Effects of Changes in Federal Funding for Academic Life Sciences R&D: Crowding-In versus Crowding-Out in the Post-Doubling Era

Margaret E. Blume-Kohout[a]

Krishna B. Kumar[b]

Neeraj Sood[c]

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[a] Corresponding author. Robert Wood Johnson Foundation Center for Health Policy and Department of Economics, MSC05 3060, University of New Mexico, Albuquerque, NM 87131-0001. Tel. 505-277-5304. Email: megbk@unm.edu.

[b] Senior Economist and Director of Labor and Population, RAND Corporation. 1776 Main Street, Santa Monica, CA 90401. Tel. 310-393-0411 x7589. Email: kumar@rand.org.

[c] Schaeffer Center for Health Policy and Economics, and Department of Pharmaceutical Economic and Policy, University of Southern California, 3335 South Figueroa Street, University Park Campus, UGW-Unit A, Los Angeles, CA 90089. Tel. 213-821-7949. Email: nsood@healthpolicy.usc.edu.

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Abstract

This paper evaluates effectiveness of federal research funding in stimulating universities' total research and development (R&D), over a period of dramatic change in the federal funding environment. Instrumental variables estimation reveals that during the NIH budget doubling, 1998—2003, each federal research dollar spurred an additional $0.27 in subsequent research funding from non-federal sources. In contrast, in the more competitive post-doubling environment, any increase in universities' federal funding was typically offset by nearly equal decrease in funding from non-federal sources. However, for non-PhD-granting and less research-intensive institutions, federal R&D funding continued to yield larger, positive effects, indicative of signaling.

I. INTRODUCTION

Federal agencies spend billions of taxpayer dollars funding academic research each year. In 2009, total federal obligations for life sciences research and development (R&D) at academic institutions totaled $15.8 billion, of which 90%, or $14.2 billion, was provided by the National Institutes of Health (NIH).[1] The American Recovery and Reinvestment Act of 2009 (ARRA) provided an additional $8.2 billion to NIH to fund extramural life sciences R&D. Public expenditures for scientific R&D are generally justified by assuming there exists market failure, that is, inadequate incentive for private investment in R&D (Arrow 1962). To the extent that research investments lead to discoveries and innovations that improve population health and productivity, support of R&D may also help fuel economic growth (see for example, Bloom, Canning, & Sevilla (2004)). Under ARRA, increases in federal funding for R&D were further justified as a means to speed economic recovery, by increasing overall spending and employment opportunities at grantee institutions and in those institutions' local economies.

The effectiveness of federal R&D funding in generating innovative medical treatments—or, for that matter, in generating productivity-enhancing innovations more broadly—depends in large part on how non-federal funders and recipient institutions themselves respond to changes in the institutions' federal R&D funding levels. For example, a federal grant that subsidizes investment in facilities, capital or equipment, or that supports investigators' skill development, might increase the university's research productivity, thereby making investigators at that institution more competitive in attracting subsequent non-federal research support. Such investments in physical and human capital at a university—sometimes nominally in support of a particular research project, investigator, or group of investigators—may also have spillover effects,

increasing resources available to other investigators at that institution. Pharmaceutical firms might observe certain faculty receiving federal funds for basic biomedical research with potential spillovers to those firms' own pipelines, spurring those firms to form alliances and invest in collaborative research. And for universities and colleges with less well-established research reputations, successful applications for federal R&D funding may bestow legitimacy, serving as a signal of research quality for non-federal funders. In any of these cases, a one-dollar increase in federal R&D subsidies to U.S. universities could yield more than a dollar increase in total R&D expenditures, due to complementary funding from non-federal sources.

On the other hand, because applying for funding is time-consuming, investigators may be deterred by high opportunity cost from submitting additional applications to non-federal funders, once their federal funding application is approved. In a static sense, if investigators' marginal utility is diminishing in income, they may simply be less inclined to pursue additional funds once some target income is achieved (Andreoni and Payne 2003, 2010). Similarly, fully-informed philanthropic funders may be reluctant to invest additional funding if they perceive an individual investigator or university is already awash in funds, as the impact of the gift may appear diminished (Duncan 2004). For any or all of these reasons, a one-dollar increase in federal R&D funding could be offset by a decrease in non-federal research funds (i.e., "crowd-out"), and thus ultimately yield less than a dollar increase in total university R&D expenditures.[2]

From a dynamic perspective, due to their binding time constraints, investigators must trade off time spent on current research progress versus time spent on applications for funding to support their future work. Both of these activities have uncertain outcomes, but the expected payoff is generally presumed to be increasing with effort, and success in either endeavor may also increase their

chances of receiving future research funding from both public and private sources. A rational investigator's decision whether to pursue federal funds should then take into account two probabilities, each of which will likely take on different values depending both on the overall funding environment and on specific characteristics of the investigator and his or her institution. First, investigators should consider the probability that a federal grant application they submit will get funded. When federal funding is relatively loose, if we assume the number of eligible applicants remains reasonably constant, then each investigator can spend less time and write fewer applications to achieve the same target level of funding. On the other hand, if application success rates are relatively high, that looser funding environment might also encourage investigators to pursue higher overall levels of funding, from all sources.

The second probability relevant to their decision relates to the signaling power or complementarity of federal funding. Institutions aiming to attract more R&D funding should consider whether an additional successful federal application would increase their institution's probability of receiving funding from non-federal sources in the future. In a looser federal R&D funding environment, successful federal applications may not be viewed by non-federal funders as conveying much information about the quality of the research conducted. Put another way, the prestige effect may be lower, and thus non-federal funders may not be as responsive to the signal. On the other hand, if federal R&D funds are used to improve an institution's research infrastructure, physical and human capital, such improvements may have longer-term impact on the institution's research quality and productivity, both of which would likely increase the probability of successful future applications for federal and non-federal funding.

As this informal theoretical analysis suggests, the expected payoffs—and thus individual investigators' time allocation decisions and resulting institutional funding outcomes—are likely to differ for investigators at smaller and less highly-ranked universities or colleges with lower total R&D expenditures, versus those at larger and more established research institutions. Specifically, when funding is loose, historically less research-intensive institutions may pursue more federal funding and seek to build infrastructure and human capital, to improve their likelihood of future funding from all sources. These institutions have higher incentive to seek additional federal funding than larger well-established sources, due to the combined potential benefits of increased research productivity and quality and the reputation or signaling effect.

In contrast, for larger, well-established research institutions that have a track record of successful applications for both federal and non-federal R&D funds, there may be little or no signaling benefit from yet another successful federal grant application. Furthermore, the marginal benefit of additional federal dollars in building the institution's long-run research productivity and quality, via increased investment in physical and human capital, may also be lower. As a result, when obtaining federal R&D funding became more competitive in the post-doubling era, investigators at smaller or less prestigious institutions had still greater incentive to pursue federal funds, as the increased rarity of successful applications provided a stronger signal of quality and thus likely increased the institution's probability of subsequent funding from non-federal sources. In contrast, for investigators at well-established research institutions which gain little or no signaling benefits and likely lower long-run marginal increases in research productivity from receipt of federal funding, the lower probability of success per federal application submitted created incentive to substitute towards other research activities. Specifically, because applying for research funding from other

(non-federal) sources and focusing on their current projects' research production became *relatively* more likely to affect their future payoffs, investigators had less incentive to spend time on additional federal applications.

