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 $3 \ {\rm November} \ 2022$

Online at https://mpra.ub.uni-muenchen.de/115373/ MPRA Paper No. 115373, posted 16 Nov 2022 09:50 UTC



Growth Effects of European Monetary Union: A Synthetic Control Approach

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November 2022

Abstract

After more than 20 years of European Monetary Union (EMU), surprisingly few scientific studies exist which study the growth effects of introducing a common currency in large parts of the European Union. I do so using a large panel (NUTS3 data) of regional data for the EU-15. Some 800 (treated) regions were subject to a policy intervention when their country joined the Euro, while some 200 control regions were not. In a synthetic control approach as explored e. g. by Abadie, Diamond and Hainmueller (ADH, 2010), I estimate the causal effects of EMU both with the standard ADH-methodology and with a novel approach which estimates counterfactuals from the control group in *post*-treatment time. The results from both approaches are very similar: EMU has benefited regions with export-oriented and highly competitive companies e. g. in Germany, while it has had sizable detrimental growth effects on most French and Mediterranean Eurozone regions. Over eighteen years, these losses in growth cumulate to losses in per-capita income of between 15% and 30% vis-à-vis the non-EMU counterfactual.

Keywords: European Monetary Union, synthetic control methods JEL: C12, C13, C21, C23, E65, F33, N14

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I. Introduction

European Monetary Union (EMU) is certainly one of the most important economic policy measures in recent history. The Euro, formally introduced in 1999, has survived major crises like the global financial crisis and the European sovereign debt crisis and has - until very recently - delivered on its pledge to safeguard price stability in the Eurozone. It is less clear, though, if and to what effect the Euro has had an impact on economic growth.

In fact, after more than 20 years of EMU, surprisingly few academic papers have tried to assess its success in terms of real per-capita growth directly. Rather, many studies investigated the effect of EMU on intermediate variables commonly expected to affect economic growth. Such intermediate variables involved trade, capital accumulation and foreign direct investment (FDI), financial integration, political integration, the volatiliy of the real exchange rate, international price elasticities and institutional development. The final effect of a positive effect on growth is usually taken for granted and not demonstrated by econometric means.

For instance, many papers have found EMU to have a positive effect on trade (Bun and Klassen (2002), Barr et al. (2003), Micco et al. (2003), Baldwin et al. (2008), Kunroo et al. (2016), Camarero et al. (2018)). But Bun and Klaassen (2007) warn that this effect may not be "as large as commonly thought". Also, Berger and Nitsch (2008) present results which suggest that increasing trade intensity is an effect of economic integration within the EU and that EMU did not add anything to it.

Disappointing results have been found for the financial integration channel: Barr et al. (2003) and Bekaert et al. (2013) did not find significant positive effects of EMU on financial integration while Brezigar Masten et al. (2014) argued that it may be predominantly the less developed EU member states outside the currency union which receive growth stimuli from financial development. Gehringer (2013), finally, finds that European monetary integration has no substantial effect on productivity, capital accumulation and economic growth.

On the other hand, several papers identify a positive effect of EMU on FDI flows, e. g. Schiavo (2007), Petroulas (2007), Brouwer et al. (2008), Baldwin et al. (2008), Abbott and de Vita (2011). But all of these papers use data prior to the financial crisis. No similar investigations with more recent data seem to have emerged.

Holtemöller and Zeddies (2013) study international price elasticities to analyze if EMU has increased competition. Their result, derived in a very careful panel data investigation of trade statistics, is negative. Janus and Riera–Crichton (2015) study real effective exchange rate volatility finding that EMU decreased this volatility, but only prior to the financial crisis. Schönfelder and Wagner (2016) report that EMU membership seems to slow down and even reverse institutional development in the Eurozone while progress is made for EU accession candidates. Overall, many studies concerned with intermediate variables fail to find a significant effect of EMU on the variable of interest. Even if they do, the effect may be small or other channels which do not operate as expected may have potential to affect growth adversely.

Only a few studies tackle EMU and its effect on economic growth directly. Conti (2014) uses a differences-in-differences (DiD) framework for seventeen European countries and finds positive effects of EMU on GDP per capita, but to a lesser extent for countries with high initial debt levels. Kalaitzoglou and Durgheu (2016) report ambiguous results of EMU which they attribute to easier access to finance on the one hand and a tendency to overborrowing on the other hand. Dreyer and Schmid (2016) employ panel GMM methods and conclude that Eurozone membership has had no significant effect on growth 1999 through 2012. Ioannotos (2018), in another DiD approach, comes to the same conclusion for a sample covering the Eurozone till 2016. Finally, a group of studies exists which is closest in spirit to this paper as they also target GDP per capita and use the synthetic control method, cf. Fernandez and Garcia Perea (2015), Verstegen et al. (2017), Puzzello and Gomis-Porqueras (2018) and Gabriel and Pessoa (2020). Results are quite different for pre- and post-crisis time periods but mostly find little overall benefit of EMU membership.

All of these studies use data on country level. Hence both the cross section and the time dimension of the data is small. Eleven EU countries adopted the Euro in 1999, with Greece joining two years later. Often, these 12 countries are compared to a similarly small set of non-Euro countries or to their own performance in the years prior to 1999. Great heterogeneity between countries and very dissimilar macroeconomic shocks in the 1990s as opposed to the 2000s make it hard to identify the causal effects of EMU in small samples. Observed and unobserved heterogeneity may, in fact, negatively affect the robustness of the estimates or even induce sizable biases.

In this study, I use regional data from the EU's ARDECO (Annual Regional Database of the European Commission) data base. The classification is NUTS3, i. e. the cross section dimension comprises some 1027 regional units for which real GDP per capita and a host of other structural information is available. While this does, of course, not reduce the heterogeneity of the sample, it makes it much more likely that regions in the Eurozone can be matched with a "synthetic twin" region with similar economic characteristics outside the Eurozone. As far as I know, no other study has previously used this rich data set with the aim of identifying the causal effects of EMU on economic growth.

For this purpose, I use the Rubin causal model (cf. Rubin (1974), Holland (1986)) and apply modern synthetic control methods (SCM, e. g. Abadie et al. (2010), Malo et al (2020), Abadie (2021)) to construct the potential outcome of a Eurozone region under the counterfactual scenario that no change in currency had taken place. Such methods have attracted much interest in recent years and are, in the words of Athey and Imbens (2017), "arguably the most important innovation in the policy evaluation literature in the last 15 years." In fact, some authors have previously applied SCM to the adoption of the Euro, albeit only on country level.

Methodologically, the standard approach is due to Abadie et al. (2010, henceforth ADH). ADH solve an optimistic bilevel minimization problem in order to determine the optimal weights for the construction of synthetic controls. This type of problem is mathematically complex and a number of unwelcome issues (which initially went unnoticed), have been detected and discussed in the more recent literature. For instance, Ferman and Pinto (2016) noted that the ADH-estimator is asymptotically biased under plausible assumptions. Also, a number of authors have reported numerical problems: The commonly used Synth-algorithm proposed by ADH is unstable and may not converge to the global minimum of the objective function, cf. Becker and Klößner (2017), Becker et al. (2018) and Klößner et al. (2018).

