Balancing Incentives: The Tension Between Basic and Applied Research

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March 2002

The cooperation and assistance of the firms that contributed the data used in this study is greatly appreciated. Jeff Furman, Pierre Azoulay, and Mercedes Delgado provided outstanding research assistance. We thank Robert Gibbons, Tom Hubbard, Jonathan Levin, Dan Levinthal, Fiona Scott Morton, David Mowery, and seminar participants at numerous universities and conferences for helpful comments; however the conclusions and opinions expressed in this paper are our own, and any errors or omissions remain entirely our responsibility. This research was funded by POPI, the Program for the Study of the Pharmaceutical Industry at MIT and by the MIT Center for Innovation in Product Development under NSF Cooperative Agreement Number EEC-9529140. Their support is gratefully acknowledged. Stern completed this research while a Health and Aging Fellow at the National Bureau of Economic Research, whose hospitality is gratefully acknowledged. All correspondence to: Professor Iain Cockburn, School of Management, Boston University, 595 Commonwealth Avenue, Boston, MA 02215, cockburn@bu.edu.

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ABSTRACT

When effort is multi-dimensional, firms will optimally "balance" the provision of incentives. Setting high-powered incentives along one dimension raises the returns to providing high-powered incentives along other dimensions which compete for the worker's effort and/or attention (Holmstrom and Milgrom, 1991). We test for this effect in the context of for-profit pharmaceutical laboratories using detailed data on individual research programs. Consistent with this complementarity hypothesis, there is both cross-sectional and time-series evidence that firms providing strong promotion-based incentives for scientists to invest in basic research are more likely to provide strong incentives to supply effort towards applied research.

JEL Classification Numbers: L2, L65, O32.

Keywords: innovation, incentives, multitasking, complementarity.

I. INTRODUCTION

Recent studies on work practices, productivity, and incentives has emphasized the role of complementarities between the elements of a "system" of distinct, yet interdependent, organizational design elements. In order to realize productivity gains, firms may need to adopt a "bundle" of work practices and incentives, rather than implementing them piecemeal. This idea has been developed theoretically in the economics of organizations (Milgrom and Roberts, 1990; Holmstrom and Milgrom, 1994) and in empirical studies, such as Ichniowski, Shaw and Prennushi (1997), who provide persuasive evidence for these complementarities in specific cases and illustrate the range of activities and practices which may constitute the bundle.[1] Yet relatively little evidence has been gathered as to the *source* of these complementarities. They may simply be inherent in the nature of the work activity required by a firm's production technology. But in many contexts, complementarities between organizational practices may arise from the contracting problems inherent in a multi-task agency setting. When output is generated by workers (or work groups) exerting effort across two or more different tasks, the firm will optimally "balance" incentives across these tasks. If it does not, workers will inefficiently allocate too much effort towards those tasks with the highest marginal return to them (Holmstrom and Milgrom, 1991, henceforth "H&M"). This idea provides valuable insight into the observation that adopting a specific organizational practice in isolation often fails to pay off.

Despite the power of this idea, and its significance for our understanding of the economics of the firm, empirical characterizations of multi-dimensional incentive systems are surprisingly scarce. In part, this is because, in internal organizational settings where multiple dimensions of effort matter, measuring comparable incentive instruments across firms and over time requires detailed firm-level data that is difficult to obtain and interpret. Anderson and Schmittlein (1984) provide an early study of how the incentives of sales agents relates to factors such as the degree of monitoring and whether the salesperson is a long-term employee. More recent cross-sectional studies explicitly test for complementarity in incentives across firm boundaries (Slade, 1996; Brickley, 1999). For

[1] Ichniowski, Shaw and Prennushi (1997) explore seven elements of organizational design which are potentially complementary with one another: incentives, recruiting and selection, work teams, employment security, flexible job assignment, skills training and labor management communication.

example, in a study of gasoline retailers, Slade (1996) provides evidence that differences in non-gasoline service offerings, such as a convenience store, influence the incentives provided by gasoline wholesalers. This paper models and tests the complementarity hypothesis in the context of pharmaceutical research laboratories, using a rich and detailed dataset compiled from extensive fieldwork and internal records of a sample of nine representative firms over 15 years. Moving beyond cross-sectional approaches common to prior studies, this data allows us to evaluate the complementarity hypothesis exploiting variation both within and across firms in our sample.

The long-run level of research productivity in pharmaceutical drug discovery depends on the level of effort devoted towards two distinct activities: basic research directed towards the solution of fundamental scientific problems, and applied research directed towards the discovery of potentially marketable drugs. Starting in the late 1970s, the pharmaceutical industry experienced a significant exogenous shock to the technology of drug discovery which changed the returns to the basic research component of the drug discovery process. While the returns to basic research were almost certainly fairly low prior to the late 1970s, several advances in (university-based) biochemistry and molecular biology resulted in a new "technology" for drug discovery in which applied research productivity depended on the research team's prior experience in and connection to relevant basic research. Firms responded heterogeneously to this shock: while some firms quickly adopted a research organization which provided high-powered promotion-based incentives designed to encourage efforts in basic research, other firms were much slower to do this, eschewing internal incentives based upon basic research outputs (such as scientific publications) well into the 1990s.

We use this heterogeneity among firms and over time to evaluate whether firms offering high-powered incentives for basic research were more likely to provide higher-powered incentives for applied research. As such, we are evaluating an important implication of the multi-task model: in response to an exogenous shock which shifts incentive intensities along one dimension, do firms increase the incentive intensity for other tasks competing for workers' time?

Pharmaceutical research provides a particularly interesting setting in which to explore multi-task agency problems. In the first place, although the question of exactly how incentives are provided for basic and applied research has important implications for the rate of technological innovation (Romer, 1990; Lazear, 1996), there is little systematic empirical evidence about how such incentives

are provided in industrial laboratories (Hauser, 1998). For IO and organizational economists, understanding how firms provide incentives to *internal* researchers (who relinquish intellectual property claims on discoveries made during their employment) is important for understanding the conditions under which R&D will take place in the confines of an integrated firm (Holmstrom, 1989; Aghion and Tirole; 1994; Lerner and Merges, 1998; Gans and Stern, 2000).

Using data from nine firms over fifteen years we establish three results. First, we demonstrate substantial variation among firms and across time in the intensity with which they provide incentives for basic research. The primary mechanism used to do this appears to have been the internal labor market of the firm. By actively rewarding research workers' participation in "open science" through practices such as using publication in the refereed literature as a criteria in promotion decisions, some firms provided powerful incentives to supply effort along this dimension. Other firms did not use these practices, or applied them less intensively. Second, we find evidence for significant variation in the provision of high-powered incentives to do applied research: some firms rewarded research teams with substantially higher budgets following better-than-expected patent output, while in others this effect was much more muted. Third, we find evidence in a variety of "cuts" of the data for a quantitatively and statistically significant positive association between the use of these two instruments.

The correlation between the incentives for basic and applied research may, of course, be due to factors unrelated to the firm's response to the multitask agency problem. Rather than rely on a single argument for identification (for example, by simply assuming the exogeneity of certain instruments), our approach is to identify the most likely sources of bias and to provide direct controls for these effects. For example, after showing the presence of a positive correlation in the context of a pooled data analysis, we demonstrate an even stronger positive correlation in a more demanding "differences-in-differences" estimator, including fixed effects for each individual research program along with time trends for each therapeutic area. While the limited number of firms in our sample makes us cautious about overinterpreting these results, we view them as supporting the H&M hypothesis about the role of "balance" in the provision of research incentives.

The paper begins with a discussion of the shock to drug discovery research in the 1970s and its implications for the management and organization of pharmaceutical research. Section III reviews

the H&M multitask agency model and derives its empirical implications. In Section IV we develop our measures of the intensity of incentives for workers to supply effort in "basic" versus "applied" research activities. Section V reviews our empirical findings, and Section VI concludes. Two supporting appendices discuss data sources and the construction of each of the incentive measures in greater detail.

II. BASIC AND APPLIED PHARMACEUTICAL RESEARCH

The process of drug discovery and development is complex and extends over several years. In the "research" phase, also referred to the drug discovery process, researchers attempt to find compounds that may plausibly be developed into drugs by demonstrating their therapeutic effects in animals. In the second, or "development" phase, these compounds are tested in humans and undergo rigorous review by the Food and Drug Administration. The two phases require distinct skills and knowledge and are nearly always carried out by quite different people. In this paper, we focus only on the research phase.

For much of this century, the technology of drug research was dominated by a technique commonly described as "random" drug discovery. Under this regime, large numbers of candidate compounds would be tested for pharmaceutical activity in an "animal model" or a relatively crude cell culture or assay. For example, a search for hypertensive therapies might involve injecting large numbers of candidate compounds into hypertensive dogs to explore the degree to which they reduced blood pressure, while a search for therapies effective against anxiety might involve administering compounds to rats and then observing their behavior in stressful situations.[2] Molecules showing pharmaceutical activity would then be subjected to further testing, and modified to improve their pharmacological properties. In most cases, the "mechanism of action" — the specific biochemical and molecular pathways responsible for a compound's therapeutic effect — was not well understood. While random drug discovery was not entirely divorced from more fundamental scientific research conducted within the public sector, in general it was not critical that pharmaceutical researchers be at the leading edge of their respective disciplines.

[2] For example, one test involved throwing rats into buckets of water and observing how long they continued to struggle.

This changed in the late 1970s. In 1978, Squibb announced the discovery of the anti-hypertensive drug Captopril. This marked a watershed in the technology of drug discovery, since it was the first drug to be discovered through the use of an *in-vitro* screen that duplicated a particular mechanism of action, rather than through the use of an animal model.[3] This technology, commonly called "rational" or "mechanism based" drug discovery, offered a new and powerful research tool, and was gradually adopted across the industry over the course of the next fifteen years.[4]

This change is critically important for this paper because it greatly increased pharmaceutical firms' returns to investment in "pure" or "basic" research. The ability to find drugs by screening compounds against mechanisms identified at the cellular level greatly increased the potential returns to understanding these mechanisms and — most importantly — to identifying them prior to competitors. Research-oriented pharmaceutical companies therefore moved to make substantial investments in basic research in disciplines such as biochemistry or cell biology, and to invest much more heavily in understanding and accessing publicly funded science.

