Healthcare Innovation Across the Racial Divide: The Interplay of Public and Private Financing*

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Abstract

U.S. pharmaceutical companies invest nearly $100 billion annually in R&D, while the National Institutes of Health (NIH) allocates around $50 billion to biomedical research. Experts argue these investments should reflect public health needs and scientific opportunity, yet systematic evidence on allocation patterns remains limited. Using machine learning, we compile a novel panel dataset linking NIH funding and industry clinical trials across major diseases over two decades to disease-specific characteristics: burden, scientific opportunity, revenue potential, and the race and gender composition of affected populations. We find that both sectors invest more in high-burden diseases, but private firms also prioritize high-revenue conditions. Strikingly, the private sector underinvests in diseases that disproportionately affect Black Americans—even after adjusting for burden, revenue, and opportunity. NIH funding partially offsets this gap but compensates for only about 10% of the dollar shortfall and 80% of the "missing" trials for conditions affecting Black Americans. We identify disease areas where gaps remain largest, offering a road-map for targeted policy action. Our findings highlight NIH's essential—but incomplete—role in correcting racial disparities in biomedical innovation and underscore how recent cuts to NIH and Medicaid risk deepening these inequities.

Classification: Social Sciences, Economic Sciences
Keywords: Biomedical R&D, NIH Funding, Racial Disparities

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1 Introduction

Biomedical research has transformed human health. In the U.S., this progress relies on two complementary innovation engines: the public sector—led by the National Institutes of Health (NIH)—which primarily funds foundational science, and the private sector, which advances drug development through clinical trials and commercialization. In 2023, U.S. pharmaceutical firms invested $96 billion in R&D, yielding 55 new drug approvals [1–5]. The NIH, with a $47.7 billion budget, backed discoveries ranging from HIV therapies to mRNA vaccines. Public investment also stimulates private-sector innovation: 41% of private clinical projects build on NIH-supported work, and every $10 million NIH funding generates an estimated 2.7 additional private patents [6–8]. Yet despite these investments, health disparities persist. Black Americans experience 25% higher morbidity and live 5.6 fewer years than White Americans [9]. This study asks two central questions. First, what are the main drivers of research funding allocations across diseases? Do these drivers differ between the public and private sectors, and how do their investments relate? Second, are resources adequately directed toward diseases disproportionately affecting underserved populations? Does the biomedical innovation system reduce—or reinforce—health inequities? Existing research offers only partial answers. Some studies show that disease burden predicts public funding [10, 11] , but often overlook competing and correlated drivers and do not examine whether private funding responds similarly. Other work finds that private-sector R&D reacts to changes in expected returns, such as those driven by Medicare expansions [12, 13]. However, these studies typically examine one sector and one investment driver at a time, missing interactions across determinants and sectors. Several studies document underinvestment in diseases affecting underrepresented groups, such as women and low-income populations [14–16]. Much of this work focuses on biological, behavioral, or access-based explanations [17–26], while the upstream allocation of R&D has received less attention [27, 28]. Moreover, many analyses rely on cross-sectional data, small samples, or fail to control for correlated investment drivers like scientific feasibility or commercial potential. Simply noting that diseases like sickle cell anemia or lupus receive less funding does not establish misallocation, as these diseases may systematically differ in burden, opportunity, or profitability. A rigorous analysis must account for all major investment drivers to assess disparities accurately. We address this gap by providing the first systematic, integrated, longitudinal analysis of how disease-level characteristics—including racial and gender disparities in burden—shape public and private biomedical investment. Using supervised machine learning [29, 30], we construct a new panel dataset spanning 96 major diseases from 2000 to 2019. These diseases offer comprehensive coverage of the U.S. disease landscape: each is listed by the NIH, maps clearly to ICD-10 codes, and causes more than 40 deaths annually (see *Materials and Methods* and *Supporting Information* for full details on variables, sample, design, and analysis). We measure public-sector activity using NIH research funding by disease-year [31]. Because private firms do not disclose R&D spending at the disease level, we proxy private investment using the number of industry- sponsored clinical trials [32]. For each disease-year, we also collect mortality rates (as a proxy for burden) [33], scientific publication volume (scientific opportunity) [34], and total medical spending by patients and insurers (revenue potential; see Table S1). To quantify disparities, we compute the ratio of mortality rates for Black versus White Americans for each disease-year [35, 36].

We use a similar method to calculate gender-based mortality ratios (see Figs. S1 and S2). We focus on Black Americans and women as key examples of historically underserved groups who represent a large share of the population and may be important targets for equity-focused policy. We report four main findings. First, in the raw data, we find that—conditional on disease burden—private investment favors diseases affecting White Americans, while public funding leans toward those affecting Black Americans. For example, among two diseases with similar mortality, the private sector is more likely to invest in the one affecting more White patients; the public sector in the one affecting more Black patients. Second, using Ordinary Least Squares (OLS) models [37], we estimate the importance of investment drivers across sectors. Both sectors invest more in high-burden diseases, but only the private sector prioritizes high-revenue conditions. Even after adjusting for burden, opportunity, and profitability, private investment is systematically lower for diseases affecting Black Americans. Public funding, in contrast, favors diseases with relatively higher Black mortality and partially offsets the private-sector gap. We find no significant evidence that either sector prioritizes diseases affecting men over those affecting women [38]. Third, we simulate a counterfactual race-neutral world by setting racial mortality ratios to 1 in our model. This exercise quantifies how funding would change if race played no role in investment decisions. We find that, in a race-neutral system, private investment in "Black diseases" would rise by 12%, while public funding would fall by nearly 18%—highlighting NIH's compensatory function. Fourth, we assess the extent to which NIH funding offsets private underinvestment. We find it compensates for only 10% of the dollar shortfall and roughly 80% of the missing clinical trials. We also identify specific disease areas with the largest gaps, offering a road-map for targeted policy action. Our findings suggest that inequities in biomedical R&D may contribute to persistent racial health disparities. In the Discussion, we argue that market forces alone are unlikely to close these gaps and that more targeted policy tools are needed to steer innovation toward underserved populations. Finally, we warn that recent policy shifts may exacerbate these disparities. Cuts to the NIH budget could weaken the public sector's compensatory role, while reductions in Medicaid coverage may further erode private-sector incentives to invest in diseases disproportionately affecting low-income populations.

2 Results

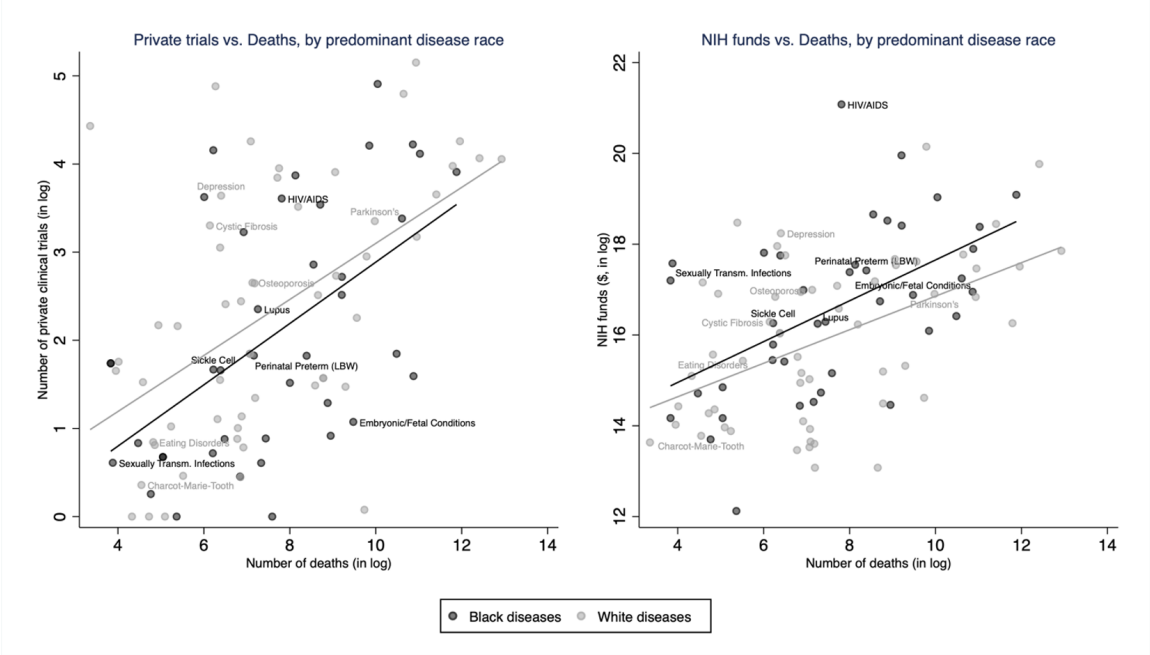
We organize our results in four parts. First, we examine how public and private investments correlate with key disease characteristics. Second, we estimate sector-specific regressions to identify the drivers of research investment. Third, we simulate race-neutral counterfactuals to assess the role of racial disparities in shaping observed allocations. Fourth, we quantify the extent to which NIH funding offsets private-sector underinvestment.

2.1 Descriptive investment patterns: Divergent responses to race

We begin by analyzing raw data to assess whether public and private sector investments are similarly distributed across diseases. Specifically, we compare funding intensity for diseases that disproportionately affect Black versus White Americans, conditional on overall mortality. Figure 1 (and Fig.

S3) shows that both sectors allocate more resources to high-mortality diseases. However, investment patterns diverge sharply by race: private investment tends to favor diseases affecting White Americans, while public funding leans toward diseases affecting Black Americans. This asymmetry holds across the mortality distribution. Figure S4 further illustrates this divergence. Among the bottom 20% of diseases by private funding, 48% disproportionately affect Black Americans (i.e., have a Black-to-White mortality ratio greater than one; see *Materials and Methods*). Among the top 20%, only 35% do. The opposite pattern holds for public sector investment. These patterns motivate a more formal investigation of the drivers underlying public and private investment decisions.

Figure 1: Public and Private Research Investment by Disease Mortality and Racial Disparity.



Notes: This figure plots the relationship between average annual disease-specific mortality and corresponding U.S. research investment from 2000–2019. Public investment is measured by NIH funding (USD); private investment is proxied by the number of industry-sponsored clinical trials. Each point represents a disease, with mortality on the x-axis and investment on the y-axis. Mortality data are cumulative and not disaggregated by race. We estimate linear fits separately for diseases with a Black-to-White mortality ratio above one ("Black diseases") and those below or equal to one. For clarity, we label the six diseases with the highest and lowest racial mortality ratios. Disease classifications follow CDC mortality data.

