

Killer Acquisitions*

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This paper argues incumbent firms may acquire innovative targets solely to discontinue the target’s innovation projects and preempt future competition. We call such acquisitions “killer acquisitions.” We develop a model illustrating this phenomenon. Using pharmaceutical industry data, we show that acquired drug projects are less likely to be developed when they overlap with the acquirer’s existing product portfolio, especially when the acquirer’s market power is large due to weak competition or distant patent expiration. Conservative estimates indicate 5.3 percent to 7.4 percent of acquisitions in our sample are killer acquisitions. These acquisitions disproportionately occur just below thresholds for antitrust scrutiny.

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1. Introduction

Innovation drives economic growth and firm profitability. Innovating firms are often acquired by incumbents, typically in the early stages of product development. Economists traditionally view this positively: firms 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

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

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

²The 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 (LVAD), 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 US LVAD market. By acquiring Heartware, Thoratec willfully seeks to maintain its LVAD monopoly, thereby denying patients the potentially life-saving benefits of competition between Thoratec and HeartWare” (FTC Administrative Complaint Docket No. 9339: <https://www.ftc.gov/sites/default/files/documents/cases/2009/07/090730thorateadminccmpt.pdf>).

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). 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 the entrepreneur, 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 than 16,000 drug projects originated by more than 4,000 companies in the past two-and-a-half decades and

³Research and development (R&D) intensity in the pharmaceutical industry is second only to semiconductors in the US manufacturing sector, at 11.3 percent in 2014 (US NSF, NCSSES, 2018).

follow each drug from initiation. We collect relevant acquisition events from comprehensive data sources. Importantly, we observe development milestones of drug projects independent of project ownership, meaning we can follow the same projects pre- and post-acquisition.⁴

To finely categorize acquirer overlap with the target’s project, and thus identify potentially competing products, we use pharmaceutical categories based on 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 we are capturing potential substitutes (i.e., companies developing drugs that, if successful, would directly compete with the acquirer).

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

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

of our baseline results. Most importantly, we find decreased development post-acquisition for overlapping acquired projects is driven by drugs that have no further development activity post-acquisition (i.e., by immediate and permanent terminations). Projects acquired by overlapping acquirers are 20.9 percent more likely to cease development immediately compared to those acquired by non-overlapping 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 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

⁵In 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.

there is no such pattern for non-overlapping acquisitions. In addition, these below-threshold deals exhibit much higher termination rates and much lower launch rates.

We employ several additional tests to address potential alternative explanations for lower 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 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—i.e., 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 post-acquisition projects of the acquirer, finding no evidence supporting the idea that acquired technologies are integrated into acquirers' new drug development projects. We also do not find that acquirers are more likely to cite acquired and terminated projects' patents. To explore human capital redeployment, we examine inventor mobility and productivity around the acquisition events. We show that only 22 percent of inventors from target firms eventually work for the acquiring firm and further that those inventors do not become more productive post-acquisition. These results are inconsistent with explanations regarding technology or human capital redeployment.

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 post-acquisition development activity. Contrary to the salvage explanation, however, we find no evidence that overlapping acquisitions have either pre-acquisition declines in development or lower valuations, on average, compared to non-overlapping acquisitions.

Our conservative estimates indicate that between 5.3 percent and 7.4 percent of all acquisitions in our sample (or about 46 to 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 percent. 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 non-overlapping acquisitions, which are only motivated by development intentions. We find an increase in acquisition probability and a decrease in post-acquisition 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

⁶Protective 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 (2019a,b) specifically analyze the role of antitrust limits on start-up acquisitions by dominant firms.

(Roll, 1986; Morck et al., 1990), synergies (Bena and Li, 2014; Maksimovic and Phillips, 2001), and increasing existing market power (Baker and Bresnahan, 1985). This paper adds to this literature in two ways. First, in our model, acquisitions are not driven by synergies or by incentives to increase current market power. Instead, we argue that incumbents acquire innovative targets to terminate nascent innovation that may threaten their profits in the future. This new mechanism combines two classic effects in the innovation literature: the “replacement effect” (Arrow, 1962), 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. Federico et al. (2017, 2018), Motta and Tarantino (2017), Cabral (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 et al. (2019) empirically document an innovation-reducing effect of mergers, whereas Guadalupe et al. (2012) find that a target company’s innovation increases following an acquisition by a multinational firm. Our paper provides a theoretical and empirical analysis of a new channel through which acquisitions impact innovation. By using detailed project-level data on acquisition and development decisions, we 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 tradeoffs involved in promoting competition while fostering innovation, through investigating the product market interactions between patented and generic drugs (Caves et al., 1991; Grabowski and Vernon, 1992; Scott Morton, 2000; 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

⁷Henderson (1993) and Igami (2017) empirically show that such cannibalization makes incumbents reluctant to innovate in the photolithographic alignment equipment and the hard disk drive manufacturing industries. More broadly, the slow response to new technologies by incumbent firms is explored in the large literature on competition and innovation. See Holmes et al. (2012) and Schmutzler (2013) for unified theoretical treatments and Cohen (2010) for a comprehensive survey.

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

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 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 meets the needs of some consumers more effectively.¹² We also allow for differential capabilities in project development. If $\rho^A > \rho^E$, 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 (Berndt et al., 2011; Ellison et al., 1997; Duggan and Scott Morton, 2010).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.

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

Consider first the product market choices of the entrepreneur when her project is not acquired ($\neg acq$). If the project is successful (S), the resulting newly-developed product competes against n other single-product incumbent firms, and the entrepreneur maximizes $p^E q^E$. This

¹²Although 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.

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

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

yields profits for the entrepreneur and the potential acquirer and all of the other incumbents) such that $\pi_{-acq,S}^E \geq \pi_{-acq,S}^A > 0$ 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 $\pi_{-acq,F}^E = 0$. The n incumbent firms each have a single existing (horizontally differentiated) product to sell, and thus the acquirer's profit is equal to $\pi_{-acq,F}^A > 0$. Profits for the acquirer are now higher, $\pi_{-acq,F}^A > \pi_{-acq,S}^A$, for two reasons. First, the acquirer only has to compete against $n - 1$ (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 $\pi_{acq,F}^A > 0$. This is exactly the same as when no acquisition occurs and the entrepreneur's project fails; hence $\pi_{acq,F}^A = \pi_{-acq,F}^A$.

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, $p_{old}^A q_{old}^A + p_{new}^A q_{new}^A$, whereas the remaining $n - 1$ other single-product incumbent firms maximize single-product profits. The profit of the multiproduct incumbent acquirer is $\pi_{acq,S}^A$. This profit is higher than when he sells only a single product with the same $n - 1$ competitors; hence $\pi_{acq,S}^A > \pi_{-acq,F}^A$.

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

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

2.3. Development Decision ($t = 1$)

2.3.1. 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 $\pi_{acq,S}^A$ and $\pi_{acq,F}^A$ for the incumbent and the difference between $\pi_{-acq,S}^E$ and $\pi_{-acq,F}^E$ for the entrepreneur. As long

as the acquirer's existing product and the new project are imperfect substitutes, we have

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

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

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

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

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

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 $\frac{\Delta^E}{\Delta^A} > \frac{\rho^A}{\rho^E}$.*

Thus, whether the entrepreneur or the acquirer has stronger incentives to continue development depends on the relative magnitudes of the replacement effect $\frac{\Delta^E}{\Delta^A}$ and the synergy effect $\frac{\rho^A}{\rho^E}$.

¹⁵If 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.

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

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

Proposition 2 (Project Development and Competition). *For any 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 only from his own existing product) than when he already faces many other existing competitors (i.e., cannibalization losses are spread over many firms). As a result, as the number of existing competitors increases, the replacement effect decreases and the acquirer's development decisions become more similar to those of the entrepreneur.

2.3.3. Patent Life and Future Competition. Until now, we have only considered 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 by T^E and those of the acquiring incumbent's existing product by T^A where $T^E > T^A \geq 0$. We assume, for simplicity, that the firms earn their static game profits every year 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 et al., 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 . Thereafter, the profits for the acquirer’s existing product drop to zero due to 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 $\Delta_{gen} = \Delta_{gen}^E = \Delta_{gen}^A$.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., $\frac{\rho^A}{\rho^E} \leq \frac{\Delta^E - \Delta_{gen}}{\Delta^A - \Delta_{gen}}$). 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 of 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

¹⁶This 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, due to its low price, also reduces the profits of the other products that are differentiated from it.

¹⁷Unlike 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 only reduces the time during which the new product can earn positive profits arising from patent protection.

¹⁸This 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

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 .

2.4. Acquisition Decision ($t=0$)

2.4.1. Single Incumbent Acquirer. We now show that killer acquisitions can only occur when the entrepreneur's project overlaps with the acquirer's existing product.

To compensate the entrepreneur for selling the project, the acquirer must pay an endogenously determined takeover price P equal to (or greater than) the expected payoff of the project when the entrepreneur remains independent.¹⁹ Recall that for any product market overlap, we have $k^E > k^A$ if and only if $\frac{\Delta^E}{\Delta^A} > \frac{\rho^A}{\rho^E}$. 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

$$\underbrace{\rho^E (\pi_{acq,F}^A - \pi_{-acq,S}^A)}_{\text{efficiency effect}} \geq \underbrace{\rho^E \Delta^E - k - L}_{\text{replacement effect}}. \quad (5)$$

Acquiring and shutting down the entrepreneur's project yields a profit equal to $\pi_{acq,F}^A$ while not acquiring it yields $\pi_{-acq,S}^A$. 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.

competitors (Ellison et al., 1997).

¹⁹This 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 Section 5, in a more general model, the existence of the acquisition exit option may be valuable enough to increase ex-ante innovation incentives.

Third, for $k \leq k^A$, both acquired and non-acquired firms develop the project. Such an acquisition (“Acquire to Continue”) occurs if

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

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 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 $\frac{\Delta^E}{\Delta^A} \leq \frac{\rho^A}{\rho^E}$, 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, $\frac{\Delta^E}{\Delta^A} < \frac{\rho^A}{\rho^E}$. 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

²⁰Under 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.

²¹Although the acquirer only has a strictly positive incentive to acquire the entrepreneur when project development is sufficiently profitable ($k \leq k^E$ so $\rho \Delta^E - k$ is positive), the acquirer has a weaker incentive to develop than the entrepreneur. This is because whenever the acquirer has a strictly positive incentive to acquire, the entrepreneur always develops any project she retains, whereas the acquirer only ends up developing a subset of his acquired projects ($k \leq k^A$).

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.

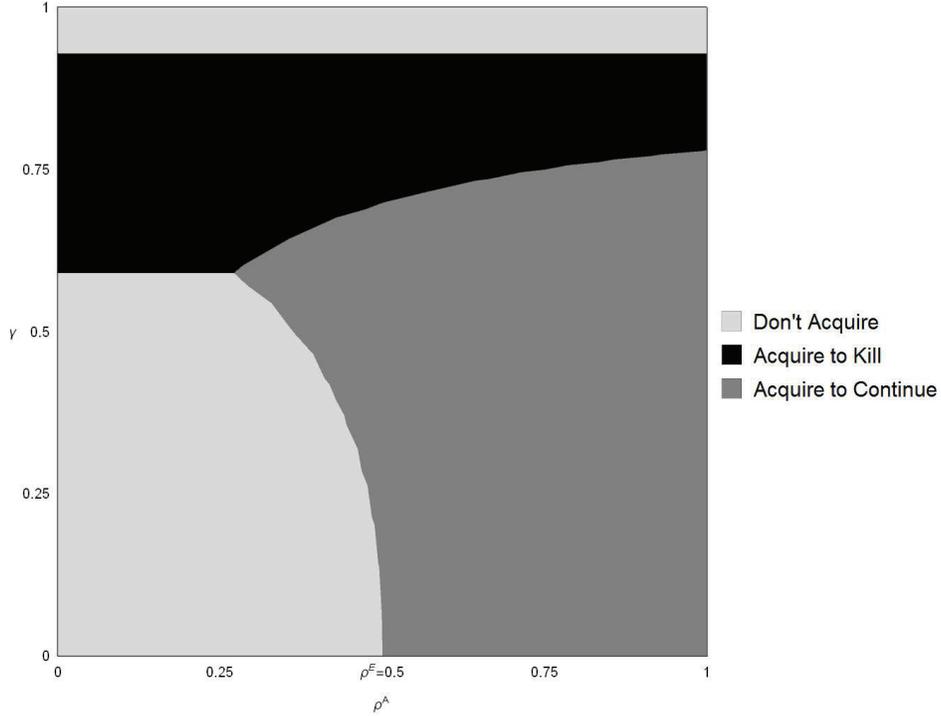
Figure 2 illustrates the two forces driving acquisitions. It plots the acquirer’s optimal acquisition strategies as a function of his development capability (ρ^A) and the degree of product overlap (γ). First, when both ρ^A and γ are low, the incumbent chooses not to acquire. If he acquires and continues 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.

