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Public support intensity and company R&D performance: evidence from a dose-response model

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Abstract

This paper presents an original econometric model for estimating a dose-response function through a regression approach when: (i) treatment is continuous, (ii) individuals may react heterogeneously to observable confounders, and (iii) selection-into-treatment may be potentially endogenous. After describing the model, two estimation procedures are suggested: one based on OLS under Conditional Mean Independence (or CMI), and one based on Instrumental-Variables (IV) under selection endogeneity. The paper goes on by presenting `ctreatreg`, an author-written Stata routine for an easy implementation of such a model, thereby performing a Monte Carlo experiment to test the reliability of the model and of its software implementation. Finally, an application to real data for assessing the effect of public R&D support on companies' R&D expenditure is presented and results briefly commented. The usefulness of such a model for program evaluation is clearly stressed.

Keywords: treatment effects, dose-response function, continuous treatment, Monte Carlo, R&D support

JEL classification: C21, C87, D04

S_E1: Counterfactual methods for regional policy evaluation

1. Introduction

In many socio-economic contexts, policy interventions take the form of a continuous exposure to a certain type of treatment. For instance, individuals getting a grant to set-up a new business, or to escape some poverty threshold are typical examples in which the amount of support can vary by individual, thereby providing ground for a different response to policy.

In public policies to support business R&D – the topic of this paper – companies are not only selected for treatment, but also awarded a different amount of support. Thus, from a program evaluation perspective, what is relevant is not only the binary treatment status, but also the level of R&D support exposure (or “dose”) provided by the public agency. This is in tune with the language of epidemiology, where dose-response functions are usually estimated in order to check patients’ resilience to different levels of drug administration (Robertson et al., 1994; Royston and Sauerbrei, 2008).

Moreover, usually firms enjoy more than one public financial support and it is relevant to understand if the curve dose-response is increasing or not and if and when there is a turn-around. The risk from a welfare point of view is a resource waste, i.e. allocation of funds which produce a substitution between public and private funds for some level of subsidy. The continuous treatment frame allows to investigate the form of the function of average treatment effects over all possible values of the treatment levels and to study whether effects change when the level of treatment changes. Although based on different econometric approaches (Adorno et al., 2007; Marino et al., 2011), recent literature on public program evaluation using a dose-response frame has found out a convergent result of decreasing relation between the level of subsidy and the outcome (capital or R&D capital), as well as a notable substitution between private and public funds occurring for high intensity of public subsidy. However, such empirical evidence has not found yet a sound theoretical explanation of these results, and this study aims at filling this gap.

In this paper we use an econometric application based on the continuous treatment model proposed by Cerulli (2014) and implemented in Stata 13 via the user-written command `ctreatreg`. As background analytical reference, we rely on the literature on adjustment costs explaining the relation between investments and cash-flows. More precisely, we assume that public subsidy may be considered as an additional injection of cash-flow for treated firms, which may face a low sensitivity of R&D investments due to frictions, such as capital adjustment costs, as resulting from the literature on investment decision. As reference we take also into consideration some studies on R&D program evaluation and in particular those studying public subsidies’ efficiency in reducing firms’ financial constraints.

The dose-response approach employed in this study is suited when: (i) treatment is continuous, (ii) individuals may react heterogeneously to observable confounders, and (iii) selection-into-treatment may be potentially endogenous.

To fix ideas, consider a policy program where the treatment is assigned not randomly (i.e., according to some “structural” rule), and where – after setting who is treated and who is not – the program provides a different “level” or “exposure” to treatment ranging from 0 (no treatment) to 100 (maximum treatment level). Two groups of units are thus formed: (i) *untreated*, whose level of treatment (or dose) is zero, and (ii) *treated*, whose level of treatment is greater than zero.

We are interested in estimating the causal effect of the treatment variable t on an outcome y within the observed sample, by assuming that treated and untreated units may respond differently both to specific observable confounders and to the “intensity” of the treatment t . In this context, the dose-response function is shown to be equal to the “Average Treatment Effect, given the level of treatment t ” (i.e. $ATE(t)$). But also other causal parameters of interest, such as the unconditional Average Treatment Effect (ATE), the Average Treatment Effect on Treated (ATET), the Average Treatment Effect on Non-Treated (ATENT) are estimated, along with these effects conditional on the vector $(\mathbf{x}; t)$.

Compared with similar models - and in particular the one proposed by Hirano and Imbens (2004) implemented in Stata by Bia and Mattei (2008)¹ - this model does not need a full normality assumption and it is well-suited when many individuals have a zero-level of treatment. Additionally, it may account for treatment “endogeneity” by exploiting an Instrumental-Variables (IV) estimation in a continuous treatment context. In this paper we present only the case of control function approach to handling observable endogeneity, and not endogeneity due to unobservable factors.

When many units are not exposed to treatment, the distribution of t has a “spike” or no-nil probability mass at zero, i.e. $\Pr(t=0)>0$. This means that assuming that the distribution of $t|\mathbf{x}$ comes from a normal (or mixtures of normal) distribution, as assumed in the Generalized Propensity Score proposed by Hirano and Imbens, is untenable, as in the presence of a spike at zero this distribution is clearly discontinuous and thus non normal.

Within the epidemiological literature, Royston et al. (2010) have proposed a dose-response model for continuous exposures with a spike at zero based on fractional polynomials. For fractional polynomials functions of t to be defined at $t=0$, the authors shift the origin of t by adding a small constant, c , before analysis. They take c as the smallest difference between successive observed

¹ See also Bia, Flores and Mattei (2011) generalizing the Hirano-Imbens (2004) model by allowing for a nonparametric estimation of the Dose-Response Function.

positive values of t , although other choices are suggested. The authors propose a model for the response variable y having a jump in zero with y equal to a constant β in $t=0$ and to a fractional polynomial of $t+c$ in $t>0$. They estimate this model in a single regression by adding an indicator variable z (taking value one if $t=0$ and zero if $t>0$) as additional predictor. In this way they are able to estimate the response at $t=0$ (i.e., recovering a consistent estimation of β) by exploiting a standard regression model.

Differently from Royston et al.(2010), our model is embedded into the potential outcome setting, the typical framework of counterfactual modeling, and it solves the zero-inflation problem by directly modeling the potential outcome in $t=0$. In this way, we avoid ad hoc assumptions, as that for choosing a reliable constant c .

Differently from Hirano and Imbens, we do not need to specify a generalized propensity score, as we work within a control-function model. Moreover, we are able to take into account both zero-inflation at $t=0$ and treatment observable endogeneity under reasonable assumptions.

