

Is there complementarity or substitutability between internal and external R&D strategies?

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IS THERE COMPLEMENTARITY OR SUBSTITUTABILITY BETWEEN INTERNAL AND EXTERNAL R&D STRATEGIES?

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Is There Complementarity or Substitutability between Internal and External R&D Strategies?

Abstract

The mixed picture of extant research on the relationship between internal and external R&D prompts us to ask such a question: under what conditions is there complementarity or substitutability between different R&D strategies? The goal of this paper is to contribute to the empirical literature by advancing and testing the contingency of the relationship between internal and external R&D strategies in shaping firms' innovative output. Using a panel sample of incumbent pharmaceutical firms covering the period 1986–2000, our empirical analysis suggests that the level of in-house R&D investments, which is characterized by decreasing marginal returns, is a contingency variable that critically influences the nature of the link between internal and external R&D strategies. In particular, internal R&D and external R&D, through either R&D alliances or R&D acquisitions, turn out to be complementary innovation activities at higher levels of in-house R&D investments, whereas at lower levels of in-house R&D efforts internal and external R&D are substitutive strategic options. These findings are robust to alternative specifications and estimation techniques, including a dynamic perspective on firm innovative performance.

Key words: Complementarity; Substitutability; Internal R&D; External R&D; Innovative output; Pharmaceutical Industry; Biotechnology

INTRODUCTION

The ability of firms to continually update their technological know-how and capabilities is becoming an imperative for competitive survival (Christensen, 2000; Foster and Kaplan, 2001). In that context, firms can pursue multiple approaches to innovative renewal. One path is to develop and nurture in-house research and development (R&D) competencies. In technologybased industries, perhaps the most salient mechanism of firms' innovative renewal is investing in internal R&D (Knott and Posen, 2009). Another path is found in the use of external technology sourcing. Powell *et al.* (1998, 1996) point out that in industries characterized by complex and rapidly expanding knowledge bases, the locus of innovative renewal lies within a broad 'network' of learning rather than within the boundaries of individual firms (see also Breschi and Malerba, 1997; Edquist, 1997). As a consequence, incumbent firms frequently need to leverage their external networks to source new technology and capabilities, especially within an emerging, new technological regime (Bettis and Hitt, 1995; Kranenburg and Hagedoorn, 2008; Nicholls-Nixon and Woo, 2003; Rothaermel and Hess, 2007).

Recent years have witnessed more and more companies pursuing an 'open innovation' (Chesbrough, 2003) strategy by leveraging internal and external knowledge flows in parallel to build and hone their innovative capabilities. Concurrent with the open innovation approach, a rather voluminous literature has emerged that examines the relationship between in-house R&D and external technology sourcing. At the heart of this literature is the discussion of the complementarity or substitutability between internal and external R&D strategies for managing innovation, a debate that has been accompanied by mixed empirical evidence¹. On the one hand,

¹ The study of complementarity between activities can be traced back to the theory of supermodularity (Milgrom and Roberts, 1990, 1995). Complementarity implies that adding one activity while the other is already being performed will result in higher incremental innovative performance than when adding the activity in isolation, whereas

a diverse set of studies demonstrate that internal R&D and external technology sourcing are complementary innovation activities, suggesting the interrelatedness of internal and external R&D in improving innovative performance (Brown and Eisenhardt, 1997; Caloghirou *et al.*, 2004; Cassiman and Veugelers, 2006; Lokshin *et al.*, 2008; Schmiedeberg, 2008; Tripsas, 1997; Tsai and Wang, 2008; Veugelers, 1997). And on the other, a host of empirical work identifying the linkage between internal and external sources of innovation have found substitutability instead (Audretsch *et al.*, 1996; Blonigen and Taylor, 2000; Higgins and Rodriguez, 2006; Laursen and Salter, 2006; Watkins and Paff, 2009). The above mixed picture of extant research on the relationship between internal and external R&D prompts us to ask the question: under what conditions is there complementarity or substitutability between different R&D strategies? The goal of this paper is to contribute to the empirical literature by advancing and testing the contingency of the relationship between internal and external and external R&D strategies in shaping firms' innovative output.

Our study focuses on the global pharmaceutical industry as it provides an ideal setting for us to explore how incumbent firms organize R&D strategies in their quest for innovation within a new biotechnology-based technological regime. Using a panel sample of 83 incumbent pharmaceutical firms covering the period 1986–2000, our empirical analysis suggests that the level of in-house R&D investments, which is characterized by decreasing marginal returns, is a contingency variable that critically influences the nature of the link between internal and external R&D strategies. In particular, internal R&D and external R&D, through either R&D alliances or R&D acquisitions, turn out to be complementary innovation activities at higher levels of in-house

substitutability refers to the case in which adding one activity can actually decrease the marginal or incremental innovative performance of other activities (Cassiman and Veugelers, 2006; Mohnen and Röller, 2005).

R&D investments, whereas at lower levels of in-house R&D efforts internal and external R&D are substitutive strategic options.

The paper proceeds as follows. After an overview of the growing literature on internal and external R&D strategies of firms, we formulate the hypotheses to be tested. We then turn to methodology, describing the sample, the model, and the estimation techniques. The empirical results are presented in the next section, followed by a variety of robustness tests. The paper concludes with a discussion of the results and some implications for future research.

THEORY AND HYPOTHESES

To the extent that R&D is an important, and perhaps even the most important, contributor to productivity growth and innovation (Griliches, 1979; Scherer, 1982), the impact of R&D investments on firm innovation has attracted enormous attention in the literature. In their seminal work on the R&D-patents relationship, Hausman *et al.* (1984) maintain that, rather than the propensity to patent just declining exogenously over time, firms are getting less patents from their more recent R&D investments, implying a decline in the 'effectiveness'or productivity of R&D. The role of R&D in patenting, as a major indicator of innovation, has later on been studied extensively (Blundell *et al.*, 1995; Blundell *et al.*, 2002; Crépon and Duguet, 1997; Guo and Trivedi, 2002; Montalvo, 1997), with the overall R&D elasticity of patents varying from 0.4 to 0.7 and thereby suggesting decreasing returns to scale (Gurmu and Perez-Sebastian, 2007).

