Killer Acquisitions

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This paper argues that 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 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 because of weak competition or distant patent expiration. Conservative estimates indicate that 5.3%–7.4% of acquisitions in our sample are killer acquisitions. These acquisitions disproportionately occur just below thresholds for antitrust scrutiny.

I. 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

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that 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 the 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, began development for the US market. Questcor acquired the US development rights for Synacthen in 2013. Following the logic of killer acquisitions (i.e., shutting down competition even before there is a marketable product), Questcor did not develop Synacthen. As the Federal Trade Commission (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]

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[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 another example of an acquisition aimed at preemptively eliminating innovative competition. At the time, Thoratec had a monopoly in the US market for left ventricular assist devices, a life-sustaining technology for end-stage heart failure patients, and Heartware ran clinical trials for its own potentially competing device (the "HVAD") but had yet to receive Food and Drug Administration (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. left ventricular assist device ('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

This paper theoretically and empirically studies killer acquisitions. To motivate the empirical analysis, we first build a parsimonious model that combines endogenous acquisition decisions, innovation choices, and product market competition. Our model formalizes the seemingly counterintuitive phenomenon of incumbents acquiring innovative potential entrants to shut down the entrants' innovative endeavors. It also highlights the conditions under which killer acquisitions are particularly prevalent.

We model acquisitions that occur when the innovative 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., substitutes for) a product or project in the incumbent's portfolio. This is a general, well-known result, "the monopolist's disincentive created by his preinvention monopoly profits" (Arrow 1962, 622). We show that this disincentive to innovate can be so strong that an incumbent firm may acquire an innovative start-up simply to shut down the start-up's projects and thereby stem the "gale of creative destruction" of new inventions (Schumpeter 1942). Importantly, some degree of acquirer-target overlap is necessary for the killer-acquisition motive to exist. However, both existing and future competition reduce the difference in project development decisions between acquirers and entrepreneurs and thereby diminish the incentive for killer acquisitions. Finally, we show that killer acquisitions continue to exist even when the entrepreneur's new project is qualitatively superior to the incumbents' existing projects or products, when incumbents benefit from development synergies relative to entrepreneurs, and when there are multiple (asymmetric) potential acquirers.

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 portfolio and the target's project and to quantify competition in the relevant product market.

Pharmaceutical drug development offers features to resolve all of these challenges. 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

Complaint, Docket No. 9339; https://www.ftc.gov/sites/default/files/documents/cases/2009/07/090730thorateadminccmpt.pdf).

[3] Research and development (R&D) intensity in the pharmaceutical industry is second only to that for semiconductors in the US manufacturing sector, at 11.3% in 2014 (NCSES 2018).

than 16,000 drug projects originated by more than 4,000 companies in the past two-and-a-half decades and follow each drug from initiation. We collect relevant acquisition events from comprehensive data sources. Importantly, we observe development milestones of drug projects independent of project ownership, meaning that we can follow the same projects before and after acquisition.[4]

To finely categorize acquirer overlap with the target's project, and thus identify potentially competing products, we use pharmaceutical categories based on disease and mechanism. 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) as a drug product or project in the acquirer's portfolio, we consider that acquisition to be an overlapping acquisition. Measuring overlap this way helps to ensure that we are capturing potential substitutes (i.e., companies developing drugs that, if successful, would directly compete with the acquirer's).

Our main empirical analyses focus on the development of drug projects. We compare projects acquired by overlapping incumbents to those acquired by nonoverlapping incumbents and to nonacquired projects. The baseline regression uses a project-year panel to estimate the annual probability of development activity (i.e., lack of project termination). Following the logic of killer acquisitions, we expect a decreased likelihood of the development of overlapping projects after acquisition. Correspondingly, we find that projects acquired by an incumbent with an overlapping drug are 23.4% less likely to have continued development activity, compared to drugs acquired by nonoverlapping incumbents. Reassuringly, the development patterns for overlapping acquired drugs are statistically indistinguishable from those for nonoverlapping acquired drugs and nonacquired drugs in the years before acquisition.

This finding is robust to controlling for a variety of economic forces. We control for project vintage and age and subsequently for drug development life cycles, using therapeutic class–mechanism of action–age fixed effects, which effectively help us compare drugs in the same stage along the same development trajectory. We also include project fixed effects to account for any unobservable time-invariant project characteristics. In addition, to control for selection into acquisition based on observable, time-varying characteristics, we implement a propensity score reweighting estimator.

We use several alternative specifications, subsamples, and analyses to confirm the robustness of our baseline results. Most importantly, we find

[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 after 2006, regardless of its change in ownership.

that decreased development after acquisition for overlapping acquired projects is driven by drugs that have no further development activity after acquisition (i.e., by immediate and permanent terminations). Projects acquired by overlapping acquirers are 20.9% more likely to cease development immediately, compared to those acquired by nonoverlapping incumbents. Further, we find no evidence that acquiring firms purposefully delay development or are simply slower at developing overlapping projects. Additionally, supplementary analysis of clinical trial phase progression confirms that overlapping acquired projects are less likely to move to the next phase.

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). We find that the decrease 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.

Despite the difficulties associated with testing for strategic motives, our empirical results suggest that killer acquisitions are both strategic and intentional. First, as our model predicts, we find that acquisitions are more likely when the incumbent acquirer's products overlap with the target project by almost four times.[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 premerger review and thereby avoid antitrust scrutiny. Acquisitions of overlapping targets bunch just below the FTC acquisition transaction value threshold, while there is no such pattern for nonoverlapping acquisitions.

[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 rather because they allow the acquirer to change the target's behavior (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 nonmonotonicity of investment with respect to market size, whereas in ours, it is identified by the lack of development of overlapping acquired projects.

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 development rates of overlapping acquired drugs. One alternative explanation is optimal project selection. Specifically, for multiproject targets, the acquirer could strategically and optimally choose to continue only the most promising projects while discontinuing those that are less promising. However, our results hold in the subsample of acquisitions of single-drug companies, implying that optimal project selection does not explain our results.

Another alternative explanation is capital redeployment, in which the acquiring firm's intention is to acquire and redeploy the acquired target's core assets—that is, its underlying technology or human capital—to more productive uses. If this were the case, our results on decreased development of overlapping acquired projects could be explained simply as a by-product. To address this, we separately consider technology and human capital redeployment. To explore technology redeployment, we track the chemical similarity of acquired drugs to pre- and postacquisition 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 after acquisition. These results are inconsistent with explanations regarding technology or human capital redeployment.

A related alternative explanation is "salvage" acquisitions, in which overlapping acquirers buy already-failing targets to (cheaply) acquire the target's valuable assets. Following this logic, decreases in development would predate the relevant acquisition, which would also have lower postacquisition development activity. Contrary to the salvage explanation, however, we find no evidence that overlapping acquisitions have either preacquisition declines in development or lower valuations, on average, compared to nonoverlapping acquisitions.

Our conservative estimates indicate that between 5.3% and 7.4% of all acquisitions in our sample (or about 46–63 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 more than 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 increase ex ante

incentives for the creation of new drug projects but also distort the direction of innovation.[6]

Our goal is to uncover an unobservable strategic motive, killer acquisitions, from observable outcomes. To do so, we empirically compare development probabilities of overlapping acquisitions, which are, in our theory, motivated by a mix of killer and development intentions, and nonoverlapping acquisitions, which are motivated only by development intentions. We find an increase in acquisition probability and a decrease in postacquisition development for overlapping acquisitions and interpret that as evidence for killer acquisitions. Importantly, killer acquisitions necessarily involve a combination of the choice to acquire an overlapping target and the resulting reduced incentives to develop drug projects that cannibalize the acquirer's existing profits. These combined effects are quite different from the effects arising from random allocation of new drug projects to incumbent firms with or without product market overlap, and thus we do not analyze quasi-random acquisitions and outcomes. Instead, we combine empirical evidence consistent with our theory and various analyses to rule out plausible alternative explanations.

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, Shleifer, and Vishny 1990), synergies (Maksimovic and Phillips 2001; Bena and Li 2014), 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,

[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). Bryan and Hovenkamp (2020a, 2020b) specifically analyze the role of antitrust limits on start-up acquisitions by dominant firms.

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), reducing the incentives of an incumbent to introduce new products that are substitutes for existing products,[7] and the "efficiency effect" (Gilbert and Newbery 1982), giving an incumbent strong incentives to acquire the property rights to a new innovation to preempt entry.[8]

Second, we focus on the implications of acquisitions and increasing concentration on innovation. Cabral (2017), Federico, Langus, and Valletti (2017, 2018), Motta and Tarantino (2017), and Gilbert (2018) present theoretical models in which merging parties have diminished innovation incentives and acquisitions can be used to cement the dominance of incumbents. Ornaghi (2009) and Haucap, Rasch, and Stiebale (2019) empirically document an innovation-reducing effect of mergers, whereas Guadalupe, Kuzmina, and Thomas (2012) find that a target company's innovation increases after an acquisition by a multinational firm. Our paper provides a theoretical and empirical analysis of a new channel through which acquisitions affect innovation. By using detailed project-level data on acquisition and development decisions, we can 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 trade-offs involved in promoting competition while fostering innovation, through investigating the product market interactions between patented and generic drugs (Caves, Whinston, and Hurwitz 1991; Grabowski and Vernon 1992; Scott Morton 2000; Reiffen and Ward 2005), the role of pricing (Howard et al. 2015) and price controls (Filson 2012), internal R&D policies (Cockburn and Henderson 1994), and mergers and acquisitions (M&As; Ornaghi 2009; Haucap, Rasch, and Stiebale 2019; Meder 2019). 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 and that incumbents may abuse this mechanism to impede innovative competition.

[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 Holmes, Levine, and Schmitz (2012) and Schmutzler (2013) for unified theoretical treatments and Cohen (2010) for a comprehensive survey.

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

II. 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 (apps. A–F are available online).

A. *Setup*

The model has the following time line, depicted in figure 1. In $t = 0$, an entrepreneur E (she) with a single project is born.[9] There are also $n \geq 1$ incumbent firms, each possessing an existing differentiated 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 use "¬acq" otherwise.

In $t = 1$, the project's owner—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.[11] Let ρ^{A} and ρ^{E} be the probabilities that the project will ultimately be successful if the acquirer or the entrepreneur develops it, k be the cost of developing the project, and L be the project's liquidation value 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 and very costly, and the eventual success is uncertain. We allow for two benefits of innovation: vertical and horizontal differentiation. If the new project is successfully developed, it expands the size of the market both because it is superior in terms of objective quality and because it meets the needs of some consumers more effectively.[12] We also allow for differential capabilities in project development. If $\rho^{A} > \rho^{E}$,

[9] We assume that the entrepreneur does not have to exert any effort to generate the idea for this project. For a detailed discussion of this assumption, see sec. V.C.

[10] In sec. II.D.2, we relax this assumption and allow for multiple incumbent acquirers. However, we do not allow for mergers between incumbents, as in Gowrisankaran (1999), and we abstract away from contracting difficulties in the sale of ideas, as in Anton and Yao (2002).

[11] We assume that the new project is the only source of innovation. We therefore do not consider the impact of acquisitions on innovation by the acquirer's existing competitors. See Haucap, Rasch, and Stiebale (2019) for a model along those lines.

[12] Although it is realistic in our empirical setting to assume that the new project is objectively superior to existing products, all of the results of our model remain unchanged if vertical differentiation is absent.

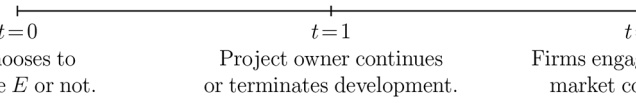

FIG. 1.—Model time line.

the acquirer has an advantage in developing the project relative to the entrepreneur.[13]

Finally, in $t = 2$, uncertainty about the project's success is resolved, and all firms engage in product market competition with imperfect substitutes. We model competition using horizontally and vertically differentiated Bertrand competition because price-setting behavior by firms with differentiated products best captures strategic interactions in the branded drug market (Ellison et al. 1997; Duggan and Scott Morton 2010; Berndt, McGuire, and Newhouse 2011).[14] We assume that if the project is successfully developed in $t = 2$, the drug has a product market payoff that depends on the degree of competition (i.e., the number of active firms/products in the market) and product differentiation (i.e., quality and taste differences) in the market. If the project is unsuccessful, the payoff is zero. We assume that the values of ρ^{A}, ρ^{E}, k, and L and the extent of vertical and horizontal differentiation are commonly known by all of the involved parties.

B. *Product Market Competition ($t = 2$)*

Consider first the product market choices of the entrepreneur when her project is not acquired (¬acq). If the project is successful (S), the resulting newly developed product competes against n other single-product incumbent firms, and the entrepreneur maximizes $p^{E}q^{E}$. This yields profits for the entrepreneur and the potential acquirer (and all of the other incumbents) such that $\pi^{E}_{\neg acq,S} \geq \pi^{A}_{\neg acq,S} > 0$ because the new product is (weakly) superior to all existing products and all products are horizontally differentiated from each other.

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 (horizontally differentiated) product to sell, and thus the acquirer's profit is equal to $\pi^{A}_{\neg acq,F} > 0$. Profits for the acquirer are now higher, $\pi^{A}_{\neg acq,F} > \pi^{A}_{\neg acq,S}$, for two reasons. First,

[13] For example, this could be due to synergies with the expertise from developing his existing product (Teece 1986; Gans and Stern 2000).