This model is applied to assess the effect of public R&D support (measured as a share of total company R&D expenditure) on business R&D outlay. Via our model, we are able to look at the pattern of the policy effect over treatment intensity, thus going beyond the typical “average effect” analysis. This suitably allows us for a better inspection into the causal relation between the policy instrument and the policy target. Nevertheless, given the unavailability of a reliable instrumental variable in our dataset, our application concerns only the case running under conditionally exogenous treatment (or CMI). Future works endowed by better data and information may solve this limitation by tackling an endogenous treatment as suggested in our approach.

The paper is organized as follows: section 2 presents the rationale of this study in the context of existing literature; section 3 sets out the model and the related estimation techniques; section 4 presents an econometric assessment of the impact of R&D public support intensity on company R&D performance; section 5, finally, concludes the paper.

2. This study in the context of existing literature

“From a welfare perspective too little R&D may be realized in (competitive) markets due to positive externalities and information asymmetries in lending and investing relationships. Both types of market failures are usually regarded as justification for government intervention” (Czarnitzki and Hottenrott, 2010). We chose the financial constraints as main reference for studying the effect of subsidy level on firms’ R&D expenditure response. Investments in R&D require financial resources and are characterized by high and firm specific investment costs, with

low collateral value. This means that establishing an R&D project involves significant sunk costs, i.e. an expense which cannot be capitalized in the balance sheet. Information asymmetries between investors and managers additionally create uncertainty that affects financing conditions. The definition of financial constraints in our frame is the inability of a firm to raise the necessary funds to finance its (target or desired) R&D investments.

In principle, there are two sources for financing innovation projects: external sources, such as bank loans or other debt contracts, and internal sources, that is retained profits or (new) equity. Firms decide about their optimal levels of investments while choosing a capital structure in such a way as to minimize the long run cost of capital.

If capital markets are imperfect and information asymmetries influence lending and investment decisions, the cost of different kinds of capital may vary by type of investment. Hall (1992), Himmelberg and Petersen (1994) and Czarnitzki and Hottenrott (2009) show that internal sources of funds are indeed more important (accessible and less costly) for R&D than for ordinary investment. Because the knowledge asset created by R&D investment is intangible, partly embedded in human capital, and ordinarily very specialized to the particular firm in which it resides, the capital structure of R&D-intensive firms exhibits considerably less leverage than that of other firms (Hall and Lerner, 2010).

Moreover, besides moral hazard problems between the management of the firm and external investors or lenders, also information asymmetries between management and owners may impact investment in innovation projects and financing conditions (Jensen and Meckling 1976, Grossman and Hart 1982, Czarnitzki and Kraft 2004). In particular (Jensen and Meckling 1976) an entrepreneur or manager in a firm which has a mixed financial structure (containing both debt and outside equity claims) will choose a set of activities for the firm such that the total value of the firm is less than it would be if the entrepreneur were the sole owner.

All these are sources of financing constraints, which can restrict the investment behaviors of firms and, more interestingly for the scope of our work, may influence R&D investments in presence of public financial subsidies, if: (i) there is a high firm's cost in participating to the public policy; (ii) the subsidy efficiency in reducing the firm's financial constraints is low; (iii) the firm may become financial constrained as consequence of the public intervention, for instance when selected as financially unconstrained but with good unexploited innovation potentiality.

When studying the effect of public subsidy level on liquidity constraints many factors should be taken into consideration, both for a more complete "coeteris paribus" condition and for a more transparent possibility of result interpretation, such as intra-firm organizational structures and other institutional factors that may increase the risk of firms to face financing constraints.

The (still small) present evaluation literature adopting a dose–response approach converges in finding a decreasing relation between funding level and firms’ R&D expenditures. Unfortunately there is also a lack of explication of these relation, which we look for by referring to (i) the capital adjustment cost models of investment (Eisner and Strotz, 1963; Lucas, 1967; Gould, 1968; Treadway, 1971; Hall, 2004; Caballero and Engel, 1999; Abel and Eberly, 1998) and the investment-cash-flow sensitivity models (Kaplan and Zingales, 1997; Alt, 2003; Coad, 2010; for a survey Carreira and Silva, 2010); (ii) the R&D investment equation, based on the demand for R&D investment and supply of funding heuristic (Hall and Lerner, 2010).

The adjustment cost models of investments, in the two different versions of convex and non convex adjustment costs, have given a theoretical explication to the empirical evidence of (i) an only partial adjustment of capital (or of other factors) towards the chosen/desired capital levels or (ii) the infrequent, lumpy changes in investment behavior. In the first case (convex adjustment costs) the firms, beyond other costs associated with the acquisition of new capital, incurred output costs due to the act of adjusting the capital stock. These costs would be increasing with the distance between the chosen new level and the current level of capital, thereby implying a smoothly rising marginal adjustment cost. In the second case (non convex adjustment costs) they are fixed costs the firm incurs at any time it adjusts its capital stock. Firms with the same current gap between actual and target (R&D) capital do not necessarily behave identically, since those drawing low cost are more likely to alter their capital than those who draw high cost of investment. This can be due also to the (R&D) capital heterogeneity. This literature in its recent development takes also in consideration different forms of firms expectations within a dynamic frame. For the scope of this paper is anyway sufficient to take into account that the sensitivity of firms’ investment to an addition of cash-flow (through public subsidy) could be low, if the available liquidity doesn’t cover all (input and output) costs of (R&D) investment. Even in presence of liquidity constrained firms, “investment cannot increase with marginal increase in cash-flow for some firms”. Cash-flow should be above a certain threshold (Pratap, 2001, p. 3). But also, the higher is the distance between the present level of (R&D) capital and the target one and the higher are the adjustment costs, therefore it is possible to find empirically that firms which are highly liquidity constrained, show little sensitivity of investment to cash-flow.

Referring now to the R&D investment equation (David et al., 2000; Hall and Lerner, 2010) this approach allows putting attention to the marginal costs of financial resources for adjusting the level of the R&D projects. Internal funds are available at a constant cost of capital until they are exhausted, at which point it becomes necessary to issue debt or equity in order to finance more investment. When the demand curve cuts the supply curve in the horizontal portion, a shock that

increases cash-flow (and shifts supply outward) has no effect on the level of investment. It could mean that the public agency has selected unconstrained firms, which have reached their desired level of R&D capital. This case could show an investment sensitivity to the new liquidity represented by public subsidy if the selected firms had some unexploited investment opportunities or high innovating potential capacity and the adjustment cost is not so high to bring a new liquidity constraint.

In sum, without liquidity constraints, investment would be insensitive to additional cash-flow; with liquidity constraints, but in a world without adjustment costs, firms would invest the incremental increase in its liquidity; the interaction of liquidity constraints, fixed cost of adjustment and public subsidy can result in an R&D investment increase, but as function of adjustment costs.

Generally speaking, firms put high relevance in maintaining a financial flexibility, therefore are not interested in drying up all the liquidity/cash-flow available, and this can influence the R&D investment behavior also in presence of additional cash-flow through public subsidies.

