Appendix:

If you had only one shot: Scale and herding in innovation experiments

A A Case Study on Approaches to Experimentation: Psoriasis

Psoriasis[17] is a chronic inflammatory skin condition affecting approximately 2-3% of the global population (Ayala-Fontanez et al., 2016). A common symptom of the disease is the appearance of raised, silvery plaques (Nestle et al., 2009). Although treatments for psoriasis have been available for decades, the disease has many forms, and the causes of some less common types are still not fully understood (Guo et al., 2023). Many hypotheses exist, and some examples include: (i) overactive T-cells triggering inflammation and rapid skin cell production, (ii) genetic factors, such as mutations in the HLA-Cw6 gene, (iii) involvement of cytokines like IL-23 and IL-17, (iv) inflammatory lipid molecules called leukotrienes, and (v) abnormalities in keratinocytes that result in excessive skin cell production. This section describes examples of firms initiating pre-clinical trials for two distinct psoriasis drug development projects within the same year. These examples are sourced directly from Pharmaprojects and supplemented with details from Trialtrove.

Astrazeneca. In 2004, AstraZeneca began preclinical trials for two psoriasis treatments. One was a humanized antibody called Sifalimumab, which targeted interferon-alpha (IFN-α). The underlying hypothesis was that in genetically predisposed individuals, the immune system is primed, and exogenous IFN-α may trigger psoriasis development.

At the same time, pre-clinical trials began for Certolizumab pegol, a recombinant humanized high-affinity anti-TNFalpha antibody fragment, developed by UCB (Celltech before the acquisition), for the treatment of chronic inflammatory conditions, including Crohn's disease (CD), rheumatoid arthritis (RA), psoriatic arthritis and ankylosing spondylitis

[17] https://www.niams.nih.gov/health-topics/psoriasis

The anti-TNF alpha psoriasis hypothesis suggests blocking Tumor Necrosis Factor-alpha (TNF-alpha). It has since been shown, however, that this approach can lead to the development or worsening of psoriasis, primarily due to an uncontrolled increase in type 1 interferons produced by plasmacytoid dendritic cells (pDCs), which are key players in psoriasis pathogenesis.

Stiefel Laboratories (GlaxoSmithKline). In 2006, Stiefel Laboratories began pre-clinical trials for two psoriasis drugs. One was called Primolux, which was a 0.05% topical formulation of the corticosteroid clobetasol, developed using its proprietary VersaFoam-EF technology, that targeted the nuclear receptor subfamily 3 group C member 1.

The second drug, Calcipotriol VersaFoam, was a vitamin D receptor antagonist. The treatment consisted of a 0.005% topical formulation of calcipotriol, a vitamin D3 analog.

In 2009, GlaxoSmithKline acquired Stiefel Laboratories for $2.9 billion to create a specialists dermatogolgy branch.[18] Consequently, in Pharmaprojects these projects are described as being originated by GlaxoSmithKline. This example highlights the complications in measurement. Pharmaprojects assigns the "company developing a drug" to each drug–treatment. Importantly, this would suggest that the listed focal company is both funding development and the primary decision maker.

Identifying the impact of such measurement error is hard to do. In the example of Stieffel laboratories, this measurement error would not affect ecometric estimates in our core results, at least. We would still consider the experiments to be done by a multi-experiment firm. If, however, we are on average more likely to incorrectly assign multi-experiment status to a large incumbent such as GlaxoSmithKline, then our analysis in Section 6.4 may inaccurately estimate the relationship between ownership type, the scale of experimentation, and the introduction of novelty (Figure 4 panels

[18] https://www.gsk.com/en-gb/media/press-releases/glaxosmithkline-completes-acquisition-of-stiefel

(a), (b), and (d))

However, measurement error in general would create an attenuation bias. In particular, if measurement error is also correlated with the target diversity dependent variable. If firm X acquired two single experimenters, each with a distinct approach, and both experiments are attributed to firm X, then this would be a positive correlated measurement error leading to an upward bias on the estimated coefficient. Given our data, it is not feasible to scrutinize the history of each drug development project and the accuracy of originator firm assignment. This should be recognised as an empirical limitation of this study.

