitions.

3.2. Drug Development Data

To build our dataset at the drug project level, we use Pharmaprojects from Pharma Intelligence, which has been used in earlier research studying drug development (for example, Kyle (2007); Blume-Kohout and Sood (2013); Adams and Brantner (2006); Branstetter et al. (2014)). Pharmaprojects is a comprehensive dataset that tracks drug projects from early stage development through to launch or discontinuation, using data collected directly from pharmaceutical companies and researchers (Blume-Kohout and Sood, 2013), and from public sources (press releases, patent filings, conference proceedings, regulatory agencies' reports, and the medical literature). Pharmaprojects tracks 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.[20]

Importantly for our purposes, the dataset also includes information about each drug's intended therapeutic market (e.g., "hypertension") and mechanism of action (e.g., "calcium channel antagonist"), which we use to identify overlapping and competing projects and products. Pharmaprojects also documents the occurrence and timing of key development

[19] DiMasi et al. (2003), Adams and Brantner (2006) and Dubois et al. (2015) estimate that a new drug incurs approximately $800 million to $1 billion in development costs with average expenditure on drugs in human clinical trials in Phases I, II, and III amounting to around $27 million per year (Adams and Brantner, 2010).

[20] In 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 other fields in the dataset. More details are provided in Appendix B.

milestones (e.g., "new patent application," "target identified," "first launch," and "additional registration for clinical trial"), including drug discontinuations (if publicly disclosed). 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 (or termination) of drug development. Development events include both research and development milestones and important steps in the commercialization process for the underlying drug project. Pharmaprojects therefore allows us to observe a broad set of milestones that indicate development of a drug, including, but not limited to, progress through clinical trials.

[Insert TABLE 1 Here.]

Our sample covers projects initiated between 1989 and 2010, with a focus on projects for which we observe some active development after initiation, or 16,015 projects originated by 4,637 firms.[21] Pharmaprojects data starts from 1989, and we exclude projects initiated in 2011 or after to ensure we observe project development events, discontinuations, and any acquisitions for each project in our sample for at least five full years from initiation. Table 1 provides descriptive information about our main sample. Over the period of our analysis, drug project initiations increase from around 500 per year in the 1990s to around 1,000 projects per year in more recent periods. Table 1 also tabulates projects by broad disease groups. The largest disease areas include therapies targeting cancer and neurological conditions (2,579 and 2,573 projects respectively, each comprising about 16% of the sample).

Figure 4 plots the distribution of the total number of new drugs originated by a company between 1989 and 2010. We find that more than half of companies originate only one drug over this period and nearly 70% originate two projects or fewer. These patterns align with general perceptions of drug development over this period: small firms initiate innovative drug projects that are subsequently developed by large, commercialization-focused incumbent firms (Cockburn, 2004).

[Insert FIGURE 4 Here.]

[21] If we include projects for which we do not observe any active development after initiation, the sample would consist of 35,712 drug projects originated by 6,709 firms. Our results are consistent across the wider sample.

We supplement the Pharmaprojects data with Pharma Intelligence's Trialtrove data on clinical trial phases, 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 (hundreds 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 Trialtrove 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 projects first 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).

3.3. Acquisition Data

We collect acquisition data from three sources. We first extract all announced and completed M&As (with complete information on acquirer and target firms) and announced and effective dates from Thomson Reuters SDC Platinum. To supplement the SDC M&A data, we use Thomson Reuters RecapIQ (now Cortellis Deals Intelligence) data. RecapIQ documents deals in the biotechnology industry using 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,

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

since VentureXpert does not provide details on the acquirer and dates of the acquisition, we manually collect that information.

Armed with acquisition events compiled from multiple data sources, we then conduct a multi-step cleaning process to ensure acquisition events are correctly linked to target and acquirer firms. We first standardize company names (for both acquirers and targets) and collect demographic information for each company. Second, since the same firm could appear in different databases with slightly different names, we create a unique firm identifier by grouping firms with highly similar standardized names and identical demographic characteristics (such as location). Third, using cleaned names of acquirers and targets and deal dates, we drop duplicate 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 combined with 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 in the years pre- and post-acquisition.

The merged drug development and acquisition data show an active acquisition market in the pharmaceutical industry, with nearly 24% of drug projects acquired during development. As tabulated in Table 1, the rate of acquisition is lower for drugs originated more recently. This pattern is likely because acquisitions often occur several years into drug development, and for more recent projects, some acquisitions may have not yet been realized at the time of data construction (i.e., right truncation).

4. Empirical Analysis

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

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

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

We measure overlap between a drug project and the acquiring firm based on a combination of its intended therapeutic class and mechanism of action. The therapeutic class is the disease or condition the therapy targets (e.g., hypertension). We use Pharmaprojects' therapeutic categories, which are based on European Pharmaceutical Market Research Association product categorizations (Kyle, 2007). These categories represent 230 possible therapeutic categories. Within each therapeutic category we also identify drug's mechanism of action, meaning the biological interaction involved in the drug achieving its desired end, including both the molecular target (e.g., beta adrenoreceptor, angiotensin I converting enzyme) and the intended effect (e.g., agonist, antagonist, reducer, inhibitor). The median number of mechanisms of action per therapeutic class in our sample is 7. In our main analyses, we categorize a project as overlapping if the acquiring firm has an existing product in the same therapeutic class which uses the same mechanism of action as that of the acquired drug project. As outlined in Table 1, about one fifth of acquired drug projects overlap with their acquirer. We also measure competition using this categorization. Our competition measure is the number of products in the same therapeutic class using the same mechanism of action.

The logic for measuring overlap narrowly is to ensure that we capture only potential substitute drugs rather than a mixture of substitutes and complements. If we were to instead use same therapeutic class regardless of mechanism of action, we might capture drugs that complement the target's project, either because they treat different sub-markets (i.e., different patient segments with the same disease) or because they are used in treatment for the same patients. By using the same therapeutic class and the same mechanism of action, we lessen the chance of capturing complements. This also allows us to investigate separately the effects of overlap measured more broadly as the same therapeutic class, which we report in supplementary analyses.

Our measure of overlap necessarily differs from measures of competition used by related but distinct streams of the pharmaceutical economics literature. First, the vast literature

that explores generic competition and the effects of generic entry on branded products define competing products as those that are the same chemical entity (Ellison and Ellison, 2011; Arcidiacono et al., 2013; Branstetter et al., 2014). Since we are comparing the development of potentially competing innovative pharmaceuticals, which by definition must be different chemical entities, we cannot use this as our measure. Second, prior research exploring market competition between branded products has defined overlap as the same FDA-approved primary indication or using prescription or usage patterns (Howard et al., 2015). However, because we analyze projects under development, many of which are never approved, let alone marketed, we cannot use approval-contingent categories or usage patterns. Last, some prior research has used the broader measure of same therapeutic class (e.g., Kyle (2007)); we use a narrower measure for the reasons discussed above.

For our main empirical analyses, we use panel data of drug development events. 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,

$$\begin{aligned} Development_{i,t} = \beta \cdot I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i + \gamma_1 \cdot I(Acquired)_i \times I(Post)_{i,t} \\ + \gamma_2 \cdot I(Acquired)_i \times I(Overlap)_i + \gamma_3 \cdot I(Acquired)_i \\ + \alpha_{FE} + \varepsilon_{i,t}, \end{aligned} \tag{9}$$

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 ever 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 confounding effects using a vast array of fixed effects (described below), and standard errors are clustered at the drug project level. We report our results estimated using a linear probability model, but the main results are similar when use logit models.