Some scholars dealing with the high adjustment costs which can accompany R&D spending (Hall and Lerner, 2010; Hall, Griliches, and Hausman, 1986; Lach and Schankerman, 1988) allow to consider that the impact of changes in the costs of capital is difficult to measure, because such effects can be weak in the short run, due to the slow response of R&D to any changes in its cost.

Other results from the literature on program evaluation can complement the above references. Hottenrott and Peters (2009) introduced the concept of innovation capacity, based on the skill structure of firms' employees, innovation experience and firms' efforts to train their employees. Firms with higher innovation capacity are more likely to have unexploited innovation projects, independent of their financial background. Firms with high innovation capacity but low financial resources turn out to be the most likely constrained.

Size and age can be factors influencing firms' sensitivity to liquidity constraints and therefore a subsidy dose-response effect. Several studies find evidence that smaller and younger firms are more likely to face financing constraints as they usually cannot provide as much overall collateral value compared to larger, more capital intensive firms: Himmelberg and Peterson (1994), Petersen and Rajan (1995), Berger and Udell (2002), Czarnitzki (2006), Ughetto (2008) and Czarnitzki and Hottenrott (2009), Müller and Zimmermann (2009). Problems of asymmetric information may be less severe for older firms that have established a long and stable relationship with their bank: Canepa and Stoneman, 2002, Savignac, 2008, Schneider and Veugelers, 2008. Financing constraints are less severe for large firms (Savignac, 2008), which therefore can react better to the fact that an additional dose of public fund, enabling new R&D capital and projects may ask for additional liquidity or external market funding.

There is a large literature evaluating the effects of public RD subsidies on firm's RD spending and innovation performance. In general the studies addressing selection bias have found evidence of an average additionality effect upon RD investment: Czarnitzki (2003), Duguet (2004), Gonzales et al. (2005), Czarnitzki and Licht (2006), Aerts and Schmidt (2008), Hussinger (2008), Czarnitzki and Lopez Bento (2013), Czarnitzki and Hussinger (2013). Nonetheless some studies (Cerulli, Potì, 2010) have shown a largely heterogeneous distribution of the additionality effect for treated firms. Finally, there can be a difference in the public subsidy efficiency also between short and long term. Some authors (Silva and Carreira, 2010) find that at short term subsidies increase firms' financial capacity, while the effect on firm's long term capacity is less clear-cut given the unclear relation between a positive certification effect vs. a firm's subsidy inertia.

3. The model

Consider two different and exclusive outcomes: one referring to a unit i when she is treated, y_{1i} ; and one referring to the same unit when she is untreated, y_{0i} . Define w_i as the treatment indicator, taking value 1 for treated and 0 for untreated units, and $\mathbf{x}_i = (x_{1i}, x_{2i}, x_{3i}, \dots, x_{Mi})$ as a row vector of M exogenous and observable characteristics (confounders) for unit $i = 1, \dots, N$. Let N be the number of units involved in the experiment, N_1 be the number of treated units, and N_0 the number of untreated units with $N = N_1 + N_0$.

Define two distinct functions, $g_1(\mathbf{x}_i)$ and $g_0(\mathbf{x}_i)$, as the unit i 's responses to the vector of confounding variables \mathbf{x}_i when the unit is treated and untreated respectively. Assume μ_1 and μ_0 to be two scalars, and e_1 and e_0 two random variables having zero unconditional mean and constant variance. Finally, define t_i – taking values within the continuous range $[0;100]$ – as the continuous-treatment indicator, and $h(t_i)$ as a general derivable function of t_i . In what follows, in order to simplify notation, we'll get rid of the subscript i when defining population quantities and relations.

Given previous notation, we assume a specific population generating process for the two exclusive potential outcomes²:

$$\begin{cases} w = 1: & y_1 = \mu_1 + g_1(\mathbf{x}) + h(t) + e_1 \\ w = 0: & y_0 = \mu_0 + g_0(\mathbf{x}) + e_0 \end{cases} \quad (1)$$

where the $h(t)$ function is different from zero only in the treated status. Given this, we can also define the causal parameters of interests. Indeed, by defining the treatment effect as the difference

² Such a model is the representation of a *treatment random coefficient regression* as showed by Wooldridge (1997; 2003). See also Wooldridge (2010, Ch. 18). For the sake of simplicity, as we refer to the population model, here we avoid to write the subscript i referring to each single unit i 's relationships.

$TE = (y_1 - y_0)$, We define the causal parameters of interests, as the population Average Treatment Effects (ATEs) conditional on \mathbf{x} and t , that is:

$$\begin{aligned} ATE(\mathbf{x}; t) &= E(y_1 - y_0 \mid \mathbf{x}, t) \\ ATET(\mathbf{x}; t > 0) &= E(y_1 - y_0 \mid \mathbf{x}, t > 0) \\ ATENT(\mathbf{x}; t = 0) &= E(y_1 - y_0 \mid \mathbf{x}, t = 0) \end{aligned} \quad (2)$$

where ATE indicates the overall average treatment effect, ATET the average treatment effect on treated, and ATENT the one on untreated units. By the law of iterated expectation (LIE), we know that the population unconditional ATEs are obtained as:

$$\begin{aligned} ATE &= E_{(\mathbf{x}; t)} \{ATE(\mathbf{x}; t)\} \\ ATET &= E_{(\mathbf{x}; t > 0)} \{ATE(\mathbf{x}; t > 0)\} \\ ATENT &= E_{(\mathbf{x}; t = 0)} \{ATE(\mathbf{x}; t = 0)\} \end{aligned} \quad (3)$$

where $E_{\mathbf{z}}(\cdot)$ identifies the mean operator taken over the support of a generic vector of variables \mathbf{z} . By assuming a linear-in-parameters parametric form for $g_0(\mathbf{x}) = \mathbf{x}\boldsymbol{\delta}_0$ and $g_1(\mathbf{x}) = \mathbf{x}\boldsymbol{\delta}_1$ the Average Treatment Effect (ATE) conditional on \mathbf{x} and t becomes:

$$ATE(\mathbf{x}, t, w) = w \cdot [\mu + \mathbf{x}\boldsymbol{\delta} + h(t)] + (1 - w) \cdot [\mu + \mathbf{x}\boldsymbol{\delta}] \quad (4)$$

where $\mu = (\mu_1 - \mu_0)$ and $\boldsymbol{\delta} = (\boldsymbol{\delta}_1 - \boldsymbol{\delta}_0)$ and the unconditional Average Treatment Effect (ATE) related to model (1) is equal to:

$$ATE = p(w = 1) \cdot (\mu + \bar{\mathbf{x}}_{t > 0} \boldsymbol{\delta} + \bar{h}_{t > 0}) + p(w = 0) \cdot (\mu + \bar{\mathbf{x}}_{t = 0} \boldsymbol{\delta})$$