[14] Our results are not sensitive to this particular form of competition. They also hold for (differentiated) Cournot competition, as we show in app. A.2.

the acquirer has to compete against only $n - 1$ (rather than n) single-product firms. Second, none of these $n - 1$ firms sell a superior new product.

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 $t = 2$, and he competes against the other $n - 1$ single-product incumbents. The resulting profit for the acquirer is $\pi^{A}_{acq,F} > 0$. This is exactly 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, the acquirer 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, $p^{A}_{old} q^{A}_{old} + p^{A}_{new} q^{A}_{new}$, whereas the remaining $n - 1$ other single-product incumbent firms maximize single-product profits. The profit of the multi-product incumbent acquirer is $\pi^{A}_{acq,S}$. This profit is higher than when he sells only a single product with the same $n - 1$ competitors; hence, $\pi^{A}_{acq,S} > \pi^{A}_{\neg acq,F}$.

To summarize, we obtain the following profit rankings for the acquirer and the entrepreneur:

$$\begin{aligned} &\pi^{A}_{acq,S} > \pi^{A}_{acq,F} = \pi^{A}_{\neg acq,F} > \pi^{A}_{\neg acq,S} > 0, \quad \text{and} \\ &\pi^{E}_{\neg acq,S} > \pi^{E}_{\neg acq,F} = 0. \end{aligned} \tag{1}$$

C. *Development Decision ($t = 1$)*

1. Product Market Overlap

We now investigate the development decision in $t = 1$, 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. As long as the acquirer's existing product and the new project are imperfect substitutes, we have

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

The acquirer gains strictly less from developing a new product than an entrepreneur would. This is due to 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.[15]

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

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

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

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

Comparing these thresholds shows two reasons for the difference in product development decisions of the entrepreneur and the acquirer. First, because of the replacement effect ($\Delta^{E} > \Delta^{A}$), the entrepreneur is more willing to develop the product. Any form of product market overlap (i.e., substitutability) with the existing drug in the acquirer's portfolio reduces his propensity to continue the development of the acquired project relative to the case in which the project remains independent. Second, the acquirer is more willing to continue if he benefits from important synergies in product development ($\rho^{A} > \rho^{E}$). Depending on the relative magnitude of these two effects, either the entrepreneur or the acquirer has a stronger incentive to develop the product.

Proposition 1 (Project development and market overlap). An incumbent firm that acquires a project continues development if $k \leq k^{A}$, while an entrepreneur continues if $k \leq k^{E}$. For any product market overlap, we have $k^{E} > k^{A}$ if and only if $\Delta^{E}/\Delta^{A} > \rho^{A}/\rho^{E}$.

Thus, whether the entrepreneur or the acquirer has stronger incentives to continue development depends on the relative magnitudes of the replacement effect Δ^{E}/Δ^{A} and the synergy effect ρ^{A}/ρ^{E}. If the acquirer does not have a development advantage or if this advantage is not large enough to outweigh the replacement effect, the entrepreneur will always be more willing to continue development. 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}, also highlighting 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 (and absence of large synergy effects), is what

[15] 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.

generates lower development incentives of the incumbent acquirer relative to the entrepreneur.

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 product market overlap, the difference $k^E - k^A$ is strictly decreasing in n.

Successfully developing a new product draws consumer demand and profits away 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 from only 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.

3. Patent Life and Future Competition

Until now, we have considered only the impact of competition from branded drugs (i.e., imperfect substitutes). 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 T^E and those of the acquiring incumbent's existing product T^A, where $T^E > T^A \geq 0$. We assume, for simplicity, that the firms earn their static game profits every year and use the same discount factor $\delta \leq 1$.

We also assume that as soon as a product's patent expires, an identical, undifferentiated product (e.g., a generic drug) enters the market (Berndt, McGuire, and Newhouse 2011). 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 and the entrepreneur earn the per-period development gains Δ^A and Δ^E, respectively. Thereafter, the profits for the acquirer's existing product drop to zero because of undifferentiated generic competition, but the profits of the newly developed products remain positive. Therefore, the acquirer faces no more cannibalization losses from the development of the new product, and hence his incentives to develop coincide with those of the entrepreneur. Specifically,

the entrepreneur's and acquirer's per-period development gains after the expiration of the acquirer's existing product's patent in T^A years are $\Delta_{gen} = \Delta^E_{gen} = \Delta^A_{gen}$.[16]

PROPOSITION 3 (Project development and patent life). For any product market overlap, the difference $k^E - k^A$ is strictly increasing in T^A if the acquirer's development synergies are not too large.

The longer the patent life T^A of the acquirer's existing product, the weaker his incentives are to continue development relative to those of the entrepreneur if his development synergies are not too large relative to the replacement effect (i.e., $\rho^A/\rho^E \leq (\Delta^E - \Delta_{gen})/(\Delta^A - \Delta_{gen})$). When the acquirer's existing overlapping product has only little remaining patent life (T^A close to zero), 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 for proposition 2. Generic entry is just a particularly intense form of competition that destroys all of the profits of the acquirer's existing product and eliminates cannibalization losses from new product development.[17]

Proposition 3 remains unchanged if we assume that generic entry does not drive the profits of the old existing product to zero.[18] As long as generic entry destroys some of the existing profits of the incumbent's old drug, the replacement effect for the incumbent will be smaller after patent expiration, and thus the difference $k^E - k^A$ will be increasing in T^A.

D. Acquisition Decision ($t = 0$)

1. Single Incumbent Acquirer

We now show that killer acquisitions can occur only 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

[16] 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 but is differentiated from all other products, including the new product) enters, it not only drives profits of that product to zero but, because of its low price, also reduces the profits of the other products that are differentiated from it.

[17] Unlike in Conner (1988), in our model with generic competition the acquirer will never find it profitable to delay the introduction of the new product, because delayed introduction reduces only the time during which the new product can earn positive profits arising from patent protection.

[18] This will be the case if the cross-price elasticity of demand between the branded drug and its generic substitutes is high but not infinite, because consumers may still prefer the branded drug to its generic competitors (Ellison et al. 1997).

remains independent.[19] Recall that for any product market overlap, we have $k^E > k^A$ if and only if $\Delta^E/\Delta^A > \rho^A/\rho^E$. If this condition holds, there are three cases to consider.

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

Second, for $k^E \geq k > k^A$, the acquirer terminates the project, but the entrepreneur continues development. Such an acquisition ("acquire to kill") occurs if

$$\underbrace{\rho^E(\pi^A_{acq,F} - \pi^A_{\neg acq,S})}_{\text{efficiency effect}} \geq \underbrace{\rho^E\Delta^E - k - L}_{\text{replacement effect}}. \tag{5}$$

Acquiring and shutting down the entrepreneur's project yields a profit equal to $\pi^A_{acq,F}$, while not acquiring it yields $\pi^A_{\neg acq,S}$. The difference between these (multiplied by the probability ρ^E with which the entrepreneur develops the project) is the efficiency effect. However, the expected marginal profit for the entrepreneur from continuing development ($d^E = 1$) given by $\rho^E\Delta^E - k$ 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 nonacquired firms develop the project. Such an acquisition ("acquire to continue") occurs if

$$\underbrace{\rho^E(\pi^A_{acq,F} - \pi^A_{\neg acq,S})}_{\text{efficiency effect}} \geq \underbrace{\rho^E\Delta^E - \rho^A\Delta^A}_{\text{replacement effect}}. \tag{6}$$

Here, the replacement effect is the difference in expected marginal project development gains because both parties develop the project.[20] Despite developing the project, the acquirer still benefits from reducing competition through (less aggressive) multiproduct pricing.

Thus, when incumbent synergies are not too large, acquisitions take place if $k \leq k^E$ and if the efficiency effect is sufficiently large relative to

[19] 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. The price is also the same 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 she would if acquisitions were impossible. As we discuss in sec. V, in a more general model, the existence of the acquisition exit option may be valuable enough to increase ex ante innovation incentives.

[20] Under symmetric (differentiated) Bertrand competition with equal development probabilities, 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.

the replacement effect. Even though the entrepreneur has a higher propensity for developing a project (because the replacement effect is stronger than the incumbent synergies), acquisitions occur because they prevent the entrepreneur from reducing the existing profits of the acquirer (efficiency effect).[21]

Finally, in the case where $\Delta^E/\Delta^A \leq \rho^A/\rho^E$, there are only two cases to consider, because $k^E \leq k^A$. If $k > k^A$, 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. If $k \leq k^A$, the acquirer always acquires and develops the project, because the incumbent's development synergies outweigh the replacement effect.

PROPOSITION 4 (Acquisition). The acquirer may have strictly positive incentives to acquire the entrepreneur if there is product market overlap or if the acquirer's development synergies are sufficiently large, $\Delta^E/\Delta^A < \rho^A/\rho^E$. Otherwise, the acquirer never has strictly positive incentives to acquire the entrepreneur.

Proposition 4 highlights that either product market overlap or significant development synergies are required for (killer or continuing) acquisitions to occur, immediately implying 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.

Figure 2 illustrates the two forces driving acquisitions. It plots the acquirer's optimal acquisition strategies as a function of his development capability (ρ^A) and the degree of product overlap (γ). First, when both ρ^A and γ are low, the incumbent chooses not to acquire. If he acquires and continues development, he is unlikely to succeed, and acquiring to kill is not worthwhile because his profits are not hurt very much by the successful development of the entrepreneur's project. Second, when ρ^A is high and γ is low, the acquirer chooses to continue because developing the project is likely to be successful and does not cannibalize his existing profits very much. Third, when ρ^A is low and γ is high, a killer acquisition is optimal because continuing development is likely unsuccessful, but not acquiring the entrepreneur leads to a significant destruction of the acquirer's existing profits. Fourth, when both ρ^A and γ are high, either a killer acquisition or acquiring to continue is optimal. Fifth, when γ is very high, the acquirer does not need to acquire the entrepreneur. Even the entrepreneur does not develop the project, because competition is too intense.

[21] Although the acquirer has a strictly positive incentive to acquire the entrepreneur only 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 ends up developing only a subset of his acquired projects ($k \leq k^A$).

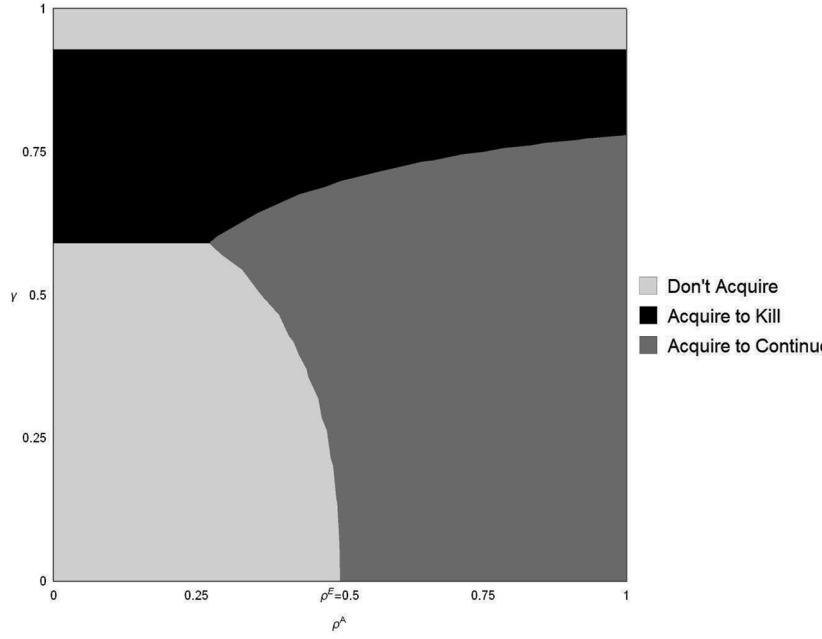

FIG. 2.—Optimal acquisition strategies. This graph plots the optimal acquisition decisions—"Don't Acquire" (light gray), "Acquire to Kill" (black), and "Acquire to Continue" (dark gray)—as functions of the acquirer's development capability ρ^A and the degree of substitutability γ. The other parameter values are $\alpha^A = \alpha^E = 100$, $\rho^E = 0.5$, $L = 20$, $k = 80$, and $n = 2$.

Figure 2 also illustrates the inefficient project ownership arrangements that can occur in our model that arise from the incentives to protect existing profits. First, killer acquisitions can materialize even when the incumbent benefits from strong development advantages ($\rho^A > \rho^E$). Second, acquiring to continue can occur even when the acquirer is much worse at developing the project than the entrepreneur ($\rho^A < \rho^E$).[22]

2. Multiple Incumbent Acquirers

Our analysis so far has made two simplifying assumptions about the acquisition process. First, we assumed that only one of the incumbents is

[22] Figure 2 shows that under differentiated Bertrand competition, as long as $\rho^A \geq \rho^E$, the acquirer strictly prefers to acquire the entrepreneur if the entrepreneur would otherwise continue development ($k^E \geq k$), and hence the inequalities (5) and (6) are always satisfied. However, these inequalities are not always satisfied under Cournot competition, even for $\rho^A \geq \rho^E$, because the acquirer may sometimes find it too costly to acquire the entrepreneur, especially when there are many existing incumbents and the products are not very differentiated. This nonacquisition result is closely related to the "Cournot merger paradox" (Salant, Switzer, and Reynolds 1983).

chosen at random to be the potential acquirer of the entrepreneur's project. Second, we assumed that all of the incumbents' existing products are equally differentiated from the entrepreneur's project and that all have the same project development capabilities. In appendix A.3, we relax these assumptions. Here we sketch out the main implications of allowing for multiple (asymmetric) incumbent acquirers.