Economically, the ADH-method has been a source of concern because covariates which are believed to have predictive power for the outcomes of interest, have empirically been found to have very little (or even zero) impact on the construction of the synthetic controls (SC). For instance, Kaul et al. (2015) showed that covariates always receive zero weight when they compete with all pre-treatment outcomes - irrespective of the predictive power of the covariates.

This is a trivial consequence of the mathematical structure of the bilevel problem. But even if – informationally inefficient - only a subset of the pre-treatment outcomes is used in the bilevel problem, several researchers have noted that the influence of covariates on the constructed synthetic control is usually surprisingly small.

Some authors have suggested improvements or modifications of the ADH approach, e. g. Ferman and Pinto (2016), Chernozhukov et al. (2020) and Malo et al. (2020). In Lucke (2022) I have argued that one should aim at minimizing the expected post-treatment synthetic control error. Under the static common factor model underlying much of the literature, the required parameters can be estimated consistently by least squares and the optimal weights can be found by solving a standard quadratic minimization problem rather than the complex bilevel problem. To show how this method compares with the standard ADH approach, I derive the synthetic control weights under both approaches.

The sequel of the paper is organized as follows. In section II I present the Rubin causal model in terms of a static factor model for potential outcomes. Section III reviews the standard ADH approach and Section IV briefly describes the alternative method laid out in Lucke (2022). In section V I describe the regional data available in the ARDECO data set. Section VI presents the estimates of the counterfactual trajectories for per-capita GDP of Eurozone NUTS3-regions. I discuss differences and similarities in the results of the two SCM approaches. Section VII concludes.

II. The Model

Suppose we observe a balanced panel of J + 1 units over $T = T_0 + T_1$ periods of time. Unit 1 has been randomly assigned to a policy intervention (the treatment) from period $T_0 + 1$ onward. The policy has no impact on units 2,..., J + 1 and no impact on unit 1 prior to period $T_0 + 1$. We are interested in the effect the treatment has had on a specific cardinal variable *y*, which we call the outcome. The observed outcome for region *i* in period *t* is denoted y_{it} and is either the outcome under treatment or under non-treatment, whatever applies.

We assume that for each unit *i* and period $t > T_0$ the *potential* outcome in the case of nontreatment can be expressed as a linear function of *R*-dimensional, random vector z_i of observables and of an *F*-dimensional random vector λ_i of unobservable shocks. The observable, unit-specific variables z_i have deterministic, but time-variant coefficients $\theta_i \in \mathbb{R}^R$, $t = T_0 + 1, ..., T$, and the unobservable time-variant shocks have deterministic, but unitspecific loading coefficients $\mu_i \in \mathbb{R}^F$, i = 1, ..., J + 1:

$$y_{it}^{N} = z_{i} \,' \theta_{t} + \lambda_{t} \,' \mu_{i} \tag{1}$$

Here the superscript N indicates that y_{it}^N is the potential outcome in the case of nonintervention. I assume that there are C common factors and J + 1 idiosyncratic shocks, i. e. F = C + J + 1. I sometimes use the partition $\lambda_t = (\lambda_t^{C'} \lambda_t^{I'})'$ where λ_t^C contains the common factors and λ_t^I contains the idiosyncratic shocks. I partition $\mu_i = (\mu_i^{C'} \mu_i^{I'})'$ accordingly. A shock is idiosyncratic for unit *i* iff μ_i is nonzero only in row C + i and all other μ_j 's are zero in this row.

The observable variables z_i are called the predictors. Note that the predictors may contain a constant term $z_{1i} = 1 \quad \forall i = 1, ..., J + 1$. Further, z_i is assumed to involve only variables unaffected by the policy intervention, i. e. either variables which are strictly exogenous or variables which have been determined prior to treatment. For instance, in a macroeconomic application where outcomes are functions of GDP, z_i may involve endogenous variables like investment, human capital, infrastructure etc., provided these variables were determined not later than period T_0 .

Note that the right-hand side of (1) involves three unobservables, θ_t , λ_t and μ_i . Many observationally equivalent choices for these unobservables exist. To see this, let G_1 be any nonzero $R \times F$ matrix and let G_2 be any nonsingular $F \times F$ matrix. Then for any given θ_t , λ_t and μ_i it is easy to find an observationally equivalent representation with, in general, different unobservables $\tilde{\theta}_t$, $\tilde{\lambda}_t$ and $\tilde{\mu}_i$:

$$y_{it}^{N} = z_{i}' \theta_{t} + \lambda_{t}' \mu_{i} = z_{i}' \left(\theta_{t} - \underbrace{G_{1}}_{R \times F} \lambda_{t} + G_{1} \lambda_{t} \right) + \mu_{i}' \lambda_{t}$$

$$= z_{i}' (\theta_{t} - G_{1} \lambda_{t}) + z_{i}' G_{1} \lambda_{t} + \mu_{i}' \lambda_{t}$$

$$\Rightarrow z_{i}' \tilde{\theta}_{t} + (z_{i}' G_{1} + \mu_{i}') \lambda_{t}$$

$$\Rightarrow z_{i}' \tilde{\theta}_{t} + \tilde{\mu}_{i}' \lambda_{t}$$

$$= z_{i}' \tilde{\theta}_{t} + \left(\tilde{\mu}_{i}' \underbrace{G_{2}}_{F \times F} \right) \left(G_{2}^{-1} \lambda_{t} \right)$$

$$\Rightarrow z_{i}' \tilde{\theta}_{t} + \tilde{\mu}_{i}' \lambda_{t}$$

Here, $\tilde{\mu}_i \coloneqq G_2'(\mu_i + G_1'z_i)$ depends on the predictors. Hence, $\tilde{\mu}_i'\lambda_i$ will, in general, correlate with z_i . In order to uniquely identify θ_i and $\eta_{it} \coloneqq \mu_i'\lambda_i$ in (1) I therefore impose the identifying assumption

A0: Orthogonality condition

For all $t > T_0$ and all i = 1, ..., J we have $E(z_i \eta_{ii}) = 0_R$.

Note that for the purpose of this paper we are not interested in identifying a "true" shock λ_i or a "true" unit-specific shock η_{it} . This would require a structural analysis and specific, probably controversial identifying assumptions. But since we do not aim at an economic interpretation of the shocks, any identification will do. We just need to ensure that the shocks we work with are indeed uniquely identified.

The most convenient identification for θ_i is achieved by assumption A0, provided $E(z_i z_i')$ is non-singular $\forall i$. This can be seen by premultiplying (1) by z_i

$$z_i y_{it}^N = z_i z_i \, '\theta_t + z_i \eta_{it} \, ,$$

taking expectations and solving for θ_t :

$$\theta_t = E\left(z_i z_i'\right)^{-1} E\left(y_{it}^N z_i\right)$$

This implies that θ_t can be consistently estimated by least squares – a fact I will use below.