where $p(\cdot)$ is a probability, and $\bar{h}_{t > 0}$ is the average of the response function taken over $t > 0$. Since, by LIE, we have that $ATE = p(w=1) \cdot ATET + p(w=0) \cdot ATENT$, we obtain from the previous formula that:

$$\begin{cases} \text{ATE} = p(w=1)(\mu + \bar{\mathbf{x}}_{t>0}\boldsymbol{\delta} + \bar{h}_{t>0}) + p(w=0)(\mu + \bar{\mathbf{x}}_{t=0}\boldsymbol{\delta}) \\ \text{ATET} = \mu + \bar{\mathbf{x}}_{t>0}\boldsymbol{\delta} + \bar{h}_{t>0} \\ \text{ATENT} = \mu + \bar{\mathbf{x}}_{t=0}\boldsymbol{\delta} \end{cases} \quad (5)$$

where the dose-response function is given by averaging $\text{ATE}(\mathbf{x}, t)$ over \mathbf{x} :

$$\text{ATE}(t) = \begin{cases} \text{ATET} + (h(t) - \bar{h}_{t>0}) & \text{if } t > 0 \\ \text{ATENT} & \text{if } t = 0 \end{cases} \quad (6)$$

that is a function of the treatment intensity t . The estimation of equation (6) under different identification hypotheses is the main purpose of next sections.

3.1 The regression approach

In this section we consider the conditions for a consistent estimation of the causal parameters defined in (2) and (3) and thus of the dose-response function in (6). What it is firstly needed, however, is a consistent estimation of the parameters of the potential outcomes in (1) – we call here “basic” parameters – as both ATEs and the dose-response function are functions of these parameters.

Under previous definitions and assumptions, and in particular the form of the potential outcomes in model (1), to be substituted into Rubin’s potential outcome equation $y_i = y_{0i} + w(y_{1i} - y_{0i})$, the following *Baseline random-coefficient regression* can be obtained (Wooldridge, 1997; 2003):

$$y_i = \mu_0 + w_i \cdot \text{ATE} + \mathbf{x}_i \boldsymbol{\delta}_0 + w_i \cdot (\mathbf{x}_i - \bar{\mathbf{x}}) \boldsymbol{\delta} + w_i \cdot (h(t_i) - \bar{h}) + \eta_i \quad (7)$$

where $\eta_i = e_{0i} + w_i \cdot (e_{1i} - e_{0i})$.

The equation sets out in (12), provides the baseline regression for estimating the basic parameters $(\mu_0, \mu_1, \boldsymbol{\delta}_0, \boldsymbol{\delta}_1, \text{ATE})$ and then all the remaining ATEs. Both a semi-parametric or a parametric approach can be employed as soon as a parametric or a non-parametric form of the function $h(t)$ is assumed. In both cases, however, in order to get a consistent estimation of basic parameters, we need some additional hypotheses. We start by assuming first *Unconfoundedness* or *Conditional*

Mean Independence (CMI), showing that it is sufficient to provide parameters' consistent estimation. Then we remove this hypothesis and introduce other identifying assumptions.

3.2 Estimation under Unconfoundedness

Unconfoundedness states that, conditional on the knowledge of the true exogenous confounders \mathbf{x} , the condition for randomization are restored, and causal parameters become identifiable. Given the set of random variables $\{y_{1i}, y_{0i}, w_i, \mathbf{x}_i\}$ as defined above, Unconfoundedness (or CMI) implies that:

$$E(y_{ij} | w_i, \mathbf{x}_i) = E(y_{ij} | \mathbf{x}_i) \text{ with } j = \{0, 1\}$$

CMI is a sufficient condition for identifying ATEs and the dose-response function in this context. Indeed, this assumption entails that, given the observable variables collected in \mathbf{x} , both w and t are exogenous in equation (7), so that we can write the regression line of the response y simply as:

$$E(y_i | w_i, t_i, \mathbf{x}_i) = \mu_0 + w_i \cdot \text{ATE} + \mathbf{x}_i \boldsymbol{\delta}_0 + w_i \cdot (\mathbf{x}_i - \bar{\mathbf{x}}) \boldsymbol{\delta} + w_i \cdot (h(t_i) - \bar{h}) \quad (8)$$

and Ordinary Least Squares (OLS) can be used to retrieve consistent estimation of all parameters. Once a consistent estimation of the parameters in (8) is obtained, we can estimate ATE directly from this regression, and ATET, ATENT and the dose-response function by plugging the estimated basic parameters into formula (5) and (6). This is possible because these parameters are functions of consistent estimates, and thus consistent themselves. Observe that standard errors for ATET and ATENT can be correctly obtained via bootstrapping (see Wooldridge, 2010, pp. 911-919).

To complete the identification of ATEs and the dose-response function, we finally assume a parametric form for $h(t)$:

$$h(t_i) = at_i + bt_i^2 + ct_i^3 \quad (9)$$

where a , b , and c are parameters to be estimated in regression (8).

Under CMI, an OLS estimation of equation (8) produces consistent estimates of the parameters, we indicate as $\hat{\mu}_0, \hat{\boldsymbol{\delta}}_0, \hat{\text{ATE}}, \hat{\boldsymbol{\delta}}, \hat{a}, \hat{b}, \hat{c}$. With these parameters at hand, we can finally estimate consistently the dose-response function as:

$$\hat{ATE}(t_i) = w[\hat{ATE}T + \hat{a}(t_i - \frac{1}{N} \sum_{i=1}^N t_i) + \hat{b}(t_i^2 - \frac{1}{N} \sum_{i=1}^N t_i^2) + \hat{c}(t_i^3 - \frac{1}{N} \sum_{i=1}^N t_i^3)] + (1-w) \hat{ATE}T \quad (10)$$

where:

$$\hat{ATE}T(t_i) = \hat{ATE}(t_i)_{t_i > 0}$$

A simple plot of the curve $\hat{ATE}(t_i)_{t_i > 0}$ over the support of t returns the pattern of the dose-response function. Moreover, for each level of the dose t , it is also possible to calculate the α -confidence interval around the dose-response curve. Indeed, by defining $T_1=t-E(t)$, $T_2=t^2-E(t^2)$ and $T_3=t^3-E(t^3)$, the standard error of the dose-response function is equal to³:

$$\hat{\sigma}_{\hat{ATE}(t)} = \left\{ T_1^2 \hat{\sigma}_a^2 + T_2^2 \hat{\sigma}_b^2 + T_3^2 \hat{\sigma}_c^2 + 2T_1T_2 \hat{\sigma}_{a,b} + 2T_1T_3 \hat{\sigma}_{a,c} + 2T_2T_3 \hat{\sigma}_{b,c} \right\}^{1/2} \quad (11)$$

This means that the α -confidence interval of $\hat{ATE}(t)$ for each t is then given by:

$$\left\{ \hat{ATE}(t) \pm Z_{\alpha/2} \cdot \hat{\sigma}_{\hat{ATE}(t)} \right\}$$

that can be usefully plotted along the dose-response curve for detecting visually the statistical significance of the treatment effect along the support of the dose t .