First, when several of the n incumbents[23] can acquire the entrepreneur, this leads to a situation akin to the "volunteer's dilemma" (Diekmann 1985) or, more broadly, the costly private provision of a public good (Bliss and Nalebuff 1984), because all of the incumbents benefit from the acquirer eliminating a future competitor. We show that all pure-strategy equilibria of this acquisition game are essentially identical to the single-acquirer case. Moreover, there exists a symmetric mixed-strategy equilibrium in which the entrepreneur is acquired with only some probability because the potential acquirers try to free ride on the privately costly decision to acquire the entrepreneur. Just as in the volunteer's dilemma, when the number of potential bidders increases, this free-riding incentive increases. However, in our setting it is counteracted by a second effect. Because any potential acquirer who bids is less likely to be (randomly) chosen as the "winning bidder," the expected cost of bidding decreases. Although the combination of these two effects on each potential acquirer's individual probability to bid is ambiguous, the overall probability of acquisition by at least one of the acquirers unambiguously increases as the number of potential incumbent acquirers increases.

Second, potential acquiring incumbents can differ in overlap and development ability. We show that when potential incumbent acquirers differ in their degree of product differentiation from the entrepreneur's project, a killer acquirer or continuing acquirer will always be the incumbent with the product that is the least differentiated from the entrepreneur's project. This is because the efficiency effect is largest for that acquirer, both in absolute terms and relative to the replacement effect. However, when incumbent acquirers also differ in terms of development capabilities, a continuing acquirer with a more differentiated product, but with a higher development capability, will acquire the project. This happens when his development success probability, and hence his expected gain from developing the entrepreneur's project, is sufficiently high to raise his valuation of the entrepreneurial firm above that of the acquirer with the least differentiated product.

[23] Outside bidders are irrelevant in our model because they always have exactly the same valuation for the entrepreneur's project as the entrepreneur herself. This is in contrast to Gans (2005), who shows that in a contracting setup à la Grossman and Hart (1986) and Hart and Moore (1990) with an acquisition stage, disinterested outside parties may end up owning firms.

To summarize, our theory shows that killer acquisitions arise from the combination of the choice to acquire a particular firm with the intention to terminate and the reduced incentive to develop acquired projects that cannibalize the acquirer's existing profits. As long as the acquirer's development synergies are not too large, our theoretical framework predicts that (1) after an acquisition, overlapping drug projects should be less likely to be developed; (2, 3) when existing (2) or future (3) competition is low, this difference in development choices between overlapping acquired drugs and their nonoverlapping acquired or nonacquired counterparts should be more pronounced; and (4) acquisitions by incumbents should target entrepreneurial firms developing drug projects that overlap with the incumbent's existing drugs.

III. 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 nonacquired 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 health care spending, innovative activity, and M&A transactions. It is an economically and socially important industry of ongoing interest to economists (see Lakdawalla 2018 for a summary).

A. 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 before any clinical trials in humans. After these preclinical evaluations, drugs undergo three phases of clinical trials (phases I, II, and III).[24] In tandem with these regimented clinical tests, firms engage in additional commercialization activities, including patent filing during the preclinical and/or discovery stage, regulatory filings in the United States and abroad, applications for coverage to various

[24] Drug developers must submit an Investigational New Drug application to the FDA before 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.

public and private insurance agencies, and launching and marketing the product in various countries around the world. Given the lengthy process before FDA approval and marketing, patented drugs usually have only a few years after approval of monopoly profits before patent expiration and generic entry.

Each component of drug development represents significant expenditure and time.[25] Because development is regulated and standardized and reaching development milestones is typically very costly, we can interpret observed development events and activities as credible evidence of purposeful and significant project-level development (or lack of project termination). Further, we observe this project-level development, or lack thereof, regardless of ownership, which is crucial to identifying killer acquisitions.

B. Drug Development Data

To build our data set at the drug project level, we use Pharmaprojects from Pharma Intelligence, which has been used in earlier research studying drug development (e.g., Adams and Brantner 2006; Kyle 2007; Blume-Kohout and Sood 2013; Branstetter, Chatterjee, and Higgins 2014). Pharmaprojects is a comprehensive data set 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 US market, along with the originating firm associated with each drug project.[26]

Importantly for our purposes, Pharmaprojects documents the occurrence and timing of development milestones and ongoing development activities (e.g., "new patent application," "target identified," "additional registration for clinical trial," and "development ongoing"), including both research (i.e., science) milestones and important steps in the commercialization process. The data set therefore allows us to observe a broad set of activities that indicate the development of a drug, including, but not limited to, progress through clinical trials. We use the occurrence of a development event or any development activity (i.e., a lack

[25] Dubois et al. (2015) estimate that a new drug incurs approximately $800 million–$1 billion in development costs, with average expenditure on drugs in human clinical trials of around $27 million per year (Adams and Brantner 2010).

[26] In the raw data set, Pharmaprojects typically updates the "originator" firm name associated with each project when and if it is acquired. We therefore reconstructed the historical originator firm using text descriptions included in other fields in the data set. We provide more details in app. B.

of project killing) in a given year as our core dependent variable.[27] Also crucial for our analyses, Pharmaprojects 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 projects and products as well as competition.

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.[28] Pharmaprojects data start from 1989, and we exclude projects initiated in 2011 or after to ensure that we observe project development activities 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). More than half of the companies originate only one drug over this period, and 70% originate two or fewer (see fig. F1; figs. A1, F1, and F2 are available online), which aligns with common perceptions of drug development: small firms initiate innovative drug projects, some of which are subsequently developed by large, commercialization-focused incumbent firms (Cockburn 2004).

C. Acquisition Data

We collect acquisition data from Thomson Reuters SDC Platinum, Thomson Reuters RecapIQ (now Cortellis Deals Intelligence), and the SDC VentureXpert database. We then conduct a multistep cleaning process to ensure that acquisition events are correctly linked to target and acquirer firms. First, we 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 under 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 (largely due to using multiple data sets).

[27] Full details are in app. B.

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

TABLE 1
DESCRIPTION OF DRUG DEVELOPMENT PROJECT ACQUISITIONS

	N	Nonacquired (%)	Nonoverlapping Acquired (%)	Overlapping Acquired (%)
Whole sample	16,015	78	17	5
By time period:				
Beginning–1995	2,684	60	31	9
1996–2000	2,854	68	25	7
2001–5	4,716	79	16	4
2006–10	5,761	90	8	2
By high-level disease group (top 5):				
Anticancer (13 TCs; 783 TC-MOAs)	2,579	80	16	4
Neurological (27 TCs; 986 TC-MOAs)	2,573	77	19	4
Anti-infectives (28 TCs; 452 TC-MOAs)	1,946	77	16	7
Biotechnology (26 TCs; 209 TC-MOAs)	1,493	79	16	5
Alimentary/metabolism (24 TCs; 498 TC-MOAs)	1,380	81	15	4

NOTE.—This table provides descriptive statistics on drug projects. The table describes the number of drugs originated over time and by consolidated disease groups as well as the proportions of projects that are nonacquired, acquired by nonoverlapping acquirers, and acquired by overlapping acquirers (i.e., acquired by an incumbent with a project in the same therapeutic class [TC] and mechanism of action [MOA] as the focal project). For illustrative purposes, we present the top five broad disease groups by number of projects (out of 16 total groups). Disease groups are high-level categorizations, and each disease group includes a number of TCs and a large number of TC-MOA pairs. These narrower TC-MOA 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.

We then combine our 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 can identify whether it went through any acquisition event during its development life cycle and, if it did, the acquirer, the timing of acquisition, and development activity in the years before and after acquisition. The merged drug development and acquisition data show active acquisition activities in our analytical sample, with 22% of drug projects having an acquisition recorded in our acquisition database. 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).

IV. Empirical Analysis

A. *Empirical Design*

The first main implication of the theoretical framework (building from proposition 1) is that if the target project overlaps with acquirer projects or products, the acquirer has weaker incentives to continue development. We therefore need a measure of overlap between the target's projects and the acquirer to test for differences in the likelihood of development across overlapping acquired, nonoverlapping acquired, and nonacquired projects.

We measure overlap between a drug project and the acquiring firm on the basis of a combination of its intended therapeutic class and mechanism of action. The therapeutic class (TC) is the disease or condition the therapy targets (e.g., hypertension). We use Pharmaprojects's therapeutic categories, which are based on the European Pharmaceutical Market Research Association product categorizations (Kyle 2007). These categories represent 230 possible TCs. Within each TC, we also identify the drug's mechanism of action (MOA), 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 MOAs per TC in our sample is seven. In our main analyses, we categorize a project as overlapping if the acquiring firm has an existing project or product in the same TC that uses the same MOA as the acquired drug project (i.e., treats the same disease or condition in the same way). As outlined in table 1, nearly one-quarter of acquired drug projects overlap with their acquirer's projects. We measure competition using this same categorization (i.e., the number of products in the same TC using the same MOA). The mean number of products in a TC-MOA pair in a given year is 13.

The logic for measuring overlap narrowly is to ensure that we capture only potential substitute drugs. If we were to instead use same TC regardless of MOA, we would be more likely to capture drugs that complement the target's project, either because they treat different submarkets (i.e., different patient segments with the same disease) or because they are used in parallel in treatment for the same patients. We investigate separately the effects of overlap measured more broadly as the same TC, which we report in supplementary analyses.[29]

Because we are studying innovative drug development before commercialization, our measure of overlap necessarily differs from measures of competition used in the literature on generic or branded drugs. First, the vast literature that explores generic competition and the effects of generic

[29] In our analytical sample, of acquired projects, 23% overlap in TC and MOA, 27% overlap in TC only, and 50% do not overlap with the acquirer.

entry on branded products defines competing products as those that are the same chemical entity (Ellison and Ellison 2011; Arcidiacono et al. 2013; Branstetter, Chatterjee, and Higgins 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 having 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 (including, e.g., estimated substitution elasticities). Given that we are analyzing premarket products, a big advantage of the pharmaceutical industry context is that categorizations of intended markets and mechanisms are readily available from a project's early stage. Finally, some prior research has used the broader measure of the same TC (e.g., Kyle 2007); we use a narrower measure for the reasons discussed above but include analyses with the broader measure.

For our main empirical analyses, we use panel data of drug projects. A project is included in the sample from the origination year and is removed from the sample after a successful US launch, if any. The empirical specification is as follows:

$$\begin{aligned}\text{Development}_{i,t} = {} & \beta \cdot I(\text{Acquired})_i \times I(\text{Post})_{i,t} \times I(\text{Overlap})_i \\ & + \gamma_1 \cdot I(\text{Acquired})_i \times I(\text{Post})_{i,t} + \gamma_2 \cdot I(\text{Acquired})_i \\ & \times I(\text{Overlap})_i + \gamma_3 \cdot I(\text{Acquired})_i + \alpha_{\text{FE}} + \varepsilon_{i,t},\end{aligned} \tag{7}$$

where the dependent variable $\text{Development}_{i,t}$ is a dummy variable indicating whether drug i has a development event in year t, $I(\text{Acquired})_i$ indicates whether drug i ever undergoes an acquisition event, $I(\text{Post})_{i,t}$ indicates whether the drug-year (i, t) observation is after the drug is acquired, and $I(\text{Overlap})_i$ indicates whether drug i overlaps with any existing product or project of the acquirer firm. We control for the potential confounding effects, using a vast array of fixed effects (α_{FE}; described below), and standard errors are clustered at the drug project level. We report our results estimated using linear probability models, but the results are similar when we use logit models.

In this panel specification, the interaction term $I(\text{Acquired})_i \times I(\text{Post})_{i,t}$ captures the change in development activity for all acquired drug projects in the years after the acquisition. The term $I(\text{Acquired})_i \times I(\text{Overlap})_i$ 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 $I(\text{Acquired})_i \times I(\text{Post})_{i,t} \times I(\text{Overlap})_i$, 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.[30]

Ideally, if terminations were comprehensively reported in a timely manner, we would use a survival analysis to test whether and when drug projects are shut down. However, project terminations are rarely observed or voluntarily reported, either at a specific point in time or at all.[31] Hence, in our main specification, we use a lack of development activity as a proxy for termination. We test for the likelihood of observed, active development of a project, using a project-year panel. There are also several advantages to a panel structure that are not possible in a survival analysis, including the ability to account for time-invariant project-level differences between acquired and nonacquired projects and preacquisition differences between overlapping and nonoverlapping acquired projects. To investigate whether we are accurately capturing drug terminations with our project-year analysis, we run additional analyses predicting any postacquisition development activity (described in detail below).

The following subsections detail our empirical analyses. First, we compare drug development rates for nonacquired, acquired nonoverlapping, and acquired overlapping projects (table 2). We then deepen our analyses of proposition 1 by focusing on single-project targets and by separately analyzing projects that are "never developed" after an acquisition (table 3). To test propositions 2 and 3, we analyze the effects of competition (table 4) and acquirer patent life (table 5). Next, we examine how overlap determines acquisitions, following from proposition 4 (table 6). To further probe strategic intent, we document acquisition and development patterns around

[30] 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 nonmonotonic otherwise. Analogously, in our case, if the incentives to discontinue projects due to potential cannibalization of existing drugs are absent, ceteris paribus, overlapping acquired drug projects should have equal or even higher development rates than nonacquired or nonoverlapping acquired ones. This is because only drug candidates that are particularly valuable to an acquirer (e.g., because of synergies resulting from prior experience of developing similar drugs) should be acquired. Thus, in the absence of strategic killing motives, the triple interaction term $I(\text{Acquired})_i \times I(\text{Post})_{i,t} \times I(\text{Overlap})_i$ should be positive. In contrast, our theory of strategic acquisition and discontinuation of overlapping projects predicts that this interaction term should be negative.

