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Working Paper 18-043



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Funding for this research was provided in part by Harvard Business School.

# Trials and Terminations: Learning from Competitors' R&D Failure

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November 22, 2019

## Abstract

I analyze project continuation decisions where firms may resolve uncertainty through news about competitors' research and development (R&D) failures, as well as through their own results. I examine the trade-offs and interactions between product-market competition and technological learning from parallel R&D projects. Leveraging the biopharmaceutical industry's unique characteristics to overcome barriers to measuring project-level responses, I employ a difference-in-differences strategy to evaluate how competitor exit news alters a firm's own project discontinuation decisions. The findings reveal that technological learning dominates competition effects. Firms are most sensitive to competitor failure news from within the same market and same technology area—more than doubling their propensity to terminate drug development projects in the wake of this type of information. Finally, I explore how levels of competition, uncertainty, and opportunities to learn moderate the response to competitor failure news.

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\*I am thankful to Pierre Azoulay, Alessandro Bonatti, Fiona Murray, and Scott Stern for their invaluable guidance and comments throughout this project. I am also grateful for insightful conversations with Michael Bikard, Daniel Fehder, Roberto Fernandez, Lee Fleming (discussant), Jeff Furman, Bob Gibbons, Matt Higgins, Brian Hubbard, Danielle Li, Manuel Hermosilla, Marvin Lieberman (discussant), Kyle Myers, Abhishek Nagaraj, Ramana Nanda, Tom Nicholas, Ray Reagans, Tim Simcoe, Chris Stanton, Ariel Stern, Carolin Haussler (discussant), Heidi Williams, and Yufei Wu. Many other colleagues and seminar participants at the NBER Summer Institute (Innovation; Aging), various MIT seminars, the Munich Summer Institute, the Roundtable for Engineering Entrepreneurship Research (REER), and the NBER Productivity Lunch also provided very helpful comments. Amanda Liu, Descartes Holland, and Matt D'Amico provided excellent research assistance. Research reported in this publication was supported by the Kauffman Foundation and the National Institute on Aging under Award Number R24AG048059 to the National Bureau of Economic Research. The content is solely the responsibility of the author and does not necessarily represent the official views of the National Institutes of Health or NBER.

# 1 Introduction

How should a firm respond to a competitor’s product development failure? On the one hand, the loss of a competitor is positive news, leaving greater potential market share for the remaining players. On the other hand, a rival’s failure might contain important, cautionary information about technological roadblocks that limit the likelihood of success. Interpreting these types of competitor outcomes is a key consideration for firms making capital-intensive investments in oligopolistic settings. Yet, empirical studies of research and development (R&D) spillovers have not addressed how competitive and technological pressures influence project investments in the wake of competitor failure.<sup>1</sup>

In this paper, I examine project-level spillovers and tease apart the different types of information contained in a competitor’s project exit: (i) knowledge spillovers, (ii) product-market competition effects, and (iii) the combination of both. Empirically, I evaluate how biopharmaceutical firms alter their project investments following competitors’ clinical trial failures. I measure how different types of competitor news (same vs. different *market*, same vs. different *technology*) influence the likelihood that a firm “pulls the plug” on its own drug development project. To overcome barriers to measuring the project-level response to competitor discontinuation news, I use unique features of the pharmaceutical R&D setting, including the observability of development milestones, the staggered timing of entry and outcomes, and the separability of product markets and technologies. I find that technological learning dominates competition effects.

Before investigating empirically, I develop a theoretical framework that adds learning from competitors’ R&D exits to the investment decision. To do so, I evaluate R&D investments as real options, combined with the possibility of both payoff and knowledge externalities. In this framework, the extent to which firms update their beliefs following competitor exits depends on how the “focal” project relates to the failed competitor project: *same market, different technology* (SM-DT); *same market, same technology* (SM-ST); or *different market, same technology* (DM-ST). I describe a belief updating process in which the interaction of these two effects need not equal the sum of the separate market and technology responses. Furthermore, the framework suggests that the project’s level of competition and uncertainty should moderate how firms respond to competitor exits. Last, I predict that competition makes a project *more* attractive when the focal and competitor projects share the same technology. Such competitors provide additional signals about the technology’s underlying quality—effectively lowering the cost of experimentation.

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<sup>1</sup>See Griliches (1991), Cockburn & Henderson (1996), Bloom et al. (2013), Schnitzer & Watzinger (2015), Manresa (2016), Colino (2017), and Lucking et al., (2018) for examples of how R&D successes create spillovers at the *firm*-level.

The choice of an empirical setting is crucial for R&D spillover analyses. While innovation scholars have argued that disclosure and the ability to assimilate external information have positive effects on the rate of innovation (Henderson & Cockburn, 1994; Cockburn & Henderson, 1994; Ederer, 2013; Bloom et al., 2013; Boudreau & Lakhani, 2015), we have little evidence on whether and when competitors respond directly to each other’s R&D project outcomes.<sup>2</sup> One reason for the scant empirical work is that making valid inferences about competitor spillovers requires a special type of setting. The analyst must be able to observe the market structure and entire set of projects, track the timing of project development and failure events, and follow the developers’ subsequent choices. Further, a suitable setting must involve project continuation decisions with large capital expenditures, so that the corresponding investments have the potential to shift firm performance. The timing of failure news must be surprising and disclosed promptly and publicly, generating well-defined decision points and learning opportunities (i.e., signals from competing projects). Perhaps most importantly, the setting must provide metrics to assess technological and product market relatedness between competing projects. Without these ingredients, one cannot isolate knowledge spillovers and competition effects.

The pharmaceutical drug development setting is uniquely well-suited to fulfill these requirements because its regulatory structure and disclosure environment generate observable project-level data in the context of high-stakes decisions. Project selection and termination decisions are central to firm performance in drug development. Failure is a frequent occurrence and interpreting competitor outcomes is a game played by executives, scientists, journalists and investors alike.<sup>3</sup> In an instant, a failure event can shift the frontier of technical knowledge as well as the competitive landscape for the remaining players. Additionally, drug development provides an ideal laboratory for studying R&D spillovers because human trials are typically the last stop in resolving scientific uncertainty about a compound or therapeutic hypothesis. Outside of trial results, common shocks are rare due to the slow-moving nature of disease markets and translational science. Finally, strong intellectual property protection, long development timelines, and the high cost of trials creates a racing environment with substantial capital commitments and very little incentive to “wait and see” how competitors fare (Schulze & Ringel, 2013).

I use this structured and highly competitive setting to evaluate how different types of competitor failure news influence project termination decisions. I construct a dataset that comprises all clinical development projects over the period 1997-2014, along with each project’s development milestones.

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<sup>2</sup>Economists have studied how events like bankruptcies and product recalls influence stock market valuations of competitor firms (Lang & Stultz, 1990; Ahmed et al., 2012), or sales performance of related products (Freedman et al., 2010). However, these studies focus on third-party valuations and market demand, rather than competitors’ own investment decisions. Closer to the setting of this study, Magazzini et al. (2012) evaluate how subsequent drug developers build off the knowledge generated by previously failed vs. successful drug candidates, by comparing citations to patents associated with those drug candidates.

<sup>3</sup>Tracking rival projects has grown into its own profitable business with considerable resources and time spent on external competitive intelligence databases and pipeline consultants.

I focus on projects undergoing phase II clinical trials, which are typically the first major test of a drug’s effectiveness and safety (vs. a placebo or standard of care). I estimate the effect of competitor news on project termination patterns by linking each focal project to its competitors’ project exit events, and by using a difference-in-differences survival model approach to control for the typical life cycle of development projects. I distinguish between product-market and technological competitors, and take advantage of variation in the (plausibly exogenous) timing of entry and failure to identify the effect of competitor failure news on project exit rates.

Using this setup, I show how the relationship between competitors across both market and technology categories dictates how competitor failure news affects the focal firm’s decision to abandon R&D projects. While the average effect of any type of competitor failure news is negligible, same technology competitor failure news results in a 23% jump in focal projects’ exit rates. More specifically, the combination of same market and same technology (SM-ST) competitor failures leads to the largest increase (more than doubling) in project exit rates, while DM-ST competitor discontinuations results in a smaller (18%) increase in exit rate. This difference between the two same technology groups suggests that disease-specific knowledge spillovers dominate the positive effects of reduced competition. On average, news of SM-DT competitor failure does not impact project survival rates.

I test the additional theoretical predictions by evaluating key subgroups of competing projects. I find that only when the level of competition is low does SM-DT failure decrease other companies’ propensity to exit. Using focal projects in the final stage of trials, I show that more advanced projects are less sensitive to all types of competitor news. Finally, I explore how having greater technological learning opportunities (i.e., more ST competitors) influences project investments and reactions to competitor news. The results are consistent with firms being more willing to test more risky (likely to fail) projects when projects have more same technology competitors. Furthermore, firms that have more competitor learning opportunities are more likely to continue following SM-DT news, and less likely to exit following DM-ST news. Overall, the evidence supports a model in which more ST competitors *increase* project value at the margin, and firms privately benefit from herding into SM-ST R&D races.

To ensure that the main empirical strategy captures responses to competitor news, rather than the independent failure of competing projects with a “shared fate,” I employ a variety of robustness checks. Using subsamples and alternative variable definitions, these analyses further isolate competitor information effects by focusing on projects less likely to have “died of natural causes.” Section 5.5 describes these robustness checks, which together suggest the results are not driven by simultaneous and independent competitor exits.

The main results highlight the importance of both separating and interacting the different dimensions of R&D competition when evaluating spillovers. Moreover, a simple back of the envelope

calculation shows that “turning off” the competitor learning channel might have resulted in 1,683 (3.7%) additional quarters of active phase II clinical trials—suggesting the reallocation of more than two billion dollars in R&D investments attributable to competitor learning.

Both the theoretical and empirical analyses focus on *project*-level spillovers of competitor exit. By measuring firm-level performance outcomes and cumulative failure experience, prior studies are not able to capture when rivals’ outcomes directly enter project investment decisions.<sup>4</sup> My approach uses a crucial distinction between product-market competitors and technology competitors first applied by Bloom et al. (2013), who separate the countervailing knowledge and competition effects at the *firm* level. This paper makes a distinct contribution to the R&D spillovers literature by applying these market and technological competitor distinctions at the project level, and adds one additional layer: their interaction effect. By allowing technological spillovers to vary depending on the particular product-market application, the econometric results illuminate whether different types of project spillovers are equally informative.

This paper also contributes to the literature studying real options. Prior work highlights how real options provides flexibility to experiment and explore more uncertain paths (Dixit & Pindyck, 1994; McGrath, 1997; Grenadier, 1999; Miller & Folta, 2002; McGrath & Nerkar, 2004; Adner & Levinthal, 2004; Manso, 2011; Nanda & Rhodes-Kropf, 2016), but does not capture cross-competitor learning and spillovers. This paper shows how competitor news can be an essential component of real options valuation because competitor failures resolve both market and technological uncertainty.

The paper proceeds as follows. I begin with the theoretical predictions that combine insights from real options theory with competition and knowledge spillovers. Next I discuss the drug development setting and how learning from failure plays out in the pharmaceutical industry. Third, I describe the main empirical approach and results. By comparing the competitor responses across projects in different competitive and technological contexts, I test additional predictions from the theoretical framework and validate robustness. Finally, I discuss the implications of the results and conclude.

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<sup>4</sup>Bennett & Snyder (2017) demonstrate how focusing on cumulative success and cumulative failure leads to biased estimates of learning. Two recent working papers apply variants of measuring cumulative (failure and success) experience within the context of drug development (Garzon-Vico, 2012; Rao, 2017). However, their focus is not on the temporal dynamics of failure entering competitors’ decisions, or separating market and technological forces. This paper focuses on how the most recent failure events impact organizational actions, rather than probability of success.

## 2 Theoretical Framework

### 2.1 R&D Projects as Real Options

How should firms update R&D investment decisions in response to changing information about competitors? I analyze these decisions by adding competitor learning into a real options framework. This framework builds on prior work that describes option value of experimentation in entrepreneurship (e.g., Nelson, 1961; Kerr et al., 2014; Nanda & Rhodes-Kropf, 2016; Manso, 2016), while adding the prospect of both payoff and information externalities.<sup>5</sup> Unlike these prior approaches, here firms must evaluate uncertainty regarding if and when competitor information will be revealed, as well as the relevance of technological or market signals. While existing real options models (e.g., Dixit & Pindyck, 1994; Grenadier, 1999; Kellogg & Charnes, 2000; Kellogg, 2014, Décaire et al., 2019) recognize that flexibility is a major source of investment value, they do not allow competitor outcomes to separately change beliefs about “technical risk” (probability of moving on to the next stage) and expected payoffs (conditional on technical success).

In addition to drug development, the analysis below is relevant for other industries with high project uncertainty, large capital commitments, correlated technological outcomes (i.e., competitors testing related hypotheses), publicly observable actions and outcomes, and potential payoff externalities. These production settings will also have well-established demand.<sup>6</sup> Outside of life science-based businesses, applicable settings might include the automotive industry, mineral exploration, energy production, aerospace technology, venture capital investing, and medical devices.

### 2.2 Structure of the Game

Traditional real options models represent decision points as pre-specified opportunities to reevaluate an investment occurring at some regular interval (i.e., monthly valuation, annual planning meeting, etc.). An option is more valuable when the investment has more opportunities to reevaluate (more flexibility), and more volatility between those intervals. I add to that general framework by considering competitor news as a type of belief updating that might occur *in-between* those traditional stages—making all stages (potentially) more valuable. I define investments as a single experiment or “stage” of R&D project (e.g., phase II clinical trial), and consider a firm’s choice to

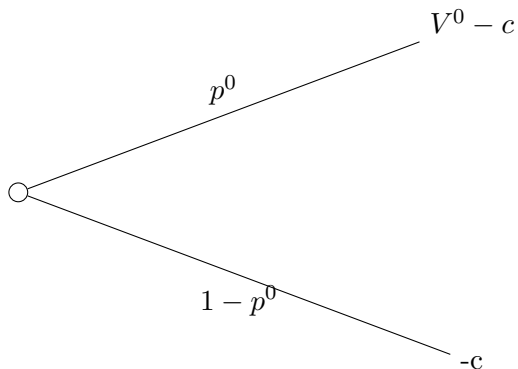
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<sup>5</sup>This approach also relates to models of strategic experimentation (Malueg & Tsutsui, 1997; Keller et al., 2005; Akcigit & Liu, 2015; Bryan & Lemus, 2017; Bonatti & Horner, 2017) and the analysis of static auctions with correlated signals (Dasgupta & Maskin, 1987; Kagel et al., 1987; Hendricks & Porter, 1988). In contrast to prior work, I do not assume that no news (about a rival’s project) is bad news. In the clinical trials setting, no news might actually be a positive sign (about the underlying technologies), because failures are hard to hide.

<sup>6</sup>If a product is the first of its kind, then failure signals may include information about demand, as well as information about the technology and competition. For example, when Google discontinued sales of its controversial wearable device Google Glass, the failure might have conveyed as much about consumer preferences and marketing as the technology itself.



Figure 1: Traditional Single Stage R&D Valuation



stop or continue any given “focal” project. A simple way to view this choice would be to estimate the expected probability of success ( $p^0$ ), cost ( $c$ ), payoff conditional on success ( $V^0$ ), and outside option ( $\bar{V}$ ). The firm will then choose to invest if the expected value is greater than the reservation value:

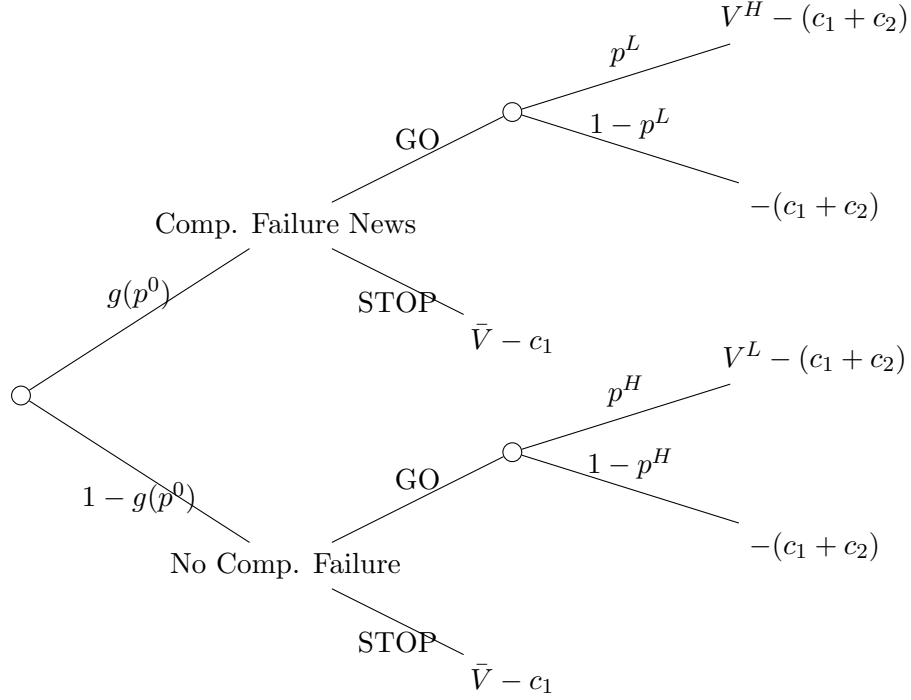
$$p^0(V^0 - c) - (1 - p^0)c > \bar{V}. \quad (1)$$

This simple approach is attractive since the firm need only generate predictions for it’s own projects (based on historical averages and/or extant experimental and market data), and wait for the next round of experiments to finish.

Next, I incorporate the possibility of vicarious learning into the model. In a standard model, more (expected) competitors would have a strictly negative affect on perceived project value (lower  $V^0$ ). Here, competitor signals introduce a countervailing force: more competitors increase the likelihood of receiving competitor news and resolving uncertainty earlier. In particular, competitor failures create new decision points—allowing firms to abandon their project early and recover  $\bar{V}$ , rather than continuing to spend on a project that was unlikely to be profitable. For illustration in Figure 2, I consider the competitive scenario with only two projects competing for the same market, both of which share the same underlying technology (i.e., SM-ST competitors).

The timing in this alternative setup is as follows. Rather than committing the entire investment amount  $c$  at the start of the stage, the focal firm invests  $c_1$  to enter the stage, where  $c_1 + c_2 = c$ . The focal firm expects to get a failure signal (“bad news”) from the competitor with probability  $g(p^0)$ , which is decreasing in  $p^0$  because the competitor failure probability is positively correlated with the focal project’s success. If competitor failure is realized, then the focal project reaches a new intermediate decision point where the firm can stop and recover  $\bar{V} - c_1$ , or continue at cost  $c_2$

Figure 2: Single R&D Stage with Competitor Learning



and face an updated gamble, with lower probability of success ( $p^L < p^0$ ) and a monopoly payoff ( $V^H > V^0$ ). If competitor failure signals do not arrive, the focal firm still reserves the option to stop the project and recover its opportunity cost of continuation. However, no signs of competitor failure also result in updating of a different type, where beliefs about the likelihood of the success increase ( $p^H > p^0$ ) and expectations shift closer to oligopoly competition ( $V^L < V^0 < V^H$ ). It is due to this decreased payoff expectation (conditional on technical success) that the firm may decide to drop out after no competitor news.

In order to compare this decision tree to the simple valuation in Equation (1), the continuation paths (“GO”) must yield an expected value equivalent to the gamble in Equation (1):

$$\begin{aligned}
 E[V|GO] &= g(p^0) \times p^L(V^H) + [1 - g(p^0)] \times p^H(V^L) - c \\
 &= p^0(V^0) - c
 \end{aligned}
 \tag{2}$$

By allowing the termination option (“STOP”) under this alternative game structure, the investment’s valuation is more complicated, but potentially higher:

$$\begin{aligned}
E[V] = & g(p^0) \times \underbrace{\max[(p^L V^H - c) - (1 - p^L)c, \bar{V} - c_1]}_{\text{Updated Payoff Post Comp. Failure}} \\
& + [1 - g(p^0)] \times \underbrace{\max[(p^H V^L - c) - (1 - p^H)c, \bar{V} - c_1]}_{\text{Updated Payoff w/o Comp. Failure}}
\end{aligned} \tag{3}$$

The project is weakly more attractive in Equation (3) than in Equation (1) because of the option value generated by competitor learning. Either of the two scenarios (competitor fails, or no competitor fails) has the potential to be a more attractive bet than the version with no competitor learning. Meanwhile, if either scenario generates a less attractive gamble, the firm can abandon the project early—saving both direct costs ( $c_2$ ) and opportunity costs ( $\bar{V}$ ). The updating opportunity from competitors effectively creates an additional stage of the game and adds flexibility due to the abandonment option.<sup>7</sup>

This logic extends to cases with more than one competitor. More same technology competitors increase option value while potentially limiting expected payoffs (if they also have the same target market). As the number of expected market competitors ( $q$ ) increases, three aspects of the model are exaggerated. First, the probability of competitor failure news goes to  $g(p^0, q)$ , which is decreasing in  $p^0$  but increasing in  $q$ . The more competitors, the higher the chance that one fails. Multiple potential competitor failures create additional intermediate branches in the decision tree (e.g., Competitor A fails then Competitor B fails; Competitor A fails, but no news from Competitor B; no competitor failure news from Competitor A or Competitor B). Due to the increased expectation of competitor news, the relative amount of belief updating on  $p$  (following competitor failure news) increases in  $q$  for “no news” and decreases in  $q$  for competitor failure news.

Second, more competition increases the chance of learning *early* and increases the value of the stopping option since the direct costs incurred are lower the earlier one drops out (i.e.,  $c_1$  becomes smaller).<sup>8</sup> Third, belief-updating about payoffs ( $V$ ) should be less sensitive as  $q$  increases. When a market competitor drops out, the expected number of competitors decreases (e.g.,  $q - 1$ ) and the potential payoff may increase. Since the impact of less competition on profits is greater as the market approaches monopoly (Bresnahan & Reiss, 1991), the increase in continuation value will be greater if the focal project has fewer competitors at baseline, and the effect may be negligible if competitors are numerous.

Thus, the exact value of competitor learning depends on the correlation between competitor signals as well as the level of competition. If project quality is uncorrelated across competitors

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<sup>7</sup>All else equal, firms prefer cheaper experimentation and more information about their technology’s potential—all of which they effectively get from technologically similar competitor news. That preference also implies more entry (“herding”) into more crowded technological areas, on the margin. Such entry would be privately optimal for the firm, but might not be socially optimal if it reduces the diversity of technological experimentation.

<sup>8</sup>Similar to an American put option on a stock, the investor gets more option value when volatility is higher, and the investor can sell early when the expected value of the asset decreases.

and the number of competitors is quite high, then dropout signals won't lead to to much (if any) updating, and the firm is effectively back to using Equation (1). So understanding the updating process is essential to pricing the real option with competitor learning.

### 2.3 Updating Process

A firm's initial expectations about an R&D project's technical success ( $p^0$ ) reflects its belief that the technology will succeed in a given product market application ("the hypothesis"). That belief is formed by three components: 1) a common (shared) belief about the performance of technology  $j$  for application  $k$  based off public knowledge (e.g., scientific publications on the role of pathway X for causing disease Y; consensus beliefs about the strength of material Z at high temperatures), 2) common beliefs about the reliability and general usefulness of technology  $j$  for *any* application, and 3) a private signal that is specific to the focal project and formed from proprietary information and experiments with one's own intellectual property. The private belief is positively, but not perfectly correlated with the common components:

$$p_{ijk}^{private} = \beta \times D_{jk}^{common} + \gamma \times D_j^{common} + \delta \times D_{ijk}^{private} \quad (4)$$

Here,  $i$  indexes the specific project in a given stage,  $j$  is the technology, and  $k$  is the product-market application.  $D_{jk}^{common}$  and  $D_j^{common}$  are scores representing the shared knowledge about the promise of technology  $j$  for application  $k$  specifically, and of technology  $j$  more generally.  $\beta$  and  $\gamma$  are both positively correlated with project-specific beliefs, and  $\beta > \gamma > 0$  due to the additional market-specific relevance of  $D_{jk}^{common}$ . Competitor news (or lack thereof) changes  $p_{ijk}^{private}$  because of the Bayesian updating process applied to  $D_{jk}^{common}$  and  $D_j^{common}$ . Thus, firms will only update  $p_{ijk}^{private}$  when relevant competitors shares the same technology, and will update more when the competitor shares the same technology and market application.

$\delta \times D_{ijk}$  represents the firm's idiosyncratic private information that may diverge from the common signals.  $D_{ijk}^{private}$  moves up and down depending on the strength of (internal) experimental safety and efficacy results (e.g., known side effects).  $\delta$  is positive and higher (relative to  $\beta$  and  $\gamma$ ) for more mature projects. For example, as scientists gather more of their own proprietary evidence, they place more weight on their own results and less on common and competitor signals. If a late-stage project has lots of internal test results and a particularly high or low  $D_{ijk}^{private}$ , then competitor news (good or bad) will not lead to a qualitatively different private posterior belief. Furthermore, firms might have higher  $\delta$ , regardless of project stage, based on their perceived expertise in running experiments, choosing applications and assimilating external knowledge.