Shifting drug discovery research towards rational drug design changes the firm's incentive provision problem. For an individual researcher, effort devoted towards understanding fundamental biological principles is a substitute for "applied" effort devoted towards translating scientific knowledge into the discovery of potential drugs. Staying at the leading edge of the discipline requires devoting substantial effort to publication, basic laboratory work and remaining connected to the wider research community. Translating this knowledge into the discovery of potentially commercializable new drugs, however, requires devoting effort to working in an interdisciplinary applied research team. Rewarding researchers solely on the basis of their ability to work as part of this team and to generate immediate output increases the risk that the researchers will either continue to use the older methods of "random" drug discovery, failing to make the time-consuming effort

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[3] For a fuller discussion of the discovery of Captopril, see Henderson (1994). Note that researchers had used speculation about drugs' mechanism of action as a research tool long before the discovery of captopril. Sir James Black, for example, discovered the first of the beta-blockers in the early 1960s by exploiting his hypothesis that blocking the heart's beta receptors would lower blood pressure. But he did not make this discovery by screening compounds against isolated beta receptors.

[4] Note that this is <u>not</u> the same as the transition to "biotechnology" or the search for large molecular weight drugs. For a fuller discussion of this transition and its relationship to the techniques of biotechnology, see Henderson, Orsenigo and Pisano (1999).

intensive actions required to be at the leading edge of fundamental science, or that they will attempt to free ride on the scientific work of others. Similarly, only rewarding effort devoted to basic research might lead researchers to focus solely on advancing their own careers at the expense of effort that might be productively invested in the search for new drugs.

In principle, pharmaceutical firms might have been able to address this dilemma by allocating the tasks of "basic" and "applied" work to different groups within the firm, with incentives tailored to each task. Some firms did indeed experiment with this approach. Hoffman-La Roche, for example, created the "Roche Institute" to pursue fundamental research in biological systems. Our fieldwork suggests that this approach had significant drawbacks: such groups tended to degenerate into "ivory towers" – producing a large number of scientific papers but contributing little to the process of drug discovery. Effective adoption of "rational" drug discovery seems to depend on a tight integration between basic and applied research (Gambardella, 1995; Henderson and Cockburn, 1996). The dominant means used to accomplish this integration was to organize researchers into small teams (4-7 PhDs), responsible both for staying at the leading edge of their particular disciplines and for working together to translate this fundamental knowledge into promising compounds.[5]

We believe that this organizational design created exactly the kinds of tension that are captured in the H&M model. Managers of these research groups had to encourage workers to supply effort in both basic and applied research activities. The flavor of the tension that this created in individual researchers is well captured by the comment of a senior researcher at a large pharmaceutical firm who, following a long discussion of the measures that he was taking to ensure that his team was at the leading edge of the elucidation of the structure of cellular receptors remarked "*and of course this is all very well, but if we can't use (this knowledge) to discover new drugs, they'll fire me...*" (Personal communication to one of the authors).

[5] There are, of course, exceptions to this generalization. For example many pharmaceutical firms currently maintain small groups of researchers charged with the development of expertise in genomics, a new area of science that will probably have a very significant impact on the drug discovery process.

III. A MODEL OF "BALANCE" BETWEEN INCENTIVES FOR BASIC AND APPLIED RESEARCH

Theoretical work on incentive contracting has generated a number of important propositions about the structure of contracts between principals and agents in situations where the agent is required to perform multiple tasks (Holmstrom and Milgrom, 1991, 1994; Baker, 1992). One of the most salient propositions is that in these multitask agency settings incentive intensities are *complementary* with one another, with the consequence that the optimal incentive regime is "balanced" — the degree to which high-powered incentives are offered along any one dimension will depend on whether high-powered incentives are offered along other relevant dimensions. To see this more clearly, we briefly review the H&M model and then adapt their general framework to the specific setting of basic and applied research in pharmaceutical drug discovery.

We begin with a simple model of the provision of incentives for research workers in an employment relationship (i.e., the firm hires the workers and owns the output of their research). Consider a simple environment where the firm's profits are dependent on two distinct research activities, applied and basic research. For each dimension of effort i (A=applied, B=basic), the researcher chooses an effort level, e_i, yielding output $Y(e_A, e_B)$ with Y increasing in e_A, and e_B. Assume that, in each period, the firm observes a contractible signal, $\boldsymbol{x}=(x_A, x_B)$:[6]

$$\begin{aligned} x_A &= f(e_A) + \eta_A \\ x_B &= f(e_B) + \eta_B \qquad \boldsymbol{\eta} \sim N(\mathbf{0}, \Sigma) \end{aligned} \tag{1}$$

The firm's problem is to offer incentives according to the vector of observed signals to elicit the optimal (feasible) level of effort. By placing structure on the agent's preference function (specifically on the cost function for supplying effort), it is possible to solve for the firm's optimal incentive scheme.

$$U(w) = -e^{-r(w(x(e)))} - c(e) \tag{2}$$

Following H&M, assume that the (risk-averse) agent trades off expected income against the cost of

[6] Importantly, we assume that the signal vector is observable, contractible and unbiased. In a model which allows for subjective signals or incorporates the role of reputation, the relationship between signals and optimal incentives will be more subtle, and empirical predictions more difficult to come by (Baker, et al, 2001).

effort, that effort is costly ($c_i > 0$), the cost function is supermodular for effort along each dimension ($c_{ij} > 0, \forall\ i \neq j$) .[7] We further assume that the incentive scheme imposed by the firm takes the form of a linear reward structure relating the agent's wage to the observable signals:[8]

$$w = \alpha^0 + \alpha^A x_A + \alpha^B x_B \qquad (3)$$

where α^A and α^B are the incentive intensities implemented by the firm for applied and basic research, respectively and α^O is the fixed component of salary. Given that the firm chooses among linear incentive schemes, the optimal incentives provided for basic and applied research are complementary with one another. We can rewrite the firm's objective function as

$$\underset{\alpha}{Max}\ \pi = Y(e_A, e_B) - \alpha^0 - \alpha^A(e_A + \eta_A) - \alpha^B(e_B + \eta_B) \qquad (4)$$

Without loss of generality, we let $Y(e_A, e_B) = \theta^A e_A + \theta^B e_B + \theta^{AB} e_A e_B \quad (\theta^i \geq 0)$. While complementarity is not required, this functional form is consistent with our earlier discussion of why multitasking is inherent in the nature of drug discovery. Taking the cross-partial with respect to α^A and α^B yields

$$\begin{aligned}\frac{\partial^2 \pi}{\partial \alpha^A \partial \alpha^B} = {} & (\theta^A + \theta^{AB} e_B^* - \alpha^A)\frac{\partial^2 e_A^*}{\partial \alpha^A \partial \alpha^B} + (\theta^B + \theta^{AB} e_A^* - \alpha^B)\frac{\partial^2 e_B^*}{\partial \alpha^A \partial \alpha^B} \\ & - \frac{\partial e_A^*}{\partial \alpha^B} - \frac{\partial e_B^*}{\partial \alpha^A} + \theta^{AB}\left(\frac{\partial e_A^*}{\partial \alpha^B}\frac{\partial e_B^*}{\partial \alpha^A} + \frac{\partial e_A^*}{\partial \alpha^A}\frac{\partial e_B^*}{\partial \alpha^B}\right) > 0\end{aligned} \qquad (5)$$

where e_i^* is the agent's optimized effort level for a given pair (α^A, α^B). The sign of (5) follows from the fact that the effort supply function is supermodular in α^A and α^B: the marginal cost of effort along one dimension is increasing in the level of effort along the other dimension. Consequently, an exogenous shock resulting in the firm raising incentive intensity along one dimension will increase

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[7] Assuming that effort at the margin is costly to the agent does not rule out the possibility that agents expend some level of effort in the absence of explicit incentives (H&M, 1991). As such, this formulation is consistent with the hypothesis that agents place value on participating in scientific research either intrinsically or because it increases their external employment options (Stern, 1999).

[8] Rather than following the detailed (and familiar) derivation under which linearity is in fact optimal (Holmstrom and Milgrom, 1987) , we assume linearity to focus on the relationship among incentive instruments.

the returns to the firm of increasing incentive intensity along the other dimension. This theoretical prediction holds a key empirical implication: if the (stochastic) factors determining the optimized levels of α^A and α^B are independent of each other, then α^A and α^B will be positively correlated with each other within a cross-section of firms choosing these incentive intensities (Holmstrom and Milgrom, 1991; Athey and Stern, 1998).[9] It is important to note that this prediction depends only on the supermodularity of the effort supply function (i.e., different tasks are substitutes in effort), the assumption of linearity in the incentive scheme (i.e., there is no interaction between signals in the wage function), and an assumption that the covariance of η_A and η_B are not too strongly *negatively* correlated with each other.

This paper builds on this theoretical insight to offer an empirical test for the presence of complementarity of incentive instruments. Specifically, we examine the degree to which measures of α^A and α^B are correlated with each other in order to infer whether there is complementarity among these incentive mechanisms.

To argue that positive covariation between incentive elements implies complementarity requires that we address potential alternative statistical sources of positive covariation -- namely positive correlation among the factors driving the adoption of each incentive element (Arora, 1996; Athey and Stern, 1998). To do so, we note that, under the complementary hypothesis, this covariation test should be robust to conditioning on observable factors which may be associated with the adoption process of each incentive element. As such, rather than imposing exclusion restrictions (Arora, 1996) or estimating a full structural model of adoption (Athey and Stern, 1998), our empirical approach is to evaluate the covariation test using several different "cuts" of the data, each chosen to control for the most likely alternative sources of positive correlation between the two incentive instruments. As the first step towards implementing such a test we next adapt this simple single agent framework to our specific institutional setting and discuss the measurement of incentive intensity.