[31] Only 14% of the projects in our sample are reported as terminated at some point (rather than inferred as discontinued on the basis of a lack of activity), contrasting with prior research that implies a drug discontinuation rate of more than 80% (DiMasi et al. 2010; Takebe, Imai, and Ono 2018).

antitrust review thresholds (table 7; fig. 3). Finally, we investigate several alternative explanations (tables 8, 9).

B. Development of Drug Projects after Acquisition

Table 2 presents the regression results from model (7) comparing nonacquired, acquired nonoverlapping, and acquired overlapping projects. We include various combinations of fixed effects to ensure that the variation in development across overlapping and nonoverlapping acquisitions is not driven by confounding economic, scientific, or firm effects (i.e., to narrow the comparison projects to those otherwise similar except for differences in acquisition status).

In column 1, we include project age and vintage fixed effects to focus our estimates on the development of drug projects that are initiated in the same year and at the same stage of development. Vintage fixed effects also account for right truncation for more recently initiated projects, given the long development time lines for pharmaceuticals (US FDA 2017). In column 1, the estimate of β is -0.037 and is statistically significant at the 1% level, meaning that acquired drug projects that overlap with the acquirers' portfolio are 3.7 percentage points less likely to have a development event in the years after acquisition than nonoverlapping acquired projects and that they are 5.7 percentage points ($0.037 + 0.020$) less likely to experience a development event than nonacquired projects. Given that the unconditional probability of having a development event is 19.9%, being acquired by a firm with an overlapping project is associated with a 20.7% ($0.037/(0.199 - 0.020)$) lower development probability than otherwise similar drugs that are acquired by a nonoverlapping acquirer.

In column 2, we include age-TC-MOA fixed effects to 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 in the size and geography of patient pools, physician capacity, or patient follow-up times, which can vary greatly across different drug markets (ASPE 2014). For example, Budish, Roin, and Williams (2015) argue that 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. Further, within TCs, certain MOAs may be "older" (i.e., more established), which also may lead to differential rates of development across TC-MOAs. These fixed effects ensure that we are comparing similar drugs at similar points in the life cycle.

In column 3, we add firm 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,

TABLE 2
OVERLAPPING ACQUISITIONS AND PROJECT DEVELOPMENT

	DEVELOPMENT EVENT = 1					
	(1)	(2)	(3)	(4)	(5)	(6)
I(Acquired) × I(Post) × Overlap	−.037***	−.033**	−.029*	−.041**	−.043**	−.054**
	(.013)	(.014)	(.015)	(.019)	(.021)	(.024)
I(Acquired) × I(Post)	−.020***	−.016**	−.017**	−.024**	−.018	−.018
	(.006)	(.007)	(.009)	(.010)	(.011)	(.013)
I(Acquired) × Overlap	.004	.009	.026**			
	(.008)	(.009)	(.011)			
I(Acquired)	−.002	−.004	−.011			
	(.004)	(.005)	(.012)			
Before(−3) × Overlap						−.031
						(.032)
Before(−2) × Overlap						.012
						(.032)
Before(−1) × Overlap						−.040
						(.030)
Before(−3)						.015
						(.017)
Before(−2)						.020
						(.017)
Before(−1)						−.003
						(.016)
Observations	143,569	143,569	143,569	143,569	134,662	143,569
R^2	.038	.252	.289	.366	.662	.370
Vintage FE	Y	Y	Y			
Age FE	Y					
Age × TC × MOA FE		Y	Y	Y	Y	Y
Originator (target company) FE			Y			
Project FE				Y	Y	Y
Propensity score reweighted					Y	

NOTE.—This table presents the likelihood of postacquisition development events for drug projects, using a drug-year panel sample. The empirical specification uses the following model: $\text{Development}_{i,t} = \beta \cdot I(\text{Acquired})_i \times I(\text{Post})_{i,t} \times I(\text{Overlap})_i + \gamma_1 \cdot I(\text{Acquired})_i \times I(\text{Post})_{i,t} + \gamma_2 \cdot I(\text{Acquired})_i \times I(\text{Overlap})_i + \gamma_3 \cdot I(\text{Acquired})_i + \alpha_{\text{FE}} + \varepsilon_{i,t}$, where the dependent variable $\text{Development}_{i,t}$ is a dummy variable indicating whether drug i has a development event in year t; FE = fixed effects. $I(\text{Acquired})_i$ indicates whether drug i is acquired during the study period, and $I(\text{Post})_{i,t}$ indicates whether the drug-year (i, t) observation is after the drug is acquired. $I(\text{Overlap})_i$ is a dummy variable indicating that 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.

* Significant at the 10% level.
** Significant at the 5% level.
*** Significant at the 1% level.

$I(\text{Acquired})_i \times I(\text{Post})_{i,t} \times I(\text{Overlap})_i$ estimates the difference in development between acquired overlapping and nonoverlapping projects for the same originator (or target). These results show that even within the same originator firm, acquired overlapping projects are significantly less

likely to have a postacquisition development event, compared to acquired nonoverlapping projects.

In column 4, we include drug-level fixed effects to absorb variation due to unobservable drug-specific characteristics, subsuming vintage fixed effects. We find that the estimate of β is statistically significant and of similar economic magnitude to that in column 1: being acquired by a firm with an overlapping project is associated with a 23.4% decrease in development rate.

In column 5, we apply propensity score reweighting to the column 4 analyses. Following Guadalupe, Kuzmina, and Thomas (2012), this allows us to further account for observable differences in the probability of being acquired. To calculate the propensity score for each firm, we run the following analysis. For each year, we consider firms acquired in that year as treated and firms that are never acquired as control observations. We pool treated and control observations across all years to estimate the probability (or propensity), p, that a firm is acquired as a function of lagged productivity (total number of development events in the past three years), firm size (the total number of projects under development), drug vintage, a year trend, and TC-MOA fixed effects. We then transform p into weights (weighting each treated firm by $1/p$ and each control firm by $1/(1 - p)$) and restrict the analysis to firms that fall within the common support.[32] The propensity score reweighting estimator allows us to control for selection stemming from both time-invariant characteristics of firms (as in the equal-weighted fixed effects regression) and time-varying characteristics.[33] Our core results are robust to the propensity score reweighting.

In column 6, we explore whether there are any differences in drug development trajectories across projects acquired by overlapping versus nonoverlapping incumbents. Given that we find decreased development after acquisition for overlapping acquired projects, one concern is that such projects were on a slower (or faster) development path before acquisition, compared to other acquired projects, and that such preacquisition differences at least partially explain postacquisition differences. To investigate this, we include indicator variables for the three years before the acquisition, separating overlapping and nonoverlapping acquired projects. The associated estimated coefficients are insignificant, suggesting that different development trajectories are not driving our results.

[32] Figure F2 shows similar propensity distributions for acquired and nonacquired projects after weighting.

[33] The underlying assumption in the estimation is that, conditional on observable time-invariant and time-varying characteristics, differences in outcomes are attributable only to being acquired by an overlapping incumbent (typically referred to as the ignorability assumption, or selection on observables). Busso, DiNardo, and McCrary (2014) show that the finite sample properties of this propensity score reweighting estimator are superior to propensity score matching techniques (where each treated firm is matched to one or several controls).

Beyond our main finding on overlap, table 2 also includes several other results that warrant discussion. First, reassuringly, the coefficients on $I(\text{Acquired})_i \times I(\text{Overlap})_i$ and $I(\text{Acquired})_i$ are both small in magnitude and insignificant, meaning that acquired drugs do not appear to have a different unconditional likelihood of development before acquisition.

Second, the γ_1 coefficient associated with $I(\text{Acquired})_i \times I(\text{Post})_{i,t}$ is negative and significant across specifications, implying a lower probability of development activity after acquisition. One reason for this pattern could be that our measure of overlap (same TC and same MOA) leads to potentially overly tight market definitions, and therefore even some nonoverlapping acquisitions may actually be killer acquisitions (i.e., substitute projects that are acquired and terminated). To investigate this, we separated out projects that overlap in TC only and found that the omitted category of nonoverlapping acquired projects now is consistently insignificant, albeit still negative (table F1; tables B1, B2, E1, E2, and F1–F8 are available online).[34]

We also run several additional analyses to supplement table 2's main results. Our results are robust to clustering standard errors at both the market (TC-MOA) and firm-market levels (table F2); measuring the acquired drug as overlapping only if the relevant acquirer's project or product is more advanced (i.e., older) than the target's (table F3); using age-TC fixed effects instead of the more narrow age-TC-MOA fixed effects to control for differences in drug development life cycles (table F4); and controlling for any preacquisition codevelopment or licensing deals, which are common in the pharmaceutical industry, by augmenting our data with comprehensive RecapIQ data on technology-related codevelopment and licensing deals (table F5). Finally, following prior literature on drug development (Guedj and Scharfstein 2004; Krieger 2017), we perform supplementary analyses, using clinical trial progression as our outcome variable, that replicate the table 2 results (app. E).

C. *Alternative Subsamples and Specifications*

Overall, table 2 provides 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. We also include two sets of

[34] The prior literature does not provide a clear expectation for the sign of this coefficient. If we were to assume that projects are acquired only if they are of high quality and that development synergies resulting from economies of scale and scope materialize after acquisition (Henderson and Cockburn 1996; Andrade, Mitchell, and Stafford 2001), 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).

TABLE 3
OVERLAPPING ACQUISITIONS AND PROJECT DEVELOPMENT: ALTERNATIVE SPECIFICATIONS

	DEVELOPMENT EVENT = 1			NO DEVELOPMENT = 1
	(1)	(2)	(3)	(4)
I(Acquired) × I(Post) × Overlap	−.050**	−.121***	.005	.149***
	(.023)	(.060)	(.035)	(.033)
I(Acquired) × I(Post)	−.024	−.041	−.095***	.401***
	(.015)	(.025)	(.013)	(.021)
Observations	27,784	19,651	7,916	9,227
R^2	.445	.249	.155	.477
Sample	Acquired projects only	Single-project target only	Excluding "never developed"	
TC × MOA FE				Y
Age × TC × MOA FE	Y	Y	Y	
Project FE	Y	Y	Y	Y

NOTE.—This table presents the postacquisition development likelihood of acquired drug projects (col. 1), single-project targets (col. 2), acquired drug projects with some postacquisition development (col. 3), and the likelihood of never experiencing a development event (col. 4). The general empirical specification is $\text{Development}_{i,t} = \beta \cdot I(\text{Acquired})_i \times I(\text{Post})_{i,t} \times I(\text{Overlap})_i + \gamma_1 \cdot I(\text{Acquired})_i \times I(\text{Post})_{i,t} + \alpha_{\text{FE}} + \varepsilon_{i,t}$, where the dependent variable $\text{Development}_{i,t}$ is a dummy variable indicating whether or not drug i has a development event in period t in cols. 1–3 (or has no development event in period t in col. 4); FE = fixed effects. I(Acquired) × I(Post) indicates whether the drug-period (i, t) observation is after the drug is acquired, and I(Acquired) × I(Post) × Overlap also indicates that the acquired drug overlaps with the acquirer's product portfolio. Standard errors clustered at the drug project level are displayed in parentheses.

** Significant at the 5% level.

*** Significant at the 1% level.

additional analyses that provide further supportive evidence for our interpretation of the table 2 results.

Table 3 includes these additional analyses. Column 1 duplicates our main results focusing on the acquired sample, confirming our main analyses (the unconditional development probability in this sample is 18.7%, with a 39.6% lower development rate for overlapping targets). In column 2, we examine postacquisition development when the target has only one drug at the time of acquisition, to address concerns that our findings could be the result of acquirer firms acquiring multiproject targets and developing only the most promising while discontinuing the others. If this mechanism is driving our results, we should expect the effect of overlap on postacquisition development to be smaller and/or insignificant in this sample. However, we find that it is both significant and larger in magnitude, alleviating these concerns.

Our second set of additional analyses aim to ensure that our main results on the decreased likelihood of development are due to project termination rather than to changes in development patterns. To do so, we perform

two different analyses. First, in table 3, column 3, we rerun our column 1 analysis on acquired projects, removing projects that are never developed after acquisition. If immediate project termination is driving our main findings, as we contend, then we should find no significant difference between acquired overlapping and acquired nonoverlapping projects after we take out projects that are never developed after acquisition. Second, in column 4, we examine the likelihood that a project is never developed after acquisition, which we expect to be significantly higher for overlapping acquisitions. For this second test, we collapse our panel into two time periods, before and after acquisition, and the outcome variable becomes no development.

The results from both analyses are consistent with immediate termination. First, in table 3, column 3, we find no significant difference in likelihood of development between acquired overlapping and acquired nonoverlapping projects after removing the never-developed projects. Second, in column 4, we find that overlapping projects are 14.9 percentage points, or 20.9% (0.149/0.711), more likely to have no postacquisition development events (i.e., to be immediately terminated) compared to nonoverlapping projects. Together, these results support termination, rather than delayed development, as the primary driver behind our results.[35]

The results from table 3 also help to alleviate any concerns that our main results might be driven by strategic reactions of nonacquired firms. We also investigate this possibility directly (in table F7) and find no evidence of increased development by nonacquired firms.