In parallel, firms adjust their beliefs about the expected level of market competition ( $q$ ):

$$q_{i,k} = q_{j,k} + q_{-j,k} \quad (5)$$

When a competing project that was targeting the same product market ( $k$ ) as the focal project ( $i$ ) drops out, the expected number of competitors,  $q_{ik}$ , decreases and the potential payoff,  $V(q)$ , increases. Since the impact of less competition on profits is greater as the market approaches monopoly, the increase in expected rewards will be greater when the focal project has fewer competitors at baseline, and the effect may be negligible if competitors are numerous. In other words, I assume  $V(q)$  is a decreasing convex function of the number of competitors:  $V'(q) < 0, V''(q) > 0$ .

Generalizing to scenarios with multiple competitors (of different types), baseline payoff expectations are a function of both the number of R&D competitors targeting the same market (i.e., SM-DT and SM-ST) and priors about the probability of success for those projects (e.g.,  $p_{jk}, p_{j'k}, p_{j''k}$ , etc.). If the exiting competitor is targeting a different product market, then the expected competition is unchanged. Regardless of whether the failing competitor shares the same technology as the focal project, the payoff updating should be the same: increased expected payoff following competitor failure news ( $V^+$ ) and gradually decreasing expected rewards in the absence of failure news ( $V^-$ ).

The updating process following a competitor failure can be summarized in the below 2x2's for the scenarios with and without a competitor failure:

Figure 3: Updating

		<u>Competitor Failure News</u>		<u>No Failure News</u>		
		Product Market		Product Market		
		<i>SameMkt</i>	<i>DiffMkt</i>	<i>SameMkt</i>	<i>DiffMkt</i>	
Tech.	<i>SameTech</i>	$p^{--}, V^+$	$p^-, V^0$	Tech.	$p^{++}, V^-$	$p^+, V^0$
	<i>DiffTech</i>	$p^0, V^+$	$p^0, V^0$		$p^0, V^-$	$p^0, V^0$

## 2.4 Empirical Predictions

Applying this general framework to the empirical setting of drug development yields four main predictions.

**Prediction #1:** *The impact of competitor failure news on project continuation decisions will differ based on both product-market and technology similarity.*

As described above, belief updating should be stronger when the competitor shares the same technology and market ( $\beta + \gamma > \gamma$ ). Therefore, reactions to SM-ST competitors will not equal the sum of the separate responses for pure market competitors (SM-DT) and pure technology competitors (DM-ST).

To empirically characterize competitor responses, one cannot simply lump all types of competitors together. If projects have more same market than same technology competitors, or vice versa, then grouping all failure types together will produce misleading results. Comparing competitor effects using the two separate dimensions (market and technology) is consistent with prior work (Bloom et al., 2013). However, the simple two-way split is also insufficient. The value of learning from same technology competitor results may be context-dependent. Differences in product-market applications should moderate the relevance of competitor signals ( $\gamma < \beta$ ). For example, doctors might be willing to accept nasty side effects for some, but not other, diseases and patient populations (e.g., children vs. adults, slow vs. fast progressing diseases). Both safety and efficacy standards may vary across diseases. Therefore, failure in one market application (disease) does not necessarily rule out the drug for other conditions.<sup>9</sup>

However, the additional knowledge effects associated with SM-ST competitor failures do not necessarily overpower the “good news” of one less competitor. Ultimately, the net response to SM-ST competitor failures is an empirical question. Comparing SM-ST, SM-DT and DM-ST competitor responses will reveal how much market-specific factors moderate technological learning.

**Prediction #2:** *The extent of competitor responses to failure news will vary with the level of competition.*

High levels of market competition can greatly diminish the payoff externalities of competitor failure. In markets with many existing products and/or intense R&D competition for the next generation products, losing a single pipeline competitor does little to the expected rewards of success. This prediction follows from the assumption that payoffs are a decreasing convex of competition.

**Prediction #3:** *Competitor learning is only relevant when the degree of project uncertainty is relatively high.*

Competitor signals are only useful for belief updating when firms still have a great deal of uncertainty about their own project’s quality (i.e.,  $\frac{\delta}{\beta+\gamma}$  is relatively low). After many stages of testing and data gathering, firms may have strong (or precise) beliefs about their own project’s quality. If the firm holds strong positive beliefs in these late stages, then it will likely view ST competitor

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<sup>9</sup>The same could be true in other contexts. For example, the reliability and stress tolerance needs of materials is different for aerospace than it is in bicycle manufacturing. So failure of a new material to meet the standards in one area does not preclude the introduction of this technology for other transportation uses.

failures as idiosyncratic to their competitor’s approach, rather than as a signal about the true quality of its own technology.

**Prediction #4:** *All else equal, firms will be more likely to continue projects when the project has more remaining same technology learning opportunities.*

These learning opportunities effectively speed up experimentation by resolving some uncertainty earlier. Competitor failures provide new decisions points where firms can cut losses and abandon projects. To measure the magnitude of ST learning opportunities’ value, the ideal experiment would suddenly introduce more ST competitors for a randomly selected set of R&D projects.<sup>10</sup> Unfortunately, that type of experiment is not feasible, so I am limited to an indirect approach. Empirically, I assess how termination decisions correlate with learning opportunities (see Section 5.4.3 and Table 4). I test whether firms are more likely to advance risky projects into phase II when ST learning opportunities are high. Next, I compare how low vs. high levels of remaining competitor learning opportunities affect responses to competitor failure. The theory suggests that continuing will be more attractive when additional ST competitors are likely to provide more experimental signals.<sup>11</sup> These additional future data points increase the project’s option value, and might reduce the weight on any one (potentially noisy) competitor signal because additional signals are likely to arrive.

## 3 Setting

### 3.1 The Drug Development Process

Drug development is a multi-stage journey with a regulatory process consisting of preclinical experimentation and clinical trials. Trials are designed to test a drug for a particular disease (indication) in humans, and developers may initiate trials for different indications (either sequentially or in parallel).<sup>12</sup> I study both small molecule drugs and large molecule drugs (biologics) but exclude vaccines, which undergo a different regulatory and clinical testing process. Drug development typically begins with lab (*in-vitro*) and animal testing before drugs reach three sequential phases of human clinical trials. Appendix A provides more detail about these development phases.

The focus of this study is the firm’s decision to stop development for drugs that have progressed into phase II trials, which serve as a drug’s first real test of both safety and efficacy in humans.

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<sup>10</sup>For the factors described above, even better would be randomly airdropping SM-ST competitors for some projects and DM-ST competitors for others.

<sup>11</sup>The low vs. high remaining learning opportunities split can only indirectly measure option value because entry is endogenous. The presence of more same technology competitors may reflect high hopes for a particular new technology solution. In my empirical analyses, I control for total entry into market and technology areas, but cannot create the ideal experiment with exogenous shocks to both entry and exit.

<sup>12</sup>I refer to the development of a compound for a given indication as a drug “project.”

These trials may last months or years,<sup>13</sup> may involve several hundred people who have the condition (disease) of interest, and typically require randomized and blinded assignment into a control group “treated” by a placebo or the existing standard of care. Data safety monitoring boards—made up of independent scientific, medical, and statistical experts—are assigned to review intermediate results and stop trials early when they deem it unsafe and/or unethical to continue. A great deal of uncertainty remains when starting phase II trials: about 32% of phase II projects will progress to phase III, and only 16% will make it to FDA approval (Hay et al., 2014).

The competitor signals are rival project failures in either phase II or phase III trials. In principle, information about projects in earlier stages of development is not relevant for rival projects that have already cleared earlier safety and efficacy hurdles. The analysis focuses on competitor failure events that happen prior to FDA review. This limitation ensures that the information conveyed in the discontinuations does not include any direct signals about the regulator’s level of scrutiny (Blankshain et al., 2013).

Drug development projects fail to reach approval for a variety of reasons. Safety and efficacy concerns make up the vast majority of clinical trial project closures. Cook et al. (2014) studied 142 drug development projects at AstraZeneca and investigated reasons for failure. The study found that about half of clinical trial safety failures were related to the drug’s primary biological target, while the other half of safety failures were attributable to off-target side effects.

The statistical signals from a project’s own phase II trial may be noisy and the tradeoffs between safety and efficacy are not easy to balance. Therefore, even after a project’s own trial finishes and the results are un-blinded for review, the decision to continue or halt development can be complicated. Information about rival projects may be particularly relevant for such marginal projects.

### 3.2 Disclosure

Due to disclosure requirements, firms are well-aware of competing projects’ progress. Early entry is disclosed through a combination of patent filings, scientific publications or company documents. These disclosures usually reveal a drug compound’s key features, including its molecular mechanism of action (if known) and potential therapeutic uses. Once the company has completed preclinical investigations of a drug compound, it must file an investigational new drug application (IND) with the FDA before starting human clinical trials. Various policies also require firms to disclose clinical trial information, including the drug compound and disease application, by pre-registering in public trial registries like the National Library of Medicine’s [clinicaltrials.gov](https://clinicaltrials.gov).

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<sup>13</sup>The length of a trial depends on the disease and endpoint (e.g., mortality, blood pressure, tumor growth, etc.) of interest. Some diseases might take longer to progress and require years of monitoring to infer a therapy’s efficacy. The median time spent in phase II trials is two and a half years.



The decision to halt a drug development project is one that affects potential consumers, employees, investors, and competitors. Firms reveal discontinuation news through a variety of mechanisms and with different degrees of detail. The shut-down decision is most often reported in company press releases, updated drug development pipeline documents (usually posted on the firm’s website), and financial filings. Competitive intelligence services monitor progress and these discontinuation disclosures—alerting subscribers to new disclosure events.<sup>14</sup> These announcements contain statements about the events leading up to the decision, and only a small fraction of the discontinuation announcements are preceded by premature clinical trial terminations. When the rationale for discontinuation is disclosed, the most commonly cited reasons for stoppage are (disappointing) efficacy and safety issues. On a few occasions, termination announcements cite disappointing results from competitors’ projects as a reason for stoppage (see IDO example below). Appendix A presents examples of discontinuation disclosure statements that have different levels of transparency.

One empirical concern is that firms of different sizes or experience have different incentives to publicly report their trial failures (see Appendix A for a discussion of materiality and Regulation Fair Disclosure). However, I find that official discontinuation rates are similar for relatively large and small firms alike. In the analysis sample, large firms that had ever developed 10 or more projects officially terminate 33% of phase II projects, vs. 29% for smaller firms. I also do not find much of a difference in firm experience/size for how often phase II drugs turn into “zombie” projects (those never officially discontinued but also never advanced).

### 3.3 The Ripple Effects of Trial Failures

Trial failures may have repercussions throughout the industry. With great uncertainty surrounding cutting-edge drug trials, competitors anxiously await news about relevant rival projects. For example, in the long quest to find an effective Alzheimer’s treatment, a number of firms have pursued  $\beta$ -secretase (BACE) inhibitors, aimed at reducing the production of amyloid plaques in the brain. In October 2016, leading up to Eli Lilly’s expected announcement of their BACE inhibitor (solanuzemab) phase III trial, *STAT News* described how a late-stage trial success “would go a long way in validating the idea that amyloid plaques are integral to disease progression, bolstering the odds of success for Biogen, Merck, Roche, and others working in the same [class of drugs].” However, the article also noted that poor results could “have a chilling effect on other drug developers targeting amyloid plaques,” and that a single late-stage failure could have “ripple effects in other companies,” who might question the direction of their own drug development approach (Garde, 2016).

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<sup>14</sup>This paper’s primary data source, Cortellis, tracks these disclosures and links them to drug development projects (drug-indications) identifiers and company information.

A recent high-profile failure in the burgeoning field of immuno-oncology had a very public influence over rivals' investments. In the Spring of 2018, pharmaceutical company Incyte announced the failure of its cancer drug, epacadostat, an indoleamine-pyrrole 2,3-dioxygenase (IDO) inhibitor. To map the epacadostat example onto the terminology of this paper: IDO is the drug's primary "technology" and Incyte was developing it for a variety of therapeutic markets (melanoma, glioblastoma, ovarian cancer, and others). Following the epacadostat failure news, NewLink Genetics stopped a melanoma trial of their own IDO inhibitor (i.e., a SM-ST project), explaining the decision as made "in the context of the failure of a competitor's trial of its enzymatic IDO inhibitor in a similar clinical setting." Bristol-Myers followed suit, pulling the plug on three different trials it was sponsoring using its own IDO inhibitor, which the company had acquired in a \$1.25 billion acquisition. Bristol-Myers changed the status of its trials on clinicaltrials.gov and credited the "emerging data on the IDO pathway" as their motivation for closing trial registration early.<sup>15</sup>

Yet, the signal from competitor exits is not always clear. Interpreting trial results can take months or years, and rival firms might not have much information to work with after the initial disclosure. Scientific publications with the detailed results can provide more guidance. For example, after a safety failure, analyzing the trial data might reveal if the drug was unable to properly interact with a molecular target, or if the safety issues were the result of collateral damage ("off-target" effects). However, firms need not publish their findings (reporting requirements are scant), and if they do, the publication may not emerge until years later. In the empirical analyses below, I use the drug pipeline data to measure when failure news has ripple effects.

## 4 Data and Sample Construction

The main goal of the empirical portion of this paper is to identify whether and when R&D failure news influences competitors' project continuation decisions. Measuring project-level spillovers requires a different level of granularity than classic studies of R&D spillovers.<sup>16</sup> For this study, I assemble a comprehensive dataset with project development histories.

### 4.1 Drug Pipeline Data

The starting point for my sample construction is the drug development records in Cortellis, which contains development information for 64,067 drugs (as of May 2016). The Cortellis platform aggregates information from public records (e.g., patent filings, company press releases, financial filings,

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<sup>15</sup>See <https://endpts.com/bristol-myers-dumps-late-stage-ido-studies-in-wake-of-incytes-pivotal-implosion-in-yet-another-setback/>; <https://www.fiercebiotech.com/biotech/bristol-myers-drops-phase-3-trials-800m-ido-drug>

<sup>16</sup>Patent data are most commonly used to study innovation spillovers and measure the relatedness of R&D projects (see Griliches, 1991; Lerner & Seru, 2017). However, their effectiveness in capturing project spillovers is limited because patents are not matched one-to-one with development projects, their scope and citations often reflect legal or patent office idiosyncracies, and their snapshot-like content lacks information about project investments or progress.

clinical trial registries, FDA submissions, etc.), and employs professional curators. Cortellis links each milestone event to its applicable disclosure information (e.g., press release, company investor literature or pipeline documentation, financial filings, etc.). Most records also have detailed data summarizing the drug’s development history and milestones.

This paper’s analyses use those milestones to construct full drug development histories for each drug-indication (development “project”). These histories include which firms were actively developing the drug, and what stage of development (discovery, preclinical, phase I/II/III clinical trials, registration, approval, launch) the project was in at any given point in time. They also include event dates for development discontinuation, suspension, and product withdrawal announcements.

## 4.2 Market and Technology Groups

In my analysis of competitor reactions, I separate drugs according to two different dimensions of relatedness: therapeutic indication (“market”) and molecular target-actions (“technology”).<sup>17</sup> Characterizing drug development projects along these two distinct and non-mutually exclusive dimensions is in-line with how researchers at major drug developers categorize projects (Cook et al., 2014; Shih et al., 2017).

**Therapeutic Indications (Market).** A therapeutic indication is the medical condition treated by a drug. Firms may develop a single drug to treat a number of separate indications—though one is usually the “lead” indication. Approximately 28% of all drugs in the Cortellis data have more than one development indication. Of the drugs that reached phase II clinical trials, 34% of started phase II trials for more than one indication. Sharing an indication does not mean that two drug compounds are similar in structure or share molecular mechanisms. Appendix Figure E1 shows that merely sharing an indication tells us very little about any two drugs’ structure. Furthermore, drug development for the median indication spans more than 10 molecular mechanisms (target-actions).

Cortellis indication names are usually quite specific (e.g., “dry age related macular degeneration”), but also include some more vague categories (e.g., “joint pain,” “stomach pain”). In some cases, two or more distinct Cortellis indication categories are actually referring to the same or highly similar conditions. For example, a drug treating “liver disease” is likely in competition with drugs treating “liver cirrhosis.” To account for these category issues, I map Cortellis indications to their International Statistical Classification of Diseases and Related Health Problems ICD-9 condition codes, and use these ICD-9 groups to delineate different therapeutic markets.<sup>18</sup>

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<sup>17</sup>Bloom et al. (2013) looks at market and technology at the *firm*-level, using industry codes for product markets and patent classes for technologies.

<sup>18</sup>Assigning ICD-9 codes to the Cortellis indication names is a challenge that requires knowledge about both the medical conditions, and about how health care providers classify conditions. A professional medical biller coded the concordance between Cortellis indications and ICD-9 codes in the Fall of 2015. ICD-9 codes have different levels of granularity, where each number represents a different medical conditions and with sub-categories denoted by decimals.

**Target-Actions (Technology).** A biological target is a molecule in the body upon which a drug acts and influences its function. For example, a drug may bind to and inhibit the function of a specific receptor (e.g., tropomyosin kinase receptor inhibitors), or a drug might function as an “agonist” by activating and increasing function in a protein (e.g., andrenoreceptor agonists). In these cases the target is defined by the biological pathway, and the action is determined by the functional change (e.g., “inhibitor,” “agonist,” “antagonist” etc.). Roughly 65% of Cortellis drug candidate records (70% of projects in this paper’s analysis sample) contain information about the drug’s primary (and secondary, if applicable) biological mechanisms of action. Though two drugs may differ in their compounds’ molecular structures or delivery mechanism, by attempting to treat a condition through the same target-action, the drugs are essentially testing the same hypothesis about how altering a biological process influences a clinically desirable outcome. Drugs may also have “off-target” effects, which represent the collateral damage incurred to other biological functions in the process of trying to act on the intended target. Often, these off-target effects are the source of drug safety/toxicity issues.

Many target-actions are useful for more than one medical condition. Sometimes, those medical conditions are naturally related through overlapping biological causes and symptoms. For example, the drug Humira (adalimumab) is a tumor necrosis factor-alpha inhibitor used to treat a range of inflammatory and autoimmune conditions such as rheumatoid arthritis, psoriasis, Crohn’s disease, and ulcerative colitis. In other cases, experimentation (or serendipity) reveals that a biological target-action may have multiple, seemingly unrelated disease applications. Angiogenesis inhibitors such as Genetech’s drug Avastin (bevacizumab) were originally explored as cancer drugs, but scientists realized (and later proved) that by blocking the formation of new blood vessels, the target-action was also promising for treating age-related macular degeneration. 59% percent of target-actions in Cortellis have development activity for more than one medical condition, with a mean of 4.2 indications per target-action (median=2). A single drug compound may also act on multiple known targets. In the set of Cortellis drug records that have at least one target, approximately one out of five has more than one primary target-actions (with a maximum of 13).

Same “technology” (target-action) drugs are not only similar in their therapeutic pathways, they are also more likely to be similar in their chemical structure. Using chemical informatics techniques, Krieger, Li, and Papanikolaou (2019) show that small-molecule drug candidates within the same target-action group are more likely to be structurally similar. Similar compounds will, on average, behave similarly in the human body (see Appendix E for more detail); however, small differences can lead to drastically different efficacy or adverse effects. Furthermore, the failure to effectively treat one disease does not necessarily rule that drug (or similar drugs) out for different diseases. This feature is reflected in belief  $D_j^{common}$  in Section 2.3. A failure within a certain

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The medical biller assigned codes to integer categories (e.g., an indication with ICD-9 of 202.5 is categorized as 202). I’m grateful to Manuel Hermosilla for providing the mapping from Cortellis indication names to ICD-9 codes.

market and technology is certainly not good news for other disease applications, but the extent of any competitor learning is not (*ex-ante*) obvious: different patient populations might respond better to a drug or tolerate certain side effects better.

I apply the chemical similarity techniques to the competitor pairs in my analysis sample, and report the results in Appendix E. Drugs that share a target-action have greater average similarity than drugs that merely share a therapeutic market. However, the distributions show that ST pairs are rarely clones and that ST compounds still have plenty of chemical diversity. So while trial outcomes signal the validity of the target-action’s treatment hypothesis, competing drugs’ idiosyncratic features make their signals imperfect substitutes for any drug’s own trial results.

### 4.3 Sample Inclusion Criteria

To estimate the project-level response to competitor failure news, one needs data that captures competitor failure disclosures and their timing. The first step is to use the development history events to create a full panel data set of drug-indication-date for all drugs. Drug projects are eligible for inclusion in the final analysis data set starting with the earliest date after entering phase II clinical trials, until they exit or begin phase III trials. I use phase II development projects because phase II trials are the first real test of a drug’s efficacy in humans, require major capital investments,<sup>19</sup> and have levels of uncertainty much higher than phase III projects.<sup>20</sup>

Next, I focus on failure disclosures that are potentially relevant to competitors. I identify the potential set of “treating” failures by defining “frontier discontinuations.” Frontier project discontinuations are those occurring after phase II trials began, and before any drug projects within the given indication and target-action combination had reached approval and market launch.<sup>21</sup> This frontier criteria is important, because it excludes early (e.g., preclinical, phase I) failures that are unlikely to influence decisions for later stage projects, as well as failures in technology areas that are already validated through the regulatory process and in the product market.

To establish competitor failure news events, I merge the frontier discontinuation dates with the full set of phase II development histories. A phase II project experiences a competitor failure if it shares either a market or technology with the failing frontier competitor, the focal project was active in phase II for at least one quarter at the time of the competitor news, and they entered phase

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<sup>19</sup>Cost estimates vary, with average Phase II trial cost reported as anywhere from \$13 million to \$80 million, while phase I cost estimates range from \$4 to \$8 million (Sertkaya et al., 2014; Adams & Brantner, 2006; <https://aspe.hhs.gov/report/examination-clinical-trial-costs-and-barriers-drug-development>; <https://lifescivc.com/2014/11/a-billion-here-a-billion-there-the-cost-of-making-a-drug-revisited/>).

<sup>20</sup>16% of phase II and 50% of phase III projects eventually reach approval, according to Hay et al. (2014).

<sup>21</sup>The main findings are robust to further excluding observations where the treating and treated drugs share at least one known target-action (even if not an indication) with a previously launched (i.e., FDA approved) drug. See Appendix Table B4.

II within 10 years of one another.<sup>22</sup> A frontier discontinuation event may only “treat” competitor projects if its discontinuation date was prior to the discontinuation of the competitor project.<sup>23</sup>

The analysis data is 1997-2014. Project-quarter observations are censored out of the panel once the project was discontinued, graduated to phase III, or after 32 quarters elapsed since the project entered phase II. Some projects are never officially discontinued, and continue to be listed as though they continue in phase II despite no development reported for long periods of time. These “zombie projects” are responsible for many of projects that persist in the panel for 32 quarters before I censor out their subsequent project-quarters. Section 5.6 summarizes the analyses using this more liberal definition of project discontinuation, and reports similar overall results.

#### 4.4 Analysis Data

The final analysis data set contains 6,183 drugs and 325 ICD9 indications (markets), which combine to form 10,639 drug-indications (projects). Projects may experience relevant competitor failure events in three different ways: 1) SM-DT, 2) SM-ST, and 3) DM-ST. Ninety-five percent of the projects eventually experience at least one competitor failure within the same market, 11% experience a competitor failure within the same market and same technology, and 43% experience a competitor failure within a different market but same technology.

I generate a set of variables for the number of times a given phase II project has experienced a competitor failure within each category, as of any given date. Next, I create treatment “window” indicator variables that equal one when a project is within a defined time range (one, two, or three quarters) following the competitor failure news. Tables 1 and 2 summarize the descriptive statistics for the analysis sample.

[Tables 1 & 2 Here]

The average likelihood of a project being discontinued in a given quarter is 1.2% (conditional on surviving up until that point). Thirty-seven percent of the projects which were not right-censored by the end of the analysis period (i.e., had the opportunity to complete 32 quarters in phase II, exit, or graduate to phase III before the third quarter of 2014) are actively discontinued during the sample period, while 20% of those projects graduated to phase III development.

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<sup>22</sup>While 10 years may seem like a long window, the median time in phase II is two and a half years, and the 90th percentile is over six years. The median length of a phase III trial is four years (90th percentile over 10 years). Therefore, a relevant frontier competitor project may still be under development 10 years after starting its own phase II trials.

<sup>23</sup>Since the analysis panel is at the quarter level, some “treating” and “treated” projects share a discontinuation quarter, but only if the “treating” project disclosed failure at an earlier date within the quarter.

## 5 Results

### 5.1 Overall Exit Rates

The econometric approach that follows isolates the impact of competitor failure news on firms’ continuation decisions. But as a first step, I establish the “typical life cycle” for projects in phase II. Figure 4A shows the cumulative hazard rate of projects by the number of quarters spent in phase II (up to 32 quarters). Intuitively, this figure represents the average project death rate for a given project age, conditional on surviving until that point in time or later. The curve has a slight “S” shape—indicating that the rate of project death starts slowly but increases through roughly 15 quarters into phase II. After that point, the death rate slows for the projects that remain in phase II (without exiting or graduating to phase III) after four years. Appendix Figure D evaluates how this baseline hazard rate differs across firm and portfolio characteristics.

Figure 4B shows the average quarterly project survival rate in phase II. This figure distinguishes between projects that are proximate (in time) to SM-ST competitor failure news, and projects which have not recently experienced such news. The figure shows that the survival is almost always lower for projects in the time window after a SM-ST failure news events.