Amongst others, Griliches (1990) suggests that the estimates for R&D elasticity vary and depend on firm size, with larger firms showing a lower R&D productivity² (see also Acs and Audretsch, 1991). In line with this thinking, Graves and Langowitz (1993) show that firms in the

 $^{^{2}}$ Griliches (1990) also points to the possibility of a reverse causality from R&D to patents in the knowledge production function. Consequently, R&D is unlikely to be strictly exogenous.

pharmaceutical industry experience decreasing returns to scale in R&D as the level of their R&D expenditures rises. Their central argument is that innovative effectiveness decreases with increasing firm R&D investments and, by association, with firm size. Cohen and Klepper (1996) explain why larger firms can still prosper despite the lower average productivity of their R&D, as the greater output over which large firms can apply their R&D enables them to profit more from R&D than smaller firms, which leads them to undertake more R&D projects at the margin. A large number of recent studies examine the impact of firm R&D investments on innovation as well (e.g., Griffith *et al.*, 2004; Hagedoorn and Duysters, 2002; Lokshin *et al.*, 2008; Love and Roper, 2002; Rothaermel and Hess, 2007) and find diseconomies of scale in R&D in most cases. The above leads us to a baseline hypothesis on the effect of internal R&D on innovative output, which is to be seen as a starting point for our analysis of the simultaneous interactions between different R&D strategies.

Hypothesis 1: There are decreasing returns to scale in internal R&D. Firm innovative output initially increases with investment in internal R&D, this rate of increase diminishes at higher levels of internal R&D investments though.

Openness of firms to external knowledge sources is another key element for knowledge creation and innovation (Arora and Gambardella, 1990; Cohen and Levinthal, 1990). A diverse set of studies emphasize that access to external R&D can be leveraged to enhance the efficiency of internal R&D investments. Brown and Eisenhardt (1997) demonstrate that firms that leverage external technology sourcing as a complement to internal R&D investments in order to probe and access cutting-edge knowledge are most successful in their new product introductions. Tripsas (1997) finds, while studying the continued survival of incumbent firms when confronted with

radical technical change, that a combination of internal R&D and external technology sourcing through alliances positively reinforces firms' innovative output³. In a similar fashion, Caloghirou *et al.* (2004) show the parallel positive role of internal R&D capabilities and their interaction with external sources of knowledge in raising innovative performance. Focusing on the choice between make or buy decision by firms, Cassiman and Veugelers (2006) find that internal R&D and external knowledge acquisition are complementary innovation activities. The moderating effect of internal R&D is also emphasized by Tsai and Wang (2008). Their findings suggest that the contribution of external technology acquisition to firm performance increases with the level of internal R&D efforts⁴. In their dynamic panel study of Dutch manufacturing firms, Lokshin *et al.* (2008) also find that combing internal and external R&D significantly contributes to productivity growth, with a positive impact of external R&D only evident in case of sufficient internal R&D.

However, a number of other papers identifying the relationship between in-house and external sources of innovation have found a substitutability instead of a complementarity effect. Audretsch *et al.* (1996) suggest that internal and external R&D are substitutes in low-technology industries but not in high-technology industries. Blonigen and Taylor (2000) find a substantial inverse relationship between R&D intensity and acquisition activities among electronic and electrical equipment firms. Higgins and Rodriguez (2006) demonstrate that firms that are experiencing deterioration in internal R&D productivity are more likely to engage in technology acquisitions, implying that firms may pursue one mode of innovation to compensate for their ineffectiveness in another mode. In their investigation into the role of firm openness in explaining

³ Also see the study by Schmiedeberg (2008), who provides further evidence for significant complementarities between internal R&D and R&D cooperation, but casts doubts on the complementarity between internal and contracted R&D.

⁴ This is consistent with the findings by Veugelers (1997) who shows that external knowledge sources stimulate the innovative productivity of internal research activities at lease for firms with internal R&D departments.

innovative performance, Laursen and Salter (2006) report evidence of a substitution effect between internal R&D and external search strategies. Consistent with previous studies, mentioned in the above, though in a rather different research context, Watkins and Paff (2009) find a substitute relationship between in-house R&D and external basic research when the relative tax prices of each category of research change⁵.

By and large, these various strands of the empirical literature indicate the inconclusive nature of the debate about the complementarity or substitutability between internal and external R&D strategies in shaping firms' innovative performance. We therefore are faced with two opposing hypotheses:

Hypothesis 2-a: Internal and external R&D strategies complement each other and hence increase a firm's innovative output.

Hypothesis 2-b: Internal and external R&D strategies substitute for each other and hence decrease a firm's innovative output.

METHODOLOGY

Research Setting

The research setting for testing our hypotheses is the global pharmaceutical industry, which is on the verge of profound mutations as a new biotechnology-based technological regime has emerged. More in particular, we will focus on incumbent pharmaceutical companies attempting to build up innovative capabilities within this new technological regime. Incumbent pharmaceutical firms are defined as pharmaceutical firms that were in existence prior to the emergence of biotechnology.

⁵ R&D policies such as R&D grants, subsidies, and tax incentives often treat different types of R&D differently, which tends to affect the composition of firms' R&D budgets (Watkins and Paff, 2009). See similar studies by Cappelen *et al.* (2008), Czarnitzki *et al.* (2004), Parsons and Phillips (2007).

These companies, such as Bayer, Hoffmann-La Roche, Merck, and Pfizer, are generally mature and very large firms that dominated the industry since the 1940s. Since the mid 1970s, new biotechnology brought along significant scientific and technological breakthroughs in genetic engineering (recombinant DNA, 1973) and hybridization (monoclonal antibodies, 1975). These advances have served as a radical process innovation for established pharmaceutical companies in the way new drugs are discovered and developed (Pisano, 1997) and they are even to affect the core capabilities needed to remain competitive (Nagarajan and Mitchell, 1998). This development provides an ideal setting for us to explore how incumbents organize R&D strategies in their quest for innovation within a new technological regime.

Data and Sample

We use data from several major sources. (1) The source for the patent data is the Technology Profile Report maintained by the U.S. Patent and Trademark Office (USPTO), an agency of the U.S. Department of Commerce. We obtained detailed data comprising the complete set of all biotechnology patents granted to global pharmaceutical firms annually since the emergence of biotechnology⁶. (2) The MERIT-CATI database, which is developed and maintained by researchers at the Maastricht Economic Research Institute on Innovation and Technology (MERIT). The CATI (Cooperative Agreements and Technology Indicators) database documents

⁶ The complete set of biotechnology patents refers to the biotechnology patents, as identified by the USPTO, in the following patent classes: 424 [Drug, bio-affecting and body treating compositions (424/9.1-424/9.2, 424/9.34-424/9.81, 424/85.1-424/94.67, 424/130.1-424/283.1, 424/520-424/583, 424/800-424/832)], 435 [Chemistry: Molecular biology and microbiology (435/1.1-435/7.95, 435/40.5-435/261, 435/317.1-435/975)], 436 [Chemistry: Analytical and immunological testing (436/500-436/829)], 514 [Drug, bio-affecting and body treating compositions (514/2-514/22, 514/44, 514/783)], 530 [Chemistry: Natural resins or derivatives; peptides or proteins; lignins or reaction products thereof (530/300-530/427, 530/800-530/868)], 536 [Organic compounds (536/1.11-536/23.74, 536/25.1-536/25.2)], 800 [Multi-cellular living organisms and unmodified parts thereof and related processes], 930 [Peptide or protein sequence], PLT [Plants].

technological partnerships⁷ in multiple industries around the globe. In this study we specifically focused on R&D alliances between established pharmaceutical companies and new biotechnology ventures. (3) The SDC (Securities Database Corporation) Platinum of Thomson Financial is used for data on R&D acquisitions of new biotechnology companies by incumbent pharmaceutical firms. This database contains information on the year of acquisition, details on the (parent) acquirer and the (parent) target, and a brief description of the acquisition transaction. (4) Financial data were cross-tracked through Compustat and Datastream (Thomson Financial). All financial data were converted to U.S. dollars and inflation-adjusted.