Note that predictors may contain endogenous variables determined in period T_0 or earlier. Hence, an identifying assumption analogous to A0 is not possible for periods $1, ..., T_0$ since endogenous predictors would typically depend on some of the unobserved shocks.

This is important because in many models outcomes of period t can be written as functions of variables which were determined in the current or a previous period. However, (1) does not provide for any right-hand side observable variable dated $T_0 + 1$ or later. It is useful to think of (1) as representing a dynamic model which has been solved backwards in time until all observable variables werde determined in period T_0 or earlier. Therefore, θ_t must be time-dependent since it depends on the time difference $t - T_0$, while z_i describes initial conditions prior to treatment.

The initial conditions joint with unobservable shocks in subsequent periods eventually give rise to y_{it}^N . Needless to say, the effects of such unobservable shocks accumulate in a complicated way over time. Their cumulative effect is expressed in the λ_i -vector, which is, therefore, very likely autocorrelated and whose variance probably increases over time.

I have defined (1) only for $t > T_0$. Extending (1) to hold for $1 \le t \le T_0$ would be problematic: Since predictors z_i are constant over time, (1) would imply that all predictors are known already in the initial period 1. This would greatly limit the predictive power of z_i for the treatment period if the time span T_0 prior to treatment is substantial.

On the other hand, if (1) is defined only for $t > T_0$, predictors may involve functions of pretreatment outcomes y_{is} , $s \le T_0$. In this case the number of pre-treatment outcomes used for the construction of synthetic controls is held fixed and is not increased when asymptotic arguments are invoked.

The potential outcomes in the case of treatment are denoted y_{it}^{Tr} and modeled as the potential outcome in the case of non-treatment plus a treatment effect α_{it} which is unit and time specific:

$$y_{it}^{Tr} = \alpha_{it} + y_{it}^{N} \tag{2}$$

Since y_{it}^N is, by definition, independent of treatment, it follows directly that α_{it} is uncorrelated with the random variables z_i and λ_t .

Denoting the treatment status of unit *i* in period *t* by d_{it} , where $d_{it} = 1$ in the case of treatment and $d_{it} = 0$ otherwise, observed outcomes are

$$y_{it} = d_{it} y_{it}^{Tr} + (1 - d_{it}) y_{it}^{N}$$
(3)

The primary object of causal analysis is knowledge of α_{1t} for $t > T_0$ or of its average over the treatment period $\overline{\alpha}_1 := T_1^{-1} \sum_{t=T_0+1}^{T} \alpha_{1t}$. More generally, knowledge of any treatment effect α_{it} may be desired. Since either y_{it}^{Tr} or y_{it}^{N} is unobserved for unit *i* in period *t*, the key question is how observations on y_{it} , d_{it} and z_i can be used to estimate the unobserved components of (2) as well as possible.

For the following, let us introduce the following notation: Collect all covariates of the control regions in the $R \times J$ matrix $Z_0 := (z_2 \dots z_{J+1})$ and collect all factor loadings of the control regions in the $F \times J$ matrix $M_0 := (\mu_2 \dots \mu_{J+1})$. Denote by $\Theta^{post} := (\theta_{T_0+1} \dots \theta_T)'$ the $T_1 \times R$ matrix of time-dependent coefficients for the covariates and by $\Lambda^{post} := (\lambda_{T_0+1} \dots \lambda_T)'$ the $T_1 \times F$ random matrix of shocks. Moreover, let $y_i^{post} := (y_{iT_0+1} \dots y_{iT})' \forall i = 1, \dots, J+1$ and collect all observations for the controls in the post-treatment period in the $T_1 \times J$ matrix $Y_0^{post} := (y_2^{post} \dots y_{J+1}^{post})$. Finally, define y_i^{pre} and Y_0^{pre} analogously for the pre-treatment periods $1, \dots, T_0$.

Using (3) we can then rewrite (1) with observable variables on the left hand side. For the J control units, we have

$$Y_0^{post} = \Theta^{post} Z_0 + \Lambda^{post} M_0 \tag{4}$$

while for the treated unit 1 (1), (2) and (3) yield

$$y_1^{post} = \alpha_1 + \Theta^{post} z_1 + \Lambda^{post} \mu_1$$
(5)

where $\alpha_1 := (\alpha_{1T_0+1} \cdots \alpha_{1T})'$ is the parameter of interest.

III. The Standard SC-Approach

The standard synthetic control approach has been popularized by Abadie and Gardeazabal (2003) and Abadie, Diamond and Hainmueller (ADH) (2010). The key idea is that y_{1t}^N , $t > T_0$, the potential output of unit 1 in the counterfactual case of non-treatment, can be approximated by a weighted average of the observed contemporaneous outcomes of the control units. Formally, if $y_1^{N,post} \coloneqq (y_{1T_0+1}^N \cdots y_{1T}^N)'$, the ADH approach aims at finding a suitable nonnegative vector of weights $w^* \in \Delta_J \coloneqq \{w \in \mathbb{R}^J | t_J 'w = 1 \land w_i \ge 0 \quad \forall i = 1, ..., J\}$ such that

$$y_1^{N,post} \approx Y_0^{post} w^* \tag{6}$$

Here, ι_I is a vector $J \times 1$ vector of ones.

To find the desired weights w^* , ADH's approach relies on the predictors z_1, Z_0 and on all pretreatment outcomes y_1^{pre}, Y_0^{pre} , where the predictors may also include functions of some or all of the pre-treatment outcomes. Since not all predictors may be equally informative for potential outputs, let $v \in \Delta_R$ be a vector of non-negative predictor weights and let V := diag(v) be the corresponding diagonal $R \times R$ matrix.

ADH propose to solve the following optimistic bilevel minimization problem:

$$\min_{v \in \Delta_{R}, w \in \Delta_{J}} L_{out}(v, w) \coloneqq \frac{1}{T_{0}} \left(y_{1}^{pre} - Y_{0}^{pre} w \right)' \left(y_{1}^{pre} - Y_{0}^{pre} w \right)$$

$$w \in \Psi(v) \coloneqq \operatorname{argmin}_{w \in \Delta_{J}} L_{in}(v, w) \coloneqq (z_{1} - Z_{0}w)' V(z_{1} - Z_{0}w)$$

$$V = \operatorname{diag}(v)$$

$$(7)$$

This formulation is due to Malo et al. (2020). For a given VI call

$$\min_{w \in \Delta_J} L_{in}(v, w) = (z_1 - Z_0 w)' V(z_1 - Z_0 w)$$
(8)

the inner minimization problem and

s. t.

$$\min_{v \in \Delta_R, w \in \Psi(v)} L_{out}(v, w) = \frac{1}{T_0} (y_1^{pre} - Y_0^{pre} w)' (y_1^{pre} - Y_0^{pre} w)$$
(9)

the outer maximization problem. Note that the outer problem requires w to be from $\Psi(v)$.