Prior empirical estimates of the effects of federal R&D funding on university productivity, grant-seeking behavior, and private R&D investment have found mixed results. For example, in a recent survey of literature on effects of public subsidies and grants on private sector investment, García-Quevedo (2004) reports that out of 45 U.S.-based studies, 23 studies find evidence of complementarities between public and private funding, 12 studies find evidence of substitution, and the remaining 10 find no significant effects. Focusing specifically on universities, Payne (2001) finds a dollar increase in federal R&D funding increases philanthropic funding—a specific form of complementary funding – by $0.64 to $0.68. On the other hand, Andreoni and Payne (2010) find that government grants to private charitable organizations crowd out private donations, mainly due to reduced fundraising efforts by the organizations themselves. This finding is consistent with evidence from Jacob and Lefgren (2011), who show only modest differences in publication productivity for successful federal grant applicants versus marginal unsuccessful applicants. The authors suggest their result may be explained by fundraising crowd out: marginal unsuccessful applicants for federal funding may attract funding from other sources, while successful applicants may exert less effort to obtain additional non-federal funding.

The mixed results observed in the literature to date may be due to differences in the studies' funding recipients, time periods and associated funding environments, and datasets analyzed, or to differences in the authors' econometric identification strategies. It also seems plausible that heterogeneity in the funding

mechanisms and recipient organizations studied could also contribute to heterogeneity in the estimated effects.

In this paper, we assess the effects of changes in federal funding levels on subsequent non-federal funding in support of life sciences R&D expenditures at U.S. universities and colleges. We also examine differences in these effects due to the changing funding environment during and after the NIH budget doubling, and due to heterogeneity in size, reputation or research intensity across institutions. Our analysis addresses possible omitted variable bias in several ways, but most notably by implementing and validating two instrumental variables for federal life sciences R&D funding.

We find that, during the NIH budget doubling, each additional dollar of federal funding for life sciences research increased universities' non-federally-funded R&D expenditures by $0.27. In contrast, in the post-doubling period, each dollar increase in federal funding was associated with a roughly equal decrease in funding from non-federal sources. Put another way, although real federal funding for academic life sciences R&D declined overall post-2006, universities' total life sciences R&D expenditures remained relatively constant, due to increased funding from non-federal sources. Our results also indicate that universities in the lowest tercile of historical federal funding, on average, experience greater increases in non-federal funding for each federal dollar, both during the budget doubling and today. These larger effects for universities with smaller research portfolios may indicate complementarity or signaling effects, as discussed above.

The rest of this paper proceeds as follows. In Section II, we discuss the data and present our empirical strategy. We present the results in Section III, and conclude with a discussion in Section IV.

II. DATA AND IDENTIFICATION STRATEGY

Data Sources

Data for this paper are derived from three sources: the National Science Foundation (NSF) Survey of Research and Development Expenditures at Universities and Colleges, administrative records maintained by the Office of Extramural Research at NIH, and historical data on House and Senate appropriations subcommittee representation.

The NSF Survey population includes all U.S. institutions granting bachelors or higher degrees in science and engineering (S&E) fields, and spending at least $150,000 annually in S&E research and development (R&D). Surveyed institutions report their S&E R&D expenditures by funding source and field. For example, these data include the amount of life sciences R&D funding received from federal versus all other sources. Unfortunately, prior to 2010, these data did not contain a detailed breakdown for each R&D field by type of non-federal funding source, for example whether due to philanthropic donations, state government, or industry. However, beginning with fiscal year 2010, the NSF's new Higher Education Research and Development (HERD) Survey provides this information. We observe that in FY2010, 62% of life sciences R&D expenditures were federally funded, with institutional (18%), nonprofit (7%), state and local government (6%), industry (5%), and other sources making up the remainder.

For this analysis, we extracted R&D expenditures by year, field and source for 1998 through 2010. We restrict our analysis to this period because, prior to FY 1998, in some years only a sample of institutions from the target population was surveyed. However, since FY1998, NSF has conducted a population survey, with very high response rates. For example, for FY2009, the NSF reports a response rate of 97.6%. Using population survey data presents a

clear advantage for our inference, as any point estimates represent true population averages rather than sample averages.

Our dependent variable is life sciences R&D funding from all non-federal sources, and our key explanatory variable is federally-funded life sciences R&D expenditures, lagged by one year. Life sciences R&D includes research in agricultural, biological, and medical sciences, as well as allied health professions; however, since 1998 over half of academic R&D expenditures in the life sciences have been for medical research, and this share has continued to grow over time. Finally, in some models we include covariates for universities' federally- and non-federally funded R&D in fields other than life sciences. Our dataset also includes institutional characteristics such as whether the institution is public or private, whether it grants PhDs in S&E fields, and its 2005 Carnegie Classification.

NIH administrative data include, for each grant awarded from 1975 onwards, the grant or contract's unique ID number, the fiscal year of the award, principal investigator's institution (including institution name, city, and state), and the financial amount of the award. As discussed below, we use NIH award data for 1993 through 1996 to calculate each university's base-period share of funding from each NIH Institute or Center (NIC). These base-period shares permit us to construct one of our two instruments for federal life sciences R&D funding, *predicted NIH funding*. Note that the sum of actual NIH awards for each university-year differs from our key explanatory variable in that, although NIH is the lead federal agency funding academic life sciences research, other federal agencies such as the Centers for Disease Control (CDC) and Food and Drug Administration (FDA) also provide life sciences R&D funding to universities. In addition, while it is true that NIH extramural R&D funding primarily supports basic and applied life sciences research, the NIH also funds research in other fields, such as social and behavioral sciences. Thus, while these measures do

overlap, they are not identical. For consistency, we rely on the NSF Survey data for both our dependent variable and our key explanatory variables.

We matched institutions across these two datasets in an iterative process. First, we found all exact matches by institution name and state. Then, we extracted all remaining awardees in the NIH data that were coded as domestic institutions of higher education, and matched these institutions by hand with those listed in the NSF survey. Finally, we included in our analytic dataset only those institutions for which actual (non-imputed) NSF survey data were available for each year in our study period, 1998-2010, and for which NIH awards were found in the administrative data during the base period 1993-1996. This process limited our panel to 228 institutions. In 2009, the universities included in our panel spent over $29 billion on life sciences R&D, representing approximately 86% of all U.S. university life sciences R&D expenditures that year.[3] Thus, despite our restrictions, these data nonetheless capture the majority of total funded university research activity in the life sciences.

Table 1 provides descriptive statistics for our analytic dataset. Among the universities and colleges in our panel, average annual federal life science funding was $64.0 million (2010 dollars), and average annual non-federal life science funding per university was $46.2 million (2010 dollars), with federal funding representing about 60% of total life sciences R&D funding. Average annual federal funding per university for all other S&E fields was $44.8 million (2010 dollars), accounting for a similar fraction of total R&D funding in these fields. Approximately one-third of the 228 institutions in our panel are private, and three-quarters were classified as Doctorate-granting Universities by the Carnegie Foundation in 2005, indicating they awarded at least 20 research doctorates that year.

Figure 1 shows the trends in federal and non-federal life sciences R&D funding for our panel. From 1998 to 2000, growth in funding from non-federal sources kept pace with increases in federal funding, but university life sciences R&D funding from both sources grew at about the same rate, but from 2001 through 2004, growth in federal funding significantly outpaced growth in non-federal funding. From 2006 onwards, real life sciences R&D expenditures at the universities in our panel remained fairly constant overall, despite a declining share of federal funding, due to increased funding from non-federal sources.