[Figure 4 Here]

### 5.2 Empirical Strategy: Measuring Project Updating After Competitor Failure

The main analyses evaluate project-level response to a competitor’s project termination. Other studies address how *cumulative failures* within the firm or industry affect the likelihood of a project or firm’s success.<sup>24</sup> However, these prior analyses either do not leverage the timing of disclosure announcements, assert strong assumptions about the decaying value of competitor news over time, or they are limited to cross-sectional correlations. My method focuses on the dynamics of updating project investments after competitor news. Here, the timing of both competitor exits and the focal project’s termination are key to identifying the role of vicarious learning in decision-making.<sup>25</sup>

The baseline specification is a panel difference-in-differences proportional hazard model. The dependent variable is an indicator for whether or not the focal project was terminated as of a given period. Using hazard models on panel data helps account for natural death rates at different project

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<sup>24</sup>For example, Ingram & Baum, 1998; Haunschild & Sullivan, 2002; Baum & Dahlin, 2007; Kim & Miner, 2007; Madsen & Desai, 2010; Rao, 2017.

<sup>25</sup>Overall experience with failures within an industry, firm or department may result in organizational changes and learning. However, long-term failure experience may also correlate with strategic choices regarding market entry and risk tolerance, as well as scientific or technical evolution. In order to link specific competitor news events to project-level decisions, it is important that my econometric framework accounts to timing of exogenous (surprising) events, as well as the changing market environment.

ages.<sup>26</sup> Variation in the timing of the information shocks (competitor discontinuation events) allows for the not-yet-treated observations to serve as a plausible control group for the treated groups. Furthermore, by stratifying the baseline hazard rate by therapeutic market, I estimate treatment effects relative to the most relevant counterfactual exit rates. In other words, the survival model framework allows one to ask “how does recently learning of a competitor failure influence the propensity to exit, when compared to ‘untreated’ projects of the same stage, age, and market?”

This approach requires three identification assumptions. The first is the “no-anticipation” assumption (Abbring & Van Den Berg, 2003), which is satisfied in the clinical trials setting since firms believe that their rivals are unlikely to invest in high-stakes clinical trials with the expectation of failure. Any public (or insider) information about disappointing trial outcomes that circulates before discontinuation announcements should bias the treatment coefficients towards zero. To the extent that any such leakage occurs, one might consider the information effects in this approach as conservative estimates of competitor response to failure news. I also test this assumption empirically, by assessing the extent of any pre-trends leading up to competitor discontinuation events (see Column 4 of Table 3, and Figure 6).

The second identification assumption is that firms do not delay their trials in order to free-ride on competitor’s results. As previously mentioned, these types of “wait and see” strategies are costly due to wasting patent protection time. Nonetheless, I empirically test for differential responses for competing projects that entered later (“follower” projects; see Appendix Figure C1).

Third, this approach assumes no unobserved common opportunity shocks. Other studies of R&D spillovers (e.g., Bloom et al., 2013; Schnitzer & Watzinger, 2015; Lucking et al., 2018) instrument for R&D spending (using state tax credits) to address a classic “reflection problem” (Manski, 1993). In measuring continuous flows of R&D activity (at the firm level), those studies aim to measure how rivals’ patent production influences the focal firm’s output and performance. Ideally, an instrument provides an exogenous change in the level of rivals’ R&D activity, so that the analyst can distinguish true firm spillovers from outcomes driven by common external shocks.

An important feature of this study’s analysis sample is that such common opportunity shocks are highly unlikely. By the time a project reaches clinical trials, typically many years have passed since the pioneering scientific work that led to the drug (e.g., drug target identified by an academic lab). In order to reach phase II clinical trials, the compounds have already gone through rigorous preclinical lab (*in-vitro*) and animal studies, as well as phase I testing in humans. At that point, remaining scientific uncertainty will be determined after relevant trial results are unblinded. Moreover, by limiting the project sample to “frontier” drug development projects (in trials), I minimize

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<sup>26</sup>The hazard models here are similar to a discrete time logistic regression specification. Running the equivalent logit model (with controls for therapeutic indication and project age) yields nearly identical results.



the risk of unobserved common signals coming from regulators and marketed drugs.<sup>27</sup> It is unlikely that regulators send any common signals that would kill a new drug class before trial results play out. Any general macroeconomic shocks should impact different drug development areas equally, and are captured in the calendar time fixed effects in the regressions.

Unlike other product development areas with trends in consumer preferences (e.g., software, self-driving cars, food and beverage, etc.), disease markets stay relatively stable and predictable over time. One exception is infectious diseases, where the market for vaccines can skyrocket with an outbreak (e.g., Ebola, Zika, etc.). Outbreaks should not impact the analyses because I remove vaccines from the analysis sample. Moreover, pharmaceutical demand shocks are quite rare, and negative demand shocks even rarer.<sup>28</sup> In contrast to other R&D activities, clinical trial outcomes are distinct and discontinuous events (i.e., failure disclosures) at the *product* level. Firms have little control over the timing of their trial outcome news, and the information content does not depend on the previous stock of R&D activity.

My main specification is analogous to hazard models in Gans et al. (2008), Rao & Dutta (2012), and Aral & Walker (2012), where the timing of both treatment and response is of central importance in interpreting the results. In practice, I employ a Cox proportional hazard model specification, using drug-market-quarter level data:

$$\begin{aligned}
 h_{i,q,k}(t, X) = & h_{0k} \times \exp[\beta_1(SAME\ MKT, DIFF\ TECH\ NEWS)_{k,-q,t} \\
 & + \beta_2(SAME\ MKT, SAME\ TECH\ NEWS)_{k,q,t} \\
 & + \beta_3(DIFF\ MKT, SAME\ TECH\ NEWS)_{-k,q,t} + \gamma_t]
 \end{aligned}$$

In this specification  $h_{0k}$  is the baseline hazard rate of project exit, stratified by therapeutic market, and  $\gamma_t$  represents calendar time (quarter) fixed effects;  $i$  represents the focal drug project,  $k$  represents the therapeutic indication (market); and  $q$  represents the drug target-actions (technology) of the focal project.  $\beta_1$ ,  $\beta_2$ , and  $\beta_3$  are the coefficients on the three different types of competitor project discontinuation news : 1) SM-DT, 2) SM-ST, and 3) DM-ST.

In the main specifications (Table 3), the competitor discontinuation (“treatment”) variables are equal to one if the focal observation is within two quarters since the competitor failure news—allowing for treatment to turn on and off for multiple treatment spells. I also consider specifications where competitor discontinuation news is an absorbing state (that is, where the variable takes on the value of one after the first competitor discontinuation event in that category), or varies by

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<sup>27</sup>See Appendix Table B4 for robustness checks using more restricted analysis samples, in which no drugs that share the same technology have reached the market.

<sup>28</sup>Prescription drug spending in the US (as a % of GDP) has risen consistently since the late 1970s, with only a slight dip following the 2008 recession (see <https://www.brookings.edu/blog/up-front/2017/04/26/the-hutchins-center-explains-prescription-drug-spending/>). Policies expanding insurance coverage for prescription drugs have created occasional positive demand shocks (e.g., the 2006 enactment of Medicare Part D).

“treatment intensity” (cumulative number of competitor failure events in each category since the focal project entered phase II). Appendix Table B1 displays those modified specifications. In addition, I evaluate a dynamic version, where each competitor failure event type is interacted with indicator variables for number of quarters until the competitor news event (see Figure 6). Appendix C also outlines alternative regression approaches. The alternative models and data structures produce the same overall patterns of response to competitor exit news.

### 5.3 Impact of Failure News Results

Table 3 presents the estimates from the main regression specifications for project exit rates—all using the “treatment window” that takes on a value of one within two quarters after each type of competitor failure news. In the first column, I report the results of a naive specification, in which I group competitor discontinuation news events into a single variable. Under this grouped competitor news variable, the results show no significant change in the propensity of project exit following competitor discontinuation. However, they story shifts once I separate different types of competitor discontinuation news.

Separating competitor news into the (not mutually exclusive) SM and ST groups reveals that recent competitor failures do, in fact, significantly increase exit rates when the competitors share a technology (Column 2). The coefficient (0.274) implies a 23% increase in the likelihood of exit in the window following a ST competitor failure. This effect was hidden in the Column 1 version because the number of SM competitor news events far outweighs ST failure events (see Table 1). The two category version is the (binary) analog to the similarity measures used in Bloom et al. (2013) and Lucking et al. (2018). However, this two-way split still does not capture the full picture of competitor responses.

Columns 3 through 6 in Table 3 further divide the competitor news into the three competitor news types: SM-DT, SM-ST, and DM-ST. Column 4 additionally tests the “no-anticipation” assumption by including indicator variables for the window before each type of treatment, and shows that competitor all failure news has no significant impact on discontinuation rates before the announcement period. Columns 5 and 6 add control variables for the number of each type of competitor and firm characteristics.

[Table 3 Here]

The impact of each type of failure news is quite similar across these preferred models (Columns 3-6).<sup>29</sup> On average, competitor news from the SM-DT group yields no significant change in hazard rate of project exit, with magnitudes close to zero.

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<sup>29</sup>Testing the proportional-hazards assumption yields non-significant results (i.e., the proportionality assumption holds.)

SM-ST competitor discontinuations lead to large and highly significant ( $p < 0.01$ ) increases in the hazard rate of project exit, while DM-ST discontinuation news lead to a smaller but still statistically significant increase in the probability of project exit. Wald tests confirm that the SM-ST coefficient is significantly larger than the DM-ST coefficient in each model ( $p < 0.01$ ). Focusing on column 3, the SM-ST coefficient represents a 0.739 increase in the log hazard (109% increase in probability) of project exit following a closely related competitor’s project discontinuation, and the DM-ST coefficient implies a 0.158 increase in the log hazard (17% increase in the probability) of project exit following a technological competitor’s discontinuation disclosure in a different therapeutic area. The coefficients from Column 3 are also depicted as bars in Figure 5.

[Figure 5 Here]

Figure 6 shows the event study of experiencing a competitor discontinuation event, by interacting the treatment event status with with indicator variables for time before (or after) the project’s earliest treatment event using six-month increments. The same general pattern of relative treatment magnitude holds. While the pre-treatment estimates do not appear perfectly flat around zero, they do not reveal any clear trends as the first competitor discontinuation date approaches. The SM-ST effect seems to occur exclusively in the six-month window after the competitor news, while the the DM-ST effect lingers a bit longer, despite being smaller.

[Figure 6 Here]

Even though the DM-ST effect lasts longer, one cannot simply add up the significant six-month coefficients in Figure 6 and compare them to the first post-treatment SM-ST coefficient. Since the coefficients relate to the exit rate, earlier increases to the hazard rate of exit have more of an impact than later increases—because a compounding effect kicks in over time and the base of active projects is smaller in later periods.<sup>30</sup>

At first look, the different magnitudes of the the two ST coefficients may seem surprising. How can the SM-ST news have a greater impact on exit rates if it also triggers the benefits from reduced competition? The first explanation is that market-specific factors (e.g., side effects) produce greater belief updating following SM-ST news. This view is consistent with the theoretical framework:  $\beta + \gamma > \gamma$  (i.e., SM-ST news leads to much stronger downward updating,  $p^{--}$ ). Furthermore, if

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<sup>30</sup>Using a two-period difference-in-differences model, where treatment is binary for post-competitor news (absorbing state version), I still find that the SM-ST coefficient is significantly larger than the DM-ST effect (see Appendix Table B1). In general, as more time elapses after the initial competitor news “treatment” event, the more likely other news or own project results are to confound the competitor effects. Concerns of “shared fate” for similar technologies also come into play when one extends the treatment window. Therefore, the comparison of information effects across news types is best identified when the treatment window is smallest. For that reason, the main specification focuses on the first few quarters after competitor news.

SM-DT effects are weak (on average), then reduced competition does not produce a formidable “opposing force.”

A related explanation is that SM-ST news is more salient, even if not more valuable, than DM-ST news. The additional time it takes for DM-ST news to affect decisions (Figure 6) suggests that salience might play a role in the differential responses. Both stories highlight the importance of identifying the separate forces and the interaction effects of market and technological similarity.

## 5.4 Heterogeneous Effects and Testing Theoretical Predictions

The average response to competitor failure news (Table 3) substantiates that competitors learn from one another, and the importance of separating the three different types of competitors when analyzing spillovers. The theoretical framework also suggests key moderating factors: competition, uncertainty, and remaining learning opportunities. Below I explore each of these areas using project-level variation in the phase II project analysis data.

### 5.4.1 Level of Competition

Figure 7 shows the results when I group projects by the level of competition and evaluate their response to competitor failure within each subset. First, I run the main specification comparing the bottom half (low) vs. top half (high) of projects in terms of number of potential market competitors in active development. This split reveals that the SM-DT effect is negative and significant when the level of market competition is low (first bar in Figure 7, Panel A).<sup>31</sup> In other words, when potential competition is low, firms are significantly more likely to *continue* after a SM-DT competitor failure. For the high-competition group, the SM-DT negative effect is statistically insignificant (despite a similar number of observations), with a much smaller and noisier coefficient.

Next, I evaluate the subset of phase II projects in therapeutic markets with low vs. high levels of previously approved drugs. Similar to the result for development market competitors, the projects below the median in number of on-the-market competitors are significantly less likely to terminate following a SM-DT competitor failure event. Appendix Figures D4 and D5 show the event study versions of these regressions for the low and high competition subgroups, respectively.

[Figure 7 Here]

The set of competitive environment findings confirm the theoretical intuition that facing fewer competitors will influence decision-making under certain market structures. The magnitude of the negative SM-DT coefficient is greater when the baseline level of market competition is smaller

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<sup>31</sup>Appendix Figure D3 shows the bottom vs. top quartile split. The patterns are similar but with an even greater negative effect for the low competition SM-DT response.

(across both definitions of number of competitors). This result supports the notion that payoffs have a nonlinear relationship with the number of competitors (Bresnahan & Reiss, 1991). The potential for monopoly or duopoly profits, if the drug development project reaches the market, is more likely with few product development or market competitors. That said, low levels of competition do not seem to matter as much in moderating the technological learning effects in the ST news scenarios.

#### 5.4.2 Project Stage, Uncertainty, and Relevance of Signal

An important assumption of the empirical approach is that competitor outcomes are only relevant when the competing project is in the same or a more advanced stage of development. According to this approach, projects that significantly lag in development are irrelevant to the decision-making process. Furthermore, more advanced focal projects should have already resolved enough uncertainty to ignore earlier stage projects. To test this prediction, I evaluate whether phase III projects are more or less likely to exit following news of a phase II competitor’s failure. This analysis sample contains 3,195 phase III projects that are ever “treated” by phase II failure news. The breakdown across competitor news types is quite similar to the main analysis: 92% experience SM-DT news, 9% SM-ST news, and 43% DM-ST news.

In Appendix Figure D6, I present the event studies. For each competitor news type, the trends are fairly flat and none of the coefficients are significantly different from zero. Phase III project investments do not appear to respond to phase II competitor failures, regardless of the market and technology relationships.<sup>32</sup> The lack of response seen in Figure D6 implies that developers only update their expectations about R&D success when their own project uncertainty is high (i.e.,  $\frac{\delta}{\beta+\gamma}$  is relatively low). If developers have already cleared certain development hurdles, they do not update beliefs based on earlier stage projects.

#### 5.4.3 Learning Opportunities and Response to Competitor Failure

To explore whether competitor responses are different with more or less remaining learning opportunities, I interact each of the three treatment types with an indicator for whether the focal project had relatively low or high ST learning opportunities. I define low vs. high learning opportunities as below or above the median number of ST competitors (five projects).

When the information environment is relatively rich with competitor learning opportunities, firms advance more marginal projects into phase II testing. Table 4 reports the coefficients from the hazard models. The correlation between high learning opportunities and the hazard rate of exit is positive and significant—implying that having above-the-median ST competitors is associated

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<sup>32</sup>The primary analyses use both phase II and phase III failure news as relevant signals for phase II projects. I also tested whether competitor failure news originating from only phase III competitor had a different effect than the same type of news stemming from a phase II. This comparison produced no statistically significant differences.

with a 24% higher (Column 2) relative probability of termination at any point in phase II.<sup>33</sup> Even after accounting for the increase in exit during the window after competitor failures, projects with more ST peers are more prone to termination. The presence of competitors that have correlated technology signals may initially strengthen the firm’s belief in their own project ( $p_{ijk}^{private}$ ) through stronger public signals about the technology ( $D_j^{common}$  and/or  $D_{j,k}^{common}$ ), while also effectively reducing the cost of exploration through the promise of additional relevant trial outcomes.

Table 4 also reports coefficients for each type of competitor failure news interacted with the remaining ST learning opportunities. The SM-DT coefficients show that having more ST competitors corresponds with being more likely to forge ahead following a “pure” market competitor failure. I find no significant differences between the high and low learning opportunity groups following SM-ST competitor news. The DM-ST effect almost disappears for projects with high remaining technology competitor learning opportunities. Essentially, the exits driving the overall DM-ST effect (Table 3) disproportionately involve low remaining learning opportunities. Projects with more learning opportunities were more likely to continue following DM-ST competitor news.

[Table 4 Here]

These findings are consistent with the theoretical framework in that more technological learning opportunities increases the attractiveness of continuing a project. Firms appear to be more likely to advance risky projects when similar competitors will provide correlated signals. A developer’s belief about a project’s probability of success is likely unchanged after a SM-DT failure. If the project has zero or very few same technology competitors remaining, then the forward-looking developer does not expect to gain any more relevant technology insight from rivals. However, a large number of remaining technology competitors may provide further information as their experiments complete over time. The combination of a more attractive competitive environment (one less potential *market* rival) with many potential learning opportunities still intact makes the continuation option even more attractive than before SM-DT competitor failure.

A similar logic helps explain the DM-ST results, where the existence of more remaining competitors using the same target-action diminishes the response DM-ST failures news. Here, developers might be more hesitant to pull the plug quickly if they believe more competitor signals may arrive in the future—down-weighting any one given competitor failure while other peers remain. Observing that other ST competitors remain committed to their projects might also lead to positive feedback loops, such that developers believe their investment or clinical hypothesis is validated by others (e.g.,  $D_j^{common}$  remains quite high). Notably, I did not find any significant difference across learning opportunity levels following SM-ST competitor news. The information shock of

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<sup>33</sup>By comparison, Budish et al. (2015) find that a 10 percentage point decrease in trial length (five-year survival rate) is associated with a 8.7 percent increase in trial entry. Both Budish et al. (2015) and the results here suggest that decreases in the cost of experimentation allow for project investments that otherwise might not happen.

these most similar competitor failures appears to overpower any secondary effects of remaining competitor outcomes (i.e., downward updating on  $D_{j,k}^{common}$  outweighs remaining option value).

#### 5.4.4 Additional Heterogeneity: Portfolio Characteristics

The theoretical predictions and empirical analyses above all focus on the characteristics of the project and competitive environment. However, as mentioned in Section 2, the model also implies that firm characteristics should moderate the updating process. Appendix D provides additional exploration of such firm-level differences. These analyses underscore how experience, and R&D portfolio choices shape firms’ absorptive capacity and flexibility. I find that that more experienced and more diversified firms are more likely to terminate projects, in general. Firms with more expertise and portfolio concentration are more likely to react to DM-ST signals, which is consistent with common signals ( $D_{j,k}^{common}, D_j^{common}$ ) being more actionable for such firms. Finally, I find that high commitment to a given drug—i.e., having a high private value for the particular compound-technology ( $D_{i,j}^{private}$ )—is associated with less reaction to DM-ST competitor news.

#### 5.5 Robustness Checks

Appendix B details alternative regression specifications. The same qualitative patterns persist in the alternative specifications, though the magnitudes differ depending on the level of analysis and treatment definition. Below, I detail key checks of the main identification strategy and the informational content of “failure news” events.

**Identification Assumptions.** First, one might be concerned about how entry order affects the results. In drug development, the looming patent expirations create a sense of urgency for development. “Wait and see” strategies may be too slow, given that development periods are long and expensive. However, firms might still choose particular entry timing in order to capitalize on information, regulatory, or first-mover advantages. For example, firms might engage in “me-too” or copycat innovation, sacrificing first-mover advantage in exchange for reduced risk and allowing the leaders to establish regulatory and marketing pathways (Stern, 2017).

To test whether entry order influences the results, I interact each competitor failure type with the focal (treated) firm’s phase II entry position relative to its first treating project: follower, neck-and-neck, leader. Appendix Figure C1 displays the results of this regression as a bar graph. The relative magnitudes of the competitor failure news coefficients are quite similar for followers, leaders, and projects that are neck-and-neck (entering phase II within a year of one another). The magnitudes of the SM-ST and DM-ST coefficients are actually greatest among the leader group.

A related concern is that independent simultaneous failure might be driving the main effects for SM-ST and DM-ST news. Under this logic, ST projects have a “shared fate” and may learn

about their own disappointing results around the same time. To address this concern, I limit the analysis sample to smaller project age ranges (e.g., the first 6/8/10/12/16 quarters in phase II, 4-12 quarters into phase II, etc.) and apply the primary regression specification. These regressions yield competitor news coefficients with magnitudes very similar to the primary regression specifications.<sup>34</sup> The median phase II trial in Cortellis lasts for more than two years (10 quarters), so the “treated” projects in these age-limited samples were unlikely to have completed their first phase II trials, let alone completed their entire battery of phase II investigations (usually involving multiple trials)—implying that simultaneous “bad news” is not likely to be driving the results.

I further restrict the analysis sample to ensure that the competitor responses are not driven by common shocks to related approved drugs. Appendix Table B4 reports the results of the main specification applied to more limited samples that remove projects which share target-actions with any previously approved drugs. The restricted sample results are very similar to the main analysis—confirming that ST common signals from post-approval drugs are not influencing the results.

Additionally, the results do not significantly differ when I compare discontinuation signals accompanied by a press release to those without press releases, and when I exclude the set of “treated” projects that were proximate in time to their own trial end dates. These analyses help to rule out the possibility that competitors are independently failing within a few quarters of each other, and that more-publicized events drive competitor response.

**Competitor Signal Strength.** I employ three different tests of how the strength of the competitor failure signals moderate the focal projects’ responses. These regressions help test issues of network interference, and confirm the intuition that firms pay greater attention to “stronger” DM-ST signals. First, I find no significant differences when comparing the focal project’s response to the first treatment vs. subsequent failure news (Appendix Figure C2A). These results help rule out concerns about multiple signals leading to mis-measurement of dyadic peer influence (Aral, 2016).

Similarly, I compare “solo” treatment events (a single competitor exit) to “clusters” of treatment for each type of failure news (Appendix Figure C2B), and when the competitor drug’s failure news involved only one indication discontinuation vs. multiple indications discontinued (Appendix Figure C3). Both of these additional splits show that DM-ST responses are significantly stronger ( $p < 0.01$ ) following the “stronger” competitor signals. Appendix C provides an in-depth discussion of the single- vs. multiple-indication failure results (Figure C3). In short, the multiple-indication failures are more likely to involve safety concerns that catalyze learning ripple effects across projects.

Together, these results suggest that firms have a higher bar for reacting to DM-ST news. Developing their drug for different conditions, these rival projects appear less sensitive to any one

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<sup>34</sup>For brevity, results not shown. In the smaller samples limited to the first 6, 8, 10 or 12 quarters or phase II, the DM-ST coefficients were not significant, despite having magnitudes quite similar to the main specification (0.17, 0.10, 0.12, and 0.15, respectively). The SM-ST coefficient remains significant in all these samples, with a magnitude between 0.7–0.8.



project termination. In contrast, a single SM-ST competitive failure may be enough reason to reconsider one’s own related investments.

## 5.6 Alternative Definitions of Project “Death” and Technological Similarity

**More Inclusive Project “Death.”** The main survival models use a conservative definition of project exit: when firms *officially* announce project stoppage or when Cortellis codes a discontinuation event.<sup>35</sup> That definition does not account for obviously stagnant projects that continue on paper even though the firm does not commit any further resources to the project (e.g., no additional trials). One might call such cases “zombie” projects. If firms use competitor events as an excuse to officially cancel such already-defunct projects, the mis-characterization of stoppage would threaten this paper’s empirical results. To evaluate this possibility, I construct an alternative project “death” variable that includes projects that never report any additional development (e.g., no new trials and no progression) as “discontinued” observations. Under this more inclusive project discontinuation definition, the average likelihood of a project being discontinued in a given quarter is 4% (conditional on surviving up until that point), and 51% of the non-right-censored projects are discontinued during my phase II sample period). Appendix Table B2 presents these regression results. These hazard models yield results very similar to the main results in Table 3.

**Chemical Similarity as Technological Distance** Appendix E uses the same discontinuation definition as the main analyses, but uses compounds’ structural overlap as an alternative measure of technological similarity. I use the similarity measure developed in Krieger, Li and Papanikolaou (2019) to quantify chemical distance between project pairs. I interact compound similarity with different types of competitor discontinuation news.

Appendix Table E1 shows that project exit rates are significantly increasing in the structural similarity to discontinuing competitors (Column 1). However, when comparing the compound similarity effect across different types of competitor news (Columns 2 and 3), that relationship partially breaks down because sharing a biological target-actions is correlated with the pair having higher chemical structure similarity. Overall, structurally similar compounds have greater exit responses; but those relationships are secondary to sharing the same target-action technology. Thus, in addition to adding a layer of granularity, these results supply further justification for using the same/different technology categories as the primary groupings for knowledge spillovers.

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<sup>35</sup>In addition to official announcements triggering the change to “discontinued” status, Cortellis also records exits based on the removal of a project from the firm’s active pipeline information on the website or investor documents.