2.2 Drivers of investment: Formal analysis

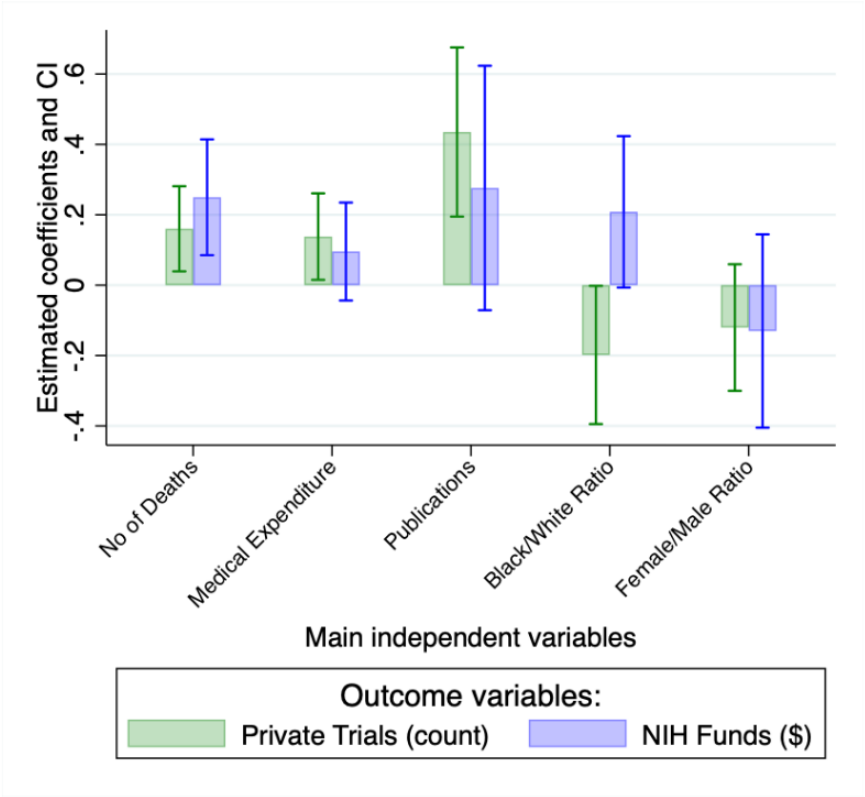
We estimate OLS regressions of investment levels by sector. The dependent variables are NIH funding and the number of privately sponsored clinical trials at the disease-year level. Key explanatory variables (lagged two years) include:

- Mortality (proxy for disease burden)

- Medical spending (proxy for revenue potential)
- Research publications (proxy for scientific opportunity)
- Relative mortality ratios (race and gender disparities)

As shown in Figure 2, both NIH funding and private trials rise with disease burden. A 1% increase in mortality corresponds to a 0.25% increase in NIH funding and a 0.16% increase in private trials. Private investment also responds to revenue potential and scientific opportunity, while NIH funding is largely unresponsive to these commercial indicators. Critically, a one-unit increase in the Black-to-White mortality ratio—about one standard deviation—is associated with a 20% drop in private trials and a 21% increase in NIH funding (see Fig. S5). In contrast, gender-based mortality ratios show no consistent relationship with investment. In other words, conditional on burden, opportunity, and revenue, private firms are less likely to invest in diseases affecting Black Americans, while NIH is more likely to do so. Neither sector appears to systematically prioritize male over female diseases (see *Supporting Information* for full regression tables). In the *Supporting Information* file, we document that these results are robust across specifications, including alternative lag structures, a post-2008 subsample, and models controlling for lagged investment. We also test and reject alternative explanations, such as racial differences in access to clinical trials. Moreover, we rule out confounding by omitted factors such as global demand, foreign investment, philanthropic funding, or logistical complexity of trials.

Figure 2: Drivers of Public and Private Research Investment across Diseases.



Notes: This figure shows estimated associations between key predictors and research investment at the disease-year level. The green bars report effects on private investment (industry-sponsored clinical trials); the blue bars on public investment (NIH funding). All continuous predictors are log-transformed and lagged two years: disease burden (total deaths), revenue potential (medical expenditure), and scientific opportunity (publications). Race and gender are captured by relative mortality ratios (Black-to-White and female-to-male, respectively). Models include year fixed effects and cluster standard errors at the disease level. As a robustness check (see *Supporting Information*), we control for lagged NIH funding when predicting private trials, and for lagged private trials when predicting NIH funding. Error bars represent 95% confidence intervals.

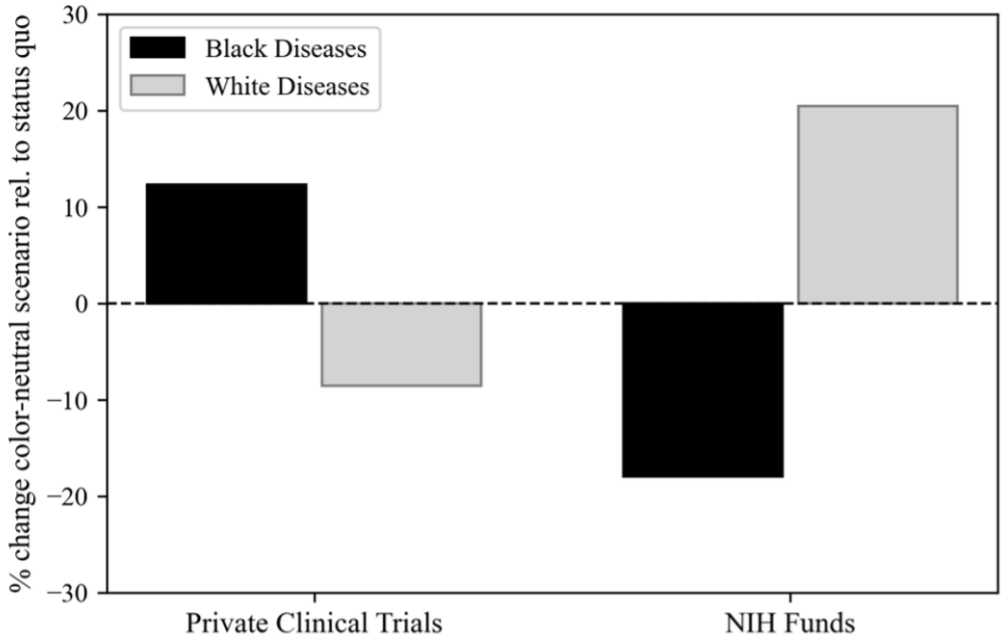
2.3 Simulating a race-neutral investment landscape

To quantify the role of racial disparities, we simulate a race-neutral scenario by setting the Black–White mortality ratio coefficient to zero in our regression models—while holding other parameters constant. We preserve overall NIH funding and trial counts to ensure budget and trial-count neutrality. Figure 3 summarizes the counterfactual allocations. In a race-neutral system:

- Private trials for diseases disproportionately affecting Black Americans would rise by 12.3% annually, while trials for White diseases would fall by 8.5%.
- NIH funding for Black diseases would fall by 17.9%, and funding for White diseases would rise by 20.4%.

These results confirm that the racial composition of affected patients plays a significant role in investment patterns, and that allocations would change materially in the absence of these disparities.

Figure 3: Counterfactual Distribution of Research Investment Under Race-Neutral Allocation.



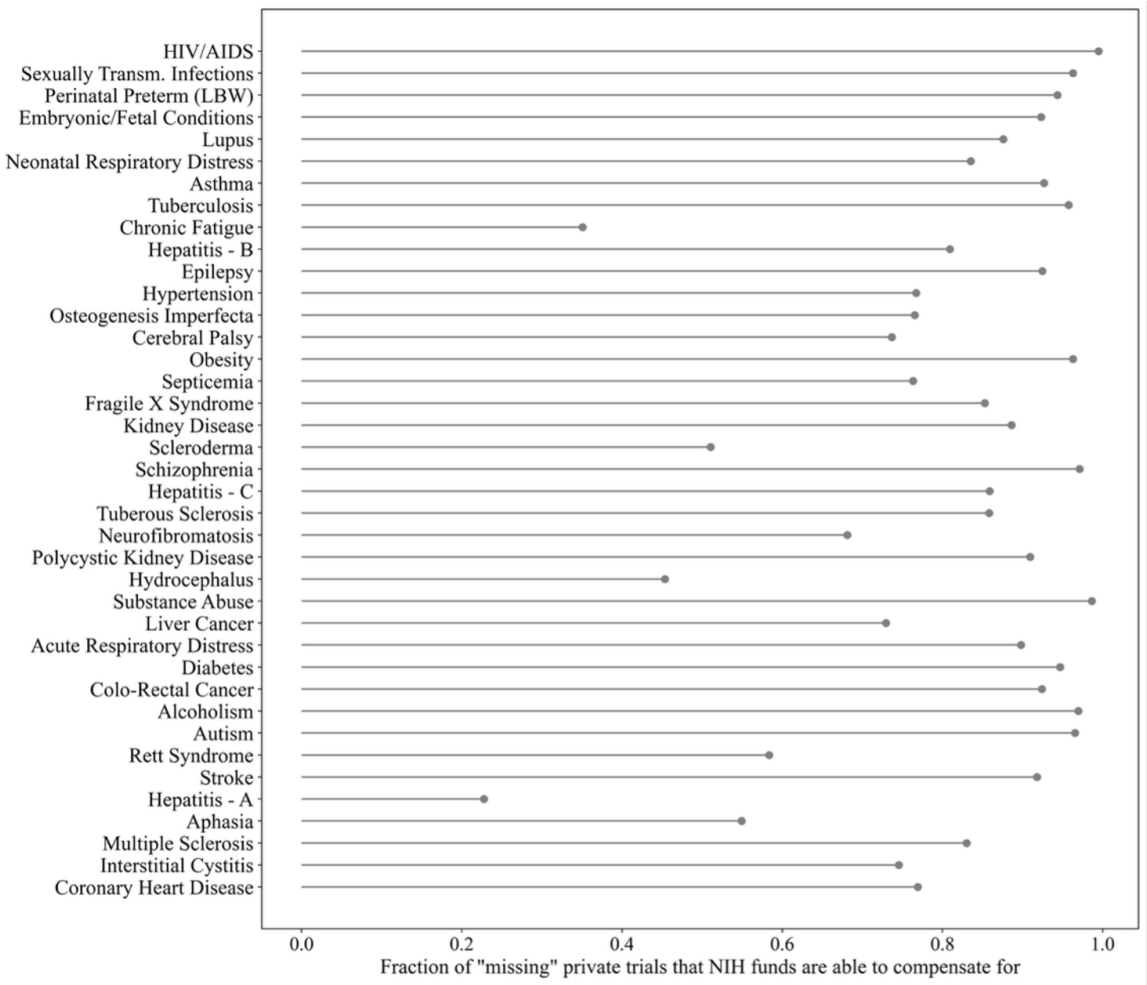
Notes: This figure shows predicted changes in NIH funding and private-sector clinical trials if race-based disparities in investment were removed. We simulate a race-neutral scenario by setting the coefficient on the Black-to-White mortality ratio to zero in our baseline regression models, while holding all other parameters constant. Total NIH spending and the aggregate number of private trials are held fixed to preserve budget and trial count neutrality. Diseases with a Black-to-White mortality ratio > 1 are labeled "Black diseases"; those < 1 are labeled "White diseases." Results highlight shifts in investment that would occur under a race-neutral allocation framework.

2.4 NIH's compensatory role: Incomplete but significant

Given the private sector's lower investment in diseases affecting Black Americans, we assess the extent to which NIH offsets these gaps through two exercises. First, we compare NIH's "excess" funding—defined as actual funding minus predicted funding under the race-neutral scenario—to the dollar value of the private shortfall. To estimate the shortfall, we multiply the number of "missing" private trials (under actual vs. race-neutral scenarios) by the average cost of a clinical trial [1]. We find that NIH offsets only about 10% of the monetary gap in private investment. Second, we estimate the number of future private trials that NIH's excess funding may induce, using our estimated elasticity from Table S10. Figure 4 shows that NIH could offset about 80% of the missing

private trials, on average. However, this varies substantially by disease. For HIV, schizophrenia, and substance use disorders, NIH nearly closes the gap; for hepatitis A and hydrocephalus, it offsets less than half. Together, these results suggest that while NIH plays a vital compensatory role in correcting racial disparities in research investment, it falls short of fully bridging the gap left by the private sector.

Figure 4: NIH Compensation for Private-Sector Shortfalls in Diseases affecting Black Americans.



Notes: This figure quantifies the extent to which NIH funding offsets underinvestment by the private sector in diseases disproportionately affecting Black Americans. For each disease, we calculate "excess" NIH funding—defined as the difference between actual NIH support and predicted funding under a race-neutral scenario. We then predict the number of additional private trials this excess might induce, based on the estimated elasticity between NIH funding and private trial activity. Offset percentages are computed as the ratio of predicted NIH-induced trials to the estimated private-sector shortfall. For example, NIH funding nearly fully offsets the gap for HIV, schizophrenia, and substance use disorders ($\geq 95\%$), but covers less than half the shortfall for conditions like hepatitis A and hydrocephalus.