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 the 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, β, consistent with the prediction that when acquired projects overlap with the acquirer's portfolio, they are more likely to be terminated.[24]

Ideally, if terminations were comprehensively reported in a timely manner, we would use a survival analysis to test if and when drugs projects are shut down. However, project terminations are rarely observed, either at a specific point in time or at all. Hence, in our main specification, we use a lack of ongoing development as a proxy for termination. We test for the likelihood of observed, active development of a project using a project-year panel. There are several advantages to a panel structure which are not possible in a survival analysis, including the ability to account for project-level differences between acquired and non-acquired projects, as well as for pre-acquisition differences between overlapping and non-overlapping acquired projects. To investigate whether we are accurately capturing drug terminations, we perform an additional analysis predicting any post-acquisition development event, described in detail below.

The following subsections detail our empirical analyses. First, we compare drug development rates for non-acquired, acquired non-overlapping, and acquired-overlapping projects (Table 2), including results on an expanded definition of overlap (Table 3). We then deepen our analyses of Proposition 1 by running tests on acquired projects only, single project targets, and by separately analyzing projects which are "never developed" after an acquisition (Table 4). We then analyze the effect of competition (Table 5) and acquirer patent life (Table 6) to test Propositions 2 and 3. After supplementary analysis on clinical trial progression (Table

[24]It is helpful to examine our empirical strategy through the lens of testing strategic entry-deterrence models in the pharmaceutical industry. Ellison and Ellison (2011) show that investments will be monotonic in market size if entry-deterrence motives are absent, but non-monotonic otherwise. Analogously, in our case, if the incentives to discontinue projects due to potential cannibalization of existing drugs are absent and all acquisitions are made with the intent to continue development, overlapping acquired drugs should have equal or even higher continuation rates. This is because only those drug candidates should be acquired which are particularly valuable to an acquirer (e.g., due to synergies resulting from prior experience of developing similar drugs). Thus, in the absence of strategic killing motives the triple interaction term $I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i$ should be positive. In contrast, our theory of strategic acquisition and discontinuation of overlapping projects predicts that this interaction term should be negative.

7), we examine how overlap determines acquisitions, following from Proposition 4 (Table 8). We document acquisition and development patterns around antitrust review thresholds (Table 9 and Figure 5). Finally, we investigate several alternative explanations (Table 10).

4.1. Development of Drug Projects Post-Acquisition

Table 2 presents the regression results. In column (1), the estimate of β is -0.037 and statistically significant at the 1% level, meaning that acquired drug projects that overlap with the acquirers' pipelines are 3.7 percentage points less likely to have a development event in the years post-acquisition compared to non-overlapping acquired projects. Overlapping acquired projects are 5.7 percentage points ($= 0.037 + 0.020$) less likely to experience a development event compared to non-acquired projects. The unconditional probability of having a development event is 19.9%, hence being acquired by a firm with an overlapping project is associated with a 28.6% ($= \frac{0.057}{0.199}$) lower development probability. In column (1), we include project age and vintage fixed effects. Age fixed effects control for the drug development life cycle. Vintage fixed effects focus our estimates on drug projects that are initiated in the same year, and address a potential concern relating to right truncation for more recently initiated projects, especially given the long development timelines for pharmaceuticals (US Food and Drug Administration, 2017).

[Insert TABLE 2 Here.]

In column (2), we include age-therapeutic class-MOA fixed effects. These fixed effects control for potential heterogeneities in the development life-cycle of drugs targeting different diseases, including differences in the stage and complexity of the underlying science, and factors such as the size and geography of patient pools, physician capacity, or patient follow-up times, which can vary greatly across drug markets (US HHS ASPE, 2014). For example, Budish et al. (2015) exposit the logic of how differences in clinical trial lengths and development trajectories arise for different types of cancer treatments caused by varying difficulty of demonstrating effectiveness which is in turn caused by differences in patient survival rates. By controlling for those heterogeneities, we guard against the possibility that acquisitions of overlapping drugs concentrate in areas with a relatively slow development

rates or higher likelihood of failure. The results remain similar with these additional fixed effects.

In column (3), we add originator fixed effects. In doing so, we are effectively using the sample of firms with two or more projects and exploiting variations in development for projects with the same originator firm. In this analysis, $I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i$ estimates the difference in development between acquired overlapping and non-overlapping projects for the same originator (or target). These results show that even within the same target firm, overlapping projects are significantly less likely to have a positive development event compared to non-overlapping projects.

In column (4), we include drug-project-level fixed effects to absorb variation due to unobservable drug-project-specific characteristics, which also subsumes vintage fixed effects. We find that the estimate of β is statistically significant and of similar economic magnitude to column (1): being acquired by a firm with an overlapping project is associated with a 32.7% decrease in development rate.

In column (5), we expand our analysis to explore any differences between drug development trajectories across projects acquired by overlapping versus non-overlapping acquirers that might even partially confound our main results. Given we find decreased development for overlapping acquired projects, one concern is that such projects were on a slower or declining development path compared to other acquired projects pre-acquisition. To investigate this, we include dummy variables for each of the three years prior to the acquisition and allow these pre-acquisition trends to vary across overlapping and non-overlapping acquisitions. The estimated coefficient on the time-trend dummies are insignificant, suggesting that different development trajectories are not driving our results.[25]

Beyond our main finding on overlap, Table 2 also includes several other results that warrant discussion. First, reassuringly, the dummy variables on $I(Acquired)_i \times I(Overlap)_i$ and $I(Acquired)_i$ do not carry loads in the regressions, meaning that acquired drugs do not appear to have a significantly different unconditional likelihood of development pre-acquisition.

[25]Further, in unreported analyses we also control for possible differential development trajectories arising from pre-acquisition co-development or licensing deals, which are common in the pharmaceutical industry. Augmenting our data with comprehensive RecapIQ data on technology-related co-development and licensing deals, we find no differences in our main results after explicitly controlling for pre-acquisition co-development or licensing arrangements.

Second, the γ_1 coefficient associated with $I(Acquired)_i \times I(Post)_{i,t}$ is negative and significant across specifications, implying a lower probability of development or milestone events post-acquisition. One reason for this pattern could be that our measure of overlap (same therapeutic class *and* same mechanism of action) leads to potentially overly tight market definitions and therefore even some non-overlapping acquisitions may actually be killer acquisitions (i.e., substitute projects are acquired and terminated). In particular, drug projects with the same therapeutic class but different mechanisms of action may also be potential substitutes. To empirically investigate this explanation, we re-run our main analysis including a separate triple-interaction term for projects that overlap in terms of therapeutic class only (i.e., have a different mechanism of action) with the acquirer's portfolio of drugs, in Table 3. We find that the therapeutic class-overlapping acquisitions have a lower likelihood of development (statistical significance depends on the fixed effects), but the omitted category of non-overlapping acquired projects now is consistently insignificant, albeit still negative.[26]

[Insert TABLE 3 Here.]

Overall, Table 2 and Table 3 provide evidence that acquired drug projects are less likely to be developed by an acquirer with competing projects, consistent with Proposition 1 of our theoretical model. To deepen our analysis of Proposition 1 we now include some additional Proposition 1 focused analyses with alternative subsamples and specifications.

[Insert TABLE 4 Here.]

First, we re-run our analysis on acquired projects only (i.e., $I(Acquired) = 1$) to partially mitigate any concern that acquired projects may be unobservably different from non-acquired projects. The results in column (1) confirm that projects are much less likely to be developed subsequent to acquisitions by an overlapping acquirer. In terms of economic magnitude, the

[26]The prior literature does not provide a clear expectation for the sign of this coefficient. If we were to assume that projects are only acquired if they are of high quality and that development synergies resulting from economies of scale and scope materialize post-acquisition (Andrade et al., 2001; Henderson and Cockburn, 1996), we would expect a positive coefficient. However, a negative coefficient is consistent with faster termination and lower development rates of larger (acquiring) firms due to the absence of private benefits from continuing development (Guedj and Scharfstein, 2004) or due to agency problems inherent in the organization of large firms (Seru, 2014).

unconditional development probability in this sample is 18.7% and there is a 39.6% lower development rate for overlapping targets.