D. Product Market Competition

To investigate proposition 2, we examine how our empirical results differ across levels of competition. We measure competition as the count of launched products in the same TC using the same MOA as the focal project (our measure of "existing product" competition).[36] We include TC-MOA fixed effects to control for differences in baseline development rates

[35] In addition to focusing on using never-developed projects to capture immediate termination, if we look at postacquisition development, we find that the rate of having just one more postacquisition development event is 17.0% for overlapping acquired projects, compared to 21.8% for nonoverlapping acquired projects. In other words, overlapping acquired drugs are less likely to successfully complete the development stage in which they were at the time of acquisition, but the difference between overlapping and nonoverlapping acquired projects is smaller in this analysis than in the never-developed-again analysis.

[36] Each drug product can fall into multiple technologies (MOAs) and multiple intended markets (TCs). In the Pharmaprojects data set, drug projects have, on average, 1.3 MOAs (median 1; 81% have 1) and, on average, 1.9 TCs (median 2; 46% have 1). In constructing our aggregate counts of competitors, we count each project in all possible TC-MOAs in which it falls. For our measures of competition for the focal projects, we use the TC-MOA with the most competition. That is, if a project falls into two categories, one with no competitors and one with five, we use the latter.

TABLE 4
OVERLAPPING ACQUISITIONS AND PROJECT DEVELOPMENT: MARKET COMPETITION

	DEVELOPMENT EVENT = 1			NO DEVELOPMENT = 1		
	Low (1)	High (2)	Interacted (3)	Low (4)	High (5)	Interacted (6)
$I(\text{Acquired}) \times I(\text{Post}) \times$ Overlap	−.065**	.017	.017	.219***	.038	.038
	(.026)	(.035)	(.035)	(.054)	(.070)	(.070)
$I(\text{Acquired}) \times I(\text{Post}) \times$ Overlap × LowCompetition			−.082*			.181**
			(.044)			(.089)
Observations	74,261	69,308	143,569	5,991	3,236	9,227
R^2	.415	.399	.408	.497	.474	.489
TC × MOA FE				Y	Y	Y
Age × TC × MOA FE	Y	Y	Y			
Project FE	Y	Y	Y	Y	Y	Y

NOTE.—This table presents the development likelihood of drug projects, using a drug-year panel sample. The empirical specification uses the following model: $\text{Development}_{i,t} = \beta \cdot I(\text{Acquired})_i \times I(\text{Post})_{i,t} \times I(\text{Overlap})_i + \gamma_1 \cdot I(\text{Acquired})_i \times I(\text{Post})_{i,t} + \gamma_2 \cdot I(\text{Acquired})_i \times I(\text{Overlap})_i + \gamma_3 \cdot I(\text{Acquired})_i + \alpha_{\text{FE}} + \varepsilon_{i,t}$, where the dependent variable $\text{Development}_{i,t}$ is a dummy variable indicating drug i has a development event in year t for cols. 1–3 (or has no development event in period t for cols. 4–6). FE = fixed effects. $I(\text{Acquired})_i$ indicates that drug i undergoes an acquisition event, and $I(\text{Post})_{i,t}$ indicates that 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 TC-MOA) as the focal drug. In cols. 1–3, the analysis predicts likelihood of development across all projects in a project-year panel, while in cols. 4–6, the analysis predicts likelihood of no development for acquired projects, comparing pre- and postacquisition periods and overlapping and nonoverlapping acquired projects. Drug development projects are categorized into high and low competition by the median of competition measures (product count). In cols. 3 and 6, we present results in which we interact $I(\text{Acquired})_i \times I(\text{Post})_{i,t} \times I(\text{Overlap})_i$ with the dummy indicating low competition. Standard errors clustered at the drug project level are displayed in parentheses.

* Significant at the 10% level.
** Significant at the 5% level.
*** Significant at the 1% level.

across markets that could lead to both lower competition and lower development rates. Table 4 presents the regression results that examine whether the postacquisition development pattern of acquired projects varies under different competition environments. We categorize drug development projects into high and low competition by the sample median (which is 2). In columns 1–3, we examine the role of competition in predicting development activity in the project-year panel, and in columns 4–6, we predict no development activity after acquisition across different levels of competition.

The results suggest that the decreased likelihood of development of overlapping projects during the postacquisition period concentrates in product markets with relatively low competition. Comparing columns 1 and 2, we observe that the development of an overlapping acquired drug under low competition 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 the unreported coefficient −0.015 associated with $I(\text{Acquired}) \times I(\text{Post})$, and the benchmark of the unconditional development rate in the subsample (19.4%) imply that acquired overlapping projects are 41.2% ((0.065 + 0.015)/0.194) less likely to be developed than nonacquired drugs and are 36.3% (0.065/(0.194 − 0.015)) less likely to be developed than acquired nonoverlapping drugs. In column 3, we test the difference between high and low competition, using an interaction term.[37] The results in columns 4–6 for no development as the outcome (following table 3) show similar findings. Further, we find the same patterns in our supplementary analysis of clinical trial outcomes (app. E) for both product market and pipeline competition.

E. 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. For each of those projects, we identify the patents associated with the acquiring firm's relevant (overlapping) approved drugs. We source patent data matched at the drug level via Pharmaprojects (which uses the FDA Orange Book data) and link patent filing dates from US Patent and Trademark Office data.

Table 5 presents these results. The key coefficient is for $I(\text{Post}) \times I(\text{NearPatExpiry})$, which contrasts those with patents near expiration (i.e., within 5 years) with those with longer remaining patent life. Consistent with our predictions, we find that when relevant acquirer patents are near expiration, the decrease in development associated with acquisition is mitigated. Specifically, among overlapping acquired drugs, those for which the acquirer patents are near expiration are more likely to have development events after acquisition, compared to projects for which the relevant acquirer patents are relatively far from expiration. These results are sensitive to whether we allow age fixed effects to vary across TC-MOAs (as in col. 2), suggesting that development life cycles vary across TC-MOAs in ways that affect how salient the remaining patent life is in shaping killer-acquisition motives. Further, these results rely on a relatively small sample (i.e., drugs for which we have information on the acquirer's overlapping patent via Pharmaprojects).

[37] We also perform these analyses using the count of projects under development as our measure of competition (i.e., pipeline competition). The results are robust and are shown in table F8.

TABLE 5
OVERLAPPING ACQUISITIONS AND PROJECT DEVELOPMENT: ACQUIRER'S PATENT LIFE

	DEVELOPMENT EVENT = 1	
	(1)	(2)
I(Post) × I(NearPatExpiry)	.013	.406***
	(.133)	(.090)
I(Post)	−.173*	−.210***
	(.092)	(.067)
I(NearPatExpiry)	−.104**	−.147***
	(.043)	(.043)
Observations	6,398	6,398
R^2	.212	.450
Vintage FE	Y	Y
Age FE	Y	
TC × MOA FE	Y	
Age × TC × MOA FE		Y

NOTE.—This table presents the development likelihood of drug projects, using a drug-year panel of 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: $\text{Development}_{i,t} = \beta_O \cdot I(\text{Post})_{i,t} + \beta \cdot I(\text{NearPatExpiry})_i + \gamma_O \cdot I(\text{NearPatExpiry})_i \times I(\text{Post})_{i,t} + \alpha_{FE} + \varepsilon_{i,t}$, where the dependent variable $\text{Development}_{i,t}$ is a dummy variable indicating whether drug i has a development event in year t. FE = fixed effects. $I(\text{Post})_{i,t}$ indicates whether the drug-year (i, t) observation is after the drug is acquired. I(NearPatExpiry) is a dummy variable indicating whether the overlapping acquirer drug is within 5 years of patent expiry. Standard errors clustered at the drug project level are displayed in parentheses.

* Significant at the 10% level.
** Significant at the 5% level.
*** Significant at the 1% level.

F. Acquisition Decisions

1. Determinants of Acquisitions

Our empirical analysis so far has focused on drug development, finding that (1) a project is less likely to be developed after being acquired by a firm with an overlapping existing drug (consistent with proposition 1) and that (2) these results are concentrated in markets with low levels of competition (proposition 2) and (3) when relevant acquirer patents are far from expiration (proposition 3).

Our theoretical model also predicts that acquiring incumbents should acquire target firms with overlapping drugs (i.e., overlap will positively predict acquisition; proposition 4). 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 acquirer-target project, we construct two different samples of potential acquisition deals (the pseudodeals). 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 that have ever performed an acquisition before the deal year. For each of those pseudoacquirers, we then form pseudoacquisitions with target project i. Second, we form a size-matched control sample: we match each acquirer in each deal to five control firms on the basis of the total number of drug projects in the year of the deal.

The analysis is performed using the following model:

$$\text{Acquirer-Target}_{ijd,t} = \beta \cdot I(\text{Overlap})_{ijd,t} + \alpha_{\text{FE}} + \varepsilon_{ijd,t}. \tag{8}$$

The dependent variable, $\text{Acquirer-Target}_{ijd,t}$, is equal to one if firm-project pair ij is a real acquirer-target pair and is zero otherwise (i.e., a pseudopair). The key explanatory variable $I(\text{Overlap})$ 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.

Table 6 presents the marginal effects from a conditional logit estimation evaluated at the mean, separately for each control sample: randomly matched in columns 1–4 and matched by size in columns 5–8. In column 1, the estimated marginal effect of 0.626 implies that acquisitions are almost four times as likely to occur 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 TC). 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 5–8 duplicate these results for the size-matched control sample. Collectively, these results suggest that overlap significantly influences the acquisition decisions of incumbents.

A higher propensity to undertake overlapping acquisitions does not isolate a strategic killer-acquisition motive on its own. However, alternative theories of corporate acquisition and development cannot explain both our acquisition and our 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 nonoverlapping 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,

TABLE 6
PRODUCT OVERLAP AND ACQUISITION DECISIONS

	ACQUISITION = 1				ACQUISITION = 1			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Overlap (TC-MOA)	.626***		.577***		.194***		.209***	
	(.009)		(.015)		(.010)		(.015)	
Overlap (TC)		.356***		.300***		.214***		.200***
		(.005)		(.008)		(.008)		(.011)
Overlap (TC-MOA) × LowCompetition			.088***				−.027	
			(.019)				(.020)	
Overlap (TC) × LowCompetition				.103***				.025*
				(.011)				(.015)
Observations	55,374	55,374	38,430	38,430	34,005	34,005	34,005	34,005
Pseudo R^2	.118	.119	.098	.097	.052	.064	.052	.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

NOTE.—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 randomly matched sample (cols. 1–4) and a size-matched sample (cols. 5–8). The empirical specification uses the following model: $\text{Acquirer-Target}_{ijd,t} = \beta \cdot I(\text{Overlap})_{ijd,t} + \alpha_{\text{FE}} + \varepsilon_{ijd,t}$, where the dependent variable $\text{Acquirer-Target}_{ijd,t}$ is a dummy variable indicating that drug i is acquired by firm j in year t (and is otherwise a pseudopair). $I(\text{Overlap}_{ijd,t})$ is a dummy variable indicating that the target drug overlaps the potential acquirer firm. LowCompetition is a dummy variable indicating that the target drug is in a low-competition market. We include deal-level fixed effects (FE; for both realized deals and pseudodeals). We report marginal effects from the estimation. Standard errors clustered at the deal level are displayed in parentheses.

* Significant at the 10% level.

*** Significant at the 1% level.

rather than less. Hence, a synergy motive contrasts sharply with our empirical findings of decreased development in section IV.B.

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 FTC's premerger review rule under the Hart-Scott-Rodino Antitrust Improvements Act (HSR) of 1976 because they fall below the acquisition deal size threshold.[38] 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 or just below the threshold. If incumbent firms conduct killer acquisitions that are intentionally under the FTC's radar, 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.

In figure 3, we plot the distribution of acquisition sizes for a narrow window around the HSR review threshold, specifically, just below it ($[-5\%, 0]$) and just above it ($[0, 5\%]$). Acquisition size is proxied by the deal amount. We categorize acquisitions into acquisitions of nonoverlapping targets (*left*) and acquisitions of overlapping targets (*right*). We observe clear bunching of deals right below the review threshold, but this pattern is apparent only for deals in which the target has projects that overlap with the acquirer (i.e., killer-acquisition suspects).

In table 7, we compare the termination and launching rates of acquisitions around the HSR threshold. We construct two buckets, which include all acquisitions with a transaction value just below or above the FTC review

[38] 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 premerger 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 have 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 (2019) 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.