[9] More generally, the incentive intensities will be positively correlated if the stochastic shocks are statistically *associated* (a strong form of positive correlation).

IV. MEASURING THE INTENSITY OF BASIC AND APPLIED RESEARCH INCENTIVES IN DRUG DISCOVERY

While the canonical model of incentive contracting assumes the presence of a single agent whose wages are established according to an incentive scheme set by the principal, such a model cannot be applied immediately to the specific case of providing incentives for drug discovery research workers. While direct "cash" incentives provided through bonuses and stock options were used by some of our sample firms during the period of study, they do not appear to have played a major role in shaping employee behavior.[10] Effort in research activities is exceptionally difficult to monitor, given the nature of the task and the very long time periods over which "output" gradually becomes apparent, making piece-rate incentive systems difficult to implement.

Instead, our previous research on the management of R&D in the pharmaceutical industry, including extensive fieldwork extending over nearly a decade, leads us to believe that the principle sources of incentives for research workers lie in the operation of firms' internal labor and capital markets: that is, in promotion decisions and project funding choices. The specific mechanisms through which these work are discussed below, but, as a first step, note that these alternative sources of compensation are easily incorporated into the formal framework above by rewriting (3) in terms of rewards provided through the internal labor and capital markets:

$$w = \varphi_0 + \varphi_L P(x_A, x_B) + \varphi_C B(x_A, x_B) \qquad (6)$$

where $P(x_A, x_B)$ is the promotion benefit to the individual, and $B(x_A, x_B)$ is the "group-level" bonus associated with signals (x_A, x_B); φ_L and φ_C are parameters translating each of these incentive effects into their monetary equivalent at the level of an individual researcher. Assuming a linear structure for the promotion and bonus incentives,

$$\begin{aligned} P(x_A, x_B) &= \alpha_L^A x_A + \alpha_L^B x_B \\ B(x_A, x_B) &= \alpha_C^A x_A + \alpha_C^B x_B \end{aligned} \qquad (7)$$

then (6) can be re-written as a linear relationship between the wage and signals of effort in the two

[10] This stands in contrast to the new small "biotech" firms which entered the industry in large numbers towards the end of our sample period.

different activities. Given the linearity assumption, pairwise complementarity between the basic and applied research incentive instruments still holds. This is convenient, since as will be apparent from the discussion below, we can only easily observe two elements of the incentive contract, α_I^B or the promotional incentive associated with basic research and α_C^A the "bonus" incentive associated with applied work.

Basic Research Incentives

The conduct of fundamental, or "basic" research is a difficult activity for an employer to monitor and reward. Effort directed towards research is difficult to measure, and must be inferred from noisy measures of output. Institutions engaged in research may therefore use a variety of mechanisms to induce appropriate effort, from "up or out" promotion policies to rank order tournaments to efficiency wages (Doeringer and Piore, 1971; Lazear and Rosen, 1981; Gibbons and Waldman, 1998). In the context of drug discovery research, firms seem to rely on these deferred compensation mechanisms, with the largest financial rewards accruing to those researchers who climb *internal* career ladders.[11]

In such an environment, workers have incentives to exert effort towards the generation of signals observable to those managers who control the promotion process. By basing promotion policies on the "right" signals, the firm can induce appropriate effort, even if effort itself is difficult to monitor. The problem, of course, is to define the right signals. Evaluating the quality of basic research is prohibitively difficult for managers who are not themselves at the cutting edge of the relevant science. Pharmaceutical firms attempting to provide incentives to perform basic research rely instead on the set of institutions that have evolved to evaluate publicly funded biomedical researchers.

The reward system of "open science" is based on publication, peer review and priority, with a clearly established public rank hierarchy in most disciplines. (Merton, 1973; Dasgupta and David, 1994; Stephan, 1996; Henderson and Cockburn, 1998; Stern, 1999). Firms who encourage their

[11] During the period of our study, there is little evidence for a high level of mobility by drug discovery researchers employed by the established pharmaceutical firms within our sample (Rees, 1999).

research workers to participate in "open science" can benefit from this system in two ways. First, they can use worker's success in publishing in peer-reviewed journals and in garnering respect from their scientific peers as informative signals of the level of effort devoted towards basic research. Second, the rank order tournament aspects of this reward system translate straightforwardly into the tournament internal to the firm. By promoting researchers on the basis of their publication record and on their standing in the public rank hierarchy of their field, or on the criteria used by the publicly funded scientific community, a firm provides high-powered incentives to supply effort towards basic research.[12]

To measure the intensity with which firms provide these incentives (i.e., α_I^B), we use a variable, PROPUB, that is derived from over a hundred interviews with senior managers and scientists at our sample of pharmaceutical firms. In order to minimize the problem of retrospective bias, the interviews were focused around the development of a comprehensive history of the development of cardiovascular drugs at each firm.[13] Each respondent was questioned in detail about the ways in which research was organized over time, but the questions were linked to specific events in the history of the firm (e.g., who worked on the development of this beta-blocker? what happened? were they rewarded? why or why not?). PROPUB was then constructed by assigning each firm in each year a value on a 5-point Likert scale based on the degree to which the firm's promotion policies are based on a researcher's standing in the external scientific community, where a value of 1 indicated that the firm placed no value at all on a researcher's reputation in the external community in rewarding his or her efforts and a value of 5 indicated that it was a central criteria in such decisions.

PROPUB has been found to discriminate effectively among firms in terms of their R&D

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[12] In addition to its impact on incentives, firms have at least two potential reasons for adopting pro-publication policies. First, researchers may have intrinsic preferences for interacting with discipline-specific scientific communities and for receiving recognition from their peers for discoveries. Simply put, scientists may have a "taste" for science, leading firms to be able to attract higher-quality researchers for lower wages (Merton, 1973; David and Dasgupta, 1994; Stern, 1999). A second motivation may be the direct productivity benefits. Firms who adopt a pro-publication orientation may gain earlier and more detailed access to new scientific discoveries and so may be purchasing a "ticket of admission" which pays itself off in terms of higher R&D productivity and a higher rate of technological innovation (Cohen and Levinthal, 1990; Rosenberg, 1990; Henderson and Cockburn, 1996).

[13] Cardiovascular drugs were chosen as the focus for the interview protocol because they are amongst the most important classes of drugs, and every firm in the sample invested heavily in their development over the study period.

productivity and is also correlated with several alternative measures of a firm's commitment to the world of public science and of its rate and extent of scientific publication activity (Henderson and Cockburn, 1996; Cockburn, Henderson, and Stern, 2000). However since the use of a subjectively constructed Likert scale will always raise questions, we also employ an alternative measure ("HIGH" PROPUB DUMMY) which is equal to 1 after a firm has increased its PROPUB level and is zero otherwise. While this measure exploits less of our qualitative information than PROPUB, it provides a more unambiguous index of the changing incentives for basic research within each firm in the sample.[14] Our results are robust to the use of either measure.

Across firms, differences in PROPUB reflect significant differences in the promotion policies of the firm (ranging from strong restrictions on scientific publishing and the active discouragement of basic research initiatives to the use of a promotion system not dissimilar to that of a university biology department – promotion based on publication record and external recommendation letters from leading scientific researchers in the public sector). Within a firm, "switches" in the PROPUB regime reflect a significant change in the firm's use of promotion incentives to encourage basic research. Over the sixteen year and nine firm sample, there are fourteen distinct "firm / basic research incentive level" regimes (i.e., five "switches" from a lower to a higher regime are observed during the sample period).[15]

Applied Research Incentives

To assess the internal incentives provided to supply effort towards applied research, we look to the firms' internal capital market, and to research funding decisions. Internal capital markets can play an important role as a reward mechanism for workers, ameliorating agency problems within the firm (Hart, 1995; Aghion and Tirole, 1997; Stein, 1997). In the context of pharmaceutical firms, we

[14] In other work, we explore several alternatives, such as PUBFRAC (the percentage of patent authors who also publish in the referred literature). Though less subjective, these quantitative measures suffer from two limitations. First, they measure *outcomes* rather than incentive policies, and, second, they cannot be constructed for the full period covered by our detailed R&D investment data.

[15] Since adopting a higher level of PROPUB may take time, for firms in which we observe a switch from a lower to a higher level of PROPUB, we allow for a "transition" period during the first year of implementation by excluding these "switching" periods from our sample. All of the results presented in Section V are robust to various different treatments of the adjustment process, such as creating a one-year "band" around the switching dates (including the year before and after) and ignoring the adjustment process altogether.

observe drug discovery teams in different therapeutic areas[16] competing with one another for resources, with variation in project funding decisions interpretable as a highly visible reward for success. By varying a research team's budget in response to observed output, a firm provides incentives for the team's workers to supply effort to generate positive signals.

This mechanism does not, of course, directly affect individual researchers. As noted above, successful new drugs are typically the result of the joint effort of a research team composed of 4-7 PhD scientists.[17] Since in general the firm cannot observe the separate contribution of each member of these teams, it may optimally choose to provide a "group-level" incentive, or a "bonus" to the group's overall budget. Nonetheless, this may still provide powerful incentives for individuals: team members can then allocate this bonus among themselves, within the constraints established by the internal procedures of each firm choosing to increase wages, to hire new researchers or to purchase expensive capital equipment. Since the teams are so small, the firm can ameliorate the problem of rewarding team production by providing rewards for successful applied research at the *group* level, giving each research group discretion in how to allocate this "bonus" (Holmstrom, 1982) while at the same time remaining confident that the small size of the group will prevent any significant free riding by individual researchers who might otherwise seek to maximize the effort that they devote to basic research at the expense of the group.

We measure the intensity of incentives to supply effort in applied research by estimating the sensitivity of drug discovery team research budgets to observed success in producing "applied" output in the form of potentially marketable compounds, where we measure this output in terms of the number of "important" patents applied for in a given year. We define a patent as important if it was subsequently granted in two of the three major patent jurisdictions (the USA, Europe and Japan). Important patents provide a particularly useful measure of applied output in this setting since the pharmaceutical industries is one of the few industries in which patents both correspond to particular products (a drug is a single patentable molecule) and in which they are central to

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[16] A "therapeutic area" is a sub-market within the pharmaceutical industry. For example depression, anxiety and hypertension are all separate therapeutic areas.