Let $\Phi_1^Z := \{w \in \Delta_J | z_1 = Z_0 w\}$ and $\Phi_1^{Y^{pre}} := \{w \in \Delta_J | y_1^{pre} = Y_0^{pre} w\}$ denote the set of weights which solve the inner and the outer minimization problem, respectively, with optimal value zero. ADH assume that the intersection $\Phi_1^Z \cap \Phi_1^{Y^{pre}}$ is nonzero, i. e. there exist weights w^0 such that linear combinations of the columns of Z_0 can exactly reproduce z_1 and the same

linear combinations of columns of Y_0^{pre} can exactly reproduce y_1^{pre} . ADH show that under this (and some additional) assumptions $Y_0^{post}w^0$ is an asymptotically unbiased estimator of $y_1^{N,post}$ when the number of pre-treatment observations T_0 approaches infinity.²

Unfortunately, the ADH approach is problematic in multiple regards: First, if λ_t contains at least one idiosyncratic shock which affects the treated unit, then, asymptotically, $\Phi_1^{\gamma^{pre}} = \emptyset$ with probability 1. This was noticed by Ferman and Pinto (2016). Since this idiosyncratic shock would be independent of the shocks hitting the control units, no linear combination of control units can exactly reproduce the pre-treatment time series of the treated unit for $T_0 >> J$. Hence, in this case, there is no reason to believe that the ADH-estimator is asymptocially unbiased.

Second, *V* is not identified at the optimum, since Φ_1^Z would be non-empty by assumption and, therefore, the inner problem can be solved with optimal value zero for any matrix *V*. Third, suboptimal weights *w* are chosen if all pre-treatment outcomes are included in the matrix of predictors: Then, a solution to the bilevel problem (7) is given by any $w \in \Phi_1^{\gamma^{pre}}$ along with $v = T_0^{-1} (\iota_{T_0} - 0_R)'$, i. e. with a *V*-matrix which has equal nonzero elements for the T_0 pre-treatment outcomes on the main diagonal and is zero everywhere else. In words: The choice of weights is solely driven by the pre-treatment outcomes and all other predictors have no impact at all. This result is due to Kaul et al. (2015). See Lucke (2022) for further problems which arise in model (1) if potential outcomes are autocorrelated.

² Some papers (e. g. Malo et al. (2020)) state that ADH prove the "consistency" of the SC-estimator. This is not true. ADH's proof shows asymptotic unbiasedness. ADH do not claim that the variance of $y_1^{N,post} - Y_0^{post} w^*$ converges to zero.

IV. Consistent estimates for synthetic controls weights

The overarching aim of synthetic control analysis is a good estimate of the counterfactual $y_1^{N,post}$. For this, let us focus on the synthetic control error $\varepsilon_1^{post}(w) := y_1^{N,post} - Y_0^{post}w, w \in \Delta_J$. A standard optimality criterion would be the least-squares criterion, i. e. we may want to minimize the mean squared error, defined as the conditional expectation

$$MSE_{1}^{post} \coloneqq \frac{1}{T_{1}} E\left(\varepsilon_{1}^{post}\left(w\right)'\varepsilon_{1}^{post}\left(w\right)\middle|I_{0}\right)$$

where $I_0 := \{ y_1^{pre}, Y_0^{pre}, Y_0^{post}, z_1, Z_0 \}$ is the relevant information set.

We have

$$\begin{split} \varepsilon_{1}^{post}(w)'\varepsilon_{1}^{post}(w) &= \left(y_{1}^{N,post} - Y_{0}^{post}w\right)'\left(y_{1}^{N,post} - Y_{0}^{post}w\right) \\ &= \left(\Theta^{post}(z_{1} - Z_{0}w) + \Lambda^{post}(\mu_{1} - M_{0}w)\right)'\left(\Theta^{post}(z_{1} - Z_{0}w) + \Lambda^{post}(\mu_{1} - M_{0}w)\right) \\ &= (z_{1} - Z_{0}w)'\Theta^{post}'\Theta^{post}(z_{1} - Z_{0}w) + (\mu_{1} - M_{0}w)'\Lambda^{post}'\Lambda^{post}(\mu_{1} - M_{0}w) \\ &+ 2(z_{1} - Z_{0}w)'\Theta^{post}'\Lambda^{post}(\mu_{1} - M_{0}w) \end{split}$$

i. e. the appropriate problem to solve is

$$\min_{w \in \Delta_{J}} MSE_{1}^{post} = (z_{1} - Z_{0}w)'E(T_{1}^{-1}\Theta^{post}'\Theta^{post}|I_{0})(z_{1} - Z_{0}w) \\
+ (\mu_{1} - M_{0}w)'E(T_{1}^{-1}\Lambda^{post}'\Lambda^{post}|I_{0})(\mu_{1} - M_{0}w) \\
+ 2(z_{1} - Z_{0}w)'E(T_{1}^{-1}\Theta^{post}'\Lambda^{post}|I_{0})(\mu_{1} - M_{0}w)$$
(10)

While (10) is quite different from the bilevel problem (7), it is apparent that under ADH's assumption $\Phi_1^Z \cap \Phi_1^{Y^{pre}} \neq \emptyset$, a solution to (7) is also a solution to (10). But the non-emptyness assumption is not innocuous.

It would, therefore, be desirable to solve (10) directly. This approach has so far been discarded in the discipline because the counterfactual potential outcome $y_1^{N,post}$ is unobserved. But, as shown in Lucke (2022), Θ^{post} , Λ^{post} , μ_1^C and M_0^C can be estimated consistently and this is sufficient to approximate (10) arbitrarily well with increasing *J* or *T* (or both).

To this end I proceed in four steps. First, suppose that J >> R and estimate equation (1) for each period $t > T_0$ as a cross section regression over the *J* control units for which the potential outcome in the case of non-treatment is observed:

$$\begin{pmatrix} y_{2t}^{N} \\ \vdots \\ y_{J+1,t}^{N} \\ \vdots \\ \vdots \\ y_{J+1,t}^{N} \end{pmatrix} = \begin{pmatrix} z_{2}' \\ \vdots \\ z_{J+1}' \\ J \times R \end{pmatrix} \underbrace{\theta_{t}}_{R \times 1} + \eta_{t} = Z_{0}' \theta_{t} + \eta_{t}$$
(11)

where $\eta_{jt} \coloneqq \lambda_t \prime \mu_j \quad \forall j = 2, ..., J + 1$ and $\eta_t \coloneqq (\eta_{2t} \cdots \eta_{J+1t})'$. The error terms η_{jt} are linear combinations of the λ_t 's, some of which are common factors, and, hence, the covariance matrix of η_t will not be diagonal, i. e. we will have non-zero covariances $E(\eta_{kt}\eta_{jt}) \neq 0, \ k \neq j$. By virtue of assumption A0 the estimate $\hat{\theta}_t$ is *J*-consistent for $\theta_t \quad \forall t > T_0$. Obviously, the associated estimate $\hat{\eta}_t$ is consistent for η_t .