To mitigate a potential survivor bias, we started with a comprehensive set of global pharmaceutical firms alive in 1986 according to various industry sources⁸. In this manner we identified 89 incumbent pharmaceutical firms, defined as pharmaceutical firms that were in existence prior to the emergence of biotechnology in the mid 1970s. We then proceeded with a family tree analysis⁹ on each of the 89 firms for the 1986–2000 time period. Through this analytical process, 6 horizontal mergers and acquisitions were ascertained among the incumbent pharmaceutical firms. As a horizontal merger or acquisition took place, we added to the survivor the historical data of the acquired company (i.e. the inactive) and then tracked the surviving entity forward¹⁰. Such scrutiny procedure ultimately renders a slightly unbalanced panel sample of 83 firms covering the 15-year period from 1986 to 2000 (i.e., 1139 firm-year observations).

⁷ CATI only collects cooperative agreements in which a combined innovative activity or an exchange of technology is engaged by at least two industrial partners. The first phase of data collection is described in Hagedoorn (1993), Duysters and Hagedoorn (1993).

⁸ To draw the sample for this study we tracked Compustat, Datastream, Amadeus, SIC reports, Ernst and Young's Annual Biotech Industry Reports, Scrip's Pharmaceutical Yearbook, amongst others.

⁹ For the rationale for constructing family trees, see Rothaermel and Hess (2007). When firms were acquired by outsiders, they were included as unbalanced data. A variety of industry sources were checked throughout the family tree analysis, including Compustat/Standard and Poor's, Encyclopedia, and company homepage.

¹⁰ In Compustat, the treatment of mergers and acquisitions seeks to preserve historical company data for the acquiring company whenever possible. It is rare that a new company is added as a result of a merger. There are usually no changes to the surviving company's company-level identifiers, whereas the acquired company subsequently becomes inactive in the database the month following the merger or acquisition. Two basic accounting

It is important to indicate that while we collected patent data until the end of 2003, we purposefully ended the study in 2000 to overcome a potential right truncation bias. This measure was taken in that all patent data used in the study are based upon patent applications granted, and the application date is only reported when the patent is actually granted. A grant lag as such is generally three years after the patent was originally applied for¹¹ (Biotechnology Industry Organization, 2008). In contrast, there is essentially no lag time between the completed invention and the patent application date, which is on average no more than three months (Darby and Zucker, 2003). Thus, the application date of a granted patent is closely tied to the timing of the new knowledge creation and should be used as the relevant time placer for patents (Hall *et al.*, 2001; Trajtenberg, 1990).

Measures

Dependent Variable

Innovative Output We focus on the patenting frequency, i.e., the number of annual biotechnology patents (*Biotech Patents*) granted to incumbent pharmaceutical firms to proxy for their innovative output within the new technological regime. Annual patent counts are generally accepted as one of the most appropriate indicators that enable researchers to compare the inventive or innovative output of companies in terms of new technologies, new processes, and new products (Hagedoorn and Cloodt, 2003). Patents are directly related to inventiveness

methods are used to account for mergers and acquisitions, the "purchase method" and the "pooling of interest method". In this study we principally followed the "purchase method" since this is the way that the majority of mergers and acquisitions are accounted for. In June 2001 the Financial Accounting Standard Board eliminated the "pooling of interest" and requires that all business combinations be accounted for by the "purchase method" (FASB Statement 141).

¹¹ The USPTO evaluates patent applications and grants patents. Patents usually last 20 years from the date on which the patent application is filed (not when it is issued). Thus, the enforceable term of a patent is between 17 and 20 years; exactly how long depends on how long the application is under PTO review (i.e., the grant lag). The PTO provides a three-year period for the agency to issue a patent.

(Walker, 1995), and represent an externally validated measure of technological novelty (Griliches, 1990). Studies also show that patents are well-correlated with other indicators of innovative output (Hagedoorn and Cloodt, 2003; Stuart, 2000).

Independent Variables

Internal R&D We measure a pharmaceutical company's internal R&D investments by its *R&D expenditures*. As highlighted in prior research, R&D expenditures are primarily taken as an input indicator of a firm's efforts in establishing R&D that might eventually lead to innovative output (Griliches, 1990, 1998; Hitt *et al.*, 1997). R&D efforts can also signal innovative competences that are found to affect the performance of companies, particularly in high-technology industries (Duysters and Hagedoorn, 2001; Henderson and Cockburn, 1994). To accurately identify the magnitude of in-house R&D investments by the global pharmaceuticals, we excluded in-process R&D spending¹², which is subsumed within R&D expense in the Compustat database. Meanwhile, the quadratic terms of internal R&D, *R&D expenditures squared*, are enclosed in the regression analysis as a test for decreasing returns to scale in R&D¹³ (Blundell *et al.*, 1995; Blundell *et al.*, 2002; Cohen and Klepper, 1996; Crépon and Duguet, 1997; Graves and Langowitz, 1993; Griffith *et al.*, 2004; Griliches, 1990; Guo and Trivedi, 2002; Hagedoorn and Duysters, 2002; Hall *et al.*, 1986; Hausman *et al.*, 1984; Lokshin *et al.*, 2008; Montalvo, 1997; Rothaermel and Hess, 2007).

¹² Acquisition-related in-process R&D charges are defined in Compustat as the portion of R&D considered to be 'purchased' and written off immediately upon acquisition if the R&D items are deemed not to have an alternative use.

¹³ Exclusion of the quadratic terms is nontrivial as in the presence of declining returns to scale in R&D one may expect firms to avoid this by combing internal and external R&D strategies (Lokshin *et al.*, 2008).

External R&D We include a dummy variable, rather than a count measure¹⁴, indicating whether or not a pharmaceutical firm pursued external R&D strategies. This dummy variable takes on the value of 1 if the company sourced R&D externally through either R&D alliances or R&D acquisitions (1 = R & D sourced externally through R&D alliances/R&D acquisitions), and 0 if the company solely undertook internal R&D. We identified R&D alliances through a detailed content analysis of each and every alliance the pharmaceutical firms entered since 1986 in biotechnology, 3,362 alliances in total, of which 1,205 alliances pertained to a clear R&D component. Likewise, we focused merely on R&D acquisitions (Ahuja and Katila, 2001; Higgins and Rodriguez, 2006), for which the business description in SDC Platinum suggested that the acquired biotechnology company was indeed targeted for its R&D capabilities. Through this process, 638 R&D acquisitions were found for the study period 1986–2000.