## 5.7 Overall Impact of Competitor Learning

A series of “back of the envelope” calculations help illustrate the overall magnitude of the competitor learning effects. I use the regression results to predict the overall rate of project terminations, with and without the two significant learning channels (SM-ST and DM-ST). Since entry behavior would also change in a regime with no competitor disclosure, this exercise can only represent a crude characterization of a counterfactual zero disclosure regime.

Appendix Figure D2 displays the learning channel’s magnitude. The figure graphs the predicted probability of project discontinuation by project age (quarters in phase II), compared to the hypothetical discontinuation rate—if one were to “turn off” the ability to learn from competitors (e.g., if firms were not required to disclose trial starts and project terminations). The “predicted” discontinuation is based off the main econometric specification’s average predicted discontinuation value (corresponding to Table 3, Column 3) for observations “treated” by SM-ST or DM-ST news. This exercise shows that the discontinuation rates would be roughly 25% lower if one were to “shut off” the ability to learn from any technological competitor news.

Another way to think about the magnitude of learning is to ask how many terminations might have occurred without learning from competitor disclosures? In the analysis sample of 10,637 projects, 2,550 projects exited in the first 32 quarters of phase II, 1,658 project terminations occurred within two quarters on any type of competitor exit, and 463 discontinuations happened after a ST competitor exit. Assuming the same level of entry, I use the regression results to generate a back of the envelope prediction of the exit rate, but *without* competitor learning. Many projects would eventually exit in both scenarios, but might be terminated sooner with competitor learning. Without a full structural model, one cannot estimate a true counterfactual here. Again, the no-learning scenario ignores how entry and continuation decisions might also change in a regime without competitor failure disclosures. For the sake of illustrating the role of learning on exit decisions, the back of the envelope estimates hold entry and the number of competitors constant.

I find that turning off the learning channel results in 5.1% (129) fewer overall project exits. Simulating the predicted timing of project exits yielded 1,683 (3.7%) additional active project-quarters in the version without competitor learning. This estimate is from the average results of 1000 iterations of a Monte Carlo simulation, in which the main regression estimates predict the timing of each project termination, both with and after “shutting off” the competitor learning coefficients. While these estimates cannot reveal whether additional terminations are wise choices, they illustrate how the disclosure channel has a potentially large impact on R&D investment decisions and the fate of R&D project teams. Even with a conservative estimate of trial costs, they suggest that competitor learning accounts for more than \$2 billion in reallocated R&D funds.<sup>36</sup>

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<sup>36</sup>The US Department of Health & Human Services has estimated an average Phase II trial cost of \$10-\$16 million. See <https://aspe.hhs.gov/report/examination-clinical-trial-costs-and-barriers-drug-development>.

## 6 Conclusion

The ability to assimilate external knowledge and update effectively is of central importance to R&D managers (Cohen & Levinthal, 1990; Cockburn et al., 2000). Developing new technologies is an inherently uncertain process and requires judgments about both the expected value of the innovation and the potential of a given R&D investment to reduce uncertainty. This paper analyzes R&D project investments through the lens of real options with competitor learning. This framework highlights how competitor news can serve as additional decision points for reevaluating project investments. Competitor failure news can have payoff externalities, knowledge spillovers, or both. Furthermore, additional same technology competitors might actually increase a risky project’s attractiveness, by serving as an additional (noisy) signal of technology quality.

The empirical results demonstrate that competitor outcomes directly enter project selection decisions. I exploit the unique features of pharmaceutical R&D, where competing projects are developed in-parallel and have distinct market and technology categories. I find that competitor discontinuations influence the probability of project exit, but the nature of this response depends on project relatedness in both market and technology space. The response to competitor failure is sensitive to the competitive environment and project characteristics. I find that market competition effects are stronger when competition is low. However, market competition considerations are subordinate to technological learning effects from highly similar competitors. Additionally, I find that project-specific uncertainty, and the potential for future competitor learning both influence the magnitude of responses to competitor failure news.

The findings contribute to the literatures on real options and R&D spillovers. Failure disclosures are distinct learning opportunities and may function as additional experimental stages. The project exit analysis reveals that the interaction of product market and knowledge spillovers is not simply the sum of the two component effects. Managers and innovation scholars should account for this interaction, in addition to the separate effects. Finally, the results suggest that learning spillovers might encourage (rational) herding both into and away from an R&D sub-field. This type of herding may be privately optimal but result in an overall lack of diversity in R&D (Dixit, 1989; Acemoglu, 2011) as firms prioritize information opportunities and industry trends over society’s optimal mix of experiments.

While drug development lends itself well to the study of R&D failure, other industries pose more difficulties for tracking R&D efforts, as well as pinpointing and decoding failure events. Future studies might address how variation in observability of competitor projects, disclosure regimes, or product complexity impacts competitor learning. When R&D failures are less public, might

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Using the average of these cost approximations (\$13 million), an average trial length of 10 quarters, and the estimate of 1,683 additional active project-quarters, yields a total of \$2.2 billion in investments reallocated (or canceled) due to competitor learning.

alternative signals (e.g., patent filings, hiring, rumors etc.) substitute as effective sources of competitor learning? How informative are R&D failures when products involve complex combinations of numerous technologies (e.g., smart phones, self-driving cars, satellites, supersonic jets, etc.)?

Furthermore, future analyses need not be limited to failure events, as firms also learn from their rivals' successes. The challenge in studying R&D successes is that firms may not disclose good outcomes in a single news event, but rather over the course of multiple announcements. And while this paper's analysis is limited to publicly available knowledge, R&D organizations have finer-grained information about their own projects and competitors. The flow of information about competitor projects combined with the deluge of internal data (e.g., experimental results and forecasts) should allow the modern R&D organization to continuously update the valuation of its portfolio projects.

While the nature of the information and disclosure may vary, reacting to competitor outcomes is of principal importance in industries where firms are juggling uncertain projects and information externalities. In these settings, novel information may drastically change the direction of investments. How firms vary in their response to external signals continues to be an exciting question for scholars examining firm performance and the supply of new technologies.

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**Table 1: Descriptive Statistics, Phase II Projects that Experienced Competitor Failures**

	Count	Mean	Std. Dev.
Discontinued in First 32 Quarters of Phase II	10,637	0.24	0.43
Ever Experience Competitor Failure in the...			
... <i>Same Market, Different Technology</i>	10,637	0.95	0.21
... <i>Same Market, Same Technology</i>	10,637	0.10	0.31
... <i>Different Market, Same Technology</i>	10,637	0.43	0.49
Active Quarters in Phase II	10,637	21.91	9.01

**Note:** The analysis data set contains 10,637 phase II drug-indications (projects) that entered phase II between 1997 and 2014. The projects consist of 6,182 drugs, and 325 therapeutic markets (ICD-9 codes). Approximately 28% of all drugs in the Cortellis data list more than one development market (34% of drugs that reached phase II clinical trials undergo phase II trials for more than one indication). 72% of drugs have at least one technology (target-action) assigned in the Cortellis database. A phase II project experiences a competitor discontinuation if it shares either a market or technology with the failing competitor, if the pair of projects were ever simultaneously active for at least one quarter, and if they entered phase II within 10 years of one another. A project can only experience a competitor discontinuation event if the competitor's discontinuation date was prior to the discontinuation of the focal project.

**Table 2: Descriptive Statistics, Phase II Project-Quarter Panel**

	Count	Mean	Std. Dev.
Within 2 Quarters of Competitor Failure in the...			
... <i>Same Market, Different Technology</i>	254,069	0.54	0.50
... <i>Same Market, Same Technology</i>	254,069	0.02	0.14
... <i>Different Market, Same Technology</i>	254,069	0.13	0.34
Sponsor Firm's Number of Development Projects To-Date	251,077	164.32	304.26

**Note:** The panel data of phase II projects consists of 254,069 project-quarters. Table 1 presents descriptive statistics at the project (drug-indication) level, and Table 2 displays information about the drug-indication-quarter panel data set. In Table 2, the sponsor firm is assigned using the drug development history data by company. When multiple firms are involved in developing a drug during a given quarter, the larger of the companies, as determined by total development projects to-date, is assigned. In 1% of observations, the sponsor company was ambiguous and, therefore, not assigned.

**Table 3: Competitor Failure News Impact on Hazard Rate of Exit**

“Treatment” Window: within 2 quarters since competitor failure news	(1)	(2)	(3)	(4)	(5)	(6)
<b>Competitor Failure Type:</b>						
<i>Any Competitor</i>	0.014 (0.050)					
<i>Same Market Competitor</i>		-0.014 (0.050)				
<i>Same Technology Competitor</i>		0.274** (0.052)				
<i>Same Market, Different Technology</i>			-0.048 (0.050)	-0.029 (0.051)	-0.046 (0.050)	-0.040 (0.051)
<i>Same Market, Same Technology</i>			0.739** (0.100)	0.613** (0.126)	0.790** (0.101)	0.686** (0.102)
<i>Different Market, Same Technology</i>			0.158** (0.056)	0.159* (0.066)	0.259** (0.060)	0.220** (0.061)
<b>3 Quarter Window Prior to Competitor News:</b>						
<i>Same Market, Different Technology</i>				-0.054 (0.051)		
<i>Same Market, Same Technology</i>				0.042 (0.142)		
<i>Different Market, Same Technology</i>				-0.007 (0.068)		
<hr/>						
Controls:						
Number of Each Competitor Type					✓	✓
Firm Characteristics						✓
Nb. Drug-Indications	10,639	10,639	10,639	10,639	10,639	10,274
Nb. of Drugs	6,183	6,183	6,183	6,183	6,183	6,002
Nb. of Observations	213,302	213,302	213,302	213,302	213,302	202,080
Log Likelihood	-10,527	-10,514	-10,496	-8,013	-10,487	-9,902

Note: Estimates stem from Cox proportional hazard model specifications using panel data on drug projects by quarter. The outcome of interest is the focal project’s discontinuation. All models include a full set of year indicator variables and stratify the estimates by therapeutic indication. The competitor failure news variables are indicator variables that take on the value of one when the focal project is within two financial quarters since the given type of competitor failure event. Column 4 tests the “no-anticipation” assumption by including indicator variables for the three quarters leading up to each type of competitor failure event. Column 5 includes control variables with the number of competitor drug projects of each type that were active in clinical trials. Column 6 further includes control variables for firm size and experience. Coefficients may be interpreted as an increase in the log hazard rate. Standard errors in parentheses.†  $p < 0.10$ , \*  $p < 0.05$ , \*\*  $p < 0.01$

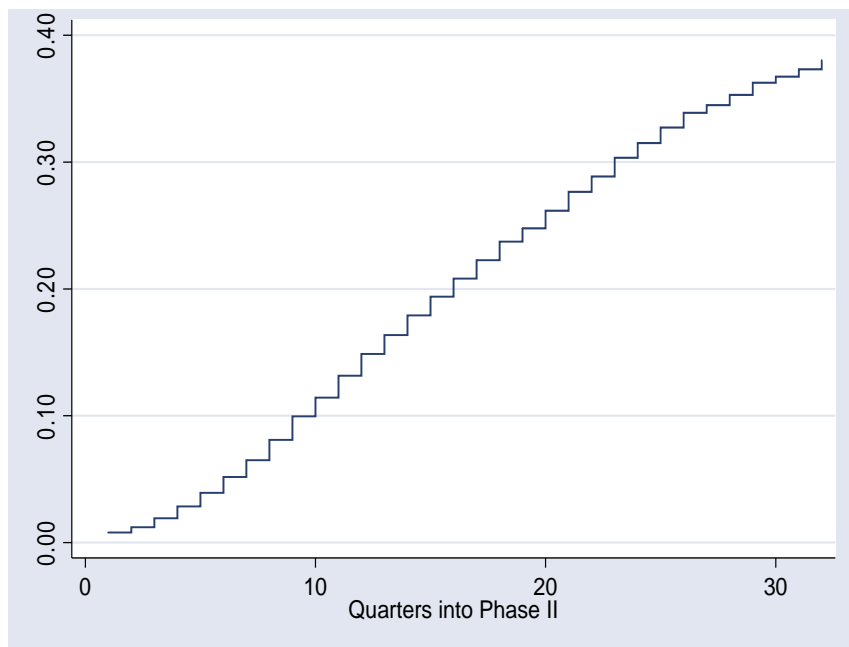
**Table 4: Competitor Failure News Impact on Exit Rates, by Remaining Technological Learning Opportunities**

	(1)	(2)
Tech. Learning Opportunities High (Above Median)	0.143 <sup>†</sup> (0.080)	0.212 <sup>**</sup> (0.081)
<b>Competitor Failure Type (Within 2 Quarter Treatment Window):</b>		
<i>Same Market, Diff. Technology</i> × Low Learning Opp.	0.002 (0.087)	-0.025 (0.088)
<i>Same Market, Diff. Technology</i> × High Learning Opp.	-0.119 <sup>†</sup> (0.068)	-0.127 <sup>†</sup> (0.068)
<i>Same Market, Same Technology</i> × Low Learning Opp.	0.803 <sup>**</sup> (0.227)	0.805 <sup>**</sup> (0.226)
<i>Same Market, Same Technology</i> × High Learning Opp.	0.687 <sup>**</sup> (0.110)	0.736 <sup>**</sup> (0.111)
<i>Diff. Market, Same Technology</i> × Low Learning Opp.	0.616 <sup>**</sup> (0.162)	0.629 <sup>**</sup> (0.162)
<i>Diff. Market, Same Technology</i> × High Learning Opp.	0.066 (0.063)	0.151 <sup>*</sup> (0.065)
<hr/>		
Controls: Number of Each Competitor Type		✓
<hr/>		
Nb. Drug-Indications	8,069	8,069
Nb. of Drugs	4,448	4,448
Nb. of Treating-Treated Quarter Obs.	158,817	158,817

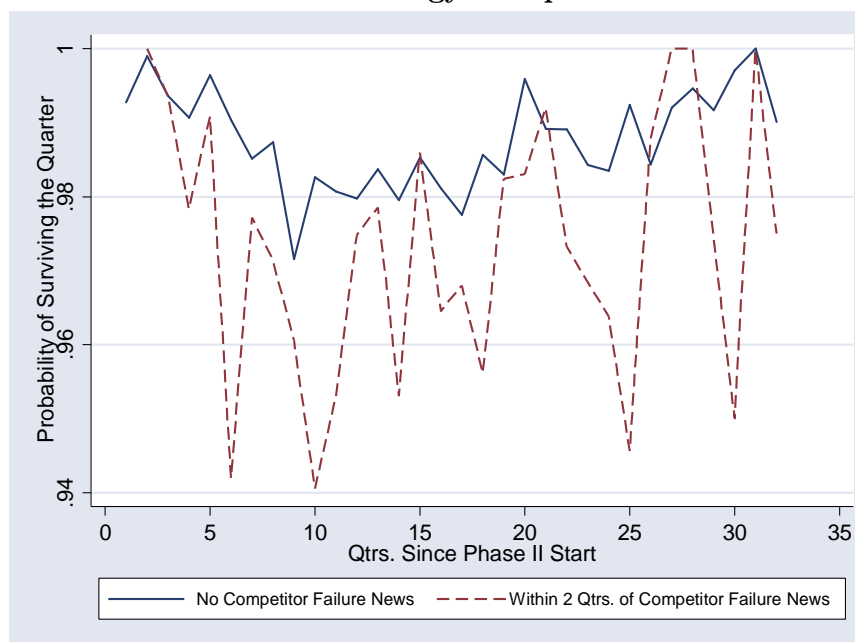
Note: Estimates stem from Cox proportional hazard model specifications using panel data of drug projects by quarter. The outcome of interest is the focal project's discontinuation. The sample excludes all projects without a primary mechanism of action (target-action) assigned in the Cortellis data. The regressions include a full set of year indicator variables and stratify the estimates by therapeutic indication. The high/low learning opportunity splits are based on the median number (5) of *same technology* competitor projects remaining in phase II or phase III trials. The competitor failure news variables are indicator variables that take on the value of one when the focal project is within two financial quarters since the given type of competitor failure event. Coefficients may be interpreted as an increase in the log hazard rate. Standard errors in parentheses. <sup>†</sup>  $p < 0.10$ , <sup>\*</sup>  $p < 0.05$ , <sup>\*\*</sup>  $p < 0.01$

**Figure 4: Project Death Rates in Phase II**

**A. Cumulative Hazard of Discontinuation**

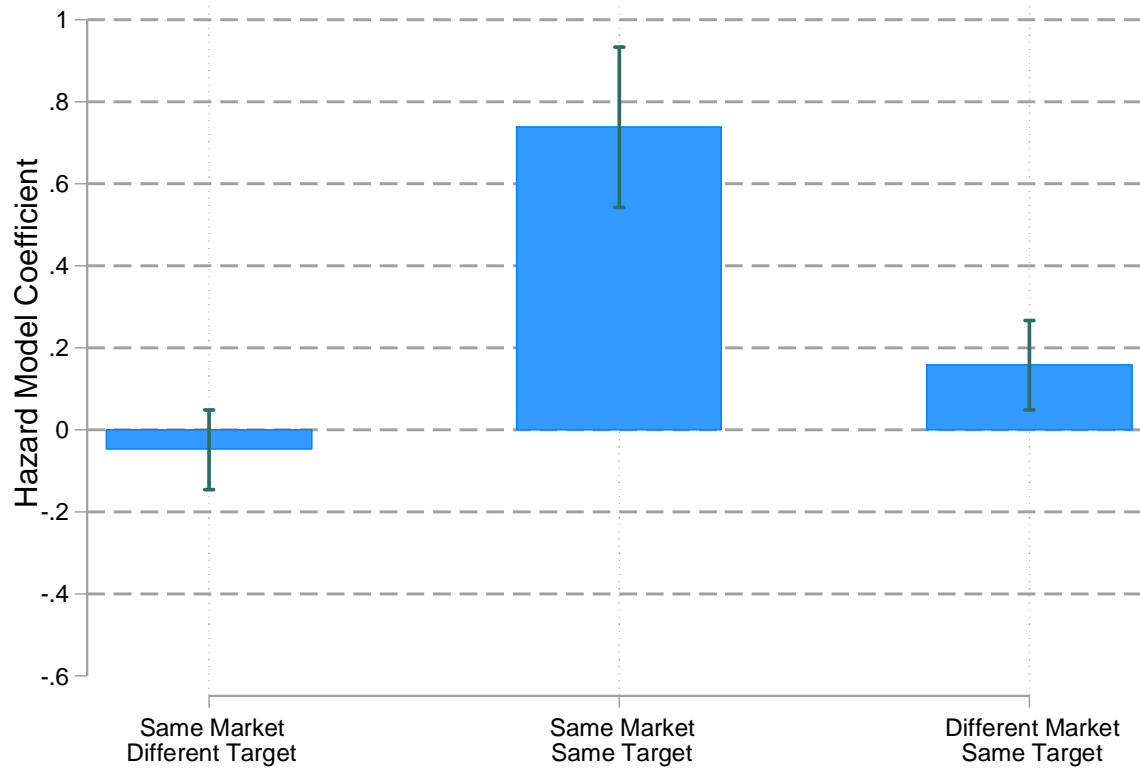


**B. Project Survival Rates with and without Same Market and Technology Competitor Failure News**



Note: Each panel represents a different way of tracking the overall rates of project exit throughout. Panel A graphs the cumulative hazard rate of project discontinuation by number of quarters since entering phase II. The intuition for the cumulative hazard rate is that it represents the project death rate for a given project age given that the project survived until that point or later. Panel B shows the probability of surviving a given quarter, conditional on entering that quarter (e.g., the likelihood that a project which enters its 8<sup>th</sup> quarter of phase II clinical trials is not officially discontinued during that period). The hashed line is the average survival rate for project-quarter observations that are within two quarters since a same market, same technology competitor disclosed project discontinuation. The solid line is the average survival rate for projects that are not within this window since the close competitor failure event.

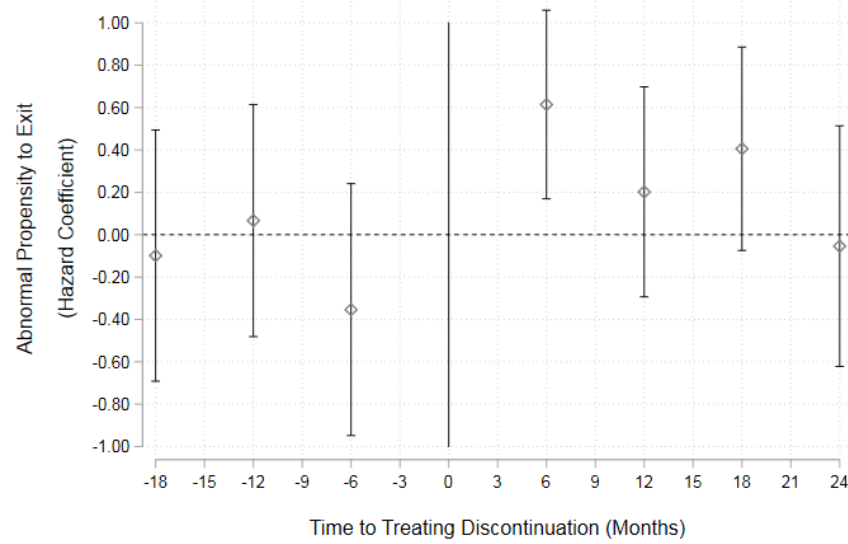
Figure 5: Competitor Failure News and Propensity to Exit



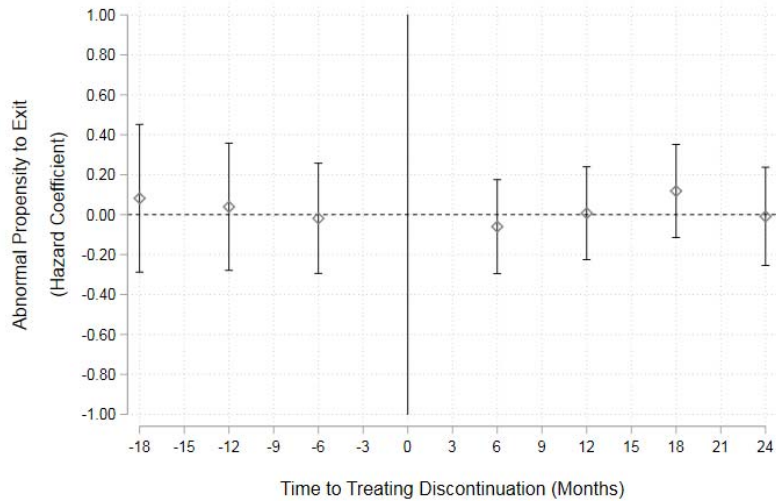
Note: The bars display the coefficients of interest from the main Cox proportional hazard model specification, which stratifies the sample by market and contains indicator variables for calendar time. The analysis sample includes 215,142 project-quarter observations (discontinued projects are censored out after exit). The magnitude of each bar represents the change in hazard rate of project exit when a focal project is within a two quarter window after a competitor failure. The left bar displays this coefficient for same market, different technology competitor discontinuations, the middle bar shows the same market, same technology coefficient, and the right bar represents the different market, same technology effect. The capped cover the 95% confidence intervals for each regression coefficient

# Figure 6: Dynamics of Response to Competitor Failure

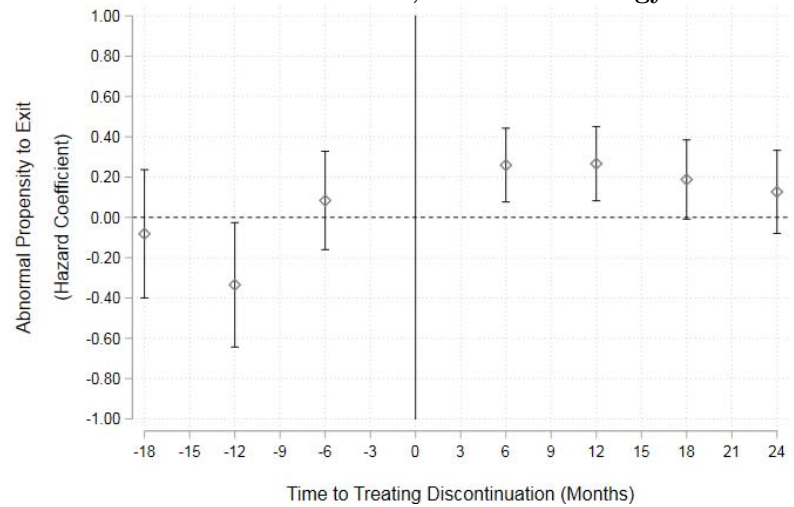
## A. Same Market, Same Technology



## B. Same Market, Different Technology



## C. Different Market, Same Technology

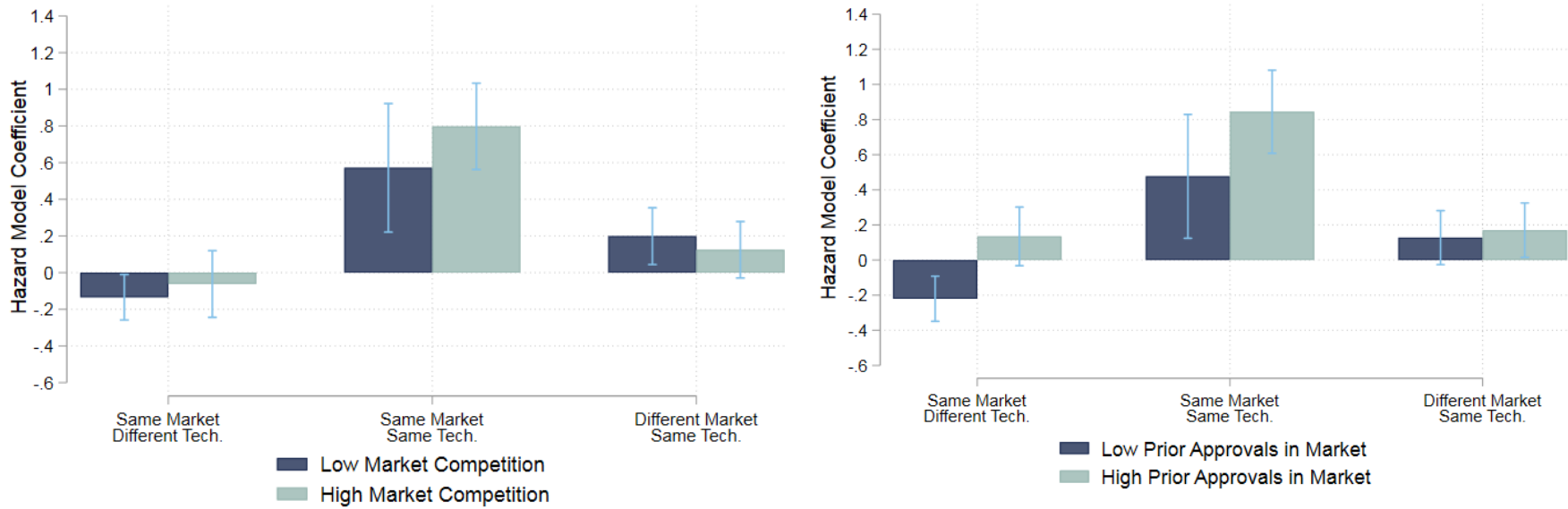


Note: The points in the above plots correspond to coefficient estimates stemming from the Cox proportional hazard model, where the variable for treatment status is interacted with the time (in six month increments) since the first competitor failure event (of each type). The six months prior to the first competitor termination event is the omitted variable. The 95% confidence intervals (corresponding to robust standard errors) are plotted with capped spikes.