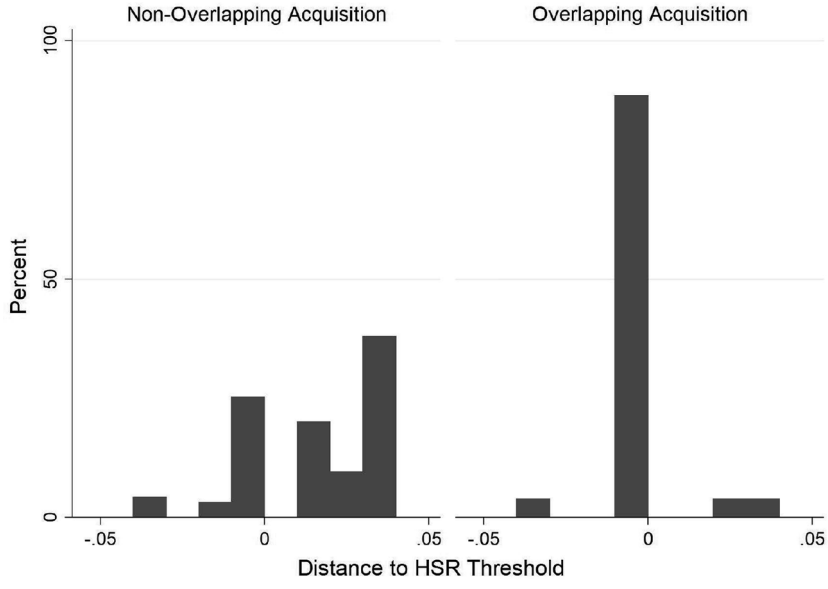

FIG. 3.—Distribution of acquisition size near the Hart-Scott-Rodino (HSR) review threshold. Acquisitions that fall into the interval [−5%, 5%] around the threshold are kept, and the horizontal axis represents the distance to the review threshold (from −5% to 5%). Nonoverlapping acquisitions are reported on the left, and overlapping acquisitions are reported on the right.

threshold. We find that the eventual product launch rate is much lower (1.8% vs. 9.1%) and the discontinuation rate is much higher (94.6% vs. 83.3%) for below-threshold acquisitions, compared to those right above the threshold. Thus, table 7 provides supporting evidence of strategic behavior by acquiring firms that is consistent with killer-acquisition motives.

However, one might reasonably expect that part of the observed decrease in success rates for acquisitions that are a few millions of dollars smaller is explained by a positive relationship between deal values and eventual development success. We therefore examine the difference in development rates around an HSR "pseudothreshold." To compare with the real HSR threshold (an annually adjusted value set using a base of $200 million in 2000), we construct a pseudothreshold (2000 base value of $150 million). This pseudothreshold is comparable in value to the real threshold but is sufficiently different that we should not observe strategic behavior in its vicinity. The results of this analysis (panel B of table 7) suggest that small differences in deal value do not produce significant development differences except when they occur at the real HSR threshold. Furthermore, they are not sensitive to the particular pseudothreshold we choose and are therefore consistent with the idea that acquirers conduct more

TABLE 7
INTENSITY OF PROJECT DISCONTINUATION AROUND FTC REVIEW THRESHOLD

	5% below Threshold (%)	5% above Threshold (%)	Difference (%)	*T*-Statistics	Statistical Significance
	A. Real HSR Threshold				
Active	3.57	7.58	−4.00	−1.18	Not significant
Launched	1.79	9.09	−7.31	−2.29	5% level
Discontinued	94.64	83.33	11.31	2.51	5% level
	B. Pseudothreshold				
Active	7.41	2.63	4.78	1.20	Not significant
Launched	3.70	4.39	−.69	−.16	Not significant
Discontinued	88.88	92.98	−4.10	−.71	Not significant

NOTE.—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 rates of being active, being discontinued, and being fully launched separately for the two samples and the difference between them. *T*-statistics of the sample means and the significance levels are reported. In panel B, we report the same analysis with a pseudo–HSR threshold. To construct the pseudothreshold, we set the base value at $150 million in 2000 and follow the same adjustment schedule (to account for the change in gross national product).

killer acquisitions in situations in which they can expect to avoid FTC scrutiny.

V. Discussion

A. *Alternative Explanations*

In this section, we address several potential alternative explanations that 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 specifically why overlapping acquired drug projects are more likely to be terminated than nonacquired or nonoverlapping acquired drug projects.

1. Informational Asymmetries in the Acquisition Market

Focusing on overlapping acquired projects means that asymmetric information, or "market for lemons"–type arguments, is 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 caused by informational asymmetries.

2. Optimal Project Selection

Given that some targets are multiproject firms, our results could reflect acquirers optimally choosing 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 3, we found results similar to those in the full sample. Our main results are therefore unlikely to be driven by optimal project selection.

3. Redeployment of Technologies

Another alternative explanation for our results is that firms acquire targets not for their projects but for their technologies. Under this logic, acquirers would shut down the target's projects and redeploy the technologies to more productive ends (i.e., to start more-promising projects). Such a rationale is less relevant in the pharmaceutical industry, compared to the tech context, as in pharmaceutical development the underlying technology (and associated patents) is closely tied to the specific drug. Nonetheless, in principle, technology redeployment is consistent with our findings, as overlapping projects are more likely to be underpinned by redeployable technologies.

We investigate technology redeployment by exploiting molecule-level information for each drug project. We compare the chemical structure of acquired projects to that of those developed by the acquirer before and after acquisition and assess whether acquirer firms' projects initiated after acquisition are more similar to the acquired project than their preacquisition drugs (consistent with technology redeployment). To measure similarity, we follow recent research in economics by Krieger, Li, and Papanikolaou (2017) and use the Tanimoto distance—the proportion of chemical fragments shared by any two chemicals—to measure similarity between two molecules (Nikolova and Jaworska 2003).

Table 8, panel A, presents chemical similarities to the acquired drug for drugs initiated by the acquirer after acquisition, compared to preacquisition drugs. In columns 1–3, 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. Contrary to a redeployment explanation, drugs initiated by acquirer firms after the acquisition of a drug are not significantly more similar to the acquired overlapping drug than preacquisition 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.

TABLE 8
POSTACQUISITION ASSET REDEPLOYMENT

	(1)	(2)	(3)	(4)	(5)	(6)
	A. Project Similarities to Acquired Drugs before and after Acquisition					
	Chemical Similarity			Citation to Targets		
I(Post) × Overlap	.001	.000	.002	−.002	−.002	−.000
	(.003)	(.003)	(.002)	(.002)	(.002)	(.000)
I(Post)	−.002	−.001	−.004	.000	.001	.000
	(.004)	(.004)	(.003)	(.000)	(.001)	(.000)
Overlap	.004	.004		.002	.002	
	(.003)	(.003)		(.002)	(.002)	
Observations	154,896	154,896	154,896	154,896	154,896	154,896
R^2	.001	.014	.361	.001	.094	.154
Acquirer FE		Y			Y	
Case FE			Y			Y

	B. Inventor Mobility and Patent Productivity before and after Acquisition		
	Before Acquisition	After Acquisition	Difference
Inventors who move to acquirer after acquisition (22%)	4.572	3.160	−1.412***
Inventors who move to other firms after acquisition (78%)	4.357	4.089	−.267*
Difference	−.215	.929***	1.144***

NOTE.—Panel A studies chemical similarities (via Tanimoto distance) and patent citations of drug projects originated by the acquirer firm. An observation is a pair of an acquired drug and a drug from the acquirer originated within the 5-year window around the acquisition event. Standard errors clustered at the drug project level are displayed in parentheses. Panel B presents inventor mobility and productivity of target firm inventors before and after acquisitions. We compare counts of new patent applications for target inventors who moved to the acquirer to those who moved to other firms. We report *t*-tests for subsample differences. FE = fixed effects.

* Significant at the 10% level.

*** Significant at the 1% level.

In columns 4–6, we adopt the same analytical structure to study an alternative measure of technology redeployment, patent citations to acquired projects. Echoing columns 1–3, we find no evidence of technology redeployment.

4. Redeployment of Human Capital

Our results could also be due to acquisitions motivated by human capital redeployment (Lacetera, Cockburn, and Henderson 2004; Ouimet and Zarutskie 2011). Such "acqui-hiring"—that is, acquiring start-ups, jettisoning the core business, and retaining the employees (Chatterji and Patro 2014; Kim 2018)—is likely less common in the pharmaceutical industry, which is almost exclusively project driven (Gompers et al. 2016), with strong project-specific intellectual property rights protection, compared to many other industries in which start-ups are valued more for their human capital. However, as in the case of technology redeployment, we would expect that the human capital underpinning overlapping projects would be useful for the acquiring firm, and so this alternative explanation could apply to our main analyses.

To formally investigate human capital redeployment, we examine target firm inventor mobility and productivity after acquisitions. To do so, we use data on disambiguated inventor names and organizational affiliations (via patent assignees) from Lai, D'Amour, and Fleming (2009) to track individuals over time and across organizations, following Bernstein (2015) and Brav et al. (2018). Specifically, we construct a list of preacquisition target firm inventors by identifying those who filed at least one patent within the 5-year window before the acquisition. We then track whether target firm inventors stay with acquiring firms and whether those who remain appear to be efficiently redeployed.

Table 8, panel B, shows the human capital results. First, only 22% of preacquisition inventors move to the acquirer after acquisition, while 78% move to other firms. Second, while those who stay and those who leave are statistically comparable before the acquisition, patenting roughly 4.5 times in the 5 years leading up to the acquisition, we find no 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 over the 5 years after acquisition. In contrast, inventors who move to other firms have a smaller productivity drop (<10%).[39]

[39] 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. Relevant patents at the drug level are typically disclosed late in drug development, when required by the FDA. Further, patents linked at a product level are available systematically in the FDA Orange Book only for approved drugs. As a result, we cannot identify whether inventors are associated with not-yet-approved projects that are shut down. However, if we focus on single-drug targets, we find that an even larger proportion of inventors leave the combined firm after the acquisition (although the sample becomes quite small).

5. Salvage Acquisitions

Another alternative explanation for our results could be that project failure itself might drive acquisitions and in particular overlapping acquisitions. In other words, firms might be motivated to acquire firms because their projects are failing, driven by the potential salvage value of the (likely cheap) reusable assets.

Our prior analyses provide some evidence that salvage acquisitions are unlikely to explain our results. First, the development rate of otherwise similar drugs does not differ significantly before acquisition (in table 2, col. 6), and second, we do not find evidence that the overlapping acquired drugs' technology is more likely to be redeployed (in table 8).

To directly investigate the salvage-acquisition explanation, we compared reported acquisition values across overlapping and nonoverlapping acquisitions. A salvage-acquisition explanation would predict relatively low valuations for overlapping acquisitions. Alternatively, killer acquisitions should involve paying fair value, and hence we should expect no difference. To perform this analysis, we collected additional information on the value of each acquisition from SDC Platinum and RecapIQ. One note of caution is that this is a selected sample of acquisitions, as it is conditional on the availability of deal value information.

The dependent variable in this analysis is the natural logarithm of the total disclosed acquisition amount (USD). The key explanatory variable is the dummy variable on whether the acquisition is conducted by an overlapping acquirer. Table 9 presents the results. We find that acquisitions conducted by overlapping acquirers are not of significantly lower value, compared to nonoverlapping acquisitions. Combined with the above-mentioned results, these results suggest that salvage acquisitions by overlapping firms are not driving our results.

B. Frequency and Importance of Killer Acquisitions

Our findings on differential project development allow us to roughly calculate the pervasiveness of killer acquisitions as well as their impact on industry-wide development decisions.[40] In our main analyses, we show 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 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 (22.7% of acquired

[40] Our back-of-the-envelope calculations use development parameters from our estimates alongside counts of annual projects and acquisition rates from the full Pharmaprojects data set.

TABLE 9
PROJECT OVERLAP AND ACQUISITION VALUE

	ln(Acquisition Value)		
	(1)	(2)	(3)
Overlap	.126	.025	−.082
	(.101)	(.067)	(.114)
Observations	14,660	14,660	14,660
R^2	.844	.905	.940
Acquirer FE	Y	Y	Y
Age FE	Y	Y	
TC × MOA FE		Y	
Age × TC × MOA FE			Y

NOTE.—This table investigates whether overlapping firms are acquired for lower transaction sums than nonoverlapping firms. The sample for this analysis is acquired firms where the deal value amount is available in one of our three main acquisition data sets (Thomson Reuters SDC Platinum, RecapIQ, and VentureXpert). The empirical specification uses the following model: $\ln(\text{AcquisitionValue})_i = \beta \cdot I(\text{Overlap})_i + \alpha_{\text{FE}} + \varepsilon_i$, where the dependent variable $\ln(\text{AcquisitionValue})_i$ is the natural logarithm of the total disclosed acquisition amount (USD) and $I(\text{Overlap}_i)$ is a dummy variable indicating the target overlaps the potential acquirer firm. Robust standard errors are displayed in parentheses. FE = fixed effects.

projects) continue at an adjusted rate of 13.4%, while acquired projects without overlap (77.3% of acquired projects) continue development at an adjusted rate of 17.5%.

To roughly calculate the number of killer acquisitions, we assume that there are two types of acquisitions that fall into the acquired overlapping category: killer acquisitions that are purely intended to shut down future competitors (and thus have a 0% probability 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 5.3% ($\nu \times 22.7\,\%$) of all acquisitions, or about 46 ($5.3\,\% \times 856$) acquisitions every year, are killer acquisitions. If instead we assume the nonkiller acquisitions to have the same development likelihood as nonacquired projects (19.9%), we estimate that 7.4% of acquisitions, or 63 per year, are killer acquisitions.

Alternatively, we can estimate the number of killer acquisitions by using the results of table 3. Specifically, we find that the share of acquired projects for which no positive development event is observed after acquisition is 14.9 percentage points higher for overlapping acquired projects than for nonoverlapping acquired projects. If this higher share of projects that are never developed is due to killer acquisitions, it implies a minimum of 29 ($14.9\,\% \times 22.7\,\% \times 856$) killer acquisitions per year.

