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Excess Commitment in R&D

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We document that firms exhibit "excess" commitment to R&D projects and examine its consequences for innovation outcomes. Using detailed data on pharmaceutical firms' clinical trial projects, we find that trial delays, empirically uncorrelated with multiple project-quality measures, substantially reduce firms' subsequent project-termination propensity. This result remains robust when we use variation in clinical trial site congestion to instrument for unexpected delays. Excess commitment intensifies when CEO compensation has greater stock-price sensitivity and the CEO is responsible for the project's initiation. Our findings have broader implications: delay-driven commitment reduces new drug project initiations, with further evidence suggesting efficiency losses for firms. (*JEL* G31, O32, L65, D91, D22)

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Research and development (R&D) is a primary driver of innovation. A defining feature of R&D is its reliance on long-term commitment to a chosen course of action. Such persistence and path dependence can arise

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for various reasons, with prior work highlighting factors such as trapped resources that cannot be redeployed (Bloom et al. 2013); commercialization capital that locks firms into specific markets (Krieger, Li, and Thakor 2022); and R&D leverage, where early-stage spending necessitates later funding (Harrington 2012; Myers 2015). In fact, scientific breakthroughs often hinge on long-run R&D commitment, that is, on the sustained allocation of effort and resources to a particular strategy. For example, in new drug development—our focus—it typically takes 10 to 15 years of extensive research, development, and testing for a new drug to reach the market (Wong et al. 2014; Sertkaya et al. 2016).[1]

At the same time, seminal work from the lab by Staw (1976) finds that decision-makers' commitment to R&D projects tends to increase, and often escalate, as they allocate more resources to a project, even when faced with costly setbacks and the availability of superior alternatives. If corporate managers behave similarly, displaying what might be termed "excess commitment" (i.e., an excessive sensitivity of R&D decisions to prior investments, shaped by signaling incentives or psychological forces), the implications for firms and consumers can be substantial. For one, the high-effort, high-cost nature of R&D often implies constraints on the number of projects a firm can pursue simultaneously. Additionally, excess commitment can affect not only which drugs firms develop and progress, but may also induce firms to pursue inferior or even less safe innovation relative to a no-excess-commitment counterfactual.

In this paper, we study how firms' commitment to ongoing R&D projects varies with the amount of resources already expended and the extent to which R&D decisions reflect excess commitment characterized by excessive sensitivity to prior investments. Specifically, we study how firms' propensity to continue versus abandon existing R&D projects is affected by the occurrence of project delays that are unanticipated, plausibly exogenous, and costly. Our setting enables us to also examine spillover effects on other R&D investment decisions, particularly new drug project initiations.

In studying the extent to which firms' R&D decisions are (overly) influenced by prior temporal, and financial,[2] investments made, we face two key empirical challenges. First, the analysis requires granular,

[1] We note that our notion of commitment, encompassing the (potentially time-varying) inclination of firms to maintain a particular R&D strategy, is different from how the term is used in the contracting literature. In that context, commitment typically refers to the ex-ante promise and adherence to contracts within multi-period principal-agent frameworks (e.g., Rey and Salanie 1990). In line with our notion, R&D firms such as Merck and Pfizer frequently emphasize their "long-term commitment to research and development strategies," as well as their sustained focus on specific areas, such as a "long-term commitment to oncology" (Merck & Co. 2018; Pfizer Inc. 2016).

[2] As prior work documents, drug development delays are inherently linked to higher project costs, as longer trial timelines lead to increased labor expenses (e.g., extended compensation for investigators and staff) and greater patient monitoring and care costs (Wong et al. 2014; Sertkaya et al. 2016; Shadbolt et al. 2023).

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project-level data on R&D activities, including observable information on milestone achievements and final outcomes. Second, it requires not only reliable measures of firms' degree of commitment to R&D projects as these projects progress, but also a setting with plausibly exogenous variation in the intensity of initial project-specific investments made.

The focus on the economically and socially important setting of new drug development allows us to overcome these twin challenges related to data and identification. Specifically, we assemble a new data set of clinical trials initiated by U.S.-based companies, comprising more than 10,000 clinical trial projects launched between 1985 and 2019. The underlying data are drawn from multiple sources, including Cortellis Clinical Trial Intelligence and web-scraped and hand-matched records from ClinicalTrials.gov. Crucially for identification, our data include both anticipated timelines for each trial phase, filed on ClinicalTrials.gov, as well as the corresponding realized timelines. This allows us to isolate unanticipated delays incurred in already completed trial phases, that is, variation in unanticipated temporal investments made in the project. Moreover, the setting provides observable project decisions that directly capture firms' commitment, namely whether they advance or terminate a given trial at a given stage.

We preface our empirical analysis with a conceptual discussion that outlines the key predictions and implications of excess commitment to R&D, characterized by excessive sensitivity to project delays. We begin with a benchmark setting without frictions, in which delays are uninformative and should not influence firms' continuation decisions. However, the presence of incentive-based or psychological frictions may lead managers to persist with delayed projects—either to avoid reputational costs or because they fail to treat delays as sunk. These frictions give rise to our main testable prediction: the likelihood of project continuation increases with the duration of experienced delays. Moreover, this notion of excess commitment carries direct implications for efficiency. As we discuss in more detail in Section 1, delay-driven continuation implies suboptimal firm investment when lower-value, delayed projects displace higher-value alternatives. Our discussion also clarifies that excess commitment is distinct from overinvestment in the sense of undertaking too many projects in aggregate.

Guided by these conceptual insights, we organize our empirical analysis into four main parts. In the first part, we establish a new baseline fact: a strong positive relationship between unanticipated delays in completing a clinical trial phase and firms' subsequent commitment to the project, measured by their propensity to continue the project through the next trial phase rather than terminate it. Using the information on anticipated trial end dates, we document that the average trial phase in our sample is completed with a delay of nearly 1 year, with the 25th (75th) percentile

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completed with no delay (a delay of 1.6 years). A one standard deviation increase in completion delay is associated with a 4 percentage point reduction in the probability of project termination, which represents a 15% decline relative to the baseline suspension rate of 31%.

In the second part of the analysis, we relate this baseline finding to our notion of excess commitment. A key question is whether unanticipated project delays might produce information that increases a project's expected value and thus should lead firms to increase their commitment and likelihood of continuation. We assess this possibility through three sets of empirical tests: incorporating additional data, implementing an instrumental variables (IV) approach, and examining an extended sample.

First, we find that experienced delays are empirically uncorrelated with a range of independent project quality measures obtained from GlobalData, the extent of drug dosage experimentation, and expected drug sales. These patterns are consistent with delays being "as good as randomly assigned."

Second, we instrument for unanticipated trial phase completion delays using a measure of clinical trial site congestion. The IV strategy exploits the fact that trial sites have limited capacity, with increases in the number of concurrent trials at a given location raising the likelihood of delays due to logistical bottlenecks. To account for the fact that firms do not randomly select trial sites, we construct the congestion measure to capture changes in site busyness relative to trial start. We find a strong positive relationship between increased trial site congestion and trial delays, indicating a strong first stage. In the second stage, congestion-instrumented delays continue to significantly predict project continuation, reinforcing the excess commitment interpretation. The identifying assumption is that changes in trial site congestion affect firms' drug continuation decisions only through their effect on trial delays. We argue that this exclusion restriction is plausible, as the congestion measure is empirically uncorrelated with a broad set of potential confounders—including the same drug quality and experimentation measures as above, as well as detailed trial site quality measures assessing hospital care quality in a given region. We conduct a series of further tests regarding the IV analysis, including an alternative instrument that isolates trial site congestion solely from drugs in unrelated disease fields. Our IV results remain robust.

Third, we assess the possibility that our sample construction introduces selection, as firms may endogenously respond to unfolding delays, thus potentially affecting which projects complete a trial phase and enter our sample of completed trials with achieved endpoints.[3] This concern gives rise to two testable predictions, which we assess using an expanded data set

[3] As we elaborate below, restricting the sample to completed trials with achieved endpoints is necessary to ensure that observed suspension decisions are not mechanically driven by unfavorable outcomes in the preceding phase.

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that includes withdrawn projects, that is, those that did not achieve their endpoints. Delay distributions are statistically indistinguishable between withdrawn and completed projects, and observable drug quality measures do not predict withdrawal, providing evidence against endogenous selection into trial phase completion.

In the third part of the analysis, we provide evidence on underlying mechanisms, with the results further substantiating the excess commitment interpretation. Our findings are not driven by firms lacking alternative drug projects to invest in (Guedj and Scharfstein 2004) nor by financial constraints that prevent them from pivoting. Moreover, the results are difficult to reconcile with reporting incentives or firms' (potentially misspecified) beliefs about anticipated trial completion dates, given our IV design and the fact that the results remain unchanged when directly controlling for firms' reported anticipated trial duration.

Instead, the effect of unexpected trial delays on subsequent project continuation is amplified in the presence of management-related frictions and when the chief executive officer's (CEO's) personal stakes in the project are higher. Specifically, delays are substantially more predictive of continuation among CEOs with greater wealth exposure to the firm's stock price, consistent with such CEOs being particularly concerned that suspending a delayed project may be interpreted as a negative signal by investors. In addition, delays predict continuation nearly twice as strongly when the project-initiating CEO remains in office at the completion of the (possibly delayed) trial phase, underscoring the role of personal responsibility.

In the final part of the analysis, we turn to the implications of our findings for firm investment. While unanticipated trial delays increase firms' subsequent commitment to the affected project, we find that they crowd out the initiation of alternative projects, especially in other therapeutic areas. Additional analyses point to efficiency costs for firms. Continued delayed projects tend to have lower projected sales than comparable nondelayed projects. Moreover, foregone projects, even though unobserved, appear likely to have created value on average, as indicated by investors' positive reaction to announcements of plausibly similar projects. We conclude with a brief discussion of potential consumer externalities, as firms' investment behavior in this setting has unusually direct consequences for consumers, most notably through the availability and quality of new therapies—an avenue we highlight for future research.

Broadly, our findings contribute to a large literature and ongoing debate about the adequacy of innovation activity in the economy. For example, seminal prior work has identified a range of reasons for potential aggregate underinvestment in innovation, including limited spillovers, weak competition, patent protection, infringement lawsuits, and risk aversion (Hall and Lerner 2010). Other work highlights potential overinvestment due

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to business-stealing and technological obsolescence (Aghion and Howitt 1992). Our contribution is distinct in that we focus on firms' commitment to existing, in-process R&D endeavors, rather than on the adequacy of R&D adoption at the extensive margin.

Our paper makes three main contributions to the literature. First, we contribute to the literature on the economics of innovation,[4] particularly on the notion of commitment in R&D investment. A growing body of work highlights why firms persist in R&D despite changing circumstances. Beyond trapped resources, commercialization capital, and R&D leverage (Bloom et al. 2013; Krieger, Li, and Thakor 2022; Harrington 2012; Myers 2015), Manso (2011) shows that long-term tolerance for failure and commitment to a long-term incentive scheme can optimally incentivize innovation, and Li, Liu, and Taylor (2023) demonstrate that institutional ownership, cost pressures, and product market competition can reinforce sustained R&D activity. We extend this literature by documenting a novel form of (excess) commitment in R&D: firms are more likely to continue a project when it experiences longer delays in already completed trials. We show that this behavior arises, in part, as an organizational response to management-related frictions. We also examine broader implications, highlighting the opportunity costs and investment crowding out associated with continuing delayed projects.