3 Discussion

3.1 Policy implications

Despite major advances in biomedical research, Black Americans continue to face poorer health outcomes than White Americans—including higher morbidity and shorter life expectancy. Prior studies have pointed to factors such as genetics, behavior, and access to care. Our findings suggest that persistent racial disparities in biomedical R&D investment may also play a consequential role—and that public funding plays a critical, though insufficient, part in addressing these gaps. Market forces alone are unlikely to close the racial investment gap. A back-of-the-envelope calculation shows that achieving race-neutral private-sector investment would require implausible shifts: a 52% increase in mortality, an 18% rise in publications, or a 64% jump in medical spending for diseases affecting Black Americans. Instead, strengthening targeted policy levers—such as extended market exclusivity or expanding the Priority Review Voucher program—could help redirect private R&D toward underserved populations. Recent policy developments risk reversing progress. First, NIH budget cuts may undermine the agency's corrective role. Because cuts are not uniformly distributed, reductions disproportionately affecting diseases that burden minority populations may shrink the very funding most needed to offset market failures [39]. Second, provisions in the One Big Beautiful Bill Act related to Medicaid are projected to increase the number of uninsured Americans by 7.8 million by 2034—roughly 2% of the population [40–42]. This would reduce revenue potential, especially for diseases concentrated in Medicaid populations. Even under limited price changes—or price cuts aimed at reducing drug costs [43]—our model predicts lower private-sector willingness to invest in these diseases. We estimate that, under conservative assumptions, these Medicaid-related changes could further reduce private investment in diseases affecting Black Americans by 0.4% to 1.4% (Fig. S6). This would exacerbate existing disparities and further limit innovation in areas already underserved by market incentives [44]. Taken together, these findings suggest that without sustained public investment and stronger equity-oriented policy tools, racial gaps in biomedical R&D are likely to persist—or worsen.

3.2 Limitations and future research

Our study responds to a notable gap in the literature: the lack of robust, system-wide analysis of biomedical R&D allocation across diseases. Still, it has several limitations. First, while we follow prior literature [45] in using mortality, publication volume, and medical spending as proxies for disease burden, scientific opportunity, and revenue potential, respectively, each is an imperfect measure [46]. Disease burden is multidimensional: should a rare but deadly disease be weighted more heavily than a common but less severe one? Likewise, scientific opportunity is difficult to measure precisely, and clinical trial counts—our proxy for private investment—do not fully capture firms' financial commitments. If measurement errors correlate with disease racial composition, our estimates could be biased. As discussed in [46, 47], every proxy comes with trade-offs, and researchers must operate under inherent data constraints. Second, while we test and rule out omitted variable bias as a major concern (see *Supporting Information*), including additional controls—such as global demand, philan-

thropic support, or logistical barriers to trials— could offer further reassurance. Our study adopts a U.S.-centric lens, appropriate for U.S.-based firms given their pricing power in the domestic market. However, this scope does not capture how diseases affect minorities globally. Third, we document complementary investment patterns by NIH and private firms with respect to race, but we do not take a stand on the origins of this asymmetry. For example, do NIH investments exhibit affirmative action toward diseases affecting Black Americans [48], or is it strategically filling gaps left by the private sector? While these are important questions, our goal is to quantify disparities and rule out competing explanations—such as differential access to clinical trials among races or differential revenue potential among diseases (see *Supporting Information*). Future work could model strategic interactions between the public and private sectors to better understand investment dynamics. Another avenue is to evaluate whether NIH's allocations align with a social welfare optimum, potentially by recovering its underlying objective function. These remain important areas for further research.

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A Materials and Methods

We describe how we assemble multiple data sources, construct key outcome variables, apply supervised machine learning to classify research projects by disease, and estimate the main statistical models. Full details are provided in the *Supporting Information* file.

A.1 Data Construction

We construct a panel dataset at the disease-year level covering 96 disease areas over two decades (2000–2019). Summary statistics are reported in Table S1. To measure public investment, we use the full universe of NIH research grants from the ExPORTER- RePORTER platform [31]. While NIH began assigning disease-area designations algorithmically in 2008, we extend this classification retrospectively to the early 2000s using supervised machine learning (see below and *Supporting Information*). As private companies do not disclose disease-level R&D expenditures, we proxy for private investment by the annual number of industry-sponsored clinical trials listed on ClinicalTrials.gov, maintained by the U.S. National Library of Medicine (NLM) [32]. Each trial includes sponsor-reported disease descriptors from a controlled vocabulary thesaurus, which we map to NIH disease categories using ICD-10 codes and custom crosswalks (see *Supporting Information* for details). We complement these data with time-varying, disease-specific characteristics: mortality data from CDC WONDER [33], publications from PubMed [34], medical expenditures by insurers and patients from the Medical Expenditure Panel Survey (MEPS), and disease-specific patient demographics from CDC's National Vital Statistics. (see *Supporting Information* for details). Following prior literature [35, 36], we compute disease-specific mortality ratios to capture racial and gender disparities. We focus on Black Americans and women as examples of key minority groups who represent a large share of the U.S. population and may therefore constitute a substantial target for policy. A disease is considered "Black-skewed" if the share of total Black deaths attributable to it is higher than the analogous share for White Americans. Specifically, for each disease d, we first compute the number of deaths due to disease d among Black individuals (D_d^{Black}) and White individuals (D_d^{White}). Second, we compute the total deaths among Black individuals (T^{Black}) and White individuals (T^{White}), no matter the cause of death. Finally, we compute the race-based relative mortality ratio for disease d as follows:

$$r_d = \frac{D_d^{Black}/T^{Black}}{D_d^{White}/T^{White}}$$

The higher the ratio, the more the disease accounts for deaths among the Black population relative to the White population. For completeness, we also construct the corresponding time-varying measure of the same race- based relative mortality ratio for each disease–we observe very limited within-disease variation. Similarly, we define a disease d as primarily affecting females if its mortality rate, relative to other diseases, is higher among females than it is among males. First, for each disease d, we compute the number of deaths due to disease d among females (D_d^{Female}) and males (D_d^{Male}). Second, we compute the total deaths among females (T^{Female}) and males (T^{Male}), regardless of the

cause of death. Then, we compute the gender-based relative mortality ratio for disease d as follows:

$$g_d = \frac{D_d^{Female}/T^{Female}}{D_d^{Male}/T^{Male}}$$

Values of both race-based and gender-based mortality ratios by disease are in Figures S1 and S2.

A.2 Supervised Machine Learning Exercise

Since 2008, NIH has used a machine-based algorithm—the Research, Condition, and Disease Categorization (RCDC) system—to classify funded research projects by disease area. This classification draws on project titles, abstracts, and key sections, matching extracted concepts to predefined disease categories using a thesaurus curated and validated by NIH experts. Projects funded prior to 2008 are not classified under this system. To extend disease classifications to the 2000–2007 period, we implement a supervised machine learning model trained on post-2008 RCDC-labeled data. Our goal is to assign earlier projects to one or more of the same disease categories used by RCDC. We begin by restricting the sample to U.S.-based, extramural NIH projects and verify the stability of disease categories over time. Following prior literature [11], we exclude non- disease research areas (e.g., risk factors, chemical agents), and 17 high-level "supercategories" that overlap almost entirely with more specific disease types. This yields a consistent set of 100 non-overlapping disease categories. We clean each project's title and abstract, removing stop words and non-discriminating terms (e.g., "and," "health"). We split the post-2008 RCDC-labeled data into a training set (80%) and validation set (20%). Using the FastText library [29, 30], we train a multi-label classification model to assign projects to one or more disease categories. FastText is well-suited to our task because it supports multi-label predictions and handles noisy, high-dimensional text efficiently. We test different classification models by varying the prediction threshold– i.e. the minimum probability required to assign a disease label–and the input–i.e., we use either projects' titles or abstracts to train the model. Model performance is evaluated on the validation sample using standard metrics:

- Precision (P): Correct predictions among predicted labels
- Recall (R): Correct predictions among true labels
- F1 Score: Harmonic mean of P and R, i.e.

$$F1 = \frac{2PR}{P+R}$$

We find that predictions based on abstracts perform similarly to those based on titles alone for intermediate prediction threshold levels. We use the latter as our main model specification, as all research projects are assigned a title, while some lack an abstract (see *Supporting* Information for results). Applying this model to pre-2008 projects, we generate disease classifications for the full 2000–2019 period. This produces a consistent 20-year panel of NIH-funded research projects, each mapped to specific disease areas.

A.3 Statistical Analysis

We estimate baseline regression models to quantify how public and private research activity respond to key drivers of investment across diseases and over time—and to test whether these drivers differ by sector. Our primary outcomes are:

1. the number of privately sponsored clinical trials, and
2. NIH funding in dollars, for each disease-year observation.

We estimate the following Ordinary Least Squares (OLS) model:

$$ln(Y_{d,t}) = \beta_0 + \beta_1 ln(Burden_{d,t-2}) + \beta_2 ln(ScientificOpportunity_{d,t-2}) + \beta_3 ln(RevenuePotential_{d,t-2}) + \beta_4 r_d + \beta_5 g_d + I_t + \epsilon_{d,t}$$

For each disease (d) and year (t), the outcome variables are privately sponsored trials (count, in log) and NIH funding (in dollars, in log), respectively. Burden is the total number of U.S. deaths caused by disease d two years before time t. Similarly, scientific opportunity is proxied as the volume of publications related to disease d two years prior to t. Disease d's revenue potential is expressed as the total annual medical expenditure incurred by insurers and patients at time t-2 to treat disease d. Race- and gender-based relative mortality ratios for disease d are indicated by r_d and g_d, as defined above. We include year fixed effects It to control for year- specific shocks to investment across diseases and we cluster standard errors at the disease level. The identification of our coefficients of interest arises from variations in private clinical trials and NIH funding across diseases, keeping the other observables constant. For example, the identification of β_4 arises from variation in trials and NIH funding across diseases that differ in their race-based mortality ratios, but share similar values of burden, scientific opportunity, revenue potential, and gender-based ratios. In the *Supporting Information*, we show that our results are robust to several alternative specifications, e.g., including different lag structures (e.g., 3-year moving averages), inclusion of lagged NIH funding (in models predicting private clinical trials) or lagged private clinical trials (in models predicting NIH funding). Moreover, we display full regression tables in which controls are added one at a time, to transparently assess coefficient relationships and stability.

B Supporting Information

We compile a panel dataset covering 96 diseases from 2000–2019 by harmonizing multiple biomedical research data sources. Below, we detail our approach to dataset construction, harmonization, and validation. Summary statistics are reported in Table S1.

B.1 Dataset and Variable Construction: Further Details

B.1.1 NIH funding

Our primary source is the NIH ExPORTER-RePORTER database, containing all NIH-funded projects from 2000–2019, including titles, abstracts, funding amounts, and fiscal years. Begin-

ning in 2008, NIH implemented an algorithmic disease-classification scheme mandated by the U.S. Congress. To classify grants consistently prior to 2008, we trained a supervised machine learning model on post-2008 data, enabling retrospective categorization of earlier projects into standardized NIH disease categories. We mapped each NIH disease category to ICD-10 codes using CMS codebooks, validating against prior research [S1]. Following established practice, we excluded non-disease-specific research areas (e.g., substances, general risk factors) due to incompatibility with standard burden metrics like mortality. This process resulted in approximately 100 distinct disease categories. We distinguish NIH grants based on their initiation: NIH-solicited (targeted programs representing agency priorities) versus investigator-initiated grants. Our main analyses use NIH-solicited grants, comprising roughly 30placebos for robustness analyses.

B.1.2 Privately sponsored clinical trials

We collected clinical trial data from ClinicalTrials.gov, maintained by the National Library of Medicine (NLM). This database includes comprehensive records of all U.S. clinical trials since 2000, irrespective of outcomes. We focused specifically on privately sponsored trials (primarily pharmaceutical companies), utilizing public or academic-sponsored trials solely for robustness checks. Trials were categorized via NLM's Medical Subject Headings (MeSH), cross-referenced to ICD-10 codes through official NLM mappings, then harmonized with NIH disease categories. We employed fuzzy matching followed by manual verification for unmatched cases.

B.1.3 Scientific opportunity

To quantify scientific opportunity, we measured annual disease-specific publication counts from PubMed, utilizing its MeSH-based indexing system. Consistent with prior research [S3], we interpret higher publication counts as indicative of greater scientific opportunity or recent advancements in understanding the disease.