**Figure 7: Level of Competition and Response to Competitor Failure**

**A. Level of Market Competition (in Development)**

**B. Level of Market Competition (Approved Drugs)**



Note: The bars display the coefficients of interest from the main Cox proportional hazard model specification, split the median number of competitors, defined in two different ways. In Panel A, the level of competition is defined as the number of development projects working on the same therapeutic indication. In Panel B, the competition split is based off the number of previously approved drugs within the same therapeutic indication. The 95% confidence intervals are plotted with capped spikes.

## Appendix A: The Drug Development Process and Disclosure Requirements

**The Phases of Development.** The FDA defines five steps in the drug development process. The first is discovery and development, where scientists may screen thousands of existing compounds, create new chemical structures, and use biological assays to determine what drug candidates might be effective in treating a disease. In the second step, preclinical research, developers engage in both *in vitro* (i.e., in a test tube) and *in vivo* (in a living organism) tests of their drug candidates—looking for signs that the compound interacts with the biological target of interest, as well as any potential toxicity success in fighting the disease of interest. If a drug project survives through the first two stages, the developer may begin clinical research, where humans become the test subjects. This involves three phases.

Each phase of these pre-approval clinical trials include increasing degrees of rigor and cost. Phase I trials typically have between 20 and 100 healthy volunteers, last a short period of time, and are intended to test safety and dosage. Phase II trials are larger (several hundred people who have the condition of interest), and typically require randomization into treatment and placebo (or the existing standard of care) groups. Phase III is essentially a larger scale version of the phase II trials, usually involving more participants (often thousands) who are tracked over a longer period of time. Trial costs range from thousands of dollars in phase I, to hundreds of millions in phase III (FDA website; Manhattan Institute, 2012).

Before the Federal Drug Administration Modernization Act in 1997, there was no centralized resource for publicly accessible information on current and past clinical trials. This act led to the formation of [clinicaltrials.gov](http://clinicaltrials.gov), a central repository for clinical trial information that required developers to post information about phase II and later clinical trials.<sup>i</sup> Trial registries now provide the bulk of information about where drug development projects stand in the process, though companies also release information about development time lines and trial outcomes via company documents (e.g. “pipeline tables” with the status of drug candidates), investor relations materials, financial files, and conferences.

Often, the earliest steps of drug development are conducted in smaller firms or in collaboration with academic researchers, while only larger pharmaceutical or biotechnology firms can provide the scale and capital needed for later stage clinical trials and marketing activities. The size of the firm and origin of the project has been shown to impact how the developer decides whether to continue a project (Guedj & Sharfstein, 2004; Hermosilla, 2015).<sup>ii</sup>

After completing the gauntlet of clinical testing, the developer can file a New Drug Application (NDA) that presents the full set of preclinical and clinical testing results. At this point, the FDA assigns a review team to evaluate the findings, inspect clinical trial sites, and eventually make a recommendation regarding approval. Despite the years of testing and data collection, these decisions often remain quite uncertain. Surprising rejections may reverberate throughout the industry, and

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<sup>i</sup>While initial compliance with trial registration rules was low, registration rates accelerated after the International Committee of Medical Journal Editors (ICMJE) initiated a policy whereby trials must be registered as a prerequisite for journal publication (Gil, 2012).

<sup>ii</sup>Contract research organizations are also retained at different points throughout the development process to run experiments and trials on developers’ behalf. The decision to outsource projects depend on both the specialized knowledge required for the R&D and the governance structure of the firm (Azoulay, 2004; Guedj, 2005).



change firms' subsequent willingness to invest in and continue projects (Blankshain et al., 2013). In the fifth and final step, developers continue to provide post-approval safety and monitoring data (e.g., Phase IV clinical trials, adverse event reporting, and other post-marketing surveillance).

**Disclosing Discontinuation.** Drug development firms disclose the discontinuation of a development project through various announcement mechanisms. The decision to shut down a project is often reported in a company press release (especially when the company is public and the discontinuation news may be considered material to the company's market valuation), in updated drug development pipeline documents (usually posted on the firm's website), and in financial filings. Cortellis for Competitive Intelligence tracks these disclosures and links them to drug development projects (drug-indications). For public companies, these disclosures often have drastic and immediate impact on stock price. A number of studies have documented how negative clinical trial news consistently leads to large statistically significant stock drops for the developer firm (Sharma & Lacey, 2004; Girotra, 2007; Perez-Rodriguez & Valcarecel, 2012). These reactions corroborate that the disclosures qualify as a "shock" or surprise to market participants. To illustrate how these disclosures appear, I include two representative examples in the next section.

Public companies have more incentives to report their project disclosures to the entire market in a timely fashion. Regulation Fair Disclosure, enacted by the U.S. Securities and Exchange Commission in 2000, requires that publicly traded firms disclose all material information to investors. In practice, this regulation means that firms must disclose via press releases (or social media, as of April 2013) any information that might impact the stock price. In the Supreme Court case of *Matrixx Initiatives, Inc. v. Siracusano* (2011), the limits of this rule were clarified after a pharmaceutical company failed to disclose news that its drug may cause adverse events. In the unanimous decision, Justice Sotomayor wrote in favor of the existing standard that the "materiality requirement is satisfied when there is 'a substantial likelihood that the disclosure of the omitted fact would have been viewed by the reasonable investor as having significantly altered the "total mix" of information made available.'" While this decision did not result in a "bright-line rule" regarding materiality of clinical trial results or adverse event information, it did reaffirm that public companies need to announce significant negative events involving drug development.

While I cannot ascertain the exact coverage of discontinuation disclosure, one can expect that public firms (who run the vast majority of phase II trials) will err on the side of caution and disclose project exits in a timely fashion. Small biotech firms may be theoretically less likely to make official announcements shortly after the decision to abandon the project is made; however, the Cortellis database includes discontinuation disclosures from all types of firms. As mentioned at the end of Section 3.2, small firms do not have a greater proportion of "zombie" projects, which remain listed as active, despite having no development activity for a substantial period of time.

**Disclosure Examples.** It is common for firms to disclose that they are shuttering a drug development project via press release. For example, Evolutec Group announced it would discontinue development of their drug Votucalis on January 3, 2007. Evolutec had been developing the compound to treat conjunctivitis, lacrimal gland disease, and ocular inflammation. The drug had entered phase II clinical trials in December 2003. At the time of discontinuation, the company put out a press release:

*Evolutec Group plc (AIM: EVC), the biopharmaceutical company developing novel products for the treatment of allergic, inflammatory and autoimmune diseases, announces that rEV131 did not meet its*

*primary endpoint in the Phase II post-cataract inflammation trial.*

*In the trial, rEV131, dosed twice-a-day, was compared to prednisolone, the standard of care, dosed four times-a-day, and placebo. The primary endpoint was inflammation 14 days after cataract surgery. There were no significant differences between rEV131 and placebo whereas prednisolone performed as anticipated.*

*Evolutec will announce its preliminary results for 2006 on 27 February 2007 and expects to report cash and held-to-maturity investments as at 31 December 2006 of STG8.7 million. Mark Carnegie Brown, Chief Executive of Evlutec, said: "Following this disappointment, no more investment will be made by Evlutec in rEV131. Whilst we continue to explore partnering opportunities with rEV576, all strategic options to realise value for shareholders are under consideration. rEV576 has demonstrated exciting preclinical results in myasthenia gravis and Guillain-Barré Syndrome where there are currently no curative therapies."*

While some discontinuation disclosures are quite specific about the reasons for failure (like the above), others are more vague about the rationale for stoppage. In November 2010, QLT Inc. discontinued its development of compound QLT091568 for flaucoma and ocular hypertension. The drug had entered phase II trials in September 2008, but November 3, 2010, the company mentioned in its Q3 earnings announcement that development would halt, stating simply:

*The Company has discontinued development of QLT091568, a prodrug of a beta adrenergic antagonist (a novel beta blocker), that was under investigation for its potential ability to lower intra-ocular pressure in glaucoma and ocular hypertension patients.*

On occasion, companies disclose when their project termination decisions are influenced by competitor outcomes. For example, in April 2012 Newlink Genetics used a press release to announce that due to a competitor failure, it was stopping a drug project (a combination therapy) for treating melanoma:

*...the Company has determined that it will not initiate the randomization portion of Indigo301, its study of indoximod in combination with pembrolizumab or nivolumab for patients with advanced melanoma. NewLink's clinical team will evaluate the design, trial size and feasibility of an alternative randomized evaluation of indoximod in melanoma **in the context of the failure of a competitor's trial of its enzymatic IDO inhibitor in a similar clinical setting.** The evaluation will include analysis of the full data set from the Company's single-arm Phase 2 melanoma study, the differentiated mechanism of action of indoximod, and the opinions of experts in the field. The Company will present final results from its Phase 2 trial in melanoma and its single-arm Phase 2 trial in pancreatic cancer at an upcoming medical conference in the first half of 2018. [emphasis added]*

## Appendix B: Alternative Econometric Specifications

In this section, I describe the alternative econometric specifications employed to estimate the effect of competitor news on project exit decisions. In the main results, I present a survival model difference-in-differences approach to measuring firms’ response to competitor project discontinuation news on a panel of development project-quarters. I favor this survival model framework because it accounts for the natural death rate of projects as they move through their development timeline, and allows one to control for different life cycles across therapeutic areas. Some of these same analysis qualities can be applied through alternative regression specifications. Below I describe three of these alternative specifications.

### OLS with Project-Level Panel

This specification uses the same drug-indication-quarter panel as the survival model specifications in the main body of the paper. The outcome variable is an indicator variable for whether or not the focal drug-indication (“project”)  $i$  was discontinued in quarter  $t$ . Unlike the hazard model, which censors drugs out of the analysis sample after their discontinuation, I keep all projects in the analysis sample for their entire time in phase II (up to 32 quarters). To avoid right-censoring, I drop projects that had neither reached 32 quarters, exited, or graduated to phase III as of the end of 2014. While the hazard models contain a baseline hazard rate,  $h_0$ , in the OLS version, I control for natural death rates through time using indicator variables for each value of phase II age (quarters since entered phase II) as an independent variable. As with the hazard models, I include time (financial quarter) fixed effects and control for therapeutic area (market),  $k$ , and define “treatment” groups based on relationship to competitors’ discontinued projects both in terms of market ( $k$ ) and drug target-action ( $q$ ). The resulting regression equation is as follows:

$$\begin{aligned} DISCONTINUED_{ikt} = & \beta_0 + \beta_1(SAME\ MKT, DIFF\ TECH\ NEWS)_{k,-q,t} \\ & + \beta_2(SAME\ MKT, SAME\ TECH\ NEWS)_{k,q,t} \\ & + \beta_3(DIFF\ MKT, SAME\ TECH\ NEWS)_{-k,q,t} \\ & + f(AGE_{ikt}) + \gamma_t + \delta_k + \epsilon \end{aligned}$$

As with the main hazard model specifications, the different treatment groups can be defined as absorbing states (“turned on” after the first competitor project of a given type discloses an exit), counts of the associated event type, or as “treatment” windows (“turning on and off” depending on proximity in time to the competitor exit news).

Column 3 in Table B3 reports the coefficients corresponding to  $\beta_1$ ,  $\beta_2$ , and  $\beta_3$  in the two-period (before and after competitor news), absorbing state treatment version equation above. At a high level, the results are qualitatively the same as the hazard model specifications: SM-ST news leads to the biggest change in propensity to exit, SM-DT news leads to a small and possibly negative impact on exit rates, and DM-ST leads to a modest increase in pulling the plug.

## Dyadic Data Construction

An alternative way to evaluate the impact of competitor exit on project continuation decisions is to analyze each event in project pairs. Under this approach, a unit of observation includes the relevant exiting drug-indication project  $i$  (“treating,” or signal “sender”), a focal project  $j$  that experiences the discontinuation news as a market or technology competitor (“treated,” or signal “receiver”), and the time period  $t$  (financial quarter). Within each dyad’s panel data, there may be only one “treating” event, and the “treating” project must exit prior to the “treated” project’s exit. The resulting data set has 11,017,691 observations with 151,840 treating-treated pairs.

## Dyadic OLS

The baseline OLS specification is

$$\begin{aligned} DISCONTINUED_{ijkt} = & \alpha_0 + \alpha_1(SAME\ MKT, DIFF\ TECH\ NEWS)_{k,-q,t} \\ & + \alpha_2(SAME\ MKT, SAME\ TECH\ NEWS)_{k,q,t} \\ & + \alpha_3(DIFF\ MKT, SAME\ TECH\ NEWS)_{-k,q,t} \\ & + f(AGE_{jkt}) + g(EntryLag_{ij}) + \gamma_t + \delta_k + \theta_{ik} + \epsilon \end{aligned}$$

The regression results corresponding to the equation above are reported in Table B3, Column 1. Again, we see the largest effect corresponds to the SM-ST news, with a smaller increase (about one half the magnitude) associated with DM-ST news. The major divergence from the single-level data set results is that the SM-DT coefficient is positive and significant, though smaller than the coefficients from the other two groups.

Figure B1 shows the dynamic event study version of these results, splitting each coefficient out by number of quarters before and after the competitor news event. The one key difference in interpreting the OLS version instead of the hazard model version is that the “treatment effect” is cumulative in the OLS version. An positive coefficient the OLS model event study means that the probability of a project being discontinued in  $t$  quarters since treatment is higher than the probability of a same age and same indication untreated project being discontinued in that same time period; though the divergence in their exit rates may have occurred at an earlier point in time (e.g.,  $t - 1$ ).

## Dyadic Hazard Models

The last version of the alternative regression specifications applies the Cox proportional hazard model to the dyadic data. As in the main specification in the paper, I stratify the baseline hazard by therapeutic indication (market) and evaluate each type of competitor news’ impact on the hazard rate of project discontinuation. As in the dyadic OLS specification, I add in control variables for the “treating” project  $ik$  and the time elapsed between the “treating” and “treated” project’s entries into phase II development:

$$\begin{aligned}
h_{i,j,k}(t, X) = & h_{0k} \times \exp[\alpha_1(SAME\ MKT, DIFF\ TECH\ NEWS)_{k,-q,t} \\
& + \alpha_2(SAME\ MKT, SAME\ TECH\ NEWS)_{k,q,t} \\
& + \alpha_3(DIFF\ MKT, SAME\ TECH\ NEWS)_{-k,q,t} \\
& + g(EntryLag_{ij}) + \gamma_t + \theta_{ik}]
\end{aligned}$$

Table B3, column 2 reports the results of the two-period version of this model. The results are in-line with the dyadic OLS results, except that the SM-DT coefficient is only marginally significant (at the 10% level), and smaller in magnitude relative to the other two coefficients of interest. The event study version, depicted in Figure B2, shows large spikes in the hazard rate of own project discontinuation in the first quarter after the competitor exit for both the SM-ST and DM-ST groups. The SM-DT coefficients are mainly flat, with a small increase right immediately following the competitor news, but are all not significantly different from zero.

## Appendix C: Additional Robustness Checks

**Exploring Treatment Intensity and Potential Cascading.** The history of competitor failure within a particular market or technology area could potentially amplify or moderate the reaction to project discontinuation. To understand how the sequencing of failure events affects the response to competitor failure, I use two separate subsample analyses. In the first, I address how firms respond to the first competitor failure (of each type), compared to subsequent failure events. Second, I separate cases of lone project failures (“singletons”) from those where multiple projects within a given market and/or technological area exit within a short window of time (“clusters”). The goal of these analyses is to evaluate whether multiple signals catalyze information cascades and produce competitor responses that differ from the average effects summarized above.

To compare initial to subsequent treatment events, I interact the treatment variables from the main regression specification (two quarter window since the given type of competitor failure news) with indicator variables for whether the particular competitor failure window was the first of its kind experienced by the focal project. The results from this regression are reported in Appendix Figure C2.A. The magnitude of each treatment effect is quite similar and not statistically distinguishable between first and subsequent treatments.

In the second analysis, I compare instances where a cluster of competitors abandon their project in a short period of time, to cases where the failure signal is from a sole competitor project. The motivating intuition is that multiple failure events might represent a stronger signal, and exit decisions might be influenced by watching other firms respond to an initial event (e.g., “if they think it’s bad news, then we should too”). The results (displayed in Appendix Figure C2.B) show some differences between the two scenarios. Most notably, the DM-ST coefficient for the clustered failure treatment scenario is relatively large (0.31) and statistically significant from zero ( $p < 0.01$ ), and significantly larger than its solo treatment coefficient counterpart ( $p < 0.05$ ). The magnitude of the solo SM-ST coefficient appears larger than the clustered treatment version, but they are not statistically different since the rarity of the clustered SM-ST treatments results in a very noisy estimate.

**Type of Failure and Signal Strength.** In addition to market and technology categories, the nature of a failure signal may vary along other dimensions. Ideally, one would want to characterize each competitor failure event into detailed reasons for failure and strength of signal. In practice, such characterization is difficult due to both the volume of failure events, and the limited and heterogeneous information that firms provide about their discontinuation events. That said, one can use features of the informational content as proxies for different types of failure signals. I investigate two of these informational features that might provide meaningful proxies for the type of failure event.

First, I use actions involving a drug’s other development projects as surrogates for the type of failure. As described above, firms may develop a single drug for multiple therapeutic indications. Whether a firm discontinues the development of a drug for multiple indications or just a single indication may reveal (or correlate with) the reason for the discontinuation decisions.

For example, in July 2013, AstraZeneca stopped development of the drug Pasireotide for the indication of meningioma, but continued developing the drug for other indications. In contrast, Pfizer halted all development of Figitumaumab for seven different conditions in February 2011, after

it terminated trials early for safety concerns. While the former example may have involved safety concerns of its own, such concerns would have been indication-specific since the firm continued development for the other diseases. Furthermore, if AstraZeneca stopped the meningioma project due to efficacy concerns, then the efficacy signal was not strong enough for the firm to cancel related projects.

To test whether the single- vs. multiple-indication discontinuation does indeed reveal information about the nature of a failure event, I first looked at a sub-sample for which I could determine the reason for failure and examine the correlation between their reasons for failure and multiple indications discontinuations. Clinicaltrials.gov requires that trial sponsors enter the reason for early stoppage when trials do not run until completion. Failure reasons are available for 300 of the “treating” projects in my sample because they involved early terminated trials. I found a statistically significant positive correlation between multiple-indication discontinuations and “safety” reasons for failure. However, I found no such correlation for “efficacy” or “strategic” reasons for project termination. These results make intuitive sense, because safety issues are more likely to effect all patients, while efficacy issues are likely disease-specific.

The results of this analysis are displayed in Appendix Figure C3. In each case the magnitudes are greater for the multiple-indication failure group (within each competitor news type). The biggest difference is in the DM-ST failure news type, where the multi-indication failure response is significantly larger than the single-indication failure response ( $p < 0.05$ ), and the single-indication failure response is not statistically different from zero. The difference between the two subgroups shows that multiple-indication (likely safety) events are driving the main effects found for DM-ST news, despite being the minority of such events.<sup>iii</sup> Safety concerns could be a sufficient signal for same technology learning, but efficacy problems only spark competitor response when the projects also share the same disease group.

I also investigated the proximity in time between discontinuation announcements and the end of the drug project’s last trial. If discontinuation announcements are completely decoupled from one’s own R&D results, they might reveal strategic concerns rather than information about the discontinued project’s quality.

To address these concerns, I compared the response to competitor discontinuations announced close (in time) to the end of the focal project’s last clinical trial to those more than one year following the last trial outcome. When I interact the main treatment specifications with whether or not the failure news was within one year of the last trial date, I found no statistically significant differences between the sub-groups (results not shown). My primary interpretation of this result is that firms are indeed responding to competitor failure news rather than other types of information, because the reaction is so similar for both the trial-linked news (whose timing is unlikely to coincide with other news types) and decisions made further away from trial end dates.

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<sup>iii</sup>Thirty-seven percent of the DM-ST competitor events involve multiple-indication competitor failure events.

## Appendix D: Firm Heterogeneity and The Impact of Failure News

**Portfolio Characteristics.** Organizational characteristics might also influence the response to competitor failure news. Drug developers can be characterized along numerous dimensions, but here I focus on factors that are likely to alter a firm’s opportunity cost for continuing a given project, or moderate the firm’s ability to accurately interpret competitor failure signals. One of the attractive features the analyzing drug pipeline data is the ability to assess a firm’s entire portfolio of project investments. I focus on two characteristics that are particularly relevant to firm agility, and expertise. First, I evaluate how firms’ overall concentration of projects influences their continuation decisions. Second, I test how firm experience in a given market or technology area impacts response to competitor failure news. Though a firm’s level of portfolio diversity and expertise reflect strategic choices regarding organizational structure and value creation, these choices may also have systematic impact on a firm’s agility and decision-making.

I measure portfolio concentration using a simple Herfindahl index of projects by company-quarter. Snapshots of different portfolios in time help to illustrate the variation in firm concentrations and commitments. On one extreme, the data includes small biotechnology firms like Discovery Genomics, who in 2003 had two active development projects for a blood disease (Hemophilia B) using two different targets. This portfolio yields a market Herfindahl index of 1, and technology Herfindahl index of 0.5. In contrast, Schering-Plough had a large and diverse set of over 100 active pipeline projects in 2000, resulting in a market index of 0.01 and a technology index of 0.02. Since small firms like Discovery Genomics do not have enough development drugs to allow for much variation in market and technological concentration, I limited the analysis to firms with more than 10 development projects.

Appendix Figure D shows the overall exit rates for phase II projects in the sample, breaking the sample down into subgroups of interest. Consistent with prior work (Guedj & Sharfstein, 2004; Guedj, 2005; Lowe & Ziedonis, 2016), I find that smaller (and inexperienced) firms are much less likely to abandon projects (Figure D1.A). On average, firms with higher market or technology portfolio concentrations are less likely to terminate their phase II projects (Figure D1.B and D1.C). This propensity to move forward with development may reflect higher levels of commitment to a particular therapeutic mission, sales channels, collaborations, or scientific hypothesis. Since the relationship of interest is how competitor news alters project continuation decisions, I control for the different groups’ baseline hazard rates of project exit by splitting the sample into low and high concentration firms using a median cutoff, and run the main regression specification on each group.

The regression results are displayed in Figure D7. These regressions exclude firms with less than 10 projects in order to eliminate very small biotech firms that are unlikely to have a choice of pipeline market or technology diversity. The results for each group (below and above median market/technology project concentration) are quite similar to the overall average effects, but with one exception. For both the market and technology definitions of portfolio concentration, the low project concentration group exhibited no significant project updating following a DM-ST competitor failure, with effect magnitudes right around zero. Meanwhile, the estimates for the high concentration groups indicate that following DM-ST, their propensity to terminate their own project increased 27% for the high market concentration group and 52% for the high technology concentration group.



Focused firms appear more likely to act on DM-ST competitor results news, while the more diverse firms do not deem that type of news to be as actionable. The most intuitive explanation for this difference is that focused firms monitor their technological rivals more studiously, since they have so much at stake in their main technology areas. Another explanation is that firms with less project concentration prefer to maintain diversified portfolios and do not mind fluctuations in the risk of any one part of their portfolio. Under this perspective, a focused firm cannot afford to see large (correlated) portions of its portfolio go down together, while the diversified firm can absorb new risk that is isolated from its other bets.