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

2.4.2. Multiple Incumbent Acquirers. Our analysis so far made two simplifying assumptions about the acquisition process. First, we assumed that only one of the incumbents is 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 all have the same project development capabilities. In Appendix A.3 we relax these

²²Figure 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 non-acquisition result is closely related to the “Cournot merger paradox” (Salant et al., 1983).

Figure 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 a function 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 $n = 2$.

assumptions. Here we sketch out the main implications of allowing for multiple (asymmetric) incumbent acquirers.

First, when several of the n incumbents²³ 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 only acquired with some probability because the potential acquirers try to free ride on the privately costly decision to acquire the entrepreneur. Just like in the volunteer’s dilemma, when the number

²³Outside 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.

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.

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 (i) following an acquisition, overlapping drug projects should be less likely to be developed; when (ii) existing or (iii) future competition is low, this difference in development choices between overlapping acquired drugs and their non-overlapping acquired or non-acquired counterparts should be more pronounced; and (iv) acquisitions by incumbents should target entrepreneurial firms developing drug projects that overlap with the incumbent’s existing drugs.

3. Background and Data

To empirically document the phenomenon of killer acquisitions, we use the setting of drug development. Testing the predictions of our theoretical framework requires comprehensive

data on project-level outcomes for both acquired and non-acquired projects. We also need to measure overlap between acquirer and target firms, and market and technological competition. As described in detail below, pharmaceutical project development offers these features. Further, the pharmaceutical industry represents a significant and growing amount of healthcare spending, innovative activity, and M&A transactions. It is an economically and socially important industry of ongoing interest to economists (see [Lakdawalla \(2018\)](#) for a summary).

3.1. Drug Development Background

The development of innovative pharmaceutical products, often known as branded or patented drugs, involves a standard set of structured milestones en route to commercialization. First, firms identify potential drug compounds through routine discovery processes. Then, for any promising compounds, firms run preliminary screening in vitro and/or in vivo to explore both efficacy and toxicity before any clinical trials in humans. Following 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 US and abroad, applications for coverage to various public and private insurance agencies, and launching and marketing the product in various countries around the world. Given the lengthy process prior to FDA approval and marketing, patented drugs usually have only a few years post-approval of monopoly profits before patent expiration and generic entry.

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.

²⁴Drug developers must submit an Investigation New Drug (IND) 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.

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

3.2. Drug Development Data

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

[Insert TABLE 1 Here.]

Our sample covers projects initiated between 1989 and 2010, with a focus on projects for which we observe some active development after initiation, or 16,015 projects originated by 4,637 firms.²⁸ Pharmaprojects data start from 1989, and we exclude projects initiated in 2011 or after

²⁶In the raw dataset, 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 dataset. We provide more details in Appendix B.

²⁷Full details are in Appendix B.

²⁸If 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.

to ensure 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 percent of the sample). More than half of companies originate only one drug over this period and 70 percent originate two or fewer (see figure F1), 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).

3.3. 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 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 datasets).

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 pre- and post-acquisition. The merged drug development and acquisition data show active acquisition activities in our analytical sample, with 22 percent 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).

4. Empirical Analysis

4.1. 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, non-overlapping acquired, and non-acquired projects.

We measure overlap between a drug project and the acquiring firm based on a combination of its intended therapeutic class and mechanism of action. The therapeutic class (TC) is the disease or condition the therapy targets (e.g., hypertension). We use Pharmaprojects’s therapeutic categories, which are based on 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 that of 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. 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 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 entry on branded products define competing products as those that are the same chemical entity (Ellison and Ellison, 2011; Arcidiacono et al., 2013; Branstetter et al., 2014). Since we are comparing the development of potentially competing innovative pharmaceuticals, which by definition must be different chemical entities, we cannot use this as our measure.

Second, prior research exploring market competition between branded products has defined overlap as 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, for example, 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. Last, 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:

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

where the dependent variable $Development_{i,t}$ is a dummy variable indicating whether drug i has a development event in year t . $I(Acquired)_i$ indicates whether drug i ever undergoes an acquisition event, and $I(Post)_{i,t}$ indicates whether the drug-year (i,t) observation is after the

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

drug is acquired. $I(Overlap)_i$ 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 (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 $I(Acquired)_i \times I(Post)_{i,t}$ captures the change in development activity for all acquired drug projects in the years after the acquisition. The term $I(Acquired)_i \times I(Overlap)_i$ captures the overall development conditions for drugs acquired by overlapping buyers in the years before the acquisition. The key term for our test is the triple interaction term $I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i$, which captures the additional change in development event probability for acquisition cases when the target and the acquirer overlap. Our model predicts a negative coefficient, β , consistent with the prediction that when acquired projects overlap with the acquirer’s portfolio, they are more likely to be terminated.³⁰

Ideally, if terminations were comprehensively reported in a timely manner, we would use a survival analysis to test if and when drugs projects are shut down. However, project terminations are rarely observed 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 non-acquired projects and pre-acquisition differences between overlapping and non-overlapping acquired projects. To investigate whether we are accurately capturing

³⁰It 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 non-acquired or non-overlapping acquired ones. This is because only drug candidates that are particularly valuable to an acquirer (e.g., due to synergies resulting from prior experience of developing similar drugs) should be acquired. Thus, in the absence of strategic killing motives, the triple interaction term $I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i$ should be positive. In contrast, our theory of strategic acquisition and discontinuation of overlapping projects predicts that this interaction term should be negative.

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

drug terminations with our project-year analysis, we run additional analyses predicting any post-acquisition development activity (described in detail below).

The following subsections detail our empirical analyses. First, we compare drug development rates for non-acquired, acquired non-overlapping, and acquired overlapping projects (table 2). 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 effect 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 antitrust review thresholds (table 7 and figure 3). Finally, we investigate several alternative explanations (tables 8 and 9).

4.2. Development of Drug Projects Post-Acquisition

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

[Insert TABLE 2 Here.]

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 timelines for pharmaceuticals (US FDA, 2017). In column 1, the estimate of β is -0.037 and statistically significant at the 1 percent 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 post-acquisition compared to non-overlapping acquired projects, and they are 5.7 percentage points ($=0.037+0.020$) less likely to experience a development event compared to non-acquired projects. Given that the unconditional probability of having a development event is 19.9 percent, being acquired by a firm with an overlapping

project is associated with a 20.7 percent ($=0.037/(0.199 - 0.020)$) lower development probability compared to otherwise similar drugs that are acquired by a non-overlapping 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 (US HHS ASPE, 2014). For example, Budish et al. (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 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, $I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i$ estimates the difference in development between acquired overlapping and non-overlapping projects for the same originator (or target). These results show that even within the same originator firm, acquired overlapping projects are significantly less likely to have a post-acquisition development event compared to acquired non-overlapping 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 column 1: being acquired by a firm with an overlapping project is associated with a 23.4 percent 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 if there are any differences in drug development *trajectories* across projects acquired by overlapping versus non-overlapping incumbents. Given that we find decreased development post-acquisition for overlapping acquired projects, one concern is that such projects were on a slower (or faster) development path pre-acquisition compared to other acquired projects, and such pre-acquisition differences at least partially explain post-acquisition differences. To investigate this, we include indicator variables for the three years before the acquisition separating overlapping and non-overlapping acquired. The associated estimated coefficients are insignificant, suggesting that different development trajectories are not driving our results.

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 pre-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 post-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 non-overlapping 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 the omitted category of non-overlapping acquired projects now is consistently insignificant, albeit still negative (Appendix table F1).³⁴

³²Figure F2 shows similar propensity distributions for acquired and non-acquired projects post-weighting.

³³The 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).

³⁴The prior literature does not provide a clear expectation for the sign of this coefficient. If we were to assume

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 (Appendix 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 (Appendix table F3); using age-TC fixed effects instead of more narrow age-TC-MOA fixed effects to control for differences in drug development life cycles (Appendix table F4); and controlling for any pre-acquisition 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 (Appendix 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 which replicate the table 2 results (Appendix E).

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

[Insert TABLE 3 Here.]

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 percent, with a 39.6 percent lower development rate for overlapping targets). In column 2, we examine post-acquisition 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

that projects are only acquired if they are of high quality and development synergies resulting from economies of scale and scope materialize post-acquisition (Andrade et al., 2001; Henderson and Cockburn, 1996), we would expect a positive coefficient. However, a negative coefficient is consistent with faster termination and lower development rates of larger (acquiring) firms due to the absence of private benefits from continuing development (Guedj and Scharfstein, 2004) or due to agency problems inherent in the organization of large firms (Seru, 2014).

of overlap on post-acquisition development to be smaller and/or insignificant in this sample. However, we find it is both significant and larger in magnitude, alleviating these concerns.

Our second set of additional analyses aim to ensure our main results on the decreased likelihood of development are due to project termination rather than due 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 post-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 non-overlapping projects after we take out projects that are never developed post-acquisition. Second, in column 4, we examine the likelihood that a project is never developed post-acquisition, which we expect to be significantly higher for overlapping acquisitions. For this second test, we collapse our panel into two time periods, pre- and post-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 non-overlapping projects after removing the never-developed projects. Second, in column 4, we find that overlapping projects are 14.9 percentage points, or 20.9 percent ($= \frac{0.149}{0.711}$), more likely to have no post-acquisition development events (i.e., to be immediately terminated) compared to non-overlapping 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 non-acquired firms. We also investigate this possibility directly (in Appendix table F7), and find no evidence of increased development by non-acquired firms.

4.4. 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

³⁵In addition to focusing on using never-developed projects to capture immediate termination, if we look at post-acquisition development, we find the rate of having just one more post-acquisition development event is 17.0 percent for overlapping acquired projects compared to 21.8 percent for non-overlapping 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 non-overlapping acquired projects is smaller in this analysis than the never-developed-again analysis.

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 across markets that could lead to both lower competition and lower development rates. Table 4 presents the regression results that examine whether the post-acquisition development pattern of acquired projects varies under different competition environments. We categorize drug development projects into high and low competition by the sample median (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 post-acquisition across different levels of competition.

[Insert TABLE 4 Here.]

The results suggest that the decreased likelihood of development of overlapping projects during the post-acquisition period concentrates in product markets with relatively low competition. Comparing columns 1 and 2, we observe that 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 unreported coefficient -0.015 associated with $I(Acquired) \times I(Post)$, and benchmark of the unconditional development rate in the subsample (19.4 percent), implies that acquired overlapping projects are 41.2 percent ($= \frac{0.065+0.015}{0.194}$) less likely to be developed than non-acquired drugs and are 36.3 percent ($= \frac{0.065}{0.194-0.015}$) less likely to be developed than acquired non-overlapping 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 (Appendix E) for both product market and pipeline competition.

³⁶Each drug product can fall into multiple technologies (MOAs) and multiple intended markets (TCs). In the Pharmaprojects dataset, drug projects have, on average, 1.3 mechanisms of action (median 1; 81 percent have 1) and, on average, 1.9 TCs (median 2; 46 percent 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 0 competitors and one with 5, we use 5.

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

4.5. 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 the United States Patent and Trademark Office (USPTO) data.

[Insert TABLE 5 Here.]

Table 5 presents these results. The key coefficient is for $I(Post) \times I(Near Patent Expiry)$, which contrasts those with patents near expiration (i.e., within five years) with those with longer remaining patent life. Consistent with our predictions, we find that 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 post-acquisition compared to projects for whom 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 column 2), suggesting that development life cycles vary across TC-MOAs in ways that impact how salient the remaining patent life is 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).

4.6. Acquisition Decisions

4.6.1. Determinants of Acquisitions. Our empirical analysis so far has focused on drug development, finding that (i) a project is less likely to be developed after being acquired by a firm with an overlapping existing drug (consistent with Proposition 1), (ii) these results are concentrated in markets with low levels of competition (Proposition 2), and (iii) 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 pseudo deals). First, we form a random control sample: for each pair of acquirer firm j and target drug i , we randomly draw five firms from the pool of firms that have ever performed an acquisition before the deal year. For each of those pseudo acquirers, we then form pseudo acquisitions with target project i . Second, we form a size-matched control sample: we match each acquirer in each deal to five control firms based on total number of drug projects in the year of the deal.

The analysis is performed using the following model:

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

The dependent variable, $Acquirer-Target_{ijd,t}$, is equal to one if firm-project pair ij is a real acquirer-target pair and is zero otherwise (i.e., a pseudo pair). The key explanatory variable $I(Overlap)$ is constructed for each firm pair and captures whether firm j has any product that overlaps with the acquired project i . Fixed effects are at the deal level (indexed by d) for each real acquirer-target and its control pairs. Our goal is to examine whether overlapping projects in the target’s pipeline drive the acquirer’s purchase decision.

[Insert TABLE 6 Here.]

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 more 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 percent. 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 drug development results. First, in contrast to our empirical finding of acquisitions of overlapping targets, acquisition motives based on empire building or managerial risk diversification theories would imply that incumbent acquirers should target non-overlapping projects. Second, although our results showing that overlap predicts acquisitions are, on their own, consistent with acquisitions motivated by project development synergies, such a synergy-based theory would predict that acquired overlapping projects are subsequently more likely to be developed rather than less. Hence, a synergy motive contrasts sharply with our empirical findings of decreased development in Section 4.2.