B.1.4 Disease burden

We measured disease burden via disease-specific mortality rates from CDC's Underlying Cause-of-Death files, disaggregated by race and gender. From these data, and as in prior studies [S4, S5], we computed disease- specific mortality ratios for Black versus White populations and female versus male populations, establishing "Black" and "female" diseases based on disproportional mortality ratios (> 1). Given limited comprehensive incidence data across demographic groups, mortality rates provide the most consistently available measure of disease burden. Our analysis includes diseases averaging ≥ 40 deaths annually; we tested alternative inclusion thresholds for robustness.

B.1.5 Revenue potential

Revenue potential was proxied by disease-specific medical expenditures, calculated from the nationally representative Medical Expenditure Panel Survey (MEPS). Using ICD-10 matched expenditure records, we aggregated annual spending per disease-year, including patient and insurer payments.

We tested robustness using alternative spending metrics (e.g., per-patient costs). In the following section, we detail our supervised machine learning approach and validation procedures to ensure rigorous and consistent disease classifications.

B.2 Supervised Machine Learning Procedure: Further Details

We employed supervised machine learning techniques to classify NIH-funded research grants from 2000–2007 into standardized disease categories, aligning them with post-2008 NIH classifications. Below, we detail our methodological approach, validation strategy, and model performance.

B.2.1 Training and Validation

Our objective was to retrospectively classify NIH-funded projects prior to 2008 using titles and abstracts. We began by preprocessing and cleaning the titles and abstracts of all NIH projects. We then split the data from 2008 onwards into a training set (80%) and a validation set (20%). We trained a multi-label classification model using FastText, a machine learning tool developed by Meta AI [S6, S7, S8], well-suited for short-text classification and multiple-label assignments.

B.2.2 Model Performance Test 1: Out-of-Sample Validation

We use the validation sample to benchmark the model on out-of-sample data. For each project, we compare the predicted disease labels to the true NIH-assigned ones. Performance is measured using:

- Precision: Share of predicted labels that are correct.
- Recall: Share of true labels that are successfully predicted.
- F1 Score: Harmonic mean of precision and recall.

We vary:

- Input type: Using either titles or abstracts to train the model for predictions.
- Prediction threshold: The minimum probability required to assign a disease label (e.g., 0.1, 0.5, 0.8).

Key results (see Tables S2–S4):

- Precision increases with tighter thresholds (fewer, more confident predictions).
- Recall increases with looser thresholds (more inclusive predictions).
- The F1 score captures the Precision-Recall trade-off and is maximized at intermediate threshold levels.
- For intermediate threshold levels, title- and abstract-based models perform similarly in terms of F1. For more extreme threshold levels, titles outperform abstracts.

B.2.3 Model Performance Test 2: Temporal Backcasting

Our core use case is to use post-2008 data to predict disease categories for pre-2008 projects. To simulate this, we train models on 2014–2019 data and validate on 2008–2013 projects. As in Test 1, we evaluate precision, recall, and F1 scores, comparing abstract- and title-based models across thresholds (see Tables S5–S6). Results are consistent:

- Higher thresholds improve precision, lower ones improve recall.
- F1 scores favor intermediate threshold levels.
- Abstracts slightly outperform titles here, likely due to richer detail, although this was not obvious a priori.

B.2.4 Model Selection and Deployment

We select the title-based model for primary use due to consistent title availability across all grants. Both title- and abstract-based models perform similarly at optimal thresholds. We apply this model to classify NIH-funded projects from 2000–2007, providing consistent categorization across our full study period (2000–2019). Abstract-based classifications are reserved for robustness checks.

B.3 Statistical Analyses: Further Details

This section outlines our main regression framework, interprets the central findings, and presents robustness checks and tests of alternative mechanisms that could explain our results.

B.3.1 Main regression results

Tables S7 and S8 present regression estimates of how private- and public-sector research activity responds to disease characteristics over time. Table S7 examines the number of clinical trials sponsored by the private sector, while Table S8 analyzes NIH funding. The core independent variables of interest are race- and gender- based relative mortality ratios, along with controls for disease burden (measured by lagged total deaths), scientific opportunity (measured by lagged publications), and year fixed effects. Standard errors are clustered at the disease level in all panel regressions. We begin by regressing private-sector clinical trial activity on the relative mortality measures. We then progressively enrich the model by introducing additional controls, including disease burden, scientific opportunity, and year fixed effects. Later specifications incorporate time-varying mortality ratios and a cross- sectional analysis for 2015 to test robustness outside the panel structure. Across all specifications, we observe that private clinical trial activity increases with both disease burden and scientific opportunity. For example, in our preferred specification (Column 3), a one percent increase in total deaths or publication volume corresponds to a 0.16 percent and 0.48 percent increase in private trials, respectively. The estimated coefficient on the race-based relative mortality ratio is consistently negative and statistically significant, suggesting that diseases disproportionately affecting Black Americans receive less private investment. A one-unit increase in this ratio—roughly equivalent to a one standard deviation increase—leads to a 19 percent decline in the number of

private-sector clinical trials. By contrast, the gender-based mortality ratio has no significant effect on private investment. If anything, diseases more prevalent among women appear slightly less likely to attract private trials, but the estimates are not statistically different from zero. We conduct a parallel analysis of NIH funding in Table S8. As with the private sector, public-sector funding responds positively to disease burden and publication volume. In the preferred specification, a one percent increase in deaths or publications results in a 0.27 percent and 0.39 percent increase in NIH funding, respectively. Crucially, the coefficient on the race-based mortality ratio is positive and significant: diseases that disproportionately affect Black Americans receive more NIH funding. A one-unit increase in the race-based ratio is associated with a 27 percent increase in public research investment. Again, we find no statistically significant effect of the gender-based ratio on NIH funding. Together, these results reveal a striking divergence: the private sector underinvests in "Black" diseases, while the public sector appears to prioritize them.

B.3.2 Alternative mechanism 1: disease revenue potential

To explore whether differential revenue potential explains the observed racial disparity in research investment, we incorporate medical expenditure data from the Medical Expenditure Panel Survey (MEPS). This dataset provides annual treatment costs by disease, which we interpret as a proxy for profitability. Diseases predominantly affecting Black patients may be associated with lower revenue potential due to reduced healthcare access and greater reliance on lower-paying insurers such as Medicaid. We augment our baseline regressions by including total medical expenditure as a control. In the private-sector regressions (Table S7, Columns 6–7), expenditure is a strong and significant predictor of clinical trial activity. However, in the public-sector regressions (Table S8, Columns 6–7), NIH funding is uncorrelated with expenditure, consistent with the notion that public funding is not primarily guided by expected commercial returns. Importantly, controlling for medical expenditure does not attenuate the racial disparity. The coefficient on the race-based mortality ratio remains stable in magnitude and significance. Even when accounting for revenue potential, the private sector continues to underinvest in diseases that disproportionately affect Black Americans, while the public sector remains disproportionately responsive.

B.3.3 Alternative mechanism 2: differential access to clinical trials

A second potential mechanism is differential access to trials. Prior research suggests that Black patients may be harder to recruit for clinical studies due to systemic mistrust [S9] and lower proximity to major medical centers [S10, S11]. If recruitment barriers were driving the observed disparity, both privately- and publicly- sponsored clinical trials should be less likely to target "Black" diseases. We test this possibility by re-running our baseline analysis on the subset of publicly funded clinical trials, which we extract from ClinicalTrials.gov. Table S9 presents the results. We find no significant relationship between race-based mortality and public clinical trial activity. This stands in contrast to our earlier findings for private clinical trials and suggests that differential patient access is unlikely to be the primary driver of the observed investment gap.

B.3.4 Interpretation of results

To contextualize our estimates, we compute what it would take for "Black" diseases to receive the same level of investment as "White" diseases, holding all else equal. Our calculations show that private-sector parity would require Black diseases to be 52 percent more deadly, 18 percent more published in the scientific literature, or associated with 64 percent higher revenue potential. By contrast, NIH funding parity could be achieved if Black diseases were 48 percent less deadly or generated 45 percent fewer publications. These counterfactuals reinforce the asymmetry in how public and private sectors respond to racial disparities in disease burden.

B.3.5 Robustness checks

We conduct a series of additional analyses to assess the robustness of our findings to alternative specifications, data restrictions, and measurement strategies. These checks confirm that the observed divergence between public and private investment in "Black" diseases is not an artifact of modeling choices, omitted variables, or data limitations.

B.3.6 Robustness 1: controlling for lagged research activity

To address the possibility that public and private research efforts influence each other over time, we augment our baseline regressions by including lagged values of the other sector's investment. Specifically, we regress private-sector clinical trials on lagged NIH funding, and vice versa. These regressions (Table S10) reveal a positive relationship between lagged public and current private investment, and vice versa—consistent with prior research suggesting that public research can stimulate private R&D, and that private sector progress may also inform future public allocations [S12, S13]. However, this interdependence does not account for our main result. The race-based mortality coefficient remains significant and unchanged in direction. If anything, the magnitude slightly increases, suggesting that the negative (positive) direct effect of race on private (public) investment persists even after accounting for indirect effects via the other sector's lagged behavior. This pattern supports the view that public and private investment decisions act, at least in part, as substitutes along the racial dimension.

B.3.7 Robustness 2: restricting the sample to post-2008 NIH projects

Our NIH disease classifications before 2008 are based on supervised machine learning. To ensure that misclassification does not bias our findings, we replicate the main regressions using only post-2008 data, where disease categorization was conducted by NIH's official RCDC system. The results (Tables S11 and S12) closely mirror those from the full sample. The direction, magnitude, and significance of the race-based mortality coefficient remain stable for both private and public investment models, reinforcing the reliability of our classification strategy and confirming that the findings are not driven by model- predicted labels.

B.3.8 Robustness 3: changing the lag structure

In our baseline specification, we use two-year lags for key covariates such as mortality, publication volume, and medical expenditure. To ensure that results are not sensitive to this timing assumption, we test an alternative lag structure using three-year moving averages. Again, the results (Tables S13 and S14) are robust. The key coefficients retain their sign and significance, with only modest changes in magnitude. This suggests that the precise lag structure does not materially affect the observed disparities in investment patterns.

B.3.9 Robustness 4: using alternative measures for racial disease prevalence

Our primary measure of racial disparity is a relative mortality ratio—specifically, the mortality rate for Black Americans relative to Whites, normalized across diseases. As a robustness check, we adopt a more direct measure: the absolute number of Black and White deaths per disease-year. In Tables S15 and S16, we regress private and public investment, respectively, on both Black and White deaths (controlling for scientific opportunity, revenue potential, and gender-based mortality). Because total deaths is mechanically a sum of Black and White deaths, we exclude it to avoid collinearity. The results reveal a striking divergence: holding other factors constant, an increase in Black deaths is associated with a decline in private investment, while an increase in White deaths predicts more private trials. A formal Wald test rejects the null hypothesis of equal coefficients for Black and White deaths. This confirms that private investors respond differently depending on the racial composition of disease mortality. The NIH response differs. Black deaths are positively associated with NIH funding, while the coefficient on White deaths is close to zero and often statistically insignificant. The Wald test does not consistently reject equality in this case—likely due to greater imprecision in the estimate for White deaths—but the core insight holds: NIH investment rises with Black disease mortality, while private investment falls.