B Model Extensions

While simulations are best suited to generalizing our model, we can formally extend the model to analyze the decision of the n^{th} experiment after $n-1$ experiments in approach a. For simplicity, we set $\pi_a = \pi_b = \pi$. Instead, approach a is assumed to be more promising than b because $p_a > p_b$ for all experimenters.

In addition, we relax our rent-sharing assumption. In the baseline model in the two-firm case, we assume that if both experiments are successful, only one experiment will capture value (with probability 0.50). In this extension, we consider the scenario where the probabilities of value capture can be lower. Concretely, this permits the scenario where two firms are successful, and they can both commercialize their innovations, but due to competition, the value they capture is less than $V/2$. We model rent dissipation with the parameter k to analyze the effect of rent dissipation on the choice of approach.

Suppose there are n firms and n experiments in approach a and 0 experiments in b, such that each firm is a single-experiment firm. A potential entrant in approach a has a payoff of:

$$\begin{aligned} & p_a\pi\left((1-p_a)^n + \frac{1}{2+k}\binom{n}{1}a(1-p_a)^{n-1} + \frac{1}{3+k}\binom{n}{2}(p_a)^2(1-p_a)^{n-2}...\frac{1}{n+1+k}(p_a)^n\right) - c \\ & = p_a\pi X(n,k) - c \end{aligned} \tag{4}$$

Note that $X(n,k)$ is strictly less than unity, and decreases with the rent dissipation parameter k, because each of the terms $\frac{1}{n+1_k}p_a^n$ decreases with k. Note also that $X(n,k) - (1-p_a)X(n-1,k)$ is positive but falls with k. Formally,

$$\begin{aligned} & X(n,k) - (1-p_a)X(n-1,k) = \\ & \frac{1}{2+k}\left(\binom{n}{1} - \binom{n-1}{1}\right)a(1-p_a)^{n-1} + \frac{1}{3+k}\left(\binom{n}{2} - \binom{n-1}{2}\right)p_a^2(1-p_a)^{n-2} \\ & .. + \frac{1}{n+k}(p_a)^n > 0 \end{aligned} \tag{5}$$

That $X(n,k) - (1-p_a)X(n-1,k)$ decreases with k follows upon noting that each terms, $\frac{1}{r+k}\left(\binom{n}{r-1} - \binom{n-1}{r-1}\right) p_a^{r-1}(1-p_a)^{n-1-(r-1)}$, decreases with k.

B.1 Incentives to herd and scale of experimentation

Entering b instead has a payoff of:

$$p_b\pi\left(\pi X + (1-\pi)\right) = p_b\pi^2 X(n,k) + p_b\pi(1-\pi) - c \tag{6}$$

The incentive to herd into approach a for the single-experiment entrant is given by:

$$\Delta_s = \pi(p_a - \pi p_b)X(n,k) + \pi(1-\pi p_b) \tag{7}$$

Now consider a potential multi-experiment firm, which has one experiment in a along with $n-1$ other firms i.e., an incumbent. The payoff to this firm of a second experiment in a is:

$$\begin{aligned} &\pi(p_a(2-p_a)\left((1-p_a)^{n-1} + \frac{1}{2+k}\binom{n-1}{1}p_a(1-p_a)^{n-2} + ...\frac{1}{n+k}p_a^{n-1}\right) \\ &= \pi X(n-1,k)p_a(2-p_a) - c \end{aligned} \tag{8}$$

And their payoff from entering with approach b is given by:

$$\pi X(n-1,k)(a + \pi(1-a)b) + b\pi(1-\pi) - c \tag{9}$$

Thus the incentive to herd in approach a for the incumbent firm can be expressed:

$$\Delta_M = \pi(1-p_a)X(n-1,k)(p_a - \pi p_b) + \pi(1-\pi)p_b \tag{10}$$

It follows that $\Delta = \Delta_s - \Delta_M = \pi(1-\pi p_b)\left[X(n,k) - (1-p_a)X(n-1,k)\right] > 0$. That is, if the $(n+1)^{th}$ experiment is conducted by a new entrant, they will be more likely to

experiment with approach a compared to a multi-experiment firm who is incumbent with an existing experiment in approach a. Furthermore, Δ falls with k because $X(n,k) - (1-p_a)X(n-1,k)$ falls with k. **That is, small-scale experimenters are more likely to herd than large-scale experimenters, but less so when rents are dissipated.**