Figure 2 shows that, although the NIH budget doubling benefited both highly research-intensive and less research-intensive institutions in relatively equal proportions, as funding became more competitive in the post-doubling era, cutbacks disproportionately affected institutions that historically received lower levels of NIH funding. In particular, institutions in the highest tercile of historical funding saw little change in their real, federally-funded R&D expenditures, whereas institutions in the lowest tercile experienced significant declines in federal funding overall.

Empirical Methods

We employ several empirical strategies to estimate the relationship between federal and non-federal R&D funding at universities. For all analyses, the unit of observation is the university-year, and standard errors are clustered at the university level to accommodate possible within-cluster autocorrelation. We first conduct descriptive analyses to examine the association between federal and non-federal funding for life sciences at research universities. Then, we estimate four different sets of multivariate linear regression models to investigate the possibility of a causal relationship between federal and non-federal R&D funding. Each set

of multivariate regression models incrementally controls for observed and unobserved university characteristics that could bias our estimates of the causal relationship between federal and non-federal funding.

Our first, descriptive regression estimates the simplest model:

$$NonFederal_{u,t} = \alpha_0 + \alpha_1 Federal_{u,t-1} + \tau + \varepsilon_{u,t} \quad (1)$$

where $Federal_{u,t-1}$ is the one-year lag of federally-funded life science R&D expenditures at university u in year t - 1; $NonFederal_{u,t}$ is current non-federal funding for life sciences at university u in year t, τ is a vector of year fixed effects that non-parametrically controls for secular changes in non-federal funding over time (for example, due to changes in economic conditions); α_0 is a constant; and $\varepsilon_{u,t}$ is the error term. The key coefficient of interest is α_1, which estimates the change in non-federal funding associated with a dollar increase in federal funding the previous year.

We have two reasons for using lagged federal funding as our key explanatory variable. First, as discussed in the introduction, we anticipate there may exist a lag between universities' receipt of federal R&D funding and dissemination of that information to non-federal funders. Second, to some extent, lagging federal funding insulates us from exogenous shocks that might simultaneously increase both federal and non-federal funding, for example, if the institution recruits a new senior faculty member who brings diverse, already-established sources of research funding.

However, α_1 may nonetheless suffer from omitted variables bias. For example, universities with more faculty members or stronger research reputations may attract both greater federal and greater non-federal funding over time. These omitted variables would yield a positive association between federal and non-federal funding, even if no causal relationship exists, biasing the estimate α_1

upward. Our second regression model addresses this source of bias by including university fixed effects.

We estimate:

$$NonFederal_{u,t} = \beta_0 + \beta_1 Federal_{u,t-1} + \tau + \mu + \varepsilon_{u,t} \quad (2)$$

where μ is a vector of university fixed effects that control for time-invariant differences across universities in their average levels of non-federal life sciences R&D funding. In contrast to equation (1) which exploited variation in funding levels both across and within universities, this model exploits only the variation within individual universities' prior year federal funding to estimate the effect on non-federal funding. In effect, this model estimates whether a university that received more federal funding (relative to its average over the study period) in year *t* receives more (or less) non-federal funding in year *t*+1.

In this second specification, β_1 could be biased if time-varying university characteristics are correlated with growth or decline in federal and non-federal funding. For example, as discussed by Lawler (2003), growth in non-federal (specifically, industry) funding at top research universities such as the University of California—Berkeley and MIT may reflect strategic initiatives by university administration to diversify funding sources. If such initiatives occurred simultaneously with increases in federal fundraising activity, we might erroneously conclude that federal funding *caused* growth in non-federal funding.

Our third regression model is designed to control for additional bias arising from such time-varying university characteristics or initiatives, through inclusion of covariates controlling for federal and non-federal R&D funding the university received in other S&E fields:

$$\begin{aligned} NonFederal_{u,t} = {} & \gamma_0 + \gamma_1 Federal_{u,t-1} + \tau + \mu \\ & + \gamma_2 OtherNonFed_{u,t} \\ & + \gamma_3 OtherFed_{u,t-1} \end{aligned} \quad (3)$$

In this third model we include contemporaneous non-federal R&D funding in other S&E fields to control for year-to-year differences in the extent to which universities seek non-federal funding. Because university policies with respect to non-federal funding may also be correlated with other measures of research quality and productivity, failure to control for these efforts could yield biased estimates of γ_1. We also include lagged federal funding for other S&E fields as a covariate to test the counterfactual. That is, given that federal funding for non-life sciences R&D is not expected to impact non-federal funding for life sciences, as long as our estimation strategy adequately controls for time-varying university characteristics that impact the university's overall R&D funding, we should see no significant correlation between changes federal funding levels for other S&E fields and life sciences R&D funding from non-federal sources.

Instrumental Variables Estimation

Finally, we employ instrumental variables (IV) estimation to mitigate potential bias from any remaining omitted or unobserved time-varying university characteristics. The challenge in implementing the IV estimator in this context is to find one or more instruments that are strongly correlated with changes in federal research funding at universities, but uncorrelated with other types of shocks that might affect non-federal research funding levels at a given university over time. We propose and validate two types of instruments for federal life sciences R&D funding, as described below: predicted NIH funding, and Congressional representation. Then, we use these instruments to estimate the following model, via two-stage least squares:

$$\begin{aligned}\overline{Federal}_{u,t} = {} & \theta_0 + \theta_1 \overline{NIH}_{u,t} + \theta_2 CongRep_{u,t} \\ & + \theta_3 OtherNonFed_{u,t} + \theta_4 OtherFed_{u,t-1}\end{aligned} \tag{4}$$

$$NonFederal_{u,t} = \delta_0 + \delta_1 \overline{Federal}_{u,t-1} + \tau + \mu + \delta_2 OtherNonFed_{u,t} + \delta_3 OtherFed_{u,t-1} \quad (5)$$

Instrument 1: Predicted NIH Funding

We construct the predicted NIH funding instrument as a function of the share of a given university's funding received from each NIH Institute or Center (NIC) in our base period, 1993 through 1996, and the overall growth (or decline) each NIC's budget, each year. For sensitivity testing, we also constructed an alternative instrument using an earlier period, 1975 through 1984, for the base shares. Specifically, predicted NIH funding, $\overline{NIH}_{u,t}$, is given by:

$$\overline{NIH}_{u,t} = NIH_{u,1997} \sum_i \left(\frac{Budget_{i,t}}{Budget_{i,1997}} \right) * share_{i,u,b} \quad (6)$$

where $NIH_{u,1997}$ is the actual NIH funding obligated for university u in 1997; $Budget_{i,t}$ is the total annual budget appropriation for NIC i in year t; and $share_{i,u,b}$ is the share of university u's total NIH funding that came from NIC i during the base period, b. Equation (6) shows that annual percentage changes in a given university's predicted NIH funding are equal to the weighted average annual percentage change across the NIC budgets, where the weight applied for each NIC equals the share of the university's total NIH R&D funding provided by that NIC during the base period. This equation can equivalently be derived by keeping university u's share of NIC i's budget constant over time. Because each of the NICs specializes in particular diseases, areas of human development, or aspects of research support (see Smith (2006)), each university's distribution of base-period shares across the NICs reflects its particular historical research strengths.

Instrument 2: Congressional Representation

Congressional appropriations for each NIC are determined through political negotiations in House and Senate appropriations subcommittees, together with other federal spending plans for health, education, and so on. After the House and Senate have passed their individual versions of the appropriations bill, differences are resolved in conference, resulting in a final product called the conference report. This conference report provides budget appropriations for each NIC, and although the appropriations bill and conference reports almost never earmark funds for specific universities, the conference report frequently includes non-binding "report language" encouraging the NICs to pursue research in specific areas. By urging the NICs to support certain specific areas of research in which their constituencies specialize, committee members may indirectly increase opportunities for their represented universities' research funding (Hegde 2009).