B.3.10 Robustness 5: assessing omitted variable bias

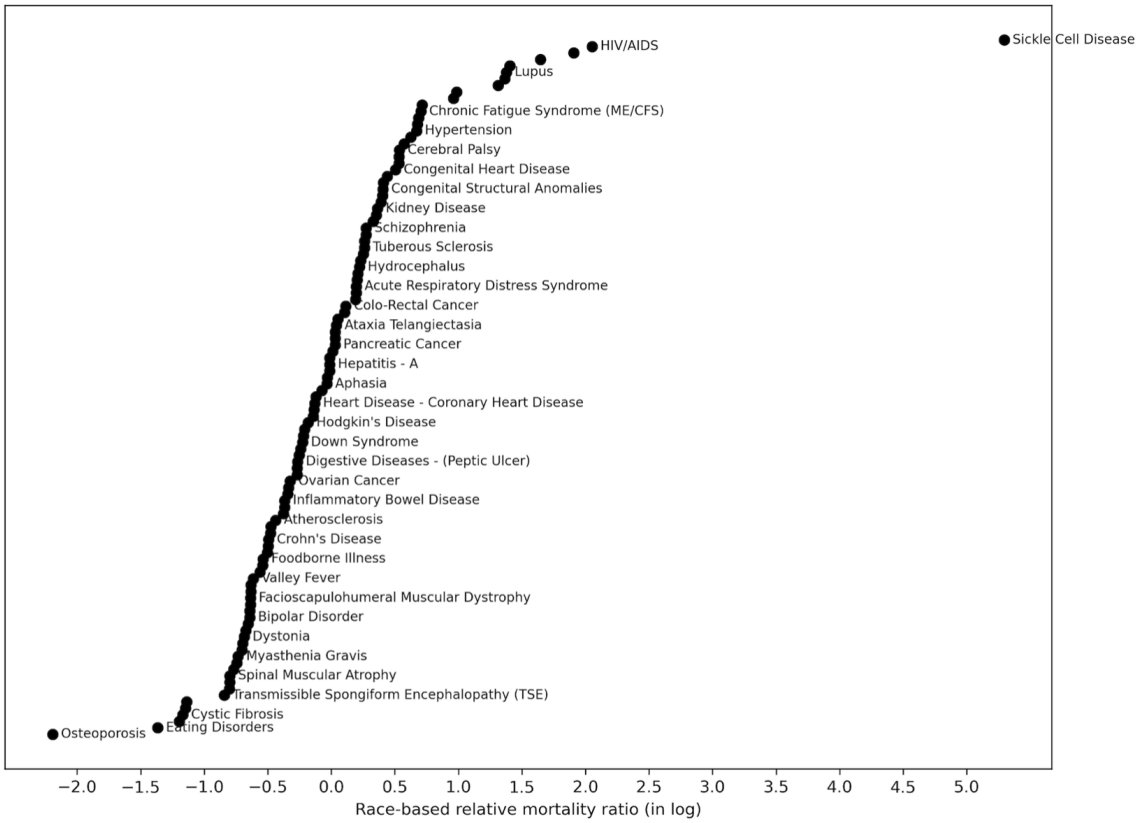
To evaluate the potential impact of unobserved confounding, we applied the Oster method [S14]. This test computes how strongly unobserved variables would need to correlate with both the treatment and the outcome to fully explain away the observed relationship. We focus on the race-based mortality ratio coefficients in our richest specifications: NIH funding (Table S7, Column 6) and private trials (Table S8, Column 6). For each specification, we compute the selection ratio δ under two scenarios for the hypothetical R-squared of the full model R_{max}: (1) R_{max} equals 1; and (2) R_{max} is twice the R-squared of the model with all the observed control variables. Table S17 shows the results.

- For NIH funding, δ ranges from 6.7 to 10.1, meaning selection on unobservables would need to be 6.7 to 10.1 times stronger than selection on observables to nullify the race-based coefficient.
- For private trials, δ ranges from -2.7 to -3.1. The negative sign reflects that, since observed controls move the coefficient away from zero, unobservables would need to move it in the opposite direction to eliminate the effect–and thus the negative sign of δ.

Because $|\delta| > 1$ in all cases, we conclude that omitted variable bias is unlikely to explain our main findings. These results confirm the robustness of our estimates linking racial disparities in mortality to divergent patterns of public and private investment. Together, these analyses robustly demonstrate persistent racial disparities in biomedical R&D investments, highlighting distinct roles of public versus private sector funding strategies.

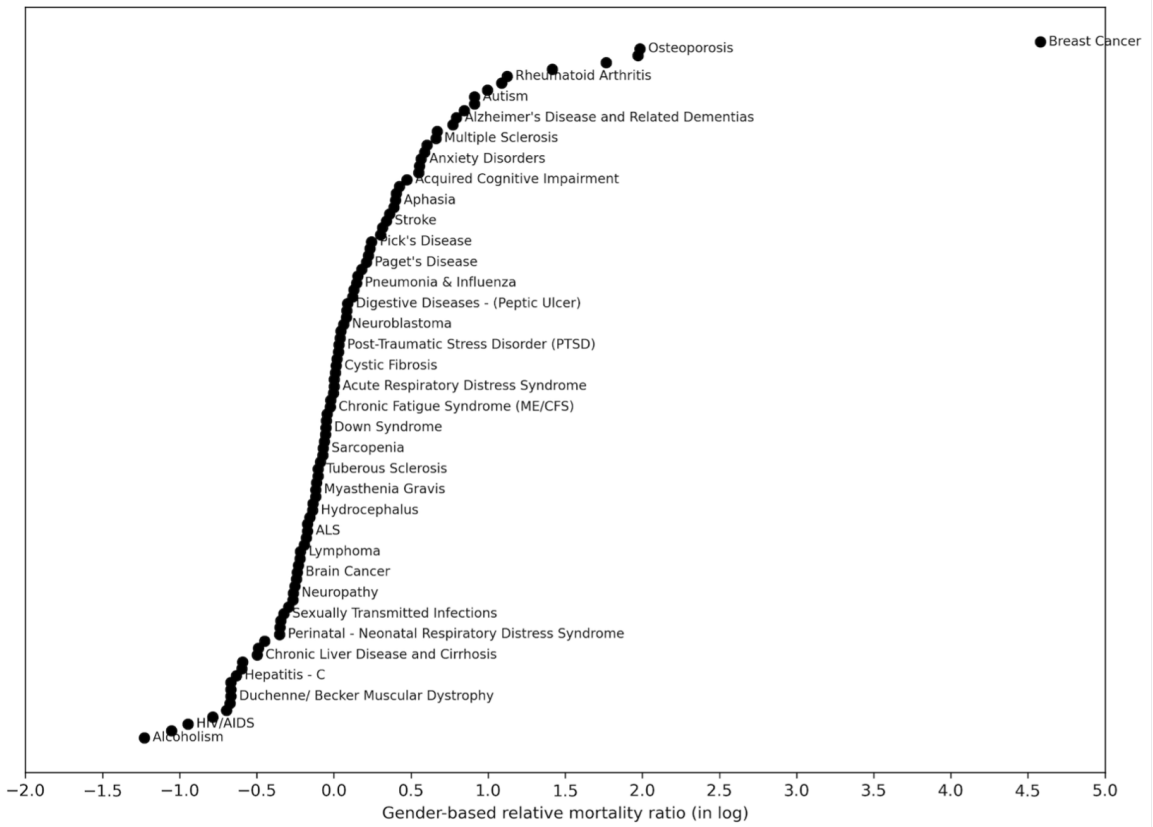
C Additional Figures

Figure S1: Relative Mortality Burden by Race Across Diseases.



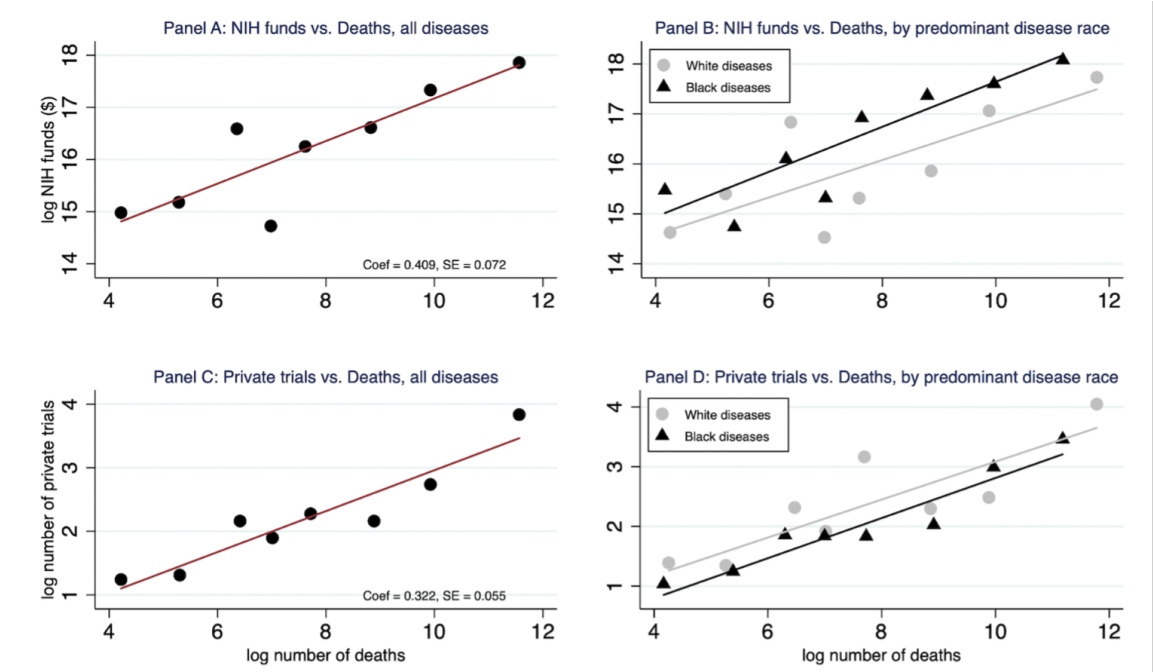
Notes: This figure plots the average (log) race-based mortality ratio by disease, calculated as the mortality rate among Black Americans divided by that among White Americans (see Methods). Each point represents a disease; for clarity, only a subset is labeled. Conditions such as sickle cell disease and HIV disproportionately affect Black Americans, while osteoporosis and eating disorders affect White Americans more heavily.

Figure S2: Relative Mortality Burden by Gender across Diseases.



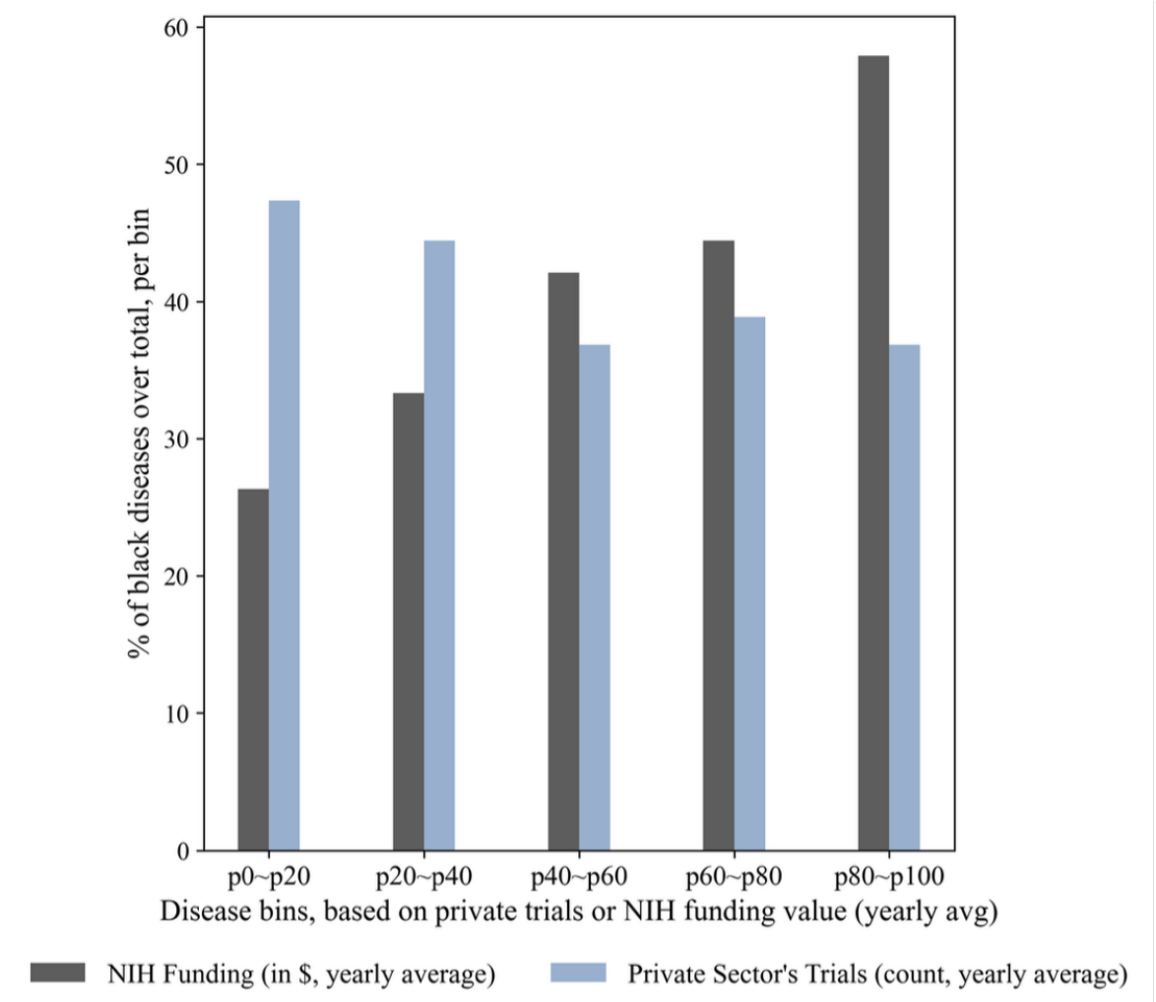
Notes: This figure displays the average (log) gender-based mortality ratio by disease, computed as the mortality rate among females divided by that among males (see Methods). Each point represents a disease; a subset is labeled for clarity. Diseases affecting only one sex (e.g., prostate or vaginal cancer) are excluded. Breast cancer and osteoporosis disproportionately affect women, while alcoholism and HIV are more prevalent causes of death among men.