[17] The mean level of funding for a single therapeutic area is $1.6m (1985 $). The detailed headcount data that we obtained from a few of the firms in our sample suggest that this is roughly sufficient to employ 4-7 PhD level researchers, when overhead and support costs are factored in.

competitive advantage (Levin, et al, 1987; Cohen, et al, 2001).[18]

We estimate this sensitivity by constructing a simple model of R&D investment at the research program level, which allows the team's research budget (and thus observed expenditures) to be driven both by the need to provide incentives and by technological opportunity. The key assumption of the model is that changes in the research budget for a given drug discovery team from year *t-1* to year *t* reflect *both* a "bonus" payment reflecting the team's "excess" productivity over and above the expected level of applied research output in year *t* ($\tilde{I}_{i,j,t} = \alpha_C^A x_{i,j,t-1}^A$) where $x_{i,j,t-1}^A$ s the "shock" to applied research productivity by a research team in year t-1, *and* changes that reflect "efficient" investment insofar as the firm adjusts its research expenditures according to "news" from period *t-1* about underlying technological and market opportunities (Pakes, 1981; Abel, 1984),

$$I_{i,j,t}^* = I_{i,j,t-1}^* + \beta^X X_{i,j,t-1} + \beta^Z Z_{i,j,t-1} \qquad (8)$$

where $X_{i,j,t-1}$ is the shock to technological opportunity realized by program *i* in firm *j* in period *t-1*, $Z_{i,j,t-1}$ are opportunity shocks external to this program but observed by the firm, and $I^*_{i,j,t-1}$ is the optimal level of expenditure in the prior period.

In addition, we assume that the firm's internal measure of technological opportunity cannot be distinguished from the signal it receives about the team's applied research output shock (i.e., $X_{i,j,t-1} = x_{i,j,t-1}^A$) and that $Z_{i,j,t-1}$ can be partitioned into "news" observable to both the firm and econometrician ($z_{i,j,t-1}$) and a shock observable to the firm but not to the econometrician ($\zeta_{i,j,t-1}$). Subtracting $I^*_{i,j,t-1}$ from both sides and modeling total investment $I_{i,j,t} = I_{i,j,t}^* + \tilde{I}_{i,j,t}$ yields an expression for the overall change in expenditure after accounting for both the group-based incentive payment and the firm's response to technological opportunity:

$$\Delta I_{i,j,t} = (\alpha_C^A + \beta^X)\, x_{i,j,t-1}^A + \beta^Z z_{i,j,t-1} + \zeta_{i,j,t-1} \qquad (9)$$

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[18] Advances in molecular biology have spawned a number of developments that are "basic" in the sense of being fundamental to advances in the science but that have nonetheless proved to be patentable (Stokes, 1997). However, during the time covered by our sample, only a small share of total research expenditures were devoted to such areas. For example, though the average firm in our sample produced several hundred U.S. patents per year, Kaplan, Murray and Henderson (2001) estimate that the average established firm produced less than five biotechnology patents per year through 1990.

Under this model, data on research program investment and applied research output can be used to estimate $\gamma^A = \alpha_C^A + \beta^X$, the sensitivity of research program budgets to the prior period's unanticipated applied research output.[19]

To estimate (9), we must derive a measure for $x_{i,j,t-1}^A$ the observed shock to applied research output for a given therapeutic program in a given year. The details of our derivation are provided in Appendix B, but essentially, we first calculate the expected level of patents (our measure of applied research output) for each team for each year by regressing the level of patents as a function of the historical patent production rate of the team and $I_{i,j,t-1}$ .We then use the fitted values from this regression as our measure of the "predicted" level of patents for that team for that year. Finally, we define PATENT SHOCK as the difference between the observed and predicted level of patenting for that research program and

$$x_{i,j,t-1}^A = SHOCK_{i,j,t} = \left(\frac{PATS_{i,j,t-1} - E\left[PATS_{i,j,t-1}\right]}{E\left[PATS_{i,j,t-1}\right]} \right) * I_{i,j,t-2} \qquad (10)$$

which is PATENT SHOCK adjusted for the scale of the research program.[20] Since this measure is observed by the firm when choosing the research team's budget for year t, the firm is able to implement the investment equation (9).

Table 3 presents an estimate of γ^A obtained by estimating (9) using data on annual research expenditures and patents at the level of individual research programs conducted using a sample of nine pharmaceutical firms during the period 1975-90. (See Tables 1 and 2 for variable definitions

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[19] Overall, the sign and magnitude of γ^A are ambiguous theoretically since, while we expect $\alpha_c^A \geq 0$, the firm's optimal investment response to applied research output shocks depends on whether there are increasing or diminishing returns to effort in a particular therapeutic area. In applying this model to data , we must ensure that the estimate of γ^A controls for unobserved factors correlated with applied research output "shocks" and increases in R&D funding problem, which we largely address through the use of a differences-in-differences estimator with firm-program fixed effects. We describe the empirical strategy in detail in Section V.

[20] As described in Appendix B, we scale $x_{i,j,t-1}^A$ so that therapeutic programs of different sizes have "proportional" reactions to applied research outputs shocks of similar sizes.

and descriptive statistics, and Appendix A for a more detailed discussion of the construction of the sample and the variables). These data have a number of desirable features. First, the unit of observation, the "research program", corresponds closely to the organization of research activity into groups, and, second, only expenditures on "discovery" or "research" are measured, not the very different activity of clinical development. On average, each firm in the sample has just above 10 distinct drug discovery teams spending $1.58 million per year (in constant 1986 dollars) and obtaining 3.30 important patents per year (Table 2).

In Table 3, the first difference of research expenditures $\Delta I_{i,j,t}$ or $\Delta RESEARCH_{i,j,t}$ is regressed against (a) our measure of the applied research output "shock" (i.e., $x^{A}_{i,j,t-1}$ represented as SHOCK), (b) our measures of external technological opportunity shocks, (i.e., $z_{i,j,t-1}$) and (c) controls for an overall time trend, the scale of the program ($I_{i,j,t-1}$, or $RESEARCH_{i,j,t-1}$) and "momentum" in the research funding process ($\Delta I_{i,jt-1,}$ or $\Delta RESEARCH_{i,j,t-1}$). Since $z_{i,j,t-1}$, is difficult to measure directly, we use "news" in the patent applications of related research programs both inside the firm and at a sample of competitor firms to proxy for changes in technological opportunity. I_{t-1} is included as a control for size and to capture any higher-order time series properties.

The very high variance of the dependent variable (and the starkness of our investment model) is reflected by the low R^2 for the regression,[21] but our main variable of interest, the "shock" to observed applied research output has a positive coefficient, as expected, and is strongly significant. The magnitude of this coefficient is sensible: it implies that a one-standard-positive "surprise" in SHOCK has about a $140,000 (or approximately 9%) impact on the budget of the average program. Finally, there is a great deal of variation in γ^{A} both across firms and across basic research incentive "regimes" within a firm (recall that our measure of basic research incentives is a categorical variable with specific "switch" dates for individual firms): we can conclusively reject homogeneity of γ^{A} along each of these dimensions (these results are available from the authors upon request). This result holds with or without the other covariates in the model, and whether or not their coefficients

[21] Another way to think of this is to recognize that research program budgets are highly autocorrelated. While firms do adjust these expenditures, either through marginal changes to program budgets or by opening or closing programs, year-on-year the average changes are quite small. (See Cockburn and Henderson, 1994).

are allowed to be regime-specific, or are constrained to be equal across subsamples.

These results suggest that firms do indeed respond heterogeneously to unexpected shocks. Recall, however, that $\gamma^A = \alpha_C^A + \beta^X$. The firm's budgetary response to unexpected shocks reflects both its rewards to effort and its responsiveness to technological opportunity. The remainder of the paper is devoted to evaluating whether this variation in γ^A can be tied to the provision of basic research incentives (i.e., to the level of PROPUB). In other words, is α_C^A correlated with PROPUB?

V. CORRELATION OF BASIC AND APPLIED RESEARCH INCENTIVES

We begin with two very simple reduced-form summary analyses. The goal of these preliminary exercises is to explore whether a correlation between basic and applied research incentives can be found even using relatively crude measures and quite aggregate data, and so to motivate the more nuanced panel data analysis that follows.

In Table 4, we compute the average change in research funding for individual firm-program-years depending on whether the firm-program receives a positive or negatively signed applied research output shock (SHOCK > or < 0) and on whether the firm is associated with a "HIGH" or "LOW" PROPUB regime. The differences are dramatic: in low PROPUB regimes, a positive SHOCK is associated with a budget "boost" of $180,000 relative to a negative SHOCK. In contrast, in high PROPUB regimes, the budget boost almost doubles, to over $350,000 (the conditional means in all four boxes are statistically significant from each other at the 1% level). In other words, drug discovery programs operating in a high PROPUB regime are associated with a much higher sensitivity to patent output shocks.

A second method for evaluating the overall presence of a correlation between γ^A and PROPUB involves a simple two-step procedure. In the first stage, we estimate the budget's sensitivity to SHOCK for each of our firm-basic research "regime" combinations (recall that there are a total of 14 "regimes" across the sample); or in other words, following (9), we estimate 14 individual γ^A estimates, one for each firm across the span of time over which that firm maintains a constant level of basic research incentives. We then evaluate the correlation between this regime-

specific estimate of the sensitivity to research outputs shocks and PROPUB.[22] Though there are only 14 distinct regimes, the results are encouraging: the Pearson correlation coefficient between PROPUB and $\hat{\gamma}^A$ is 0.499 (significant at 5%). In Table 5, we present two simple regressions of $\hat{\gamma}^A$ on PROPUB; even after controlling for a time trend, PROPUB has a positive coefficient (significant at the 10% level).[23] Figure A presents this finding graphically; consistent with Table 5, there is a strong positive trend across PROPUB regimes.