Over 80 percent of total NIH appropriations are awarded as competitive extramural research grants, primarily to support life sciences research at U.S. universities. Prior work by Payne and Siow (2003) indicates that universities with alumni serving on relevant Congressional appropriations committees tend to receive more federal R&D funding. Hedge and Mowery (2008) also show that each additional representative on the Congressional subcommittee responsible for NIH appropriations is associated with a 5.9% increase in NIH funding for institutions in their state overall, and with an even stronger 8.8% increase in NIH funding for public institutions in their state.

For this paper, we investigated three alternative political representation instruments, based on the state and Congressional district for each university in each year: district-level representation on the House appropriations subcommittee, state-level representation on the House appropriations subcommittee, and state-level representation on the Senate appropriations subcommittee. Like Hegde and

Mowery (2008), we find that changes in Senate appropriations subcommittee representation are generally not a significant predictor of changes in funding. However, we do find that state-level House appropriations subcommittee representation is a useful predictor of university funding. The state-level representation variable comprises both any district-level representation the university may have, as well as political representation for other districts in the same state. Since the majority of Congressional representatives attend universities in their home state, albeit rarely in the same district they ultimately represent, the state-level representation instrument also largely encompasses alumni representation. The relevance of this instrument is thus consistent with prior literature.

Instrument Validity and Sensitivity Analysis

The relevance condition is easily tested, by calculating the partial F-statistic for the excluded instruments in the first-stage regression shown in equation (5). We do so, and find strong evidence of relevance. For example, for the full panel IV regression presented in Table 2, the F-statistic exceeds 50, well above the Stock & Yogo (2002) critical value. Because Hegde (2009) found that Congressional representation most affects research institutions with historically lower R&D funding levels and public universities, we also test to see that our excluded instruments pass the relevance condition in our subpanel regressions for universities with historically lower or higher levels of federal R&D funding.

To validate our exogeneity assumption, we performed several additional analyses. As shown in equation (6), variation over time in the predicted NIH funding instrument derives from differences across universities in the shares of total NIH funding they received from each NIC during the base period, and differences in growth rates across the NICs' aggregate budget appropriations in

subsequent years. The validity of our exogeneity assumption for the predicted NIH funding instrument therefore relies on the following conditions: (1) base-period specialization across research fields, as represented by the share of research funding a university receives from each NIC, must be uncorrelated with other institutional characteristics that make a university more or less likely to obtain non-federal funding in later periods; and (2) relative changes in individual NIC budget appropriations year-to-year cannot be correlated with other unobserved factors affecting universities' non-federal funding in subsequent years. We believe these are plausible assumptions, for the reasons discussed below.

First, we considered the possibility that universities might have chosen their research specializations strategically during the base period, 1993-1996, anticipating faster growth in the future for particular NICs. To address this, we constructed and implemented a second predicted NIH funding instrument, using a much earlier base period, 1975-1984. Given the long lag between this earlier base period and our analytic time series, and given that year-to-year appropriations for the NICs are determined by political processes that are clearly sensitive to current changes in political representation, it seems implausible that institutions could have anticipated and acted strategically two decades prior, to take advantage of the differential growth rates across the NICs during our study period 1998-2009. This alternative instrument yields very similar results when used alone, and also improves first-stage prediction for some types of institutions. In the Results section, we therefore present models employing both of these predicted NIH funding instruments.

Second, we considered whether disease-specific technological shocks might simultaneously increase both federal and non-federal R&D for particular diseases, thus increasing availability of both types of funding for universities

specializing in a given disease category. Within each NIC, research funding for each field (and, therefore, the amount of funding available to universities specializing in one research field versus another) is highly decentralized, and funding levels by disease or specialized research area reflect not only the social and economic costs of particular diseases, but also the quality of investigator-initiated proposals received by each NIC and their potential for scientific progress. Thus, while it is certainly conceivable that technological opportunity might simultaneously increase both federal and non-federal funding for one particular disease (and thus potentially provide increased availability of both types of funding for a particular university), such within-NIC shifts in research priorities are unlikely to be reflected in the NIC's total appropriation by Congress, determined in budget negotiations the previous year.

We tested this notion empirically, investigating whether increases in NIH funding by NIC were associated with contemporaneous increases in industry R&D for the diseases each NIC represents. Due to lack of publicly available data on industry R&D expenditures by disease, we use drugs entering Phase I trials for each disease as a proxy. Phase I trials—the first trials of new investigational compounds conducted in humans to establish safety and metabolism—have previously been shown to be associated with lagged changes in NIH disease-specific funding (Blume-Kohout 2012). We matched the clinical indication for each drug investigation with the lead NIC for each disease, as identified on the National Library of Medicine's MedlinePlus website. This assignment was supplemented by analysis of the fraction of grants awarded by each NIC that were classified to each disease in 2006, following the algorithm in Blume-Kohout (2009). Estimating Poisson models with NIC and year fixed effects, and with robust standard errors clustered on NIC, we found no significant relationship between contemporaneous NIH funding and pharmaceutical R&D. These results

support our assumption that shocks to federal R&D funding of universities due to changes in NIC budgets are unlikely to be accompanied by contemporaneous shocks to university funding from industry sources; however, we cannot rule out contemporaneous shocks to university funding from state governments or other non-federal sources, including strategic funding by not-for-profit private foundations (Feldman and Graddy-Reed 2012).

Third, we assessed possible correlation between universities' base-period specializations and other observable characteristics associated with higher levels of non-federal R&D. For example, if PhD-granting universities were more likely to specialize in research fields that were funded by NICs that grew most rapidly after 1997, that could invalidate our instrument. To investigate this possibility, we tested whether the share of funding each university received in the base period from each NIC was correlated with other observable characteristics, including Carnegie Classification, public versus private control, and so on. Applying the Bonferroni correction α/N for multiple regressions, where $\alpha=.05$ and N represents the 20 NIH Institutes and Centers over which we calculate institution base shares, we found only two significant correlations: non-PhD-granting colleges and universities received a significantly higher baseline share of funding from the National Institute of General Medical Sciences (NIGMS), and a significantly lower baseline share of funding from the National Heart, Lung, and Blood Institute (NHLBI). However, t-tests comparing year-to-year percentage changes in NIGMS and NHLBI appropriations versus average change across all NICs combined reveals no significant correlation. That is, growth rates in appropriations for NHLBI and NIGMS do not significantly differ from that for other NICs.

Fourth, we estimated "placebo regressions" to test whether changes in universities' federal life sciences funding are associated with changes in non-federal funding for other S&E fields, including engineering, political science, other social sciences, psychology, computer science, mathematics, and environmental sciences. Assuming our IV estimation approach controls adequately for changes over time in universities' fundraising effort, reputation, and so on, we should find little or no relationship between federal life sciences funding and non-federal funding for these largely unrelated fields. As expected, we find no significant effects of federal funding for other fields in any of our regressions.

Finally, for each IV model we estimate, we calculate Hansen's J statistic—a test of overidentifying restrictions—exploiting the availability of multiple instruments, and in all models presented we find no evidence to support rejecting the null of exogeneity. This battery of tests persuades us that predicted NIH funding and Congressional representation are useful instruments for the total federal life sciences R&D funding a university receives, which in turn permits us to attribute causality.