As a second step, define the $T_1 \times J$ matrix $H := (\eta_{T_0+1} \cdots \eta_T)^{\prime 3}$. We have $H = \Lambda M_0$, a decomposition which is unique if we impose the conventional restrictions that $M_0 M_0^{\prime}$ be diagonal and $T_1^{-1} \Lambda \Lambda = I_F$, i. e. all shocks are orthogonal to each other. We can partition $M_0^{\prime} = (M_0^{C}, M_0^{\prime})$, where M_0^{\prime} is $J \times J + 1$ and its first column is a column of zeros reflecting the fact that the idiosyncratic shock of the treated unit 1 does not affect any of the control units.

Moreover, we can partition $\Lambda = (\Lambda^C \quad \Lambda^I)$ with the common factors $\Lambda^C := (\lambda_{T_0+1}^C \quad \cdots \quad \lambda_T^C)$ and the idiosyncratic shocks $\Lambda^I := (\lambda_{T_0+1}^I \quad \cdots \quad \lambda_T^I)$ being of dimensions $C \times T_1$ and $J + 1 \times T_1$, respectively. Note that the first column of Λ^I is the idiosyncratic shock of the treated unit 1.

We obtain

$$H = \Lambda M_0 = \left(\Lambda^C \quad \Lambda^I\right) \left(M_0^C \quad M_0^I\right)' = \Lambda^C M_0^C + \Lambda^I M_0^I + =: \Lambda^C M_0^C + \Omega$$
(12)

where Ω is a $T_1 \times J$ matrix of the idiosyncratic shocks with typical element ω_{ti} . Let $\omega_t := (\omega_{t2} \cdots \omega_{tJ+1})'$ be the period-*t* column of Ω' . Clearly, $\omega_t = M_0^T \cdot \lambda_t^T$ and $E(\omega_t \omega_t') = E(M_0^T \cdot \lambda_t^T \lambda_t^T \cdot M_0^T) = M_0^T \cdot E(\lambda_t^T \lambda_t^T \cdot) M_0^T = M_0^T \cdot M_0^T \quad \forall t$ since the *J* idiosyncratic shocks in each λ_t^T vector are, by definition, orthogonal to each other and have unit variance.

Note that the covariance matrix $M_0^I M_0^I$ is diagonal and, therefore, the least squares criterion requires to choose Λ^C and M_0^C such that

$$tr(\Omega'\Omega) = tr((H - \Lambda^{C}M_{0}^{C})'(H - \Lambda^{C}M_{0}^{C}))$$

³ We often suppress the superscript *post* in the following derivations, since all variables are from the post-treatment period.

is minimal. This is achieved by the standard principal components estimator, i. e. when $M_0^C = T_1^{-1} \Lambda^C H$ and Λ^C equals $\sqrt{T_1}$ times the $T_1 \times F$ matrix of those orthonormal eigenvectors of *HH* which correspond to the *C* greatest eigenvalues of *HH*.

The common factors Λ^{C} and their factor loadings M_{0}^{C} can be consistently estimated by the principal components estimator even if T_{1} is fixed and only *J* approaches infinity, cf. Bai (2003).

Suppose that we estimate (11) by OLS for all $t > T_0$. We can collect the estimated coefficients in the $T_1 \times R$ matrix $\hat{\Theta}^{post} := \hat{\Theta} := (\hat{\theta}_{T_0+1} \dots \hat{\theta}_T)'$ and the residuals in the $T_1 \times J$ matrix $\hat{H} := (\hat{\eta}_{T_0+1} \dots \hat{\eta}_T)'$. The number of common factors *C* is not known, but it can be consistently estimated, e. g. by using the information criterion of Bai and Ng (2002). In this procedure both *J* and T_1 are required to go to infinity for the estimate of *C* to be consistent, but no specific relation between *J* and T_1 must hold. In particular, *J* may be much larger than T_1 .

Hence, the principal components estimators provides consistent estimates $\hat{\Lambda}^C$ and \hat{M}_0^C . Let us now, as the third step, estimate μ_1^C , i. e. unit 1's loading coefficients for the common factors. For all $t > T_0$ we have

$$y_{1t}^{Tr} = \alpha_{1t} + z_1' \theta_t + \lambda_t' \mu_1 = \overline{\alpha}_1 + z_1' \theta_t + \lambda_t^C' \mu_1^C + \underbrace{\lambda_t^{I'} \mu_1^I + (\alpha_{1t} - \overline{\alpha}_1)}_{=\omega_{1t}}$$
(13)

Note that α_{1t} is the treatment effect. If z_1 and Z_0 contain a constant (i. e. a one) in their first row, then this functions as a time dummy, since θ_{1t} is different for every period *t* but constant across the units *i*.

For fixed *C*, suppose that T_1 goes to infinity. Since z_1 is known and consistent estimates of θ_t and λ_t^C have been derived, we can run a regression across time with regression coefficients $\overline{\alpha}_1$ and $\mu_1^{C,post}$

$$y_{1}^{Tr,post} - \hat{\Theta}^{post} z_{1} = \overline{\alpha}_{1} u_{T_{1}} + \hat{\Lambda}^{C,post} \mu_{1}^{C,post} + u_{1}^{post}, \qquad (14)$$

where $y_1^{Tr, post} := (y_{1T_0+1} \dots y_{1T})'$ and $u_1^{post} := (u_{1T_0+1} \dots u_{1T})'$ collect the respective post-treatment periods.

If z_1 contains a constant term, then the dependent variable is adjusted for the mean of the potential outcomes in the non-treatment case. Therefore, the estimate of $\overline{\alpha}_1$ is a (first) estimate of the average treatment effect over time for unit 1.

By construction, the error term u_1^{post} does not correlate with the regressors $\hat{\Lambda}^{C, post}$, so a simple OLS-estimate of (14) is T_1 – consistent for μ_1^C . However, the idiosyncratic shock and the

nonconstant component of the treatment effect $\alpha_{1t} - \overline{\alpha}_1$ may be autocorrelated and therefore we may encounter serial correlation in u_1^{post} .