Second, we contribute to the corporate finance literature on the effects of top management on firm investment. Guenzel (2025) documents distortions in firm investment due to managerial sunk cost effects—one form of path-dependent decision-making—within the context of M&A, where plausibly exogenous increases in acquisition costs make acquired businesses less likely to be abandoned through divestiture.[5] In relation to aggregate efficiency and welfare, Barrero (2022) and Ma et al. (2024) analyze the macroeconomic consequences of managerial belief distortions for firms' resource-allocation decisions, including hiring and capital investment. In contrast, we focus on R&D, a setting in which project-level decisions carry distinctive implications for product markets. We extend the scope of management-related frictions by showing that

[4] Moser (2016) and Bryan and Williams (2021) provide comprehensive surveys of recent research on market failures in innovation and intellectual property. Related to pharmaceutical innovations, Acemoglu and Linn (2004), Budish, Roin, and Williams (2015), and Azoulay et al. (2019) investigate market size, short-termism, and funding structures as drivers of R&D dynamics. In biomedical R&D, Lo and Thakor (2022) and Lo and Thakor (2023) review work on financing frictions. Related studies include Lerner, Shane, and Tsai (2003), Robinson and Stuart (2007), and Lerner and Malmendier (2010) on funding models; Cunningham, Ederer, and Ma (2021), Aghamolla and Thakor (2022a), and Liu (2021) on M&A and IPO decisions; Cohen, Gurun, and Li (2021) on internal deadlines and innovation quality; and Higgins and Rodriguez (2006), Krieger, Li, and Papanikolaou (2022), Thakor and Lo (2022), and Bonaimé and Wang (2024) on the role of outsourcing, competition, and risk in shaping R&D investment.

[5] Using standard firm-level R&D data from Compustat, Guenzel (2025) also presents suggestive correlational evidence consistent with a sunk cost mechanism in R&D investment, indicating potential excess investment following prior R&D spending.

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project delays generate excess commitment and crowd out alternative investments, thereby contributing to the literature on nonstandard firm behavior (see Malmendier [2018] and Guenzel and Malmendier [2020] for recent literature surveys).

Third, we contribute to the literature on commitment and escalation of commitment. Following Staw's (1976) lab evidence of escalation in R&D decision-making, subsequent work has documented similar patterns in field settings such as loan write-offs and NBA draft picks (Staw, Barsade, and Koput 1997; Staw and Hoang 1995; Camerer and Weber 1999). Much of this literature, however, lacks exogenous variation in initial investment, leaving room for confounding explanations related to ex ante information, beliefs, or selection. We advance it by examining how unanticipated delays influence within-firm R&D commitment and by tracing their broader effects on investment, including crowding out.[6]

1. Hypothesis Development

To guide the empirical analysis, we first discuss the main hypothesis, the notion of excess commitment, as well as the associated counterfactuals and efficiency implications.

Hypothesis Our setting can be summarized by a manager undertaking a multi-stage project with a positive net present value (NPV) at the outset. After completing the first project phase—potentially with delays—the manager must decide whether to continue or abandon the project. In the most basic benchmark case, there are no frictions, and delays in completing the first phase are uninformative about either the project's NPV or the manager's quality. Under these conditions, the NPV-optimal behavior dictates that the manager will continue the project if and only if the NPV is positive at the time of the continuation decision, irrespective of any delays incurred. Terminating the project may become optimal if the first phase provides negative signals about the project's prospects or if superior opportunities arise, either of which could render the project's NPV negative. Consequently, in the benchmark case, delays do not marginally affect the likelihood of project continuation.

Alternatively, the presence of frictions may induce the manager to factor incurred delays into their decision-making. One possibility is incentive-based frictions, where the manager's compensation is partially linked to the market's perception of their ability, and abandoning a delayed project may adversely affect this perception. A second possibility

[6] See Augenblick (2016) and Ho, Png, and Reza (2018) on sunk cost effects using quasi-random variation among consumers, and Guenzel (2025) on quasi-random sunk M&A costs. Our focus differs from studies on interfirm path dependence (e.g., Manez et al. 2009) by emphasizing within-firm dynamics.

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involves psychological frictions, where the manager fails to recognize delays as sunk costs (in terms of time and money already spent) and becomes attached to projects with delays, with the degree of attachment increasing in the extent of delay. In both cases, delays may induce the manager to persist with the project. While quantifying the precise relative importance of different microfoundations is not the focus of this paper (and it is plausible that multiple mechanisms operate simultaneously), we provide some tests on this in Section 6.2. Most importantly, both of the above mechanisms converge on the same prediction: delays increase the likelihood of project continuation, with longer delays leading to higher continuation probabilities.

Our notion of excess commitment derives from comparing this predicted positive slope to the base-case prediction of a flat relationship. In essence, it captures an excess sensitivity to delays, where the manager becomes increasingly committed to a multi-stage project as delays accumulate.

Efficiency Implications Excess commitment, in the sense of excess sensitivity to delays, entails efficiency implications for firms. We discuss the various predictions conceptually in turn and indicate how we assess them empirically whenever feasible given our data.

Delay-induced commitment leads to the continuation of projects that, under counterfactual conditions would have been abandoned. Here, counterfactual conditions refer to the absence of commitment-inducing frictions, which we empirically capture through the absence of delays. Two interrelated aspects shape the precise nature of the efficiency implications: (i) whether the firm is constrained (e.g., operationally or financially) and (ii) whether, in the counterfactual scenario without excess sensitivity to project delays, it invests optimally (as in the most basic benchmark case described above) or suboptimally in R&D, conditional on any potential constraints the firm faces.[7]

If the firm is unconstrained, delay-induced project commitment and continuation leads to efficiency losses whenever, in the counterfactual scenario, the firm invests optimally or overinvests, that is, the marginal project that is undertaken has a zero or negative NPV. In these cases, delay-driven commitment induces the manager and firm to continue projects with an even lower, and strictly negative, NPV. Alternatively, if the firm is constrained, delay-induced commitment leads the manager to continue delayed projects with a relatively lower NPV instead of nondelayed or less delayed projects with a relatively higher NPV, resulting in efficiency costs through suboptimal project prioritization. Consequently, the only scenario

[7] The counterfactual scenario that real-world firms face in the absence of excess sensitivity to project delays may involve other frictions that lead to suboptimal investment, such as risk aversion (resulting in underinvestment) or empire-building motives (resulting in overinvestment).

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where delay-induced project commitment may not involve efficiency costs (but instead gains) for firms arises when they would otherwise underinvest in R&D, that is, undertake too few projects, and delay-induced commitment does not crowd out other projects.

This discussion highlights that our notion of excess commitment, reflecting an excessive sensitivity to prior outcomes, is potentially often, but not always, synonymous with excess investment by firms, with the extent to which this is the case ultimately an empirical question. Our analyses in Section 7 examine the efficiency implications for firms by comparing the projected quality (cash flows) of delayed versus nondelayed continued projects and by testing for crowding-out effects. We also include tests that provide suggestive evidence on the foregone incremental value of crowded-out projects, while noting the empirical challenge that arises because such projects are inherently unobserved, as they are never initiated.

2. Institutional Background and Data

2.1 Institutional background

The pharmaceutical industry is highly R&D intensive. For example, in 2019 alone, the industry spent $83 billion dollars on R&D, with R&D intensity (defined by R&D spending as a share of net revenues) reaching 25%, surpassing that of the software and semiconductor industries (Austin and Hayford 2021). A substantial share of pharmaceutical R&D spending is devoted to clinical trials, which constitute a major component of the total cost of bringing a new drug to market, frequently exceeding $1 billion (DiMasi, Hansen, and Grabowski 2003; DiMasi, Grabowski, and Hansen 2016; Wouters, McKee, and Luyten 2020).

Clinical trials, regulated by the FDA, proceed in three stages: Phase I, II, and III. Phase I trials assess the safety of a drug candidate in a small group of humans and examine how the drug is absorbed, distributed, and metabolized in the body. Phase II trials involve a larger patient pool and aim to identify optimal drug dosage and preliminary effectiveness. Phase III trials evaluate safety and efficacy across diverse populations, typically involving large-scale enrollment. All trial phases require adherence to a strict protocol—a scientific plan detailing patient recruitment, inclusion and exclusion criteria for eligible patients, testing procedures, data collection methods, defined endpoints (outcomes of interest), and criteria for trial discontinuation. Successful completion of each phase is required for progression to the next phase. Upon successful completion of all three phases, drug developers may submit a New Drug Application to the FDA for market approval.

Clinical trials across all phases are frequently conducted at multiple sites and typically require several years to complete. As we discuss below in more detail, in our sample, comprising Phase I and II trials, the median

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Table 1
Summary statistics

	N	Mean	SD	P25	Median	P75
Suspension	11,228	0.31	0.46	0.00	0.00	1.00
Trial Duration (in months)	11,228	35.65	29.45	12.00	29.00	51.00
Anticipated Trial Duration	9,948	22.83	17.79	9.00	20.00	32.00
Delay in Trial Completion	9,948	11.78	19.19	0.00	3.00	19.00
Number of Clinical Trial Sites	11,179	13.65	40.62	1.00	3.00	13.00
Trial Participants (in hundreds)	11,195	1.00	1.62	0.25	0.49	1.06
Clinical Trial Phase (I or II)	11,228	1.59	0.49	1.00	2.00	2.00
Number of Drug Projects	10,998	136.14	179.07	11.00	41.00	230.00
Number of Competing Drug Projects	11,182	402.07	812.80	81.00	206.00	406.00
Change in Trial Site Congestion	9,948	5.30	10.13	0.00	0.91	4.18
Drug Likelihood of Approval (LOA %)	3,046	11.24	6.19	7.20	10.95	14.38
Drug Phase Transition Success Rate (PTSR %)	3,046	45.63	14.36	36.60	42.35	54.89

This table reports summary statistics at the drug-project level. The main sample contains all projects that completed Phase I or II trials with detailed clinical trials records.

trial phase duration is 2.4 years (Table 1; Section 2.3). Wong, Siah, and Lo (2019) report comparable figures, with median durations of 1.6, 2.9, and 3.8 years for Phase I, II, and III trials, respectively.

Reflecting the time-intensive nature and complexity of clinical trials, their costs are substantial. Key cost drivers include clinical procedures, administrative staff, site monitoring, site retention, and central laboratory services. Total trial costs are typically categorized into per-patient and per-site components. Per-patient costs include expenses related to patient retention, compensation for medical personnel such as nurses and physicians, laboratory services, and clinical procedures. Per-site costs generally comprise site recruitment and retention, administrative staffing, and site monitoring (Wong et al. 2014; Sertkaya et al. 2016).

Notably, these cost components are highly sensitive to trial duration, with longer timelines driving up drug development costs through both per-patient and per-site expenses (Wong et al. 2014; Sertkaya et al. 2016). For example, extended trials raise labor costs, as investigators and staff must be compensated over longer periods. They also require additional time and resources to care for trial participants, further contributing to overall expenditures for drug developers.

2.2 Data and sample construction

Clinical Trials Data We focus on clinical trials initiated by U.S.-based companies. The trials data come from Cortellis Clinical Trial Intelligence and allow us to observe detailed trial information, including the trial title, trial phase, start and completion dates, number of sites and site countries, trial protocol, eligibility criteria, interventions, adverse outcomes (if applicable), and whether endpoints are achieved, along with information on the drug developer ID and name, its role in the trial (e.g., sponsor), and details on drug candidates, drug indications,[8] drug dosages tested during

[8] In all analyses, we use the most granular, three-level categories of the International Classification of Diseases (ICD) codes. We sometimes refer to them simply as ICD categories or ICD codes for conciseness.

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the trial, and associated technology. All our analyses are implemented at the trial-phase-by-drug-indication level, that is, a drug project in our data is defined as a drug-by-indication combination.