III. RESULTS

In Table 2, we present results from our multivariate regressions. The dependent variable in each case is non-federally-funded life sciences R&D expenditures, and the key explanatory variable is prior-year federally-funded life sciences R&D expenditures. The results from model 1 (corresponding to equation (1)) show that each additional dollar of federal life sciences R&D funding is associated with a \$0.51 increase ($p<.01$) in funding from non-federal sources. However, as discussed above, the lack of any controls for university characteristics in this model likely bias this estimate upwards. For example, universities with larger faculties and/or reputations for higher quality research might receive both more federal and more non-federal funding for life sciences R&D. To address this concern, subsequent models include university fixed effects to control for time-invariant university characteristics.

Results from model 2 (corresponding to equation (2)) indicate that, even after controlling for time invariant university characteristics and secular time trends, a dollar increase in federal funding for life sciences R&D is still associated with a \$0.26 increase ($p<.01$) in non-federal funding for life sciences R&D. Adding covariates for federal and non-federal R&D funding in other fields to control for unobserved time-varying differences in university fundraising efforts, per equation (3), yields a nearly identical estimate, \$0.25 ($p<.01$).

In column 4 of Table 2, we present results from the IV estimation described in equations (5) and (6), for the entire period 1998 through 2009. Taking into account the opposing trends of complementary funding universities received during the NIH budget doubling, followed by substitution effects at many universities after NIH funding stagnated, the average overall effect is further muted: a dollar increase in NIH funding yields only \$0.12 in non-federal

funds. However, this conclusion ignores the strong structural break we observe in Figures 1 and 2 for the post-doubling era.

Table 3 presents results for models estimated separately for the budget doubling period (1998—2003), the funding transition period (2004—2006), and the post-doubling period (2007—2009). Similar to earlier results reported by Blume-Kohout, Kumar, and Sood (2009), we find that during the NIH budget doubling period, a one dollar increase in a university's federal life sciences R&D funding yielded an average $0.27 complementary increase in university life sciences R&D funding from non-federal sources the following year. The partial F-statistic for the excluded instruments again exceeds 50, and we observe no evidence on Hansen's overidentification test to suggest failure of the exogeneity assumption. On the other hand, an endogeneity test for the lagged federal life sciences R&D variable fails to reject the null hypothesis of exogeneity ($p>.78$), suggesting that during the budget doubling period OLS estimation is adequate. Estimating an OLS model equivalent to model 3 in Table 2, but restricting the time series to years prior to 2004, yields a nearly identical result: each federal dollar for life sciences R&D funding increases non-federal funding, on average, by $0.26 ($p<.01$).

The second column of Table 3 provides similar results for the transition period, 2004 through 2006: a one dollar increase in federal life sciences R&D funding during this period yielded an average $0.30 ($p<.05$) increase in funding from non-federal sources the following year. Once again, the first-stage F-statistic exceeds critical values, and there is no evidence to suggest failure of the exogeneity assumption.

In contrast, in the post-doubling period (2007—2009), we find strong evidence of partial to complete substitution. Simple OLS estimation (results not

shown) indicates that, for each federal dollar lost, universities increased life sciences R&D funding from non-federal sources by $0.25. However, IV estimation indicates a much stronger substitution effect, with each federal dollar lost fully compensated by R&D funding from non-federal sources (coeff. estimate -1.11, $p<.01$). Although first-stage identification is somewhat weaker for the post-doubling period (F-statistic for the excluded instruments is 8.3), statistical evidence suggests the IV estimate is still preferable to OLS. Specifically, both a test for endogeneity of the federal life sciences R&D variable and a test for robust inference in the presence of weak instruments—the latter testing whether the the second-stage coefficient is non-zero for the endogenous variable under valid orthogonality conditions—strongly reject their respective null hypotheses ($p<.001$).

Heterogeneous Effects by University Characteristics

As shown in Figure 3, universities responded to the NIH budget doubling and its aftermath differently. We investigated possible heterogeneity in effects of level changes in federal R&D funding based on the following university characteristics: (1) whether the university was above or below the panel median for actual NIH funding in 1997, prior to the budget doubling; (2) whether the university's Carnegie classification for 2005 was as a doctoral research university; and (3), private versus public institutional control. We found that PhD-granting Carnegie classification yielded similar results to simply having higher baseline levels of federal life sciences R&D funding, but private versus public institutional control had little effect after controlling for these other characteristics. Therefore, in this section we focus on the differential effects by historical NIH funding level.

Table 4 compares institutions that historically received relatively lower levels of NIH funding (specifically, those that were below the median for NIH

funding in our panel in 1997), versus those institutions historically above the median for federal funding, during the NIH budget doubling period. We find the lower-volume research institutions experience much greater non-federal return per federal dollar invested. Controlling non-parametrically for differences in overall availability of funding year-to-year for above-median versus below-median institutions, via year fixed effects with and without the above-median interaction term, we find that each federal dollar yielded \$0.76 to \$0.88 ($p<.05$) in non-federal life sciences R&D funding the following year at less research-intensive institutions. However, for historically heavily-funded research universities, we find a significantly smaller effect of each federal dollar on non-federal funding, with the sum of the coefficients estimated at \$0.13 to \$0.20 (Model 1 $p=.144$, Model 2 $p<.01$).

Models 3 and 4 in Table 4 differ from the first two, in that we remove the above- and below-median groups' year fixed effects. So, whereas Models 1 and 2 allowed for differences in total year-to-year funding trends for above- versus below-median institutions—permitting us to disentangle possible signaling effects from secular differences across these two groups in their non-federal funding trends—the latter models show the net effects of changes in federal R&D funding on universities' total life sciences R&D. We find that each federal dollar received at historically less research-intensive institutions yielded \$0.45 to \$0.59 ($p<.10$) in non-federal R&D funding the following year. For the more research-intensive institutions, the estimated effects are again significantly smaller, \$0.21 to \$0.30 ($p<.01$). However, the *difference* between above- and below-median institutions is also smaller in these models, which supports the notion of accumulative advantage, aka the Matthew effect. Overall, non-federal life sciences R&D funding at the historically more research-intensive institutions appears to have grown relatively faster over time.

Table 5 considers differences in effects of changes in federal R&D funding after NIH funding stagnated, from 2007 through 2009. First, we note that for historically less research-intensive institutions, the effect of receiving a dollar in federal R&D funds on funding from non-federal sources is remarkably similar to that during the budget doubling period, $0.73 to $0.76 per federal dollar ($p<.10$). In addition, whereas these universities' overall success in attracting non-federal funding for R&D across S&E fields was not a significant determinant of their non-federal life sciences R&D funding in previous years, now we find less research-intensive universities' propensity to seek non-federal funding more generally is significant as a predictor of their ability to attract non-federal funding for life sciences R&D ($p<.05$).

The apparent complete substitution of declining federal funds with R&D funding from non-federal sources that we observe in Table 3 obscures the dramatic differences for highly-funded versus less-funded institutions as NIH funding became more competitive. As noted above, Figure 2 shows that the lowest tercile institutions by NIH funding were disproportionately impacted during this period. That is, on the margin, these institutions were even less likely to receive federal funding than in the past. Models 1 and 2 in Table 5 suggest that, after controlling for year-to-year differences in non-federal funding trends for below- versus above-median institutions, the signaling effect of a federal dollar received remained largely the same as before for less research-intensive institutions. However, taking into account more research-intensive institutions' greater propensity to attract non-federal funding overall, for these institutions each federal dollar resulted in a $1.37 to $1.49 *decrease* ($p<.01$) in funding from non-federal sources the following year.