To measure how experience in a particular technological area influences the response to competitor failure news, I split the sample into companies that were above and below the median of project experience (three prior drugs developed) within the focal technology area at the time of competitor discontinuation news. The results are displayed in Figure D8. Here, the response to SM-DT and SM-ST news is fairly similar for both groups. The response to DM-ST news is quite different across levels of experience. The high experience group accounts for all the increased propensity to discontinue following DM-ST competitor discontinuations, while the low experience group does not respond to this type of competitor news, on average. Again, inattention is a possible explanation if the low experience firms are less knowledgeable, or are not as tied to the network of scientists and doctors working in these target areas. Another interpretation is that the low experience areas tend to be more novel therapeutic approaches, so developers put less stock in their (equally inexperienced) competitors' project failures.

**Firm's Commitment to Focal Drug.** Another project feature that might influence the firm's response to competitor failure news is the firm's level of commitment to that project. Firms may be more committed to a particular project for a number of reasons. For example, senior leadership may have particularly strong beliefs in a given idea. Here, I evaluate how project commitment influences the response to competitor failure news by using variation in the number of therapeutic indications for which the firm is developing a given drug. In my analysis of competitor reactions, my unit of analysis is the drug-indication level. However, a firm may develop a single drug for more than one indication at a time—though one indication is usually the “lead” indication. By doing so, the firm is committing more resources and effort to develop that particular drug.<sup>iv</sup>

Appendix Figure D9 displays the results of this analysis. I divide treatment events into events where the focal project had one or less additional indications in development (low commitment), and projects where the firm was also developing the drug for two or more additional indications (high commitment). I find that the SM-DT and SM-ST news have no statistically significant differences in average response to the failure news. However, the low commitment group has a significantly higher response than the high commitment group to DM-ST news ( $p < 0.01$ ). In fact, the low commitment group accounts for all of the positive response that I find in the average full sample response to this type of news. Taken together, these splits imply that commitment does not completely mitigate learning effects, but it does correlate with firms' willingness to alter investments in response to DM-ST news. Once again, the DM-ST response is quite sensitive to the organizational or competitive context—and those effects seem driven by a smaller, yet highly susceptible subgroup—while the SM-ST learning effects are quite robust across different different scenarios.

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<sup>iv</sup>Economies of scale are possible when developing a drug for different conditions. For example, some preclinical testing of toxicity in animals might be applicable for multiple human indications.

## Appendix E: Molecular Similarity and Competitor Failure News

**Chemical Similarity Data.** While shared target-actions serve as the primary measure of technological similarity in this paper and in related analyses from industry researchers (Cook et al., 2014; Shih et al., 2018), I also evaluate technological distance using chemical informatics techniques comparing the structural makeup of small molecule drugs. Rather than directly measuring functional similarity, this alternative approach quantifies the physical similarity of any two drug compounds.

Cortellis has chemical structure information for roughly 40% of Cortellis drug projects (drug-indications) and 60% of projects that reach phase II clinical trials. Those percentages rise to approximately 60% and 85%, respectively, when restricting to small molecule drugs (i.e., excluding biologic drugs and vaccines).

Pairwise chemical similarity measures are quantitative measures for comparing the structural elements of compounds. Chemists and drug developers often use drug similarity to predict chemical properties and to screen and compare drug candidates. For the analyses in this paper, I used the “tanimoto” chemical similarity data collected in Krieger, Li and Papanikolaou (2019). That paper takes the full set of SMILES codes associated with Cortellis drugs and uses the open source ChemmineR package<sup>v</sup> to calculate all pairwise chemical similarity scores (where similarity score is greater than or equal to 0.1).

**Chemical Similarity Results.** Figure E1 describes the pairwise similarity data in this paper’s analysis data. These pairs include all the phase II focal projects and their relevant “frontier” competitors (see Section 4 for more detail on the sample). The three panels in Figure E1 separate drug dyads into the three types of competitor relationships: 1) SM-ST, 2) SM-DT, and 3) DM-ST.

The median same technology pair has a chemical similarity of less than 0.23 (where a score of 1 means 100% chemical structure overlap), and fewer than 4% have similarity scores over 0.9.<sup>vi</sup> These distributions confirm that, despite sharing more structural features, that same target-action compounds still have plenty of chemical diversity <sup>vii</sup>

While group has plenty of variation in pairwise similarity scores, the mean similarity scores are at least double in the same technology groups than they are in the SM-DT group. This pattern is in line with Krieger, Li and Papanikolaou (2019) which finds that sharing the same target-action is associated with doubling in chemical similarity (on average), while merely sharing the same indication is associated with an approximately 25% increase in compound similarity score. In other words, same target-action (“technology”) drugs are more likely to share structural features, even though many of these same target-action drugs are chemically quite different from one another.

Using the subset competitor pairs that are both small molecules, I analyze how compound similarity moderates the response to competitor failure news. For this survival analysis, I use the dyadic level data described in Appendix C, and Cox proportional hazard models similar to the main

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<sup>v</sup>Full documentation: <https://www.bioconductor.org/packages/release/bioc/vignettes/ChemmineR/inst/doc/ChemmineR.html>

<sup>vi</sup>Krieger, Li, and Papanikolaou (2019) define “me-too” drugs as those with similarity to previously developed drugs of greater than or equal to 0.9.

<sup>vii</sup>Aspects of the patent system also prevent too many copy-cat drugs. For a given new compound, firms can patent a “Markush” structure which covers very closely related chemical compounds.

results (Table 3), with three quarter treatment windows (“within two quarters since competitor failure news”).

The results show that chemically similar compounds are far more reactive to competitor discontinuation than chemically dissimilar focal projects. First, I pool the competitor dyads and separately estimate the treatment coefficient and the treatment interacted with compound similarity of the the focal project to the “treating” competitor (Column 1). The former is insignificant, while the latter is highly significant and implies that a one standard deviation increase in failing competitor’s chemical similarity (0.14) results in a 40% increase in the likelihood of project exit. Said differently, a jump from the 25th percentile to the 75th percentile in project similarity increases the competitor response (likelihood of exit) by 61%.

However, the relationship between similarity and competitor response is not uniform. When I further split the treatment variables by “type” of failure news and interact each type with focal and competitor project compound similarity, the results show more nuance (Column 2). On average, focal projects with low similarity to SM-DT competitors do not respond to low similarity competitor failure news. However, for the SM-DT news, a high level of structural similarity leads to roughly the same response as to competitors with SM-ST and low structural similarity. Even with low levels of compound similarity, SM-ST competitor failures induce significant competitor exit, and that effect increases with compound similarity. However, the additional impact of compound similarity does not show up for the DM-ST group. For this group, learning from more similar compound’s trial outcomes appears secondary to the relevance of sharing the same technology (target-action). Column 3 further shows that same technology competitor signals are influential regardless of chemical similarity, while on average, the effect of same market signals is highly dependent on the level of chemical similarity.

Overall, the compound similarity data provides two helpful insights. First, these results confirm that chemical similarity is positively correlated with sharing a target-action, while the descriptive data still shows plenty of compound variation within target-action categories. Second, the regression results suggest that the degree of chemical distance is relevant for responses to competitor failure, but less relevant for same target-action competitor news. Together, these results suggest that chemical similarity provides an additional interesting layer of heterogenous spillovers, but is unlikely to be a better (more relevant) way of defining competitor sets than the straightforward same/different target-action distinction used in this paper and in Shih et al. (2018).

**Table B1: Competitor News Effects on Exit Rate, Alternative Treatment Definitions**

	(1)	(2)	(3)	(4)	(5)	(6)
After Any Competitor Failure News	-0.005 (0.077)					
After Competitor Failure News ( <i>Same Market</i> )		-0.083 (0.070)				
After Competitor Failure News ( <i>Same Tech.</i> )		0.204** (0.044)				
After Competitor Failure News ( <i>Same Market, Different Tech.</i> )			-0.106 (0.070)			
After Competitor Failure News ( <i>Same Market, Same Tech.</i> )			0.421** (0.076)			
After Competitor Failure News ( <i>Different Market, Same Tech.</i> )			0.113* (0.047)			
# Competitor Failure Events ( <i>Any Type</i> )				0.009 <sup>†</sup> (0.005)		
# Competitor Failure Events ( <i>Same Market</i> )					-0.003 (0.006)	
# Competitor Failure Events ( <i>Same Tech.</i> )					0.025** (0.006)	
# Competitor Failure Events ( <i>Same Market, Different Tech.</i> )						-0.006 (0.006)
# Competitor Failure Events ( <i>Same Market, Same Tech.</i> )						0.154** (0.037)
# Competitor Failure Events ( <i>Different Market, Same Tech.</i> )						0.012 (0.007)
Nb. of Observations	213,302	213,302	213,302	213,302	213,302	213,302
Log Likelihood	-10,527	-10,516	-10,504	-10,525	-10,520	-10,514

Note: Estimates stem from Cox proportional hazard model specifications using panel data on drug projects by quarter. The sample includes 6,183 drugs and 10,639 drug-indications. The first six variables are binary treatment variables that take on the value of one after the first competitor exit (of each type). Wald tests confirm that the coefficients in Column 3 are significantly different from one another. The bottom six variables are counts of each treatment type. All models include a full set of year indicator variables and stratify by therapeutic indication. Standard errors in parentheses. <sup>†</sup>  $p < 0.10$ , \*  $p < 0.05$ , \*\*  $p < 0.01$

**Table B2: Competitor Failure News Impact on Hazard Rate of Exit (Alternative Project “Death” Outcome)**

“Treatment” Window: within 2 quarters since competitor failure news	(1) Project Death (Inclusive Definition)	(2) Project Death (Inclusive Definition)	(3) Project Death (Inclusive Definition)
<b>Competitor Failure Type</b>			
<i>Same Market, Different Technology</i>	0.058 (0.036)	0.058 (0.036)	0.057 (0.037)
<i>Same Market, Same Technology</i>	0.521** (0.080)	0.529** (0.082)	0.571** (0.081)
<i>Different Market, Same Technology</i>	0.114** (0.042)	0.124** (0.043)	0.211** (0.045)
<b>3 Quarter Window Prior to Competitor News:</b>			
<i>Same Market, Different Technology</i>		-0.016 (0.032)	
<i>Same Market, Same Technology</i>		-0.038 (0.092)	
<i>Different Market, Same Technology</i>		-0.033 (0.040)	
<b>Controls: Number of Each Competitor Type</b>			✓
Nb. Drug-Indications	10,639	10,639	10,639
Nb. of Drugs	6,183	6,183	6,183
Nb. of Observations	143,992	143,992	143,992
Log Likelihood	-22,110	-22,109	-22,095

Note: Estimates stem from Cox proportional hazard model specifications using panel data on drug projects by quarter. The outcome is project discontinuation; however, the alternative definition of discontinuation used here includes both officially discontinued projects and projects that completed their final phase II trial for a given drug-indication and do not have any subsequent development. Under this more liberal definition of project “death,” nearly 51% of the (non-right-censored) phase II projects in the sample are discontinued, as opposed to 18% in the more conservative definition used in the main specifications. All models include a full set of year indicator variables and stratify the estimates by therapeutic indication. The competitor failure news variables are indicator variables that take on the value of one when the focal project is within two financial quarters since the given type of competitor failure event. Column 2 tests the “no-anticipation” assumption by including indicator variables for the three quarters leading up to each type of competitor failure event. Column 3 includes control variables with the number of competitor drug projects of each type that were active in clinical trials. Coefficients may be interpreted as an increase in the log hazard rate. Standard errors in parentheses.†  $p < 0.10$ , \*  $p < 0.05$ , \*\*  $p < 0.01$

**Table B3: Comparing Regression Specifications for Competitor News Effects**

	(1)	(2)	(3)	(4)
	Dyadic OLS	Dyadic Cox	Single-Level OLS	Single-Level Cox
After Competitor Failure News (Same Market, Different Tech.)	0.041** (0.002)	0.050 <sup>†</sup> (0.026)	-0.021** (0.008)	-0.106 (0.070)
After Competitor Failure News (Same Market, Same Tech.)	0.119** (0.019)	0.531** (0.086)	0.050** (0.015)	0.421** (0.076)
After Competitor Failure News (Different Market, Same Tech.)	0.062** (0.008)	0.228** (0.051)	0.015 <sup>†</sup> (0.009)	0.113* (0.047)
Nb. Observations	5,069,504	7,725,658	223,249	213,302
Nb. Drug-Indications	8,436	11,145	8,217	10,639
Log-Likelihood		-1,313,324		-10,504

Note: Estimates in columns 1 and 3 stem from ordinary least squares regression specifications, while estimates in columns 2 and 4 are from cox proportional hazard models. The regression models in columns 1 and 2 analyze the dyadic panel data at the level of treating-treated project level. Column 2 contains more observations because the OLS analysis in Column 1 is limited to projects that had either exited or graduated to Phase II by the end of 2014 (in order to avoid right-censoring issues). The regression models in columns 3 and 4 use the same single-level data described in the main body of the paper (project-quarter). The outcome variable is project discontinuation. In each model, the variables of interest are indicators with absorbing states, such that they take on the value of one after the first instance of the given type of competitor exit news experienced by the focal project. Standard errors in parentheses. <sup>†</sup>  $p < 0.10$ , \*  $p < 0.05$ , \*\*  $p < 0.01$

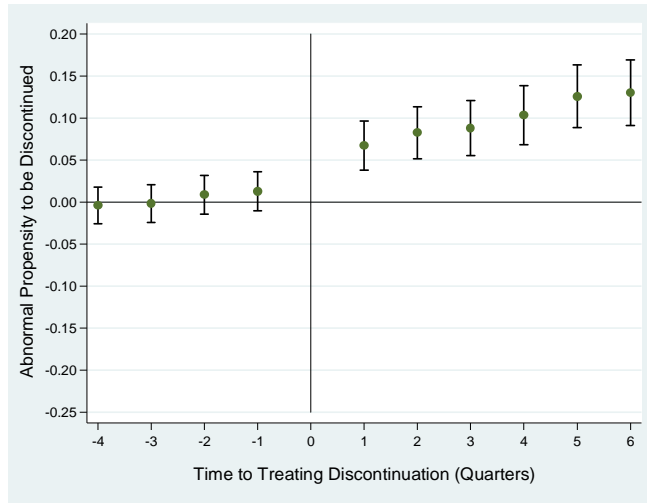
**Table B4: Competitor Failure News Impact on Hazard Rate of Exit, Restricted Sample with No Previously Approved Target-Actions**

	(1)	(2)	(3)
	Full Analysis Sample	Excluding Projects with One or More Previously Approved Target- Actions	Excluding Projects where Exact Combination of Target-Actions Previously Approved
<b>“Treatment” Window:</b> within 2 quarters since competitor failure news			
Same Market, Different Technology	-0.048 (0.050)	0.003 (0.062)	-0.006 (0.057)
Same Market, Same Technology	0.739** (0.100)	0.714** (0.144)	0.708** (0.116)
Different Market, Same Technology	0.158** (0.056)	0.333** (0.087)	0.322** (0.067)
Nb. Drug-Indications	10,639	6,664	7,919
Nb. of Drugs	6,183	4,065	4,661
Nb. of Observations	213,302	134,263	158,093
Log Likelihood	-10,496	-6,038	-7,461

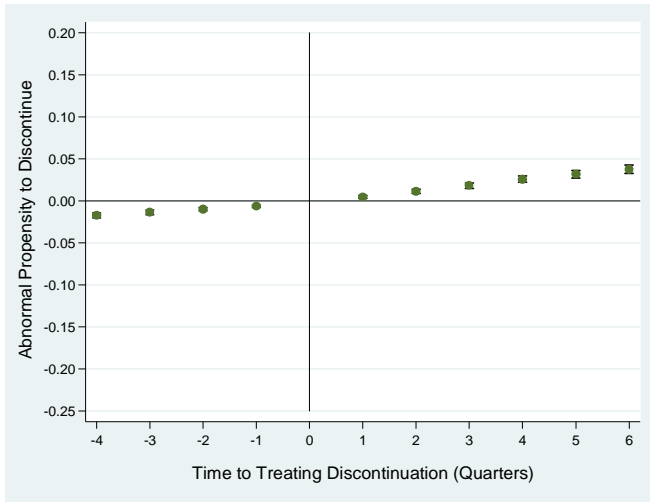
Note: Estimates stem from Cox proportional hazard model specifications using panel data on drug projects by quarter. The outcome of interest is the focal project’s discontinuation. All models include a full set of year indicator variables and stratify the estimates by therapeutic indication (ICD9). The competitor failure news variables are indicator variables that take on the value of one when the focal project is within two quarters since the given type of competitor failure event. Column 1 reports the full sample version (same as Table 3, Column 3). Column 2 applies the same specification, but limits the analysis sample to drug projects that do not share any known mechanisms (target-actions) with any previously approved drugs. Similarly, Column 3 excludes drugs that share the exact same set of target-actions with at least one previously approved drug. The sample used to generate the Column 3 results is less restrictive than the sample in Column 2 because some drugs have multiple target-actions, where only one of those target-actions was associated with a previously approved drug. In all three models, Wald tests confirm that *same market, same technology* and the *same market, different technology* coefficients are statistically different from one another ( $p < 0.05$ ). Coefficients may be interpreted as an increase in the log hazard rate. Standard errors in parentheses. †  $p < 0.10$ , \*  $p < 0.05$ , \*\*  $p < 0.01$

# Figure B1: Dynamics of Response to Competitor Failure (Dyadic OLS Version)

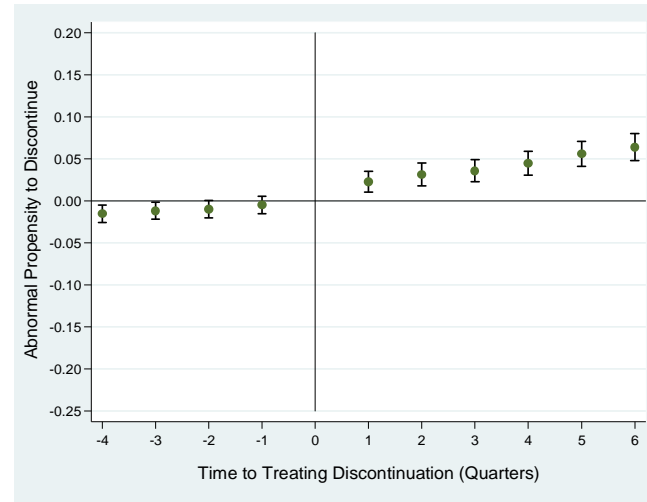
## A. Same Market, Same Technology



## B. Same Market, Different Technology



## C. Different Market, Same Technology

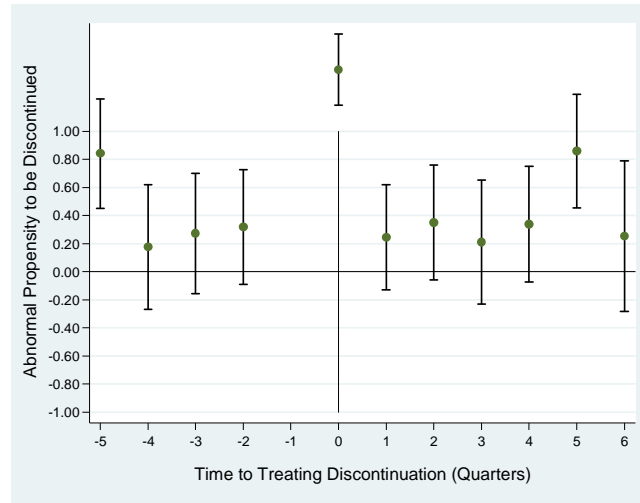


Note: The dots in the above plots correspond to coefficient estimates stemming from the ordinary least squares regression model, where the variables for treatment status are interacted with the time (in quarterly increments) until the first competitor failure event (of each type). The quarter of the first competitor termination event is the omitted variable. The analysis uses the dyadic version of the panel data (treating-project, treated-project, quarter level). The 95% confidence intervals (corresponding to robust standard errors) are plotted with the help of capped spikes.

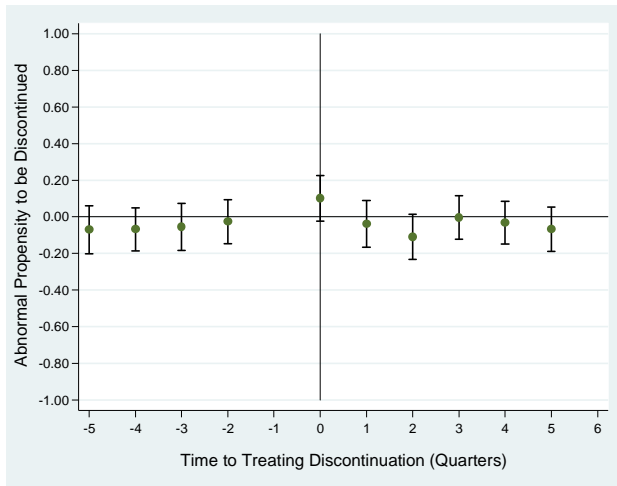


# Figure B2: Dynamics of Response to Competitor Failure (Dyadic Hazard Model Version)

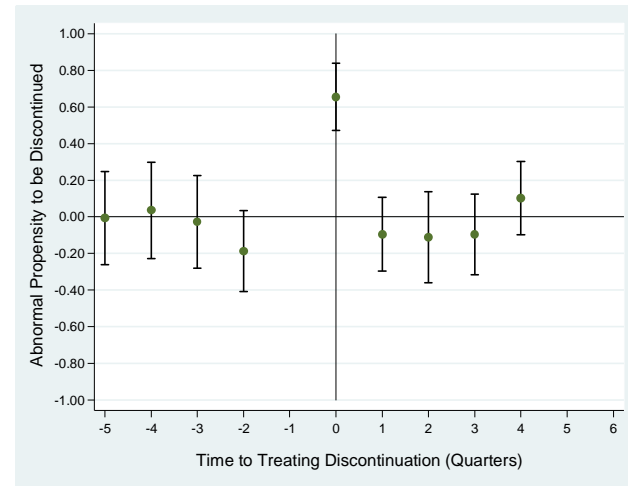
## A. Same Market, Same Technology



## B. Same Market, Different Technology

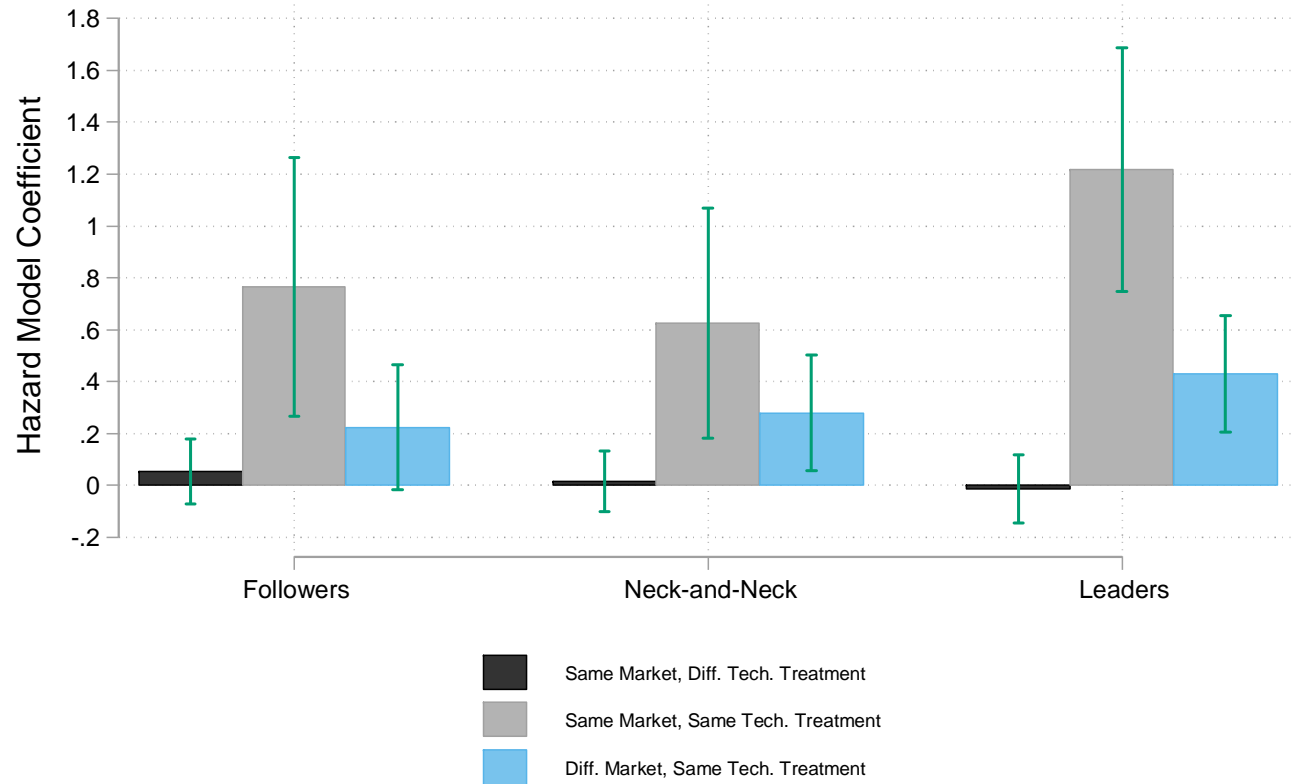


## C. Different Market, Same Technology



Note: The dots in the above plots correspond to coefficient estimates stemming from the Cox proportional hazard model, where the variables for treatment status are interacted with the time (in quarterly increments) until the first competitor failure event (of each type). The quarter prior to the first competitor termination event is the omitted variable. The analysis uses the dyadic version of the panel data (treating-project, treated-project, quarter level). The 95% confidence intervals (corresponding to robust standard errors) are plotted with the help of capped spikes.