Control Variables

Presample Innovation In all models we attempted to capture the unobserved heterogeneity in firm innovation. As stressed by Blundell *et al.* (1995), the 'permanent' capabilities of companies to successfully commercialize new products and processes should be reflected in the pre-sample history of innovative output. Hence, we included both the *pre-sample average patent count* (i.e., the average number of biotech patents by the firm in the period from 1974^{15} to 1985) and a dummy (1 = if the firm had ever innovated prior to 1986)¹⁶ to proxy for the unobserved heterogeneity in the innovation models of incumbent pharmaceutical firms (Blundell *et al.*, 1995; Blundell *et al.*, 2002).

¹⁴ See Ahuja and Katila (2001), a simple count measure could possibly lead to measurement concerns. Instead of considering the scale of external R&D activities, we focus on whether firms use external R&D or not. The average of this dummy variable is 0.43.

¹⁵ Due to the emergence of the new biotechnology in the mid 1970s.

¹⁶ The dummy variable captures the fact that firms who sometimes innovate may be qualitatively different from those who never innovate (Blundell *et al.*, 1995).

Firm Merged The global pharmaceutical industry has witnessed a continuing trend of consolidation and concentration, especially in the late 1990s. To account for horizontal mergers and acquisitions among sample firms, as previously mentioned in the data section, we pursued a detailed family tree analysis by tracing all incumbent pharmaceutical firms back to their forerunners alive in 1986. Along with this analytical approach, we explicitly inserted a dummy variable indicating whether or not the sample firm was the outcome of a horizontal merger or acquisition over the study period 1986–2000 (1 = Firm Merged).

Pharmaceutical Firm The global pharmaceutical industry is made up of both specialized pharmaceutical firms and more diversified, mainly chemical, conglomerates. A firm's level of diversification indicates its previous experience in entering new businesses (Chang and Singh, 1999; Yip, 1982), which is likely to influence to what extent it attempts to innovate within the new biotechnology regime. Therefore, we controlled for a firm's degree of diversification by coding a firm as 1 if it is a specialized company (1 = Pharma Firm), and 0 otherwise. Specialized pharmaceutical companies are those firms that are active in SIC 2834 (Pharmaceutical Preparations Manufacturing), whereas a conglomerate might engage, for instance, in both SIC 2834 and SIC 2890 (Chemical Products Manufacturing). About one half of the firms are fully specialized (45%).

Firm Nationality To control for country-specific institutional configurations that are significant in shaping patenting propensities (Jaffe and Trajtenberg, 1999) as well as internal and external R&D strategies, we included two indicator variables based upon the location of company headquarters. One indicator variable is coded as 1 if the pharmaceutical firm is located in the United States (1 = U.S. *Firm*); the other indicator is coded as 1 if the pharmaceutical firm is headquartered in Europe (1 = European Firm), with a Japanese location as the reference category.

The global nature of our dataset is reflected in the fact that 36% of the firms are U.S. based, 28% are European based, with the remaining 36% headquartered in Japan.

Firm Size Previous work has examined how factors associated with firm size impact on a firm's incentive to invest in R&D (Cohen and Levinthal, 1989; Schumpeter, 1942; Teece, 1982, 1992). Moreover, firm size has been shown to exert a direct effect on innovative output (Acs and Audretsch, 1988; Cohen and Klepper, 1996; Freeman and Soete, 1997). Researchers also observe that small firms are more likely to restrict themselves to a simple innovation strategy, whereas large firms tend to combine various R&D sources (Beneito, 2006; Cassiman and Veugelers, 1999). We thus controlled for firm size by taking into account the total number of employees (*Employees*). The average number of employees of individual firms is 28,970, suggestive of their large size.

Firm Performance Previous research suggests that there is a trade-off between the size of a firm and its performance (Cubbin and Leech, 1986; Dobson and Gerrard, 1989; Reid, 1993, 1995). Companies with higher performance may as well be better positioned to finance their R&D investments. These influences were captured by including *Net Income* and *Total Revenues*¹⁷ in the regression analysis to allow for financial status of large pharmaceutical companies.

Year Fixed Effects In order to control for factors other than firm-specific characteristics that may influence all firms, such as economic-wide changes, we inserted *year dummies*, with 2000 being the reference year.

¹⁷ Total revenue is a measure of firm size as well and might account for firm size speedily increased through horizontal mergers and acquisitions. Besides, control for firm revenue is necessary for isolating the effect of R&D expenditures on innovative output (Rothaermel and Hess, 2007).

Estimation Technique

The dependent variable of this study, innovative output, as measured by biotech patent counts, is a count variable taking only non-negative integer values. To model count data the linear exponential or log-link family is a good alternative (Cameron and Trivedi, 1986). On account of the overdispersion phenomenon displayed by patent counts, namely the preponderance of zero and small values of firm patenting, the negative binomial estimation provides a better fit for the data than the more restrictive Poisson model (Hausman *et al.*, 1984).

The common first moment condition for the negative binomial model is,

$$\lambda_{it} = \mathbf{E}(\mathbf{Y}_{it}) = \mathbf{e}^{\mathbf{X}'_{it}\beta} \tag{1}$$

In our innovation model, we write as

$$\begin{aligned} X'_{it}\beta &= \theta_0 + \theta_1 R D_{I_{it-1}} + \theta_2 R D_{I_{it-1}}^2 + \theta_3 R D_{E_{it-1}} + \theta_{13} R D_{I_{it-1}} R D_{E_{it-1}} \\ &+ \theta_{23} R D_{I_{it-1}}^2 R D_{E_{it-1}} + \sum \gamma_n Control_{n_{it-1}} \end{aligned}$$
(2)

where λ_{it} is the expectation value of biotech patent counts for firm i in period t, RD_I and RD_I² are R&D expenditures and R&D expenditures squared, respectively, RD_E represents the dummy variable of external R&D through R&D alliances or R&D acquisitions, Control_n is a vector of control variables, and the parameters β , θ , γ are coefficients of various explanatory variables. A one-period lag is employed on all explanatory (key and control) variables to alleviate a potential simultaneity bias.