C Simulation

C.1 Logic

In the model developed in Section 4, there are two important assumptions underlying its basic intuition. First, the total value that can be captured by successful firms is independent of the number of successful firms - meaning the total profit from successful experiments by independent firms stays constant. Specifically, a firm with two successful experiments earns the same payoff as if it had only one successful experiment. This implies that competition among successful firms does not dissipate rents but merely distributes the total payoff in some fashion. The second assumption is that the validity of approaches is uncertain. Firms choosing the same approach are more likely to succeed or fail together than firms choosing different approaches, meaning value diversion is more likely when firms choose the same approach. In other words, firms choosing the same approach have correlated outcomes, whose effect is not fully integrated into individual firms' decisions. However, this correlation arises only if $\pi \neq 1$; if $\pi = 1$, the probability of success depends solely on implementation. Thus, firms will choose the approach with which they have the highest probability of implementation success. Target diversity at the market level would simply reflect differences in implementation ability for different approaches.

Generalizing beyond two approaches and two possible experiments to multiple approaches and multiple firms involves considerations of competition and order of entry into experimentation. While this is not analytically tractable, simulations permit us to explore the robustness of the model's intuition. We use Monte Carlo simulations to generalize the results of our model. In the baseline simulation, we define a pool of 50 firms, of which $\phi \in [0, 1]$ have a high cost of experimenting (and $1 - \phi$ incur a low cost). In each period, a firm is randomly selected and can choose to experiment. Firms experiment with the approach that returns the largest non-negative expected

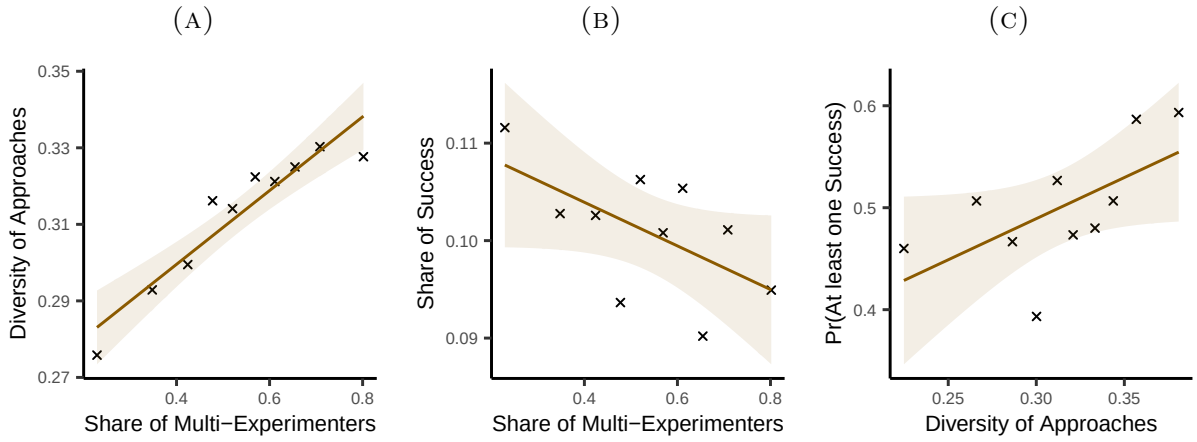
payoff and do not enter if all expected payoffs are negative. This payoff depends on the ongoing experiments and whether the firm has an ongoing experiment. Once a firm has entered twice we remove them from the pool of firms. We run simulations for different shares ϕ to exogenously create variation in the share of firms that enter with two experiments (a higher share of high-cost firms leads to fewer firms entering with two experiments). A simulation ends when the first low-cost firm chooses not to enter since this indicates that there will be no more entry (high-cost firms are less likely to enter than low-cost firms, given their higher cost). We run 400 simulations for $\phi = \{0, 0.1, 0.2, 0.3, 0.4, 0.5, 0.6, 0.7, 0.8, 0.9, 1\}$

