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Daylight Saving Time and incidence of myocardial infarction: Evidence from a regression discontinuity design

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HIGHLIGHTS

- We estimate the effect of Daylight Saving Time (DST) on myocardial infarction (AMI).
- We use daily mortality data provided by the Brazilian Ministry of Health.
- We use a regression discontinuity design to estimate the effect of interest.
- Results show that this abrupt disturbance increases incidence of AMI by 7.4%–8.5%.

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ABSTRACT

Limited evidence suggests that Daylight Saving Time (DST) shifts have a substantial influence on the risk of acute myocardial infarction (AMI). Previous literature, however, lack proper identification necessary to vouch for causal interpretation. We exploit Daylight Saving Time shift using non-parametric regression discontinuity techniques to provide indisputable evidence that this abrupt disturbance does affect incidence of AMI.

1. Introduction

Empirical researchers in epidemiology and related disciplines have for long been interested in estimating the causal effect of circadian variations in the incidence of acute myocardial infarction (hereafter, AMI).[1] Most literature on the subject, however, lack proper identification strategies needed to vouch for causal interpretation. Since randomization is unfeasible, generally owing to ethical constraints, the natural experiment induced by Daylight Saving Time (hereafter, DST) serves as an alternative potentially as good as randomization to identify the effect of interest (Imbens and Lemieux, 2008; Angrist and Pischke, 2014).

This sudden disturbance caused by DST to individuals' daily routine has recently been shown to affect outcomes such as fatal vehicle crashes (Smith, 2014), criminal activity (Doleac and Sanders, forthcoming) and individual well-being (Kountouris and Remoundou, 2014) using robust econometric techniques. In the medical literature, Janszky and Ljung (2008) provide one of the first recognized piece of evidence relating DST and the incidence of AMI. Since then, other papers using specific-hospital admissions have flourished. Their analyses are usually based on incidence ratios calculated by dividing the incidence just after transition by the incidence two weeks before transition, therefore considering small (and selected) samples, ad-hoc definitions for bandwidths around the discontinuity and strategies that strongly depend on almost ideal unconfoundedness conditions. Our aim in this paper

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[1] According to experimental evidence, circadian variations caused by disturbance in sleep patterns may increase high-sensitivity C-reactive protein (CRP) levels, a stable marker of inflammation, which has been shown to be predictive of cardiovascular morbidity (Meier-Ewert et al., 2004).

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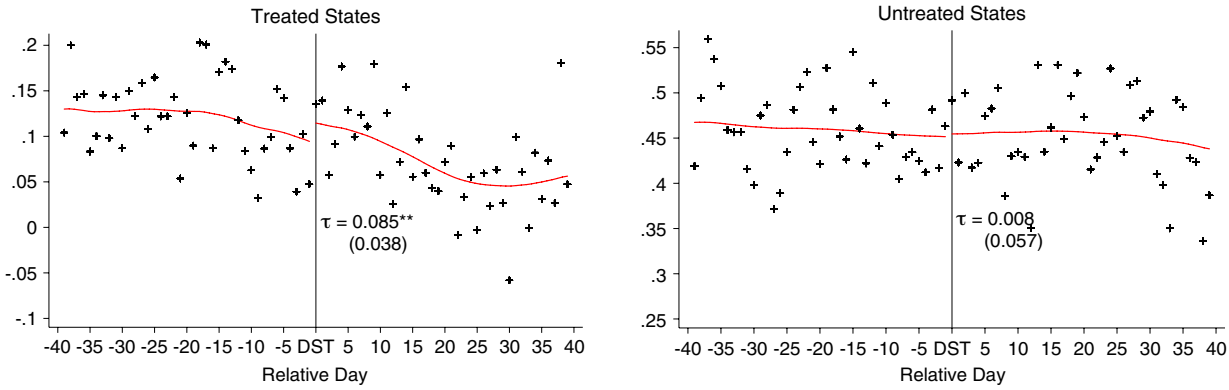

Note: Residuals are generated from a regression of ln(infarction) on day-of-week, State and year dummies. Fitted lines represent locally weighted regression.

Fig. 1. DST entrance transition—residuals plot.

is therefore to provide a well-founded and accurate estimate of the effect of DST on AMI using Brazilian data and a regression discontinuity design.