Models 3 and 4 in Table 5 again summarize the net effects on total life sciences R&D funding at historically above- and below-median institutions,

including any effects of year-to-year secular changes in total non-federal funding. For less research-intensive institutions, the beneficial signaling effect we observe for those that continued to attract federal funds was offset by the overall decline in availability of federal life sciences R&D funding among institutions in this group. Many institutions in this group likely became abruptly less successful in attracting federal funds, and attempted to compensate for those losses with R&D funds from non-federal sources. The negative relationship for these institutions, which sought to "substitute" non-federal funds for the federal funds they lost, offsets the positive signaling effect we observe for the luckier few, so that overall we detect no significant effect of changes in federal funding on non-federal funding for these institutions. In contrast, at the more research-intensive institutions, the substitution effect clearly dominates, so that overall these universities' total life sciences R&D expenditures remained essentially constant. Each federal dollar for life sciences R&D received by these universities was associated with a $1.12-$1.13 ($p<.001$) decrease in non-federal R&D funding the following year.

IV. DISCUSSION

The efficacy and productivity of public research funding is a controversial issue, especially in periods of fiscal crisis. If increases in public research funding during the budget doubling years had negatively impacted universities' funding from other sources—whether by discouraging private funders from investing, or because researchers put forth less fundraising effort—one might then argue for reducing government investment in biomedical life sciences R&D. Instead, we find that the period of accelerated NIH funding growth, 1998 through 2003, yielded complementary increases in non-federal investment in academic life sciences R&D.

As discussed by Korn et al. (2002), over half of NIH funding goes to fund investigator-initiated research project grants, with average non-competing duration of four years. By FY2007, four years after the doubling ended, with real funding stagnant and research costs continuing to rise, institutions that had been below median for NIH R&D funding a decade earlier (prior to the doubling) experienced disproportionate reductions in their federal R&D support. On the positive side, institutions in this historically less-funded group that were successful in their applications for federal funding continued to attract significantly greater subsequent R&D funding from non-federal sources. For these institutions, each dollar of federal life sciences R&D funding continued to yield over $0.73 in non-federal R&D funds the following year. The strong and continuing impact of federal research support for these historically less research-intensive institutions may be due to signaling effects, as successful application vetted through the federal funding agencies' peer review process may be viewed by non-federal funders as a signal of research quality. In addition, if federally-funded R&D expenditures build these universities' productive capacity, facilities,

and human capital, this too would make recipient universities more attractive to non-federal funders.

In contrast, highly research-intensive PhD-granting institutions experienced little real decline in federal support in the post-doubling era. But, to the extent that federal funding for life sciences R&D did decline at any given institution, each dollar lost appears to have been completely offset by increased funding from non-federal sources. Specifically, whereas during the NIH budget doubling period these institutions might have attracted an additional \$0.20 ($p<.01$) in non-federal funding per federal dollar received, in the post-doubling era each federal dollar these institutions lost was replaced with over \$1.10 ($p<.01$) in non-federal funding.

The nonlinearity we observe in non-federal responses is consistent with prior literature. As discussed by Borgonovi (2006), if additional federal support at low levels of total (public and private) expenditure permits recipients to expand their set of activities and undertake higher quality projects, this expanded set of opportunities may yield, on the margin, higher utility for non-federal funders, encouraging crowd-in of non-federal funds. In addition, it is conceivable that universities with higher total research funding may exhibit greater crowding out of fundraising effort. Similar empirical research on nonprofit arts organizations likewise found that at lower total funding levels government support stimulates private donations, while at high levels of total funding crowd-out effects may dominate (Brooks 2000).

In summary, using annual life sciences R&D expenditures by funding source for a panel of 228 U.S. universities, followed over a decade of dramatic increase and subsequent stagnation, we find that the NIH budget doubling was successful in stimulating complementary research investment from non-federal

sources. By enabling universities to attract greater private-sector investment, federal life sciences R&D funding may also influence downstream commercialization of university research. However, to understand the mechanisms by which federal funding results in commercial products such as life-saving drugs, one should also look at broader outcomes such as university patenting and licensing behavior, and alliances between universities and the private sector. Through its direct and indirect support of graduate students and postdoctoral trainees, increases in federal funding may also increase the number and quality of the scientific workforce, thereby creating benefits that transcend the university's boundaries. Finally, qualitative research with not-for-profit foundations and other non-federal funders may serve to confirm the suggested signaling effect; however, a structural approach is needed to disentangle this effect from complementarity between federal and non-federal funds in the production of knowledge. These are subjects for our future research.

FOOTNOTES

[1] Source: NSF Survey of Federal Funds for Research and Development

[2] It is also conceivable that federal dollars may be the sole source of financial support for some types of R&D, and thus neither substitute for nor complement non-federal R&D funding. Theoretically, if we consider basic science to be a public good – that is, if universities and other researchers are unable to appropriate the full social value of their biomedical inventions – we would expect underinvestment in those areas of basic science by the private sector. For these research areas, changes in federal funding may have no short-run impact on non-federal funding: a dollar increase in federal funding would simply increase total university R&D expenditures by one dollar.

[3] The NSF estimated total life sciences R&D expenditures at U.S. universities and colleges as $32.8 billion in 2009. See: http://www.nsf.gov/statistics/nsf11313/pdf/tab48.pdf

Table 1
Descriptive Statistics for University R&D Expenditure Data

Variable	Mean	Standard Deviation
Federal Funding for Life Sciences, $ Millions	64.0	94.0
Non-Federal Funding for Life Sciences, $ Millions	46.2	65.3
Federal Funding for Other Fields, $ Millions	44.8	78.9
Non-Federal Funding for Other Fields, $ Millions	29.6	43.5
PhD-Granting Institutions	74.6% (n=170)	
Private Institutions	32.9% (n=75)	
Number of Institutions in Panel	228	

Note: Authors' calculations based on data from National Science Foundation (NSF) Survey of Research and Development Expenditures at Universities and Colleges. All amounts reported in constant 2010 dollars, inflated using the Biomedical Research and Development Price Index. Percent of institutions granting PhDs based on institution's 2005 Carnegie Classification. Reported standard deviations are calculated between panel institutions.

Table 2
Effects of Changes in Federal Funding on Non-Federal Funding for Life Sciences Research and Development at U.S. Universities

	(1)	(2)	(3)	(4)
Federal funding, life sciences	0.511***	0.258***	0.247***	0.120**
	(0.0613)	(0.0549)	(0.0537)	(0.0542)
Non-federal funding, other non-life-sciences fields			0.264*	0.276**
			(0.139)	(0.135)
Federal funding, other non-life-sciences fields			0.0104	0.0668
			(0.0459)	(0.0546)
Observations	2508	2508	2508	2508
Number of institutions	228	228	228	228
R^2 ; Model 4: First-Stage F-stat	0.54	0.54	0.62	69.42
Hansen's J-statistic Overid Test				0.157
p-value				0.924

* significant at 10%; ** significant at 5%; *** significant at 1%

Standard errors are robust to heteroskedasticity and clustered on university, and are reported in parentheses below each coefficient estimate.

Results from multivariate regression with non-federal life sciences funding as the dependent variable, and with the university-year as unit of observation. All federal funding amounts are lagged one year. Model 1 includes year fixed effects (estimates not shown); Model 2 adds university fixed effects. Model 3 adds controls for non-life-sciences funding received by the university from both federal and non-federal sources. Model 4 employs instrumental variables estimation, with predicted NIH funding (both 1993-1996 and 1975-1984 base period) and state-level representation on the House appropriations subcommittee as instruments for federal life sciences funding. Like Model 3, Model 4 also includes university and year fixed effects, as well as federal- and non-federal non-life-sciences funding covariates.