Figure C.1 presents results from the baseline simulation, where we simulate firms choosing between two approaches, a and b, where $\pi_a < \pi_b$. Firms continue to enter until they become unprofitable with either approach. In panel C.1a, we link the market-level share of multi-experiment firms to the diversity of approaches used. Maximum diversity would be an even split between approach a and b, with target diversity defined as one minus the share of experiments using approach a. Our simulations support the analytical prediction that greater approach diversity occurs when firms conduct multiple experiments on average. In panels C.1b and C.1c, we show results from simulations predicted by Propositions 2 and 3. Markets with a greater share of multi-experiment firms have a lower individual experiment success rate. However, in markets with greater approach diversity—associated with more multi-experiment firms—we observe a higher probability of at least one experiment succeeding.

C.2 Extension: Discovery of new Approaches

A feature of our empirical context is that new approaches are discovered. Additionally, in practice, more than two approaches will be available to the experimenter. In our empirical context, for example, the average number of targets observed in a therapeutic class–year is 9.09 (median=5, std.dev.=14.21, min=2, max=210. Here, we extend our

FIGURE C.1: SIMULATION RESULTS



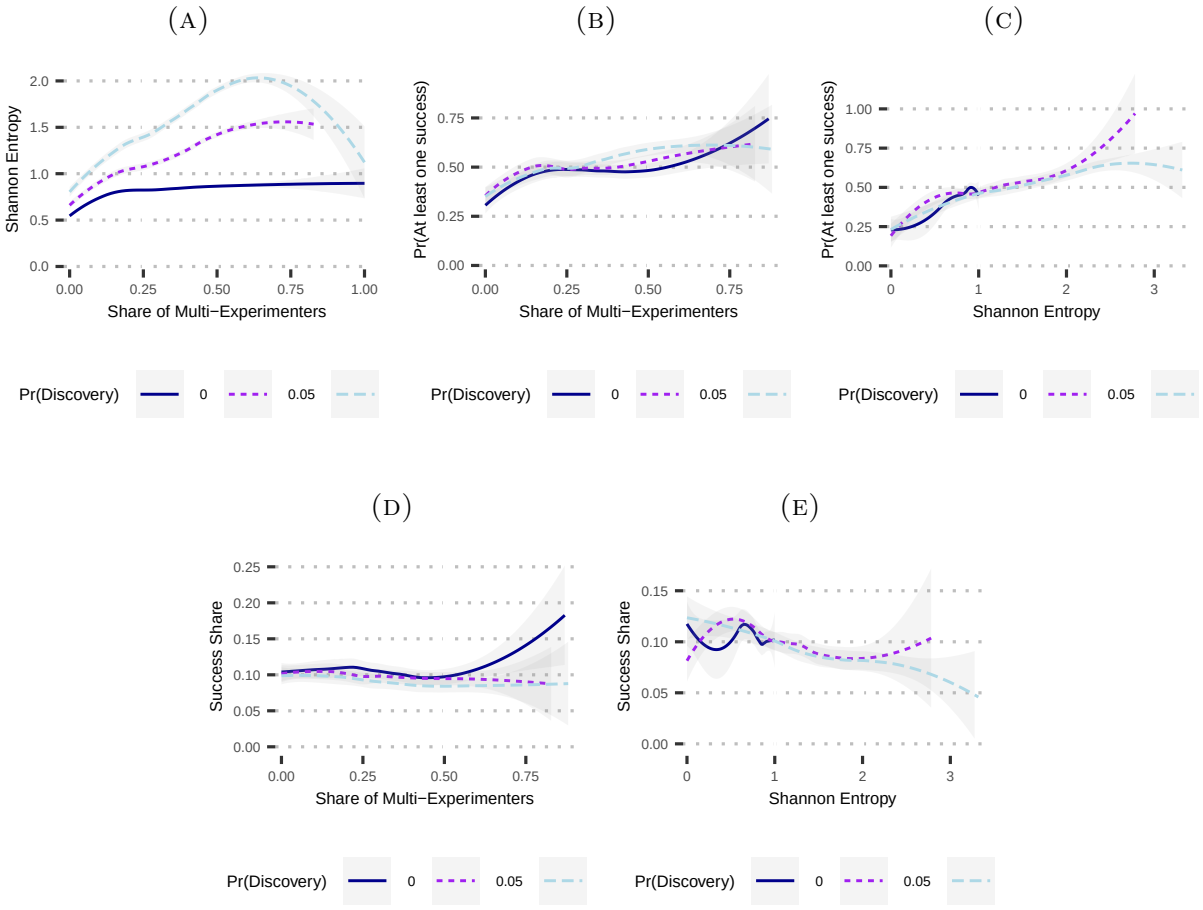
Notes: These figures report binned scatter plots of our simulation results, illustrating the relationships predicted by each proposition. We simulate 1500 markets, creating variation in the share of large-scale experimenters by exogenously shifting the share of firms that have a high cost of experimenting. A detailed explanation of the simulation logic is provided in Appendix C. This simulation uses the following parameters: $\pi_a = 0.4$, $\pi_b = 0.2$, $p_a = p_b = 0.3$.