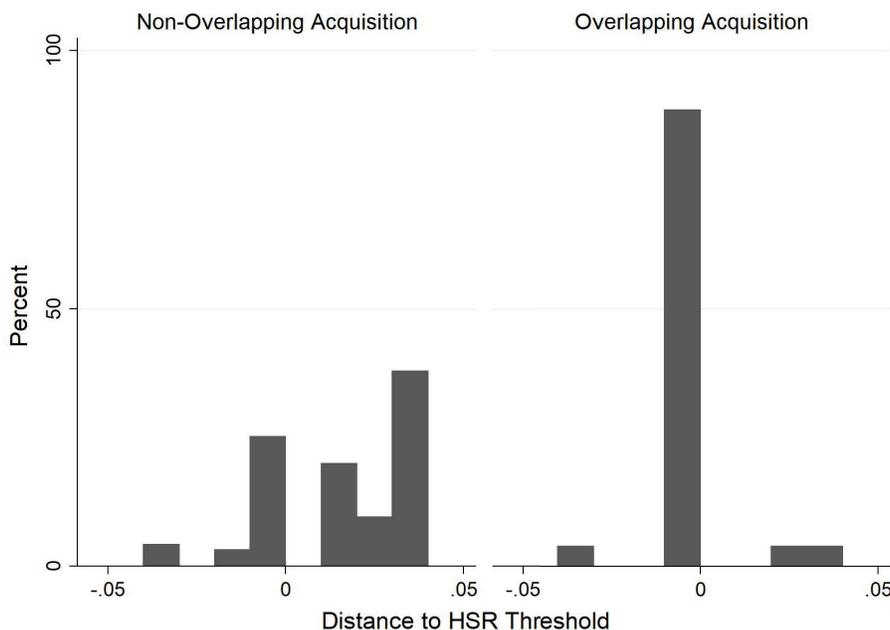
4.6.2. Antitrust and FTC Review Thresholds. In the pharmaceutical industry incumbents often conduct acquisitions when the target’s technology or project is still at a nascent stage. As a result, some of these deals are exempted from the FTC’s premerger review rule under the Hart-Scott-Rodino (HSR) Antitrust Improvements Act 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 and below the threshold. If incumbent

³⁸In 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 need to be reviewed by the FTC. Because in the pharmaceutical industry the size-of-the-person test is typically not satisfied for the smaller (target) party, acquisitions below \$200 million will usually not be investigated. [Wollmann \(2019\)](#) 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.

firms conduct killer acquisitions 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.

Figure 3. Acquisition Size Distributions Around HSR Review Threshold



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

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

[Insert TABLE 7 Here.]

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 threshold. We find that the eventual product launch rate is much lower (1.8 percent versus 9.1 percent) and the discontinuation rate is much higher (94.6 percent versus 83.3 percent) 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 “pseudo threshold.” To compare with the real HSR threshold (an annually adjusted value set using a base of \$200 million in 2000), we construct a pseudo threshold (2000 base value of \$150 million). This pseudo threshold is comparable in value to the real threshold but is sufficiently different such that we should not observe strategic behavior in its vicinity. The results of this analysis (lower panel 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 pseudo threshold we choose and are therefore consistent with the idea that acquirers conduct more killer acquisitions in situations in which they can expect to avoid FTC scrutiny.

5. Discussion

5.1. Alternative Explanations

In this section we address several potential alternative explanations 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 non-acquired or non-overlapping acquired drug projects.

5.1.1. Informational Asymmetries in the Acquisition Market. Focusing on overlapping acquired projects means that asymmetric information, or “market for lemons” type arguments, are an implausible candidate explanation. Although an acquiring firm likely knows less than the target about the quality of the target’s projects and may therefore sometimes buy

lemons, this asymmetry should be lower when the acquirer has its own overlapping projects and therefore has knowledge of both the underlying science and the eventual market of the drug candidate. Our main results are therefore unlikely to be caused by informational asymmetries.

5.1.2. 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.

5.1.3. 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 those developed by the acquirer pre- and post-acquisition, and assess whether acquirer firms’ projects initiated post-acquisition are more similar to the acquired project than their pre-acquisition drugs (consistent with technology redeployment). To measure similarity, we follow recent research in economics by [Krieger et al. \(2017\)](#) 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](#)).

[Insert TABLE 8 Here.]

Table 8, panel A presents chemical similarities to the acquired drug for drugs initiated by the acquirer post-acquisition compared to pre-acquisition drugs. In columns 1–3, each observation is a pair consisting of an acquired drug and a drug that was initiated by the acquirer within

the ten-year window (i.e., \pm five 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 pre-acquisition projects. The economic magnitude of 0.001 is also negligible compared to the global similarity mean of 0.133. Overall, these results do not support a technology redeployment explanation.

In columns 4–6, 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.

5.1.4. Redeployment of Human Capital. Our results could also be due to acquisitions motivated by human capital redeployment (Lacetera et al., 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, similar to technology redeployment, we would expect that 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 following acquisitions. To do so, we use data on disambiguated inventor names and organizational affiliations (via patent assignees) from Lai et al. (2009) to track individuals over time and across organizations following Bernstein (2015) and Brav et al. (2018). Specifically, we construct a list of pre-acquisition target firm inventors by identifying those who filed at least one patent within the five-year window before the acquisition. We then track whether target firm inventors stay with acquiring firm and if those who remain appear to be efficiently redeployed.

Table 8, panel B shows the human capital results. First, only 22 percent of pre-acquisition inventors move to the acquirer post-acquisition, while 78 percent 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 five years leading up to the acquisition, we find no evidence that the re-

tained inventors become more productive in the combined firm. In fact, their average patenting quantity drops by 30 percent from 4.57 to 3.16 patents over the five years post-acquisition. In contrast, inventors who move to other firms have a smaller productivity drop (< 10 percent).³⁹

5.1.5. 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 pre-acquisition (in table 2, column 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 non-overlapping 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 this is a selected sample of acquisitions, as it is conditional on the availability of deal value information.

[Insert TABLE 9 Here.]

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 non-overlapping acquisitions. Combined with the above-mentioned results, these results suggest that salvage acquisitions by overlapping firms are not driving our results.

³⁹One 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).

5.2. 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 percent. Using the estimates from our tightest specification reported in table 2, column 4, we find that acquired projects with overlap (22.7 percent of acquired projects) continue at an adjusted rate of 13.4 percent, while acquired projects without overlap (77.3 percent of acquired projects) continue development at an adjusted rate of 17.5 percent.

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 percent probability of development) and acquisitions that have the same development likelihood as acquisitions without overlap (17.5 percent). 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 percent)? 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 non-killer acquisitions to have the same development likelihood as non-acquired projects (19.9 percent), we estimate that 7.4 percent 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 non-overlapping 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

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

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 percent 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 share of follow-on drugs are 35 percent, 66 percent, and 84 percent, respectively. Using those numbers as an estimate of the risk set of potential killer acquisition targets, and using an estimate of 1.5 percent of all drugs—including those not at risk—as killer acquisitions,⁴¹ we estimate that 6.3 percent/ 3.3 percent/ 2.6 percent 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 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 percent. 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 percent) as non-acquired projects (47.5 percent of all projects). The number of total drug projects for which development continues would increase by 4.3 percent ($= \frac{19.9\% - 13.4\%}{18.2\%} \times (1 - 47.5\%) \times 22.7\%$) 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).

⁴¹This is computed using the above estimates of 5.3 percent to 7.4 percent of all acquired drug projects as killer acquisitions, which implies 1.3 percent to 1.8 percent, or roughly 1.5 percent of all drug projects.

We can compare them 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 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 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 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 non-zero 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.

5.3. Ex-Ante Innovation Incentives and Welfare

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

⁴²Pay-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 et al., 2015](#); [Helland and Seabury, 2016](#); [Ghili and Schmitt, 2017](#); [Hartman et al., 2019](#)).

patients suffer because there are fewer drugs and the drugs that are developed and brought to market are sold at higher prices.⁴³

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 et al. \(2020\)](#). 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 or those that do not, increased takeover activity and killer acquisitions by incumbents may spur innovation of very similar me-too drugs ([Garattini, 1997](#); [Arcidiacono et al., 2013](#)) at the expense of the origination of

⁴³Although 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 the loss in producer surplus of the existing incumbents as in [Mankiw and Whinston \(1986\)](#). In [Appendix A](#) we derive a sufficient condition under which the loss in consumer surplus resulting from killer acquisitions outweighs the producer surplus gains and thus reduces social welfare overall. As long as there are few existing incumbents and the entrepreneur’s drug project is not too similar to the 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 [Appendix A](#), it is precisely in cases in which killer acquisitions do not harm welfare that they are also unlikely to take place.

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

Given these competing forces the overall effect of killer acquisitions on ex-ante innovation (and, therefore, welfare) remains unclear. However, based on 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 it also deters incumbents from engaging in killer acquisitions of future competitors, thus increasing future competition and further deterring killer acquisitions.

6. 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

⁴⁴A variety of evidence (Adams and Brantner, 2006; Budish et al., 2015) suggests that intellectual property protection, most notably patents, plays a key role in motivating innovation and influencing the direction of innovative efforts in the pharmaceutical industry.

⁴⁵Rasmusen (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.

⁴⁶For 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/>.

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 valuable human capital, and the relative data scarcity poses 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.

⁴⁷A first attempt in quantifying potential adverse effects of acquisitions by tech giants on start-up innovation is by Kamepalli et al. (2019), 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 (2019) 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 last decade: https://www.ftc.gov/system/files/documents/public_statements/1566385/statement_by_commissioners_wilson_and_chopra_re_hsr_6b.pdf.

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Table 1
Description of Drug Development Project Acquisitions

This table provides descriptive statistics on drug projects categorized into non-acquired, acquired by non-overlapping acquirers, and acquired by overlapping acquirers. The table describes the number of drugs originated over time and by consolidated disease groups as well as the proportion of projects that are non-acquired, acquired by non-overlapping 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.

	N	Non-Acquired	Non-overlap Acquired	Overlap 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%
2001-2005	4,716	79%	16%	4%
2006-2010	5,761	90%	8%	2%
By high-level disease group (top 5)				
Anti-cancer (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%

Table 2
Overlapping Acquisitions and Project Development

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

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

where the dependent variable $Development_{i,t}$ is a dummy variable indicating whether drug i has a development event in year t . $I(Acquired)_i$ indicates whether drug i is acquired during the study period, and $I(Post)_{i,t}$ indicates whether the drug-year (i,t) observation is after the drug is acquired. $I(Overlap)_i$ is a dummy variable indicating the acquired drug overlaps with the product portfolio of the acquirer. $Before(-t)$ indicates that the drug-year is t years before an acquisition and takes zero otherwise. Standard errors clustered at the drug project level are displayed in parentheses. ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent levels, respectively.

	Development Event = 1					
	(1)	(2)	(3)	(4)	(5)	(6)
$I(Acquired) \times I(Post) \times Overlap$	-0.037*** (0.013)	-0.033** (0.014)	-0.029* (0.015)	-0.041** (0.019)	-0.043** (0.021)	-0.054** (0.024)
$I(Acquired) \times I(Post)$	-0.020*** (0.006)	-0.016** (0.007)	-0.017** (0.009)	-0.024** (0.010)	-0.018 (0.011)	-0.018 (0.013)
$I(Acquired) \times Overlap$	0.004 (0.008)	0.009 (0.009)	0.026** (0.011)			
$I(Acquired)$	-0.002 (0.004)	-0.004 (0.005)	-0.011 (0.012)			
$Before(-3) \times Overlap$						-0.031 (0.032)
$Before(-2) \times Overlap$						0.012 (0.032)
$Before(-1) \times Overlap$						-0.040 (0.030)
$Before(-3)$						0.015 (0.017)
$Before(-2)$						0.020 (0.017)
$Before(-1)$						-0.003 (0.016)
Observations	143,569	143,569	143,569	143,569	134,662	143,569
R-squared	0.038	0.252	0.289	0.366	0.662	0.370
Vintage FE	Y	Y	Y			
Age FE	Y					
Age \times TC \times MOA		Y	Y	Y	Y	Y
Originator [Target company] FE			Y			
Project FE				Y	Y	Y
Propensity score reweighted					Y	

Table 3

Overlapping Acquisitions and Project Development: Alternative Specifications

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

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

where the dependent variable $Development_{i,t}$ is a dummy variable indicating whether or not drug i has a development event in period t in columns 1–3 (or has no development event in period t in column 4). $I(Acquired) \times I(Post)$ indicates whether the drug-period(i,t) observation is after the drug is acquired, and $I(Acquired) \times I(Post) \times I(Overlap)$ also indicates the acquired drug overlaps with the acquirer’s product portfolio. Standard errors clustered at the drug project level are displayed in parentheses. ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent levels, respectively.