To identify the outcome of trials and the corresponding drug projects, we merge the trials data with drug development data from Cortellis Competitive Intelligence. We classify a project as suspended at a given trial phase if it is explicitly coded as suspended, discontinued, withdrawn, or coded as "no development" for over 3 years. We also consider a project suspended if it remains in Phase I, II, or III for over 5, 8, or 10 years, respectively, without further progression (Li, Liu, and Taylor 2023).

Data Filters We apply several filters to construct our main analysis sample. First, we focus on completed Phase I or II trials and nonmissing records on outcomes after these trials, that is, suspension or continuation. We exclude Phase III trials, as our focus is on whether firms suspend or advance trials through the subsequent phase, necessitating the possibility of a follow-up phase. Second, we drop any trials completed after 2020, as these projects may not have had sufficient time to generate observable outcomes. Third, we exclude Phase I and II trials that did not achieve their endpoints to ensure any observed suspension is not solely attributable to unfavorable readouts in the preceding trial phase. Finally, we retain only projects with both realized trial start and completion dates, excluding those with estimated completion dates. These filters yield a sample of 11,228 projects initiated between 1985 and 2019 (with trial completions between 1991 and 2020), with trial sites spanning across 91 countries, including the United States.

Match with ClinicalTrials.gov We manually match our sample with additional trial data scraped from ClinicalTrials.gov, maintained by the U.S. National Library of Medicine. Drug developing firms are required to submit both initial and follow-up reports for each clinical trial to ClinicalTrials.gov. Crucially for our purposes, firms must report the anticipated trial completion date in their initial submission. This information allows us to construct the key variable in our analysis—the trial completion delay—measured as the difference between the anticipated trial completion date in the initial submission to ClinicalTrials.gov and the actual trial completion date.[9] By matching the data set with the ClinicalTrials.gov reports, we also obtain trial-specific information on clinical holds imposed by the FDA, as well as detailed site-level location data for each trial, including street addresses, cities, states, countries, and zip codes. Internet Appendix Section A.1

[9] Throughout the paper, we use the terms "trial phase completion delay," "trial completion delay," and "trial delay" interchangeably.

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provides further details on the matching procedure of the data set with the ClinicalTrials.gov reports. Out of the 11,228 projects, we successfully match 9,448 to the ClinicalTrials.gov reports.

Drug Quality Data We collect several proxies for drug quality from the GlobalData Pharma database, including the drug-specific likelihood of approval (LOA) and the drug's phase transition success rate (PTSR). Both are forward-looking indicators intended to capture a drug project's underlying quality and prospects. The LOA reflects the probability that a pipeline drug will successfully progress through clinical stages and obtain regulatory approval, conditional on its current status and stage. GlobalData estimates LOA using proprietary machine learning algorithms that incorporate over 30 attributes related to the company, drug, and regulatory events. Similarly, PTSR estimates the probability that a drug will successfully advance from its current phase to the next. GlobalData derives this measure using similar machine learning models. We also collect data on expected drug revenues at market launch from Cortellis Competitive Intelligence.

Other Data We merge in various supplementary data. We collect data on U.S. hospital quality from various CMS-managed quality programs, including 30-day mortality rates, 30-day readmission rates, and patient safety indicators, from various CMS-managed quality programs. We also obtain financial accounting and management data for a subset of the drug developers that are public companies traded on U.S. exchanges from Compustat, BoardEx, and Execucomp.

2.3 Summary statistics

Table 1 presents summary statistics for our main sample comprising completed Phase I and II clinical trial projects. Approximately one in three projects is suspended, that is, not advanced through the subsequent phase (Phase II or III, respectively). The average trial takes about 3 years to complete. (We discuss additional variables related to trial duration in Sections 3.1 and 3.2 when introducing the empirical design.)

The average trial in our data is conducted across 14 sites, with considerable variation in site count, and involves 100 participants. Just under 60% of trials are Phase II trials, with the remainder being Phase I trials. The average firm in our sample has 136 drug projects in development, indicating that these are *not* single-product firms, in contrast to the firms studied in Guedj and Scharfstein (2004). Each project faces, on average, competition from approximately 400 other projects within the same ICD code. The average likelihood of approval and phase transition success rate in our sample are 11.24% and 45.63%, respectively. This aligns with

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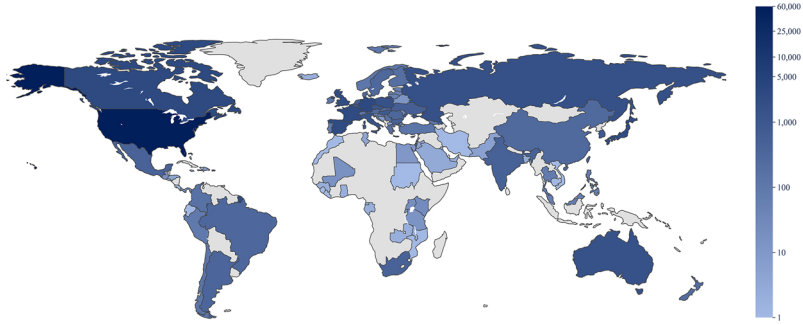

Figure 1
Geographic distribution of trial sites
This figure shows the geographic distribution of clinical trial sites in the sample, plotting the number of sites by country.

our sample consisting primarily of drugs in the earlier stages of drug development.

Figure 1 shows the geographic distribution of clinical trial sites in our sample. A substantial share of clinical trials is conducted in the United States, which accounts for 63% of all trial sites. At the same time, trial activity is distributed across many countries. Outside the United States, the largest contributors are Canada, Germany, France, and Japan, which together account for an additional 11% of trial sites. These patterns remain consistent when tabulating the number of trials, rather than trial sites, across countries (Figure IA.1 of the Internet Appendix). In terms of trial counts, the United States accounts for 45% of observations. Canada, Germany, France, and Japan together account for 14%.

3. Empirical Approach

Our empirical approach focuses on pharmaceutical firms' decision to continue or suspend drug development projects. Specifically, we test whether plausibly exogenous delays in completing the preceding clinical trial phase make firms more likely to advance the project through the next trial phase. Figure 2 illustrates the key elements of the project timeline and empirical design.

3.1 Delays in trial phase completion and project continuation versus suspension

To examine how firms factor unexpected delays in trial phase completion into their subsequent R&D decision-making, and, specifically, whether to continue the project through the next trial phase, we first estimate:

$$Suspension_i = \beta DelayInTrialCompletion_i + \gamma Controls_i + \text{FEs} + \eta_i, \quad (1)$$

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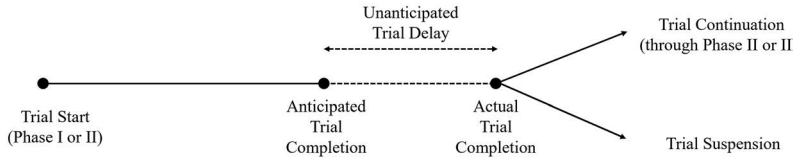

Figure 2
Project timeline
This figure illustrates the project and decision timeline, which motivates our empirical test of the relationship between trial continuation decisions and unanticipated delays in completing the preceding phase.

using the sample of all projects *i* that have completed Phase I or II clinical trials with detailed trials records. $Suspension_i$ is an indicator variable equal to one if a project that completes a Phase I (Phase II) trial is subsequently suspended and does not advance through Phase II (Phase III). The independent variable of interest, $DelayInTrialCompletion_i$, is the difference (in months) between the actual and the anticipated completion date of the trial phase, with the anticipated date as indicated by the firm at the start of the trial phase. As control variables, we include the number of clinical trial sites (in logs), the number of enrolled participants (in hundreds), the number of drug projects in the company's pipeline (in logs), and the number of competing projects in the same ICD category (in logs). We also include a rich set of fixed effects to account for unobserved heterogeneity. These include fixed effects for the drug company, the drug's ICD category, and the preceding trial phase (Phase I or II), as well as year-by-quarter fixed effects based on the trial phase start date.[10] Our estimation therefore isolates differences in the probability of suspension versus continuation among projects initiated at the same time (within the same year-quarter) but experiencing different levels of delay. Standard errors are two-way clustered at the ICD category and year-by-quarter levels.

Discussion of the Delay Measure Inspecting the key variable of interest, the delay in completing the preceding trial phase, we find that, on average, trials are completed nearly 1 year later than initially expected (Table 1). There is also substantial heterogeneity in delays, which is useful from an estimation and identification perspective. The 25th-percentile trial phase is completed without delay, whereas the 75th-percentile trial phase experiences a delay of approximately 1.5 years.

Two additional aspects are worth highlighting in terms of the measurement of the delay variable and its interpretation in relation to

[10] Table IA.2 in the Internet Appendix examines how the included control variables and fixed effects relate to a trial's anticipated duration, that is, the firm's expectation regarding trial duration at trial start. The analysis points to the disease field, the trial initiation year, and firm identity as key determinants of expected trial duration.

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the key prediction concerning project continuation. First, in measuring trial completion delays as the difference between the actual and the anticipated trial end dates, it is important to emphasize that firms face clear incentives and institutional guidance to report anticipated trial end dates truthfully. For one, firms are required to update anticipated completion dates on ClinicalTrials.gov whenever changes occur, which imposes additional compliance costs if firms were to misreport expected trial end dates at the outset. In addition, ClinicalTrials.gov reporting guidelines explicitly state that "it is important these dates are accurate."[11] These reporting requirements reduce the possibility of measurement error—which would plausibly bias the estimates toward zero—or distortions in the delay variable. Moreover, our IV strategy, discussed in Section 3.2, further mitigates concerns about potential strategic reporting, as long as any possibly remaining reporting incentives are uncorrelated with the instrument.

Second, building on the discussion in Section 2.1, trial delays do not only extend the temporal investment in a project but also increase its associated costs. As prior research highlights, prolonged trial timelines lead to higher labor expenses (since investigators and staff must be compensated over a longer period), as well as increased costs related to patient monitoring and care (Wong et al. 2014; Sertkaya et al. 2016; Shadbolt et al. 2023). These elevated project costs can reinforce the key prediction that, as project delays increase, firms become increasingly inclined to continue rather than abandon the project.

3.2 Increase in trial site congestion as an instrument for trial phase completion delays

As discussed in the previous section, the *DelayInTrialCompletion* measure captures the unanticipated component of a clinical trial's duration as the difference between the realized and anticipated trial end dates. To further isolate the effects of plausibly exogenous variation in trial length on firms' continuation-versus-suspension decisions, we employ an IV strategy that uses trial site congestion as an instrument for trial completion delay. More specifically, we focus on changes in trial site congestion over the course of the trial. By focusing on changes in congestion since the trial start, we account for the fact that trial sites, and thus initial levels of site busyness, are not randomly selected by firms.

Intuitively, the instrument builds on the idea that trial sites have limited capacity to accommodate clinical trials. When a site becomes overburdened, it becomes more difficult to, for example, recruit patients on time, maintain adequate staffing to monitor multiple trials simultaneously,

[11] See, for example, https://www.dfhcc.harvard.edu/crs-resources/ODQ_Documents/02_CT.GOV_CTRP/DFHCC_ClinicalTrialsGov_Results_Reporting_Training.pdf.

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and ensure timely access to specialized medical devices, diagnostic tools, laboratory equipment, or other technology required for data collection and analysis. These constraints increase the likelihood of operational bottlenecks and delays in trial completion. Importantly, the instrument captures variation that is largely outside the focal firm's control, as changes in congestion depend on decisions made by external parties—such as other sponsors independently initiating trials at the same sites—rather than the firm's own actions.