Differentiating equation (1) with respect to RD_I and dividing both sides of the resulting equation by λ_{it} yields,

$$\frac{\partial \lambda_{it} / \lambda_{it}}{\partial RD_{I_{it-1}}} = \theta_1 + 2\theta_2 RD_{I_{it-1}} + \theta_{13}RD_{E_{it-1}} + 2\theta_{23}RD_{I_{it-1}}RD_{E_{it-1}}$$
(3)

The expected marginal innovative output is herein given by the relative change¹⁸ in λ_{it} associated with a one unit change in RD_I. We then differentiate equation (3) with respect to RD_I a second time,

$$\frac{\partial \left(\frac{\partial \lambda_{it} / \lambda_{it}}{\partial R D_{I_{it-1}}}\right)}{\partial R D_{I_{it-1}}} = 2\theta_2 + 2\theta_{23} R D_{E_{it-1}} = \begin{cases} 2\theta_2 & \text{when } R D_{E_{it-1}} = 0\\ 2\theta_2 + 2\theta_{23} & \text{when } R D_{E_{it-1}} = 1 \end{cases}$$

$$\tag{4}$$

The above model explains the concavity of marginal returns to internal R&D, indicating whether there are decreasing returns (i.e., concave whenever $2\theta_2 < 0$ or $2\theta_2 + 2\theta_{23} < 0$) or increasing returns (i.e., convex whenever $2\theta_2 > 0$ or $2\theta_2 + 2\theta_{23} > 0$) to scale in internal R&D. Further, the existence of an interactive effect between RD₁ and RD_E is defined by requiring that the relative change in λ_{it} associated with a change in RD₁ depends on RD_E and vice versa (Winkelmann, 2008). Differencing equation (3) with respect to RD_E, given a dummy variable of RD_E, we obtain¹⁹,

$$\left\{\frac{\partial \lambda_{it} / \lambda_{it}}{\partial R D_{I_{it-1}}} \middle| R D_{E_{it-1}} = 1\right\} - \left\{\frac{\partial \lambda_{it} / \lambda_{it}}{\partial R D_{I_{it-1}}} \middle| R D_{E_{it-1}} = 0\right\} = \theta_{13} + 2\theta_{23} R D_{I_{it-1}}$$
(5)

Equation (5) clarifies the sense in which complementarity or substitutability between internal and external R&D strategies is contingent on the level of in-house R&D. RD_I and RD_E are considered (strict) complementary when the interactive effects are positive ($\theta_{13} + 2\theta_{23}RD_{I_{it-1}} > 0$), while RD_I and RD_E are considered (strict) substitutive when the interactive effects are negative ($\theta_{13} + 2\theta_{23}RD_{I_{it-1}} > 0$).

$$\begin{cases} \frac{\partial \lambda_{it}}{\partial RD_{I_{it-1}}} \Big| RD_{E_{it-1}} = 1 \Big\} - \left\{ \frac{\partial \lambda_{it}}{\partial RD_{I_{it-1}}} \Big| RD_{E_{it-1}} = 0 \right\} = \\ \left\{ \left(\theta_1 + 2\theta_2 RD_{I_{it-1}} + \theta_{13} + 2\theta_{23} RD_{I_{it-1}} \right) e^{\left(\theta_1 RD_{I_{it-1}} + \theta_2 RD_{I_{it-1}}^2 + \theta_3 + \theta_{13} RD_{I_{it-1}} + \theta_{23} RD_{I_{it-1}}^2 + \sum \gamma_n Control_{n_{it-1}} \right)} \right\} - \\ \left\{ \left(\theta_1 + 2\theta_2 RD_{I_{it-1}} \right) e^{\left(\theta_1 RD_{I_{it-1}} + \theta_2 RD_{I_{it-1}}^2 + \sum \gamma_n Control_{n_{it-1}} \right)} \right\} \right\}$$

¹⁸ See the interpretation of parameters in the Poisson regression by Winkelmann (2008, p.70).

¹⁹ Mullahy (1999) discusses the difficulties that arise if interactive effects in nonlinear models are defined in terms of absolute, rather than relative, changes. In our model the interactive effects under absolute change would be otherwise as complicated as follows,

In our count panel data model, either fixed or random effects specification can, in theory, be used to control for firm-specific unobserved heterogeneity (Greene, 2003). One difficulty with the random effects specification is its underlying assumption that time invariant unobserved heterogeneity is uncorrelated with regressors of interest. In other words, this specification rules out the existence of time invariant unobserved factors that influence both a firm's R&D strategies and its innovative output, which is untenable since managers make their 'strategic' organizational choices with certain performance expectations in mind (Hamilton and Nickerson, 2003; Leiblein *et al.*, 2002). Hence, we used the fixed-effects specification so as to estimate the model parameters consistently (Hamilton and Nickerson, 2003; Winkelmann, 2008). Additionally, following Blundell *et al.* (1995, 2002), we allowed for time invariant unobserved heterogeneity by explicitly taking into account the pre-sample history of firm innovative output.

RESULTS

Table 1 shows descriptive statistics and a bivariate correlation matrix. The table indicates a high degree of variance on most of the variables such as *Biotech Patents*, *R&D Expenditures*, *Employees*, and *Presample Mean*. The bivariate correlations are, with the exception of R&D expenditures and employees²⁰, low and thereby indicate sound validity. Moreover, variance inflation factors (VIFs) were computed to assess the severity of multicollinearity. The average VIF value is 2.19, with the maximum VIF value of 5.29, which are well below the cut-off point

²⁰ As mentioned previously, factors associated with firm size tend to influence a firm's incentive to invest in R&D (Cohen and Levin, 1989; Schumpeter, 1942; Teece, 1982, 1992). In addition, employees and total revenue, as close measures of firm size, are also highly correlated in our dataset (r = 0.80).

of 10 (Cohen *et. al.*, 2003), implying that multicollinearity does not pose a problem for our estimation models.²¹

Insert Table 1 about here

Table 2 provides results for all models using fixed-effects negative binomial estimation. Model 1 is the baseline specification including the control variables only. Model 2 estimates R&D expenditures and the quadratic terms thereof. Model 3 presents the full model with the interactive effects between internal and external R&D. Each subsequent model demonstrates a significant (p < 0.001) improvement over the baseline specification. We use the full Model 3 to discuss the regression results of the hypothesis tests. Overall, the estimated coefficients suggest a curvilinear relationship between R&D expenditures and biotech patenting. The estimated second derivative, $2\theta_2$ when RD_E = 0 given by equation (4), is negative and statistically significant (p < 0.001). By comparison, when $RD_E = 1$, the estimated second derivative, $2\theta_2 + 2\theta_{23}$ given by equation (4), becomes less negative (and significant at p < 0.10), implying that marginal returns to internal R&D decrease less in the presence of external R&D. Taken together, these results provide strong support for hypothesis 1 that there are decreasing returns to scale in internal R&D. Firm innovative output initially increases with investment in internal R&D, this rate of increase diminishes at higher levels of internal R&D spending though. This finding is consistent with previous studies clearly suggesting diseconomies of scale in internal R&D (Graves and

²¹ As an additional test, we centered the independent variables before creating their squares and cross products (Cohen, 2003). The estimated coefficients turned out to show a very similar empirical pattern. Besides, even if multicollinearity does not bias coefficient estimates, it may affect the stability of the estimated coefficients, thereby omitting even a few observations can change the sign or the significance of the affected variables (Greene, 2003). To ensure the robustness of our results, we performed a sensitivity analysis by drawing random samples of 90% of the total observations and estimating the full model for each of these random samples (Ahuja and Katila, 2001). The corresponding results are similar to the results reported below.