simulation framework to allow for discovery and, thus, multiple approaches.

The logic for these simulations is identical to that deribed in the baseline simulations, but we make the following changes to introduce discovery. In each period, a new approach is discovered with probability γ. In the case of discovery, a new entrant enters with the approach, and the approach enters the domain of available approaches that subsequent experiments can choose from. We keep all parameter values the same as the baseline simulation (i.e., $\pi_a = 0.4; \pi_b = 0.2; p_a = p_b = 0.3$). All new approaches k have a lower viability probability $\pi_k = 0.1$, but the same implementation probability $p_k = 0.3$ as existing approaches a and b. We assume that only new entrants discover new targets and that this is their only experiment. This is to match our empirical finding, that the introduction of new approaches is more likely to happen with single-experiment firms. Keeping all other parameters constant, we vary the probability of discovery and observe the effect of discovery on the diversity of targets and success. We run 400 simulations for each combination of high-cost experimenter share $\phi = \{0, 0.2, 0.4, 0.6, 0.8, 1\}$ and discovery probability $\gamma = \{0, 0.05, 0.1\}$.

In Figure C.2, we report simulation results grouped by each discovery probability.

FIGURE C.2: Simulating the Effect of Discovery on the Diversity of Approaches and the Outcomes of Experiments



In each plot we show binned scatter plots, with 50 bins. We measure the diversity of approaches with Shannon entropy, consistent with our empirical method. In panel (a), our simulations show that the probability of discovery positively moderates the relationship between the share of multi-experimenters and the diversity of approaches. In other words, for a fixed share of multi-experiment firms, when the likelihood of a new approach being discovered is higher, our simulations predict a greater diversity of approaches.

Turning to outcomes, the results are less clear-cut. In panels (b) and (c), we look at the probability of at least one success, and in panels (d) and (e), we look at the average success rate. In panel (b), the simulations suggest a small benefit from discovery on

the probability of at least one success. This is consistent with our previous results: a higher discovery rate is associated with a greater diversity of approaches, which is associated with a higher probability of the market finding at least one success. Panel (c) is harder to interpret, but our results suggest a declining benefit to higher discovery rates. Holding the diversity of approaches constant, a higher probability of at least one success is achieved when the discovery rate is lower.

Panel (d) also conforms with prior results. The share of successful experiments is lower when a market consists of a greater share of multi-experiment firms, especially when the discovery rate is higher. Discovery leads to new approaches becoming available, but individually, they have a smaller probability of success. Panel (e) suggests results consistent with the intuition of the panel (c). Holding the diversity of approaches constant, the share of experimental successes will be lower when the discovery rate is higher.

