Effect of the Career on Premature Retirement. Using Propensity-Score-Matching to Estimate Causality

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Data

For the analysis longitudinal data must be used. The FDZ-RV holds different types of datasets. The data of the insured people and their insurance account were used for the following analysis. This data not only includes a sample of the retired population, it also contains a sample of none-retired persons and their biography. The biography began with the year a person gets 15 years old and ends latest with the year the person went 67. For each individual the dataset includes the whole biography till the reference year (in our case the year 2007). The biography part of this data holds for each month information's of the Social Employment Situation (SES). The following figure gives an overview of the enclosed information.

Table 1: SES in longitudinal data (FDZ-RV)

Social Employment Situation (SES)
School / University
Apprenticeship / Training
Care Giving
Homeproduction
Invalidity / Sickness
Unemployment (support, benefit or creditable periods)
Military / Civilian Service
Marginal Employment
Self Employment
Other Activities
Employment Subject to Social Insurance Contributions
Invalidity Pension
Old-Age Pension

(Himmelreicher/Stegmann, 2008, p.654)

With the SES it is possible to isolated different episodes in the biography. This allows taking a closer look to the career of the individuals till they retire (or the reference year is reached).

This set of longitudinal data is useful for the Rubin-Causal-Model, because we are able to analyze the transitions of individuals from work to retirement and built treatment and control groups of retired and still working people.

Methods

The analysis of the longitudinal dataset allows to identify the causal effects of the career with different methods. For the analysis there were two techniques of interest. First the Rubin-Causality-Model (RCM) and second the Propensity-Score-Matching (PSM). The requirements and derivations will be shown in the following parts.

Rubin-Causality-Model

To Analyze the Causal Effect we use a counterfactual approach to causality, the RCM. In the simplest example we have a binary factor for which we measure a causal effect. There it is the difference between the event $Y_{1i} = Y \mid T = 1$ that appears at T and the alternative $Y_{0i} = Y \mid T=0$. With this we are able to assume the causal effect of T as a unit effect on the level of individuals with value X_i.

$$\delta_i = Y_i(X_i, T = 1) - Y_i(X_i, T = 0) = Y_{1i} - Y_{0i}$$

To measure Causal Effects the individual I had to be measured with and without treatment, but this is impossible, only one of the measures is available, the unavailable is the counterfactual. The causal effect of the average treatment effect (ATE) and the Average Treatment effect of the treated (ATET) are the favored measures for such effects. The ATE is the difference between the expected outcome when receiving the treatment and the expected outcome when not receiving the treatment (for any individual randomly drawn from the population), this can be measured by

$$\alpha = E[Y^1 - Y^0]$$

The similar for the ATET,

$$\alpha = E[Y^1 - Y^0 | D = 1]$$

This is the expected outcome difference for any individual randomly drawn from the subpopulation of the treatment recipients. The Term $D_i \in \{0,1\}$ says if an individual received the treatment or not. (Fröhlich 2002b, p.3f)

There are three assumptions which had to comply; the formula below shows these assumptions for the difference of the events $Y_{1i,i\in t} \big| T = 1 \; and \; Y_{0i,i\in C} \big| T = 0$.

$$Y_{1i,i\in E} - Y_{01,i\in C} = \delta T + \left(Y_{0i,i\in E} - Y_{0i,i\in C}\right) + (\delta_{i\in E} - \delta_{i\in C})$$

The difference between treatment and Control persons = “true” causal Effect of δ from Factor T + difference in event Y without the effect of T + difference in the effect of T between treatment and control individuals.

It is also possible to show the difference between groups also as before and after treatment. This is the difference-in-difference estimator (DID).

$$\left(y_{i1}^1 - y_{i0}^1\right) - \left(y_{i1}^0 - y_{i0}^0\right) = \beta + \left(\varepsilon_{i1}^1 - \varepsilon_{i0}^1\right) - (\varepsilon_{i1}^0 - \varepsilon_{i0}^0)$$

Under the assumption that

$$E = [\left(\varepsilon_{i1}^1 - \varepsilon_{i0}^1\right) - \left(\varepsilon_{i1}^0 - \varepsilon_{i0}^0\right)]$$

is equal zero, we can unbiased estimate β and then we have a sample average of

$$\left(y_{i1}^1 - y_{i0}^1\right) - \left(y_{i1}^0 - y_{i0}^0\right)$$

for the estimation of the DID.

The made explanations for measuring the causal effects are only works, as long we can differ between treatment and control groups. To select the individuals there exist several approaches, we chose the PSM method. The reasons are given below as well as the equations to build the propensity score.

Propensity-Score-Matching

To select treatment on the observables, matching estimators of treatment effects are used. Through this method the control and treated population have comparable observed characteristics. The PSM is a non-parametric technique used for the estimating average treatment effects, but it can also be used to decompose effects due to observables and to unobservable and for DID treatment evaluation.

The Propensity Score is the probability of a unit being assigned to a particular condition in a study given a set of known covariates. With the PSM we can reduce the selection bias by equating groups based on these covariates. Let T any given binary Treatment, Y is the outcome and X the background variables. The propensity score is defined as the conditional probability of treatment given background variables:

$$p(x) = \Pr(D = 1 \,|X = x)$$

To evaluate treatment the assumption of balancing condition plays an important role. It says that

$$\mathrm{D} \perp \mathrm{x}|\mathrm{p}(\mathrm{x})$$

In other words, any individual with the same propensity score for the assignment to treatment is random and is identical in terms of their x vector. (Cameron/Trivedi 2005, p.865)

To build the propensity score we chose several items like gender, age of retirement, federal state, sum of earning points. This will allow to identify (the most) equal individuals. With the logistic Regression the propensity score can be calculated as the predicted probability of the Treatment. A simple one-to-one matching would reduce the statistical power and could raise selection bias; the use of caliper and nearest-neighbor techniques can decrease these effects. With the caliper there is the closest neighborhood around the term p(x) chosen. The nearest-neighbor method selects for every individual *i* in the treatment group, the set

$$Ai(x) = \{j|min_j \|x_i - x_j\|\}$$

In Terms of the propensity Score Nearest-Neighbor-Matching can be define as

$$A_i(p(x)) = \{p_j|min_j \|p_i - p_j\|\}$$

The term ‖ ‖ stands for the Euclidean distance between vectors. (Cameron/Trivedi 2005, p.875)

With this method we are able to analyze the causal effect of the career to premature retirement. This allows to show causal effects of the career on PMR and minimize the bias through PSM.

Result of Propensity Score Matching

To Estimate the PS the following Variables were used:

Year of birth, Gender, Location, Sum of earning Points, Contribution Period, Creditable Periods (overall, disability and unemployment)

The Result of the Matching Process is shown below:

Estimate... 20.06

AI SE..... 48.463

T-stat..... 0.41392

p.val..... 0.67893

Original number of observations..... 9441

Original number of treated observations..... 2630

Matched number of observations..... 2630

Further analyses were made and will show the effect of the career on premature retirement.

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