Bias Reduction in a Matching Estimation of Treatment Effect

Sulla Riduzione della Distorsione nella Stima Matching dell'Effetto del Trattamento

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Abstract The traditional matching methods for the estimation of the treatment parameters are often affected by selectivity bias due to the endogenous joint influence of latent factors on the assignment to treatment and on the outcome, especially in a cross-sectional framework. In this study, we show that the influence of unobserved factors involves a cross-correlation between the endogenous components of the propensity scores and causal effects. A correction for the effects of this correlation on matching results leads to a reduction of bias. A Monte Carlo experiment supports this finding.

Abstract *I tradizionali stimatori matching dei parametri del trattamento spesso producono stime affette da selettività dovuta all'influenza endogena di fattori latenti, specialmente nelle analisi cross-section. In questo studio, mostriamo che l'influenza di fattori non osservabili mette in correlazione la propensione a sottoporsi al trattamento e gli effetti causali prodotti da quest'ultimo. La correzione delle stime matching per gli effetti di questa correlazione consente di ridurre la distorsione dovuta alla selettività. Questo risultato è supportato dalle evidenze di una serie di esperimenti Monte Carlo.*

Key words: propensity score matching, endogenous treatment, measurement and transition equation.

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1 Introduction

A typical assumption of models for treatment effects is based on the hypothesis that the decision of a subject to receive a certain treatment depends on the difference in the outcomes potentially gained by the subject under the two alternative regimes of treatment and control, respectively (see, e.g., Winship and Morgan, 1999). Starting from this assumption, the decision of a subject to undergo the treatment is endogenous with respect to the potential outcome. The non-random selection of the units into the treatment regime, due to the endogeneity of treatment, involves that important unobserved covariates influence jointly the propensity of a subject to undergo the treatment and the outcomes. As a consequence, matching estimation of the treatment effect, based on the comparison of treated and untreated units with the same propensity score, is biased (e.g. Austin, 2011).

In this context, a natural solution, as the detection of new statistically significant covariates in the treatment choice equation, could not reduce the bias; in fact, Heckman and Navarro-Lozano (2004) show that this is the case when these variables are not exogenous with respect to the outcome.

In this study, we try to circumvent the problem of misspecification of the selection equation in matching methods based on propensity score, assuming that the potentially omitted endogenous factors can be represented by a stochastic component correlated with the causal effects of the treatment. This implies that the causal effect of each subject is correlated with the causal effect of another subject with similar propensity score; moreover, the stochastic component is autocorrelated, as causal effects relative to similar propensity scores will be more similar. In order to assess this endogenous relationship, we model the causal effects adopting a sort of state-space model (see, for example, Harvey, 1990), where a common latent factor is detected in correspondence of the endogenous stochastic component of the propensity score sorted in an ascending (or descending) order. State-space models are generally adopted for time series; the extension to this framework is simple, substituting the ordering of the observations in terms of dating with the order in terms of increasing propensity score. The predictions of these components are used as correction terms in the matching procedure. The estimation method proposed, called State-Space Corrected Matching (*SSCM*), is based on the Kalman filter (see Harvey, 1990) and possesses the nice characteristic of not imposing conditions of identification of the probability to undergo the treatment as in the randomized experiments.

We verify the performance of this method comparing its bias with respect to the bias occurring with traditional propensity score matching (cf., among others, Rosenbaum and Rubin, 1983) by Monte Carlo experiments. In the Monte Carlo experiment we generate data in a cross-sectional context, adopting a two-regime model whose data generation process (*DGP*) is affected by endogeneity. Applying our correction method, we obtain a marked reduction of bias in the estimated average treatment effect for the treated (*ATT*) in comparison with the traditional Propensity Score Matching estimator (*PSME*, Rosenbaum and Rubin, 1983).

In next Section we describe this new procedure, whereas in Section 3 we show the results of the Monte Carlo experiments comparing the performance of *SSCM* estimation method and traditional *PSME* in terms of prediction of the *ATT* parameter.

2 The Model

In this paper we insert several novelties with respect to the present literature. The most relevant is the individuation of an autoregressive process that characterizes, jointly, individual propensity scores and causal effects. As a consequence, another important novelty is given by the correction term based on the estimation of a State-Space model in which the endogenous component common to causal effects and propensity scores is specified by a "measurement" equation and a "transition" equation, respectively. In addition, in our model it is not necessary to reproduce conditions of identification of the probability to undergo the treatment such as in a randomized experiment.