D Data

D.1 Description of Variables

Notes: All variables are defined at the therapeutic class–year level (i, t)

Variable	Description
Target Diversity	Shannon entropy of the relative abundance of targets employed in pre-clinical experiments.
Average Experimenter Scale	Average number of pre-clincial experiments started by firms.
Average Experimenter Scale $(t-1)$	Average number of pre-clincial experiments started by firms in the previous year.
Multi-Experiment Share	Share of pre-clincial experiments started by firms who themselves launched two or more experiments.
Multi-Experiment Share $(t-1)$	Share of pre-clincial experiments started by firms in the previous year who themselves launched two or more experiments.
ln(Discovery)	Natural logarithm of the count of publications in GWAS that report a novel target.
Average Firm Age	Average age of distinct experimenters (firms).
At least 1 Pre-Clinical Success	A dummy variable equal to 1 if at least one pre-clinical trial is successful and is followed by a Phase 1 clinical trial; 0 otherwise.
Share of Pre-Clinical Success	Share of pre-clinical trials that are followed by a Phase 1 clinical trial.
At least 1 Drug Launch	A dummy variable equal to 1 if at least one pre-clinical trial is successful and ultimately results in the launch of a drug product, 0 otherwise.
Share of Drug Launch	Share of pre-clinical trials that eventually result in the launch of a drug product.
Project Start Year	Year in which experimentation begins. We define an experiment as the first pre-clinical trial in a drug development project.
Number of Firms	Total number of firms experimenting.
Number of Projects Started	Total number of pre-clinical experiments initiated.
Number of Targets	Total number of targets being drugged in experiments.

D.2 Matching Pharmaprojects to Pitchbook

We match data on firm founding year and ownership from Pitchbook to Pharmaprojects by firm name. Matching by firm name is challenging, because datasets often use different naming conventions. For example, one dataset may list "Eli Lilly & Co.", while the other simply states "Lilly". Here we briefly describe our approach to this inherently noisy matching task and report results from manual validation.

We first filter firms in Pitchbook by their primary industry group, selecting industries that contain the phrases pharmaceuticals, healthcare, drugs, medical, surgical, hospitals, and clinics. We then clean firm names, removing punctuation, special characters, and common legal suffixes, e.g., ltd., inc., co.

Matching on these cleaned firm names produces multiple matches for each firm in Pharmaprojects. For example, we find that Pfizer matches to many subsidiaries or overseas business units. To identify the focal firm in each match, we keep the matched firm with the oldest founding year. If this data is missing, we select the matched firm with the largest number of employees.

Out of 2,804 pharmaceutical firms, we matched 2,150 to Pitchbook. To validate our match, we took a random sample of 50 firms from those that were matched and another random sample of 50 firms from those that were not matched. For those that were matched, we compared the matches across other observable dimensions, such as HQ location. These manual checks revealed one error in the random sample of 50 firms. As for the unmatched firms, we repeated the same process but with the unfiltered Pitchbook data (i.e., not conditioning on pharmaceutical-relevant firms). For the 654 unmatched firms, we found a match for 76 firms. However, we found that only half of these were accurate upon manual inspection.

In sum, our matching approach is highly accurate, but the main limitation is the coverage of the data.

D.3 Identifying ATC-1 groups

In our analyses, we control for unobserved time and industry varying trends using $ATC1 \times Year$ fixed effects (Branstetter et al., 2022). Our data, however, do not include the ATC-1 code to which the therapeutic class belongs. Fortunately, there are only 14 ATC-1 categories (`https://www.who.int/tools/atc-ddd-toolkit/atc-classification`). We exclude ATC P: Antiparasitic products, insecticides and repellents, which leaves 13 codes. In a similar approach to matching our sample to GWAS, we loop through our 200 therapeutic class names and use GPT-4.0 to identify the closest ATC-1 match. We match 139 out of 200 therapeutic class designations to an ATC-1 code. Importantly, the therapeutic classes which we match are those pervasive, as we only lose 268 (out of 2,791) observations when we exclude therapeutic classes that we are unable to match to an ATC-1. We ran all of our analyses including these 268 observations grouped under a pseudonym ATC-1 'X'. Though not reported here, all of these results were very similar to those reported in the main paper and this Appendix.