Langowitz, 1993; Hagedoorn and Duysters, 2002; Lokshin *et al.*, 2008; Rothaermel and Hess, 2007).

Insert Table 2 about here

In hypothesis 2, we postulated that internal and external R&D strategies may reinforce or substitute for each other in predicting firms' innovative output. We tested the interactive effect between internal and external R&D by examining the sign and statistical significance of the values of the cross-partial derivative, $\theta_{13} + 2\theta_{23}RD_{I_{it-1}}$ given by equation (5). To this purpose, a graphical analysis, as exhibited in Figure 1, was conducted to manifest the nature and significance of the interactive effect by plotting its value and the implied z-statistic value over the range of internal R&D investments by incumbent pharmaceutical firms (Ai and Norton, 2003; Hoetker, 2007; Wiersema and Bowen, 2009). In Figure 1, the bullet symbols (•) depict values of the interactive effect between internal and external R&D, recorded on the left axis, while the diamond shaped symbols (\diamond) depict z-statistic values, recorded on the right axis. As seen in Figure 1, the sign, magnitude, and statistical significance of the interactive effect varies with the level of in-house R&D. The interactive effect is negative and significant (p < 0.10) when internal R&D is less than 886.14, whereas the effect is positive and significant (p < 0.10) when internal R&D is in the range from about 1364.34 to 2689.31, with the interactive effect not statistically significant in between. These findings highlight the complexity of understanding the relationship between internal and external R&D strategies in shaping firms' innovative performance, and clear-cut results, either complementarity (hypothesis 2-a) or substitutability (hypothesis 2-b), are not always to be expected. In contrast to conventional wisdom, our overall findings suggest that a

complete picture is to be found in the contingency of the relationship between internal and external R&D strategies, where complementarity or substitutability is contingent on the level of in-house R&D.

Insert Figure 1 about here

Turning to the effects of control variables, we found largely consistent results across various models. The pre-sample patent variables, i.e., pre-sample mean patent count and the dummy, are both positive and statistically significant, indicating that it is important to control for the unobserved differences in the innovative capabilities with which firms entered our sample. Employees, as a measure of firm size, are positively associated with patenting frequency (p < 0.05). The implied gains from large horizontal merger or acquisition, however, have not been identified. Incumbent pharmaceutical companies that speedily expand through horizontal mergers and acquisitions appear to degrade their innovative output significant(p < 0.001) over the study period. The estimated coefficient of firm revenues is significant (p < 0.001) but, contrary to expectations, in a negative direction. With regard to the year fixed effects (included but not shown), the year dummies for 1993 – 1999 are positive and significant, while all the other calendar year indicators are not statistically significantly accelerated in the later sample period (1993 – 1999), in contrast with its earlier years (1986 – 1992).

Robustness of Results

To ensure the robustness of our results presented above, we also ran models including a measure of firm knowledge stock²², effectively a lagged dependent variable, to partially control for firm-specific unobserved heterogeneity that is not fixed throughout time (Blundell *et al.*, 1995; Blundell *et al.*, 2002; Dushnitsky and Lenox, 2005). Since the lagged dependent variable is predetermined, the conditional maximum likelihood estimator for count panel data models is inconsistent (Blundell *et al.*, 1995; Blundell *et al.*, 2002; Crépon and Duguet, 1997; Montalvo, 1997; Wooldridge, 1997). Following Blundell *et al.* (1995, 2002), we used the pre-sample mean (PSM) estimator²³, which is a good alternative to standard estimators when there are predetermined regressors. The results under PSM estimation are reported in the first and third columns of Table 3. These estimated results set a very similar pattern as what we observed in Table 2. The additional message is that pharmaceutical firms with higher knowledge stock exhibit a significantly greater number of biotechnology patents (p < 0.001). The strong effects of presample dummy and firm merged, though, are driven into insignificance in this dynamic analysis.

Furthermore, as our study focuses on a dummy, rather than a count, variable of external R&D, we disaggregate external R&D into R&D alliances and R&D acquisitions so as to capture the scale of different types of external R&D investments. The corresponding results are shown in the last two columns of Table 3. Both estimation methods — fixed-effects negative binomial and PSM — produce fairly close findings to those presented before. The robust empirical patterns overall suggest that the complementarity or substitutability between internal R&D and R&D

²² See Blundell *et al.* (1995), knowledge stock (K_{it}) is the depreciated sum of past innovations and defined as,

 $K_{it} = Y_{it} + (1-\delta) K_{it-1}$ where the depreciation rate δ is taken to be 30%, Y_{it} is biotech patent in our models. ²³ Another alternative in the case of predetermined regressors is the quasi-differenced GMM estimator, which can however be severely biased in small samples, particularly when regressors are highly persistent and the instruments are therefore weak predictors of the endogenous variables in the differenced model (Blundell *et al.*, 2002).

alliances, or between internal R&D and R&D acquisitions, is contingent on the level of in-house R&D investments. The only noteworthy difference between these two methods of estimation, fixed-effects negative binomial and PSM, is that R&D acquisitions exhibit more significant interactive effects with internal R&D under PSM estimation. In sum, these results corroborate our findings from the more parsimonious models of Table 2.

Insert Table 3 about here

DISCUSSION AND CONCLUSION

A substantial body of literature has examined the relationship between internal R&D and external technology sourcing through external R&D, so far focusing on either complementarity or substitutability. Yet there is still considerable confusion over the conditions under which there may in fact be complementarity or substitutability between different R&D strategies. Following Cassiman and Veugelers (2006), success in innovation will depend not only on combing various innovation activities, but also on creating the right context.²⁴ The potential benefits from complementarity are therefore context-specific or contingent. One implication of our analysis is that the level of in-house R&D investments, which is characterized by decreasing marginal returns, is a contingency variable that critically influences the nature of the link between internal and external R&D strategies. In particular, internal R&D and external R&D, through R&D alliances or R&D acquisitions, are complementary innovation activities at higher levels of in-house R&D investments, whereas at lower levels of in-house R&D efforts internal and external R&D and external and external R&D and external R&D

²⁴ In their study, Cassiman and Veugelers (2006) identify reliance on more basic R&D, i.e., the use of universities and research centers as information sources for the innovation process, as an important context variable that affects the strength of the complementarity between innovation activities.