Another benchmark for understanding the frequency of killer acquisitions is the share of drug projects that actually are at risk of being the target of a killer acquisition. We first define and identify the risk set. It comprises those drug projects that potential acquirers would plausibly want to acquire to kill that come from plausibly acquirable targets. Thus, we define the killer-acquisition risk set as all "follow-on" drugs initiated by "new" firms. We define follow-on drugs as the second (or later) drug project initiated in a TC-MOA category. Such drugs constitute 70% of our total sample. We then identify those follow-on drugs that are initiated by firms that are plausible acquisition targets (i.e., new firms), using three different categorizations: (1) firms with no prior projects, (2) firms with no prior projects in the TC, and (3) firms with no prior projects in the TC-MOA. In our data, the corresponding shares of follow-on drugs are 35%, 66%, and 84%, respectively. Using those numbers as an estimate of the risk set of potential killer-acquisition targets, and using an estimate of 1.5% of all drugs—including those not at risk—as killer acquisitions,[41] we estimate that 6.3%, 3.3%, or 2.6% of potential killer-acquisition targets are acquired to kill.

These back-of-the-envelope calculations provide a lower bound for the actual number of killer acquisitions, as 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 the development of acquired projects with overlap.

How would overall development rates in the pharmaceutical industry be affected if antitrust policy directly targeted killer acquisitions? The average development probability in our sample is 18.2%. 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 nonacquired projects (47.5% of all projects). The number of total drug projects for which development continues would increase by 4.3% ($[(19.9\,\% - 13.4\,\%)/18.2\,\%] \times (1 - 47.5\,\%) \times 22.7\,\%$), or by about 13 drug projects per year ($18.2\,\% \times 4.3\,\% \times 1,630$, where 1,630 is the yearly average number of projects).

We can compare these to estimates of the effects of targeted innovation policies in the pharmaceutical industry. One policy—considered successful but also very costly—is the Orphan Drug Act, which focused

[41] This is computed by using the above estimates of 5.3%–7.4% of all acquired drug projects as killer acquisitions, which implies 1.3%–1.8%, or roughly 1.5% of all drug projects.

on encouraging the 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 per trial), grants, and extended market exclusivity. Economic analysis by Yin (2008, 2009) suggests that the policy resulted in roughly 25 more clinical trials per year from 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 those from the Orphan Drug Act.

It is also instructive to compare killer acquisitions to reverse-payment patent settlements ("pay-for-delay"), a common and related phenomenon in the pharmaceutical industry whereby incumbents pay to temporarily thwart the entry of generic competitors.[42] Helland and Seabury (2016) estimate that over the next 25 years, pay-for-delay settlements in pharmaceuticals will generate a deadweight loss of at least $21 billion. Although our analysis does not allow us to compute similar welfare estimates, we believe that killer acquisitions likely cause at least as much anticompetitive harm as pay-for-delay settlements. The FTC reports that in the period from 2004 to 2016, there were between 10 and 20 pay-for-delay settlements per year: 20 if one counts any patent infringement settlement that includes restriction on generic entry and some nonzero payment and only 13 if one excludes cases where the payment was solely for litigation costs above $7 million. In contrast, our analyses suggest that during approximately the same time frame there were 46–63 killer acquisitions per year.

C. 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 of the other incumbents, are better off when such acquisitions are allowed. But consumers are hurt by both the lack of competition and the elimination of innovative new products. In other words, patients suffer because there are fewer drugs and because the drugs that are developed and brought to market are sold at higher prices.[43]

[42] Pay-for-delay has been the subject of substantial theoretical (Shapiro 2003; Bulow 2004; Lemley and Shapiro 2005; Hemphill 2006) and empirical research (FTC 2010; Bokhari 2013; Drake, Starr, and McGuire 2015; Helland and Seabury 2016; Ghili and Schmitt 2017; Hartman, Drake, and McGuire 2019).

[43] Although killer acquisitions 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 for the loss in producer surplus of the existing incumbents, as in Mankiw and Whinston

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 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 this paper, but two points are worthy of discussion.

The presence of an acquisition channel may have a countervailing 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) and Letina, Schmutzler, and Seibel (2020). 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. If the entrepreneur is left with no surplus relative to the standalone value of her project, she will not increase 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 affect the direction of innovation. If entrepreneurs can choose between originating projects that overlap with existing products and 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 truly novel products.[44] This distortion of the direction of innovation in response to the prospect of acquisition will add to the negative welfare impact of killer acquisitions.[45]

(1986). In app. 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 incumbents' existing drugs, killer acquisitions reduce not only consumer surplus but also total welfare. Put differently, killer acquisitions of "me-too" drugs (drugs that are very close substitutes) in markets in which there is more than a single incumbent need not be welfare reducing because they destroy producer surplus of existing incumbents by more than they increase consumer surplus. However, as we show in app. A, it is precisely in cases in which killer acquisitions do not harm welfare that they are also unlikely to take place.

[44] A variety of evidence (Adams and Brantner 2006; Budish, Roin, and Williams 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.

[45] 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.

Given these competing forces, the overall effect of killer acquisitions on ex ante innovation (and, therefore, welfare) remains unclear. However, on the basis of our analysis, 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 spur ex ante innovation. In fact, we document a 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 well-known immediate benefits for social welfare but also deters incumbents from engaging in killer acquisitions of future competitors, thus increasing future competition and further deterring killer acquisitions.

VI. Conclusion

In this paper, we document that incumbent firms acquire innovative targets and terminate their innovative projects in order to preempt future competition. Empirically, we exploit the setting of drug development, in which we are able to track project development before and after acquisitions. Consistent with the killer-acquisitions motive, we show that incumbents acquire firms with overlapping drug projects and that overlapping acquired drugs are less likely to be developed, particularly when the acquirer has strong incentives to protect his existing market power. Alternative interpretations, such as optimal project selection, delayed development, the redeployment of technological or human capital, and salvage acquisitions, 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 start-up exit and have become increasingly popular as an exit strategy over time across various industries, suggesting that the potentially damaging consequences reach beyond pharmaceuticals.[46] Our results caution against interpreting acquisitions of nascent technologies solely as incumbents' efforts to integrate and foster entrepreneurial innovation. Instead, a substantial part of what is fueling this trend may actually be killer acquisitions that potentially harm innovation and competition. In particular, the large number of acquisitions of small entrepreneurial start-ups by large incumbents in the tech sector would suggest a fruitful opportunity for investigating whether killer acquisitions extend beyond the pharmaceutical industry. However, the lack of strong intellectual property protection afforded by drug patents, the importance of acquiring and retaining

[46] For example, following recent reports about an alleged killer acquisition in the medical ventilator industry, some FTC officials have called for a retrospective antitrust investigation: https://promarket.org/the-danger-of-no-antitrust-enforcement-how-a-merger-led-to-the-us-ventilator-shortage/.

valuable human capital, and the relative data scarcity pose new challenges for any future theoretical or empirical analysis.[47]

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 start-ups' inventions topple entrenched and less innovative incumbents—may be smaller than previously documented. Innovation and the share of young firms in economic activity may have declined (Akcigit and Ates 2019) not only because incumbents are more reluctant to innovate but also because incumbent firms with market power acquire innovators to eliminate future competition and thereby inhibit technological progress.

References

Adams, Christopher P., and Van V. Brantner. 2006. "Estimating the Cost of New Drug Development: Is It Really $802 Million?" *Health Affairs* 25 (2): 420–28.

———. 2010. "Spending on New Drug Development." *Health Econ.* 19 (2): 130–41.

Akcigit, Ufuk, and Sina T. Ates. 2019. "Ten Facts on Declining Business Dynamism and Lessons from Endogenous Growth Theory." Working Paper no. 25755 (April), NBER, Cambridge, MA.

Andrade, Gregor, Mark Mitchell, and Erik Stafford. 2001. "New Evidence and Perspectives on Mergers." *J. Econ. Perspectives* 15 (2): 103–20.

Anton, James J., and Dennis A. Yao. 2002. "The Sale of Ideas: Strategic Disclosure, Property Rights, and Contracting." *Rev. Econ. Studies* 69 (3): 513–31.

Arcidiacono, Peter, Paul B. Ellickson, Peter Landry, and David B. Ridley. 2013. "Pharmaceutical Followers." *Internat. J. Indus. Org.* 31 (5): 538–53.

Arrow, Kenneth. 1962. "Economic Welfare and the Allocation of Resources for Invention." In *The Rate and Direction of Inventive Activity: Economic and Social Factors*, 609–26. Princeton, NJ: Princeton Univ. Press (for NBER).

ASPE. 2014. "Examination of Clinical Trial Costs and Barriers for Drug Development." Technical report, Office of the Assistant Secretary for Planning and Evaluation, US Dept. Health and Human Services, Washington, DC.

[47] A first attempt at quantifying potential adverse effects of acquisitions by tech giants on start-up innovation is by Kamepalli, Rajan, and Zingales (2020), who theoretically and empirically study "kill zones" in which new tech ventures are no longer worth funding after the acquisition of an entrant. However, Cabral (2020) cautions that a regulatory tightening of merger policy in the high-tech space could also lead to a significant discouragement effect on innovation. Nevertheless, as of February 2020, the FTC is conducting a 6(b) study of potentially anticompetitive acquisitions by large technology companies over the past decade: https://www.ftc.gov/system/files/documents/public_statements/1566385/statement_by_commissioners_wilson_and_chopra_re_hsr_6b.pdf.

Baker, Jonathan B., and Timothy F. Bresnahan. 1985. "The Gains from Merger or Collusion in Product-Differentiated Industries." *J. Indus. Econ.* 33 (4): 427–44.

Bena, Jan, and Kai Li. 2014. "Corporate Innovations and Mergers and Acquisitions." *J. Finance* 69 (5): 1923–60.

Berndt, Ernst R., Thomas McGuire, and Joseph P. Newhouse. 2011. "A Primer on the Economics of Prescription Pharmaceutical Pricing in Health Insurance Markets." *Forum Health Econ. and Policy* 14 (2): 10.

Bernstein, Shai. 2015. "Does Going Public Affect Innovation?" *J. Finance* 70 (4): 1365–403.

Bliss, Christopher, and Barry Nalebuff. 1984. "Dragon-Slaying and Ballroom Dancing: The Private Supply of a Public Good." *J. Public Econ.* 25 (1–2): 1–12.

Blume-Kohout, Margaret E., and Neeraj Sood. 2013. "Market Size and Innovation: Effects of Medicare Part D on Pharmaceutical Research and Development." *J. Public Econ.* 97:327–36.

Bokhari, Farasat A. S. 2013. "What Is the Price of Pay-to-Delay Deals?" *J. Competition Law and Econ.* 9 (3): 739–53.

Branstetter, Lee, Chirantan Chatterjee, and Matthew J. Higgins. 2014. "Starving (or Fattening) the Golden Goose? Generic Entry and the Incentives for Early-Stage Pharmaceutical Innovation." Working Paper no. 20532 (September), NBER, Cambridge, MA.

Brav, Alon, Wei Jiang, Song Ma, and Xuan Tian. 2018. "How Does Hedge Fund Activism Reshape Corporate Innovation?" *J. Financial Econ.* 130 (2): 237–64.

Bryan, Kevin, and Erik Hovenkamp. 2020a. "Antitrust Limits on Startup Acquisitions." *Rev. Indus. Org.* 56 (4): 615–36.

———. 2020b. "Startup Acquisitions, Error Costs, and Antitrust Policy." *Univ. Chicago Law Rev.* 87 (2): 331–56.

Budish, Eric, Benjamin N. Roin, and Heidi Williams. 2015. "Do Firms Underinvest in Long-Term Research? Evidence from Cancer Clinical Trials." *A.E.R.* 105 (7): 2044–85.

Bulow, Jeremy. 2004. "The Gaming of Pharmaceutical Patents." *Innovation Policy and Econ.* 4:145–87.

Busso, Matias, John DiNardo, and Justin McCrary. 2014. "New Evidence on the Finite Sample Properties of Propensity Score Reweighting and Matching Estimators." *Rev. Econ. and Statis.* 96 (5): 885–97.

Cabral, Luís. 2017. "Standing on the Shoulders of Dwarfs: Dominant Firms and Innovation Incentives." Working paper, New York Univ. Stern.

———. 2020. "Merger Policy in Digital Industries." *Information Econ. and Policy.* https://doi.org/10.1016/j.infoecopol.2020.100866.

Caves, Richard E., Michael D. Whinston, and Mark A. Hurwitz. 1991. "Patent Expiration, Entry, and Competition in the U.S. Pharmaceutical Industry." *Brookings Papers Econ. Activity: Microeconomics* 1991:1–66.

Chatterji, Aaron, and Arun Patro. 2014. "Dynamic Capabilities and Managing Human Capital." *Acad. Management Perspectives* 28 (4): 395–408.

Cockburn, Iain, and Rebecca Henderson. 1994. "Racing to Invest? The Dynamics of Competition in Ethical Drug Discovery." *J. Econ. and Management Strategy* 3 (3): 481–519.

Cockburn, Iain M. 2004. "The Changing Structure of the Pharmaceutical Industry." *Health Affairs* 23 (1): 10–22.

Cohen, Wesley M. 2010. "Fifty Years of Empirical Studies of Innovative Activity and Performance." In *Handbook of the Economics of Innovation*, vol. 1, edited by Bronwyn H. Hall and Nathan Rosenberg, 129–213. Amsterdam: North-Holland.