Second, we examine post-acquisition development only for the cases in which the target has only one drug at the time of acquisition. This analysis investigates concerns that our findings could be the result of acquirer firms acquiring multi-project targets and developinging only the most promising of the acquired projects while discontinuing the other projects. If this mechanism is driving our results, we should expect our focal results to be much less prevalent among single-project acquisitions. As shown in Table 4 column (2), we find the effect of overlap on the post-acquisition development rate to be significant and larger in magnitude that in Table 2, alleviating these concerns.

Last, we perform two different analyses to ensure our main results on decreased likelihood of development are due to project termination rather than to changes in development patterns. First, we re-run our column (1) analysis on acquired projects, removing those projects which are "never-developed" post-acquisition. If terminations are driving our main findings, we should find no significant differences between acquired-overlap and acquired-non-overlap projects in this analysis. In other words, a null result after we take out projects that are never developed post-acquisition is consistent with our predictions. Second, we directly examine the likelihood that a project is "never developed" post-acquisition, which we expect to be significantly higher for overlapping acquisitions. To test this conjecture, we use the sample of acquired projects and create two time periods: pre- and post-acquisition. For the first test, the outcome variable measures any development event during each period; for the second test, the outcome variable is no development event. Correspondingly, in Table 4 column (3), we find no significant differences in likelihood of development events between acquired-overlap and acquired-non-overlap projects. And for the second test, in column (4), we find that overlapping projects are 32.9 percentage points more likely to have no post-acquisition development events—that is, to be effectively terminated—compared to non-overlapping projects. Together, these results further support termination, rather than delayed development, as the primary driver behind our main results.

4.2. Product Market Competition

To investigate Proposition 2 we examine how our empirical development results differ across levels of competition. We measure competition as the count of launched products in the same therapeutic class using the same mechanism of action as the focal project (our measure of "existing product" competition) or similarly overlapping projects under development (our measure of "pipeline" competition).[27]

[Insert TABLE 5 Here.]

Table 5 presents the regression results that 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, with existing product competition measures used in columns (1) to (3) and pipeline competition in columns (4) to (6).[28]

The results suggest that the decreased likelihood of development of overlapping projects during the post-acquisition period concentrates in product markets with relatively low competition. Comparing columns (1) and (2), we observe that development of an overlapping acquired drug in the low competition environment decreases by 6.5 percentage points, while under high competition, the coefficient is 0.017 and statistically insignificant. The coefficient estimate of -0.065, together with unreported coefficient -0.015 associated with $I(Acquired) \times I(Post)$, means that acquired overlapping projects are 41.2% ($= \frac{0.065+0.015}{0.194}$) less likely to be developed than non-acquired drugs as benchmarked by the unconditional development rate in the subsample (19.4%). In column (3), we test the difference between high and low competition using an interaction term. The results in columns (4) to (6) for competition among pipeline products show similar findings.

[27]Note 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.

[28]As 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 competitors' pipelines are observable, we use pipeline data as a measure of future or expected competition.

Beyond providing supportive evidence for Proposition 2, these analyses highlight a positive reinforcement loop from competition. If incumbents face significant existing competition, acquired projects are not significantly more frequently discontinued than independent projects. Thus, more competition deters incumbents from acquiring and terminating the projects of potential future competitors, which leads to more competition in the future.

4.3. Heterogeneity across Patent Expiration

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 findings in Table 2. We perform this analysis on overlapping acquired projects, and thereby focus solely on how variation in remaining patent life (of the acquiring firm's relevant patent) affects our baseline results. For each of those projects, we identify the patents associated with the relevant (overlapping) approved drugs of the acquiring firm. We source patent data matched at the drug-level via Pharmaprojects (which uses the FDA Orange Book data), and link patent filing dates from United States Patent and Trademark Office (USPTO) data.

[Insert TABLE 6 Here.]

Table 6 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 expiration (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 expiration, the decrease in development associated with acquistion appears to be mitigated. That is, among overlapping acquired drugs, those for which the acquirer patents are near expiration are more likely to have development events post-acquisition compared to projects for whom the relevant acquirer patents are relatively far from expiration.

4.4. Evidence from Clinical Trials

To supplement the preceding analyses of development events writ large, we examine the likelihood that a project continues in the clinical trials process. In addition to providing

robustness for our main results, analyzing progression through the clinical trial phases allows us to focus, albeit narrowly, on drugs in the exact same phase of clinical development and examine progress to the next necessary phase. It also more closely replicates related research on drug development (Guedj and Scharfstein, 2004; Krieger, 2017). Focusing in this way helps to alleviate concerns that our main results are driven by differences in the stage of development across projects that might remain even after including various age, project, and firm related fixed effects. We treat the clinical trial analysis as supplementary evidence because our main analyses include a larger sample of projects, additional key development events besides trial starts (e.g., patent applications), and the panel structure allows for project-level fixed effects.

In this analysis, we focus on the subsample of drugs that start Phase I clinical trials and are subsequently acquired. That is, $I(Acquired) = 1$ holds for all observations in this subsample. We test whether drug projects acquired by firms with overlapping products are less likely to start Phase II trials than drug projects that are acquired by non-overlapping acquirers, using the following specification,

$$PhaseII_i = \beta \cdot I(Acquired)_i \times I(Overlap)_i + \alpha_{FE} + \varepsilon_i. \tag{10}$$

Note that we focus on those projects started before 2011 to ensure sufficient time to observe an acquisition and to give the analyzed projects sufficient time to enter Phase II trials. $PhaseII_i$ indicates whether drug i ever entered the Phase II trial. As before, the key coefficient of interest is $I(Acquired)_i \times I(Overlap)_i$, which indicates that the drug was acquired by an incumbent with an overlapping product.

[Insert TABLE 7 Here.]

Table 7 presents the clinical trial regression results. We find projects that are acquired by firms with overlapping products are 17.7 percentage points less likely to progress to Phase II than non-overlapping acquired projects (column (1)). In terms of economic magnitude, this represents a decrease of 46.6% from the base rate of starting Phase II for acquired projects (38.0%). Columns (2) through (5) examine how competition conditions these results. We find that the decreased likelihood of acquired overlapping projects progressing in clinical trials is

concentrated in markets with low existing product market or pipeline competition, akin to our main analyses.

4.5. Acquisition Decisions

4.5.1. Determinants of Acquisitions. Our empirical analysis so far has focused on drug development, finding that a project is less likely to be developed after being acquired by a firm with an overlapping existing drug (consistent with Proposition 1), that these results are concentrated in markets with low levels of competition (Proposition 2), and when relevant acquirer patents are far from expiration (Proposition 3). Additionally, our theoretical model predicts that the incentives to discontinue the development of threatening innovation should lead firms to exhibit a particular selection pattern in their search for possible targets. In particular, Proposition 4 states that acquiring incumbents should acquire target firms with overlapping drugs (i.e., that overlap will positively predict acquisition).

To test this prediction, we compare completed deals with pseudo control deals and employ a conditional logit regression (McFadden, 1974) using cross-sectional data. Following Bena and Li (2014), for each completed pair of acquirer-target project, we construct two different control samples as pools of potential acquisition deals (the pseudo deals). First, we form a random control sample: for each pair of acquirer firm j and target drug i, we randomly draw five firms from the pool of firms which ever performed an acquisition prior to the deal year. For each of those pseudo acquirers we then form pseudo acquisitions with target project i. Second, we form a size-matched control sample: we match each acquirer in each deal to five control firms based on their total number of drug projects in the year of the deal.

The analysis is performed using the following model:

$$Acquirer\text{-}Target_{ijd,t} = \beta \cdot I(Overlap)_{ijd,t} + \alpha_{FE} + \varepsilon_{ijd,t}. \tag{11}$$

The dependent variable, $Acquirer\text{-}Target_{ijd,t}$, is equal to one if firm-project pair ij is a real acquirer-target pair, and zero otherwise (i.e., a pseudo-pair). The key explanatory variable $I(Overlap)$ is constructed for each firm pair and captures whether firm j has any product that overlaps with the acquired project i. Fixed effects are at the deal level (indexed by d) for

each real acquirer-target and its control pairs. Our goal is to examine whether overlapping projects in the target's pipeline drive the acquirer's purchase decision.