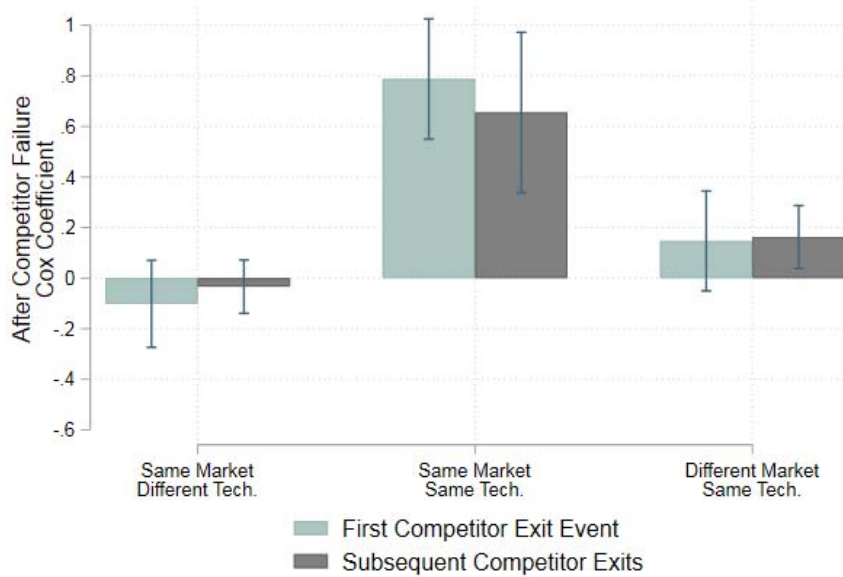
**Figure C1: Entry Order and Competitor Failure Response (Abnormal Propensity to Exit)**



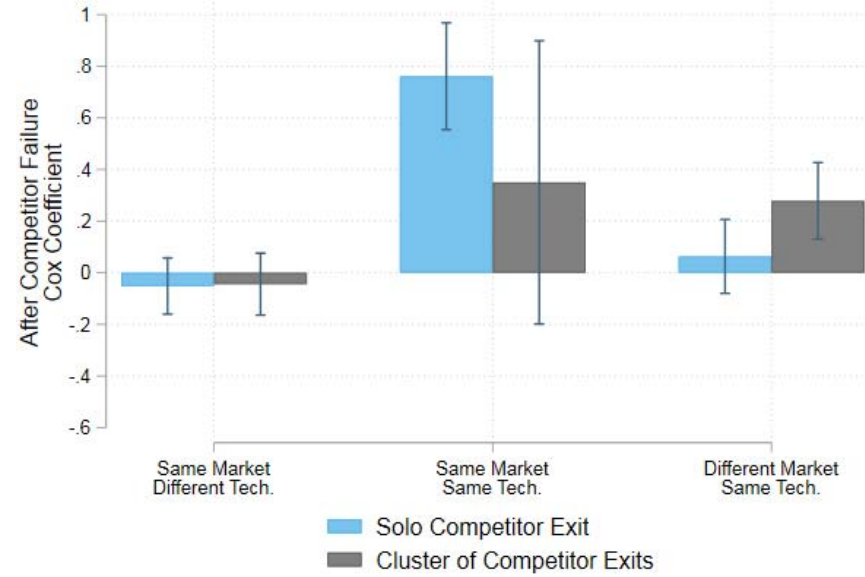
Note: The bars display the coefficients of interest from the Cox proportional hazard model specification, using analysis data which contains 212,759 project-quarter observations (discontinued projects are censored out after exit). Here, the coefficients of interest are the absorbing state of post-competitor failure news (as opposed to the within two quarter window that may turn on and off). To capture the projects entry position relative to the first failed competitor (of each type), the regression includes interaction terms for each treatment effect type with the focal (treated) firm's phase II entry position relative to their first treating project (follower, neck-and-neck, leader), and stratifies the baseline hazard by overall treatment groups (e.g. ever treated by same market, same technology) as well as therapeutic market. The capped spikes cover the 95% confidence intervals for each regression coefficient.

**Figure C2: Treatment Order and Clustering**

**A. First vs. Subsequent Treatment Events**

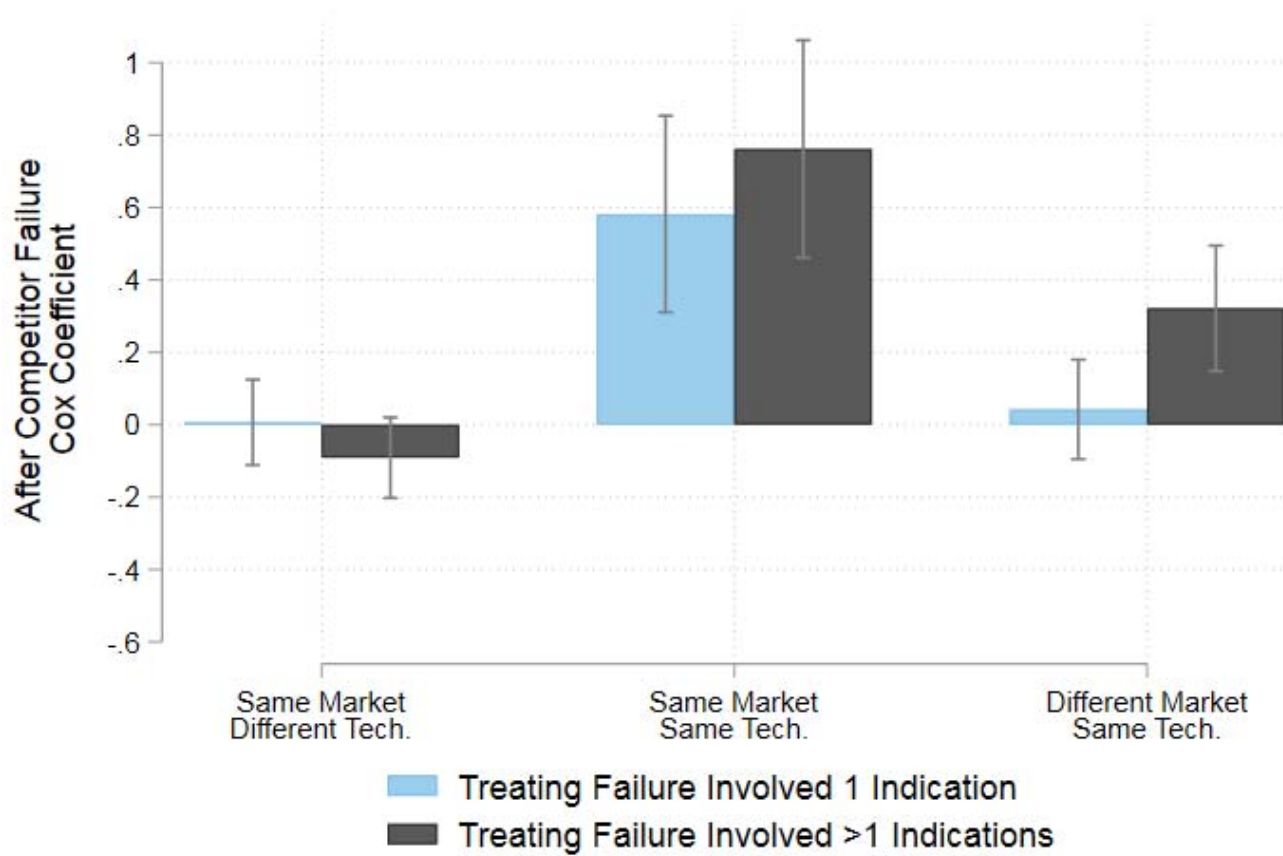


**B. Solo vs. Clustered Treatment Events**



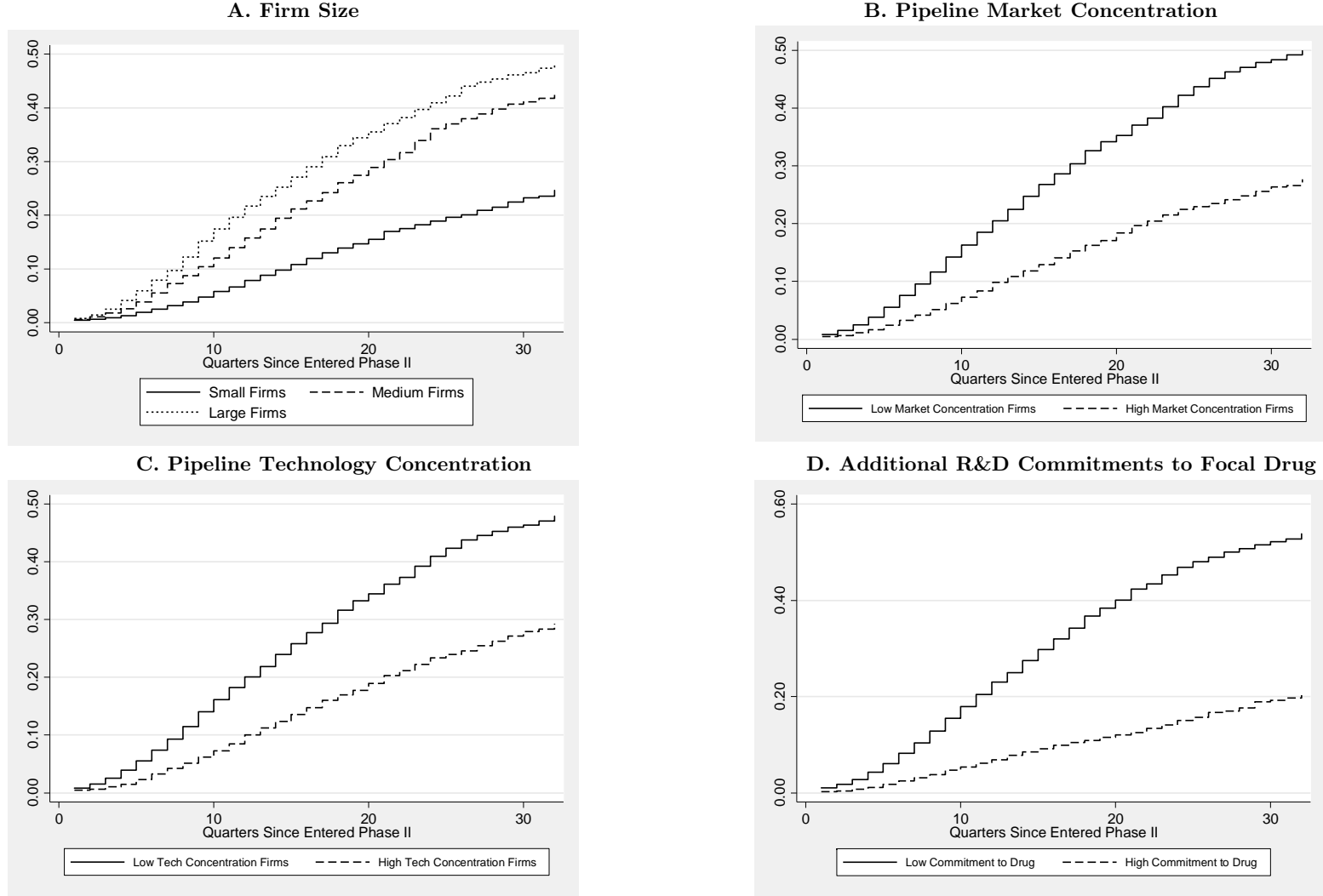
Note: The bars display the coefficients of interest from the main Cox proportional hazard model specification, where the type of competitor failure news event is interacted with whether the news event is the first of its kind or a subsequent event (Panel A), and whether the competitor failure news events involved a single competitor discontinuation or a cluster of multiple competitor discontinuations within three financial quarters (Panel B). The capped spikes cover the 95% confidence intervals for each regression coefficient.

Figure C3: Treating Failure Type and Response to Competitor Exit News



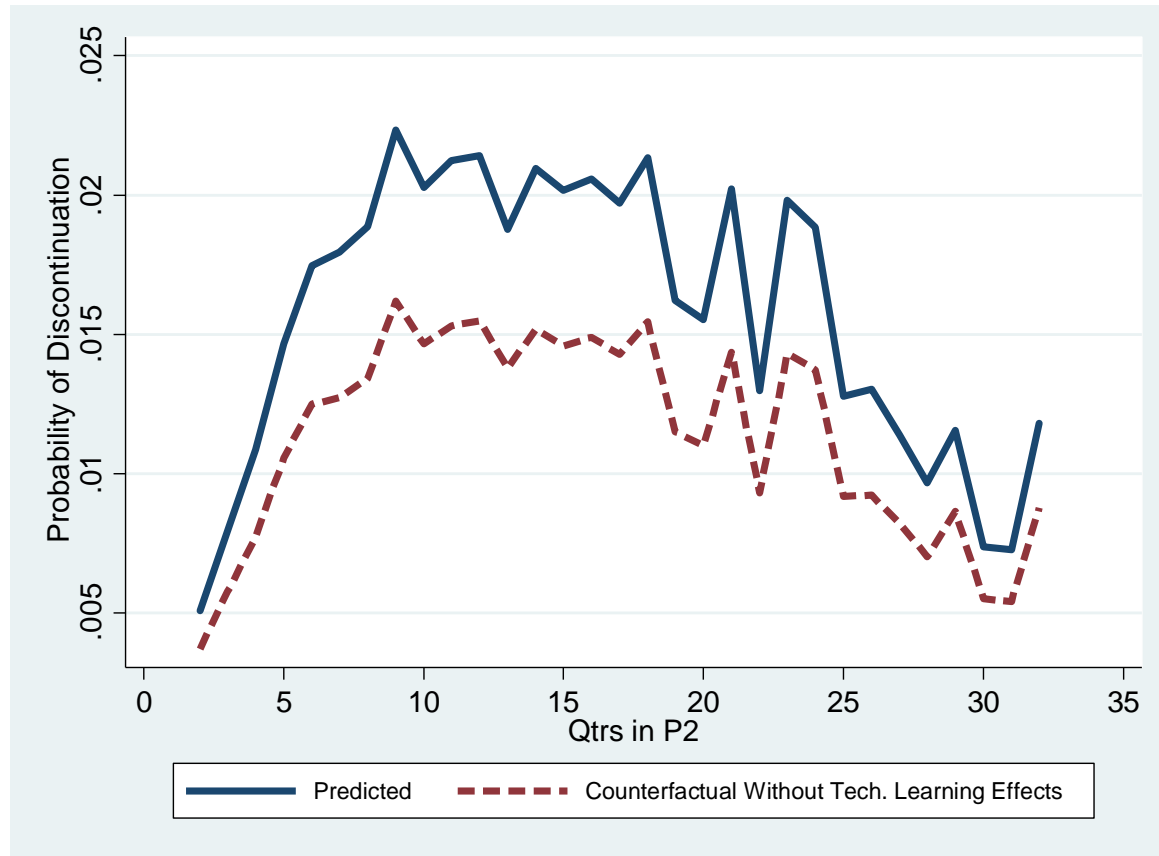
Note: The bars display the coefficients of interest from the main Cox proportional hazard model specification, with interactions for whether the competitor drug's failure news involved only one indication discontinuation or multiple indications. The magnitude of each bar represents the change in hazard rate of project exit when a focal project is within a two quarter window after a competitor failure. The capped spikes cover the 95% confidence intervals for each regression coefficient.

**Figure D1: Cumulative Hazard of Project Exit, by Project Type**



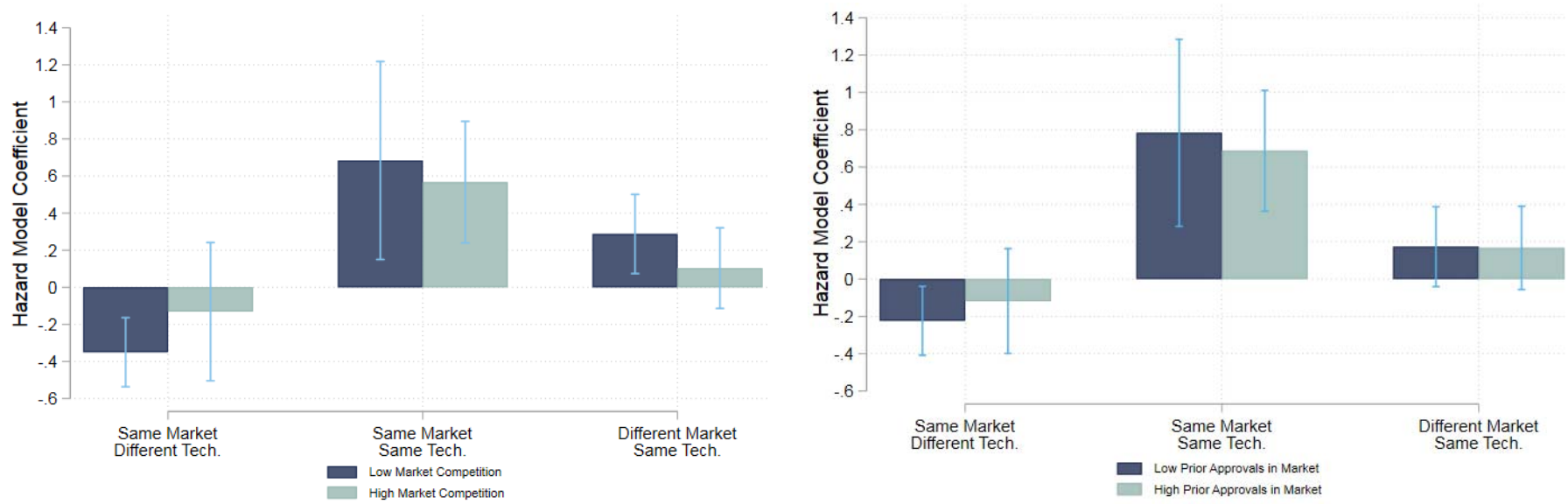
Note: The graphs plot the cumulative hazard estimate for project exit by time elapsed since the start of phase II trials. In Panel A, the hazard rate of exit is split by firm size—as proxied by the firm’s number of projects developed (low is the bottom quartile, medium is the middle two quartiles, and high is the top quartile). Panels B and C split the sample by level of therapeutic market or technological concentration of the focal firms’ projects as of the time period (median split). Panel D divides the analysis sample into drug projects for which the developing firm is concurrently developing the drug for zero or one other medical indication (low commitment) and drugs being developed for more than one other indication (high commitment).

Nm Figure D2: Discontinuation Rate by Quarter for Projects that Experience Technological Competitor Failure News



Note: The graph shows the average predicted probability that a phase II project is terminated, given that it survived until a given project age (quarters into phase II) and did not graduate to phase III. The solid line is the predicted values generated by the main econometric model (corresponding to Table 3, Column 4) for projects that experience technological competitor failure news at each project age. The dashed line is the back-of-the-envelope “counterfactual” prediction, which is calculated by removing the technological competitor learning effects from the predicted termination value.

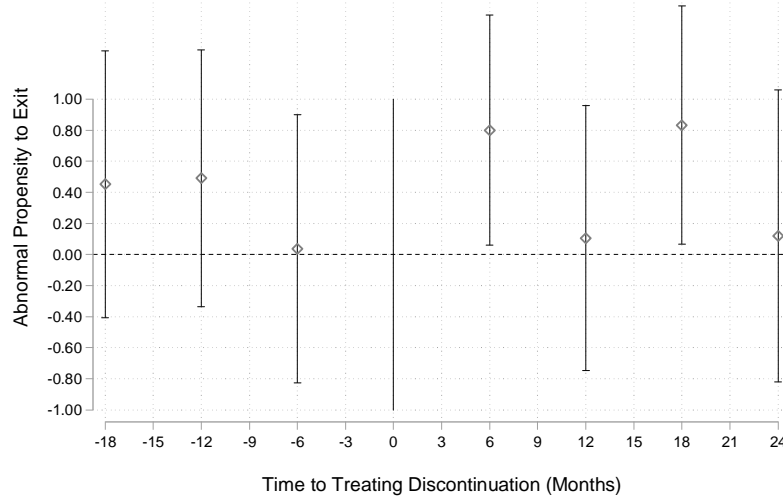
**Figure D3: Level of Competition and Competitor Failure Response, Bottom vs. Top Quartile**  
**A. Level of Market Competition (in Development)**      **B. Level of Market Competition (Approved Drugs)**



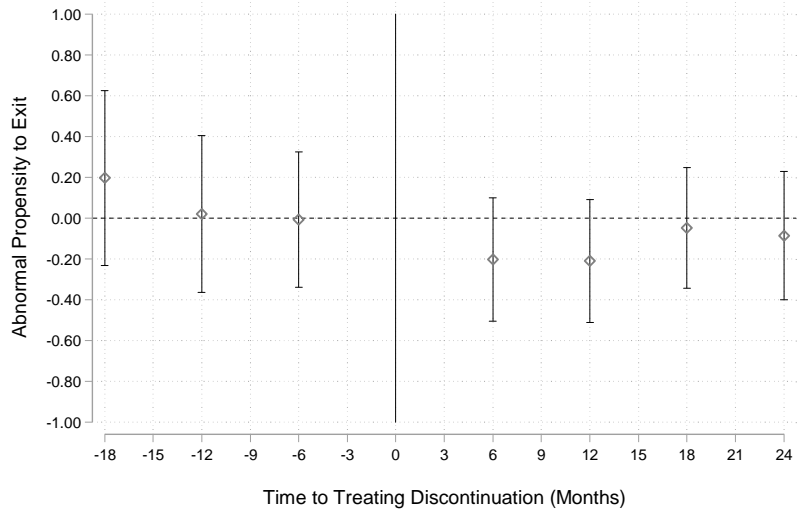
Note: The bars display the coefficients of interest from the main Cox proportional hazard model specification, split by bottom and top quartile of competition as defined in two different ways. In Panel A, the level of competition is defined as the number of development projects working on the same therapeutic indication. In Panel B, the competition split is based off the number of previously approved drugs within the same therapeutic indication. The capped spikes cover the 95% confidence intervals for each regression coefficient.

**Figure D4: Dynamics of Response to Competitor Failure, Low Competition Scenario**

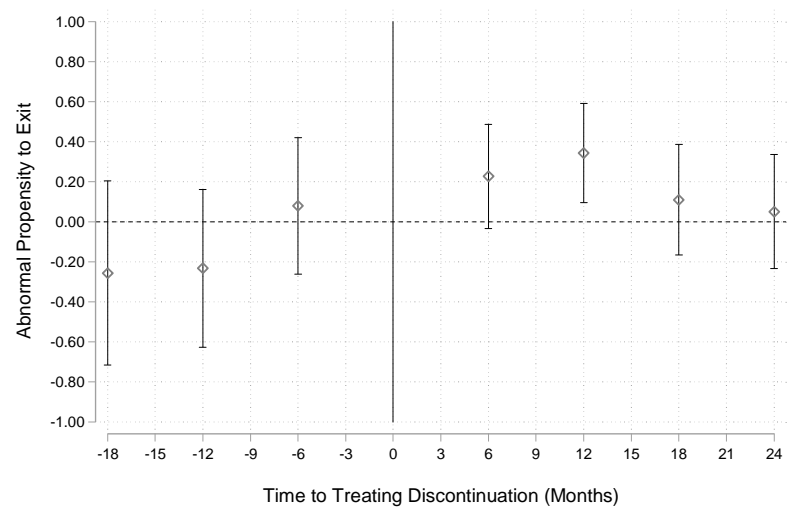
**A. Same Market, Same Technology**



**B. Same Market, Different Technology**



**C. Different Market, Same Technology**

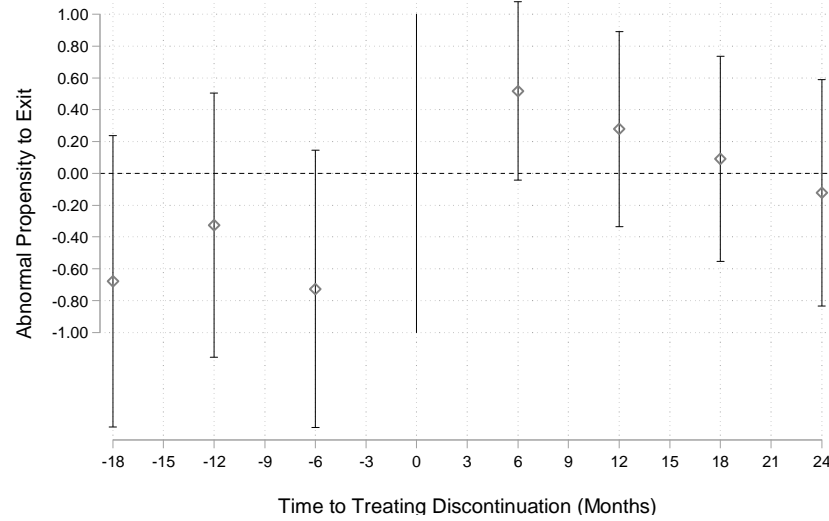


Note: The points in the above plots correspond to the event study coefficients stemming from the Cox proportional hazard model. The sample is limited to projects with low (below median) number of approved competitor drugs within the same therapeutic indication. The six months prior to the first competitor termination event is the omitted variable. The 95% confidence intervals (corresponding to robust standard errors) are plotted with capped spikes.