4. The R&D impact of public support intensity

In this section, we present an application of our model on real data. We aim at estimating the effect of public research and development (R&D) support on company R&D performance. The level of the public support to R&D, in fact, is a typical continuous treatment variable showing a large number of zeros for non supported companies and a positive value for all supported units. As dataset we employ the 8th, 9th and 10th Unicredit surveys collecting a large body of information on various characteristics and activities of a sample of Italian companies, including innovation and R&D (public and private) financing for around 5,000 companies in each wave. The timing is: 1998-2000 for the 8th survey, 2001-2003 for the 9th survey, and 2004-2006 for the 10th one. All surveys

³ This comes from the variance/covariance properties where T_1 T_2 T_3 are taken as constant and a , b and c as random variables.

are built by stratifying on sector, size and location, thus being representative of Italian manufacturing companies with more than 10 employees.

The three surveys are then combined in a unique repeated cross-section of 14,106 companies, since building a longitudinal dataset (or panel) would have caused a sharp reduction in the sample size (only 451 companies appear in all the three surveys). Furthermore, since only relatively few businesses present information on R&D financing, being this section of the questionnaires very rich of missing values, exploiting a repeated cross-section guarantees a larger (final) sample size. The final dataset is then merged with companies' balance sheet data coming from the AIDA archive.

Table 4 reports the model's specification for this application. Here a description of the outcomes, binary treatment, treatment level (or dose), and control covariates is compactly reported. This specification of the outcome equation comes from a widely accepted view of the main drivers of company R&D performance as maintained in the R&D policy evaluation literature (David et al. 2000; Cerulli and Potì, 2012).

In each survey, variables are calculated through a three-year average so to have common time consistency. Therefore, given the time structure of the Unicredit surveys, we perform a three-period analysis with each period made of an average over three years. In sum, in this application we cover nine years, from 1998 to 2006.

An usual problem in the impact evaluation of company R&D public support is the difficulty in singling out good candidates as instrumental variables. Our application shares this limitation with the previous literature. Therefore, in this exercise we only apply the OLS control-function approach by leaving the IV approach to be applied in future developments.

Before commenting the results, it seems firstly useful to inspect into the dataset we obtain after combining and merging these different data sources. Our regression's specification, unfortunately, leads to a huge drop of observations. Indeed, due to a great number of crossing missing values and after deleting influential observations, we are left with a sample of 932 observations. Nevertheless, Table 5 shows that the representativeness of the population is quite well kept in the sample both in terms of sector and location. As for the size, on the contrary, we have a higher presence of companies having between 51-250 employees and a lower presence of smaller firms. This might generate a little bias towards less financial constrained companies, thus probably magnifying the average effect of the R&D support considered here.

Another important descriptive statistics is the distribution of the R&D publicly supported, measured as the share of total firm R&D expenditure. Table 6 shows that in our sample this average

share is around 40% with a median equal to 30%. It means that, among the supported companies, a large quota of R&D is generally covered by public financing.

Table 4. Variables used in the specification of the outcome regression model.

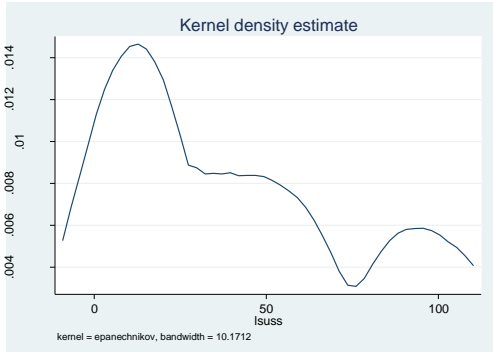
| <i>Covariates</i> | |
|-------------------------|---|
| Treatment | Binary treatment indicator taking value 1 for supported and 0 for non-supported firms |
| Treatment level or dose | Intensity of treatment varying between 0 and 100 |
| N. of employees | Number of company employees, as proxy of company size |
| Debt | Stock of company total stock of debt (long, medium and short term) on total turnover |
| Cash-flow | Rate of profitability, as proxy of company liquidity constrain |
| Labour-intensity | Labor cost to turnover, used as measure of |
| Capital-intensity | Stock of firm material assets to turnover, as |
| Knowledge Stock | Stock of firm immaterial assets, as measure of accumulated R&D experience |
| Group | Binary indicator taking value 1 if the company is part of a group and 0 otherwise |
| Export | Binary indicator taking value 1 if the company exports and 0 otherwise |
| Size | Six categories for company size, using the number of employees |
| Sector | Four categories representing Pavitt sector taxonomy |
| Location | Twenty Italian regions |
| <i>Outcomes</i> | |
| Gross R&D expenditure | Total R&D expenditure |
| Net R&D expenditure | Total R&D expenditure minus the public support received by the firm |
| Gross R&D per capita | Total R&D expenditure on total number of employees |
| Net R&D per capita | Net R&D expenditure on total number of employees |
| Gross R&D intensity | Total R&D expenditure on company turnover |
| Net R&D intensity | Net R&D expenditure on company turnover |

Table 5. Representativeness of the final sample employed in this application.

| | <i>Sample</i> | <i>Population</i> |
|-----------------------|---------------|-------------------|
| <i>Size</i> | | |
| 0-10 | 8.78 | 3.08 |
| 11-20 | 12.24 | 29.48 |
| 21-50 | 18.2 | 33.98 |
| 51-250 | 50.05 | 25.67 |
| 251-500 | 6.39 | 4.05 |
| >500 | 4.33 | 3.74 |
| <i>Location</i> | | |
| North | 73.13 | 67.83 |
| Center | 16.68 | 18.09 |
| South & Islands | 10.21 | 14.06 |
| <i>Sector</i> | | |
| Traditional | 42.47 | 51.23 |
| Scale intensive | 15.6 | 18.03 |
| Specialized suppliers | 36.19 | 25.93 |
| High-tech | 5.74 | 4.8 |
| Number of obs. | 932 | 14,106 |

The number of supported companies is rather small, with 235 units in the final sample: this is due to the fact that very few companies respond to the financing section of the Unicredit questionnaires. Nevertheless, at least in Italy, Unicredit surveys are the only available datasets providing the level of the R&D public support at firm level (Cerulli and Potì, 2012).