[Insert TABLE 8 Here.]

Table 8 presents the marginal effects from a conditional logit estimation evaluated at the mean, separately for each control sample: random-matched in columns (1) to (4) and size-matched in columns (5) to (8). In column (1), the estimated marginal effect of 0.626 implies that acquisitions are almost four times more likely when the incumbent acquirer's products narrowly overlap with the target's development projects, compared to the baseline acquisition rate of 16.7%. In column (2), we find similar patterns if the overlapping measure is more broadly defined (same therapeutic class). In columns (3) and (4) we study the effect of market competition on the acquisition decision. The results suggest that target drugs in low competition markets are more likely to be acquired by an overlapping buyer. Columns (5) to (8) duplicate these results for the size-matched control sample. Collectively, these results suggest that overlap significantly influences the acquisition decisions of incumbents.

Thus, acquisitions disproportionately involve target firms with projects that could potentially develop into products that overlap and compete with the acquirer's products. On its own, the propensity to undertake overlapping acquisitions does not demonstrate a strategic "killer acquisition" motive. However, alternative theories of corporate acquisition and development choices cannot satisfactorily explain both our acquisition and drug development results. First, in contrast to our empirical finding of acquisitions of overlapping targets, acquisition motives based on empire building or managerial risk diversification theories would imply that incumbent acquirers should target non-overlapping projects. Second, although our results showing that overlap predicts acquisitions are, on their own, consistent with acquisitions motivated by project development synergies, such a synergy-based theory would predict that acquired overlapping projects are subsequently more likely to be developed rather than less. Hence, a synergy motive contrasts sharply with our empirical findings of decreased development in Section 4.1.

4.5.2. Antitrust and FTC Review Thresholds. In the pharmaceutical industry incumbents often conduct acquisitions when the target's technology or project is still at a nascent

stage. As a result, some of these deals are exempted from the pre-merger review rule of the FTC under the Hart-Scott-Rodino (HSR) Antitrust Improvements Act of 1976 because they fall below the acquisition deal size threshold.[29] To further strengthen the claim that "killer acquisitions" are the driving force behind our empirical results, we now present evidence that incumbent acquirers conduct overlapping acquisition deals that do not trigger FTC reporting requirements under HSR and thereby avoid antitrust scrutiny.

Specifically, we examine acquisitions around the HSR review threshold and compare the project development decisions for transactions just above and below the threshold. If incumbent 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 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 $([0, 5\%]$. Acquisition size is proxied by the deal amount. We categorize acquisitions into acquisitions of non-overlapping targets (left panel) and acquisitions of overlapping targets (right panel). We observe clear bunching of deals right below the review threshold, but this pattern is only apparent for deals in which the target has projects that overlap with the acquirer (i.e., "killer acquisition" suspects).

[Insert TABLE 9 Here.]

In Table 9, we compare the termination and launching rates of acquisitions around the threshold. We construct two buckets, which include all acquisitions with a transaction

[29] 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 with a target valuation 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: If the larger party has less than $100 million in assets or sales or the smaller party has less than $10 million in assets, the deal does not need to be reviewed by the FTC. Because in the pharmaceutical industry the size-of-the-person test is typically not satisfied for the smaller (target) party, 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.

value just below or above 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.

5. Discussion

5.1. Alternative Explanations

In this section we address several potential alternative explanations which a priori could be consistent with our main findings. Importantly, a plausible alternative explanation would have to explain not just why acquired drug projects are more likely to be terminated, but also why overlapping acquired drug projects are more likely to be terminated than non-acquired or non-overlapping acquired drug projects.

5.1.1. Informational Asymmetries in the Acquisition Market. Focusing on overlapping acquired projects means that asymmetric information or "market for lemons" type arguments are an implausible candidate explanation. Although 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 lower when the acquirer has its own overlapping projects and therefore has knowledge of both the underlying science and the eventual market of the drug candidate. Our main results are therefore unlikely to be a simple market for lemons story.

5.1.2. 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 4), we found results similar to those in the full sample. Our main results are therefore unlikely to be driven by optimal project selection.

5.1.3. 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 more likely to be underpinned by 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 initiated 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, commonly used to measure similarity between two molecules in the chemical informatics literature (Nikolova and Jaworska, 2003). It is the proportion of chemical fragments shared by any two chemicals, bounded by 0 and 1, with 0 indicating the pair share no common chemical fragments. If acquired drugs are redeployed, one would expect acquirer firms' post-acquisition projects to be more similar to the acquired project than their pre-acquisition projects.

[Insert TABLE 10 Here.]

Table 10 Panel A presents chemical similarities to the acquired drug for drugs initiated by the acquirer post-acquisition compared to pre-acquisition drugs. In columns (1) to (3), 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 initiated by acquirer firms after the acquisition of a drug do not become more similar to the acquired overlapping drug compared to pre-acquisition projects. 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.

In columns (4) to (6), we adopt the same analytical structure to study an alternative measure of technology redeployment, namely patent citations to acquired projects. We investigate whether the patents for drugs developed in acquirer firms after the acquisition of an overlapping drug cite the target's patents. Echoing columns (1) to (3), we find no evidence of redeployment.

5.1.4. Redeployment of Human Capital. Similar to technology redeployment, the key motivation behind an acquisition could be to acquire the target firm's valuable human capital such as its research team or other key individuals (Lacetera et al., 2004; Ouimet and Zarutskie, 2011). Under this view, the lack of development of acquired, overlapping projects could be a byproduct 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 more useful for the acquiring firm, and so this alternative explanation could apply to our main analyses.

Before empirically addressing human capital redeployment, it is worth highlighting that "acqui-hiring"—the practice of acquiring startups, jettisoning the core business, and retaining the employees (Chatterji and Patro, 2014; Kim, 2018)—might not be as common in pharmaceuticals as in other industries. This is because the pharmaceutical industry is almost exclusively project-driven (Gompers et al., 2016) with strong project-specific intellectual property rights protection, in contrast to many other industries in which startups are valued more for their human capital. However, to formally investigate human capital redeployment independent of technology redeployment, we explore inventor mobility and productivity following acquisitions.

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 HBS 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 pre-acquisition target firm inventors by identifying those who filed at least one patent within the five-year window prior to the 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 target firm inventors should be retained and that those who stay should become more productive as they are redeployed away from the terminated projects.

Table 10 Panel B shows the human capital results. First, only 22% of pre-acquisition inventors move to the acquirer after the acquisition, while 78% move to other firms. Second, while those who stay and those who leave 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 directly link target firm patents to a specific drug project because of their early stage.[30] As a result, we are not able to identify 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).

5.2. Frequency and Importance of Killer Acquisitions

Our empirical estimates document effects of acquisitions on project development when there is overlap with acquirers' existing product portfolios. 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. The

[30]That 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.

unconditional probability of having a development event is 19.9%. Using the estimates from our tightest specification reported in Table 2 column (4), we find that acquired projects with overlap (25.5% of acquired projects) continue at an adjusted rate of 13.4%, while acquired projects without overlap (74.5% of acquired projects) continue development at an adjusted rate of 17.5%. 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 (17.5%). 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 (13.4%)? Specifically, we solve the equation $13.4\% = \nu \times 0 + (1 - \nu) \times 17.5\%$ for ν which yields $\nu = 23.4\%$. Therefore, we estimate that 6% ($= \nu \times 25.5\%$) of all acquisitions or about 45 ($= 6\% \times 758$) 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 probability in our sample is 18.4%. Consider first the case in which acquisitions of overlapping projects are no longer allowed and that all such projects instead have the same development probability (19.9%) as non-acquired

projects (56.1% of all projects). In that case, the number of total drug projects for which development continues would increase by 4.0% ($= \frac{19.9\%-13.4\%}{18.4\%} \times (1 - 56.1\%) \times 25.5\%$) or by about 13 drug projects per year ($= 18.4\% \times 4.0\% \times 1,727$ 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, as large as half of the size of the Orphan Drug Act.