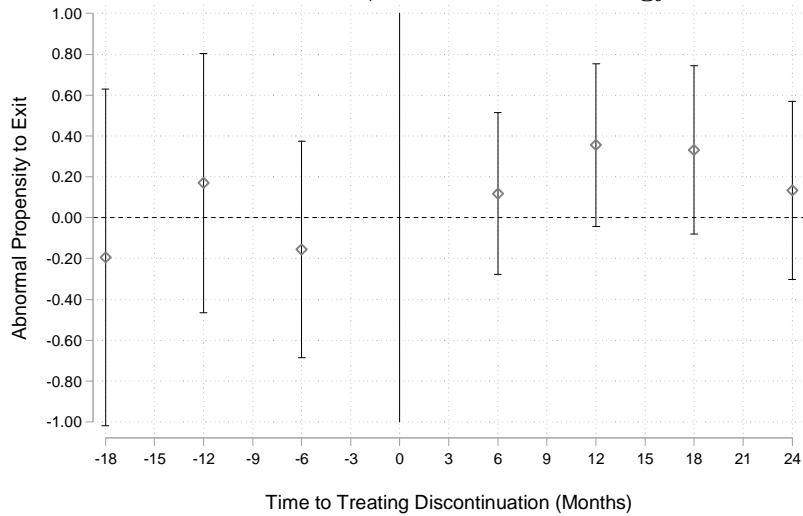


# Figure D5: Dynamics of Response to Competitor Failure, High Competition Scenario

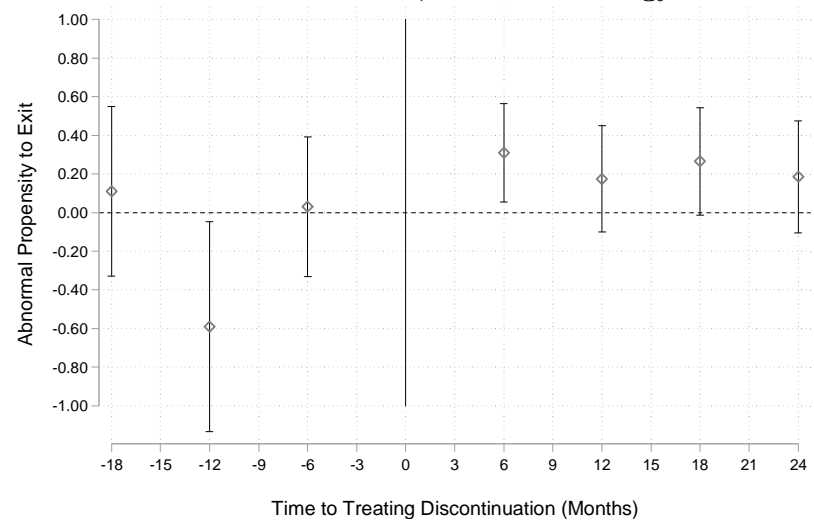
## A. Same Market, Same Technology



## B. Same Market, Different Technology



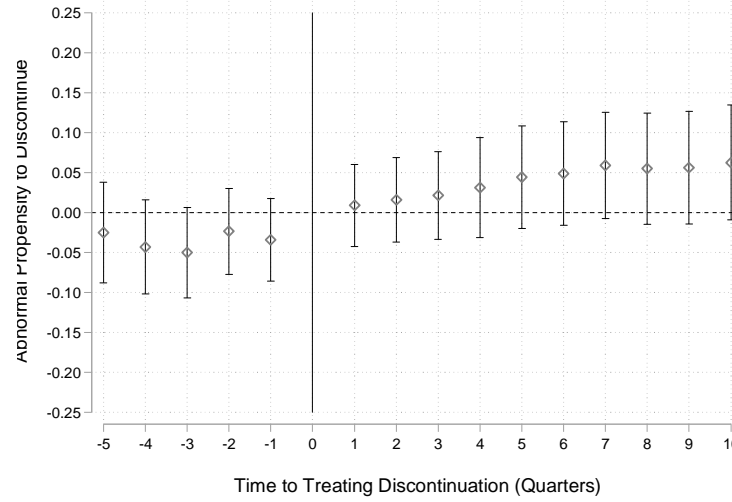
## C. Different Market, Same Technology



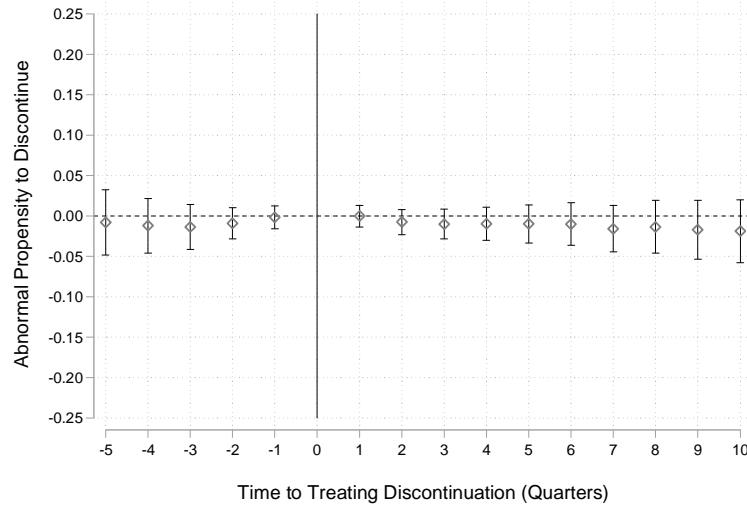
Note: The points in the above plots correspond to the event study coefficients stemming from the Cox proportional hazard model. The sample is limited to projects with high (above median) number of approved competitor drugs within the same therapeutic indication. The six months prior to the first competitor termination event is the omitted variable. The 95% confidence intervals (corresponding to robust standard errors) are plotted with capped spikes.

**Figure D6: Placebo Test, Phase III Projects and Phase II Failure News (Dyadic Data)**

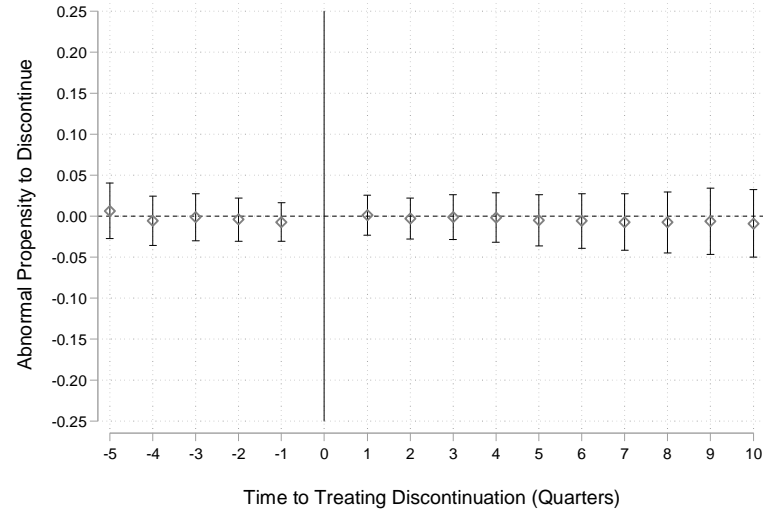
**A. Same Market, Same Technology**



**B. Same Market, Different Technology**

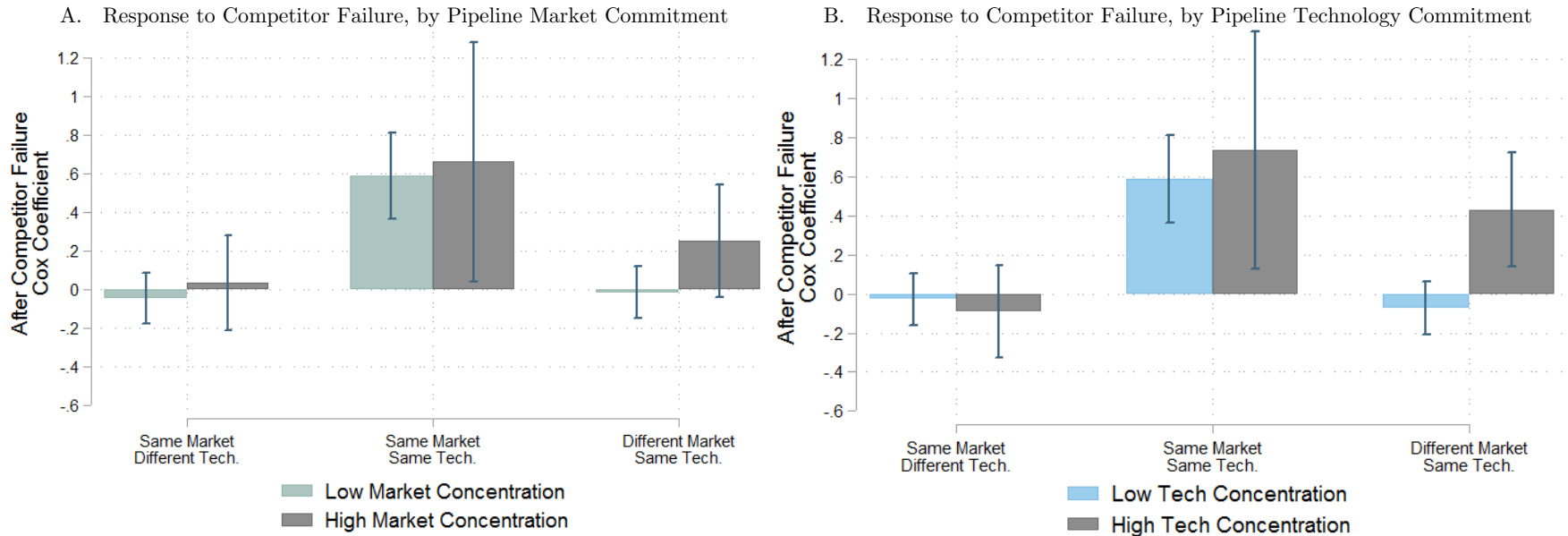


**C. Different Market, Same Technology**



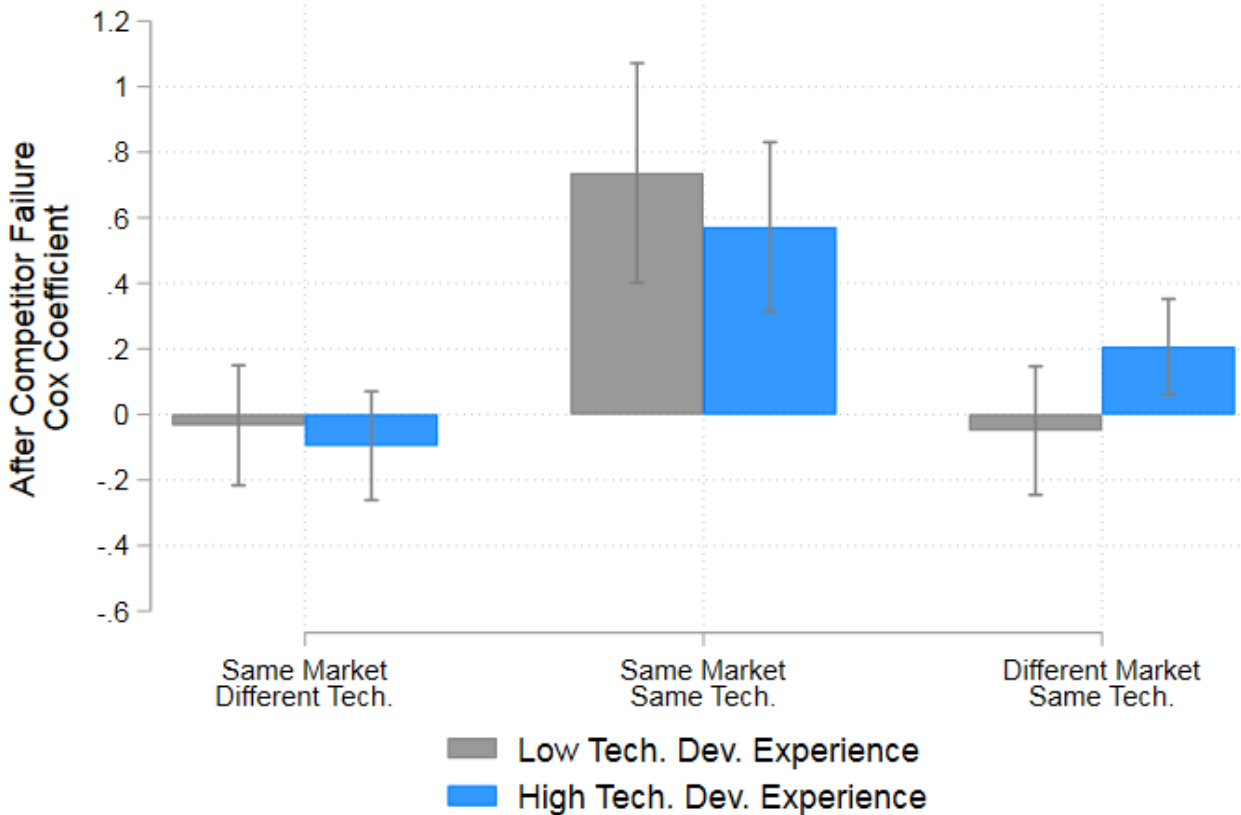
Note: The points in the above plots correspond to the event study coefficients stemming from the Cox proportional hazard model using the dyadic data panel setup (see Appendix B). Unlike the main analysis sample, the focal projects in this analysis are Phase III projects and are “treated” by Phase II competitor failures of each type. The sample includes 3,195 focal projects---2,920 (92%) experience SM-DT news, 272 (9%) experience SM-ST news, and 1,368 (43%) experience DM-ST news. The 95% confidence intervals (corresponding to robust standard errors) are plotted with capped spikes.

**Figure D7: Pipeline Concentration and Propensity to Learn from Competitor Failures**



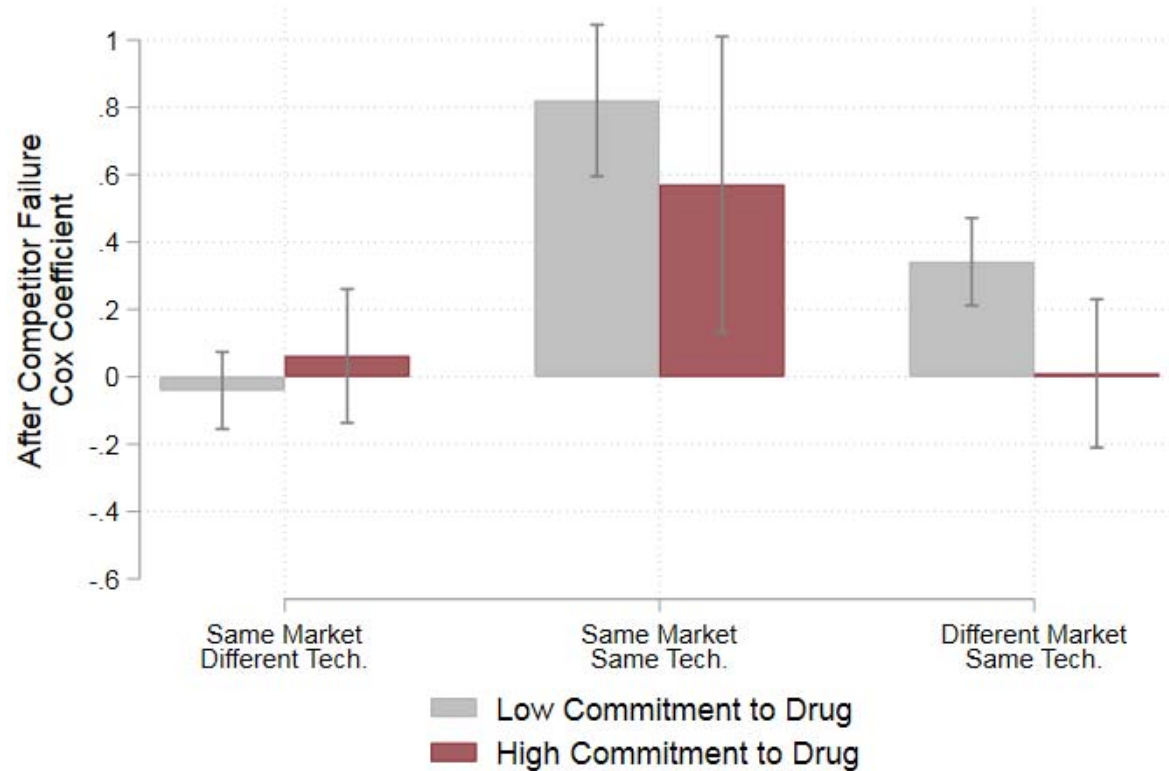
Note: The bars display the coefficients of interest from the main Cox proportional hazard model specification, split level of pipeline project concentration by therapeutic markets (Panel A) and technologies (Panel B). Concentration is measured by a Herfindahl index of a given firm’s projects across markets or technologies by company and time period (financial quarter). These analyses exclude firms with less than 10 projects in order to eliminate very small biotech firms that are unlikely to have a choice of pipeline market or technology diversity. The capped spikes cover the 95% confidence intervals for each regression coefficient. The difference in the coefficients’ magnitude is not statistically significant for the first two types of news in each panel, but the *different market, same technology* group coefficients are significantly different across the two groups in both panels ( $p < 0.05$ ).

Figure D8: Technological Experience and Response to Competitor Exit News



Note: The bars display the coefficients of interest from the main Cox proportional hazard model specification, split by the firm’s level of experience with the focal technology. The magnitude of each bar represents the change in hazard rate of project exit when a focal project is within a two quarter window after a competitor failure. To capture the amount of technological experience, the sample is split between those below and above the median level of experience (number of development projects) within the technology area. The capped spikes cover the 95% confidence intervals for each regression coefficient

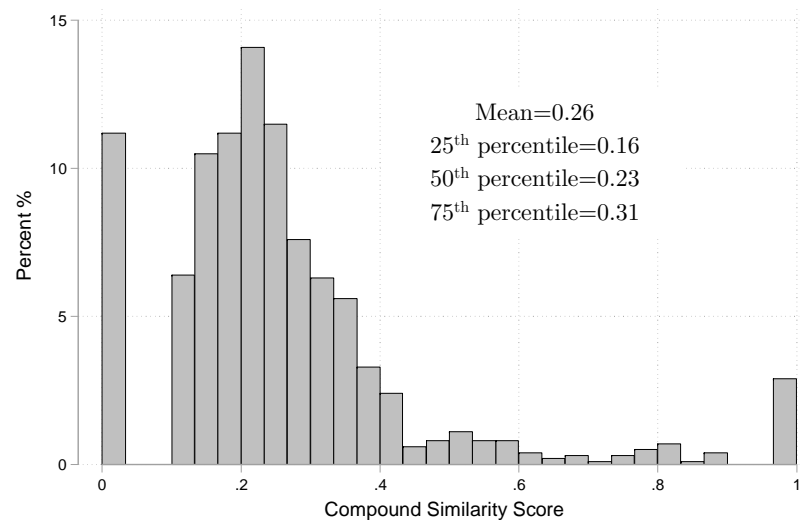
Figure D9: Additional R&D Commitments to Drug and Response to Competitor Exit News



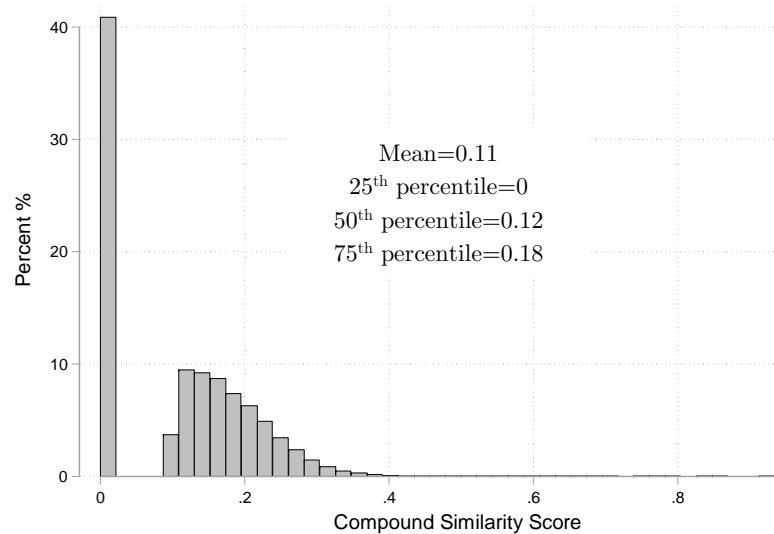
Note: The bars display the coefficients of interest from the main Cox proportional hazard model specification, with interactions for the firm’s level of additional R&D commitments to the focal drug. The magnitude of each bar represents the change in hazard rate of project exit when a focal project is within a two quarter window after a competitor failure. The sample is split between those below and above the median level of additional project commitments (number of active development projects) for a given drug. Low commitment is defined as having zero or one additional active development (for different therapeutic indications) projects for the focal drug, while high commitment is when the developers have more than one additional development project for the same focal drug. The regression controls for the underlying differences in project continuation rates, by stratifying the baseline hazard rate discontinuation by commitment level (in addition to therapeutic indications). The low and high commitment groups are not statistically different for the first two types of news, but are statistically different for the different market, same technology group ( $p < 0.05$ ). The capped spikes cover the 95% confidence intervals for each regression coefficient.

# Figure E1: Dyadic Molecular Similarity, Treating-Treated Drug Pairs

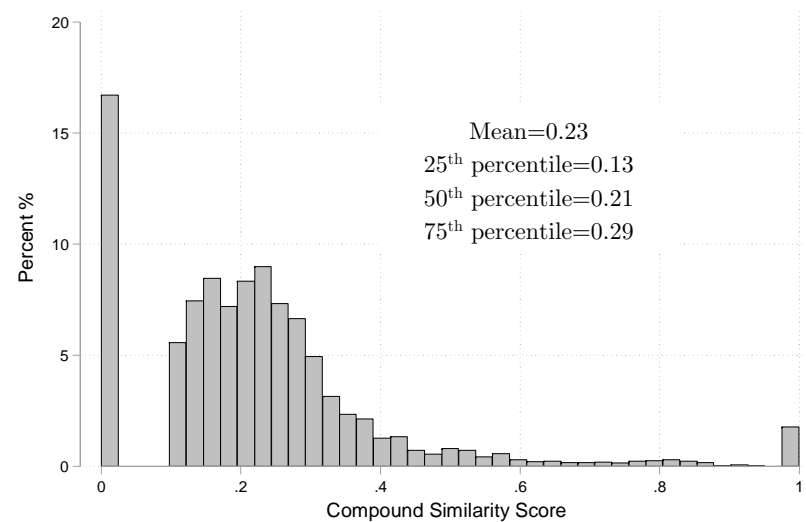
A. Same Market, Same Technology (N=1001)



B. Same Market, Different Technology (N=48,236)



C. Different Market, Same Technology (N=13,226)



**Table E1: Competitor Failure News Impact on Exit Rates, by Molecular Similarity**

“Treatment” Window: within 2 quarters since competitor failure news	(1) Project Exit (Dyadic Cox)	(2) Project Exit (Dyadic Cox)	(3) Project Exit (Dyadic Cox)
Competitor Discontinuation ( <i>Any</i> )	0.051 (0.036)		
Competitor Discontinuation ( <i>Any</i> ) × Similarity	1.370** (0.167)		
Competitor Discontinuation ( <i>Same Mkt, Diff. Tech.</i> )		-0.017 (0.043)	
Competitor Discontinuation ( <i>Same Mkt, Diff. Tech.</i> ) × Similarity		0.819** (0.290)	
Competitor Discontinuation ( <i>Same Mkt, Same Tech.</i> )		0.903** (0.209)	
Competitor Discontinuation ( <i>Same Mkt, Same Tech.</i> ) × Similarity		0.831 <sup>†</sup> (0.429)	
Competitor Discontinuation ( <i>Diff. Mkt, Same Tech.</i> )		0.829** (0.093)	0.828** (0.093)
Competitor Discontinuation ( <i>Diff. Mkt, Same Tech.</i> ) × Similarity		0.232 (0.284)	0.230 (0.284)
Competitor Discontinuation ( <i>Same Mkt</i> )			-0.040 (0.042)
Competitor Discontinuation ( <i>Same Mkt</i> ) × Similarity			1.207** (0.248)
Nb. Drug-Indications	5,015	5,015	5,015
Nb. of Drugs	2,819	2,819	2,819
Nb. of Treating-Treated Quarter Obs.	1,476,105	1,476,105	1,476,105
Log Likelihood	-115,357	-115,275	-115,292

Note: Estimates stem from Cox proportional hazard model specifications, where the outcome is own project discontinuation. To allow for pairwise chemical compound comparisons, all models are at the dyadic (treating-treated) level, and is limited to pairs of small molecule drugs (i.e., no biologic drugs). All models include a full set of year indicator variables and stratify the hazard specification by therapeutic indication. The competitor failure news events (treatment variables) take on a value of one when the focal project is within two financial quarters since the given type of competitor failure event. Additional independent variables interact these treatment states with the pairwise Tanimoto chemical similarity between the treating and treated drugs. These similarity values range from zero (no chemical structure overlap) to one (100% structure overlap, not accounting for stereochemistry). Coefficients may be interpreted as an increase in the log hazard rate. Standard errors in parentheses.

<sup>†</sup>  $p < 0.10$ , \*  $p < 0.05$ , \*\*  $p < 0.01$