	(1)	(2)	(3)	(4)
		Development Event = 1		No Development = 1
$I(Acquired) \times I(Post) \times Overlap$	-0.050** (0.023)	-0.121*** (0.060)	0.005 (0.035)	0.149*** (0.033)
$I(Acquired) \times I(Post)$	-0.024 (0.015)	-0.041 (0.025)	-0.095*** (0.013)	0.401*** (0.021)
Observations	27,784	19,651	7,916	9,227
R-squared	0.445	0.249	0.155	0.477
Sample:	Acquired projects only	Single-project target only	Excluding “never developed”	
TC × MOA				Y
Age FE × TC × MOA	Y	Y	Y	Y
Project FE	Y	Y	Y	Y

Table 4
Overlapping Acquisitions and Project Development: Market Competition

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

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

where the dependent variable $Development_{i,t}$ is a dummy variable indicating drug i has a development event in year t for columns 1–3 (or has no development event in period t for columns 4–6). $I(Acquired)_i$ indicates drug i undergoes an acquisition event, and $I(Post)_{i,t}$ indicates the drug-year (i,t) observation is after the drug is acquired. We count the number of firms with a drug or drug project that is in the same market (same TC-MOA) as the focal drug. In columns 1–3, the analysis predicts likelihood of development across all projects in a project-year panel, while in columns 4–6, the analysis predicts likelihood of no development for acquired projects, comparing pre- and post-acquisition periods and overlapping and non-overlapping acquired projects. Drug development projects are categorized into high and low competition by the median of competition measures (product count). In columns 3 and 6 we present results in which we interact $I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i$ with the dummy indicating low competition. Standard errors clustered at the drug project level are displayed in parentheses. ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent levels, respectively.

	Development Event = 1			No Development = 1		
	(1) Low	(2) High	(3) Interacted	(4) Low	(5) High	(6) Interacted
$I(Acquired) \times I(Post) \times Overlap$	-0.065** (0.026)	0.017 (0.035)	0.017 (0.035)	0.219*** (0.054)	0.038 (0.070)	0.038 (0.070)
$I(Acquired) \times I(Post) \times Overlap \times LowCompetition$			-0.082* (0.044)			0.181** (0.089)
Observations	74,261	69,308	143,569	5,991	3,236	9,227
R -squared	0.415	0.399	0.408	0.497	0.474	0.489
TC \times MOA	Y	Y	Y	Y	Y	Y
Age FE \times TC \times MOA	Y	Y	Y	Y	Y	Y
Project FE						

Table 5
Overlapping Acquisitions and Project Development: Acquirer’s Patent Life

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:

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

where the dependent variable $Development_{i,t}$ is a dummy variable indicating whether drug i has a development event in year t . $I(Post)_{i,t}$ indicates whether the drug-year (i,t) observation is after the drug is acquired. $I(NearPatentExpiry)$ is a dummy variable indicating whether the overlapping acquirer drug is within five years of patent expiry. Standard errors clustered at the drug project level are displayed in parentheses. ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent levels, respectively.

	(1)	(2)
	Development Event = 1	
I(Post) × I(Near patent expiry)	0.013 (0.133)	0.406*** (0.090)
I(Post)	-0.173* (0.092)	-0.210*** (0.067)
I(Near patent expiry)	-0.104** (0.043)	-0.147*** (0.043)
Observations	6,398	6,398
R-squared	0.212	0.450
Vintage FE	Y	Y
Age FE	Y	
TC × MOA FE	Y	
Age × TC × MOA FE		Y

Table 6
Product Overlap and Acquisition Decisions

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

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

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

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Acquisition = 1				Acquisition = 1			
Overlap (TC-MOA)	0.626*** (0.009)		0.577*** (0.015)		0.194*** (0.010)		0.209*** (0.015)	
Overlap (TC)		0.356*** (0.005)		0.300*** (0.008)		0.214*** (0.008)		0.200*** (0.011)
Overlap (TC-MOA) × LowCompetition			0.088*** (0.019)				-0.027 (0.020)	
Overlap (TC) × LowCompetition				0.103*** (0.011)				0.025* (0.015)
Observations	55,374	55,374	38,430	38,430	34,005	34,005	34,005	34,005
Pseudo R -squared	0.118	0.119	0.098	0.097	0.052	0.064	0.052	0.065
Deal FE	Y	Y	Y	Y	Y	Y	Y	Y
Matching method		Random matching				Matched by pipeline size		
No. of deals	9,229	9,229	9,229	9,229	9,229	9,229	9,229	9,229
No. of control deals	46,145	46,145	46,145	46,145	46,145	46,145	46,145	46,145

Table 7
The Intensity of Project Discontinuation Around FTC Review Threshold

This table presents univariate survival tests on the drugs that are acquired just below $[-5\%, 0]$ and just above $[0, 5\%]$ the FTC review threshold. Specifically, we examine the rates of being active, being discontinued, and being fully launched using the development status of each project as of June 2017. To ensure that we leave adequate room for acquisitions to occur, we focus on drug projects originated before 2011. We report the rate of being active, being discontinued, and being fully launched separately for the two samples and the difference between them. T -test of the sample means and the significance levels are reported. ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent levels, respectively. In panel B, we report the same analysis with a pseudo HSR threshold. To construct the pseudo threshold, 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).

Real HSR Threshold						
	5% Below Threshold	5% Above Threshold	Diff	T-Statistics	Stat. Significance	
Active	3.57%	7.58%	-4.00%	-1.176	Not Sig.	
Launched	1.79%	9.09%	-7.31%	-2.293	**	
Discontinued	94.64%	83.33%	11.31%	2.509	**	

Pseudo Threshold						
	5% Below Threshold	5% Above Threshold	Diff	T-Statistics	Stat Significance	
Active	7.41%	2.63%	4.78%	1.20	Not Sig.	
Launched	3.70%	4.39%	-0.69%	-0.16	Not Sig.	
Discontinued	88.88%	92.98%	-4.10%	-0.71	Not Sig.	

Table 8
Post-Acquisition Asset Redeployment

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 five-year windows 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 -test for subsample differences. ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent levels, respectively.

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

	(1)	(2)	(3)	(4)	(5)	(6)
	Chemical Similarity			Citation to Targets		
I(Post) \times Overlap	0.001 (0.003)	0.000 (0.003)	0.002 (0.002)	-0.002 (0.002)	-0.002 (0.002)	-0.000 (0.000)
I(Post)	-0.002 (0.004)	-0.001 (0.004)	-0.004 (0.003)	0.000 (0.000)	0.001 (0.001)	0.000 (0.000)
Overlap	0.004 (0.003)	0.004 (0.003)		0.002 (0.002)	0.002 (0.002)	
Observations	154,896	154,896	154,896	154,896	154,896	154,896
R -squared	0.001	0.014	0.361	0.001	0.094	0.154
Acquirer FE		Y			Y	
Case FE			Y			Y

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

	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	-0.267*
Difference	-0.215	0.929***	1.144***

Table 9
Project Overlap and Acquisition Value

This table investigates if overlapping firms are acquired for lower transaction sums than non-overlapping firms. The sample for this analysis is acquired firms where the deal value amount is available in one of our three main acquisition datasets (Thomson Reuters SDC Platinum, RecapIQ, and VentureXpert). The empirical specification uses the following model:

$$\ln(\text{AcquisitionValue})_i = \beta \cdot I(\text{Overlap})_i + \alpha_{FE} + \varepsilon_i,$$

where the dependent variable $\ln(\text{AcquisitionValue})_i$ is the natural logarithm of the total disclosed acquisition amount (USD). $I(\text{Overlap})_i$ is a dummy variable indicating the target overlaps the potential acquirer firm. Robust standard errors are displayed in parentheses. ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent levels, respectively.

	(1)	(2)	(3)
	Ln(Acquisition Value)		
Overlap	0.126 (0.101)	0.025 (0.067)	-0.082 (0.114)
Observations	14,660	14,660	14,660
R-squared	0.844	0.905	0.940
Acquirer FE	Y	Y	Y
Age FE	Y	Y	
TC × MOA FE		Y	
Age × TC × MOA FE			Y

Online Appendix

A. Proofs and Extensions of the Theoretical Framework

A.1. Bertrand Competition

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

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

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

where q^i is the quantity of product i , $\alpha^i > 0$ measures product quality in a vertical sense, and γ represents the degree of substitutability between products. $1 > \gamma > 0$ ensures that the products are (imperfect) substitutes. The higher the γ , the more alike are the products. The resulting consumer maximization problem yields linear inverse demand for each product i given by $p^i = \alpha^i - q^i - \gamma \sum_{j \neq i} q^j$, where p^i is the price of product i . For simplicity, let us assume that the product quality of all the incumbents is identical, α^A for all the incumbents' existing products and α^E for the entrepreneur's potential product.

A.1.2. No Acquisition. Consider first the product market choices of an entrepreneur that is not acquired ($-acq$). If the project is successful (S), the resulting newly developed product competes against n other single-product incumbent firms. The entrepreneur's objective function is $\max_{p^E} p^E q^E$, and the acquirer's (and all of the other incumbents') objective function is $\max_{p^A} p^A q^A$. The n incumbents have product quality α^A , and the entrepreneur has product quality α^E . The entrepreneur's product market profit is given by

$$\pi_{-acq,S}^E = \frac{\gamma(n-1)+1}{(1-\gamma)(\gamma n+1)} (p_{-acq,S}^E)^2,$$

where

$$p_{-acq,S}^E = \frac{\alpha^E \{ \gamma^2 [(n+1)^2 - 5(n+1) + 5] + 3\gamma(n-1) + 2 \} - \gamma [\gamma(n-1) + 1] \alpha^A n}{[\gamma(n-2) + 2] \{ \gamma [2(n+1) - 3] + 2 \}}.$$

The acquirer's profit is equal to

$$\pi_{-acq,S}^A = \frac{\gamma(n-1) + 1}{(1-\gamma)(\gamma n + 1)} (p_{-acq,S}^A)^2,$$

where

$$p_{-acq,S}^A = \frac{\alpha^A \{ \gamma^2 [(n+1)^2 - 5(n+1) + 5] + 3\gamma(n-1) + 2 \} - \gamma [\gamma(n-1) + 1] [\alpha^A(n-1) + \alpha^E]}{[\gamma(n-2) + 2] \{ \gamma [2(n+1) - 3] + 2 \}}.$$

In the special case of no vertical differentiation ($\alpha^A = \alpha^E$), these expressions simplify to

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

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

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

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

$$\max_{p_{old}^A, p_{new}^A} p_{old}^A q_{old}^A + p_{new}^A q_{new}^A,$$

whereas the remaining $n-1$ other single-product firms maximize single-product profits.

The profit of the multi-product incumbent acquirer is

$$\pi_{acq,S}^A = p_{old}^A q_{old}^A + p_{new}^A q_{new}^A,$$

where

$$p_{old}^A = \frac{1}{2} \left[\frac{\alpha^A - \alpha^E}{2} + \frac{[2 + \gamma(2n - 1)][2 + \gamma(n - 2)]}{[2 + \gamma(2n - 2)][2 + \gamma(n - 1)] - 2\gamma^2} \tilde{p} \right]$$

$$p_{new}^A = \frac{1}{2} \left[\frac{\alpha^E - \alpha^A}{2} + \frac{[2 + \gamma(2n - 1)][2 + \gamma(n - 2)]}{[2 + \gamma(2n - 2)][2 + \gamma(n - 1)] - 2\gamma^2} \tilde{p} \right]$$

and

$$q_{old}^A = \frac{1}{2} \left[\frac{1}{1 - \gamma} \frac{\alpha^A - \alpha^E}{2} + \frac{[2 + \gamma(2n - 1)][2 + \gamma(n - 2)]}{[2 + \gamma(2n - 2)][2 + \gamma(n - 1)] - 2\gamma^2} \frac{1 + \gamma(n - 2)}{1 + \gamma(n - 1)} \tilde{q} \right]$$

$$q_{new}^A = \frac{1}{2} \left[\frac{1}{1 - \gamma} \frac{\alpha^E - \alpha^A}{2} + \frac{[2 + \gamma(2n - 1)][2 + \gamma(n - 2)]}{[2 + \gamma(2n - 2)][2 + \gamma(n - 1)] - 2\gamma^2} \frac{1 + \gamma(n - 2)}{1 + \gamma(n - 1)} \tilde{q} \right],$$

where $\tilde{p} = p_{-acq,S}^A + p_{-acq,S}^E$ and $\tilde{q} = \frac{\gamma(n-1)+1}{(1-\gamma)(\gamma n+1)} \tilde{p}$ denote the sum of prices and the sum of quantities of the acquirer's two products if these two products were produced by two separate single-product competitors.

In the special case of no vertical differentiation ($\alpha^A = \alpha^E$), the profit of the multiproduct acquirer simplifies to

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

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

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

Comparing these expressions yields the profit ranking for the acquirer

$$\pi_{acq,S}^A > \pi_{acq,F}^A = \pi_{-acq,F}^A > \pi_{-acq,S}^A > 0$$

and for the entrepreneur

$$\pi_{-acq,S}^E > \pi_{-acq,F}^E = 0.$$

In addition, with these expressions, we obtain the central inequality characterizing development gains of the new product for the entrepreneur and the acquirer, which is given by

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

which holds for $1 > \gamma > 0$.