5.3. 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.[31]

[31] Although 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 partly duplicates development costs, but does not provide a sufficiently large increase in consumer surplus to fully compensate the loss in producer surplus of the existing incumbents as in Mankiw and Whinston (1986). 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

A comprehensive welfare analysis of the impact of killer acquisitions is, however, much 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). In our model, entrepreneurs are born with a project and thus do not have to exert effort to come up with an idea, but it is plausible that the prospect of later acquisition may motivate the origination of entrepreneurial ideas. Yet, killer acquisitions will motivate such idea origination only if the entrepreneur receives some of the surplus that accrues to the incumbent through the acquisition.[32] 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 (Garattini, 1997; Arcidiacono et al., 2013) at the expense of the origination of

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.

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

truly novel products.[33] This response to the prospect of acquisition would likely add to the negative welfare impact of killer acquisitions.[34]

Because killer acquisitions may motivate ex-ante innovation, the overall effect of such acquisitions on social welfare remains unclear. However, we think it unlikely that this acquisition channel, which generates significant ex-post inefficiencies resulting from the protection of market power, is the most effective way to motivate ex-ante innovation. In particular, this is because our analysis emphasizes the positive reinforcement loop of competition: Because killer acquisitions are less likely to occur when incumbents face significant existing competition, 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, thus increasing future competition.

6. 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 incumbents acquire firms with overlapping drug projects and that these 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, delayed development, and the redeployment of technological or human 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, suggesting that the potentially damaging consequences reach beyond pharmaceuticals. Our results caution against

[33] A variety of evidence (Adams and Brantner, 2006; Budish et al., 2015) suggests that intellectual property protection, most notably patents, plays a key role in motivating innovation and influencing the direction of innovative efforts in the pharmaceutical industry.

[34] 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.

interpreting acquisitions of nascent technologies solely as incumbents' efforts to integrate and foster entrepreneurial innovation. Instead, a significant part of what is fueling this trend may actually be killer acquisitions that potentially harm innovation and competition.

Our results also suggest that antitrust policy should continue to closely scrutinize the 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 appear to routinely avoid regulatory scrutiny by acquiring entrepreneurial ventures at transaction values below the HSR review thresholds exacerbates the concern.

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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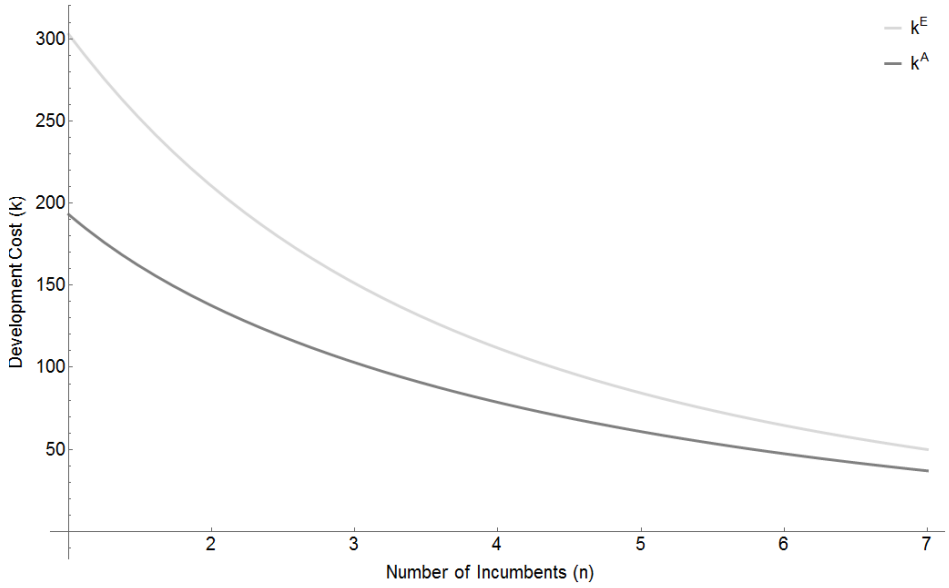
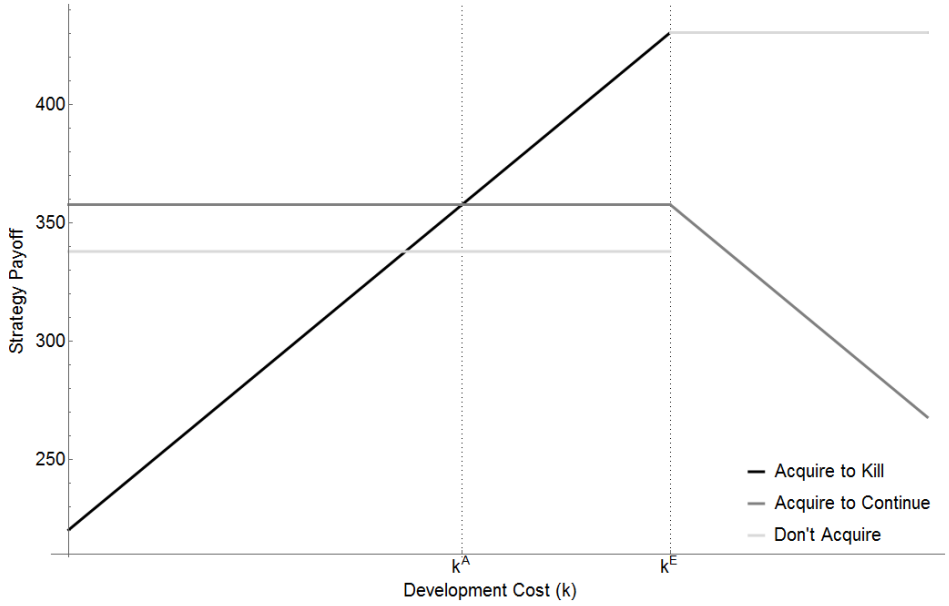
Figure 2. Development Cost Thresholds and Competition



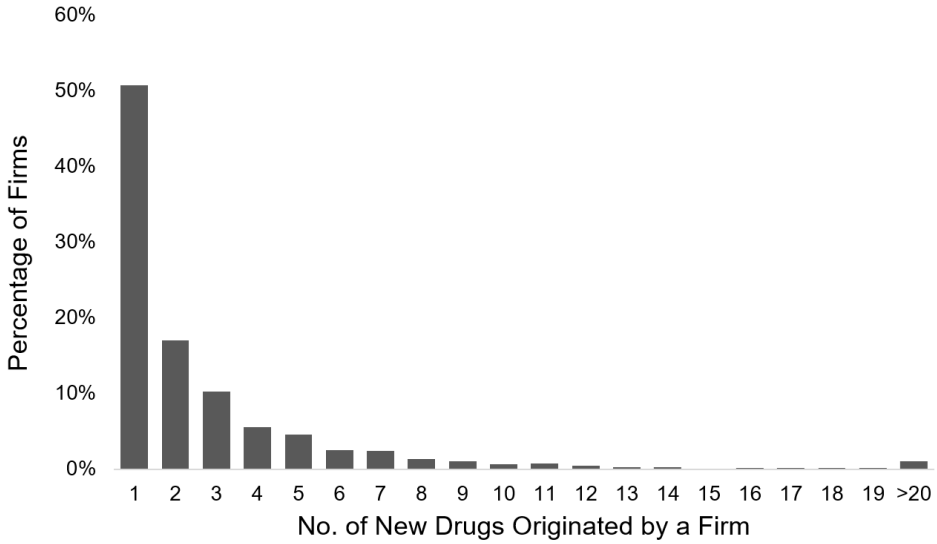
This graph plots the optimal development cost thresholds of the entrepreneur (k^E, light gray) and the acquirer (k^A, dark gray) as a function of the number of incumbents n. The other parameter values are $\alpha = 100$, $\beta = 4$, $\gamma = 1.5$, $\rho = 0.75$, and $L = 20$. α represents overall product quality, β measures the concavity of the utility function, and γ represents the degree of substitutability. See Appendix A for more details.