These results are highly suggestive, and are certainly consistent with our core hypothesis: under high PROPUB regimes, firms offer more aggressive incentives for the generation of applied output. However, our analysis so far has not accounted for the potential impact of unobserved heterogeneity on the correlation between PROPUB and γ^A. If the levels of these variables are jointly determined by an unobserved factor (or if the factors determining each variable are correlated with each other), then the correlation among incentive intensities may be due to unobserved heterogeneity rather than to complementarity. At the same time, if these unobserved factors are independent of each other, this will introduce "noise" into the observed correlation of incentive intensities, and so weaken the power of a correlation test for inferring complementarity among incentive instruments.

To see this more clearly, consider the following examples. First, suppose there is an unanticipated positive shock in basic science which both increases the returns to drawing on basic science and raises the informativeness of patent output as a signal of technological opportunity. Then, we might observe increased sensitivity of R&D investment to applied research output alongside increases in PROPUB, independent of any complementarity among incentive instruments. Alternatively, if the intensity of incentives for applied research is determined by factors unrelated to the intensity of incentives for basic research (e.g., because of corporate culture or liquidity constraints), then the observed correlation between them will provide a downward-biased estimate of the importance of complementarities in the provision of incentives.

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[22] Note that if β^{γ} were a constant, so that variation in $\hat{\gamma}^A$ reflected variation in α_c^A, this would provide a clean test of the H&M hypothesis.

[23] Of course, because both of these variables are measured with substantial error (a problem we address below), the estimated coefficient is likely downward-biased.

To address these concerns, we exploit the panel structure of our data to estimate the conditional correlation between basic and applied research incentives under several alternative assumptions about the nature of unobserved heterogeneity within our sample. Specifically, the remainder of the analysis is conducted at a more disaggregated level – taking the "firm-program-year" as the unit of observation. This allows us to take advantage of the full richness of our research program data and to introduce controls for both potential changes in β^X and for possible alternative drivers of correlation between basic and applied research incentives.

To understand this empirical strategy more precisely, recall that we defined $\gamma^A_{i,j,t}$ to be the total response of the research budget (of research program i in firm j in year t) to the "surprise" in applied research output: $\gamma^A_{i,j,t} = \alpha^A_{C\,i,j,t} + \beta^X_{i,j,t}$. We test for correlation between $\alpha^A_{C\,i,j,t}$ and $\alpha^B_{I\,i,j}$ by letting $\alpha^A_{C\,i,j,t}$ be a function of $\alpha^B_{I\,i,j}$ (i.e. $\alpha^A_{C\,i,j,t} = \rho^0 + \rho^{A,B}\alpha^B_{I\,j,t}$) yielding:

$$\gamma^A_{i,j,t} = \beta^X_{i,j,t} + \rho^0 + \rho^{A,B}\alpha^B_{I\,j,t}$$

Substituting back into (9) results in an empirical model to test for the presence of correlation using firm-program-year data:

$$\Delta I_{i,j,t} = (\beta^X_{i,j,t} + \rho^0 + \rho^{A,B}\alpha^B_{I\,j,t})\, x^A_{i,j,t-1} + \beta^Z z_{i,j,t-1} + \zeta_{i,j,t-1} \qquad (11)$$

where the test for complementarity is simply $\rho^{A,B} > 0$. As discussed above, the key challenge in estimating this parameter (and therefore performing a consistent test for complementarity) is accounting for the impact of variation in β^X.

To begin, we assume that β^X is unobservable and is uncorrelated with $\alpha^B_{I\,i,j}$. Under this assumption, we can implement (11) by using PROPUB as a measure of $\alpha^B_{I\,i,j}$ and regressing ΔRESEARCH on SHOCK and interactions of SHOCK with PROPUB. As in Table 3, we also include a time trend and controls for technological opportunity and other drivers of ΔRESEARCH. Table 6 reports these results. In model (6-1) we reconfirm our results from Table 3, with a

regression showing a significant relationship between ΔRESEARCH and SHOCK. Model (6-2) provides our first detailed evidence that the overall sensitivity to SHOCK is positively associated with the level of PROPUB. Not only does the inclusion of a PROPUB interaction decrease the quantitative and statistical importance of SHOCK, but the coefficient suggests that the impact of PROPUB is quite large. Whereas a one-standard deviation in SHOCK is associated with less than a 5% increase in investment when PROPUB is at its lowest level, this same shock is associated with over a 19% increase when PROPUB is at its highest level. In (6-3), we include several controls associated with $z_{i,j,t-1}$ – two measures of information about technological opportunity ("NEWS" in COMPETITOR PATENTS and "NEWS" in RELATED PATENTS) as well as measures to account for the scale of the research program ($RESEARCH_{t-1}$) and potential serial correlation in the dependent variable ($\Delta RESEARCH_{t-1}$). Though these additional regressors enter significantly, their inclusion does not change our key result: the coefficient on SHOCK*PROPUB remains positive, of a similar magnitude, and with a similar standard error. In (6-4), we replace the time trend with year fixed effects, each interacted with SHOCK. These are jointly significant and result in a modest increase in the estimated parameter on SHOCK*PROPUB.[24]

These results are consistent with the findings from Tables 4 and 5. On the one hand, they provide evidence consistent with the presence of complementarity between basic and applied research incentives. On the other hand, this interpretation is conditional on our assumption that variation in β^X is uncorrelated with PROPUB. We therefore turn to more detailed analyses which control for the likely sources of correlation between β^X and PROPUB across programs, firms, and time. Three alternatives stand out. First, as discussed above, spurious correlation would be introduced if the use of both incentive instruments simply increased over time. Between the late 1970s and early 1990s, the use of promotion-based basic research incentives diffused widely

[24] We also have explored several robustness checks on these relatively "sparse" specifications (available from the authors). These include: using therapeutic class-specific fixed effects, incorporating several controls for changes in the firm's management structure (such as changes in the CEO, R&D Vice President, or changes in the process used in the capital budgeting process, and introducing additional lags of the dependent variable into the specification. As well, as discussed in Appendix B, we have explored specifications calculating SHOCK with alternative models of the firm's expectations process and using the "levels" version of SHOCK rather the percentage version used in Table 6. While several of these additional results contribute modestly to the regression's explanatory power, none is associated with a substantial change in either the magnitude or statistical significance of the SHOCK*PROPUB coefficient.

throughout the pharmaceutical industry. The results in Table 6 indicate that though overall changes over time are statistically significant, whether captured by a time trend or by year fixed effects. Though these variables have little or no impact on the coefficient of interest, we continue to include them in subsequent regressions in order to control for any omitted trends over time industry-wide variables. Second, there may be heterogeneity across therapeutic classes. It is possible that firms with higher levels of PROPUB are concentrated in therapeutic areas which tended to increase their sensitivity to SHOCK at a faster rate than the average. For example, the benefits from providing incentives for basic research seems to have increased especially rapidly in hypertension (Henderson, 1994; Cockburn, Henderson, and Stern, 2000). To the extent that patents (or a "surprise" in patenting) in these therapeutic areas also became more informative about applied research effort and technological opportunity, the correlation between PROPUB and the level of applied research incentives will reflect heterogeneity among firms in terms of their participation in different therapeutic areas. Third, it is possible that high PROPUB regimes are associated with firms and research programs which have "intrinsically" higher sensitivity to patents in the research budgeting process. For example, perhaps firms with higher levels of PROPUB have more "active" R&D managers who also tend to be more sensitive to applied research output in capital budgeting, or who simply have a taste for high powered incentives. In such an environment, exploiting the cross-sectional variation in the data will confound evidence of complementarity with evidence of a "taste" for incentives.

We address each of these concerns by including controls that directly account for each factor.[25] Specifically, we interact $x^{A}_{i,j,t-1}$ with firm-program fixed effects, a time trend for each research program, yielding a richer specification:

$$\Delta I_{i,j,t} = (\gamma^{0}_{i,j} + \gamma^{t}_{i}(t - t_0) + \rho^{A,B}\alpha^{B}_{I,j,t})x^{A}_{i,j,t-1} + \beta_{i,j} + \beta^{Z} z_{i,j,t-1} + \zeta_{i,j,t-1} \qquad (12)$$

Interacting $x^{A}_{i,j,t-1}$ with a firm-program fixed effect controls for any cross-sectional variation in the "intrinsic" sensitivity of different research programs to applied research output. For example, if

[25] This approach can be contrasted with more "structural" solutions, such as imposing cross-equation restrictions regarding adoption drivers (Arora, 1996) or the estimation of a simultaneous equations model integrating the adoption and performance implications of complementarity (Athey and Stern, 1998).

patents in a particular hypertension program are inherently more informative than patents in a particular depression program, (12) will control for these effects. As well, these fixed effects will control for the potential variation among managers in their "taste" for providing high-powered incentives. Controlling for changes over time, including year-specific and therapeutic class/year-specific dummies and interactions with x^{A}_{ijt-1} nets out both an overall and class-specific trend in unobserved components of β^{X}. In other words, in (12), $\rho^{A,B}$ s the correlation between changes in the sensitivity to x^{A}_{ijt-1} and changes in the level of PROPUB relative to the trend. This estimator is essentially a differences-in-differences estimator. However, in contrast to the classic differences-in-differences estimator, the hypothesis tested here concerns an interaction effect and so we require each of the individual effects to be interacted with x^{A}_{ijt-1}

Table 7 reports the results. In all of these regressions, we include a complete set of firm-program fixed effects and interactions of these with SHOCK. These interaction effects are jointly significant and substantially increase the explanatory power of the regression, indicating a high degree of heterogeneity among firm-programs in their average investment response to applied research output. To be consistent with a differences-in-differences estimator, these specifications rely exclusively on within-program variation in PROPUB and the presence of "switches" in the incentives provided for basic research. In this table, rather than using the Likert scale variable PROPUB, we use the "HIGH" PROPUB dummy, which is equal to one only for those years after the firm has "switched" from a lower level of PROPUB to a higher level of PROPUB, and is otherwise set equal to zero.[26] This measure is equal to one for a little more than one quarter of the sample, suggesting that it may be possible to identify our test exclusively on the "within" dimension.