In line with the two roles, or faces, of in-house R&D²⁵ (Cohen and Levinthal, 1989, 1990), a minimum level of absorptive capacity, derived from in-house R&D activities, is required for effectively incorporating external R&D (Catozzella and Vivarelli, 2007; Lokshin et al., 2008; Tsai and Wang, 2008). Higher levels of internal R&D investments, as a consequence, may enable incumbent firms to maintain their ability to react adequately to technological changes by leveraging external networks. As Arora and Gambardella (1990) demonstrate in the context of pharma-biotechnology, large pharmaceutical firms with higher internal research activities are more active in using external knowledge sources. In this light, sufficient investments in in-house R&D contribute substantially to a spiral of success and thereby sustainable innovative capabilities. By contrast, at lower levels of in-house R&D investments, our findings point to the idea that the simultaneous pursuit of internal R&D and external R&D sourcing strategies would actually decrease a firm's innovative output, at least at the margin. Part of the explanation may lie in the fairly constrained resource set of firms, especially in terms of their absorptive capacity, as a firm may fail to reap economies of scope when ineffectively scanning, selecting, and assimilating external technological knowledge. Alternatively, incumbent firms with low research capabilities tend to exhibit overreliance on external R&D alliances or acquisitions for promising technologies in the face of radical technological change. This would explain, to a certain extent, why some established pharmaceutical firms are obtaining or choose to obtain their most technologically advanced projects from alliance partners or through the market for know-how.

In attempting to characterize the contingency of the relationship between internal and external R&D strategies in shaping firms' innovative output, a number of econometric problems arising from the count panel nature of the data have to be considered. In essence, we adopted an

²⁵ As Cohen and Levinthal (1989, 1990) point out, the two roles, or faces, of internal R&D are reflected in directly generating new knowledge and indirectly contributing to absorptive capacity.

estimation approach that places the simultaneous interactions between internal R&D and two alternative modes of external R&D investments, R&D alliances and R&D acquisitions²⁶, at the core of our analysis. It is readily apparent that firms do not randomly choose a specific way to organize R&D, either rely exclusively on in-house R&D or systematically combine with R&D alliances and/or R&D acquisitions. The effects that drive firms to choose ex ante a specific R&D strategy towards superior performance outcomes²⁷, when not corrected for, would introduce a serious endogeneity bias²⁸ (Hamilton and Nickerson, 2003; Leiblein *et al.*, 2002). Possible remedies are available, nevertheless, if endogeneity is caused by time invariant correlated unobserved heterogeneity, for which one can estimate the model parameters consistently by fixed effects panel data methods (Winkelmann, 2008). Accordingly, we employed fixed effects specification in our panel count data models. In addition, to partially control for firm-specific unobserved heterogeneity that is not fixed throughout time (Blundell *et al.*, 1995; Blundell *et al.*, 2002; Dushnitsky and Lenox, 2005), a dynamic analysis was performed by taking into account the measure of firm knowledge stock.

So far the inconsistent literature on the complementarity or substitutability between R&D strategies, as emphasized by Watkins and Paff (2009), leaves considerable room for improving the consistency in defining and measuring complementarity, particularly at the boundaries

²⁶ In some cases large pharmaceutical firms also hope to establish a 'preferential link' with the new biotechnology venture by acquiring part of its capital stocks. Such acquisition of minority stocks would likely translate into a formal alliance agreement once a specific new discovery needs to be implemented and possibly commercialized (Arora and Gambardella, 1990).

²⁷ Understanding how firms choose ex ante a specific way to organize R&D is beyond the scope of our study.

²⁸ Another concern is the sample selection bias. A varied set of sample selection models are available in econometric literature to deal with such self-selection bias — the foremost model being Heckman two-stage approach. However, while Heckman and similar approaches are most commonly used within the domain of continuous-response models, they are not appropriate for count response models (Cameron and Trivedi, 1998; Hilbe, 2007). The development of methodology in count panel data models to correct for sample selection bias is unfortunately still an open task (Winkelmann, 2008).

between disciplinary traditions²⁹. The cloud of confusion over what is and what conditions complementarity thus suggests further research work to disentangle the rationales for combining different R&D sourcing arrangements. Moreover, there appears to be increasing variation among the largest pharmaceutical firms in terms of their strategies to extract value from new technologies, each trying to achieve a preferred balance between in-house R&D and externally sourced R&D. It may consequently no longer be useful to talk about Big Pharma as a homogenous sector (Mittra, 2007). An interesting avenue for future work would be to investigate these issues, for instance, through a fine-grained analysis of firm's optimal balance between internal and external R&D strategies at various stages of innovation process or towards different types of innovation. We believe that a fuller appreciation of the origins of innovativeness is to be found in a better understanding of the alignment between R&D strategies and the internal and external environment of firms, be it in either a high-technology or a low-technology industry.

²⁹ In the standard economic literature, partial substitution elasticity among inputs theoretically measures the relative change in the ratio of inputs when their relative prices change. By contrast, a very different measure is recently used in the management literature, comparing changes in average output ratios under different combinations of inputs. For the latter measure input prices play no role, nor do margins (Watkins and Paff, 2009).

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	Variable	Mean	S. D.	Min	Max	1	2	3	4	5	6	7	8	9	10	11	12
1	Biotech Patents	7.60	14.42	0.00	207.00	1.00											
2	R&D Expenditures (MM\$) _{t-1} *	492.17	557.26	0.30	2869.31	0.38	1.00										
3	R&D External t-1	0.43	0.50	0.00	1.00	0.27	0.33	1.00									
4	Firm Merged t-1	0.07	0.26	0.00	1.00	0.31	0.34	0.17	1.00								
5	European Firm t-1	0.28	0.45	0.00	1.00	0.09	0.21	0.01	0.14	1.00							
6	US Firm t-1	0.36	0.48	0.00	1.00	0.09	0.19	0.14	-0.02	-0.46	1.00						
7	Pharma Firm t-1	0.45	0.50	0.00	1.00	0.19	-0.06	0.21	0.22	-0.08	-0.06	1.00					
8	Employees (1,000) t-1	28.97	32.99	0.32	173.00	0.18	0.77	0.14	0.15	0.28	0.25	-0.31	1.00				
9	Net Income (MM\$) *	595.69	963.44	-6680.33	7948.49	0.28	0.69	0.23	0.16	0.05	0.33	-0.09	0.57	1.00			
10	Revenues (MM\$) _{t-1} *	9377.54	10729.70	15.83	53885.29	0.07	0.61	0.03	0.03	0.21	0.21	-0.42	0.80	0.57	1.00		
11	Presample Mean _{t-1}	3.64	5.73	0.00	27.38	0.56	0.40	0.29	0.45	-0.08	0.21	0.28	0.15	0.34	0.05	1.00	
12	Presample Dummy _{t-1}	0.91	0.29	0.00	1.00	0.04	-0.17	-0.12	-0.07	-0.28	0.09	-0.04	-0.13	-0.06	-0.01	0.20	1.00

 Table 1 Descriptive Statistics and Bivariate Correlation Matrix

Notes: N = 1,139 firm-year observations. *In constant, inflation-adjusted year 2000 US \$.