Figure 3. Strategy Payoffs



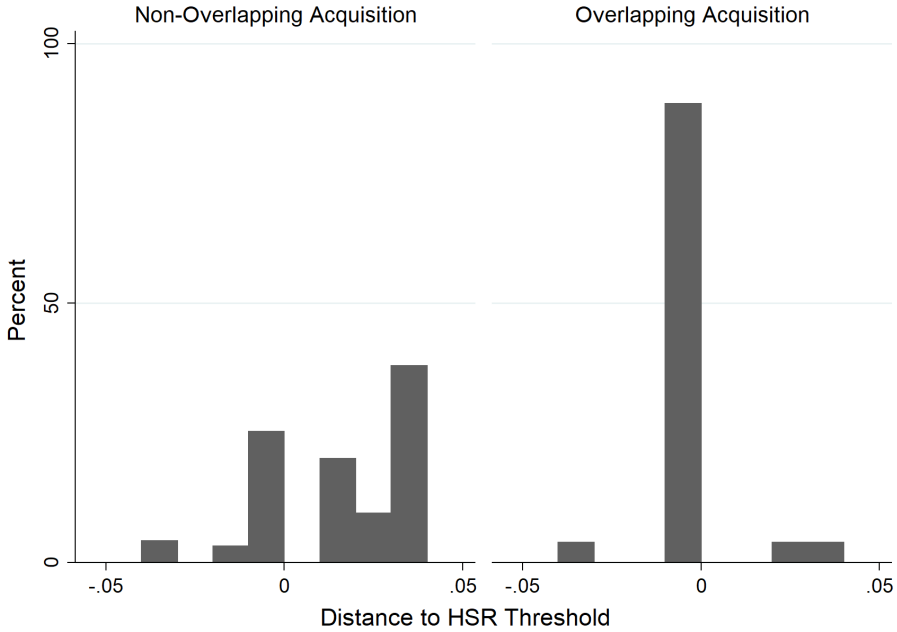
This graph plots the incumbent's payoff from pursuing one of the three acquisition strategies—"Don't Acquire" (light gray), "Acquire to Kill" (black), and "Acquire to Continue" (dark gray)—as a function of the development cost k. The other parameter values are $\alpha = 100$, $\beta = 4$, $\gamma = 1.5$, $\rho = 0.75$, $L = 20$, 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 overlappling 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	16,015	78%	17%	5%
By Time Period				
Beginning-1995	1,605	60%	31%	9%
1996-2000	1,933	68%	25%	7%
2001-2005	3,739	79%	16%	4%
2006-2010	5,208	90%	8%	2%
By High-level Disease Group (top 5)				
Anti-cancer (13 therapeutic classes; 783 ThC/MoA)	2,579	80%	16%	4%
Neurological (27 therapeutic classes; 986 ThC/MoA)	2,573	77%	19%	4%
Anti-infectives (28 therapeutic classes; 452 ThC/MoA)	1,946	77%	16%	7%
Biotechnology (26 therapeutic classes; 209 ThC/MoA)	1,493	79%	16%	5%
Alimentary/Metabolism (24 therapeutic classes; 498 ThC/MoA)	1,380	81%	15%	4%

Table 2
Overlapping Acquisitions and Project Development

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

$$Development_{i,t} = \beta \cdot I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i + \gamma_1 \cdot I(Acquired)_i \times I(Post)_{i,t} + \gamma_2 \cdot I(Acquired)_i \times I(Overlap)_i + \gamma_3 \cdot I(Acquired)_i + \alpha_{FE} + \varepsilon_{i,t},$$

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 is acquired during the study period and $I(Post)_{i,t}$ indicates whether the drug-year (i,t) observation is after the drug is acquired. $I(Overlap)$ is a dummy variable indicating the acquired drug overlaps with the product portfolio of the acquirer. Before(-t) indicates that the drug-year is t years before an acquisition and takes zero otherwise. 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)
I(Acquired) × I(Post) × Overlap	-0.037***	-0.033**	-0.029*	-0.041**	-0.054**
	(0.013)	(0.014)	(0.015)	(0.019)	(0.024)
I(Acquired) × I(Post)	-0.020***	-0.016**	-0.017**	-0.024**	-0.018
	(0.006)	(0.007)	(0.009)	(0.010)	(0.013)
I(Acquired) × Overlap	0.004	0.009	0.026**		
	(0.008)	(0.009)	(0.011)		
I(Acquired)	-0.002	-0.004	-0.011		
	(0.004)	(0.005)	(0.012)		
Before(-3) × Overlap					-0.031
					(0.032)
Before(-2) × Overlap					0.012
					(0.032)
Before(-1) × Overlap					-0.040
					(0.030)
Before(-3)					0.015
					(0.017)
Before(-2)					0.020
					(0.017)
Before(-1)					-0.003
					(0.016)
Observations	143,569	143,569	143,569	143,569	143,569
R-squared	0.038	0.252	0.289	0.366	0.370
Vintage FE	Y	Y	Y		
Age FE	Y				
Age FE X Therapeutic Class X MOA		Y	Y	Y	Y
Originator [Target Company] FE			Y		
Project FE				Y	Y

Table 3
Overlapping Acquisitions and Project Development: Measures of Overlap

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

$$\begin{aligned} Development_{i,t} = {} & \beta_1 \cdot I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i \\ & + \beta_2 \cdot I(Acquired)_i \times I(Post)_{i,t} \times I(OverlapDiseaseOnly)_i \\ & + \gamma_1 \cdot I(Acquired)_i \times I(Post)_{i,t} + \gamma_2 \cdot I(Acquired)_i \times I(Overlap)_i \\ & + \gamma_3 \cdot I(Acquired)_i \times I(OverlapDiseaseOnly)_i + \gamma_4 \cdot I(Acquired)_i + \alpha_{FE} + \varepsilon_{i,t}, \end{aligned}$$

where the dependent variable $Development_{i,t}$ is a dummy variable indicating drug i has a development event in year t. $I(Acquired)_i$ indicates drug i is acquired during the study period and $I(Post)_{i,t}$ indicates whether the drug-year (i,t) observation is after the drug is acquired. $I(Overlap)$ is a dummy variable indicating the acquired drug overlaps with the product portfolio of the acquirer. $I(Overlap)$ (Disease Only) is a dummy variable indicating the acquired drug overlaps with the product portfolio of the acquirer through the same therapeutic class only (not same mechanism of action). 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)
	Development Event = 1			
I(Acquired) × I(Post) × Overlap	-0.052***	-0.037**	-0.036**	-0.051**
	(0.014)	(0.015)	(0.016)	(0.020)
I(Acquired) × I(Post) × Overlap (Disease Only)	-0.046***	-0.018	-0.022	-0.036*
	(0.012)	(0.017)	(0.018)	(0.021)
I(Acquired) × I(Post)	-0.005	-0.012	-0.010	-0.013
	(0.007)	(0.009)	(0.010)	(0.012)
I(Acquired) × Overlap	0.009	0.007	0.034**	
	(0.008)	(0.009)	(0.013)	
I(Acquired) × Overlap (Disease Only)	0.013*	-0.007	0.015	
	(0.007)	(0.010)	(0.013)	
I(Acquired)	-0.007	-0.001	-0.015	
	(0.005)	(0.006)	(0.013)	
Observations	143,569	143,569	143,569	143,569
R-squared	0.037	0.252	0.289	0.366
Vintage FE	Y	Y	Y	
Age FE	Y			
Age FE X Therapeutic Class X MOA		Y	Y	Y
Originator [Target Company] FE			Y	
Project FE				Y