Conner, Kathleen Reavis. 1988. "Strategies for Product Cannibalism." *Strategic Management J.* 9 (S1): 9–26.
Diekmann, Andreas. 1985. "Volunteer's Dilemma." *J. Conflict Resolution* 29 (4): 605–10.
DiMasi, J. A., L. Feldman, A. Seckler, and A. Wilson. 2010. "Trends in Risks Associated with New Drug Development: Success Rates for Investigational Drugs." *Clinical Pharmacology and Therapeutics* 87 (3): 272–77.
Drake, Keith M., Martha A. Starr, and Thomas G. McGuire. 2015. "Do 'Reverse Payment' Settlements Constitute an Anticompetitive Pay-for-Delay?" *Internat. J. Econ. Bus.* 22 (2): 173–200.
Dubois, Pierre, Olivier de Mouzon, Fiona Scott Morton, and Paul Seabright. 2015. "Market Size and Pharmaceutical Innovation." *RAND J. Econ.* 46 (4): 844–71.
Duggan, Mark, and Fiona Scott Morton. 2010. "The Effect of Medicare Part D on Pharmaceutical Prices and Utilization." *A.E.R.* 100 (1): 590–607.
Ellison, Glenn, and Sara Fisher Ellison. 2011. "Strategic Entry Deterrence and the Behavior of Pharmaceutical Incumbents Prior to Patent Expiration." *American Econ. J.: Microeconomics* 3 (1): 1–36.
Ellison, Sara Fisher, Iain Cockburn, Zvi Griliches, and Jerry Hausman. 1997. "Characteristics of Demand for Pharmaceutical Products: An Examination of Four Cephalosporins." *RAND J. Econ.* 28 (3): 426–46.
Federico, Giulio, Gregor Langus, and Tommaso Valletti. 2017. "A Simple Model of Mergers and Innovation." *Econ. Letters* 157:136–40.
———. 2018. "Horizontal Mergers and Product Innovation." *Internat. J. Indus. Org.* 59:1–23.
Filson, Darren. 2012. "A Markov-Perfect Equilibrium Model of the Impacts of Price Controls on the Performance of the Pharmaceutical Industry." *RAND J. Econ.* 43 (1): 110–38.
FTC (Federal Trade Commission). 2010. "Pay-for-Delay: How Drug Company Pay-Offs Cost Consumers Billions." Staff study, FTC, Washington, DC.
Gans, Joshua S. 2005. "Markets for Ownership." *RAND J. Econ.* 36 (2): 433–45.
———. 2017. "Negotiating for the Market." In *Entrepreneurship, Innovation, and Platforms*, edited by Jeffrey Furman, Annabelle Gawer, Brian S. Silverman, and Scott Stern, 3–35. Bingley, UK: Emerald.
Gans, Joshua S., and Scott Stern. 2000. "Incumbency and R&D Incentives: Licensing the Gale of Creative Destruction." *J. Econ. and Management Strategy* 9 (4): 485–511.
Garattini, Silvio. 1997. "Are Me-Too Drugs Justified?" *J. Nephrology* 10 (6): 283–94.
Ghili, Soheil, and Matt Schmitt. 2017. "A Framework for Estimating Damages in Reverse Payment Cases." *Antitrust Law J.* 81 (3): 873–901.
Gilbert, Richard. 2018. "Mergers and R&D Diversity: How Much Competition is Enough?" Working paper, Dept. Econ., Univ. California, Berkeley.
Gilbert, Richard J., and David M. G. Newbery. 1982. "Preemptive Patenting and the Persistence of Monopoly." *A.E.R.* 72 (3): 514–26.
Gompers, Paul, William Gornall, Steven N. Kaplan, and Ilya A. Strebulaev. 2016. "How Do Venture Capitalists Make Decisions?" Working Paper no. 22587 (September), NBER, Cambridge, MA.
Gowrisankaran, Gautam. 1999. "A Dynamic Model of Endogenous Horizontal Mergers." *RAND J. Econ.* 30 (1): 56–83.
Grabowski, Henry G., and John M. Vernon. 1992. "Brand Loyalty, Entry, and Price Competition in Pharmaceuticals after the 1984 Drug Act." *J. Law and Econ.* 35 (2): 331–50.

Grossman, Sanford J., and Oliver D. Hart. 1986. "The Costs and Benefits of Ownership: A Theory of Vertical and Lateral Integration." *J.P.E.* 94 (4): 691–719.

Guadalupe, Maria, Olga Kuzmina, and Catherine Thomas. 2012. "Innovation and Foreign Ownership." *A.E.R.* 102 (7): 3594–627.

Guedj, Ilan, and David Scharfstein. 2004. "Organizational Scope and Investment: Evidence from the Drug Development Strategies and Performance of Biopharmaceutical Firms." Working Paper no. 10933 (November), NBER, Cambridge, MA.

Hart, Oliver, and John Moore. 1990. "Property Rights and the Nature of the Firm." *J.P.E.* 95 (4): 1119–58.

Hartman, Raymond S., Keith M. Drake, and Thomas G. McGuire. 2019. "Event Study Analysis in Cases with Multiple Brand-Generic Reverse-Payment Settlements." *Internat. J. Econ. Bus.* 26 (3): 399–410.

Haucap, Justus, Alexander Rasch, and Joel Stiebale. 2019. "How Mergers Affect Innovation: Theory and Evidence." *Internat. J. Indus. Org.* 63:283–325.

Helland, Eric, and Seth Seabury. 2016. "Are Settlements in Patent Litigation Collusive? Evidence from Paragraph IV Challenges." Working Paper 22194 (April), NBER, Cambridge, MA.

Hemphill, C. Scott. 2006. "Paying for Delay: Pharmaceutical Patent Settlement As a Regulatory Design Problem." *New York Univ. Law Rev.* 81 (5): 1553–623.

Henderson, Rebecca. 1993. "Underinvestment and Incompetence As Responses to Radical Innovation: Evidence from the Photolithographic Alignment Equipment Industry." *RAND J. Econ.* 24 (2): 248–70.

Henderson, Rebecca, and Iain Cockburn. 1996. "Scale, Scope, and Spillovers: The Determinants of Research Productivity in Drug Discovery." *RAND J. Econ.* 27 (1): 32–59.

Holmes, Thomas J., David K. Levine, and James A. Schmitz. 2012. "Monopoly and the Incentive to Innovate When Adoption Involves Switchover Disruptions." *American Econ. J.: Microeconomics* 4 (3): 1–33.

Howard, David H., Peter B. Bach, Ernst R. Berndt, and Rena M. Conti. 2015. "Pricing in the Market for Anticancer Drugs." *J. Econ. Perspectives* 29 (1): 139–62.

Igami, Mitsuru. 2017. "Estimating the Innovator's Dilemma: Structural Analysis of Creative Destruction in the Hard Disk Drive Industry, 1981–1998." *J.P.E.* 125 (3): 798–847.

Kamepalli, Sai Krishna, Raghuram Rajan, and Luigi Zingales. 2020. "Kill Zone." Working Paper no. 2020-19, Becker Friedman Inst., Chicago.

Katz, Michael L., and Carl Shapiro. 1985. "On the Licensing of Innovations." *RAND J. Econ.* 16 (4): 504–20.

Kim, J. Daniel. 2018. "Predictable Exodus: Startup Acquisitions and Employee Departures." Working paper, Wharton School Bus.

Krieger, Joshua, Danielle Li, and Dimitris Papanikolaou. 2017. "Developing Novel Drugs." Working Paper no. 18-056, Harvard Bus. School.

Krieger, Joshua L. 2017. "Trials and Terminations: Learning from Competitors' R&D Failures." Working Paper no. 18-043, Harvard Bus. School.

Kyle, Margaret K. 2007. "Pharmaceutical Price Controls and Entry Strategies." *Rev. Econ. and Statis.* 89 (1): 88–99.

Lacetera, Nicola, Iain M. Cockburn, and Rebecca Henderson. 2004. "Do Firms Change Capabilities by Hiring New People? A Study of the Adoption of Science-Based Drug Discovery." In *Business Strategy over the Industry Life Cycle*, edited by Joel A. C. Baum and Anita M. McGahan, 133–60. Bingley, UK: Emerald.

Lai, Ronald, Alexander D'Amour, and Lee Fleming. 2009. "The Careers and Co-Authorship Networks of US Patent-Holders, since 1975." Working paper, Harvard Univ.

Lakdawalla, Darius N. 2018. "Economics of the Pharmaceutical Industry." *J. Econ. Literature* 56 (2): 397–449.

Lemley, Mark A., and Carl Shapiro. 2005. "Probabilistic Patents." *J. Econ. Perspectives* 19 (2): 75–98.

Lerner, Josh, and Robert P. Merges. 1998. "The Control of Technology Alliances: An Empirical Analysis of the Biotechnology Industry." *J. Indus. Econ.* 46 (2): 125–56.

Letina, Igor, Armin Schmutzler, and Regina Seibel. 2020. "Killer Acquisitions and Beyond: Policy Effects on Innovation Strategies." Discussion Paper no. 2003, Dept. Econ., Univ. Bern.

Maksimovic, Vojislav, and Gordon M. Phillips. 2001. "The Market for Corporate Assets: Who Engages in Mergers and Asset Sales and Are There Efficiency Gains?" *J. Finance* 56 (6): 2019–65.

Mankiw, N. Gregory, and Michael Whinston. 1986. "Free Entry and Social Inefficiency." *RAND J. Econ.* 17 (1): 48–58.

McFadden, Daniel. 1974. "Conditional Logit Analysis of Qualitative Choice Behavior." In *Frontiers in Econometrics*, edited by Paul Zarembka, 105–42. New York: Academic Press

Meder, Hendrik. 2019. "Post-M&A Synergies and Changes in Development Trajectory of Pharmaceutical Firms." Working paper.

Morck, Randall, Andrei Shleifer, and Robert W. Vishny. 1990. "Do Managerial Objectives Drive Bad Acquisitions?" *J. Finance* 45 (1): 31–48.

Motta, Massimo, and Emanuele Tarantino. 2017. "The Effect of Horizontal Mergers: When Firms Compete in Prices and Investments." Working Paper no. 1579, Dept. Econ. and Bus., Univ. Pompeu Fabra.

NCSES (National Center for Science and Engineering Statistics). 2018. "Business R&D and Innovation: 2014. Detailed Statistical Tables." Technical Report no. NSF 18-302, National Science Foundation, Alexandria, VA.

Nikolova, Nina, and Joanna Jaworska. 2003. "Approaches to Measure Chemical Similarity: A Review." *Molecular Informatics* 22 (9–10): 1006–26.

Ornaghi, Carmine. 2009. "Mergers and Innovation in Big Pharma." *Internat. J. Indus. Org.* 27 (1): 70–79.

Ouimet, Paige Parker, and Rebecca Zarutskie. 2011. "Acquiring Labor." Working Paper no. CES-WP-11-32, Center Econ. Studies, US Census Bur., Washington, DC.

Phillips, Gordon, and Alexei Zhdanov. 2013. "R&D and the Incentives from Merger and Acquisition Activity." *Rev. Financial Studies* 26 (1): 34–78.

Rasmusen, Eric. 1988. "Entry for Buyout." *J. Indus. Econ.* 36 (3): 281–99.

Reiffen, David, and Michael Ward. 2005. "Generic Drug Industry Dynamics." *Rev. Econ. and Statis.* 87 (1): 37–49.

Roll, Richard. 1986. "The Hubris Hypothesis of Corporate Takeovers." *J. Bus.* 59 (2): 197–216.

Salant, Stephen, Sheldon Switzer, and Robert Reynolds. 1983. "Losses from Horizontal Merger: The Effects of an Exogenous Change in Industry Structure on Cournot-Nash Equilibrium." *Q.J.E.* 98 (2): 185–99.

Schmutzler, Armin. 2013. "Competition and Investment—A Unified Approach." *Internat. J. Indus. Org.* 31 (5): 477–87.

Schumpeter, Joseph Alois. 1942. *Socialism, Capitalism and Democracy*. New York: Harper.

Scott Morton, Fiona. 2000. "Barriers to Entry, Brand Advertising, and Generic Entry in the US Pharmaceutical Industry." *Internat. J. Indus. Org.* 18 (7): 1085–104.
Segal, Ilya, and Michael Whinston. 2007. "Antitrust in Innovative Industries." *A.E.R.* 97 (5): 1703–30.
Seru, Amit. 2014. "Firm Boundaries Matter: Evidence from Conglomerates and R&D Activity." *J. Financial Econ.* 111 (2): 381–405.
Shapiro, Carl. 2003. "Antitrust Limits to Patent Settlements." *RAND J. Econ.* 34 (2): 391–411.
Takebe, Tohru, Ryoka Imai, and Shunsuke Ono. 2018. "The Current Status of Drug Discovery and Development as Originated in United States Academia: The Influence of Industrial and Academic Collaboration on Drug Discovery and Development." *Clinical and Translational Sci.* 11 (6): 597–606.
Teece, David J. 1986. "Profiting from Technological Innovation: Implications for Integration, Collaboration, Licensing and Public Policy." *Res. Policy* 15 (6): 285–305.
US FDA (Food and Drug Administration). 2017. "The Drug Development Process." https://www.fda.gov/patients/learn-about-drug-and-device-approvals/drug-development-process.
Wollmann, Thomas G. 2019. "Stealth Consolidation: Evidence from an Amendment to the Hart-Scott-Rodino Act." *A.E.R. Insights* 1 (1): 77–94.
Yin, Wesley. 2008. "Market Incentives and Pharmaceutical Innovation." *J. Health Econ.* 27 (4): 1060–77.
———. 2009. "R&D Policy, Agency Costs, and Innovation in Personalized Medicine." *J. Health Econ.* 28 (5): 950–62.