In this analysis, we start to consider the potential outcome gained by choosing one of the two treatment status as a relevant (endogenous) determinant of the decision undergoing the treatment. In particular, we specify the model assuming that the difference between the potential (expected) outcomes, y_{1i} and y_{0i}, obtainable, respectively, under the regimes T_i =1 (if the subject belongs to the treatment group) and T_i=0 (if the subject belongs to the comparison group), determines, at least in part, the choice of the regime.

We specify a Probit (or Logit) model, where the (latent) propensity to undergo the treatment of the *i-th* subject, T^*_i, depends linearly on the covariates in **Z**:

$$T^*_i = \mathbf{z}'_i \boldsymbol{\beta} + v_i \tag{1}$$

where $\mathbf{z}'_i$ is the *i*-th row of the matrix **Z**, β is a vector of unknown coefficients and v_i is a zero-mean random disturbance with unit variance. If $T^*_i > 0$, $T_i = 1$ (the subject is undergone to treatment), otherwise $T_i = 0$ (the subject is not undergone to treatment).

Assuming that the assignment to treatment is endogenous, T^*_i will depend on the causal effect $\Delta_i = y_{1i} - y_{0i}$.

Formally, we can explain autocorrelation and endogeneity specifying our model similarly to a generalized Roy model (e.g., Carneiro et al., 2003). In doing this, we add to the above selection equation (Eq. 1) two equations that specify the outcome of treated and untreated subjects, as follows:

$$y_{1i} = \mu_{1i} + u_{1i} \qquad \text{if } T_i = 1; \tag{2a}$$

$$y_{0i} = \mu_{0i} + u_{0i} \qquad \text{if } T_i = 0; \tag{2b}$$

In Eqs. (2a) and (2b) μ_{1i} and μ_{0i} are the expected outcomes, respectively, of treated and untreated subjects, depending on the decision to undergo the treatment ($T = 1$) or not ($T = 0$). The error terms u_{1i} and u_{0i} are normally distributed with zero mean and variances equal to σ^2_1 and σ^2_0 respectively. The covariances σ_{1v} and σ_{0v} of the disturbances of both outcome equations, u_{1i} and u_{0i}, with the disturbances of the selection equation (1), v_i, can be different from zero as a consequence of endogeneity. The covariances σ_{1v} and σ_{0v} are measurements of the endogeneity of the propensity to undergo the treatment, T^*_I with respect to the outcome gained under T =1 and $T = 0$.

Correlation between outcomes and propensity scores, as well as the autocorrelation of the causal effects, may be specified starting from the definition of causal effects, Δ_i. Hence, subtracting Eq. (2b) from Eq. (2a), we obtain:

$$\Delta_i = y_{1i} - y_{0i} = \mu_{1i} - \mu_{0i} + (u_{1i} - u_{0i}) \tag{3}$$

Imposing a linear relationships between the error terms of the outcome equations and the selection equation, we have:

$$u_{1i} = \sigma_{1v} v_i + \varepsilon_{1i} \tag{4a}$$

$$u_{0i} = \sigma_{0v} v_i + \varepsilon_{0i} \tag{4b}$$

where ε_{1i} and ε_{0i} are i.i.d. disturbance with zero mean. By substituting (4a) and (4b) into Eq. (3):

$$\Delta_i = y_{1i} - y_0 = \mu_{1i} - \mu_0 + (\sigma_{1v} - \sigma_{0v}) v_i + (\varepsilon_{1i} - \varepsilon_{0i}) \tag{5}$$

Putting $\mu_{1i} - \mu_{0i} = \mu_i$; $(\sigma_{1v} - \sigma_{0v}) v_i = \sigma v_i$ and $(\varepsilon_{1i} - \varepsilon_{0i}) = \varepsilon_i$, Eq. (5) can be written as a *measurement equation* of a state-space model, as follows (cf., among others, Harvey, 1990):

$$\Delta_i - \mu_i = \sigma v_i + \varepsilon_i \tag{6}$$

In Eq. (6), ε_i is a vector of $n \times 1$ disturbance terms uncorrelated across i. The variable v_i can be considered as the state variable whose elements are not observable, but are assumed to be generated by a first-order Markov process, such as the following transition "equation":

$$v_i = \rho v_{i-1} + \eta_i \tag{7}$$

The dependent variable of Eq. (6), $\Delta_i - \mu_i$, may be considered as the stochastic component of the causal effect Δ_i, endogenous with respect to the decision to undergo to treatment. Starting from this result, the selectivity effect due to the endogeneity of the decision to undergo the treatment may be corrected by estimating σv_i in Eq. (6), and using the predicted values, $\sigma \hat{v}_i$, as a correction term in the matching estimation of the causal effects. In doing this, a preliminary estimation of

causal effects Δ_i is obtained at a first stage by applying a propensity score matching procedure. Then, at a second stage, matching is replicated using the corrected outcomes $y_{1i} - \sigma\hat{v}_i$ so as to obtain the corrected causal effects $\Delta_i - \sigma\hat{v}_i = \hat{\mu}_i$. We call this estimator the State-Space Corrected Matching (*SSCM*) estimator.