Table 4
Overlapping Acquisitions and Project Development: Alternative Specifications

This table presents the post-acquisition development likelihood of acquired drug projects (column 1), single project targets (column 2), acquired drug projects with some post-acquisition development (column 3), and the post-acquisition likelihood of never experiencing a development event (column 4). The general empirical specification is,

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

where the dependent variable $Development_{i,t}$ is a dummy variable indicating whether or not drug i has a development event in period t (or has no development event in period t for the column 4 analyses). $I(Acquired) \times I(Post)$ indicates whether the drug-period(i,t) observation is after the drug is acquired and $I(Acquired) \times I(Post) \times I(Overlap)$ also indicates the acquired drug overlaps with the acquirer's product portfolio. 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)
	Development Event = 1			No Development = 1
I(Acquired) × I(Post) × Overlap	-0.050**	-0.121***	0.005	0.329***
	(0.023)	(0.060)	(0.035)	(0.015)
I(Acquired) × I(Post)	-0.024	-0.041	-0.095***	0.123***
	(0.015)	(0.025)	(0.013)	(0.032)
Observations	27,784	19,651	7,916	8,970
R-squared	0.445	0.249	0.155	0.237
Sample:	Acquired Projects Only	Single-Project Target Only	Excluding "never developed"	
Age FE X Therapeutic Class X MOA	Y	Y	Y	Y
Project FE	Y	Y	Y	Y

Table 5
Overlapping Acquisitions and Project Development: Market Competition

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

$$Development_{i,t} = \beta \cdot I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i + \gamma_1 \cdot I(Acquired)_i \times I(Post)_{i,t} + \gamma_2 \cdot I(Acquired)_i \times I(Overlap)_i + \gamma_3 \cdot I(Acquired)_i + \alpha_{FE} + \varepsilon_{i,t},$$

where the dependent variable $Development_{i,t}$ is a dummy variable indicating drug i has a development event in year t. $I(Acquired)_i$ indicates drug i undergoes an acquisition event and $I(Post)_{i,t}$ indicates 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 market (same therapeutic class and mechanism of action) as the focal drug. In columns (1) to (3), the competition measure is calculated using existing launched products, while in columns (4) to (6), the measure is calculated using the pipeline (pre-launched projects). Drug development projects are categorized into high and low competition by the median of competition measures. In columns (3) and (6) we present results in which we interact $I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i$ with the dummy indicating low competition. 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)
	Low	High	Interacted	Low	High	Interacted
I(Acquired) × I(Post) × Overlap	-0.065**	0.017	0.017	-0.083***	-0.012	-0.012
	(0.026)	(0.035)	(0.035)	(0.030)	(0.026)	(0.027)
I(Acquired) × I(Post) × Overlap × LowCompetition			-0.082*			-0.071*
			(0.044)			(0.040)
Competition Measure	Product Competition			Pipeline Competition		
Observations	74,261	69,308	143,569	72,782	70,787	143,569
R-squared	0.415	0.399	0.408	0.425	0.348	0.385
Age FE X Therapeutic Class X MOA	Y	Y	Y	Y	Y	Y
Project FE	Y	Y	Y	Y	Y	Y

Table 6
Overlapping Acquisitions and Project Development: Acquirer's Patent Life

This table presents the development likelihood of drug projects using a drug-year panel sample. The sample for this analysis is acquired projects where the project overlaps with the portfolio of the acquiring firm. The analysis investigates how the remaining patent term length of the acquirer's relevant patent (the "overlapping" patent) influences the effect of acquisition on the likelihood of development. The empirical specification uses the following model,

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

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. $I(NeatPatentExpiry)$ is a dummy variable indicating whether the overlapping acquirer drug is within 5 years of patent expiry. 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)	0.013	0.406***
	(0.133)	(0.090)
I(Post)	-0.173*	-0.210***
	(0.092)	(0.067)
I(Near Patent Expiry)	-0.104**	-0.147***
	(0.043)	(0.043)
Observations	6,398	6,398
R-squared	0.212	0.450
Vintage FE	Y	Y
Age FE	Y	
Therapeutic Class X MOA FE	Y	
Age X Therapeutic Class X MOA FE		Y

Table 7
Overlapping Acquisitions and Project Development: Clinical Trials

This table presents the likelihood of starting Phase II trials. An observation is a drug project that is acquired after starting Phase I clinical trials. We test whether drug projects acquired by firms with overlapping products are less likely to subsequently start Phase II trials than drug projects that are acquired by non-overlapping acquirers, using the following specification,

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

where the dependent variable $PhaseII_i$ is a dummy variable indicating whether drug i enters Phase II. $I(Acquired)_i$ indicates whether the drug (i) is acquired in Phase I. Note that $I(Acquired) = 1$ holds for every observation in this sample. $I(Overlap)$ is a dummy variable indicating whether the acquired drug overlaps with the portfolio of the acquirer. Standard errors are displayed in parentheses. ***, **, and * indicate significance at the 1%, 5%, and 10% levels, respectively.

		Phase II = 1			
	(1)	(2)	(3)	(4)	(5)
		Low Competition	High Competition	Low Competition	High Competition
I(Acquired by Overlapping Firms)	-0.177***	-0.356***	-0.142***	-0.396***	-0.068**
	(0.028)	(0.071)	(0.031)	(0.049)	(0.034)
Competition Measure		Existing Product		Pipeline	
Observations	1,860	511	1,348	631	1,228
R-squared	0.151	0.286	0.156	0.321	0.145
Phase I Start Year FE	Y	Y	Y	Y	Y

Table 8
Product Overlap and Acquisition Decisions

This table presents estimates of conditional logit models to explain the likelihood of an acquisition of a drug project. The sample for this analysis includes all completed project-firm pairs and one (of two) control samples: a random-matched sample (columns (1) to (4)) and a size-matched sample (columns (5) to (8)). The empirical specification uses the following model,

$$Acquirer\text{-}Target_{ijd,t} = \beta \cdot I(Overlap)_{ijd,t} + \alpha_{FE} + \varepsilon_{ijd,t}.$$

where the dependent variable $Acquirer\text{-}Target_{ijd,t}$ is a dummy variable indicating drug i is acquired by firm j in year t (and is otherwise a pseudo-pair). $I(Overlap_{ijd,t})$ is a dummy variable indicating the target drug overlaps the potential acquirer firm. *LowCompetition* is a dummy variable indicating the target drug is in a low competition market. We include deal-level fixed effects (for both realized and pseudo deals). We report marginal effects from the estimation. Standard errors clustered at the deal level are displayed in parentheses. ***, **, and * indicate significance at the 1%, 5%, and 10% levels, respectively.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Acquisition = 1				Acquisition = 1			
Overlap	0.626***		0.577***		0.194***		0.209***	
	(0.009)		(0.015)		(0.010)		(0.015)	
Overlap (Disease Only)		0.356***		0.300***		0.214***		0.200***
		(0.005)		(0.008)		(0.008)		(0.011)
Overlap × LowCompetition			0.088***				-0.027	
			(0.019)				(0.020)	
Overlap (disease only) × LowCompetition				0.103***				0.025*
				(0.011)				(0.015)
Observations	55,374	55,374	38,430	38,430	34,005	34,005	34,005	34,005
Pseudo R-squared	0.118	0.119	0.098	0.097	0.052	0.064	0.052	0.065
Deal FE	Y	Y	Y	Y	Y	Y	Y	Y
Matching Method	Random Matching				Matched by Pipeline Size			
No of Deals	9,229	9,229	9,229	9,229	9,229	9,229	9,229	9,229
No of Control Deals	46,145	46,145	46,145	46,145	46,145	46,145	46,145	46,145