But this can be dealt with in the usual way. Suppose $u_{1t} = \rho_1 u_{1t-1} + \varepsilon_{1t}$ and ε_{1t} is i.i.d., then multiplying the period t-1 equation of (14) by ρ_1 and subtracting the result from the period t equation yields

$$y_{1t}^{Tr} - z_{1} \hat{\theta}_{t} = (1 - \rho_{1})\overline{\alpha}_{1} + \rho_{1} (y_{1t-1}^{Tr} - z_{1} \hat{\theta}_{t-1}) + \lambda_{t}^{C} \mu_{1}^{C} - \lambda_{t-1}^{C} \rho_{1} \mu_{1}^{C} + \varepsilon_{1t}$$
(15)

In the case of a unit root this simplifies to

$$\Delta y_{1t}^{Tr} - z_1 \left(\hat{\theta}_t - \hat{\theta}_{t-1} \right) = \left(\lambda_t^C - \lambda_{t-1}^C \right) \mu_1^C + \varepsilon_{1t}$$

As the fourth and last step we show that it is possible to solve (10) without knowledge of μ_1^I and M_0^I . For this define

$$\varepsilon_1^{C,post}\left(w\right) \coloneqq \left(y_1^{N,post} - \Lambda^{I,post}\mu_1^I\right) - \left(Y_0^{post} - \Lambda^{I,post}M_0^I\right)w, \ w \in \Delta_J$$

where $\Lambda^{I,post}$ and the $J + 1 \times J$ matrix M_0^I are the submatrices of Λ^{post} and M_0 , respectively, which correspond to the idiosyncratic shocks. Hence,

$$\varepsilon_{1}^{post}\left(w\right) = \varepsilon_{1}^{C,post}\left(w\right) + \Lambda^{I,post}\left(\mu_{1}^{I} - M_{0}^{I}w\right)$$

and (10) becomes

$$\begin{split} \min_{w \in \Delta_{J}} & MSE_{1}^{post} = \frac{1}{T_{1}} E\left(\left(\varepsilon_{1}^{C, post}\left(w\right) + \Lambda^{I, post}\left(\mu_{1}^{I} - M_{0}^{I}w\right)\right)'\left(\varepsilon_{1}^{C, post}\left(w\right) + \Lambda^{I, post}\left(\mu_{1}^{I} - M_{0}^{I}w\right)\right)\middle|I_{0}\right) \\ & = \frac{1}{T_{1}} E\left(\varepsilon_{1}^{C, post}\left(w\right)'\varepsilon_{1}^{C, post}\left(w\right) + \left(\mu_{1}^{I} - M_{0}^{I}w\right)'\underline{\Lambda^{I, post}}\underline{\Lambda^{I, post}}\left(\mu_{1}^{I} - M_{0}^{I}w\right)\right|I_{0}\right) \\ & + \frac{2}{T_{1}} E\left(\varepsilon_{1}^{C, post}\left(w\right)'\Lambda^{I, post}\left(\mu_{1}^{I} - M_{0}^{I}w\right)\middle|I_{0}\right) \\ & = \frac{1}{T_{1}} E\left(\varepsilon_{1}^{C, post}\left(w\right)'\varepsilon_{1}^{C, post}\left(w\right) + \mu_{1}^{I'}\mu_{1}^{I'} + T_{1}^{-1}w'\Omega'\Omega w\middle|I_{0}\right) \\ & + \frac{2}{T_{1}} E\left(\varepsilon_{1}^{C, post}\left(w\right)'\Lambda^{I, post}\left(\mu_{1}^{I} - M_{0}^{I}w\right)\middle|I_{0}\right) \end{split}$$

since $\mu_1^I M_0^I = 0$ and $M_0^I M_0^I = T_1^{-1} E(\Omega' \Omega | I_0)$. Note that $\mu_1^I \mu_1^I$ is independent of *w* and can therefore be neglected.

Note further that $\varepsilon_1^{C,post}(w)$ is stochastically independent of the idiosyncratic shocks and hence

$$E\left(\varepsilon_{1}^{C,post}\left(w\right)'\Lambda^{I,post}\left(\mu_{1}^{I}-M_{0}^{I}w\right)\middle|I_{0}\right)=E\left(\varepsilon_{1}^{C,post}\left(w\right)\middle|I_{0}\right)'E\left(\omega_{1}-\Omega w\middle|I_{0}\right)=0$$

where $\omega_1 \coloneqq \Lambda^{I, post} \mu_1^I$.

Setting $\hat{\varepsilon}_{1}^{C,post}(w) := \hat{\Theta}^{post}(z_1 - Z_0 w) + \hat{\Lambda}^{C,post}(\hat{\mu}_{1}^{C} - \hat{M}_{0}^{C} w)$, it follows that solving

$$\min_{w \in \Delta_J} \hat{\varepsilon}_1^{C,post}(w)' \hat{\varepsilon}_1^{C,post}(w) + T_1^{-1} w' \hat{\Omega}' \hat{\Omega} w$$
(16)

is asymptotically equivalent to solving (10). Note that J and T_1 may approach infinity along some arbitrary path and that $\hat{\Omega}$ is the matrix of residuals from (12).

We have to write (16) as a standard constrained quadratic minimization problem. We have

$$\hat{\varepsilon}_{1}^{C,post}\left(w\right) = \left(\hat{\Theta}^{post} z_{1} + \hat{\Lambda}^{C,post} \hat{\mu}_{1}^{C}\right) - \left(\hat{\Theta}^{post} Z_{0} + \hat{\Lambda}^{C,post} \hat{M}_{0}^{C}\right) w$$

so that minimizing $\hat{\varepsilon}_{1}^{C,post}(w)$ ' $\hat{\varepsilon}_{1}^{C,post}(w)$ is equivalent to minimizing

$$\frac{1}{2}w'\left(\hat{\Theta}^{post}Z_{0}+\hat{\Lambda}^{C,post}\hat{M}_{0}^{C}\right)'\left(\hat{\Theta}^{post}Z_{0}+\hat{\Lambda}^{C,post}\hat{M}_{0}^{C}\right)w-w'\left(\hat{\Theta}^{post}Z_{0}+\hat{\Lambda}^{C,post}\hat{M}_{0}^{C}\right)'\left(\hat{\Theta}^{post}Z_{1}+\hat{\Lambda}^{C,post}\hat{\mu}_{1}^{C}\right)+\frac{1}{2T_{1}}w'\hat{\Omega}'\hat{\Omega}w$$

If w^* denotes the solution to (16), the synthetic control for unit 1 is given by $\hat{y}_1^{N,post} := Y_0^{post} w^*$ and the estimated causal effects of treatment are $\hat{\alpha}_1 := y_1^{Tr,post} - \hat{y}_1^{N,post}$.

V. Data

In the following, I use regional data of the Annual Regional Database of the European Commission (ARDECO). The time span is 1980-2018, the level of disaggregation is NUTS3, the lowest level available. For instance, at NUTS3 level the former West Germany (excluding Berlin) is disaggregated into 324 regional units. I exclude all regions from formerly socialist economies, since no data is available prior to 1990 (and often later) and because growth in these regions is strongly driven by transitional catch-up growth not found in the Western part of the EU.

I therefore confine the analysis to regions from Austria, Belgium, Germany, Denmark, Greece, Spain, Finland, France, Ireland, Italy, Luxembourg, Netherlands, Portugal, Sweden and the United Kingdom (UK), i. e. the former EU 15. Of these countries, Denmark, Sweden and the UK kept their national currencies, while all others formed the Eurozone.

Two countries (and the NUTS3 regions therein) deserve special attention: Denmark never adopted the Euro, but it joined the Exchange Rate Mechanism II (ERM II) which involved fixing the exchange rate of the national currency to the Euro. Since Denmark never adjusted

this fixed exchange rate, its economy operated under essentially the same conditions as any other Eurozone country. The second country is Greece which joined the Eurozone only in 2001, i. e. two years later than the other Eurozone countries. But similar to Denmark, Greece was in the ERM II in the years before 2001 and thus had a fixed Euro-exchange rate already since 1999.