Table 6. Some descriptive statistics for public financed company R&D share.

| | | |
|----------------|-------|--|
| Number of obs. | 235 |  |
| Mean | 40.32 | |
| Std. Dev. | 33.67 | |
| Min | 1 | |
| Max | 100 | |
| Median | 30 | |

Given this picture, we can assess the impact of public R&D support on the six outcome measures reported in Table 4. We have to observe, however, that the most relevant outcome for our purposes is the level of “net R&D expenditure” (R&D expenditure *minus* the subsidy received) as it returns the actual amount of additional R&D that a company has been able to perform. Nevertheless, also the effect on “gross” (or “total”) R&D outlay will be estimated. Moreover, as scale effects can be relevant even if controlling for firm size as we do here, we calculate also the effect on both gross

and net R&D expenditure either on turnover and per employee. Results are set out in Table 7 and 8, for all variables, although it seems more interesting the graphical patter of the estimated dose-response function and of the distributions of $ATE(\mathbf{x},t)$, $ATET(\mathbf{x},t)$ and $ATENT(\mathbf{x},t)$. These are visible in Figure 2.

Table 7. Baseline regression for assessing the effect of public support intensity on firm R&D outcomes.

| | (1) Gross R&D | (2) Net R&D | (3) Gross R&D per capita | (4) Net R&D per capita | (5) Gross R&D intensity | (6) Net R&D intensity |
|-------------------|----------------------|----------------------|--------------------------------|------------------------------|-------------------------------|-----------------------------|
| Treatment | 308.39** (142.48) | 325.74** (137.25) | 1.66 (1.22) | 1.22 (1.19) | 0.01 (0.01) | 0.01 (0.01) |
| N. of employees | 2.14*** (0.26) | 2.11*** (0.25) | -0.00 (0.00) | -0.00 (0.00) | -0.00 (0.00) | -0.00 (0.00) |
| Debt | -3.27* (1.95) | -3.38* (1.88) | 0.01 (0.02) | 0.01 (0.02) | 0.00 (0.00) | 0.00 (0.00) |
| Cash-flow | -7.49** (2.99) | -7.44*** (2.88) | 0.02 (0.03) | 0.01 (0.02) | 0.00* (0.00) | 0.00* (0.00) |
| Labour-intensity | 1.65 (3.41) | 1.78 (3.29) | -0.02 (0.03) | -0.02 (0.03) | 0.00 (0.00) | 0.00 (0.00) |
| Capital-intensity | 0.96 (0.80) | 0.95 (0.77) | 0.01* (0.01) | 0.01* (0.01) | -0.00 (0.00) | -0.00 (0.00) |
| Knowledge Stock | 4.53 (16.85) | 4.86 (16.23) | 0.13 (0.14) | 0.14 (0.14) | 0.00 (0.00) | 0.00 (0.00) |
| Group | 159.25** (69.39) | 124.34* (66.84) | 0.86 (0.59) | 0.66 (0.58) | 0.01 (0.00) | 0.00 (0.00) |
| Export | -1.74 (79.93) | 4.06 (76.99) | 0.96 (0.69) | 0.81 (0.66) | -0.01 (0.00) | -0.01 (0.00) |
| Parameter a | -3.01 (19.39) | -12.27 (18.68) | 0.01 (0.17) | -0.01 (0.16) | -0.00 (0.00) | -0.00 (0.00) |
| Parameter b | 0.00 (0.48) | 0.01 (0.46) | -0.00 (0.00) | -0.00 (0.00) | -0.00 (0.00) | -0.00 (0.00) |
| Parameter c | 0.00 (0.00) | 0.00 (0.00) | 0.00 (0.00) | 0.00 (0.00) | 0.00 (0.00) | 0.00 (0.00) |
| <i>N</i> | 932 | 932 | 932 | 932 | 928 | 928 |
| adj. R^2 | 0.399 | 0.345 | -0.004 | -0.011 | 0.041 | 0.034 |
| r^2 | 0.43 | 0.38 | 0.04 | 0.04 | 0.09 | 0.08 |
| F | 14.75 | 11.90 | 0.93 | 0.77 | 1.88 | 1.72 |

Standard errors in parentheses. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Sector, size and location dummies not reported.

Table 8. Baseline regression for assessing the effect of public support intensity on firm R&D outcomes. Standardized coefficients.

| | (1) Gross R&D | (2) Net R&D | (3) Gross R&D per capita | (4) Net R&D per capita | (5) Gross R&D intensity | (6) Net R&D intensity |
|-------------------|---------------------|---------------------|--------------------------------|------------------------------|-------------------------------|-----------------------------|
| Treatment | 0.118** (142.48) | 0.136** (137.25) | 0.096 (1.22) | 0.073 (1.19) | 0.096 (0.01) | 0.076 (0.01) |
| N. of employees | 0.390*** (0.26) | 0.417*** (0.25) | -0.043 (0.00) | -0.038 (0.00) | -0.084 (0.00) | -0.082 (0.00) |
| Debt | -0.052* (1.95) | -0.059* (1.88) | 0.036 (0.02) | 0.029 (0.02) | 0.059 (0.00) | 0.051 (0.00) |
| Cash-flow | -0.071** (2.99) | -0.076*** (2.88) | 0.022 (0.03) | 0.019 (0.02) | 0.070* (0.00) | 0.069* (0.00) |
| Labour-intensity | 0.015 (3.41) | 0.018 (3.29) | -0.026 (0.03) | -0.028 (0.03) | 0.014 (0.00) | 0.012 (0.00) |
| Capital-intensity | 0.038 (0.80) | 0.041 (0.77) | 0.070* (0.01) | 0.076* (0.01) | -0.056 (0.00) | -0.052 (0.00) |
| Knowledge Stock | 0.009 (16.85) | 0.010 (16.23) | 0.038 (0.14) | 0.041 (0.14) | 0.028 (0.00) | 0.031 (0.00) |
| Group | 0.066** (69.39) | 0.056* (66.84) | 0.054 (0.59) | 0.043 (0.58) | 0.054 (0.00) | 0.051 (0.00) |
| Export | -0.001 (79.93) | 0.002 (76.99) | 0.051 (0.69) | 0.045 (0.66) | -0.043 (0.00) | -0.059 (0.00) |
| Parameter a | -0.057 (19.39) | -0.252 (18.68) | 0.022 (0.17) | -0.034 (0.16) | -0.005 (0.00) | -0.094 (0.00) |
| Parameter b | 0.006 (0.48) | 0.019 (0.46) | -0.064 (0.00) | -0.209 (0.00) | -0.156 (0.00) | -0.138 (0.00) |
| Parameter c | 0.012 (0.00) | 0.090 (0.00) | 0.056 (0.00) | 0.142 (0.00) | 0.187 (0.00) | 0.133 (0.00) |
| N | 932 | 932 | 932 | 932 | 928 | 928 |
| adj. R^2 | 0.399 | 0.345 | -0.004 | -0.011 | 0.041 | 0.034 |
| r ² | 0.43 | 0.38 | 0.04 | 0.04 | 0.09 | 0.08 |
| F | 14.75 | 11.90 | 0.93 | 0.77 | 1.88 | 1.72 |

Standard errors in parentheses. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Sector, size and location dummies not reported.