A.1.4. Product Market Overlap.

Proof of Proposition 1. From inequality (3), it immediately follows that an incumbent firm acquires a project and continues development if $k \leq k^A$ and that an independent entrepreneur continues if $k \leq k^E$. Equation (4) shows that the thresholds $k^E > k^A$ if and only if $\rho^E \Delta^E > \rho^A \Delta^A$. Rearranging the inequality yields the proposition.

Using the expressions above for any positive product market overlap $1 > \gamma > 0$, we have $\Delta^E > \Delta^A$. For $\gamma = 0$ and $\gamma = 1$, we have $\Delta^E = \Delta^A$. Therefore, for any product market overlap, even if the incumbent has a development advantage (i.e., $\rho^A > \rho^E$), he still may have a lower willingness to develop the project than the entrepreneur. \square

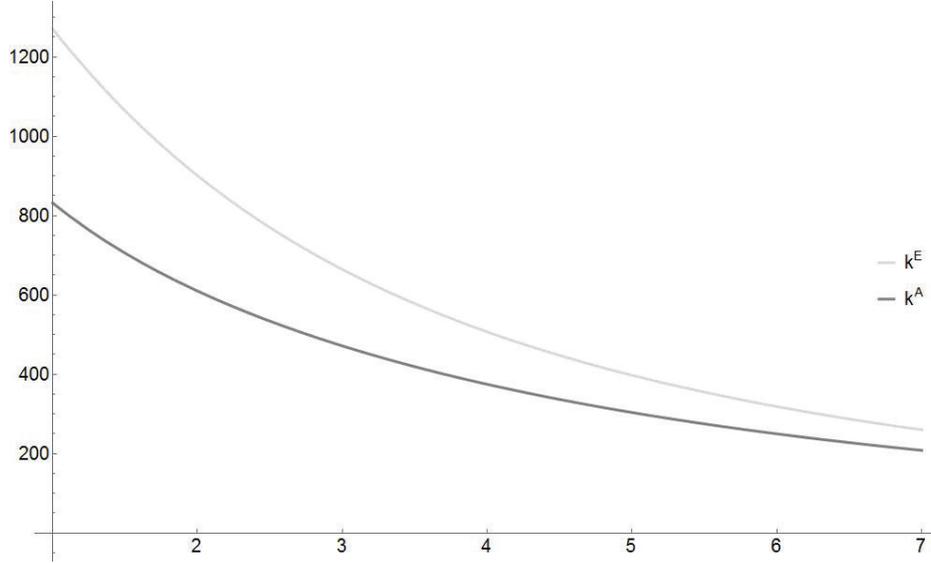
A.1.5. Competition.

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

Figure A1 illustrates this point by plotting the development thresholds k^E and k^A as a function of the number of incumbents for the case where $\rho^E = \rho^A$. These thresholds are closer together when there are more existing incumbents because the difference $\Delta^E - \Delta^A$ shrinks as n increases. In the limit in which the number of existing products n goes to infinity, the acquiring incumbent has a vanishingly small share of the existing market and thus all of the

cannibalization losses fall on the other competitors. As a result, for $n \rightarrow \infty$, Δ^E and Δ^A are the same and hence the continuation policies of the entrepreneur and the acquirer are identical.

Figure A1. Development Cost Thresholds and Competition



This graph plots the optimal development cost thresholds of the entrepreneur (k^E , light gray) and the acquirer (k^A , dark gray) as a function of the number of incumbents n . The other parameter values are $\alpha^A = \alpha^E = 100$, $\gamma = 0.375$, $\rho^A = \rho^E = 0.75$, and $L = 20$.

A.1.6. Patent Life and Future Competition.

Proof of Proposition 3. After T^A years, the patent on the incumbent's existing product expires and generic undifferentiated entry for this product occurs. Due to undifferentiated Bertrand competition, profits of the acquirer's existing product drop to zero after T^A . Until that time T^A , the acquirer's development gain from developing the new product is equal to $T^A \Delta^A$. Similarly, the entrepreneur's development gain over that time span is $T^A \Delta^E$.

In addition, after T^A and until T^E , the newly developed product still earns positive profits because it is differentiated from the generic entry that drives the profits of the existing drug to zero. Denote the per-period development gains of the new product for the entrepreneur and the acquirer in the presence of generic competition for the acquirer's existing product by Δ_{gen}^E and Δ_{gen}^A .

First, these development gains are smaller than the previous expressions, $\Delta_{gen}^E < \Delta^E$ and $\Delta_{gen}^A < \Delta^A$. When a generic product that is undifferentiated from the acquirer's existing product enters, it not only drives profits of that existing product to zero, but it also reduces the profits

of the newly developed product. This occurs because the generic product is a perfect substitute for the acquirer's existing product and an imperfect substitute for the newly developed product.

Second, the development gains of the new product under generic competition for the existing product are equal to each other; $\Delta_{gen} = \Delta_{gen}^E = \Delta_{gen}^A$. This is because generic competition for the acquirer's existing drug has already reduced all of the profits of the acquirer's existing drug, and thus development of the new product does not replace any of the existing product's profits for the acquirer.

After T^E , generic entry also occurs for the newly developed product occurs, thereby reducing all profits to zero.

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

$$\rho^E \left(\frac{1-\delta^{T^A}}{1-\delta} \Delta^E + \frac{\delta^{T^A} - \delta^{T^E}}{1-\delta} \Delta_{gen} \right) - k \geq L \quad (9)$$

$$\rho^A \left(\frac{1-\delta^{T^A}}{1-\delta} \Delta^A + \frac{\delta^{T^A} - \delta^{T^E}}{1-\delta} \Delta_{gen} \right) - k \geq L, \quad (10)$$

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

Therefore, the resulting difference in the development thresholds is given by $\rho \frac{1-\delta^{T^A}}{1-\delta} (\Delta^E - \Delta^A)$. This difference is increasing in T^A if $\frac{\rho^A}{\rho^E} \leq \frac{\Delta^E - \Delta_{gen}}{\Delta^A - \Delta_{gen}}$, which establishes the proposition. \square

A.1.7. Acquisition Decision.

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

$$d^A [\rho^A \pi_{acq,S}^A + (1-\rho^A) \pi_{acq,F}^A - k] + (1-d^A)(L + \pi_{acq,F}^A) - P \geq d^E [\rho^E \pi_{acq,S}^A + (1-\rho^E) \pi_{acq,F}^A] + (1-d^E) \pi_{acq,F}^A, \quad (11)$$

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

To compensate the entrepreneur for selling the project, the acquirer must pay a price P that

is equal to the expected payoff of the project when the entrepreneur remains independent. Thus,

$$P = d^E(\rho^E \Delta^E - k) + (1 - d^E)L. \quad (12)$$

Substituting the takeover price (12) into the inequality for the acquisition decision (11) and solving for each of the cases of k establishes the acquisition choices outlined in the main text for the case where $1 > \gamma > 0$.

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

A.1.8. Welfare. We now show under what conditions killer acquisitions are welfare-decreasing. This is the case whenever $k^E \geq k > k^A$ and the social surplus resulting from no acquisition (and continued development) is higher than when there is no acquisition (and termination). Under a social welfare standard that uses the unweighted sum of consumer surplus and producer surplus, this is given by the following inequality

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

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

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

The first term in parentheses on the left-hand side is the entrepreneur's net expected profit gain from continuing development. This is positive in the killer acquisitions region $k^E \geq k > k^A$. The second term is the expected increase in consumer surplus due to continued development, which is also positive both because there is more product variety and prices are lower. The term on the right-hand side of the inequality is the expected loss in profit for the n incumbents, and it is also positive. We can derive a sufficient condition for killer acquisitions to be welfare-reducing by setting the first term to zero (i.e., $k = k^E$ so the entrepreneur is indifferent between developing and not developing). This yields the following condition:

$$CS_{-acq,S} - CS_{-acq,F} \geq n(\pi_{-acq,F}^A - \pi_{-acq,S}^A). \quad (13)$$

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

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

where q_n and q_{n+1} denote the equilibrium quantities when there are n and $n+1$ firms in the market.

It is now straightforward to show that the sufficient condition for killer acquisitions to be welfare-reducing given by inequality (13) is always satisfied for any degree of product substitution under differentiated Bertrand competition with a single incumbent ($n=1$). For $n \geq 2$, even without vertical differentiation, the inequality is satisfied under differentiated Bertrand competition as long as the entrant's product is sufficiently differentiated (i.e., $\gamma < \gamma^W$) from the existing incumbents.

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

A.2. Cournot Competition

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

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

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

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

$$\pi_{acq,F}^A = \frac{(\alpha^A)^2}{[2+\gamma(n-1)]^2}$$

and

$$\pi_{acq,S}^A = p_{old}^A q_{old}^A + p_{new}^A q_{new}^A,$$

where $p_{old}^A = q_{old}^A + \gamma q_{new}^A$ and $p_{new}^A = q_{new}^A + \gamma q_{old}^A$ and

$$\begin{aligned}q_{old}^A &= \frac{1}{2} \left[\frac{\alpha^A - \alpha^E}{2(1-\gamma)} + \frac{2+\gamma(n-2)}{2+\gamma(n-\gamma)} \left(\frac{\alpha^A + \alpha^E}{2} - \gamma \frac{(n-1)\alpha^A}{2+\gamma(n-2)} \right) \right] \\ q_{new}^A &= \frac{1}{2} \left[\frac{\alpha^E - \alpha^A}{2(1-\gamma)} + \frac{2+\gamma(n-2)}{2+\gamma(n-\gamma)} \left(\frac{\alpha^A + \alpha^E}{2} - \gamma \frac{(n-1)\alpha^A}{2+\gamma(n-2)} \right) \right].\end{aligned}$$

In the special case of no vertical differentiation ($\alpha^A = \alpha^E$), the profit for the multiproduct acquirer simplifies to

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

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

A.3. The Market for Ownership

A.3.1. Multiple Potential Acquirers. In the baseline version of our model we assume that one of the n incumbents is chosen at random to be the potential acquirer of the entrepreneur's project. Assume the development cost k is in the region in which killer acquisitions can occur in the case of a single bidder, $k^E > k > k^A$, and that $\rho^E(\pi_{acq,F}^A - \pi_{-acq,S}^A) \geq \rho^E \Delta^E - k - L$ such that an acquirer acquires the entrepreneur (and does not develop the project). The acquirer has to pay the acquisition price $P = \rho^E \Delta^E - k$ (i.e., at least the entrant's expected profits under competition), but the benefits from eliminating a new competitor are diffuse and also accrue to the other incumbent. In particular, the acquirer earns $\pi_{acq,F}^A + L - P$, while all the other $n - 1$ incumbents earn $\pi_{acq,F}^A > \pi_{acq,F}^A + L - P$ because for $k^E > k > k^A$, we have $\rho^E \Delta^E - k > L$. This leads to a potential free-riding problem known as the "volunteer's dilemma" (Diekmann, 1985) or, more broadly, to the problem of costly private provision of a public good (Bliss and Nalebuff, 1984).

Let us now assume that $n_b \leq n$ incumbents can acquire the entrepreneur.⁴⁸ It is never optimal for any of the n_b bidders to bid any price other than $P = \rho^E \Delta^E - k$ because paying a higher price reduces the bidder's profits and bidding less means that the entrepreneur would reject the bid. First, there are therefore multiple pure-strategy equilibria in which one of the n_b incumbents bids $P = \rho^E \Delta^E - k$, all the other incumbents do not bid, and the bidding incumbent acquires the entrepreneur. All these pure-strategy equilibria are essentially identical to the single bidder case analyzed in our baseline model. Second, for $n_b \geq 2$ there exists a mixed-strategy equilibrium in which each of the n_b bidders chooses to bid $P = \rho^E \Delta^E - k$ with probability p and to not make any bid with probability $1 - p$. In case of a tie in which several bidders make the same highest bid, one of them is chosen randomly.

If the bidder chooses not to make a bid, he obtains

$$(1-p)^{n_b-1}[\rho^E \pi_{-acq,S}^A + (1-\rho^E)\pi_{-acq,F}^A] + [1 - (1-p)^{n_b-1}]\pi_{acq,F}^A,$$

which is the sum of the expected payoffs when no bidder bids and when at least one bidder makes a bid.

⁴⁸Outside bidders are irrelevant in our model because they always have exactly the same valuation for the entrepreneur's project as the entrepreneur herself.

If the bidder chooses to bid $P = \rho^E \Delta^E - k$, he obtains

$$\sum_{j=0}^{n_b-1} \binom{n_b-1}{j} p^j (1-p)^{n_b-1-j} \left[\frac{1}{j+1} (\pi_{acq,F}^A + L - P) + \frac{j}{j+1} \pi_{acq,F}^A \right],$$

which can be rewritten in the following way

$$\pi_{acq,F}^A - \sum_{j=0}^{n_b-1} \binom{n_b-1}{j} p^j (1-p)^{n_b-1-j} \frac{1}{j+1} (\rho^E \Delta^E - k - L),$$

where the expected payment that the bidder makes depends on the number of other bidders who also bid.