Table 3
Dynamic Effects of the Rise and Fall in Federal R&D Funding, During and After the NIH Budget Doubling

	Pre-2004	2004-2006	Post-2006
Federal Funding for Life Sciences R&D	0.267***	0.303**	-1.111***
	(0.0769)	(0.123)	(0.324)
Non-Federal R&D Funding, Other Fields	0.579***	0.290**	0.363***
	(0.214)	(0.125)	(0.133)
Federal R&D Funding, Other Fields	0.116*	-0.0252	-0.0809
	(0.676)	(0.0992)	(0.107)
Observations	1140	684	684
Number of institutions	228	228	228
First-Stage F-statistic	54.74	20.30	8.304
Hansen's J-statistic	2.179	0.728	1.448
p-value	0.336	0.394	0.485

* significant at 10%; ** significant at 5%; *** significant at 1%

Standard errors are robust to heteroskedasticity and clustered on university, and are presented in parentheses below each coefficient estimate.

All results are from instrumental variable estimation with non-federal life sciences funding as the dependent variable, with the university-year as unit of observation, and all federal funding amounts are lagged one year. Models include university fixed effects, and use both predicted NIH funding instruments (base periods 1993-1996 and 1975-1984) and state-level Congressional representation as instruments for federal life sciences R&D funding.

Table 4
Heterogeneous Effects of Changes in Federal Life Sciences R&D Funding in the Budget Doubling Period, 1998—2003

	(1)	(2)	(3)	(4)
Lagged Federal R&D Funding, Life Sciences	0.761**	0.884***	0.446*	0.588**
	(0.369)	(0.266)	(0.229)	(0.271)
Lagged Federal R&D Funding, Life Sciences * Above Median	- 0.630*	- 0.681**	- 0.236	- 0.286
	(0.379)	(0.275)	(0.237)	(0.288)
Non-Federal R&D Funding, Other Fields	0.0278	0.0355	0.0713	0.0543
	(0.0879)	(0.0873)	(0.101)	(0.0955)
Non-Federal R&D Funding, Other Fields * Above Median	0.511**	0.533**	0.495*	0.559**
	(0.248)	(0.247)	(0.253)	(0.253)
Observations	1140	1140	1140	1140
Number of institutions	228	228	228	228
First-Stage F-statistic & Above Median F-stat	3.980 29.04	5.262 35.51	4.908 17.54	6.763 72.61
Hansen's Overid Test p-value	2.292 0.514	2.181 0.536	2.124 0.547	0.607 0.436

* significant at 10%; ** significant at 5%; *** significant at 1%

Standard errors are robust to heteroskedasticity and clustered on university, and are presented in parentheses below the coefficient estimates. All results are from IV models with dependent variable non-federal life sciences R&D funding and university fixed effects (not shown). Excluded instruments for lagged federal life sciences R&D funding and its interaction with above-median historical funding level include: base period 1993-1996 predicted NIH funding, the interaction of base 1993-1996 predicted NIH funding with the above-median historical NIH funding indicator variable, and state-level Congressional representation. Models (1) and (3) also include federal non-life-sciences R&D funding as a covariate (not shown) and employ the alternative predicted NIH funding instrument (base period 1975-1984) and its above-median interaction as instruments. Models (1) and (2) also include year fixed effects, with and without above-median interactions.

Table 5
Heterogeneous Effects of Changes in Federal Life Sciences R&D Funding in the Post-Doubling Era, 2007—2009

	(1)	(2)	(3)	(4)
Lagged Federal R&D Funding, Life Sciences	0.755*	0.727*	0.111	0.114
	(0.438)	(0.410)	(0.264)	(0.264)
Lagged Federal R&D Funding, Life Sciences * Above Median	- 2.242***	- 2.097***	- 1.245***	- 1.235***
	(0.637)	(0.635)	(0.411)	(0.415)
Non-Federal R&D Funding, Other Fields	0.0897**	0.0914**	0.0917***	0.0872***
	(0.0374)	(0.0358)	(0.0319)	(0.0323)
Non-Federal R&D Funding, Other Fields * Above Median	0.387**	0.369**	0.357**	0.348**
	(0.183)	(0.187)	(0.172)	(0.174)
Observations	684	684	684	684
Number of institutions	228	228	228	228
First-Stage F-statistic & Above Median F-stat	1.824 3.535	3.636 6.822	3.109 6.240	6.229 11.28
Hansen's Overid Test p-value	1.836 0.607	0.128 0.720	1.510 0.680	0.092 0.761

* significant at 10%; ** significant at 5%; *** significant at 1%
Standard errors are robust to heteroskedasticity and clustered on university, and are presented in parentheses below the coefficient estimates. All results are from IV models with dependent variable non-federal life sciences R&D funding and university fixed effects (not shown). Excluded instruments for lagged federal life sciences R&D funding and its interaction with above-median historical funding level include: base period 1993-1996 predicted NIH funding, the interaction of base 1993-1996 predicted NIH funding with the above-median historical NIH funding indicator variable, and state-level Congressional representation. Models (1) and (3) also include federal non-life-sciences R&D funding as a covariate (not shown) and employ the alternative predicted NIH funding instrument (base period 1975-1984) and its above-median interaction as instruments. Models (1) and (2) also include year fixed effects, with and without above-median interactions.

Figure 1

University Life Sciences R&D Expenditures by Funding Source,

Panel of 228 Universities, 1998-2010

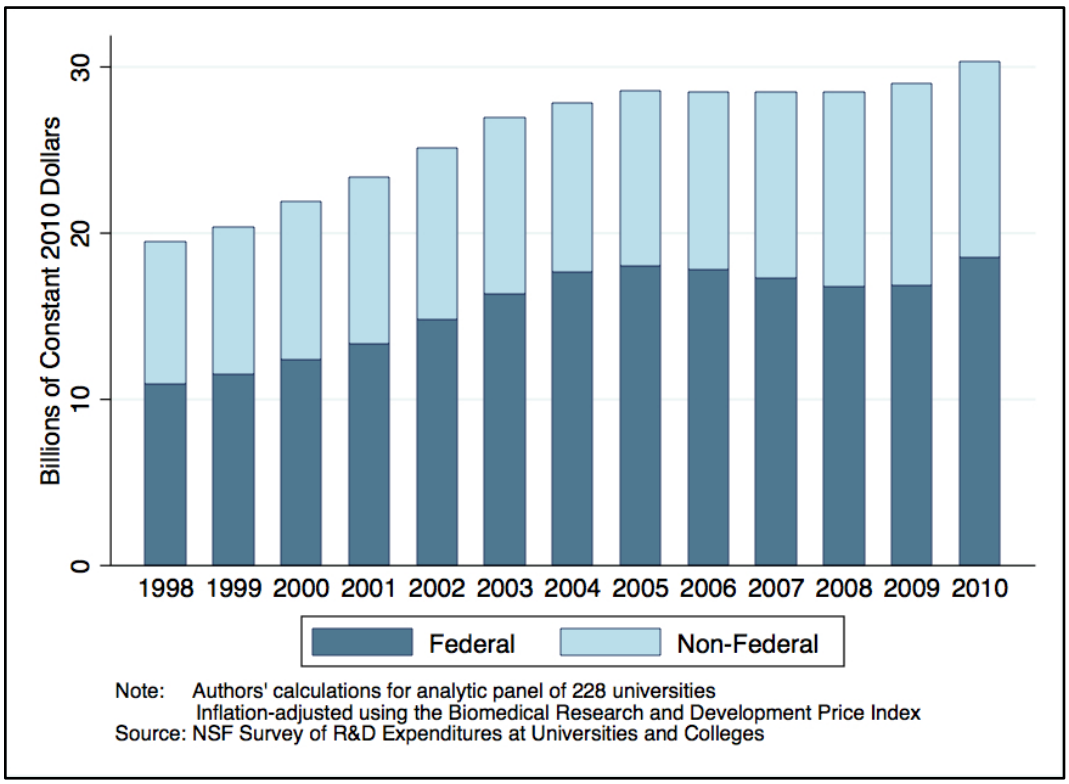


Figure 2

Differences in Federal Life Sciences R&D Funding Trends for Highest vs Lowest Tercile Funded Universities, 1998 – 2010