Results in Table 7 show that public support to both gross and net R&D is effective at least at the 5% significant level. Quite surprisingly, the effect on net R&D is a bit higher, around 325 thousand euro. Other significant predictors are *size* (measured as number of employees, with a positive sign), *cash-flow* (although with a negative sign) and belonging to a group of firms (with a positive value). As to cash-flow, since such a measure is not specifically referring to R&D, as it can be devoted by firms to different uses, a negative sign can mean that in the presence of public subsidies firm liquidity is used to sustain other company functions not strictly related to R&D (for instance, marketing, fixed capital, etc.). In terms of coefficients' magnitude, measured by standardized coefficients, Table 8 reveals that *size* is the most relevant predictor followed by the *Treatment* having however a coefficient three times lower.

In the R&D literature, it is well recognized that company size has generally a great impact on R&D spending; thus, having found a positive and significant effect of R&D support even by controlling for company size, suggests that public financing has been effective in fostering the level of both net and gross R&D expenditure. Nevertheless, when considering both gross and net R&D per capita and R&D intensity (obtained in dividing by firm turnover) results show no significant (although positive) effect of R&D public support. This might indicate that the additional level of R&D induced by the public support has been not comparatively higher than the growth in company employees and turnover. As such, previous conclusions on support's effectiveness might be carefully reconsidered, as scale-neutral R&D performance indicators do not show such a result. Anyway, if the level of R&D is the main policy objective, moderately optimistic conclusions on the achievements of the policy thus evaluated can be drawn.

In the future, we intend to estimate this dose-response model grouping firms by size and sector, either to take into account differential easiness in raising funds in the market of capital (size), or a different distribution of technological opportunities among firms (sector).

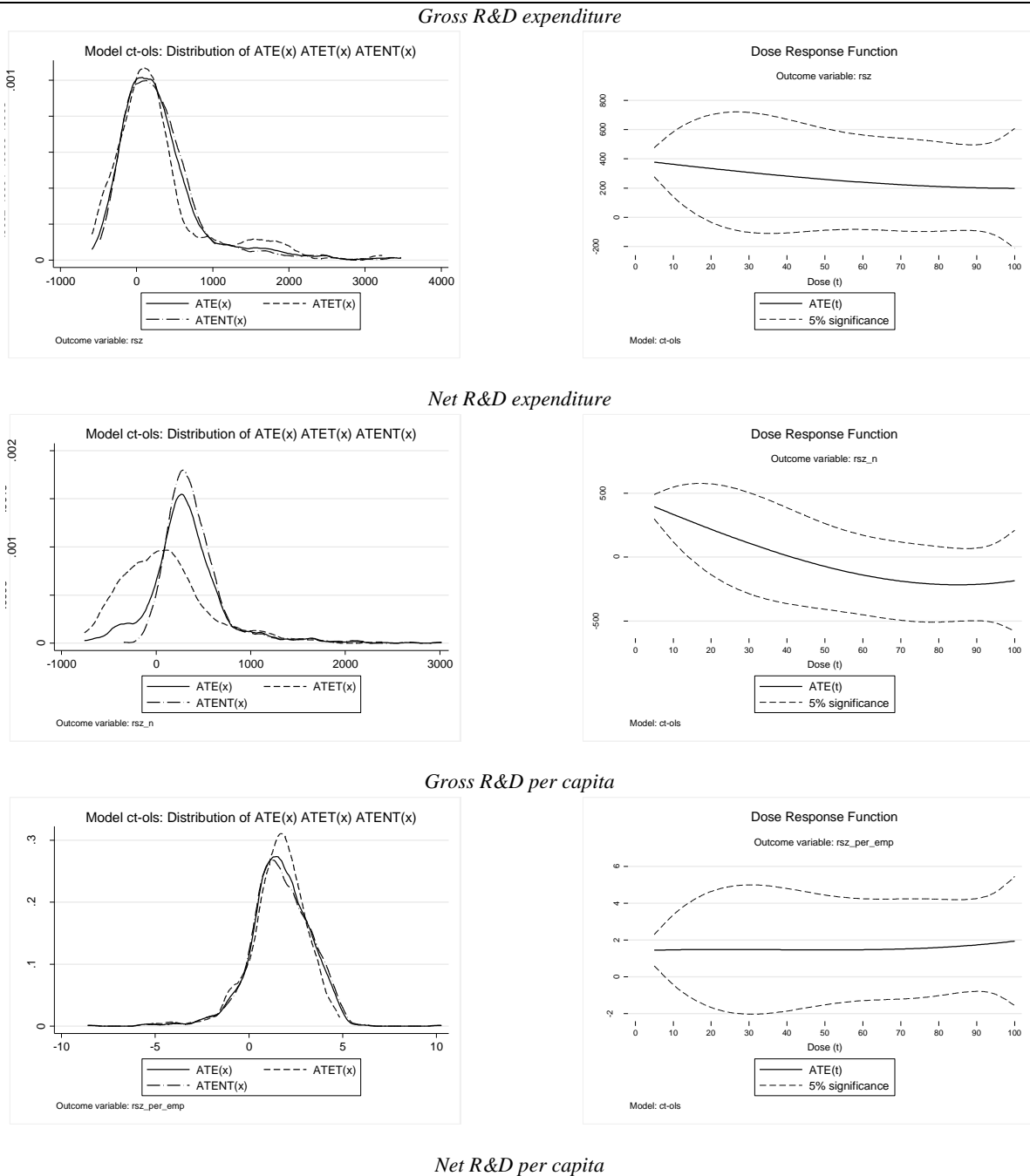
Figure 2 shows the kernel estimation of the distribution of $ATE(\mathbf{x},t)$, $ATET(\mathbf{x},t)$ and $ATENT(\mathbf{x},t)$ and the plot of the dose-response function with 95% confidence intervals. As for the distributions, it is immediate to see that the net R&D performance shows, in each graph, a much more disperse distribution for $ATET(\mathbf{x},t)$ compared with $ATE(\mathbf{x},t)$ and $ATENT(\mathbf{x},t)$. Moreover, $ATET(\mathbf{x},t)$ appears much more concentrated on lower values, thus indicating that the effect on treated units seems surprisingly not only less regular, but also weaker for treated than for untreated units. This might question the selection process adopted by the public agency (although, on average, differences are not strong). An analysis by sector will allow us to understand whether the better performance of untreated firms can be attributed to better technological opportunities.