Variable	Model 1	Model 2	Model 3
Constant	0.1134	-0.0927	-0.2953
	(0.3374)	(0.3473)	(0.3545)
Year Fixed Effects	Included	Included	Included
Firm Merged t-1	-0.9764 ^{***} (0.2746)	-1.1353 ^{***} (0.2669)	-1.1584 ^{***} (0.2650)
European Firm t-1	-1.3036***	-1.2720****	-1.2603****
US Firm t-1	(0.2726) -0.8015 ^{**} (0.2764)	(0.2803) -0.9207** (0.2854)	(0.2828) -0.9156 ^{**} (0.2865)
Pharma Firm t-1	0.0839	-0.0305	0.0153
Employees t-1	(0.2270) 0.0166 ^{***}	(0.2232) 0.0076 [*]	(0.2236) 0.0079 [*]
Net Income _{t-1}	(0.0034) 1.49E-05 (4.05E-05)	(0.0039) -1.32E-05 (3.90E-05)	(0.0038) -1.40E-05 (3.93E-05)
Revenues t-1	-3.91E-05** (1.25E_05)	-4.77E-05	-4.93E-05
Presample Mean _{t-1}	(1.23E-05) 0.0528^{**} (0.0161)	(1.34E-03) 0.0291^{\dagger} (0.0171)	(1.32 ± -0.5) 0.0321^{\dagger} (0.0176)
Presample Dummy t-1	1.3161***	1.3288***	1.3900 ^{***}
R&D Expenditures t-1	(0.2755)	(0.2)20) 1.70E-03*** (3.23E 04)	2.30E-03*** (2.81E-04)
R&D Expenditures squared t-1		(3.33E-04) -4.07E-07***	(3.81E-04) -6.99E-07***
R&D External t-1		(1.14E-07)	(1.49E-07) 0.2555*
R&D Expenditures*R&D External t-1			(0.0993) -9.36E-04**
R&D Expenditures Squared* R&D External t-1			(2.82E-04) 4.36E-07 ^{**} (1.41E-07)
Ν	1125	1125	1125
Log Likelihood	-2273.8789	-2258.3008	-2252.8549
Chi Square	286.36***	337.51***	351.57***

Table 2 Fixed-Effects Negative Binomial Regression Predicting Biotech Patenting

Notes: Standard errors are in parentheses; year dummies are included but not shown. The fixed-effects specification eliminates firms who never patent, thereby reducing the effective sample to 1125 firm-year observations. Two tailed-tests significant at:

† * p<0.10

p<0.05

** p<0.01

*** p<0.001



Figure 1 Interactive Effects between Internal and External R&D Under Relative Change

Notes: z-statistic value (-1.645, 1.645), 10% two-tailed test. The solid symbols depict values of the interaction effect between internal and external R&D, recorded on the left axis, while the diamond shaped symbols depict z-statistic values, recorded on the right axis. The values of the interaction effect, as given by equation (5), range from -0.0009 to 0.0016, with a mean value of -0.0005; the z-statistic values range from -3.3142 to 2.8260, with a mean value of -2.3453.

	External R&D (dummy)	<u>External R&D (count)</u>					
Variable	PSM	Negative Binomial (FE)	PSM				
Constant	0.2136	-0.1925	0.1899				
	(0.4545)	(0.3504)	(0.4184)				
Year Fixed Effects	Included	Included	Included				
Firm Merged t-1	-0.0527	-1.1806***	-0.0431				
	(0.1908)	(0.2712)	(0.1693)				
European Firm t-1	0.0996	-1.2490***	0.0370				
	(0.247)	(0.2752)	(0.2322)				
US Firm t-1	-0.1470	-0.9321**	-0.2045				
	(0.2339)	(0.2807)	(0.2249)				
Pharma Firm _{t-1}	-0.0883	-0.0282	-0.1152				
	(0.1932)	(0.2258)	(0.1870)				
Employees t-1	0.0086^{*}	0.0100^{*}	0.0087^{*}				
	(0.0040)	(0.0040)	(0.0042)				
Net Income t-1	-3.20E-06	-8.15E-06	3.73E-05				
	(6.82E-05)	(3.88E-05)	(5.84E-05)				
Revenues t-1	-3.61E-05**	-5.08E-05***	-3.66E-05**				
	(1.28E-05)	(1.33E-05)	(1.32E-05)				
Presample Mean t-1	0.0393**	0.0394*	0.0469**				
	(0.0137)	(0.0178)	(0.0147)				
Presample Dummy t-1	-0.0623	1.2789***	0.0276				
	(0.2749)	(0.2935)	(0.2806)				
Knowledge Stock _{t-1}	0.0098^{***}		0.0094***				
	(0.0017)		(0.0017)				
R&D Expenditures t-1	2.12E-03***	1.93E-03***	1.97E-03***				
	(6.31E-04)	(3.58E-04)	(5.32E-04)				
R&D Expenditures squared t-1	-9.52E-07***	-5.57E-07***	-8.60E-07***				
	(2.56E-07)	(1.25E-07)	(2.07E-07)				
R&D External t-1	0.6121**						
	(0.2191)						
R&D Expenditures*	-1.22E-03*						
R&D External t-1	(5.64E-04)						
R&D Expenditures Squared*	6.28E-07*						
R&D External t-1	(2.67E-07)	**	***				
R&D Alliance _{t-1}		0.1120	0.1964				
		(0.0390)	(0.0561)				
R&D Expenditures*		-2.05E-04	-3.02E-04				
R&D Alliance _{t-1}		(6.15E-05)	(1.17E-04)				
R&D Expenditures Squared*		9.11E-08	1.27E-07				
R&D Alliance _{t-1}		(2.35E-08)	(4.68E-08)				
R&D Acquisition _{t-1}		0.1076	0.3139				
		(0.0736)	(0.0995)				
R&D Expenditures*		-2.04E-04	-5.56E-04				
R&D Acquisition $_{t-1}$		(1.19E-04)	(1.64E-04)				
R&D Expenditures Squared*		7.88E-08	2.03E-07				
R&D Acquisition _{t-1}	1120	(4.74E-08)	(6.73E-08)				
	1139	1125	1139				
Log Likelihood		-2247.5552					
Chi Square		360.39					

Table 3 Robustness Test

Notes: Standard errors are in parentheses; year dummies are included but not shown. The fixed-effects (FE) specification eliminates firms who never patent, thereby reducing the effective sample to 1125 firm-year observations. Two tailed-tests significant at: $\dagger p < 0.10$, $\star p < 0.05$, $\star p < 0.01$, $\star \star p < 0.001$.

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