This more stringent test provides further support for the "balance" hypothesis. After accounting for individual firm/program-specific interactions and program-specific time trend interactions with SHOCK, the magnitude of $\rho^{A,B}$ (our key parameter) increases substantially and

[26] All of the results in Table 7 are robust to using the five-point PROPUB variable instead of this differenced version.

remains at a similar level of statistical significance ($p < 0.01$ for all specifications). According to the "richest" specification, (7-4), for an average-sized program which realizes a one standard deviation SHOCK, there is a $390,000 incremental "boost" in the research budget after the firm switches to a higher level of PROPUB. This amount is more than 25% of the size of the average research program. In other words, after accounting for several sources of potential spurious correlation, the estimated relationship between basic and applied research incentives is stronger than in pooled data analysis conducted in Table 6.

Indeed, our evidence in favor of the complementarity hypothesis is somewhat strengthened when we consider the results from Tables 6 and 7 in concert. Recall that the key concern about the pooled analysis was the possible presence of a positive correlation between $\beta^{X}_{i,j,t}$ and PROPUB. However, in Table 7, after controlling for several sources of heterogeneity, we find that the magnitude on our key parameter increases and that a substantial share of the overall variation in ΔRESEARCH is associated with firm program-specific fixed effects. As such, the evidence from Table 7 is consistent with the hypothesis that the coefficient on PROPUB*SHOCK in Table 6 is, if anything, *underestimated*. If there was unobserved heterogeneity which was strongly and positively correlated with PROPUB, then either the "within" estimate of the coefficient in Table 7 would be much smaller in magnitude, or the fixed effects would have to account for a only a small fraction of the total variance.

For our result to be biased by any omitted independent variables driving incentive intensities, these would have to have a significant explanatory power above and beyond firm-program fixed effects and therapeutic class-specific trends. Meeting this challenge weakens the appeal of alternative interpretations of this result, since they must hold true both in the "pooled" or "between" dimensions of the data, and in the "within" dimension. Suppose, for example, that PROPUB and $\hat{y}^{A}$ were driven by a common general organizational response to science-driven drug discovery, with changes in PROPUB reflecting the outcome of "doing science" in terms of actual tasks performed by workers, and the nature of human capital employed by the firm, and changes in the sensitivity of research budgets to patent signals reflecting higher quality inventions, or a "science driven" capital budgeting process. This would certainly result in PROPUB and $\hat{y}^{A}$ being correlated in the cross-

section. But for the same to be true in the "within" dimension of the data there would have to be both (a) enough heterogeneity at the research program level in these effects that their "true" variation would not be accounted for by the fixed effects and therapeutic class-specific trends, and (b) sufficient co-movements over time in the "true" residual impact of adopting science-driven drug discovery on PROPUB and $\hat{\gamma}^{A}$ (as opposed to just noise) to generate a strongly positive association in the data. Absent effective instruments for the adoption of science driven drug discovery as distinct from pro-publication incentives we cannot definitively reject this hypothesis, but nonetheless we believe it to be unlikely. By and large, while firms adopted uniform incentive policies, the rate at which they adopted the particular techniques of science-driven drug discovery varied significantly across programs and thus we think it is very unlikely that the second condition holds in these data.

VI. CONCLUDING THOUGHTS

The principal finding of this paper is the presence of a positive correlation between measures of the use of promotion-based incentives for basic research and of team-based incentives for applied research. This correlation is both economically and statistically significant in a variety of different "cuts" of a panel dataset on R&D investment behavior of pharmaceutical companies. As in Inchniowski, Shaw and Prenusshi (1997), our empirical strategy has been to exploit the full range of variation contained within a micro-level dataset to rule out a variety of potential sources of unobserved heterogeneity. The positive correlation between basic and applied research incentives exists whether we aggregate the data into a small number of distinct firm-regimes, exploit cross-sectional variation among individual research programs, or subject the hypothesis to a differences-in-differences test using only within-program variation over time.

This result is consistent with a key proposition of modern agency theory – that when a principal prefers agents to balance their effort across multiple tasks, incentives will also be balanced, with increases in incentive intensity on one dimension associated with increases in incentive intensity on competing dimensions. Our interpretation of our results as providing novel empirical support for this "complementarities" proposition is, however, tempered by our inability thus far to obtain data which would allow us to directly identify incentive intensity choices, and to rule out other potential explanations.

An interesting aspect of our investigation is the degree to which the types of incentives discussed in the abstract in contract theory are embedded in the design of the firm's internal organizational processes. We do not discount the efficacy of unidimensional monetary incentive schemes in environments where output is easily monitored and there is opportunity for specialization. But, to understand incentives in a complex environment such as an R&D laboratory, it is critical to account both for the possibility that incentives may be multidimensional, and for the firm's ability to provide these incentives through mechanisms such as the operation of its internal labor and capital markets. Aligning agency theory with the use of incentives in real organizations is likely to require quite careful tailoring of the empirical content of contract theory to concrete organizational and institutional settings.

APPENDIX A: DATA SOURCES AND CONSTRUCTION

Our results are obtained from a unique data set built from detailed internal records of a sample of nine research-oriented pharmaceutical companies who, taken together, spend about 25% of the total amount of privately funded pharmaceutical research conducted worldwide.[27] These data on individual research programs expenditures are supplemented by patent data and a measure of the degree to which the firm provides incentives for basic research in its promotion policies. This appendix reviews the sources of this data, the construction of the sample, and summary statistics (Cockburn and Henderson (1994) and Henderson and Cockburn (1994; 1996) discuss the construction of this data set in greater detail, Table 1 provides variable definitions, and Table 2 reports the summary statistics).

A.1. Data Sources

FUNDING VARIABLES. Our data on research investment are taken from a database on research expenditures for several hundred individual research programs conducted by firms in this sample between 1975-1990. These data were assembled from confidential internal records, and great care was taken to treat data consistently across firms and over time. Pharmaceutical research takes place in two distinct phases: pre-clinical (or "discovery" research) and clinical (i.e., development); here we focus exclusively on the former.[28] RESEARCH is thus the level of expenditures on pre-clinical discovery research in a given firm-program-year, deflated to 1986 dollars by the NIH biomedical research deflator. We measure the "bonus" to the research budget, ΔRESEARCH, as the first difference of RESEARCH. Similarly, FIRM RESEARCH is just the sum of RESEARCH over all observed programs of a firm in a given year.

PATENTING VARIABLES. Our measure of the objective signal of research output is based on the number of patents produced by a given firm-program-year. Patents correspond quite closely to the output of the "discovery" phase of pharmaceutical research, in the sense that they are generated by the identification of candidate compounds and represent the end of the pre-clinical phase of the research process. Of course, patents are a notoriously noisy measure of inventive activity and effort: there is enormous variation across patents in their technological and economic significance; patents are the result of a stochastic process; and there may be only a weak link between the realized level of patenting in a given year and the level of effort provided by a research group. However, despite these qualifications, we believe that patenting rates are a useful and utilized "objective" performance

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[27] The data are provided under guarantees of strict confidentiality and anonymity so we can discuss the makeup of the sample only in broad terms. The sample is relatively representative of the industry as whole, in terms of size, technical or commercial performance, and geographic distribution (with firms headquartered in both the United States and Europe).

[28] By focusing exclusively on the discovery phase of pharmaceutical research, we avoid the complexities of modeling the multi-year multi-stage development phase whereby individual drugs are moved through clinical development and testing for regulatory approval. Also note that external research grants and licensing or joint-venture payments are sometimes included in the data (as appropriate); however, these types of funding arrangement represent a very small share of the total during the period of our sample.

measure. To ensure comparability across firms, we restrict ourselves to a measure of "important" patent counts, that is inventions for which patent applications were filed in at least two of three major jurisdictions (the U.S., Europe, and Japan.) This controls for variation across firms in their propensity to patent "marginal" discoveries or in their national environment (patent counts based on single country grants will tend to be biased towards domestic firms).[29] Moreover, we assume that the timing of the firm's patent filings is a good measure of the time at which decision-makers acquire objective information about a research group's recent production of potentially commercializable compounds. Finally, we match these patents to underlying research expenditures using a classification scheme based on standard therapeutic class codes (such as the IMS Worldwide Therapeutic Classification Scheme) modified to reflect the organizational structure of the firms in the sample.[30] All patents are counted by earliest world-wide priority date of the invention.

PRO-PUBLICATION PROMOTION POLICIES. We measure this aspect of organizational design at the firm-year level. The extent to which firms reward effort devoted to the pursuit of excellence in fundamental science, is measured by a variable which we label PROPUB for "pro-publication". This is a Likert scale variable coded (1-5) which measures the degree to which the firm promotes individuals based on their standing in the external scientific community. PROPUB was constructed from extensive interviews with each firm's senior managers and scientists, covering various aspects of the firm's history of organizational structure and incentives. Firms were scored according to the extent to which they encourage their scientists to participate in conferences and publish in the open scientific literature, use publication in peer-reviewed journals as an explicit criteria for promotion or other rewards, or otherwise link their internal research effort to the wider scientific community. Through appropriate selection of interview candidates and cross-checking of responses we were able to construct these scores for each firm back to 1975. As we discuss in the main text, we overcome some of the potential problems with the use of the Likert measure PROPUB by using an alternative measure in some of the analysis based on changes in PROPUB within a given firm (allowing a one-year adjustment period). We define "HIGH" PROPUB DUMMY to be equal to one for all years after a firm has increased its level of PROPUB and zero otherwise.

TECHNOLOGICAL OPPORTUNITY INDICES. We construct several measures of the technological opportunity present in the environment by controlling for patenting both in related therapeutic areas and by other firms in the industry. In particular, we calculate three "shocks" to opportunity, exploiting the "POISSON" methodology described in Appendix B below, along the following dimensions: (a) the number of patents granted to competitor firms in the focus category (COMPETITOR PATENTS) and (b) the number of patents granted to a focus firm in therapeutic

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[29] Derwent's *World Patent Index* compiles comprehensive data on international patent filings, allowing us to identify those granted in multiple jurisdictions. Application costs rise roughly proportionately with the number of jurisdictions, and firms rarely file in all possible jurisdictions, let alone all major markets (e.g. all OECD countries.) By excluding inventions where the firm does not file in at least two out of three major jurisdictions, we are therefore left with a count of "important" patents. Derwent's database goes back to 1962, though much less comprehensive data is available before 1970.