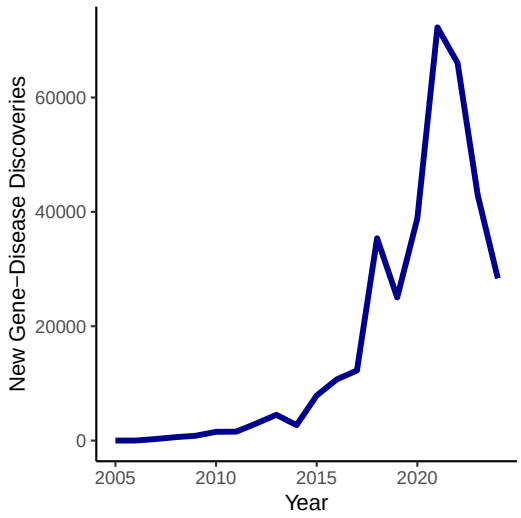
D.4 Matching Pharmaprojects to GWAS

A key feature of our analysis includes controlling for the frequency of discovery. The variable we construct—ln(Discovery)—is the natural logarithm of the number of GWAS publications reporting a new target relevant to therapeutic class i in year t.

Founded in 2008 by the National Human Genome Research Institute, GWAS (`https://www.ebi.ac.uk/gwas`)—or the Catalog of human Genome-Wide Association Studies—is a record of all scientific publications in top-tier journals reporting the discovery of a new target–disease correspondence. An in-depth overview of GWAS is provided by Tranchero (2023).

The GWAS catalog only reports the disease and/or trait that a particular study is concerned with, so to match these data to Pharmaprojects, we need to make a correspondence between disease/traits and therapeutic class. We create two lists of the

Figure D.1: New Gene-Disease Discoveries Over Time



200 distinct therapeutic class names and 28,540 disease names from GWAS. We then wrote a Python script that looped through each disease name and used OpenAI's GPT-4.0 API to identify any and all (at most three) corresponding therapeutic class names. We matched 22,381/28,840 disease names to 179/200 therapeutic classes. Then, for each therapeutic class–year, we count the number of new discoveries in corresponding diseases published in GWAS in that year. As Figure D.1 shows, the coverage of GWAS is considerably weaker for the first three-quarters of our sample period (GWAS was only founded in 2008, after all). Consequently, we have a measure of discovery for 811 out of 2791 market observations. After dropping observations where we don't have an ATC-1 code, this becomes 809.

E Robustness Analysis

Table E.1: Replication of baseline results with unproven targets only

	Target Diversity		Share of Pre-Clinical Success		At least 1 Pre-Clinical Success	
	(1)	(2)	(3)	(4)	(5)	(6)
Average Experimenter Scale	0.879***	0.802***	-0.028***	-0.029***		
	(0.217)	(0.131)	(0.005)	(0.007)		
Target Diversity					0.243***	0.325***
					(0.023)	(0.025)
Market Structure Controls	No	Yes	No	Yes	No	Yes
ATC-1×Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Observations	2,243	2,243	2,243	2,243	2,243	2,243
Adj R-squared	0.139	0.743	0.069	0.069	0.201	0.217

Notes: These table replicate the baseline results from Table 5, where the underlying dataset has been modified to exclude projects using targets which have been targeted by at least one launched drug in the same therapeutic class. all models are estimated with OLS. Robust standard errors clustered at the ATC-1 level are shown in parentheses. Significance codes: * p<.1, ** p<.05, *** p<.01