In Brazil, DST is governed annually by means of Federal enactments based on information of technical reports provided by The Electric System National Operator (ONS). The National Operator indicates the States that should adopt DST as well as the duration of the regime, which usually starts on the third Sunday of each October, when clocks skip forward from 12 am to 1 am, and extends to midnight of the third Sunday of each February. This policy favors our identification since it provides variation in DST adoption within and across States. Having non-adopter States helps us in designing a robust placebo test, given other factors affecting AMI, besides DST, must evolve smoothly around the transition date.[2]

The remainder of the article is organized as follows. In Section 2 we present the data set and methodological approaches. Section 3 discusses the results. Finally, conclusions are presented in Section 4.

2. Data and empirical strategy

In this section we present the data and the empirical strategy we adopt to identify the causal effect of DST on AMI. In particular, we compare the incidence of AMI on the day before entering DST with the incidence of AMI on the first day after its initiation for States that adopted DST using a regression discontinuity (RD) design. For that, consider the following reduced-form model

$$\ln AMI_{isy} = \tau I(Transition_{isy} \geq 0) + g(Transition_{isy}) + \varepsilon_{isy} \qquad (1)$$

where $\ln AMI_{isy}$ is the logarithm of AMI in day i, state s and year y, $Transition_{isy}$ is defined as the number of days to transition to DST, which is equal to zero on the first day after transition and is positive (negative) after (before) then, g is a non-parametric function and ε is a random term. To eliminate persistent day-of-week effects (it might be the case that AMI incidence is higher on weekends than weekdays, for example), State differences and long-term time trends, we follow Smith (2014) and demean the log of AMI incidence by day-of-week, State and year.

We utilize local-polynomial regression-discontinuity point estimators with robust bias-corrected non-parametric confidence intervals (Calonico et al., 2014). Instead of ad-hoc bandwidths adopted by previous literature, we rely on two optimal data-driven bandwidth selectors outlined in Imbens and Kalyanaraman (2012, hereafter, IK) and Calonico et al. (2014, hereafter, CCT) and on an alternative cross-validation method, as done by Ludwig and Miller (2007, hereafter, CV). We also propose falsification tests on the basis of (a) a well defined control group, namely untreated States, and (b) placebo diseases that in principle should not be affected by the transition.

We use individual-level mortality data from the Mortality Information System (SIM), compiled by the Brazilian Ministry of Health. The system provides almost global coverage within the Brazilian territory, containing daily information on cause of death following International Classification of Diseases (ICD) codes. To ensure the best reliability of our data, we consider the years from 2007 to 2012. During this period, all states within Midwestern, Southern and Southeastern region, where light incidence vary the most during the year, adopted DST. Bahia (Northeastern region) and Tocantins (Northern region) adopted DST only in 2011 and 2012, respectively.[3]

3. Results

Fig. 1 presents our main results graphically. Demeaned values of log(AMI) by day-of-week, State and year are plotted, centered on the DST transition date. The graph on the left, which considers States that adopted DST, shows that points to the right of the cutoff are slightly shifted above, implying higher incidence of AMI after transition. This is not observed when looking at untreated States.[4] Table 1 presents formal results considering all bandwidth selection procedures for treated and untreated States. After transition, AMI incidence increased by about 7.4%–8.5% for treated States.[5] For untreated States, results are precisely zero.

A common practice in causal inference literature suggests support for the identifying assumption can be offered by estimation of the causal effect of a treatment that, under the hypothesis of identification, is supposed not to have any effect (Imbens, 2004).

[2] Doleac and Sanders (forthcoming) and Smith (2014), for example, consider law changes to DST policy in the US to account for endogeneity, since DST occurs simultaneously across 48 states (Arizona and Hawaii do not observe DST) and at approximately the same time each year.

[3] Results are unchanged if we exclude these two States.

[4] For untreated States, we consider as if DST was adopted in the same time period as treated States. Hence, in this falsification test, we estimate equation (1) using data on $\ln AMI_{isy}$ for untreated States.

[5] These numbers are slightly higher than the ones previously obtained in this literature. For example, Janszky and Ljung (2008) find that incidence of myocardial infraction increased by 5.1% using data from the Swedish registry of acute myocardial infarction.