Figure S3: Public and Private Research Investment by Disease Mortality, Overall and by Racial Disparity.



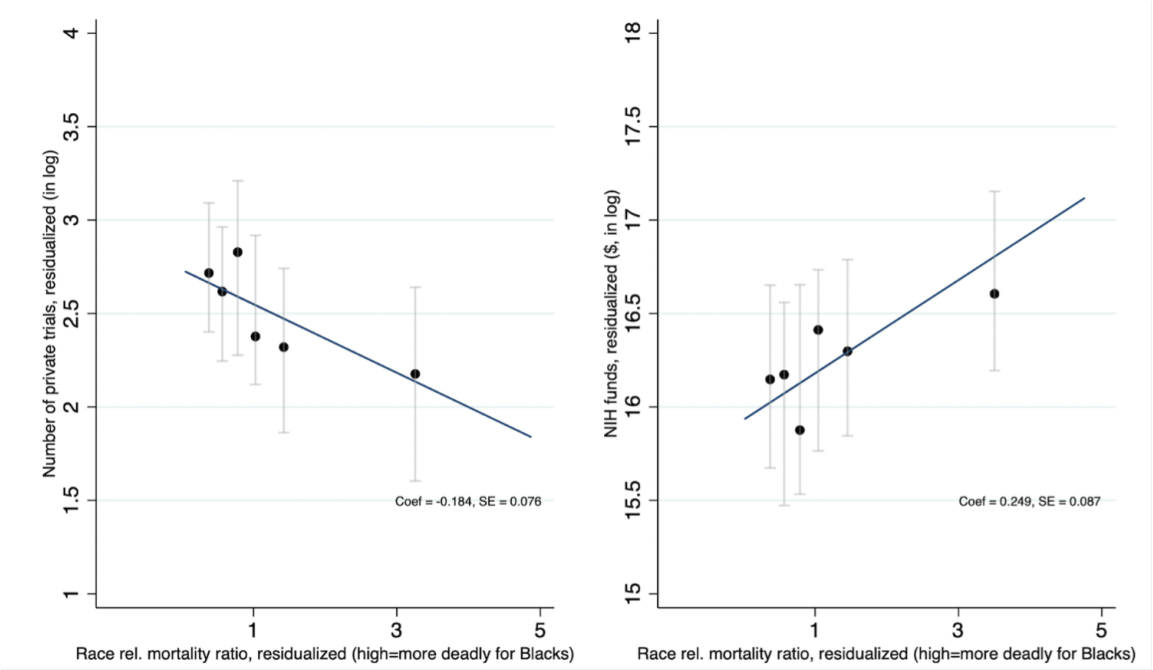
Notes: This figure depicts the relationship between disease-specific mortality and U.S. research investment using binscatter plots. Public investment is measured by NIH funding (USD); private investment by the number of industry-sponsored clinical trials. Death counts are cumulative and not disaggregated by race. For each disease, we compute average annual mortality, NIH funding, and clinical trials from 2000–2019. Diseases are grouped into eight bins by mortality, and each point represents the mean investment and mortality within a bin. Panels A and C show linear fits for all diseases. Panels B and D separate predictions by racial disparity: diseases with a Black-to-White mortality ratio > 1 ("Black diseases") versus ≤ 1 ("White diseases") [35, 36].

Figure S4: Inverse Relationship between Public and Private Investment in Diseases affecting Black Americans.



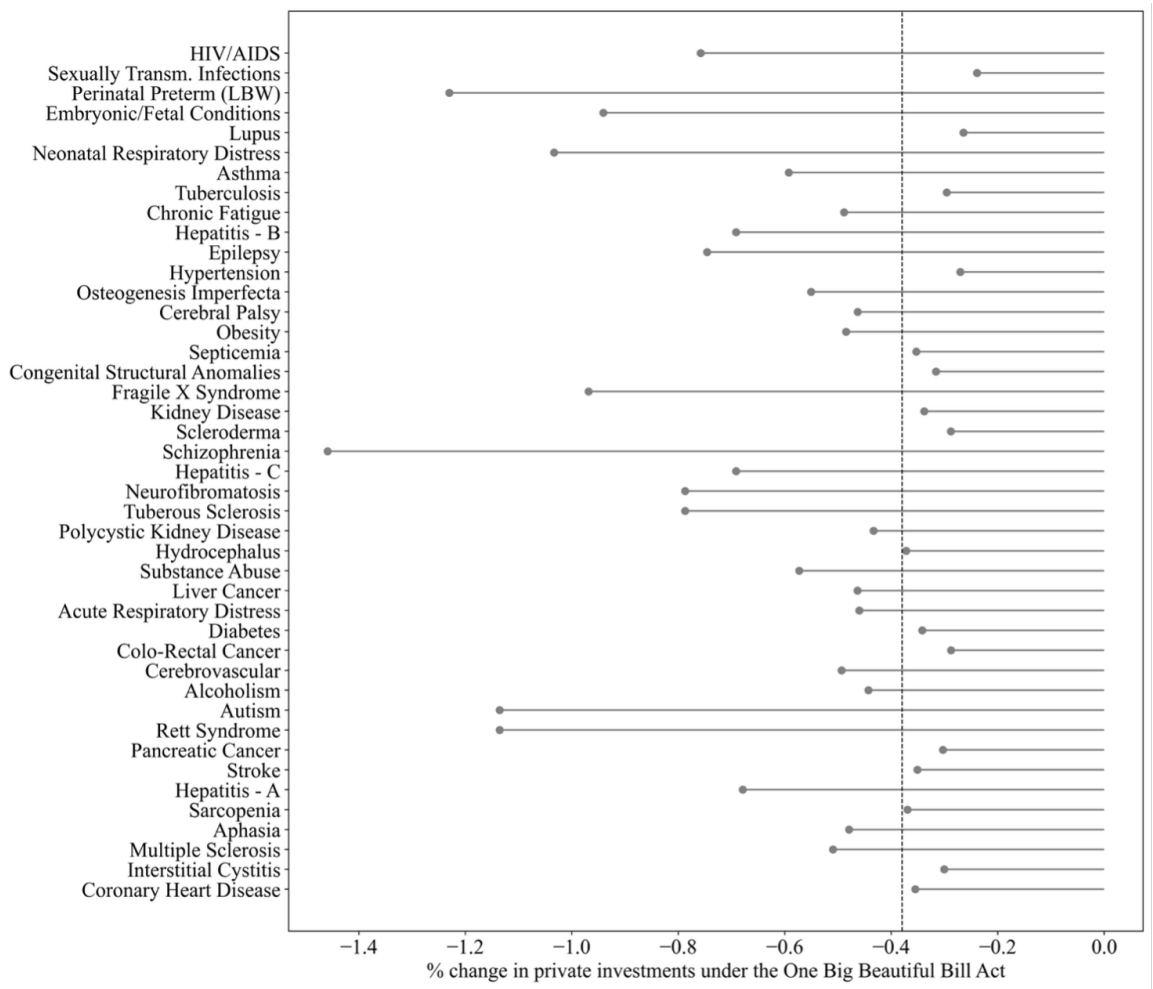
Notes: This figure shows the share of diseases disproportionately affecting Black Americans across quintiles of research investment. Diseases are ranked separately by average annual NIH funding and number of industry-sponsored clinical trials (2000–2019), then grouped into quintiles. The x-axis represents increasing levels of investment, from the lowest (p0–p20) to the highest (p80–p100). For each quintile, we report the percentage of diseases classified as "Black diseases," defined by a Black-to-White mortality ratio greater than one (11, 12). Results reveal an inverse relationship: public investment is more concentrated in diseases affecting Black Americans at lower private-sector investment levels.

Figure S5: Relationship between Race-based Disease Burden and Residualized Research Investment.



Notes: This figure plots the residual relationship between disease-specific investment and race-based mortality disparities. Public investment is measured by NIH funding (USD); private investment by the number of industry-sponsored clinical trials. For each disease-year, we residualize both investment outcomes and the race-based mortality ratio by removing variation explained by other investment determinants—disease burden, scientific opportunity, revenue potential, and gender-based mortality ratio (see Methods). The race-based mortality ratio is computed as the mortality rate among Black Americans divided by that among White Americans. Diseases are grouped into six bins by this ratio; each point represents the average residualized investment and mortality ratio within the bin. Error bars show 95% confidence intervals. The figure illustrates that, conditional on other drivers of investment, private funding declines with Black mortality burden, while public investment increases.

Figure S6: Predicted Change in Private-Sector Investment Following the One Big Beautiful Bill Act.



Notes: This figure quantifies the extent to which private investment would change as a result of the Medicaid-related provisions in the recent One Big Beautiful Bill Act. According to the Congressional Budget Office, the number of uninsured people is projected to increase by 7.8 million in 2034 following these provisions, as many people would lose access to Medicaid. This is a conservative projection, since other provisions in the Act are expected to raise the number of uninsured even further. With more people remaining uninsured, total medical spending would decline—especially for diseases that affect the Medicaid population. This is true assuming limited price changes, or even in case prices fall (as proposed by current initiatives to lower U.S. pharmaceutical costs). For each disease, we use our empirical model and estimates to predict the private-sector investment level under the new scenario and compute the percentage change relative to the status quo. We present results for diseases disproportionately affecting Black Americans. We report the average across all diseases in our sample (i.e., affecting disproportionally either Black and White Americans) as a dotted vertical line. Unsurprisingly, the Medicaid-related provisions would reduce private investment particularly in "Black diseases", since many of them heavily affect the Medicaid population. For example, HIV and schizophrenia would be much less attractive for the private sector than in the status quo, while diabetes and hydrocephalus would be negatively affected as much as the average disease.

D Additional Tables

Table S1: Summary statistics of the key variables of interest.

				Percentile				
	Count	Mean	SD	5th	25th	50th	75th	95th
Dependent Variables								
NIH-initiated funding (million $)		51.4	150.2	0.1	2.0	10.1	42.1	188.8
Privately sponsored clinical trials (count)		25.8	42.3	0.0	2.0	7.0	33.0	109.0
Independent Variables								
Black deaths		2,101.1	5,666.4	5.0	29.0	169.0	1,208.0	10,108.0
White deaths		16,518.1	47,979.4	46.0	310.0	1,112.0	7,868.0	88,119.0
Male deaths		9,554.2	28,118.4	20.0	186.0	727.0	5,826.0	37,764.0
Female deaths		9,580.7	27,406.5	32.0	182.0	654.0	4,805.0	59,977.0
Publications		5,391.5	10,628.5	114.0	426.0	1,463.0	5,098.0	22,767.0
Race-based relative mortality ratio		1.3	1.3	0.3	0.6	0.9	1.4	4.0
Gender-based relative mortality ratio		1.2	1.1	0.5	0.8	1.0	1.3	3.2
Potential market size (million $)		839.9	1,853.9	5.6	44.0	169.3	763.6	3,658.0
Sample Overview								
Year	20							
Numbers of diseases	96							

Table S2: Model performance, test 1–Precision.