3 Monte Carlo Experiment

We propose a Monte Carlo experiment to compare the performance of the *SSCM* procedure with that of the *PSME* in terms of bias reduction under both the conditions of heterogeneous and homogeneous covariates between regimes. For this purpose, we generate 500 data sets of 2,000 units from the Two-Regime model above in Eqs. (1), (2a) and (2b). The exogenous covariates Z are generated in order to reproduce the very frequent condition of heterogeneity in observed covariates between treatment and comparison group, and the condition of homogeneity in the observed covariates between regimes. We consider two different *DGPs*, with and without endogeneity, so as to fix two distinct set of population parameters under the condition of endogeneity and exogeneity, respectively.

Table 1 summarizes the estimated *ATT* values obtained by embedding different endogeneity conditions into the *DGP*. Computing the bias with respect to the population *ATT* value (set equal to 5), the *SSCM* estimator performs better than the *PSME* procedure. The bias resulting from the application of *SSCM* is markedly smaller than the one resulting from *PSME*.

Table 1: Simulation Results. Estimated ATT parameters. Population *ATT* value = 5. Generated sample size: n = 2000. No of reps. 500. Simulated endogeneity by setting σ_{1v} and σ_{0v}.

	SSCM			*PSME*		
Endogeneity	***ATT***	*95% CI*		***ATT***	*95% CI*	
σ_{1v} 5.4; σ_{0v} 2.4	4.974	*4.930*	*5.018*	7.996	*7.970*	*8.022*
σ_{1v} 5.4; σ_{0v} -2.4	4.320	*4.279*	*4.362*	6.814	*6.782*	*6.846*
σ_{1v} 5.4; σ_{0v} 0.8	4.983	*4.939*	*5.027*	7.571	*7.534*	*7.607*
σ_{1v} 5.4; σ_{0v} -0.8	4.729	*4.688*	*4.770*	7.572	*7.536*	*7.608*
	% BIAS*	*St.Dev.*	*t***	***% BIAS****	*St.Dev.*	*t***
σ_{1v} 5.4; σ_{0v} 2.4	-0.51%	*0.022*	222.160	59.92%	*0.013*	*607.170*
σ_{1v} 5.4; σ_{0v} -2.4	-13.59%	*0.021*	205.720	36.28%	*0.016*	*414.280*
σ_{1v} 5.4; σ_{0v} 0.8	-0.35%	*0.022*	222.480	51.41%	*0.018*	*410.660*
σ_{1v} 5.4; σ_{0v} -0.8	-5.42%	*0.021*	225.710	51.45%	*0.018*	*412.000*

Note: * % of Bias [(Est. ATT-5)/5]%; ** t-ratio: ATT/St.Dev.)

We can observe, in particular, that, if we reproduce the "more common" endogeneity conditions (characterized by covariances, σ_{1v} and σ_{0v}, with the same sign) in the *DGP*, the confidence intervals obtained by the *SSCM* estimates include the population *ATT* value. In the less frequent case, in which the propensity to

undergo the treatment is endogenously affected in the two regimes with opposite sign, confidence intervals of the *SSCM* estimates do not include the population parameter. However, the percentage of bias of *SSCM* estimation does not exceed 15% in absolute value.

4 Conclusion

The aim of this study is to improve the propensity-score matching approach so that estimation results do not overly suffer from the influence of the endogeneity of treatment. We show that, applying a state-space model, we can estimate the endogenous component of the causal effects, so as to use the result of this estimate as a correction term. In particular, the results of the Monte Carlo experiments here reported confirm that, simulating endogeneity of the selection into treatment in a Two-Regime model, the predicted components of causal effects can be successfully used, at a second stage of the estimation procedure, to correct the matches outcomes.

As the results of our Monte Carlo analysis show, this method allows us to reduce the selectivity bias in matching without imposing, to the data or the model, any restriction usually adopted to reproduce a condition comparable to the randomization. At this stage of our research, we have deepened the characteristics of the SSCM estimator only through Monte Carlo experiments. However the inferential properties must still be investigated. This will be the next aim of this research.

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