[30] Where we were not confident about this matching, research programs and patents are assigned to a "Misc/NEC" class and not used in the analysis.

areas similar to the focus class (RELATED PATENTS). The control measures for competitors' patents are drawn from a broader cross-section of 29 leading worldwide pharmaceutical firms.

SAMPLE SELECTION. With a complete, balanced data set (all firms participating in all programs in all years from 1975-1990), the data set would consist of 7040 firm-program-year observations. The data set is unbalanced, however, affecting the size of the sample. First, and most importantly, firms initiate and discontinue research programs throughout the sample. We only include observations for which a research program is "active" in the sense that the firm actively engaged in at least some research in a particular therapeutic area (resulting in the loss of 2319 potential observations). As well, some firms are involved in mergers and some firms' discovery spending is not observed continuously between 1975-1990 (resulting in a net loss of 978 observations). Further, 1164 observations are removed because both ΔRESEARCH and PATENT SHOCK*RESEARCH$_{t-1}$ are equal to 0. Finally, since we are interested in whether firms who have a given level of PROPUB tend to be more responsive to applied research outputs shocks in their capital budgeting, we allow a one-year "adjustment" for those firms who switch PROPUB during the sample period, resulting in the loss of 139 observations. Taken together, these sampling rules result in a final data set of 2417 observations which we use throughout our empirical analysis.

A.2. Descriptive Statistics

Table 2 presents the summary statistics for this final data set. Beginning with the FUNDING variables, the average annual budget for an active research program is $1.58 million, with the average firm spending $38 million on drug discovery research annually (note all financial measures are in 1986 dollars, using the BEA Biomedical Research Price Deflator). On average, program receive a modest "boost" over time; however, ΔRESEARCH varies widely across programs and over time.

In return for this investment in research, the programs in this sample yield an average of 3.3 patents per year and firms are each granted over 90 patents per year. Although some programs produce more than 15 patents per year, no patents are produced in 30 percent of program-years, and, for 76% of the annual observations, fewer than five patents are produced.

While the promotion policy variable PROPUB is centered around the mean of the 5-point Likert scale, there exists substantial variation along these dimensions both across firms and across time (ANOVA reveals that the variance is evenly divided across the within-firm and between-firm dimensions). The measure capturing the presence of a "switch" in PROPUB ("HIGH" PROPUB DUMMY) captures more than one quarter of the sample.

APPENDIX B: ESTIMATING THE APPLIED RESEARCH OUTPUT SHOCK

"Surprises" in patenting play a key role in this paper as signals of effort supplied by research workers in applied research. This section discusses a variety of possible methods for constructing this "surprise" variable.

Our measure of applied research output is "important patents" attributed to the research program. Pharmaceutical firms file patent applications on discoveries which show commercial promise promptly, and we believe that at least in this context they are a good measure of successful outcomes in applied research projects.[31] Using time series on each research group's patenting (PATENTS), one could construct a simple measure of the applied research output "shock" as the difference between the research group's observed and expected patenting rate,

$$PATENT\ SHOCK_{i,j,t} = PATENTS_{i,j,t} - \mu^{PATS}_{i,j,t} \qquad \text{(B1)}$$

where μ^{PATS} is an estimate of expected patent output from program *i* in firm *j* in year *t*. Two issues arise in adapting this formula to the investment sensitivity equation estimated in Section IV. First, this measure must be made comparable across programs, and so we need to take account of systematic technological differences across programs in the number of patents generated by a given amount of research spending. These differences may be large: a million dollars spent on screening for antibiotics may generate as much as five times as many patentable candidate compounds as a similar level of resources devoted towards cancer research. As well, programs vary widely in terms of their absolute size, and we need to adjust our shock measure so that the "budget sensitivity" parameter implies a proportional impact on the budget for programs of different size. Consequently, though the results do not depend on whether we control for these two "proportionality" problems, we address these issues in the empirical work by expressing the applied research output shock as:

$$SHOCK_{i,j,t-1} = \frac{PATENTS_{i,j,t-1} - \mu^{PATS}_{i,j,t-1}}{\mu^{PATS}_{i,j,t-1}} * I_{i,j,t-2} \qquad \text{(B2)}$$

To use (B2), we require a consistent estimate of μ^{PATS}, the expected level of patenting for a given firm-program-year. Obviously, the econometrician cannot construct an exact measure of this expectation; each firm has access to richer information about its own programs than outside

[31] We recognize that patents may be filed on discoveries which are quite far from commercial application: in this context, putting a candidate compound into clinical trials. There is also the possibility that strategic considerations may lead firms to delay filing applications, or to pursue large numbers of otherwise insignificant applications in an effort to construct a protective "thicket" around a core discovery. However prior work with these data, as well as interviews with firm personnel lead us to believe that these problems are unlikely to be a serious source of systematic bias. Note also that we count only "important" patents filed in two out of three major jurisdictions worldwide, and we date applications by their worldwide priority date.

observers. However, it is feasible to attempt to estimate the firm's expectation of patent output by making assumptions about what the firm pays attention to in this process. Here we discuss three alternatives, which attempt to "span the space" of reasonable models. Results presented in the body of the paper use only the third (most sophisticated) of these expectations models.

In the first, most naive, of these models, we assume that the firm's expectation is simply the level of patenting in the immediate prior year (i.e., annual patent counts follow a first-order Markov process):

$$\mu^{M}_{i,j,t} = PATENTS_{i,j,t-1} \qquad \text{(B3)}$$

This measure has obvious shortcomings, since it assumes that decision makers have only a very limited "memory" and are basing decisions on an extraordinarily limited information set. To construct our second measure, we model the firm's expectations about each research group's performance as being based on the assumption that patent counts follow a Poisson process whose rate parameter, $\mu^{POISSON}$ can be estimated from past realizations of patent output. Specifically, $\mu^{POISSON}$ is just the mean number of patents per period over all observed periods to date,

$$\mu^{P}_{i,j,t} = \frac{1}{t - T_0} \sum_{s=T_0}^{t-1} PATENTS_{i,j,s} \qquad \text{(B4)}$$

While data are only available for the period 1975-1990 for some of our other variables, we have much longer time series on the patent output of each research program, and $\mu^{POISSON}$ is constructed from as many as 30 years of data on patent counts.

While it incorporates the historical trend in patenting in the research program, $\mu^{POISSON}$ takes no account for the level of funding provided to each research program. It is reasonable to suppose that managers base their expectations about the level of patent output on both the history of patenting and the amount of resources currently available. To allow for this, we compute a third measure of expected patents, $\mu^{ADAPTIVE}$ based on the notion that manager's expectations are updated adaptively in response to both of these factors. $\mu^{ADAPTIVE}$ is constructed using a two-stage procedure based on a regression-based weighting of the $\mu^{POISSON}$ measure and $RESEARCH_{t-1}$, the level of funds provided to the research program in the previous period. To do this, we first compute $\mu^{POISSON}$, and then in the second stage run a Poisson regression of observed patenting on the level of research by each research group and $\mu^{POISSON}$ (this amounts to estimating the regression with a distributed lag on the dependent variable). $\mu^{ADAPTIVE}$ is thus the fitted value of the level of patenting resulting from this Poisson regression,

$$\mu^{A}_{t} = \exp(\hat{\lambda}_0 + \hat{\lambda}_P \mu^{P}_{t-1} + \hat{\lambda}_R RESEARCH_{t-1}) \qquad \text{(B5)}.$$

Of course, it would be possible to extend the logic of $\mu^{ADAPTIVE}$ to take the fitted value from any model of the drivers of patenting productivity. Though $\mu^{ADAPTIVE}$ is a marked improvement over just using past realizations of the patenting process, the data we have available is only a small subset of the information available to the decision-maker in reality. However, we are reluctant to impose an overly sophisticated model (e.g., a vector autoregression model which minimizes ex-post forecasting error) for two reasons. In the first place, it is simply counterfactual. Research managers can and do use sophisticated quantitative tools (Nichols, 1994), but a considerable body of research has demonstrated that practicing managers rely extensively on heuristics and rules of thumb. In the second place, an overly specified model is actually unhelpful in this context: a fully saturated statistical model will result in "shocks" which contain less and less "signal" about unanticipated performance and more and more true random noise! Consequently, $\mu^{ADAPTIVE}$ is our preferred measure of expectations, since we believe that it incorporates a realistic amount of information. While we doubt that research managers would update expectations without taking into account the amount of funds that had been invested in a program, we are skeptical that they account (in a consistent way) for factors such as the size or structure of the firm's overall research activities.

In Table B1 we present the results of estimating the Poisson regression which forms the basis for $\mu^{ADAPTIVE}$. The dependent variable is the count of patents applied for, and the explanatory variables are $\mu^{POISSON}$ and the log of RESEARCH. Estimated coefficients on both variables are highly significant and have the anticipated positive sign.

Table B2 summarizes the three alternative measures of expected patents. Note again that they are calculated from much longer time series on patenting and research expenditures than the sample used in the regressions in the body of the paper. For the MARKOV and POISSON measures, calculation is based only on years for which the program is "alive" i.e., the MARKOV measure is missing for those programs which were not at least minimally active in the immediately prior year, and the POISSON measure requires that the firm-program is minimally active in at least one year in the past. The final column of reports the sample statistics for the ADAPTIVE measure. In all cases, the reported descriptive statistics are for the sample of 2417 observations used in the main regressions. It is useful to note that the expectation for these three measures are similar though not identical (the average MARKOV expectation is 3.11, the average POISSON expectation is a lower value of 2.43, and the average ADAPTIVE expectation lies in between at 2.89).