Year	**P(A-0.1)**	**P(A-0.5)**	**P(A-0.8)**	**P(T-0.1)**	**P(T-0.5)**	**P(T-0.8)**
2008	0.58	0.89	0.95	0.73	0.89	0.93
2009	0.65	0.91	0.96	0.78	0.91	0.95
2010	0.62	0.91	0.96	0.75	0.89	0.93
2011	0.60	0.89	0.95	0.73	0.88	0.93
2012	0.60	0.90	0.96	0.73	0.88	0.93
2013	0.59	0.89	0.95	0.73	0.88	0.93
2014	0.58	0.89	0.95	0.72	0.88	0.93
2015	0.59	0.89	0.95	0.73	0.89	0.93
2016	0.59	0.89	0.96	0.73	0.88	0.93
2017	0.59	0.89	0.95	0.73	0.89	0.94
2018	0.62	0.90	0.96	0.74	0.88	0.92
2019	0.62	0.90	0.96	0.74	0.88	0.93

Table S3: Model performance, test 1–Recall.

Year	R(A-0.1)	R(A-0.5)	R(A-0.8)	R(T-0.1)	R(T-0.5)	R(T-0.8)
2008	0.79	0.57	0.43	0.71	0.61	0.54
2009	0.84	0.65	0.51	0.75	0.66	0.60
2010	0.82	0.63	0.50	0.70	0.60	0.54
2011	0.82	0.61	0.48	0.70	0.60	0.53
2012	0.82	0.62	0.49	0.70	0.60	0.53
2013	0.80	0.59	0.45	0.70	0.60	0.53
2014	0.80	0.59	0.46	0.70	0.59	0.53
2015	0.81	0.59	0.46	0.70	0.59	0.53
2016	0.80	0.60	0.47	0.70	0.60	0.53
2017	0.81	0.62	0.50	0.72	0.61	0.56
2018	0.82	0.63	0.52	0.72	0.62	0.56
2019	0.84	0.66	0.54	0.73	0.64	0.58

Table S4: Model performance, test 1–F1 Score.

Year	F1(A-0.1)	F1(A-0.5)	F1(A-0.8)	F1(T-0.1)	F1(T-0.5)	F1(T-0.8)
2008	0.67	0.69	0.60	0.72	0.72	0.68
2009	0.73	0.76	0.67	0.76	0.77	0.73
2010	0.71	0.75	0.66	0.72	0.72	0.69
2011	0.69	0.73	0.64	0.71	0.72	0.68
2012	0.70	0.73	0.65	0.71	0.71	0.67
2013	0.68	0.71	0.61	0.72	0.71	0.67
2014	0.67	0.71	0.62	0.71	0.71	0.68
2015	0.68	0.71	0.62	0.72	0.71	0.68
2016	0.68	0.71	0.63	0.71	0.71	0.68
2017	0.69	0.73	0.66	0.72	0.72	0.69
2018	0.70	0.75	0.67	0.73	0.73	0.69
2019	0.71	0.76	0.69	0.74	0.74	0.71

Table S5: Model performance, test 2–F1 Score, based on research projects' abstracts.

Threshold	Precision	Recall	F1
0.1	0.66	0.88	0.75
0.3	0.75	0.83	0.79
0.5	0.79	0.79	0.79
0.7	0.81	0.75	0.78
0.9	0.84	0.68	0.75

Table S6: Model performance, test 2–F1 Score, based on research projects' titles.

Threshold	**Precision**	**Recall**	**F1**
0.1	0.68	0.74	0.71
0.3	0.75	0.70	0.72
0.5	0.77	0.68	0.72
0.7	0.80	0.65	0.72
0.9	0.82	0.61	0.70

Table S7: Privately-funded Clinical Trials vs. Main Investment Drivers.

	Log privately sponsored clinical trials						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Log total deaths (2-year lagged)		0.159**	0.164**	0.167***	0.155***	0.160**	0.164***
		(0.0615)	(0.0634)	(0.0620)	(0.0556)	(0.0608)	(0.0595)
Log publications (2-year lagged)		0.482***	0.476***	0.472***	0.498***	0.435***	0.430***
		(0.111)	(0.117)	(0.115)	(0.0798)	(0.121)	(0.119)
Race-based rel. mortality ratio	-0.0933	-0.191**	-0.191**		-0.153*	-0.198**	
	(0.107)	(0.0789)	(0.0799)		(0.0866)	(0.0987)	
Race-based rel. mortality ratio (time varying)				-0.184**			-0.187**
				(0.0760)			(0.0935)
Gender-based rel. mortality ratio	-0.103	-0.105	-0.101		-0.111	-0.121	
	(0.101)	(0.0912)	(0.0942)		(0.113)	(0.0905)	
Gender-based rel. mortality ratio (time varying)				-0.0836			-0.0967
				(0.0749)			(0.0697)
Log total expenditure (2-year lagged)						0.138**	0.138**
						(0.0618)	(0.0611)
Observations	1,368	1,243	1,243	1,243	82	1,031	1,031
Sample	Full	Full	Full	Full	2015	Full	Full
Year FEs	No	No	Yes	Yes	-	Yes	Yes
Clusters	Yes	Yes	Yes	Yes	-	Yes	Yes
Adjusted R sq	0.00899	0.417	0.420	0.418	0.500	0.454	0.451
E[Y]	2.444	2.506	2.506	2.506	2.670	2.585	2.585
sd[Y]	1.501	1.507	1.507	1.507	1.468	1.534	1.534
E[race ratio]	1.245	1.243	1.243	1.238	1.324	1.219	1.219
sd[race ratio]	1.156	1.150	1.150	1.160	1.324	1.046	1.046

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table S8: Public NIH Funds vs. Main Investment Drivers.

	Log NIH funding						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Log total deaths (2-year lagged)		0.256***	0.273***	0.272***	0.267***	0.250***	0.252***
		(0.0775)	(0.0786)	(0.0777)	(0.0780)	(0.0827)	(0.0817)
Log publications (2-year lagged)		0.440***	0.393**	0.395**	0.394***	0.276	0.276
		(0.156)	(0.159)	(0.158)	(0.112)	(0.175)	(0.173)
Race-based rel. mortality ratio	0.317***	0.263***	0.272***		0.351***	0.208*	
	(0.110)	(0.0949)	(0.0942)		(0.122)	(0.108)	
Race-based rel. mortality ratio (time varying)				0.249***			0.190*
				(0.0866)			(0.0956)
Gender-based rel. mortality ratio	-0.0874	-0.0796	-0.0606		-0.0556	-0.130	
	(0.106)	(0.146)	(0.143)		(0.159)	(0.138)	
Gender-based rel. mortality ratio (time varying)				-0.0572			-0.108
				(0.115)			(0.109)
Log total expenditure (2-year lagged)						0.0955	0.0920
						(0.0700)	(0.0694)
Observations	1,564	1,392	1,392	1,392	83	1,109	1,109
Sample	Full	Full	Full	Full	2015	Full	Full
Year FEs	No	No	Yes	Yes	-	Yes	Yes
Clusters	Yes	Yes	Yes	Yes	-	Yes	Yes
Adjusted R sq	0.0423	0.364	0.399	0.397	0.401	0.383	0.382
E[Y]	16.16	16.25	16.25	16.25	16.55	16.43	16.43
sd[Y]	1.948	1.943	1.943	1.943	1.928	1.891	1.891
E[race ratio]	1.285	1.287	1.287	1.289	1.313	1.266	1.266
sd[race ratio]	1.235	1.231	1.231	1.288	1.320	1.148	1.148

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table S9: Alternative Mechanism–*Publicly-funded* Clinical Trials vs. Main Investment Drivers.

	Log publicly sponsored clinical trials						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Log total deaths (2-year lagged)		0.144***	0.158***	0.158***	0.153***	0.142***	0.144***
		(0.0474)	(0.0480)	(0.0473)	(0.0519)	(0.0478)	(0.0471)
Log publications (2-year lagged)		0.477***	0.440***	0.439***	0.468***	0.397***	0.394***
		(0.106)	(0.109)	(0.108)	(0.0747)	(0.122)	(0.120)
Race-based rel. mortality ratio	-0.0257	-0.100	-0.0899		-0.0485	-0.140	
	(0.0768)	(0.0818)	(0.0782)		(0.0808)	(0.0854)	
Race-based rel. mortality ratio (time-varying)				-0.0878			-0.128*
				(0.0690)			(0.0726)
Gender-based rel. mortality ratio	-0.0202	-0.0447	-0.0300		-0.0290	-0.0540	
	(0.0623)	(0.0971)	(0.0962)		(0.106)	(0.0856)	
Gender-based rel. mortality ratio (time-varying)				-0.0257			-0.0423
				(0.0804)			(0.0722)
Log total expenditure (2-year lagged)						0.0290	0.0293
						(0.0531)	(0.0526)
Observations	1,507	1,341	1,341	1,341	82	1,101	1,101
Sample	Full	Full	Full	Full	2015	Full	Full
Year FEs	No	No	Yes	Yes	-	Yes	Yes
Clusters	Yes	Yes	Yes	Yes	-	Yes	Yes
Adjusted R sq	-0.000617	0.455	0.499	0.499	0.503	0.503	0.502
E[Y]	2.824	2.908	2.908	2.908	3.220	2.980	2.980
sd[Y]	1.418	1.396	1.396	1.396	1.360	1.391	1.391
E[race ratio]	1.293	1.293	1.293	1.294	1.313	1.271	1.271
sd[race ratio]	1.243	1.235	1.235	1.291	1.320	1.151	1.151

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table S10: Robustness 1–controlling for lagged research activity.

	Log privately sponsored clinical trials					Log NIH funding				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Log total deaths (2-year lagged)		0.135***	0.136**	0.140***	0.123**		0.199**	0.228***	0.227***	0.204**
		(0.0511)	(0.0535)	(0.0527)	(0.0516)		(0.0800)	(0.0826)	(0.0811)	(0.0818)
Log publications (2-year lagged)		0.428***	0.432***	0.428***	0.402***		0.198	0.195	0.199	0.0852
		(0.114)	(0.116)	(0.114)	(0.112)		(0.209)	(0.213)	(0.211)	(0.208)
Race-based rel. mortality ratio	-0.227***	-0.226***	-0.226***		-0.226**	0.375***	0.331***	0.328***		0.227*
	(0.0853)	(0.0673)	(0.0690)		(0.0900)	(0.0870)	(0.108)	(0.108)		(0.122)
Race-based rel. mortality ratio (time varying)				-0.217***					0.324***	
				(0.0657)					(0.109)	
Gender-based rel. mortality ratio	-0.0422	-0.0806	-0.0816		-0.0913	-0.117	-0.0729	-0.0635		-0.108
	(0.121)	(0.0937)	(0.0957)		(0.0925)	(0.121)	(0.146)	(0.144)		(0.136)
Gender-based rel. mortality ratio (time varying)				-0.0663					-0.0565	
				(0.0768)					(0.119)	
Log total expenditure (2-year lagged)					0.125**					0.0174
					(0.0601)					(0.0743)
Log NIH funding (2-year lagged)	0.384***	0.156*	0.157	0.154	0.188*					
	(0.0674)	(0.0915)	(0.0986)	(0.0983)	(0.101)					
Log privately sponsored trials (2-year lagged)						0.538***	0.295*	0.245	0.239	0.309*
						(0.0939)	(0.174)	(0.183)	(0.183)	(0.183)
Observations	1,197	1,197	1,197	1,197	998	1,161	1,161	1,161	1,161	953
Year FEs	No	No	Yes	Yes	Yes	No	No	Yes	Yes	Yes
Clusters	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Adjusted R sq	0.221	0.454	0.456	0.454	0.500	0.246	0.322	0.340	0.337	0.345
E[Y]	2.532	2.532	2.532	2.532	2.614	16.58	16.58	16.58	16.58	2.776
sd[Y]	1.504	1.504	1.504	1.504	1.525	1.837	1.837	1.837	1.837	1.462
E[race ratio]	1.260	1.260	1.260	1.255	1.229	1.254	1.254	1.254	1.240	1.225
sd[race ratio]	1.165	1.165	1.165	1.174	1.058	1.160	1.160	1.160	1.136	1.038

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table S11: Robustness 2 (Private Sector)–restricting sample to avoid classification errors.