Construction of the Congestion Measure To construct our instrument, we download the universe of trial records across all phases from ClinicalTrials.gov—that is, all available Phase I, II, and III trials, forming a superset of our main sample. We then standardize zip codes for the 20 most frequent countries in which these trials are conducted, covering approximately 90% of all trials on ClinicalTrials.gov. We define each unique zip code within a country as a distinct trial location, z, and drop any trials with missing addresses, participant counts, or start and completion dates.

In a first step, we construct a congestion measure at the zip code level. For any trial k conducted across N_k sites, with a start year τ_1 and completion year τ_2, we calculate the average patient enrollment per year and site, E_k, as a trial's total number of enrolled patients divided by the number of sites, N_k, and the number of years to complete the trial, $(\tau_2 - \tau_1)$. The congestion measure for a focal trial i in location z of year t, denoted G^i_{zt}, is then defined as the sum of E_k across all trials with a site located in zip code z and year t, excluding E_i itself (in a leave-one-out fashion):

$$G^i_{zt} = \sum_{\{k \in I \setminus i : N_k \cap z = z, t \in [\tau_1, \tau_2]\}} E_k,$$

where $I \setminus i$ denotes the set of all clinical trials excluding trial i. The key intuition is that a larger G^i_{zt} implies that location z is hosting more trial participants in year t.

In a second step, we calculate the change in congestion at location z between the start year τ_1 and the completion year τ_2 of trial i. We scale this change by the mean level of congestion in location z across years, $\overline{G}_z$, so that the resulting measure can be interpreted as the relative change in congestion.

Our instrument for trial completion delay is then defined as

$$z_i = CongestionChange_i = \max_{z \in N_i} \left\{ \frac{G^i_{z\tau_2} - G^i_{z\tau_1}}{\overline{G}_z} \right\},$$

that is, the instrument uses the maximum change in congestion across trial i's locations. This reflects the notion that a clinical trial cannot be completed until results are finalized across all sites. For the average trial in our sample, the maximum normalized increase in site-level congestion between the start and end of the trial is slightly more than 5% (Table 1).

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Discussion of the Exclusion Restriction Apart from instrument relevance condition—that is, the requirement that changes in trial site congestion strongly affect trial completion delays (which we establish in Section 4.2)—the key identifying assumption for the IV strategy is that changes in trial site congestion influence project suspension decisions only through their effect on trial delays.

One argument supporting this exclusion restriction is that changes in site-level congestion are an aggregate measure of trial activity across drug developers and drug fields, and are therefore unlikely to correlate with unobserved shocks to a given firm's decision to advance a specific clinical trial, which is a firm-level outcome.

At the same time, there are plausible concerns regarding the exclusion restriction. One concern is that more crowded trial locations, with larger participant volumes, may indicate higher service quality—such as better monitoring, more robust data collection, and stricter adherence to clinical protocols. Trials conducted at such sites might, despite potentially experiencing delays, yield higher-quality results, thereby making firms more likely to advance them. To alleviate this concern, we collect detailed hospital quality data as a proxy for trial site quality (see Section 2.3) and examine their correlation with our congestion instrument. As shown in Figure 3, we find no evidence of a meaningful correlation—positive or negative—between the congestion instrument and trial site quality, proxied by the average clinical score across hospitals in a zip code, spanning 26 distinct hospital quality measures.

A second concern is that, although the instrument captures aggregate, rather than project-specific variation, there may exist aggregate shocks that simultaneously increase trial site activity and the prospects of drugs under development, such as innovations or drug demand shocks within a specific disease field. We address this concern in detail in Section 4.2 when we present the IV results. To preview, the results are robust to controlling, both linearly and nonparametrically, for the number of competing drug projects in the same ICD category. In addition, we construct an alternative version of the instrument that excludes patient flows from drugs in the same ICD category. This adjustment purges the instrument of potential confounding from correlated disease-field shocks and isolates variation in trial site congestion stemming from unrelated drug projects. Our IV results remain highly similar when using this alternative measure.

Finally, we also find that the congestion instrument is empirically uncorrelated with a range of project quality and experimentation measures, further alleviating concerns related to the exclusion restriction. We discuss these additional (non)correlations in more detail in Section 5.1.[12]

[12] Yet another plausible concern is that, even if the instrument captures changes in congestion after trial start driven by decisions from other parties, firms may still predominantly use the same sites across trials,

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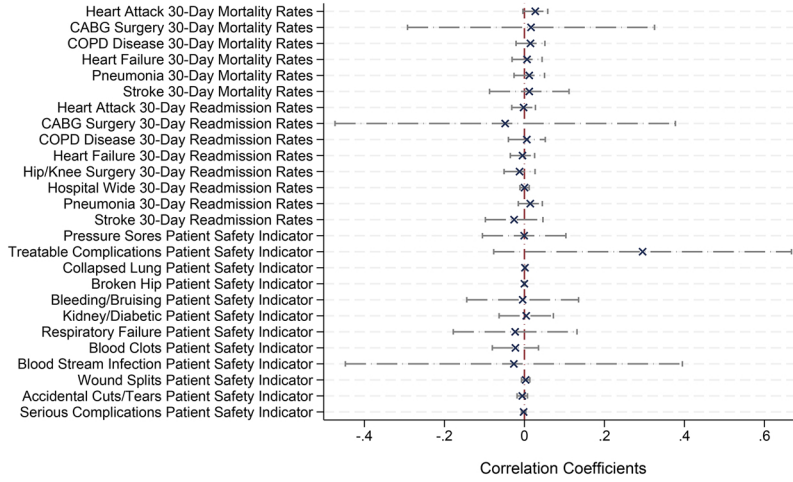

Figure 3
Association between change in trial site congestion and hospital care quality
This figure shows the association between the trial site congestion measure and hospital care quality measures at the zip code level. The plotted coefficients, β, are estimated from the regression

$$Q_{zt} = \beta G_{zt} + \gamma_z + \tau_t,$$

where Q_{zt} is the average hospital care quality in zip code z in year t, G_{zt} is the corresponding congestion measure, and γ_z and τ_t are zip code and year fixed effects, respectively. The y-axis lists the quality measures. Standard errors are two-way clustered at the zip code and year levels. Capped lines indicate 95% confidence intervals.

4. Main Results

4.1 Delays in trial phase completion and project continuation versus suspension

Table 2 presents the baseline results on how a firm's decision to continue versus suspend a clinical trial following a completed phase depends on the experienced delay in trial phase completion. Column (1), which compares trials initiated within the same year and quarter, shows a strong negative effect of delay on the likelihood of suspension, both economically and statistically. A one standard deviation increase in delay in the preceding trial phase reduces the suspension probability by 4.4 percentage points, or 15% relative to the baseline suspension probability of 31% (Table 1).

The effect of trial phase completion delay on trial continuation remains stable across specifications. In column (2), the estimates are unchanged when we add firm fixed effects. Column (3) shows that the result also holds when we further include fixed effects for the drug's ICD category

potentially complicating the validity of the instrument. To address this, Figure IA.2 examines the overlap in trial site zip codes across successive trials within firms and shows consistently low overlap. Moreover, the degree of site overlap is empirically uncorrelated with both experienced delays and changes in site congestion.

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Table 2
Delays in trial phase completion and project continuation versus suspension

	Dependent Variable: *Suspension*			
	(1)	(2)	(3)	(4)
Delay in Trial Completion	–0.229***	–0.210***	–0.206***	–0.200***
	(–5.77)	(–6.92)	(–5.70)	(–5.41)
Controls	N	N	N	Y
Year × Quarter FE	Y	Y	Y	Y
Firm FE	N	Y	Y	Y
ICD FE	N	N	Y	Y
Trial Phase FE	N	N	Y	Y
Observations	9,912	9,433	9,397	9,161
Adj. *R*-squared	0.1558	0.2711	0.3086	0.3099

This table reports OLS estimates of Equation (1), examining the relationship between delays in trial phase completion and project continuation versus suspension. The unit of observation is a drug project. The sample includes all projects that completed Phase I or II trials with detailed clinical trials records. The dependent variable, $Suspension_i$, is an indicator variable (multiplied by 100 for ease of exposition) for whether project i was suspended after completing Phase I or II trials. The key independent variable, $DelayinTrialCompletion_i$, measures the gap in months between the realized and expected trial completion dates. Fixed effects are reported in the bottom rows. Control variables include the log number of trial sites, number of enrolled participants (in hundreds), log number of projects in the company's pipeline, and log number of competing projects in the same ICD category. Standard errors are two-way clustered at the ICD category and year-by-quarter levels, and t-statistics are reported in parentheses. $^{*}p<.1$; $^{**}p<.05$; $^{***}p<.01$.

and for the trial phase (i.e., whether the completed phase was a Phase I or II trial). Finally, in column (4), the effect remains robust to the inclusion of additional control variables, namely the number of sites, the number of enrolled participants, the number of drug projects in the company's pipeline, and the number of competing projects in the same ICD category.[13]

Figure 4 visualizes the negative relationship between trial completion delay and subsequent trial suspension, after residualizing both variables with respect to the control variables and fixed effects from the final specification in Table 2. The figure reveals an almost monotonic decline in the probability of suspension as delay increases.

Overall, the results in Table 2 and Figure 4 provide initial evidence that firms' prior investments to a given clinical trial—in the form of time required to complete the trial beyond initial expectations—increases subsequent project commitment, as measured by the decision to subsequently continue rather than suspend the project.

4.2 Increase in trial site congestion as an instrument for trial phase completion delays

Table 3 presents the main IV results, estimating the effect of trial phase completion delays that are driven by changes in trial site congestion on

[13] The coefficients on these control variables (unreported) are in line with our prior. A larger pipeline and more competing projects are positively (though insignificantly) associated with suspension. Trials with more sites are more likely to be suspended, while those with more enrolled participants are less likely to be suspended.

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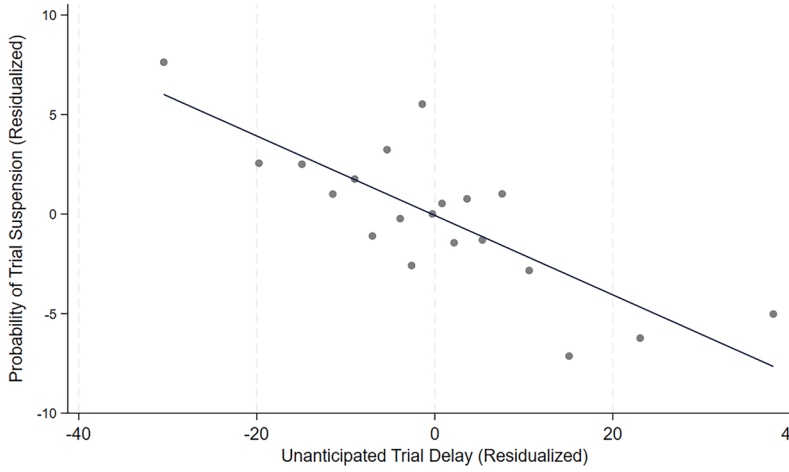

Figure 4
Delays in trial phase completion and project continuation versus suspension
This figure shows a binscatter plot of the relationship between unanticipated clinical trial delays and firms' subsequent decision to suspend versus advance the trial through the next phase. Both the delay variable and the suspension indicator are residualized on the control variables and fixed effects from the final column of Table 2.

the subsequent decision to continue versus suspend the project. Columns (1) and (2) report the first-stage results, that is, the relation between delay and change in trial site congestion, with and without the control variables from the final column of Table 2. Both specifications reveal a strong positive association between congestion and delay. Economically, a one standard deviation increase in the congestion instrument is associated with an increase in trial completion delay of nearly 4 months. Statistically, the Kleibergen and Paap (2006) F-statistic is well above the conventional weak instrument threshold of 10 (Stock, Wright, and Yogo 2002). Figure IA.3 in the Internet Appendix visually confirms this relationship: a binned scatterplot shows a strong and nearly monotonic positive association between increases in trial site congestion and resulting unanticipated trial delays.