Table 9
The Intensity of Project Discontinuation around FTC Review Threshold

This table presents univariate survival tests on the drugs that are acquired just below $[-5\%, 0]$ 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			

Table 10
Post-Acquisition Asset Redeployment

Panel A: Project Similarities to Acquired Drugs Pre- and Post-Acquisition

This table studies patent citations and 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). To measure citations we use USPTO data. 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)
	Chemical Similarity			Citation to Targets		
I(Post) × Overlap	0.001	0.000	0.002	-0.002	-0.002	-0.000
	(0.003)	(0.003)	(0.002)	(0.002)	(0.002)	(0.000)
I(Post)	-0.002	-0.001	-0.004	0.000	0.001	0.000
	(0.004)	(0.004)	(0.003)	(0.000)	(0.001)	(0.000)
Overlap	0.004	0.004		0.002	0.002	
	(0.003)	(0.003)		(0.002)	(0.002)	
Observations	154,896	154,896	154,896	154,896	154,896	154,896
R-squared	0.001	0.014	0.361	0.001	0.094	0.154
Acquirer FE		Y			Y	
Case FE			Y			Y

Panel B: Inventor Mobility and Patent Productivity Pre- and Post-Acquisition

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***

Appendix

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

$$U(q) = \alpha \sum_{i=1}^{n} q_i - \frac{1}{2}\left(\beta \sum_{i=1}^{n} q_i^2 + 2\gamma \sum_{i \neq j} q_i q_j\right) \tag{12}$$

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

$$\max_{p_E} p_E q_E \tag{13}$$

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

$$\pi^E_{\neg acq,S} = \frac{\alpha^2(\beta - \gamma)(\beta + (n-1)\gamma)}{(2\beta + (n-2)\gamma)^2(\beta + n\gamma)} = \pi^A_{\neg acq,S}. \tag{14}$$

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

$$\pi^A_{\neg acq,F} = \frac{\alpha^2(\beta-\gamma)(\beta+(n-2)\gamma)}{(2\beta+(n-3)\gamma)^2(\beta+(n-1)\gamma)} = \pi^I_{\neg acq,F}. \tag{15}$$

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

$$\max_{p_1,p_2} p_1 q_1 + p_2 q_2 \tag{16}$$

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

$$\pi^A_{acq,S} = \frac{\alpha^2(\beta-\gamma)(\beta+(n-2)\gamma)(2\beta+\gamma(2n-1))^2}{2(\beta+n\gamma)(2\beta^2+(3n-4)\beta\gamma+(1+(n-3)n)\gamma^2)^2}. \tag{17}$$

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

$$\pi^A_{acq,F} = \frac{\alpha^2(\beta-\gamma)(\beta+(n-2)\gamma)}{(2\beta+(n-3)\gamma)^2(\beta+(n-1)\gamma)}. \tag{18}$$

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

$$\pi^A_{acq,S} > \pi^A_{acq,F} = \pi^A_{\neg acq,F} > \pi^A_{\neg acq,S} = \pi^E_{\neg acq,S} > \pi^E_{\neg acq,F} = 0 \tag{19}$$

as well as the following inequality

$$\Delta^E \equiv \pi^E_{\neg acq,S} - \pi^E_{\neg acq,F} > \pi^A_{acq,S} - \pi^A_{acq,F} \equiv \Delta^A. \tag{20}$$

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 $\Delta^E \equiv \pi^E_{\neg acq,S} - \pi^E_{\neg acq,F}$ and $\Delta^A \equiv \pi^A_{acq,S} - \pi^A_{acq,F}$. It is immediately apparent that for $\gamma = 0$ 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. □

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 $\pi^E_{\neg acq,S}$, $\pi^E_{\neg acq,F}$, $\pi^A_{acq,S}$, and $\pi^A_{acq,F}$ and differentiation of $\Delta^E - \Delta^A$ with respect to n establishes the result. Furthermore, we have $\lim_{n\to\infty}(k^E - k^A) = 0$. □

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 $\Delta_{gen} = \Delta^E_{gen} = \Delta^A_{gen}$. These (equal) development gains are different from the previous expressions Δ^E and Δ^A. This is because when a generic product (that is undifferentiated from the acquirer's existing product) enters, it not only drives profits of that product to zero, but due to its low price it also reduces the profits of the other products that

are differentiated from it. Thereafter, the profits for the acquirer's existing product drop to 0, and hence his incentives to develop coincide with those of the entrepreneur.

Thus, the development decisions of the entrepreneur d^E_{gen} and the acquiring incumbent d^A_{gen} 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. □

A.1.7. Acquisition Decision.

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

$$d^A[\rho\pi^A_{acq,S} + (1-\rho)\pi^A_{acq,F} - k] + (1-d^A)(L + \pi^A_{acq,F}) - P \geq \\ d^E[\rho\pi^A_{\neg acq,S} + (1-\rho)\pi^A_{\neg acq,F}] + (1-d^E)\pi^A_{\neg acq,F} \quad (21)$$

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,

$$P = d^E(\rho\Delta^E - k) + (1-d^E)L. \quad (22)$$

Substituting the takeover price (22) into the inequality for the acquisition decision (21) and solving for each of the three cases of ρ establishes the acquisition choices outlined in the main text for the case where $\beta > \gamma > 0$.

In the case in which there is no product market overlap ($\gamma = 0$), there is no replacement effect. As a result, the incremental profit from developing the product is the same for the incumbent acquirer and the entrepreneur, their continuation thresholds coincide ($k^A = k^E$), and the incumbent acquirer's profit is unaffected by the entrepreneur's development success. Therefore, the two players value the project exactly the same and the incumbent acquirer is always indifferent between acquiring or not acquiring the project. This establishes the proposition. □

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

$$\rho\pi^E_{\neg acq,S} - k + n[\rho\pi^A_{\neg acq,S} + (1-\rho)\pi^A_{\neg acq,F}] + \rho CS_{\neg acq,S} + (1-\rho)CS_{\neg acq,F} \geq P + (\pi^A_{acq,F} + L - P) + CS_{acq,F} \quad (23)$$

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

$$(\rho\pi^E_{\neg acq,S} - k - L) + \rho(CS_{\neg acq,S} - CS_{\neg acq,F}) \geq n\rho(\pi^A_{\neg acq,F} - \pi^A_{\neg acq,S}). \quad (24)$$

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

$$CS_{\neg acq,S} - CS_{\neg acq,F} \geq n(\pi^A_{\neg acq,F} - \pi^A_{\neg acq,S}). \quad (25)$$

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

$$CS_{\neg acq,S} - CS_{\neg acq,F} = \frac{(n+1)[\beta + \gamma n]}{2} q^2_{n+1} - \frac{n[\beta + \gamma(n-1)]}{2} q^2_n \quad (26)$$

It is now straightforward to show that the sufficient condition for killer acquisitions to be welfare-reducing given by inequality (25) 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

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

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

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

Defining Δ^E and Δ^A with these new payoffs 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 teach 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 Genetech. 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 orginal 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 Trialtrove 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

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

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 Trialtrove 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.[35] 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).

[35]More details on the timeline of publicly available clinical trials database can be found at http://www.clinicaltrials.gov.

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 HBS patent databases to provide patent assignee information. After this step, each company in the drug development and acquisition database will have its original name, standardized name, and a stem name; 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 HBS 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 if identified, and the two companies are located in the same city and state OR the two companies are located in the same state and the earliest patenting year in NBER and HBS databases is later than the founding year in the drug data, we consider this as a "*successful match*." The company is removed from the set of names waiting to be matched on both sides.

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

   (c) Otherwise, next step.

3. For the remaining companies, each stem originator and owner name is matched with up to 3 close stem names from the USPTO data using a fuzzy-matching method based on the Levenshtein edit distance.[36] 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.

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