	Log privately sponsored clinical trials						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Log total deaths (2-year lagged)		0.158**	0.157**	0.161**	0.155***	0.154**	0.159***
		(0.0648)	(0.0653)	(0.0632)	(0.0556)	(0.0608)	(0.0587)
Log publications (2-year lagged)		0.482***	0.486***	0.479***	0.498***	0.455***	0.446***
		(0.112)	(0.115)	(0.111)	(0.0798)	(0.120)	(0.117)
Race-based rel. mortality ratio	-0.0832	-0.186**	-0.186**		-0.153*	-0.206*	
	(0.109)	(0.0868)	(0.0878)		(0.0866)	(0.109)	
Gender-based rel. mortality ratio	-0.0754	-0.0954	-0.0975		-0.111	-0.0995	
	(0.122)	(0.0953)	(0.0964)		(0.113)	(0.0933)	
Race-based rel. mortality ratio (time varying)				-0.175**			-0.187*
				(0.0862)			(0.111)
Gender-based rel. mortality ratio (time varying)				-0.0815			-0.0794
				(0.0689)			(0.0626)
Log total expenditure (2-year lagged)						0.175***	0.177***
						(0.0562)	(0.0553)
Observations	931	761	761	761	82	572	572
Sample	from 2008	from 2008	from 2008	from 2008	2015	from 2008	from 2008
Year FEs	No	No	Yes	Yes	-	Yes	Yes
Clusters	Yes	Yes	Yes	Yes	-	Yes	Yes
Adjusted R sq	0.00503	0.431	0.428	0.426	0.500	0.486	0.481
E[Y]	2.588	2.593	2.593	2.593	2.670	2.712	2.712
sd[Y]	1.490	1.480	1.480	1.480	1.468	1.515	1.515
E[race ratio]	1.263	1.271	1.271	1.262	1.324	1.212	1.212
sd[race ratio]	1.229	1.233	1.233	1.216	1.324	1.077	1.077

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table S12: Robustness 2 (Public Sector)–restricting sample to avoid classification errors.

	Log NIH funding						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Log total deaths (2-year lagged)		0.288***	0.289***	0.283***	0.267***	0.270***	0.273***
		(0.0789)	(0.0796)	(0.0769)	(0.0780)	(0.0860)	(0.0831)
Log publications (2-year lagged)		0.415***	0.413***	0.418***	0.394***	0.281	0.275
		(0.148)	(0.150)	(0.147)	(0.112)	(0.174)	(0.169)
Race-based rel. mortality ratio	0.398***	0.362***	0.364***		0.351***	0.274**	
	(0.112)	(0.0932)	(0.0940)		(0.122)	(0.121)	
Gender-based rel. mortality ratio	-0.0872	-0.0810	-0.0816		-0.0556	-0.179	
	(0.110)	(0.142)	(0.142)		(0.159)	(0.128)	
Race-based rel. mortality ratio (time varying)				0.370***			0.292**
				(0.0963)			(0.130)
Gender-based rel. mortality ratio (time varying)				-0.0721			-0.132
				(0.115)			(0.107)
Log total expenditure (2-year lagged)						0.0418	0.0374
						(0.0693)	(0.0690)
Observations	1,000	815	815	815	83	575	575
Sample	from 2008	from 2008	from 2008	from 2008	2015	from 2008	from 2008
Year FEs	No	No	Yes	Yes	-	Yes	Yes
Clusters	Yes	Yes	Yes	Yes	-	Yes	Yes
Adjusted R sq	0.0723	0.419	0.417	0.416	0.401	0.360	0.357
E[Y]	16.57	16.60	16.60	16.60	16.55	16.94	16.94
sd[Y]	1.922	1.933	1.933	1.933	1.928	1.808	1.808
E[race ratio]	1.294	1.302	1.302	1.285	1.313	1.249	1.249
sd[race ratio]	1.280	1.285	1.285	1.245	1.320	1.144	1.144

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table S13: Robustness 3 (Private Sector)– changing lag structure.

	Log privately sponsored clinical trials						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Log total deaths (3-year MA lagged)		0.164**	0.167**	0.170***	0.151**	0.161**	0.166***
		(0.0624)	(0.0643)	(0.0628)	(0.0584)	(0.0618)	(0.0603)
Log publications (3-year MA lagged)		0.488***	0.485***	0.480***	0.500***	0.440***	0.436***
		(0.114)	(0.120)	(0.118)	(0.0800)	(0.128)	(0.126)
Race-based rel. mortality ratio	-0.0933	-0.190**	-0.189**		-0.153*	-0.206*	
	(0.107)	(0.0777)	(0.0786)		(0.0877)	(0.113)	
Gender-based rel. mortality ratio	-0.103	-0.106	-0.104		-0.117	-0.125	
	(0.101)	(0.0912)	(0.0933)		(0.115)	(0.0923)	
Race-based rel. mortality ratio (time varying)				-0.182**			-0.195*
				(0.0749)			(0.107)
Gender-based rel. mortality ratio (time varying)				-0.0849			-0.0993
				(0.0740)			(0.0713)
Log total expenditure (3-year MA lagged)						0.154**	0.154**
						(0.0712)	(0.0702)
Observations	1,368	1,171	1,171	1,171	80	906	906
Sample	Full	Full	Full	Full	2015	Full	Full
Year FEs	No	No	Yes	Yes	-	Yes	Yes
Clusters	Yes	Yes	Yes	Yes	-	Yes	Yes
Adjusted R sq	0.00899	0.427	0.427	0.425	0.492	0.459	0.456
E[Y]	2.444	2.531	2.531	2.531	2.670	2.661	2.661
sd[Y]	1.501	1.511	1.511	1.511	1.465	1.545	1.545
E[race ratio]	1.245	1.248	1.248	1.244	1.339	1.192	1.192
sd[race ratio]	1.156	1.152	1.152	1.157	1.337	0.980	0.980

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table S14: Robustness 3 (Public Sector)– changing lag structure.

	Log NIH funding						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Log total deaths (3-year MA lagged)		0.257***	0.272***	0.271***	0.266***	0.237***	0.239***
		(0.0792)	(0.0804)	(0.0793)	(0.0799)	(0.0866)	(0.0856)
Log publications (3-year MA lagged)		0.437***	0.398**	0.400**	0.379***	0.273	0.273
		(0.157)	(0.161)	(0.159)	(0.110)	(0.181)	(0.180)
Race-based rel. mortality ratio	0.317***	0.270***	0.278***		0.373***	0.186	
	(0.110)	(0.0932)	(0.0931)		(0.120)	(0.118)	
Gender-based rel. mortality ratio	-0.0874	-0.0839	-0.0669		-0.0699	-0.141	
	(0.106)	(0.146)	(0.144)		(0.158)	(0.138)	
Race-based rel. mortality ratio (time varying)				0.254***			0.160
				(0.0860)			(0.103)
Gender-based rel. mortality ratio (time varying)				-0.0613			-0.116
				(0.115)			(0.109)
Log total expenditure (3-year MA lagged)						0.112	0.107
						(0.0832)	(0.0824)
Observations	1,564	1,304	1,304	1,304	81	968	968
Sample	Full	Full	Full	Full	2015	Full	Full
Year FEs	No	No	Yes	Yes	-	Yes	Yes
Disease FEs	No	No	No	No	-	No	No
Clusters	Yes	Yes	Yes	Yes	-	Yes	Yes
Adjusted R sq	0.0423	0.363	0.391	0.388	0.403	0.363	0.361
E[Y]	16.16	16.30	16.30	16.30	16.51	16.49	16.49
sd[Y]	1.948	1.937	1.937	1.937	1.930	1.884	1.884
E[race ratio]	1.285	1.290	1.290	1.290	1.328	1.239	1.239
sd[race ratio]	1.235	1.229	1.229	1.282	1.333	1.093	1.093

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table S15: Robustness 4 (Private Sector)– using different measures for Black vs. White diseases.

	Log privately sponsored clinical trials						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Log black deaths (2-year lagged)	-0.0286	-0.291**	-0.292**	-0.288**	-0.241	-0.284*	-0.280*
	(0.169)	(0.136)	(0.138)	(0.138)	(0.158)	(0.159)	(0.159)
Log white deaths (2-year lagged)	0.338*	0.449***	0.455***	0.454***	0.400**	0.443***	0.443***
	(0.175)	(0.130)	(0.132)	(0.132)	(0.158)	(0.160)	(0.160)
Log publications (2-year lagged)		0.493***	0.488***	0.484***	0.500***	0.446***	0.442***
		(0.113)	(0.119)	(0.116)	(0.0800)	(0.122)	(0.121)
Gender-based rel. mortality ratio	0.0478	-0.126	-0.121		-0.118	-0.146	
	(0.0967)	(0.0960)	(0.0993)		(0.114)	(0.0936)	
Gender-based rel. mortality ratio (time varying)				-0.101			-0.116
				(0.0780)			(0.0715)
Log total expenditure (2-year lagged)						0.141**	0.139**
						(0.0616)	(0.0612)
Observations	1,242	1,242	1,242	1,242	82	1,031	1,031
Sample	Full	Full	Full	Full	2015	Full	Full
Year FEs	No	No	Yes	Yes	-	Yes	Yes
Clusters	Yes	Yes	Yes	Yes	-	Yes	Yes
Adjusted R sq	0.209	0.417	0.420	0.420	0.500	0.455	0.453
E[Y]	2.508	2.508	2.508	2.508	2.670	2.585	2.585
sd[Y]	1.507	1.507	1.507	1.507	1.468	1.534	1.534
Wald Stat	1.175	8.148	8.083	7.967	4.244	5.408	5.336
P-Value	0.281	0.00530	0.00548	0.00581	0.0428	0.0224	0.0233

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table S16: Robustness 4 (Public Sector)– using different measures for Black vs. White diseases.

	Log NIH funding						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Log black deaths (2-year lagged)	0.528**	0.314*	0.326*	0.327*	0.407*	0.195	0.199
	(0.207)	(0.179)	(0.183)	(0.181)	(0.230)	(0.210)	(0.209)
Log white deaths (2-year lagged)	-0.145	-0.0784	-0.0737	-0.0744	-0.167	0.0387	0.0372
	(0.215)	(0.166)	(0.171)	(0.170)	(0.230)	(0.191)	(0.191)
Log publications (2-year lagged)		0.458***	0.412**	0.411**	0.425***	0.300*	0.297*
		(0.159)	(0.162)	(0.160)	(0.117)	(0.178)	(0.176)
Gender-based rel. mortality ratio	0.0929	-0.0786	-0.0589		-0.0704	-0.127	
	(0.123)	(0.149)	(0.147)		(0.166)	(0.138)	
Gender-based rel. mortality ratio (time varying)				-0.0545			-0.105
				(0.118)			(0.109)
Log total expenditure (2-year lagged)						0.0910	0.0893
						(0.0714)	(0.0712)
Observations	1,390	1,390	1,390	1,390	83	1,108	1,108
Sample	Full	Full	Full	Full	2015	Full	Full
Year FEs	No	No	Yes	Yes	-	Yes	Yes
Clusters	Yes	Yes	Yes	Yes	-	Yes	Yes
Adjusted R sq	0.233	0.344	0.379	0.379	0.355	0.369	0.368
E[Y]	16.26	16.26	16.26	16.26	16.55	16.43	16.43
sd[Y]	1.941	1.941	1.941	1.941	1.928	1.890	1.890
Wald Stat	2.603	1.357	1.344	1.367	1.602	0.157	0.169
P-Value	0.110	0.247	0.249	0.245	0.209	0.693	0.682

Robust standard errors in parentheses

*** p<0.01, ** p<0.05, * p<0.1

Table S17: Oster Test to Evaluate the Importance of Omitted Variable Bias.

	NIH funds		Private clinical trials	
	Rmax=1	Rmax=2R	Rmax=1	Rmax=2R
Relative Selection on Unobservables	6.7	10.1	-2.7	-3.1