Columns (3) and (4) present the second-stage results. Trial completion delays, when instrumented using trial site congestion, continue to have a significant effect on firms' decision to advance versus suspend the trial project. The economic magnitudes of the IV estimates are somewhat larger than the OLS point estimates from Table 3, which we discuss further below. Statistically, the coefficients on instrumented delay remain significant at 5% or 1%, where we continue to use two-way clustered standard errors.

Overall, the IV results reinforce the interpretation that unanticipated, plausibly exogenous delays in trial phase completion lead to excessive

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Table 3
Delays in trial phase completion and project continuation versus suspension: Increase in trial site congestion as an instrument for delay

	Dependent Variable: *Delay*		Dependent Variable: *Suspension*	
	First Stage		Second Stage	
	(1)	(2)	(3)	(4)
Trial Site Congestion	0.361***	0.359***		
	(4.27)	(4.39)		
Delay in Trial Completion			-0.335**	-0.528***
			(−2.33)	(−2.98)
Controls	N	Y	N	Y
Year × Quarter FE	Y	Y	Y	Y
Firm FE	Y	Y	Y	Y
ICD FE	Y	Y	Y	Y
Trial Phase FE	Y	Y	Y	Y
F-stat	18.24	19.24	–	–
Observations	9,397	9,161	9,397	9,161

This table reports IV estimates of Equation (1), examining the association between delays in trial phase completion and project continuation versus suspension, and using changes in trial site congestion as an instrument for trial completion delays. The unit of observation is a drug project. The sample includes all projects that completed Phase I or II trials with detailed clinical trials records. The second-stage dependent variable, $Suspension_i$, is an indicator variable (multiplied by 100 for ease of exposition) for whether project i was suspended after completing Phase I or II trials. The key independent variable, $DelayinTrialCompletion_i$, measures the gap in months between the realized and expected trial completion dates. The instrument, $TrialSiteCongestion_i$, is the normalized, zip-code-level change in the number of participants in the universe of trials on ClinicalTrials.gov between the trial start and end dates, taking the maximum across all trial sites of a given trial in the sample. Fixed effects are reported in the bottom rows. Control variables include the log number of trial sites, number of enrolled participants (in hundreds), log number of projects in the company's pipeline, and log number of competing projects in the same ICD category. Standard errors are two-way clustered at the ICD category and year-by-quarter levels, and t-statistics are reported in parentheses. $^{*}p<.1$; $^{**}p<.05$; $^{***}p<.01$.

project commitment, as reflected in firms' increased propensity to continue delayed trials.

IV Versus OLS Magnitudes Several observations are worth noting when comparing the estimated OLS and IV magnitudes in Tables 2 and 3, respectively. First, while the IV estimates are larger in magnitude than the corresponding OLS estimates, they remain economically plausible. For example, a 1 year increase in delay during the preceding trial phase is associated with a 4.0–6.3 percentage point decrease in suspension probability, corresponding to a 13–20% change relative to the baseline suspension rate. Moreover, the 95% confidence intervals of both IV estimates in fact include the OLS point estimates. Nonetheless, it is useful to consider potential reasons for the observed gap in point estimates.

One possibility is residual measurement error in the *Delay* variable, for example, due to remaining reporting incentives (see Section 3.1). In this case, the OLS estimates may be attenuated, whereas IV regressions can recover the true effect. To assess this possibility, we follow Pancost and Schaller (2024) in computing the measurement error ratio, which

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quantifies how much measurement error weakens the estimated effect.[14] We compute this ratio using two IV-OLS pairs with the same regressor, *Delay*, but different dependent variables: suspension probability (as in Table 3) and progress probability (defined as 100 times an indicator for a project advancing to the next stage).[15] A value closer to one indicates less attenuation. The resulting measurement error ratio is 0.725, suggesting only modest measurement error.

Second, the larger IV estimates may partly reflect an omitted variable in the OLS regressions. One candidate is variation in the quality of contract research organizations (CROs), which support trial execution. More experienced CROs may reduce delays and suspension risk through more efficient management and higher-quality data, potentially attenuating the OLS estimates. The IV estimates, by contrast, would remain valid so long as CRO quality is uncorrelated with changes in trial site congestion.

Finally, it is worth noting that IV estimates identify a local average treatment effect, the effect of delay on the subset of projects whose duration is affected by variation in congestion (i.e., compliers). If compliers respond more strongly to delay, this too could help explain the larger IV estimates.

Taken together, these points highlight several plausible, non–mutually exclusive explanations for the observed gap between the OLS and IV estimates.

IV Robustness We conclude this section with a discussion of several IV robustness tests. These tests address a remaining concern with the congestion-based IV approach: that, despite the instrument's lack of correlation with trial site quality proxies (Figure 3; see also Section 3.2), disease-category-wide shocks could simultaneously influence trial site activity and project viability.

Three pieces of evidence alleviate this concern. First, as in the final column of the OLS results in Table 2, the final IV specification in Table 3 directly controls for the number of competing drug projects in the same ICD category, accounting for broader disease-field-wide effects. Second, Internet Appendix Table IA.3 shows that the IV results are virtually unchanged when controlling for the number of competing drug projects in the same ICD category nonparametrically, using number-of-competing-drug decile fixed effects, rather than parametrically. The results also remain quantitatively similar in subsamples with below-median and above-median drug project competition, respectively. Third, in Internet Appendix Table IA.4, we go one step further and reestimate the IV model using an

[14] See also an application of this method in Lerner, Li, and Liu (2023).

[15] We note that progress and suspension are not mechanical complements, since nonsuspension differs from having progressed in trials that remain active but have not yet completed the next phase.

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alternative congestion instrument that excludes patient flows from drugs in the same ICD category. This alternative instrument, described in detail in Internet Appendix Section A.2, captures trial site congestion driven only by unrelated drug projects, thereby eliminating possible confounding effects from disease-field-specific shocks. The IV results remain unchanged under this alternative specification, both economically and statistically.[16]

5. Alternative Interpretations

The previous section establishes that experiencing unanticipated trial delays is associated with a significant increase in firms' subsequent project continuation propensity. A key question for interpreting this result as evidence of excessive sensitivity to delays is whether delays may convey information that increases a project's value, either because delays are more likely among higher-quality projects or because firms respond endogenously to delays, thereby justifying continued investment. This section presents a series of tests that, individually and collectively, argue against this alternative interpretation.

5.1 Key concern 1: Delays not as good as randomly assigned

One key concern for why a positive correlation between project quality and delays might arise is if delays are not as good as randomly assigned to projects but instead occur more frequently among higher-quality projects. Our setting allows us to provide three tests and arguments to alleviate this concern.

First, the IV results from Section 4.2 address the concern, provided that changes in trial site congestion are uninformative about, and uncorrelated with, project quality and information. In this case, trial site busyness isolates variation in project delays driven by factors unrelated to project quality. As discussed in Section 3.2, consistent with this, we find no evidence that increasingly congested sites are associated with better service quality or aggregate disease field shocks, either of which could plausibly enhance project quality.

Second, we can analyze direct measures of project quality; specifically, the collected third-party measures of drug quality from the GlobalData Pharma database described in Section 2.2: the predicted drug-specific likelihood of FDA approval and the drug's predicted phase transition success rate. Table 4 examines the association between experienced trial phase delays and these drug quality measures, as well as the association

[16] As shown in Internet Appendix Table IA.4, both the first- and second-stage results are stable under the alternative instrument. The Kleibergen and Paap (2006) F-statistic remains well above 10, confirming the relevance of the instrument even when congestion is driven solely by trials in other ICD categories—for instance, due to limited staff, constrained physical space, or slower patient recruitment.

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Table 4
Association between trial delays and drug quality

	Dependent Variable:		Dependent Variable:	
	Delay in Trial Completion		*Trial Site Congestion*	
	(1)	(2)	(3)	(4)
Drug Likelihood of Approval (LOA)	0.083		0.011	
	(0.79)		(0.21)	
Phase Transition Success Rate (PTSR)		0.017		0.002
		(0.38)		(0.08)
Controls	Y	Y	Y	Y
Year × Quarter FE	Y	Y	Y	Y
Firm FE	Y	Y	Y	Y
ICD FE	Y	Y	Y	Y
Trial Phase FE	Y	Y	Y	Y
Observations	2,884	2,884	2,925	2,925
Adj. *R*-squared	0.3584	0.3582	0.4881	0.4881

This table examines the association between trial delays and drug quality measures. Columns (1) and (2) use $DelayinTrialCompletion$ as the dependent variable, while columns (3) and (4) use $TrialSiteCongestion$. The two drug quality measures, *Drug Likelihood of Approval* and *Phase Transition Success Rate (PTSR)*, capture the predicted probabilities that a drug candidate will receive FDA approval and successfully progress from its current trial phase to the next, respectively. Both measures are obtained from GlobalData. Fixed effects are reported in the bottom rows. Control variables include the log number of trial sites, number of enrolled participants (in hundreds), log number of projects in the company's pipeline, and log number of competing projects in the same ICD category. Standard errors are two-way clustered at the ICD category and year-by-quarter levels, and *t*-statistics are reported in parentheses. $^{*}p<.1$; $^{**}p<.05$; $^{***}p<.01$.

between changes in trial site congestion and these same measures. In columns (1) and (2), the correlation between the LOA and PTSR measures with the experienced trial phase completion delay is insignificant, both economically and statistically. Next, in columns (3) and (4), the correlation between the LOA and PTSR measures with the instrument, the change in trial site congestion, is also insignificant. These results further alleviate the concern that omitted variables related to project quality affect the baseline or instrumented delay results from the previous section.

The Internet Appendix presents related supporting results. For one, Internet Appendix Table IA.5 shows that the main effect on the relationship between trial delays and project continuation is unaffected when including the LOA and PTSR measures directly in the estimation, rather than examining their correlation with delay, as in Table 4.[17] In addition, the Internet Appendix examines the relationship between experienced trial delays and additional trial quality and profitability proxies beyond LOA and PTSR. Panel A of Internet Appendix Table IA.6 shows that trial delays are uncorrelated with expected drug sales conditional on drug launch, using the data obtained from Cortellis Competitive Intelligence. This holds across multiple expected sales measures, including the sum of expected

[17] The difference in coefficient estimates in Table IA.5 is entirely driven by the fact that the LOA and PTSR measures are only available for a subsample. Column (1) repeats the main specification on this restricted sample but omits these controls, yielding estimates that are nearly identical to those in columns (2) and (3), where LOA and PTSR are included as additional controls.