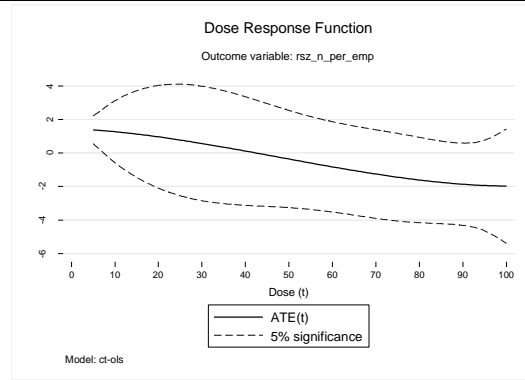
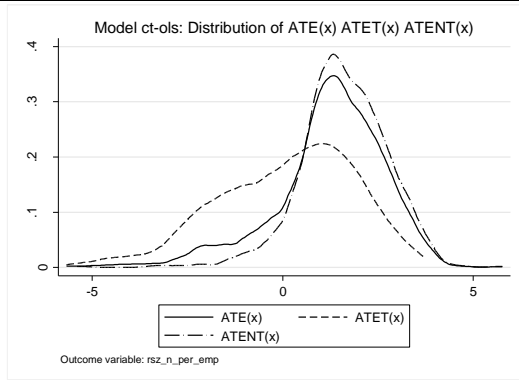
More interesting for the aim of this paper is the pattern of the dose-response functions. As for both gross and net R&D, it is easy to see that the dose-response function has a negative slope with significant confidence intervals lying in between (0;20] for gross R&D spending, and in between (0;15] for net R&D. This result says that the overall positive effect of the policy found in the previous regression results (Table 7 and 8) is mainly driven by those supported companies getting a comparatively lower share of R&D covered by public support (no more than around 20% or 15% for gross and net R&D respectively). A similar finding has been found in Marino et al (2011), using the Hirano and Imbens' approach on company R&D support in Denmark. No effect seems to emerge for higher shares of publicly financed R&D, as the dose-response functions decrease slowly with very large confidence intervals. Interestingly, company net R&D expenditure

becomes negative around a threshold of 40%: this finding might have remarkable policy implications.

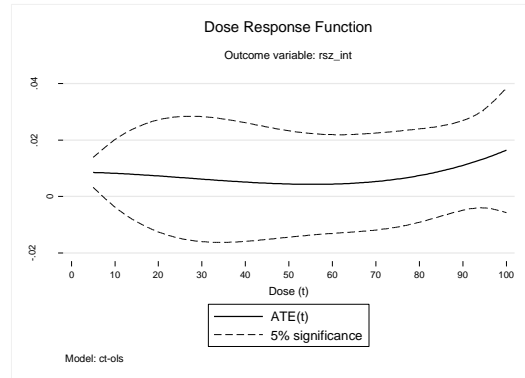
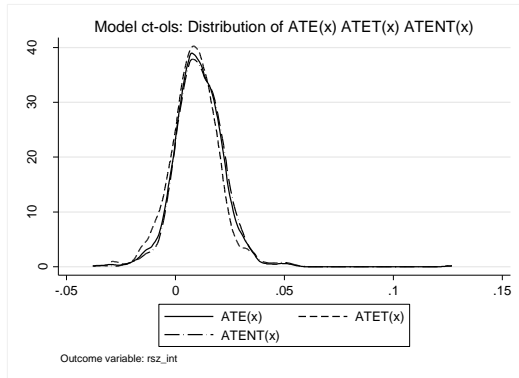
Results on R&D per capita and R&D intensity seem similar and in tune with previous regression output: confidence intervals are almost uniformly large, with the pattern of net R&D appearing decisively more decreasing than that of gross R&D.

Figure 2. Distribution of $ATE(x,t)$, $ATET(x,t)$ and $ATENT(x,t)$ and dose-response function with confidence intervals.

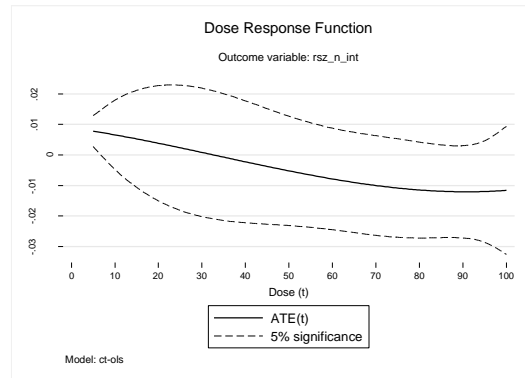
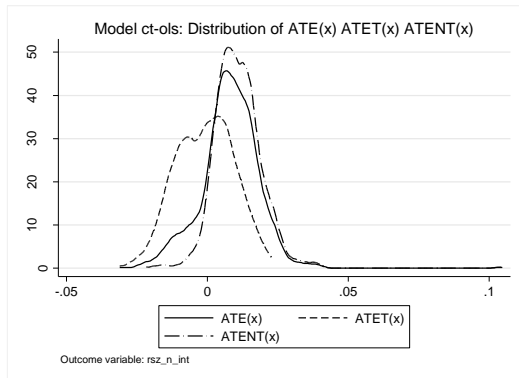




Gross R&D intensity



Net R&D intensity



5. Conclusion

In this paper we assessed the effect of the intensity of public R&D support (measured as the share of total company R&D expenditure) on business gross and net R&D outlay using a dose-response model. Results seem to shed some new light on the relation between public support and its effect on company R&D behavior⁴. In particular, by exploiting the proposed model, we are able to look at the pattern of the policy effect over treatment intensity, thus going beyond the typical average-effect analysis. The pattern of the dose-response function, along with the plot of its confidence intervals, allows us for a better inspection into the causal relation between the policy instrument and the

⁴ In relation to this, see also Cerulli (2010) for an econometric review of the treatment models used in this field of study.

policy target. Additionally, a joined plot of the distributions of the average treatment effects conditional on the covariates is also reported and commented.

Our results show, coherently with other papers on policy evaluation based on a continuous treatment frame, a reliable positive and decreasing relation between subsidy and firm R&D investment for a relatively low level of financial support and a negative effect for high doses of public funding. This could be explained on the basis of the adjustment cost model of investment as due to the marginal increase of adjustment costs accompanying higher level of R&D investment, which are not fully compensated by public intervention. Of course, a more detailed model, one including observable factors impacting on firms R&D capital adjustment costs for instance, could offer a more clear-cut explanation of the phenomenon.

Finally, it is worth stressing that, given the unavailability of reliable instrumental variables in our dataset, this application concerned only dose-response estimation under conditionally exogenous treatment (CMI). Future works with richer data and information may solve this limitation by providing an estimation of the policy effect also in the case of treatment endogeneity due to selection on unobservable factors.

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