In equilibrium, each bidder is indifferent between bidding P and not bidding, and thus we have

$$\begin{aligned} \pi_{acq,F}^A - \sum_{j=0}^{n_b-1} \binom{n_b-1}{j} p^j (1-p)^{n_b-1-j} \frac{1}{j+1} (\rho^E \Delta^E - k - L) = \\ (1-p)^{n_b-1} [\rho^E \pi_{-acq,S}^A + (1-\rho^E) \pi_{-acq,F}^A] + [1 - (1-p)^{n_b-1}] \pi_{acq,F}^A, \end{aligned}$$

which we can rearrange to obtain

$$p = 1 - \left[\frac{\rho^E \Delta^E - k - L}{\rho^E (\pi_{acq,F}^A - \pi_{-acq,S}^A)} \sum_{j=0}^{n_b-1} \binom{n_b-1}{j} p^j (1-p)^{n_b-1-j} \frac{1}{j+1} \right]^{\frac{1}{n_b-1}}.$$

This equation defines the individual equilibrium mixing probability p . It has a very intuitive interpretation. As in the single acquirer case, the main driving force is the ratio of the replacement effect ($\rho^E \Delta^E - k - L$) to the efficiency effect ($\rho^E (\pi_{-acq,F}^A - \pi_{-acq,S}^A)$). The larger the efficiency effect relative to the replacement effect is, the more likely an acquirer is to make an acquisition bid for the entrepreneur. This is essentially the same as the inequality in (5), which requires that the ratio of $\rho^E \Delta^E - k - L$ (replacement effect) to $\rho^E (\pi_{-acq,F}^A - \pi_{-acq,S}^A)$ (efficiency effect) is smaller than one.

In addition, the number of potential acquirers n_b also plays a crucial role now. There are two forces at work here that work in opposite directions. First, as the number of potential bidders increases, the incentive to free ride on another bidder's bid (and killer acquisition) increases.

This can be seen by looking at the exponent $\frac{1}{n_b-1}$ on the right-hand side of the equation that reduces the right-hand side because the term inside the brackets is positive and strictly smaller than one. This is the familiar free-riding effect in the volunteer's dilemma. Second, as the number of potential acquirers increases, the expected payment that an acquirer has to make when he decides to make a bid decreases. This occurs because he is less likely to be chosen as the "winning bidder" who has to pay the acquisition price to the entrepreneur.⁴⁹ The term inside the summation on the right-hand side captures this effect; it decreases with n_b and thus raises the probability that a potential acquirer will make a bid for the entrepreneur.

We can further rewrite the equation defining p to obtain

$$p = 1 - \left[\frac{\rho^E \Delta^E - k - L}{\rho^E (\pi_{acq,F}^A - \pi_{acq,S}^A)} \frac{1 - (1-p)^{n_b}}{n_b p} \right]^{\frac{1}{n_b-1}}.$$

Therefore the probability that there will be a killer acquisition (i.e., that at least one bidder will acquire the entrepreneur) is defined by

$$1 - (1-p)^{n_b} = 1 - \left[\frac{\rho^E \Delta^E - k - L}{\rho^E (\pi_{acq,F}^A - \pi_{acq,S}^A)} \frac{1 - (1-p)^{n_b}}{n_b p} \right]^{1 + \frac{1}{n_b-1}}.$$

Using implicit differentiation, we can show that this probability strictly increases as the number of potential bidders n_b increases. In other words, the increase in the number of potential acquirers n_b together with the above-mentioned second effect (i.e., the effect arising from an acquirer having to make a lower expected payment) dominate the first effect (i.e., the conventional free-riding effect in the volunteer's dilemma).

Finally, note that these comparative statics of the mixed-strategy equilibrium are for the case where we hold the number of incumbents n fixed and focus on the region in which killer acquisitions occur. Changing the number of incumbents n , however, also affects the ratio of the replacement effect to the efficiency effect and, more importantly, shrinks the region $k^E \geq k > k^A$ in which killer acquisitions are possible. Therefore, comparative statics of the mixed-strategy equilibrium with respect to the number of incumbents who are all allowed to

⁴⁹This effect would be absent in an (albeit unrealistic) all-pay auction setup in which each bidder always has to pay his bid.

bid lead to ambiguous predictions.

A.3.2. Asymmetries between Potential Acquirers. Consider now the case in which there are multiple potential acquirers denoted by A_i whose products can differ in their degree of differentiation from the entrepreneur's project (i.e., different degree of substitutability $\gamma^{A_i E}$) and can have different synergy advantages (i.e., different development probability ρ^{A_i}). For simplicity, we assume that there is no vertical differentiation. The linear inverse demand for the existing product of potential acquirer A_i is now given by $p^{A_i} = \alpha - q^{A_i} - \gamma \sum_{A_j \neq A_i}^n q^{A_j} - \gamma^{A_i E} q^E$. We assume that the development synergies are not too large such that k^{A_i} , which denotes the development threshold of the potential acquirer A_i defined by $k^{A_i} = \rho^{A_i} \Delta^{A_i} - L$, is smaller than k^E for each potential acquirer A_i . Formally, this is the case if $\frac{\Delta^E}{\Delta^{A_i}} > \frac{\rho^{A_i}}{\rho^E}$.

First, as discussed before, the parties are indifferent as to who owns the projects if development costs are such that $k > k^E$ because everybody chooses not to develop the project in that case and has the same valuation.

Second, if development costs are in the killer acquisitions region (i.e., $k^E > k > k^{A_i}$) for all A_i potential acquirers, the potential acquirer who is the least differentiated from the entrepreneur will have the highest valuation for the entrepreneur's project among all potential acquirers. Recall that in this region an acquirer is willing to acquire the entrepreneur if

$$L + \pi_{acq,F}^{A_i} - (\rho^E \Delta^E - k) \geq \rho^E \pi_{-acq,S}^{A_i} + (1 - \rho^E) \pi_{-acq,F}^{A_i},$$

which we can rewrite to obtain the familiar condition

$$\underbrace{\rho^E (\pi_{acq,F}^{A_i} - \pi_{-acq,S}^{A_i})}_{\text{efficiency effect}} - \underbrace{(\rho^E \Delta^E - k - L)}_{\text{replacement effect}} \geq 0.$$

The replacement effect is the same for all the acquirers because they all shut down the project upon acquisition. The efficiency effect, however, is largest for the potential acquirer A_i with the product that is the least differentiated from the entrepreneur's project (i.e., the highest $\gamma^{A_i E}$). To see this, note that in case of a killer acquisition, all incumbents earn the same profit $\pi_{acq,F}^{A_i} = \pi_{acq,F}$. If the killer acquisition does not occur and the entrepreneur successfully

develops the project, $\pi_{-acq,S}^{A_i}$ will be the lowest profit for the incumbent with the least degree of differentiation from the entrepreneur. Thus, the incumbent with the most closely related product to the entrepreneur's project will have the highest valuation to acquire it. Importantly, this result holds even if potential acquirers differ in their development capabilities ρ^{A_i} because none of the potential acquirers would want to continue the project.

Third, this unambiguous result that the least differentiated potential acquirer has the highest valuation among all potential acquirers continues to hold if the potential acquirers have the same development capabilities (i.e., $\rho^{A_i} = \rho^A$ for all A_i) but differ in their degree of differentiation such that at least some (or all) of the potential acquirers have development thresholds $k^{A_i} > k$ (and thus they would continue development following an acquisition), while the remaining potential acquirers have thresholds $k^{A_i} < k$ (and thus they would kill the project following an acquisition). Because the potential acquirers only differ in their degree of differentiation from the entrepreneur's project, the potential acquirer with the highest overlap $\gamma^{A_i E}$ (i.e., the most closely related product) has the lowest k^{A_i} . Denote this potential acquirer by A_h . As we showed above, all other potential killer acquirers with $k^{A_i} > k$ have a strictly lower net gain from acquisition than this acquirer with his overlap $\gamma^{A_h E}$. For this acquirer, the net gain from acquisition is equal to

$$G^{A_h} = \rho^E (\pi_{acq,F}^{A_h} - \pi_{-acq,S}^{A_h}) - (\rho^E \Delta^E - k - L) \geq \rho^E (\pi_{acq,F}^{A_h} - \pi_{-acq,S}^{A_h}) - (\rho^E \Delta^E - \rho^A \Delta^{A_h}),$$

where we used the fact that for $k + L = \rho^A \Delta^{A_i}$, this potential acquirer is just indifferent between developing and not developing the project. The expression after the inequality is the same as the net gain from acquisition for all potential acquirers with $k^{A_i} < k$:

$$G^{A_i} = \rho^E (\pi_{acq,F}^{A_i} - \pi_{-acq,S}^{A_i}) - (\rho^E \Delta^E - \rho^A \Delta^{A_i}).$$

This net gain is again a trade-off between the efficiency effect (first term) and the replacement effect (second term). It remains to show that the net gain for potential acquirer A_h is larger than for any other acquirer A_i . Both the efficiency effect as well as the replacement effect are largest for the acquirer A_h , and thus it is not a priori clear whether this acquirer will have the highest valuation for the entrepreneur's project. However, under both Bertrand and Cournot competition, G^{A_h} is strictly larger than any other G^{A_i} . This is because the impact of increasing product over-

lap is greater on the efficiency effect than on the replacement effect. Thus, the potential acquirer with the largest product overlap generally has the strongest incentive to acquire the entrepreneur if potential acquirers only differ in the degree of product differentiation from the entrepreneur.

Fourth, this acquisition value result no longer holds if potential acquirers also differ with respect to their development capabilities ρ^{A_i} . If development capabilities ρ^{A_i} and degrees of product differentiation from the entrepreneur's project $\gamma^{A_i E}$ are such that at least some (or all) of the potential acquirers have development thresholds $k^{A_i} > k$, it is possible that a continuing acquirer with less overlap (lower $\gamma^{A_i E}$) but higher development capability (higher ρ^{A_i}) will have the highest valuation for the entrepreneur's project. This can be seen by writing out the net gain from acquisition for a continuing acquirer A_i with project development probability ρ^{A_i} :

$$G^{A_i} = \rho^E (\pi_{acq,F}^{A_i} - \pi_{-acq,S}^{A_i}) - (\rho^E \Delta^E - \rho^{A_i} \Delta^{A_i}).$$

For ρ^{A_i} sufficiently high (but still low enough such that $\frac{\Delta^E}{\Delta^{A_i}} > \frac{\rho^{A_i}}{\rho^E}$ is satisfied), this net gain is larger than that of the potential (killer or continuing) acquirer with the highest overlap. Conversely, as we showed above it is also possible that a killer acquirer with more overlap (higher $\gamma^{A_i E}$) but a slightly lower development capability (lower ρ^{A_i}) will have the highest valuation for the entrepreneur's project. As in the case with a single potential acquirer, the relative magnitudes of the replacement effect and the efficiency effect play a crucial role because they determine the identity of the potential acquirer with the highest valuation.

B. Cleaning Pharmaprojects Data

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

B.1. Identifying Originators of Drug Projects

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

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

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

To extract the original originator firm from these fields, we used regular expressions and phrases such as “X acquired by Y” or “developed by X.” We algorithmically created a list of original originators and the acquiring firms, and we checked them against our M&A datasets from SDC and Recap IQ.

B.2. Merging Pharmaprojects with Acquisition Data

Once we had a dependable measure of the true originator firms, our second challenge in using Pharmaprojects was to standardize originator firm names for matching with other datasets, including M&A events. We did so first by using the Stata program “stnd_compname” (Wasi and Flaaen, 2015), which isolated the stem name for each originator firm associated with each project in Pharmaprojects. We then checked all non-exact matching manually to confirm accuracy.

B.3. Categorizing Development Milestones

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

Examining the patterns in development events and activities over the life cycle of active drug projects, we see that commercialization-related activity increases as a share of development activity over time (see table “Share of Development Events, by Project Age”). Therefore capturing such activities over and above clinical trial progress is especially important for inferring post-acquisition development activity.

In addition to our main results using development milestones, we also run supplementary analysis using progression through phases of clinical trials as the dependent variable, which involves supplementing the Pharmaprojects data with Pharma Intelligence’s Trialrove data on clinical trials, linked at the project level. The data construction and results of this analysis are described in detail Appendix E.