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Table 5
Association between trial delays and drug experimentation

	Dependent Variable: *Delay in Trial Completion* (1)	Dependent Variable: *Trial Site Congestion* (2)
Num of Doses in a Trial	0.389	0.009
	(1.42)	(0.08)
Controls	Y	Y
Year × Quarter FE	Y	Y
Firm FE	Y	Y
ICD FE	Y	Y
Trial Phase FE	Y	Y
Observations	3,853	3,905
Adj. *R*-squared	0.3563	0.5247

This table examines the association between trial delays and drug experimentation, measured by the number of doses tested in a clinical trial. Column (1) uses *DelayinTrialCompletion* as the dependent variable, while column (2) uses *TrialSiteCongestion*. The key independent variable, *Num of Doses in a Trial*, counts the number of drug doses tested. Fixed effects are reported in the bottom rows. Control variables include the log number of trial sites, number of enrolled participants (in hundreds), log number of projects in the company's pipeline, and log number of competing projects in the same ICD category. Standard errors are two-way clustered at the ICD category and year-by-quarter levels, and *t*-statistics are reported in parentheses. $^{*}p<.1$; $^{**}p<.05$; $^{***}p<.01$.

sales in the first 5 years and the expected average annual sales after FDA drug approval (based on all available years). Panel B of Internet Appendix Table IA.6 shows that trial delays are also uncorrelated with the frequency of severe adverse reactions during the completed trial phase, as another project quality proxy.[18]

Third, we can also examine measures of drug project experimentation. One possible concern is that firms testing multiple dosages within a trial might be more likely to experience (unanticipated) delays, while also generating valuable scientific data on optimal dosing. This, in turn, could enhance project value and increase firms' willingness to continue the project. While this concern is more relevant for the OLS estimates than for the IV approach, which relies on instrumented delay, we examine it further using project-level information on number of dosages tested. In Table 5, we estimate the association between both experienced delays and the instrument with the number of dosages tested. Column (1) shows that trial phase delays are not significantly correlated with the number of dosages tested, and column (2) shows that the instrument is similarly uncorrelated with dosage experimentation. This evidence is not easily consistent with the notion that drug experimentation is the primary driver of the observed relationship between delay and project continuation.

[18] We examine both the total and maximum percentage of life-threatening reactions relative to the total number of enrolled trial participants. Specifically, for each life-threatening reaction (e.g., acute myelogenous leukemia, basal cell carcinoma, or brain hemorrhage), we calculate the proportion of trial participants experiencing that reaction. The total percentage is the sum of these proportions across all life-threatening reactions, while the maximum percentage represents the highest proportion observed for any single reaction.

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Overall, the evidence in this section speaks against an omitted variable related to project quality or information as the underlying explanation for our findings.

5.2 Key Concern 2: Endogenous selection into trial phase completion

A second key concern is that firms might endogenously respond to delays as they unfold, even if the onset of delay is not correlated with project quality. This could introduce selection in terms of which projects ultimately complete a trial phase. Such selection is relevant because our sample is restricted to completed trials that achieved their endpoints (see Section 2.2), a necessary condition for constructing precise delay measures based on realized trial completion dates and for ensuring that observed suspension decisions are not mechanically driven by unfavorable readouts in the preceding trial phase.[19] We discuss two versions of this selection concern, and implement a two-pronged approach that includes additional robustness tests as well as the collection of an additional sample of drug projects that were abandoned prior to reaching their endpoints.

Firm Heterogeneity in Drug Commitment A first version of the above selection concern is that some drug developers may be inherently more committed to their projects from the outset and, as a result, more willing to sustain project delays and successfully complete the trial phase while achieving all endpoints. A higher baseline level of commitment would, in turn, predict a greater likelihood of project continuation.

To alleviate this concern, we include in all analyses firm fixed effects, which absorb any potential differences in ex-ante project commitment across firms. In addition, our results remain robust when adopting a more stringent specification that includes firm-by-ICD-category fixed effects. This specification further accounts for any potential differences in ex-ante commitment within the same firm across disease fields and project types—for example, if a firm has differing ex-ante commitment levels for cancer drugs compared to pneumonia drugs. As shown in Internet Appendix Tables IA.7 and IA.8, both the OLS and IV results remain similar with the inclusion of the more stringent firm-by-ICD-category fixed effects.[20]

Project-Specific Heterogeneity in Drug Commitment A second version of the selection concern is that lower-quality projects may be

[19] This restriction removes 3,397 Phase I or II projects that did not achieve their endpoints from the raw sample.

[20] While the statistical significance weakens in column (3) of Internet Appendix Table IA.8, all results remain significant at 5% or 1% in the most stringent specifications, which include the full slate of fixed effects and control variables.

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more likely to be withdrawn when facing delays before achieving their endpoints. This type of selection could occur within a given firm or within firm-by-disease-fields, and thus would not be accounted for by the fixed effects in the previous paragraph. Such behavior could systematically exclude struggling projects from our final sample in a nonrandom manner, potentially leading to an overestimation of the sensitivity of project continuation to delays among completed trials.[21]

This concern gives rise to two testable predictions that we can assess by assembling an expanded data set that includes withdrawn projects, that is, those that did not achieve their endpoints. First, if lower-quality projects are more likely to be withdrawn when facing delays, the withdrawn projects should exhibit shorter delays on average. Second, withdrawn projects should be of lower quality on average.

To assemble the expanded data set and test these predictions, we follow a similar approach to that described in Section A.1 of the Internet Appendix for the sample of completed projects, and scrape anticipated completion dates from ClinicalTrials.gov's "History of Changes" records for the subsample of withdrawn projects. Because withdrawn projects, by definition, lack actual completion dates, we compute the delay measure for these projects as the difference between the withdrawal date and the anticipated completion date filed on ClinicalTrials.gov at project initiation.

Internet Appendix Figure IA.4 tests the first prediction above, comparing delays between projects that are withdrawn and those that achieve their endpoints (and are therefore included in the main sample analyzed in Section 4). The delay distributions for withdrawn and completed projects are very similar, with a p-value of 0.55 for a test of equality of distributions. Thus, there is no evidence that projects are selectively abandoned in response to ensuing delays. Next, Internet Appendix Table IA.9 tests the second prediction above, examining whether observable measures of drug quality differ between withdrawn projects and those that achieve their endpoints. As before, we use GlobalData's drug likelihood of approval and phase transition success rate as proxies for project quality. The results show that neither measure—statistically nor economically—predicts project withdrawal, providing further evidence against endogenous sample selection as a confounding factor.

Overall, the evidence in this section speaks against endogenous selection into trial phase completion as the underlying explanation for our findings.

[21] As a first way to gauge and bound the severity of this concern, we collect and tabulate the reasons for discontinuation among the projects excluded for not achieving their endpoints. Only 12% explicitly cite reasons related to "Adverse Events" or "Lack of Activity or Efficacy," that is, reasons indicative of lower quality. The additional tests presented in the remainder of this section further help to alleviate this type of selection concern.

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5.3 Further concerns and robustness tests

This section examines additional potential confounders and presents further robustness tests, both reinforcing the evidence against the alternative explanations discussed in Sections 5.1 and 5.2 and addressing remaining threats to our excess commitment interpretation. It also presents corroborating evidence from an entirely separate strategy—based on exchange-rate–induced variation in trial costs—that supports our main hypothesis of an excess relationship between unanticipated project investment and project continuation.

Trial Delays Reflecting Real Options First, directly related to the prior discussion about trial delays and drug experimentation and quality, one potential concern is that delays may reflect firms exercising a real option in response to favorable new information (Pindyck 1991; Dixit, Dixit, and Pindyck 1994; McAfee, Mialon, and Mialon 2010). However, the analysis presented above provides evidence against this channel as the driver of our findings. In particular, the instrumented, congestion-induced delay is plausibly unrelated to a "learning-by-doing" channel, and neither project delay nor congestion is significantly associated with any observable experimentation and quality measures (drug dosages tested, project success probabilities, expected sales, or adverse events during trials). These patterns are difficult to reconcile with a real options explanation.

FDA Clinical Trial Holds Second, we address possible concerns that the observed relationship between trial delays and continuation may reflect the effect of FDA clinical trial holds, which are orders issued by the FDA requiring sponsors to delay a proposed clinical investigation.[22] During a hold, drug developers typically work with the FDA to revise the clinical protocol and address deficiencies. As a result, trials subject to holds may be prolonged but could also benefit from improved design or oversight, potentially increasing the likelihood of continuation. We note that this concern mostly applies to the OLS-based delay results in Table 2, rather than the IV-based delay results in Table 3 and, to address it further, Internet Appendix Table IA.10 shows that the presence of clinical holds is not significantly associated with trial duration, trial delays, or the trial site congestion instrument. We conclude that FDA clinical holds are unlikely to be driving our results.

Further Miscellaneous Tests Third, we implement additional miscellaneous robustness tests. In Internet Appendix Table IA.11, we show that

[22] Common reasons for such holds include impurity profiles suggesting potential health hazards, missing information on dosing or exposure, or insufficient detail in the clinical protocol (Lapteva and Pariser 2016).

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our results are driven by trials that experience a positive (that is, ≥ 0) delay, whereas negative delays—projects completed earlier than anticipated—do not significantly predict the decision to continue versus suspend.[23] Internet Appendix Table IA.12 provides several further tests. Column (1) shows that our results remain robust when we exploit variation in trial completion delays within trials initiated in the same quarter and targeting the same ICD category, using time-by-ICD-category fixed effects. In columns (2) and (3), we reestimate the model separately for U.S. and non-U.S. trials to account for potential geographic differences in regulatory environments and cost structures; the results are consistent across both subsamples. To address concerns arising from Cortellis data having weaker coverage prior to 2000, column (4) restricts the sample to trials initiated in 2000 or later; the results remain robust. Finally, column (5) focuses on trials beginning after the 2007 passage of the Food and Drug Administration Amendments Act, which altered disclosure requirements and may have affected reporting quality (Aghamolla and Thakor 2022b; Hsu et al. 2025); again, the findings hold.

Additional Validation from Exchange-Rate-Induced Variation in Trial Costs Finally, moving beyond temporal trial investments made (and their associated increased trial costs), we conduct an additional validation test using variation in trial costs driven by exchange rate fluctuations. We briefly summarize the approach here and provide details in Internet Appendix Section A.3.

The key idea is that if firms increase their commitment following unanticipated trial delays and time-based investments, we might observe similar behavior in response to unexpected financial investments. To test this, we leverage the institutional feature that many clinical trials are conducted outside the United States (Figure 1), where contracts and payments are typically denominated in local currencies. When foreign currencies appreciate relative to the U.S. dollar, drug developers face unexpected cost increases due to exchange rate volatility.[24]

Internet Appendix Table IA.13 examines how these exchange-rate-induced cost shocks affect firms' decisions to continue versus suspend trials. Columns (1) and (2) focus on foreign-based trials, with column (2) adding controls. Columns (3) and (4) use the full sample, interacting exchange rate variation and covariates with an indicator for foreign trials. In all specifications, higher exchange-rate-induced costs significantly reduce suspension likelihood. A one standard deviation increase in costs

[23] Only about 15% of trials in our sample are completed prior to the originally anticipated completion date.

[24] See, for example, https://www.appliedclinicaltrialsonline.com/view/mastering-currency-fluctuation, which notes that "a study's financial obligations may include absorbing fluctuations in exchange rates" and that payments occur "typically in the currencies of the countries where the trials are held."

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lowers the suspension probability by 1.75 percentage points (6% relative to baseline), which is somewhat smaller but still economically meaningful compared to the effect of delays in our main analysis. We further assess robustness through a placebo test that randomly reassigns trial countries and recomputes the cost shocks. Figure IA.5 shows that the distribution of placebo coefficients is centered near zero and far smaller in magnitude than the true estimates.

Overall, the exchange rate analysis offers additional support for our interpretation: plausibly exogenous increases in trial-specific investments—whether temporal or financial—elevate firms' (excess) propensity to continue project development.

6. Underlying Economic Channels

Having established the main results and addressed alternative interpretations related to omitted variables and endogenous selection into project phase completion, we now examine potential underlying economic mechanisms that may contribute to excess project commitment and continuation in response to trial delays. We begin by examining firm- and project-level channels, including agency conflicts, initial firm expectations about trial duration, financial constraints, heterogeneities by trial phase, and accumulated knowledge capital. We then turn to mechanisms related to the ultimate decision-maker responsible for R&D investment—the CEO of the pharmaceutical firm.