I therefore consider regions from Denmark and Greece as "treated", i. e. being subject to a policy intervention which was very similar to full participation in EMU since 1999. For the purpose of constructing a synthetic control I just use regions of those two EU-15 countries which retained and actually experienced exchange rate flexibility during 1999-2018, i. e. Sweden and the United Kingdom. These two countries consist of exactly 200 regions at NUTS3 level⁴. Since some of the British regions underwent a territorial redefinition in the year 2000 which left major traces in their recorded GDP growth rate of this year, I discard the first two treatment years 1999 and 2000 from the analysis of treatment effects. All results below refer to the eighteen post-treatment years 2001-2018.

On a country level, comparing countries like Italy and Greece to Sweden or the UK would be very hazardous. On the regional level, chances that some regions here and there are economically similar are much higher. Some regions are intensive in agriculture or in fisheries both in northern and in southern countries, or depend in a similar way heavily on traditional industries, or have lots of tourism or other services or have similar strengths and weaknesses in infrastructure. If the predominant view is that Sweden and the UK are more competitive than Italy and Greece, say, than this only says that a greater share of regions in the former countries is home to competitive enterprises than in the latter. It does not say that Sweden and the UK do not have some regions which are as traditional or as underdeveloped or as rusty as perhaps many such regions are in Italy and Greece. For the construction of synthetic controls it suffices to have some regions in the control group which are similar to many regions in the treatment group.

The ARDECO data base provides data on nominal GDP per capita on NUTS3 level. I transform this data into consumption units by dividing through the respective national consumer price index and take the log difference, i. e. the growth rate of GDP, as the outcome variable. I use a set of 20 covariates in the analysis below, some of which are directly taken from ARDECO, others are constructed as follows:

From ARDECO, I obtain information on the sectoral distribution of activities in each NUTS3 region. Specifically, I use the 1998 shares of gross value added (GVA) for five sectors, Agriculture, Industry, Construction, Trade, Financial and Business Services. For the same sectors, I also use the 1998 shares of labor costs in sectoral GVA as predictors.

⁴ I delete region UKN15 (Mid and East Antrim) from the donor pool because this region was redefined with sizable increases in territory in 2015. Hence the growth rate of GDP in 2015 is artificially great (22%) which disqualifies UKN15 as a control region in treatment time.

The ARDECO data were compiled by a third party (Cambridge Econometrics) and then transferred to the European Commission. Cambridge Econometrics also compiled sectoral capital stock data at NUTS2 level. This data has not yet been made available through ARDECO, but Cambridge Econometrics kindly supplied it to me. The sectoral disaggregation of capital stocks is the same as for GVA at NUTS3 level, so I broke down the sectoral NUTS2 capital stock data proportional to the GVA shares in order to construct measures of total capital stocks at NUTS3 level. Their 1998 values are also used as predictors.

Data from the EU census 2011 provide information on dwellings, their age and the number of flats per dwelling on NUTS3 and NUTS2 level. Lacking data for 1998, I use the available information on dwellings constructed in 2000 or earlier, reasoning that this is very similar to dwellings in 1998. Dwellings are indicative of infrastructure, as inhabitants need roads, gas, electricity, telecommunication and so on. I use the ratio of dwellings per square kilometer as a proxy for infrastructure and the average age of the dwellings as an indicator of the modernity of infrastructure. Further, I use the ratio of dwellings with three or more flats over all dwellings as an indicator of the degree of urbanization of the NUTS3 region.

Finally, ARDECO also supplies some simple statistics like the population in 1998, the area in square kilometers and dummy variables indicating whether a NUTS3 region is a mountain region or a region bordering on some other country. These variables are also included among the set of predictors.

VI Synthetic Controls for EMU regions

This section desribes the results obtained from estimating synthetic controls for EMU regions 2001-2018. As mentioned above, regions from Denmark and Greece are also considered as treated throughout this time period.

I use two competing methods to estimate the synthetic controls: The standard ADH method with predictor weights which minimize the mean squared prediction error for pre-treatment outcomes, cf. ADH and Abadie (2021). Predictors are the "structural" predictors described above and three statistics of pre-treatment GDPs: Pre-treatment averages of growth rates and levels (in logs) plus the 1998 log GDP. Alternatively, I use the approach outlined in Section IV which constructs the counterfactuals from the common factor model (1) estimated on the control group data in post-treatment time. I denote the results produced by both approaches by ADH and BL, respectively.

Let us first have a look at the average growth effect (across all Eurozone regions) over time. Figure 1 displays for each year from 2001 to 2018 the sum of all the estimated causal effects of Eurozone membership.

< insert Figure 1 about here >

ADH and BL estimates suggest broadly the same conclusion: There were, on average, sizable losses in GDP growth in the years prior to the financial crisis. However, during and immediately after the financial crisis, Eurozone regions may, on average, actually have benefited from the existence of a common currency. This positive effect came to an end and turned deeply into negative territory with the Eurozone's sovereign debt crisis.

Interestingly, much of the first two developments may have been driven by Germany, the EU's largest economy, cf. Figure 2. First, Germany suffered from exceptionally low growth in the late 1990s and early 2000s. Sinn (2003, 2014) argues that the introduction of the common currency has greatly contributed to Germany becoming "the laggard of Europe" during these years:

The EU's southern periphery was poor in capital when the Euro was introduced and exchange rates were permanently fixed. Attracted by a high marginal product of capital and greatly reduced inflationary risks, investors shifted capital from Germany and other northern EU countries to the southern periphery, competing down its interest rates to almost the German level. As a result, German companies lost their competitive edge in terms of access to cheaper credit and also suffered from weaker aggregate demand since capital and investors were moving south. Consequently, German growth was weaker than it had been under the German mark. The causal effects estimated for Germany in the early 2000s are well in line with this reasoning.

< insert Figure 2 about here >

But, second, the tide turned with the financial crisis. Investors worried about financial institutions and debt sustainability in the south, moving back capital to Europe's core economies. Germany, in particular, was seen as a safe haven so that interest spreads vis-à-vis Germany increased greatly in the runup to the sovereign debt crisis 2010-2013. As a consequence, Germany recovered quickly from the financial crisis, whereas the Eurozone's southern countries got deeper and deeper into trouble. European capital market integration propelled by the common currency was in the crisis years quite benefitial for Germany. Until about 2011 the benefitial effects for German regions may actually have dominated the detrimental effects for southern European countries so that the Eurozone average is estimated to have had a positive growth effect from the Euro.

But in the years from 2012 onward, the negative causal effects in southern Europe became so strong that even the aggregate Eurozone effects turn into negative territory (with a slow recovery over the next several years). These negative aggregate effects are clearly not driven by Germany, which, according to the estimates, experienced neither great harm nor great benefit from the common currency in these years.