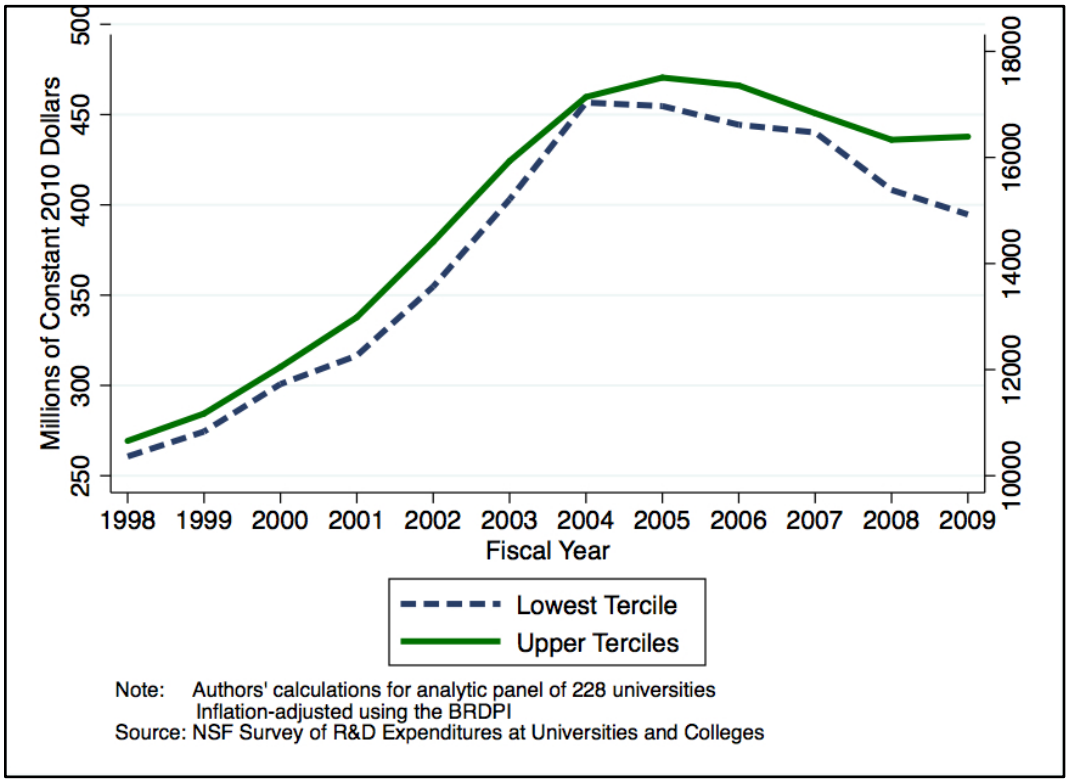
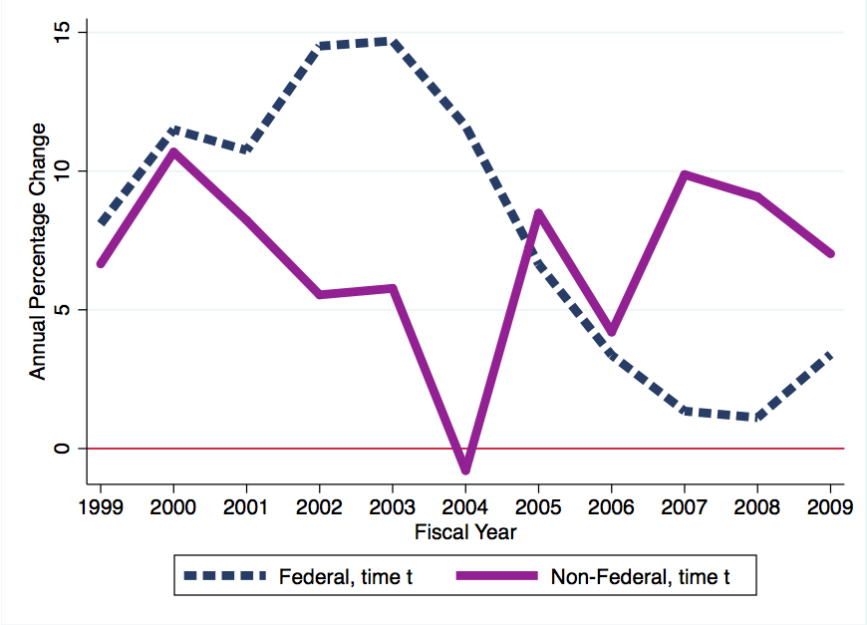
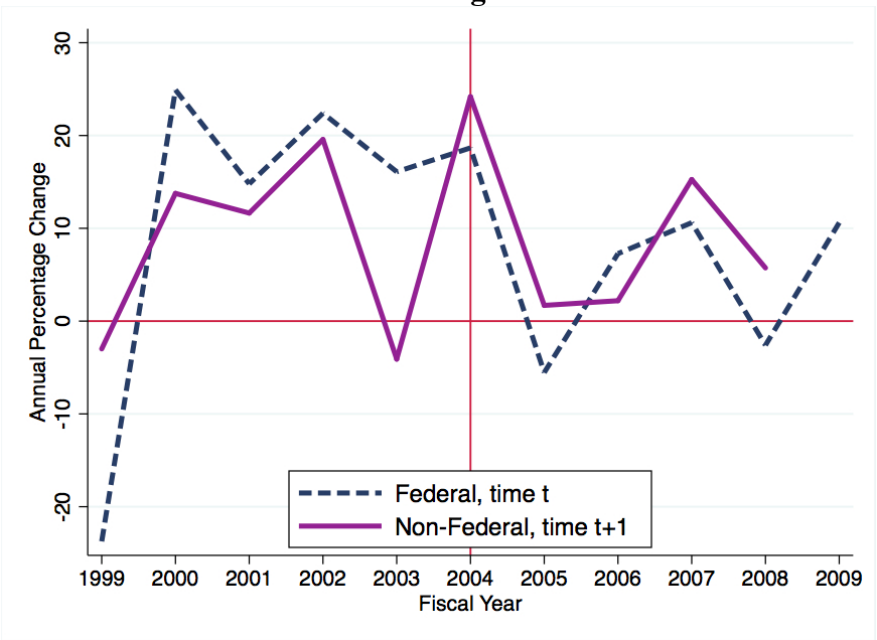


Figure 3

A. Non-Federal Life Sciences R&D Funding Substitutes for Federal at Carnegie Doctoral High / Very High Research Universities



B. Complementarity and/or Signaling Effects Seem to Dominate at Non-PhD-Granting Institutions



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