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TABLE 1
VARIABLES AND DEFINITIONS

Variable Name	Definition	Unit of Observation
FUNDING VARIABLES		
$RESEARCH_{i,j,t}$	Annual expenditure on drug discovery in program *i* by firm *j* in year *t* in $M 1986, excluding clinical development	program-firm-year
$\Delta RESEARCH_{i,j,t}$	$RESEARCH_{i,j,t}$ - $RESEARCH_{i,j,t-1}$	program-firm-year
FIRM $RESEARCH_{j,t}$	Annual overall expenditure on drug discovery by firm *j* in year *t* in $M 1986, excluding clinical development	firm-year
PATENTING VARIABLES		
$PATENTS_{i,j,t}$	Annual number of patent applications in year t granted in at least two of U.S., Japan, EU; by worldwide priority date	program-firm-year
FIRM $PATENTS_{j,t}$	Annual overall number of patent applications in year t granted in at least two of U.S., Japan, EU; by worldwide priority date	firm-year
ORGANIZATIONAL DESIGN VARIABLES		
$PROPUB_{j,t}$	Likert scale variable between 1 and 5, where higher values indicate that the firm promotes individuals on the basis of their standing in the scientific community	firm-year
"HIGH" PROPUB $DUMMY_{j,t}$	Dummy equals 1 for firm j for all years after "switch" to higher level of PROPUB by firm j, 0 else	firm-year
MEASURES OF SHOCK TO APPLIED RESEARCH OUTPUT		
PATENT $SHOCK_{i,j,t}$	$\frac{PATS_{i,j,t-1} - E[PATS_{i,j,t-1}]}{E[PATS_{i,j,t-1}]}$	program-firm-year
SHOCK $(x^A)_{i,j,t}$	$PATENT\ SHOCK_{i,j,t} * I_{i,j,t-2}$	program-firm-year
MEASURES OF TECHNOLOGICAL ACTIVITY		
COMPETITOR $PATENTS_{i,j,t}$	Annual number of patent applications granted to 29 competitor firms	program-firm-year
RELATED $PATENTS_{i,j,t}$	Annual number of patent applications granted in classes related to a given program	program-firm-year

TABLE 2
MEANS AND STANDARD DEVIATIONS

Variable	N	Mean	Standard Deviation
RESEARCH BUDGET VARIABLES			
RESEARCH	2417	1.58	3.07
ΔRESEARCH	2417	0.10	1.03
FIRM RESEARCH	2417	38.20	26.86
# OF RESEARCH PROGRAMS	2417	10.10	4.37
PATENTING VARIABLES			
PATENTS	2417	3.30	4.60
FIRM PATENTS	2417	90.56	60.29
ORGANIZATIONAL DESIGN VARIABLES			
PROPUB	2417	3.35	1.46
"HIGH" PROPUB DUMMY	2417	0.26	0.44
MEASURES OF SHOCK TO APPLIED RESEARCH OUTPUT			
PATENT SHOCK	2417	0.08	1.30
SHOCK (x^A)	2417	0.26	3.22
MEASURES OF TECHNOLOGICAL OPPORTUNITY			
COMPETITOR PATENTS	2417	40.79	40.79
RELATED PATENTS	2417	7.58	7.58

TABLE 3
SENSITIVITY OF RESEARCH BUDGETS TO APPLIED RESEARCH OUTPUT SHOCKS (x^A)

DEPENDENT VARIABLE = ΔRESEARCH	
YEAR	0.011 (0.005)
SHOCK	0.043 (0.007)
TECHNOLOGICAL OPPORTUNITY CONTROLS	
COMPETITOR PATENTS	0.008 (0.008)
RELATED PATENTS	-0.007 (0.012)
SCALE & MOMENTUM CONTROLS	
$RESEARCH_{t-1}$	-0.018 (0.008)
$\Delta RESEARCH_{t-1}$	0.122 (0.022)
CONSTANT	-0.789 (0.380)
N	2417.00
R-squared	0.04
H_0: $\gamma_{i,j} = \gamma_{i,j}$ for all i,j F(13, 2396) = 8.23, rejected at 1% level	

TABLE 4
RESEARCH FUNDING CHANGE
BY PATENT SHOCK & BASIC RESEARCH INCENTIVE INTENSITY

	LOW PROPUB (PROPUB = 1, 2, or 3)	HIGH PROPUB (PROPUB = 4 or 5)
PATENT SHOCK < 0	-0.02	0.03
PATENT SHOCK > 0	0.16	0.38
"Boost" in Research Funding for Positive Shock	0.18	0.35
Difference in "Boost" by Organizational Form	.17 = 94%	

TABLE 5
γ^A AND PROPUB
THE "REGIME" LEVEL

DEPENDENT VARIABLE = $\hat{\gamma}^A$ **N=14**		
	(5-1)	**(5-2)**
PROPUB	0.034 (0.016)	0.034 (0.019)
YEAR		-0.0006 (0.0065)
CONSTANT	-0.092 (0.058)	-0.138 (0.489)

TABLE 6
RESEARCH BUDGET SENSITIVITY TO APPLIED RESEARCH OUTPUTS SHOCKS: INTERACTION WITH PROPUB
PROGRAM-FIRM-YEAR "POOLED" SAMPLE

DEPENDENT VARIABLE = ΔRESEARCH N=2417				
	(6-1)	**(6-2)**	**(6-3)**	**(6-4)**
SHOCK	0.059 (0.020)	0.006 (0.025)	0.008 (0.025)	
SHOCK INTERACTION TERMS				
SHOCK*PROPUB ($\rho^{A,B}$)		0.015 (0.004)	0.015 (0.004)	0.017 (0.005)
SHOCK* YEAR	-0.001 (0.002)	-0.001 (0.002)	-0.001 (0.002)	
SHOCK*[Year Fixed Effects]				Significant
DIRECT EFFECTS OF PROPUB AND YEAR				
PROPUB		0.018 (0.014)	0.021 (0.015)	0.021 (0.015)
YEAR	0.011 (0.004)	0.0092 (0.0047)	0.0087 (0.0048)	
[Year Fixed Effects]				Significant
TECHNOLOGICAL OPPORTUNITY CONTROLS				
COMPETITOR PATENTS			0.009 (0.008)	0.008 (0.008)
RELATED PATENTS			-0.007 (0.012)	-0.010 (0.012)
PROGRAM SCALE AND MOMENTUM CONTROLS				
$RESEARCH_{t-1}$			-0.020 (0.008)	-0.009 (0.008)
$\Delta RESEARCH_{t-1}$			0.122 (0.022)	0.112 (0.022)
CONSTANT	-0.028 (0.050)	-0.070 (0.062)	-0.048 (0.062)	-0.033 (0.056)
R-Squared	0.022	0.028	0.041	0.071

TABLE 7
RESEARCH BUDGET SENSITIVITY TO APPLIED RESEARCH OUTPUTS SHOCKS: INTERACTION WITH PROPUB. PROGRAM-FIRM FIXED EFFECTS

DEPENDENT VARIABLE = ΔRESEARCH N=2417				
	(7-1) Program-Firm FEs and Controls	**(7-2)** (7-1) w/ PROPUB CHANGE	**(7-3)** Program-Firm FEs, Program-specific trends, and PROPUB	**(7-4)** (7-3) w/ Controls
[Program-Firm FE]	Insignificant	Insignificant	Insignificant	Insignificant
SHOCK INTERACTION TERMS				
SHOCK*[Program-Firm FE]	Significant	Significant	Significant	Significant
SHOCK*HIGH PROPUB DUMMY ($\rho^{A,B}$)		0.151 (0.040)	0.115 (0.045)	0.121 (0.044)
SHOCK* YEAR	0.001 (0.002)	-0.004 (0.003)		
SHOCK*YEAR*[Program FE]			Significant	Significant
DIRECT EFFECTS OF PROPUB AND YEAR				
"HIGH" PROPUB DUMMY		0.007 (0.088)	-0.113 (0.089)	-0.012 (0.088)
YEAR	0.022 (0.005)	0.023 (0.007)		
[Year Fixed Effects]			Significant	Significant
TECHNOLOGICAL OPPORTUNITY CONTROLS				
COMPETITOR PATENTS	0.013 (0.012)	0.013 (0.012)		0.015 (0.012)
RELATED PATENTS	-0.004 (0.015)	-0.002 (0.015)		-0.008 (0.014)
PROGRAM SCALE AND MOMENTUM CONTROLS				
$RESEARCH_{t-1}$	-0.156 (0.016)	-0.159 (0.016)		-0.171 (0.016)
$\Delta RESEARCH_{t-1}$	0.145 (0.024)	0.138 (0.024)		0.131 (0.024)
CONSTANT	0.701 (0.957)	0.656 (0.956)	0.572 (1.027)	0.191 (1.000)
R-Squared	0.291	0.296	0.289	0.331

TABLE B1
"ADAPTIVE" MODEL FOR EXPECTED PATENT PRODUCTION

Poisson regression:

$$\ln E(PATENTS_t) = \lambda_0 + \lambda_P \mu_{t-1}^P + \lambda_R \ln(RESEARCH_{t-1})$$

Where μ_t^P is given by $\mu_t^P = \frac{1}{t - T_0} \sum_{s=T_0}^{t-1} PATENTS_s$

DEPENDENT VARIABLE= PATENTS$_t$ N=3446	
μ_{t-1}^P	0.146 (0.002)
ln(RESEARCH$_{t-1}$)	0.121 (0.007)
CONSTANT	0.666 (0.017)
Log-Likelihood	-8511.30

TABLE B2
SUMMARY STATISTICS FOR ALTERNATIVE MEASURES OF EXPECTED PATENTS

Expected Patent Production Measure	μ_t^M	μ_t^P	μ_t^A
Definition	PATS$_{t-1}$	$\frac{\sum_{s=0}^{t-1} PATS_s}{t-1}$	$e^{\hat{\lambda}_0 + \hat{\lambda}_P \mu_{t-1}^P + \hat{\lambda}_R \ln(RESEARCH_{t-1})}$
Mean Expectation:	3.11	2.43	2.89
Std. Deviation of Expectation	4.34	2.95	3.47
N	2417	2417	2417.00