6.1 Project-characteristics- and firm-related channels

Lack of Other Viable Drug Candidates Prior work by Guedj and Scharfstein (2004) argues that early-stage biopharmaceutical firms may distort trial continuation decisions due to a reluctance to abandon their only viable drug candidate.[25] To test whether agency conflicts in early-stage firms can help explain our findings, we restrict the sample to trials conducted by firms with more than 10 drug projects in their pipeline at the time of trial completion, that is, firms with ample alternative investment opportunities. This restriction removes less than 25% of the sample, indicating that the results in previous sections are largely driven by firms with many ongoing projects, leaving limited scope for an early-stage firm channel. Consistent with this, column (1) of panel A in Table 6 shows that unexpected delays continue to predict trial continuation decisions with nearly identical magnitude within this subsample. Thus, our effects

[25] Hermosilla (2021) documents a related pattern among large pharmaceutical firms, showing that they increase licensing activity to sustain their drug development pipeline following unexpected Phase III failures of in-house R&D projects that leave them without other viable internal drug candidates. In contrast, we focus on firms' decisions regarding projects within their existing development pipeline.

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are not driven by early-stage firms lacking alternative drug candidates as in Guedj and Scharfstein (2004).

Trial Duration Expectations Another potential mechanism is that firms that are more optimistic about a project's prospects may be both more inclined to advance it and more likely to understate its anticipated duration. This could generate a correlation between delays and continuation decisions, as more optimistic firms would report shorter timelines and continue projects at higher rates. In the data, we do observe a positive correlation between a trial's anticipated duration and its realized delay, which is consistent with this possibility. However, as discussed in Section 3.1, two arguments are at odds with this explanation. First, firms are required to report anticipated trial end dates truthfully. Second, our IV strategy isolates variation in delays that is plausibly orthogonal to firms' expectations, as long as they are uncorrelated with the congestion instrument. Further supporting this, when we directly control for firms' reported anticipated duration in column (2) of panel A in Table 6, the results remain unchanged.[26] Together, this evidence suggests that an optimism-based mechanism operating through trial duration expectations is unlikely to explain our findings.[27]

Financial Constraints Next, we test whether firms' sustained commitment to drug projects in response to trial delays may be influenced by financial constraints. Once delays have been incurred in a preceding trial phase—which can heighten financial pressures—it may become more difficult for firms to pivot and initiate new projects. In this sense, continuation may be the relatively more attractive and less costly choice for financially constrained firms compared to suspension and reallocation. Mace (2023) finds that financial constraints can lead firms to abandon early-stage developments; an alternative possibility in our setting is that, after delays, such constraints may instead push firms to persist. To test for such a channel, we augment our specifications with various financial constraints measures (thereby restricting the sample to public firms). Specifically, the *Constrained* variable in the third column of panel A in Table 6 is an indicator variable equal to one if the firm's Whited and Wu (2006) (WW) index at trial end is in the top quartile of the index's sample distribution. Column (3) shows that the estimated effect of delay on project continuation remains unchanged with this added

[26] In unreported tests, we include second- and third-order polynomials of anticipated duration and find results nearly identical to those reported in Table 6.

[27] We note that controlling for firms' anticipated trial duration also helps further address concerns about strategic reporting incentives. Conditioning on reported timelines accounts for potential strategic behavior and helps isolate the residual variation in realized delays.

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Table 6
Firm- and project-characteristics-related channels

A: Firm-characteristics-related channels

	Dependent Variable: Suspension				
	Has Other Inv. Opportunities (1)	Controlling for Expectations (2)	Controlling for Fin. Constr. (3)	Phase I Only (4)	Phase II Only (5)
Delay in Trial Completion	–0.208***	–0.224***	–0.196***	–0.152**	–0.199***
	(–5.15)	(–5.87)	(–4.08)	(–2.63)	(–5.08)
Anticipated Trial Duration		–0.112**			
		(–2.57)			
Constrained (WW)			0.238		
			(0.06)		
Controls	Y	Y	Y	Y	Y
Year × Quarter FE	Y	Y	Y	Y	Y
Firm FE	Y	Y	Y	Y	Y
ICD FE	Y	Y	Y	Y	Y
Trial Phase FE	Y	Y	Y	Y	Y
Observations	7,052	9,161	4,746	3,356	5,414
Adj. *R*-squared	0.2726	0.3107	0.2703	0.3655	0.3294

B: Knowledge capital channel

	Dependent Variable: Suspension	
	(1)	(2)
Delay in Trial Completion	–0.202***	–0.249***
	(–5.27)	(–3.23)
$\#PriorProjects$	–0.055	
	(–0.52)	
$Delay \times \#PriorProjects$	0.001	
	(0.13)	
$\mathbf{1}\{PriorProjects\}$		–3.449
		(–1.54)
$Delay \times \mathbf{1}\{PriorProjects\}$		0.059
		(0.80)
Controls	Y	Y
Year × Quarter FE	Y	Y
Firm FE	Y	Y
ICD FE	Y	Y
Trial Phase FE	Y	Y
Observations	9,161	9,161
Adj. *R*-squared	0.3098	0.3101

This table reports OLS estimates examining underlying firm- and project-characteristics-related channels. The unit of observation is a drug project. The sample includes all projects that completed Phase I or II trials with detailed clinical trials records. Panel A focuses on channels related to investment opportunities, expectations, financial constraints, or development stages, while panel B focuses on firms' prior knowledge capital. The dependent variable, $Suspension_i$, is an indicator variable (multiplied by 100 for ease of exposition) for whether project i was suspended after completing Phase I or II trials. The key independent variable, $DelayinTrialCompletion_i$, measures the gap in months between the realized and expected trial completion dates. In panel B, $\#PriorProjects$ counts the number of drug projects initiated by the developer in prior years sharing the same ICD category, and the indicator variable $\mathbf{1}\{\cdot\}$ equals 1 if there is at least one such project in the same ICD category. Fixed effects are reported in the bottom rows. Control variables include the log number of trial sites, number of enrolled participants (in hundreds), log number of projects in the company's pipeline, and log number of competing projects in the same ICD category. Standard errors are two-way clustered at the ICD category and year-by-quarter levels, and t-statistics are reported in parentheses. $^{*}p<.1$; $^{**}p<.05$; $^{***}p<.01$.

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constraint measure.[28] Thus, the documented path dependence in firm decision-making is unlikely to be explained by financially constrained firms lacking the flexibility to shift focus to alternative projects.

Phase I versus II Trials The last two columns of panel A in Table 6 separate the main result by whether the preceding trial phase was Phase I or Phase II. The effect of trial delay on subsequent continuation versus suspension is robust and large for both Phase I and II trials. This indicates that our findings are not driven by trial-phase-specific confounders but instead reflect a robust pattern in early-stage drug development.

The robust relationship across both phases is worth emphasizing. For Phase I trials, where the probability of eventual approval is lowest and cost increases may be most likely to render a project unviable, the presence of excess commitment is particularly striking. For Phase II trials, the point estimates are slightly larger, which is consistent with path dependence effects becoming stronger as projects progress. Together, these patterns align with the broader idea that commitment becomes increasingly difficult to reverse as more resources and information are invested over time.

Knowledge Capital of Drug Developers Finally, we examine built-up knowledge capital as an underlying mechanism, that is, whether firms with greater accumulated knowledge in the ICD area of a delayed drug are less likely to suspend the project when faced with a delay. As Krieger, Li, and Thakor (2022) show, firms' "commercialization capital"—including investments in manufacturing and distribution centers in the supply chain, as well as knowledge gained through postmarketing research—can significantly influence R&D decisions and create path dependencies, even following negative product pipeline shocks.

To test this potential mechanism, we proxy for firms' existing knowledge capital by the number of previously initiated drug projects in the same ICD category as the focal project. Panel B of Table 6 presents the results. Column (1) uses the count of prior projects, whereas column (2) uses an indicator variable for whether any such prior projects exists. Across both specifications, the interaction term between delay and knowledge capital is statistically insignificant and small in magnitude, indicating that firms with more built-up knowledge in a given area are not significantly less likely to suspend delayed projects. This implies that accumulated

[28] Internet Appendix Table IA.14 shows robustness to alternative financial constraint measures, including the Hadlock and Pierce (2010) (HP) and Kaplan and Zingales (1997) (KZ) indices, as well as to constraint measures defined at the time of trial start to capture ex-ante proneness to financial constraints. Across all specifications and constraint definitions, we continue to find a strong, positive effect of trial delay on subsequent continuation probabilities.

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knowledge capital is unlikely to explain the excess continuation behavior we document. At the same time, the coefficient on prior knowledge capital is negative, consistent with a general effect on project continuation irrespective of delay, though we cannot statistically reject the null. Table IA.15 in the Internet Appendix confirms these findings using alternative proxies for firms' existing knowledge capital.

6.2 CEO-related channels

Next, we examine CEO-related mechanisms. Specifically, we explore how the effect of drug delays on suspension decisions varies with the CEO's personal stake in the project and the firm. We focus on two dimensions: the CEO's pay sensitivity to stock price movements and their personal responsibility for the project, proxied by whether they were in charge of the firm at the time of trial initiation.

CEO Pay Sensitivity to Firm Value Changes We first test whether CEO pay-for-performance sensitivity (delta) mediates the relationship between trial delays and continuation decisions.[29] Intuitively, CEOs with higher delta and more nonlinear compensation contracts often have stronger incentives to avoid sending negative signals to the market, as adverse reactions more directly affect their personal wealth. Consistent with this, column (1) of Table 7 finds that the effect of trial delay on the decision to suspend versus continue the project is substantially more pronounced among CEOs with greater exposure to stock price changes. A one standard deviation increase in CEO delta is associated with a 41% larger effect of delay on project continuation.

CEO Changes Second, we examine how firms' project continuation decisions following trial delays vary with CEOs' personal responsibility for having initiated the project. In the context of M&A, Guenzel (2025) shows that managers exhibit sunk-cost thinking toward acquired targets, distorting their subsequent decisions to abandon acquired businesses through divestiture. Notably, Guenzel (2025) finds that sunk cost effects are concentrated among CEOs who made the initial acquisition.

To test for a similar CEO effect in our setting, we estimate the relationship between delay and trial continuation separately for two subsamples: the subsample where a new CEO was appointed between trial

[29] We obtain the CEO delta data from Lalitha Naveen's website at https://sites.temple.edu/lnaveen/data/ (Coles, Daniel, and Naveen 2006; Core and Guay 2002). We use the natural logarithm of delta in the analysis in Table 7 as in Coles, Daniel, and Naveen (2006), and standardize the variable to have a mean of zero and standard deviation of one for ease of interpretation. Our findings below are similar when using alternative measures, including the scaled wealth-performance sensitivity measure by Edmans, Gabaix, and Landier (2009).