Table E.2: Replication of key results with market defined in longer time windows

(a) Unit of Observation = Therapeutic Class—Two Year Period

	Target Diversity		Share of Pre-Clinical Success		At least 1 Pre-Clinical Success	
	(1)	(2)	(3)	(4)	(5)	(6)
Average Experimenter Scale	0.730***	0.569***	-0.026***	-0.030***		
	(0.205)	(0.096)	(0.005)	(0.007)		
Target Diversity					0.204***	0.288***
					(0.017)	(0.020)
Market Structure Controls	No	Yes	No	Yes	No	Yes
ATC-1×Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Observations	1,557	1,557	1,557	1,557	1,557	1,557
Adj R-squared	0.148	0.741	0.070	0.069	0.237	0.259

(b) Unit of Observation = Therapeutic Class—Five Year Period

	Target Diversity		Share of Pre-Clinical Success		At least 1 Pre-Clinical Success	
	(1)	(2)	(3)	(4)	(5)	(6)
Average Experimenter Scale	0.954***	0.484***	0.005	0.008		
	(0.162)	(0.051)	(0.011)	(0.013)		
Target Diversity					0.090***	0.157***
					(0.012)	(0.024)
Market Structure Controls	No	Yes	No	Yes	No	Yes
ATC-1×Year FE	Yes	Yes	Yes	Yes	Yes	Yes
Observations	398	398	398	398	398	398
Adj R-squared	0.233	0.760	0.064	0.058	0.111	0.132

Notes: These table replicate the baseline results from Tables 5, where the underlying dataset has been modified to allow for longer time windows. In the original results, we defined the unit of observation—a market—as a therapeutic class–year. In Panel (a), the unit of observation is a therapeutic class–two year period i.e., 1996-1997 is period 1, 1998-1999 is period 2, and so on. In Panel (b), the unit of observation is a therapeutic class–five year period i.e., 1996-2000 is period 1, 2001-2005 is period 2, and so on. Robust standard errors clustered at the ATC-1 level are shown in parentheses. Significance codes: * p<.1, ** p<.05, *** p<.01

Table E.3: Replication of key results with HHI and Drug Launch

(a) Measuring Diversity with HHI

	HHI		At least 1 Pre-Clinical Success	
	(1)	(2)	(3)	(4)
Average Experimenter Scale	-0.111***	-0.132***		
	(0.021)	(0.012)		
HHI			-1.204***	-1.117***
			(0.066)	(0.073)
Market Structure Controls	No	Yes	No	Yes
ATC-1×Year FE	Yes	Yes	Yes	Yes
Observations	2,503	2,503	2,503	2,503
Adj R-squared	0.112	0.480	0.184	0.198

(b) Measuring Success with Drug Launch

	Share of Drug Launch		At least 1 Drug Launch	
	(1)	(2)	(3)	(4)
Average Experimenter Scale	-0.008***	-0.008*		
	(0.002)	(0.004)		
Target Diversity			0.101***	0.098***
			(0.023)	(0.028)
Market Structure Controls	No	Yes	No	Yes
ATC-1×Year FE	Yes	Yes	Yes	Yes
Observations	2,503	2,503	2,503	2,503
Adj R-squared	0.059	0.059	0.096	0.096

Notes: Panel (a) replicates the baseline results from Table 5, where the variable Target Diversity has been replaced with HHI. A higher HHI value indicates that experiments are concentrated in a handful of approaches, and thus the market is less diverse. Panel (b) replicates the baseline results from Table 5 where we define success as a drug development project ultimately leading to a drug product launch. Robust standard errors clustered at the ATC-1 level are shown in parentheses. Significance codes: * p<.1, ** p<.05, *** p<.01

Table E.4: Two-stage least-squares (2SLS)

	Average Experimenter Scale (t)	Target Diversity
	(1) First Stage	(2) 2SLS
Average Experimenter Scale (t-1)	0.086***	
	(0.019)	
Average Experimenter Scale		1.747**
		(0.599)
Market Structure Controls	Yes	Yes
ATC-1×Year FE	Yes	Yes
Observations	2,362	2,362
Adj R-squared	0.271	0.564
First Stage F-test		39.559

Notes: This table replicate the baseline results from column (2) of Table 5, using lagged experimenter scale as an instrument. Robust standard errors clustered at the ATC-1 level are shown in parentheses. Significance codes: * p<.1, ** p<.05, *** p<.01