Table B1
Measuring Drug Development

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

Events	Development Event?
Additional launches	Yes
Additional registrations	Yes
Change in disease status	–
Change in global status	–
Change in licensee status	–
Compounds identified	Yes
Development continuing	Yes
Discontinued products	No
First launches	Yes
First registrations	Yes
Global status reversion	–
Licenses discontinued	–
Licensing opportunities	–
Mechanism identified	Yes
Names granted	Yes
New chemical structure	Yes
New disease	Yes
New licensees	Yes
New patent applications	Yes
New product	–
New therapeutic activity	Yes
No development reported	–
Novel target reported	Yes
Orphan drug status granted	Yes
Registration submissions	–
Suspended products	No
Target identified	Yes
Withdrawn products	No

Table B2
Share of Development Events, by Project Age

This table presents a list of the share of development event/activities that comprise the dependent variable in our main analyses, across different ages of drug projects. The top panel presents the fully disaggregated tabulation. To highlight changes in the type of events over time, the bottom panel collapses event/activities into commercialization-related activity (first launches, additional launches, first registrations, additional registrations, names granted, new licensees, new patent applications, orphan drug status granted) and science-related activity (compounds identified, mechanisms identified, new chemical structure, new disease, new therapeutic activity, target identified, development continuing).

Events	0–2 years	3–5 years	6+ years	TOTAL
Additional launches	0.01	0.03	0.07	0.03
Additional registrations	0.01	0.03	0.06	0.03
Compounds identified	0.09	0.03	0.01	0.05
Development continuing	0.12	0.20	0.11	0.14
First launches	0.01	0.03	0.03	0.02
First registrations	0.01	0.03	0.03	0.02
Mechanism identified	0.04	0.02	0.02	0.03
Names granted	0.02	0.06	0.06	0.04
New chemical structure	0.20	0.07	0.05	0.12
New disease	0.13	0.18	0.22	0.17
New licensees	0.11	0.12	0.12	0.12
New patent applications	0.07	0.02	0.03	0.04
New therapeutic activity	0.10	0.10	0.09	0.10
Novel target reported	0.02	0.00	0.00	0.01
Orphan drug status granted	0.03	0.06	0.09	0.06
Target identified	0.03	0.02	0.01	0.02

Events	0–2 years	3–5 years	6+ years	TOTAL
Science related	0.70	0.62	0.50	0.62
Commercialization related	0.30	0.38	0.50	0.38

C. Data on Acquisitions

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

Armed with acquisition events compiled from multiple data sources, we then conduct a multistep cleaning process to ensure acquisition events are correctly linked to target and acquirer firms. We first standardize company names (for both acquirers and targets) and collect demographic information for each company. Second, since the same firm could appear in different databases 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 (which appear possibly due to using multiple datasets). To the best of our knowledge, this combination provides the most comprehensive database on acquisitions in the pharmaceutical industry.⁵⁰

We 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 pre- and post-acquisition.

The merged drug development and acquisition data show active acquisition activities in our analytical sample, with 22 percent of drug projects having an acquisition recorded in our acquisition databases. As tabulated in table 1, the rate of acquisition is lower for drugs

⁵⁰Each of the three data sources, SDC M&A Database, RecapIQ, and VentureXpert, independently contributes at least 10 percent of cases in the final database.

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)

D. Merging Drug Development and Acquisition Data with Patent Databases

In this section, we describe the process to merge drug development and acquisition data with USPTO patent databases through matching company names with assignee names in the USPTO patent database. To minimize potential problems introduced by the minor discrepancy between different versions of the USPTO database, we use both NBER and HBS patent databases to source patent assignee information. After this step, each company in the drug development and acquisition database will have its original name, standardized name, and a stem name; it is similar for USPTO assignees.

D.1. Name Standardization

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

D.2. The Matching Procedure

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

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

2. Each stem drug originator and owner name is matched with stem names from the NBER data and HBS data.
 - (a) If an exact match of stem names is identified, and the two companies are located in the same city and state OR the two companies are located in the same state and the earliest patenting year in NBER and HBS databases is later than the founding year in the drug data, we consider this as a “*successful match*.” The company is removed from the set of names waiting to be matched on both sides.
 - (b) If an exact match of stem names is identified, but the two companies do not satisfy the location and chronology criteria above, we consider this as a “*potential match*.” The company is moved to a pool of firms waiting for manual checks.
 - (c) Otherwise, we go to the next step.
3. For the remaining companies, each stem originator and owner name is matched with up to three close stem names from the USPTO data using a fuzzy matching method based on the Levenshtein edit distance.⁵¹ The criterion is based on the length of the strings and the Levenshtein distance, and the threshold is determined through a random sampling procedure.
 - (a) If the fuzzy matched pair is located in the same city and state OR the two companies are located in the same state and the earliest patenting year in NBER and HBS databases is later than the founding year in the drug data, we consider this as a “*potential match*.”
 - (b) Otherwise, the companies are categorized as “*failed to match*.”
4. The “*potential matches*” set identified in the procedures above are reviewed by hand, incorporating information from both data sources, including full patent abstracts and company business descriptions.
 - (a) Pairs confirmed as successful matches through the manual check are moved to the “*successful match*” set.

⁵¹The Levenshtein edit distance measures the degree of proximity between two strings and corresponds to the number of substitutions, deletions, or insertions needed to transform one string into the other one (and vice versa).

E. Clinical Trials

To supplement our main analyses, which use panel of all drug development events, we also examine the likelihood that a project continues to the next phase in the clinical trials process. In this section, we describe the process of creating the clinical trials dataset, outline the analysis, and present our clinical trial results.

E.1. Clinical Trials Data

To perform this supplementary analyses, we link the Pharmaprojects data with Pharma Intelligence’s Trialtrove data on clinical trial phases at the project level. Drug clinical trials comprise three main phases: Phase I trials, which are small (20–100 healthy volunteers) and short and are intended to test safety; Phase II trials, which are larger (hundreds of affected patients), typically randomized control trials lasting up to two years and are intended to test efficacy; and Phase III trials, which are expanded versions of Phase II trials, involving hundreds or thousands of participants and typically lasting one to four years (US FDA, 2017). Following successful trials, firms may submit a New Drug Application (NDA) to the FDA, which then decides if, and under what conditions, the drug can be marketed to US patients. We use Trialtrove data to identify the initiation of clinical trials by phase, including the timing of trial initiation.

Notably, clinical trial phase data are widely available only from 1997 onward, when the US federal government first mandated the National Institutes of Health (NIH) to collect and make publicly available a comprehensive clinical trials database.⁵² Therefore, we have comprehensive trial phase data only for projects first initiated after 1997. Within this limited sample, we identify projects for which we observe the start date of Phase I trials and track their progression, following prior studies that use progression through phases of clinical trials as a measure of project development (Krieger, 2017; Guedj and Scharfstein, 2004). We use trial progression as the dependent variable in these supplementary analyses.

In addition to providing robustness for our main results, analyzing progression through the clinical trial phases allows us to focus, albeit narrowly, on drugs in the exact same phase of clinical development and to examine progress to the next necessary phase. Focusing in this way helps

⁵²More details on the timeline of publicly available clinical trials database can be found at <http://www.clinicaltrials.gov>.

to alleviate concerns that our main results are driven by differences in the stage of development across projects that might remain even after including various age, project, TC-MOA, and firm-related fixed effects. We treat the clinical trial analysis as supplementary evidence because our main analyses include a larger sample of projects and additional key development events besides trial starts (e.g., patent applications) and the panel structure allows for project-level fixed effects.

E.2. Evidence from Clinical Trials

In this analysis, we focus on the subsample of drugs that start Phase I clinical trials and are subsequently acquired and on projects started before 2011. We do this to ensure there is sufficient time to observe an acquisition and to give the analyzed projects enough time to enter Phase II trials. Using the following specification, we test whether drug projects acquired by firms with overlapping products are less likely to start Phase II trials than drug projects acquired by non-overlapping incumbents:

$$PhaseII_i = \beta \cdot I(Acquired)_i \times I(Overlap)_i + \alpha_{FE} + \varepsilon_i. \quad (14)$$

Table E1 presents the clinical trial regression results. We find that projects acquired by firms with overlapping products are 9.0 percentage points less likely to progress to Phase II than non-overlapping acquired projects (column 1). In terms of economic magnitude, this represents a decrease of 23.7 percent from the base rate of starting Phase II for acquired projects of 38 percent. Or, put in terms of project termination, 62 percent of non-overlapping acquired projects are stopped before starting Phase II versus 71 percent of overlapping acquired projects. Columns 2–4 examine how competition conditions these results. Akin to our main analyses, we find that the decreased likelihood of acquired overlapping projects progressing in clinical trials is concentrated in markets with low existing product market competition. We also replicate table E1 using “pipeline” competition (i.e., number of competing projects under development) in table E2 and find similar results to both table E1 and the pipeline version of the main competition analysis (Appendix table F8).

Table E1
Overlapping Acquisitions and Project Development: Clinical Trials

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

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

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

	Phase II = 1			
	(1)	(2)	(3)	(4)
		Low Competition	High Competition	Interacted
I(Acquired) × Overlap	-0.090* (0.049)	-0.287** (0.112)	-0.065 (0.064)	-0.037 (0.055) -0.232** (0.109)
I(Acquired) × Overlap × LowCompetition				
Observations	1,860	511	1,348	1,860
R-squared	0.548	0.815	0.548	0.554
Phase I start year FE	Y	Y	Y	Y
TC × MOA FE	Y	Y	Y	Y

Table E2
Overlapping Acquisitions and Project Development:
Clinical Trials (Pipeline Competition)

This table presents the likelihood of starting Phase II trials, replicating the analysis in table E1. The key difference is the measure of competition in this table which is “pipeline competition.” An observation is a drug project that is acquired after starting Phase I clinical trials. We test whether drug projects acquired by firms with overlapping products are less likely to subsequently start Phase II trials than drug projects that are acquired by non-overlapping acquirers, using the following specification:

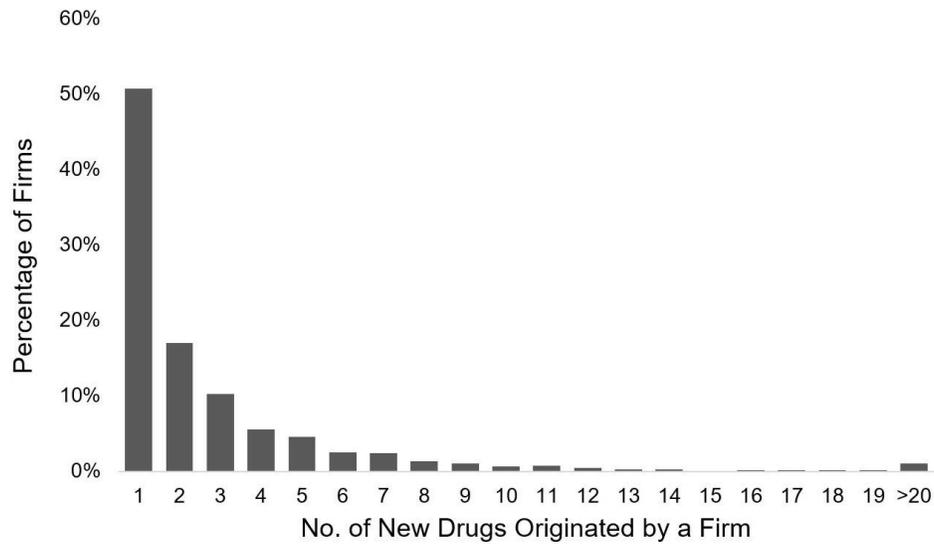
$$PhaseII_i = \beta \cdot I(Acquired)_i \times I(Overlap)_i + \alpha_{FE} + \varepsilon_i,$$

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

	Phase II = 1			
	(1)	(2)	(3)	(4)
		Low Competition	High Competition	Interacted
$I(Acquired) \times \text{Overlap}$	-0.090* (0.049)	-0.260*** (0.086)	-0.085 (0.076)	-0.033 (0.063) -0.189* (0.103)
$I(Acquired) \times \text{Overlap} \times \text{LowCompetition}$				
Observations	1,860	631	1,228	1,860
R-squared	0.548	0.836	0.518	0.561
Phase I start year FE	Y	Y	Y	Y
TC \times MOA FE	Y	Y	Y	Y

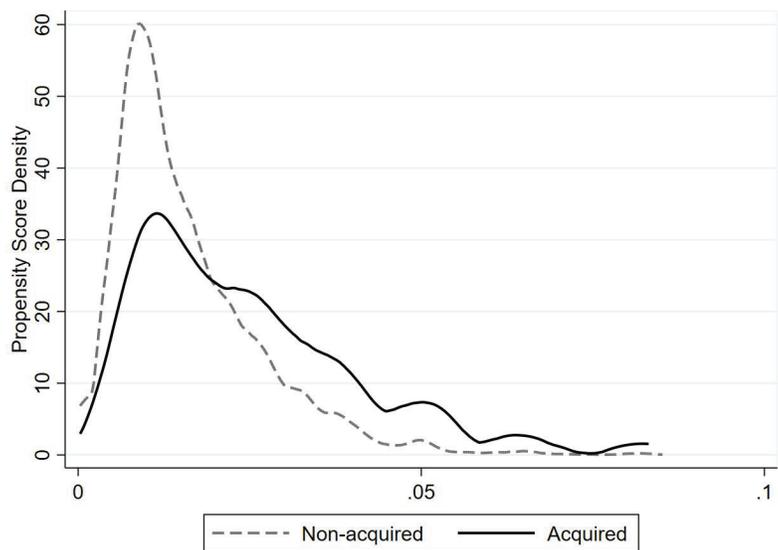
F. Additional Results

Figure F1. Firm Size (Number of New Drugs Originated) Distribution

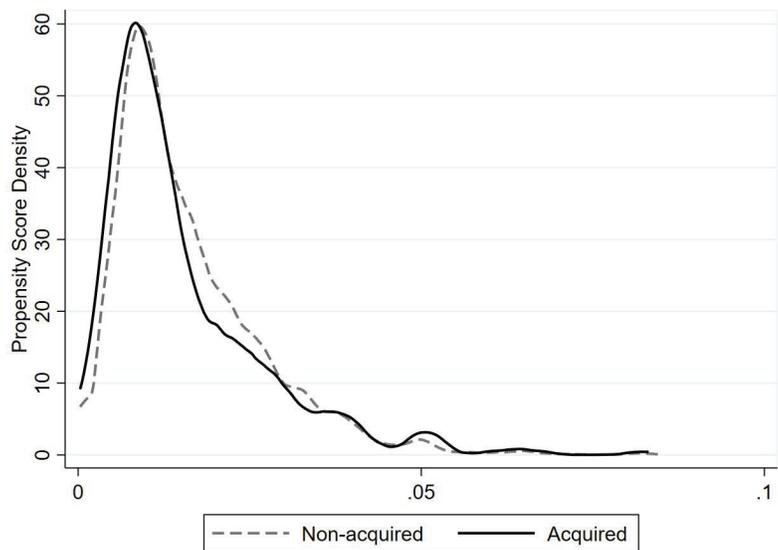


This graph plots the distribution of the number of new drugs originated by a company between 1989 and 2010. We assign a drug to a company if the company was the first to own the drug development project, but we do not assign the drugs that were obtained through acquisitions. The drug origination data are from the Pharmaprojects database.