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Table 7
CEO-related channels

	Dependent Variable: *Suspension*		
	Full CEO Sample (1)	New CEO Subsample (2)	Project-Initiating CEO Subsample (3)
Delay in Trial Completion	−0.222***	−0.188***	−0.359***
	(−4.42)	(−3.36)	(−3.12)
CEO Delta	0.423		
	(0.27)		
Delay × *CEO Delta*	−0.092**		
	(−2.36)		
p-value for difference of coefficients on *Delay*, columns (2) vs. (3): 0.057			
Controls	Y	Y	Y
Year × Quarter FE	Y	Y	Y
Firm FE	Y	Y	Y
ICD FE	Y	Y	Y
Trial Phase FE	Y	Y	Y
Observations	2,913	1,207	2,143
Adj. *R*-squared	0.2590	0.2226	0.3079

This table reports OLS estimates examining underlying CEO-related channels. The unit of observation is a drug project. The sample includes all projects that completed Phase I or II trials with detailed clinical trials records. The dependent variable, $Suspension_i$, is an indicator variable (multiplied by 100 for ease of exposition) for whether project i was suspended after completing Phase I or II trials. The key independent variable, $DelayinTrialCompletion_i$, measures the gap in months between the realized and expected trial completion dates. *CEO Delta* is the natural logarithm of delta as in Coles, Daniel, and Naveen (2006), standardized to have a mean of zero and standard deviation of one for expositional purposes. Fixed effects are reported in the bottom rows. Control variables include the log number of trial sites, number of enrolled participants (in hundreds), log number of projects in the company's pipeline, and log number of competing projects in the same ICD category. Standard errors are two-way clustered at the ICD category and year-by-quarter levels, and *t*-statistics are reported in parentheses. $^{*}p<.1$; $^{**}p<.05$; $^{***}p<.01$.

phase start and completion (column (2)) and the subsample where the CEO remained the same between trial phase start and completion (column (3) of Table 7). The estimated commitment effects are substantially more pronounced when the CEO at trial end, when projects may be advanced or discontinued, is the same as the CEO at trial start, consistent with a personal responsibility channel. Specifically, the effect of unexpected trial delays on subsequent continuation is roughly twice as large in the no-CEO-change subsample, with the difference statistically significant ($p=$ 0.057).

Additionally, we explore CEO age as a possible mediating factor within the project-initiating CEO subsample. We find no statistically significant difference in the effect of delay sensitivity between younger CEOs—who have longer career horizons—and older CEOs closer to retirement. This pattern is consistent with the managerial responsibility channel in Table 7 reflecting not only externally oriented career or reputation concerns (which are expected to be stronger for younger CEOs; Fama [1980]; Holmström [1982]; Gibbons and Murphy [1992]), but also internally driven, psychological motives, such as cognitive dissonance avoidance (Festinger 1962) or aversion to realizing losses (Kahneman and Tversky 1979).

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Overall, the results in Table 7 support the role of management-induced frictions as a key underlying mechanism for the observed delay-induced (excess) commitment to R&D projects. These heterogeneity results also help to further alleviate concerns about omitted variables related to project quality or about issues related to sample selection.

7. Implications

In the final part of the paper, we examine how delay-induced commitment to drug projects affects firms' broader investment behavior. We conclude with a brief discussion of potential externalities for consumers, highlighting promising directions for future research.

7.1 Implications for firm investment

Building on the discussion of efficiency implications in Section 1, we examine the broader consequences of delay-induced project commitment for firms by testing for spillover effects on other investments, specifically, investment crowding out. As discussed in Section 1, such crowding out would imply direct efficiency losses from the firm's private perspective whenever the crowded-out projects are of a higher NPV than the continued, delayed project.

Concretely, we construct three crowding-out-related outcome variables: (i) the total number of new projects (in logs) initiated by a company within 1 year of completing the focal project phase, (ii) the number of new projects that share the same ICD indication as the focal project, and (iii) the number of new projects that do not share the same ICD indication. As before, projects are defined at the drug-by-indication level, and we include all newly initiated projects with development status of "Discovery" or "Preclinical."

Table 8 presents the results for the effect of trial delays on investment crowding out. Specifically, following the (better-identified) IV strategy from Table 3, we relate instrumented delays, driven by changes in trial site congestion, to the rate of new project initiation. The table reports the second-stage estimation results, with the first-stage Kleibergen and Paap (2006) F-statistics for the instrument shown at the bottom.[30] As columns (1) and (2) show, congestion-induced trial completion delays significantly reduce the number of newly initiated projects. A 1-year instrumented delay is associated with approximately 1 fewer new project started, relative to a median of 4 new projects. The remaining columns show that this effect is concentrated among new projects targeting other diseases (i.e., drugs with

[30] The sample size is somewhat reduced compared to Table 3, as this test requires 1-year forward-looking information on each project, resulting in the loss of some observations at the end of our sample period.

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Table 8
Effects on other firm investments

	Dependent Variable: *Project Initiation* Second Stage					
	All Projects		Same-ICD Projects		Different-ICD Projects	
	(1)	(2)	(3)	(4)	(5)	(6)
Delay in Trial Completion	−0.011***	−0.011***	0.002	0.002	−0.011***	−0.011***
	(−3.39)	(−3.24)	(1.02)	(0.85)	(−3.35)	(−3.24)
Controls	N	Y	N	Y	N	Y
Year × Quarter FE	Y	Y	Y	Y	Y	Y
Firm FE	Y	Y	Y	Y	Y	Y
ICD FE	Y	Y	Y	Y	Y	Y
Trial Phase FE	Y	Y	Y	Y	Y	Y
First-stage *F*-stat	26.88	25.83	26.88	25.83	26.88	25.83
Observations	7,838	7,636	7,838	7,636	7,838	7,636

This table reports IV estimates examining effects on new drug project initiation, using changes in trial site congestion as an instrument for trial completion delays. The unit of observation is a drug project. The sample includes all projects that completed Phase I or II trials with detailed clinical trials records. The second-stage dependent variable is the total number of new projects (in logs) initiated by the company within 1 year of completing the focal project (i.e., the project included in the main sample). New projects are defined as a drug by indication, and include newly initiated drug projects with development status "Discovery" or "Preclinical." Columns (1) and (2) consider all new projects, while columns (3) to (4) (columns (5) to (6)) calculate the total number of new projects that share (do not share) the same ICD indication as the focal project. The key independent variable, $DelayinTrialCompletion_i$, measures the gap in months between the realized and expected trial completion dates. The instrument, $TrialSiteCongestion_i$, is the normalized, zip-code-level change in the number of participants in the universe of trials on ClinicalTrials.gov between trial start and end date, taking the maximum across all trial sites of a given trial in the sample. Fixed effects are reported in the bottom rows. Control variables include the log number of trial sites, number of enrolled participants (in hundreds), log number of projects in the company's pipeline, and log number of competing projects in the same ICD category. Standard errors are two-way clustered at the ICD category and year-by-quarter levels, and *t*-statistics are reported in parentheses. $^{*}p<.1$; $^{**}p<.05$; $^{***}p<.01$.

nonoverlapping ICD indications), suggesting that crowding out primarily occurs with respect to investment in alternative fields.[31]

As discussed in detail in Section 1 and reiterated above, the documented crowding out effects imply efficiency costs for firms whenever the crowded-out projects have higher NPV than the continued, delayed projects. Empirically, however, crowded-out projects are unobserved, as they are never initiated, and we lack data on "considered but not executed" projects. This makes a comprehensive empirical assessment of efficiency costs inherently difficult.

Internet Appendix Table IA.16 provides one data point suggestive of potential inefficiencies. Conditional on project continuation, delayed projects have lower projected sales than nondelayed projects. Of course, projected sales differ from NPV (though they are our best available proxy in terms of projecting cash flows), and a precise counterfactual comparison

[31] While our tests in Section 6.1 do not support a primary role for knowledge capital in explaining our main findings, the cross-ICD pattern in Table 8 is broadly consistent with such a form of path dependence, as delays appear to shift firms toward continuing in familiar therapeutic areas over exploring new ones. Over time, crowding out spillovers to other new drug development may not only negatively affect firm profits but also the range of treatment options available to consumers.

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would involve the unobserved, crowded-out projects. Nonetheless, this pattern suggests that delay-induced continuation leads firms to pursue lower-value projects, making it more likely that the forgone projects would have been more promising.[32]

To complement this suggestive evidence, we also conduct a market-based test aimed at shedding light on the extent of value creation or destruction from implementing projects comparable to those crowded out in our setting. Specifically, we examine stock market announcement returns for a random sample of 100 newly initiated projects when existing projects are not delayed. The logic for focusing on this sample is that these projects are the closest observable analogue to the type of projects that are foregone when delay induces crowding out. The logic for the event study approach is that investors' reactions to new project announcements capture the full NPV effect—including indirect effects on other projects, for instance through competition over resources—and thereby provide a benchmark for the value of projects that firms forego when delay induces crowding out. We find announcement returns of roughly 2% in the [−1,+3] and [−1,+5] event windows (Table IA.17 in the Internet Appendix), consistent with the interpretation that such projects tend to create value. While this approach necessarily rests on the assumption that crowded-out projects are on average similar to projects initiated in the absence of delay, and entail similar competition for resources, the results provide additional suggestive evidence of efficiency costs from project crowding out.

7.2 Consumer externalities

While our analysis focuses on firms' investment behavior and efficiency implications, we close with a brief discussion of potential consumer externalities. These externalities are particularly relevant in our context, where the investment decisions we study directly shape both the availability and quality of new therapies. Broadly, our findings suggest that excess commitment to R&D not only affects firms' internal capital allocation but also influences the pace and composition of innovation reaching patients.

In principle, we have data on several consumer outcomes—such as postapproval adverse events and measures of drug novelty—but we refrain from analyzing these outcomes or conducting a formal welfare assessment for two main reasons. First, welfare effects are difficult to infer from these measures alone: for example, a drug with frequent adverse events may

[32] We note and reiterate three additional points. First, while the estimates in Internet Appendix Table IA.16 are economically meaningful, they are statistically marginal. Second, the conceptual framework in Section 1 clarifies and predicts that additional efficiency losses would result, even without crowding out, from some of the lower-value projects that are continued following delays being negative-NPV. Third, the results in Table IA.16 should not be conflated with the unconditional patterns in Table IA.6. Taken together, the findings are fully consistent with delays being uncorrelated with project quality unconditionally and firms selectively advancing projects based on both project quality and delays.

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still be life-saving for many patients. Second, a full welfare analysis would require richer information, such as data on pricing and market structure, since these and other factors shape how the benefits of innovation are distributed.

Overall, the consumer welfare implications of excess R&D commitment are likely nuanced, heterogeneous, and context dependent, highlighting the importance of future work that comprehensively connects firm-level decision-making to downstream consumer outcomes.

8. Conclusion

This paper examines how firms' commitment to ongoing R&D projects is influenced by the temporal (and financial) resources already invested, and explores the resulting broader implications. Using project-level data from the pharmaceutical industry, we show that firms are more likely to continue drug projects following unanticipated delays, despite delays being empirically unrelated to project quality. These effects are economically meaningful and are more pronounced when the CEO who initiated the project remains in place or has stronger pay-performance incentives, consistent with managerial commitment or sunk-cost motives. Delay-induced commitment appears to crowd out the initiation of new drug projects, potentially limiting future treatment options, with additional tests suggesting resulting efficiency losses for firms.

Our findings suggest several avenues for future research. One promising direction is to explore heterogeneity in excess R&D commitment across organizational settings—for example, how such behavior varies with decision-making structures (e.g., centralized vs. decentralized) or organizational cultures (e.g., tolerance for failure). Another important direction concerns the consumer implications of excess commitment. While our analysis focuses on firm-level efficiency, excess R&D commitment inevitably has downstream consequences for consumers by shaping the availability, quality, and pricing of new therapies. Assessing these welfare effects, however, would require richer data beyond the variables available for this study, including, at a minimum, comprehensive information on drug launches, postlaunch adverse events, market structure, competition, and pricing, together with a framework to translate clinical outcomes into welfare. Finally, an important open question is how competitors respond to firms' excess commitment to R&D projects and the resulting general equilibrium effects.

More broadly, while our setting focuses on clinical trials, we view excess commitment as a general phenomenon not confined to drug development or even R&D. In any environment with path-dependent investment and uncertainty, prior investments can affect the likelihood of continuation even when doing so is not optimal. Such dynamics may also arise in

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industries where projects are less capital-intensive but unfold over time, suggesting that the mechanism we document could shape firm behavior across a wide range of contexts.

The replication code is available in the Harvard Dataverse at https://doi.org/10.7910/DVN/30WFIH.

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