As this discussion makes clear, focusing on the Eurozone average is of only limited value, since growth in European regions was quite differently affected by the common currency. Still, before turning to more country-specific results, a last visualization for the whole Eurozone may be interesting: Cumulating the estimated causal effects on growth over time,

Figure 3 illustrates the total estimated gain or loss in per capita income at each point in time between 2001 and 2018.

< insert Figure 3 about here >

According to these results both ADH and BL estimates suggest that prior to the financial crisis the Eurozone had, on average, lost about 8% of per capita GDP compared to a counterfactual with national currencies. But ADH and BL results differ somewhat in the sequel: BL results find the benefitial effects of the Euro during the financial crisis somewhat greater than ADH, and the detrimental effects (for the Eurozone average) somewhat worse than ADH. This may be due to the fact that the predictor weights of the BL-method take common shocks in post-treatment time into account, while ADH estimates do not. But despite this difference, both methods come up with the conclusion of a sizable loss in GDP after twenty years of common currency (2018): About 9 percent according to ADH and about 12 percent according to BL⁵.

While Figures 1-3 give an impression of average treatment effects (ATTs) across regions for different points in time, we will now turn to ATTs across time for different regions. Clearly, it is suggestive to look at all regions in a particular country.

An interesting case is Belgium, cf. Figure 4. Here, the first region is Brussels, the next 22 regions are located in Flanders, and regions 24-44 are part of mostly French-speaking Wallonia. It is well known that companies in Wallonia are, on average, less competitive than companies in Flanders, see e. g. OECD (2020) which presents evidence that regional productivity in Wallonia is quite a bit lower than in Flanders and that Flanders was quicker and more successful in transitioning from manufacturing to services. Both the ADH and BL estimates of the causal effects of EMU suggest that, by and large, the less competitive Wallonian regions have suffered more from EMU than the Flemish regions. This is quite plausible since under EMU problems with competitiveness cannot be ameliorated by a depreciation of the national currency.

< insert Figure 4 about here >

Note that synthetic controls are estimated separately for each treated region. No information is supplied on the geographic location of a treated region or on the country to which it belongs or which language is spoken in the region. The computer does not know which region is Flemish or Wallonian, French, Italian or German. Yet the results are very much in line with the view that Wallonian companies would urgently need a devaluation of the currency to improve their competitiveness and that this is less so for Flemish companies. They also suggest that the German economy does not have a competitiveness problem at prevailing exchange rates, but that the French and the Italian economy might actually greatly have benefited from a devaluation.

⁵ Note that these results refer to 2001-2018 and thus implicitly assume zero causal effects in the first two years of currency union.

To see this, let us look at Germany first, cf. Figure 5. The causal effects estimated for German regions fluctuate around zero, most of them within a range of one percentage point loss or gain in annual growth due to EMU. For some regions sizably greater effects are estimated.

Interestingly, many of the most positive causal effects are associated with the German car industry. According to both methods, the highest positive effect is found for Ingolstadt (Audi), with Wolfsburg (VW), Dingolfing (BMW) and Germersheim (Daimler) among the top ten. Additionally, ADH finds Neckarsulm (Audi) on rank 4. As German car manufacturers are strongly export-oriented, these results may be due to an undervalued Euro (compared to a counterfactual German mark) and, hence, favorable export conditions for German exporters in general. In fact, German net exports as a share of GDP have risen to record levels of between six and eight percent since the introduction of the Euro (up from about two percent prior to 1999).

< insert Figure 5 about here >

A completely different picture emerges for French regions, cf. Figure 6. Both methods broadly agree in the sizes and signs of the estimated causal effects – and indicate that EMU has not done much good for France. Most of the estimated effects are clearly negative, fluctuating around a loss of one percentage point of annual growth. Paris is the far left region and the next seven regions all belong to the Département Île-de-France, i. e. the Greater Paris region. This rather modern area achieves slightly better results than the rest of France (with the exceptions of two overseas territories (Guadeloupe and Réunion) at the far right end of the graph). But for basically all the rest of France (some 90 regions), both methods find that over the years EMU has caused a substantial loss of per-capita income, between 15% (ADH) and 20% (BL).

< insert Figure 6 about here >

Things are even worse for Italy and Greece, cf. Figures 7 and 8, respectively. The causal effects of EMU are estimated as negative throughout and by both methods. In the case of Italy, BL estimates are substantially lower than ADH estimates, but the correlation between BL and ADH is clearly very strong. For Greece, on the other hand, BL and ADH estimates are not only highly correlated, but also have practically the same mean. Both methods suggest that, over a time span of 18 years, Greek regions have lost about two percentage points of annual growth each year due to EMU. For Italy, BL estimates suggest a similar loss on average, whereas ADH finds that Italy suffered not quite as badly (losses in growth due to EMU fluctuating around -1.5% annually.)⁶

VII Conclusions

After more than 20 years of European Monetary Union (EMU), surprisingly little empirical research has been devoted to the important question if the adoption of a common currency in

⁶ Results for other EMU countries are available upon request.

many EU member states has, on balance, benefited the participating economies. While a number of studies have found evidence for increases in trade and FDI, it is far less clear if and to what extent the loss of exchange rate flexibility has impeded governments from responding optimally to negative aggregate shocks and has disadvantaged companies which had trouble to withstand the competitive pressure of the common market.

This paper is the first attempt to analyze how EMU has impacted on regional economic growth. Using regional data facilitates comparisons between regions in and outside the Eurozone, since it is easier to construct comparable units and thereby isolate the effect of the common currency. The introduction of the Euro can be thought of as a natural experiment where treatment is assigned as good as randomly. For not the structural characteristics of a certain region determined if the region joined the Eurozone but rather the political will of the country's government (in the case of the UK) or of the people opposing the will of the government (in the case of Sweden).

Therefore, synthetic control methods are a suggestive methodology to quantify the growth effects of EMU. I have used the standard ADH (2010) approach to the construction of synthetic controls along with a novel method which estimates the counterfactuals from the large sample of control regions in post-treatment time. While this is a completely different approach, results do not differ by much. Essentially, both methods lead to the conclusion that most (or even all) regions in France, Italy and Greece would have had much stronger growth had their country not joined the Eurozone. Regions with competitive industries, e. g. German car manufacturers, however, may have benefited greatly from an undervaluation of the common currency. Similar conclusions emerge for Belgium regions.

Hence, there is no uniformly positive or negative effect of EMU on regional growth, neither across regions nor over time. But there are many regions in the Eurozone which seemingly were disfavored by the common currency and these regions are seen clustering in certain countries like France, Italy, Greece or the French speaking part of Belgium. Also, increased capital mobility due to EMU has likely had heterogeneous effects across the Eurozone. In particular, in times of exogenous crises like the financial crisis, it seems that EMU cushioned a fairly competitive country like Germany even better than a national currency would have done, whereas regions in poorer countries adversely affected an would have fared better had their country still had exchange rate flexibility to counter a negative shock.

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