Figure F2. Propensity Score Distribution: Acquired and Non-Acquired Drugs



(a) Unweighted



(b) Weighted

This graph plots the kernel density distribution of propensity scores for the acquired and non-acquired samples. Panel (a) uses the raw acquisition propensity, and Panel (b) uses the propensity score reweighting method described in section 4.2 and used for results in table 2 column 5.

Table F1
Overlapping Acquisitions and Project Development: Measures of Overlap

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

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

where the dependent variable $Development_{i,t}$ is a dummy variable indicating drug i has a development event in year t . $I(Acquired)_i$ indicates drug i is acquired during the study period, and $I(Post)_{i,t}$ indicates whether the drug-year (i,t) observation is after the drug is acquired. $I(OverlapTC - MOA)_i$ is a dummy variable indicating the acquired drug overlaps with the product portfolio of the acquirer through the same TC and MOA. $I(OverlapTC)_i$ is a dummy variable indicating the acquired drug overlaps with the product portfolio of the acquirer through the same TC only (not same MOA). Standard errors clustered at the drug project level are displayed in parentheses. ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent levels, respectively.

	(1)	(2)	(3)	(4)
	Development Event = 1			
I(Acquired) × I(Post) × Overlap (TC-MOA)	-0.052*** (0.014)	-0.037** (0.015)	-0.036** (0.016)	-0.051** (0.020)
I(Acquired) × I(Post) × Overlap (TC)	-0.046*** (0.012)	-0.018 (0.017)	-0.022 (0.018)	-0.036* (0.021)
I(Acquired) × I(Post)	-0.005 (0.007)	-0.012 (0.009)	-0.010 (0.010)	-0.013 (0.012)
I(Acquired) × Overlap (TC-MOA)	0.009 (0.008)	0.007 (0.009)	0.034** (0.013)	
I(Acquired) × Overlap (TC)	0.013* (0.007)	-0.007 (0.010)	0.015 (0.013)	
I(Acquired)	-0.007 (0.005)	-0.001 (0.006)	-0.015 (0.013)	
Observations	143,569	143,569	143,569	143,569
R-squared	0.037	0.252	0.289	0.366
Vintage FE	Y	Y	Y	
Age FE	Y			
Age FE × TC × MOA		Y	Y	Y
Originator [Target company] FE			Y	
Project FE				Y

Table F2**Overlapping Acquisitions and Project Development: Standard Error Clustering**

This table presents the likelihood of post-acquisition development events for drug projects using a drug-year panel sample, replicating columns 3 and 4 of table 2 but clustering the standard errors at the market (TC-MOA) (columns 1 and 2) and firm-market (columns 3 and 4) levels.

	Development Event = 1			
	(1)	(2)	(3)	(4)
I(Acquired) × I(Post) × Overlap	-0.029* (0.015)	-0.041* (0.021)	-0.029* (0.015)	-0.041** (0.020)
I(Acquired) × I(Post)	-0.017** (0.009)	-0.024** (0.010)	-0.017* (0.009)	-0.024** (0.011)
I(Acquired) × Overlap	0.026** (0.011)		0.026** (0.011)	
I(Acquired)	-0.011 (0.012)		-0.011 (0.012)	
Observations	143,569	143,569	143,569	143,569
<i>R</i> -squared	0.289	0.366	0.289	0.366
Vintage FE	Y		Y	
Age FE × TC × MOA	Y	Y	Y	Y
Originator [Target company] FE	Y		Y	
Project FE		Y		Y

Table F3
Overlapping Acquisitions and Project Development: Overlapping Definition

This table presents the likelihood of post-acquisition development events for drug projects using a drug-year panel sample, replicating table 2. The key difference is the definition of *Overlap*. In this table, an acquired drug is considered overlapping *only* if the acquirer has an existing project in the same TC-MOA as the target *and* the acquirer's project or product is further along in development (measured using age).

	Development Event = 1	
	(1)	(2)
I(Acquired) × I(Post) × Overlap	-0.044*** (0.017)	-0.073*** (0.024)
I(Acquired) × I(Post)	-0.019*** (0.007)	-0.024*** (0.009)
I(Acquired) × Overlap	-0.001 (0.010)	
I(Acquired)	0.002 (0.05)	
Observations	143,569	143,569
R-squared	0.255	0.370
Vintage FE	Y	
Age × TC × MOA FE	Y	Y
Project FE		Y

Table F4
Overlapping Acquisitions and Project Development:
Therapeutic Class Fixed Effects

This table presents the likelihood of post-acquisition development events for drug projects using a drug-year panel sample, replicating table 2. The key difference is the use of age-TC level fixed effects instead of age-TC-MOA level fixed effects.

	Development Event = 1		
	(1)	(2)	(3)
I(Acquired) × I(Post) × Overlap	-0.030** (0.013)	-0.034*** (0.013)	-0.036** (0.015)
I(Acquired) × I(Post)	-0.020*** (0.006)	-0.021*** (0.006)	-0.031*** (0.007)
I(Acquired) × Overlap	0.006 (0.008)	0.016* (0.009)	
I(Acquired)	-0.002 (0.004)	-0.000 (0.010)	
Observations	143,569	143,569	143,569
<i>R</i> -squared	0.067	0.107	0.186
Vintage FE	Y	Y	Y
Age FE × TC	Y	Y	Y
Originator [Target company] FE		Y	
Project FE			Y

Table F5
Overlapping Acquisitions and Project Development: Licensing

This table presents the likelihood of post-acquisition development events for drug projects using a drug-year panel sample, replicating table 2. The key difference is to include controls for any pre-acquisition licensing activity by including the variable, $I(Licensed)$, which indicates the drug has undergone a licensing event or codevelopment deal. To do so, we augment our data with comprehensive RecapIQ data on technology-related codevelopment and licensing deals.

	Development Event = 1		
	(1)	(2)	(3)
I(Acquired) × I(Post) × Overlap	-0.037** (0.013)	-0.033*** (0.014)	-0.040** (0.019)
I(Acquired) × I(Post)	-0.020*** (0.006)	-0.017*** (0.008)	-0.024*** (0.010)
I(Acquired) × Overlap	0.003 (0.008)	0.008* (0.009)	
I(Acquired)	-0.003 (0.004)	-0.005 (0.005)	
I(Licensed)	0.006** (0.003)	0.006* (0.003)	0.015 (0.009)
Observations	143,569	143,569	143,569
R-squared	0.038	0.256	0.370
Vintage FE	Y	Y	
Age FE	Y		
Age × TC × MOA FE		Y	Y
Project FE			Y

Table F6
Overlapping Acquisitions and Project Development:
Matched Non-Acquired Drugs

This table presents the likelihood of not experiencing any development event, replicating table 3. The key difference is that this table includes drugs that are not acquired, whereas table 3 uses acquired drugs. The reason for using only acquired drugs in table 3 is because that analysis uses acquisition events to study differences in (pre- and) post-acquisition likelihood of “never developed.”

To run the same analyses as in table 3 but also to include non-acquired drugs, we matched acquired projects to non-acquired projects (matched based on pre-acquisition development rates up to the acquired project’s acquisition age). We randomly selected three non-acquired projects among those with the same development rates for each acquired project and created “pseudo acquisition” events for non-acquired projects using the matched acquired projects. In the regressions, $I(PseudoAcquired) \times I(Post)$ captures the pseudo acquisition event for all matched, non-acquired projects, and $I(Acquired) \times I(Post) \times Overlap$ captures the coefficient of interest.

	No Development = 1			
	(1)	(2)	(3)	(4)
I(Acquired) × I(Post) × Overlap	0.102*** (0.025)	0.105*** (0.030)	0.120*** (0.041)	0.139*** (0.038)
I(Acquired) × I(Post)	0.033** (0.015)	0.024 (0.018)	0.045* (0.025)	0.013 (0.022)
I(Pseudo Acquired) × I(Post)	0.274*** (0.008)	0.332*** (0.010)	0.355*** (0.014)	0.369*** (0.011)
I(Acquired) × Overlap	−0.023 (0.015)	−0.017 (0.015)	−0.035 (0.028)	
I(Acquired)	−0.002 (0.008)	−0.000 (0.011)	−0.001 (0.018)	
Observations	27,993	27,993	27,993	27,993
R-squared	0.074	0.265	0.368	0.442
Vintage FE	Y	Y	Y	
Acq age FE	Y	Y		
TC × MOA		Y		Y
Acq age × TC × MOA FE			Y	
Project FE				Y

Table F7
Overlapping Acquisitions and Industry-Level Project Development

This table presents TC-MOA-year level analysis drug development of “control” projects as it relates to acquisition activity in the focal market. The sample is drug projects that are neither acquired by an overlapping acquirer nor belonging to the acquirer firm. The analysis investigates if acquisitions of potential competitor drugs increase subsequent development of the non-acquired projects. The empirical specification uses the following model:

$$DevelopmentRate_{j,t} = \gamma \cdot (\#OverlappingAcquisitions)_{j,t} + \alpha_j + \varepsilon_{j,t},$$

where the dependent variable $DevelopmentRate_{j,t}$ is the average number of development events for drug projects in the TC-MOA j in year t . $(\#OverlappingAcquisitions)_{j,t}$ is the number of overlapping acquisitions in the TC-MOA j in year t . ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent levels, respectively.

	(1)	(2)	(3)
	Non-Acquired Project Development		
Number of overlapping acquisitions	-0.030*** (0.007)		
ln(Number of overlapping acquisitions)		-0.082*** (0.014)	
Asinh(Number of overlapping acquisitions)			-0.095*** (0.016)
Observations	70,139	70,139	70,139
R-squared	0.183	0.183	0.183
TC × MOA FE	Yes	Yes	Yes

Table F8

Overlapping Acquisitions and Project Development: Pipeline Competition

This table presents the development likelihood of drug projects replicating the table 4 analyses. The key difference is the measure of competition in this table is “pipeline competition.” The empirical specification uses the following model:

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

where the dependent variable $Development_{i,t}$ is a dummy variable indicating drug i has a development event in year t . $I(Acquired)_i$ indicates drug i undergoes an acquisition event, and $I(Post)_{i,t}$ indicates the drug-year (i,t) observation is after the drug is acquired. We count the number of firms with a drug or drug project that is in the same market (same TC-MOA) as the focal drug. The analysis predicts likelihood of development across all projects in a project-year panel. Drug development projects are categorized into high and low competition by the median of competition measures, which is measured using the number of projects in the pipeline. In column 3 we present results in which we interact $I(Acquired)_i \times I(Post)_{i,t} \times I(Overlap)_i$ with the dummy indicating low competition. Standard errors clustered at the drug project level are displayed in parentheses. ***, **, and * indicate significance at the 1 percent, 5 percent, and 10 percent levels, respectively.

	Development Event = 1		
	(1) Low	(2) High	(3) Interacted
$I(Acquired) \times I(Post) \times Overlap$	-0.083*** (0.030)	-0.012 (0.026)	-0.012 (0.027)
$I(Acquired) \times I(Post) \times Overlap \times LowCompetition$			-0.071* (0.040)
Observations	72,782	70,787	143,569
R-squared	0.425	0.348	0.385
Age FE \times TC \times MOA	Y	Y	Y
Project FE	Y	Y	Y